Thomas Robert
Sara Bonasia
Michel W. Bojanowski
Editors

Anatomy of Cranial Arteries, Embryology and Variants

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Preface

When the idea of writing a book on the anatomy and embryology of cerebral arteries came up, we immediately found it exciting. The great interest we already had in anatomy and embryology, together with the desire to understand as much as possible the variations offered by human vascular anatomy, were the spark to embark on this path. The hard work, which lasted about 3 years and led to the completion of this book, transformed this interest into a burning passion.

The development of this project was then only possible through patience, dedication, and a love for details.

Each of us, with our own personal vision, contributed by tackling the obstacles that arose and provided our own point of view, making each chapter unique. This book encompasses in fact the three different visions of a resident, a specialist, and an established neurosurgeon, which is why it aims not only to be the starting point for knowledge of vascular anatomy, but also to be a refuge in which to find the most complex anatomical variants after having encountered them in a patient.

The strategic organization of the chapters reflects the same ambition: it makes possible to quickly find the desired arteries and their variants based on the type of vascularization they provide, while at the same time providing meticulous and detailed information. For this reason, the chapters are organized into five different sections, which progressively introduce general vascular embryology and unfold by addressing intradural, dural, skull base, and cranio-cervical junction vascularization.

We have also tried to make the complex concepts belonging to embryology more horny and visualizable through a wide range of illustrations. Although many concepts remain only hypothetical, the illustration of the developmental stages of each artery can help to understand and memorize these steps.

We hope that this book will satisfy your questions and stimulate your curiosity to discover the complexity of human beings.

Paris, France — Sara Bonasia
Lugano, Switzerland — Thomas Robert

Acknowledgments

This book is the result of the hard work of the editors, which would not have been possible without a solid education and a strong passion for neuroscience. For this reason, our main thanks go to all those who contributed to our training and who were able to transmit to us their passion for this topic, their desire to know, and their ability to work hard.

The completion of this work would not have been possible without the indispensable work of all the co-authors, who we would like to thank infinitely for their contribution and trust.

Finally, we would like to express our gratitude to all the members of Springer Group, especially to Ms. Christina Shirly, Ms. Sylvana Freyberg, and Ms. Katherine Anisha for their fundamental support.

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Abbreviations

AA	Arterial arches
ACA	Anterior cerebral artery
AccMA	Accessory meningeal artery
AccMCA	Accessory middle cerebral artery
AChoA	Anterior choroidal artery
AComA	Anterior communicating artery
AICA	Anterior inferior cerebellar artery
AOA	Aortic arch
APhA	Ascendent pharyngeal artery
APS	Anterior perforated substance
ARSA	Aberrant right subclavian artery
ASA	Anterior spinal artery
AVM	Arteriovenous malformations
BA	Basilar artery
BCT	Brachiocephalic trunk
CCA	Common carotid artery
CMA	Callosomarginal artery
cpA	Clinoid process artery
CTA	Carotico-tympanic artery
DA	Dorsal aorta
dAVF	Dural arterio-venous fistula
DiA	Diencephalic artery
DMCA	Duplicated middle cerebral artery
DSA	Digital subtraction angiography
ECA	External carotid artery
FPA	Frontopolar artery
HA	Hypoglossal artery
HyA	Hyoid artery
ICA	Internal carotid artery
ILT	Infero-lateral trunk
IMA	Internal maxillary artery
LingA	Lingual artery
LNAs	Longitudinal neural arteries
LPChoA	Lateral posterior choroidal artery
MA	Mandibular artery
MACC	Median artery of the corpus callosum
MCA	Middle cerebral artery
MesA	Mesencephalic artery
MetA	Metencephalic artery
MHT	Meningo-hypophyseal trunk
MMA	Middle meningeal artery
MPChoA	Medial posterior choroidal artery

MRI	Magnetic resonance imaging
OA	Ophthalmic artery
OccA	Occipital artery
OFA	Orbitofrontal artery
OpV	Optic vesicle
OtA	Otic artery
PA	Proatlantal artery and pericallosal artery
PAA	Posterior auricular artery
PCA	Posterior cerebral artery
PChoA	Posterior choroidal artery
PComA	Posterior communicating artery
PDOA	Primitive dorsal ophthalmic artery
PICA	Posterior inferior cerebellar artery
PMA	Primitive maxillary artery
POA	Primitive olfactory artery
PPOA	Persistent primitive olfactory artery
PVOA	Primitive ventral ophthalmic artery
RAH	Recurrent artery of Heubner
SA	Stapedial artery
SAH	Subarachnoid hemorrhage
SBA	Subclavian artery
SCA	Superior cerebellar artery
SHA	Superior hypophyseal artery
SOF	Superior orbital fissure
STA	Superficial temporal artery
TA	Trigeminal artery
TCT	Thyrocervical trunk
TGA	Thalamogeniculate arteries
ThyrA	Thyroid artery
TPA	Thalamoperforating arteries
VA	Vertebral artery
VGAM	Vein of Galen aneurysmal malformation
ViA	Vidian artery
VPhA	Ventral pharyngeal artery

Part I

General Concepts

A Brief Historical Review of Neurovascular Embryology

Lorenzo Bertulli and Thomas Robert

To introduce our long and beautiful trip into the fascinating vasculature of the human brain, we will briefly start reviewing the crucial steps that lead to a better and thorough comprehension of the complexity of its anatomy and development.

The first embryologic study, which deals with the formation and development of the human embryo and fetus, finds its roots back in ancient Egypt around 1400 BC [1].

Embryology as a science first appears though in writings by Greek philosophers such as Empedocles, Anaxagoras, and Diogenes. They were interested in the process of reproduction, development, differentiation, and regeneration. According to their beliefs, new organisms can emerge through sexual reproduction, asexual reproduction, or spontaneous generation. A widely held belief was that the fetus developed by a fire inside the embryo, which ordered the parts [1].

A great historian of embryology, Joseph Needham, referred to Hippocrates (c460 BC–c370 BC) as the first embryologist. Hippocrates believed in preformationism, the theory that all organisms are fully formed in miniaturized form within the womb before birth. According to him, the embryo received its blood supply from the placenta and developed by absorbing moisture and breath from the mother [1].

The next major development in embryology occurred under Aristotle (384 BC–322 BC). "On the Generation of Animals" was his main book of embryology, but the majority of his well-known observations about embryology can be found in the four compendiums: "The History of Animals," "The Parts of Animals," "The Respiration," and "The Motion of Animals." It is evident from his writings that he studied embryos of multiple organisms by opening bird eggs at various stages of development and dissecting mammalian embryos as well. He may also have observed human embryos, an almost impossible task in antiquity. According to Aristotle, the semen supplied a substance to the embryo that gave it form, and then the mothers provided a substance that contributed to its development [1].

In the early Middle Ages, Albertus Magnus of Cologne (c1200 AD–1280 AD) gave a great contribution to embryology. In his interpretation of the works of Aristotle and the Arabic commentaries that accompanied them, Albertus replaced speculative and theological ideas with observational techniques and attention to detail, basing his theories upon extensive studies on chick and fish embryos [1].

In the following centuries, great progress in the understanding of embryology was made thanks to the enormous work of scientists of the caliber of Leonardo da Vinci (April 15th, 1452–May 2nd, 1519) (Fig. 1), Hieronymus Fabricius (May 20th, 1537–May 21st, 1619), William Harvey (April 1st, 1578–June 3rd, 1657) (Fig. 2), and Marcello Malpighi (March 10th, 1628–November 29th, 1694) (Fig. 3), with their colossal dissection studies and illustrations on several embryos, including human ones [1].

During the next 150 years, the discourse and debate between preformationists and epigeneticists continued throughout the age of enlightenment, best exemplified by the debate between Albrecht von Haller (October 16th, 1708–December 12th, 1777), a Swiss anatomist, physiologist, and naturalist, and Caspar Friedrich Wolff (January 18th, 1733–February 22nd, 1794), a German physiologist considered to be the father of modern embryology [1].

As early as the nineteenth century, human embryology went forward with the study of human embryo samples derived from maternal deaths, abortions, or surgeries. Because animal experimental biology developed in the twentieth century cannot and should not be applied to human embryology on ethical grounds, nothing has changed in the twenty-first century. Human embryology has advanced very

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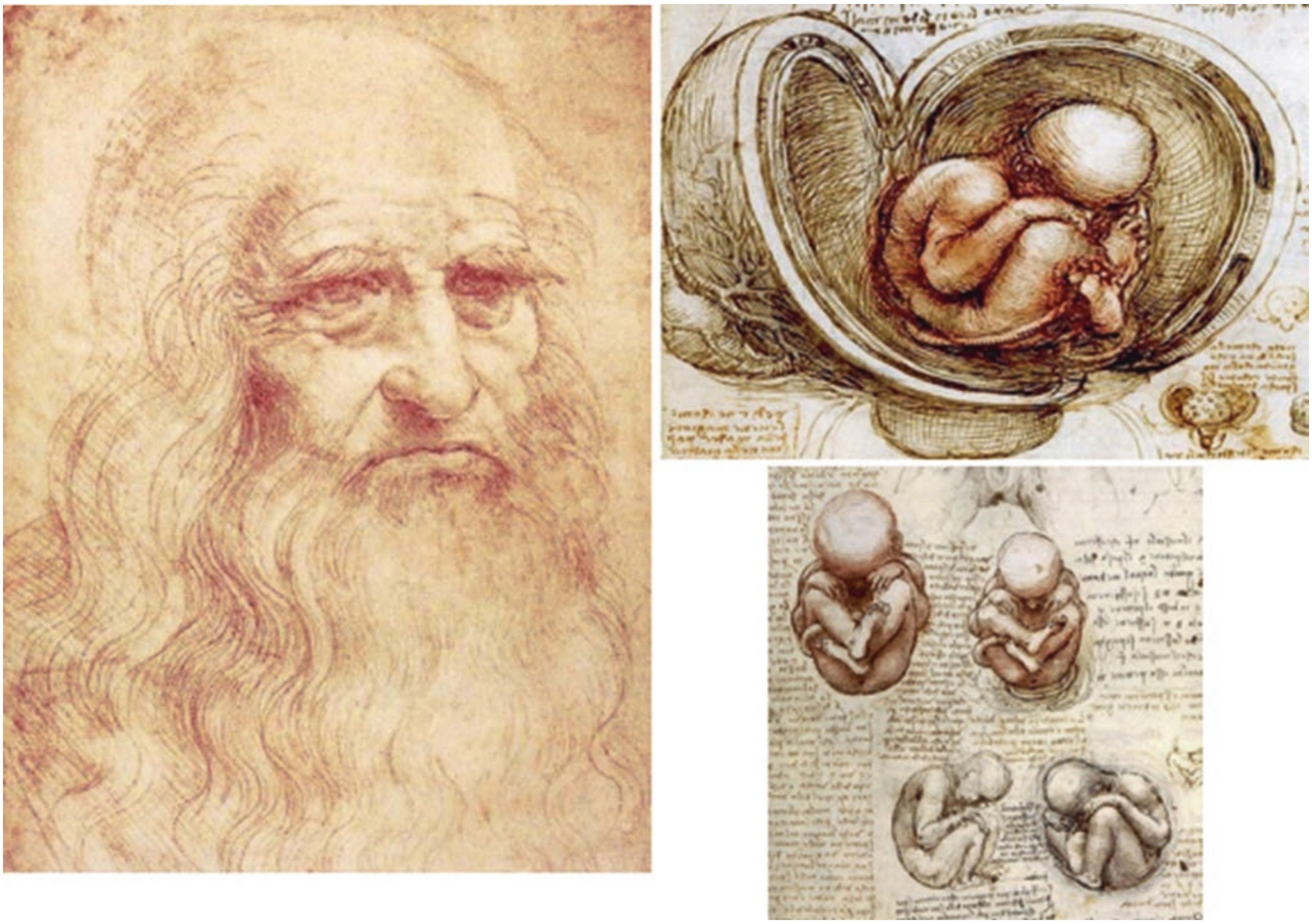

Fig. 1 Self-portrait of Leonardo Da Vinci (left), drawings of the *Studies of the Foetus in the Womb*, approx. 1511 (right). Adapted from [1]

little during the last 100 years, with only a few molecular studies on limited numbers of human tissue being conducted [2]. On the other hand, recent studies using destructive and non-destructive imaging techniques on existing collections have enabled many morphological measurements to be made on these embryos [2, 3].

Human embryology has greatly benefited from the establishment and study of key embryological collections during its history. The biggest and most known are the Carnegie Collection, the Kyoto Collection, the Blechschmidt Collection, and the Madrid Collection [2].

Of these, surely the biggest and probably the most well-known is the Carnegie Collection. Began by Franklin P. Mall (1862–1917) and Franz Keibel (1861–1929) in the early 1900s, the embryo's collection grew at a rate of about 400 specimens a year, donated by clinicians and researchers, and the number of samples reached over 8000 by the early 1940s. The research of Mall and his co-workers was documented in Contributions to Embryology of the Carnegie Institution of Washington between 1915 and 1966. They are still considered foundational in human embryology even today [2].

Although the first systematic descriptions of the neurovascular embryology have been reported in the early 1920s, the first descriptions of blood vessel formation and CNS invasion date back to the mid-1940s. Several investigators have examined fetal brains injected with India ink in order to observe patent vessels to describe the vascularization of the central nervous system [4, 5]. They discovered that peri-neural vascular plexus (PNVP) vessels invaded the brain at specific times, and that vessels form stereotypical patterns once they enter the brain. Although it was thought that patterns would ultimately reflect functional domains, how early blood vessel patterns within a developing brain developed was unknown [6]. Feeney and Watterson first described the formation of the PNVP around the developing spinal cord in chick embryos in 1946, as well as the stereotypical vessel ingression patterns as the embryos developed [4].

Fig. 2 Portrait of William Harvey. Wellcome Collection

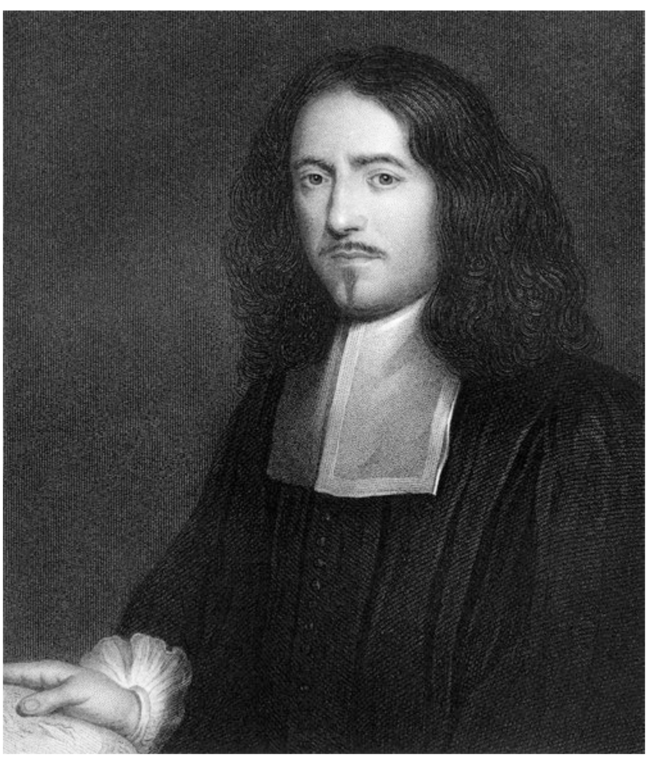

Fig. 3 Portrait of Marcello Malpighi. Now in the Royal Society

Historically, five great authors are known for their thorough studies concerning cerebral arteries development, and three of them based their work on the embryos collection of the Carnegie University.

George L. Streeter (1873–1948), who replaced F.Mall as director of the Department of Embryology at the Carnegie Institution of Washington after his premature death in 1917, was the first to define the 23 Carnegie Stages (currently used to classify the developmental embryological stages) [2]. In 1921, he described the principles leading to cerebral vascular system formation, dividing it into 5 phases: (1) angioblastic stage, (2) arterial, venous, and capillary development, (3) penetration of perforators, (4) construction of more large vessels from a "web," and (5) histological differentiation [7].

E.D. Congdon in 1922 defined for the first time the complete embryology of the aortic arches [8].

Dorcas Hager Padget (1906–1973) (Fig. 4) continued the descriptive work of Streeter working on a series of embryos at the Carnegie University, publishing in 1948 the most thorough study about the embryology of the whole cerebral arterial system starting from the appearance of the internal carotid artery to the final fetal configuration [9, 10]. Having completed only three years of college early, she moved on to train as a medical artist at Johns Hopkins. Her experience working for Walter Dandy honed her artistic skills, earning her fame as a neurosurgical illustrator. At the beginning of her career as a medical researcher, D. Padget's scientific curiosity and Dandy's confidence led to her first foray into the field. Her studies of human embryo sections led to the first description of the embryological development of intracranial vessels, with separate treatises detailing the development of arteries and veins. D. Padget's publications on neuroembryology are unrivaled in the medical literature because of her scientific approach, meticulous attention to detail, and exceptional artistic ability [9].

Franz Altmann, an Austrian ENT emigrated to the USA in the late 1930s, gave his contribution to the field with several works on arterial variants and comparative anatomy [11].

Finally, in the more modern era, Pierre Lasjaunias (1948–2008) (Fig. 5) from France is one of the most influential and important names in the field of neurovascular anatomy of the past century, with his immense work published as articles and books on cerebrovascular anatomy, embryology, and comparative anatomy [12–14]. He started from the rigorous study of his large number of angiographies, comparing them to draw up several embryological hypotheses to explain physiological development, anatomical variants, and pathological entities [15, 16].

The modern technologies nowadays integrate molecular analyses and complex imaging modalities to try to help us better understand the complex and beautiful processes that

Fig. 4 Dorcas H. Paget. Left, portrait by Audrey Juliet Arnott, student of Brödel; right, picture with (from left to right) Mrs. Sadie Dandy, Dr. Walter E. Dandy, Mrs. Dorcas Hager Padget, Mrs. Frances C. Woodhall, and Dr. Barnes Woodhall. Brödel Archives, adapted from [9]

Fig. 5 Pierre Lasjaunias, from [16]

lead to the formation of the incredibly fascinating vascular structure of the central nervous system, but there's still a lot of work to do to have a complete comprehension of it.

References

1. Amjad B. A brief history of embryology: historical vignettes in embryology. Clinical embryology: an atlas of congenital malformations. Cham: Springer; 2019. p. 1–9.
2. Yamada S, Hill M, Takakuwa T. Human embryology. New discoveries in embryology. 2015: 97–124.
3. de Bakker BS, de Jong KH, Hagoort J, de Bree K, Besselink CT, de Kanter FE, et al. An interactive three-dimensional digital atlas and quantitative database of human development. Science. 2016;354(6315):aag0053. https://doi.org/10.1126/science.aag0053.
4. Feeney JF Jr, Watterson RL. The development of the vascular pattern within the walls of the central nervous system of the chick embryo. J Morphol. 1946;78:231–303. https://doi.org/10.1002/jmor.1050780205.
5. Strong LH. The early embryonic pattern of internal vascularization of the mammalian cerebral cortex. J Comp Neurol. 1964;123:121–38. https://doi.org/10.1002/cne.901230111.
6. Bautch VL, James JM. Neurovascular development: the beginning of a beautiful friendship. Cell Adhes Migr. 2009;3(2):199–204. https://doi.org/10.4161/cam.3.2.8397.
7. Streeter GL. The developmental alterations in the vascular system of the brain of the human embryo. Contrib Embryol Carnegie Inst Wash. 1921;8:7–38.
8. Congdon ED. Transformation of the branchial-arch system during the development of the human embryo. Contrib Embryol. 1922;68:47–110.

9. Kretzer RM, Crosby RW, Rini DA, Tamargo RJ. Dorcas Hager Padget: neuroembryologist and neurosurgical illustrator trained at Johns Hopkins. J Neurosurg. 2004;100(4):719–30. https://doi.org/10.3171/jns.2004.100.4.0719.
10. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–62.
11. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
12. Lasjaunias PL, Berenstein A, Brugge KG. Surgical neuroangiography Vol. 1: Clinical vascular anatomy and variations, vol. 1. 2nd ed. Berlin/New York: Springer; 2001.
13. Lasjaunias PL, Berenstein A, Brugge KG. Surgical neuroangiography Vol. 2: Clinical and endovascular treatment aspects in adults, vol. 2. Berlin/New York: Springer-Verlag; 2004.
14. Berenstein A, Brugge KG. Surgical neuroangiography Vol. 3: Clinical and interventional aspects in children, vol. 3. Berlin/New York: Springer-Verlag; 2006.
15. Leonardi M, Picard L, Rodesch G. Pierre lasjaunias. Interv Neuroradiol. 2008;14(Suppl 1):81–92. https://doi.org/10.1177/15910199080140S115.
16. Picard L. Pierre Lasjaunias: his messages. J Radiol. 2008;89(7–8 Pt 1):839–41. https://doi.org/10.1016/s0221-0363(08)73871-x.

Embryological Development of the Human Cranio-Facial Arteries

Lorenzo Bertulli and Thomas Robert

The embryological development of the cerebral vasculature is very complex. The knowledge and understanding of such embryological development are important for physicians interested in neurovascular pathologies. Indeed, all vascular variants and almost all vascular pathologies, such as aneurysms, dolichoectasia, atherosclerosis, and neurovascular conflicts could be explained by an alteration during the embryological life. Understanding the process which leads to the development of the normal cerebral arterial system in humans is therefore very important to have a better knowledge of the possible clinical and surgical implications of these anomalies.

In this chapter, we will review the embryological development of the cranio-facial arterial vasculature from its beginning at approximately day 21 to day 50 of intrauterine life, with pictures illustrating each developmental phase.

After some definitions and a historical overview, we will describe the general principles of the vascular embryology of the whole cranio-facial arterial system in human. Each paragraph describes one of the seven stages of Padget, to make the reading easier [1, 2]. For each stage, general developmental changes, evolution of the anterior circulation and, in the end, the evolution of the posterior and extracranial circulation will be successively addressed. Each stage is summarized by a picture with all the main vessels showing their relationship with each other and with the developing neural and somatic structures.

Terminology

- **Days:** The embryo implants in the uterus to start its intrauterine life around 1 week after fertilization. Normally, the embryonic life is divided into weeks: the gestational age (or clinical age) is calculated from the last menstrual period; the post-conception age, on the other hand, refers to days after fertilization. In this chapter, we will refer to "days" as days after fertilization.
- **Stages:** With the word "stages" we will only refer to the 7 stages of Padget [1, 2]. They do not correspond to the "Carnegie stages", which is a standardized system of 23 stages used to provide a unified developmental chronology of the vertebrate embryo [3–7]. For the sake of completeness, we specified the corresponding Carnegie stage(s) to the Padget stage in each paragraph.
- **Millimeters:** The size of the embryos during the various phases is expressed in millimeters of length from the rostral to the caudal point.

In her work, Padget refers to the two main primitive components of the ophthalmic artery as "ventral" and "dorsal", where ventral is anterior and dorsal posterior [1]. However, if we analyze the true disposition of the vessels, that might be confusing. In fact, the so-called "primitive dorsal ophthalmic artery" comes off more proximally from the primitive internal carotid artery (and, consequently, in a ventral position with respect to the developing head); the so-called "primitive ventral ophthalmic artery" comes off more distally (and so in a dorsal position). In this chapter we will maintain the original terminology from Padget, specifying the correct position of the arterial branches when necessary.

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Development of the Cranio-Facial Arterial System

The cerebral vasculature begins to develop quite early in the embryo, during the third week of intrauterine life (21 days ≃2 mm), slightly before the heart starts beating [8, 9]. At this stage, the neural tube is not closed yet. The prosencephalon is irrigated initially by the two dorsal aortas (DA), whose rostral part will become the internal carotid arteries (ICA); the mesencephalon and rhombencephalon vasculature develop after, as the vertebro-basilar system appears later [1, 10, 11].

The vascular development consists of 2 main stages: vasculogenesis and angiogenesis [8, 12]. Vasculogenesis is the process by which hemangioblasts are differentiated into angioblasts and the formation of a primitive vascular network; angiogenesis refers to the formation of new capillary vessels from pre-existing blood vessels [8, 9, 13]. The main mechanism of angiogenesis is sprouting, which is mainly driven by hypoxia/ischemia mechanism and related growth factors from the target tissue [8, 9, 14, 15]. As more capillaries are formed, the impedance to flow is reduced in larger arteries; this way, flow-induced remodeling of the vessels supplying a specific parenchymal area is promoted [8, 16].

The development of the circulatory system supplying blood to the brain begins with the formation of the 6 pairs of primitive branchial arch arteries, subsequently undergoing heavy modifications during development [8, 10].

The Arterial Arches and Proximal Arterial Branches Development

The human embryo has six pairs of visceral arterial arches (AA) [1, 8, 10, 17–20]. Each AA arises ventrally from the bulbus arteriosus (aortic sac) and courses in the corresponding visceral branch, to end dorsally in the DA [1, 8, 10, 17, 20]. They are present until about the 13 mm stage; the development starts from front to back, but they are never all present at the same time [1, 10, 18, 20]. For example, the first AA regresses during the completion of the fourth, and the second disappears before the sixth is completely closed [1, 17] (Figs. 1, 2, and 3).

Controversies exist about the existence of a fifth arterial arch: only the first four pharyngeal arches, grooves, and pouches are distinct structures. Nevertheless, arch arteries develop caudal to the fourth arch. The pulmonary arch cau-

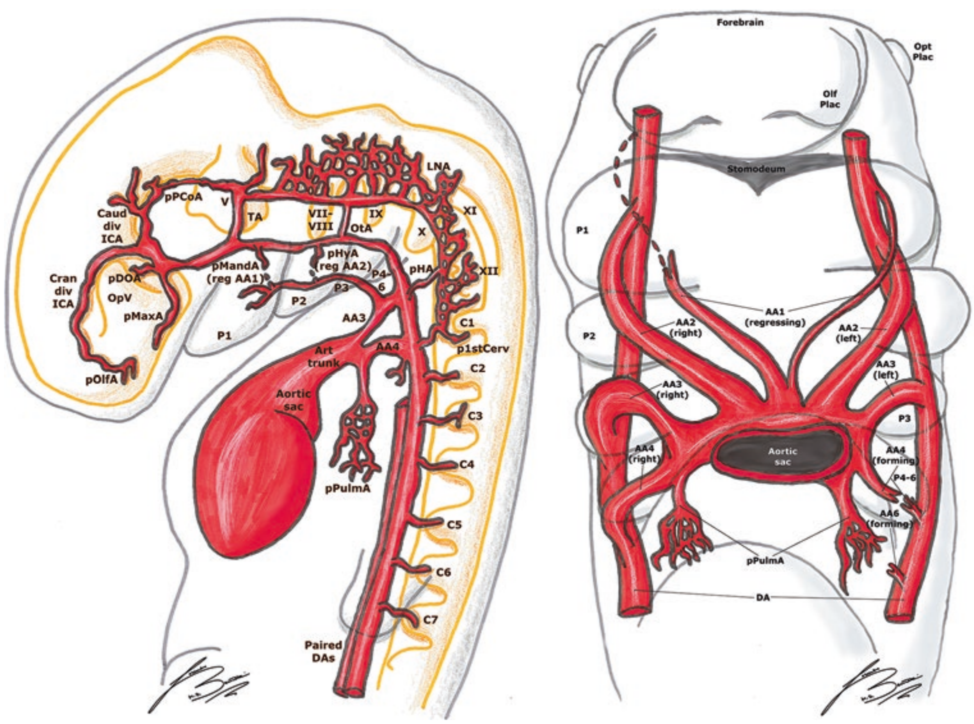

Fig. 1 Padget stage 1; lateral (left) and frontal (right) view of a 4–5 mm, ≃30 days old embryo. In gray, the outer structure of the embryo is outlined; in yellow, the developing nervous system is represented. Developing cranial nerves are numbered from V to XII, developing spinal roots are listed from C1 to C7 and pharyngeal arches from P1 to P4–6

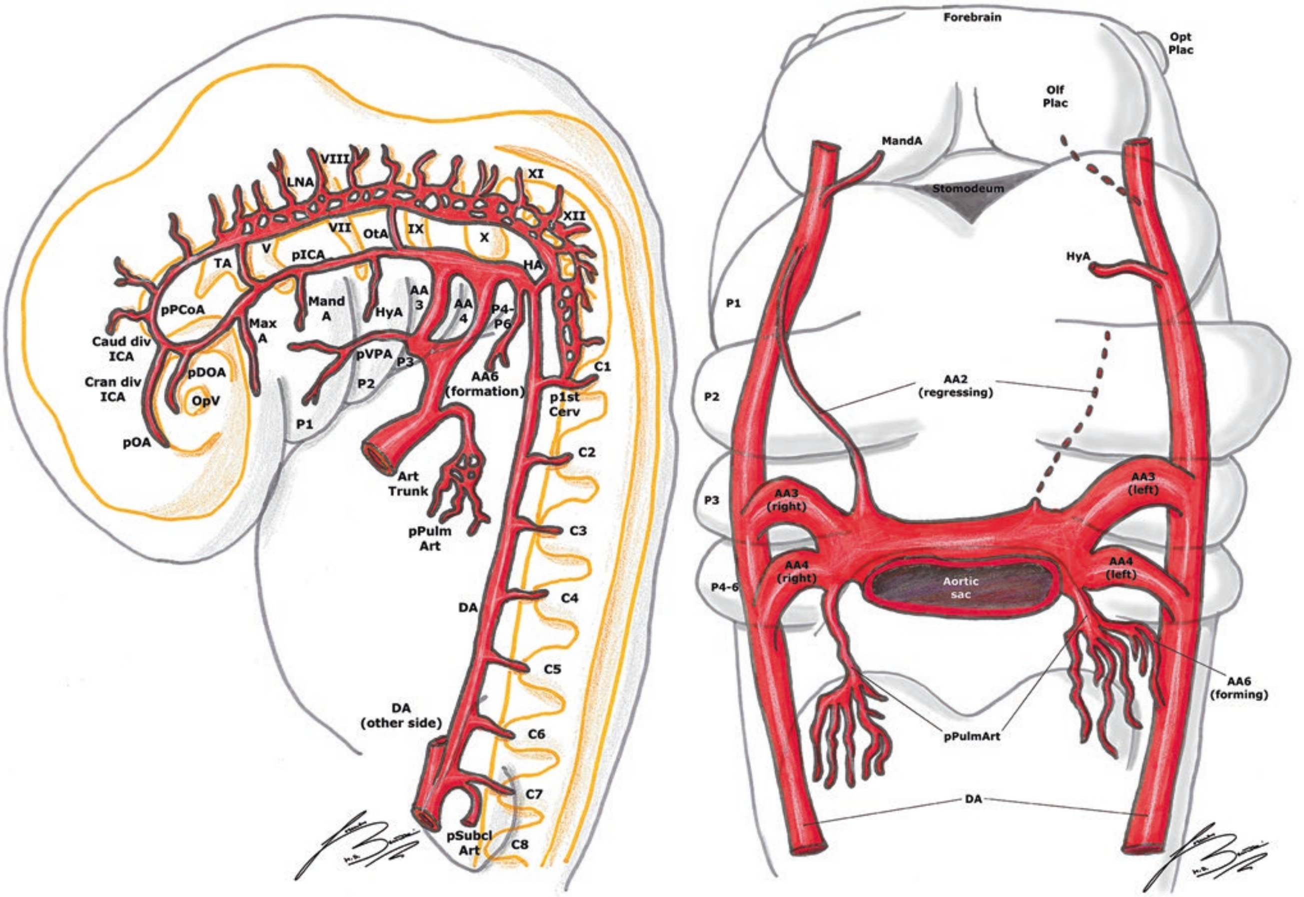

Fig. 2 Padget stage 2; lateral (left) and frontal (right) view of a 5–6 mm, ≃31 days old embryo. In gray, the outer structure of the embryo is outlined; in yellow, the developing nervous system is represented. In gray, the outer structure of the embryo is outlined; in yellow, the developing nervous system is represented. Developing cranial nerves are numbered from V to XII, developing spinal roots are listed from C1 to C8 and pharyngeal arches from P1 to P4–6

dal to the fourth arch artery is named the "sixth arch artery" because of its phylogeny, even when a fifth arch artery is not present [17]. Some recent works have tried to explain several anomalies, such as double-barrel aorta, with the presence of a persistent fifth arch artery; despite in the most cases other mechanisms seem to explain these anomalies, in rare cases "true" fifth arterial arches have been described [21–23].

In embryos longer than 13 mm, the portion of DA between the third and fourth AA disappears on both sides of the embryo [1, 10]. The same does the segment between the fourth AA and the junction with left DA, but only on the right side [1, 10]. This way, the left horn of the aortic sac and the fourth AA form definitive aortic arch (AOA), and the right horn of the aortic sac becomes the arteria anonyma (future brachio-cephalic trunk). The third AA will become the ICA [1, 8, 10]. Its main trunk is formed by the segment of the DA between the first and third AA. The external carotid arteries (ECA) develop later from the ventral pharyngeal arteries [1, 8, 10, 11] (Figs. 4 and 5).

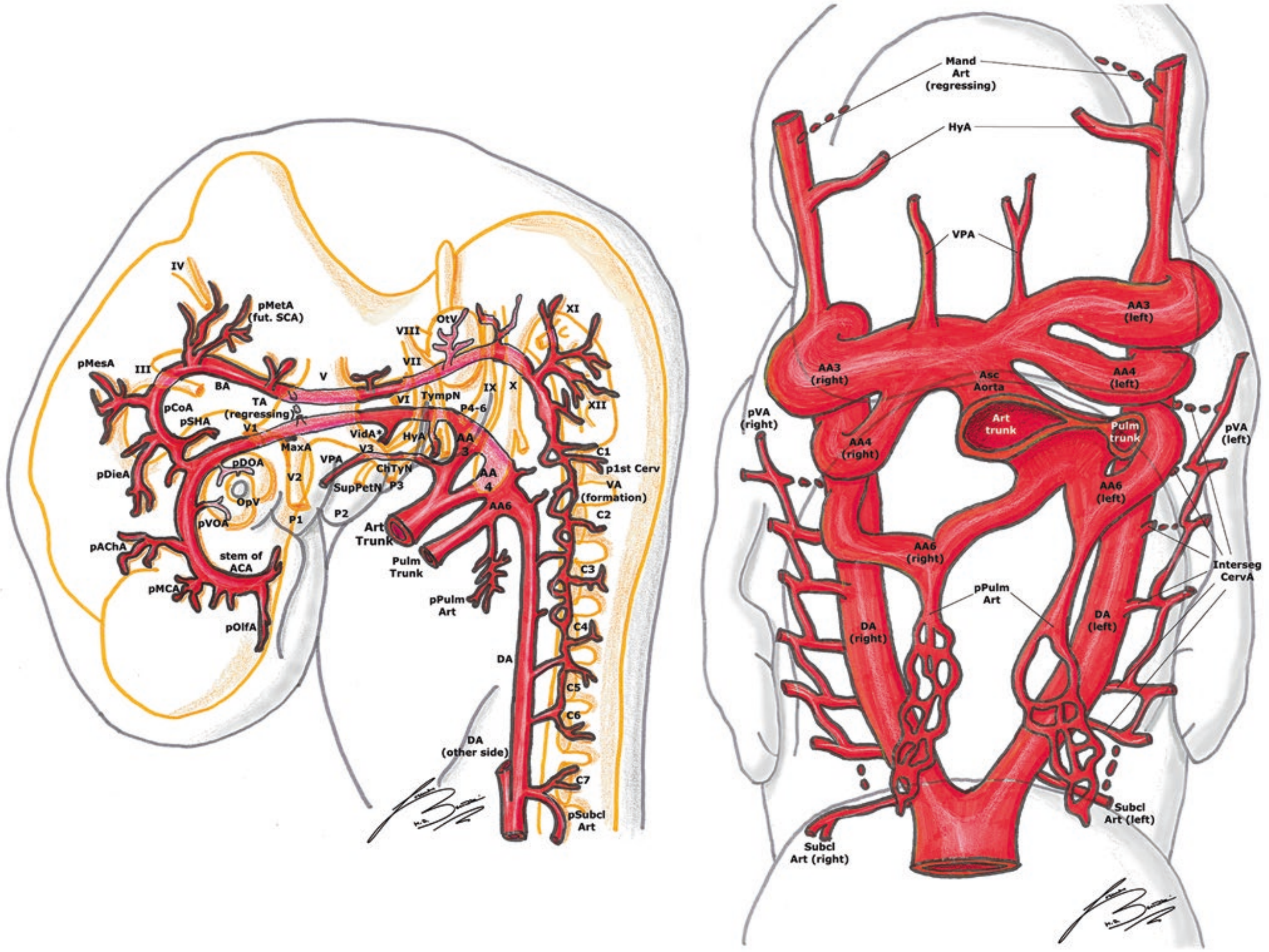

Fig. 3 Padget stage 3; lateral (left) and frontal (right) view of a 7–12 mm, ≃33 days old embryo. In gray, the outer structure of the embryo is outlined; in yellow, the developing nervous system is represented. In gray, the outer structure of the embryo is outlined; in yellow, the developing nervous system is represented. Developing cranial nerves are numbered from III to XII, developing spinal roots are listed from C1 to C7 and pharyngeal arches from P1 to P4–6

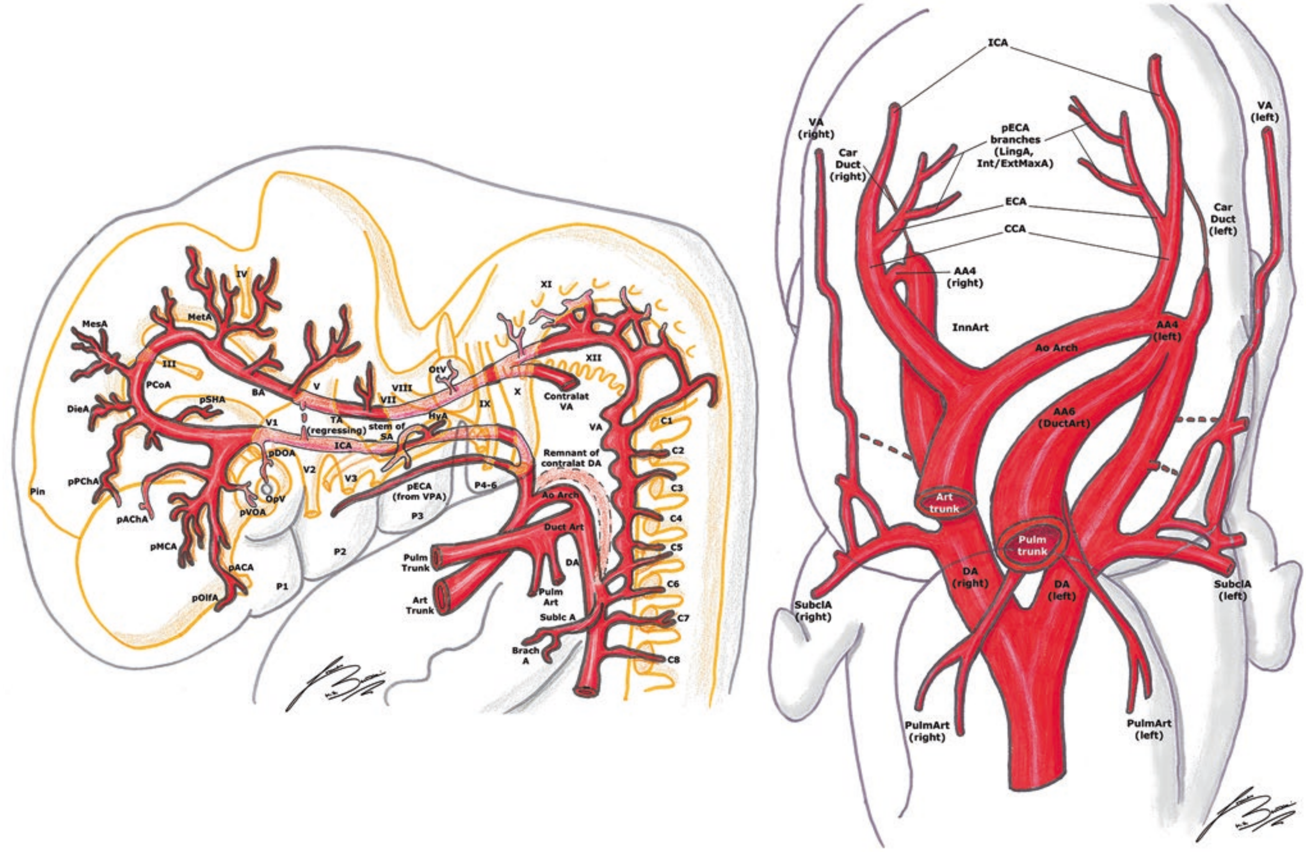

Fig. 4 Padget stage 4; lateral (left) and frontal (right) view of a 12–14 mm, ≃36 days old embryo. In gray, the outer structure of the embryo is outlined; in yellow, the developing nervous system is represented. In gray, the outer structure of the embryo is outlined; in yellow, the developing nervous system is represented. Developing cranial nerves are numbered from III to XII, developing spinal roots are listed from C1 to C8 and pharyngeal arches from P1 to P4–6

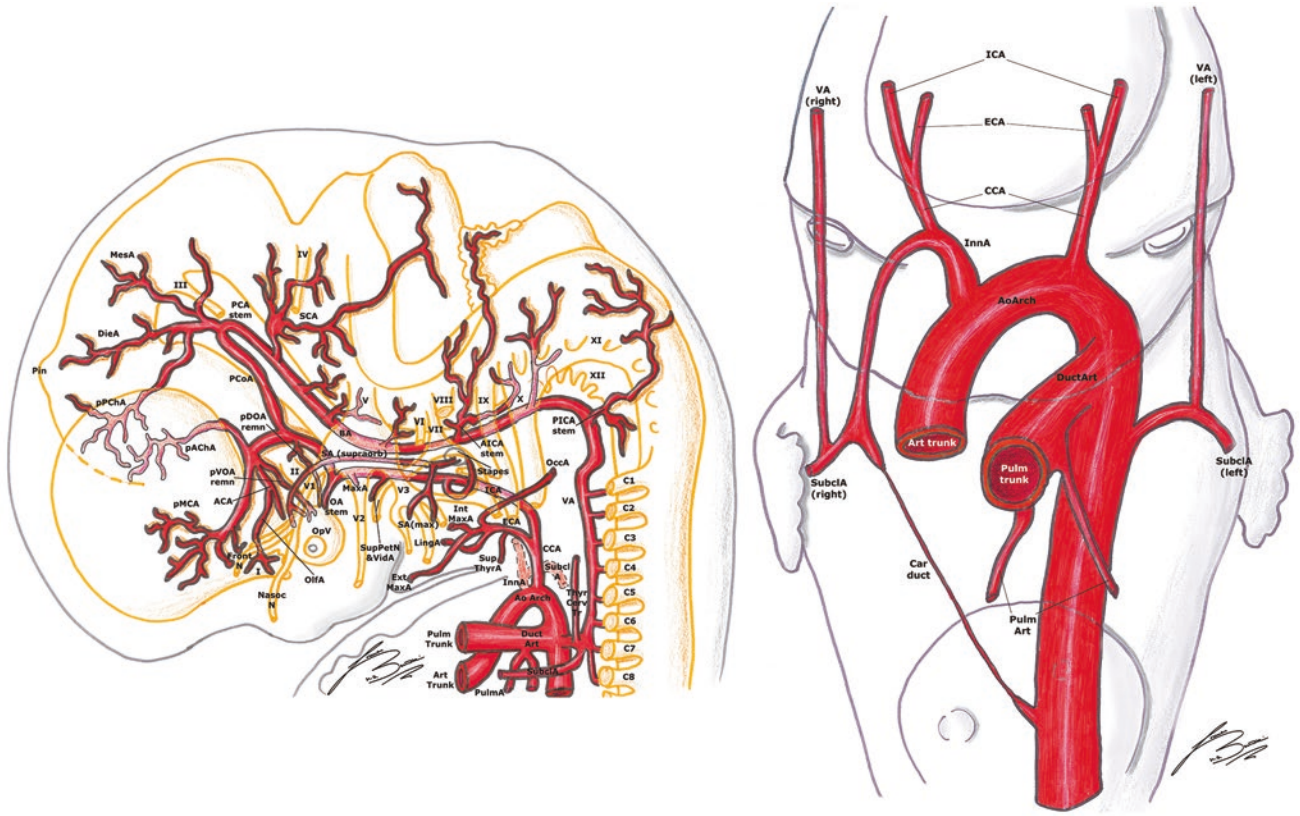

Fig. 5 Padget stage 5; lateral (left) and frontal (right) view of a 16–18 mm, ≃40 days old embryo. In gray, the outer structure of the embryo is outlined; in yellow, the developing nervous system is represented. In gray, the outer structure of the embryo is outlined; in yellow, the developing nervous system is represented. Developing cranial nerves are numbered from I to XII, developing spinal roots are listed from C1 to C8. Pharyngeal arches are becoming the respective fetal derivatives

The Development of the Cranio-Facial Arteries (Stage by Stage Development)

"Pre-Padget" Stage (≃21 to ≃28 Days, from 1.5–2 to 4 mm; Carnegie Stages 10–12)

At the beginning of the "pre-Padget" stage, after about 21 days of intrauterine life, both the ventral aortas (from the aortic bulb) and DAs are connected by the first AA [1, 10, 11]. In the next days, the second AA develops and the first two AAs are thus well visible [1, 17]. The two DAs course along the whole embryo, giving segmental branches to each somite [1, 17]. The primitive heart starts beating after about 23 days [1, 11, 17].

At about 4 mm length, ≃28 days of intrauterine life, the two ventral aortas (or the two horns of the aortic bulb) are still present; the DAs are beginning to fuse, starting from their caudal end in a "zipper-like" fashion to form the definitive aorta [1, 11]. The first four AAs are visible; the first one is beginning to disappear while the fourth is being completed [1, 10].

4–5 mm Stage (≃30 Days), Padget Stage 1; Carnegie Stage 13 (Fig. 1)

At this stage, the optic vesicle (OpV, whose formation began around the 18th day) is well visible. The first AA is completely regressed, and the regression of the second has begun; they will become the mandibular (MA) and hyoid arteries (HyA), respectively [1, 11, 18–20, 24] (Fig. 1).

As mentioned before, the primitive ICA is beginning its development from the DA located between the first three AAs and from the third AA itself [1, 8, 11, 18, 19]. At the level of the trigeminal ganglion, there is a first division into two branches: 1. the primitive trigeminal artery (TA), which extends dorsally to join the longitudinal neural artery (LNA) on the hindbrain wall; 2. the continuation of the carotid, extending cranially and medially toward Rathke's pouch [1, 8, 11, 18–20, 25, 26]. This branch joins its counterpart on the opposite side, which is connected by a plexus [1, 11, 25]. The primitive maxillary artery (PMA) is also developing at the base of Rathke's pouch, with a ventral course along the forebrain wall toward the OpV [1, 27].

Near to the most cranial part of the OpV, the ICA bifurcates into two branches: the caudal branch will become the posterior communicating artery (PComA) and, in the future stages, the posterior cerebral artery (PCA); the cranial branch (future anterior cerebral artery, ACA) curves around the frontal part of the vesicle to terminate in the olfactory area [1, 8, 11, 25] (Figs. 1 and 2).

The primitive ophthalmic artery (OA) appears like a small branch sprouting near the carotid division at the medial side of the optic vesicle, but the supply to the vesicle itself is highly plexiform in this stage and contributed by the carotid and the PMA [1, 11, 25, 27] (Figs. 1 and 2).

In the posterior cerebral region (hindbrain), the two LNAs appear, with a strip of non-vascular tissue in the midline [1, 8, 11, 18, 25, 28]. They are supplied cranially by the TA and otic artery (OtA) and caudally by the hypoglossal artery (HA) and first cervical segmental artery (p1stCerv, also called proatlantal artery) [1, 8, 11, 18, 25, 28] (Figs. 1 and 2).

5–6 Mm Stage (≃31 Days), Padget Stage 2; Carnegie Stage 14 (Fig. 2)

The ICA, extending from the third AA, is now well delineated. The MA (remnant of the first aortic arch) is now regressing [1, 11, 18–20, 24]. The HyA (remnant of the second aortic arch) is quite thick and will become the stem of the stapedial artery (SA) in the future stages [1, 11, 18–20]. In the median part of the embryo, between the first two AAs, there is a big vessel, called the ventral pharyngeal artery (VPhA). This vessel extends from the aortic bulb to a cranial and lateral direction toward the mandibular root of the fifth nerve [1]. Once the AAs regress, this branch becomes the primitive ECA [1, 8, 11, 19, 20, 24]. The PMA follows the lateral margin of Rathke's pouch to reach the ventromesial aspect of the tip of the prosencephalon, terminating in a plexus [1, 27] (Fig. 2).

Lateral to the origin of the primitive ventral OA (PVOA, the more distal branch), more proximal to the primitive carotid bifurcation, a longer branch develops passing over the dorso-caudal margin of the optic disc to the primitive lens. This is the so-called primitive dorsal OA (PDOA, the more proximal branch) [1, 11, 27, 29]. Consequently, at this stage, the optic supply is dual (PVOA plus PDOA) [1, 27, 29] (Fig. 2).

The caudal division of the ICA has formed a second anastomosis with the cranial end of the LNA at the mesencephalon, forming the definitive PComA [1, 8, 11, 18, 19, 25] (Fig. 2).

Consequently, the TA begins to involve, being replaced by the PComA, and the two LNAs begin to fuse to form the primitive basilar artery (BA). The hypoglossal arteries (HA) also begin their regression; this is because the most part of the blood supply to the caudal LNAs and BA comes from the first cervical segmental branches. A temporary vessel supplied cranially by the lateral branch of the BA (i.e., TA) and caudally by the first cervical segment artery often becomes an accessory anastomosis between BA and vertebral arteries (VAs) [1, 11, 18, 19, 28] (Fig. 2).

7–12 Mm Stage (≃33 Days), Padget Stage 3; Carnegie Stages 15–16 (Fig. 3)

The ICA is quite large in the cerebral part, while is thinner at the level of the third aortic arch. The HyA courses caudally, laterally, and ventrally to reach the area between the seventh nerve root and the glossopharyngeal ganglion (between the second and third branchial arches). The MA is now regressed almost completely. The VPhA supplies the first two pharyngeal arches [1, 11, 19, 24] (Fig. 3).

The PDOA is now fully developed, supplying a plexus to the OpV; the shorter PVOA is also well recognizable, rising near the junction between the ICA and PComA [1, 11, 27, 29]. Several branches now appear from the cranial division of the ICA: the most important of these is the primitive anterior choroidal artery (AChoA), coursing along the outlining choroid fissure in the diencephalon [1, 11, 25, 30]. Some small sprigs arise distally to this branch representing the stem of the MCA [1, 11]. More distally, just before the termination of the ICA at the olfactory sac, the primitive anterior cerebral artery (ACA) is beginning its development [1, 11, 18, 19, 25] (Fig. 3).

From the caudal end of the primitive PComA, two branches emerge right next to the third nerve: one supplies the diencephalon (diencephalic artery, DiA) and gives a posterior choroidal branch (PChoA) which is directed toward the AChA, while the other one supplies the mesencephalon (mesencephalic artery, MesA) [1, 11] (Figs. 3 and 4).

The PChoA from the PComA anastomoses with the AChoA and the choroidal branches of the ACA in the choroidal plexus at the level of the interventricular foramen. Later, the ACA pericallosal branches to the choroidal plexus elongate and regress. This AChoA-PChoA anastomosis is named "limbic arterial arch" and in some cases may persist in the adult life [1, 30, 31] (Figs. 3, 4, 5, and 6).

At this stage, the VA begins its formation by a transverse anastomosis between the six upper cervical segmental arteries; meanwhile, the aortic end of each cervical segmental branch is progressively occluding and each segmental branch regresses [1, 8, 11, 18, 19, 25]. It is notable that each transverse anastomosis courses from the more proximal end of one segmental branch to the more distal end of the next branch (in a cranial direction) (Figs. 3 and 4).

From the cranial end of the BA, which is still quite irregular in shape and formed by some "islands", rises the metencephalic artery (MetA) or primitive superior cerebellar artery (SCA), supplying the region of the fourth nerve and metencephalon. Some symmetrical branches leaving from the BA supply the developing cranial nerves [1, 11] (Figs. 3 and 4).

12–14 mm Stage (≃36 Days), Padget Stage 4; Carnegie Stage 17 (Fig. 4)

This stage is important from a developmental point of view for the higher vertebrates. The definitive conformation of the cranial division of the ICA is well identifiable. The first collateral of this cranial division is the AChoA [1, 11, 25, 30]. The second one is the primitive MCA, and the next and more distal branch is the stem of the ACA, at the terminal end of the cranial ICA division (the primitive olfactory artery, POA) [1, 11, 19, 27]. This artery divides into a branch to the nasal fossa, and another and more medial one to the emerging olfactory nerve root; this second branch is the future definitive ACA, and in some embryos is joined with its contralateral homolog by plexiform anastomoses in the midline, the "sketch" of the anterior communicating artery (AComA) [1, 11, 18, 19, 25, 27]. It is notable that, in this phase, the terminal branch of the ICA appears to be the ACA, whereas in the adult configuration, the MCA is the most important branch of this artery, and the ACA seems to be a "collateral" (Figs. 3 and 4).

The PDOA is now longer and gives two branches: the common temporal ciliary artery (ComTempCilA, Fig. 6), to the caudo-dorsal aspect of the optic cup, and the hyaloid artery (HyalA, future central retinal artery), which enters the ocular cleft. The PVOA is also elongated and its distal part will form the common nasal ciliary artery (ComNasCilA, Fig. 6). The definitive stem of the OA has not yet begun its development [1, 11, 27, 29].

This stage is characterized by the formation of the SA. The formation of this branch starts from the HyA, which gives a collateral branch passing through the primitive stapes, between the first pharyngeal pouch to the seventh nerve, to descend in the mandibular substance [1, 11, 20] (Fig. 4).

The thyroid (ThyrA) and lingual (LingA) arteries, branches of the ECA, arise from the VPhA and begin their development in most of the embryos at this stage (Fig. 5). The obliteration of the two ventral aortas between the third and fourth AA is almost complete, giving rise to the common carotid artery (CCA) [1, 11, 24] (Figs. 4 and 5).

16–18 Mm Stage (≃40 Days), Padget Stage 5; Carnegie Stages 18–19 (Fig. 5)

At this stage, we can see the ongoing clarification of the adult vascular configuration. The descent of the heart and large proximal arterial trunks into the thorax is now almost completed (Figs. 5 and 6).

The CCA is nearly fully elongated, the VA is more regular and straightened and its origin from the subclavian artery (SBA), shifting cranially and opposite to the pulmonary arch (the sixth AA), is defined (Fig. 5).

The POA (the lateral terminal part of the anterior division of the ICA, as seen in the previous stage) is quite large and gives off a branch for the olfactory nerve, but now its medial counterpart, the stem of the definitive ACA, is clearly bigger [1] (Figs. 5 and 6).

The MCA is now a quite big trunk with branches spreading out to the growing hemisphere. The AChoA and PChoA terminate in the choroidal fissure at the diencephalic roof [1, 30, 31] (Figs. 5 and 6).

The permanent stem of the OA appears; although the exact formation of this artery is still debated and controversial, we here report the most accepted hypothesis. The SA is now entirely developed, giving two major branches. The first one is the maxillo-mandibular branch (ventral), from which the MA and MaxA (or infraorbital) arteries arise. The second

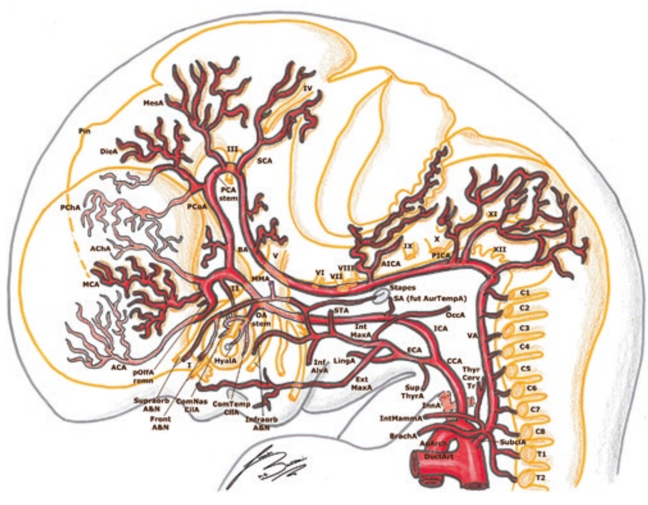

Fig. 6 Padget stage 6; lateral view of a 20–24 mm, ≃45 days old embryo. In gray, the outer structure of the embryo is outlined; in yellow, the developing nervous system is represented. In gray, the outer structure of the embryo is outlined; in yellow, the developing nervous system is represented. Developing cranial nerves are numbered from I to XII, developing spinal roots are listed from C1 to T2. The arterial "ring" or loop around the optic nerve is now visible

one is the supraorbital branch (dorsal), which passes lateral to the trigeminal ganglion to reach the primitive orbit. Here, this branch anastomoses with branches of the PDOA (ComTempCilA and HyalA) and of the PVOA (ComNasCilA). Therefore, the definitive OA consists of two different parts of the ICA: 1) the supraorbital branch of the SA, which gives the orbital arteries and 2) PDOA and PVOA, which give the ocular branches. As these anastomoses form, the original stem of the PDOA regresses (becoming part of the inferolateral trunk) and the artery itself makes a sort of caudal "migration" along the ICA. As a result, the definitive stem of the OA develops in its final position [1, 11, 20, 27, 29] (Figs. 5 and 6).

It is usually found as a dorsal remnant of the MA which runs along the future greater petrosal nerve in the pterygoid (or Vidian) canal, to reach the pterygopalatine fossa: these are the adult Vidian artery (ViA) and nerve. There usually is also a primitive maxillary branch, directed medially and cranially toward the caudal border of the hypophysis, which contributes to the inferior hypophyseal artery [1, 11, 19, 24] (Figs. 5 and 6).

Together with the appearance of the definitive CCA, several ECA branches begin their development: the thyroid (ThyrA), lingual (LingA), occipital (OccA), and external maxillary (ExtMaxA) arteries appear. The internal maxillary artery (IMA) anastomoses with the mandibulo-maxillary branch of the SA [1, 11, 20, 24, 27] (Figs. 5 and 6).

The diencephalon is supplied by two large branches: the more anteroventral is the PChoA, the other one is the DiA, which courses toward the primitive pineal region. The mesencephalon is also supplied by two branches (MesA and lateral branch of PChoA) [1] (Figs. 4, 5, and 6).

At this stage, the PCA starts to develop as the caudal segment of the PComA and can be considered a branch of the PComA such as the PChoA or the hindbrain arteries. During the development of the cerebral hemispheres, the PCA progressively enlarges, and the other smaller arteries become PCA branches, as we can see in the adult configuration [1, 11, 18, 19, 25] (Fig. 5).

The BA branches also develop as the mesencephalon and the fourth ventricle region differentiate. The SCA divides in a mesial branch coursing caudal to the future fourth nerve and a lateral one continuing over the cerebellar lip. The stem of the future anterior inferior cerebellar artery (AICA) arises at the level of the eighth nerve, ending at the choroid plexus of the fourth ventricle. More caudally, many small branches emerging from the VA with many anastomoses pass through the rootlets of the vagus and accessory nerves. One bigger branch of the VA runs cranially to the choroid plexus along the medulla, constituting the stem of the future posterior inferior cerebellar artery (PICA) [1, 11, 25] (Figs. 5 and 6).

20–24 Mm Stage (≃45 Days), Padget Stage 6; Carnegie Stages 20–21 (Fig. 6)

In this stage, as the head begins to lift away from the chest, acquiring more human features, and the cerebral hemispheres expand, the final configuration of the adult circle of Willis is almost perfectly recognizable.

The IMA (a branch of the ECA, as mentioned before) now anastomoses with the lower division of the SA (maxillo-mandibular branch), going lateral to V3 nerve. When the ventral (infraorbital) division of the SA becomes surrounded by the auriculotemporal nerve, the part remaining over the anastomosis with the IMA becomes the stem of the middle meningeal artery (MMA). The dorsal (supraorbital) division of the SA, laterally to the trigeminal ganglion, constitutes the extension of the MMA. At this point, the proximal segment of the SA (medial to the stapes) begins its regression: thus, the maxillo-mandibular branch of the SA (and so the MMA too) becomes an ECA branch [1, 11, 20, 27, 29] (Fig. 6).

At the level of the OA, the primitive ComTempCilA and HyalA anastomose with the ComNasCilA of the primitive ventral OA to constitute a sort of "arterial loop" around the optic nerve. This loop is also connected with the supraorbital branches of the SA (which has become the MMA, as seen above). As a result, the OA gains a more dorsal position to the optic nerve and gets its orbital branches (that come from the SA) [1, 11, 20, 27, 29] (Fig. 6).

The two ACAs approximate and course upwards between the two expanding cerebral hemispheres. In some embryos, a small branch to the choroid plexus can be found, before the development of the corpus callosum [1, 19]. The persistence of such branch in the human adult is extremely rare, as it regresses while the corpus callosum develops and the artery moves more cranially and dorsally [1, 18, 19]. The AComA is much less plexiform and almost completed, with a branch to the commissural plate; the course of this branch is more caudal than ACA's, and it can become an unpaired or accessory ACA in the adult [1, 8]. The POA is now a small branch which follows the olfactory nerve in the nasal cavity; a larger branch, arising from the primitive stem of the olfactory artery, has a more lateral course to supply the forming basal ganglia, called the medial striate branch. This is the so-called "recurrent artery" (or Heubner's artery). This artery is formed by components of the anastomosis between the primitive olfactory artery and its "collateral" ACA branch; it leaves the ACA at the level of the AComA (or slightly more distally), extends toward the anterior perforated substance and enters it to supply the anteroinferior part of the striatum and internal capsule [1, 11, 27, 32, 33] (Fig. 7).

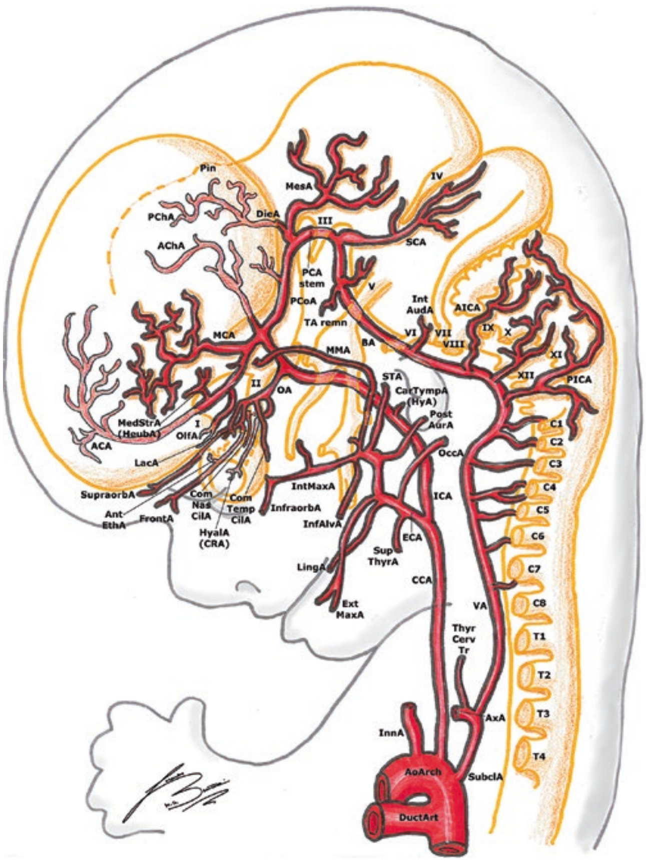

Fig. 7 Padget stage 7; lateral view of a 40 mm, ≃50 days old embryo. In gray, the outer structure of the embryo is outlined; in yellow, the developing nervous system is represented. In gray, the outer structure of the embryo is outlined; in yellow, the developing nervous system is represented. Developing cranial nerves are numbered from I to XII, developing spinal roots are listed from C1 to T4. The definitive pre-term vessel configuration is almost completed

The PComA (which initially was a direct continuation of the ICA to the BA) now moves cranially and posteriorly becoming a true "collateral" leaving the ICA with a 90° angle.

The AChoA and PChoA are connected by several anastomoses at the level of the diencephalon and choroid plexus, as explained before [1, 11, 30, 31]. With the progressive enlargement of the basal ganglia, the contribution of the AChoA for the plexus becomes smaller, as this artery's main supply remains the pallidus, the posterior limb of the internal capsule, the optic tract, the caudate tail, and the amygdala, with some cortical branches to the uncinate gyrus (Figs. 6 and 7).

The stems of both AICA and PICA are still difficult to recognize among many arterial branches supplying the posterior part of the hindbrain, which still have a quite plexiform configuration. This is because the cerebellar hemispheres have not developed yet. The great number of variants in the vertebrobasilar circulation is explained by the late formation of the cerebellar hemispheres, which leads to this highly plexiform arterial configuration with persistence of remnants of the vertebro-basilar anastomoses described from stage 2 [1, 8, 11, 18, 19, 25].

40 Mm Stage (≃50 Days), Padget Stage 7; Carnegie Stages 22–23 (Fig. 7)

In this stage, in almost all the embryos the adult stem of nearly all the main arterial branches can be identified. The circle of Willis is now fully recognizable also from the basal view thanks to the definitive position of the PComA. As mentioned before, the length, size, and direction of the posterior branches (PCA, AICA, PICA) are determined by the development of the cerebellum [1, 25].

As seen before, we can find an accessory branch of the ACA close to the midline (the former "median artery of the corpus callosum") [1, 8].

In this phase, the OA takes the blood supply of the orbit as the SA stem has disappeared during the previous stage. The lacrimal artery (LacA) is the last appearing orbital branch of the OA, originating from the orbital segment of the supraorbital division of the SA. The extraorbital part of the supraorbital SA division becomes a collateral of the anterior branch of the MMA (which is now an ECA branch, as seen before). In the adult, this is a site of frequent anastomoses between ICA (LacA) and ECA (anterior branch of the MMA). In the latter stage, the HyalA becomes the central artery of the retina and takes charge of the blood supply to the deep ocular structures [1, 11, 20, 27, 29] (Fig. 7).

The PComA is quite large in this stage, and in this phase the adult configuration known as "fetal PComA" can take shape: the PComA can remain bigger (as the original caudal end of the posterior division of the ICA) and give off the homolateral PCA, with a very small branch connecting the divisional branch of the basilar (the first segment of the PCA in the normal configuration) [1, 8, 11, 19].

Beginning of the Fetal Stage

At 7–8 weeks of gestational age, the embryological phase is over, and we begin to speak about "fetus" [34, 35].

The cerebrum is now a "privileged" organ, being composed by an almost complete arterial system and receiving a more oxygenated blood compared to the other organs.

In fact, well-oxygenated blood coming from the placenta passes from the umbilical vein through the *ductus venosus* (or left half of the liver), bypassing the inferior vena cava to directly reach the left atrium and ventricle through the foramen ovale, and then goes up the ascending aorta to the ICAs. Deoxygenated blood from the superior and inferior vena cava, forming the so-called "via dextra" through the right atrium and ventricle, passes in the pulmonary trunk and *ductus arteriosus* (DuctArt), to mix with the rest of the oxygenated blood coming from the ascending aorta, to supply the other organs and limbs [1, 36–38].

Conclusion

In this chapter, we tried to sum up the main stages and concepts of the embryological development of the blood supply to the brain, in order to have a better understanding of the normal anatomy and, doing so, make it easier to recognize and understand some of the most important anatomical variants or pathologies. This is supposed to be a comprehensive introduction for the following chapters, that will address each artery in detail.

References

1. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–62.
2. Padget DH. The cranial venous system in man in reference to development, adult configuration, and relation to the arteries. Am J Anat. 1956;98(3):307–55. https://doi.org/10.1002/aja.1000980302.
3. Streeter GL. Developmental horizons in human embryos. Description of age group XIII, embryos about 4 or 5 millimeters long, and age group XIV, period of indentation of the lens vesicle. Contrib Embryol Carnegie Inst Wash. 1945;557(31):27–63.
4. Streeter GL. Developmental horizons in human embryos. Description of age groups XV, XVI, XVII, and XVIII, being the third issue of a survey of the Carnegie collection. Contrib Embryol Carnegie Inst Wash. 1948;575(32):133–203.
5. Streeter GL. Developmental horizons in human embryos. Description of age groups XIX, XX, XXI, XXII, and XXIII, being the fifth issue of a survey of the Carnegie collection (prepared for publication by Heuser CH. And corner GW.). Contrib Embryol Carnegie Inst Wash. 1951;592(34):165–96.
6. Streeter GL. Developmental horizons in human embryos description or age groups XIX, XX, XXI, XXII, and XXIII, being the fifth issue of a survey of the Carnegie collection. Contrib Embryol Carnegie Inst Wash. 1957;611(36):167–96.
7. Streeter GL. Developmental horizons in human embryos. Description of age group XI, 13 to 20 somites, and age group XII, 21 to 29 somites. Contrib Embryol Carnegie Inst Wash. 1942;541(30):211–45.
8. Menshawi K, Mohr JP, Gutierrez J. A functional perspective on the embryology and anatomy of the cerebral blood supply. J Stroke. 2015;17(2):144–58. https://doi.org/10.5853/jos.2015.17.2.144.
9. Plate KH. Mechanisms of angiogenesis in the brain. J Neuropathol Exp Neurol. 1999;58(4):313–20. https://doi.org/10.1097/00005072-199904000-00001.
10. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
11. Lazorthes G. Vascularisation et circulation cérébrale. Paris: Masson et Cie; 1966.
12. Leblanc GG, Golanov E, Awad IA, Young WL. Biology of vascular malformations of the brain NWC. Biology of vascular malformations of the brain. Stroke. 2009;40(12):e694–702. https://doi.org/10.1161/STROKEAHA.109.563692.
13. Vailhe B, Vittet D, Feige JJ. In vitro models of vasculogenesis and angiogenesis. Lab Investig. 2001;81(4):439–52. https://doi.org/10.1038/labinvest.3780252.
14. Beck L Jr, D'Amore PA. Vascular development: cellular and molecular regulation. FASEB J. 1997;11(5):365–73.
15. Risau W. Mechanisms of angiogenesis. Nature. 1997;386(6626):671–4. https://doi.org/10.1038/386671a0.
16. Faraci FM, Heistad DD. Regulation of large cerebral arteries and cerebral microvascular pressure. Circ Res. 1990;66(1):8–17. https://doi.org/10.1161/01.res.66.1.8.
17. Congdon ED. Transformation of the branchial-arch system during the development of the human embryo. Contrib Embryol. 1922;68:47–110.
18. Kathuria S, Gregg L, Chen J, Gandhi D. Normal cerebral arterial development and variations. Semin Ultrasound CT MR. 2011;32(3):242–51. https://doi.org/10.1053/j.sult.2011.02.002.
19. Okahara M, Kiyosue H, Mori H, Tanoue S, Sainou M, Nagatomi H. Anatomic variations of the cerebral arteries and their embryology: a pictorial review. Eur Radiol. 2002;12(10):2548–61. https://doi.org/10.1007/s00330-001-1286-x.
20. Silbergleit R, Quint DJ, Mehta BA, Patel SC, Metes JJ, Noujaim SE. The persistent stapedial artery. AJNR Am J Neuroradiol. 2000;21(3):572–7.
21. Gupta SK, Bamforth SD, Anderson RH. How frequent is the fifth arch artery? Cardiol Young. 2015;25(4):628–46. https://doi.org/10.1017/S1047951114002182.
22. Gupta SK, Gulati GS, Anderson RH. Clarifying the anatomy of the fifth arch artery. Ann Pediatr Cardiol. 2016;9(1):62–7. https://doi.org/10.4103/0974-2069.171392.
23. Bamforth SD, Chaudhry B, Bennett M, Wilson R, Mohun TJ, Van Mierop LH, et al. Clarification of the identity of the mammalian fifth pharyngeal arch artery. Clin Anat. 2013;26(2):173–82. https://doi.org/10.1002/ca.22101.
24. Osborn AG. The vidian artery: normal and pathologic anatomy. Radiology. 1980;136(2):373–8. https://doi.org/10.1148/radiology.136.2.7403513.
25. Raybaud C. Normal and abnormal embryology and development of the intracranial vascular system. Neurosurg Clin N Am. 2010;21(3):399–426. https://doi.org/10.1016/j.nec.2010.03.011.
26. Tubbs RS, Verma K, Riech S, Mortazavi MM, Shoja MM, Loukas M, et al. Persistent fetal intracranial arteries: a comprehensive review of anatomical and clinical significance. J Neurosurg. 2011;114(4):1127–34. https://doi.org/10.3171/2010.11.JNS101527.
27. Gregg L, San Millan D, Orru E, Tamargo RJ, Gailloud P. Ventral and dorsal persistent primitive ophthalmic arteries. Oper Neurosurg (Hagerstown). 2016;12(2):141–52. https://doi.org/10.1227/NEU.0000000000001066.
28. Gregg L, Gailloud P. The role of the primitive lateral Basilovertebral anastomosis of Padget in variations of the vertebrobasilar arterial system. Anat Rec (Hoboken). 2017;300(11):2025–38. https://doi.org/10.1002/ar.23633.
29. Roland J, Georges JL, Bracard S, Picard L. Persistence of the perioptic ring of the ophthalmic artery. Anatomical and embryological observations. Bull Assoc Anat (Nancy). 1984;68(200):65–70.
30. Yu J, Xu N, Zhao Y, Yu J. Clinical importance of the anterior choroidal artery: a review of the literature. Int J Med Sci. 2018;15(4):368–75. https://doi.org/10.7150/ijms.22631.

31. Galatius-Jensen F, Ringberg V. Anastomosis between the anterior choroidal artery and the posterior cerebral artery demonstrated by arteriography. Radiology. 1963;81:942–4. https://doi.org/10.1148/81.6.942.
32. Maga P, Tomaszewski KA, Skrzat J, Tomaszewska IM, Iskra T, Pasternak A, et al. Microanatomical study of the recurrent artery of Heubner. Ann Anat. 2013;195(4):342–50. https://doi.org/10.1016/j.aanat.2013.03.011.
33. Vasovic L, Ugrenovic S, Jovanovic I. Human fetal medial striate artery or artery of Heubner. J Neurosurg Pediatr. 2009;3(4):296–301. https://doi.org/10.3171/2008.12.PEDS08258.
34. O'Rahilly R, Müller F. Developmental stages in human embryos. Contrib Embryol Carnegie Inst Wash. 1987:637.
35. Findlay JK, Gear ML, Illingworth PJ, Junk SM, Kay G, Mackerras AH, et al. Human embryo: a biological definition. Hum Reprod. 2007;22(4):905–11. https://doi.org/10.1093/humrep/del467.
36. Degani S. Fetal cerebrovascular circulation: a review of prenatal ultrasound assessment. Gynecol Obstet Investig. 2008;66(3):184–96. https://doi.org/10.1159/000143157.
37. Kiserud T, Acharya G. The fetal circulation. Prenat Diagn. 2004;24(13):1049–59. https://doi.org/10.1002/pd.1062.
38. Kiserud T. Physiology of the fetal circulation. Semin Fetal Neonatal Med. 2005;10(6):493–503. https://doi.org/10.1016/j.siny.2005.08.007.

Part II

Intradural Arterial System

Embryological Development of the Internal Carotid Artery

Sara Bonasia and Thomas Robert

The internal carotid artery (ICA) is considered one of the most "stable" of the cranio-facial arteries because the frequency of its anatomical variations is very low and evaluated as less than 0.1% [1]. However, different types of anatomical variations of the ICA were described in these last decades in the literature, as segmental agenesis, duplication, and aberrant course [2]. All of these anatomical variations partially find their explanation in the knowledge of the embryological development of the ICA [1]. In this chapter, we will describe the different embryological steps of the ICA development. These steps will be fundamental to understand the developing of the other cerebral arteries, which will be described in the following chapters.

History

The first observation about the carotid artery could be attributed to Artistote early before the first human cadaveric dissections. Galen, with its galenic theory about vascular system circulation, considered the artery to contain blood and not air [3]. Based on this concept, Harvey and Da Vinci highlighted the importance of the carotid artery in the vascularization of the brain. The first publications based on human cadaveric dissections were proposed by Vesalius (1543), Willis (1664), Scarpa (1794), or Quain (1844) who proposed masterworks that gave great contribution to the human vascular system's understanding [3].

In the first half of the twentieth century, the phenomenal publication of Dorcas Padget, based on the dissections of 22 human embryos of the Carnegie collection, gave a lot of information about the embryological development of the cranio-facial arteries [4]. In the same period, brilliant authors such as Altmann (1947) paid a particular attention to furnish a comprehensive explanation about the development of the embryologic aortic arches and about the variations of the carotid system [5]. Pioneers of the interventional neuroradiology also furnished a fantastic work in the understanding of all anatomical variations of the cranio-facial arteries. In this contest, P. Lasjaunias was able to give a comprehensive explanation of all variations implicating the internal carotid artery and proposed the most accomplished hypothesis concerning the development of the ICA in seven distinct segments [1, 2]. This hypothesis, first described in 1984 and taken as reference by almost all authors, will be explained in this chapter.

Embryology

The embryological vascular development starts early in the embryological life (Fig. 1).

- The primitive heart is already recognizable at approximately 19 days (embryos of 2 mm) and paired ventral and dorsal aortas are formed and already have a cranial continuity, which is the first cranial arch [3].
- From 21 to 32 days (embryos of 3 to 12 mm), the other aortic arches are successively formed from front to back. At the same time, the dorsal aortas give an extension cranial to the first aortic arch [4].
- At 28 days (embryos of 4–5 mm), the first aortic arch initiates to involute, followed by the second aortic arch [3]. Consequently, the six pairs of aortic arches are not all present at the same time [5].

Each aortic arch has a different function, the two first one involute completely, the third arch persists and will become a part of the internal carotid artery, and the fourth one persists and will give the adult aortic arch on the left and the

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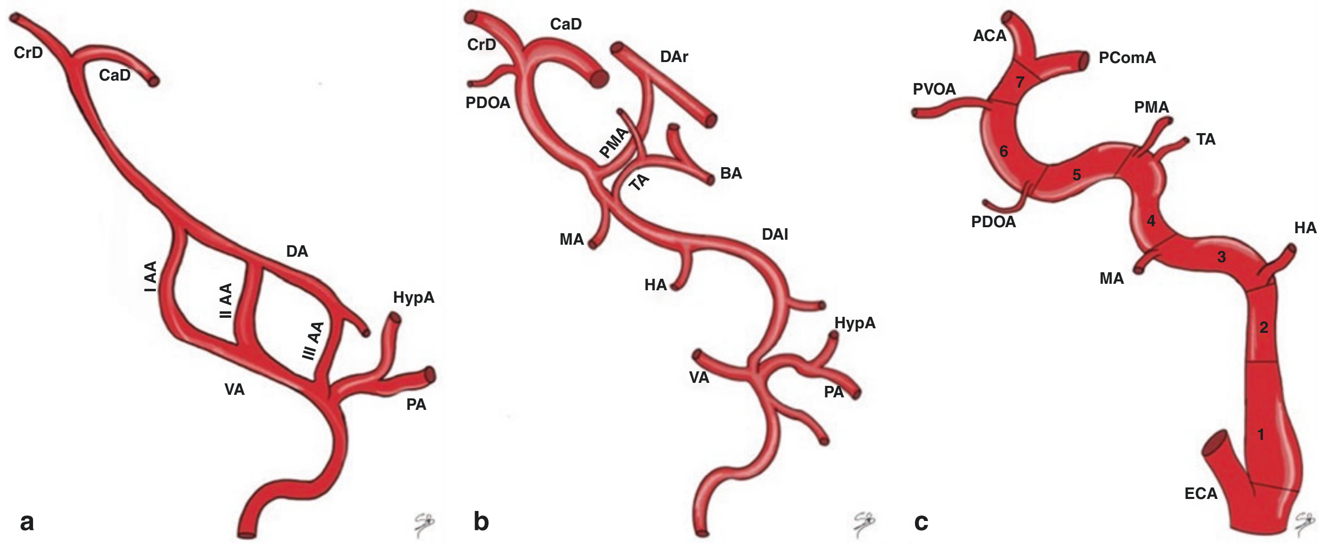

Fig. 1 Embryology and embryological segments of the internal carotid artery. The illustrations (**a**, **b**, and **c**) show consecutive stages of ICA embryological development. The first stages of development (**a**) are characterized by the presence of three aortic arches that link the ventral and dorsal aorta (VA, DA). The VA regresses together with the ventral part of the aortic arches. The dorsal remnants of the aortic arches persist as embryonic arteries. These embryonic arteries divide the ICA into 7 embryological segments: (*1*). The cervical segment: It derives from the remnant of the third aortic arch (III AA). (*2*). The ascending intrapetrous segment: It is the remnant of the dorsal aorta (DA) between the second (II AA) and third aortic arches. The division point between the segment 2 and 3 is at the point of origin of the Hyoid artery (HA), that is the dorsal remnant of the second aortic arch. (*3*) The horizontal intrapetrous segment: It is the remnant of the DA between the first (I AA) and second aortic arches. The division point is at the point of origin of the mandibular artery (MA), which corresponds to the dorsal remnant of the first aortic arch. (*4*) The intracavernous ascending segment: It origins from the DA between the first aortic arch and the primitive maxillary artery (PMA), that connects the DA of the two sides (DAl: dorsal aorta left; DAr: dorsal aorta right). At the junction between segments 4 and 5 also the trigeminal artery (TA) takes its origin. This latter represents a primitive connection between the cavernous ICA and the basilar artery (BA). (*5*) The horizontal intracavernous segment: It derives from the DA between the PMA and the primitive dorsal ophthalmic artery (PDOA). (*6*) The clinoid segment: corresponds to the DA between the PDOA and the primitive ventral ophthalmic artery (PVOA). (*7*) The terminal segment: the terminal ICA between (PVOA) and the primitive ICA bifurcation into the future anterior cerebral artery (ACA) and future posterior communicating artery (PComA). The figure also shows the hypoglossal artery (HypA) and the proatlantal artery (PA), that originate proximal to the third aortic arch and will contribute to the formation of external carotid artery (ECA) branches

brachiocephalic artery on the right. The fifth aortic arch will completely involute and the sixth one will give origin to the pulmonary arteries [6].

Common Carotid Artery

The embryological origin of the common carotid artery is not clear. Altmann in 1947 postulated two different hypotheses [5]. According to the first one, the common carotid artery derivates from the ventral aorta between the third and fourth aortic arch and from the third aortic arch. The second hypothesis says that the common carotid artery derivates directly from the aortic sac a second time after the formation of the aortic arches. In her monograph, Padget (1948) did not insist on the embryological formation of the common carotid artery and considered it as the development of the ventral aorta between the third and fourth aortic arches [4]. Lasjaunias (1984), based on the fact that a segmental agenesis of the cervical internal carotid artery never influences the formation of the common carotid artery, considered that the third aortic arch could not participate in the formation of the distal common carotid artery [2]. In conclusion, the strongest hypothesis concerning the formation of the common carotid artery is that it originates from the ventral aorta between the insertion of the third and fourth aortic arches, which have an important extension after the involution of the other arches as previously described [6, 7].

External Carotid Artery

Also for the external carotid artery (ECA), different hypotheses were postulated to explain its embryological development but the most probable is its direct origin from the aortic sac [4, 5]. The ventral pharyngeal artery (which is the principal precursor of the external carotid artery) extends craniolaterally from the aortic sac as early as the 5–6 mm stage [4].

The ventral pharyngeal artery, which develops cranially to give almost all branches of the future external carotid artery, annexes later (24 mm stage) the maxillomandibular branch of the stapedial artery to become the external carotid artery [6, 8, 9]. At approximately 40 days, the descent of the aortic sac favorites the cranial migration of the external carotid artery origin along the common carotid artery to give the adult configuration [3]. Lasjaunias strongly argued against a common embryological origin of internal and external carotid arteries, based on different types of segmental agenesis of the ICA always sparing the ECA development [1, 2].

Internal Carotid Artery

Early in the development (first stage of Padget, embryos of 4–5 mm), the two first aortic arches initiate their natural regression allowing the internal carotid artery to be individualized. The embryological segments of the ICA are derived from the third aortic arch and from the dorsal aorta cranial to the third aortic arch [1, 4, 5]. The dorsal aorta also regresses at the same time between the third and fourth aortic arches. In the 4–5 mm embryos, the primitive ICA bifurcation is already visible with its two divisions: anterior and posterior that will become in adult, the anterior cerebral artery and the posterior communicating artery. The ICA, cranial to the posterior communicating origin, derivates from the anterior division of the ICA; and not from the ICA itself. Consequently, the primitive ICA bifurcation is considered to be at the origin of the posterior communicating artery and not at the origin of the middle cerebral artery. Concomitantly to the formation of the ICA, other arteries of the circle of Willis have their own development and the circle of Willis is completely formed in the 24 mm embryos (with the formation of the anterior communicating artery).

Embryologically, we could divide the internal carotid artery into seven different segments according to Lasjaunias [2].

These segments, which are represented in Fig. 1 together with the main embryological steps of ICA development, could be described as follows:

1. The first one corresponds to the third aortic arch from the origin of the ventral pharyngeal artery (future external carotid artery) to the junction between the third aortic arch and the dorsal aorta. In the adult, it corresponds to the cervical internal carotid artery.
2. The second segment is the dorsal aorta between the third and second aortic arch. Its distal part is the hyoid artery in the embryo and the caroticotympanic artery in the adult (ascending intrapetrous segment).
3. The third segment is the dorsal aorta between the second and first aortic arch. It corresponds in the embryo to the segment between the hyoid and the mandibular arteries, and, in the adult, between caroticotympanic artery and the Vidian artery (horizontal intrapetrous segment).
4. The fourth segment is the dorsal aorta between the first aortic arch and the origin of the trigeminal artery (and the primitive maxillary artery). In the adult, it corresponds to the segment between the Vidian artery and the origin of the meningo-hypophysary trunk (ascending foramen lacerum segment).
5. The fifth segment is the dorsal aorta between the origin of the trigeminal artery (and the primitive maxillary artery) and the origin of the primitive dorsal ophthalmic artery (PDOA, future inferolateral trunk-ILT). It corresponds in the adult to the horizontal cavernous segment between the meningo-hypophysary and infero-lateral trunks.
6. The sixth segment is between the origin of the PDOA and the anastomotic point of the primitive ventral ophthalmic artery (PVOA) on the internal carotid artery. This segment in the adult is the clinoidal segment between the origin of the ILT and the OA.
7. The seventh segment is between the anastomotic point of the PVOA and the primitive carotid bifurcation. In the adult, this is the supraclinoid ICA from the origin of the OA to the origin of the posterior communicating artery.

As described, each segment of the embryonic ICA will give origin to a specific segment in the adult. The correlations between the embryological and the adult segment are summarized in Table 1.

It is important to note that the carotid bulb has not the same embryological origin than the other segments of the ICA. It originates from the pharyngo-occipital system that easily explains variations in the origin of the ascending pharyngeal and occipital arteries.

Table 1 Embryological segments of the ICA with their corresponding segments in the adult

Embryological segment	Embryological origin	Adult segment	Proximal limit	Distal limit
First segment	Third aortic arch	Cervical ICA	Carotid bulb	Junction cervical-petrous ICA
Second segment	Dorsal aorta Between second and third aortic arches	Ascending petrous segment	Junction cervical-petrous ICA	Caroticotympanic artery
Third segment	Dorsal aorta Between first and second aortic arches	Horizontal petrous segment	Caroticotympanic artery	Vidian artery
Fourth segment	Dorsal aorta Between first aortic arch and the primitive maxillary artery	Ascending lacerum segment	Vidian artery	Meningo-hypophysary trunk
Fifth segment	Dorsal aorta Between the primitive maxillary artery and the PDOA	Horizontal cavernous segment	Meningo-hypophysary trunk	Infero-lateral trunk
Sixth segment	Dorsal aorta Between the PDOA and the PVOA	Clinoidal segment	Infero-lateral trunk	Ophthalmic artery
Seventh segment	Dorsal aorta Distal to the PVOA	Ophthalmic segment	Ophthalmic artery	Posterior communicating artery

Clinical Implications

The knowledge of the embryological development of the ICA is mandatory to understand developmental variations and anomalies of this artery. Agenesis or abnormal regression of one or more segments of the carotid artery explains an intratympanic course of the ICA and also the different type of "reperfusion" in case of ICA agenesis [5, 10]. All anatomical variations in the course of the ICA will be described in detail in the respective chapters. The intratympanic course of the ICA also named as "aberrant flow of the ICA" is the consequence of the abnormal regression of the first and second segments with anastomosis between the inferior tympanic artery (from the ascending pharyngeal artery) and the carotico-tympanic artery (from the carotid artery) that infuses distally the carotid artery. The pseudo-ICA has consequently an intratympanic course without passing through the stapes [1, 10, 11].

References

1. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer edition; 2001.
2. Lasjaunias P, Santoyo-Vazquez A. Segmental agenesis of the internal carotid artery: angiographic aspects with embryological discussion. Anat Clin. 1984;6(2):133–41.
3. Dungan DH, Heiserman JE. The carotid artery: embryology, normal anatomy, and physiology. Neuroimaging Clin N Am. 1996;6(4):789–99.
4. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–62.
5. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
6. Li S, Hooda K, Gupta N, Kumar Y. Internal carotid artery agenesis: a case report and review of literature. Neuroradiol J. 2017;30(2):186–91. https://doi.org/10.1177/1971400917692162.
7. Vasovic L, Trandafilovic M, Vlajkovic S, Radenkovic G. Congenital absence of the bilateral internal carotid artery: a review of the associated (ab)normalities from a newborn status to the eighth decade of life. Childs Nerv Syst. 2018;34(1):35–49. https://doi.org/10.1007/s00381-017-3629-1.
8. Lasjaunias P, Moret J, Manelfe C, Theron J, Hasso T, Seeger J. Arterial anomalies at the base of the skull. Neuroradiology. 1977;13(5):267–72.
9. Lasjaunias P, Moret J. Normal and non-pathological variations in the angiographic aspects of the arteries of the middle ear. Neuroradiology. 1978;15(4):213–9.
10. Steffen TN. Vascular anomalites of the middle ear. Laryngoscope. 1968;78(2):171–97. https://doi.org/10.1288/00005537-196802000-00001.
11. Margolis MT, Newton TH. Collateral pathways between the cavernous portion of the internal carotid and external carotid arteries. Radiology. 1969;93(4):834–6. https://doi.org/10.1148/93.4.834.

Segmental Classifications of the Internal Carotid Artery

Sara Bonasia and Thomas Robert

The internal carotid artery (ICA) is one of the most studied arteries in the human, since it interests a lot of physicians: ENT, neuroradiologists, neurologists, anatomists, and neurosurgeons. During the last century, a lot of classifications of the internal carotid artery segmental division have been proposed, with the purpose to be helpful during the clinical practice. Since each specialist who treats pathologies of or around the internal carotid artery needs different details, a universally shared classification is difficult to propose. Historically, the classifications that spread the most had some important characteristics: simplicity, easy to use, easy to remember, and reproducibility. In this chapter, we will present the most used classifications of the internal carotid artery with their respective clinical implications, pros and cons.

History

The term "carotid" was already used by Greek physicians like Aristotle and Galen who, even with an incomplete idea of the human vascular circulation, understood the vital role of this vessel. After them, a lot of physicians, anatomists, and humanists gave their theories or hypothesis about the role of the carotid artery. To cite some of them, Da Vinci (1504), Vesalius (1543), William Harvey (1628), Thomas Willis (1664), and more recently Quain (1844) marked their respective time contributing to the knowledge of the human vascular anatomy [1–7].

In 1938, Fischer, a German anatomist, was the first to propose a classification in segments of the internal carotid artery [8, 9]. However, only at the end of the twentieth century, several authors became more interested in the internal carotid artery [9–11]. Depending on the interest of each author, each classification was based on different elements. Lasjaunias (1984) described different segments based on their embryological development to explain segmental agenesis of the internal carotid artery [12]. Gibo and Rhoton also like Bouthilier proposed their classification principally based on the surgical anatomy of the artery [9, 13]. On the other hand, surgeons experienced in endonasal surgery proposed some classifications focused on the cavernous and petrous segments of the carotid artery [11, 14].

Fischer's Classification

In 1938, Erich Fischer proposed the first segmental classification concerning the internal carotid artery and its two major branches: the anterior and middle cerebral arteries. In its classification, the ICA segments are numbered in the opposite direction of the flow [9]. Fischer did not consider the anatomical relationship of the artery but segmented the ICA depending on the different arterial displacement in case of extrinsic tumoral compression. This classification was based on angiographic studies. From its termination to its cavernous region, the ICA was segmented into five different segments, which are illustrated in Fig. 1 [15].

1. The first segment (C1) is the portion of the ICA from its termination to the origin of the posterior communicating artery.
2. The second segment (C2) is the part of the ICA between the origin of the posterior communicating artery and the origin of the ophthalmic artery (OA).
3. The third segment (C3) is the anterior genu of the ICA that corresponds to the clinoidal portion of the artery.
4. The fourth segment (C4) is the cavernous portion of the artery that ends at the posterior genu of the ICA.

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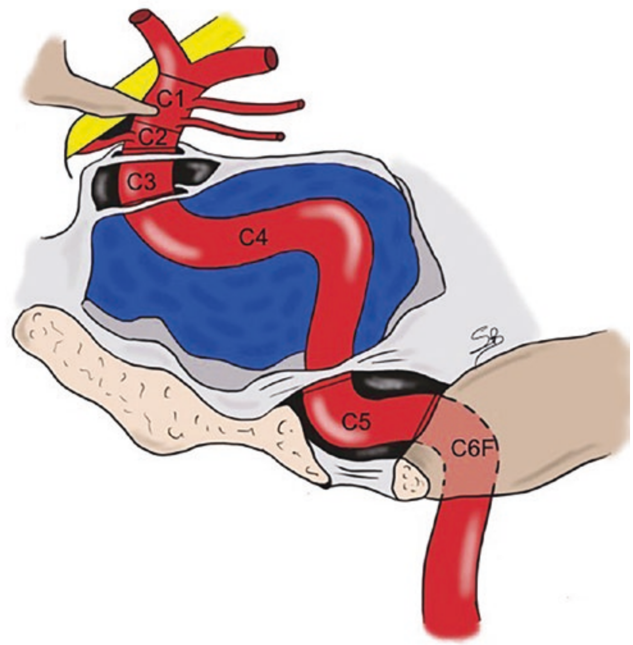

Fig. 1 Fischer's classification

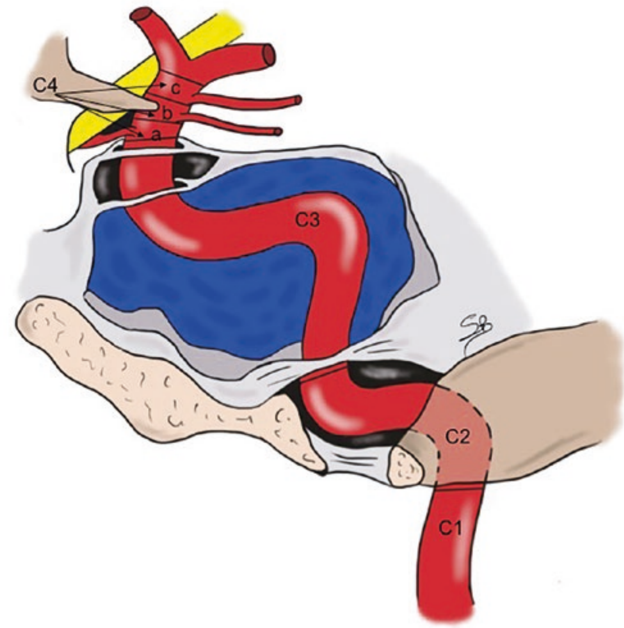

Fig. 2 Rhoton's classification

5. The fifth segment (C5) is the part of the ICA latero-inferior to the cavernous sinus in the lacerum foramen. This segment does not include the petrous segment.

This classification has the merit to be the first one and to have helped physicians in the understanding of tumors location before the computerized tomography era. The disadvantages of this classification were the numbering opposite to the arterial flow, the exclusion of the petrous and cervical portions of the artery, and the absence of correspondence to anatomical landmarks. In 1994, Fukushima, in a personal communication, proposed a change of this classical classification adding a sixth segment (C6F) that corresponds to the petrous portion of the artery.

Gibo and Rhoton's Classification

This classification, proposed in 1981, is actually the most used. It was proposed after the cadaveric dissection of 25 brains [9]. This is the first anterograde segmentation of the internal carotid artery. Each segment of the artery has a different anatomical compartment that makes this classification easy to use and reproducible. The different segments are illustrated in Fig. 2 and were described as follows:

1. C1 is the cervical portion of the ICA from its origin to its entry into the external orifice of the carotid canal.
2. C2 is the petrous portion of the artery from the external orifice of the carotid canal to the entry of the ICA into the cavernous sinus.
3. C3 corresponds to the cavernous portion of the artery from its entrance in the posterior part of the cavernous sinus to its exit near the anterior clinoidal process. The origin of the ophthalmic artery is assimilated to the junction between C3 and C4 (cavernous and supraclinoid) segments.
4. C4 is the supraclinoid portion of the ICA that was divided by Gibo et al. into three distinct parts: the ophthalmic segment (C4-a) from the origin of the OA to the origin of the posterior communicating artery, the communicating part (C4-b) from the origin of the posterior communicating artery to the origin of the anterior choroidal artery, and the choroidal part (C4-c) from the origin of the anterior choroidal artery to the ICA bifurcation.

This classification highlights the precise anatomy of perforating arteries from the ICA on its supraclinoid portion and was oriented in the surgical clipping of ICA aneurysms with precise description of anatomical relationship between aneurysmal sac and perforating arteries.

Embryological Classification (Lasjaunias)

This classification was first proposed by Pierre Lasjaunias in 1984 and is only based on the embryological knowledge of the carotid artery system [4, 12]. The aim of this classification is to explain and understand the different types of segmental agenesis and other anatomical variations encountered in the clinical practice. The internal carotid artery development depends on the third aortic arch and on the dorsal aorta cranial to the third aortic arch. During the embryological life, the ICA gives origin to different branches that delimitate different segments of this classification. It is important to remember that, from an embryological point of view, the ICA bifurcates in two cranial and caudal divisions that correspond to the future anterior cerebral artery and to the future posterior communicating artery.

According to P. Lasjaunias (1984), the different segments of the ICA, represented in Fig. 3, could be identified as follows [12]:

1. The first one corresponds to the third aortic arch from the origin of the ventral pharyngeal artery (future external carotid artery) to the junction between the third aortic arch and the dorsal aorta. In the adult, it corresponds to the cervical internal carotid artery.
2. The second segment is the dorsal aorta between the second and third aortic arch. Its distal part is the hyoid artery in the embryo and the caroticotympanic artery in the adult (ascending intrapetrous segment).
3. The third segment is the dorsal aorta between the second and first aortic arch. It corresponds in the embryo to the segment between the hyoid and the mandibular artery, and, in the adult, between caroticotympanic artery and the Vidian artery (horizontal intrapetrous segment).
4. The fourth segment is the dorsal aorta between the first aortic arch (future mandibular artery) and the origin of the trigeminal artery (and the primitive maxillary artery). In the adult, it corresponds to the segment between the Vidian artery and the origin of the meningo-hypophysary trunk (ascending foramen lacerum segment).
5. The fifth segment is the dorsal aorta between the origin of the trigeminal artery (and the primitive maxillary artery) and the origin of the primitive dorsal ophthalmic artery (PDOA, future inferolateral trunk-ILT). It corresponds in the adult to the horizontal cavernous segment between the meningo-hypophysary and infero-lateral trunks.
6. The sixth segment is between the origin of the PDOA and the anastomotic point of the primitive ventral ophthalmic artery (PVOA) on the internal carotid artery. This segment in the adult is the clinoid segment between the origin of the ILT and the OA.
7. The seventh segment is between the anastomotic point of the PVOA and the primitive carotid bifurcation. In the adult, this is the supraclinoid ICA from the origin of the OA to the origin of the posterior communicating artery.

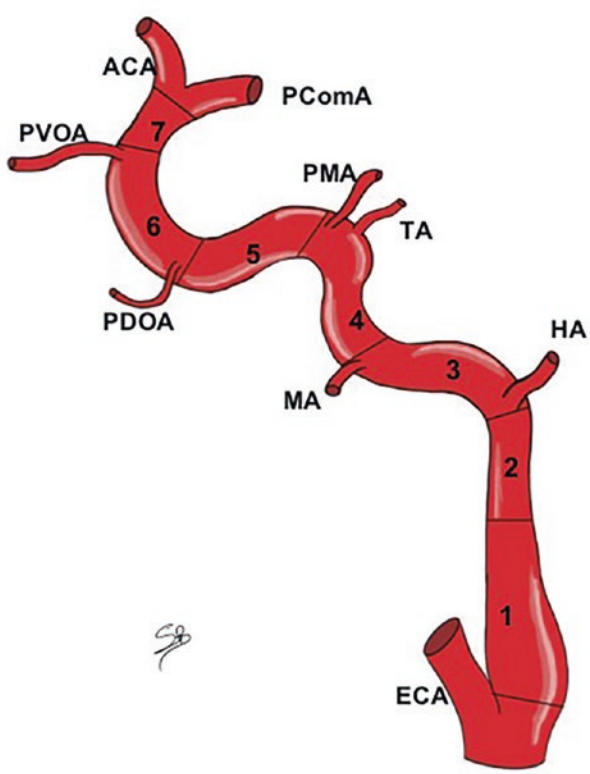

Fig. 3 Lasjaunias' classification

It is important to note that the carotid bulb has not the same embryological origin than the other segments of the ICA. It originates from the pharyngo-occipital system that easily explains variations in origin of the ascending pharyngeal and occipital arteries [4].

This classification allows to understand and explain quite all forms of agenesis and variations of the ICA and is based on two fundamental principles: first, each segment is considered to have a distinct embryological development; second, the agenesis of one segment of the ICA leads to the regression of all segments proximal to the anomaly.

However, in 2004, Gailloud presented a case of ICA agenesis distal to the posterior communicating artery origin [16]. Consequently, he discussed the presence of an eighth embryological ICA segment. This hypothesis was clearly refused by P. Lasjaunias who considered the adult ICA distal to the posterior communicating artery origin not as an ICA segment [17].

Bouthillier's Classification

In 1996, Bouthillier et al. proposed another segmental classification based on a cadaveric study of 10 specimens [13]. This classification is illustrated in Fig. 4 and a clinical case subdivided according to this classification is presented in Fig. 5.

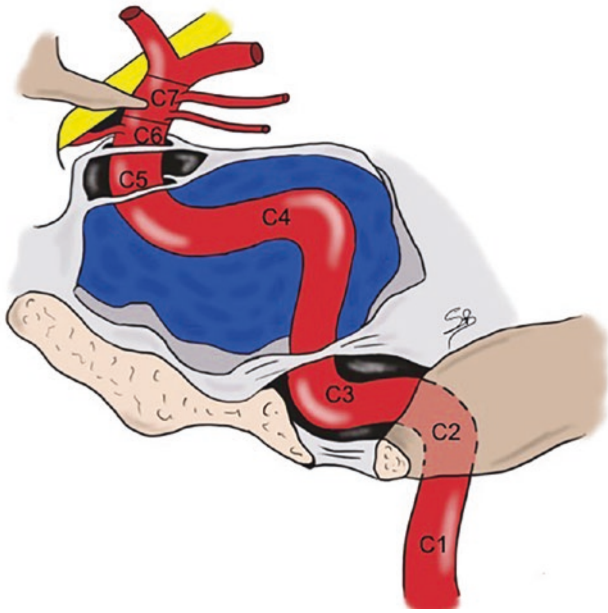

Fig. 4 Bouthillier's classification

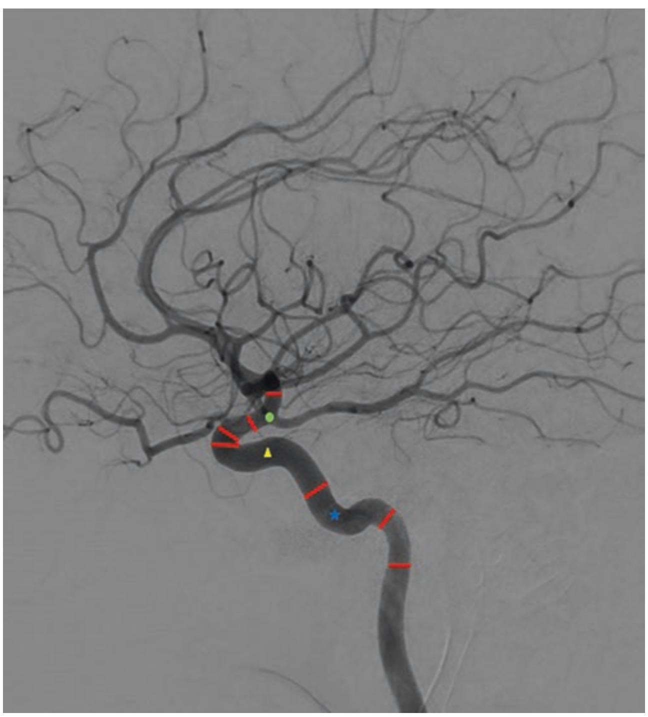

Fig. 5 The figure shows a left ICA angiogram in lateral view, in which the ICA is divided according to Bouthillier's classification. The blue star indicates the petrous segment of the ICA, the yellow triangle indicates the cavernous segment, and the green circle corresponds to the communicating segment at the origin of the posterior communicating artery (that has a fetal configuration in this case)

They divided the petrous segment in petrous and lacerum segment because they observed that in this lacerum segment, the carotid artery is not completely englobed into the petrous bone but into a dural ligament. They argued this added segment for its clinical implication for surgical approaches of the Meckel's cave. They also added the clinoidal segment that is the little part of the internal carotid artery between the proximal and distal dural rings.

Consequently, the ICA is segmented into seven portions as follows:

1. The cervical segment (C1) from the carotid bifurcation to the external extremity of the carotid canal.
2. The petrous segment (C2) from the external orifice of the carotid canal to the posterior edge of the foramen lacerum.
3. The lacerum segment (C3) extends from the end of the carotid canal (posterior edge of the foramen lacerum) to the entry point of the carotid artery into the cavernous sinus. In this portion, the petro-lingual ligament only covers the carotid artery.
4. The cavernous portion (C4) begins at the entry point of the carotid artery into the cavernous sinus and ends at the exit point of the artery at the level of the inferior dural ring of the anterior clinoid process.
5. The clinoid segment (C5) extends from the inferior to the superior dural rings. In this segment, the carotid artery is in a fibro-osseous canal before entering into the subarachnoidal compartment.
6. The ophthalmic segment (C6) begins at the distal dural ring and ends just proximal to the origin of the posterior communicating artery.
7. The communicating segment (C7) is the distal portion of the ICA from the origin of the posterior communicating artery to its bifurcation.

Other Classifications

Recently, numerous authors proposed different classifications depending on their specialty and depending on their interest in their respective clinical practice. The most interesting classifications are summarized in Table 1. Ziyal et al. (2005) proposed another anatomical classification based on cadaveric dissections of 15 human heads. In this classification, authors highlighted the importance of the clinoidal segment, as described by Bouthilier et al. 10 years before [13, 18]. This classification is helpful for skull base surgeons in case of surgeries of the cavernous sinus or in case of anterior clinoidectomy [18]. Shapiro et al. (2014)

Table 1 Summary of the different classifications of the internal carotid artery

ICA classifications	Fisher	Lasjaunias	Gibo-Rhoton	Bouthilier	Ziyal	Shapiro	Abdelrauf
Cervical	x	1	C1	C1	C1	Cervical	x
Petrous	C6 (add by Fukushima)	2 and 3	C2	C2-C3	C2	Petrous	Cochlear petrous clival
Cavernous	C4-C5	4 and 5	C3	C4	C3	Cavernous	Sellar
Clinoidal	C3	6	C3	C5	C4	Peri-ophthalmic	Sphenoid
Ophthalmic	C2	7	C4-opht	C6	C5	Peri-ophthalmic	Cisternal
Communicating	C1	x	C4-com	C7	C5	Communicating	Cisternal
Choroidal	C1	x	C4-chor	C7	C5	Choroidal and terminus	Cisternal

proposed another classification of the ICA in seven segments without differentiation between clinoidal and ophthalmic segments but adding a terminal segment distal to the choroidal segment [10]. This classification was made regarding different possible location of ICA aneurysms and depending on the choice of endovascular technique. Other than a segmental classification, Abdelrauf et al. published a study focused on different angles and curvature of the ICA [11]. This description is very helpful for endoscopic surgeons to avoid vascular damage during these approaches. In the same idea, Wang et al. gave a precise description of the shape and curvature of the ICA in its cavernous segment in order to help identifying it during trans-sphenoidal surgery [14].

Conclusion

Each of these classifications brings important information about the anatomy of the ICA and finds its utility in the clinical practice [9]. Depending on the specialty of each physician, the use of one of these classifications is more useful. For example, anatomists and neurosurgeons often use the classification of Gibo et al. or the classification of Bouthillier et al., which are the two most "anatomical" classifications. The knowledge of the history and of the major classifications are important to have all together the same language in the definition of the ICA.

References

1. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
2. Lie TA. The congenital anomalies of the carotid arteries. Amsterdam/New York: Excerpta Medica Foundation; 1968.
3. Dilenge D. Bilateral agenesis of internal carotid artery. J Can Assoc Radiol. 1975;26(2):91–4.
4. Lasjaunias P, Berenstein A, ter Brugge KG. Surgical neuroangiography. Berlin: Springer Edition; 2001.
5. Tobenas-Dujardin AC, Duparc F, Ali N, Laquerriere A, Muller JM, Freger P. Embryology of the internal carotid artery dural crossing: apropos of a continuous series of 48 specimens. Surg Radiol Anat. 2005;27(6):495–501. https://doi.org/10.1007/s00276-005-0018-3.
6. Hills J, Sament S. Bilateral agenesis of the internal carotid artery associated with cardiac and other anomalies. Case report. Neurology. 1968;18(2):142–6. https://doi.org/10.1212/wnl.18.2.142.
7. Dungan DH, Heiserman JE. The carotid artery: embryology, normal anatomy, and physiology. Neuroimaging Clin N Am. 1996;6(4):789–99.
8. Legait E. The admirable carotid network. Biol Med (Paris). 1947;36(9–10):139–65.
9. Mele MV, Puigdellivol-Sanchez A, Mavar-Haramija M, Juanes-Mendez JA, Roman LS, De Notaris M, et al. Review of the main surgical and angiographic-oriented classifications of the course of the internal carotid artery through a novel interactive 3D model. Neurosurg Rev. 2018;43:473–82. https://doi.org/10.1007/s10143-018-1012-7.
10. Shapiro M, Becske T, Riina HA, Raz E, Zumofen D, Jafar JJ, et al. Toward an endovascular internal carotid artery classification system. AJNR Am J Neuroradiol. 2014;35(2):230–6. https://doi.org/10.3174/ajnr.A3666.
11. Abdulrauf SI, Ashour AM, Marvin E, Coppens J, Kang B, Hsieh TY, et al. Proposed clinical internal carotid artery classification system. J Craniovertebr Junction Spine. 2016;7(3):161–70. https://doi.org/10.4103/0974-8237.188412.
12. Lasjaunias P, Santoyo-Vazquez A. Segmental agenesis of the internal carotid artery: angiographic aspects with embryological discussion. Anat Clin. 1984;6(2):133–41. https://doi.org/10.1007/bf01773165.
13. Bouthillier A, van Loveren HR, Keller JT. Segments of the internal carotid artery: a new classification. Neurosurgery. 1996;38(3):425–32; discussion 32–3. https://doi.org/10.1097/00006123-199603000-00001.
14. Wang C, Xie J, Cui D, Cheng Y, Zhang S. A new classification of cavernous segment of the internal carotid artery. J Craniofac Surg. 2013;24(4):1418–22. https://doi.org/10.1097/SCS.0b013e318292c970.
15. Rhoton AL Jr. The supratentorial arteries. Neurosurgery. 2002;51(4 Suppl):S53–120.
16. Gailloud P, Clatterbuck RE, Fasel JH, Tamargo RJ, Murphy KJ. Segmental agenesis of the internal carotid artery distal to the posterior communicating artery leading to the definition of a new embryologic segment. AJNR Am J Neuroradiol. 2004;25(7):1189–93.
17. Krings T, Lasjaunias PL. Segmental agenesis of the internal carotid artery distal to the posterior communicating artery leading to the definition of a new embryologic segment. AJNR Am J Neuroradiol. 2006;2:246–7. author reply 7
18. Ziyal IM, Ozgen T, Sekhar LN, Ozcan OE, Cekirge S. Proposed classification of segments of the internal carotid artery: anatomical study with angiographical interpretation. Neurol Med Chir (Tokyo). 2005;45(4):184–90; discussion 90–1. https://doi.org/10.2176/nmc.45.184.

Branches and Variations of the Internal Carotid Artery

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The internal carotid artery (ICA) has the principal role to bring high-flow blood to a major part of the cerebral hemisphere. During the species evolution, the ICA takes always more importance [1–3]. Its secondary roles as dural, middle ear, and hypophysis supplies are less known [4, 5]. Even if the internal carotid artery is considered as a "stable" artery, with a very low rate of anatomical variations, some variants have been described in the literature [1, 6–8]. In this chapter, all anatomical variations encountered, and all branches of the ICA will be presented. Persistent carotido-vertebral arteries will be developed and explained in chapter "Carotido-Vertebral Anastomoses." Anatomical variations as ICA origin of the occipital or ascending pharyngeal arteries will also be presented in chapters "Embryology and Variations of the Occipital Artery" and "Embryology and Variations of the Ascending Pharyngeal Artery".

History

For more than 200 years, the internal carotid artery is one of the arteries that caught the most attention [4, 6, 9, 10]. In the beginning, scientists, anatomists, and also philosophers based their knowledge only on animal's dissection and only in a second time, on human cadaveric dissection [1, 10]. In the last 50 years, angiographic studies always gave a more detailed analysis of little branches of the internal carotid artery [8, 9, 11–14]. Among the physicians who dedicated their studies to this artery, we can mention Malacarne, Stattin, Parkinson, and Rhoton for their studies and cadaveric dissections about ICA branches [3, 8, 15–17]. As for all cranio-facial arteries, Congdon and Padget allowed to understand the embryologic basis of the ICA [2]. Altmann and Barry also furnished amazing works about the evolution of the aortic arches [1, 18]. Finally, Lasjaunias illuminated other physicians in the understanding of almost all anatomic variations of the ICA based on his embryologic knowledge [7, 19–23].

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Embryology

The embryological vascular development starts early in the embryological life. The primitive heart is already recognizable at approximately 19 days (embryos of 2 mm) and paired ventral and dorsal aortas are formed and already have a cranial continuity, forming the first cranial arch. From 21 to 32 days (embryos of 3 to 12 mm), the other aortic arches are successively formed from front to back [1, 2, 10]. At the same time, the dorsal aortas give a cranial extension to the first aortic arch. At 28 days (embryos of 4–5 mm), the first aortic arch initiates to involute, followed by the second aortic arc [1, 2, 18]. Consequently, the six pairs of aortic arches are not all present at the same time. Each aortic arch has a different function, the two first one involute completely, the third arch persists and will become a part of the internal carotid artery, the fourth one persists and will give the adult aortic arch on the left and the brachiocephalic artery on the right. The fifth aortic arch will completely involute and the sixth one will become the pulmonary arteries [1, 2].

Common Carotid Artery

The embryological origin of the common carotid artery is not clear. Altmann in 1947 already postulated two different hypotheses [1]. The first one is that the common carotid artery derivates from the ventral aorta between the third and fourth aortic arch and from the third aortic arch. The second hypothesis is that the common carotid artery derivates

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directly from the aortic sac a second time after the formation of the aortic arches [1]. In her monograph, Padget (1948) did not insist on the embryological formation of the common carotid artery and considered it as the development of the ventral aorta between the third and fourth aortic arches [2]. Lasjaunias (1984), based on the fact that a segmental agenesis of the cervical internal carotid artery never influences the formation of the common carotid artery, considered that the third aortic arch could not participate in the formation of the distal common carotid artery [22]. In conclusion, the strongest hypothesis about the formation of the common carotid artery is that it origins from the ventral aorta between the insertion of the third and fourth aortic arches which have an important extension after the involution of the different other arches as described before [5, 7, 20, 22, 24].

External Carotid Artery

Also, for the external carotid artery, different hypotheses were postulated to explain its embryological development but the most probable is its direct origin from the aortic sac [9]. The ventral pharyngeal artery (which is the principal precursor of the external carotid artery) extends cranio-laterally from the aortic sac as early as the 5–6 mm stage. The ventral pharyngeal artery annexes later (24 mm stage) the maxillomandibular branch of the stapedial artery to become the external carotid artery. At approximately 40 days, the descent of the aortic sac favorites the cranial migration of the external carotid artery origin along the common carotid artery to give the adult configuration [1, 2]. Lasjaunias strongly argued against a common embryological origin of internal and external carotid arteries, based on different types of segmental agenesis of the ICA always sparing the ECA development [7].

Internal Carotid Artery

Early in the development (first stage of Padget, embryos of 4–5 mm), the two first aortic arches initiate their natural regression allowing the internal carotid artery to be individualized [1, 2, 10, 18]. The embryological segments of the ICA are derived from the third aortic arch and from the dorsal aorta cranial to the third aortic arch. The dorsal aorta also regresses at the same time between the third and fourth aortic arches. In the 4–5 mm embryos, the primitive ICA bifurcation is already visible with its two divisions: anterior and posterior that will become in adult, the anterior cerebral artery and the posterior communicating artery [2]. The ICA, cranial to the posterior communicating origin, derivates from the anterior division of the ICA; and not from the ICA itself. Consequently, the primitive ICA bifurcation is considered to be at the origin of the posterior communicating artery and not at the origin of the middle cerebral artery.

Embryologically, we could divide the internal carotid artery into seven different segments according to Lasjaunias [22].

These segments could be individualized as follows:

1. The first one corresponds to the third aortic arch from the origin of the ventral pharyngeal artery (future external carotid artery) to the junction between the third aortic arch and the dorsal aorta. In the adult, it corresponds to the cervical internal carotid artery.
2. The second segment is the dorsal aorta between the third and second aortic arch. Its distal part is the hyoid artery in the embryo and the caroticotympanic artery in the adult (ascending intrapetrous segment).
3. The third segment is the dorsal aorta between the second and first aortic arch. It corresponds in the embryo to the segment between the hyoid and the mandibular arteries, and, in the adult, between caroticotympanic artery and the Vidian artery (horizontal intrapetrous segment).
4. The fourth segment is the dorsal aorta between the first aortic arch and the origin of the trigeminal artery (and the primitive maxillary artery). In the adult, it corresponds to the segment between the Vidian artery and the origin of the meningo-hypophysary trunk (ascending foramen lacerum segment).
5. The fifth segment is the dorsal aorta between the origin of the trigeminal artery (and the primitive maxillary artery) and the origin of the primitive dorsal ophthalmic artery (PDOA, future inferolateral trunk-ILT). It corresponds in the adult to the horizontal cavernous segment between the meningo-hypophysary and infero-lateral trunks.
6. The sixth segment is between the origin of the PDOA and the anastomotic point of the primitive ventral ophthalmic artery (PVOA) on the internal carotid artery. This segment in the adult is the clinoid segment between the origin of the ILT and the OA.
7. The seventh segment is between the anastomotic point of the PVOA and the primitive carotid bifurcation. In the adult, this is the supraclinoid ICA from the origin of the OA to the origin of the posterior communicating artery.

It is important to note that the carotid bulb has not the same embryological origin than the other segments of the ICA. It originates from the pharyngo-occipital system that easily explains variations in origin of the ascending pharyngeal and occipital arteries [7, 22].

Fenestration and Duplication

Fenestrations and duplications involving the internal carotid artery are extremely rare anatomical variations [25, 26]. A fenestration is defined as the presence of two different vessel lumens corresponding to the same vessel (same embryologic

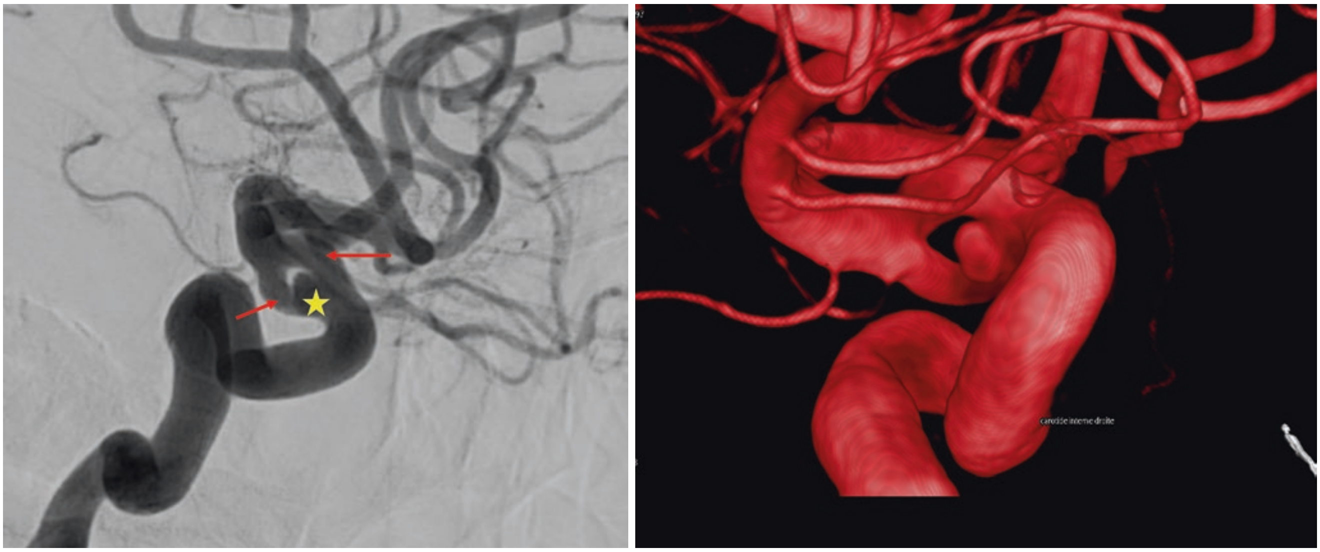

Fig. 1 Case of ICA fenestration associated with aneurysm. The lateral and 3D view of ICA DSA show a case of sovraclinoidal internal carotid artery fenestration (red arrows), associated with aneurysm formation on its proximal part (yellow star)

origin) [7]. On the other hand, a duplication is the presence of two different vessels from two different embryologic origins (persistence of an embryologic vessel) [7, 19]. Cases of duplication of the internal carotid artery are limited to its two first segments [19]. These cases correspond to the presence of an intra-tympanic flow of the ICA (aberrant ICA) associated with the persistence of the normal ICA [27]. Fenestrations could involve each segment of the ICA. The two most involved segments are the cervical and the supraclinoid segments [28]. Supraclinoid segment fenestration of the ICA is recognized as an anatomical predisposition for the formation of anterior circulation aneurysm, as shown in a case in Fig. 1 [29]. Isolated cases of fenestration involving petrous and cavernous segments of the ICA are also described [30].

Branches of the Internal Carotid Artery

The cervical segment of the ICA usually does not offer any branches. The possible branches originating from this segment are usually the result of a persistent embryonic artery or other embryological anomalies. The branches arising from the petrous, cavernous, and supraclinoid segment are listed below and illustrated in Fig. 2.

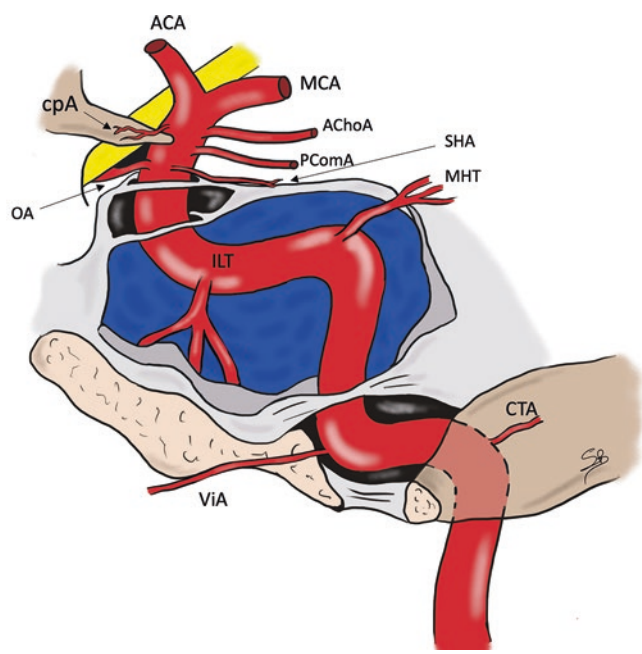

Fig. 2 Illustration of ICA branches. *CTA* carotico-tympanic artery, ViA vidian artery, *MHT* meningo-hypophyseal artery, *ILT* infero-lateral trunk, *OA* ophthalmic artery, *SHA* superior hypophyseal artery, *PComA* posterior communicating artery, *AChoA* anterior choroidal artery, *cpA* clinoid process artery, *ACA* anterior cerebral artery, *MCA* middle cerebral artery

Cervical Segment of the Internal Carotid Artery

Separate Origin ICA-ECA

The absence of the common carotid artery or separate origin of the external and internal carotid arteries is a rare anatomical variation with an incidence estimated inferior than 0.1% [15]. Only 87 cases have been described in the literature, among which almost all are unilateral (75 cases) and the majority is associated with a normal aortic arch (58% of cases) [15]. Lie (1968) proposed three different theories to explain this anatomical variation depending on the hypothesis on the development of the external carotid artery supported [31]. The first theory is the involution of the third aortic arch with persistence of the ductus caroticus (dorsal aorta between the third and fourth aortic arches). The second hypothesis is the failure of migration of the ECA origin which remains caudal to the third aortic arch. The third theory is the persistence of the ductus caroticus with involution of the distal part of the third aortic arch (its proximal part is the precursor of the ECA).

Aberrant ICA with Normal ICA Course

Aberrant ICA or intra-tympanic flow of the ICA is largely developed in the chapter "Embryology and Anatomy of the Internal Maxillary Artery." The agenesis of the first two segments of the ICA could be complete or partial [32]. In rare cases, the agenesis of the cervical and petrous ICA is only partial and consequently, the cervical and petrous ICA seems duplicated. The normal course of the ICA is generally hypoplastic in these cases [27].

Pharyngo-Tympano-Stapedial Artery

In this variant, the cervical ICA presents another branch, which is the MMA. This was first described by Lasjaunias (1977) in its original publication [5]. The same case served as illustration in the textbook "Surgical Angiography" and only one similar case was published by Baltsavias et al. (2012) [33]. The MMA arises from the cervical portion of the ICA, ascends along the cervical ICA, enters the tympanic cavity through the inferior tympanic canal, and follows the usual course of the stapedial artery. The two cases described were presented as "partial" persistence of the SA with only the MMA arising from the SA and the absence of the foramen spinosum. In this variant, an annexation of the SA by the inferior tympanic artery (branch of the ascending pharyngeal artery) with regression of the proximal part of the SA explains this vascular configuration. Therefore, the SA arises from the cervical instead of the petrous segment of the ICA [5, 7, 22, 24].

Petrous Segment of the Internal Carotid Artery

Vidian Artery

The vidian artery (ViA), also called the artery of the pterygoid canal, is an anastomotic vessel between the petrous internal carotid artery and the third segment of the internal maxillary artery [16]. The vidian artery is the remnant of the first aortic arch and courses in the pterygoid canal [34]. Its function is limited to the periosteal supply of the pterygoid canal and to the supply of the vidian nerve. The vidian artery is usually not seen on normal angiography but could be enlarged in case of internal carotid artery stenosis or tumoral process of the skull base [34]. Figure 3 shows a rare case of ViA visible on DSA.

Carotico-Tympanic Artery

The carotico-tympanic artery is a little artery arising from the ascending petrous segment of the ICA and corresponds to the carotid remnant of the stapedial artery (second aortic arch) [7]. Its supply is limited to a part of the middle ear. This artery can anastomose with the inferior tympanic artery and enlarge the inferior tympanic canal in case of intra-tympanic flow of the ICA [22].

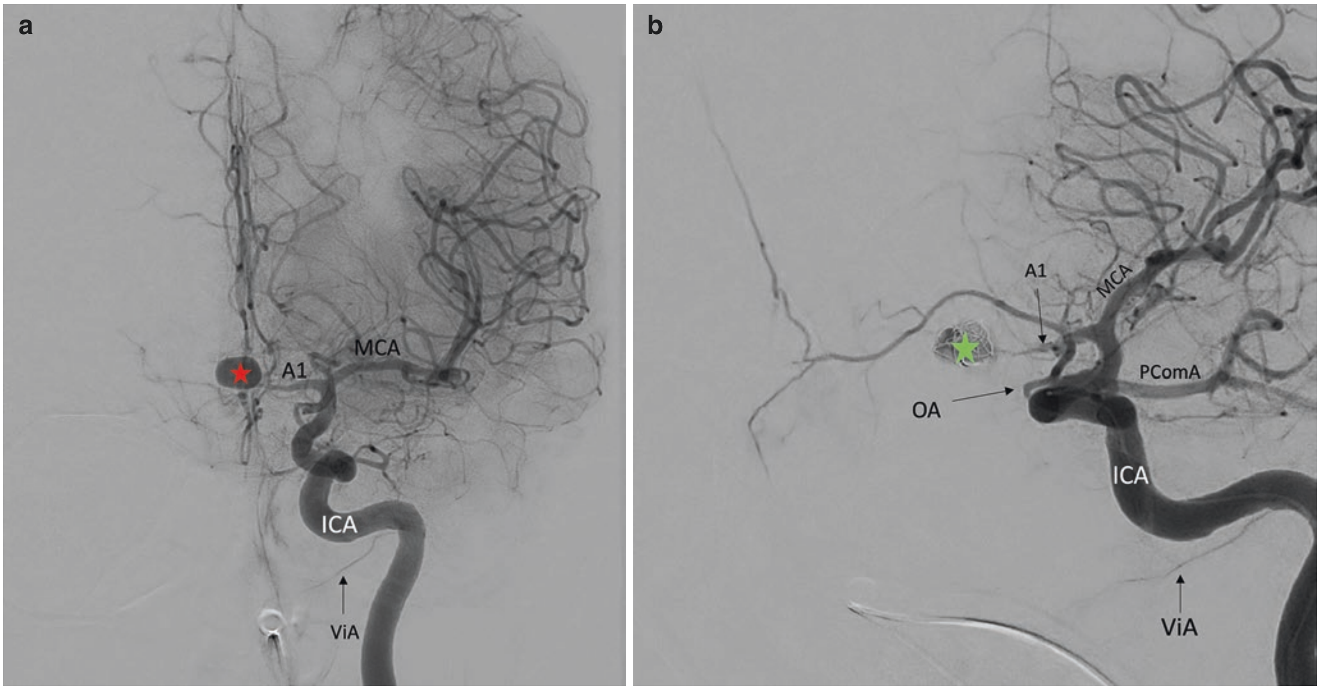

Fig. 3 DSA showing the vidian artery. The figure shows the case of a patient who arrived to our emergency department after unusual headache and the CT scan showed a subarachnoidal hemorrhage associated with multiple aneurysms. The image shows an antero-posterior (**a**) and lateral (**b**) DSA view after left internal carotid artery (ICA) injection. In Figure **a** and **b**, the first visible branch of the ICA is the vidian artery (ViA). It is also visible the ICA bifurcation into A1 and middle cerebral artery (MCA), and the ruptured aneurysm of the anterior communicating artery (red star). In Fig. B, the ViA is still visible, as well as the posterior communicating artery (PComa), the ICA bifurcation, and the aneurysm during the phase of coiling (green star)

Cavernous Segment of the Internal Carotid Artery

The cavernous segment of the ICA is very stable and usually it is not affected by major anatomic variations other than segmental agenesis, described in chapter “Segmental Agenesis of the Internal Carotid Artery.” This segment of the ICA presents two major branches, the meningo-hypophyseal and the infero-lateral trunks, that supply the dura of the central skull base [8, 16]. Their embryology, variations, and functions are well described in chapter “Dural Branches of the Internal Carotid Artery.” Figure 4 presents a case of dural arterio-venous fistulas supplied by these two ICA branches.

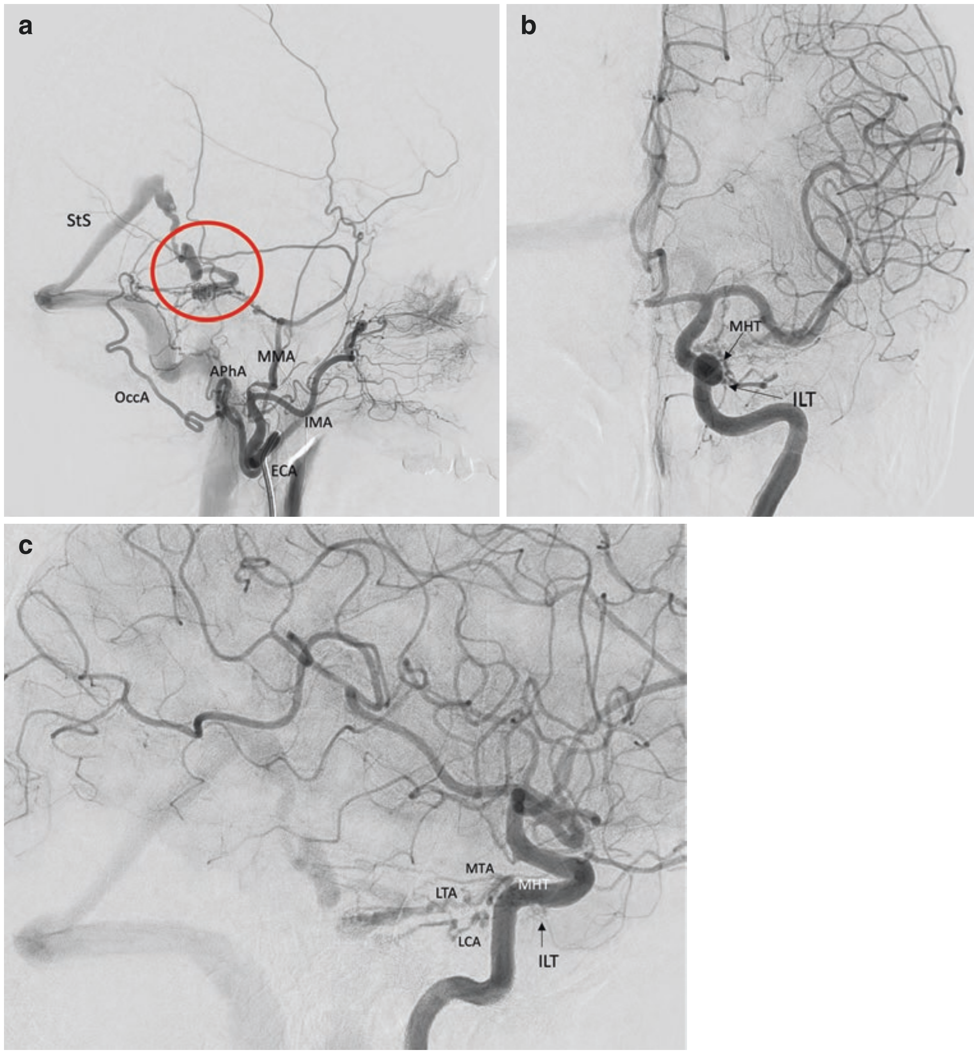

Fig. 4 Case of dural arterio-venous fistula supplied by branches of MHT. The meningo-hypophyseal trunk (MHT) and the infero-lateral trunk (ILT) are usually difficult to see in a normal DSA. However, they can be enlarged and consequently visible in case of dAVFs. Fig. **a** shows a case of dural artero-venous fistula (red circle) located at the petrous apex on the left side. The major feeders come from the external carotid artery (ECA), and especially from dural branches of the occipital artery (OccA), the ascendent pharyngeal artery (APhA), and the middle meningeal artery (MMA), whose origin from the internal maxillary artery (IMA) is also visible. The venous drainage is from the superior petrous vein, the transverse pontine vein and later into the Galen vein and the straight sinus (StS). Fig. **b** and **c** show respectively an antero-posterior and lateral view after left ICA injection. In both the projections, the MHT and ILT are visible. The MHT divides into the marginal tentorial artery (MTA), the lateral tentorial artery (LTA), and the lateral clival artery (LCA). The inferior hypophyseal artery is not visible. The ILT is visible at its origin from the internal carotid artery on its infero-lateral aspect

Supraclinoid Segment of the Internal Carotid Artery

Ophthalmic Artery

Embryologic development and anatomical variations of the ophthalmic artery are largely explained in chapter "Embryology and Variations of the Ophthalmic Artery," thus they will not be treated in this chapter.

Superior Hypophyseal Artery

The superior hypophyseal artery (SHA) is an artery or a group of arteries that arises from the medial wall of the supraclinoid portion of the ICA [11, 35]. The SHA could be composed from one to five arteries with a predilection for two distinct branches (anterior and posterior in 40% of cases) [35]. It supplies the anterior hypophysis, the pituitary stalk, and the optic nerve and chiasma [17, 36]. Four distinct branches are generally individualized: (1) recurrent optic, (2) chiasmatic, (3) infundibular, and (4) descending branch. The two last branches often present a rich anastomotic network with contralateral similar branches called the circuminfundibular anastomosis [35].

Perforators

The supraclinoid segment of the ICA is the origin of important perforator arteries that supply the optic nerve, chiasma and tract, the temporal uncus, the floor of the third ventricle, and the anterior perforating substance [17, 36]. These perforators arise from the ophthalmic and the choroidal segments of the artery, the communicating segment does not bear any perforating arteries in more than 60% of cases. These little branches often take their origin on the postero-medial wall of the ICA [37].

Posterior Communicating Artery

The posterior communicating artery is embryologically the caudal division of the primitive internal carotid artery. Its embryology, variations, and branches are exposed in chapter "Embryology and Anatomy of the Posterior Communicating Artery and Basilar Artery."

Anterior Choroidal Artery

The anterior choroidal artery has an important role in the embryologic development of brain hemispheres. This artery is largely exposed in chapter "Embryology and Variations of the Anterior Choroidal Artery."

Artery of the Anterior Clinoid Process

The artery of the anterior clinoid process is the only dural branch of the ICA that does not arise from its cavernous segment [38]. All details known about this artery are exposed in the chapter "Dural Branches of the Internal Carotid Artery."

Terminal Branches

The terminal branches are the anterior cerebral artery and the middle cerebral artery with different embryologic origin. Embryology and anatomic variations of these two arteries are respectively developed in chapters "Embryology, Anatomy, and Variations of the Anterior Cerebral Artery" and "Cortical and Perforating Branches of the Middle Cerebral Artery."

References

1. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
2. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–62.
3. Parkinson D. A surgical approach to the cavernous portion of the carotid artery. Anatomical studies and case report. J Neurosurg. 1965;23(5):474–83. https://doi.org/10.3171/jns.1965.23.5.0474.
4. Curnow J. Two instances of irregular ophthalmic and middle meningeal arteries. J Anat Physiol. 1873;8(Pt 1):155–6.
5. Lasjaunias P, Moret J, Manelfe C, Theron J, Hasso T, Seeger J. Arterial anomalies at the base of the skull. Neuroradiology. 1977;13(5):267–72.
6. Fisher AG. A case of complete absence of both internal carotid arteries, with a preliminary note on the developmental history of the Stapedial artery. J Anat Physiol. 1913;48(Pt 1):37–46.
7. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer edition; 2001.
8. Parkinson D. Collateral circulation of cavernous carotid artery: anatomy. Can J Surg. 1964;7:251–68.
9. Tode. Medizinisch-chirurgische Bibliothek. Copenhagen: 1787.
10. Legait E. Le réseau admirable carotidien. Biol Med (Paris). 1947;36(9–10):139–65.
11. McConnell EM. The arterial blood supply of the human hypophysis cerebri. Anat Rec. 1953;115(2):175–203. https://doi.org/10.1002/ar.1091150204.
12. Cortes O, Chase NE, Leeds N. Visualization of tentorial branches of the internal carotid artery in intracranial lesions other than Meningiomas. Radiology. 1964;82:1024–8. https://doi.org/10.1148/82.6.1024.
13. Manelfe C, Tremoulet M, Roulleau J. Arteriographic study of the intracavernous branches of the internal carotid. Neurochirurgie. 1972;18(7):581–98.
14. Manelfe C, Tremoulet M, Roulleau J. Intracavernous collaterals of the internal carotid artery. 2. Angiographic study. Ann Radiol (Paris). 1974;17(3):268–70.
15. Vasovic L, Trandafilovic M, Vlajkovic S. Congenital aplasia of the common carotid artery: a comprehensive review. Biomed Res Int. 2019;2019:9896138. https://doi.org/10.1155/2019/9896138.
16. Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A Jr. Microsurgical anatomy of the dural arteries. Neurosurgery. 2005;56(2 Suppl):211–51; discussion −51. https://doi.org/10.1227/01.neu.0000144823.94402.3d.
17. Rhoton AL Jr. The supratentorial arteries. Neurosurgery. 2002;51(4 Suppl):S53–120.
18. Barry A. The aortic arch derivatives in human adult. Anat Rec. 1951;111(2):221–38.
19. Chng SM, Alvarez H, Marsot-Dupuch K, Mercier P, Lasjaunias P. "Duplicated" or "multiple" cervical internal carotid and vertebral arteries from fenestration, duplication and vasa vasorum to segmental rete. Interv Neuroradiol. 2004;10(4):301–7. https://doi.org/10.1177/159101990401000403.
20. Lasjaunias P, Moret J, Doyon D, Vignaud J. C5 collaterals of the internal carotid siphon: embryology, angiographic anatomi-

cal correlations, pathological radio-anatomy (author's transl). Neuroradiology. 1978;16:304–5. https://doi.org/10.1007/bf00395282.
21. Lasjaunias P, Moret J, Mink J. The anatomy of the inferolateral trunk (ILT) of the internal carotid artery. Neuroradiology. 1977;13(4):215–20. https://doi.org/10.1007/bf00344216.
22. Lasjaunias P, Santoyo-Vazquez A. Segmental agenesis of the internal carotid artery: angiographic aspects with embryological discussion. Anat Clin. 1984;6(2):133–41.
23. Willinsky R, Lasjaunias P, Berenstein A. Intracavernous branches of the internal carotid artery (ICA). Comprehensive review of their variations. Surg Radiol Anat. 1987;9(3):201–15.
24. Lasjaunias P, Moret J. Normal and non-pathological variations in the angiographic aspects of the arteries of the middle ear. Neuroradiology. 1978;15(4):213–9.
25. Banach MJ, Flamm ES. Supraclinoid internal carotid artery fenestration with an associated aneurysm. Case report. J Neurosurg. 1993;79(3):438–41. https://doi.org/10.3171/jns.1993.79.3.0438.
26. Sgreccia A, Coskun O, Di Maria F, Rodesch G, Consoli A. Fenestration of the supraclinoid segment of the ICA and associated aneurysms: a case report with literature review. Acta Neurochir. 2018;160(6):1143–7. https://doi.org/10.1007/s00701-018-3551-7.
27. Koenigsberg RA, Zito JL, Patel M, Swartz JD, Goldofsky E, Zahtz G. Fenestration of the internal carotid artery: a rare mass of the hypotympanum associated with persistence of the stapedial artery. AJNR Am J Neuroradiol. 1995;16(4 Suppl):908–10.
28. Dey M, Awad IA. Fenestration of supraclinoid internal carotid artery and associated aneurysm: embryogenesis, recognition, and management. World Neurosurg. 2011;76(6):592 e1–5. https://doi.org/10.1016/j.wneu.2011.04.019.
29. Jha N, Crockett MT, Singh TP. Unusual right internal carotid artery supraclinoid segment fenestration associated with multiple aneurysms treated with flow diversion and coiling. BMJ Case Rep. 2018;2018 https://doi.org/10.1136/bcr-2018-227020.
30. Uchino A, Saito N, Kurita H, Ishihara S. Persistent trigeminal artery arising from the arterial ring/fenestration of the cavernous segment of the internal carotid artery. Surg Radiol Anat. 2012;34(7):651–4. https://doi.org/10.1007/s00276-011-0927-2.
31. Lie TA. The congenital anomalies of the carotid arteries. Amsterdam/New York: Excerpta Medica Foundation; 1968.
32. Aladwan A, Mack M, Gstottner W, Vogl TJ. Duplication of internal carotid artery: a rare case of tympanic mass. Eur Radiol. 2005;15(12):2525–7. https://doi.org/10.1007/s00330-004-2507-x.
33. Baltsavias G, Kumar R, Valavanis A. The pharyngo-tympano-stapedial variant of the middle meningeal artery. A case report. Interv Neuroradiol. 2012;18(3):255–8. https://doi.org/10.1177/159101991201800302.
34. Osawa S, Rhoton AL Jr, Seker A, Shimizu S, Fujii K, Kassam AB. Microsurgical and endoscopic anatomy of the vidian canal. Neurosurgery. 2009;64(5 Suppl 2):385–411; discussion −2. https://doi.org/10.1227/01.NEU.0000338945.54863.D9.
35. Doglietto F, Prevedello DM, Belotti F, Ferrari M, Lancini D, Schreiber A, et al. The superior hypophyseal arteries: anatomical study with an endoscopic endonasal perspective. Oper Neurosurg (Hagerstown). 2019;17(3):321–31. https://doi.org/10.1093/ons/opy393.
36. Joo W, Funaki T, Yoshioka F, Rhoton AL Jr. Microsurgical anatomy of the carotid cave. Neurosurgery. 2012;70(2 Suppl Operative):300–11; discussion 11-2. https://doi.org/10.1227/NEU.0b013e3182431767.
37. Marinkovic SV, Milisavljevic MM, Marinkovic ZD. The perforating branches of the internal carotid artery: the microsurgical anatomy of their extracerebral segments. Neurosurgery. 1990;26(3):472–8.; discussion 8-9. https://doi.org/10.1097/00006123-199003000-00015.
38. Salunke P, Singh A, Rekhapalli R. Dural artery from supraclinoid internal carotid artery to anterior clinoid process: origin, course, and clinical implications. World Neurosurg. 2018; https://doi.org/10.1016/j.wneu.2018.12.071.

Segmental Agenesis of the Internal Carotid Artery

Thomas Robert, Sara Bonasia, E. Ventura, S. Smajda, and Michel W. Bojanowski

Segmental agenesis of the internal carotid artery (ICA) is a very rare anatomical variation [1–3]. Even if it is difficult to give a realistic frequency of this variation, its incidence is evaluated to be lesser than 0.01% by some authors [4–6]. Larger series and reviews of the literature did not highlight a gender predominance for a segmental agenesis of the ICA [5]. This anatomical variation could be unilateral or bilateral; when unilateral, it concerns more frequently the left side, with a ratio of right side/left side/bilateral described as 1:3:1 [5, 7, 8]. In 1968, Lie gave the different definitions of agenesis (non-formation of the artery), aplasia (complete regression after normal formation of the artery), and hypoplasia (incomplete regression after normal formation of the artery) [9]. Even if most authors cited this concept, we do not consider it as an important concept to understand segmental agenesis of the ICA. Consequently, it will not be used in this chapter. After a summary of the history and embryology of the ICA, we will explain the different types of segmental agenesis of the ICA classifying them in a more comprehensive classification.

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History

The first case of internal carotid artery agenesis was described by Tode in 1787 [10]. He already noted during this cadaver dissection the absence of the carotid canal in the skull base. After this inaugural case, we have to wait for the beginning of the twentieth century and another cadaveric dissection published by Fisher (1913) that presented an agenesis of the ICA [11]. Since this case, a crescent number of publications about segmental agenesis of the ICA could be found and almost all of them are isolated case reports [8, 12–14]. The embryology of the carotid artery was described by three major works written by Padget (1946), [15] Altmann (1947) [16], and Barry (1951) [17]. The first description of angiographic features of agenesis of the ICA was proposed by Verbiest (1954) [18]. Lie (1968) proposed the most used 6-type classification based on the different cases he had seen in its clinical practice [9]. Between the 1970s and the 1990s, P.Lasjaunias gave an important contribution in the understanding of collateral circulations in case of segmental agenesis of the ICA [19–21]. First of all, he proposed an embryological classification of the ICA in 1984 that will be presented below [21]. He then tried to explain different possible collateral circulations in case of segmental agenesis of the ICA. He had already understood that the flow compensation could be from the circle of Willis, by persistence of an embryonic artery or by skull base collaterals (rete mirabile) [22]. In another publication, he classified the segmental agenesis of the ICA into 4 different types: the aberrant flow of the ICA into the tympanic cavity, a cervico-petrous agenesis with compensation by a persistent trigeminal artery, a cavernous agenesis with compensation by the circle of Willis, and a caverno-petro-cervical agenesis [22, 23]. All these different theories and classifications make this anatomical variation difficult to understand.

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Embryology

The understanding of the segmental agenesis of the ICA necessitates a detailed knowledge of the embryology of the internal carotid system and also of its embryological branches. In this chapter, after a brief definition of general concepts, we will describe the embryology of the carotid system and we will insist on the embryological phases when each carotid branch appears and disappears to understand possible flow-rerouting in case of segmental agenesis of the ICA.

General Concepts

The ICA has been divided into seven distinct segments by Lasjaunias, depending on its embryological branches. According to Lasjaunias, the agenesis of one of the seven segments of the ICA implicates the non-formation of all proximal segments [21, 22]. This concept is however not always true, and the formation of proximal ICA segments depends principally on the presence of an embryological artery at the moment of the agenesis/dysgenesis, that can maintain the flow into the proximal segments of the ICA. Moreover, each segment is considered to have an individual trigger for its own vasculogenesis [22].

From an embryological point of view, the carotid bulb does not derivate from the same origin than the proximal part of the internal carotid artery (third aortic arch) but derivates from the pharyngo-occipital system. This easily explains variations in origin of the ascending pharyngeal and occipital arteries that could arise from the internal carotid artery [20, 22, 24].

The primitive internal carotid artery has its terminal bifurcation in cranial and caudal divisions, which respectively correspond in adults to the anterior cerebral artery (ACA) and posterior communicating artery (PComA) [15, 16]. Consequently, the ICA distal to the PComA has a different embryological origin, since it derivates from the cranial division of the ICA, and not from the primitive ICA itself. Thus, the primitive ICA bifurcation is considered to be at the origin of the PComA and not at the origin of the middle cerebral artery (MCA) [17]. For this reason, anatomic variants of the segment distal to the PComA are not considered in this chapter.

Internal Carotid Artery

Early in the development (first stage of Padget, embryos of 4–5 mm), the two first aortic arches initiate their natural regression allowing the internal carotid artery to be individualized [15, 16]. The embryological segments of the ICA are derived from the third aortic arch and from the dorsal aorta cranial to the third aortic arch. The dorsal aorta also regresses in the same time between the third and fourth aortic arches [17]. In the 4–5 mm embryos, the primitive ICA bifurcation is already visible with its two divisions: cranial and caudal that will become in adult, the anterior cerebral artery and the posterior communicating artery [15].

Embryologically, we could divide the internal carotid artery into seven different segments according to Lasjaunias [21]. Figure 1 illustrates the seven embryological segments of the ICA with their respective development in the adult ICA.

These segments could be individualized as follows:

1. The first one corresponds to the third aortic arch from the origin of the ventral pharyngeal artery (future external carotid artery) to the junction between the third aortic arch and the dorsal aorta. In the adult, it corresponds to the cervical internal carotid artery.
2. The second segment is the dorsal aorta between the third and second aortic arches. Its distal part is the origin of the hyoid artery in the embryo and of the carotico-tympanic artery in the adult (ascending intrapetrous segment).
3. The third segment is the dorsal aorta between the second and first aortic arches. It corresponds in the embryo to the segment between the origins of the hyoid and mandibular arteries, and, in the adult, between the origin of the carotico-tympanic artery and Vidian artery (horizontal intrapetrous segment).
4. The fourth segment is the dorsal aorta between the first aortic arch and the origin of the trigeminal artery (and the primitive maxillary artery). In the adult, it corresponds to the segment between the origin of the Vidian artery and the origin of the meningo-hypophyseal trunk (ascending foramen lacerum segment).
5. The fifth segment is the dorsal aorta between the origin of the trigeminal artery (and the primitive maxillary artery) and the origin of the primitive dorsal ophthalmic artery (PDOA, future inferolateral trunk-ILT). It corresponds in the adult to the horizontal cavernous segment between the meningo-hypophyseal and infero-lateral trunks.
6. The sixth segment is between the origin of the PDOA and the anastomotic point of the primitive ventral ophthalmic artery (PVOA) on the internal carotid artery. This segment in the adult is the clinoidal segment between the origin of the ILT and the OA.
7. The seventh segment is between the anastomotic point of the PVOA and the primitive carotid bifurcation and the bifurcation of the ICA. In the adult, this is the supraclinoid ICA from the origin of the OA to the origin of the posterior communicating artery.

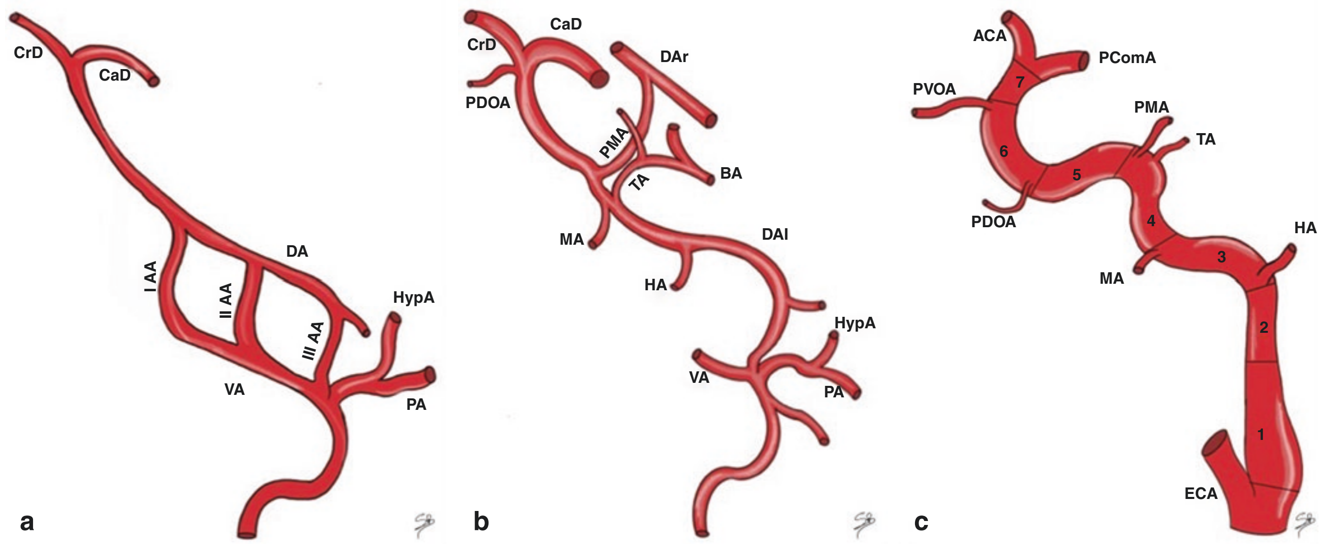


Fig. 1 Embryology and embryological segments of the internal carotid artery. The illustrations **a**, **b**, and **c** show consecutive stages of ICA embryological development. The first stages of development (**a**) are characterized by the presence of three aortic arches that link the ventral and dorsal aorta (VA, DA). The VA regresses together with the ventral part of the aortic arches. The dorsal remnants of the aortic arches persist as embryonic arteries. These embryonic arteries divide the ICA into 7 embryological segments [21]: (*1*) The cervical segment: It derives from the remnant of the third aortic arch (III AA). (*2*) The ascending intrapetrous segment: It is the remnant of the dorsal aorta (DA) between the second (II AA) and third aortic arches. The division point between the segment 2 and 3 is at the point of origin of the Hyoid artery (HA), that is the dorsal remnant of the second aortic arch. (*3*) The horizontal intrapetrous segment: It is the remnant of the DA between the first (I AA) and second aortic arches. The division point is at the point of origin of the mandibular artery (MA) that corresponds to the dorsal remnant of the first aortic arch. (*4*) The intracavernous ascending segment: It origins from the DA between the first aortic arch and the primitive maxillary artery (PMA), that connects the DA of the two sides (DAl: dorsal aorta left; DAr: dorsal aorta right). At the junction between segments 4 and 5 also the trigeminal artery (TA) takes its origin. This latter represents a primitive connection between the cavernous ICA and the basilar artery (BA). (*5*) The horizontal intracavernous segment: It derives from the DA between the PMA and the primitive dorsal ophthalmic artery (PDOA). (*6*) The clinoid segment: corresponds to the DA between the PDOA and the primitive ventral ophthalmic artery (PVOA). (*7*) The terminal segment: the terminal ICA between (PVOA) and the primitive ICA bifurcation into the future anterior cerebral artery (ACA) and future posterior communicating artery (PComA). The figure also shows the hypoglossal artery (HypA) and the proatlantal artery (PA) that originate proximal to the third aortic arch and will contribute to the formation of external carotid artery (ECA) branches

External Carotid Artery

For the external carotid artery (ECA) development, different hypotheses were postulated, even if the most probable is considered to be its direct origin from the aortic sac [16, 22]. The ventral pharyngeal artery (VPhA), the principal precursor of the ECA, develops cranio-laterally from the aortic sac as early as the 5–6 mm stage, to give almost all the branches of the future ECA [15]. The ventral pharyngeal artery annexes later (24 mm stage) the maxillomandibular branch of the stapedial artery to become the external carotid artery. At approximately 40 days, the descent of the aortic sac favorites the cranial migration of the external carotid artery origin along the common carotid artery to give its adult configuration. Lasjaunias strongly argued against a common embryological origin of ICA and ECA, since different types of segmental agenesis of the ICA always spared the ECA development [20, 22].

Internal Carotid Artery Branches

In case of segmental agenesis of the ICA, the rerouting of the vascular flow, and consequently, the type of compensation in adult, depends on two factors:

- The segment interested by the vascular insult.
- Which carotid branches are already developed when the vascular insult happens.

The knowledge of the embryological phases of development and regression for each ICA branch is consequently crucial to understand the possibility of rerouting of the carotid agenesis. Table 1 summarizes phases of development and regression of each involved artery.

1. The dorsal aorta between the third and fourth aortic arches is already present before the formation of the ICA

Table 1 Times of appearance and regression of embryonic arteries

Artery	Appearance (Embryo size)	Regression (Embryo size)
Hyoid artery	3 mm	16–18 mm
Primitive maxillary artery	2 mm	4–5 mm
Trigeminal artery	4 mm	6 mm
PDOA	4 mm	ILT
PVOA	7 mm	OA

and completely regresses at the 12–14 mm phase (stage IV of Padget) [15].

2. The hyoid artery, which is the dorsal remnant of the second aortic arch, initiates its development at the 3 mm phase and regresses at the 16–18 mm phase (stage V of Padget) [15].
3. The primitive maxillary artery, remnant of the first aortic arch is already visible at the early phase 2 mm and regresses at the 4–5 mm phase (stage I of Padget) [22].
4. The trigeminal artery is an ephemeral carotido-basilar anastomosis which is normally presents between stages I (4–5 mm) and II (6 mm) of Padget [16].
5. The primitive dorsal ophthalmic artery (PDOA) also appears early at the first stage of Padget (4–5 mm) and does not regress but becomes the remnant of the inferolateral trunk [15].
6. The primitive ventral ophthalmic artery (PVOA) appears only at the stage III of Padget (7–12 mm) and does not regress because it becomes the definitive ophthalmic artery after its second anastomosis with the internal carotid artery [15].

Formation of the Circle of Willis

Early in the embryological development, the caudal division of the internal carotid artery develops primitive branches that are precursors of the posterior choroidal arteries and of the posterior cerebral artery [15]. At the stage III of Padget (7–12 mm), the posterior division of the ICA is anastomosed with the distal part of the basilar artery and becomes the PComA. At the stage VI of Padget (24 mm), the development of the anterior communicating artery (AComA) marks the complete formation of the circle of Willis [15].

Classifications Already Described

The first author who proposed a classification for ICA agenesis was Lie in 1968, who described 6 types of ICA agenesis, which are summarized in Table 2 [9]. Lie included cases of unilateral and bilateral ICA agenesis [9]. Even if this classification is the clearest one, it is not based on a comprehensive vascular embryology to explain the vascular insult. Moreover, some cases, as for example a compensation by a persistent TA, or the aberrant flow of the ICA, cannot be included. Another weak point of this classification is the differentiation between agenesis (absence of development of one artery) and hypoplasia (incomplete regression of a formed artery) for bilateral ICA agenesis (types C and E), which does not bring an important message from an embryological point of view.

Table 2 Classification proposed by Lie for internal carotid artery agenesis (Adapted from 9. Lie TA. The congenital anomalies of the carotid arteries. Amsterdam-New York: Excerpta Medica Foundation; 1968)

Type	Description
A	Unilateral ICA agenesis MCA supplied by PcomA ACA supplied by AcomA No A1 segment
B	Unilateral ICA agenesis ACA and MCA supplied by AcomA No PComA
C	Bilateral ICA agenesis ACAs and MCAs supplied by bilateral PComA
D	Unilateral ICA agenesis ACA supplied by AcomA MCA supplied by an inter-cavernous anastomosis
E	Bilateral ICA hypoplasia MCAs supplied by bilateral PcomA ACAs supplied by hypoplastic ICAs
F	Uni or bilateral ICA agenesis Collaterals from the ECA Rete mirabile

Tsurata and Miyazaki in 1977 [25] proposed their classification, which is summarized in Table 3. Since it is limited to unilateral ICA agenesis compensated by a competent circle of Willis, it does not allow to include all cases described in the literature. Moreover, it does not offer an embryological explication for each variant. On the other hand, it helps to differentiate the risk of occurrence of AComA aneurysm depending on the type of flow compensation.

Jamous et al., in 2007, proposed a great classification for unilateral ICA agenesis, which is summarized in Table 4 [26]. The authors based their classification on four cases described and on a review of the literature. They differentiated cases of ICA agenesis with compensation from the circle of Willis and by a primitive vessel. In this classification, the type III "ECA compensation without rete mirabile" is difficult to understand because it does not exist and is very similar to type Ia "ECA compensation with rete mirabile". This classification is also specific to unilateral ICA agenesis and does not consider the embryological segment of the ICA involved.

Table 3 Classification proposed by Tsurata and Miyazaki limited to unilateral ICA agenesis with circle of Willis compensation (Adapted from 25. Tsuruta J, Miyazaki Y. [A case of complete absence of the left internal carotid artery associated with an aneurysm of the anterior communicating artery (author's transl)]. No Shinkei Geka. 1977;5(8):895–900)

Type	Description
I	Unilateral ICA agenesis MCA supplied by PcomA ACA supplied by AcomA No A1 segment
II	Unilateral ICA agenesis ACA and MCA supplied by PComA AcomA not competent or absent
III	Unilateral ICA agenesis ACA and MCA supplied by AcomA No PComA

Table 4 Classification of Jamous et al. for unilateral ICA agenesis. Adapted from 26. Jamous MA, Abdel-Aziz H, Kaisy F. Collateral blood flow patterns in patients with unilateral ICA agenesis and cerebral aneurysm. Neuro Endocrinol Lett. 2007;28(5):647–51.

Type	Sub-type	Description
I	a	Unilateral ICA agenesis Collaterals from the ECA Rete mirabile
	b	Unilateral ICA agenesis ACA and MCA supplied by a persistent trigeminal artery No PComA
	c	Unilateral ICA agenesis ACA supplied by AcomA MCA supplied by an inter-cavernous anastomosis
II	a	Unilateral ICA agenesis MCA supplied by PcomA ACA supplied by AcomA No A1 segment
	b	Unilateral ICA agenesis ACA and MCA supplied by AcomA No PComA
	c	Unilateral ICA agenesis ACA and MCA supplied by AcomA and PComA
III		Unilateral ICA agenesis Collaterals from the ECA

Proposition of a New Comprehensive Classification

The classifications previously described do not consider two important factors involved in this pathology: the embryological segment involved by the vascular insult and the stage when the insult happens [9, 25, 26]. For this reason, we propose a new classification of ICA agenesis based on these two factors that allows to understand the embryology of this anatomical variation and to include all cases of literature described until now. This classification is summarized and illustrated in Table 5.

Type I: Compensation by the Circle of Willis

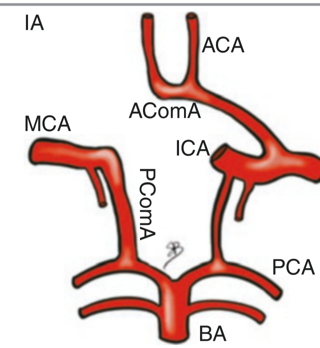
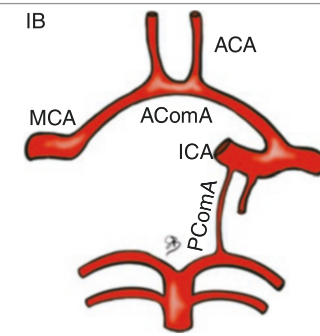
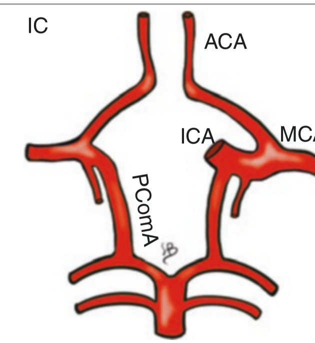
In most cases of ICA agenesis described in the literature, the flow compensation to the cerebral arteries is provided by the contralateral ICA and the basilar artery through the circle of Willis.

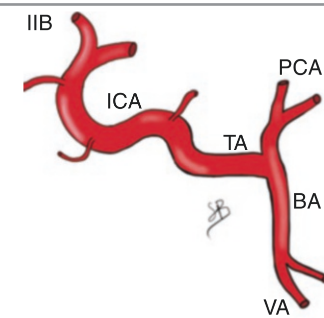
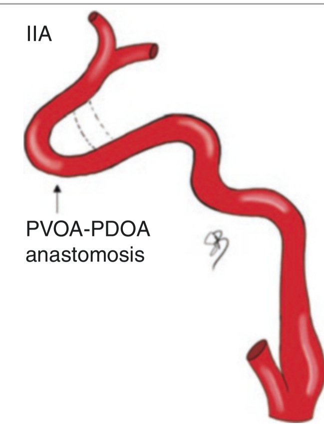
This type of compensation is only possible if the vascular insult happens after the connection of the caudal division of the ICA with the posterior circulation and consequently, after the formation of the PComA (stage III of Padget, 7–12 mm). In case of participation of the AComA, the vascular insult happens after the complete formation of the circle of Willis (Stage VI, 24 mm) [15]. In all these cases described in the literature, the agenesis involves the last segment of the ICA (VIIth segment) with proximal regression of other segments [15]. We could identify three different types of flow compensation as follows:

1. **Type IA:** This is the most frequent type which corresponds to the type A of Lie [9]. The MCA is perfused by an enlarged PComA and the ACA is perfused by an enlarged AComA. The A1 segment is always absent. Lie called this type "fetal type" since the compensation is provided by a fetal variant of the PComA [9]. From an embryological point of view, the insult happens immediately below the bifurcation of the primitive ICA. Thus, the caudal division of the primitive ICA (future PComA) will compensate the flow of the cranial division (from which the MCA origins), thanks to the already-formed connection with the BA.

   These cases are always unilateral and are more than 75 in the literature [27–56]. The pathology most frequently encountered in association with this case of ICA agenesis is an AComA aneurysm (in approximately 20% of cases) [10, 57–63]. An aneurysm located on another artery of the circle of Willis was discovered on the homolateral posterior cerebral artery (PCA, 2 cases) [34, 44], on the contralateral ICA (2 cases) [35, 64], or the contralateral MCA (1 case) [31]. Few authors had also observed other associated vascular variations as an infra-optic course of the ACA (3 cases) [28, 65, 66] or as an ophthalmic artery arising from the contralateral ICA (1 case) [67]. Figure 2 shows a case of this type of ICA agenesis, with the ophthalmic artery arising from the MCA.
2. **Type IB:** This type of ICA agenesis with flow compensation by the circle of Willis corresponds to type B of Lie [9]. Both MCA and ACA are supplied by the contralateral ICA through an enlarged AComA. The PComA is not visible, the reason why this type was called the "adult type" by Lie. This type is very rare with only 9 cases described in the literature, including one case of type II neurofibromatosis [6, 67–74].

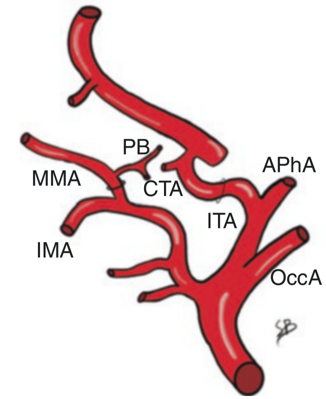
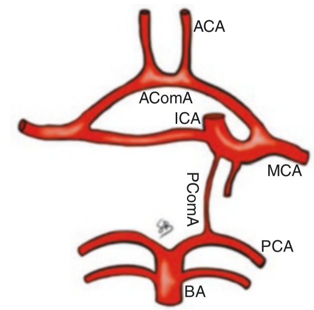
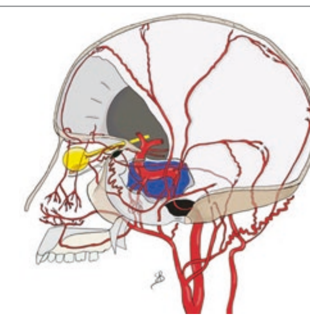
Table 5 Classification proposed by the authors

Type	Subtype	Stage	Segment insulted	Description	
I Circle of Willis	A	After formation circle of Willis 24 mm	VII with proximal regression	Unilateral ICA agenesis MCA supplied by PcomA ACA supplied by AcomA No A1 segment	
	B	After formation circle of Willis After 24 mm	VII with proximal regression	Unilateral ICA agenesis ACA and MCA supplied by AcomA No PComA	
	C	After formation PcomA 12 mm Before formation AcomA 24 mm	VII with proximal regression	Uni or bilateral ICA agenesis ACAs and MCAs supplied by uni/bilateral PComA	

Type	Subtype	Stage	Segment insulted	Description	
II Non-regression of embryonic artery	A	Before regression trigeminal artery	IV with proximal regression	Unilateral ICA agenesis ACA and MCA supplied by persistent trigeminal artery No PComA	
	B	Before regression PDOA	VI without proximal regression	Unilateral ICA agenesis Intra-orbital flow of the ICA Connection between PDOA and PVOA	

(continued)

Table 5 (continued)

Type	Subtype	Stage	Segment insulted	Description	
III Arterio-arterial anastomosis	A	Before regression of the stapedial artery	II with proximal regression	Uni or bilateral ICA agenesis Intra-tympanic flow of the ICA Anastomosis between inferior tympanic artery and caroticotympanic artery	
	B	After regression trigeminal artery	IV or V with proximal regression	Unilateral ICA agenesis ACA supplied by AcomA MCA supplied by an inter-cavernous anastomosis	
IV Rete mirabile		Before regression of the two first aortic arches	V and VI without proximal regression	Uni or bilateral ICA agenesis Collaterals from the ECA Rete mirabile	

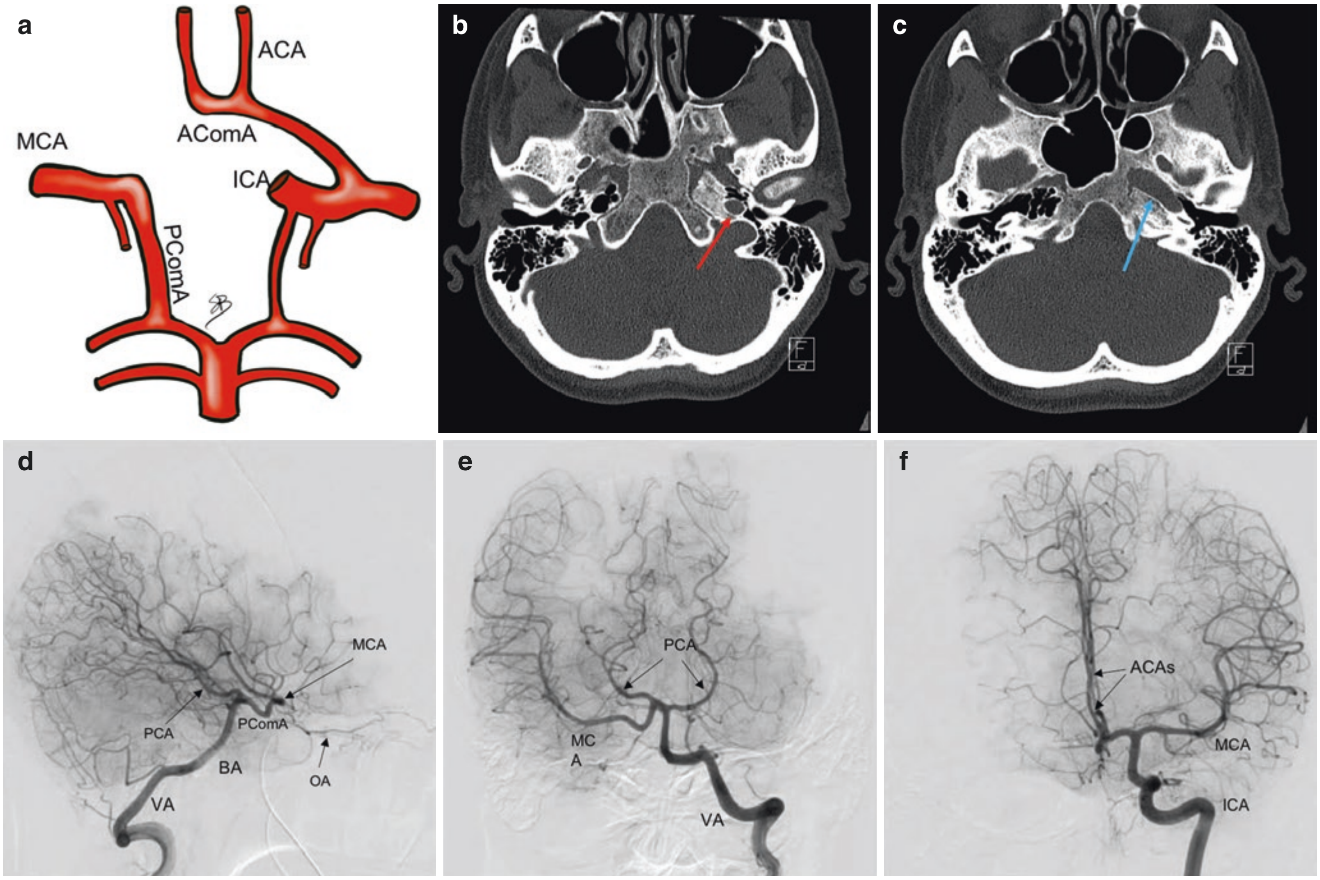


Fig. 2 Clinical case of type IA of ICA agenesis, with the ophthalmic artery origin from the MCA. In this type of compensation (**a**), the circle of Willis supplies the MCA through an enlarged PComA and the ACA through an enlarged AComA. In this case the ICA is absent on the right side. Consequently, the carotid foramen (red arrow, figure **b**) and canal (blue arrow, figure **c**) are visible on the left side but not on the side of the agenesis. After a left vertebral artery injection (figure **d**), the BA and the PCA are visible, together with the right MCA that is refilled through a fetal PComA. In absence of the right ICA, the homolateral OA arises from the MCA. The left ICA injection shows the homolateral MCA and both the ACAs, with compensation through an enlarged AComA (figure **e**)

3. **Type IC:** In this subtype, both MCA and ACA are supplied by an enlarged PComA, the AComA is not enough competent to participate in the flow compensation of the ACA. This type can be unilateral or bilateral (with bilateral enlarged PComA) and can be complete or incomplete. This subtype includes types C and E of Lie and also few cases of unilateral ICA agenesis that were erroneously classified as type B of Lie [9]. A total of 33 cases are described in the literature corresponding to this type of flow compensation [75–94]. Among these 33 cases, the majority (29 cases) concern a bilateral ICA agenesis and only 4 cases are unilateral [70]. The presence of a basilar tip aneurysm was noted in 3 cases and a dolico-ectasic BA in 1 another case [79, 87, 88]. Rare cases of associated pathologies to this subtype of ICA agenesis were described: 2 cases of cerebral ischemic event, 1 case of tongue hemangioma, and 1 case of psychomotor retard. A clinical case of type IC is shown in Fig. 3.

Type II: Compensation by the Non-regression of an Embryological Artery

In rare cases, if the segment involved by the agenesis is proximal to an embryological artery, the persistence of this artery allows the compensation. The vascular insult must occur before the natural regression of the embryonic artery. In the literature, we found two different types of ICA agenesis with flow compensation by the persistence of an embryonic artery.

1. **Type IIA:** This subtype of ICA agenesis concerns the IVth segment of the ICA with consequent proximal regression of other segments. The vascular insult happens before the regression of the trigeminal artery (TA) (stage II, 6 mm), and the loss of distal flow into the ICA provokes the reversal of flow into the TA [15–17]. This embryonic artery does not regress and participates in the supply of the ICA territory. The persistence of the TA also

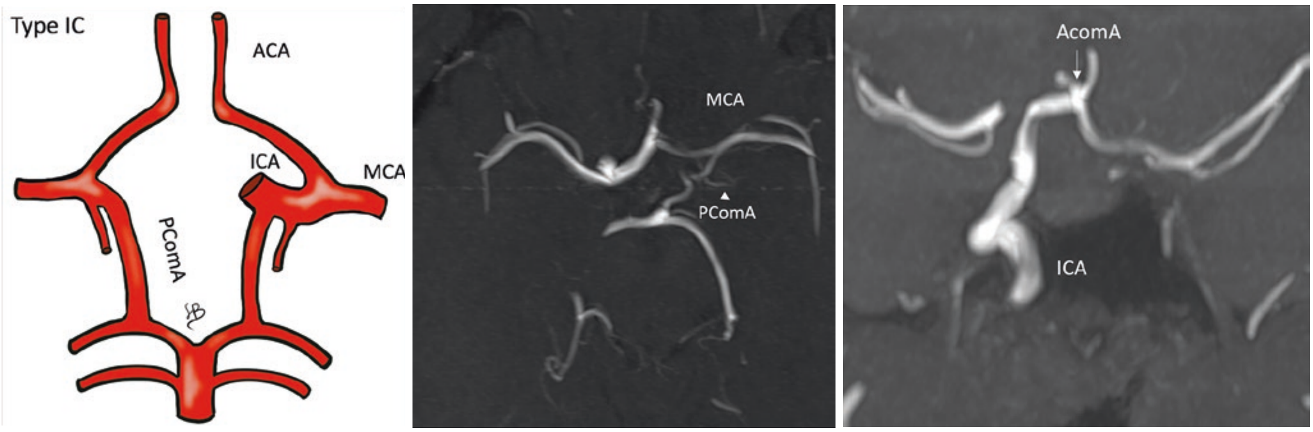


Fig. 3 Clinical case of type IC. The figure shows an example of internal carotid artery (ICA) agenesis on the left side, where the supply from the circle of Willis comes partially from the AComA, which is not able to compensate alone the flow, and from the PComA, which grant the circle to the ACA and MCA

provokes the non-formation of the PComA. This subtype of flow compensation is very rare with only 7 cases described in the literature [95–100].

2. **Type IIB:** In this case, a segmental agenesis of the VIth segment of the ICA happens before regression of the PDOA [15]. The presence of the PDOA at the moment of the vascular insult allows to maintain the flow into the proximal segments of the ICA. Consequently, only the VIth segment of the ICA is absent, and the flow of the ICA has an intra-orbital course with persistence of the 2 primitive OA (PDOA and PVOA) with anastomosis between them. This subtype of ICA agenesis with intra-orbital flow of the ICA is very rare and reported in only 1 case [101].

Type III: Compensation by Arterio-Arterial Anastomosis

In case of agenesis of one or more segments of the ICA, the flow-rerouting could be done by anastomosis and enlargement of two existing arteries. During the embryological life, some artery could be more developed during one phase before having a partial regression. The most prominent example is the hyoid artery which has a transient development in stapedial artery (SA) before a partial regression to become the carotico-tympanic artery in adult. We could find two different types of segmental ICA agenesis with compensation by arterio-arterial anastomosis.

1. **Type IIIA:** In case of agenesis of the second ICA segment with proximal regression, the arterial flow is rerouted through the inferior tympanic artery (branch of the ascending pharyngeal artery) which creates an anastomosis with the carotico-tympanic artery (carotid remnant of the SA) [19, 20, 22, 102]. This anatomic variation is the result of a vascular insult of the second segment of the ICA probably when the SA is near its maximal development (stage IV of Padget). This variant called intra-tympanic flow of the ICA or aberrant ICA is largely described in chapter "Intra-Tympanic Flow of the Internal Carotid Artery" and will not develop in the present chapter. A clinical case of this type is shown in Fig. 4.
2. **Type IIIB:** This subtype of ICA agenesis is secondary to the agenesis of segment V or VI of the ICA with proximal regression of other segments. The flow compensation of ICA territories is done by an anastomosis and enlargement of side-to-side cavernous branches of both ICAs. Consequently, the distal segments of the ICA are supplied by an inter-cavernous vessel that origins from the cavernous segment of the contralateral ICA. A case of this form of agenesis is shown in Fig. 5. In literature, more than 40

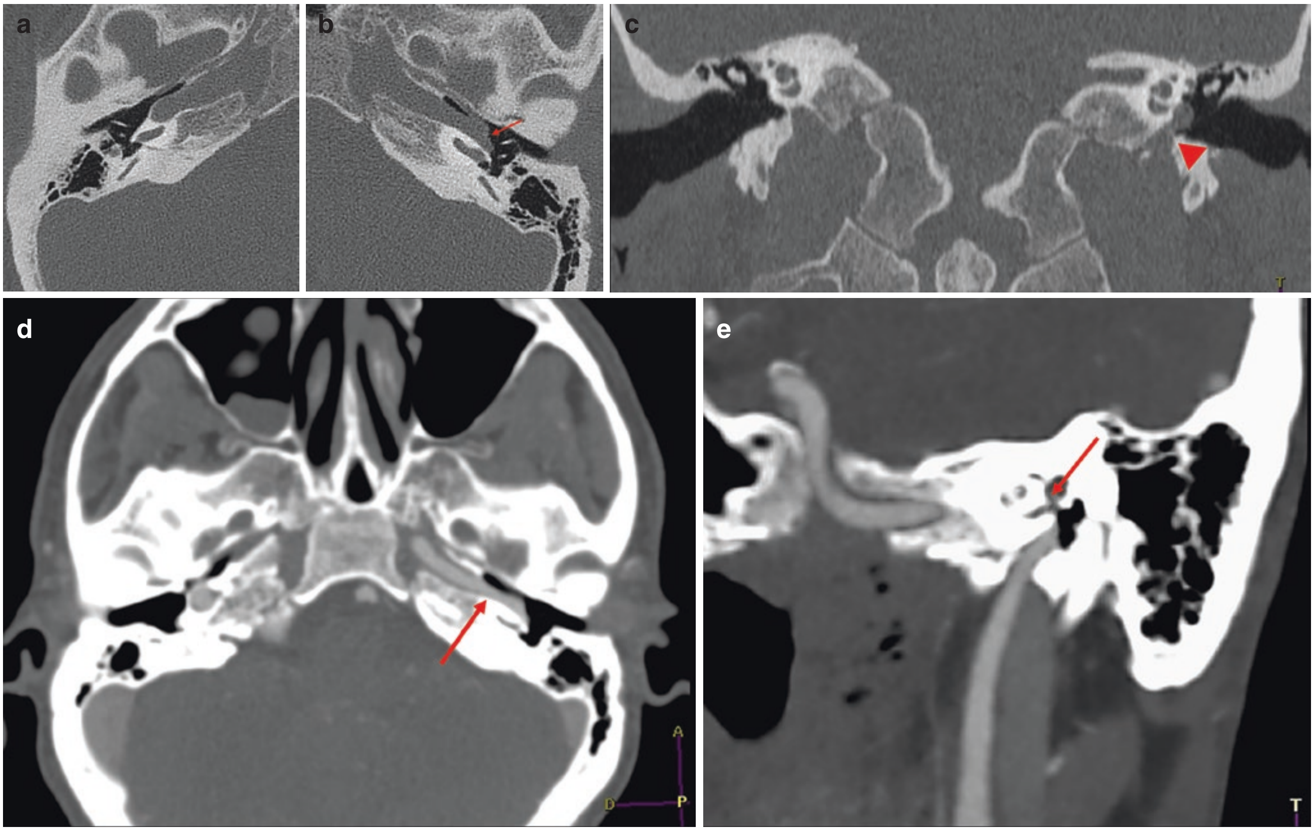


Fig. 4 Clinical case of pseudo-petrous or aberrant ICA. Compared to normal side (**a**) axial image (**b**) and coronal (**c**) projection reveal on the left side a tubular structure crossing the middle ear cavity close to the cochlear promontory. Note the caliber change of the aberrant vessel rejoining the horizontal petrous ICA (red arrow in **b**) and the enlargement of the left inferior tympanic canaliculus (red arrowhead in **c**). CTA axial (**d**) and oblique coronal reconstruction (**e**) depicts the left aberrant ICA abutting the cochlear promontory within the middle ear (red arrow)

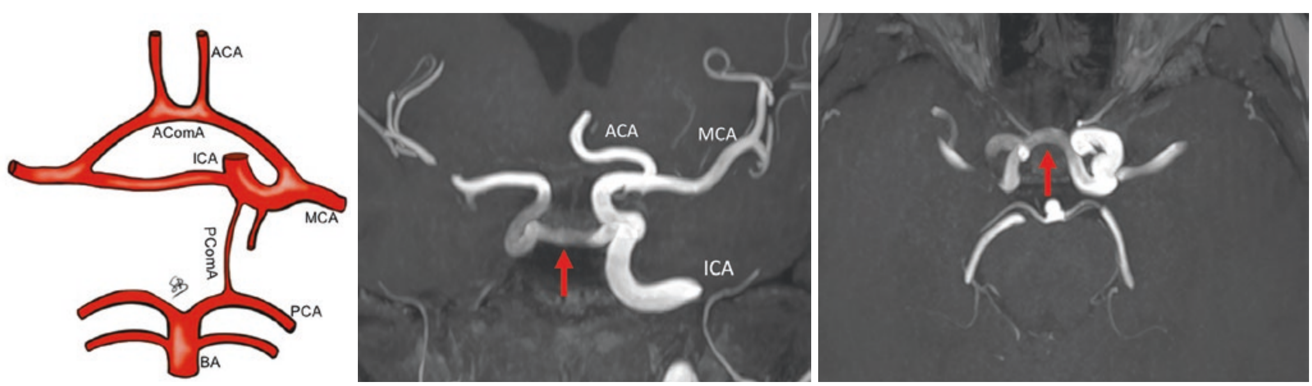


Fig. 5 Type III B intercavernous anastomosis. The figure shows a clinical case (MRI TOF sequences) of right ICA agenesis, with compensation of the distal flow into the ACA and MCA granted by intercavernous anastomosis (red arrow). The artist's illustration shows a schematic representation of this type of agenesis

cases of ICA agenesis with inter-cavernous anastomosis were described [32, 103–134]. The majority of these cases involved the Vth segment of the ICA, but few cases involved also the VIth segment [117, 121, 123, 126]. Different authors proposed some hypotheses to explain the formation of this inter-cavernous anastomosis. The first one is the fusion of the two TAs [19, 125]; the second hypothesis is the anastomosis between capsular branches of the ICA and the third one is the anastomosis between clival branches (which are branches of the meningohypophyseal trunk) [130, 133]. Based on the cadaveric dissections, these anastomoses could be intraosseous into the sphenoid bone or trans-sellar [104, 106]. These cases were described in association with an AComA aneurysm (5 cases), a hypopituitarism (5 cases), a contralateral ICA aneurysm (1 case), or a MCA aneurysm (1 case) [49, 122, 125, 135]. Two cases described by Uchino (2019) and by Park (2018) are enough particular to be cited [125, 133]. These cases of ICA agenesis with inter-cavernous anastomosis also present a supply of the MCA territory by an enlarged PComA and could be assimilated as hybrid between type IC and IIIB. Other four cases presented the particularity that the anastomotic vessel arises from the supraclinoid segment of the ICA [120, 122, 129, 132]. This reinforces the hypothesis that the inter-cavernous vessel is an anastomotic vessel between capsular arteries or hypophyseal arteries.

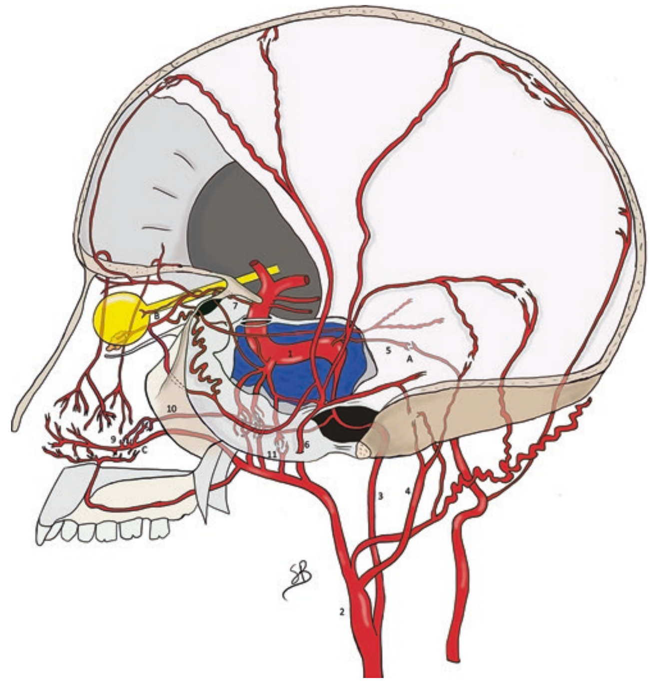


Fig. 6 Artist's illustration of Type IV (Rete mirabile). It consists in the flow compensation of the distal ICA (*1*) by trans-osseous anastomoses between branches of the ECA (*2*) and ICA. Most cases do not present a complete regression of the proximal segments of the ICA but only hypoplasia (*3*). The most common anastomoses are: *A* Between branches of the ascending pharyngeal artery (*4*) and clival branches (*5*) from the cavernous segment of the ICA. *B* Between the middle meningeal artery (*6*) and the ophthalmic artery (*7*) to the clinoidal segment of the ICA. *C* Between the internal maxillary artery (*8*) through the great palatine arteries (*9*) and the vidian artery (*10*) from the petrous segment of the ICA. *D* Between the internal maxillary artery (*8*) through the accessory meningeal (*11*) artery to the cavernous segment of the ICA

Type IV: Compensation by External-Internal Carotid Arteries Anastomoses (Carotid Rete Mirabile)

This type of ICA agenesis has been known for centuries since it represents the normal vascular disposition for some mammals [136]. It consists in the flow compensation of the distal ICA by trans-osseous anastomoses between branches of the external and internal carotid arteries. Even if Galen had already described it, the carotid rete mirabile remains complex and poorly understood. It is a very rare variant with only 22 cases described in the literature [137–155]. Carotid rete mirabile, compared to other type of compensation, presents some particularities. First, most cases (19/22 cases) do not present a complete regression of the proximal segments of the ICA but only hypoplasia. Secondly, most cases (17/22 cases) present a bilateral agenesis of the ICA. Finally, the two most involved segments are the segments V and VI of the ICA (14/22 cases). All these particularities allow us to think that the vascular insult succeed very early in the development, before the regression of the aortic arches that would explain the non-regression of the proximal segments [155]. Even if it is difficult to identify angiographically all the ECA branches that participate in the flow compensation of the ICA, the most frequent are represented and described in Fig. 6.

A carotid rete mirabile could be associated with a rete vertebralis (5 cases/22), a cerebral aneurysm (1/22), a brain AVM (1/22), or an Apert syndrome (1/22).

Our classification does not allow to understand all the embryological details to explain the flow compensation in each type described. The rarity of these variants, the incidental discovery of most of them, and the lack of a complete radiological assessment limit, of course, their analysis and their detailed knowledge. Despite the limits of our classification, it presents some advantages compared to other classifications previously presented. Firstly, it is based on our current knowledge of vascular embryology. Secondly, it is easy to understand and useful. Finally, it allows to include all cases of ICA agenesis described in the literature. Further studies with selective catheterisms would be surely helpful to add details to our knowledge of the anastomosis that support the flow in case of ICA agenesis, even if the rarity of these variants represents an important limit.

Clinical Implications

Most cases of segmental ICA agenesis are asymptomatic and discovered incidentally. Numerous non-specific neurological symptoms have been associated with cases of ICA agenesis as headache, blurred vision, seizures, or transient hemiparesis [5, 56, 133, 155]. Other symptoms or signs could be more easily imputable to the vascular variation. For example, cases of trigeminal neuralgia in case of persistent trigeminal artery (type IIA) were described [98].

With the lack of one of the three major arterial supplies of the brain, the possibility of compensation in case of stroke is limited, particularly in watershed zones. Signs of transient ischemic attack or stroke is reported in approximately 20% of cases.

The incidence of aneurysmal sub-arachnoid hemorrhage (SAH) is particularly high in case of ICA agenesis. This risk, evaluated to 25–34%, is much higher to the risk of aneurysmal SAH in the general population (3–5%) [54, 62–64, 122, 156, 157]. Aneurysms in these cases are almost all located at the AComA complex, the basilar tip or the PComA origin. Two hypotheses are postulated in the literature to explain the high risk of aneurysmal SAH in case of ICA agenesis. The first one is the hemodynamic changes due to the flow compensation. The second one is the developmental errors of the arterial wall present in association with the primary vascular insult. The fact that the majority of cases are cases of ICA agenesis with flow compensation by the circle of Willis (types IA, IB, and IC) allows us to think that the hemodynamic factor plays an important role in the formation of the aneurysm.

Differing from type I ICA agenesis, the presence of ICA agenesis with rete mirabile (type IV) is more frequently associated with a non-aneurysmal SAH. The frequency of this association is evaluated to 29% but no explanation could be found in the literature [138].

Independently to the type of flow compensation, an ICA agenesis is often reported in association with other pathology or malformation as an agenesis of the corpus callosum, a trans-sphenoidal encephalocele, a PHACE syndrome, a cavum vergae, an arachnoid cyst, or a Horner's syndrome.

The presence of an ICA agenesis and its type must be known in certain clinical situation. For example, the presence of an inter-cavernous anastomotic vessel could have dramatic consequences in case of trans-sphenoidal surgery. Another important example is the surgery of carotid ligation or carotid endarterectomy that could be dramatic in the presence of contralateral ICA agenesis.

References

1. Teal JS, Nahedy MH, Hasso AN. Total agenesis of the internal carotid artery. AJNR Am J Neuroradiol. 1980;1:435–42.
2. Quint DJ, Silbergleit R, Young WC. Absence of the carotid canals at skull base CT. Radiology. 1992;182(2):477–81. https://doi.org/10.1148/radiology.182.2.1732967.
3. Dungan DH, Heiserman JE. The carotid artery: embryology, normal anatomy, and physiology. Neuroimaging Clin N Am. 1996;6(4):789–99.
4. Meder JF, Blustajn J, Trystram D, Godon-Hardy S, Devaux B, Zuber M, et al. Radiologic anatomy of segmental agenesis of the internal carotid artery. Surg Radiol Anat. 1997;19(6):385–94. https://doi.org/10.1007/bf01628506.
5. Vasovic L, Trandafilovic M, Vlajkovic S, Radenkovic G. Congenital absence of the bilateral internal carotid artery: a review of the associated (ab)normalities from a newborn status to the eighth decade of life. Childs Nerv Syst. 2018;34(1):35–49. https://doi.org/10.1007/s00381-017-3629-1.
6. Zink WE, Komotar RJ, Meyers PM. Internal carotid aplasia/hypoplasia and intracranial saccular aneurysms: series of three new cases and systematic review of the literature. J Neuroimaging. 2007;17(2):141–7. https://doi.org/10.1111/j.1552-6569.2007.00092.x.
7. Lee JH, Oh CW, Lee SH, Han DH. Aplasia of the internal carotid artery. Acta Neurochir. 2003;145(2):117–25; discussion 25. https://doi.org/10.1007/s00701-002-1046-y.
8. Fitzgerald RT, Zuccoli G. Agenesis of the internal carotid artery: associated malformations including a high rate of aortic and cardiac malformations. Pediatr Radiol. 2012;42(11):1333–8. https://doi.org/10.1007/s00247-012-2455-6.
9. Lie TA. The congenital anomalies of the carotid arteries. Amsterdam/New York: Excerpta Medica Foundation; 1968.
10. Tode. Medizinisch-chirurgische Bibliothek. Copenhagen. 1787.
11. Fisher AG. A case of complete absence of both internal carotid arteries, with a preliminary note on the developmental history of the stapedial artery. J Anat Physiol. 1913;48(Pt 1):37–46.
12. Tasar M, Yetiser S, Tasar A, Ugurel S, Gonul E, Saglam M. Congenital absence or hypoplasia of the carotid artery: radioclinical issues. Am J Otolaryngol. 2004;25(5):339–49.
13. Gailloud P, Clatterbuck RE, Fasel JH, Tamargo RJ, Murphy KJ. Segmental agenesis of the internal carotid artery distal to the posterior communicating artery leading to the definition of a new embryologic segment. AJNR Am J Neuroradiol. 2004;25(7):1189–93.
14. Cohen JE, Gomori JM, Leker RR. Internal carotid artery agenesis: diagnosis, clinical spectrum, associated conditions and its importance in the era of stroke interventions. Neurol Res. 2010;32(10):1027–32. https://doi.org/10.1179/016164110X12767786356273.
15. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–62.
16. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
17. Barry A. The aortic arch derivatives in human adult. Anat Rec. 1951;111(2):221–38.
18. Verbiest H. Radiologic findings in a case with absence of the left internal carotid artery and compression of several cranial nerve roots in the posterior fossa by the basilar artery. Med Contemp. 1954;71:601–9.
19. Lasjaunias P, Moret J, Manelfe C, Theron J, Hasso T, Seeger J. Arterial anomalies at the base of the skull. Neuroradiology. 1977;13(5):267–72.
20. Lasjaunias P, Moret J. Normal and non-pathological variations in the angiographic aspects of the arteries of the middle ear. Neuroradiology. 1978;15(4):213–9. https://doi.org/10.1007/bf00327529.
21. Lasjaunias P, Santoyo-Vazquez A. Segmental agenesis of the internal carotid artery: angiographic aspects with embryological discussion. Anat Clin. 1984;6(2):133–41. https://doi.org/10.1007/bf01773165.
22. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer edition; 2001.

23. Krings T, Lasjaunias PL. Segmental agenesis of the internal carotid artery distal to the posterior communicating artery leading to the definition of a new embryologic segment. AJNR Am J Neuroradiol. 2006;27(2):246–7. author reply 7
24. Moret J, Delvert JC, Bretonneau CH, Lasjaunias P, de Bicetre CH. Vascularization of the ear: normal-variations-glomus tumors. J Neuroradiol. 1982;9(3):209–60.
25. Tsuruta J, Miyazaki Y. A case of complete absence of the left internal carotid artery associated with an aneurysm of the anterior communicating artery (author's transl). No Shinkei Geka. 1977;5(8):895–900.
26. Jamous MA, Abdel-Aziz H, Kaisy F. Collateral blood flow patterns in patients with unilateral ICA agenesis and cerebral aneurysm. Neuro Endocrinol Lett. 2007;28(5):647–51.
27. Lagarde C, Vigouroux R, Perrouty P. Terminal agenesis of internal carotid, aneurysm of anterior communicating; radiologic documents. J Radiol Electrol Arch Electr Medicale. 1957;38(9–10):939–41.
28. Turnbull I. Agenesis of the internal carotid artery. Neurology. 1962;12:588–90. https://doi.org/10.1212/wnl.12.9.588.
29. Lhermitte F, Gautier JC, Poirier J, Tyrer JH. Hypoplasia of the internal carotid artery. Neurology. 1968;18(5):439–46. https://doi.org/10.1212/wnl.18.5.439.
30. Moyes PD. Basilar aneurysm associated with agenesis of the left internal carotid artery. Case report. J Neurosurg. 1969;30(5):608–11. https://doi.org/10.3171/jns.1969.30.5.0608.
31. Tangchai P, Khaoborisut V. Agenesis of internal carotid artery associated with aneurysm of contralateral middle cerebral artery. Neurology. 1970;20(8):809–12. https://doi.org/10.1212/wnl.20.8.809.
32. Harispe L, Creissard P, Martinez G, Metzger J. An abnormal communicating artery uniting the two intracranial internal carotid arteries. J Radiol Electrol Med Nucl. 1970;51(12):759–61.
33. Teal JS, Rumbaugh CL, Bergeron RT, Segall HD. Congenital absence of the internal carotid artery associated with cerebral hemiatrophy, absence of the external carotid artery, and persistence of the stapedial artery. Am J Roentgenol Radium Therapy, Nucl Med. 1973;118(3):534–45.
34. Carella A, Federico F, Germinario L, Lamberti P. Angiographic finding of agenesis of one internal carotid artery associated with two aneurysms of the omolateral posterior cerebral artery. Acta Neurol (Napoli). 1975;30(3):281–5.
35. Servo A. Agenesis of the left internal carotid artery associated with an aneurysm on the right carotid syphon. Case report. J Neurosurg. 1977;46(5):677–80. https://doi.org/10.3171/jns.1977.46.5.0677.
36. Shigemori M, Kojo N, Miyagi J, Watanabe M, Kuramoto S. Agenesis of the left internal carotid artery associated with an aneurysm of the anterior communicating artery. Neurol Med Chir (Tokyo). 1980;20(1):73–9. https://doi.org/10.2176/nmc.20.73.
37. Bernini FP, Cioffi FA, Muras I, Rinaldi F, Vaino R. Agenesis of the right internal carotid artery associated with an aneurysm of the anterior communicating artery. Case report. Acta Neurochir (Wien). 1980;54(3–4):257–63. https://doi.org/10.1007/bf01407093.
38. Handa J, Matsuda I, Nakasu S, Nakano Y. Agenesis of an internal carotid artery: angiographic, tomographic and computed tomographic correlation. Neuroradiology. 1980;19(4):207–11. https://doi.org/10.1007/bf00376709.
39. Afifi AK, Godersky JC, Menezes A, Smoker WR, Bell WE, Jacoby CG. Cerebral hemiatrophy, hypoplasia of internal carotid artery, and intracranial aneurysm. A rare association occurring in an infant. Arch Neurol. 1987;44(2):232–5. https://doi.org/10.1001/archneur.1987.00520140090024.
40. Wakai S, Watanabe N, Inoh S, Ochiai C, Nagai M. Agenesis of internal carotid artery presenting with oculomotor nerve palsy after minor head injury. Neurosurgery. 1987;20(5):794–6. https://doi.org/10.1227/00006123-198705000-00022.
41. Verdeille S, Tonnelier M, Ghossain MA, Besse F, Buy JN. Agenesis of the left internal carotid artery associated with coarctation of the aorta: report on an unusual case. Cardiovasc Intervent Radiol. 1988;11(6):325–8.
42. Grand CM, Louryan S, Bank WO, Baleriaux D, Brotchi J, Raybaud C. Agenesis of the internal carotid artery and cavernous sinus hypoplasia with contralateral cavernous sinus meningioma. Neuroradiology. 1993;35(8):588–90. https://doi.org/10.1007/bf00588400.
43. Kikuchi K, Kowada M, Kojima H. Hypoplasia of the internal carotid artery associated with spasmodic torticollis: the possible role of altered vertebrobasilar haemodynamics. Neuroradiology. 1995;37(5):362–4. https://doi.org/10.1007/bf00588012.
44. Ide M, Jimbo M, Yamamoto M, Hagiwara S. Posterior cerebral artery aneurysm associated with unilateral internal carotid artery agenesis. Case report. Neurol Med Chir (Tokyo). 1995;35(11):825–9. https://doi.org/10.2176/nmc.35.825.
45. Wu CH, Hwu WL, Wang JK, Young C, Peng SS, Kuo MF. Deletion of 11q24.2-qter with agenesis of unilateral internal carotid artery and total anomalous pulmonary venous return. Am J Med Genet. 2001;103(3):245–8.
46. Ito S, Miyazaki H, Iino N, Shiokawa Y, Saito I. Unilateral agenesis and hypoplasia of the internal carotid artery: a report of three cases. Neuroradiology. 2005;47(5):311–5. https://doi.org/10.1007/s00234-003-1090-1.
47. Naeini RM, De J, Satow T, Benndorf G. Unilateral agenesis of internal carotid artery with ophthalmic artery arising from posterior communicating artery. AJR Am J Roentgenol. 2005;184(2):571–3. https://doi.org/10.2214/ajr.184.2.01840571.
48. Tubbs RS, Oakes WJ. Horner's syndrome resulting from agenesis of the internal carotid artery: report of a third case. Childs Nerv Syst. 2005;21(1):81–2. https://doi.org/10.1007/s00381-003-0893-z.
49. Wong GK, Yu SC, Antonio GE, Poon WS. A rare anatomic variant of a solitary internal carotid artery associated with moyamoya phenomenon of the middle cerebral artery. AJNR Am J Neuroradiol. 2006;27(9):2012–3.
50. Li S, Hooda K, Gupta N, Kumar Y. Internal carotid artery agenesis: a case report and review of literature. Neuroradiol J. 2017;30(2):186–91. https://doi.org/10.1177/1971400917692162.
51. Perla FM, Carbotta G, Di Nardo D, D'Avanzo M, Colaiacomo MC, Di Biasi C, et al. Agenesis of the internal carotid artery: a family pathology? G Chir. 2017;38(1):46–9.
52. Pierro A, Cilla S, Cucci E, Sallustio G. Computed tomography angiography of unilateral agenesis of the internal carotid artery: 2 cases report with focus on embryology, collateral pathways, and imaging. Radiol Case Rep. 2017;3:606–9.
53. Mahajan A, Goel G, Das B. Comments on "agenesis of the left internal carotid artery associated with Dolichoectatic intracranial arteries". J Stroke Cerebrovasc Dis. 2018;27(8):2285. https://doi.org/10.1016/j.jstrokecerebrovasdis.2018.02.024.
54. Jianu DC, Barsan C, Dan TF, Jianu SN, Motoc AGM, Cretu OM. Left internal carotid artery agenesis associated with communicating arteries anomalies. A case report. Romanian J Morphol Embryol. 2018;59(2):601–5.
55. Nomura M, Tamase A, Mori K, Seki S, Iida Y, Kawabata Y, et al. Agenesis of the left internal carotid artery associated with Dolichoectatic intracranial arteries. J Stroke Cerebrovasc Dis. 2018;27(2):e24–e6. https://doi.org/10.1016/j.jstrokecerebrovasdis.2017.09.021.
56. Uchino A, Ehara T, Kurita H. Hypoplasia of the internal carotid artery with associated fenestration and extremely long P1 segment of the ipsilateral posterior cerebral artery diagnosed by MR

angiography. Surg Radiol Anat. 2019;41(6):707–11. https://doi.org/10.1007/s00276-019-02212-z.
57. Armand JP, Dousset V, Viaud B, Huot P, Chehab Z, Dos Santos E, et al. Agenesis of the internal carotid artery associated with an aneurysm of the anterior communicating artery. J Neuroradiol. 1996;23(3):164–7.
58. Blustajn J, Netchine I, Fredy D, Bakouche P, Piekarski JD, Meder JF. Dysgenesis of the internal carotid artery associated with transsphenoidal encephalocele: a neural crest syndrome? AJNR Am J Neuroradiol. 1999;20(6):1154–7.
59. Dinc H, Gumele HR, Kuzeyli K, Baykal S. Unilateral agenesis of internal carotid artery with subarachnoid hemorrhage: report of two cases. Int J Angiol. 1999;8(3):157–60.
60. Florio F, Balzano S, Nardella M, Strizzi V, Cammisa M, Bozzini V, et al. Congenital absence of the internal carotid artery. Cardiovasc Intervent Radiol. 1999;22(1):74–8.
61. Claros P, Bandos R, Gilea I, Claros A Jr, Capdevila A, Garcia Rodriguez J, et al. Major congenital anomalies of the internal carotid artery: agenesis, aplasia and hypoplasia. Int J Pediatr Otorhinolaryngol. 1999;49(1):69–76. https://doi.org/10.1016/s0165-5876(99)00012-9.
62. Orakdogen M, Berkman Z, Ersahin M, Biber N, Somay H. Agenesis of the left internal carotid artery associated with anterior communicating artery aneurysm: case report. Turk Neurosurg. 2007;17(4):273–6.
63. Demirgil B, Tugcu B, Gunaldi O, Postalci L, Gunal M, Tanriverdi O. Agenesis of the left internal carotid artery associated with right anterior cerebral artery A1 segment bifurcation aneurysm: a case report. Minim Invasive Neurosurg. 2007;50(5):300–3. https://doi.org/10.1055/s-2007-985364.
64. Amano T, Inamura T, Matsukado K, Yoshikai S, Yuhi F, Nagata S. Ruptured saccular aneurysm of a dolichoectatic internal carotid artery in a patient with agenesis of the contralateral internal carotid artery—case report. Neurol Med Chir (Tokyo). 2004;44(1):20–3. https://doi.org/10.2176/nmc.44.20.
65. Akiyama Y, Okada T, Hayashi N, Yokoi T. Infraoptic course of the anterior cerebral artery originating from the extradural internal carotid artery associated with contralateral internal carotid artery agenesis and multiple intracerebral aneurysms. Neurol Med Chir (Tokyo). 2010;50(11):984–7. https://doi.org/10.2176/nmc.50.984.
66. Kang HJ, Lee YS, Suh SJ, Lee JH, Ryu KY, Kang DG. A ruptured aneurysm at the infraoptic azygous anterior cerebral artery with the contralateral internal carotid artery agenesis treated by Y-stent assisted coil embolization. J Cerebrovasc Endovasc Neurosurg. 2012;14(3):237–42. https://doi.org/10.7461/jcen.2012.14.3.237.
67. Given CA 2nd, Huang-Hellinger F, Baker MD, Chepuri NB, Morris PP. Congenital absence of the internal carotid artery: case reports and review of the collateral circulation. AJNR Am J Neuroradiol. 2001;22(10):1953–9.
68. Chen MC, Liu HM, Huang KM. Agenesis of the internal carotid artery associated with neurofibromatosis type II. AJNR Am J Neuroradiol. 1994;15(6):1184–6.
69. Ottaviano G, Calzolari F, Martini A. Goldenhar syndrome in association with agenesia of the internal carotid artery. Int J Pediatr Otorhinolaryngol. 2007;71(3):509–12. https://doi.org/10.1016/j.ijporl.2006.11.003.
70. Slankamenac P, Jesic A, Zivanovic Z, Gebauer-Bukurov K, Bozic K. Agenesis of internal carotid artery with unusual fetal collateral circulation. Arch Neurol. 2010;67(10):1280–1. https://doi.org/10.1001/archneurol.2010.236.
71. Lopez-Alvarez F, Bernardo-Corte MA, Gomez-Martinez JR, Suarez-Nieto C. Unilateral agenesis of the internal carotid artery. Acta Otorrinolaringol Esp. 2012;63(1):72–4. https://doi.org/10.1016/j.otorri.2010.11.010.
72. Ventura E, Ormitti F, Crisi G, Sesenna E. Goldenhar syndrome associated with contralateral agenesis of the internal carotid artery. Neuroradiol J. 2014;27(2):150–3. https://doi.org/10.15274/nrj-2014-10029.
73. David H, Dubayle P, Girodeau A, Sarrazin JL, Boyer B. Agenesis of the internal carotid artery: two cases report. J Radiol. 2000;81(2):147–50.
74. Elazab NE, Riel-Romero RM, Gonzalez-Toledo E. Internal carotid artery agenesis and basilar artery aneurysm with third nerve palsy. Pediatr Neurol. 2010;42(6):451–4. https://doi.org/10.1016/j.pediatrneurol.2010.02.002.
75. Hills J, Sament S. Bilateral agenesis of the internal carotid artery associated with cardiac and other anomalies. Case report. Neurology. 1968;18(2):142–6. https://doi.org/10.1212/wnl.18.2.142.
76. Boudin G, Chomette G, Guillard A, Hauw JJ, Der Agupian P. Bilateral aplasia of the internal carotid artery. Apropos of an anatomo-clinical case. Rev Neurol (Paris). 1972;127(3):329–40.
77. Rosen IW, Mills DF, Nadel HI, Kaiserman DD. Angiographic demonstration of congenital absence of both internal carotid arteries. Case report. J Neurosurg. 1975;42(4):478–82. https://doi.org/10.3171/jns.1975.42.4.0478.
78. Schlenska GK. Absence of both internal carotid arteries. J Neurol. 1986;233(5):263–6.
79. Cali RL, Berg R, Rama K. Bilateral internal carotid artery agenesis: a case study and review of the literature. Surgery. 1993;113(2):227–33.
80. Iaccarino V, Tedeschi E, Brunetti A, Soricelli A, Salvatore M. Congenital agenesis/aplasia of the internal carotid arteries: MRA and SPECT findings. J Comput Assist Tomogr. 1995;19(6):987–90. https://doi.org/10.1097/00004728-199511000-00028.
81. Yokochi K, Iwase K. Bilateral internal carotid artery agenesis in a child with psychomotor developmental delay. Pediatr Neurol. 1996;15(1):76–8.
82. Sunada I, Inoue T. Bilateral internal carotid artery agenesis. J Neurol Neurosurg Psychiatry. 1996;61(2):206–7. https://doi.org/10.1136/jnnp.61.2.206.
83. Rumboldt Z, Castillo M, Solander S. Bilateral congenital absence of the internal carotid artery. Eur Radiol. 2003;13(Suppl 4):L130–2.
84. Welman CJ, Khangure MS. MRI of bilateral internal carotid artery agenesis. Australas Radiol. 2007;51 Spec No:B25–7. https://doi.org/10.1111/j.1440-1673.2007.01697.x.
85. Sun J, Jiang D, Zhang S. Bilateral internal carotid artery agenesis with artery compression of the brain parenchyma. J Neurol Sci. 2008;272(1–2):191–3. https://doi.org/10.1016/j.jns.2008.05.007.
86. Steer AC, Rowe PW. Bilateral agenesis of the internal carotid artery: case report and approach to management. J Paediatr Child Health. 2008;44(9):511–3. https://doi.org/10.1111/j.1440-1754.2008.01357.x.
87. Yim NY, Ha HI, Park JH, Moon YJ, Yoon W, Kim JK. Agenesis of bilateral internal carotid artery associated with basilar artery fenestration mimicking intra-arterial thrombus: a case report. Vasc Endovasc Surg. 2010;44(1):69–74. https://doi.org/10.1177/1538574409347393.
88. Barbosa LG, Barbosa LA, Pimentel DP, Mata BE, Guerra LR, Viana LS. Bilateral agenesis of the internal carotid artery associated with basilar artery aneurysm treated via the endovascular route. A case report. Interv Neuroradiol. 2010;16(1):89–92. https://doi.org/10.1177/159101991001600112.
89. Bordet J, Larre I, Arndt C, Brugniart C, Benisty D, Ducasse A. Acute abducens nerve palsy as a result of bilateral internal carotid artery agenesis. J Fr Ophtalmol. 2016;39(4):e109–11. https://doi.org/10.1016/j.jfo.2015.01.025.
90. Tharp B, Heyman A, Pfeiffer JB, Young WG Jr. Cerebral ischemia result of hypoplasia of internal carotid artery. Arch Neurol. 1965;12:160–4. https://doi.org/10.1001/archneur.1965.00460260050006.

91. Smith KR Jr, Nelson JS, Dooley JM Jr. Bilateral “hypoplasia” of the internal carotid arteries. Neurology. 1968;18(12):1149–56. https://doi.org/10.1212/wnl.18.12.1149.
92. Austin JH, Stears JC. Familial hypoplasia of both internal carotid arteries. Arch Neurol. 1971;24(1):1–10. https://doi.org/10.1001/archneur.1971.00480310029001.
93. Guy G, Elouatassi K, Lancien G, Le Marec B, Pecker J. Bilateral hypoplasia of the internal carotid arteries. Apropos of an anatomo-clinical case. Sem Hop. 1974;50(12):809–18.
94. el Khamlichi A, Amrani F, el Azzusi M, el Oufir M, Khamlichi AM. Bilateral hypoplasia of the internal carotid arteries associated with aneurysm of the right posterior communicating artery. Apropos of a case. Neurochirurgie. 1989;35(1):23–30.
95. Dilenge D. Bilateral agenesis of internal carotid artery. J Can Assoc Radiol. 1975;26(2):91–4.
96. Fukuda M, Kameyama S, Takahashi H, Tanaka R. Trigeminal neuralgia caused by the vertebral artery associated with primitive trigeminal artery and agenesis of the internal carotid artery. Neurol Med Chir (Tokyo). 1998;38(6):367–70. https://doi.org/10.2176/nmc.38.367.
97. Garg A, Mishra NK, Gaikwad SB, Gupta V. Bilateral internal carotid artery segmental agenesis associated with persistent trigeminal artery, vascular ectasia, abnormal configuration of brain stem and cerebellum. A case report. Neuroradiol J. 2007;20(1):25–9. https://doi.org/10.1177/197140090702000103.
98. Hattori T, Kobayashi H, Inoue S, Sakai N. Persistent primitive trigeminal artery associated with absence of internal carotid artery. Surg Neurol. 1998;50(4):352–5. https://doi.org/10.1016/s0090-3019(97)00491-6.
99. Jaeger HJ, Mehring UM, Gissler HM, Mathias KD. Congenital absence of the internal carotid artery and the basilar artery with persistent trigeminal artery associated with coarctation of the aorta. Eur Radiol. 2000;10(11):1805–9. https://doi.org/10.1007/s003300000500.
100. Medhi G, Pendharkar H, terBrugge K, Prasad C, Gupta AK. Trigeminal neuralgia caused by a new variant of persistent trigeminal artery associated with agenesis of internal carotid artery. Interv Neuroradiol. 2017;23(6):566–70. https://doi.org/10.1177/1591019917715955.
101. Blackburn SL, Dannenbaum M, Schuette AJ, Dion J. Anomalous course of the intracranial internal carotid artery into the orbit: case report. J Neurointerv Surg. 2012;4(3):e9. https://doi.org/10.1136/neurintsurg-2011-010037.
102. Willinsky RA, Ter Brugge K, Lawson V, Mirich D, Lasjaunias P. Aberrant carotid artery: recognition on high resolution computed tomography. J Otolaryngol. 1990;19(4):249–51.
103. Smith RR, Kees CJ, Hogg ID. Agenesis of the internal carotid artery with an unusual primitive collateral. Case report. J Neurosurg. 1972;37(4):460–2. https://doi.org/10.3171/jns.1972.37.4.0460.
104. Faivre J, Vallee B, Carsin M, Scarabin JM, Simon J. Agenesis of the cervical and petrosal portions of the left internal carotid artery. J Neuroradiol. 1978;5(2):133–8.
105. Kishore PR, Kaufman AB, Melichar FA. Intrasellar carotid anastomosis simulating pituitary microadenoma. Radiology. 1979;132(2):381–3. https://doi.org/10.1148/132.2.381.
106. Staples GS. Transsellar intracavernous intercarotid collateral artery associated with agenesis of the internal carotid artery. Case report. J Neurosurg. 1979;50(3):393–4. https://doi.org/10.3171/jns.1979.50.3.0393.
107. Janicki PC, Limbacher JP, Guinto FC Jr. Agenesis of the internal carotid artery with a primitive transsellar communicating artery. AJR Am J Roentgenol. 1979;132(1):130–2. https://doi.org/10.2214/ajr.132.1.130.
108. Huber G. Intracranial carotid anastomosis and partial aplasia of an internal carotid artery. Neuroradiology. 1980;20(4):207–12. https://doi.org/10.1007/bf00336685.
109. Pascaud JL, Vigneu P, Hummel P, Bouchet JB, Rihouet J, Pascaud-Ged E. Partial absence of the internal carotid artery with trans-sellar intercarotid anastomosis. A case report and review of the literature (author’s transl). J Radiol. 1982;63(1):37–40.
110. Elefante R, Fucci G, Granata F, Graziussi G, Smaltino F. Agenesis of the right internal carotid artery with an unusual transsellar intracavernous intercarotid connection. AJNR Am J Neuroradiol. 1983;4(1):88–9.
111. Alexander MW, Chui MC, Gray T. Transellar intercavernous carotid anastomosis with agenesis of internal carotid artery and right-sided descending aorta: case report. J Can Assoc Radiol. 1984;35(1):92–5.
112. Tracy PT. Unusual intercarotid anastomosis associated with anterior communicating artery aneurysm. Case report. J Neurosurg. 1987;67(5):765–7. https://doi.org/10.3171/jns.1987.67.5.0765.
113. Udzura M, Kobayashi H, Taguchi Y, Sekino H. Intrasellar intercarotid communicating artery associated with agenesis of the right internal carotid artery: case report. Neurosurgery. 1988;23(6):770–3. https://doi.org/10.1227/00006123-198812000-00019.
114. Quint DJ, Boulos RS, Spera TD. Congenital absence of the cervical and petrous internal carotid artery with intercavernous anastomosis. AJNR Am J Neuroradiol. 1989;10(2):435–9.
115. Midkiff RB, Boykin MW, McFarland DR, Bauman JA. Agenesis of the internal carotid artery with intercavernous anastomosis. AJNR Am J Neuroradiol. 1995;16(6):1356–9.
116. Chen CJ, Chen ST, Hsieh FY, Wang LJ, Wong YC. Hypoplasia of the internal carotid artery with intercavernous anastomosis. Neuroradiology. 1998;40(4):252–4.
117. Kjellin IB, Kaiserman KB, Curran JG, Geffner ME. Aplasia of right internal carotid artery and hypopituitarism. Pediatr Radiol. 1999;29(8):586–8; discussion 5. https://doi.org/10.1007/s002470050654.
118. Mellado JM, Merino X, Ramos A, Salvado E, Sauri A. Agenesis of the internal carotid artery with a trans-sellar anastomosis: CT and MRI findings in late-onset congenital hypopituitarism. Neuroradiology. 2001;43(3):237–41.
119. Slaba SG, Zafatayeff SN, Chelala AS. Complete imaging study of internal carotid artery agenesis with intercavernous anastomosis. Neurochirurgie. 2002;48(1):35–8.
120. Moon WJ, Porto L, Lanfermann H, Weis R, Zanella FE. Agenesis of internal carotid artery associated with congenital anterior hypopituitarism. Neuroradiology. 2002;44(2):138–42. https://doi.org/10.1007/s002340100678.
121. Horie N, Tsutsumi K, Kaminogo M, Morikawa M, Kitagawa N, Nagata I. Agenesis of the internal carotid artery with transcavernous anastomosis presenting with an anterior communicating artery aneurysm—A case report and review of the literature. Clin Neurol Neurosurg. 2008;110(6):622–6. https://doi.org/10.1016/j.clineuro.2008.03.004.
122. Suyama K, Mizota S, Minagawa T, Hayashi K, Miyazaki H, Nagata I. A ruptured anterior communicating artery aneurysm associated with internal carotid artery agenesis and a middle cerebral artery anomaly. J Clin Neurosci. 2009;16(4):585–6. https://doi.org/10.1016/j.jocn.2008.06.001.
123. Lamine F, Kanoun F, Chihaoui M, Saveanu A, Menif E, Barlier A, et al. Unilateral agenesis of internal carotid artery associated with congenital combined pituitary hormone deficiency and pituitary stalk interruption without HESX1, LHX4 or OTX2 mutation: a case report. Pituitary. 2012;15(Suppl 1):S81–6. https://doi.org/10.1007/s11102-012-0411-y.
124. Lee EJ, Chang HW, Ha EY, Kim SH, Kim MJ, Yi HA, et al. Acute infarction in a patient with agenesis of the internal carotid artery and transcavernous anastomosis. J Clin Neurosci. 2012;19(4):597–9. https://doi.org/10.1016/j.jocn.2011.07.034.
125. Uchino A, Saito N, Kohyama S. Agenesis of the internal carotid artery with paraclinoid-supraclinoid anastomosis and basi-

lar artery-posterior communicating artery anastomosis diagnosed by magnetic resonance angiography. Surg Radiol Anat. 2015;37(6):685–7. https://doi.org/10.1007/s00276-014-1388-1.
126. Kumaresh A, Vasanthraj PK, Chandrasekharan A. Unilateral agenesis of internal carotid artery with intercavernous anastomosis: a rare case report. J Clin Imaging Sci. 2015;5:7. https://doi.org/10.4103/2156-7514.150453.
127. Okawa M, Higashi T, Komiyama M, Fukuda K, Abe H, Inoue T. Left internal carotid artery agenesis with trans-sellar collateral and a right aortic arch: case report. Interv Neuroradiol. 2015;21(6):759–64. https://doi.org/10.1177/1591019915609130.
128. Alurkar A, Karanam LS, Oak S, Kori S. Congenital absence of internal carotid artery with rare type of intercavernous anastamosis and ruptured cerebral aneurysm. J Clin Diagn Res. 2016;10(4):TD03-4:TD03. https://doi.org/10.7860/JCDR/2016/17797.7549.
129. Hong JH, Chang HW, Kim JM, Cho HC, Kim SH, Hong JH, et al. Internal carotid artery agenesis associated with a rare collateral pathway: supraclinoid-supraclinoid anastomosis. A case report. Surg Radiol Anat. 2016;38(2):261–3. https://doi.org/10.1007/s00276-015-1531-7.
130. Duan H, Li L, Zhao G, Zhang Y, Zhang J. Internal carotid artery agenesis with stenosed intercavernous anastomosis: a case report. J Biomed Res. 2016;30(4):344–7. https://doi.org/10.7555/JBR.30.20150134.
131. Erdogan M, Senadim S, Ince Yasinoglu KN, Selcuk HH, Atakli HD. Internal carotid artery agenesis with an intercavernous anastomosis: a rare case. J Stroke Cerebrovasc Dis. 2017;26(10):2442–5. https://doi.org/10.1016/j.jstrokecerebrovasdis.2017.06.055.
132. Kumagai K, Takeuchi S, Otani N, Komiyama M, Mori K. Agenesis of the internal carotid artery with transcavernous anastomosis associated with anterior communicating artery aneurysms. Asian J Neurosurg. 2017;12(4):801–3. https://doi.org/10.4103/1793-5482.181125.
133. Park YW, Yoo J, Kim DJ. Double inter-internal carotid artery communication through intercavernous anastomosis and posterior communicating artery associated with multiple intracranial artery segmental agenesis/aplasia. Surg Radiol Anat. 2018;40(2):227–31. https://doi.org/10.1007/s00276-017-1897-9.
134. Lee D, Kim JA, Choi WS. Agenesis of internal carotid artery and deformation of caudate and nucleus accumbens. Neurol Sci. 2019;40:2653. https://doi.org/10.1007/s10072-019-03983-4.
135. Hendrikse J, Hartkamp MJ, Hillen B, Mali WP, van der Grond J. Collateral ability of the circle of Willis in patients with unilateral internal carotid artery occlusion: border zone infarcts and clinical symptoms. Stroke. 2001;32(12):2768–73. https://doi.org/10.1161/hs1201.099892.
136. Legait E. Le réseau admirable carotidien. Biol Med (Paris). 1947;36(9–10):139–65.
137. Minagi H, Newton TH. Carotid rete mirabile in man. A case report. Radiology. 1966;86(1):100–2. https://doi.org/10.1148/86.1.100.
138. Hawkins TD, Scott WC. Bilateral rete carotidis in man. Clin Radiol. 1967;18(2):163–5. https://doi.org/10.1016/s0009-9260(67)80011-4.
139. Arnould G, Tridon P, Laxenaire M, Picard L, Thiriet M, Pourel J. Carotid angiomatosis presenting a rete mirabile. (Apropos of 2 cases). Rev Neurol (Paris). 1967;116(4):358–66.
140. Occleshaw JV, Garland P. Bilateral rete carotidis. Br J Radiol. 1969;42(503):851–3. https://doi.org/10.1259/0007-1285-42-503-851.
141. Jones RR, Wetzel N. Bilateral carotid vertebrobasilar rete mirabile. Case report. J Neurosurg. 1970;33(5):581–6. https://doi.org/10.3171/jns.1970.33.5.0581.
142. Picard L, Andre JM, Renard M, Montaut J, Tridon P. Rete mirabile of the human carotid. Etiologic and nosologic problems. Acta Radiol Diagn (Stockh). 1972;13(1):205–19.
143. Ernest C, Hirsch JF, Metzger J, Chahinian P, Leblanc J, Godeau P. Bilateral hypoplasia of the internal carotid with persistance of an admirable network. Apropos of a case in an adult. Nouv Press Med. 1973;2(24):1655–8.
144. Damry N, Hanquinet S, Christophe C, Janssen F, Delatte P, Perlmutter N. Bilateral congenital absence of the internal carotid artery with a primitive transmaxillary arterial anastomosis. Pediatr Radiol. 1994;24(3):200–3.
145. Mahadevan J, Batista L, Alvarez H, Bravo-Castro E, Lasjaunias P. Bilateral segmental regression of the carotid and vertebral arteries with rete compensation in a Western patient. Neuroradiology. 2004;46(6):444–9. https://doi.org/10.1007/s00234-003-1086-x.
146. Weon YC, Chung JI, Kim HJ, Byun HS. Agenesis of bilateral internal carotid arteries and posterior fossa abnormality in a patient with facial capillary hemangioma: presumed incomplete phenotypic expression of PHACE syndrome. AJNR Am J Neuroradiol. 2005;26(10):2635–9.
147. Kim MS, Lee SJ, Lee CH, Park HI. Bilateral segmental absence of the internal carotid artery with rete compensation associated with absence of basilar artery: case report. Surg Neurol. 2006;65(6):615–9; discussion 9; author reply 20. https://doi.org/10.1016/j.surneu.2005.07.072.
148. Sahin H, Cinar C, Oran I. Carotid and vertebrobasilar rete mirabile: a case report. Surg Radiol Anat. 2010;32(2):95–8. https://doi.org/10.1007/s00276-009-0531-x.
149. Hong JM, Lee JH, Yong SW. Neurological picture. Asymtomatic rete mirabile in carotid and vertebral circulation systems. J Neurol Neurosurg Psychiatry. 2010;81(9):1022–3. https://doi.org/10.1136/jnnp.2009.178368.
150. Lee SY, Cha SH. Bilateral carotid and vertebral rete mirabile presenting with a prominent anterior spinal artery mimicking a spinal dural AV fistula at MRI. Korean J Radiol. 2011;12(6):740–4. https://doi.org/10.3348/kjr.2011.12.6.740.
151. Del Zotto E, Ritelli M, Pezzini A, Drera B, Gamba M, Giossi A, et al. Clinical, neuroradiological and molecular features of a patient affected by pseudoxhantoma elasticum associated to carotid rete mirabile: case report. Clin Neurol Neurosurg. 2012;114(6):758–61. https://doi.org/10.1016/j.clineuro.2011.12.031.
152. Alexandre AM, Visconti E, Schiarelli C, Frassanito P, Pedicelli A. Bilateral internal carotid artery segmental agenesis: embryology, common collateral pathways, clinical presentation, and clinical importance of a rare condition. World Neurosurg. 2016;95(620):e9–e15. https://doi.org/10.1016/j.wneu.2016.08.012.
153. Requejo F, Strawich FR, Mouratian DM, Krings T. Isolation of right internal carotid artery, persistent proatlantal 1 artery and rete mirabile in a child with 22q11 deletion syndrome. Childs Nerv Syst. 2018;34(12):2509–13. https://doi.org/10.1007/s00381-018-3910-y.
154. Dhiman R, Gorimanipalli B, Swamy DR, Sharma S, Garg A, Saxena R. Congenital third nerve palsy associated with midbrain hypoplasia due to bilateral segmental internal carotid artery agenesis. J Neuroophthalmol. 2018;38(4):483–5. https://doi.org/10.1097/wno.0000000000000666.
155. Turk Y, Alicioglu B. Unilateral cervical and petrosal segment agenesis of the internal carotid artery with rete mirabile. Clin Imaging. 2019;57:25–9. https://doi.org/10.1016/j.clinimag.2019.04.012.
156. Wani A, Behari S, Lyndoh B, Jaiswal A, Sahu R, Jain V. Multiple aneurysms associated with agenesis of internal carotid artery. Turk Neurosurg. 2011;21(1):83–5.
157. Kawamura Y, Sayama T, Maehara N, Nishimura A, Iihara K. Ruptured aneurysm of an aberrant internal carotid artery successfully treated with simultaneous intervention and surgery in a hybrid operating room. World Neurosurg. 2017;102(695):e1–5. https://doi.org/10.1016/j.wneu.2017.03.133.

Embryology and Variations of the Anterior Choroidal Artery

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The anterior choroidal artery (AChoA) has been the subject of numerous anatomical descriptions and publications, given its crucial functional importance and its involvement in many neurovascular pathologies. Indeed, even though its size is relatively small, this vessel is responsible for the vascularization of deep-seated cerebral structures to which any damage would result in devastating clinical sequelae. This chapter will attempt to synthesize the wide variety of published data on the subject to provide a comprehensive understanding of the anatomy and embryology of this vessel.

History

The first specific anatomical description of the anterior choroidal vascularization was made by the French author Vicq-d'Azyr (1748–1794) in his remarkable anatomical books *Traité d'Anatomie et de Physiologie*, published in 1786 and dedicated to the King of France, and *Traité de l'Anatomie du Cerveau* [1, 2]. In his works, this author described the choroidal vasculature and called the AChoA the "anterior and inferior choroidal artery" or "choroidal branch of the internal carotid". By detailing that the AChoA is a "branch originating from the internal carotid", the author already mentioned the connections of this vessel with the one stemming from the posterior communicating artery (PComA), and from the posterior cerebral artery's (PCA) circulation ("posteroinferior and superior choroidal branches" or "artère choroïdienne de la cérébrale profonde", in French). Interestingly, this author stated that the AChoA is constant but variable in volume, and mostly originates directly from the internal carotid artery (ICA), even if a proximal middle cerebral artery (MCA) origin can be seen. He justified this last statement on the basis of Haller's drawings published in 1813 in his book *Anatomical Description of the Arteries of the Human Body* [3]. In the latter, Haller mentioned the presence of "branches to the choroid plexus" issuing from the "posterior carotid, or artery of the fossa sylviana", but did not describe their anatomical disposition.

Later on, other authors described the choroidal vascularization, and more specifically, its territories: Heubner (1872) [4], Duret (1874) [5], Kolisko (1891) [6], and Beevor (1908) [7, 8]. The works of the French surgeon Henri Duret are worth mentioning, as he was one of the first to carry out an anatomical study of cerebral vascularization by selective injection of colored gelatin [5]. This author described the AChoA as a terminal branch of the ICA through classic papers published in 1874 and stated that it sometimes arose from the MCA or PCA. Its course along the "lateral aspect of the optic tract" was specifically mentioned, as well as its branches irrigating the optic tract, cerebral peduncle, and *cornu ammonis*, and that its terminal branches were distributed to the choroid plexus of the inferior horn of the lateral ventricle. Heubner [4] concurred with Duret in many of his findings. Moreover, he described the basal branches, which are known for supplying the highest part of the *crus cerebri*, the posterior limb of the internal capsule, and the thalamus.

In 1891, Alexander Kolisko conducted a special study on 17 brains in Vienna, focusing on the supply mediated by the AChoA, which was injected either alone or in conjunction with the PCA or the MCA [6]. This work confirmed Duret's data concerning the territory supplied by the AChoA [6, 9]. In 1905, von Monakow [10] showed he was aware that the AChoA supplied part of the lateral geniculate body. Beevor, in 1908, perfected Duret's technique by injecting colored gelatin simultaneously into several arterial trunks, and specifically studied the vascularization of the lateral geniculate body [7, 8].

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In 1907, Blackburn was the first to describe, through the study of 220 brains, two cases where the AChoA was the main source of supply to a large part of the territory of the PCA [11]. In both brains, the PComA and PCA were hypoplastic, mainly supplying the cerebral peduncle only, whereas the AChoA was large, supplying both choroidal branches and arteries to the territory normally irrigated by the PCA. Since then, these arteries have been described by many authors [12].

A few years later, Abbie (1934) and Padget (1948) provided phylogenic and embryologic knowledge regarding the development of cerebral vasculature, respectively [9, 13–15].

In the 1950s, there has been a renewed interest in the anatomical description of this artery. Its proximal ligation (or section) was described by the American neurosurgeon Cooper as a therapeutic technique for the treatment of involuntary movements and Parkinsonism [16–20]. Indeed, during a subtemporal approach for a cerebral pedunculotomy, Cooper inadvertently tore and was therefore forced to occlude by clipping the AChA [21]. Much to Cooper's surprise, the patient's tremor and rigidity were abolished without any residual hemiparesis. One year later, in 1954, Carpenter et al. published a specific anatomical description of the AChoA [22].

Up to this day, several authors have specifically studied the microsurgical anatomy or the clinical importance of the AChoA with the help of modern imaging techniques [12, 23–29].

Embryological Development

The choroidal arteries, developing from the prechoroidal vessels, are among the earliest cerebral arteries to appear. Therefore, they morphologically constitute the basic system on which the future arterial tree will be grafted [30].

In addition to the works of Padget and Streeter, cited earlier in this book, Lasjaunias et al. have provided a detailed description of the AChoA's embryological development, which can be divided into six steps to facilitate the understanding of this relatively complex process (Table 1) [27, 30]:

Table 1 Embryology and development of the anterior choroidal artery (AChoA)

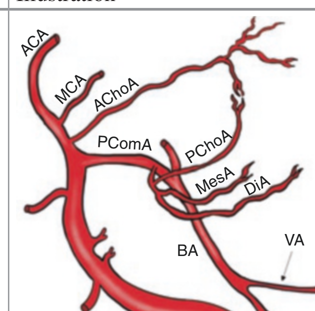
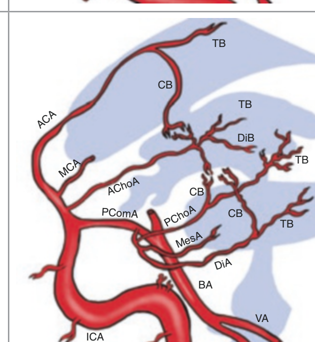
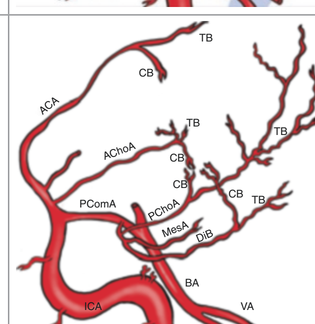
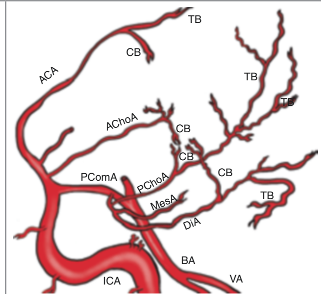
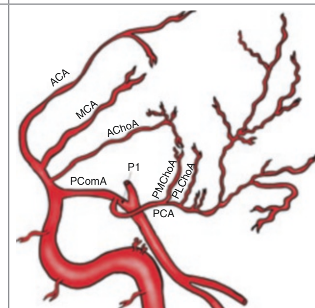
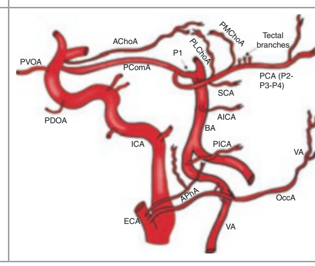
Stage		Illustration
I Prechoroidal	– Primitive ICA visible with its two divisions (CaR, CrR) – PComA completely formed – Origin from the distal PComA of a diencephalon-mesencephalic trunk (DiA-MesA). PChoA arising from the common trunk and anastomoses with the AChoA	
II Choroidal	– ACA gives a telencephalic (TB) and a choroidal branch (CB). The CB anastomoses with the CB of the AChoA at the interventricular foramen forming the "limbic arterial arch" – Development of a TB and a diencephalic branch (DiB) from the AChoA – The CB of the AChoA anastomoses with the CB of the PChoA	
III Telencephalic primitive	– Major development of the TB of the PChoA with annexation of some AChoA and ACA territories	

Table 1 (continued)

Stage		Illustration
IV Telencephalic intermediate	– Progressive development of the diencephalo-mesencephalic trunk of the PChoA with annexation of new cortical territories	
V Telencephalic final	– The cortical annexation gives relief to the ACA, facilitating the development of the MCA – The PChoA constitutes the main PCA stream and annexes the TB of the DiA – The CB branches of the PChoA and the DiA become the posteromedial and posterolateral choroidal arteries (PMChoA and PLChoA)	
VI Adult disposition	– Definitive development of the AChoA with reduction of its telencephalic territories – The MesA of the PChoA corresponds to the tectal branches of the PCA – SCA, AICA and PICA are visible	

ACA anterior cerebral artery, *AChoA* anterior choroidal artery, *BA* basilar artery, *Ca R* caudal ramus, *Cr R* cranial ramus, *ICA* internal carotid artery, *MCA* middle cerebral artery, *PCA* posterior cerebral artery, *PChoA* posterior choroidal artery, *PComA* posterior communicating artery

- *Prechoroidal (Padget stage 3/Carnegie 15–16)*: Initially, the vascular system of the brain is meningeal and consists of a deep perineural capillary network still outside the neural tube, supplied and drained by a more superficial arterial and venous network. With the continuous growth of the neural tube, the choroid plexuses will develop: the meninx primitiva will penetrate the ventricular lumen and invaginate into the ependymal layer to constitute the choroid plexuses. The choroid plexus plays a vital role in supporting early brain tissue, as the ventricles begin to develop in the following weeks. In this early stage of development, several arteries converge toward the cephalic region. The ICA divides into two branches: a caudal branch (future PCoA) and a cranial branch (telencephalic branch), the latter supplying the precursors of the anterior cerebral artery (ACA) and the AChA. Occasionally, the AChA originates from the proximal part of the caudal branch of the ICA. From the caudal end of the primitive PCoA, two branches emerge: a mesencephalic and a diencephalic artery, the latter giving rise a posterior choroidal branch directed toward the AChA.
- *Choroidal (Padget stage 4/Carnegie 17):* The development of the choroid plexuses, especially at the diencephalic–telencephalic junction, favors the extension of the ACA and AChoA. These arteries supply both the choroid plexuses and anastomose at the interventricular foramen. This AChoA–ACA anastomosis is named the “limbic arterial arch” and, in some cases, may persist into adult life. In addition, they present telencephalic branches anastomosing on the surface of the telencephalic vesicles.
- *Telencephalic Primitive* (*Padget stage 5/Carnegie 18–19*): A posterior choroidal artery (PChoA) develops from the diencephalo-mesencephalic trunk of the caudal branch and anastomoses at the atrium with the AChoA from the rostral division. During this stage, annexation of part of the AChoA’s territory by the PChoA of the caudal branch will begin and continue thereafter. Additional changes occur simultaneously at the telencephalic (cortical) level by means of anastomosis and capture, regressions, and changes in flow direction. The development of the cortex creates additional needs and opens new territories. Accordingly, the available sources of supply will provide the necessary vascularization.

- *Telencephalic intermediate (Padget stage 5/Carnegie 18–19):* The development of the patency of the vertebral artery and the resulting increasing flow in the basilar artery will give the developing diencephalo-mesencephalic trunk a high development potential. Therefore, some of the cortical territories of the ACA and AChA will be progressively annexed by the telencephalic branch of the PChA, and later by the diencephalic artery.
- *Telencephalic final (Padget stage 6/Carnegie 20–21):* At this stage, these "cortical" annexations gave some relief to the cranial branch, facilitating the development of the MCA system. The posterior choroidal branch of the caudal branch constitutes the main PCA stream. The telencephalic division of the diencephalic artery is annexed by its homolog from the PChoA.
- *Adult disposition (Padget stage 7/Carnegie 22–23):* The AChoA of the cranial branch has reduced its telencephalic territory to the uncus, pyriform cortex, and amygdaloid nucleus. Its diencephalic territory involves the thalamus, lateral geniculate body, and optic tract. As a choroidal vessel, it has preserved its supply to the anterolateral portion of the choroid plexus. Its territory may extend from the subthalamic area to part of the head of the caudate nucleus as well as some additional cortical supply. Finally, the choroidal branch of the PChoA corresponds to the posterolateral choroidal artery; the choroidal branch of the diencephalic branch will become the posteromedial choroidal artery. The mesencephalic arteries correspond to the tectal territorial branches of the PCA.

This process teaches us that the AChoA is embryologically an "old" artery. Moreover, its telencephalic branches to the temporal, parietal, and occipital lobes are annexed to the diencephalic/mesencephalic arteries or primitive PChoA of the caudal division of the primitive ICA in a process called "distal annexation" [31, 32]. The annexed cortical branches of the AChoA become the PCA distal to the PComA (P2–P4). When distal annexation does not occur or is incomplete, the AChoA keeps the cortical branches of the temporo-parieto-occipital lobes to different degrees, which are normally supplied by the P2–4 segments of the PCA. Such variants are described as "hyperplastic" variants of the AChoA. This embryologic process also explains that anterior and posterior choroidal vessels are connected by several anastomoses at the level of the diencephalon and choroid plexus [33–36].

The phylogeny of the AChoA is worth mentioning, as it is used by some authors to explain anatomical variants of the artery. According to a phylogenetic study of forebrain arteries by Abbie [9, 13, 14], the AChoA is the homolog of the inferior cerebral artery of Dendy in the sphenodon, a phylogenetically lower reptile. This small vessel runs posteriorly along the optic tract and anastomoses with the caudal division, which is the predecessor of the PCA, in a higher species of reptile: the crocodile. This vessel has no choroidal branches, and thus is not a "true" choroidal artery at this stage (it must be remembered that lateral ventricle has no inferior horn in reptiles). In mammals, because both the lateral ventricle and choroid fissure take on an arcuate form, the posterior part of the AChoA comes to lie alongside the anteroinferior end of the elongated choroid fissure, and acquires choroidal branches from the PCA, thus completing the AChoA in the true sense of the word. Hypoplasia of the plexal segment of the AChoA, a relatively rare anatomic variant, as described below, may represent an "evolutionary" variant in which the artery ceases to acquire choroidal branches, remaining in the "reptilian" stage [12, 37].

Furthermore, Tanriover et al. mentioned that the AChoA can be separated into a phylogenetically old parenchymal ramification, referred to as the cisternal segment, and its newly acquired choroidal ramification, referred to as the plexal segment [38–41]. On the basis of the results derived from the ICA developmental anatomy concept, the cisternal segment of the AChoA is also divided into two parts by this author: a proximal–preoptic part, which is arguably related to a phylogenetically older parenchymal ramification and resembles the ACA, and a distal–post-optic part, which is possibly phylogenetically related to a newer parenchymal ramification and similar to the PCA [38].

Number and Origin of the Artery

Several anatomical descriptions of the AChoA using different techniques have been published, and these works are summarized in Table 2.

Anatomists seem to agree that the presence of the AChoA looks constant. The artery was present in each of the 778 hemispheres studied by Otomo et al. and in each of the 50 hemispheres studied by Rhoton et al., although its absence was reported in one (1.7%) of the 60 hemispheres studied by Carpenter et al. in 1954 [22, 25, 42, 43]. However, Goldberg specifies that the AChoA may be very small or unrecognized if its territory is fed by branches of the PComA or PCA [40].

The AChoA usually arises from the supraclinoid segment of the ICA distal to the PComA as a single artery, and its origin serves as a landmark for the ICA's choroidal segment, extending from the AChoA's origin to the ICA's bifurcation into the ACA and MCA [25]. Several authors agree that the artery arises from the posterior or posterolateral aspect of the ICA. In their microsurgical anatomic descriptions based on cadaveric descriptions, Rhoton et al., Gibo et al., and Saeki et al. specified that the site of origin was on the posterolateral aspect of the carotid in 66% of hemispheres, on the posterior

Table 2 Summary of large series results about the AChA with the method used and the data analyzed

Author (year)	Number of hemispheres/brains	Method	Origin	Number	Diameter (mm)	Length (cisternal segment; mm)
Blackburn (1907)	220 br.	Fresh cadavers	ICA: 100%	NS	NS	NS
Beevor (1909)	87 br.	Colored gelatin injections	ICA: 100%	NS	NS	NS
Abbie (1933)	16 h.	Indian ink	ICA: 100%	NS	NS	NS
Carpenter (1954)	60 h.	Formalin-fixed	ICA: 76.6% PCoA: 6.7% Bif. ICA: 3.3% MCA: 11.7%	Single: 100%	0.6–1	26 (15–35)
Morello (1955)	100 h.	Angiography	ICA: 90.2% PCoA: 87% MCA: 1.1%	NS	NS	NS
Sjogren (1956)	100 angiographies (+ 30 specimens)	Angiography (+formalin-fixed)	ICA: 95% PCoA: 3% MCA: 2%	NS	NS	NS
Otomo (1965)	778 h.	Formalin-fixed	ICA: 99.2% PCoA: 0.4% Bif. ICA: 0.4%	NS	0.4–1.5	±25
Herman (1966)	44 h.	Indian ink and gelatin	ICA: 85% Bif. ICA 7% MCA: 8%	NS	NS	18 (11–22)
Wollschlaeger (1969)	162 br.	Barium	NS	NS	0.1–1	NS
Saeki (1977)	100 h.	Formalin-fixed	ICA: 100%	Single: 96% Double: 4%	0.5–2.3 (average: 1)	NS
Rhoton (1979)	50 h.	Latex-acrylic	ICA: 98% PCoA: 2%	Single: 96% double: 4%	0.7–2.0 (average:1.2)	24 (20–34)
Takahashi (1980)	640 h.	Angiography	NS	NS	NS	NS
Gibo (1981)	50 h.	Formalin-fixed. Colored latex or acrylic	ICA: 100%	Single: 96% double: 4%	0.5–2.1 (mean 1.0)	NS
Yasargil (1984)	200 specimens/2000 periprocedural observations	Formalin-fixed. Intraoperative observations	NS	Single: 70% 2–4 br.: 30%	Average 0.93	NS
Hussein (1988)	140 h.	Acrylic (100) + selective AChA dye injection (40)	PCoA: 2.5%	Single: 93.6% Double: 5% triple: 1.4%	0.4–1.1 (average: 0.9)	NS
Takahashi (1990)	216 h.	Angiography	ICA: 100%	Single: 100%	0.38–2.0 (average: 0.75 mm)	NS
Erdem (1993)	30 h.	Latex-silicone; formalin-fixed	ICA: 100%	Single: 100%	0.7 to 1.3 (average 0.93)	NS
Marinkovic (1994)	22 h.	Indian ink and gelatin	NS	NS	NS	NS
Morandi (1996)	50 h.	China ink and latex	ICA: 96% PCoA: 2% ICA Bif: 2%	Single: 100% *NB: Uncal a. with distinct origin in 58%*	Ø ≤ PCoA: 82% Ø = PCoA: 18%	NS
Marinkovic (1999)	30 h.	Indian ink (20) and methyl methacrylate (10)	NS	NS	NS	NS
Uz (2005)	30 h.	Colored latex	ICA: 100%	Single: 100%	0.7–1.2 (average: 0.94 mm)	NS

(continued)

Table 2 (continued)

Author (year)	Number of hemispheres/brains	Method	Origin	Number	Diameter (mm)	Length (cisternal segment; mm)
Akar (2009)	130 h.	Intraoperative observations	ICA: 100%	Single: 84.6% Double: 13% Triple: 2.4%	NS	NS
Tanriover (2014)	30 h.	Colored latex	ICA: 100%	Double: 3.3% (+1 pseudo-duplication)	NS	25.5 (18.4–36.3)
Antunovic (2017)	24 h.	Indian ink and gelatin. Paraffin embedded	NS	NS	NS	NS
Wang (2021)	54 h.	Angiography	NS	Double: 5.6%	0.3 mm to 1.6 mm (mean 0.8 mm)	NS

ACA anterior cerebral artery, *ICA* internal carotid artery, *Bif. ICA* bifurcation of the internal carotid artery (MCA/ACA), *MCA* middle cerebral artery, *PCoA* posterior communicating artery

Table 3 Anatomical characteristics of the AChA

Author	Origin (%)				Distance to PCoA (range). mm	Diameter (range). mm
	ICA	PCoA	Bif. ICA	MCA		
Beevor	100	0	0	0	–	–
Carpenter	76.6	6.7	3.3	11.7	–	–
Morello	90.2	8.7	0	1.1	–	–
Sjogren	95	3	0	2	–	–
Otomo	99.2	0.4	0.4	0	–	(0.4–1.5)
Herman	85	0	7	8	–	–
Saeki	100	0	0	0	2.7 (1.0–5.0)	1 (0.5–2.3)
Rhoton	98	2	0	0	2–5	–
Gibo	100	0	0	0	–	1 (0.5–2.1)
Hussein	97.5	2.5	0	0	3.2 (1.1–5.9)	0.9 (0.4–1.1)
Takahashi	100	0	0	0	NS	0.75 (0.38–2.0)
Erdem	100	0	0	0	4.2 (2.3–8)	0.93 (0.7–1.3)
Morandi	96	2	2	0	–	–
Uz	100	0	0	0	5.3 (3.8–8)	–
Tanriover	100	0	0	0	2.9 (2.1–3.7)	–

ACA anterior cerebral artery, *ICA* internal carotid artery, *Bif. ICA* bifurcation of the internal carotid artery (MCA/ACA), *MCA* middle cerebral artery, *PCoA* posterior communicating artery

aspect in 28%, and on the lateral side in 6%, lateral to the optic tract [25, 26, 44]. This observation is confirmed by more recent literature [38, 45].

The artery originates distal to the PComA, nearer to the origin of the PComA than to the carotid bifurcation. It is situated at a distance from the PComA which varies depending on the anatomical reports and the technique used, but which is usually a few millimeters (1–8 mm) distal to the PComA and proximal (2–7 mm) to the carotid bifurcation [38, 46]. A more distal origin of the artery, at the level of the carotid bifurcation (MCA/ACA), is described more rarely. Anatomical characteristics of the AChoA are summarized in Table 3.

Based on cadaveric descriptions, it is important to note that the choroidal segment of the ICA may also be the site of origin of other arterial branches. Tanriover et al. mentioned that as many as three branches, which may be as large as the AChoA itself, might arise from the posterior wall of the ICA, between the origin of the PComA and the AChoA [38]. More recently, Morandi et al. drew the same conclusion by referring to the possible presence of multiple branches (n = 1–4) [37]. This author specifies that the AChoA is the first branch to arise distally to the PComA origin in 40% of cases, the second in 48%, the third in 8%, and the fourth in 2% [37]. In 88% of the hemispheres studied by Erdem et al., the AChoA was the first branch of the ICA distal to the PComA, and the second in 12% [47]. In Hussein's series of cases, 8% of the specimens presented 1–3 small arteries (0.4–0.7 mm) arising from the infero-lateral carotid wall proximal to the AChoA's origin [46]. These branches were also reported in 10% and 32% of cases by Carpenter et al. and Rhoton et al., respectively [22]. They aim to supply the optic tract, the posterior perforated substance, and the medio-basal temporal lobe, but these latter were not observed in the series from Hussein et al. [46].

The duplication of the AChoA represents a minority of cases for which no consensus has been reached regarding the interpretation. The AChoA is reported as a single vessel in 89% of cases by Hussein et al. [46] and 96% according to Saeki et al., Rhoton et al., and Gibo et al. [25, 26, 44]. Rhoton et al. noted that if the AChoA can arise as two separate arteries in rare cases, its single trunk is divided immediately into two trunks in 47% of the hemispheres [25]. Hussein et al. reported a duplication in 7/140 specimens (5%) and a triplication in 4/140 specimens (2.9%) [46]. In contrast, Yasargil et al. encountered 2–4 independent choroidal vessels in 30% of their cases [48, 49]. This discrepancy may be explained by the different interpretations of the inferolateral carotid branches arising distally to the origin of the AChoA. Most of these arteries are variable in size and branch early to penetrate the uncal area. Hussein et al. used the course of the artery to define as the AchoA only such vessels running in the crural cistern and which are directed toward the optic tract or cross it [46]. Other authors consider "true" duplication as a rarity, and the previous cases as misinterpretations. Erdem et al. mentioned that they did not observe any duplication in this series [47]. However, in 20% of his cases, an early branch caused this artery to have the appearance of a duplicate origin. Tanriover et al. [38] reported a duplication in one case (3.3%) and mentions a pseudo-duplication related to a separate origin of the uncal artery. Lasjaunias et al. also refuted the assumptions of AChoA duplication. For these authors, multiple trunk origins of the artery probably represent the ICA's origin: an uncal or temporal amygdaloid branch distal to the AChoA. Like Lasjaunias et al., Morandi et al. considered it merely a pseudo-duplication, with a separation of the origin of the uncal artery [30, 37]. A clinical case of this variant is shown in Fig. 1. In more recent works, duplication of the AChoA is rare but exists and is underreported [50, 51].

Variations in AChoA origins are uncommon. In a minority of cases, AChoA can have an origin distinct from the ICA, including the PComA, bifurcation of the ICA (at the ACA/MCA level), or the MCA, the latter being controversial. In a large Japanese series of cases published by Otomo, 99.2% of AChoAs arose from the ICA, 0.4% from the PComA, and 0.4% from the junction of the ACA and MCA, but none from the MCA [42]. In two angiographic studies by Sjogren and Morello & Cooper, the AChoA originated from the PComA in 3% and 8% of the angiograms, and from the MCA in 2% and 1%, respectively [52, 53]. The artery originated from the ICA in 49 (98%) and from the PComA in one (2%) of the 50 hemispheres examined by Rhoton et al. [25].

A PComA origin of the AChoA is rare and reported in 0.2% to 8.7% of cases, the higher incidence being described in the angiographic works of Morello and Cooper [22, 25, 26, 37, 42, 46, 48, 52–54] and explained embryologically by the AChoA arising from the caudal division of the primitive ICA, rather than from the cranial one [30].

An ectopic origin from the bifurcation of the ICA (at the ACA/MCA level) represents a minority of cases and has been found in 0.3% to 7% of the observations [22, 42, 54, 55]. This variant can also easily be explained by various migrations or annexations of the AChoA distally during embryology.

The MCA origin of the AChoA can range from 2.5% to 58.5% but is a subject of controversy [22, 37, 54]. The high rates initially reported are probably incorrect and attributed to a non-use of the microscope and/or confusion of the AChoA with the uncal artery, which may arise from the proximal segment of the MCA. In the large case series of Otomo [42], none of the AChoAs originated from the MCA. For Hussein et al., this assumption also results from a false interpretation of the uncal artery, which may have a distinct MCA origin [46].

The AChoA's stem diameter is reported angiographically to be 0.38 to 2.0 mm and ranges from 0.7 to 2.0 mm in studies using anatomic dissection (Table 3) [12, 25, 37, 38, 45, 56].

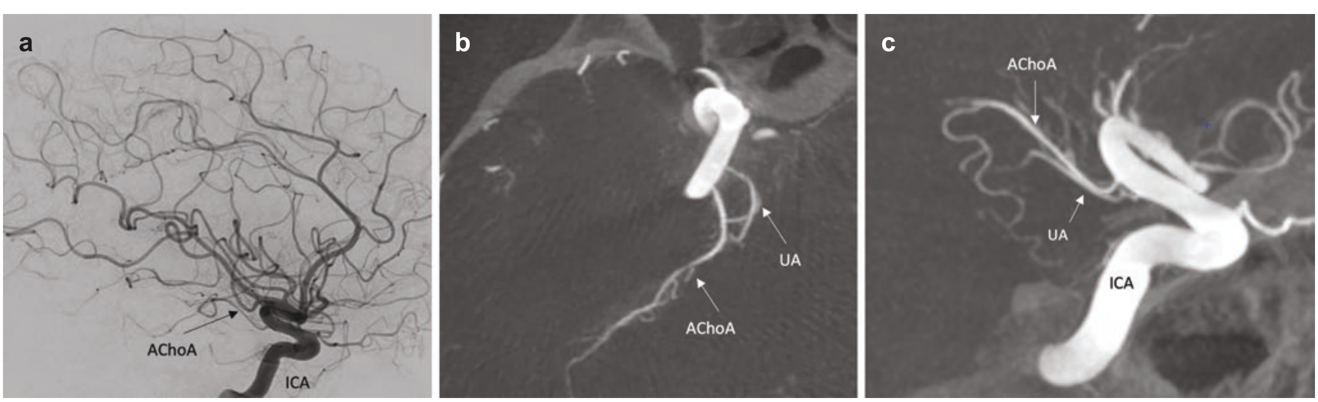


Fig. 1 Clinical case of separate origin of the AChoA and the uncal artery. the angiogram (**a**, **b** and **c**) shows a clinical case in which the anterior choroidal artery (AChoA) and the uncal artery (UA) have a distinct internal carotid artery (ICA) origin. Even if not shown in the figure, the vertebral artery injection did not show the presence of the posterior communicating artery

Course of the Artery

At its origin, the AChoA is located in the carotid cistern. It then successively goes through the crural and ambient cisterns before penetrating the choroidal fissure to supply the choroid plexus [46].

The artery's course has been subdivided into different segments for a comprehensive analysis. Goldberg and Rhoton et al. divide it into a *cisternal segment*, extending from its origin to the choroidal fissure, and a *plexal segment*, entering the choroidal fissure and penetrating the choroidal plexus of the temporal horn [25, 26, 40, 57]. An illustration of the segments of AChoA's segments is shown in Fig. 2.

Cisternal Segment

The cisternal (extra-ventricular) segment begins at the artery's origin at the ICA level and terminates at the inferior choroidal point corresponding to its entry in the choroidal fissure. Its length is ±25 mm (range: 11–36.3 mm) [22, 25, 38, 42, 55].

The artery initially runs within the carotid cistern in a posteromedial direction, posterior and lateral to the course of the PComA, behind the ICA. It reaches the lateral margin of the optic tract after passing below the anterior perforated substance. The segment of the artery crosses the optic tract inferiorly from a lateral to medial direction, and reaches its medial margin in most cases (87% and 83%, respectively), in the works of Tanriover et al. and Erdem et al. whereas this arterial segment goes further medially in a minority of cases (13.3% and 17%, respectively) [25]. During its eventual course over the anterior segment of the uncus, the artery passes along the semi-annular sulcus separating the anteriorly located ambient gyrus and the posteriorly located semilunar gyrus. After traveling posteromedially and passing under the optic tract, the artery reaches its most medial extension point. At this point, the artery diverges from the PComA and changes its course, creating an abrupt bend to enter the crural cistern. In the axial plane, the AChoA makes an abrupt turn from posteromedial to posterolateral. This medially located turning point, called the "genu" by Tanriover et al., serves as a reference point for this author to divide the cisternal segment into pre- and post-optic parts [38]. After

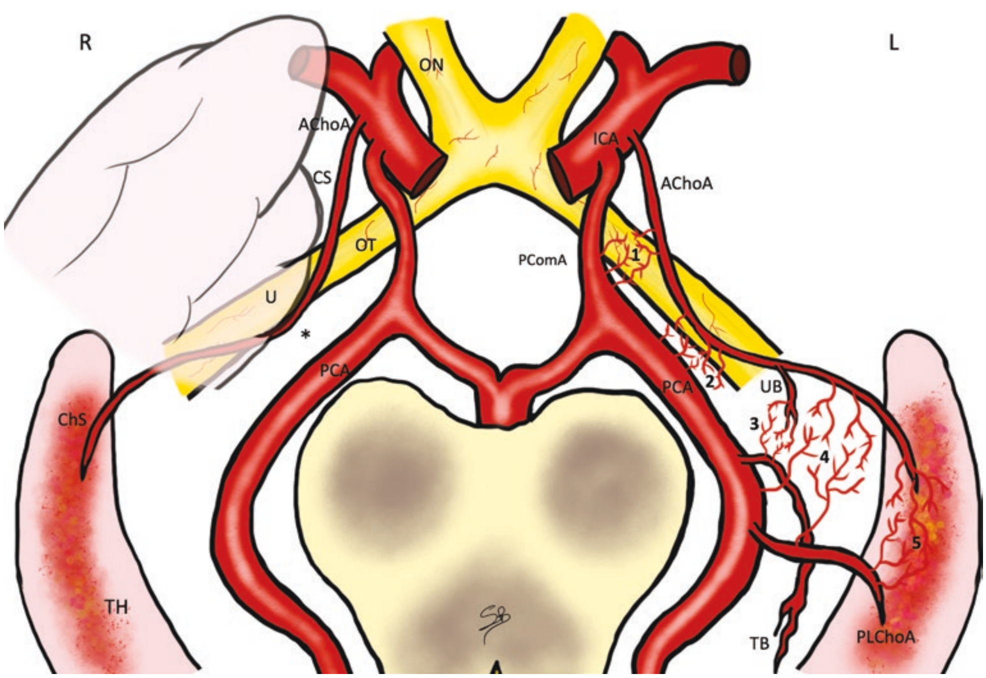


Fig. 2 Segments, branches, and possible anastomoses of the AChoA. On the right side (R), the figure shows the course and segmental division of the AChoA. After its origin from the internal carotid artery (ICA), the AChoA courses in its cisternal segment (CS) until its entry point into the temporal horn (TH) of the lateral ventricle, where it gives rise to its choroidal segment (ChS). The cisternal segment can be divided at the level of the genu (asterisk) into a pre- and post-optic part. At this point the AChoA runs along and below the optic tract (OT) and courses between the uncus (U) antero-laterally and the cerebral peduncle posteromedially. The main anastomoses of the AChoA can be summarized in 5 points. (*1*) Over the optic tract with branches of the posterior communicating artery (PComA); (*2*) Over the cerebral peduncle with the proximal posterior cerebral artery (PCA); (*3*) Over the piriform cortex with temporal (TB) and hippocampal branches of the PCA; (*4*) over and around the lateral geniculate body with PCA branches, including the lateral posterior choroidal artery (PChoA); (*5*) in the choroid plexus with posterior choroidal branches

this curve, the artery runs along and below the optic tract and passes posterolaterally within the crural cistern in the space between the posterior segment of the uncus anterolaterally and the cerebral peduncle posteromedial. The PCA and its branches, as well as the basal vein of Rosenthal, are posteromedial to the AChoA's course within the inferior portion of the crural cistern. After passing below the optic tract from a medial to lateral direction, the artery reaches the superior edge of the posterior limit of the uncus. The cisternal segment of the AChoA terminates at the inferior limit of the choroidal fissure named "the inferior choroidal point", at which point the artery passes to the temporal horn and becomes extra-cisternal to supply the choroid plexus.

As mentioned above, the cisternal extraventricular segment of the artery has itself been subdivided by some authors for a detailed and comprehensive analysis [38, 46]. Hussein et al. divided it according to its location in the carotid or crural cistern [46]. Tanriover et al. subdivided it into pre- and post-optic parts that meet at the artery's genu, which is the most medial extension point of the cisternal segment, where the artery makes an abrupt turn after passing under the optic tract [38]. When studying the microsurgical anatomy of the cisternal segment, Rhoton et al. divided it at the anterior margin of the lateral geniculate body into proximal and distal portions [25].

On an anteroposterior angiogram, the initial segment of the AChoA is medial to the ICA and takes a gentle S-shaped course on the lateral view in most cases [12].

Takahashi et al. [12, 23] evaluated the diameter of the cisternal segment to be 1 cm distal to the origin, and its size ranged from 0.38 to 2.0 mm (average: 0.75 mm) [12, 23]. It was judged as large (0.8 mm or more) in 33%, medium-sized (0.6–0.8 mm) in 51.8%, and small (less than 0.6 mm) in 14.2% of cases [12].

Plexal Segment

The plexal segment is composed of one or more branches passing through the choroidal fissure and entering the choroid plexus of the temporal horn. It courses along the medial border of the choroid plexus in close relation to the posterolateral choroidal branches of the PCA. Rhoton reports that in some cases, it can pass dorsally along the medial border of the plexus, reaching the foramen of Monro. Morandi et al. specified that most often the AChoA enters the temporal part of the choroidal fissure as a single trunk, 10 to 15 mm from the anterior end of the choroid plexus of the lateral ventricle's inferior horn [37]. At this level, the AChoA usually divides into medial and lateral plexal branches at the location designated by Morandi et al. as the "bifurcation of the AChoA" [37]. This "bifurcation" is well seen in the clinical case shown in Fig. 3. The medial branch is larger and travels along the attached border of the lateral ventricle's choroid plexus, then curves posterosuperiorly, often accompanied by the posterolateral and superior choroidal arteries [12].

Erdem et al. [47] describe specifically the course of the plexal segment and the variants observed at the level of its bifurcation. In his study, the plexal segment (named "choroid segment" by this author) bifurcates into medial perforating and lateral plexal branches in two ways. First, in 84% of the hemispheres, the plexal segment courses within the choroid fissure to supply the choroid plexus. The medial branches enter the brain around the lateral geniculate body and optic tract. In cases where the bifurcation of the choroid (plexal) segment into the medial perforating and lateral plexal branches take place within the choroid plexus, the plexal branch border the choroid plexus of the inferior horn laterally. The second branching pattern was observed

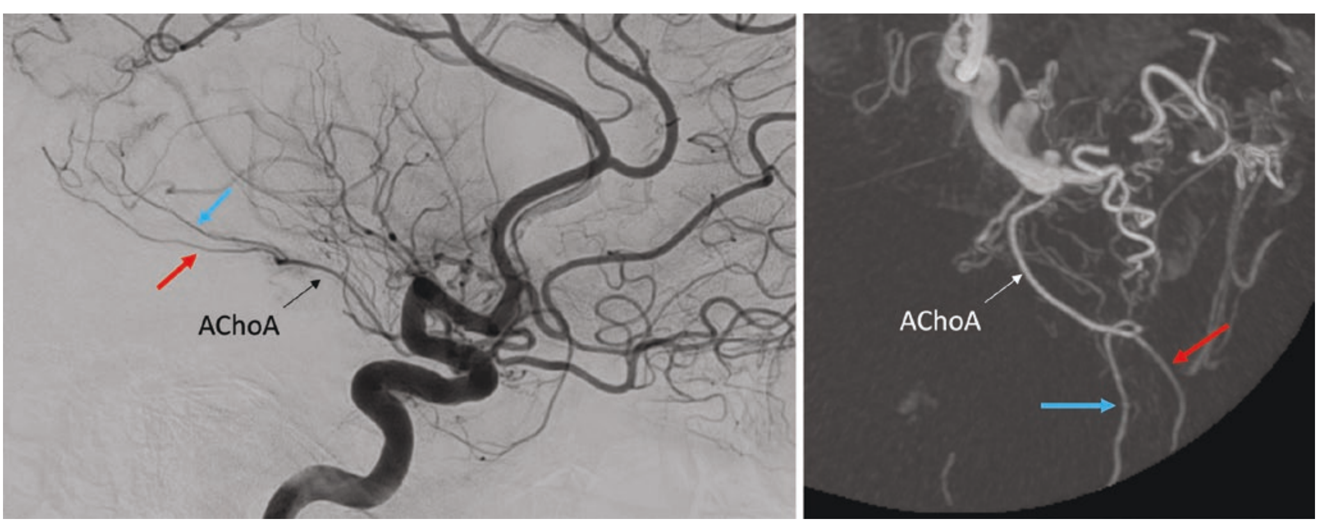


Fig. 3 The AChoA bifurcation. The figures show a patient with chronic occlusion of the middle cerebral artery. The AChoA is therefore well identified on the lateral view after selective injection of the internal carotid artery, especially the medial and lateral branches of the plexal segment (blue and red arrows)

in 16% of the hemispheres examined. In such configuration, the choroid (plexal) segment passes through the choroid fissure as a "trunk" and divides into lateral plexal and medial perforating branches within the choroid plexus. Erdem [47] mentions that in 13% of the cases the AChoA could be followed to or a few centimeters behind the foramen of Monro.

For Takahashi et al., hypoplasia of the plexal segment of the AChoA was found in 3% of cases and characterized by a lack of visualization of either the medial or lateral plexal branches on angiography [12].

The "plexal point", also known as the "ventral choroidal point", is an angiographic landmark at which the artery becomes intraventricular, appearing at the center of a checkmark-like course on lateral projections, where the vessel descends and then sharply ascends posteriorly [58].

Branches

Throughout its course, the AChoA gives origin to branches distributed to the closest structures. These branches vary in number (average: 9, range: 4–18) and size, which is why authors have categorized them according to the segment in which they originate, their location, and/or the structures they supply [25, 59]. Based on embryology, they can be categorized into three groups: diencephalic, telencephalic, and choroidal branches [30]. Tanriover et al. separated the AChoA into a phylogenetically old parenchymal ramification (branching parts of the artery), referred to as the cisternal segment, and its newly acquired choroidal ramification, referred to as the plexal segment [38].

As mentioned above, the vascularization of certain territories by the AChoA is in balance with adjacent territories. The caliber of AChoA branches will therefore depend on the presence of branches vascularizing the same territory from the ICA, MCA, or PCA [37].

AChoA branches are described based on the segment from which they originate.

Cisternal Segment

According to Morandi et al. six to 13 (average: 8) branches originate from this segment, arising mainly from its lateral aspect [37]. Their diameter ranges between 90 μm and 600 μm (mean: 317 μm) [59]. Tanriover et al. classified them into two groups based on their destination. The first gathers the branches supplying the anterior segment of the mesial temporal region, i.e., predominantly to the uncus, but also to the piriform cortex, amygdala, and head of the hippocampus. The second is composed of perforating branches supplying deep critical structures, such as the globus pallidus, posterior limb, retrolenticular part of the internal capsule, and tail of the caudate nucleus [38, 59].

The uncal artery is often reported to be the first, or sometimes the second, branch coming from the AChoA, the other being intended for the anterior perforated substance [59, 60]. Based on their observations, Hussein et al. described the uncal artery as a strong lateral branch arising from the main choroidal trunk 1–4 mm distal to its origin in 97% of the hemispheres examined [46]. This artery penetrates the uncal area and usually divides into 3–5 tiny branches supplying the head of the hippocampus. This author observes a reciprocal relationship between the uncal vessels arising from the ICA and MCA and the unco-hippocampal branches of the AChoA. In cases with developed uncal vessels from the ICA/MCA, 1–4 tiny unco-hippocampal branches from the proximal AChoA could be observed (13% of Hussein et al.'s cases) [46]. In contrast, when uncal arteries from the ICA/MCA were lacking, the AChoA supplied the unco-hippocampal area with a single larger branch (87% of Hussein et al.'s cases). The authors noted that this artery may be an important landmark for the identification of the optic tract because it originates 2–3 mm proximal to its crossing-point with the AChoA [46]. A case of uncal artery arising from the ICA instead of the AChoA is shown in Fig. 5.

Tanriover et al. aimed to detail and clarify the mesiotemporal vasculature from the AChoA by distinguishing three categories of arteries [38]. First, the anterior uncal arteries, also known as "anterosuperior parahippocampal arteries" or "rostral uncal arteries", which supply the ambient and semilunar gyri. Second, the middle uncal arteries, which supply the uncal apex. Third, the posterior uncal arteries, also named "medial uncal arteries" or "caudal uncal arteries", which supply the medial surface of the uncus's posterior segment formed by the hippocampal head's extraventricular part. These posterior uncal arteries arise from the post-optic/distal part of the cisternal segment.

The perforating arteries tend to arise from the superolateral aspect of the AChoA at its proximal cisternal segment (preoptic part) level and almost always run laterally along their course [38]. In contrast, the perforators are likely to arise from the inferomedial aspect of the AChoA at its distal cisternal segment (post-optic part) and most often course medially. The post-optic part is reported to have more perforating arteries supplying critical deep structures, including the optic tract and cerebral peduncles [38]. Marinkovic et al. described that the most distal perforator, usually single, most often originates close to the lateral geniculate body, just in front of the inferior horn of the ventricle [59]. This perforator, known as the capsulothalamic artery, is reported to be larger than other perforating vessels (200 to 610 μm, mean: 431 μm).

Several authors have classified the branching patterns and distributions of these AChoA branches. In 1974, Goldberg divided the medial vascular group into proximal branches to the substantia nigra, parts of the red nucleus, subthalamus, and the ventrolateral thalamic nuclei, and distal branches supplying the antero-lateral geniculate body [40]. Rhoton et al. described a) superior branches supplying the anterior and posterior perforate substance, optic tract, origin of the optic radiations, medial globus pallidus, and posterior limb of the internal capsule; b) inferolateral branches to the amygdaloid body, anterior hippocampus, and fascia dentata; and c) medial branches entering the cerebral peduncle and the lateral geniculate body [25]. In 1988, Hussein et al. categorized these arteries slightly differently: (a) superior branches supplying the anterior and posterior perforated substance, optic tract, origin of the optic radiations, medial globus pallidus, and posterior limb of the internal capsule; (b) inferolateral branches to the amygdaloid body, anterior hippocampus, and fascia dentata; and (c) medial branches entering the cerebral peduncle and the lateral geniculate body. These medial branches have been subdivided into two groups: proximal branches to the substantia nigra, parts of the red nucleus, subthalamus, ventrolateral thalamic nuclei, and distal branches, supplying the anterolateral geniculate body [40]. Morandi et al. categorized the perforators into superior, medial, and posterior branches [37]. The superior branches are essential to the optic tract, which they commonly perforate to supply the internal capsule and globus pallidus. The medial branches arise like the “teeth of a comb” at right angles to the distal two-thirds of the AChoA and supply the anterolateral part of the cerebral peduncle. The posterior branches arise from the distal part of the AChoA to supply the lateral geniculate body and sometimes the parahippocampal gyrus and posterior third of the optic tract.

As mentioned above, the vascularization of certain territories by the AChoA is in balance with adjacent territories. The caliber of its branches will therefore depend on the presence of branches vascularizing the same territory from the ICA, MCA, and PCA. For example, as pointed out by Tatu, the AChoA participates in the network of the superficial hippocampal arteries, which are issued from the PCA from its choroidal posterolateral, splenial, inferior, temporal branches, and from the AChoA [61]. It is rare that the arteries arise predominantly from the anterior choroidal artery [47].

Plexal (Ventricular) Segment

In most cases, the plexal segment originates as a single branch of the AChoA passing through the choroidal fissure. However, additional smaller branches from the choroid plexus may arise proximally to the choroidal fissure. The plexal branches divide into lateral and medial branches. They enter and run alongside the medial border of the choroid plexus of the temporal horn and frequently anastomose with branches of the lateral PChoAs [12]. At this level, the AChoA provides many branches to the choroid plexus of the lateral ventricle. Some branches may supply the distal part of the optic tract, lateral geniculate body, and thalamus [37, 47, 62]. Therefore, contrary to some previous reports stating that endovascular interventions on this segment are risk-free, the sacrifice of this segment represents a significant ischemic risk according to these authors [30, 63–65].

Possible Anastomoses

The AChoA has great anastomotic potential with the neighboring arteries, i.e., the PCA, PComA, and MCA.

Takhashi et al. categorized them and defined five anastomotic zones on the artery course [12] (Table 4, Fig. 2):

- Area 1. Over the optic tract with branches from the PcomA.
- Area 2. Over the cerebral peduncle with the proximal PCA.
- Area 3. Over the piriform cortex with PCA branches (temporal and hippocampal branches).
- Area 4. Over and around the lateral geniculate body with PCA branches, including the lateral PChoA.
- Area 5. In the choroid plexus with posterior choroidal branches.

Table 4 Anastomotic zones of the AChoA

Zone	Segment	Location	Anastomotic vessel
1	Cisternal (prox./preoptic)	Over optic tract	PCoA branches, ICA
2	Cisternal (prox./pre-post-optic junction)	Over cerebral peduncle	Proximal PCA branches
3	Cisternal (prox./post optic)	Over piriform cortex (uncal br.)	Temporal and hippocampal branches of PCA
4	Cisternal (distal/post-optic)	Over and around LGB	PCA branches including posterolateral choroidal artery
5	Plexal	Choroid plexus	Posterior choroidal artery

ACA anterior cerebral artery, *Ant. TA* anterior temporal artery, *ICA* internal carotid artery, *Ch. PL* choroid plexus, *LGB* lateral geniculate body, *LPCHA* lateral posterior choroidal artery, *LGB* lateral geniculate body, *OC* optic chiasm, *ON* optic nerve, *OT* optic tract, *PCA* posterior cerebral artery, *PCoA* posterior communicating artery, *PTA* posterior temporal artery, *Unc Br* uncal branch

Adapted from Takahashi, S., et al. (1990). AJNR Am J Neuroradiol **11**(4): 719–729 [12]

The two most important anastomotic zones are Areas 4 and 5. Following Morandi et al., anastomoses with the posterolateral choroidal arteries are constant and can be located on the lateral geniculate body (Area 4) or in the choroid plexus (Area 5). However, they did not find any connections with the posteromedial choroidal arteries as described by Wolfram-Gabel et al. [37, 66]. These authors consider, in agreement with Carpenter et al. but unlike Frisen et al., that anastomoses on the ventral surface of the lateral geniculate body are constant [22, 37, 67]. This rich anastomotic network was already described by Abbie in his works from 1933 detailing the vascularization of the lateral geniculate body, which was subdivided into "hilar anastomoses" and "intermediate and lateral anastomoses" [9, 13]. However, several authors have concluded that the richest anastomoses are those located on the surface of the choroid plexus with the lateral posterior choroidal branches of the PCA [25, 33, 37]. In addition, choroid plexus anastomoses can occur with the ACA branches at the interventricular foramen level [30].

Otherwise, anastomoses of small caliber are located over the anterior perforated substance, on the ventral aspect of the optic tract's proximal third, and on the anterior aspect of the cerebral peduncles. Hussein et al. noted that anastomoses with the PComA were encountered in 14% of their specimens and with ICA branches located between the origin of the PComA and the choroidal artery in 5% [46]. Anastomoses with superior branches of the proximal part of the PCA's post-communicating segment (P2 segment) on the cerebral peduncle's lateral aspect have also been reported [37]. The uncal branch of the AChoA frequently anastomoses with the anterior hippocampal artery deep inside the uncal sulcus and thus contributes to the vascularization of the hippocampal head. More occasionally, lateral branches anastomose with the lenticulo-striated arteries originating from the MCA [37].

These complex and variable anastomoses make it difficult to predict the effects of occlusion of a single AChoA but explain some of the inconsistent results of AChoA occlusion. Ligation of the AChoA, as proposed by Cooper in severe cases of Parkinsonism, was rapidly abandoned due to the high operative morbidity and mortality rates and poor long-term prognosis [16–19]. In contrast, occlusion of the AChoA at the level of the choroidal fissure may be well tolerated because there are rich anastomoses. As illustrated by Takahashi et al. [12], annexation or loss (transfer) of territory occurs more likely at these levels and will be detailed in the next paragraph [12].

Parenchymal Territories

As underlined by Padget, the AChoA supplies much more than its name implies, and throughout its course, this artery gives origin to branches distributed to the structures close to which it runs [35]. The territories supplied by the AChoA are in equilibrium with the territories of the perforating arteries (from the ICA, PCA, and MCA) and should be understood as a spectrum of possibilities with marked interchangeability in their field of supply, reinforced by the anastomotic network described above [25, 61, 68]. These points probably explain why the comparison of autopsies, pathological injection studies, and studies using neuroimaging demonstrates that the areas involved in AChoA infarctions are usually smaller than the AChoA territory inferred from experimental investigations [69]. Parenchymal territories supplied by the AChoA are summarized in Table 5.

Table 5 Parenchymal territories supplied by the AChA

Structure	Territory	Other sources of supply
Temporal lobe	Uncus, pyriform cortex, posteromedial part of the amygdaloid nucleus	ICA MCA
Visual system	Optic tract, portion of the lateral geniculate body, optic radiations	ICA PCoA
Internal capsule/ basal ganglia	Medial globus pallidus and caudate tail, genu and posterior part of the capsule	MCA P2 PCA
Diencephalon	Part of the lateral thalamic nuclear mass and subthalamus	P1-BA-PCoA
Midbrain	Middle one-third of the cerebral peduncle, substantia nigra, red nucleus	PCA-BA

BA basilar artery, *ICA* internal carotid artery, *PCA* posterior cerebral artery, *PCoA* posterior communicating artery

AChoA branches more commonly supply the optic tract, lateral part of the geniculate body, posterior two-thirds of the internal capsule's posterior limb, most of the globus pallidus, origin of the optic radiation, and middle third of the cerebral peduncle [25, 26, 43]. Less commonly supplied structures, or those showing more variability, include part of the head of the caudate nucleus, pyriform cortex, uncus, posteromedial part of the amygdaloid nucleus, substantia nigra, red nucleus, subthalamic nucleus, and superficial aspect of the ventrolateral nucleus of the thalamus. Based on Rhoton et al.'s works from 1979, none of these structures is always supplied by the artery, but in approximately two-thirds of the hemispheres, it supplies the medial part of the globus pallidus, the posterior limb, the retrolenticular part of the internal capsule, the optic tract, and the lateral geniculate body [25]. The choroid plexus of the temporal horn was the only structure to receive branches in every case. In approximately half of the hemispheres, it supplies the lateral part of the globus pallidus and the caudate tail; and in one-third, it supplies the thalamus, hypothalamus, and subthalamus.

Lasjaunias et al. categorized the supplied territory of the AChoA into two parts: one belonging to the cranial division of the ICA and supplying the paleostriatum and piriform cortex, and the other belonging to the caudal division and supplying the remainder of the anterior choroidal territories [30]. The cranial component is mainly paleostriatal regarding its distribution and supplies, in addition to the piriform cortex, part of the head of the caudate nucleus and most of its tail, the posteromedial part of the amygdaloid nucleus, and most of the globus pallidus. Variations in this territory involve the MCA branches. The caudal component supplies the optic tract and lateral geniculate body, middle third of the crus cerebri, subthalamic region, posterior two-thirds of the posterior limb of the internal capsule, optic radiation, anteroinferior part of the fascia dentata, hippocampus, and choroid plexus. Associated variations involve the PCA. Notably, Rhoton et al. stated that if there is a double AChoA, the distal branch usually terminates in the temporal lobe, and the proximal branch nourishes the remaining anterior choroidal field [25].

The vascularization of the uncus illustrates this concept of interchangeability. In the work of Isolan et al., the AChoA provided most of the branches that supply the uncus, with an average of 2.79 branches per hemisphere, or 38% of all the branches reaching this particular anatomical structure [70]. The MCA was the second largest contributor of branches to the uncus, with a more substantial contribution of ICA and PCA perforators [70].

The contribution of the AChoA to the supply of the thalamus is a matter of debate [69]. Kolisko found a supply to the superior external portion of the thalamus in some cases, and Abbie reported that the AChoA distribution frequently included the superficial part of the ventrolateral thalamus [6, 9, 13, 14]. Helgason et al. also reported an involvement of the posterolateral thalamus in some AChoA infarction cases [71, 72]. In contrast, Beevor did not demonstrate any supply from the AChoA to the thalamus, unlike some investigators who focused on its vascularization [7, 69]. In summary, Mohr et al. [69] concluded that although superficial involvement of the thalamus in AChoA infarctions may occur, extension deep into the lateral thalamus must be considered exceptional.

Finally, it is worth mentioning that the AChoA does not have any dural territory in the absence of any pathological context.

Variants

The presence of the AChoA seems to be a constant based on numerous anatomical observations. As mentioned above, the reported absence of the AChoA may correspond to the following variants: the artery considered as the PComA is a dominant AChA (i.e., an AChoA with parieto-occipital territory) or an AChoA arising from the PComA [12, 23, 25, 37, 42].

The anatomical variants reported concern the origin and the territories vascularized by the AChoA. Considering the importance of these territories, careful attention must be paid to these variations.

Origin

- Common PComA/AChoA origin: such an anatomical variant is relatively rare (0.2%–6.7%) and is due to the AChoA arising from the caudal branch of the primitive ICA [37, 73]. A clinical case of this variant is shown in Fig. 4.
- The AChoA originates from ICA (ACA/MCA) bifurcation: it represents a minority of cases (0.3%–7%). This variant can also be explained by various migrations or annexations of the AChoA distally during embryology [37].
- The AChoA originates from the ICA proximal to the PComA; this is rare and reported in only a few cases (six case reports) [74–79]. The genesis of this anomaly is unknown and unexplained from a developmental point of view.

Territories and Size

Variations in territories (and consequently in artery size) relate to AChoA-PCA connections [80]. From an embryological point of view, the AChoA initially has a large telencephalic territory, but it is lost in the early stages of embryogenesis by transfer (distal annexation) to the caudal branch of the ICA, which becomes the PCA [31, 32]. As noted by Lasjaunias et al., the distal annexations may or may not follow the truncal changes [30] and may be partial or total, explaining the wide variety of equilibria at the cortical level between the PCA and AChoA. These annexations or losses of territory occur more likely at the level of anastomotic zones [81–83].

The most commonly encountered variants are the capture of the inferior temporal branch by the AChoA, which supplies the undersurface of the temporal lobe. In such configuration, the parieto-occipital and calcarine branches typically originate from their "usual" positions. Less frequently, the AChoA can capture the PCA's entire cortical territory [12, 30, 37, 84, 85]. In this case, the AChoA should not be confused with a fetal PComA [86].

Takahashi et al. proposed a classification of these variations by dichotomizing hyperplastic and hypoplastic arteries with a prevalence of, respectively, 3% and 2.3% [12].

Hyperplastic anomalies of the AChoA supplying the inferomedial temporo-occipital region, including the territory of the AChoA and part of the PCA territory, are subdivided into four subtypes (Table 6). For this author, they

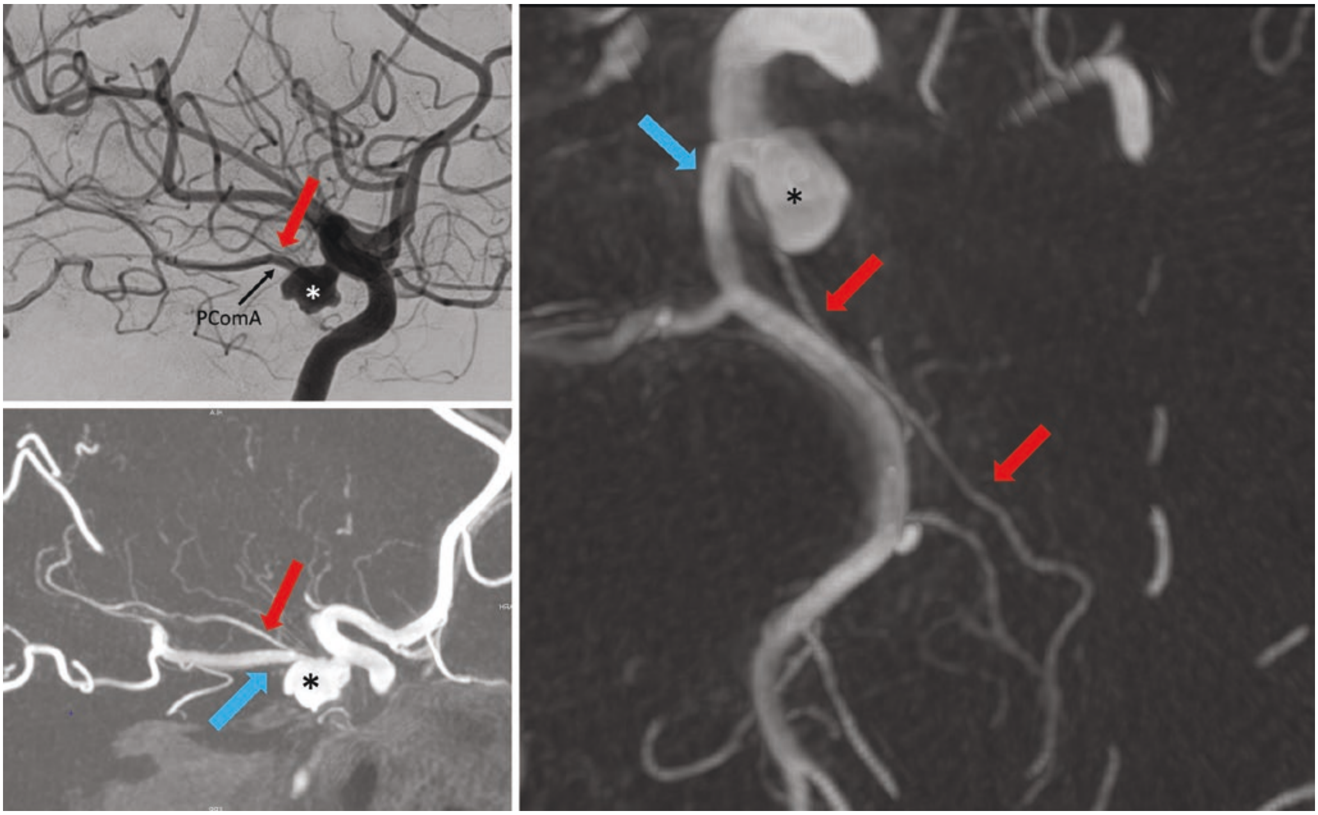


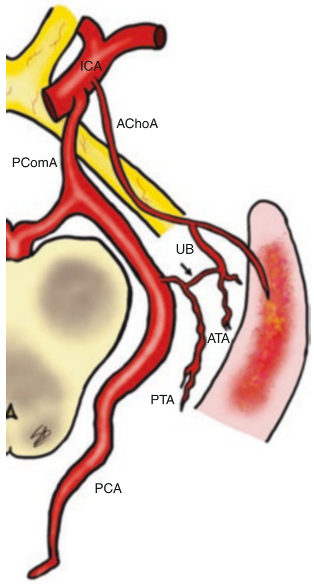
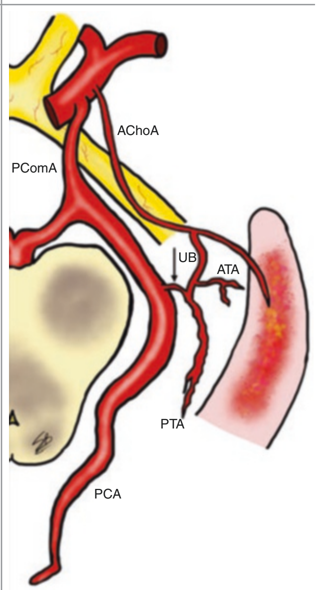
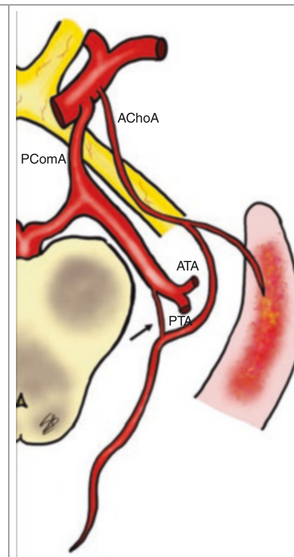
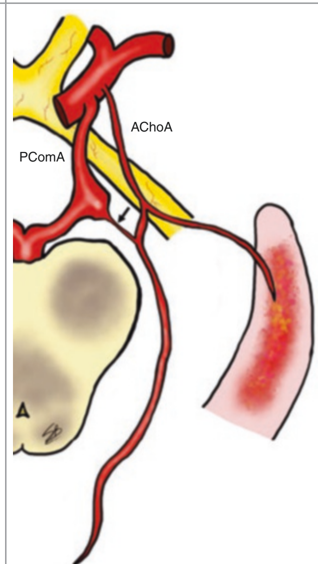
Fig. 4 Clinical case of common origin of the PComA and AChoA. Patient with fetal configuration of the posterior communicating artery (PComA, blue arrows) and PComA aneurysm. The anterior choroidal artery (red arrow) originates from the proximal segment of the PComA

represent a situation in which one of the anastomoses remains and enlarges as a main pathway of the artery, while a segment of the PCA just proximal to the anastomosis eventually attenuates. A case of hyperplastic variant of the AChoA is shown in Fig. 5.

A hypoplastic AChoA corresponds to a hypoplastic aspect, i.e., a lack of visualization on angiography, of the plexal segment [12]. In this case, the choroid vascularization is taken in charge by the PCA. According to Takahashi et al., it represents the "reptilian" stage of evolution, as mentioned above in our paragraph about embryology [12].

Doi et al. recently added a rare variant characterized by AChoA/PComA "fusion" [87]. In this variant, there is a hyperplastic aspect of the AChoA with a connection between the AChoA and PCA at the level of P2. It is considered to belong to the spectrum of the choroidal supply of the PCA territory, except that there is a persistent connection between the two at the level of the P2 segment.

Table 6 Takahashi's classification of the hyperplastic variants of the AChoA

Type 1	Hypetrophic uncal branch: Persistent anastomosis (arrow) between the uncal branch of the AChoA and the anterior temporal branch of the PCA	
Type 2	Anomalous temporal artery: 2a: Persistent anastomosis (arrow) between the uncal branch of the AChoA and a common stem of the temporal branches of the PCA 2b: Persistent anastomosis between somewhere in the surface area of the medial temporal region 2c: Persistent anastomosis deep in the choroid fissure at the inferior horn of the lateral ventricle	
Type 3	Anomalous occipito-parietal artery: Persistent anastomosis (arrow) with the PCA distal to branching of its temporal arteries	
Type 4	Anomalous temporo-occipito-parietal artery: Persistent anastomosis (arrow) at level of PComA or at anterior ambient segment of PCA	

AChoA anterior choroidal artery, *ATA* anterior temporal artery, *ICA* internal carotid artery, *PCA* posterior cerebral artery, *PComA* posterior communicating artery, *PTA* posterior temporal artery, *UB* uncal branch

Adapted from Takahashi, S., et al. (1990). AJNR Am J Neuroradiol 11(4): 719–729 [12].

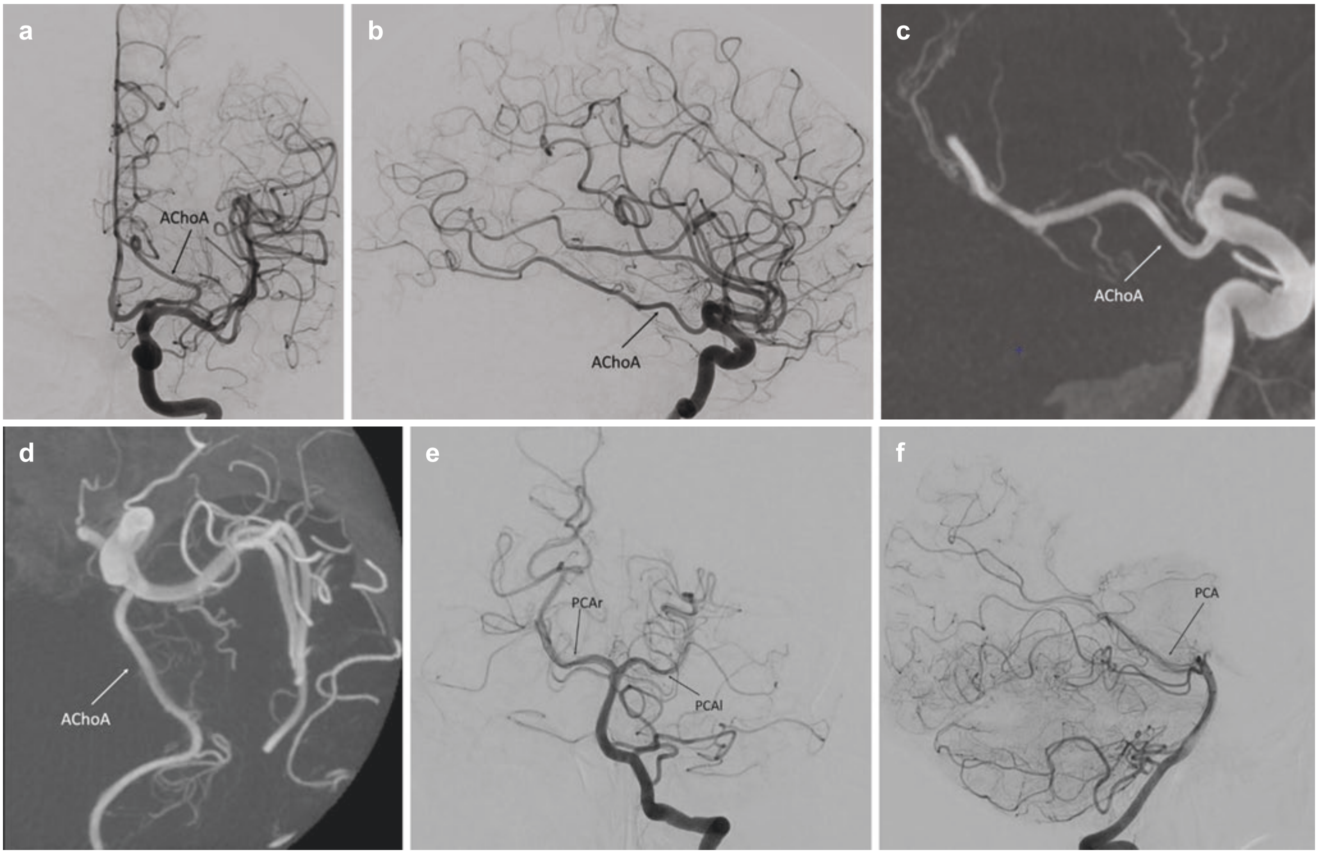


Fig. 5 Clinical case of hyperplastic variant of the AChoA. Figures(**a–f**) show a DSA image of a hyperplastic AChoA. The left AChoA has an anastomosis with the posterior cerebral artery (PCA) distal to its temporal branches and supplies its distal vascular territory. In the antero-posterior view of VA injection (figure **e**), it is visible that the left PCA (PCA l) keeps only its temporal territory. This case was interpreted as a type III according to Takahashi's classification

Clinical Implications

Despite its small size, the AChoA is a critical artery in brain physiology and function. Therefore, the clinical implications are numerous.

Stroke

Although Kolisko had already outlined the typical neurological syndrome in AChoA infarctions, the first specific description of AChoA artery stroke is attributed to Foix in 1925 [6, 39].

The possible clinical presentations of AChoA infarcts are as follows: the classical triad includes hemiplegia, hemisensory loss (hemianesthesia), and homonymous hemianopia. Lacunar syndromes, ataxic hemiparesis, neglect, language disorders, and sectoranopia have also been reported [69, 71, 72, 88]. The contralateral hemiplegia and hemianesthesia, for all sensory modalities, result from infarction in the posterior limb of the internal capsule's posterior two-thirds and the middle third of the cerebral peduncle. The homonymous hemianopsia to varying degrees results from interruption of the supply to the origin of the optic radiation, the optic tract, and part of the lateral geniculate body. Transient loss of consciousness, as well as extrapyramidal signs, may also occur following an ischemic lesion in the pallidal area. A rarer clinical presentation of hemichoreathetosis has been reported [89, 90].

Inconstant results, including the absence of deficit, have followed proximal AChoA surgical occlusion for the treatment of Parkinson's disease [16–20, 91]. On the contrary, as discussed above, the occlusion of the AChoA at the level of the choroidal fissure may be well tolerated because there are rich anastomoses between the AChoA and the posterolateral choroidal artery.

As highlighted by Mohr et al., who used CT and MRI to map the area of AChoA infarctions and study the variations in the AChoA territories, the area involved in AChoA infarction is usually smaller than the full territory inferred from experimental investigations. The restricted infarct size may reflect the effects of collateral supply to the AChoA territory from branches of the PCA, PComA, and MCA [69, 92].

Regarding the etiology of AChoA strokes, it has been suggested by some authors that those result more frequently from small-vessel disease [93, 94]. This assumption is subject to controversy and does not seem to be a diagnostic value regarding etiology [95].

Aneurysm

AChoA aneurysms represent 2% to 5% of intracranial aneurysms [36, 37, 96]. They usually arise at its origin with a dorso- or ventrolateral orientation and are thus usually intimately related to the medial part of the temporal lobe, often embedded in the uncus. Treatment of these can be achieved endovascularly or surgically. Ischemic stroke is reported to be the most common complication after clipping with a morbi-mortality varying from 5% to 50%, with lower upper rates in the contemporary case series [49, 96–98]. When compared with the surgical literature, endovascular series seem to have a lower incidence of stroke, although only a few have been reported [96, 99, 100]. Recently, the use of flow-diverting stents has been evaluated for the treatment of intracranial aneurysms. The use of this endovascular material eliminating, or at least making optional, the need to place coils in the aneurysm sac does not seem to be associated with AChoA ischemic complications [101].

Of note, distal AChoA aneurysms occurring without the context of arteriovenous malformations (AVM) or Moyamoya disease are rare but should be considered in patients with isolated medial temporal intracerebral hematoma with intraventricular extension [102].

Moyamoya Disease

When the AChoA is not involved in Moyamoya disease, the trunk and branches of the AChoA often develop compensatory dilation and extend to distal regions, thereby forming collateral vessels that increase blood flow and, consequently, the risk of distal flow-related aneurysm [103, 104]. As mentioned in the case series of Morioka, dilatation and abnormal branching of the AChoA are strong predictors of intracranial hemorrhagic events [105].

Vein of Galen Aneurysmal Malformation (VGAM) and Congenital Diseases

Vein of Galen aneurysmal malformations is particularly often associated with a full persistence of the limbic arterial arch, which bridges the cortical branch of the AChoA initially and the PCA secondarily with the pericallosal artery. This arch was first described by Moffat in 1961 and usually represents a transient stage during embryology [31, 32]. Lasjaunias et al. described two types of persisting limbic arch: the first, or “true” limbic arch, links the AChoA to the ACA around the limbic structures, and in the second, the PCA takes over the role of the AChoA [30]. In the case series of Lasjaunias et al., half of the patients diagnosed at a fetal or neonatal age or in infancy and 30% of children with VGAM demonstrate a persistent limbic arch. Both the mural and choroidal types of VGAM are associated with this abnormality, although the former is twice as frequent [106]. The circle regresses after obliteration of the VGAM by embolization.

Holoprosencephalic malformations are associated with an enlarged AChoA (and abnormal ACA fusions), but the presence of a limbic arch is not reported [107].

Arteriovenous Malformations

The AChoA is a classic feeder for AVMs located in its vascularization territory, and is reported to be a constant feeder of lateral ventricular AVMs [108]. Knowledge of the variants described above is necessary to anticipate the potential complications of endovascular or surgical treatment of these AVMs [58, 108–110]. The authors emphasize the importance of a thorough understanding of the functional anatomic structures supplied by each segment of the artery to decrease the ischemic complication rate. Regarding endovascular treatment, the importance of correct catheter positioning and changes in flow dynamics during embolization are reported to be major prognosis factors. It is worth mentioning that the plexal segment can, in some cases, provide branches for eloquent territory, such as the thalamus, when using this landmark for microcatheter positioning [111, 112]. A clinical case of choroidal AVM supplied by the AChoA is shown in Fig. 6.

Brain Tumors

Tumors that may affect the AChoA include choroid papilloma, gliomas, and, more rarely, meningiomas [36, 113–115].

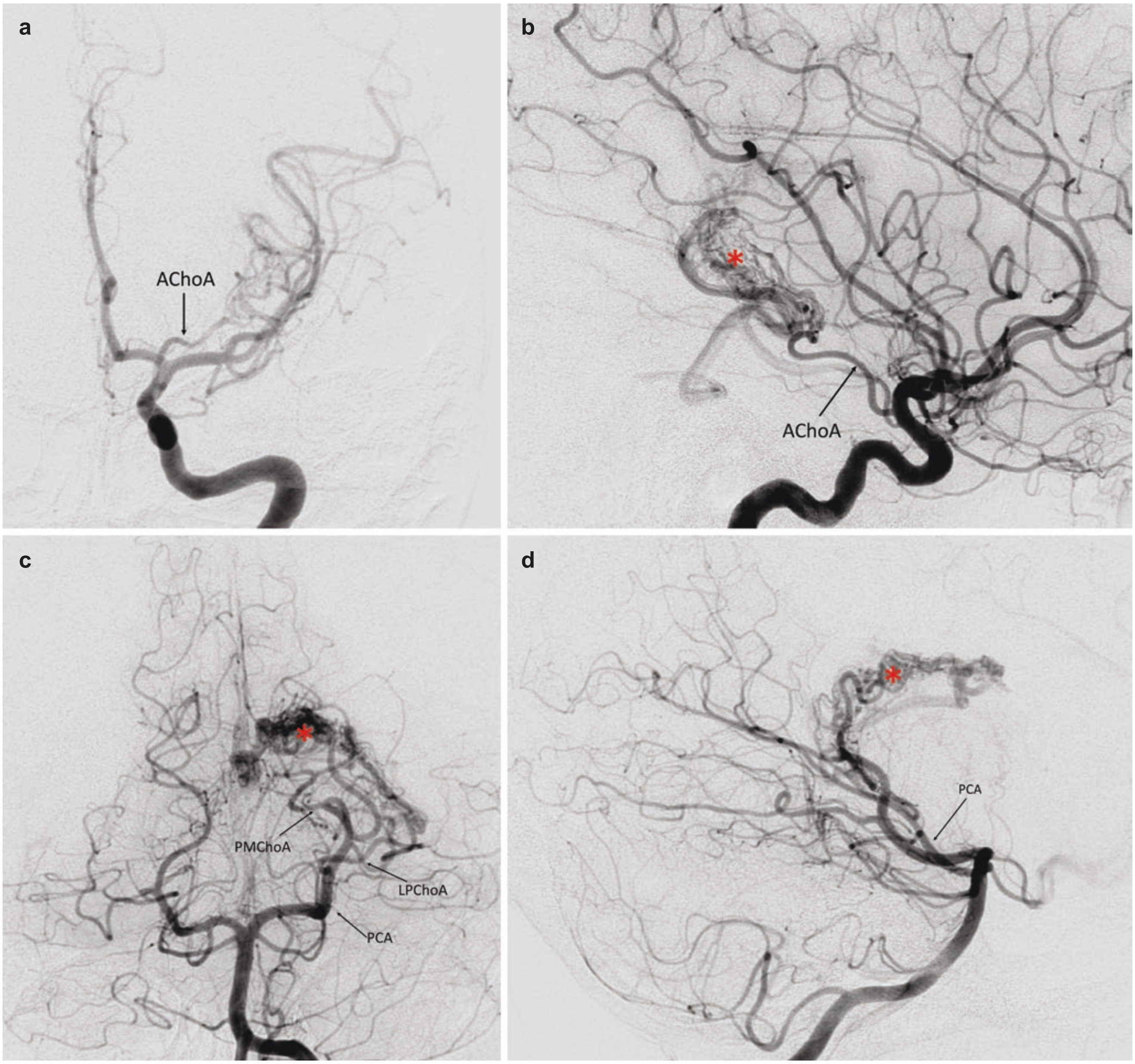


Fig. 6 Clinical case of choroidal AVM. Figures (**a–d**) show a clinical case of choroidal arterio-venous malformation. Figures a and b show respectively an antero-posterior view and a lateral view obtained after left internal carotid artery injection. Both show the supply of the AVM provided by the anterior choroidal artery (AChoA). Figures (**c** and **d**) show antero-posterior and lateral views of left vertebral artery injection. These figures show the contribution to the AVM of the medial and lateral posterior choroidal arteries (PMChoA, LPChoA)

Conclusion

The complex anatomy of the AChoA and its variants may have important therapeutic implications due to the eloquent territories vascularized by this artery. Therefore, a comprehensive understanding of its characteristics is mandatory in the management of neurovascular pathologies.

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References

1. Vicq-d'Azyr. Traité d'anatomie et de physiologie, avec des planches coloriées représentant au naturel les divers organes de l'homme et des animaux. 1786.
2. Vicq-d'Azyr. Traité de l'anatomie du Cerveau, Nouvelle Edition. Paris; 1813.
3. von Haller A. Anatomical description of the arteries of the human body, illustrated by several coloured engravings. Cox: Selected and Reduced from the Icones of Haller. E; 1813.
4. Heubner. Zur Topographie der einzelnen Hirnarterien. Centralblatt der medicinischen Wissenschaften. 1872.

5. Duret. Recherches anatomiques sur la circulation de l'encéphale. Arch Physiol. 1874;I:60–91.
6. Kolisko. Über die Beziehung der Arteria choroidea anterior zum hinteren Schenkel der innern Kapsel des Gehirnes. Wien. 1891.
7. Beevor. The cerebral arterial supply. Brain. 1908;30:403–25.
8. Beevor CE. On the distribution of the different arteries supplying the human brain. Philos Trans R Soc B. 1907;200:1–55.
9. Abbie AA. The clinical significance of the anterior choroidal artery. Brain. 1933;56(3):233–46. https://doi.org/10.1093/brain/56.3.233.
10. von Monakow C. Gehirnpathologie. Wien, 1905, 6, 1086. 1905(6):1085–92.
11. Blackburn. Anomalies of the encephalic arteries among the insane. J Comp Neurol Psychol. 1907;17:493–517.
12. Takahashi S, Suga T, Kawata Y, Sakamoto K. Anterior choroidal artery: angiographic analysis of variations and anomalies. AJNR Am J Neuroradiol. 1990;11(4):719–29.
13. Abbie AA. The blood supply of the lateral geniculate body, with a note on the morphology of the choroidal arteries. J Anat. 1933;67(Pt 4):491–521.
14. Abbie AA. The morphology of the fore-brain arteries, with especial reference to the evolution of the basal ganglia. J Anat. 1934;68(Pt 4):433–70.
15. Padget DH. The cranial venous system in man in reference to development, adult configuration, and relation to the arteries. Am J Anat. 1956;98(3):307–55. https://doi.org/10.1002/aja.1000980302.
16. Cooper IS. Ligation of the anterior choroidal artery for involuntary movements; parkinsonism. Psychiatry Q. 1953;27(2):317–9. https://doi.org/10.1007/BF01562492.
17. Cooper IS. Effect of anterior choroidal artery ligation on involuntary movements and rigidity. Trans Am Neurol Assoc. 1953;3(78th Meeting):6–7; discussion, 8–9
18. Cooper IS. Surgical alleviation of parkinsonism; effects of occlusion of the anterior choroidal artery. J Am Geriatr Soc. 1954;2(11):691–718. https://doi.org/10.1111/j.1532-5415.1954.tb02479.x.
19. Cooper IS. Surgical occlusion of the anterior choroidal artery in parkinsonism. Surg Gynecol Obstet. 1954;99(2):207–19.
20. Cooper IS. A symposium on anterior choroidal artery occlusion and chemopallidectomy in the treatment of Parkinson's disease. J Am Geriatr Soc. 1956;4(12):1167.
21. Das K, Benzil DL, Rovit RL, Murali R, Couldwell WT, Irving S. Cooper (1922-1985): a pioneer in functional neurosurgery. J Neurosurg. 1998;89(5):865–73. https://doi.org/10.3171/jns.1998.89.5.0865.
22. Carpenter MB, Noback CR, Moss ML. The anterior choroidal artery; its origins course, distribution, and variations. AMA Arch Neurol Psychiatry. 1954;71(6):714–22. https://doi.org/10.1001/archneurpsyc.1954.02320420042005.
23. Takahashi M, Arii H, Tamakawa Y. Anomalous arterial supply of temporal and occipital lobes by anterior choroidal artery: angiographic study. Am J Neuroradiol. 1980;1(6):537–40.
24. Takahashi S. Studies on choroidal blood blow with a hydrogen clearance method. II. Comparison of blood flow in the choroid and other tissues (author's transl). Nippon Ganka Gakkai Zasshi. 1981;85(9):1426–31.
25. Rhoton AL Jr, Fujii K, Fradd B. Microsurgical anatomy of the anterior choroidal artery. Surg Neurol. 1979;12(2):171–87.
26. Saeki N, Rhoton AL Jr. Microsurgical anatomy of the upper basilar artery and the posterior circle of Willis. J Neurosurg. 1977;46(5):563–78. https://doi.org/10.3171/jns.1977.46.5.0563.
27. Lasjaunias P, tBK, Berenstein A. Surgical neuroangiography: clinical and interventional aspects in children. Berlin: Springer; 2006.
28. Lasjaunias PL. Segmental identity and vulnerability in cerebral arteries. Interv Neuroradiol. 2000;6(2):113–24. https://doi.org/10.1177/159101990000600205.
29. Wiesmann M, Yousry I, Seelos KC, Yousry TA. Identification and anatomic description of the anterior choroidal artery by use of 3D-TOF source and 3D-CISS MR imaging. AJNR Am J Neuroradiol. 2001;22(2):305–10.
30. Lasjaunias PLBA, terBrugge K. Surgical neuroangiography, vol. 1. Clinical vascular anatomy and variations. Berlin: Springer; 2001.
31. Moffat DB. The development of the posterior cerebral artery. J Anat. 1961;95:485–94.
32. Moffat DB. The embryology of the arteries of the brain. Ann R Coll Surg Engl. 1962;30:368–82.
33. Galatius-Jensen F, Ringberg V. Anastomosis between the anterior choroidal artery and the posterior cerebral artery demonstrated by arteriography. Radiology. 1963;81:942–4. https://doi.org/10.1148/81.6.942.
34. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: a pictorial review. Surg Radiol Anat. 2021;43(6):961–73. https://doi.org/10.1007/s00276-021-02684-y.
35. Padget DH. The development of the cranial arteries in the human embryo. Contribution to embryology. Carnegie Institution; 1948. p. 205–61.
36. Yu J, Xu N, Zhao Y, Yu J. Clinical importance of the anterior choroidal artery: a review of the literature. Int J Med Sci. 2018;15(4):368–75. https://doi.org/10.7150/ijms.22631.
37. Morandi X, Brassier G, Darnault P, Mercier P, Scarabin JM, Duval JM. Microsurgical anatomy of the anterior choroidal artery. Surg Radiol Anat. 1996;18(4):275–80. https://doi.org/10.1007/BF01627605.
38. Tanriover N, Kucukyuruk B, Ulu MO, Isler C, Sam B, Abuzayed B, et al. Microsurgical anatomy of the cisternal anterior choroidal artery with special emphasis on the preoptic and postoptic subdivisions. J Neurosurg. 2014;120(5):1217–28. https://doi.org/10.3171/2014.1.JNS131325.
39. Foix C. Oblitération de l'artère choroïdienne antérieure: Ramollissement cérébral, hémiplégie, hémianesthésie et hémianopsie. Bull Soc Ophthalmol Paris. 1925;37:221–3.
40. Goldberg. The anterior choroidal artery. Radiology of the skull and brain. 1974.
41. Friedman JA, Pichelmann MA, Piepgras DG, Atkinson JL, Maher CO, Meyer FB, et al. Ischemic complications of surgery for anterior choroidal artery aneurysms. J Neurosurg. 2001;94(4):565–72. https://doi.org/10.3171/jns.2001.94.4.0565.
42. Otomo E. The anterior choroidal artery. Arch Neurol. 1965;13(6):656–8. https://doi.org/10.1001/archneur.1965.00470060092009.
43. Fujii K, Lenkey C, Rhoton AL Jr. Microsurgical anatomy of the choroidal arteries: lateral and third ventricles. J Neurosurg. 1980;52(2):165–88. https://doi.org/10.3171/jns.1980.52.2.0165.
44. Gibo H, Lenkey C, Rhoton AL Jr. Microsurgical anatomy of the supraclinoid portion of the internal carotid artery. J Neurosurg. 1981;55(4):560–74. https://doi.org/10.3171/jns.1981.55.4.0560.
45. Uz A, Erbil K, Esmer A. The origin and relations of the anterior choroidal artery: an anatomical study. Folia Morphol (Warsz). 2005;64(4):269–72.
46. Hussein S, Renella RR, Dietz H. Microsurgical anatomy of the anterior choroidal artery. Acta Neurochir. 1988;92(1–4):19–28. https://doi.org/10.1007/BF01401968.
47. Erdem A, Yasargil G, Roth P. Microsurgical anatomy of the hippocampal arteries. J Neurosurg. 1993;79(2):256–65. https://doi.org/10.3171/jns.1993.79.2.0256.
48. Yasargil MG. Microneurosurgery. Stuttgart/New York: Thieme; 1984.

49. Yasargil MG, Yonas H, Gasser JC. Anterior choroidal artery aneurysms: their anatomy and surgical significance. Surg Neurol. 1978;9(2):129–38.
50. Artov M, Iwanaga J, Korndorffer ML, Dumont AS, Tubbs RS. Duplicated anterior choroidal arteries: literature review and clinical implications. Cureus. 2021;13(7):e16291. https://doi.org/10.7759/cureus.16291.
51. Chenin L, Chivot C, Toussaint P, Deramond H, Peltier J. An unusual, duplicate origin of the anterior choroidal artery with aneurysm: a case report. Surg Radiol Anat. 2015;37(10):1273–5. https://doi.org/10.1007/s00276-015-1499-3.
52. Morello A, Cooper IS. Arteriographic anatomy of the anterior choroidal artery. Am J Roentgenol Radium Therapy, Nucl Med. 1955;73(5):748–51.
53. Sjogren SE. The anterior choroidal artery. Acta Radiol. 1956;46(1–2):143–57. https://doi.org/10.3109/00016925609170823.
54. Von Mitterwallner F. Variationsstatistische untersuchungen an den basalen hirngefässen. Cells Tissues Organs. 1955;24(1):51–87. https://doi.org/10.1159/000141019.
55. Herman LH, Fernando OU, Gurdjian ES. The anterior choroidal artery: an anatomical study of its area of distribution. Anat Rec. 1966;154(1):95–101. https://doi.org/10.1002/ar.1091540109.
56. Wollschlaeger G, Wollschlaeger PB, Meyer PG, Krautmann JJ. Widening or hyperplasia of the anterior choroidal artery. Criteria for the diagnosis. Radiology. 1969;93(5):1079–83. https://doi.org/10.1148/93.5.1079.
57. Rhoton AL Jr. The supratentorial arteries. Neurosurgery. 2002;51(4 Suppl):S53–120.
58. Hodes JE, Aymard A, Casasco A, Rufenacht D, Reizine D, Merland JJ. Embolization of arteriovenous malformations of the temporal lobe via the anterior choroidal artery. AJNR Am J Neuroradiol. 1991;12(4):775–80.
59. Marinkovic S, Gibo H, Brigante L, Nikodijevic I, Petrovic P. The surgical anatomy of the perforating branches of the anterior choroidal artery. Surg Neurol. 1999;52(1):30–6. https://doi.org/10.1016/s0090-3019(99)00043-9.
60. Marinkovic S, Gibo H, Erdem A. Huge Uncal branch of the anterior choroidal artery. Neurol Med Chir. 1994;34(7):423–8. https://doi.org/10.2176/nmc.34.423.
61. Tatu L, Vuillier F. Structure and vascularization of the human hippocampus. Front Neurol Neurosci. 2014;34:18–25. https://doi.org/10.1159/000356440.
62. Zhang N, Xin WQ, Yue SY, Zhong Y, Wei W, Yang XY, et al. Using a new landmark of the most external point in the embolization of distal anterior choroidal aneurysms: A report of two cases. Front Neurol. 2020;11:693. https://doi.org/10.3389/fneur.2020.00693.
63. Theron J. A reference system for arteriography based on the anterior choroidal artery (author's transl). Ann Radiol (Paris). 1976;19(2):223–6.
64. Theron J, Newton TH. Anterior choroidal artery. I. Anatomic and radiographic study. J Neuroradiol. 1976;3(1):5–30.
65. Theron J, Newton TH. Anterior choroidal artery. II. Pathologic states (tumors). J Neuroradiol. 1976;3(1):31–51.
66. Wolfram-Gabel R, Maillot C, Koritke JG. The vascular pattern in the tela choroidea of the prosencephalon in man. J Neuroradiol. 1987;14(1):10–26.
67. Frisen L, Holmegaard L, Rosencrantz M. Sectorial optic atrophy and homonymous, horizontal sectoranopia: a lateral choroidal artery syndrome? J Neurol Neurosurg Psychiatry. 1978;41(4):374–80. https://doi.org/10.1136/jnnp.41.4.374.
68. Tatu L, Moulin T, Bogousslavsky J, Duvernoy H. Arterial territories of the human brain: cerebral hemispheres. Neurology. 1998;50(6):1699–708.
69. Mohr JP, Steinke W, Timsit SG, Sacco RL, Tatemichi TK. The anterior choroidal artery does not supply the corona radiata and lateral ventricular wall. Stroke. 1991;22(12):1502–7. https://doi.org/10.1161/01.str.22.12.1502.
70. Isolan GR, Marrone ACH, Marrone LCP, Stefani MA, da Costa JC, Telles JPM, et al. Vascularization of the uncus - anatomical study and clinical implications. Surg Neurol Int. 2021;12:393. https://doi.org/10.25259/SNI_616_2021.
71. Helgason C, Wilbur A, Weiss A, Redmond KJ, Kingsbury NA. Acute pseudobulbar mutism due to discrete bilateral capsular infarction in the territory of the anterior choroidal artery. Brain. 1988;111(3):507–24. https://doi.org/10.1093/brain/111.3.507.
72. Helgason CM, Wilbur AC. Capsular hypesthetic ataxic hemiparesis. Stroke. 1990;21(1):24–33. https://doi.org/10.1161/01.str.21.1.24.
73. Tonetti DA, Andrews EG, Stabingas K, Tyler-Kabara E, Gross BA, Jadhav A. Posterior communicating artery giving rise to shared-origin anterior choroidal artery: case illustration. World Neurosurg. 2018;109:413–5. https://doi.org/10.1016/j.wneu.2017.10.098.
74. Nishio A, Yoshimura M, Yamauchi S, Masamura S, Kawakami T, Goto T, et al. Anomalous origin of the anterior choroidal artery. Interv Neuroradiol. 2009;15(1):73–6. https://doi.org/10.1177/159101990901500111.
75. Hara N, Koike T, Akiyama K, Toyama M. Anomalous origin of anterior choroidal artery. Neuroradiology. 1989;31(1):88. https://doi.org/10.1007/BF00342038.
76. Moyer DJ, Flamm ES. Anomalous arrangement of the origins of the anterior choroidal and posterior communicating arteries. Case report. J Neurosurg. 1992;76(6):1017–8. https://doi.org/10.3171/jns.1992.76.6.1017.
77. Nomura M, Kida S, Kita D, Hasegawa M, Matsui O, Yamashita J. Anomalous origin of anterior choroidal artery associated with an aneurysm. Acta Neurochir. 2000;142(9):1067–8. https://doi.org/10.1007/s007010070065.
78. Mamaliga T, White AC, Khattar NK, Hadi M. Anomalous origin of the anterior choroidal artery proximal to an ipsilateral fetal posterior cerebral artery: case report of an extremely rare anatomic variant and discussion of its clinical implications. Cureus. 2019;11(12):e6442. https://doi.org/10.7759/cureus.6442.
79. Choi CY, Lee CH. Transposition of anterior choroidal artery and posterior communicating artery origin. J Korean Neurosurg Soc. 2012;52(3):240–2. https://doi.org/10.3340/jkns.2012.52.3.240.
80. Abrahams JM, Hurst RW, Bagley LJ, Zager EL. Anterior choroidal artery supply to the posterior cerebral artery distribution: embryological basis and clinical implications. Neurosurgery. 1999;44(6):1308–14.
81. Windle BC. The arteries forming the circle of Willis. J Anat Physiol. 1888;22(Pt 2):289–93.
82. Gordon-Shaw C. Two cases of reduplication of the arteria cerebri posterior. J Anat Physiol. 1910;44(Pt 3):244–8.
83. Adachi B, Hasebe, K. Kyōto Teikoku Daigaku, Kenkyūsha. Das Arteriensystem der Japaner. Vol 1 Maruzen Kyoto; 1928. pp. 116–118.
84. Uchino A, Kamide T, Kurita H. Replaced posterior cerebral artery (PCA): origin of all branches of the PCA from the anterior choroidal artery diagnosed by MR angiography. Surg Radiol Anat. 2019;41(6):703–5. https://doi.org/10.1007/s00276-019-02209-8.
85. Hahm MH, Moon S. Anomalous hyperplastic anterior choroidal artery and its mimicker: a case series. Surg Radiol Anat. 2021;43(12):1961–5. https://doi.org/10.1007/s00276-021-02777-8.
86. Koyama T, Gibo H, Kobayashi S. A large anomalous anterior choroidal artery associated with internal carotid artery-posterior communicating artery aneurysm. Case report. Neurosurg Rev. 1998;21(4):299–301. https://doi.org/10.1007/BF01105790.

87. Doi K, Mizuno T, Shigematsu Y, Kobayashi O, Takezaki T, Muta D, et al. A new type of hyperplastic anterior choroidal artery. J Clin Neurosci. 2018;51:72–4. https://doi.org/10.1016/j.jocn.2018.02.002.
88. Hupperts RM, Lodder J, Heuts-van Raak EP, Kessels F. Infarcts in the anterior choroidal artery territory. Anatomical distribution, clinical syndromes, presumed pathogenesis and early outcome. Brain. 1994;117(Pt 4):825–34. https://doi.org/10.1093/brain/117.4.825.
89. Nadaf S, Chakor RT, Kothari KV, Patel BA. Anterior choroidal artery infarction. BMJ Case Rep. 2018;2018 https://doi.org/10.1136/bcr-2017-222414.
90. Tolla AS, Schichtel LT, Alboudi A, Sanders AK. Anterior choroidal artery infarct presenting as right hemichoreoathetosis. BMJ Case Rep. 2020;13(5):e234861. https://doi.org/10.1136/bcr-2020-234861.
91. Gullaume J, Caron JP. A case of extensive hemiathetosis treated by section of the anterior choroid artery. Rev Neurol (Paris). 1953;89(5):367–8.
92. Komiyama M, Morikawa T, Ishiguro T, Matsusaka Y, Yasui T. Anterior choroidal artery variant and acute embolic stroke. Case report. Interv Neuroradiol. 2002;8(3):313–6. https://doi.org/10.1177/159101990200800312.
93. Bruno A, Graff-Radford NR, Biller J, Adams HP Jr. Anterior choroidal artery territory infarction: a small vessel disease. Stroke. 1989;20(5):616–9. https://doi.org/10.1161/01.str.20.5.616.
94. Fisher M, Lingley JF, Blumenfeld A, Felice K. Anterior choroidal artery territory infarction and small-vessel disease. Stroke. 1989;20(11):1591–2. https://doi.org/10.1161/01.str.20.11.1591.
95. Leys D, Mounier-Vehier F, Lavenu I, Rondepierre P, Pruvo JP. Anterior choroidal artery territory infarcts. Study of presumed mechanisms. Stroke. 1994;25(4):837–42. https://doi.org/10.1161/01.str.25.4.837.
96. Piotin M, Mounayer C, Spelle L, Williams MT, Moret J. Endovascular treatment of anterior choroidal artery aneurysms. AJNR Am J Neuroradiol. 2004;25(2):314–8.
97. Bohnstedt BN, Kemp WJ 3rd, Li Y, Payner TD, Horner TG, Leipzig TJ, et al. Surgical treatment of 127 anterior choroidal artery aneurysms: a cohort study of resultant ischemic complications. Neurosurgery. 2013;73(6):933–9; discussion 9–40. https://doi.org/10.1227/NEU.0000000000000131.
98. Lee YS, Park J. Anterior choroidal artery aneurysm surgery: ischemic complications and clinical outcomes revisited. J Korean Neurosurg Soc. 2013;54(2):86–92. https://doi.org/10.3340/jkns.2013.54.2.86.
99. Kim BM, Kim DI, Shin YS, Chung EC, Kim DJ, Suh SH, et al. Clinical outcome and ischemic complication after treatment of anterior choroidal artery aneurysm: comparison between surgical clipping and endovascular coiling. AJNR Am J Neuroradiol. 2008;29(2):286–90. https://doi.org/10.3174/ajnr.A0806.
100. Kang HS, Kwon BJ, Kwon OK, Jung C, Kim JE, Oh CW, et al. Endovascular coil embolization of anterior choroidal artery aneurysms. Clinical article. J Neurosurg. 2009;111(5):963–9. https://doi.org/10.3171/2009.4.JNS08934.
101. Bhogal P, Ganslandt O, Bazner H, Henkes H, Aguilar PM. Treatment of unruptured, saccular, anterior choroidal artery aneurysms with flow diversion: A single centre experience. Clin Neuroradiol. 2019;29(3):459–65. https://doi.org/10.1007/s00062-018-0677-1.
102. Inci S, Arat A, Ozgen T. Distal anterior choroidal artery aneurysms. Surg Neurol. 2007;67(1):46–52; discussion. https://doi.org/10.1016/j.surneu.2006.05.053.
103. Yang S, Yu JL, Wang HL, Wang B, Luo Q. Endovascular embolization of distal anterior choroidal artery aneurysms associated with moyamoya disease. A report of two cases and a literature review. Interv Neuroradiol. 2010;16(4):433–41. https://doi.org/10.1177/159101991001600410.
104. Guo X, Gao L, Yu H, Chen W, Yang Y, Jin F, et al. Computed tomographic angiography may be used for assessing the dilatation of the anterior choroidal and posterior communicating arteries in patients with moyamoya syndrome. Eur Radiol. 2021;31(8):5544–51. https://doi.org/10.1007/s00330-021-07722-2.
105. Morioka M, Hamada J, Kawano T, Todaka T, Yano S, Kai Y, et al. Angiographic dilatation and branch extension of the anterior choroidal and posterior communicating arteries are predictors of hemorrhage in adult moyamoya patients. Stroke. 2003;34(1):90–5. https://doi.org/10.1161/01.str.0000047120.67507.0d.
106. Lasjaunias PL, Alvarez H, Rodesch G, Garcia-Monaco R, Ter Brugge K, Burrows P, et al. Aneurysmal malformations of the vein of Galen. Follow-up of 120 children treated between 1984 and 1994. Interv Neuroradiol. 1996;2(1):15–26. https://doi.org/10.1177/159101999600200102.
107. van Overbeeke JJ, Hillen B, Vermeij-Keers C. The arterial pattern at the base of arhinencephalic and holoprosencephalic brains. J Anat. 1994;185(Pt 1):51–63.
108. Gabarros Canals A, Rodriguez-Hernandez A, Young WL, Lawton MT, Project UBAS. Temporal lobe arteriovenous malformations: anatomical subtypes, surgical strategy, and outcomes. J Neurosurg. 2013;119(3):616–28. https://doi.org/10.3171/2013.6.JNS122333.
109. Hou K, Yu J. Role of the anterior choroidal artery in the endovascular treatment of brain arteriovenous malformations. Acta Neurol Belg. 2022;123:57–67. https://doi.org/10.1007/s13760-022-01878-4.
110. Elkordy A, Endo H, Sato K, Matsumoto Y, Kondo R, Niizuma K, et al. Embolization of the choroidal artery in the treatment of cerebral arteriovenous malformations. J Neurosurg. 2017;126(4):1114–22. https://doi.org/10.3171/2016.2.JNS152370.
111. Ghali MGZ. Choroid plexus AVM with anomalous origin of the capsulothalamic artery: A case report. Interv Neuroradiol. 2019;25(5):585–6. https://doi.org/10.1177/1591019919840005.
112. Yamauchi S, Kawakami T, Murata K, Ishiguro T, Ikeda H, Nishio A. Choroid plexus AVM with anomalous origin of the capsulothalamic artery: A case report. Interv Neuroradiol. 2018;24(1):76–81. https://doi.org/10.1177/1591019917739841.
113. Javed K, Das JM. Neuroanatomy, anterior choroidal arteries. Treasure Island, FL: StatPearls; 2022.
114. Oyama H, Noda S, Negoro M, Kinomoto T, Miyachi S, Kuwayama N, et al. Giant meningioma fed by the anterior choroidal artery: successful removal following embolization—case report. Neurol Med Chir (Tokyo). 1992;32(11):839–41. https://doi.org/10.2176/nmc.32.839.
115. Ben Nsir A, Gdoura Y, Thai QA, Zhani Kassar A, Hattab N, Jemel H. Intraventricular glioblastomas. World Neurosurg. 2016;88:126–31. https://doi.org/10.1016/j.wneu.2015.12.079.

Embryology, Anatomy, and Variations of the Anterior Cerebral Artery

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The anterior cerebral artery (ACA) is one of the most important arteries of the brain and in particular of the telencephalon [1, 2]. The ACA is also one of the most studied cerebral arteries since it is a common location for intracranial aneurysms [3]. During the species' evolution, the ACA acquires its vascular territories with the development of the telencephalon in mammals; before this event, its role is limited to the vascularization of the olfactory system and in part of the diencephalon [4]. In this chapter, the normal anatomy and functions of the ACA will be reminded with special attention to its embryological development and to its known anatomical variations.

History

The embryologic development of the ACA, as for another cerebro-facial arteries, has been mostly understood thanks to the monumental work of D.Padget (1948) and also with the studies of comparative anatomy proposed by Abbie (1934) and by Moffat (1962) [4–6]. The largest and most significant cadaveric series about the ACA were published in the 1970s and 1980s; they include the detailed works of Perlmutter and Rhoton (1976, 1978), Rosner et al. (1984), and Gomes et al. (1984) [1, 7–9]. Yasargil (1984), in his monography about clinical considerations and surgical techniques of intracranial aneurysms, described the anatomy of the ACA and its variations noted during aneurysms' microsurgical clipping [10]. The angiographic and radiological anatomy of the ACA was analyzed in detail by Salamon (1977) [11]. While studying arterial brain vascularization as seen on angiography, Lasjaunias and Berenstein (2001) described in detail all anatomical variations of the ACA and proposed hypotheses for their embryological development [12].

Embryology

Streeter (1918) and Padget (1948) described different stages in vasculature development of the brain after having analyzed 22 sectioned embryos [4, 6]. Our understanding of the embryological development of the ACA and its anatomical variants is based on the development of the cranial division of the internal carotid artery (ICA) and of the ophthalmic artery (OA), whose developmental steps are represented in Tables 1 and 2, respectively.

Cranial Division of the Primitive Carotid Artery (Table 1)

In Padget's "early stage I" (embryos of 4–5 mm), the primitive ICA divides into a cranial and a caudal branch (future ACA and posterior communicating artery) [4, 6]. The cranial branch curves above the optic vesicle and terminates in the olfactory area, thus named primitive olfactory artery (POA) [6]. Concomitantly, the primitive ICA, proximal to its bifurcation, gives off the primitive maxillary artery (PMA) which passes laterally to the Rathke's pouch and courses under the optic vesicle. This PMA seems to be a temporary supply of the optic vesicle that will regress at Padget's stage IV [5, 6]. The exact role of the PMA in the embryological development of the ophthalmic artery is not known.

During Padget's stage III (7–12 mm), some primitive branches spread out from the POA. The largest and most caudal of these is the anterior choroidal artery (AChoA); a second, directed to the optic vesicle, is the primitive ventral

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Table 1 Embryological development of the anterior cerebral artery

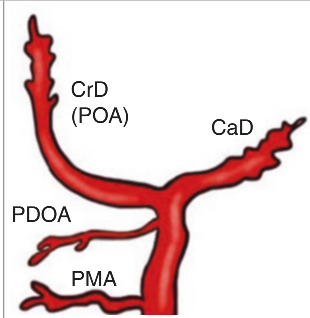
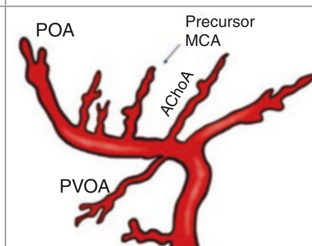
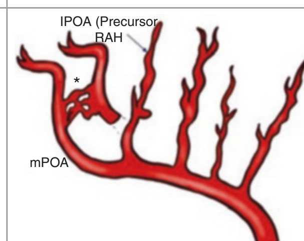
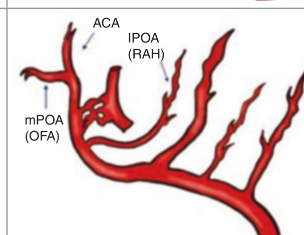
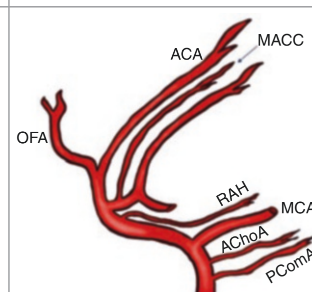
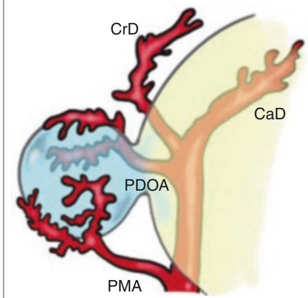
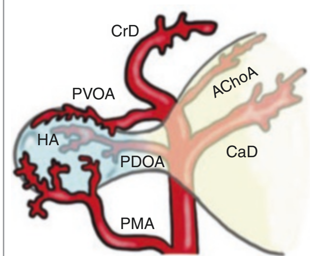
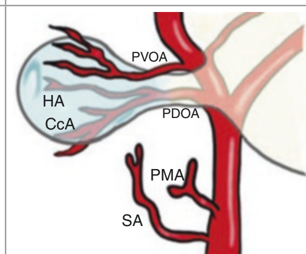
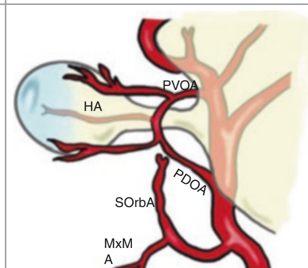
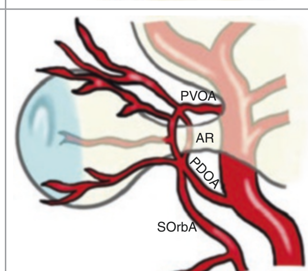
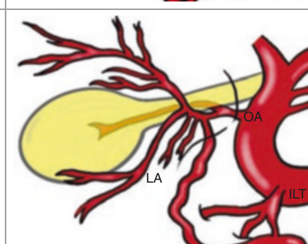
Stage of padget	Size of embryo (mm)	Events	Illustration
I	4–5	– Cranial and caudal division of the carotid artery (CrD, CaD) appearance – CrD is the primitive olfactory artery (POA) – Primitive maxillary artery (PMA) and primitive dorsal ophthalmic artery (PDOA) appearance	
III	7–12	– Branches of the POA appearance: Anterior coroidal artery (AChoA), primitive ventral ophthalmic artery (PVOA), precursor of the middle cerebral artery (MCA)	
IV	12–14	– POA division in lateral (lPOA) and medial (mPOA) branches – The mPOA borrows the future ACA – The lPOA is the precursor of the recurrent artery of Heubner (RAH) – Plexiform anastomosis between the two ACAs (*, future AcomA)	
V	16–18	– ACA development with terminal part of the medial POA appearing as a branch, future orbito-frontal artery (OFA) – Lateral extension of the lPOA (future RAH)	
VI	20–24	– Formation of the AComA (complete circle of Willis) – Medial artery of the corpus callosum visible (MACC)	

Table 2 Embryological development of the ophthalmic artery

Stage	Embryo size (mm)	Events	Graphic representation
I	4–5	– Primitive maxillary artery (PMA) as temporary branch – PDOA appearance	
II	9	– Primitive hyaloid artery (HA) as plexiform channels – PVOA appearance	
III	14	– Formation of primitive hyaloid and common ciliary arteries – Stapedial artery (SA) development	
IV	18	– Migration of the PDOA origin – Formation of the supraorbital branch (SOrbA) of the SA – Regression of the PVOA	
V	20	– Maximal development of the SA – Formation of the anastomotic ring (AR)	
VI	40	– Ventral interruption of the AR – Regression of the SOrbA	

ophthalmic artery (PVOA); a third, that is directed cranially between the two cerebral hemispheres, is the precursor of the future ACA. In some embryos, a lateral small branch from the POA is visible and is considered as the primitive middle cerebral artery (MCA) [6, 12].

At Padget's stage IV (12–14 mm), as already noted by Abbie (1934) in fishes and amphibians, two terminal branches (medial and lateral) of the POA can be seen [4]. The medial one follows the course of the olfactory nerve and is the precursor of the ACA; on each side of the midline of the brain, plexiform anastomoses between these two medial branches are already visible and are the precursors of the AComA. The lateral branch of the POA finishes its course in the nasal fossa and is considered by some authors as the precursor of the recurrent artery of Heubner (RAH) [4].

In Padget's stage V (16–18 mm), the lateral branch of the POA (future RAH), appears as the main branch, but progressively regresses to become a lateral branch of the medial POA (future ACA) [6].

In Padget's stage VI (20–24 mm), the primitive ACA is clearly seen to have an upper course between the two cerebral hemispheres and gives different anastomoses to the choroidal arteries [12]. During this time, the AComA continues its development to be completely formed at the end of this stage, concluding the formation of the circle of Willis [6]. The AComA is consequently the last artery of the circle of Willis to develop. It is important to note that at this point the presence of a superior branch from the AComA, named the medial artery of the corpus callosum (MACC), can persist or regress in the adult [6]. The medial POA now appears as a branch of the ACA following the course of the olfactory nerve, ending in the nasal fossa (future orbito-frontal artery of the ACA). The lateral branch of the POA develops and extends laterally in the region of the anterior perforated substance (future RAH) [6].

Ophthalmic Artery (Table 2)

The embryological development of the OA is intricately related to the primitive olfactory artery, and the knowledge thereof is essential in the understanding of some adult variants, like the infra-optic course of the ACA or the origin of the OA from the ACA.

The embryological development of the ophthalmic artery is a series of development and regression of primitive arteries and could be divided into six stages, which are summarized in Table 2. These six stages described by Padget are different than the stages of the general embryological development also elaborated by the same author [6].

The development of the OA depends mostly on two embryonic arteries: the primitive dorsal ophthalmic artery (PDOA) and the primitive ventral ophthalmic artery (PVOA). The PDOA originates from the bifurcation of the primitive ICA when the embryo is about 4–5 mm, while the PVOA branches off from the primitive ICA (primitive olfactory artery), when the embryo is about 9 mm.

Stage I: In the early embryologic life (4–5 mm), the optic vesicle begins its differentiation in optic stalk and optic vesicle. The primitive maxillary artery, which originated from the future cavernous segment of the primitive carotid artery, sends a temporary branch to the base of the optic vesicle. The other major event of this stage is the formation of the first primitive ophthalmic artery from the primitive carotid artery: the primitive dorsal ophthalmic artery (PDOA) which takes its origin at the bifurcation of the primitive carotid artery in its caudal and cranial division (future posterior communicating segment).

Stage II: In the 9 mm embryos, the PDOA now invests the optic cup by a large plexus without clear direct supply to the lens. The major change of this stage is the formation of another primitive ophthalmic artery from the primitive olfactory artery: the primitive ventral ophthalmic artery (PVOA).

Stage III: In the 14 mm embryos, both PDOA and PVOA are elongated, probably due to the ventral shifting of the optic cup and dorsal shifting of cerebral hemispheres. On the other hand, the stapedial artery begins its development and one of its collateral branches is captured by the ventral pharyngeal artery (future stem of the middle meningeal artery).

Stage IV: At approximately 18 mm, the elongated stem of the PDOA is gradually pulled along the primitive internal carotid artery and its origin migrates to its adult position. Streeter (1918) called this phenomenon "anastomotic progression" in reference to vessels of other organs. Concomitantly, the stapedial artery continues its progression and gives off two branches, the maxillomandibular artery and the supraorbital artery. The latter one passes into the orbital cavity through the superior orbital fissure.

Stage V: In the 20 mm embryos, the supraorbital branch of the stapedial artery develops several orbital branches and anastomoses with the primitive ophthalmic artery. An anastomotic ring is formed around the optic nerve by anastomosis between the supraorbital artery, the PDOA, and the PVOA.

Stage VI: At approximately 40 mm, the adult conformation of the ophthalmic artery could be recognizable after the ventral interruption of the anastomotic ring that forms the future second segment of the ophthalmic artery. The extra-orbital part of the supraorbital artery also regresses, and the lacrimal artery is naturally annexed by the primitive ophthalmic artery.

In the stages described two events are crucial to determine the definitive origin of the OA and, as a consequence, its relationship with the ICA and the ACA:

1. The migration of the OA's origin on the supraclinoid ICA (embryo of about 18 mm): This phenomenon is explained by Padget to be caused by the cranial elongation of the ICA during this stage, consequently resulting in the PDOA shifting to a lower position on the ICA [6]. However, Lasjaunias explained this migration to be the result of an intradural anastomosis between the PVOA

and the primitive ICA followed by the regression of the original proximal stem of the PVOA [12, 13].

2. The formation of an anastomotic ring around the optic nerve (embryo of about 20 mm), formed by the anastomosis between the PVOA, the PDOA, and the supraorbital artery (a branch of the stapedial artery): This ring will be ventrally interrupted to give the definitive configuration of the OA. The part of the anastomotic ring that regresses is critical in determining the definitive configuration of the adult OA.

Possible variants of these two phenomena can determine an infraoptic course of the ACA, or the origin of the OA from the ACA.

Segments and Course of the Artery

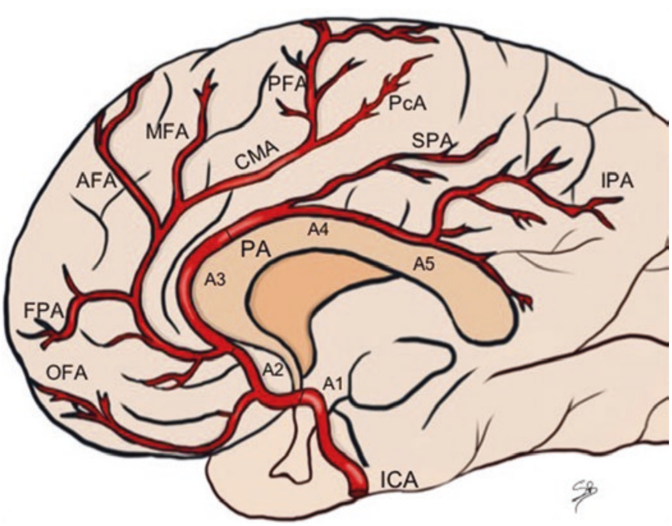
The ACA is the most important artery of the medial surface of the telencephalon and one of the most important in the diencephalic supply. Fisher (1938) was the first to propose a segmental classification of the ACA [14]. This classification in five distinct segments is nowadays the most used and will be used in this chapter (Fig. 1). Other authors proposed various classifications but none of which gained popularity. For example, Morris and Peck (1955) proposed a four segments classification of the ACA and Lin and Kircheff (1974) gave a personal segmental classification of the pericallosal artery (post-communicating ACA) [9]. The post-communicating ACA (segments A2–A5) could also be named as pericallosal artery or distal ACA.

A1 Segment

This first segment of the ACA (A1), also named precommunicating or horizontal segment of the ACA, begins at the terminal bifurcation of the ICA and ends at its junction with the AComA. This segment has an average length of 12.7 mm (7–18 mm) and an average diameter of 2.6 mm (0.3–7 mm) [2, 7]. This segment courses above the optic chiasm in 70% or above the optic nerve in 30%. Gomes et al. (1986) noted that longest is the A1 segment, more anterior it courses above the optic structures [2]. Uchino et al. (2006) noted an asymmetry in the size of A1 segments in 5–7% of the general population (in MRI realized for other pathology than AComA aneurysms) [15]. However, in patients harboring an AcomA aneurysm, Yasargil observed a frequent asymmetry (1984) [10]. This observation was confirmed by Aydin et al. (1997), who found in their series of AComA aneurysm an asymmetry of A1 segments in 27% of cases [16]. Even if there are no comparative studies, the asymmetry of A1 segments seems to be an important hemodynamic factor in the development of AComA aneurysm [1, 2, 9, 17].

A2 Segment

The second segment of the ACA (A2 segment) extends from the AComA to the point where the artery passes over the junction between the rostrum and genu of the corpus callosum [9]. This segment is into the cistern of the lamina terminalis. The median length of the A2 segment is 28 mm



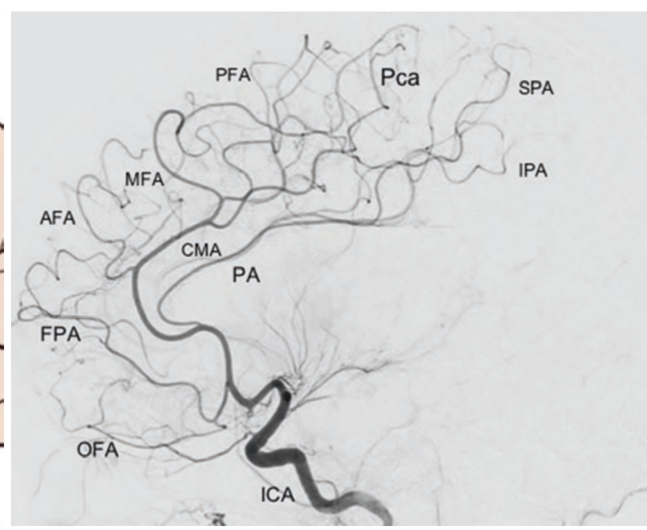


Fig. 1 Fisher's classification of the ACA and branches (Adapted from [114]). The figure shows an artist's illustration and a lateral DSA ICA injection, with the aim to highlight the division of the ACA in five segments (A1-A5) and to describe its branches. The visible branches are: the orbitofrontal artery (OFA), the frontopolar artery (FPA), the anterior middle and posterior internal frontal arteries (AFA, MFA, PFA), the paracentral artery (PcA), the superior and inferior parietal arteries (SPA, IPA). The point of origin of these branches is very variable, however the bifurcation of the distal ACA in a callosomarginal artery (CMA), that courses in the cingulate sulcus, and in a pericallosal artery (PA), that courses in the epicallosal sulcus, is almost constant

(22–30 mm) and the overall diameter is 2.4 mm (0–3.5 mm) [2, 7]. Usually, the left A2 segment is positioned posterior to the right one in 72% and inversely in 28% [10].

A3 Segment

The A3 segment of the ACA begins at the junction between the rostrum and genu of the corpus callosum, runs along the genu and terminates when the artery turns posterior above the body of the corpus callosum. This segment has an average length of 41 mm (33–48 mm) and a diameter of 2.4 mm [8, 9].

A4 and A5 Segments

The A4 and A5 segments of the ACA represent the part of the artery above the body of the corpus callosum [7, 9, 10]. The virtual limit between these two segments corresponds to the projection of the coronal suture. The average length of these segments is 76 mm and the average diameter is 2.2 mm [2]. These segments lie in the pericallosal cistern, in the epicallosal sulcus (sulcus of the corpus callosum) (86%), but they may also be found (14%) in the cingulate sulcus [10]. A clinical case of this variant is represented in Fig. 2.

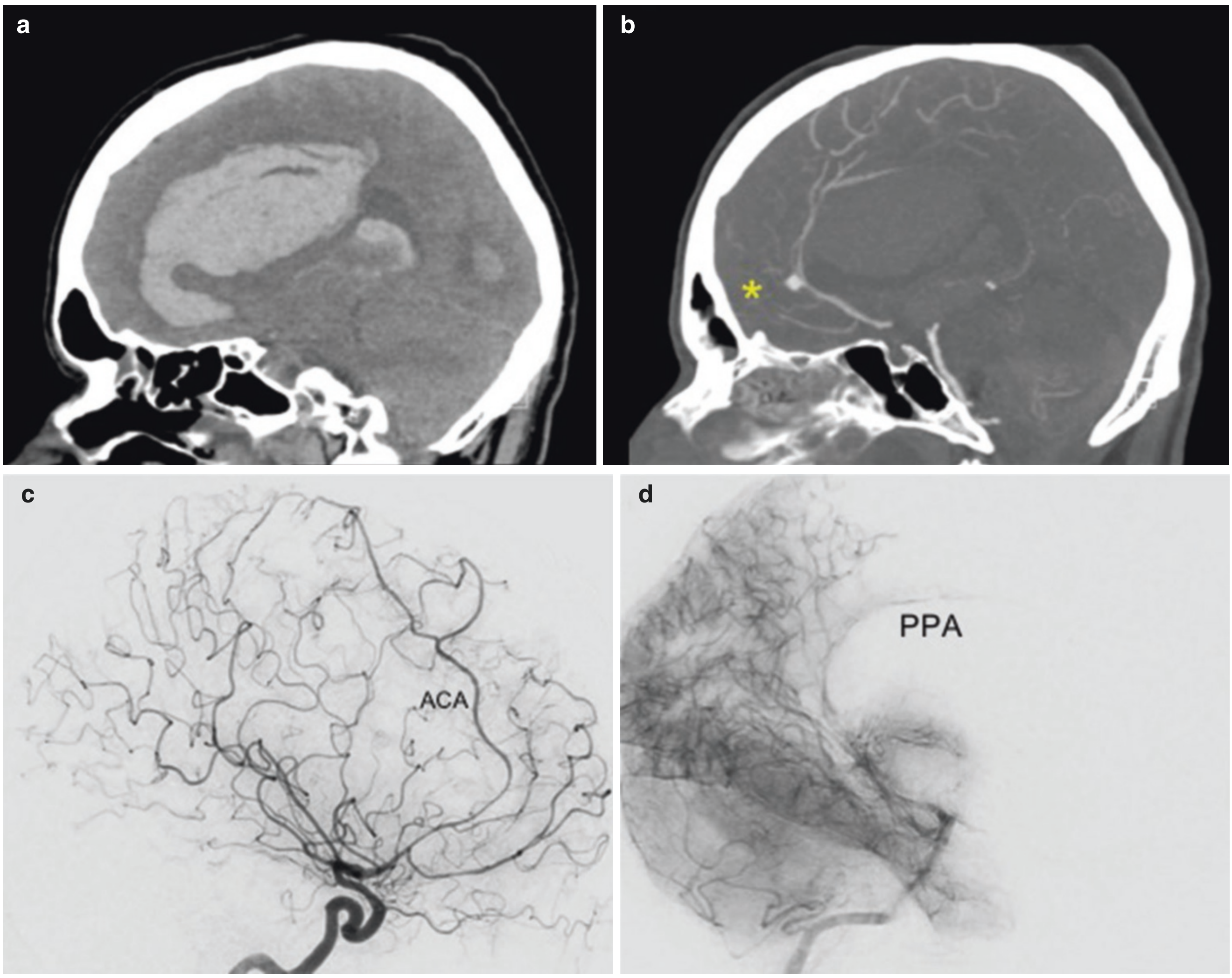


Fig. 2 Clinical case of ACA located in the cingulate sulcus. The figure shows the case of a patient admitted to the emergency room for acute loss of consciousness. The brain CT scan showed a big frontal hematoma with ventricular extension (**a**). The Angio-CT identified a pericallosal artery aneurysm (yellow asterisk in figure **b**). The patient underwent urgent surgical clipping and a post-operative DSA was performed. The exam showed a course of the distal ACA into the cingulate sulcus (**c**), instead of the epicallosal sulcus and this finding was confirmed during surgery. Figure (**d**) shows a compensation from the posterior circulation around the corpus callosum through the posterior pericallosal artery (PPA)

Branches of the Artery and Their Variations

In this paragraph, only the direct perforating branches, cortical branches, and callosal branches of the ACA will be described in detail (Table 3). The anatomy and variations of the recurrent artery of Heubner are addressed in chapter "Embryology and Variations of the Recurrent Artery of Heubner," as well as the dural branches of the ACA (chapter "Dural Branches of the Anterior and Posterior Cerebral Arteries").

Table 3 Branches of the ACA with respective parenchymal territory

ACA branches	Origin from the ACA (segment, most frequent)	Diameter at origin (mm)	Territory
Perforating arteries	A1–A2	0.2 (0.1–1.0)	Subfrontal area Optic chiasm Preoptic region Hypothalamus Internal capsule
Recurrent artery of Heubner	A2	0.7 (0.4–1.0)	Head of caudate nucleus Anterior limb internal capsule Uncinate fasciculus Hypothalamus
Orbitofrontal artery	A2	0.9 (0.4–2.0)	Gyrus rectus Olfactory bulb and tract Medial half of orbital gyri
Frontopolar artery	A2	1.3 (0.6–1.8)	Medial surface of the frontal pole
Anterior internal frontal artery	A3	1.3 (0.4–2.4)	Superior frontal gyrus (anterior part)
Middle internal frontal artery	A3	1.3 (0.9–2.9)	Superior frontal gyrus (medial and lateral surface)
Posterior internal frontal artery	A3-A4	1.4 (0.9–2.4)	Cingulate gyrus Superior frontal gyrus (posterior part)
Paracentral artery	A4	1.3 (0.7–2.0)	Paracentral lobule (superior part)
Superior parietal artery	A5	1.2 (0.8–1.7)	Paracentral lobule (posterior part)
Inferior parietal artery	A5	1.1 (0.6–1.8)	Precuneus Cuneus
Callosal arteries		0.2 (0.1–1.0)	Corpus callosum Septum pellucidum Anterior commissure Column of the fornix
Pericallosal artery		1.9 (0.9–2.6)	Corpus callosum Cingulate gyrus
Callosomarginal artery	A3	1.8 (1.2–2.7)	Cingulate gyrus Superior frontal gyrus (medial surface)

Perforating Branches From the ACA

Perforating branches of the ACA arise mainly from the A1 segment of the artery, more likely from its lateral half (68% vs. 32%) [8, 18]. The number of these branches varies from 1 to 11 (average 5.3) in the lateral half of the A1 segment and from 0 to 6 (average 2.5) in the medial half [2, 7, 8, 19]. However, according to Barry (1972), perforating branches arise from the first 5 mm of the A1 segment, making its proximal segment ideal for applying a temporary clip during AComA aneurysm surgery [20]. The origin of the perforating branches is usually on the superior surface (54%) or on the posterior surface (32%) of the A1 segment [7, 8, 10, 17]. Their average diameter is 0.2 mm (0.1–1 mm) and their average length (from the origin to the penetrating point of the artery) is 3.8 mm (0.8–8.5 mm) [19]. Perforating arteries from the A1 segment supply the subfrontal area, the optic chiasm, the preoptic region, the hypothalamus, and the internal capsule [7, 21]. In 41% of cases, these branches penetrate into the parenchyma through the medial portion of the anterior perforating surface (APS) [8].

Few perforating branches may arise from the first 5 mm of the A2 segment but are inconstant and less numerous (average 1.2 branches, 0–4) [9, 17]. These branches arise from the lateral (46%) or posterior (43%) surfaces of the A2 segment and supply the gyrus rectus (29%), the inferior frontal area (31%), or the APS (15%) [8].

Cortical Branches

The ACA provides usually eight major cortical branches that are summarized in Table 3 and illustrated in Fig. 1. Some of these cortical branches may arise not directly from the ACA but from the callosomarginal artery (CMA), which is a branch of the ACA running into the cingulate sulcus, above the cingulate gyrus [2, 9, 22]. The CMA is defined as a branch of the ACA from which two or more cortical branches of the ACA originate [9]. Present in 60–90% of hemispheres, the CMA arises from the A3 segment in 60–90% or from the A2 segment in 3–10% and its average diameter is 1.8 mm (1.2–2.7 mm) [2, 9, 22]. The CMA may also surprisingly arise from the A1 segment, as described by Krishnamoorthy et al. (2006) [23]. The cortical arteries that most frequently branch off from the CMA are the anterior (24–84%), middle (42–77%), and posterior (28–56%) internal frontal arteries [2, 9].

Orbitofrontal (OFA) and frontopolar (FPA) arteries are the two first cortical branches of the ACA. They are always present in cadaveric series and usually arise from the A2 segment, even if a rare origin from the CMA has been described [2, 9, 22]. OFA and FPA may share a common origin and the OFA may also share its origin with the recurrent artery of Heubner (5%) [1]. At their origin, OFA and FPA have a diameter of 0.9 mm and 1.1 mm, respectively [9, 22].

The internal frontal arteries (anterior, middle, and posterior) are constant branches of the ACA present in 76–100% of hemispheres [2, 7, 9, 22]. These three branches often share their origin from the CMA or arise from the A3 segment. These three branches have usually an average diameter about 1.1–1.4 mm (0.4–2.9 mm) [9, 22].

The paracentral artery is a branch of the ACA that terminates into the medial part of the central sulcus and is very constant, with an incidence estimated around 90–100% on cadaveric studies [2, 9, 22]. It arises from the A4 segment in 32% of cases, from the CMA in 16–26% or from the A3 (0–18%) or A5 (14–49%) segments [2, 9]. Its diameter at the origin has an average of 1.2 mm (0.7–2.0 mm) [9, 22].

Superior and inferior parietal arteries are also relatively common cortical branches of the ACA, found in 74–85% and 60–64%, respectively [2, 9, 22]. These branches arise from the A5 segment in 90–100% of cases [2, 9]. Their respective diameter is 1.0–1.2 mm (superior) and 0.8–1.1 mm (inferior) at origin [9, 22].

Callosal Arteries

Callosal arteries arise from the pericallosal artery (PA) and are classified as short or long callosal arteries. These arteries may be found all along the course of the post-communicating part of the ACA [7, 9, 24]. The short callosal arteries are assimilated as perforating arteries of the corpus callosum, and their penetrating point is near their origin from the ACA (98%). The medial artery of the corpus callosum can be considered as a proximal short callosal artery [24]. The long callosal arteries do not penetrate the corpus callosum immediately after their origin but rather course into the callosal cistern, parallel to the ACAs [2, 7, 24]. These arteries supply the corpus callosum from the rostum to the splenium, the anterior commissure, the septum pellucidum, and the columns of the fornix [24].

Parenchymal Territory

Table 3 summarizes the different branches of the ACA with their respective parenchymal territory. The ACA is the principal arterial supply of the medial surface of the telencephalon but also participates in the vascularization of the diencephalic structures through its perforating branches and the recurrent artery of Heubner [25]. The parenchymal territory of the ACA is in balance with the territory of the middle and posterior cerebral arteries [2, 9, 25]. It corresponds to the medial part of the orbital surface of the frontal lobe, the superior frontal gyrus, the cingulate gyrus, the paracentral lobule, and the precuneus [9]. Deep structures that are supplied by the ACA are usually the septum pellucidum, the fornix, the anterior commissure, the internal capsule, and a part of the hypothalamus [25].

Anterior Cerebral Artery Origin of the Ophthalmic Artery

An anomalous origin of the OA from the ACA was reported in few isolated cases during the last decades [26–33]. This variant is well explained by a defect in the embryological development of the OA due to the absence of an anastomosis between the PVOA and the supraclinoid ICA during Padget's stage IV (18 mm) Lasjaunias explained the embryological basis of this anatomical variation [6, 12]. Other authors concur with Padget's hypothesis, naming this variant "persistent non-migrated ventral primitive ophthalmic artery" [28, 31]. The first case was documented angiographically by Picard and Vignaud in 1975 [26]. Fewer than 10 cases were subsequently described in the literature [27–33]. In the majority, an elongated OA arises from the mid-portion of the A1 segment, coursing over the optic nerve before entering the optic foramen. This anatomical variant was found to be associated with a suprasellar cyst, a contralateral cavernous ICA aneurysm, an AComA aneurysm, an anterior clinoidal meningioma, and an ophthalmic ICA aneurysm [27, 28, 30–32]. Three particular cases were associated with an infraoptic course of the ACA, with the OA arising from the origin of the abnormal A1 segment [29, 32, 34].

Infraoptic Course of the ACA

The infraoptic course of the ACA is defined as the origin of the ACA from the ophthalmic segment of the ICA with the A1 segment that courses below the optic nerve to end at the region of the AcomA [3, 4, 12, 35]. The first case was described by Robinson (1959) during a cadaveric dissection [36]. He named it "infraoptic course of the ACA". In 1976, Nutik and Dilenge noted the possible presence of concomitant normal A1 segment and consequently introduced the term "carotid-anterior cerebral artery anastomosis" [37, 38]. Few authors wrongly defined this anatomical variation as a "low internal carotid artery bifurcation" [39–41]. Isherwood (1969) was the first to report a case diagnosed by digital subtraction angiography (DSA), while Given et al. (2002) were the first to report a case found on MRI [42, 43].

Three different hypotheses have been proposed to explain the embryological origin of this anatomical variant. The first one, proposed by Robinson (1959), is that this variant is due to the enlargement of an anastomosis between a chiasmatic branch of the inferior hypophyseal artery and a chiasmatic branch of the A1 segment [36]. The second hypothesis, postulated by Bosma (1977), involves the primitive maxillary artery (PMA) [39]. The PMA, whose exact role is not known yet, plays a role in the early development of orbital artery (Padget's stage I) and is thought to be the precursor of the inferior hypophyseal artery [6, 12]. The PMA normally regresses early in the embryologic development. Some authors explain the infraoptic course of the ACA by the non-regression of the PMA and its anastomosis with the primitive olfactory artery (future ACA). The third hypothesis was proposed by Lasjaunias, who explained the infraoptic course of the ACA by the non-regression of the proximal part of the PVOA after its anastomosis with the ICA, resulting in the adult configuration of the OA [12]. These three hypotheses are summarized in Table 4. Another hypothesis stems from a false interpretation of Lasjaunias' concept about the embryological development of the OA, in which some authors postulated that the infraoptic course of the ACA is due to a persistence of an embryologic anastomosis between PVOA and PDOA [3, 29, 35, 44, 45]. It is true that such an anastomosis between PDOA and PVOA exists during the embryologic development, but this anastomosis is intraorbital, not intracranial, and allows the formation of the second segment of the adult OA.

In the literature, less than 50 isolated cases were described, the majority of them were unilateral (79%) and few of them were bilateral (21%) [3, 34, 35, 39, 40, 45–66]. Wong et al. (2008) proposed a classification for infraoptic course of the ACA that is illustrated in Fig. 3 [67].

Table 4 Embryologic hypotheses for infraoptic ACA

Hypothesis	Description
Robinson (1959)	Anastomosis between chiasmatic branches of inferior hypophyseal and A1
Bosma (1977)	– Persistence of the PMA – Anastomosis PMA—olfactory artery
Lasjaunias (2001)	Non-regression of the proximal part of the PVOA
Hannequin (2013)	Persistence of the anastomosis between PDOA and PVOA

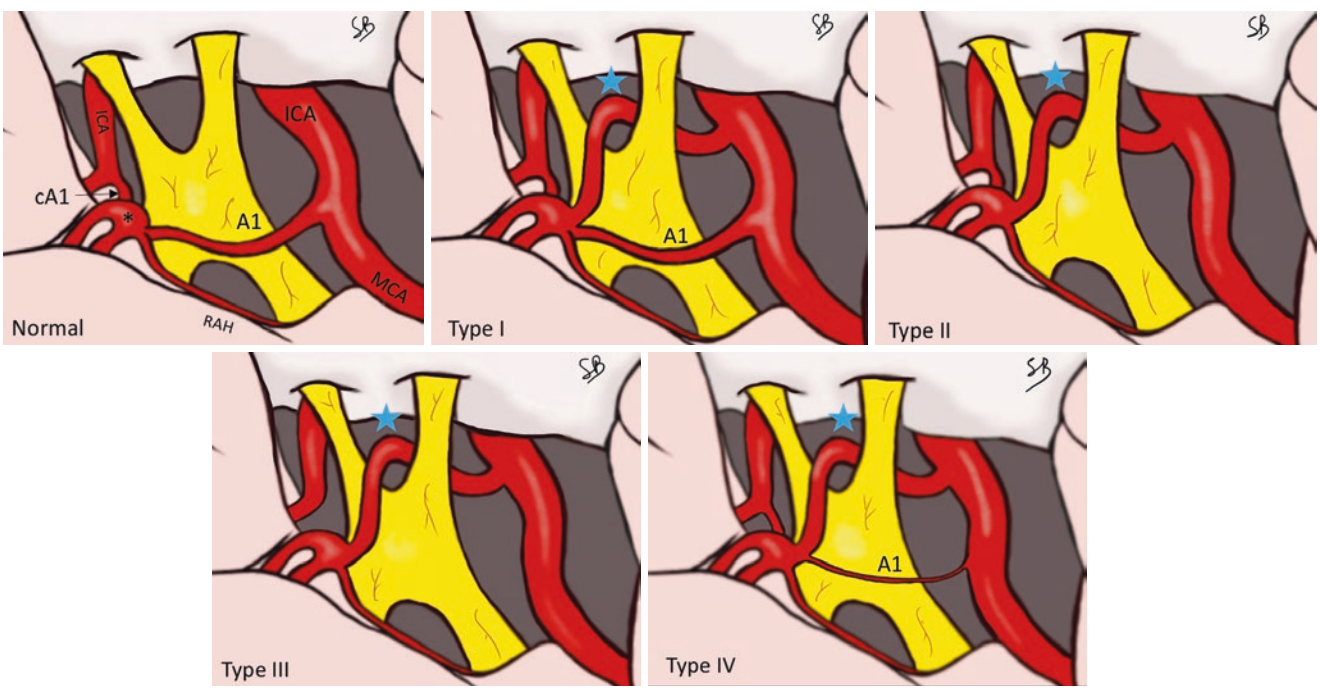


Fig. 3 Different types of infraoptic ACA (Wong's classification) (Adapted from [67]). The Figure shows the four possible types of infraoptic course of the ACA. This variant is indicated in each figure with a blue star. In the normal configuration, the internal carotid artery (ICA) bifurcates into the anterior cerebral artery with its first segment (A1) and the middle cerebral artery (MCA). The A1 joins the contralateral A1 (cA1) into the anterior communicating artery (asterisk). From this junction is also visible the origin of the recurrent artery of Heubner (RAH). Type I consists in the presence of the ICA-ACA anastomosis with concomitant presence of both A1 segments. Type II is the presence of the anastomotic vessel in the absence of the homolateral A1 segment but in the presence of the contralateral A1 segment. Type III is the presence of the carotid-anterior cerebral artery anastomosis in the presence of both A1 segments. Type IV is similar to type I but with a hypoplasia of the homolateral A1 segment

In type I, an anastomosis of the carotid-anterior cerebral artery is present with concomitant A1 segments in both hemispheres. In type II, there is absence of the ipsilateral A1 segment of the ACA but presence of the contralateral A1 segment. In type III, both A1 segments are absent. Type IV presents a particularity in which the configuration is similar to type I, but with a hypoplasia of the homolateral A1 segment. Although this classification is based on the presence or absence of the A1 segments, the absence of which may predict the risk of aneurysmal formation and seems to give an idea on the risk of distal aneurysm, this classification has also been met with some criticism. One such criticism is that bilateral anatomical cases are difficult to classify. The second is that type IV is a hybrid between types I and II, which may explain why only few of the described cases could be classified as type IV. Among the cases already described in the literature, 35% (18 cases) are type I, 33% (17 cases) type II, 27% (14) type III, and only 6% (3 cases) could be considered as type IV.

Persistent Primitive Olfactory Artery

As described by Padget and explained in the paragraph embryology, the primitive olfactory artery (POA) is the precursor of the ACA and of the RAH, and normally disappears during the embryological development [4, 6]. The persistence of the primitive olfactory artery (PPOA) is defined as the presence of an embryological artery that after birth is found to arise from the supraclinoid ICA or from the A1 segment of the ACA, and that courses anteriorly along the olfactory nerve. In some variants, the PPOA supplies the distal territories of the ACA. The incidence of the PPOA varies between 0.14 and 0.64% depending on the type of study [3, 30, 68]. The first case of PPOA was described after a cadaveric dissection by Moffat (1967) and the first angiographic report was provided by Amaura (1979) [68, 69].

Four different variants of PPOA could be identified in the literature and are summarized in Table 5 and illustrated in Fig. 4. Type I is the most frequent (84% of cases) and consists in a PPOA that makes an upward hairpin turn to supply the ACA territory [3, 30, 70–77]. Type II is very rare (3%) and difficult to see angiographically [78]. In this type II, the PPOA courses along the olfactory nerve to enter the nasal cavity through the cribriform plate and anastomoses with the anterior ethmoidal artery. Type III is a hybrid between the first and second types and was firstly described by Horie (2012) [76, 79]. In this variant, the PPOA, after following its typical course along the olfactory nerve gives 2 branches, the first one supplies the territory of the callosomarginal artery (CMA), and the second one enters into the nasal cavity. Type III represents only 6% of cases. Type IV variant is also very rare (6% of cases) [68, 70]. The PPOA, in this variant, courses along the olfactory nerve and makes a hairpin turn to supply an accessory middle cerebral artery (RAH territory). Keeping in mind the development of the primitive olfactory artery, it seems that types I, II, and III are the persistence of the medial branch of the POA, while type IV is the persistence of the lateral branch of the POA [12].

It is interesting to note that the PPOA usually arises from the ICA (ophthalmic segment) and courses below the optic nerve as do the infraoptic variants of the ACA previously described. Therefore, the infraoptic course of the ACA could be imagined as the persistence of the POA limited to its proximal segment. Almost all cases described of PPOAs involved only one side even if Uchino et al. (2011) described a bilateral PPOA [30].

Unpaired ACA

Phylogenic development of the ACA varies from one species to another [4]. For example, in lower primates there is no AComA and only one ACA supplying the medial surface of both hemispheres, while in orangutans there are often three ACAs [80, 81]. In humans, the incidence of an unpaired ACA is probably higher than might be expected. Windle (1888) reported the first case of an unpaired ACA found during a cadaveric dissection [82]. Baptista (1963), proposed a 3-type classification of unpaired ACAs based on his cadaveric study of the arteries of the circle of Willis (Fig. 5) [80]. The first type is the azygos ACA characterized by the pres-

Table 5 Different types of PPOA

Type	Proportion	Origin	Course	Terminaison	Anastomosis with	Embryo precursor
I	84%	Terminal ICA or A1	Olfactory sulcus	Interhemispheric fissure	ACA	Medial branch of the olfactory artery
II	3%	Terminal ICA or A1	Olfactory sulcus	Nasal cavity	Ethmoidal arteries	Medial branch of the olfactory artery
III	6%	Terminal ICA or A1	Olfactory sulcus	1. Nasal cavity 2. Interhemispheric fissure	1. Ethmoidal artery 2. Callosomarginal artery	Medial branch of the olfactory artery
IV	6%	Terminal ICA or A1	Olfactory sulcus	Anterior perforated substance	Perforating arteries	Lateral branch of the olfactory artery

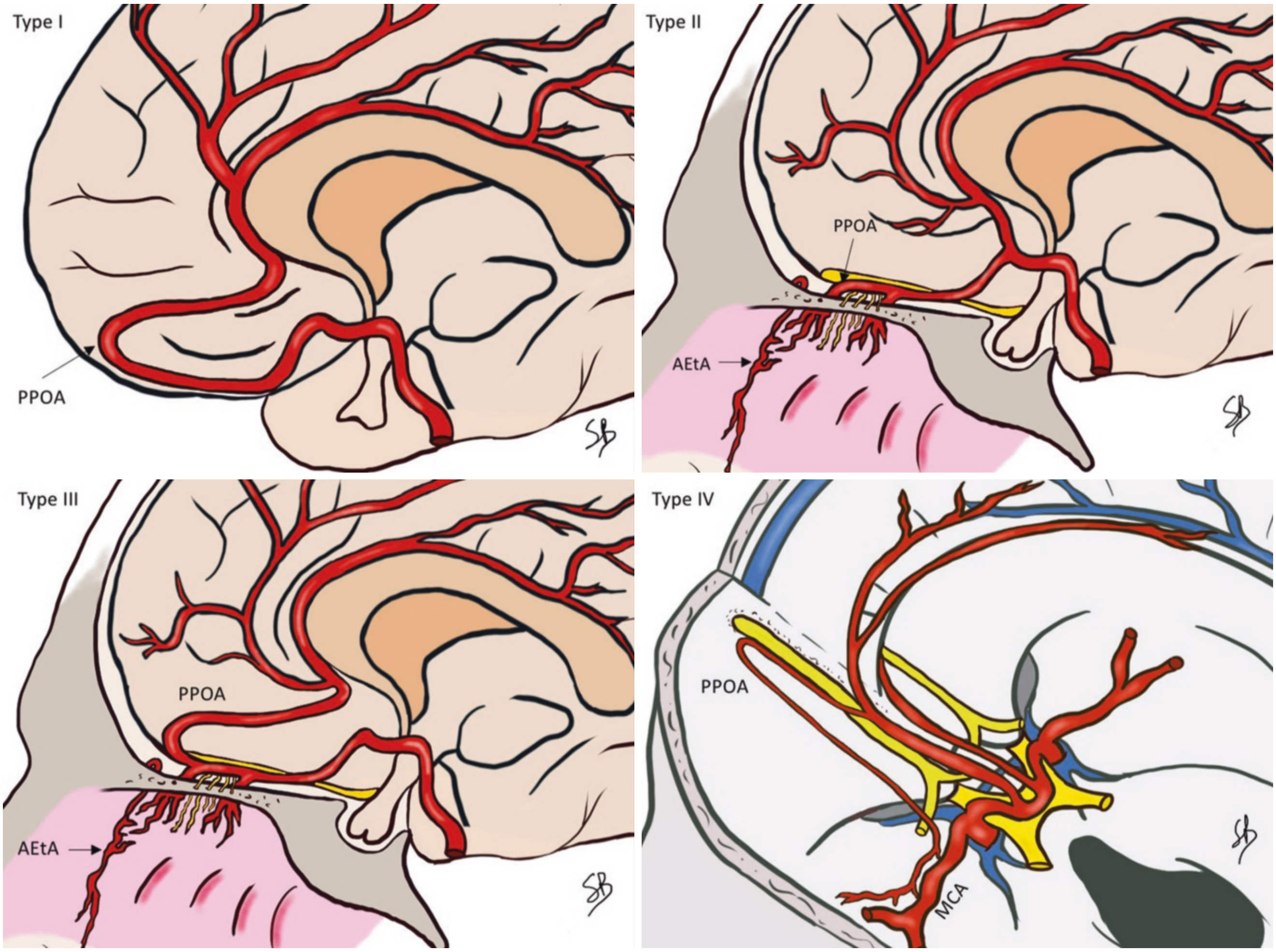


Fig. 4 Artist's illustration of the different types of the primitive persistent olfactory artery (PPOA). Type I of PPOA consists of an abnormal artery that can arise from the ICA or from the A1 segment, that courses anteriorly following the course of the olfactory nerve, and then turns back forming a hairpin to supply the whole ACA vascular territory. In type II, the PPOA courses anteriorly to follow the olfactory nerve and enters the cribriform plate of the ethmoidal bone to enter the nasal fossa and anastomoses with branches of the anterior ethmoidal artery (AEtA). In type III, the PPOA follows the same anterior course but then gives a branch that enters the nasal fossa, and another that forms the hairpin to supply the ACA territories. In type IV, the PPOA. Follows at first the course of the olfactory nerve, but then forms the hairpin to give an artery that follows the course of the recurrent artery of Heubner or of an accessory MCA

ence of only one A2 segment that bifurcates into two branches of equal diameter that supply both hemispheres. The second type is named bi-hemispheric ACA and is defined by an asymmetry in diameter of the A2 segments, the larger one giving branches to both hemispheres. Baptista's last type is the median artery of the corpus callosum (MACC) and is composed by three A2 segments, the third of which may supply only the genu of the corpus callosum (true MACC) or may also supply a cortical territory (accessory ACA). The two first types could be explained by the non-differentiation of two A2 segments during the stage V of Padget and the third type could be explained as the non-regression of the MACC visible in the stage VI of Padget [6].

Azygos ACA (Baptista's type I) is the most frequently described in the literature [9, 12, 16, 80, 81, 83, 84]. Its true incidence is difficult to know because of the disparity of different studies. In his original series, Baptista found 23 cases on 2153 dissected brains (1.1%) [80]. However, following a retrospective review of DSAs over a period of 20 years, Huber et al. (1980) found an azygos ACA in only 0.2% of cases [84]. Uchino et al. (2006), reporting variants of the ACA detected by MRI, found an azygos in 2% of cases (total of 923 cases) [15]. Many isolated cases of azygos ACA were found to be associated with a distal aneurysm (junction A2-A3), highlighting the importance of hemodynamic factors in the formation and rupture of intracranial aneurysms [83–88]. The association of an azygos ACA with a pericallosal aneurysm has an incidence between 13 and 71% in the literature, making the true incidence of this association difficult to know [89]. Huber et al. (1980) found a distal aneu-

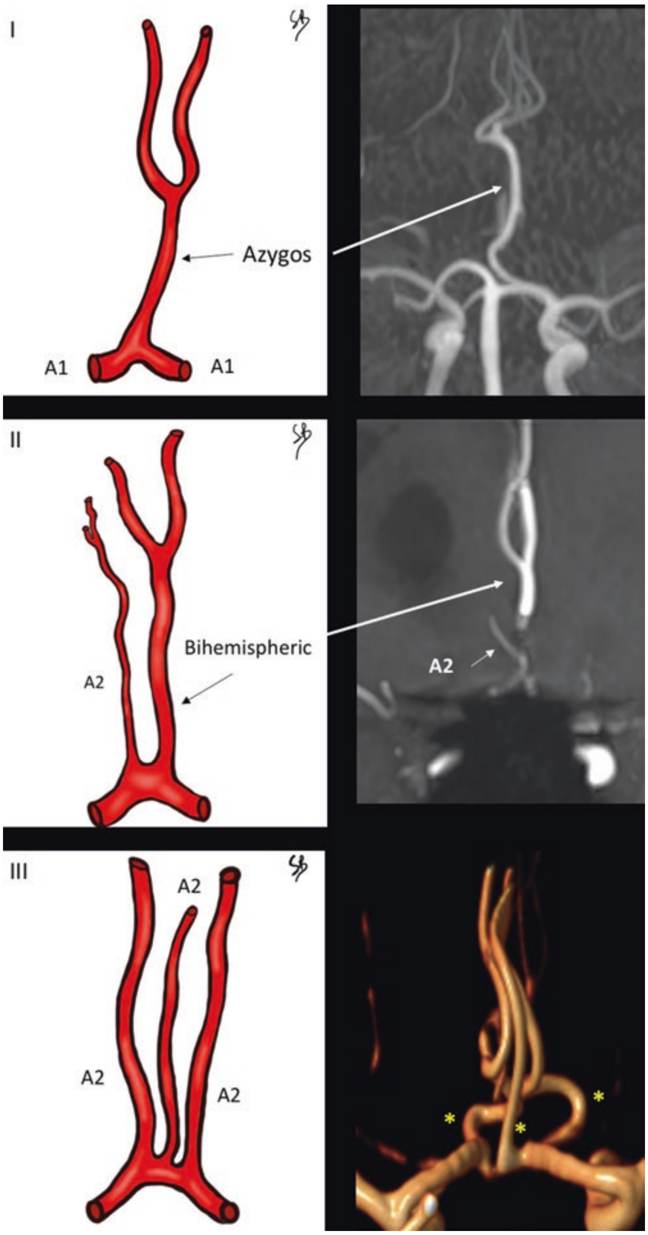


Fig. 5 Baptista's classification for unpaired ACA. (Adapted from [80]). The figure shows artist's illustrations and MRI reconstructions of the three types of unpaired anterior cerebral artery described by Baptista. Type I corresponds to the azygos variant, which consists in a single A2 segment arising from the anterior communicating artery, and that supplies both hemispheres. Type II or bihemispheric ACA is present when there is an asymmetry between the two A2 segments, and the bigger one gives branches to both the hemispheres. In type III there are three A2 segments, with the medial one corresponding to the persistence of the embryonic median artery of the corpus callosum

rysm in 41% of their cases of azygos ACA, whereas Uchino et al. (2006) found it to be 11% [15, 84]. It is well accepted that the presence of an azygos ACA increases the risk of distal ACA aneurysm formation and rupture [89]. Other pathologies were described in association with an azygos ACA as agenesis of the corpus callosum, hydranencephaly, cerebral AVM, or A1 segment fenestration [81]. One case of an azygos ACA has been found to be associated with an infraoptic ACA [90].

Bi-hemispheric ACA (Baptista's type II) is the most difficult to see angiographically or in MRI and is less frequently described in the literature. In his original report, Baptista et al. (1963) noted this variation in 12% of cadaveric dissections but in which only 1 case of a dominant ACA gave bilateral branches (true bi-hemispheric ACA) and not limited to only little branches to the contralateral hemisphere [80]. Perlmutter and Rhoton (1978) made the same observation: 64% of ACAs presented small branches that supplied the contralateral side but in only 1 case a large ACA clearly supplied a bilateral territory [9].

The presence of a third ACA (Baptista's type III) is a more common anatomical variant in which its incidence largely depends on the type of study. Authors made the difference between the MACC, which is limited to supply the genu of the corpus callosum, and the accessory ACA which has in addition a cortical territory [91, 92]. In cadaveric and radiological series, the incidence of these variants is around 2–3% depending on the technique of the study [2, 8, 9, 15]. On the other hand, surgical series for AComA aneurysms noted a higher incidence for the presence of a third A2 segment (4.4–13%) [10, 16, 93, 94].

Fenestrations and Duplications

A fenestrated artery is defined by the presence of two different lumens of the same segment of the artery. On the other hand, a duplicated artery corresponds to two arteries with distinct embryological origins and is due to the non-regression of an embryonic artery [12]. More than 40 cases of ACA fenestration have been reported, even this variant remains less frequent than fenestration of the AcomA [94–103]. The incidence of ACA fenestration varies between 0.1% and 7% according to the type of study, whether cadaveric, angiographic, or surgical [15, 16, 80, 94, 104]. The first author that described an A1 segment fenestration was Fawcett (1906) [104]. He found only 1 case in his cadaveric series of 700 brains (0.14%). On the other hand, Aydin et al. (1997) found an incidence of 7% in their surgical series of AcomA [16].

This variant may be explained by the persistence of two channels from the embryological plexus of the ACA. An anatomical study conducted on 200 fetal dissections highlighted the presence of ACA fenestration in 62.5%, supporting this hypothesis [105]. This hypothesis was not shared by Lasjaunias (2001), who believed that this variant is due to the fusion of the proximal part of the recurrent artery with an A1 perforator, and thus constituting a duplication rather than a fenestration [12].

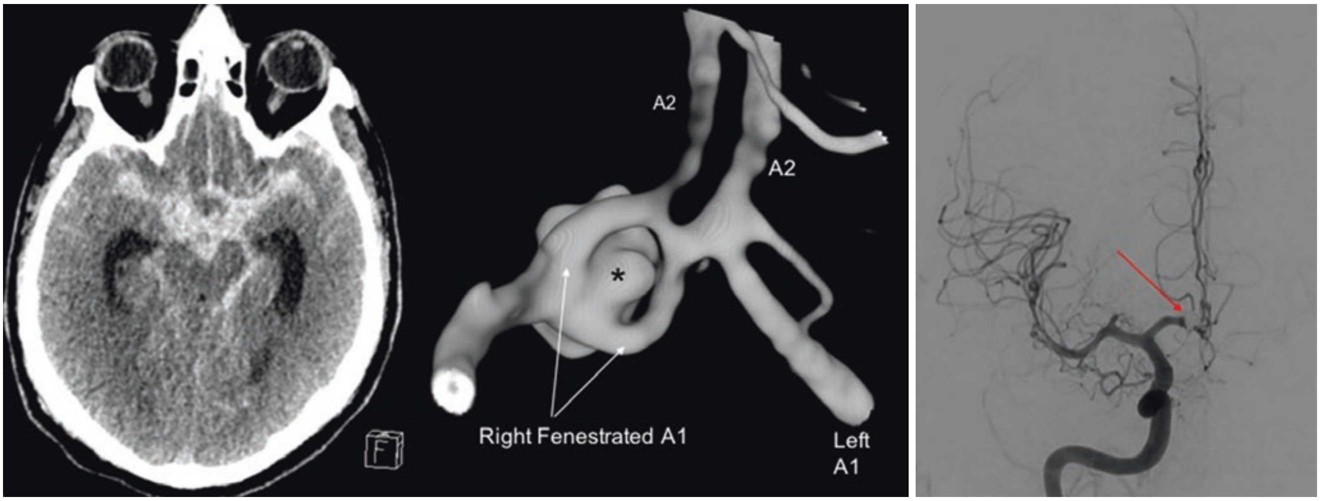


Fig. 6 Clinical case of aneurysm located on a fenestrated A1 segment. The figure shows the case of 56-year-old man, who experienced unusual headache without neurological deficits associated. The CT scan showed a massive subarachnoid hemorrhage and the diagnostic DSA showed a saccular aneurysm (asterisk) located on a right fenestrated A1 segment. The aneurysm was treated with simple coiling (red arrow)

In almost all cases, ACA fenestration involved the distal half of the horizontal segment of the artery (A1 segment). Only few cases of A2 fenestrations have been described as fenestration involving both A1 and A2 segments [12, 15, 106].

ACA fenestrations have been associated with the formation of an aneurysm, which in 27.6% were located at the proximal end of the fenestration [95, 99, 102, 103]. Figure 6 shows a ruptured aneurysm located on a fenestrated A1 segment. Crompton (1962) described a defect of the media layer of the vessel wall at the proximal end of the fenestration. A1 segment fenestration has also been associated with an azygos ACA [62, 95, 98, 100].

Clinical Implications

Knowledge of different anatomical variants of the ACA is essential in vascular neurosurgery and in interventional neuroradiology. Most of the variants described have been discovered because of their associations with other intracranial pathologies, and most commonly with intracranial aneurysms.

The association between the infraoptic course of the ACA and the presence of an intracranial aneurysm is about 60%, with a predominance on the AComA (51%) [32, 40, 44, 49, 50, 52, 53, 55, 57, 59–61, 63, 66, 107–112]. The occurrence of aneurysms in the infraoptic course of the ACA highlights the importance of hemodynamic factors in the formation of aneurysms. Other locations for intracranial aneurysms associated with this variant are the ICA bifurcation (8%) and the contralateral MCA bifurcation (2%) [45, 67, 113]. In two cases, the aneurysm was located on the anastomotic vessel [41, 54]. In one of these two cases, the anastomotic vessel was not infraoptic but transoptic, confirmed by operative photography [41]. In few cases, the OA arises from the anastomotic vessel between the carotid and the ACA [29, 32, 34]. The infraoptic course of the ACA was also associated with contralateral ICA agenesis, an azygos ACA, a persistent trigeminal artery, or an abnormal gyral segmentation [35, 46, 60, 64, 66, 90]. Other associated pathologies described are an arteriovenous malformation, a craniopharyngioma, and a pituitary tumor [51, 56, 65, 67]. The presence of an infraoptic ACA should be recognized on imaging in the planning for surgical clipping of an AComA aneurysm. In these cases, proximal control should be obtained on the ICA instead of on the A1 segment.

Some rare associations with intracranial aneurysms were also described in cases of PPOA. In 15% of PPOA type I cases, the aneurysm was located at the hairpin turn of the PPOA [3, 69, 72, 77]. The presence of such aneurysm was described in 15% of cases. Anosmia is a symptom frequently associated with the PPOA and its incidence is as high as 7% [72]. Other associated pathologies described are an aneurysm of the A1 segment (at the origin of the PPOA), a dural arteriovenous fistula (dAVF) supplied by the PPOA and moyamoya disease [71, 74, 79]. Knowledge of this anatomical variant is necessary in choosing the appropriate surgical approach.

The presence of the azygos ACA variant is strongly associated with the formation of a distal ACA aneurysm. The presence of these variants may require a shorter interval between follow-up imaging. The presence of an azygos ACA may influence the treatment strategies for a distal ACA aneurysm.

Surgical series of AComA found a high incidence of MACC and of A1 segment fenestration. When present, these variants must be recognized before and during surgery. The MACC must be identified and protected.

References

1. Gomes F, Dujovny M, Umansky F, Ausman JI, Diaz FG, Ray WJ, et al. Microsurgical anatomy of the recurrent artery of Heubner. J Neurosurg. 1984;60(1):130–9. https://doi.org/10.3171/jns.1984.60.1.0130.
2. Gomes FB, Dujovny M, Umansky F, Berman SK, Diaz FG, Ausman JI, et al. Microanatomy of the anterior cerebral artery. Surg Neurol. 1986;26(2):129–41. https://doi.org/10.1016/0090-3019(86)90365-4.
3. Tahir RA, Haider S, Kole M, Griffith B, Marin H. Anterior cerebral artery: variant anatomy and pathology. J Vasc Interv Neurol. 2019;10(3):16–22.
4. Abbie AA. The morphology of the fore-brain arteries, with especial reference to the evolution of the basal ganglia. J Anat. 1934;68(Pt 4):433–70.
5. Moffat DB. The embryology of the arteries of the brain. Ann R Coll Surg Engl. 1962;30:368–82.
6. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–62.
7. Rosner SS, Rhoton AL Jr, Ono M, Barry M. Microsurgical anatomy of the anterior perforating arteries. J Neurosurg. 1984;61(3):468–85. https://doi.org/10.3171/jns.1984.61.3.0468.
8. Perlmutter D, Rhoton AL Jr. Microsurgical anatomy of the anterior cerebral-anterior communicating-recurrent artery complex. J Neurosurg. 1976;45(3):259–72. https://doi.org/10.3171/jns.1976.45.3.0259.
9. Perlmutter D, Rhoton AL Jr. Microsurgical anatomy of the distal anterior cerebral artery. J Neurosurg. 1978;49(2):204–28. https://doi.org/10.3171/jns.1978.49.2.0204.
10. Yasargil MG. Microneurosurgery. New York: Thieme Medical Publisher; 1984.
11. Farnarier P, Michotey P, Moscow N, Salamon G. An anatomical and radiological study of the cortical branches of the anterior cerebral artery (author's transl). Ann Radiol (Paris). 1977;20(2):203–21.
12. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer edition; 2001.
13. Lasjaunias P, Moret J, Manelfe C, Theron J, Hasso T, Seeger J. Arterial anomalies at the base of the skull. Neuroradiology. 1977;13(5):267–72. https://doi.org/10.1007/bf00347072.
14. Fisher E. Die Lageabweichungen der vorderen Hirnarterie in Gefassbild. Zentralbl Neurochir. 1938;3:300–12.
15. Uchino A, Nomiyama K, Takase Y, Kudo S. Anterior cerebral artery variations detected by MR angiography. Neuroradiology. 2006;48(9):647–52. https://doi.org/10.1007/s00234-006-0110-3.
16. Aydin IH, Takci E, Kadioglu HH, Tuzun Y, Kayaoglu CR, Barlas E. Vascular variations associated with anterior communicating artery aneurysms-an intraoperative study. Minim Invasive Neurosurg. 1997;40(1):17–21. https://doi.org/10.1055/s-2008-1053407.
17. Rhoton AL Jr, Perlmutter D. Microsurgical anatomy of anterior communicating artery aneurysms. Neurol Res. 1980;2(3–4):217–51. https://doi.org/10.1080/01616412.1980.11739581.
18. Kaplan HA. The lateral perforating branches of the anterior and middle cerebral arteries. J Neurosurg. 1965;23(3):305–10. https://doi.org/10.3171/jns.1965.23.3.0305.
19. Marinkovic S, Milisavljevic M, Kovacevic M. Anatomical bases for surgical approach to the initial segment of the anterior cerebral artery. Microanatomy of Heubner's artery and perforating branches of the anterior cerebral artery. Surg Radiol Anat. 1986;8(1):7–18.
20. Barry DM. Surgical treatment of anterior cerebral-anterior communicating artery aneurysms. R I Med J. 1972;55(8):245–7.
21. Kaplan HA, Krieger AJ. Vascular anatomy of the preoptic region of the brain. Acta Neurochir. 1980;54(3–4):233–41. https://doi.org/10.1007/bf01407090.
22. Stefani MA, Schneider FL, Marrone AC, Severino AG, Jackowski AP, Wallace MC. Anatomic variations of anterior cerebral artery cortical branches. Clin Anat. 2000;13(4):231–6. https://doi.org/10.1002/1098-2353(2000)13:4<231::AID-CA1>3.0.CO;2-T.
23. Krishnamoorthy T, Gupta AK, Bhattacharya RN, Rajesh BJ, Purkayastha S. Anomalous origin of the callosomarginal artery from the A1 segment with an associated saccular aneurysm. AJNR Am J Neuroradiol. 2006;27(10):2075–7.
24. Ture U, Yasargil MG, Krisht AF. The arteries of the corpus callosum: a microsurgical anatomic study. Neurosurgery. 1996;39(6):1075–84; discussion 84-5. https://doi.org/10.1097/00006123-199612000-00001.
25. van der Zwan A, Hillen B, Tulleken CA, Dujovny M, Dragovic L. Variability of the territories of the major cerebral arteries. J Neurosurg. 1992;77(6):927–40. https://doi.org/10.3171/jns.1992.77.6.0927.
26. Picard L, Vignaud J, Lombardi G, Roland J. Radiological anatomy of the origin of the ophthalmic artery. Mod Probl Ophthalmol. 1975;14:164–9.
27. Hassler W, Zentner J, Voigt K. Abnormal origin of the ophthalmic artery from the anterior cerebral artery: neuroradiological and intraoperative findings. Neuroradiology. 1989;31(1):85–7. https://doi.org/10.1007/bf00342037.
28. Baltsavias G, Turk Y, Valavanis A. Persistent ventral ophthalmic artery associated with supraclinoid internal carotid artery aneurysm: case report and review of the literature. J Neuroradiol. 2012;39(3):186–9. https://doi.org/10.1016/j.neurad.2011.08.002.
29. Hannequin P, Peltier J, Destrieux C, Velut S, Havet E, Le Gars D. The inter-optic course of a unique precommunicating anterior cerebral artery with aberrant origin of an ophthalmic artery: an anatomic case report. Surg Radiol Anat. 2013;35(3):269–71. https://doi.org/10.1007/s00276-012-1028-6.
30. Uchino A, Saito N, Kozawa E, Mizukoshi W, Inoue K. Persistent primitive olfactory artery: MR angiographic diagnosis. Surg Radiol Anat. 2011;33(3):197–201. https://doi.org/10.1007/s00276-010-0743-0.
31. Islak C, Ogut G, Numan F, Cokyuksel O, Kuday C. Persistent nonmigrated ventral primitive ophthalmic artery. Report on one case. J Neuroradiol. 1994;21(1):46–9.
32. Honma Y, Ogawa T, Nagao S. Angiographically occult anomalous ophthalmic artery arising from the anterior cerebral artery. Acta Neurochir. 1997;139(5):480–1. https://doi.org/10.1007/bf01808888.
33. Belotti F, Ferrari M, Doglietto F, Cocchi MA, Lancini D, Buffoli B, et al. Ophthalmic artery originating from the anterior cerebral artery: anatomo-radiological study, histological analysis, and literature review. Neurosurg Rev. 2016;39(3):483–93. https://doi.org/10.1007/s10143-016-0715-x.
34. Uchino A, Saito N, Tanahashi N. Bilateral carotid-anterior cerebral artery anastomoses associated with bilateral ophthalmic arteries arising from the anastomotic arteries diagnosed by magnetic resonance angiography: a case report. Surg Radiol Anat. 2018;40(6):721–5. https://doi.org/10.1007/s00276-017-1953-5.
35. Matsuura K, Uchino A, Saito N, Ishida J, Suzuki T. Carotid-anterior cerebral artery (ACA) anastomosis associated with azygos ACA and ophthalmic artery arising from the middle meningeal artery: a case report. Surg Radiol Anat. 2020;42(2):211–4. https://doi.org/10.1007/s00276-019-02353-1.

36. Robinson LR. An unusual human anterior cerebral artery. J Anat. 1959;93(1):131–3.
37. Nutik S, Dilenge D. Carotid-anterior cerebral artery anastomosis. Case report. J Neurosurg. 1976;44(3):378–82. https://doi.org/10.3171/jns.1976.44.3.0378.
38. Mercier P, Velut S, Fournier D, Lescalie F, Guy G, Pillet J, et al. A rare embryologic variation: carotid-anterior cerebral artery anastomosis or infraoptic course of the anterior cerebral artery. Surg Radiol Anat. 1989;11(1):73–7. https://doi.org/10.1007/bf02102250.
39. Bosma NJ. Infra-optic course of anterior cerebral artery and low bifurcation of the internal carotid artery. Acta Neurochir. 1977;38(3–4):305–12. https://doi.org/10.1007/bf01401101.
40. Kessler LA. Unusual anomaly of the anterior cerebral artery: report of a case. Arch Neurol. 1979;36(8):509–10. https://doi.org/10.1001/archneur.1979.00500440079017.
41. Maurer J, Maurer E, Perneczky A. Surgically verified variations in the A1 segment of the anterior cerebral artery. Report of two cases. J Neurosurg. 1991;75(6):950–3. https://doi.org/10.3171/jns.1991.75.6.0950.
42. Given CA 2nd, Morris PP. Recognition and importance of an infraoptic anterior cerebral artery: case report. AJNR Am J Neuroradiol. 2002;23(3):452–4.
43. Isherwood I, Dutton J. Unusual anomaly of anterior cerebral artery. Acta Radiol Diagn (Stockh). 1969;9:345–51.
44. Chua MH, Thomas AJ, Fusco MR, Ogilvy CS. An unruptured anterior communicating artery aneurysm with bilateral infraoptic anterior cerebral arteries. Case report and review of the literature. J Cerebrovasc Endovasc Neurosurg. 2014;16(4):368–73. https://doi.org/10.7461/jcen.2014.16.4.368.
45. Yi Z, Sasaki T, Satoh D, Muruta T, Kitazawa K, Kobayashi S. Carotid-anterior cerebral artery anastomosis associated with Paraclinoid aneurysm and ophthalmic artery originating from middle meningeal artery: case report and consideration of terminology. World Neurosurg. 2016;88(689):e1–4. https://doi.org/10.1016/j.wneu.2015.11.021.
46. Turnbull I. Agenesis of the internal carotid artery. Neurology. 1962;12:588–90. https://doi.org/10.1212/wnl.12.9.588.
47. Brismar J, Ackerman R, Roberson G. Anomaly of anterior cerebral artery. A case report and embryologic considerations. Acta Radiol Diagn (Stockh). 1977;18(2):154–60. https://doi.org/10.1177/028418517701800202.
48. Milenkovic Z. Anastomosis between internal carotid artery and anterior cerebral artery with other anomalies of the circle of Willis in a fetal brain. J Neurosurg. 1981;55(5):701–3. https://doi.org/10.3171/jns.1981.55.5.0701.
49. Bernini FP, Cioffi FA, Muras I, Rinaldi F. Carotid syphon—pericallosal arterial anastomosis. Case report. Acta Neurochir (Wien). 1982;66(1–2):61–9. https://doi.org/10.1007/bf01809304.
50. Senter HJ, Miller DJ. Interoptic course of the anterior cerebral artery associated with anterior cerebral artery aneurysm. Case report. J Neurosurg. 1982;56(2):302–4. https://doi.org/10.3171/jns.1982.56.2.0302.
51. Sheehy JP, Kendall BE, Thomas DG. Infraoptic course of the anterior cerebral artery associated with a pituitary tumor. Surg Neurol. 1983;20(2):97–9. https://doi.org/10.1016/0090-3019(83)90456-1.
52. Bollar A, Martinez R, Gelabert M, Garcia A. Anomalous origin of the anterior cerebral artery associated with aneurysm—embryological considerations. Neuroradiology. 1988;30(1):86. https://doi.org/10.1007/bf00341953.
53. Onishi H, Yamashita J, Enkaku F, Fujisawa H. Anomalous origin of the anterior cerebral artery and congenital skull dysplasia. Case report. Neurol Med Chir (Tokyo). 1992;32(5):296–9. https://doi.org/10.2176/nmc.32.296.
54. Sakai K, Asari S, Fujisawa M, Katagi R. Ruptured aneurysm arising from the anomalous anterior cerebral artery. Case report. Neurol Med Chir (Tokyo). 1992;32(11):846–50. https://doi.org/10.2176/nmc.32.846.
55. Ladzinski P, Maliszewski M, Majchrzak H. The accessory anterior cerebral artery: case report and anatomic analysis of vascular anomaly. Surg Neurol. 1997;48(2):171–4. https://doi.org/10.1016/s0090-3019(96)00421-1.
56. Singer RJ, Abe T, Taylor WH, Marks MP, Norbash AM. Intracavernous anterior cerebral artery origin with associated arteriovenous malformations: a developmental analysis: case report. Neurosurgery. 1997;40(4):829–31; discussion 31. https://doi.org/10.1097/00006123-199704000-00033.
57. Al-Qahtani S, Tampieri D, Brassard R, Sirhan D, Mellanson D. Coil embolization of an aneurysm associated with an infraoptic anterior cerebral artery in a child. AJNR Am J Neuroradiol. 2003;24(5):990–1.
58. Burbank NS, Morris PP. Unique anomalous origin of the left anterior cerebral artery. AJNR Am J Neuroradiol. 2005;26(10):2533–5.
59. Kilic K, Orakdogen M, Bakirci A, Berkman Z. Bilateral internal carotid to anterior cerebral artery anastomosis with anterior communicating artery aneurysm: technical case report. Neurosurgery. 2005;57(4 Suppl):E400; discussion E
60. Akiyama Y, Okada T, Hayashi N, Yokoi T. Infraoptic course of the anterior cerebral artery originating from the extradural internal carotid artery associated with contralateral internal carotid artery agenesis and multiple intracerebral aneurysms. Neurol Med Chir (Tokyo). 2010;50(11):984–7. https://doi.org/10.2176/nmc.50.984.
61. Odake G. Carotid-anterior cerebral artery anastomosis with aneurysm: case report and review of the literature. Neurosurgery. 1988;23(5):654–8. https://doi.org/10.1227/00006123-198811000-00021.
62. Friedlander RM, Oglivy CS. Aneurysmal subarachnoid hemorrhage in a patient with bilateral A1 fenestrations associated with an azygos anterior cerebral artery. Case report and literature review. J Neurosurg. 1996;84(4):681–4. https://doi.org/10.3171/jns.1996.84.4.0681.
63. Fujimura M, Seki H, Sugawara T, Tomichi N, Oku T, Higuchi H. Anomalous internal carotid artery-anterior cerebral artery anastomosis associated with fenestration and cerebral aneurysm. Neurol Med Chir (Tokyo). 1996;36(4):229–33. https://doi.org/10.2176/nmc.36.229.
64. McLaughlin N, Bojanowski MW. Infraoptic course of anterior cerebral arteries associated with abnormal gyral segmentation. Case report. J Neurosurg. 2007;107(2):430–4. https://doi.org/10.3171/JNS-07/08/0430.
65. Stevenson CB, Chambless LB, Rini DA, Thompson RC. Bilateral infraoptic A1 arteries in association with a craniopharyngioma: case report and review of the literature. Surg Neurol Int. 2011;2:89. https://doi.org/10.4103/2152-7806.82371.
66. Turkoglu E, Arat A, Patel N, Kertmen H, Baskaya MK. Anterior communicating artery aneurysm associated with an infraoptic course of anterior cerebral artery and rare variant of the persistent trigeminal artery: a case report and literature review. Clin Neurol Neurosurg. 2011;113(4):335–40. https://doi.org/10.1016/j.clineuro.2010.12.009.
67. Wong ST, Yuen SC, Fok KF, Yam KY, Fong D. Infraoptic anterior cerebral artery: review, report of two cases and an anatomical classification. Acta Neurochir. 2008;150(10):1087–96. https://doi.org/10.1007/s00701-008-0016-4.
68. Kim MS, Lee GJ. Persistent primitive olfactory artery: CT angiographic diagnosis and literature review for classification and clinical significance. Surg Radiol Anat. 2014;36(7):663–7. https://doi.org/10.1007/s00276-013-1239-5.
69. Moffat DB. A case of peristence of the primitive olfactory artery. Anat Anz. 1967;121(5):477–9.

70. Kim MS. Persistent primitive olfactory artery connected with middle cerebral artery: case report. Surg Radiol Anat. 2013;35(9):849–52. https://doi.org/10.1007/s00276-013-1147-8.
71. Lin WC, Hsu SW, Kuo YL, Feekes JA, Wang HC. Combination of olfactory course anterior cerebral artery and accessory middle cerebral artery (MCA) with occluded in situ MCA and related moyamoya phenomenon. Brain and Development. 2009;31(4):318–21. https://doi.org/10.1016/j.braindev.2008.05.008.
72. Nozaki K, Taki W, Kawakami O, Hashimoto N. Cerebral aneurysm associated with persistent primitive olfactory artery aneurysm. Acta Neurochir. 1998;140(4):397–401; discussion −2. https://doi.org/10.1007/s007010050114.
73. Tsuji T, Abe M, Tabuchi K. Aneurysm of a persistent primitive olfactory artery. Case report. J Neurosurg. 1995;83(1):138–40. https://doi.org/10.3171/jns.1995.83.1.0138.
74. Tsutsumi S, Shimizu Y, Nonaka Y, Abe Y, Yasumoto Y, Ito M, et al. Arteriovenous fistula arising from the persistent primitive olfactory artery with dual supply from the bilateral anterior ethmoidal arteries. Neurol Med Chir (Tokyo). 2009;49(9):407–9. https://doi.org/10.2176/nmc.49.407.
75. Uchino A, Sawada A, Takase Y, Kudo S. Persistent primitive olfactory artery: diagnosis with MR angiography. Clin Imaging. 2001;25(4):258–61. https://doi.org/10.1016/s0899-7071(01)00294-7.
76. Vasovic L, Trandafilovic M, Vlajkovic S, Jovanovic I, Ugrenovic S. Persistent primitive olfactory artery in Serbian population. Biomed Res Int. 2013;2013:903460. https://doi.org/10.1155/2013/903460.
77. Yamamoto T, Suzuki K, Yamazaki T, Tsuruta W, Tsurubuchi T, Matsumura A. Persistent primitive olfactory artery aneurysm. Neurol Med Chir (Tokyo). 2009;49(7):303–5. https://doi.org/10.2176/nmc.49.303.
78. Signorelli F, Scholtes F, Kauffmann C, McLaughlin N, Bojanowski MW. Dural artery from the anterior cerebral artery to the planum sphenoidale. Acta Neurochir. 2010;152(10):1807–8. https://doi.org/10.1007/s00701-010-0716-4.
79. Horie N, Morikawa M, Fukuda S, Hayashi K, Suyama K, Nagata I. New variant of persistent primitive olfactory artery associated with a ruptured aneurysm. J Neurosurg. 2012;117(1):26–8. https://doi.org/10.3171/2012.3.JNS111932.
80. Baptista AG. Studies of the arteries of the brain. II. The anterior cerebral artery: some anatomic features and their clinical implications. Neurology. 1963;13:825–35.
81. LeMay M, Gooding CA. The clinical significance of the azygos anterior cerebral artery (a.C.a.). Am J Roentgenol Radium Therapy, Nucl Med. 1966;98(3):602–10. https://doi.org/10.2214/ajr.98.3.602.
82. Windle BC. The arteries forming the circle of Willis. J Anat Physiol. 1888;22(Pt 2):289–93.
83. Katz RW, Horoupian DS, Zingesser L. Aneurysm of azygous anterior cerebral artery. A case report. J Neurosurg. 1978;48(5):804–8. https://doi.org/10.3171/jns.1978.48.5.0804.
84. Huber P, Braun J, Hirschmann D, Agyeman JF. Incidence of berry aneurysms of the unpaired pericallosal artery: angiographic study. Neuroradiology. 1980;19(3):143–7. https://doi.org/10.1007/bf00342389.
85. Hashizume K, Nukui H, Horikoshi T, Kaneko M, Fukamachi A. Giant aneurysm of the azygos anterior cerebral artery associated with acute subdural hematoma. Case report. Neurol Med Chir (Tokyo). 1992;32(9):693–7. https://doi.org/10.2176/nmc.32.693.
86. Kanemoto Y, Tanaka Y, Nonaka M, Hironaka Y. Giant aneurysm of the azygos anterior cerebral artery. Case report. Neurol Med Chir (Tokyo). 2000;40(9):472–5. https://doi.org/10.2176/nmc.40.472.
87. Kondo A, Koyama T, Ishikawa J, Iwaki K, Yamasaki T. Ruptured aneurysm of an azygos anterior cerebral artery. Neuroradiology. 1979;17(4):227–9. https://doi.org/10.1007/bf00342754.
88. Niizuma H, Kwak R, Uchida K, Suzuki J. Aneurysms of the azygos anterior cerebral artery. Surg Neurol. 1981;15(3):225–8. https://doi.org/10.1016/0090-3019(81)90150-6.
89. Hayashi M, Kobayashi H, Kawano H, Handa Y, Kabuto M. Giant aneurysm of an azygos anterior cerebral artery: report of two cases and review of the literature. Neurosurgery. 1985;17(2):341–4. https://doi.org/10.1227/00006123-198508000-00020.
90. Handa J, Matsuda M, Koyama T, Handa H, Kikuchi H. Internal carotid aneurysm associated with multiple anomalies of cerebral arteries. Neuroradiology. 1971;2(4):230–3. https://doi.org/10.1007/bf00337573.
91. Yanaka K, Shirai S, Shibata Y, Nose T. Ruptured aneurysm at the origin of the median artery of the corpus callosum associated with accessory middle cerebral artery—Case report. Neurol Med Chir (Tokyo). 2000;40(10):511–4. https://doi.org/10.2176/nmc.40.511.
92. Gibbons K, Hopkins LN, Heros RC. Occlusion of an "accessory" distal anterior cerebral artery during treatment of anterior communicating artery aneurysms. Report of two cases. J Neurosurg. 1991;74(1):133–5. https://doi.org/10.3171/jns.1991.74.1.0133.
93. Kwak R, Niizuma H, Hatanaka M, Suzuki J. Anterior communicating artery aneurysms with associated anomalies. J Neurosurg. 1980;52(2):162–4. https://doi.org/10.3171/jns.1980.52.2.0162.
94. Ogawa A, Suzuki M, Sakurai Y, Yoshimoto T. Vascular anomalies associated with aneurysms of the anterior communicating artery: microsurgical observations. J Neurosurg. 1990;72(5):706–9. https://doi.org/10.3171/jns.1990.72.5.0706.
95. Crompton MR. The pathology of ruptured middle-cerebral aneurysms with special reference to the differences between the sexes. Lancet. 1962;2(7253):421–5. https://doi.org/10.1016/s0140-6736(62)90281-7.
96. Teal JS, Rumbaugh CL, Bergeron RT, Segall HD. Angiographic demonstration of fenestrations of the intradural intracranial arteries. Radiology. 1973;106(1):123–6. https://doi.org/10.1148/106.1.123.
97. Ito J, Washiyama K, Kim CH, Ibuchi Y. Fenestration of the anterior cerebral artery. Neuroradiology. 1981;21(5):277–80. https://doi.org/10.1007/bf02100160.
98. Kobayashi S, Yuge T, Sugita Y, Kuratomi A, Katayama M, Iryo O, et al. Azygos anterior cerebral artery aneurysm associated with fenestration of the anterior cerebral artery. Kurume Med J. 1986;33(3):149–53. https://doi.org/10.2739/kurumemedj.33.149.
99. Korosue K, Kuwamura K, Okuda Y, Tamaki N, Matsumoto S. Saccular aneurysm arising from a fenestrated anterior cerebral artery. Surg Neurol. 1983;19(3):273–5. https://doi.org/10.1016/s0090-3019(83)80015-9.
100. Matsumura M, Nojiri K. Ruptured anterior communicating artery aneurysms associated with fenestration of the anterior cerebral artery. Surg Neurol. 1984;22(4):371–6. https://doi.org/10.1016/0090-3019(84)90142-3.
101. Minakawa T, Kawamata M, Hayano M, Kawakami K. Aneurysms associated with fenestrated anterior cerebral arteries. Report of four cases and review of the literature. Surg Neurol. 1985;24(3):284–8. https://doi.org/10.1016/0090-3019(85)90040-0.
102. Ogasawara H, Inagawa T, Yamamoto M, Kamiya K. Aneurysm in a fenestrated anterior cerebral artery. Case report. Neurol Med Chir (Tokyo). 1988;28(6):575–8. https://doi.org/10.2176/nmc.28.575.
103. Yamada T, Inagawa T, Takeda T. Ruptured aneurysm at the anterior cerebral artery fenestration. Case report. J Neurosurg. 1982;57(6):826–8. https://doi.org/10.3171/jns.1982.57.6.0826.
104. Fawcett E, Blachford JV. The circle of Willis: an examination of 700 specimens. J Anat Physiol. 1905;40(Pt 1):632–70.

105. Vucetic RR. Segmental duplication of the fetal anterior cerebral artery. J Anat. 1998;192(Pt 3):431–4. https://doi.org/10.1046/j.1469-7580.1998.19230431.x.
106. Dimmick SJ, Faulder KC. Fenestrated anterior cerebral artery with associated arterial anomalies. Case reports and literature review. Interv Neuroradiol. 2008;14(4):441–5. https://doi.org/10.1177/159101990801400410.
107. Fujimoto S, Murakami M. Anomalous branch of the internal carotid artery supplying circulation of the anterior cerebral artery. Case report. J Neurosurg. 1983;58(6):941–6. https://doi.org/10.3171/jns.1983.58.6.0941.
108. Hillard VH, Musunuru K, Nwagwu C, Das K, Murali R, Zablow B, et al. Treatment of an anterior communicating artery aneurysm through an anomalous anastomosis from the cavernous internal carotid artery. J Neurosurg. 2002;97(6):1432–5. https://doi.org/10.3171/jns.2002.97.6.1432.
109. Yurt A, Ucar K, Ozer F, Oran I, Arda N. A rare embryologic variation: anterior communicating artery aneurysm associated with carotid-anterior cerebral artery anastomosis or infraoptic course of the anterior cerebral artery. Clin Med Case Rep. 2008;1:123–5. https://doi.org/10.4137/ccrep.s918.
110. Chakraborty S, Fanning NF, Lee SK, Terbrugge KG. Bilateral infraoptic origin of anterior cerebral arteries: a rare anomaly and its embryological and clinical significance. Interv Neuroradiol. 2006;12(2):155–9. https://doi.org/10.1177/159101990601200210.
111. Rosenorn J, Ahlgren P, Ronde F. Pre-optic origin of the anterior cerebral artery. Neuroradiology. 1985;27(3):275–7. https://doi.org/10.1007/bf00344501.
112. Kim MS, Sim SY. Infraoptic anterior cerebral artery: case series report and literature review. Surg Radiol Anat. 2016;38(8):887–91. https://doi.org/10.1007/s00276-016-1651-8.
113. Ji C, Ahn JG. Infraoptic course of both anterior cerebral arteries. J Korean Neurosurg Soc. 2010;47(1):71–3. https://doi.org/10.3340/jkns.2010.47.1.71.
114. Fisher AG. A case of complete absence of both internal carotid arteries, with a preliminary note on the developmental history of the stapedial artery. J Anat Physiol. 1913;48(Pt 1):37–46.

Anatomy and Variations of the Anterior Communicating Artery

Sara Bonasia, Thomas Robert, and Michel W. Bojanowski

The anterior communicating artery (AComA) is one of the most studied cerebral arteries because of its predisposition to bear cerebral aneurysms [1–5]. Even if its study and analysis is limited radiologically, numerous anatomical and surgical series described in detail the AcomA [2, 3, 5–8]. In this chapter, after a short reminder about the embryology of the AComA, we will describe the anatomy of the artery, its branches, and its variations.

Embryology

Our knowledge regarding the embryological development of the cerebral vasculature is mainly based on embryo dissections performed at the Carnegie Institution by Streeter (1918) and Padget (1948), who described different stages in vasculature development after having analyzed 22 sectioned embryos [9, 10]. In this chapter, we will not develop all the embryology of the anterior cerebral artery (ACA), whose main stages are resumed in Table 1 and described in chapter "Embryology, Anatomy, and Variations of the Anterior Cerebral Artery," but we will only remind the fundamental steps for the formation of the AComA.

At stage VI (20–24 mm), the primitive ACA clearly has an upper course between the two cerebral hemispheres and gives different anastomoses to the choroidal arteries [11]. The AComA starts its evolution and is completely formed in embryos of 24 mm, concluding the formation of the circle of Willis [6]. The AComA is consequently the last artery of the circle of Willis to develop. Padget noted that the AComA is in almost all embryos seen as a developed network between the two ACA that progressively fuse to give only one artery [9]. At this stage, a superior branch from the AComA is visible. Its name is medial artery of the corpus callosum (MACC) and can persist or regress in the adult [9, 10].

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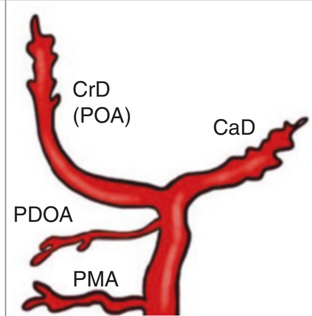
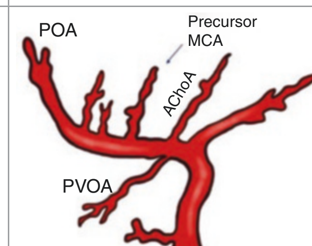
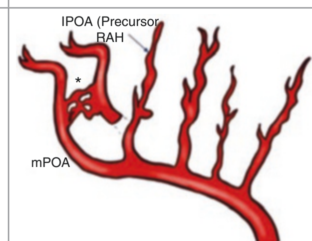
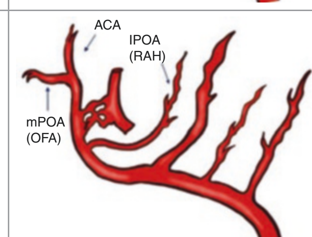
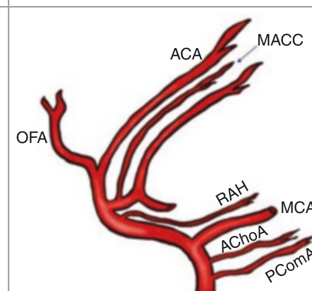
Table 1 Embryological development of the cranial division of the primitive ICA

Stage of Padget	Size of embryo (mm)	Events	Illustration
I	4–5	– Cranial and caudal division of the carotid artery (CrD, CaD) appearance – CrD is the primitive olfactory artery (POA) – Primitive maxillary artery (PMA) and primitive dorsal ophthalmic artery (PDOA) appearance	
III	7–12	– Branches of the POA appearance: Anterior coroidal artery (AChoA), primitive ventral ophthalmic artery (PVOA), precursor of the middle cerebral artery (MCA)	
IV	12–14	– POA division in lateral (lPOA) and medial (mPOA) branches – The mPOA borrows the future ACA – The lPOA is the precursor of the recurrent artery of Heubner (RAH) – Plexiform anastomosis between the two ACAs (*, future AcomA)	
V	16–18	– ACA development with terminal part of the medial POA appearing as a branch, future orbito-frontal artery (OFA) – Lateral extension of the lPOA (future RAH)	
VI	20–24	– Formation of the AComA (complete circle of Willis) – Medial artery of the corpus callosum visible (MACC)	

The Anterior Communicating Artery

The AComA is a little transversal artery located in the lamina terminalis cistern stretched between the two ACAs and represents the limit between the first (A1, horizontal) and the second (A2, vertical) segments of the ACA [7, 12]. Table 2 summarizes different anatomical variations of the AComA described in the literature. The AComA is called "normal" in textbooks if it is only one artery with a length of 0.1–3 mm and a diameter comprises between 1 and 3 mm [5]. This "normal" configuration is actually found in only 20–58% of cases depending on the type of study [4, 5, 12–14]. Already

Table 2 Anatomical variations of the AComA

Author (year)	Number of brains	Method	Mean length of ACoA (mm) (range)	Mean diameter of ACoA (mm) (range)	Number of ACoA(s) (%)
Serizawa et al. (1997)	30	Cadaveric	4.0 (1.5–8.8)	1.7 (0.2–2.5)	Single: 40 Double: 33 Multichannels: 27
Marinkovic et al. (1990)	26	Cadaveric	2.9 (0.8–4.6)	1.2 (0.7–2.4)	Fusion: 4.5 Single: 41 Double: 4.5 Triple: 18 Multichannels: 32
Perlmutter and Rhoton (1976)	50	Cadaveric	2.6 (0.3–7.0)	1.5 (0.2–3.4)	Single: 60 Double: 30 Triple: 10
Tulleken (1978)	75	Cadaveric	N.D.	N.D.	Absent: 2 Single: 57 Multichannels: 41
Tao et al. (2006)	45	Cadaveric	3.3 (0–9.5)	1.2 (0.2–3.9)	Single: 35.6 Complex: 64.4
Yasargil (1984)	200	Cadaveric	N.D.	N.D.	Single: 57 Double: 20.5 Triple: 18.5 Network: 4
Yasargil (1984)	375	Operative	N.D.	N.D.	Single: 59.7 Double: 22.4 Triple: 15.2 Network: 2.7
Windle (1888)	200	Cadaveric	N.D.	N.D.	Single: 79.5 Double: 7 Triple: 3.5

in 1930, De Almeida defined 20 anatomical variants of the AComA complex based on its cadaveric observations [15]. In almost all studies, the average diameter of the artery is between 1 and 2 mm [2, 3, 5]. A hypoplastic AComA, defined as an artery with a diameter inferior to 1 mm, is rare. Perlmutter and Rhoton (1976) found 16% of hypoplastic AComA in their cadaveric study but other authors noted a lowest rate: 1.5% for Yasargil (1984) and 5.4% for Tulleken (1978) [2, 4, 5]. An aplasia of the AComA is very rare and is defined as the absence of communication between the two ACAs. Tulleken (1978) was the lonely author who described this variation [4]. He noted only one case among 75 cadaveric brains dissection. Few authors also described a fusion of the two ACA without true AComA between them and called this variant: fused ACA. Marinkovic et al. (1990) described this variation in 4.5% of cases and Serizawa et al. (1997) in 12% [12, 13].

In case of AComA aneurysms, an asymmetry in diameter between the two A1 segments is frequent and most frequently seen than in the population without AComA aneurysm [1, 3, 16–18]. Uchino et al. (2006) noted an asymmetry in the diameter of the A1 segments in 5–7% of MRI realized for other pathologies than an AComA aneurysm [19]. On the other hand, Yasargil (1984) described an asymmetry of A1 segments in 80% of his patients with AComA aneurysm [5].

Branches of the Anterior Communicating Artery

Prior to the introduction of the operative microscope, branches of the AComA were not recognized by anatomists and surgeons [5]. For example, in cadaveric works, Critchley (1930) did not describe any branches arising from the ACA believing that the perforating branches arise only from the ACA [1]. In fact, embryologically, it is difficult to explain the presence of perforating branches arising directly from the AComA, since it forms as a plexiform complex [9]. This is why some authors consider these perforating branches as branches of the ACA instead of branches of the AcomA [10, 11]. One exception, the medial artery of the corpus callosum may be embryologically explained since this artery can be observed in the embryo at Padget's stage VI (24 mm) [9].

In the era of the operative microscope, these perforating branches were well-studied, their number varies from 2 to 7 [12, 20]. Yasargil noted that the origin of these branches depends on the symmetry of the A1 segments. In case of symmetric A1 segments, the perforating branches arise from the AComA, in case of asymmetric A1 segments, these branches arise from the A1-A2 angle ipsilateral of the larger A1 [5].

Table 3 Perforating branches of the AComA

ACoA branches	Average diameter (mm)	Origin	Territory	Bilateral supply
Subcallosal branch	0.5 (0.4–0.8)	Postero-superior aspect	Cingulate gyrus Rostrum CC Lamina terminalis Septum pellucidum	90%
Hypothalamic branch	0.19 (0.1–0.3)	Postero-inferior aspect	Preoptic area of the hypothalamus	10%
Chiasmatic branch	0.1	Postero-inferior aspect	Optic chiasm	No

AComA branches have an average diameter of 0.25 mm (0.1–0.8 mm) and arise from the posterior aspect of the artery [2, 3, 7, 12–14, 17, 21]. They share a common stem from the AComA in 65% of cases [4].

Serizawa et al. (1997) published an interesting microsurgical analysis of perforating branches from the AComA and proposed to classify them into 3 different groups, which are summarized in Table 3 [13]. The first group is subcallosal and represents the largest branch (0.4–0.8 mm) with bilateral supply. The second group represents the hypothalamic branches. These branches are 1 to 6 arteries of 0.1–0.3 mm. Serizawa et al. (1997) noted that these hypothalamic branches cross the midline in 10% of cases [13]. The third and last group of perforating arteries is the chiasmatic branches, present in 20% with an average diameter of 0.1 mm. In their series, Marinkovic et al. noted the presence of the subcallosal artery in more than 90% and the MACC in 3–20% [12]. Tulleken [4] (1978) interestingly showed that in case of duplication of the AComA, only one of the two AComAs bears the perforating branches.

The parenchymal territory of the perforating branches of the AComA is the preoptic area of the hypothalamus, the optic chiasm, the infundibulum, septal and paraolfactory nuclei, the anterior part of the septum pellucidum, the rostrum of the corpus callosum, the column of the fornix, and the lamina terminalis [3–5, 13, 14, 17].

Duplications and Fenestrations of the AComA

The AComA is formed of only one artery in not more than 40–60% of cases [2, 3, 7, 8, 12, 17]. Not less than 20 anatomical variations of the AComA were already distinguished at the beginning of the twentieth century [5]. A duplication or fenestration is discovered by almost all authors in more than 40% [1, 3–5, 7, 13–15, 17]. The AComA could be divided into 2 distinct channels (20–30%), in 3 different channels (10–18%), or could be identified as a complex network as in the embryo (4–6%) [2, 3, 5, 7, 12, 17, 21]. Different forms of the fenestration were described and Marinkovic et al. make also the distinction in case of multichannel AComA between one dominant artery and others smaller, and different arteries of equal diameter [12]. A case of AComA aneurysm located on a duplicated AComA is shown in Fig. 1.

Fig. 1 Clinical case of aneurysm located on a fenestrated AComA. The figure shows the case of a 39-year-old woman, with incidental discovery of an aneurysm located AComA complex. The figure on the right shows an oblique DSA view after left internal carotid artery injection (left ICA). The bifurcation of the ICA into the left A1 and M1 segment is visible. The round circle highlights a fenestrated anterior communicating artery, from which the A2 segments origin. The figure on the right is a 3D reconstruction of the DSA that shows an aneurysm (asterisk) located on the fenestrated AComA (black star). The contralateral A1 (right A1) is also visible origin from the AcomA

Clinical Implications

Korsakoff's Syndrome or Syndrome of the AComA

Surgical occlusion of the AComA and its perforators may cause a Korsakoff-like syndrome: acute confusion, severe anterograde amnesia, and cognitive-behavioral changes [22]. The first observation of amnesia after surgical treatment of an AComA aneurysm was described by Norlèn in 1953 [23]. However, the largest series of AComA aneurysms treatment resulting in post-operative amnesia was reported by Gade in 1982 [24]. He described a series of 48 patients with AComA aneurysms: 11 were treated with trapping, resulting in 9 cases of anterograde amnesia; 37 were treated with aneurysm clipping without trapping, resulting in only 6 post-operative amnesia [24]. Following these observations, he hypothesized an association between damage to the perforating branches of the AComA and postoperative amnesia, and he recommended avoiding the use of trapping for AComA aneurysms.

The subcallosal artery is the largest perforating branch of the AComA providing blood supply to the fornix, and its occlusion may lead to Korsakoff's-like syndrome [25].

References

1. Critchley M. Syndromes of the anterior cerebral artery. Proc R Soc Med. 1930;23(5):630–2.
2. Perlmutter D, Rhoton AL Jr. Microsurgical anatomy of the anterior cerebral-anterior communicating-recurrent artery complex. J Neurosurg. 1976;45(3):259–72. https://doi.org/10.3171/jns.1976.45.3.0259.
3. Dunker RO, Harris AB. Surgical anatomy of the proximal anterior cerebral artery. J Neurosurg. 1976;44(3):359–67. https://doi.org/10.3171/jns.1976.44.3.0359.
4. Tulleken CA. A study of the anatomy of the anterior communicating artery with the aid of the operating microscope. Clin Neurol Neurosurg. 1978;80(3):169–73. https://doi.org/10.1016/s0303-8467(78)80038-9.
5. Yasargil MG. Microneurosurgery. New York: Thieme Medical Publisher; 1984.
6. Lazorthes G, Poulhes J, Gaubert J. Vascular areas of the hypothalamus. Bull Acad Natl Med. 1956;140(32–33):607–12.
7. Rhoton AL Jr, Perlmutter D. Microsurgical anatomy of anterior communicating artery aneurysms. Neurol Res. 1980;2(3–4):217–51. https://doi.org/10.1080/01616412.1980.11739581.
8. Rosner SS, Rhoton AL Jr, Ono M, Barry M. Microsurgical anatomy of the anterior perforating arteries. J Neurosurg. 1984;61(3):468–85. https://doi.org/10.3171/jns.1984.61.3.0468.
9. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–62.
10. Moffat DB. The embryology of the arteries of the brain. Ann R Coll Surg Engl. 1962;30:368–82.
11. Abbie AA. The morphology of the fore-brain arteries, with especial reference to the evolution of the basal ganglia. J Anat. 1934;68(Pt 4):433–70.
12. Marinkovic S, Milisavljevic M, Marinkovic Z. Branches of the anterior communicating artery. Microsurgical anatomy. Acta Neurochir (Wien). 1990;106(1–2):78–85. https://doi.org/10.1007/BF01809337.
13. Serizawa T, Saeki N, Yamaura A. Microsurgical anatomy and clinical significance of the anterior communicating artery and its perforating branches. Neurosurgery. 1997;40(6):1211–6.; discussion 6–8. https://doi.org/10.1097/00006123-199706000-00019.
14. Tao X, Yu XJ, Bhattarai B, Li TH, Jin H, Wei GW, et al. Microsurgical anatomy of the anterior communicating artery complex in adult Chinese heads. Surg Neurol. 2006;65(2):155–61; discussion 61. https://doi.org/10.1016/j.surneu.2005.05.027.
15. Baptista AG. Studies of the arteries of the brain. II. The anterior cerebral artery: some anatomic features and their clinical implications. Neurology. 1963;13:825–35.
16. Aydin IH, Takci E, Kadioglu HH, Tuzun Y, Kayaoglu CR, Barlas E. Vascular variations associated with anterior communicating artery aneurysms-an intraoperative study. Minim Invasive Neurosurg. 1997;40(1):17–21. https://doi.org/10.1055/s-2008-1053407.
17. Ego H, N'Da H, Viart L, Foulon P, Le Gars D, Havet E, et al. Microsurgical anatomy of perforating branches of anterior communicating artery. Morphologie. 2015;99(324):6–13. https://doi.org/10.1016/j.morpho.2014.11.001.
18. Gomes FB, Dujovny M, Umansky F, Berman SK, Diaz FG, Ausman JI, et al. Microanatomy of the anterior cerebral artery. Surg Neurol. 1986;26(2):129–41. https://doi.org/10.1016/0090-3019(86)90365-4.
19. Uchino A, Nomiyama K, Takase Y, Kudo S. Anterior cerebral artery variations detected by MR angiography. Neuroradiology. 2006;48(9):647–52. https://doi.org/10.1007/s00234-006-0110-3.
20. Tahir RA, Haider S, Kole M, Griffith B, Marin H. Anterior cerebral artery: variant anatomy and pathology. J Vasc Interv Neurol. 2019;10(3):16–22.
21. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer edition; 2001.
22. Mosimann PJ, Saint-Maurice JP, Lenck S, Puccinelli F, Houdart E. Fornix infarction and Korsakoff dementia after coiling of a large anterior communicating artery aneurysm. Neurol Clin Pract. 2012;2(3):260–2. https://doi.org/10.1212/CPJ.0b013e31826af1cf.
23. Norlen G, Barnum AS. Surgical treatment of aneurysms of the anterior communicating artery. J Neurosurg. 1953;10(6):634–50. https://doi.org/10.3171/jns.1953.10.6.0634.
24. Gade A. Amnesia after operations on aneurysms of the anterior communicating artery. Surg Neurol. 1982;18(1):46–9. https://doi.org/10.1016/0090-3019(82)90013-1.
25. Meila D, Saliou G, Krings T. Subcallosal artery stroke: infarction of the fornix and the genu of the corpus callosum. The importance of the anterior communicating artery complex. Case series and review of the literature. Neuroradiology. 2015;57(1):41–7. https://doi.org/10.1007/s00234-014-1438-8.

Embryology and Variations of the Recurrent Artery of Heubner

Sara Bonasia, Michel W. Bojanowski, and Thomas Robert

During the last 150 years, anatomists, neurologists, and neurosurgeons (and one pediatrician) have paid particular attention to the recurrent artery of Heubner (RAH). Few fascinating particularities of this artery completely justify this interest. The first one is its singular embryological and phylogenic developments; the second one is the discrepancy between its diameter and its functional importance; the third one is the number of anatomical variants of this artery. RAH is also named in the literature as distal medial striate artery, long centralis artery, telencephalic artery, and also rostral striate artery, but the term recurrent artery of Heubner is the most used [1–4]. However, the various nomenclature found in the literature suggests the role of the RAH in the basal ganglia vascular supply. In this chapter, we focus on the description of embryology and possible anatomical variations of the RAH, and on the clinical implications derived from its occlusion during surgical approach to the anterior cranial fossa.

History

Johann Otto Leonhardt Heubner (1843–1926), a German pediatrician, was passionate about cerebral anatomy and spent a lot of his time to study the "*relationship of vascular disease with specific regions of the brain"* dissecting 30 human brain cadavers after having injected a solution of Bruke's Berlin blue [5]. In 1872, Otto Heubner wrote this sentence about the artery bearing now its name: "*From the base of the arteria cerebri anterior, which lies between the arteria cerebri media and the arteria communicans anterior, there constantly arises a very little artery, close to the latter that provides blood to the head of the corpus striatum*" [6]. After this remarkable anatomical description, Shellshear, in 1920, gave a more precise vision of the functional anatomy of the RAH illustrated by numerous drawings. In the same period, Abbie (1934) and Padget (1948) provided respectively phylogenic and embryologic knowledge in the development of cerebral vasculature [7–9]. Lazorthes (1956), in France, was one of the pioneer anatomists to describe the cerebral vasculature and in particular the arteries of the basal ganglia [10]. After, we could note different periods of interest in the literature of the RAH which could be attributed to the advent of the operative microscope, to the improvement of the digital subtraction angiography (DSA), and to the development of new cadaveric preparations. Most pertinent studies about the RAH were published by Ostrowski, Kaplan, Ahmed (1967), Rhoton Jr. (1976, 1984), Dunker (1976), Gomes (1984), and more recently by Vasovic (2009), Zunon-Kipré (2012), and Matsuda (2018) [1, 4, 11–18]. Lasjaunias was the only author to base the occurring cerebrovascular variations on DSA or cadaveric dissections on embryological and phylogenic knowledge [2].

Embryological Development

Our knowledge about the embryological development of the cerebral vasculature is principally based on the embryos dissection of the Carnegie Institution and on the writings of Streeter (1918) and Padget (1948) [8, 19]. Indeed, they described different stages in the vasculature development according to major vascular changes after having analyzed 22 sectioned embryos.

At the early stage I (4–5 mm embryo), the cranial division (also named primitive olfactory artery, future anterior cerebral artery, ACA) of the internal carotid artery (ICA) can

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already be individualized turning around the optic vesicle and ending in the olfactory area. During stage VI (20 mm), when the anterior cerebral artery extends up between the two hemispheres, the primitive olfactory artery sends an offshoot to the anterior perforated substance, which could be interpreted as the initial step of RAH development. From an embryological point of view, the middle cerebral artery (MCA) could be assimilated as a branch of the cranial division of the primitive carotid artery. This primitive middle cerebral artery could be seen at stage III (7–12 mm) and extends laterally into the primitive sylvian fissure caudal to the optic vesicle.

Few years before, Abbie (1934) realized a work of comparative anatomy dissecting brain specimens of fishes, amphibians, reptiles, sub-primates, and primates [7]. He already noted that the MCA, even if it has a large cerebral territory in adult human, appears later in the phylogenic evolution than the ACA. Regarding specifically the RAH, he wrote that it is the survivor of a series of anastomotic channels over and around the paleo-olfactorium. Main steps of RAH embryological development are resumed and illustrated in Table 1. Variations in origin or course of the RAH depend on which of this anastomotic channel is used as stem of origin. The parenchymal territory taken in charge by the RAH can also variate according to the extension of the middle cerebral artery vascular contribute, since the two systems are in balance and each one tries to compensate the other.

All variations of the RAH, and in particular the accessory MCA (AccMCA) variant, are the result of the phylogenic origins of the MCA from a group of vessels with similar potentials, including the RAH. These two arteries (MCA and RAH) with the same embryologic origin may supply the same parenchymal territory, and a balance between the two arteries during the embryological development well explains variations in the brain territory supplied by the RAH [2]. The main steps of RAH's embryological development are summarized and illustrated in Table 1.

Table 1 Embryological steps of the Recurrent artery oh Heubner

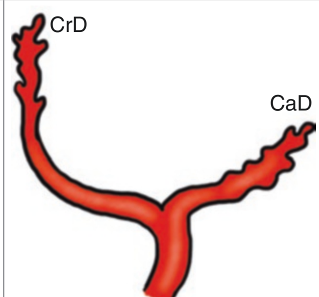
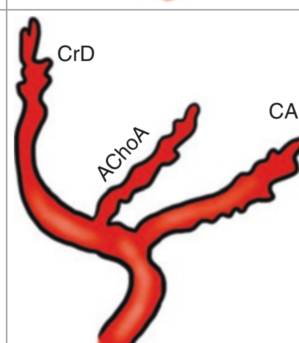
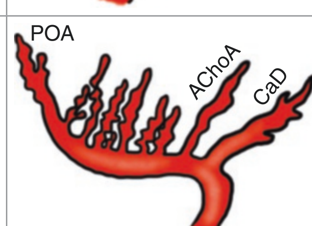
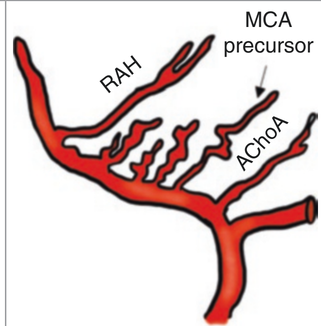
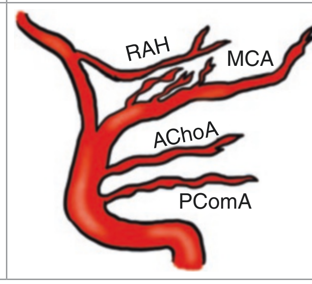
Embryo size	Critical events	Graphic illustration
4–5 mm	The caudal and cranial divisions (CaD, CrD) of the primitive ICA are visible	
9 mm	The anterior choroidal artery (AChoA) arises from the cranial division of the ICA	
10–14 mm	The primitive olfactory artery (POA), future anterior cerebral artery, gives birth to multiple plexiform vessels	

Table 1 (continued)

Embryo size	Critical events	Graphic illustration
15–18 mm	The RAH origins from the plexiform channels, while the precursor of the MCA begins to be visible more caudally	
20–30 mm	The POA transforms definitively into the ACA, from which the RAH origins. The MCA, the AChoA, and the PComA are visible in the adult configuration	

Number and Origin of the Artery

Anatomic analyses of the RAH and its variants are based on three different types of studies. The most precise and complete description is brought by cadaveric dissections. The other two types of studies are operative observations and angiographic descriptions. Even if each author tried to describe the anatomy of the artery as thoroughly as possible, a lot of discrepancies could be found in the literature. These discrepancies are easily explained by the technical differences of cadaver preparations and by the methods used to observe the artery and its branches (microscope, section cut, etc.). In the literature, we found 16 large cadaveric series, summarized in Table 2, that described the number of RAH by side with a total of more than 2300 brain hemispheres analyzed [1, 11, 12, 14, 15, 18, 20–30]. The absence of the RAH is rare and found in only 2% of cases. Frequently, only one RAH is present (79%). Two RAHs have been found in 14% of cases and three in only 4%. The incidence of variations in the number of RAH is summarized in Table 3.

The origin of the RAH is also variable but in 90% of cases the arteries arise from the ACA within 2 mm of distance from the anterior communicating artery [26]. Many cadaveric and operative studies presented a detailed description of the possible variations in RAH origin (Table 2) [1, 11–15, 18, 21–25, 27–32]. The most frequent point of origin of the RAH was the A1-A2 junction representing 44% of cases. The RAH was the first branch of the A2 segment in 43% of other cases, few millimeters distal to the A1–A2 junction. In 14% of cases, the RAH arose from the A1 segment. Perlmutter et al. (1976) and few other authors highlighted that the RAH always origin from the posterior wall of the A1 (when its origin is on the A1 segment) and on the lateral wall of the ACA if it origins from the A1-A2 junction or distally (Table 4) [1, 2, 13–15, 18, 33]. Figure 1 shows the different possible origins of the RAH as described before.

Few authors also highlighted an interesting anatomical variation that is the common trunk of the RAH with the orbito-frontal artery (which is generally the second branch of the A2 segment) [2, 10, 11, 14, 16, 18]. In this case, its origin is on the A2 segment and presents a more posterior orientation along the gyrus rectus to reach the anterior perforated substance [34]. This anatomical variation, illustrated in Fig. 1b on the left side, could be explained by the partial (distal part) persistence of the primitive olfactory artery [2].

Table 2 Summary of large series results about the RAH with the method used and the data analyzed

Author (year)	Number of hemispheres	Method	RAH origin (%)	Number of RAH (%)	RAH course (%)	Diameter (mm)	Length (mm)
Lazorthes et al. (1956)	70	Latex injection, corrosion	ND	ND	ND	ND	20
Perlmutter et al. (1976)	100	India ink	A1 segment: 14 A1-A2 junction: 8 A2: 78	Absent: 1 Single: 98 Double: 1	Type I (superior): 40 Type II (anterior): 60 Type III (posterior): 0	1	ND
Dunker et al. (1976)	40	Formalin-fixed	A1 segment: 11 A1-A2 junction: 54 A2: 35	ND	ND	ND	ND
Lemos et al. (1977)	166	Formalin-fixed	ND	Absent: 0.6 Single: 97 Double: 0.6 Triple: 1.8	ND	ND	21
Gomes et al. (1984)	60	Polyester resin injection	A1 segment: 8 A1-A2 junction: 35 A2: 57	Absent: 3 Single: 89 Double: 8	Type I (superior): 63 Type II (anterior): 34 Type III (posterior): 3	0.8	23
Rosner et al. (1984)	50	Latex injection	A1 segment: 33 A1-A2 junction: 66 A2: 0	Absent: 0 Single: 28 Double: 48 Triple: 24	ND	0.74	ND
Marinkovic et al. (1986)	66	India ink	A1 segment: 24 A1-A2 junction: 29 A2: 47	Absent: 0 Single: 73 Double: 24 Triple: 3	ND	0.66	21
Gorczyca et al. (1987)	100	India ink	A1 segment: 22 A1-A2 junction: 0 A2: 78	Absent: 0 Single: 28 Double: 48 Triple: 23	Type I (superior): 60 Type II (anterior): 36 Type III (posterior): 3	ND	ND
Avci et al. (2003)	62	Formalin-fixed	A1 segment: 8 A1-A2 junction: 28 A2: 64	Absent: 1.6 Single: 77 Double: 22 Triple: 1.6	ND	0.45	17
Ciolkowski et al. (2004)	40	Formalin-fixed	ND	ND	ND	ND	ND
Loukas et al. (2006)	88	Formalin-fixed	A1 segment: 14 A1-A2 junction: 62 A2: 24	Absent: 4.5 Single: 78 Double: 17.5	ND	0.8	23
Tao et al. (2006)	90	Formalin-fixed	A1 segment: 7 A1-A2 junction: 47 A2: 46	Absent: 1 Single: 61 Double: 32 Triple: 6	ND	0.64	30
Boongird et al. (2009)	100	Formalin-fixed	A1 segment: 8 A1-A2 junction: 32 A2: 60	Absent: 3 Single: 94 Double: 2 Triple: 1	Type I (superior): 30 Type II (anterior): 63 Type III (posterior): 7	ND	ND
Uzun et al. (2009)	108	Formalin-fixed	A1 segment: 7 A1-A2 junction: 79 A2: 14	ND	ND	0.67	ND

Table 2 (continued)

Author (year)	Number of hemispheres	Method	RAH origin (%)	Number of RAH (%)	RAH course (%)	Diameter (mm)	Length (mm)
Vasovic et al. (2009)	188	Latex injection	A1 segment: 6 A1-A2 junction: 42 A2: 52	Absent: 3 Single: 70 Double: 24 Triple: 3	ND	ND	ND
Gasca et al. (2011)	60	Formalin-fixed	A1 segment: 5 A1-A2 junction: 44 A2: 51	Absent: 7 Single: 55 Double: 38	ND	ND	24
Zunon et al. (2012)	40	Formalin-fixed /fresh cadavers	A1 segment: 30 A1-A2 junction: 12 A2: 68	Absent: 0 Single: 95 Double: 5	Type I (superior): 33 Type II (anterior): 61 Type III (posterior): 6	0.74	24
El Falougy et al. (2013)	366	Formalin-fixed	A1 segment: 6 A1-A2 junction: 44 A2: 50	Absent: 5 Single: 89 Double: 6	ND	0.55	ND
Maga et al. (2013)	140	Formalin-fixed	A1 segment: 26 A1-A2 junction: 40 A2: 34	Absent: 1 Single: 30 Double: 43 Triple: 26	Type I (superior): 61 Type II (anterior): 32 Type III (posterior): 4	ND	25
Matsuda et al. (2018)	714	Formalin-fixed	A1 segment: 7 A1-A2 junction: 76 A2: 17	Absent: 1 Single: 96 Double: 3	Type I (superior): 30 Type II (anterior): 62 Type III (posterior): 8	0.69	ND

Table 3 Incidence in the number of RAH by hemisphere

Number of RAH	Incidence
Absent	2%
One	79%
Two	14%
Three	4%

Table 4 Incidence in the origin of the RAH

Origin of the RAH	Incidence
A1–A2 junction	44%
A2	43%
A1	14%
Common trunk with OFA	Few cases described
Common trunk with OFA and FPA	Few cases described

FPA fronto-polar artery, *OFA* orbito-frontal artery

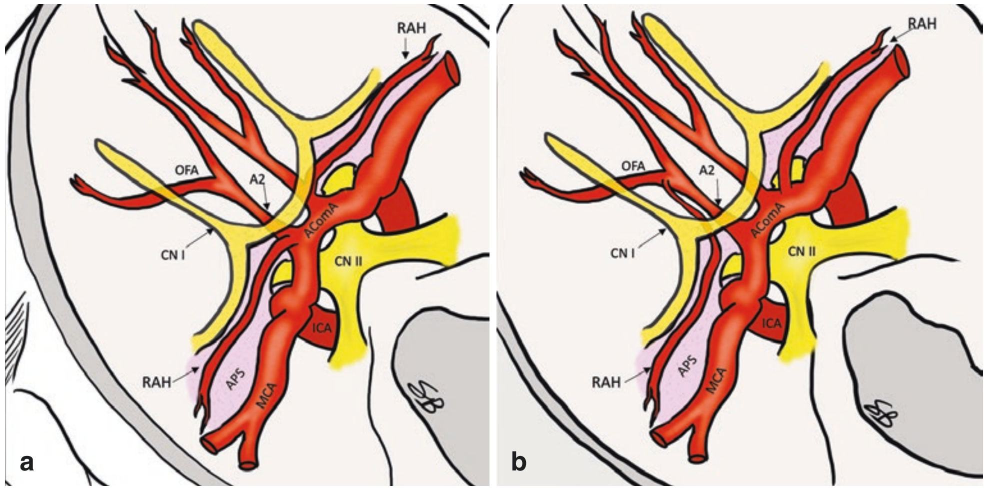


Fig. 1 Artist's drawing highlighting the possible origin of the recurrent artery of Heubner (RAH). (**a**) On the left side, the RAH arises at the A1-A2 junction and on the right side, the RAH origins from the A2 segment. (**b**) The left RAH shares a common trunk with the orbito-frontal artery (OFA) and the right RAH arises from the A1 segment. *I cn* first cranial nerve, *II cn* second cranial nerve, *APS* anterior perforated substance, *MCA* middle cerebral artery, *ICA* internal carotid artery, *AComA* anterior communicating artery

Course of the Artery

The diameter of the RAH is also very variable. It depends essentially on the number of RAH that the hemisphere bears and the importance of brain parenchymal supplied by the artery. After the analysis of all the cadaveric studies described in the literature (2370 hemispheres), the median diameter of the RAH results to be 0.68 mm and could vary from 0.45 mm to 1 mm [1, 12, 14, 15, 18, 21–25, 28, 31, 32]. Moreover, the diameter of the artery and its length are extremely variable and depend on the point of origin of the RAH, its tortuosity, and the entry point of its last perforating branches into the anterior perforated substance (APS). Most of the authors described the RAH as a very tortuous artery with a length generally more than two times the distance between its origin and its more lateral entry point in the APS [2, 10, 14–16, 18, 35, 36]. The median length of the artery is 22.9 mm but can vary from 17 mm to 30 mm [1, 10, 12, 20, 21, 23–25, 27, 28].

Gomes et al. (1984) described three different types of RAH courses as illustrated in Fig. 2: type I when the RAH courses superiorly to the A1 segment (Fig. 2b, left side), type II if the RAH courses anterior to the A1 segment (Fig. 2a), and type III when the RAH has a course posterior to the A1 segment (Fig. 2b, right side) [1]. Before the publication of Gomes et al. (1984), Perlmutter et al. (1976) studied the course of the artery on 100 brain hemispheres dissection but did not find any posterior course (type III) of the RAH [14]. Five other studies described in detail the course of the RAH [12, 18, 22, 26, 28]. Type I of RAH represents 38%; type II, 53% and type III, only 9%. Iczi et al. (2009) noted that the RAH passes across the posterior part of the gyrus rectus in 70% and posterior to the gyrus rectus in 30% [34]. Table 5 summarized the incidence of each type of RAH course.

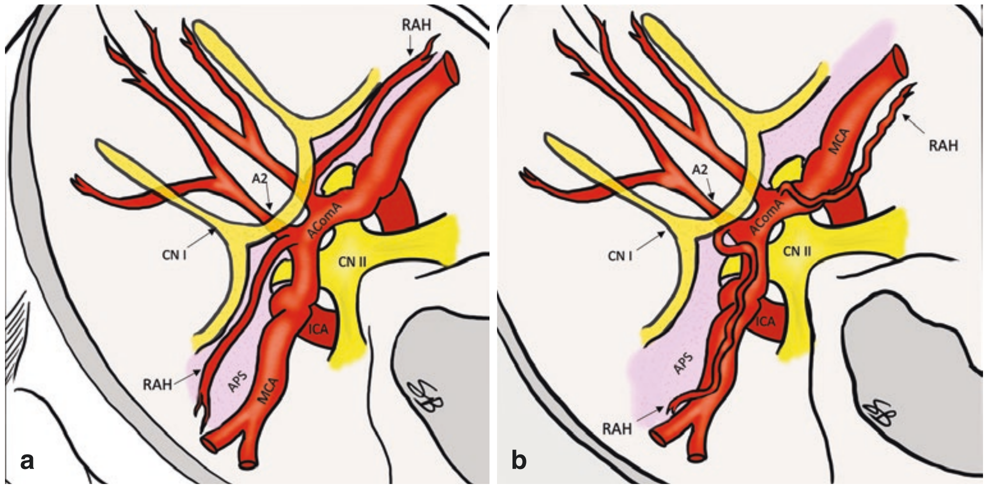


Fig. 2 Artist's drawing showing the different course of the recurrent artery of Heubner (RAH). (**a**) The two RAH arising from the A1-A2 junction (left side) and from the A2 segment (right side) have an anterior course relative to the A1 segment (type II). (**b**) On the left side, the RAH has a superior course (type I) and on the right side, a posterior course (type III). *MCA* middle cerebral artery, *ICA* internal carotid artery, *AComA* anterior communicating artery

Table 5 Different course of the RAH with their incidence

Course of the RAH	Incidence
Type I	38%
Type II	53%
Type III	9%

Branches of the Artery

In its extra-cerebral course, the RAH could give from 2 to 30 branches [14, 15, 29]. The majority of these branches are perforator vessels passing through the APS and are present in all cases. The number of perforating branches passing through the APS varies from 2 to 44 with a clear predilection to the anterior part of the APS (95%) with a full mediolateral extent on the APS. Other branches could perforate the APS in its middle portion (34%) or rarely in its posterior part (7%) [13–15]. Rosner et al. (1984) provided the most detailed analysis of the APS branches and noted that two-thirds of RAH branches enter the APS in its lateral part (lateral to the olfactory tract) and one-third in its medial part [15].

In approximately 30–40% of cases, the terminal branches of the RAH turn around the limen insulae to enter into the sylvian fissure to give branches to the vertical part of the insula [14]. In few cases, these branches could also supply the inferior frontal gyrus and could mimic the accessory middle cerebral artery variant (AccMCA) that will be discussed below.

In few cases, the RAH also gives fronto-orbital branches that supply the posterior part of the gyrus rectus, the posterior third of the olfactory tract, the olfactory trigone, and the posterior fronto-orbital gyrus. The presence of these "olfactory branches" has an incidence of 13% but is very variable from one study to another one [10, 17, 23, 37]. Ciolkowski et al. (2004) who analyzed the vascularization of the posterior part of the olfactory tract revealed a participation of RAH branches with an incidence of 50% [37].

The RAH inconstantly also gives branches that course lateral to the optic chiasm, to supply the anterior part of the hypothalamus [17, 22, 29]. Ostrowski (1960) had already noted this vascularization possibility by selectively injecting the RAH. Their incidence seems to be approximately 30% [17, 29].

Many authors highlighted the absence of branches to the optic nerve and optic tract but few recent studies with detailed and microscopic observations of little branches described very little branches (0.1 mm of diameter) in approximately one-third of cases supplying the optic tract [28–30].

Possible Anastomoses

Some authors have noted the presence of extra-cerebral anastomoses between the perforating branches of the RAH and of the MCA [12, 22, 29]. Their incidence is about 10% and the lenticulo-striate branches of the medial group (that branch off the MCA) are the perforators most frequently involved. After contemporary injection of RAH and lenticulostriate branches of the MCA, Maga et al. (2013) postulated that intracerebral anastomoses between branches of the RAH and MCA are also present in 6.5% of cases [30].

The olfactory branch of the RAH could also anastomose with cortical fronto-basal branch of the MCA. Gorczyca et al. (1987) found this type of anastomosis in 5% of cases and described them as pio-pial anastomosis [22].

The presence of these anastomoses, in particular between the perforators of the RAH and MCA, is a favorable element of Abbie's concept, which postulated that the RAH is the remnant of a series of anastomotic channels over and around the paleo-olfactorium [7].

The Accessory Middle Cerebral Artery

The term "accessory middle cerebral artery" (AccMCA) was first used in 1962 by Crompton when he examined 347 brain specimens and reported an anomalous large branch passing into the sylvian fissure in 11 cases (3.6%) [38]. In 10 of these, the artery took its origin from the internal carotid artery and in one case from the anterior communicating artery region. Jain (1964) noted this anomalous branch with a similar incidence (3.4%, 10/300 brain hemispheres) but, in contrast to Crompton's series, eight AccMCA arose from the anterior cerebral artery and only 2 from the ICA bifurcation [38]. Few years later, the term "accessory MCA" was reserved for the vessel arising from the anterior cerebral artery. In case of origin from the ICA bifurcation, the vessel was called duplicated MCA [2, 33, 36]. In 1977, Manelfe established a classification of these variants including three different types [2]. Types I and II are duplicated MCA where the duplicated artery arises at the bifurcation or at the proximal part of the A1 segment. Type III involves the RAH with an AccMCA arising from the A1-A2 junction. The different types of duplicated or accessory MCA are illustrated in Fig. 3.

The AccMCA could be defined as a vessel arising from the anterior cerebral artery, in the region of the A1-A2 junction that courses into the sylvian fissure superior to the ICA bifurcation and superior to the M1 segment and that participates in the vascularization of the frontal cortex.

After these first descriptions, few isolated cases of AccMCA were described and the incidence of this variant was estimated as 0.3%, principally based on angiographic studies [39–47]. It is generally accepted that the AccMCA has perforating branches to the APS [3, 42]. The cortical territory of an AccMCA could also be variable but the lateral part of the orbito-frontal gyri and the prefrontal region are the most frequently involved [21, 46]. Lasjaunias and Berenstein (2001) sometimes noted also its implication in the supply of the anterior temporal area [2].

Embryologically, the RAH and the MCA develop after the primitive olfactory artery (anterior division of the ICA or anterior cerebral artery) and could both be considered as branches of the ACA. Several hypotheses were formulated regarding the development of the AccMCA. Handa et al. (1970), supported by other authors, thought the AccMCA to be a hypertrophied RAH extending the Abbie's theory that the RAH is a remnant of anastomotic channels between the ACA and the MCA [3, 40, 43, 44]. Few years later, Teal et al. (1973) disagreed with Handa arguing that (1) the perforating arteries do not always originate from the AccMCA, (2) a RAH could co-exist with an AccMCA, and (3) the AccMCA passes lateral to the APS where the RAH enters into the APS [41]. The fact that the AccMCA has frequently perforating branches and that the RAH could be multiple puts the Teal's objections into question. Lasjaunias and Berenstein (2001) also explained by phylogenic analysis the AccMCA as a hypertrophic RAH with a cortical supply [2].

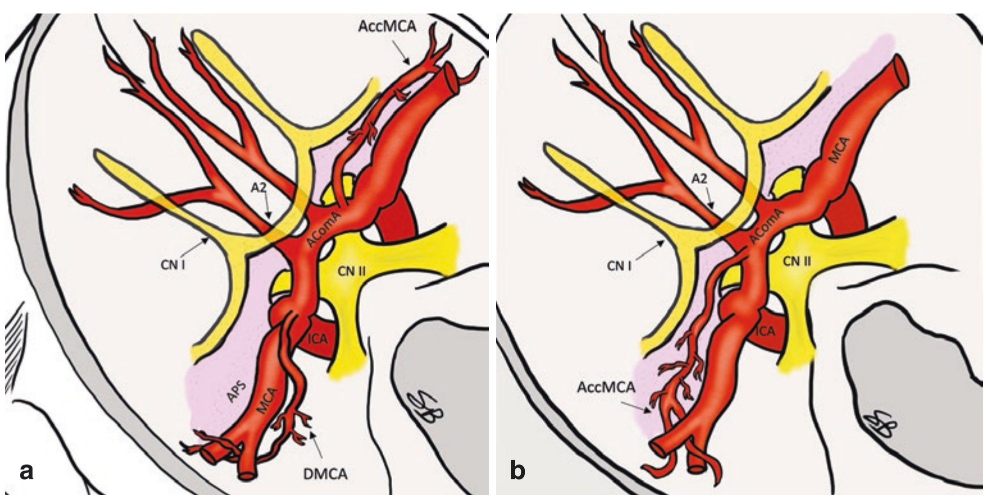


Fig. 3 Illustrations of the different variations of duplicated or accessory MCA. (**a**) On the left side, the artery origins from the ICA bifurcation and is commonly named duplicated middle cerebral artery (DMCA) or type I of Manelfe. On the right side is illustrated AccMCA arising from the proximal part of the A1 segment (type II of Manelfe). (**b**) On the left side, this AccMCA arises from the distal part of the A1 segment (type III of Manelfe). *MCA* middle cerebral artery, *ICA* internal carotid artery, *AComA* anterior communicating artery

Parenchymal Territory

The first study which gave a detailed description of the RAH parenchymal supply was published in 1960 by Ostrowski et al. [17]. The authors, ingeniously, thought to selectively inject the artery with India ink water-diluted solution permitting to color territories supplied by arteries as thin as 100–150 μm. They highlighted that the RAH constantly supplies the head of the caudate nucleus, the anterior inferior limb of the internal capsule, the hypothalamus, the uncinated fasciculus, and the olfactory regions [17]. Other studies showed that the external portion of the globus pallidum is constantly supplied by the RAH [13, 14, 48]. Recently, Maga et al. (2013) proposed another cadaveric study and noted the presence of optic tract branches in approximately 15% [29]. They also noted inconstant additional supply to the anterior thalamic nuclei (35%), to the genu of the internal capsule (26%), to the subcallosal area, to the lamina terminalis, and to the supra-optic nucleus [29]. In 2009, Mavridis et al. added the nucleus accumbens as a constant territory of the RAH [48].

Clinical Implications

The knowledge of the detailed anatomy of the RAH is mandatory during surgical approach to the anterior part of the circle of Willis since the injury of this artery can lead to several iatrogenic complications. Accidental occlusion or injury of this artery can cause infarction of the regions previously described, resulting in contralateral hemiparesis with upper extremity predominance and mild face paresis. If the dominant hemisphere is involved, also expressive aphasia with emotional symptoms can occur [1, 22]. Some cases of tongue and palate dysfunction have also been documented. Even if these symptoms could also be severe, they tend to resolve completely over weeks or months in the majority of patients. The persistence of them can be explained by the absence of balance or a hypoplasia of the ipsilateral lenticulo-striate artery [12].

The most common situation that can lead to RAH injuries is the surgical clipping of an anterior communicating artery aneurysm. An example of accidental RAH clipping is shown in the illustrative case in Fig. 4. During any approach to this region (pterional, subfrontal, or anterior hemispheric), the RAH should be identified and accurately dissected. Whatever the orientation of the AcomA aneurysm is (inferiorly, anteriorly, superiorly, posteriorly, etc.), the first step in the approach to aneurysms of this region consists in the identification and following of the A1 segment and visualizing the RAH [49].

The artery needs sometimes to be dissected from the aneurysmal dome and can be also involved in the wall of the aneurysm. In such cases also section and reimplantation of the artery into the A1 segment should be considered to preserve this vessel [1]. In case of temporary clipping of the A1 segment, particular attention should be paid to the possibility, even if present only in 10% of cases, of a posterior course of the RAH (Type III) in order to avoid its accidental clipping.

During surgery, the RAH may also be confused with the orbitofrontal artery, which is usually the second major branch of the A2 segment. These two arteries could be distinguished based on two factors: the point of origin and the course. As previously described the RAH, in case of origin from the A2 segment, arises within the proximal few millimeters from the A1-A2 junction and has a recurrent course along the A1 segment. On the other hand, the orbitofrontal artery arises more distal and courses perpendicularly over the gyrus rectus and across the olfactory tract [5]. Since the two arteries have a similar diameter, based on it alone to distinguish them, the orbitofrontal artery can be mistaken for the RAH.

In conclusion, the RAH must be always identified before a surgical clipping of anterior communicating artery aneurysms since its accidental occlusion can lead to severe neurological deficits. The knowledge of the possible variants in its origin, number, and course can help the surgeon to better understand the microsurgical anatomy.

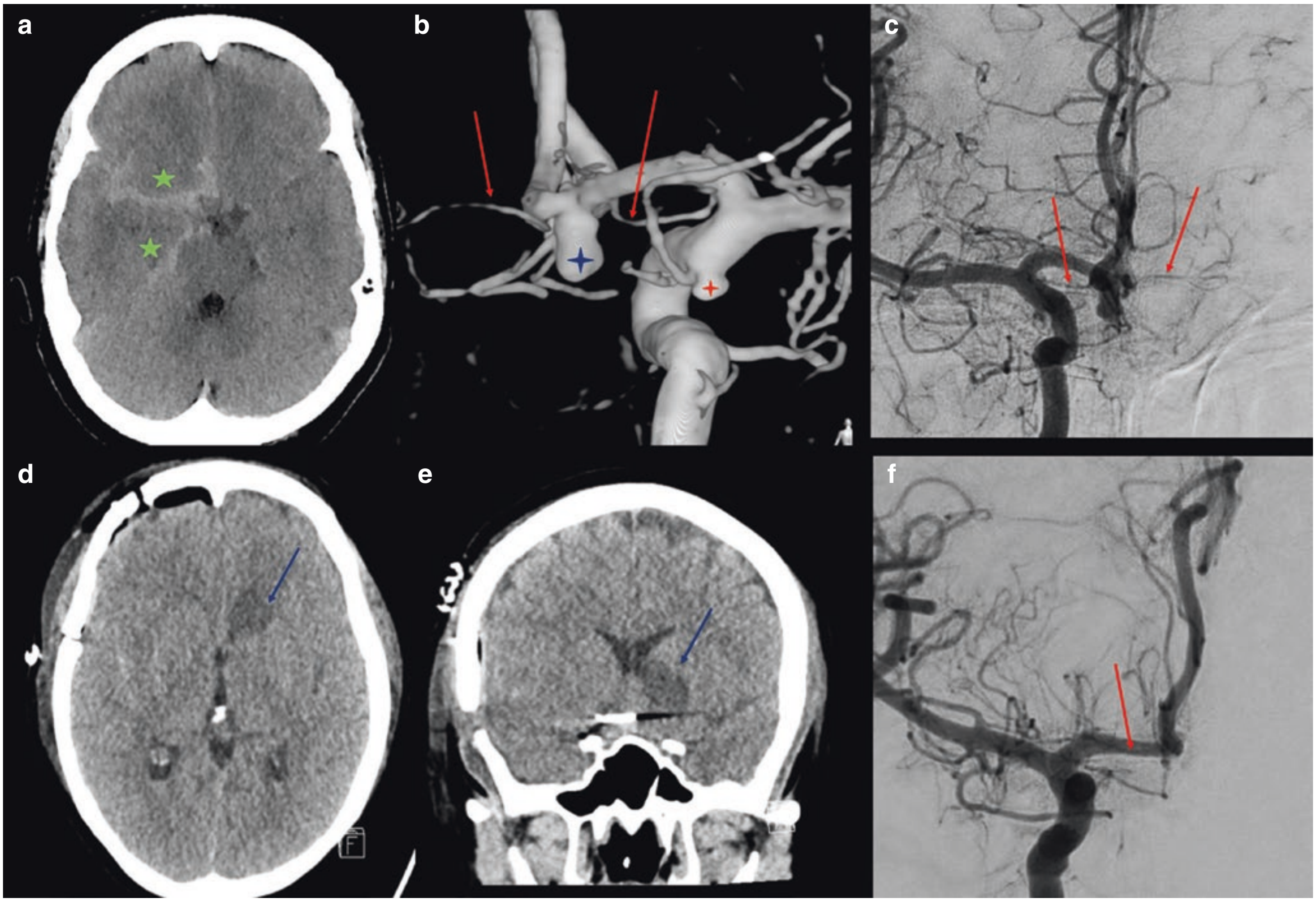


Fig. 4 Illustrative case. In March 2019, a 54-year-old woman, anamnestically known only for hypercholesterolemia under treatment, arrived at a minor hospital in Switzerland following the acute onset of explosive headache associated with nausea and vomiting. A cerebral CT scan was performed, which showed a subarachnoid hemorrhage grade Fisher III (**a**), mainly affecting the right sylvian and ambiens cisterns (green stars), associated with an aneurysm of the communicating anterior artery. The patient was then transferred to our center, where she was subjected to diagnostic cerebral angiography (**b** and **c**), which confirmed the presence of the aneurysm of the communicating anterior artery (blue cross), with high probability responsible for bleeding, and also showed an aneurysm of the anterior right posterior communicating artery (red cross). In the 3D reconstruction (**b**) and standard angiograms (**c**) the left and right RAH are visible (red arrows). We decided to surgically treat both the aneurysms using a standard right pterional approach. However, during the dissection of the aneurysm of AComA, we witnessed a massive bleeding of this latter, thus, we proceeded to clipping in urgency the sac and inducing of cardiac arrest by atropine. After the bleeding had stopped, and the patient was hemodynamically stabilized, the second aneurysm was clipped without complications. Upon awakening, the patient showed up with a GCS of 15 and without focal neurological deficits. However, although the control CT showed no post-surgical bleeding complications, it highlighted an ischemic hypodensity involving the head of the left caudate nucleus (**c**). The ischemic territories indicated by the blue arrows (**d** and **e**) correspond to the parenchyma normally vascularized by the left RAH and indicate the accidental occlusion of the left RAH during the emergency clipping. The postoperative cerebral angiography (**f**) showed a good exclusion of the two aneurysms, but the left RAH was no more visible, well explaining the hypodensity visible on the postoperative CT scan

References

1. Gomes F, Dujovny M, Umansky F, Ausman JI, Diaz FG, Ray WJ, et al. Microsurgical anatomy of the recurrent artery of Heubner. J Neurosurg. 1984;60(1):130–9. https://doi.org/10.3171/jns.1984.60.1.0130.
2. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer edition; 2001.
3. Tran-Dinh H. The accessory middle cerebral artery—A variant of the recurrent artery of Heubner (A. centralis longa)? Acta Anat (Basel). 1986;126(3):167–71.
4. Kaplan HA. The lateral perforating branches of the anterior and middle cerebral arteries. J Neurosurg. 1965;23(3):305–10. https://doi.org/10.3171/jns.1965.23.3.0305.
5. Haroun RI, Rigamonti D, Tamargo RJ. Recurrent artery of Heubner: Otto Heubner's description of the artery and his influence on pediatrics in Germany. J Neurosurg. 2000;93(6):1084–8. https://doi.org/10.3171/jns.2000.93.6.1084.
6. Heubner O. Zur Topographie der Ernahrungsgebrete der einzelnen Hirnarterien. Cent Med Wissen. 1872;52:816–21.
7. Abbie AA. The morphology of the fore-brain arteries, with especial reference to the evolution of the basal ganglia. J Anat. 1934;68(Pt 4):433–70.

8. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–62.
9. Shellshear JL. The basal arteries of the forebrain and their functional significance. J Anat. 1920;55(Pt 1):27–35.
10. Lazorthes G, Poulhes J, Gaubert J. Vascular areas of the hypothalamus. Bull Acad Natl Med. 1956;140(32–33):607–12.
11. Vasovic L, Ugrenovic S, Jovanovic I. Human fetal medial striate artery or artery of Heubner. J Neurosurg Pediatr. 2009;3(4):296–301. https://doi.org/10.3171/2008.12.PEDS08258.
12. Zunon-Kipre Y, Peltier J, Haidara A, Havet E, Kakou M, Le Gars D. Microsurgical anatomy of distal medial striate artery (recurrent artery of Heubner). Surg Radiol Anat. 2012;34(1):15–20. https://doi.org/10.1007/s00276-011-0888-5.
13. Dunker RO, Harris AB. Surgical anatomy of the proximal anterior cerebral artery. J Neurosurg. 1976;44(3):359–67. https://doi.org/10.3171/jns.1976.44.3.0359.
14. Perlmutter D, Rhoton AL Jr. Microsurgical anatomy of the anterior cerebral-anterior communicating-recurrent artery complex. J Neurosurg. 1976;45(3):259–72. https://doi.org/10.3171/jns.1976.45.3.0259.
15. Rosner SS, Rhoton AL Jr, Ono M, Barry M. Microsurgical anatomy of the anterior perforating arteries. J Neurosurg. 1984;61(3):468–85. https://doi.org/10.3171/jns.1984.61.3.0468.
16. Ahmed DS, Ahmed RH. The recurrent branch of the anterior cerebral artery. Anat Rec. 1967;157(4):699–700. https://doi.org/10.1002/ar.1091570414.
17. Ostrowski AZ, Webster JE, Gurdjian ES. The proximal anterior cerebral artery: an anatomic study. Arch Neurol. 1960;3:661–4.
18. Matsuda W, Sonomura T, Honma S, Ohno S, Goto T, Hirai S, et al. Anatomical variations of the recurrent artery of Heubner: number, origin, and course. Anat Sci Int. 2018;93(3):317–22. https://doi.org/10.1007/s12565-017-0415-9.
19. Streeter GL. The developmental alterations in the vascular system of the brain of the human embryo. Contrib Embryol. 1918;8(271):5–38.
20. Lemos VP. Recurrent branch of the anterior cerebral artery: a neuroanatomic study of the extracerebral segment (author's transl). Rev Bras Pesqui Med Biol. 1977;10(1):21–4.
21. Marinkovic S, Milisavljevic M, Kovacevic M. Anatomical bases for surgical approach to the initial segment of the anterior cerebral artery. Microanatomy of Heubner's artery and perforating branches of the anterior cerebral artery. Surg Radiol Anat. 1986;8(1):7–18.
22. Gorczyca W, Mohr G. Microvascular anatomy of Heubner's recurrent artery. Neurol Res. 1987;9(4):259–64.
23. Avci E, Fossett D, Aslan M, Attar A, Egemen N. Branches of the anterior cerebral artery near the anterior communicating artery complex: an anatomic study and surgical perspective. Neurol Med Chir (Tokyo). 2003;43(7):329–33; discussion 33
24. Loukas M, Louis RG Jr, Childs RS. Anatomical examination of the recurrent artery of Heubner. Clin Anat. 2006;19(1):25–31. https://doi.org/10.1002/ca.20229.
25. Tao X, Yu XJ, Bhattarai B, Li TH, Jin H, Wei GW, et al. Microsurgical anatomy of the anterior communicating artery complex in adult Chinese heads. Surg Neurol. 2006;65(2):155–61; discussion 61. https://doi.org/10.1016/j.surneu.2005.05.027.
26. Boongird A, Duangtongphon P. Variation of the recurrent artery of Heubner in human cadavers. J Med Assoc Thail. 2009;92(5):643–7.
27. Gasca-Gonzalez OO, Delgado-Reyes L, Perez-Cruz JC. Microsurgical anatomy of the extracerebral segment of recurrent artery of Heubner in the Mexican population. Cir Cir. 2011;79(3):201–6, 19–24
28. Maga P, Tomaszewski KA, Skrzat J, Tomaszewska IM, Iskra T, Pasternak A, et al. Microanatomical study of the recurrent artery of Heubner. Ann Anat. 2013;195(4):342–50. https://doi.org/10.1016/j.aanat.2013.03.011.
29. Maga P, Tomaszewski KA, Krzyzewski RM, Golec J, Depukat P, Gregorczyk-Maga I, et al. Branches and arterial supply of the recurrent artery of Heubner. Anat Sci Int. 2013;88(4):223–9. https://doi.org/10.1007/s12565-013-0192-z.
30. Maga P, Tomaszewski KA, Pasternak A, Zawilinski J, Tomaszewska R, Gregorczyk-Maga I, et al. Extra- and intracerebral course of the recurrent artery of Heubner. Folia Morphol (Warsz). 2013;72(2):94–9.
31. Uzun I, Gurdal E, Cakmak YO, Ozdogmus O, Cavdar S. A reminder of the anatomy of the recurrent artery of heubner. Cent Eur Neurosurg. 2009;70(1):36–8. https://doi.org/10.1055/s-0028-1119379.
32. El Falougy H, Selmeciova P, Kubikova E, Haviarova Z. The variable origin of the recurrent artery of Heubner: an anatomical and morphometric study. Biomed Res Int. 2013;2013:873434. https://doi.org/10.1155/2013/873434.
33. Huber P, editor. Cerebral angiography. 2nd ed. Stuttgart: Thieme; 1982.
34. Izci Y, Seckin H, Medow J, Turnquist C, Baskaya MK. Sulcal and gyral anatomy of the orbitofrontal cortex in relation to the recurrent artery of Heubner: an anatomical study. Surg Radiol Anat. 2009;31(6):439–45. https://doi.org/10.1007/s00276-009-0465-3.
35. Westberg G. The recurrent artery of Heubner and the arteries of the central ganglia. Acta Radiol Diagn (Stockh). 1963;1:949–54.
36. Yasargil MG, Fox JL, Ray MW. Anterior cerebral artery complex. In: Yasargil MG, editor. Microneurosurgery. New York: Thieme; 1975. p. 92–9.
37. Ciolkowski M, Michalik R, Ciszek B. Arteries to the proximal part of the olfactory tract. Folia Morphol (Warsz). 2004;63(4):455–8.
38. Jain KK. Some observations on the anatomy of the middle cerebral artery. Can J Surg. 1964;7:134–9.
39. Stabler J. Two cases of accessory middle cerebral artery, including one with an aneurysm at its origin. Br J Radiol. 1970;43(509):314–8. https://doi.org/10.1259/0007-1285-43-509-314.
40. Handa J, Shimizu Y, Matsuda M, Handa H. The accessory middle cerebral artery: report of further two cases. Clin Radiol. 1970;21(4):415–6.
41. Teal JS, Rumbaugh CL, Bergeron RT, Segall HD. Anomalies of the middle cerebral artery: accessory artery, duplication, and early bifurcation. Am J Roentgenol Radium Therapy, Nucl Med. 1973;118(3):567–75.
42. Watanabe T, Togo M. Accessory middle cerebral artery. Report of four cases. J Neurosurg. 1974;41(2):248–51. https://doi.org/10.3171/jns.1974.41.2.0248.
43. Takahashi S, Hoshino F, Uemura K, Takahashi A, Sakamoto K. Accessory middle cerebral artery: is it a variant form of the recurrent artery of Heubner? AJNR Am J Neuroradiol. 1989;10(3):563–8.
44. Mueller DP, Sato Y, Yuh WT. Accessory middle cerebral artery as a source of collateral blood flow. AJNR Am J Neuroradiol. 1991;12(6):1223–4.
45. Tacconi L, Johnston FG, Symon L. Accessory middle cerebral artery. Case report. J Neurosurg. 1995;83(5):916–8. https://doi.org/10.3171/jns.1995.83.5.0916.
46. Komiyama M, Nishikawa M, Yasui T. The accessory middle cerebral artery as a collateral blood supply. AJNR Am J Neuroradiol. 1997;18(3):587–90.
47. Komiyama M, Nakajima H, Nishikawa M, Yasui T. Middle cerebral artery variations: duplicated and accessory arteries. AJNR Am J Neuroradiol. 1998;19(1):45–9.
48. Mavridis I, Anagnostopoulou S. Comment on the brain areas whose blood supply is provided by the recurrent artery of Heubner. Surg Radiol Anat. 2010;32(1):91. https://doi.org/10.1007/s00276-009-0540-9.
49. Lawton MT. Seven aneurysms. 1st ed. New York: Thieme; 2010.

Embryology and Variations of the Posterior Choroidal Artery

A. Venier

Posterior choroidal arteries (PChoAs) represent the ventricular branches of the posterior cerebral artery (PCA) which are directed to the lateral and third ventricles to supply their choroid plexus and walls. Commonly referred to one single artery, as a matter of fact, PChoAs are multiple arteries and are divided into a medial and a lateral group according to their origins, numbers, courses, and territories of supply, accounting several variations and anomalies [1].

Interestingly, PChoA in its adult disposition is part of the posterior circulation but embryologically develops from the caudal division of the internal carotid artery (ICA) which will become the posterior communicating artery (PComA) that in turn will become the PCA, being the PChoA one of its branch [1].

Clinical consequences of PChoA occlusion aren't as devastating as those of anterior choroidal artery (AChoA) but still enough relevant to know and understand if the blood flow is compromised by a stroke or by some vascular or neoplastic lesions that may displace and stenose the artery along its course.

History

Posterior choroidal artery history is inextricably linked to choroid plexus embryology and anatomy understanding.

The first who dared to describe the posterior circulation of the choroid plexus was Duret (1874) who identified a posterolateral and a posteromedian choroidal artery coming from the PCA [2]. Then, Kolisko (1891) described some anastomoses between the AChoA and PCA branches; Beevor (1909) differentiated the areas of supply of AChoA and PCA branches, and Abbie (1933) found 3–5 choroidal branches of PCA [3–5].

A genuine leap forward in terms of comprehension of choroidal plexus' anatomy and vascularization was made only around the 1950s with Padget (1948) and its embryologic studies, Millen and Woolam (1953) and their AChoA and PChoA detailed portrait, Galloway and Greitz (1959) and their gross anatomical and radiological description, Hudson (1960) and its classification of lateral posterior choroidal arteries (LPChoAs) and finally Netsky and Shuangshoti (1970) [6–10]. Notably, Duret was the first who described a posterior, middle, and anterior system corresponding to the medial posterior choroidal artery (MPChoA) and the superior and inferior branches of LPChoA, described successively by Hudson and, based on these works, Wolfram-Gabel et al. (1987) described medial and lateral branches of MPChoA and superior and inferior branches of LPChoA [2, 9, 11].

Embryological Development

Most of our knowledge about arterial embryology comes from the meticulous studies of 22 embryos of the Carnegie Collection conducted first by Steeler and then by Padget [6, 12]. There are 8 Padget stages (from pre-Padget stage to stage 7, the adult configuration) that explain cranial arteries development and 23 Carnegie stages proposed by Streeter and revised by O'Rahilly that explain the chronological embryo development and are classified according to age (from 1 to 56 days of intrauterine life) and length (from 0.1 to 31 mm) [13–18].

Posterior choroidal artery appears at Padget stage 3 or Carnegie stages XV–XVI (7–12 mm, 33 days), when from the caudal division of PComA, just in front to the third nerve, arise the diencephalic and the mesencephalic arteries (DA, MA). From the first one emerges the PChoA. Posterior choroidal artery is directed toward the AChA with which anastomoses in the choroidal plexus at the interventricular foramen together with some anterior cerebral artery's (ACA) choroidal branches (Table 1).

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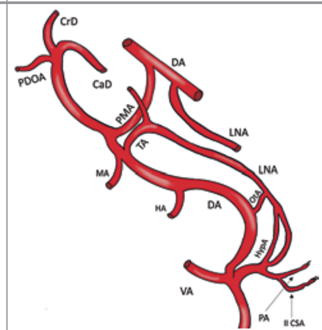
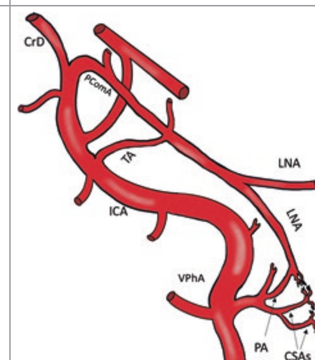
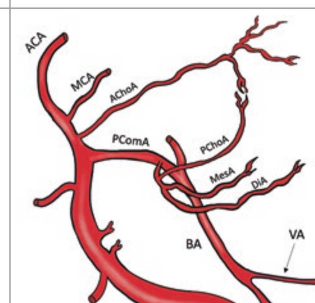
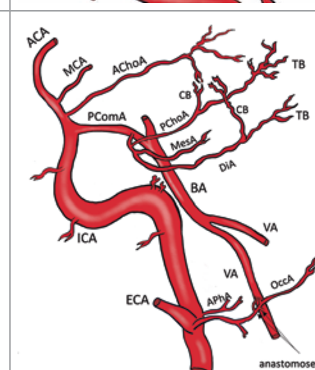
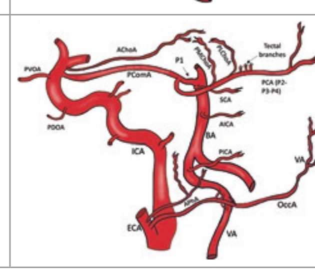
Table 1 Major embryological changes of the PChoA according to Padget. (Adapted from [6]: Padget DH. The development of the cranial arteries in the human embryo. Contrib Embryol. 1948;32:205–62)

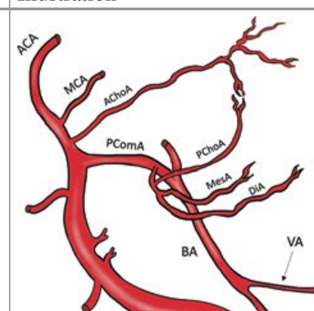
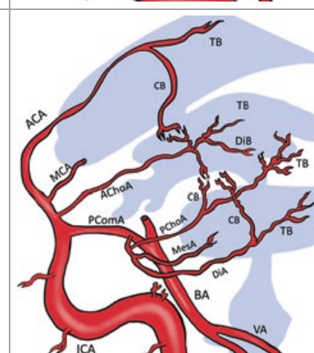
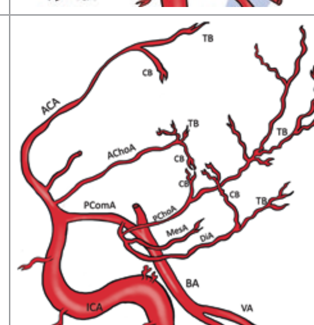
Stage	Embryo size (mm)	Major evolutions	Graphic Illustration
I	4–5	– Paired longitudinal neural arteries (LNAs) visible – Carotido-vertebral anastomoses visible: Trigeminal (TA), otic (OtA), hypoglossal (HypA), proatlantal (PA), and second cervical segmental artery (II CSA) – Primitive ICA bifurcation visible into caudal and cranial divisions (CaD, CrD) – PComA and BA not formed	
II	5–6	– Formation of the PComA – Regression of otic and hypoglossal arteries – Midline fusion of LNAs to form the basilar artery (BA)	
III	7–12	– Basilar artery (BA) completely formed – PComA completely formed – Origin from the distal PComA of a diencephalon-mesencephalic trunk (DiA-MesA). PChoA arising from the common trunk and anastomoses with the AChoA – Regression of trigeminal and proatlantal arteries – Formation of the vertebral artery VA by transverse anastomoses between the intersegmental arteries	
IV	13–15	– Development of a choroidal (CB) and telencephalic branch (TB) from the PChoA and the diencephalic artery (DiA) – Remodeling of the BA – Migration of OccA origin on the external carotid artery (ECA) – Persistence of the anastomoses between OccA and VA	
V	16–18	– The telencephalic branch of the PChoA forms the main PCA trunk – The choroidal branches of the PChoA and of the diencephalic artery form the PLChoA and PMChoA – Complete formation of the VA – Complete formation of the OccA (remnant of the proatlantal artery) – Complete formation of the ascending pharyngeal artery (APhA, remnant of the hypoglossal artery) – SCA, AICA, and PICA are visible	

At Padget stage 5 or Carnegie stages XVIII–XIX (16–18 mm, 40 days), AChoA and PChoA end in the choroidal fissure at the diencephalic roof. PChoA supplies both the diencephalon and the mesencephalon together with the diencephalic and the mesencephalic arteries, respectively [6]. At this stage, PCA develops from the caudal end of PComA, but progressively enlarges and the smaller arteries, such as PChoA, become its branches (Table 1).

At Padget stage 6 or Carnegie stages XX–XXI (20–24 mm, 45 days), AChoA and PChoA are intimately connected by several anastomoses, especially at the diencephalon and into the choroid plexus but, as the basal ganglia enlarge, the AChoA supply to the choroid plexus becomes smaller (Table 1).

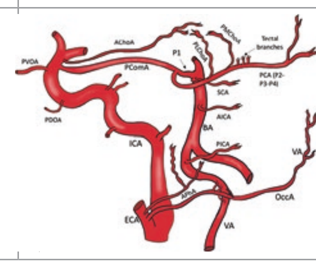
Another complementary source of embryological knowledge focused on choroidal vessels development, comes from the encompassing work of Lasjaunias et al., which is mainly based on Moffat study on rat embryos [19, 20]. Seven stages are encountered, from the prechoroidal one to adult disposition [19]. From the caudal division of ICA arise the DiA and MesA (stage I prechoroidal, Table 2). From this diencephalo-mesencephalic trunk a PChoA develops and anastomoses at the atrium level with choroidal branches of AChoA, coming from the cranial division of ICA, and ACA (stage II choroidal, Table 2). Progressively, the choroidal branches territories of AChoA and ACA are annexed by the choroidal branches of PChoA (stage III telencephalic primitive, Table 2) and subsequently, also the telencephalic branches territories of AChoA and ACA (stage IV telencephalic intermediate, Table 2) and of the diencephalic branch are annexed by the telencephalic branch of PChoA (stage V telencephalic final, Table 2). At this point, PChoA represents the main PCA stream. In the end, the caudal division of ICA becomes PComA, the choroidal branch of PChoA becomes the

Table 2 Embryology and development of the PChoA according to Lasjaunias. (Adapted from [1]: Lasjaunias P, Berenstein A, Ter Brugge KG. Surgical Neuro-angiography—Clinical Vascular Anatomy and Variations: Springer; 1987)

Stage		Illustration
I Prechoroidal	– Primitive ICA visible with its two divisions (CaR, CrR) – PComA completely formed – Origin from the distal PComA of a diencephalon-mesencephalic trunk (DiA-MesA). PChoA arising from the common trunk and anastomoses with the AChoA	
II Choroidal	– ACA gives a telencephalic (TB) and a choroidal branch (CB). The CB anastomoses with the CB of the AChoA at the interventricular foramen forming the "limbic arterial arch" – Development of a TB and a diencephalic branch (DiB) from the AChoA – The CB of the AChoA anastomoses with the CB of the PChoA	
III Telencephalic primitive	– Major development of the TB of the PChoA with annexation of some AChoA and ACA territories	

(continued)

Table 2 (continued)

Stage		Illustration
IV Telencephalic intermediate	– Progressive development of the diencephalo-mesencephalic trunk of the PChoA with annexation of new cortical territories	
V Telencephalic final	– The cortical annexation gives relief to the ACA, facilitating the development of the MCA – The PChoA constitutes the main PCA stream and annexes the TB of the DiA – The CB branches of the PChoA and the DiA become the postero-medial and postero-lateral choroidal arteries (PMChoA and PLChoA)	
VI Adult disposition	– Definitive development of the AChoA with reduction of its telencephalic territories – The MesA of the PChoA corresponds to the tectal branches of the PCA – SCA, AICA, and PICA are visible	

ACA anterior cerebral artery, *AChoA* anterior choroidal artery, *BA* basilar artery, *Ca R* caudal ramus, *Cr R* cranial ramus, *ICA* internal carotid artery, *MCA* middle cerebral artery, *PCA* posterior cerebral artery, *PChoA* posterior choroidal artery, *PComA* posterior communicating artery

LPChoA, the choroidal branch of diencephalic branch becomes the MPChoA and PCA rises from the telencephalic branches of PChoA and from diencephalic artery (stage VI adult disposition, Table 2).

Number and Origin of the Artery

Cadaveric and angiographic studies were used to analyze the anatomical details of PChoAs demonstrating a high variability in terms of number of branches and origins [8, 21–28].

In most cases, MPChoA arises as a single branch but there may be up to 3 branches per hemisphere [19, 21, 23, 25, 26]. Generally, it arises from the posterolateral surface of PCA's proximal half, distal to thalamo-perforating arteries and proximal to LPChoA, in the interpeduncular or crural cistern, above the oculomotor nerve, mostly from P2 (especially P2A), but also from P1 and P1–P2 junction [19, 21, 23, 25, 26]. Some distal origins are also reported from P3 or other PCA cortical branches, such as the parieto-occipital (POA) and the calcarine arteries (CA) [21, 25–27].

On the other hand, LPChoA in most cases arises as multiple branches and there may be up to 9 branches per hemisphere [21, 22, 25, 26]. Duplication and triplication are frequently encountered and most of the time the first artery, the most anterior one, is the longest and the largest [21, 22, 25, 26]. Generally, it arises from PCA, distal to PComA and MPChoA, mostly from P2 (especially P2P) in the ambient or quadrigeminal cistern, but also P2A, P3, or other PCA branches [21, 22, 25, 26, 29].

Course of the Artery

Cadaveric dissections and angiographic studies report detailed anatomical course of PChoAs and most authors agreed on it [8, 21, 22, 24–26, 28].

From its origin MPChoA encompasses the midbrain, medial to PCA, in the superior compartment of ambient cistern, perforates the precentral cerebellar membrane to enter the quadrigeminal cistern and then points to the pineal gland. Then it turns forward, adjacent to the internal cerebral vein, above the habenular trigone, between tela choroidea layers, to enter the third ventricle roof, then runs through the choroidal fissure and the Monro foramen to reach the choroid plexus of the lateral ventricle (Fig. 1).

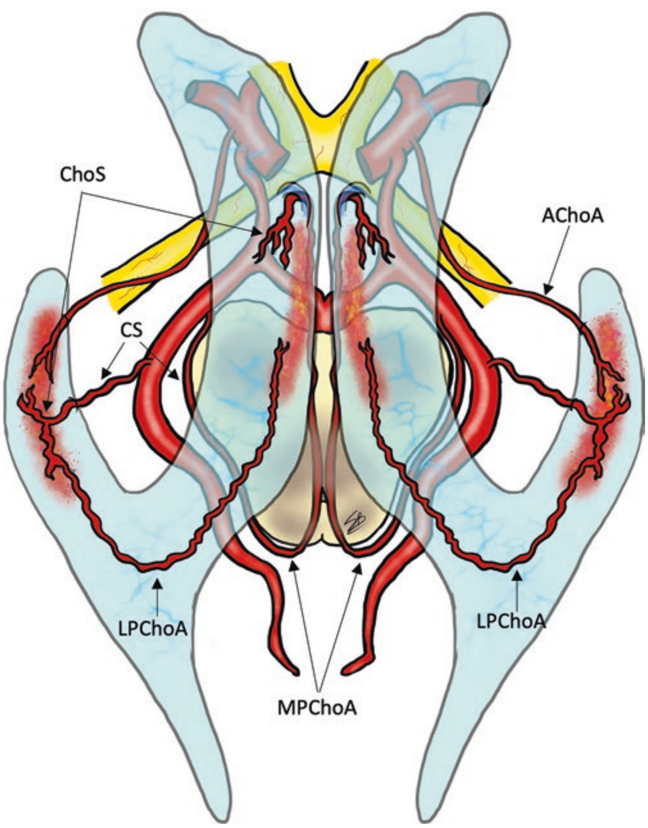


Fig. 1 Course of the medial posterior choroidal artery (MPChoA) and lateral posterior choroidal artery (LPChoA) with cisternal and choroidal segments. After its origin from the posterior cerebral artery, the MPChoA courses medially and posteriorly to reach the quadrigeminal cistern (Cisternal segment, CS). Then it bends anteriorly to the roof of the third ventricle and enters the foramen of Monro and the choroid plexus. (Choroidal segment, ChoS). The LPChoA arises from the lateral aspect of the posterior cerebral artery and points laterally (Cisternal segment, CS) to enter the temporal horn of the lateral ventricle (Choroidal segment, ChoS). The anterior choroidal artery (AChoA) is also visible in the figure

Some authors divided the MPChoA course into 2 or 3 segments: cisternal and plexal or cisternal ambient, cisternal quadrigeminal and choroidal [21, 26]. If MPChoA arises from distal PCA or other cortical branches, its course is shorter and goes retrogradely from the origin to the third ventricle roof [27].

On the other hand, LPChoA from its origin runs laterally along the upper edge of the parahippocampal gyrus, passes directly through the choroidal fissure, around the pulvinar, to enter the choroid plexus of the lateral ventricle temporal horn and atrium, toward the foramen of Monro (Fig. 1).

Some authors divided the LPChoA course into 2 segments: cisternal and choroidal (Fig. 1) [21].

A few studies analyzed the length and diameter of PChoAs [21, 25, 26]. For example, Vinas et al. in their work on 52 hemispheres reported the mean length of right MPChoA being 77.5 ± 15 mm and the left one 77.5 ± 18 mm [21]. The mean diameter of the right MPChoA being 0.8 ± 0.5 mm and the left one 0.8 ± 0.4 mm. The mean length for the first right LPChoA was 50 ± 26 mm and for the left one 58 ± 23 mm. The mean diameter of the right and left LPChoA was 0.7 ± 0.4 mm. The second and third LPChAs were shorter and thinner.

Fujii et al. distinguished the diameter at the origin of the cisternal and plexal segment: the first segment of MPChoA was on average 0.8 mm (range 0.2–1.4 mm) and the second 0.5 mm (range 0.2–1.1 mm); while the first segment of LPChoA was on average 0.6 mm (range 0.2–1.5 mm), the second was 0.4 mm (range 0.1–1.1 mm) [26]. They also identified the length of the cisternal segment, being on average 42 mm (range 8–83 mm) for the MPChoA and 23 mm (range 5–70 mm) for the LPChoA.

The possible configurations of the LPChoA and MPChoA are shown in Fig. 2.

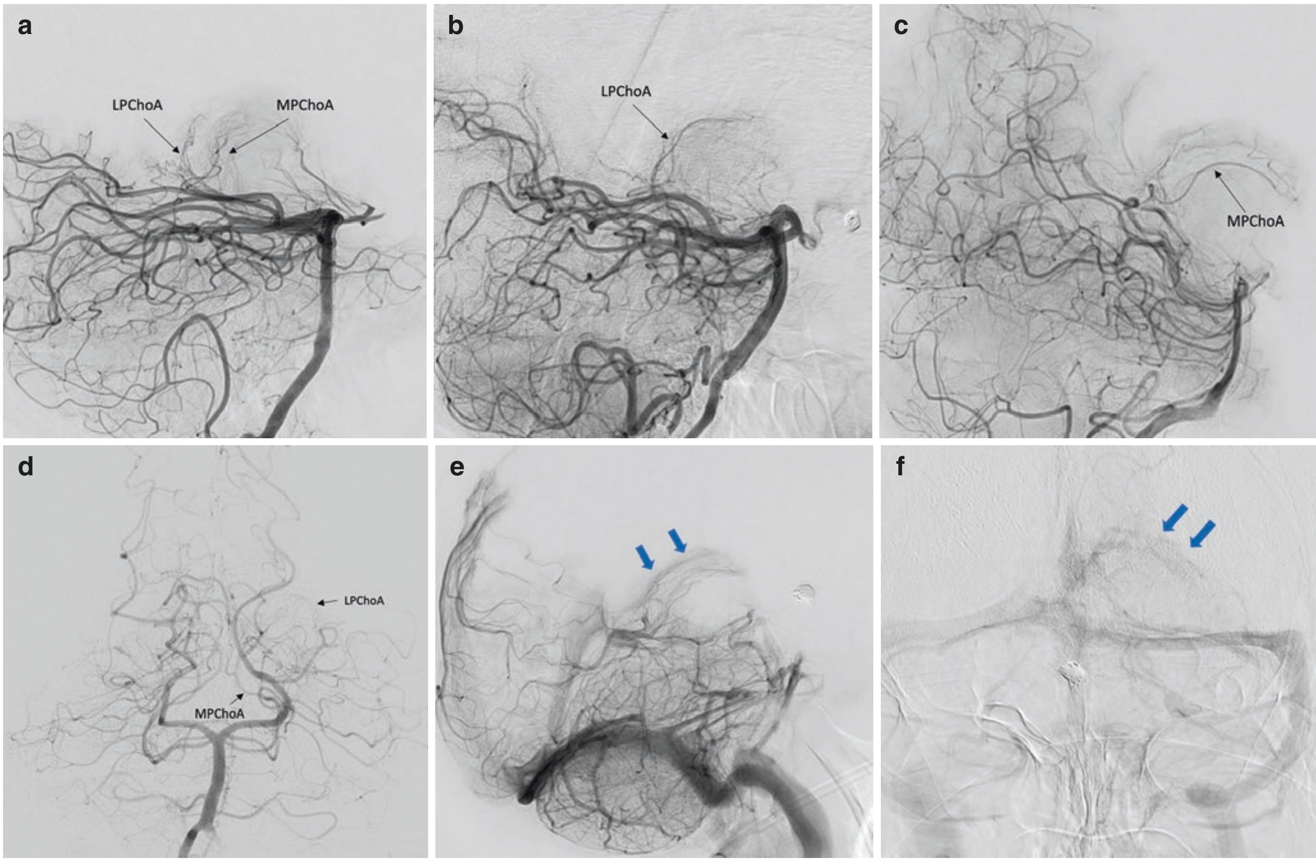


Fig. 2 Possible angiographic configurations of the medial (MPChoA) and lateral (LPChoA) posterior choroidal arteries. (**a**) The MPChoA and the LPChoA are both visible and represented in the lateral vertebral artery angiogram. (**b**) the LPChoA is dominant and the MPChoA is not visible. (**c**) The MPChoA is dominant and the LPChoA is very thiny. (**d**) Antero-posterior angiogram obtained after injection of the right vertebral artery shows the MPChoA and LPChoA. (**e** and **f**) Lateral and anteroposterior venous phases showing the "silhouette" of the choroid plexus in the venous phase (blue arrows)

Branches

Historically, Duret was the first who described different PChoA systems: the posterior one being the MPChoA and the middle and anterior ones being the LPChoA [2]. Then Hudson identified a posteromedial, posterolateral, and posteroinferior choroidal artery corresponding to the middle and anterior systems of Duret [9]. Lastly, Wolfram-Gabel defined 2 branches of MPChoA, a medial and a lateral one, and 2 branches of LPChoA, a superior and an inferior one [11].

Generally, MPChoA branches may be directed to the peduncle, tegmentum, medial geniculate body, colliculi, pulvinar, pineal gland, and medial thalamus. Vinas et al. observed 25 ± 9 branches from MPChoA while Zeal et al. counted 13 branches in total [21, 25].

On the other hand, LPChoA branches may be directed to the peduncle, posterior commissure, crura, fornix body, lateral geniculate body, pulvinar, dorsomedial thalamic nucleus, and body of caudate nucleus [21, 25–27, 30–33]. More specifically, the main trunk of LPChoA and its terminal divisions give rise to intrachoroidal, thalamic, caudate, and subependymal branches. The intrachoroidal ones form different types of vessels according to their length (short, medium, and long vessels) and according to their course and branching patterns (parallel, glomus, and bush-like vessels). Moreover, some branches may have a reverse course and some other a transverse one (centripetal) [22]. Vinas et al. observed 17 ± 8 branches from the first LPChoA, 14 ± 7 from the second, and 8 from the third while Zeal et al. counted 48 branches in total [21, 25].

Possible Anastomoses

Connections between PChoA and AChoA and between PChoAs are reported by numerous authors. They are of fundamental importance because they protect the areas of terminal supply: when ICA is occluded proximal to AChoA origin, its parenchymal territories may be supplied by the blood flow coming from PChoA, and vice versa, when PCA is occluded proximal to PChoA origin, its parenchymal territories may be supplied by the blood flow coming from AChA

[34–37]. The interdependency of these two arteries is also evident when considering the territories supplied: they are in perfect equilibrium [26].

More specifically, at first Duret identified the choroid plexus of lateral and third ventricles as the point of anastomosis between LPChoA and MPChoA [2]. Then, Beevor described several communications by injecting PChoA that opacified AChoA territories [4]. Poirier and Charpy demonstrated also the reverse [38]. Fujii et al. stated that most of anastomosis between AChoA and PChoAs are located on the surface of the choroid plexus, geniculate bodies, and uncus [26]. Vinas et al. also described most of the connections between AChoA and LPChoA at the anterior third of the temporal horn while between MPChoA and LPChoA are founded mainly at the foramen of Monro [21]. According to Carpenter et al., anastomoses between AChoA and LPChoA were present in 93% of cases and occurred: via the choroid plexus in 85% of cases, rostral to the lateral geniculate body, over its surface or at both sites in 80% of cases, directly in 6% of cases [34].

Moreover, subependymal arteries could anastomose with choroidal vessels at the third ventricle but also at the lateral ventricle, coming from thalamic or hypothalamic perforators, they merge at the attachment of the tela choroidea [9].

These connections may have an impact on clinical events, for example, Wang et al. demonstrated a correlation between LPChoA anastomosis and an increased risk of hemorrhage in Moyamoya patients [39]. The possible anastomoses of the LPChoA and the MPChoA are illustrated in Fig. 3.

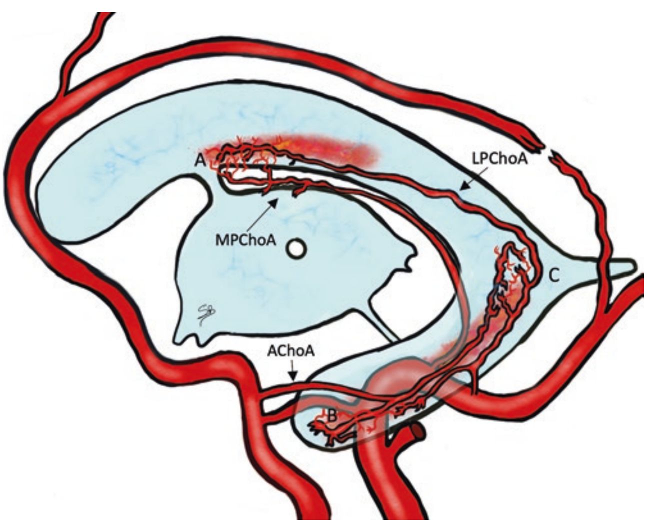


Fig. 3 Possible anastomoses between choroidal arteries. Most of the anastomoses between the lateral posterior (LPChoA) and medial posterior choroidal arteries (MPChoA) are located at the foramen of Monro (*A*). The anastomoses between the LPChoA and the anterior choroidal artery (AChoA) are mostly located at the anterior third of the temporal horn (*B*). Other anastomoses between the LPChoA and the AChoA can occur in the choroidal plexus of the lateral ventricle (*C*)

Parenchymal Territories

As we already mentioned, there is a fascinating interchangeability and interdependency among choroidal arteries, such as among cerebellar arteries. For example, the choroid plexus areas of supply of AChoA, MPChoA, and LPChoA are in equilibrium in an inversely related manner: as one enlarges, the others decrease, especially between AChoA and LPChoA, LPChoA and MPChoA, MPChoA, and MPChoA [26].

In the scientific community, there isn't an agreement about the territory supplied by the MPChoA: some authors reported that it supplies only the choroid plexus of the third ventricle, some others reported supply to choroid plexus of the third ventricle and lateral ventricles, pineal gland, and thalamus [24, 40].

Vinas et al. made a careful and detailed dissection and found a constant supply from MPChoA to [21]:

- Choroid plexus of the third ventricle
- Lateral ventricles body
- Thalamus
- Cerebral peduncles
- Tegmentum
- Medial geniculate body
- Stria medullaris
- Pineal glandOnly in some cases MPChoA supplied trigona, temporal horn, fornix, lateral geniculate body, quadrigeminal plate, posterior commissura, temporal, parietal, and occipital lobes.

Similarly, they found a constant supply from LPChoA to:

- Choroid plexus of the third ventricle
- Temporal horns
- Fornix
- Thalamus
- Splenium
- Stria medullaris
- Posterior commisura
- Temporal lobe

Only in some cases LPChoA supplied cerebral peduncles, tegmentum, lateral and medial geniculate bodies, pineal gland, quadrigeminal plate, parietal and occipital lobes [21].

More specifically, the temporal portion, the glomus, and the body portion of the choroid plexus of the lateral ventricles are supplied primarily from the AChoA and LPChoA [8, 11, 22, 25–27, 37, 41, 42]. On the other hand, the superior portion, at the level of the foramen of Monro, is continuous with third ventricle choroid plexus and is supplied primarily by MPChoA [22, 25–27, 37, 42].

Generally, MPChoA and LPChoAs send branches to their homolateral side, sporadically to the contralateral side [26].

For example, MPChoA sends branches in 22% to the contralateral side of the third ventricle and in 16% to the opposite lateral ventricle [26, 27].

Variants

Origin

Kaya et al., analyzing 28 PCA specimens, observed a more distal origin of MPChoA when P1 segment gives off more than one perforating branch: the largest one is generally the thalamo-perforating artery (42%), the MPChoA (40%) or a large trunk from which they both arise (18%), as observed also by Saeki et al. [23, 24].

Zeal et al., looking at 50 cadaveric cerebral hemispheres, described MPChoA origins from P2A segment in 50% of cases, from P2P in 21%, from P1 in 12%, from POA in 10%, from P3 in 4% and from CA in 3% [25]. On the other hand, LPChA origins from P2P in 35% of cases, from P2A in 16%, from P3 in 1%, and none from P1. The remaining 48% origin from POA (13%), anterior temporal artery (ATA) (10%), posterior temporal artery (PTA) (9%), hippocampal artery (HA) (8%), MPChoA (4%), middle temporal artery (MTA) (2%), and CA (2%). The LPChoAs arising from P2P are the largest (Table 3) [8, 25].

For Fujii et al., who studied 50 cadaveric cerebral hemispheres, MPChoA origins from PCA in 87% of cases and specifically from P2A in 54% of cases, from P1 in 14%, from P3 in 11%, from P2P in 7% [26]. In the remaining 13% of cases, it arises from PCA branches and specifically from splenial artery (SA) in 7%, from CA in 4%, from POA and PTA in 1%, from basilar artery (BA) in 1%. While LPChoA origins in 77% of cases directly from PCA and specifically from P2P in 46% of cases, from P2A in 20%, from P3 in 11%; in the remaining 23% of cases arise from PCA branches and specifically from POA in 10%, from common temporal and PTA in 3%, from ATA and CA in 2%, from HA, SA, and MPChoA in 1% (Table 3) [26].

If more than one MPChoAs or LPChoAs are present, the largest is the one with the most anterior origin [26].

As mentioned by Fuji et al., an anomalous and fascinating origin of MPChoA is from BA: it is very rare but reported [26]. Berland et al., for example, published a case report where the DSA showed a PCA originating from ICA and PChoA was absent because it arose from BA [43]. Embryologically, PCA and PChoA originate from the diencephalon-mesencephalic trunk but if this involutes after an established communication with BA, PChoA becomes a branch of BA and on the other hand PCA arises from ICA (<0.25% of cases).

Number

Although MPChoA is thought to be a single artery, in 10–40% of reported cases there are multiple MPChoAs [8, 32, 33, 44]. On the contrary, LPChoA is a single artery in only 12% of cases, most report multiple arteries [8, 25, 30, 32, 33, 45].

Zeal et al. identified from 1 to 3 MPChoAs (average 1.6) and from 1 to 9 LPChoAs (average 4) [25]. In particular, a single MPChoA was present in 54%, double in 32%, and triple in 14% [25]. Fujii et al. also identified a single MPChoA in 50% of cases, double in 30%, triple in 20%; while LPChoA was single in 18% of cases, double in 44%, and triple in 34% [26].

In most cases, LPChoA is represented by two arteries but it may range variably from 1–3 to 5–9 and could also be present as small plexal branches [7, 22, 25–27].

Nevertheless, Carpenter et al., studying 45 cadaveric hemispheres, noted the absence of LPChoAs in 8% of cases and compensation was provided by the anterior choroidal system [34].

Moreover, Marinkovic et al., analyzing 17 cadaveric brains, identifies 3 types of LPChoA: single inferior (50%), double inferior and superior (40%), and common trunk for inferior and superior (10%) [22].

Table 3 Variable origins of medial and lateral posterior choroidal artery

PChA origins													
%	P1	P2A	P2B	P3	POA	CA	ATA	MTA	PTA	HA	SA	MPChA	BA
Zeal et al. (1978)													
MPChA	12	50	21	4	10	3	–	–	–	–	–	–	–
LPChA	0	16	35	1	13	2	10	2	9	8	–	4	–
Fuji et al. (1980)													
MPChA	14	54	7	11	1	4	–	–	1	–	7	–	1
LPChA	–	20	46	11	10	2	2	–	3	1	1	1	–

Course

Variations of the course of LPChoA are more frequently reported in the literature compared to those of MPChoA.

Anomalies in the proximal course of MPChoA are observed in 17% of cases where multiple extra or prechoroidal anastomoses may occur [25, 26].

Medial posterior choroidal arteries arising from distal PCA, POA, and CA, run backward to the third ventricle [25, 26, 30, 32, 33]. On the other hand, LPChoAs arising from P2A or its cortical branches run laterally through the choroidal fissure into the choroid plexus of the temporal horn and the glomus in the atrium of the lateral ventricles to anastomose with AChoA [8, 25, 34]. Meanwhile, LPChoAs arising from P2P, P3, or their cortical branches run over the pulvinar and beneath the columns of the fornix to enter the atrium and the body of the lateral ventricles [25].

The course of LPChoAs was analyzed by Agnoli et al. in 282 angiograms and they identified 3 types of configuration in the choroidal fissure in the antero-posterior projection: slightly concave course (68%), S-form (20%), and almost straight (12%) [28].

Lopez et al. identified also 3 types of configuration in the lateral projection: concave course anteriorly (50%), convex course anteriorly (30%), and posteriorly course (20%) [29].

When a single LPChoA is present, it is longer and larger and runs along the choroidal fissure to supply the entire body of the choroid plexus [22]. When a double LPChoA is present most frequently the inferior branch approaches the posterior part of the temporal portion of the choroid plexus, while the superior one enters the choroidal fissure and then approaches the posterior part of the body portion of the choroid plexus [22]. When a common trunk LPChoA is present, it divides close to or in the choroidal fissure and the inferior branch courses along the fissure, while the superior one courses along the thalamus surface before reaching the fissure [22].

The terminal division of LPChoA is absent in 60%, bifurcates in 35%, and trifurcates in 5% of cases [22]. When it bifurcates, the medial trunk courses along the choroidal fissure or through the medial part of choroid plexus, while the lateral trunk courses through its lateral part.

Branches

Zeal et al. observed in 12% of cases a branch of MPChoA passing laterally within the quadrigeminal cistern, through the choroidal fissure and supplying an area usually own to LPChoA [25]. In 17% of cases a P2A branch, a circumflex branch of the parieto-occipital artery, joins MPChoA to form a single trunk and enters the third ventricle roof [25].

Clinical Implications

While clinical consequences of AChoA occlusion are well known, it is difficult to predict those of PChoA because of collateral circulation and choroidal anastomoses that protect the territories supplied.

In most cases, PChoA occlusion coexists with PCA or SCA involvement. It is more rarely present as an isolated stroke syndrome. Typically, when an isolated occlusion is present the damage is limited to lateral geniculate body, pulvinar, posterior thalamus, hippocampus, and parahippocampal gyrus. More in detail, if the stroke is limited to LPChoA, clinical manifestations may be represented by homonymous quadrantanopia, hemisensory loss, neuropsychological dysfunction (trans-cortical aphasia, memory troubles), and homonymous horizontal sector anopsia (lateral geniculate body). On the other hand, if the stroke is limited to MPChoA, it may be characterized by eye movement disorders, but it is less frequent [46–49].

Infarction of the PChA may be a classic ischemic stroke in origin but could also be the consequence of surgical manipulation of healthy anatomical structure [50–52].

Intraventricular or periventricular arterio-venous malformations (AVM) are frequently supplied by AChoAs and PChoAs and also those of basal ganglia, temporal lobe, midbrain, geniculate bodies, and thalamus [26, 53–60]. Choroidal arteries commonly give their contribution to aneurysms or vein of Galen aneurysmal malformations (VGAM) [61–63]. More rarely aneurysms may be located on PChoAs [64–69].

The bleeding of these vascular malformations could often cause intraventricular hematoma and consequent hydrocephalus [54, 57, 70–72]. Moreover, choroidal arteries are frequently enlarged if intraventricular masses are present [35, 73–75]. A clinical case of choroidal AVM is shown in Fig. 4.

The cisternal segment of MPChoA and LPChoA may be dislocated by PCA aneurysms, brainstem and thalamus exophytic tumors, cavernomas or peripeduncular AVMs [44]. The cisternal quadrigeminal segment of MPChoA may be displaced by pineal region tumors, quadrigeminal plate exophytic tumors, teratomas, AVMs, cavernomas, and VGAM.

The choroidal segment of MPChoA and LPChoA may be affected by intraventricular lesions, such as choroid plexus tumors, ependymomas, teratomas, and AVMs [21, 76, 77].

Medial posterior choroidal artery may be enlarged in case of AVMs, tumor of tectal region, thalamus, and third ventricle. It may be displaced anteriorly or posteriorly by pineal region tumors, anteriorly by cerebellar cyst or tumor bulging upward through tentorial notch, posteriorly by thalamic tumor, downward by ventricular enlargement or corpus callosum tumors, or upward by ventricular tumors or corpus callosum agenesis [8, 25, 32, 44, 77–81].

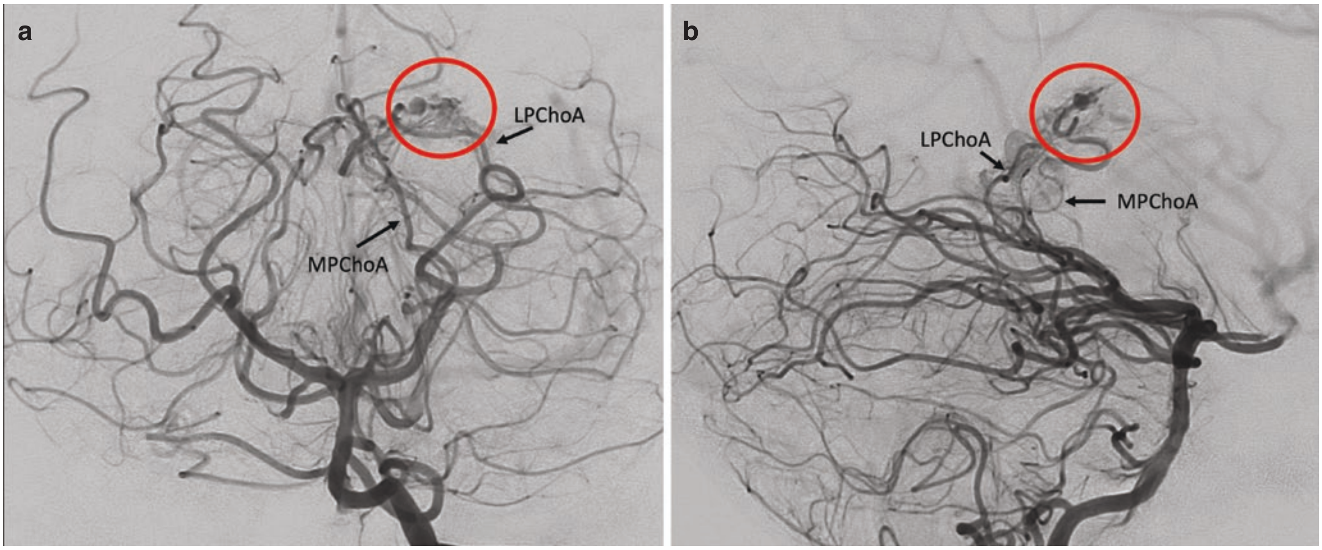


Fig. 4 Clinical case of choroidal AVM. The figure shows a clinical case of choroidal arterio-venous malformation (red circle). Figures **a** and **b** show respectively an antero-posterior view and a lateral view obtained after the left vertebral artery injection. Both show the supply of the AVM provided by the medial and lateral posterior choroidal arteries (MPChoA, LLChoA)

Lateral posterior choroidal artery defines the anterior wall of the atrium of the lateral ventricle and the posterior convexity of the thalamus on a lateral view of cerebral angiography [28] and can easily be viewed if displaced by thalamic tumors, flattened or anteriorly displaced by occipital lobe masses, posteriorly displaced by ventricular dilation, enlarged by ventricular masses such as meningiomas, ependymomas, choroid plexus papillomas, AVM of splenium, thalamus, and choroid plexus [8, 31, 44, 51, 73, 75, 78, 82–86].

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References

1. Lasjaunias P, Berenstein A, Ter Brugge KG. Surgical neuroangiography - clinical vascular anatomy and variations. Springer; 1987.
2. Duret M. Recherches anatomiques sur la circulation de l'encephale. Arch Physiol Norm Pathol. 1874;1:60–91, 11:316–53
3. Kolisko A. Uber die Beziehung der Arteria Choroidea Anterior Zuni hinteren Schenkel der inneren Kapsel des Gehirnes. 1891.
4. Beevor CE. On the distribution of the different arteries supplying the human brain. Phil Trans Roy. 1909;200:1–55.
5. Abbie AA. The blood supply of the lateral geniculate body, with a note on the morphology of the choroidal arteries. J Anat Physiol. 1933;67:491–521.
6. Padget DH. The development of the cranial arteries in the human embryo. Contrib Embryol. 1948;32:205–62.
7. Millen JW, Woollam DH. Vascular patterns in the choroid plexus. J Anat. 1953;87(2):114–23.
8. Galloway JR, Greitz T. The medial and lateral choroid arteries. An anatomic and roentgenographic study. Acta Radiol. 1960;53:353–66.
9. Hudson AJ. The development of the vascular pattern of the choroid plexus of the lateral ventricles. J Comp Neurol. 1960;115:171–86.
10. Netsky MG, Shuangshoti S. Studies on the choroid plexus. Neurosci Res. 1970;3:131–73.
11. Wolfram-Gabel R, Maillot C, Koritke JG. The vascular pattern in the Tela choroidea of the prosencephalon in man. J Neuroradiol. 1987;14(1):10–26.
12. Steeler GL. The developmental alterations in the vascular system of the brain of the human embryo. Contrib Embryology. 1918;8(271):5–38.
13. Streeter GL. Developmental horizons in human embryos. Description of age group XI 13 to 20 somites and age group CII 21 to 29 somites. Contrib Embryol. 1942;541(30):211–45.
14. Streeter GL. Developmental horizons in human embryos. Description of age group XIII embryos about 4 or 5 millimeters long and age group XIV period of indentation of the lens vescicle. Contrib Embryol. 1945;557(31):27–63.
15. Streeter GL. Developmental horizons in human embryos. Description of age groups XV XVI XVII and XVIII being the third issue of a survey of the Carnegie collection. ContribEmbryol. 1948;575(32):133–203.
16. Streeter GL. Developmental horizons in human embryos. Description of age groups XIX, XX, XXI, XXII, and XXIII, being the fifth issue of a survey of the Carnegie collection (prepared for publication by Heuser CH and Corner GW). Contrib Embryol. 1951;592(34):165–96.
17. Streeter GL. Developmental horizons in human embryos description or age groups XIX, XX, XXI, XXII, and XXIII, being the fifth issue of a survey of the carnegie collection. Contrib Embryol. 1957;611(36):167–96.
18. O'Rahilly R. Developmental stages in human embryos. Contrib Embryol. 1987;637:65–201.
19. Lasjaunias P, Berenstein A, Ter Brugge KG. Surgical Neuroangiography, vol. 1. 1987.
20. Moffat DB. The development of the posterior cerebral artery. J Anat. 1961;95:485–94.
21. Vinas FC, Lopez F, Dujovny M. Microsurgical anatomy of the posterior choroidal arteries. Neurol Res. 1995;17(5):334–44.
22. Marinkovic S, Gibo H, Milisavljevic M, Djulejic V, Jovanovic VT. Microanatomy of the intrachoroidal vasculature of the lat-

eral ventricle. Neurosurgery. 2005;57(1 Suppl):22–36; discussion 22–36
23. Kaya AH, Dagcinar A, Ulu MO, Topal A, Bayri Y, Ulus A, et al. The perforating branches of the P1 segment of the posterior cerebral artery. J Clin Neurosci. 2010;17(1):80–4.
24. Saeki N, Rhoton AL Jr. Microsurgical anatomy of the upper basilar artery and the posterior circle of Willis. J Neurosurg. 1977;46(5):563–78.
25. Zeal AA, Rhoton AL Jr. Microsurgical anatomy of the posterior cerebral artery. J Neurosurg. 1978;48(4):534–59.
26. Fujii K, Lenkey C, Rhoton AL Jr. Microsurgical anatomy of the choroidal arteries: lateral and third ventricles. J Neurosurg. 1980;52(2):165–88.
27. Timurkaynak E, Rhoton AL Jr, Barry M. Microsurgical anatomy and operative approaches to the lateral ventricles. Neurosurgery. 1986;19(5):685–723.
28. Agnoli AL, Fontana HJ, Zierski J. Posterior lateral choroidal artery in antero-posterior angiographic projection. Neuroradiology. 1982;24(1):7–12.
29. Lopez F, Braun JP. Principales tipos morfologicos de la ramas centrales de la arteria cerebral psoterior. Rev Clin Esp. 1969;114:427–30.
30. Krayenbühl HA, Yasargil MG. Cerebral angiography. 2nd ed. Philadelphia, PA: JP Lippincott; 1968.
31. Lazorthes G. Vascularisation et circulation cérébrale. Paris: Masson et Cie; 1966.
32. Margolis MT, Newton TH, Hoyt WF. Gross and roentgenologic anatomy of the posterior cerebral artery. In: Newton TH, Potts PG, editors. Radiology of the skull and brain. II. St. Louis, MO: Mosby; 1974. p. 1551–76.
33. Stephens RB, Stilwell DL. Arteries and veins of the human brain. Springfield: Charles C Thomas; 1969.
34. Carpenter MB, Noback CR, Moss ML. The anterior choroidal artery; its origins course, distribution, and variations. AMA Arch Neurol Psychiatry. 1954;71(6):714–22.
35. Goldberg HI. The anterior chroroidal artery. In: Newton TH, Potts PG, editors. Radiology of the skull and brain. II. St. Louis, MO: Mosby; 1974. p. 1628–58.
36. Galatius-Jensen F, Ringberg V. Anastomosis between the anterior choroidal artery and the posterior cerebral artery demonstrated by arteriography. Radiology. 1963;81:942–4.
37. Rhoton AL Jr, Fujii K, Fradd B. Microsurgical anatomy of the anterior choroidal artery. Surg Neurol. 1979;12(2):171–87.
38. Poirier P, Charpy A. Traité d'anatomie humaine.1911.
39. Wang J, Yang Y, Li X, Zhou F, Wu Z, Liang Q, et al. Lateral posterior choroidal collateral anastomosis predicts recurrent ipsilateral hemorrhage in adult patients with Moyamoya disease. AJNR Am J Neuroradiol. 2019;40(10):1665–71.
40. Mallory TB. Case report of the Massachusetts General Hospital: case 26061. N Engl J Med. 1940;222:191–5.
41. Hussein S, Renella RR, Dietz H. Microsurgical anatomy of the anterior choroidal artery. Acta Neurochir. 1988;92(1–4):19–28.
42. Yasargil MG. Microneurosurgery: microsurgical anatomy of basal cisterna and vessels of the brain. Stuttgart: Georg Thieme; 1984.
43. Berland LL, Haughton VM. Anomalous origin of posterior choroidal artery from basilar artery. AJR Am J Roentgenol. 1979;132(4):674–5.
44. Margolis MT, Hoffman HB, Newton TH. Choroidal arteries in the diagnosis of thalamic tumors. J Neurosurg. 1972;36(3):287–98.
45. Waddington MM. Atlas of cerebral angiography with anatomic correlation. Boston, MA: Little, Brown and Co.; 1974.
46. Neau JP, Bogousslavsky J. The syndrome of posterior choroidal artery territory infarction. Ann Neurol. 1996;39(6):779–88.
47. Takeuchi I, Takagi M, Yamagata S, Hoshino H, Takagi Y. A case of posterior choroidal artery infarction. Rinsho Shinkeigaku. 1992;32(10):1125–9.
48. Shibata K, Nishimura Y, Kondo H, Otuka K, Iwata M. Isolated homonymous hemianopsia due to lateral posterior choroidal artery region infarction: a case report. Clin Neurol Neurosurg. 2009;111(8):713–6.
49. Saeki N, Shimazaki K, Yamaura A. Isolated infarction in the territory of lateral posterior choroidal arteries. J Neurol Neurosurg Psychiatry. 1999;67(3):413–5.
50. Saito R, Kumabe T, Sonoda Y, Kanamori M, Mugikura S, Takahashi S, et al. Infarction of the lateral posterior choroidal artery territory after manipulation of the choroid plexus at the atrium: causal association with subependymal artery injury. J Neurosurg. 2013;119(1):158–63.
51. Darbari S, Meena RK, Sawarkar D, Doddamani RS, Singh M, Chandra PS. Intraventricular meningioma presenting as lateral posterior choroidal artery stroke. World Neurosurg. 2019;125:364–7.
52. Saito R, Kumabe T, Kanamori M, Sonoda Y, Mugikura S, Takahashi S, et al. Medial posterior choroidal artery territory infarction associated with tumor removal in the pineal/tectum/thalamus region through the occipital transtentorial approach. Clin Neurol Neurosurg. 2013;115(8):1257–63.
53. Perret G, Nishioka H. Report on the cooperative study of intracranial aneurysms and subarachnoid hemorrhage. Section VI. Arteriovenous malformations. An analysis of 545 cases of cranio-cerebral arteriovenous malformations and fistulae reported to the cooperative study. J Neurosurg. 1966;25:467–90.
54. Little JR, Blomquist GA Jr, Ethier R. Intraventricular hemorrhage in adults. Surg Neurol. 1977;8:143–9.
55. Crawford JV, Russel DS. Cryptic arteriovenous and venous hamartomas of the brain. J Neurol Neurosurg Psychiatry. 1956;19:1–11.
56. Margolis G, Odom GL, Woodhall B, Bloor BM. The role of small angiomatous malformations in the production of intracerebral hematomas. J Neurosurg. 1951;8(6):564–75.
57. Pia HW. The surgical treatment of intracerebral and intraventricular haematomas. Acta Neurochir. 1972;27(3):149–64.
58. Scott WG, Simril WA, Seaman WB. Intracerebral arteriovenous malformations. Their diagnosis and angiographic demostration. Am J Roentgenol Radium Therapy, Nucl Med. 1954;71:762–76.
59. Elkordy A, Endo H, Sato K, Matsumoto Y, Kondo R, Niizuma K, et al. Embolization of the choroidal artery in the treatment of cerebral arteriovenous malformations. J Neurosurg. 2017;126(4):1114–22.
60. Tahmouresie A, Quest DO. Vascular malformation of the medial posterior choroidal artery. Surg Neurol. 1979;12(3):235–7.
61. Amacher AL, Shillito J Jr. The syndromes and surgical treatment of aneurysms of the great vein of Galen. J Neurosurg. 1973;39(1):89–98.
62. Gagnon J, Boileau G. Anatomical study of an arteriovenous malformation drained by the system of Galen. J Neurosurg. 1960;17:75–80.
63. Newton TH, Troost BT. Arteriovenous malformations and fistulae. In: Newton TH, Potts PG, editors. Radiology of the skull and brain. II. St. Louis, MO: Mosby; 1974. p. 2490–565.
64. Fukuda H, Munoz D, Macdonald RL. Spontaneous thalamic hemorrhage from a lateral posterior choroidal artery aneurysm. World Neurosurg. 2013;80(6):900e1–6.
65. Weigele JB, Chaloupka JC, Lesley WS, Mangla S, Hitchon PW, VanGilder JC, et al. Peripheral aneurysms of the lateral posterior choroidal artery: clinical presentation and endovascular treatment: report of two cases. Neurosurgery. 2002;50(2):392–5; discussion 5–6
66. Muzii VF, Dashti R, Toninelli S, Hernesniemi J. Complete loop of the posterior cerebral artery with ruptured aneurysm at the origin of the medial posterior choroidal artery. A case report. Surg Neurol. 2009;72(4):403–5.
67. Alshumrani GA, Al-Qahtani S. Endovascular embolization of a giant aneurysm in medial posterior choroidal artery with associated arteriovenous malformation. Ann Saudi Med. 2013;33(3):307–9.

68. Ohta T, Ozawa H, Yamauchi T, Kubokura T. Ruptured aneurysm of the medial posterior choroidal artery first demonstrated by the microcatheter technique - case report. Neurol Med Chir (Tokyo). 2003;43(12):601–4.
69. Ungersbock K, Perneczky A. Intraventricular aneurysm of the medial posterior choroid artery clipped via the contralateral transcallosal approach. Acta Neurochir. 1986;82(1–2):24–7.
70. Schürmann K, Brock M, Samii M. Circumscribed hematoma of the lateral ventricle following rupture of an intraventricular saccular arterial aneurysm. Case report. J Neurosurg. 1968;29:195–8.
71. Bozkurt G, Yaman ME, Cevik S, Kirbas I, Yagmurlu B. Massive primary intraventricular hemorrhage due to idiopathic lateral posterior choroidal artery aneurysm: case illustration. World Neurosurg. 2018;112:199–200.
72. Vinas FC, Kazmierczak C, Coplin WM. Active intracerebral hemorrhage from the lateral posterior choroidal artery. Arch Neurol. 2000;57(6):889–90.
73. Huang YS, Araki C. Angiographic confirmation of lateral ventricle meningiomas; a report of five cases. J Neurosurg. 1954;11(4):337–52.
74. Mani RL, Hedgcock MW, Mass SI, Gilmor RL, Enzmann DR, Eisenberg RL. Radiographic diagnosis of meningioma of the lateral ventricle. Review of 22 cases. J Neurosurg. 1978;49(2):249–55.
75. Sears RA, Burnett SW. Angiographic appearance of a papilloma of the choroid plexus. A case report. J Neurosurg. 1961;18:822–5.
76. Hodge CJ Jr, King RB. Arteriovenous malformation of choroid plexus. Case report. J Neurosurg. 1975;42(4):457–61.
77. Rothman SLG, Allen VE, Simeone JF. The medial posterior choroidal arteries as an indicator od masses at the formaen of Monro. Neuroradiology. 1976;11:123–9.
78. Newton TH, Hoyt WF, Margolis MT. Pathology of the posterior cerebral artery. In: Newton TH, Potts PG, editors. Radiology of the skull and brain. II. St. Louis, MO: Mosby; 1974. p. 1580–627.
79. Greitz T. Tumours of the quadrigeminal plate and adjacent structures. Acta Radiol Diagn (Stockh). 1972;12(5):513–38.
80. Sones PJ, Hoffman JC. Angiography of tumors involving the posterior third ventricle. Am J Roentgenol Radium Therapy, Nucl Med. 1975;124(2):241–9.
81. Pachtman H, Hilal SK, Wood EH. The posterior choroidal arteries. Normal measurements and displacement by hydrocephalus or tumors of the pineal region or brainstem. Radiology. 1974;112(2):343–52.
82. Hammom WM, Kempe LG, Hayes GJ. Angiographic appearance of a papilloma of the choroid plexus of the lateral ventricle. J Neurosurg. 1963;20:711–4.
83. Rogers V. Vertebral angiography in the diagnosis of meningioma within the lateral ventricle. Br J Radiol. 1960;33:326–8.
84. Lazorthes G, Salamon G. The arteries of the thalamus: an anatomical and radiological study. J Neurosurg. 1971;34(1):23–6.
85. Tovi D, Schisano G, Liljeqvist B. Primary tumors of the region of the thalamus. J Neurosurg. 1961;18:730–40.
86. Newton TH, Hoyt WF, Margolis MT. The posterior cerebral artery. In: Newton TH, Potts PG, editors. Radiology of the skull and brain. II. St. Louis, MO: Mosby; 1974. p. 1580–627.

Cortical and Perforating Branches of the Middle Cerebral Artery

Sara Bonasia, S. Smajda, E. Ventura, and Thomas Robert

The middle cerebral artery (MCA) is the largest and the most complex cerebral artery [1]. Presenting a dual territory with a cortical supply and a deep nuclei supply, the MCA is one of the "youngest" arteries of the brain [2]. Indeed, from a phylogenic or embryologic point-of-view, MCA develops later than other cerebral arteries [3–5]. MCA is a well-studied artery because of its importance in various clinical implications: first, it bears nearly one-third of all cerebral aneurysms; second, the MCA is the most frequent location of ischemic stroke and finally, it is also involved in other pathologies as Moyamoya disease and brain arterio-venous malformations or tumors [6]. Anatomical variations of the MCA (incidence of 3.8%), even if they are less frequent if compared to other arteries, are important to be known to avoid endovascular or surgical complications [7]. In this chapter, after a brief review of its embryological development, we will discuss the normal anatomy of the MCA and its most frequent variations.

History

A lot of cadaveric studies were published in this last five decades that allowed a comprehensive knowledge of the MCA anatomy [1, 6, 8–16]. Considering the important number of pathologies involving the MCA, few aspects of the MCA anatomy are of paradigm important, as for example the anatomy of lenticulostriate arteries, the branching pattern of the MCA, and the anatomy of the "early branches". To cite only one author, A. Rhoton published and participated in different cadaveric studies that gave a structured definition of the normal anatomy of the MCA [1, 8, 13, 14]. From a radiological point-of-view, a lot of authors furnished a precise description of the MCA based on angiographic or magnetic resonance imaging analysis. One of the first of them was Salamon who described the angiographic representation of the MCA and its normal angiographic anatomy [17]. After him, of course P. Lasjaunias gave an important contribution in the radiological anatomy of the MCA and in its different anatomical variations explained by embryological knowledges [17].

Even if anatomical variations involving the MCA are rarest than for other cerebral arteries, these variations were first described by Crompton in 1962 and classified by Teal in 1973 [18, 19]. After them a consequent number of isolated cases were published that gave a strong knowledge of these variations [20–37].

Embryological Development

Our knowledge about the embryological development of the cerebral vasculature is principally based on the embryos dissection of the Carnegie Institute and on the writings of Streeter (1918) and Padget (1948) [3, 5]. Indeed, they described different stages in the vasculature development according to major vascular changes after having analyzed 22 sectioned embryos (Table 1).

At the early stage I (4–5 mm embryo), the cranial division (also named primitive olfactory artery, future anterior cerebral artery, ACA) of the internal carotid artery (ICA) can already be individualized turning around the optic vesicle and ending in the olfactory area [38].

At stage III (12–14 mm), a multiple plexiform network initiates its development at the lateral aspect of the cranial division of the ICA. This primitive middle cerebral artery

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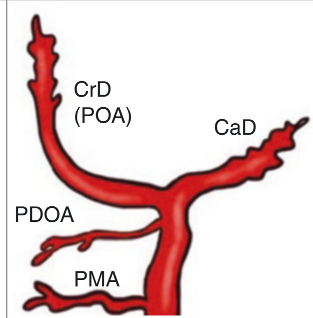
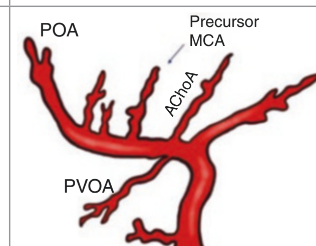
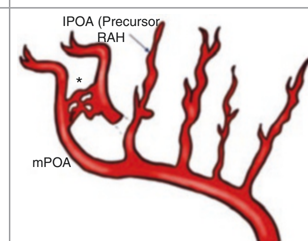
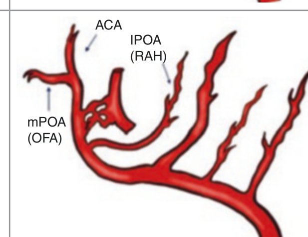
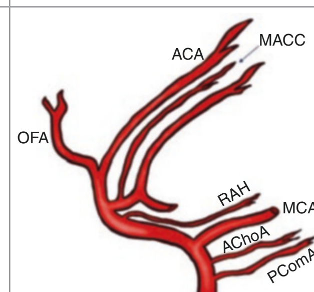
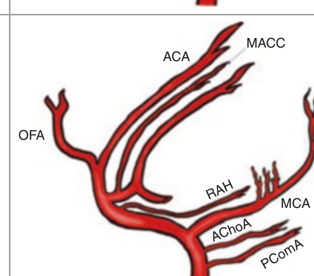
Table 1 Embryology and development of the middle cerebral artery

Stage of Padget	Size of embryo (mm)	Events	Illustration
I	4–5	– Cranial and caudal division of the carotid artery (CrD, CaD) appearance – CrD is the primitive olfactory artery (POA) – Primitive maxillary artery (PMA) and primitive dorsal ophthalmic artery (PDOA) appearance	
III	7–12	– Branches of the POA appearance: Anterior coroidal artery (AChoA), primitive ventral ophthalmic artery (PVOA), precursor of the middle cerebral artery (MCA)	
IV	12–14	– POA division in lateral (lPOA) and medial (mPOA) branches – The mPOA borrows the future ACA – The lPOA is the precursor of the recurrent artery of Heubner (RAH) – Plexiform anastomosis between the two ACAs (*, future AcomA) – MCA enlargement with lateral striate arteries development	
V	16–18	– ACA development with terminal part of the medial POA appearing as a branch, future orbito-frontal artery (OFA) – Lateral extension of the lPOA (future RAH)	
VI	20–24	– Formation of the AComA (complete circle of Willis) – Medial artery of the corpus callosum visible (MACC)	
VII	24–40	– Complete formation of the MCA with striatal and cortical branches	

extends laterally into the primitive Sylvian fissure caudal to the optic vesicle [2, 38, 39].

At stage IV (16–18 mm), by different regressions and anastomoses of the multiple channels, the MCA is formed initially with the formation of the lateral striate arteries first and with its cortical development after [2]. The MCA is completely formed at stage VII with its different perforating arteries and cortical branches [2, 38].

During stage VI (20 mm), when the anterior cerebral artery extends up between the two hemispheres, the primitive olfactory artery sends an offshoot to the anterior perforated substance, which could be interpreted as the initial step of recurrent artery of Heubner (RAH) development. From an embryologic point-of-view, the MCA could be assimilated as a branch of the cranial division of the primitive carotid artery [2].

The RAH is considered, embryologically, as an older artery that initially supplies the basal ganglion and the MCA, which is a younger artery, during its development, takes a part of the territory of the RAH by competition between these two arteries [5, 17, 19, 40, 41].

Analyses in phylogeny confirm that the MCA is one of the youngest cerebral arteries. In the study of different species, one could also highlight that the MCA is an artery appearing only in lower mammals instead of other arteries, as the RAH, that are already present in reptile's species [4].

General Anatomy of the Artery

The MCA is the artery of the lateral convexity of the cerebral hemisphere and is the largest of the cerebral arteries in 70% of cases [8]. The diameter of the MCA at its origin from the ICA is comprised between 2.4 and 5 mm with an average diameter of 3.9 mm [8, 10, 42, 43]. In almost all cases, the two MCAs are symmetric, Guerin et al. noted that an asymmetry in caliber between the two MCAs could only be in presence of asymmetric carotid pillars associated with an asymmetry of the posterior communicating arteries [44].

Different segments of the MCA, the anatomy of its perforating branches and of the "early branches" will be developed in the following and respective paragraphs. In the present paragraph, we will particularly focus on an important anatomical point which is the branching pattern of the MCA. Indeed, the branching pattern is an important aspect to be analyzed for the planning of an MCA aneurysm clipping. The most frequent branching pattern is a bifurcation in superior and inferior trunks of the MCA along its first segment [8]. This configuration is seen in 64–82% of cases depending on the studies [45]. Delion et al. noted that in case of true bifurcation, the inferior trunk is dominant in 41%, the superior trunk is dominant in 36% and the two trunks are of equal diameter in the remaining 23% [46]. A trifurcation in superior, middle, and inferior trunks is the most frequent anatomical variation and is seen in 9–12% of cases [1, 8, 14]. More than three different trunks are rare and seen in only 1–4% [1, 8, 16]. According to Yasargil, all cases of MCA need to be considered as a bifurcation, cases of trifurcation or quadrifurcation are explained by the proximity of the second bifurcation of each trunk [42]. Some authors also noted an anatomical variation of no bifurcation along the M1 segment [16, 47]. In this situation, the bifurcation is more distal along the second segment of the artery, its incidence is between 0 and 20.5% depending on the studies [45, 47–49]. Yeh (1984) tried to give a comprehensive description of the branching pattern of the MCA defining three different types [50]. Types I and II correspond to true bifurcation and the difference between the two is the location of the early branch: temporal (type I) or frontal (type II). Type III is a trifurcation to complete the different branching patterns. Even if this classification is not complete, it could help to understand the different cortical branches of the artery. Recently, Al Fauzi et al. proposed a systematic review about this argument taking into consideration all major cadaveric studies and found the following results: no bifurcation in 1.9%, bifurcation in 70%, trifurcation in 27%, and quadrifurcation in 1% [45].

Course of the Artery

The MCA has a complex course divided into four different segments which are illustrated in Fig. 1 [1].

The first segment of the MCA, called M1 or sphenoidal segment initiates at the internal carotid artery bifurcation and finishes at the limen insulae where the MCA presents a 90-degree turn called genu of the artery. The M1 segment is posterior to the sphenoidal ridge at 4–19 mm of distance [8]. The length of this segment is comprised between 14 and 20 mm long depending on the modality used to analyze the artery [46, 51].

The second segment of the MCA, M2 or insular segment, is between the genu of the artery at the limen insulae to the point where the artery presents a 180-degree turn in the circular sulcus of the insula [16]. Along this segment, the different branches, or trunks of the MCA course on the different gyri of the insula. Frontal branches of the MCA have a more vertical and shorter course than posterior branches [1, 8, 52].

The third segment of the MCA (M3) is the opercular segment and corresponds to the segment of artery between the circular sulcus to the superficial part of the Sylvian fissure. Along this segment, branches of the MCA have a superficial orientation and course on the frontal, parietal, and temporal operculum [1, 8].

The fourth or terminal segment of the MCA (M4) corresponds to the branches of the MCA from the superficial part of the Sylvian fissure to the end of the artery. Almost

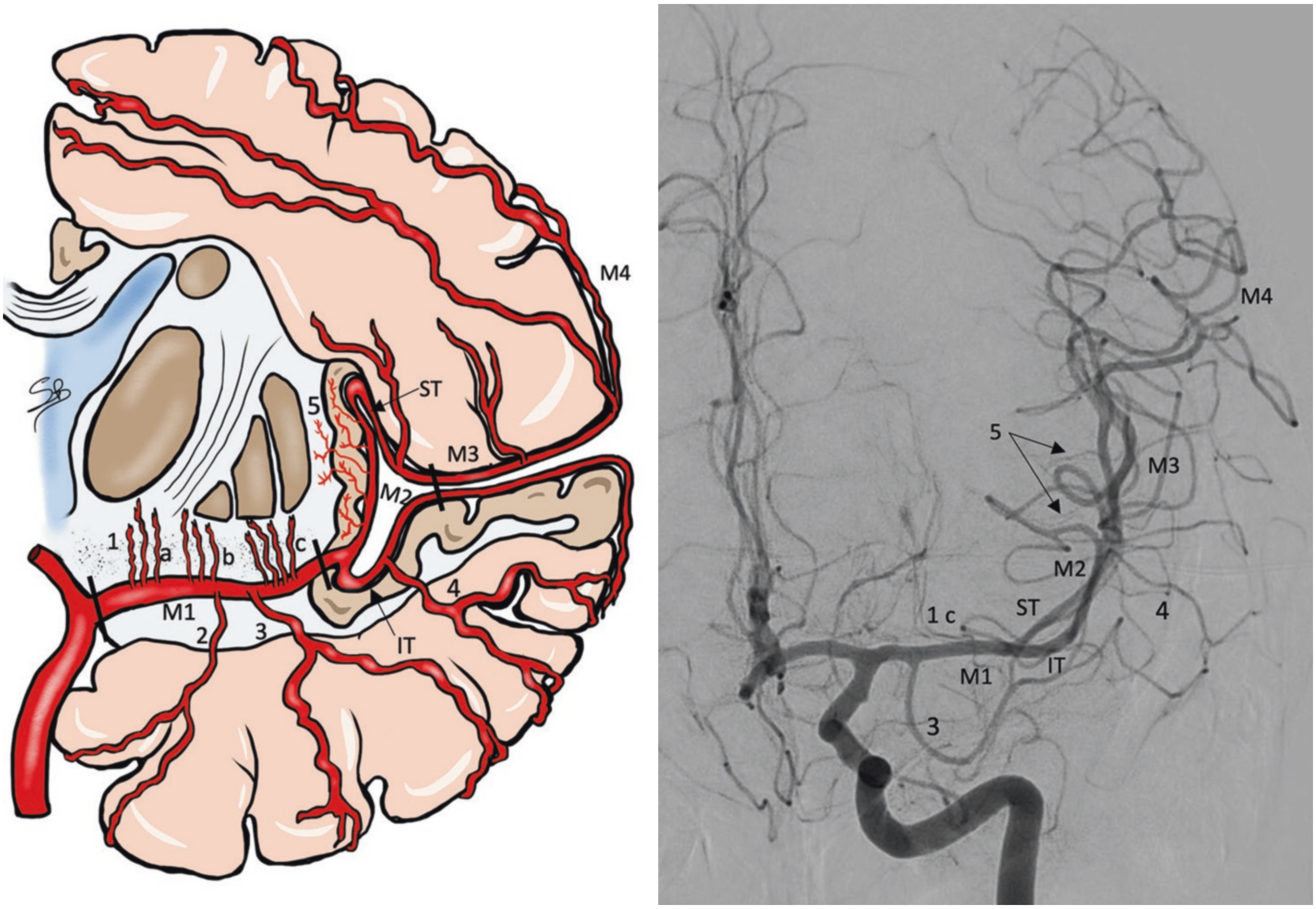


Fig. 1 Segmental division and perforating branches of the MCA. The figure shows an illustration and a DSA of an antero-posterior view of the left MCA. The M1, M2, M3, and M4 segments of the MCA are shown. The perforating branches arising from the M1 segment (*1*) represent the anterior (*a*), middle (*b*), and lateral (*c*) group of lenticulostriate arteries. The temporo-polar (*2*) and anterior temporal arteries (*3*) are shown in the illustration as early branches. In the DSA only the anterior temporal artery (*3*) is visible as early branch. After the MCA bifurcation in a superior (ST) and inferior trunk (IT), some insular perforators (*5*) origin in particular from the ST. The middle temporal artery is also shown (*4*)

all branches of the MCA are located inside the convexity sulci [8].

Branches of the Artery

As other arteries of the cerebral hemisphere, the MCA has a dual supply [1, 8, 14, 53]. The first supply is to the basal ganglia from its perforating branches and the second supply, which is the most important in size is the cortical one. This paragraph will be divided into three distinct parts to explain different branches of the MCA: perforating branches, early cortical branches, and cortical branches (Table 2).

Perforating Branches of the MCA

Perforating arteries of the MCA, also called lenticulostriate arteries, are branches of the MCA that pass through the anterior perforated substance (APS) to supply a part of the basal ganglia and of the hemispheric white matter [54–57]. Half of the perforating arteries are single vessels that arise from the MCA and enter the APS, the other half are branches arising from a common trunk [15, 58–60].

Along the MCA, the number of perforating arteries could be from one to 21 different branches (average: 9.5/hemisphere). These arteries could arise from the pre-bifurcation part of the M1 segment in 52–80% of cases, from the post-bifurcation M1 segment in 15–17% or from the M2 segment in 3–14% of cases [8, 13, 15, 42, 46, 54, 61]. Perforating arteries could also arise from the early branches in few cases,

Table 2 Branches of the MCA with their respective parenchymal territory

MCA branches	Origin from the MCA (segment, most frequent)	Diameter at origin (mm)	Territory
Medial group Perforating arteries	Proximal M1	0.1–0.6	Head of caudate nucleus Internal capsule (anterior part)
Intermediate group Perforating arteries	M1	0.1–1.7	Globus pallidus Internal capsule (anterior part)
Lateral group Perforating arteries	Distal M1- M2	0.1–2.2	Putamen Globus pallidus
Insular perforators	M2	0.1–0.8	Insula Claustrum External capsule
Orbito-frontal artery	M4	1.1	Lateral part orbital gyri Inferior frontal gyrus
Pre-frontal artery	M4	1.0	Inferior frontal gyrus Middle frontal gyrus
Pre-central artery	M4	1.3	Inferior frontal gyrus (post.Part) Middle frontal gyrus (post.Part)
Central artery	M4	1.3	Ascending frontal gyrus
Anterior parietal artery	M4	1.2	Anterior part parietal gyrus
Posterior parietal artery	M4	1.4	Posterior part parietal gyrus
Angular artery	M4	1.5	Supra-marginal gyrus Angular gyrus
Temporo-occipital artery	M4	1.4	Superior temporal gyrus Occipital gyri
Posterior temporal artery	M4	1.3	Superior temporal gyrus Middle temporal gyrus
Middle temporal artery	M4	1.2	Superior temporal gyrus Middle temporal gyrus Inferior temporal gyrus
Anterior temporal artery	M4	1.2	Superior temporal gyrus Middle temporal gyrus Inferior temporal gyrus Fusiform gyrus
Temporo-polar artery	M4	0.9	Temporal pole

most frequently from a frontal early branch [62]. Umansky et al. noted that 6% of perforating branches arise from an early branch [15].

These little but important arteries are classified differently depending on the authors; in this chapter, we choose the classification of Gibo et al. that seems the most accepted by other authors [8]. Perforating arteries of the MCA could therefore be classified into three different groups:

- Medial group: there is the least constant perforating arteries' group. These arteries branch off from the postero-superior aspect of the M1 segment, have a direct course to their entry point into the APS. Their entry point into the APS is in 90% of cases in the lateral territory and in 10% of cases in the medial territory (the limit is the posterior projection of the olfactory tract) [1, 8, 13].
- Intermediate group: these arteries also arise on the superior and posterior aspect of the MCA (M1 segment) and have a direct sub-arachnoid course. All these arteries penetrate the APS in its lateral territory, 51% in the middle zone, 35% in the posterior zone, and 14% in the anterior zone [13].
- Lateral group: this is the most constant group which could arise from the pre-bifurcation or the post-bifurcation M1 segment (equal incidence) [1]. These perforating arteries arise from the supero-posterior aspect of the principal artery and have an S-shaped course before entering the APS. They penetrate the APS principally in the postero-lateral zone (90% in the posterior zone and 10% in the middle zone) [8, 13, 14].

Depending on the anatomical pattern of its perforating arteries, Grand et al. described three distinct types of MCA anatomy: group I when the largest perforating arteries arise from the proximal M1(14% of cases), group II when they arise near but proximal to the bifurcation (39%), and group III when they are distal to the bifurcation (47%) [55].

For insisting on the presence of "distal" perforating arteries, Delion et al. described what they called the "insular perforators" which are perforating arteries that arise from the insular segment of MCA [46]. Most of these little arteries perforate the posterior part of the insula instead of passing through the APS to supply the insula and the corona radiata. Their average diameter is 0.3–0.5 mm, and their average number is 4.5/hemisphere. Most of the insular perforators arise from the superior trunk (72.5%), others from the inferior trunk in 19.8% or from the middle trunk in 7.7%.

Early Cortical Branches of the MCA

Early branch of the MCA is the name given to cortical branches that arise proximal to the first bifurcation of the MCA. Originally, Yasargil, in its monograph, described in most hemispheres the presence of three distinct early temporal arteries: the uncal artery, the temporo-polar artery, and the anterior temporal artery [42]. After its description, different large cadaveric studies meticulously presented anatomical variations concerning these early branches of the MCA [14, 16, 53, 63–68]. Gibo et al. noted the presence of an early branch in 54% of hemispheres [8]. It was a temporal branch in 88% and a frontal branch in only 12%. Other authors found a similar incidence of early branch comprised between 47% and 58% in cadaveric series [6, 16, 47, 63, 67, 69]. Tanriover et al. also noted that in case of early temporal branch, the presence of only one artery is seen in 78%, two distinct branches in 20% and three branches in 2% [14]. They also gave a comprehensive classification of the anatomical pattern concerning the early branches:

- Group I: No early branch.
- Group II: Only temporal branch.
  - A: Common trunk for temporo-polar, anterior, middle, and posterior temporal arteries.
  - B: Only temporo-polar artery.
  - C: Two distinct arteries: temporo-polar and anterior temporal arteries.
- Group III: Only frontal branch: orbito-frontal or pre-frontal artery.
- Group IV: Frontal and temporal branches.
  - A: Orbito-frontal artery and common trunk for temporo-polar and anterior temporal arteries.
  - B: Common trunk for orbito-frontal, pre-frontal and precentral arteries, and temporo-polar artery.
  - C: Common trunk frontal and two distinct temporal arteries.

In rare cases as described by Umansky et al., an early temporal cortical branch could arise from the anterior choroidal artery [16].

Cortical Branches of the MCA

Various authors described the different cortical areas of the MCA [6, 17, 22, 70]. The most used and accepted subdivision is whom of Michotey et al. who divided the cortical areas of the MCA into 12 distinct cortical branches (Fig. 2) [8].

- The orbito-frontal artery, which is an early branch in 6–23%, origins in almost all cases from the superior trunk, but could also arise from the pre-frontal artery [8, 16]. It is one of the smallest branches of the MCA with a mean caliber of 1.1 mm [9, 67]. It is an inconstant branch but seen in 83–98% of cases with a possible duplication (15%) [71, 72].

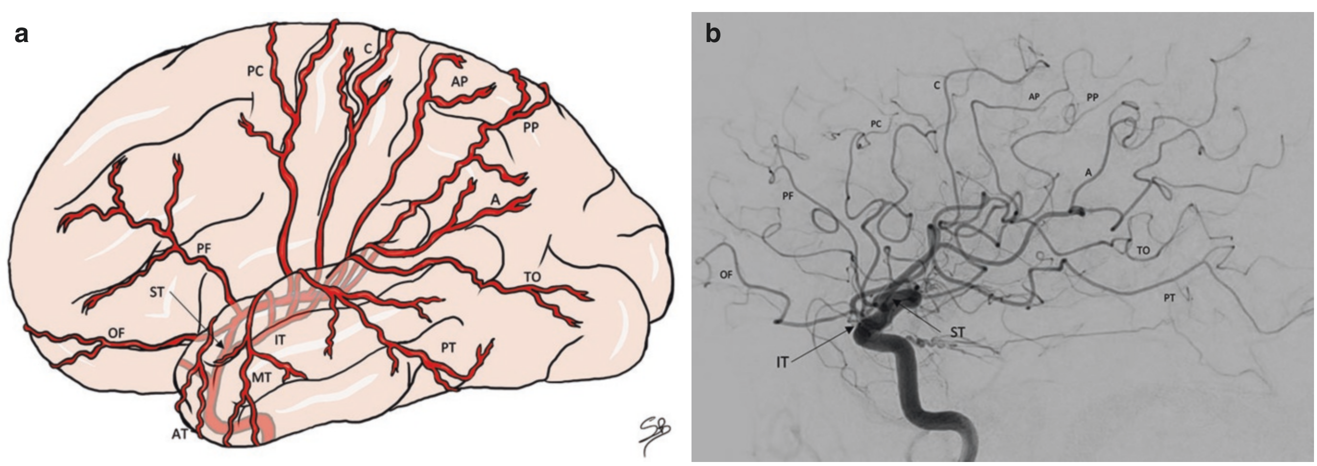


Fig. 2 Illustration of the principal branches of the MCA. *IT* inferior trunk, *ST* superior trunk, *OF* orbito-frontal branch, *PF* pre-frontal branch, *PC* pre-central branch, *C* central branch; *AP* anterior parietal branch, *PP* posterior parietal branch, *A* angular artery, *TO* temporo-occipital branch, *PT* posterior temporal artery, *MT* middle temporal branch, *AT* anterior temporal branch

- The pre-frontal artery is also a small cortical branch (diameter 1 mm) that arises from the superior trunk (96%) or as an early branch (4%) [45]. The presence of this artery is noted in 90–100% of hemispheres [46, 71].
- The pre-central artery (1.3 mm) is present in almost all hemispheres (95–100%) and could have a duplicated origin in 25% of cases [16, 17]. It arises from the superior trunk in 95% or from the central artery in 5% [71].
- The central artery is one of the most important cortical branches (1.3 mm) and is constant [9]. Its origin is from the superior trunk in 91%, from the pre-central artery in 5%, or rarely from the inferior trunk (4%) [71].
- The anterior parietal artery (1.2 mm) is present in almost all hemispheres (88–100%) and has often a double origin (30%) [1]. It arises from the superior trunk in 78%, the inferior trunk in 18%, or from the central artery in 4% [67].
- The posterior parietal artery is one of the largest branches (1.4 mm) and is constant [9]. Even if its origin is double in 15%, it branches off from the superior trunk in 50%, the inferior trunk in 41%, or from the angular artery in 9% [71].
- The angular artery could be considered as the terminal branch of the MCA (1.5 mm) and is constant [67]. It arises from the inferior trunk (79%) or from the superior trunk (21%) [71].
- The temporo-occipital artery is also a constant artery arising from the inferior (92%) or superior trunk (8%) [71].
- The posterior temporal artery (1.3 mm) is present in 90–100% of hemispheres, could have a double origin in 5% and arises from the inferior trunk (61%) or from another temporal branch (39%) [16].
- The middle temporal artery (1.2 mm) arises from the inferior trunk (47%), as an early branch (12%) or shares its origin with another temporal branch [46].
- The anterior temporal artery is frequently a little (1.2 mm) early temporal branch (22%).
- The temporo-polar artery is an inconstant branch (60–87% of cases). It is the thinnest branch of the MCA (0.9 mm) and arises as an early branch in 40% of hemispheres [71].

Parenchymal Territory

MCA, as other principal cerebral arteries, has a dual parenchymal territory [73]. These two territories are clearly separated even if some cases of common trunk between a perforating artery and a cortical artery are described, giving to this common trunk a dual parenchymal territory [54]. The first one is the deep territory supplied by the perforating arteries of the MCA. The second one is the cortical territory of the MCA supplied by its cortical branches.

Perforating Arteries Supply

The deep territory of the perforating branch of the MCA is in competition during the embryological development with the territory of the recurrent artery of Heubner and with perforators from the internal carotid artery and from the anterior cerebral artery [2, 17, 38]. The principal structures supplied by perforating arteries of the MCA are:

- The dorsal half of the internal capsule.
- The head of caudate nucleus.
- The putamen.
- The globus pallidus.
- The claustrum.
- The external capsule.
- The basal nucleus of Meynert (inconstant).
- The innominate substance and the diagonal band of Broca (inconstant).

Cortical Branches Supply

The cortical territory of the MCA represents between 60% and 80% of the cortical surface of the hemisphere [8, 46, 54]. It could be divided into 12 territories corresponding to the respective 12 distinct cortical arteries described before but this is very inconstant, each cortical territory is in balance with the territory of each other. With the aim to simplify, the cortical territory of the MCA could be seen as follows:

- Majority of the lateral surface of the hemisphere.
- Insular surface.
- Lateral part of the orbital surface of the frontal lobe.
- Lateral surface of the temporal lobe.
- Lateral part of the inferior surface of the temporal lobe.

The frontal and occipital pole of the hemisphere is not supplied by the MCA but respectively by the anterior cerebral artery and by the posterior cerebral artery [73].

Duplicated Middle Cerebral Artery

The first author who used the terminology “duplicated middle cerebral artery” was Teal in 1973 and described it as a supplementary branch arising from the internal carotid artery distal to the origin of the anterior choroidal artery (AChA) that courses in the Sylvian fissure [18]. Before it, few cases were published and the first one was described by Crompton in 1962 [19]. The incidence of duplicated MCA is between 0.2 and 2.9% depending on the modality of study (cadaveric, operative, or radiological) [30, 67, 74–80].

Two different embryological hypotheses were developed to explain this anatomical variation [17, 40, 81]. The first one is a lack of fusion of the plexiform primitive MCA living in two principal arteries instead of only one. The second hypothesis is a variation in origin of an early branch that arises from the distal ICA instead of from the M1 segment of the MCA. Based on these two different embryological explanations, Kai et al. proposed a two-group classification with [10]:

- Type A corresponds to a duplicated MCA with the same course and same diameter than the principal artery. Its origin is on the top of the ICA bifurcation. Perforating arteries could arise from both trunks.
- Type B is a supplementary vessel that arises from the ICA between the AChA and the ICA bifurcation, it courses over the temporal pole instead of inside the Sylvian fissure and is smaller in diameter than the principal MCA. This variant usually does not bear any perforating arteries [23].

Duplicated MCA is an anatomical variation without any clinical consequences, but few cases of associated aneurysms were described [7, 10, 35, 75, 82–89]. Typically, they are aneurysms located at the origin of the duplicated MCA that need to be managed as other ICA aneurysms. This highlights one more time the importance of a hemodynamic factor in the development of cerebral aneurysms.

Two different cases caught our attention, the first one was published by Tutar et al. who described a case of duplicated MCA that arises from the petrous ICA and passes medial to the temporal lobe to enter the Sylvian fissure [90]. The second particular case was published by Uchino et al. and was a case of association between a duplicated MCA and an accessory MCA on the same hemisphere [75].

Accessory Middle Cerebral Artery

The term "accessory middle cerebral artery" (AccMCA) was first used in 1962 by Crompton when he examined 347 brain specimens and reported an anomalous large branch passing into the Sylvian fissure in 11 cases (3.6%) [6]. In 10 of these, the artery took its origin from the internal carotid artery and in one case from the anterior communicating artery region. Jain (1964) noted this anomalous branch with a similar incidence (3.4%, 10/300 brain hemispheres) but, in contrast to Crompton's series, eight AccMCA arose from the anterior cerebral artery and only 2 from the ICA bifurcation. Few years later, the term "accessory MCA" was reserved for the vessel arising from the anterior cerebral artery [6]. In 1977, Manelfe established a classification of these variants including three different types [17]. Types I and II are duplicated MCA where the duplicated artery arises at the bifurcation or at the proximal part of the A1 segment. Type III involves the RAH with an AccMCA arising from the A1-A2 junction. Two cases of duplicated and accessory MCA are illustrated respectively in Figs. 3 and 4.

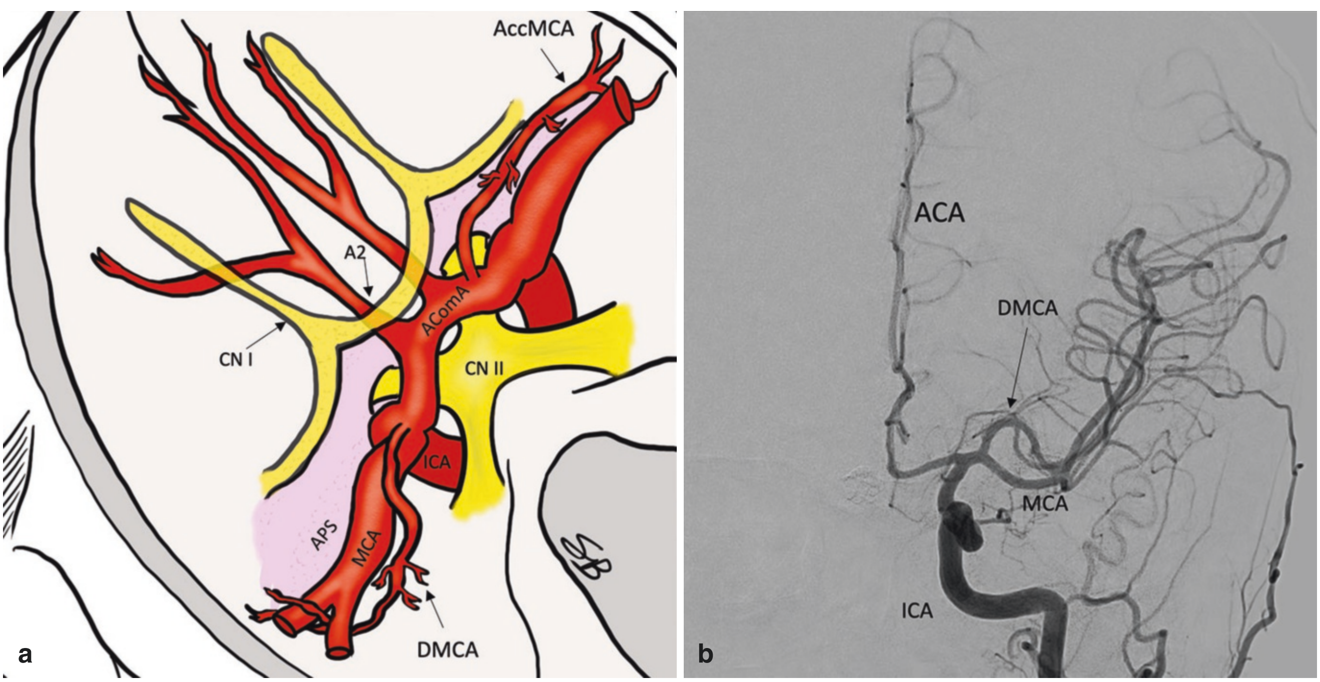


Fig. 3 Illustrations and clinical case of duplicated MCA. (**a**) On the left side, the artery origins from the ICA bifurcation and is commonly named duplicated middle cerebral artery (DMCA) or type I of Manelfe. On the right side is illustrated AccMCA arising from the proximal part of the A1 segment (type II of Manelfe). (**b**) Clinical case with DSA and left ICA injection in antero-posterior view. After the ICA bifurcation into anterior cerebral artery (ACA) and middle cerebral artery (MCA), a second MCA arises from the ICA bifurcation. This second artery is named duplicated MCA (DMCA)

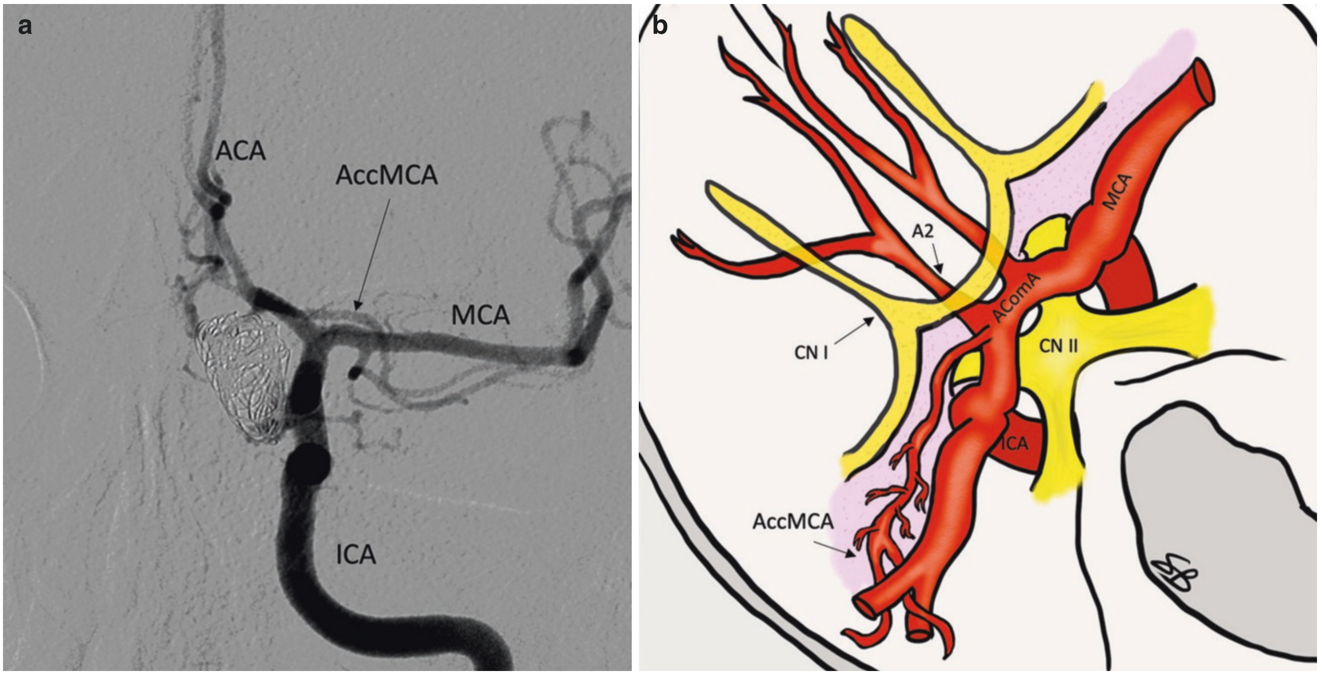


Fig. 4 Illustration and clinical case of accessory MCA (AccMCA). (**a**) Clinical case with DSA injection of a patient with AccMCA arising from the A1 segment of the anterior cerebral artery (ACA). (**b**) On the left side, this AccMCA arises from the distal part of the A1 segment (type III of Manelfe). *MCA* middle cerebral artery, *ICA* internal carotid artery, *AComA* anterior communicating artery, *CN I* olfactory nerve, *CN II* optic nerve

The AccMCA could be defined as a vessel arising from the anterior cerebral artery, in the region of the A1-A2 junction that courses into the sylvian fissure superior to the ICA bifurcation and superior to the M1 segment and that participates in the vascularization of the frontal cortex.

After these first descriptions, few isolated cases of AccMCA were described and the incidence of this variant was estimated as 0.3%, principally based on angiographic studies [18, 28, 29, 34, 37, 91–94]. It is generally accepted that the AccMCA has perforating branches destinated to the APS [25, 93]. The cortical territory of an AccMCA could also be variable but the lateral part of the orbito-frontal gyri and the prefrontal region are the most frequently involved [37, 95]. Lasjaunias and Berenstein (2001) sometimes noted also its implication in the supply of the anterior temporal area [17].

Embryologically, the RAH and the MCA develop after the primitive olfactory artery (anterior division of the ICA or anterior cerebral artery) and could both be considered as branches of the ACA. Several hypotheses were formulated regarding the development of the AccMCA. Handa et al. (1970), supported by other authors [25, 28, 29], thought the AccMCA to be a hypertrophied RAH extending the Abbie's theory that the RAH is a remnant of anastomotic channels between the ACA and the MCA [92]. Few years later, Teal et al. (1973) disagreed with Handa arguing that (1) the perforating arteries do not always originate from the AccMCA, (2) a RAH could co-exist with an AccMCA, and (3) the AccMCA passes lateral to the APS where the RAH enters into the APS [18, 92]. The fact that the AccMCA has frequently perforating branches and that the RAH could be multiple puts the Teal's objections into question. Lasjaunias and Berenstein (2001) also explained by phylogenic analysis the AccMCA as a hypertrophic RAH with a cortical supply [17].

Fenestrated Middle Cerebral Artery

Fenestrations of cerebral arteries are more frequent on the vertebro-basilar system and are defined as the division of the artery into two distinct channels with complete intimal separation [21, 31, 33, 96–99]. Fenestration of the MCA is less frequent, and its incidence is estimated to 0.17–1% [19, 32, 47, 100]. This anatomical variation could be explained by the absence of complete fusion between the multiple arterial twigs during the embryological life [17, 101]. Most of the fenestration of the MCA involved the proximal part of the M1 segment but could also involve the pre-bifurcation part of the M1 segment. Gailloud et al. noted that in almost all cases an early temporal branch arises from the inferior channel of the fenestration and postulated that the early branch plays a role in the formation of the fenestration [102]. In rare cases, an aneurysm is located at the proximal part of the fenestration highlighting the importance of the hemodynamic stress factor [23, 97].

Twig-Like Middle Cerebral Artery or Unfused Middle Cerebral Artery

This anatomical variation is not well known and is often misdiagnosed as a unilateral Moyamoya disease [103, 104]. It consists in the complete persistence of the embryological multichannel network of the proximal part of the MCA (Table 3) [11].

The twig-like MCA could be described with the following criteria to help differentiate it from a Moyamoya syndrome [104]:

- No steno-occlusive lesion of the ICA or ACA.
- Plexiform arterial network instead of MCA trunk.
- Normal anatomy of the perforating arteries originating from the plexiform network.
- Anterograde flow in the MCA branches.

The first case of twig-like MCA was described by Cekirge et al. in 2005 and nowadays, Seo et al. presented the largest series of this variant (15 cases) [104, 105]. The incidence of this variation is about 0.1% and is frequently associated with an hypertrophied RAH or an AccMCA [103–108]. The presence of a saccular aneurysm located distally to the plexiform network was already described in the series of Seo et al. [104]. In such a case, the principal technical difficulty of surgical clipping is to obtain an efficient proximal control. Two clinical cases of this variant are shown in Fig. 5.

Table 3 Principal anatomical variations involving the MCA

Anatomical variation	Definition	Incidence	Embryological explanation	Associated pathology
Duplicated MCA	Supplementary vessel from the distal ICA	0.2–2.9%	Lack of fusion of the plexiform network Early branch from the ICA	Aneurysm of the duplicated MCA
Accessory MCA	Supplementary vessel from the ACA	1.2–3.4%	Hypertrophied RAH	No associated pathology
Fenestration MCA	Double channel on proximal M1	0.17–1%	Lack of fusion of the plexiform network	Aneurysm of the proximal part of the fenestration
Twig-like MCA	Plexiform network M1	0.1%	Complete persistence of the embryological network	Distal aneurysm

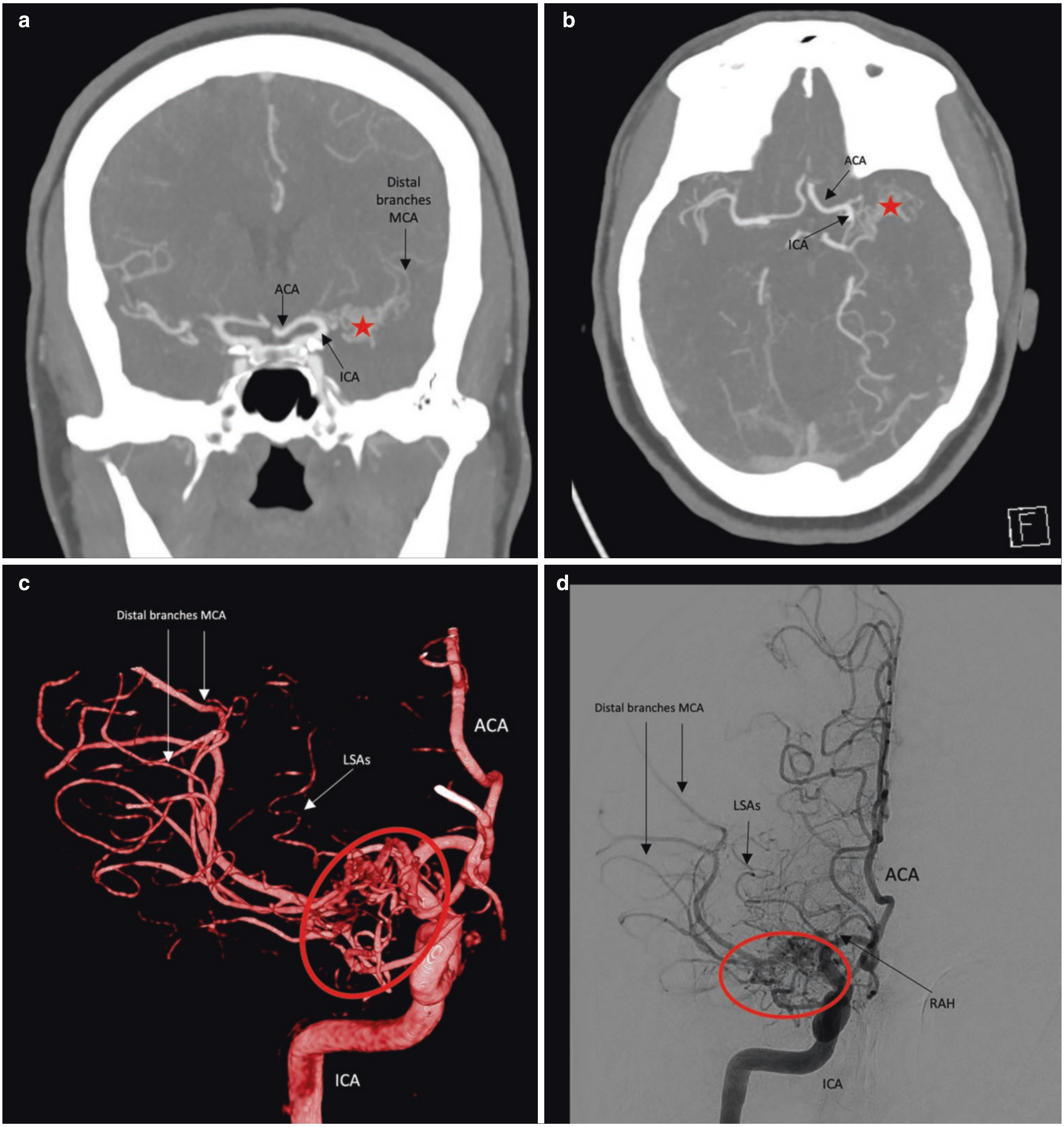


Fig. 5 Clinical cases of Twig-like MCA. Figures (**a** and **b**) show the case of a patient with a twig-like MCA on the left side, shown through an Angio-CT scan. After the internal carotid artery (ICA) bifurcation, the anterior cerebral artery (ACA) is clearly visible, while the middle cerebral artery appears as a plexiform arterial network (red star). Figures (**c** and **d**) show a second case of the same variant on the right side (red circle), through DSA after right ICA injection. The lenticulo-striate arteries (LSAs) are normally visible, and the recurrent artery of Heubner (RAH) has a hypertrophic aspect. In both cases the distal branches of MCA are normally visible and irrorated by an anterograde flow. In both cases there are no steno-occlusive lesion of the ICA or ACA

Clinical Implications

Anatomy of the MCA and knowledge of its various anatomical variations are of paramount importance for the treatment of all pathologies involving the MCA [8, 10, 17, 109]. The most important and known is the cerebral aneurysm of the MCA bifurcation but the MCA is also involved in the majority of ischemic stroke, arterio-venous malformations, and is used in case of extracranial-intracranial arterial anastomosis. In this chapter, we highlight the importance of a strong MCA anatomy knowledge to address some technical problems.

The opening of the Sylvian fissure is the first step of different treatment as for an aneurysmal clipping or an insular surgery [109]. During the opening of this arachnoid fissure, maximal attention is to minimize the retraction of frontal and temporal lobe and to avoid pia-mater transgression. The observation and recognition of the early branches of the MCA could help a lot during this step and give to the surgeon an idea of the position of the M1 segment before seeing it [52, 81, 110].

Comprehensive knowledge of the lenticulostriate arteries anatomy is also crucial in vascular surgery if the surgeon needs to clip temporarily the M1 segment [10, 111]. It avoids closure or rupture of these important little arteries. The site of origin, side of origin, and course of these perforating arteries need to be known to limit complications of these procedures.

As we have seen before, perforating arteries of the MCA arise from the proximal part of the MCA but it is frequent to see some of these arteries arising near the bifurcation. This is very important to take in mind that these little arteries could be hidden by the aneurysmal sac especially if the aneurysm is oriented superiorly or posteriorly [66]. The surgeon needs to search these arteries on the posterior wall of the aneurysm before definitive clip placement and a 360-degree dissection of the aneurysm is always the best management to avoid such a complication.

With an increasing navigability of distal access catheter this last decade, thrombectomy transformed stroke management allowing even more distal thrombus aspiration possible. These procedures ask to the physician a detailed knowledge of the distal MCA anatomy not only to diagnose distal thrombus of the MCA but also to perform microcatheter navigation safer [112].

References

1. Rhoton AL Jr. The supratentorial arteries. Neurosurgery. 2002;51(4 Suppl):S53–120.
2. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: a pictorial review. Surg Radiol Anat. 2021;43(6):961–73. https://doi.org/10.1007/s00276-021-02684-y.
3. Streeter GL. The developmental alterations in the vascular system of the brain of the human embryo. Contrib Embryol. 1918;8(271):5–38.
4. Abbie AA. The morphology of the fore-brain arteries, with especial reference to the evolution of the basal ganglia. J Anat. 1934;68(Pt 4):433–70.
5. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–62.
6. Jain KK. Some observations on the anatomy of the middle cerebral artery. Can J Surg. 1964;7:134–9.
7. Uchino A, Kato A, Takase Y, Kudo S. Middle cerebral artery variations detected by magnetic resonance angiography. Eur Radiol. 2000;10(4):560–3. https://doi.org/10.1007/s003300050960.
8. Gibo H, Carver CC, Rhoton AL Jr, Lenkey C, Mitchell RJ. Microsurgical anatomy of the middle cerebral artery. J Neurosurg. 1981;54(2):151–69. https://doi.org/10.3171/jns.1981.54.2.0151.
9. Kahilogullari G, Ugur HC, Comert A, Tekdemir I, Kanpolat Y. The branching pattern of the middle cerebral artery: is the intermediate trunk real or not? An anatomical study correlating with simple angiography. J Neurosurg. 2012;116(5):1024–34. https://doi.org/10.3171/2012.1.JNS111013.
10. Kai Y, Hamada J, Morioka M, Yano S, Kudo M, Kuratsu J. Treatment of unruptured duplicated middle cerebral artery aneurysm: case report. Surg Neurol. 2006;65(2):190–3; discussion 3. https://doi.org/10.1016/j.surneu.2005.05.032.
11. Marinkovic S, Gibo H, Milisavljevic M, Cetkovic M. Anatomic and clinical correlations of the lenticulostriate arteries. Clin Anat. 2001;14(3):190–5. https://doi.org/10.1002/ca.1032.
12. Pai SB, Varma RG, Kulkarni RN. Microsurgical anatomy of the middle cerebral artery. Neurol India. 2005;53(2):186–90. https://doi.org/10.4103/0028-3886.16406.
13. Rosner SS, Rhoton AL Jr, Ono M, Barry M. Microsurgical anatomy of the anterior perforating arteries. J Neurosurg. 1984;61(3):468–85. https://doi.org/10.3171/jns.1984.61.3.0468.
14. Tanriover N, Kawashima M, Rhoton AL Jr, Ulm AJ, Mericle RA. Microsurgical anatomy of the early branches of the middle cerebral artery: morphometric analysis and classification with angiographic correlation. J Neurosurg. 2003;98(6):1277–90. https://doi.org/10.3171/jns.2003.98.6.1277.
15. Umansky F, Gomes FB, Dujovny M, Diaz FG, Ausman JI, Mirchandani HG, et al. The perforating branches of the middle cerebral artery. A microanatomical study. J Neurosurg. 1985;62(2):261–8. https://doi.org/10.3171/jns.1985.62.2.0261.
16. Umansky F, Juarez SM, Dujovny M, Ausman JI, Diaz FG, Gomes F, et al. Microsurgical anatomy of the proximal segments of the middle cerebral artery. J Neurosurg. 1984;61(3):458–67. https://doi.org/10.3171/jns.1984.61.3.0458.
17. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer edition; 2001.
18. Teal JS, Rumbaugh CL, Bergeron RT, Segall HD. Anomalies of the middle cerebral artery: accessory artery, duplication, and early bifurcation. Am J Roentgenol Radium Therapy, Nucl Med. 1973;118(3):567–75. https://doi.org/10.2214/ajr.118.3.567.
19. Crompton MR. The pathology of ruptured middle-cerebral aneurysms with special reference to the differences between the sexes. Lancet. 1962;2(7253):421–5. https://doi.org/10.1016/s0140-6736(62)90281-7.
20. Waga S, Kojima T, Morooka Y, Sakakura M. Aneurysm of the accessory middle cerebral artery. Surg Neurol. 1977;8(5):359–60.
21. Ito J, Maeda H, Inoue K, Onishi Y. Fenestration of the middle cerebral artery. Neuroradiology. 1977;13(1):37–9. https://doi.org/10.1007/BF00335032.
22. Waddington MM. Microsurgical anatomy of the middle cerebral artery. Union Med Can. 1984;113(4):264–6.

23. Abanou A, Lasjaunias P, Manelfe C, Lopez-Ibor L. The accessory middle cerebral artery (AMCA). Diagnostic and therapeutic consequences. Anat Clin. 1984;6(4):305–9. https://doi.org/10.1007/BF01654463.
24. Ueda T, Goya T, Wakisaka S, Kinoshita K. Fenestrations of the middle cerebral artery associated with aneurysms. AJNR Am J Neuroradiol. 1984;5(5):639–40.
25. Tran-Dinh H. The accessory middle cerebral artery—a variant of the recurrent artery of Heubner (A. centralis longa)? Acta Anat (Basel). 1986;126(3):167–71.
26. Petrela M, Kurti X, Xhumari A, Leka L, Anastasi V, Vreto G. “Cross-over” duplication of middle cerebral artery, agenesis of internal carotid artery and saccular aneurysms. Acta Neurochir. 1987;84(1–2):73–6. https://doi.org/10.1007/BF01456356.
27. Kobari M, Ishihara N, Yunoki K, Togashi O, Sato S. Triplication of the middle cerebral artery associated with fenestration of the anterior cerebral artery. Keio J Med. 1988;37(4):429–33. https://doi.org/10.2302/kjm.37.429.
28. Takahashi S, Hoshino F, Uemura K, Takahashi A, Sakamoto K. Accessory middle cerebral artery: is it a variant form of the recurrent artery of Heubner? AJNR Am J Neuroradiol. 1989;10(3):563–8.
29. Mueller DP, Sato Y, Yuh WT. Accessory middle cerebral artery as a source of collateral blood flow. AJNR Am J Neuroradiol. 1991;12(6):1223–4.
30. Yamamoto H, Marubayashi T, Soejima T, Matsuoka S, Matsukado Y, Ushio Y. Accessory middle cerebral artery and duplication of middle cerebral artery—terminology, incidence, vascular etiology, and developmental significance. Neurol Med Chir (Tokyo). 1992;32(5):262–7. https://doi.org/10.2176/nmc.32.262.
31. Deruty R, Pelissou-Guyotat I, Mottolese C, Bognar L, Laharotte JC, Turjman F. Fenestration of the middle cerebral artery and aneurysm at the site of the fenestration. Neurol Res. 1992;14(5):421–4. https://doi.org/10.1080/01616412.1992.11740097.
32. Sanders WP, Sorek PA, Mehta BA. Fenestration of intracranial arteries with special attention to associated aneurysms and other anomalies. AJNR Am J Neuroradiol. 1993;14(3):675–80.
33. Nakamura H, Takada A, Hide T, Ushio Y. Fenestration of the middle cerebral artery associated with an aneurysm. Case report. Neurol Med Chir (Tokyo). 1994;34(8):555–7. https://doi.org/10.2176/nmc.34.555.
34. Tacconi L, Johnston FG, Symon L. Accessory middle cerebral artery. Case report. J Neurosurg. 1995;83(5):916–8. https://doi.org/10.3171/jns.1995.83.5.0916.
35. Koyama S, Kotani A, Sasaki J, Tazoe M, Tsubokawa T. Ruptured aneurysm at the origin of duplication of the middle cerebral artery. Case report. Neurol Med Chir (Tokyo). 1995;35(9):671–3. https://doi.org/10.2176/nmc.35.671.
36. Fujimura M, Seki H, Sugawara T, Tomichi N, Oku T, Higuchi H. Anomalous internal carotid artery-anterior cerebral artery anastomosis associated with fenestration and cerebral aneurysm. Neurol Med Chir (Tokyo). 1996;36(4):229–33. https://doi.org/10.2176/nmc.36.229.
37. Komiyama M, Nishikawa M, Yasui T. The accessory middle cerebral artery as a collateral blood supply. AJNR Am J Neuroradiol. 1997;18(3):587–90.
38. Bonasia S, Bojanowski M, Robert T. Embryology and variations of the recurrent artery of Heubner. Neuroradiology. 2020;62(4):427–37. https://doi.org/10.1007/s00234-020-02372-5.
39. Okahara M, Kiyosue H, Mori H, Tanoue S, Sainou M, Nagatomi H. Anatomic variations of the cerebral arteries and their embryology: a pictorial review. Eur Radiol. 2002;12(10):2548–61. https://doi.org/10.1007/s00330-001-1286-x.
40. Moffat DB. The embryology of the arteries of the brain. Ann R Coll Surg Engl. 1962;30:368–82.
41. Raybaud C. Normal and abnormal embryology and development of the intracranial vascular system. Neurosurg Clin N Am. 2010;21(3):399–426. https://doi.org/10.1016/j.nec.2010.03.011.
42. Yasargil MG, Fox JL, Ray MW. Anterior cerebral artery complex. In: Yasargil MG, editor. Microneurosurgery. New York: Thieme; 1975. p. 92–9.
43. Zurada A, Gielecki J, Tubbs RS, Loukas M, Maksymowicz W, Cohen-Gadol AA, et al. Three-dimensional morphometrical analysis of the M1 segment of the middle cerebral artery: potential clinical and neurosurgical implications. Clin Anat. 2011;24(1):34–46. https://doi.org/10.1002/ca.21051.
44. Guerin J, Gouaze A, Lazorthes G. The circle of Willis in children and the factors shaping it. Neurochirurgie. 1976;22(3):217–26.
45. Fauzi AA, Aji YK, Gunawan R, Suroto NS. Neuroangiography patterns and anomalies of middle cerebral artery: a systematic review. Surg Neurol Int. 2021;12:235. https://doi.org/10.25259/SNI_125_2021.
46. Delion M, Mercier P, Brassier G. Arteries and veins of the Sylvian fissure and insula: microsurgical anatomy. Adv Tech Stand Neurosurg. 2016;43:185–216. https://doi.org/10.1007/978-3-319-21359-0_7.
47. Umansky F, Dujovny M, Ausman JI, Diaz FG, Mirchandani HG. Anomalies and variations of the middle cerebral artery: a microanatomical study. Neurosurgery. 1988;22(6 Pt 1):1023–7. https://doi.org/10.1227/00006123-198806010-00008.
48. Gunnal SA, Farooqui MS, Wabale RN. Study of middle cerebral artery in human cadaveric brain. Ann Indian Acad Neurol. 2019;22(2):187–94. https://doi.org/10.4103/0972-2327.144289.
49. Srinivasa R, Shwetha K, Pravin S, Daisy S, Eilene B, Mathuriya SN, et al. Microsurgical anatomy of middle cerebral artery in northwest Indian population: a cadaveric brain dissection study. Asian J Neurosurg. 2021;16(4):785–91. https://doi.org/10.4103/ajns.ajns_189_21.
50. Yeh YS. Normal anatomy and aneurysms of the middle cerebral artery: a morphological, neuroradiological and clinical study. Neurol Res. 1984;6(1–2):41–8. https://doi.org/10.1080/01616412.1984.11739662.
51. Khatri R, Qureshi MA, Chaudhry MRA, Maud A, Vellipuram AR, Cruz-Flores S, et al. The angiographic anatomy of the sphenoidal segment of the middle cerebral artery and its relevance in mechanical thrombectomy. Interv Neurol. 2020;8(2–6):231–41. https://doi.org/10.1159/000502545.
52. Sadatomo T, Yuki K, Migita K, Imada Y, Kuwabara M, Kurisu K. Differences between middle cerebral artery bifurcations with normal anatomy and those with aneurysms. Neurosurg Rev. 2013;36(3):437–45. https://doi.org/10.1007/s10143-013-0450-5.
53. Ulm AJ, Fautheree GL, Tanriover N, Russo A, Albanese E, Rhoton AL Jr, et al. Microsurgical and angiographic anatomy of middle cerebral artery aneurysms: prevalence and significance of early branch aneurysms. Neurosurgery. 2008;62(5 Suppl 2):ONS344–52; discussion ONS52-3. https://doi.org/10.1227/01.neu.0000326018.22434.ed.
54. Donzelli R, Marinkovic S, Brigante L, de Divitiis O, Nikodijevic I, Schonauer C, et al. Territories of the perforating (lenticulostriate) branches of the middle cerebral artery. Surg Radiol Anat. 1998;20(6):393–8. https://doi.org/10.1007/BF01653128.
55. Grand W. Microsurgical anatomy of the proximal middle cerebral artery and the internal carotid artery bifurcation. Neurosurgery. 1980;7(3):215–8. https://doi.org/10.1227/00006123-198009000-00002.
56. Herman LH, Ostrowski AZ, Gurdjian ES. Perforating branches of the middle cerebral artery. An anatomical study. Arch Neurol. 1963;8:32–4. https://doi.org/10.1001/archneur.1963.00460010048005.

57. Kaplan HA. The lateral perforating branches of the anterior and middle cerebral arteries. J Neurosurg. 1965;23(3):305–10. https://doi.org/10.3171/jns.1965.23.3.0305.
58. Aydin IH, Takci E, Kadioglu HH, Kayaoglu CR, Tuzun Y. The variations of lenticulostriate arteries in the middle cerebral artery aneurysms. Acta Neurochir. 1996;138(5):555–9. https://doi.org/10.1007/BF01411176.
59. Kang HS, Han MH, Kwon BJ, Kwon OK, Kim SH, Chang KH. Evaluation of the lenticulostriate arteries with rotational angiography and 3D reconstruction. AJNR Am J Neuroradiol. 2005;26(2):306–12.
60. Ota T, Komiyama M. Embryological lateral striate artery variants : revised concept of recurrent artery of Heubner, the perforators to the anterior perforated substance and middle cerebral artery variants. Clin Neuroradiol. 2021;31(1):73–8. https://doi.org/10.1007/s00062-020-00978-z.
61. Vincentelli F, Caruso G, Andriamamonji C, Rabehanta P, Graziani N, Grisoli F. Modalities of origin of the middle cerebral artery. Incidence on the arrangement of the perforating branches. J Neurosurg Sci. 1990;34(1):7–11.
62. Marinkovic SV, Milisavljevic MM, Kovacevic MS, Stevic ZD. Perforating branches of the middle cerebral artery. Microanatomy and clinical significance of their intracerebral segments. Stroke. 1985;16(6):1022–9. https://doi.org/10.1161/01.str.16.6.1022.
63. Ciszek B, Aleksandrowicz R, Zabek M, Mazurowski W. Classification, topography and morphometry of the early branches of the middle cerebral artery. Folia Morphol (Warsz). 1996;55(4):229–30.
64. Vuillier F, Medeiros E, Moulin T, Cattin F, Bonneville JF, Tatu L. Main anatomical features of the M1 segment of the middle cerebral artery: a 3D time-of-flight magnetic resonance angiography at 3 T study. Surg Radiol Anat. 2008;30(6):509–14. https://doi.org/10.1007/s00276-008-0360-3.
65. Dimmick SJ, Faulder KC. Normal variants of the cerebral circulation at multidetector CT angiography. Radiographics. 2009;29(4):1027–43. https://doi.org/10.1148/rg.294085730.
66. Brzegowy P, Polak J, Wnuk J, Lasocha B, Walocha J, Popiela TJ. Middle cerebral artery anatomical variations and aneurysms: a retrospective study based on computed tomography angiography findings. Folia Morphol (Warsz). 2018;77(3):434–40. https://doi.org/10.5603/FM.a2017.0112.
67. Oo EM, Saw KEE, Oo HN, Than T, Thida K. Variable anatomy of the middle cerebral artery from its origin to the edge of the Sylvian fissure: a direct fresh brain study. ScientificWorldJournal. 2021;2021:6652676. https://doi.org/10.1155/2021/6652676.
68. De Long WB. Anatomy of the middle cerebral artery: the temporal branches. Stroke. 1973;4(3):412–8. https://doi.org/10.1161/01.str.4.3.412.
69. Ogeng'o JA, Njongo W, Hemed E, Obimbo MM, Gimongo J. Branching pattern of middle cerebral artery in an African population. Clin Anat. 2011;24(6):692–8. https://doi.org/10.1002/ca.21147.
70. Ring BA. Middle cerebral artery: anatomical and radiographic study. Acta Radiol. 1962;57:289–300. https://doi.org/10.3109/00016926209171758.
71. Cilliers K, Page BJ. Anatomy of the middle cerebral artery: cortical branches, branching pattern and anomalies. Turk Neurosurg. 2017;27(5):671–81. https://doi.org/10.5137/1019-5149.JTN.18127-16.1.
72. Christoforidis GA, Kollias SS, Valavanis A. Identification of the ascending middle cerebral artery branches on digital subtraction angiography. Neuroradiology. 2002;44(10):803–5. https://doi.org/10.1007/s00234-002-0804-0.
73. van der Zwan A, Hillen B, Tulleken CA, Dujovny M, Dragovic L. Variability of the territories of the major cerebral arteries. J Neurosurg. 1992;77(6):927–40. https://doi.org/10.3171/jns.1992.77.6.0927.
74. Uchino A, Saito N, Okada Y, Nakajima R. Duplicate origin and fenestration of the middle cerebral artery on MR angiography. Surg Radiol Anat. 2012;34(5):401–4. https://doi.org/10.1007/s00276-012-0936-9.
75. Uchino M, Kitajima S, Sakata Y, Honda M, Shibata I. Ruptured aneurysm at a duplicated middle cerebral artery with accessory middle cerebral artery. Acta Neurochir. 2004;146(12):1373–4.; discussion 5. https://doi.org/10.1007/s00701-004-0353-x.
76. Takahashi T, Suzuki S, Ohkuma H, Iwabuchi T. Aneurysm at a duplication of the middle cerebral artery. AJNR Am J Neuroradiol. 1994;15(6):1166–8.
77. Elmac I, Baltacioglu F, Ekinci G, Erzen C, Pamir N. Middle cerebral artery duplication associated with multiple intracranial aneurysms. Case report. J Neurosurg Sci 2001;45(4):232–234; discussion 4.
78. Nomura M, Yamashima T, Kita D, Kida S, Kajinami K, Yamashita J. Duplication of the middle cerebral artery associated with an unruptured aneurysm. Acta Neurochir. 2000;142(2):221–2. https://doi.org/10.1007/s007010050029.
79. Uchino A, Ito S, Kurita H, Tanaka M. Duplicated middle cerebral artery arising from the origin of the hyperplastic anterior choroidal artery that mimicked aneurysm on routine MR angiography. Neuroradiol J. 2016;29(2):106–9. https://doi.org/10.1177/1971400916633711.
80. Vuksanovic-Bozaric A, Vukcevic B, Abramovic M, Radunovic M. Duplication of the middle cerebral artery in a neonate: case report. Surg Radiol Anat. 2018;40(10):1173–5. https://doi.org/10.1007/s00276-018-2074-5.
81. LaBorde DV, Mason AM, Riley J, Dion JE, Barrow DL. Aneurysm of a duplicate middle cerebral artery. World Neurosurg. 2012;77(1):201e1–4. https://doi.org/10.1016/j.wneu.2011.03.038.
82. Imaizumi S, Onuma T, Motohashi O, Kameyama M, Ishii K. Unruptured carotid-duplicated middle cerebral artery aneurysm: case report. Surg Neurol. 2002;58(5):322–4. https://doi.org/10.1016/s0090-3019(02)00887-x.
83. Kaliaperumal C, Jain N, McKinstry CS, Choudhari KA. Carotid "trifurcation" aneurysm: surgical anatomy and management. Clin Neurol Neurosurg. 2007;109(6):538–40. https://doi.org/10.1016/j.clineuro.2007.04.006.
84. Perez J, Machado C, Scherle C, Hierro D. Duplicated middle cerebral artery. BMJ Case Rep. 2009;2009:bcr0620092035. https://doi.org/10.1136/bcr.06.2009.2035.
85. Miyamoto J, Mineura K. Unruptured middle cerebral artery aneurysm associated with a duplicated middle cerebral artery and a dolichoectasic anterior cerebral artery. J Stroke Cerebrovasc Dis. 2010;19(6):503–6. https://doi.org/10.1016/j.jstrokecerebrovasdis.2009.08.007.
86. Chang HY, Kim MS. Middle cerebral artery duplication : classification and clinical implications. J Korean Neurosurg Soc. 2011;49(2):102–6. https://doi.org/10.3340/jkns.2011.49.2.102.
87. Elsharkawy A, Ishii K, Niemela M, Kivisaari R, Lehto H, Hernesniemi J. Management of aneurysms at the origin of duplicated middle cerebral artery: series of four patients with review of the literature. World Neurosurg. 2013;80(6):e313–8. https://doi.org/10.1016/j.wneu.2012.10.004.
88. Gomez-Choco M, Valdueza JM. Duplication of the middle cerebral artery: a potential pitfall for transcranial Doppler examination. J Ultrasound Med. 2013;32(8):1517–9. https://doi.org/10.7863/ultra.32.8.1517.

89. Rennert J, Ullrich WO, Schuierer G. A rare case of supraclinoid internal carotid artery (ICA) fenestration in combination with duplication of the middle cerebral artery (MCA) originating from the ICA fenestration and an associated aneurysm. Clin Neuroradiol. 2013;23(2):133–6. https://doi.org/10.1007/s00062-011-0120-3.
90. Tutar NU, Tore HG, Kirbas I, Tarhan NC, Coskun M. Various origins of the duplicated middle cerebral artery. J Neuroimaging. 2008;18(4):448–50. https://doi.org/10.1111/j.1552-6569.2008.00254.x.
91. Stabler J. Two cases of accessory middle cerebral artery, including one with an aneurysm at its origin. Br J Radiol. 1970;43(509):314–8. https://doi.org/10.1259/0007-1285-43-509-314.
92. Handa J, Shimizu Y, Matsuda M, Handa H. The accessory middle cerebral artery: report of further two cases. Clin Radiol. 1970;21(4):415–6.
93. Watanabe T, Togo M. Accessory middle cerebral artery. Report of four cases. J Neurosurg. 1974;41(2):248–51. https://doi.org/10.3171/jns.1974.41.2.0248.
94. Komiyama M, Nakajima H, Nishikawa M, Yasui T. Middle cerebral artery variations: duplicated and accessory arteries. AJNR Am J Neuroradiol. 1998;19(1):45–9.
95. Marinkovic S, Milisavljevic M, Kovacevic M. Anatomical bases for surgical approach to the initial segment of the anterior cerebral artery. Microanatomy of Heubner's artery and perforating branches of the anterior cerebral artery. Surg Radiol Anat. 1986;8(1):7–18.
96. Lazar ML, Bland JE, North RR, Bringewald PR. Middle cerebral artery fenestration. Neurosurgery. 1980;6(3):297–300.
97. Nussbaum ES, Defillo A, Janjua TM, Nussbaum LA. Fenestration of the middle cerebral artery with an associated ruptured aneurysm. J Clin Neurosci. 2009;16(6):845–7. https://doi.org/10.1016/j.jocn.2008.08.039.
98. Sim KB, Lee CS, Park JC, Huh JS. Cerebral aneurysm in the long fenestration at the middle portion of m1 segment. J Korean Neurosurg Soc. 2010;48(5):434–7. https://doi.org/10.3340/jkns.2010.48.5.434.
99. Tabuchi S, Yoshioka H. Ruptured aneurysm at the fenestration of the middle cerebral artery detected by magnetic resonance angiography in a patient with systemic lupus erythematosus and renal failure: a case report. J Med Case Rep. 2014;8:30. https://doi.org/10.1186/1752-1947-8-30.
100. Jeong SK, Kwak HS, Cho YI. Middle cerebral artery fenestration in patients with cerebral ischemia. J Neurol Sci. 2008;275(1–2):181–4. https://doi.org/10.1016/j.jns.2008.07.037.
101. Uchino A, Takase Y, Nomiyama K, Egashira R, Kudo S. Fenestration of the middle cerebral artery detected by MR angiography. Magn Reson Med Sci. 2006;5(1):51–5. https://doi.org/10.2463/mrms.5.51.
102. Gailloud P, Albayram S, Fasel JH, Beauchamp NJ, Murphy KJ. Angiographic and embryologic considerations in five cases of middle cerebral artery fenestration. AJNR Am J Neuroradiol. 2002;23(4):585–7.
103. Liu HM, Lai DM, Tu YK, Wang YH. Aneurysms in twig-like middle cerebral artery. Cerebrovasc Dis. 2005;20(1):1–5. https://doi.org/10.1159/000086119.
104. Seo BS, Lee YS, Lee HG, Lee JH, Ryu KY, Kang DG. Clinical and radiological features of patients with aplastic or twiglike middle cerebral arteries. Neurosurgery. 2012;70(6):1472–80; discussion 80. https://doi.org/10.1227/NEU.0b013e318246a510.
105. Cekirge HS, Peynircioglu B, Saatci I. Endovascular treatment of an "anterior cerebral artery" aneurysm in a patient with "embryonic unfused middle cerebral artery" anomaly: a case report. Neuroradiology. 2005;47(9):690–4. https://doi.org/10.1007/s00234-005-1407-3.
106. Akkan K, Ucar M, Kilic K, Celtikci E, Ilgit E, Onal B. Unfused or twig-like middle cerebral artery. Eur J Radiol. 2015;84(10):2013–8. https://doi.org/10.1016/j.ejrad.2015.06.012.
107. Uchiyama N. Anomalies of the middle cerebral artery. Neurol Med Chir (Tokyo). 2017;57(6):261–6. https://doi.org/10.2176/nmc.ra.2017-0043.
108. Amagasaki K, Yagishita T, Kawataki T, Kase M, Nukui H. Middle cerebral artery aplasia associated with an aneurysm of the proximal anterior cerebral artery. Acta Neurochir. 1998;140(12):1313–4. https://doi.org/10.1007/s007010050256.
109. Hori E, Kurosaki K, Matsumura N, Yamatani K, Kusunose M, Kuwayama N, et al. Multiple aneurysms arising from the origin of a duplication of the middle cerebral artery. J Clin Neurosci. 2005;12(7):812–5. https://doi.org/10.1016/j.jocn.2004.08.033.
110. Otani N, Nawashiro H, Tsuzuki N, Osada H, Suzuki T, Shima K, et al. A ruptured internal carotid artery aneurysm located at the origin of the duplicated middle cerebral artery associated with accessory middle cerebral artery and middle cerebral artery aplasia. Surg Neurol Int. 2010;1:51. https://doi.org/10.4103/2152-7806.69378.
111. Grellier P, Roche JL, Duplay J. Radio-anatomical study of the main trunk of the middle cerebral artery (author's transl). Neurochirurgie. 1978;24(4):227–33.
112. Shapiro M, Raz E, Nossek E, Chancellor B, Ishida K, Nelson PK. Neuroanatomy of the middle cerebral artery: implications for thrombectomy. J Neurointerv Surg. 2020;12(8):768–73. https://doi.org/10.1136/neurintsurg-2019-015782.

Embryology and Anatomy of the Posterior Communicating Artery and Basilar Artery

Sara Bonasia and Thomas Robert

The basilar artery (BA) and the posterior communicating artery (PComA) represent the upper part of the posterior circle of Willis. Their embryological development is strictly linked and depends on two systems: the primitive carotid system through its caudal division, and the vertebro-basilar system which develops from the fusion of the longitudinal neural arteries (LNAs).

The first description of the circle of Willis is attributed to Thomas Willis in 1681, however our modern comprehension of this circle and its variant is mostly based on the embryo's dissection of D. Padget in 1948 [1, 2]. The contribution of her embryo's dissection permitted a most comprehensive interpretation of the future anatomical dissection studies and of the angiographic analysis of this circle [3–7].

In this chapter, we will describe the principal embryological events that determine the development, as well as the possible adult variants related to these two arteries. Even if the posterior cerebral artery (PCA) and the superior cerebellar artery (SCA) will be described in their respective chapters, we will report some crucial information concerning these arteries according to their embryological affinity with the PComA and the BA.

Embryology

The formation of the PComA and the BA is the result of the joining of two systems: the primitive carotid system (its caudal division) and the vertebrobasilar system. Modifications that involve these two systems during the embryological life will determine the final configuration of the PComA, the P1 segment, and the BA, and are represented in Table 1.

According to Lasjaunias et al., the formation of the BA spreads in two directions: longitudinal and axial [7]. The longitudinal system corresponds to the midline fusion of the paired longitudinal neural arteries (LNAs) in stage II of Padget (embryo of 5–6 mm) and will determine the adult configuration of the proximal BA; the axial system consists in the fusion of the caudal division of the ICA with the contralateral ICA and with the mid-basilar artery; it will determine the configuration of the PComA, the P1 segment, the distal BA, and the SCA [2]. The "pivotal point" between these two systems is represented by the trigeminal artery (TA), which represents the most cranial segmental artery, as well as the point of transition between the cranial cerebral vasculature and the simplified caudal fusion system of the spinal cord.

In the following paragraphs, we will describe how the longitudinal and axial systems influence respectively the formation of the caudal and cranial part of the BA as well as the PComA and the cerebellar arteries.

The Caudal Division of the Internal Carotid Artery (Axial System)

In stage I of Padget (embryos of 4–5 mm), the two first aortic arches start their regression, while the third aortic arch and the dorsal aorta cranial to it give origin to the primitive internal carotid artery [2, 7, 8]. In the 4–5 mm embryos, the primitive ICA bifurcation is visible with its two divisions: cranial and caudal that will become respectively in adult the anterior cerebral artery (ACA) and the posterior communicating artery (PComA).

The ICA, cranial to the posterior communicating artery origin, derivates from the anterior division of the ICA and not from the ICA itself. Consequently, the primitive ICA bifurcation is at the origin of the PComA and not at the origin of the middle cerebral artery (MCA). According to this

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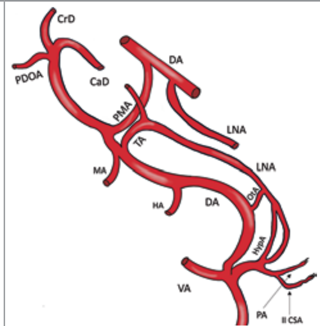
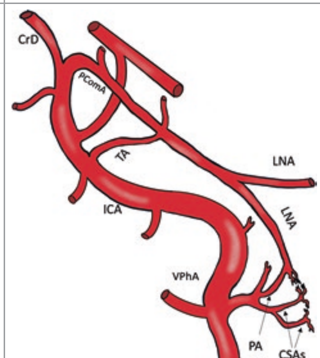
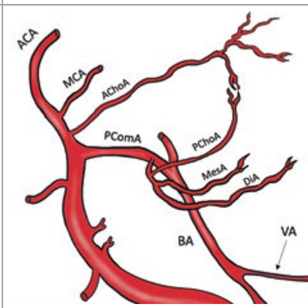
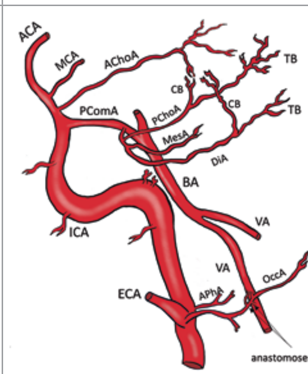
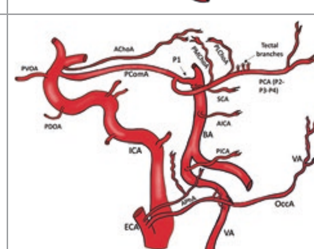
Table 1 Major embryological changes in the formation of the vertebro-basilar system

Stage	Embryo size (mm)	Major evolutions	Graphic Illustration
I	4–5	– Paired longitudinal neural arteries (LNAs) visible – Carotido-vertebral anastomoses visible: Trigeminal (TA), otic (OtA), hypoglossal (HypA), proatlantal (PA), and second cervical segmental artery (II CSA) – Primitive ICA bifurcation visible into caudal and cranial divisions (CaD, CrD) – PComA and BA not formed	
II	5–6	– Formation of the PComA – Regression of otic and hypoglossal arteries – Midline fusion of LNAs to form the basilar artery (BA)	
III	7–12	– Basilar artery (BA) completely formed – PComA completely formed – Origin from the distal PComA of a diencephalon-mesencephalic trunk (DiA-MesA). PChoA arising from the common trunk and anastomoses with the AChoA – Regression of trigeminal and proatlantal arteries – Formation of the vertebral artery VA by transverse anastomoses between the intersegmental arteries	
IV	13–15	– Development of a choroidal (CB) and telencephalic branch (TB) from the PChoA and the diencephalic artery (DiA) – Remodeling of the BA – Migration of OccA origin on the external carotid artery (ECA) – Persistence of the anastomoses between OccA and VA	
V	16–18	– The telencephalic branch of the PChoA forms the main PCA trunk – The choroidal branches of the PChoA and of the diencephalic artery form the PLChoA and PMChoA – Complete formation of the VA – Complete formation of the OccA (remnant of the proatlantal artery) – Complete formation of the ascending pharyngeal artery (APhA, remnant of the hypoglossal artery) – SCA, AICA, and PICA are visible	

theory proposed by Lasjaunias, the internal carotid artery can be embryologically divided into seven different segments, which are described in chapter "Embryologic Development of the Internal Carotid Artery" [9].

The segment of our interest for this chapter is the seventh one, which extends between the anastomotic point of the primitive ventral ophthalmic artery (PVOA) and the primitive carotid bifurcation. In the adult, this segment corresponds to the supraclinoid ICA from the origin of the ophthalmic artery (OA) to the origin of the PComA.

Even if this latter is the most accepted theory nowadays, Gailloud et al. proposed a modified ICA division in eight segments instead of seven basing on a case of "segmental agenesis of the ICA distal to the PComA" [10]. According to this classification, the seventh and eighth segments are divided by PComA, in which the eighth segment terminates at the adult ICA bifurcation [10]. This makes the PComA to have a similar role to the embryonic carotid-vertebral anastomoses. However, different form of anomalies, such as the midbasilar agenesis, which are representative of the failure of the axial fusion system, demonstrate that the PComA has a completely different role into the supply of the BA, if compared to a persistent TA. Moreover, this theory cannot be accepted because it does not explain the co-existing abnormalities present in the ICA, PComA, P1, and distal BA.

During stage II of Padget (embryos of 5–6 mm), the caudal division of the primitive internal carotid artery anastomoses with the cranial part of the LNA at the level of the mesencephalon, and usually caudal to the origin of the SCA, forming thereby the PComA [2]. The timing and level of fusion between the two systems is principally influenced by the timing of regression of the TA, since it determines the site of flow reversal in the distal basilar artery system. The later the TA involves, the more caudal will be the fusion [7]. According to Lasjaunias et al., the fusion occurs in 88% of cases at the interpeduncular fossa, in 10% at the pons level, and in 2% at the level of mammillary body [7].

During stage III of Padget (embryos of 7–12 mm), the caudal division of the ICA gives off two large branches in front of the oculomotor nerve: a diencephalic and a mesencephalic branch [2]. From the common trunk of these branches, the posterior choroidal artery (PChoA) develops and reaches the atrium to anastomose with the anterior choroidal artery (AChoA) from the cranial division [7]. At this stage, the AChoA origins from the ICA cranial division, together with the ACA. The AChoA and the PChoA supply the choroidal plexus and anastomose to each other at the interventricular foramen, forming thereby the choroidal circle. They present, moreover, a telencephalic supply, like the olfactory cortex, which will be gradually annexed by the posterior cerebral artery (PCA). These changes will give some relief to the cranial ICA division, facilitating the development of the middle cerebral artery (MCA).

The portion of the caudal segment of the ICA that remains unfused gives origin to the SCA. This artery will supply the region of the mesencephalon and of the trochlear nerve. The point of fusion of the caudal segment of the ICA determines if the SCA arises from the BA or from the first segment of the posterior cerebral artery (P1 segment).

During stage IV and V of Padget (embryos of 12–18 mm), the diencephalo-mesencephalic trunk from the caudal ICA division increases its development, by progressive annexation of some cortical territories of the ACA and AChoA. Both the PChoA and the diencephalic artery develop a choroidal and a telencephalic branch. The main trunk of the PChoA with its telencephalic branch will constitute the main posterior cerebral artery stem (P2, P3, P4 segments) and will take in charge most of the cortical territories previously supplied by the cranial division. Its choroidal branch will become the posterior lateral choroidal artery (PLChoA). On the other side, the choroidal branch of the diencephalic artery will become the posterior medial choroidal artery (PMChoA) [7]. The mesencephalic artery corresponds to the tectal territorial branch from the PCA [7]. In this stage the SCA is recognizable with its mesial division coursing caudal to the trochlear nerve and a lateral one extending over the cerebellar lip.

The Longitudinal Neural Arteries (Longitudinal System)

The development of the vertebro-basilar system starts during stage I of Padget (embryos of 4–5 mm), with the appearance of a paired vascular plexus in the ventral wall of the hindbrain called "longitudinal neural arteries" [2, 11]. At this stage, the vertebral arteries do not exist yet and the LNAs are supplied by different carotido-vertebral anastomoses. The main supply of the LNAs is provided cranially by the TA and caudally by the first primitive intersegmental cervical arteries including the proatlantal artery. This supply is reinforced by the otic artery at the level of the acoustic nerve, and by the hypoglossal artery in the region of the hypoglossal nerve [7, 12].

During stage II of Padget (embryos of 5–6 mm), the caudal division of the primitive internal carotid artery anastomoses with the cranial part of the LNA at the level of the mesencephalon, forming thereby the PComA [2].

This anastomosis determines crucial flow modifications in the LNAs that will lead to the progressive regression of the carotido-vertebral anastomoses. The first two arteries that regress are the otic and the hypoglossal arteries, followed by the trigeminal and hypoglossal arteries. At the same time, the two LNAs come closer to the midline to fuse together in a craniocaudal direction [2, 7, 12].

At stage III of Padget (embryos of 7–12 mm), the basilar artery, as well as the posterior communicating artery, is completely formed by fusion of the two LNAs. The formation of transverse anastomoses among the first six cervical intersegmental arteries, and the consequent obliteration of their aortic ends, determines the formation of the vertebral arteries (VA). The distal portion of the proatlantal intersegmental artery persists as the transverse suboccipital segment of the vertebral artery [11].

The caudal supply of the vertebral artery is now provided by the seventh segmental artery, that keeps its connection with the dorsal aorta and corresponds to the future subclavian artery [2, 7, 8].

Stage IV of Padget (embryos of 12–14 mm) is characterized by the definitive remodeling of the vertebro-basilar system in its definitive configuration [2]. This determines the definitive change from the exclusive carotid supply of all cerebral arteries to the vertebral predominance in the hindbrain region [2].

At stage V of Padget (embryos of 16–18 mm), only the first segment (P1) of the posterior cerebral artery is developed [2]. The branches of the BA and VA start to be recognizable at this stage. While the SCA belongs to the caudal ICA system, the AICA and PICA arise from the caudal BA. The primitive stem of the AICA arises at the level of the vestibulo-cochlear nerve and terminates into the choroidal plexus of the fourth ventricle. Caudal to this stem, among several branches of the VA that pass between the rootlets of the vagus and accessory nerves, one vertebral branch that courses cranially along the medulla, could be identified as the future posterior inferior cerebellar artery (PICA).

Stage VI of Padget (embryos of 20–24 mm) is characterized by the complete formation of the circle of Willis, which is completed after the development of the anterior communicating artery, when the embryo is approximately 24 mm.

Anatomy

The embryological events previously described determine the formation of cranial part of the so-called posterior circulation, including the PComA, the P1 segment, the basilar artery, the PCA, and the SCA, including their branches.

In this chapter, we will describe the anatomical features of these arteries, except the PCA and the SCA, which will be described in detail in their respective chapter.

Posterior Communicating Artery

Possible Configurations

The PComA represents the adult connection between the internal carotid artery system and the vertebro-basilar system and contributes to the formation of the circle of Willis.

Referring to the configuration of the PComA and the P1 segment, the anatomical dissection studies described different types of circle of Willis [3–5, 13]:

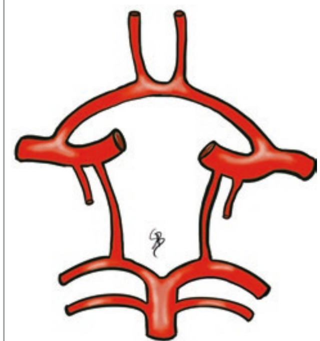
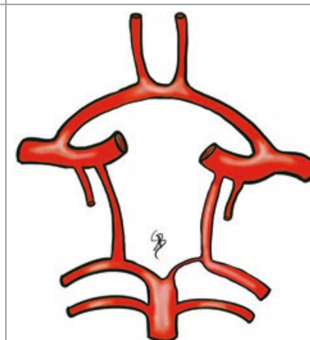
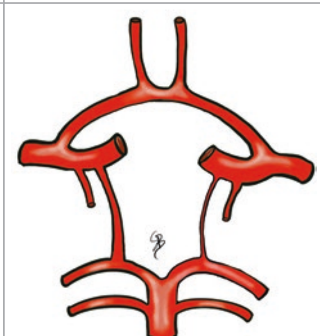
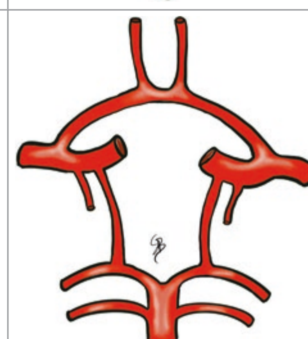
- An "adult" or "normal" configuration, in which the P1 segment has a diameter larger than the PComA, that is not hypoplastic (diameter superior to 1 mm) [6, 14]. This configuration reflects the major contribution of the vertebrobasilar system to the PCA flow, and its frequency has been reported to be between 16% and 76% [4–6].
- An "abnormal" configuration, reported with a frequency between 23% and 48%, that includes [4–6]:
- A "fetal" configuration, in which the P1 diameter is smaller than the PComA and the blood supply to the PCA is mainly provided by the ICA. In this type, the PCA keeps its embryonic origin from the ICA. The prevalence of this type is described to be 22% by Seaki and Rhoton, with a unilateral involvement in 20% and bilateral in 2%; Alpers and Brassier described a slight lower prevalence of this variant as about 14% [4, 6, 15];
- A "hypoplastic" configuration of the PComA, if the vessel has a diameter of 1 mm or lower [4, 6]. In this case, the diameter of the PComA is inferior to the P1 segment, like in the normal configuration, but the vessel is also hypoplastic (diameter < 1 mm). The presence of a hypoplastic PComA has been described with a frequency between 22–40% of cases (32% of cases by Saeki and Rhoton et al., in 40% of cases by Brassier et al. and in 22% of cases by Alpers et al.); this variant was unilateral in 26% and bilateral in 6% of cases in the Saeki's series [4, 6, 15]. This is the most common form of "abnormal" circle of Willis [3–5, 16].
- A "transitional" configuration, in which the PComA and the P1 segment have the same diameter and give an equal contribution to the PCA [2, 5, 13, 14, 17, 18]. This variant has been described in 7–20% of cases and some reports have suggested that its frequency is lower in older people as well as in older foeti [5, 14, 17, 18].

These configurations refer to a unilateral aspect, thus they can occur in combination with a different configuration on the contralateral side. Indeed, Seak and Rhoton described an incidence of 8% of cases with a hypoplastic PComA on one side and a fetal one on the other side [6]. On the other hand, the complete absence of a PComA has been described very rarely, more often unilaterally, with an incidence between 0.6% and 3% [4, 6, 13, 15, 18–20]. The data identified by the different authors about the PComA variants are represented in Table 2.

Course and Origin

The PComA has a constant origin from the posterior aspect of the supraclinoidal ICA and courses backward and slightly medially to join the P1 segment in the interpeduncular fossa

Table 2 Possible configuration of the PComA and the circle of Willis

Configuration	Characteristic	Illustration
Adult or normal	P1 larger than PComA, that is not hypoplastic (diameter superior to 1 mm)	
Fetal	P1 smaller than PComA. Blood supply to PCA mainly provided by the ICA. P1 hypoplastic or absent	
Hypoplastic	PComA smaller than P1, PComA is also hypoplastic (diameter < 1 mm)	
Transitional	PComA and the P1 segment with same diameter	

[6]. The origin is usually located on the postero-medial aspect of the ICA and has been only rarely described on the postero-lateral surface of the ICA [6, 19].

Bisaria et al. also described an isolated case of PComA origin from the ophthalmic artery (OA), with the PComA coursing into the optic canal with a recurrent trajectory [19]. We analyzed the case in detail and identified some criticisms that do not permit us to consider it as a real OA origin of the PComA. The first one is that such an origin has been previously described only in association with ICA agenesis and interpreted as "ophthalmic artery origin from the PComA" [21–23]. The fact that in the cadaveric case reported by Bisaria et al., the left ICA is not shown, does not allow to exclude the associated presence of an ICA agenesis [19].

Moreover, we are not able to find a clear embryological explication for an OA origin of the PComA, and the absence of other cases described suggests us that this case has been probably misinterpreted.

In its classic configuration, the PComA courses superiorly and medially to the oculomotor nerve. However, this relationship can change in case of fetal or tortuous configuration of the PComA [24, 25].

Considering the high variability of PComA configuration, it is difficult to determine a length or precise diameter of this artery. The average length is 15 mm with a range between 7 and 22 mm [16, 19, 20, 26, 27]. The major lengths are reported in case of fetal configuration [24]. Its average diameter is described as 1.6 mm and is reported to be slightly larger at the ICA origin than at the P1 junction, even if this difference is rare great as 1 mm [6, 16, 19, 20]. This mild enlargement of the PComA at its origin on the ICA is known as "PComA infundibular dilatation," and it is still the object of debate because of its predisposition to the formation of aneurysms.

Duplicated/Fenestrated PComA

The duplication or fenestration of the PComA has been described in literature very rarely [4, 7, 20, 28]. In the literature, there are many definitions of these two entities. In this chapter, we consider as "fenestration" of an artery the presence of two different lumen of the same vessel (with the same embryological origin) and as "duplication" the presence of two vessels with different embryological origin [7]. In the case of the PComA, the incidence of its fenestration has been described in the literature as 0.58%, and of duplication as even rarer [19, 20]. Bisaria et al. also reported an isolated case of PComA triplication [19]. The PComA fenestration and duplication can be explained by PComA-P1 junctional variations that happen during the axial fusion process [7]. However, a misunderstanding can happen in case of pseudo-duplication of the PComA. In these cases, a branch of the AChoA can keep a portion of its embryonic vascular territory, giving the appearance of a duplicated PComA and PCA with same origin from the ICA [7]. At the same time, it is also possible that the PComA and the AChoA share their origin from the ICA, giving the impression of a PComA duplication [29]. The case described by Bisaria can be interpreted in these terms [19].

P1 Segment

Even if the anatomical details of the posterior cerebral artery will be presented in the dedicated chapter, some information concerning the P1 segment should be mentioned in this contest because of its embryological implication.

The P1 segment, also known as "pre-communicant," "circular," or "mesencephalic," refers to the part of the PCA between the BA bifurcation and the PComA.

The length of this segment is longer in case of fetal pattern of the PComA, as well as in case of PComA hypoplasia [6, 15, 24]. The average P1 length has been described as 9 mm in case of fetal configuration of the PComA, and as 7 mm in case of adult configuration (range of 3–14 mm) [17, 24]. The average P1 diameter is 2.5 mm with a range of 1–4 mm and is usually constant throughout its length [6, 17].

Even if the hypoplastic configuration of the P1 segment is relatively frequent in association with a fetal PComA configuration, its complete absence has been described rarely and its incidence is estimated to be 1% unilaterally and 0.5% bilaterally [17, 18].

Other forms of anomalies related to the P1 segment are duplications or fenestrations. In case of fenestration, there is a duplicated origin of the P1 segment, but the two lumina fuse together at the connection with the PComA. The incidence of these variants has been described as 0.25% by Krayenbühl and Yasargil (1957), and similar cases were also previously described by Gordon-Shaw in 1910 [30, 31].

A real duplication of the P1 segment, with consequent duplication of the PCA, is considered even rarer and was firstly described by Windle in 1888, even if this case was later interpreted as an early bifurcation of the artery [32]. Another case was later reported by Bisaria in 1984, who reported a true bifurcation with an aneurysm located at the origin of the larger trunk [19].

Basilar Artery

Origin, Course, and Possible Configurations

The basilar artery represents the major vascular trunk of the posterior circulation and is formed in the adult by fusion of the two vertebral arteries at the ponto-medullary junction. The average length of the artery is 32 mm, with a range of 15–40 mm [6]. Its diameter, that is usually wider at its cranial bifurcation, is described as 4.1 mm below the SCA and as 4.5 mm above the SCA [6].

It courses along the ventral surface of the pons to bifurcate near the ponto-mesencephalic junction. Its bifurcation can be found caudally 1.3 mm below the ponto-mesencephalic junction and rostrally at the mammillary bodies. In fact, although fusion occurs in 72–88% in the interpeduncular fossa, it can be at the upper pons in 4–10% of cases or at the mammillary bodies level in 2–4% [6, 7, 33].

The different changes that happen during the embryological life will influence the point of fusion and the configuration of the BA tip and bifurcation. Usually, the point of fusion between the BA and the caudal division of the ICA is distal to the embryonic SCA origin, at the ponto-mesencephalic sulcus. Thus, the portion of the caudal ICA between the PCA origin and the SCA will form the P1 segment. This location is however related to the timing of regression of the TAs.

As mentioned in the preceding paragraph about embryology, a mesencephalic artery with a predominant tectal supply arising from the embryonic P1 segment (from the caudal segment of the ICA) will acquire the distal cortical supply of the AChoA becoming the posterior cerebral artery. At the same time, the BA will be formed after the fusion of the LNAs. The fusion between the caudal ICA and the BA will determine the regression of the trigeminal arteries and the consequent flow reversion into the BA with the acquisition of the posterior cerebral vascularization by the VA. Indeed, the later the TAs involute, the lower will be the role of the vertebral flow in the supply of the mesencephalic region, and the longer the mesencephalic supply will be granted by the ICA [7, 34]. The angiographical landmark that allows to distinguish the different form of fusion is the site of origin of the SCA.

Lasjaunias et al. described three possible configurations of the basilar tip, which are represented in Fig. 1 [7]:

1. Symmetrical cranial: The SCA origins from the BA bilaterally and the main supply of mesencephalon comes from the vertebro-basilar system. Campos et al. found an incidence of this variant of 30.4% [34];
2. Symmetrical caudal: The SCA origins from the P1 segment bilaterally. In such a situation, the carotid contribution to the mesencephalic supply remains dominant. The incidence is estimated to be 26.1% [34];
3. Asymmetrical: The SCA origins on one side from the P1 segment and on the other side from the BA (this can happen also in case of P1 absence). This configuration is the most common and its incidence is estimated to be 43.5% [34].

The angiographic analysis of Campos et al. identified however a higher incidence of caudal and asymmetrical fusion among patients with basilar tip aneurysms in comparison to patients without basilar tip aneurysms, suggesting that these two configurations are triggers for aneurysmal formation [34]. Moreover, the disposition of the basilar tip has an influence on the disposition of the P1 and BA perforators, which will be explained in detail in the dedicated chapter.

Fenestration/Duplication of the Basilar Artery

Fenestration of a cerebral artery refers to a division of the lumen of an artery, resulting in two distinct endothelium-lined channels, which may or may not share their adventitial layer [35]. This variant, in the case of the BA, is considered to be the expression of the failure of the longitudinal system fusion, since it derives from the LNAs fusion failure.

On the contrary, the presence of a fenestration at the vertebrobasilar junction or of the vertebral artery is considered to be the result of a vascular flow modification between two segmental arteries and the longitudinal anastomoses among them [7]. For this reason, the origin of these two entities will be considered separately and in this chapter, we will treat only the anomalies related to the BA.

Basilar artery fenestration represents the second most common site of fenestration, after the anterior communicating artery [36–38]. Its frequency is 1–5.26% in post-mortem

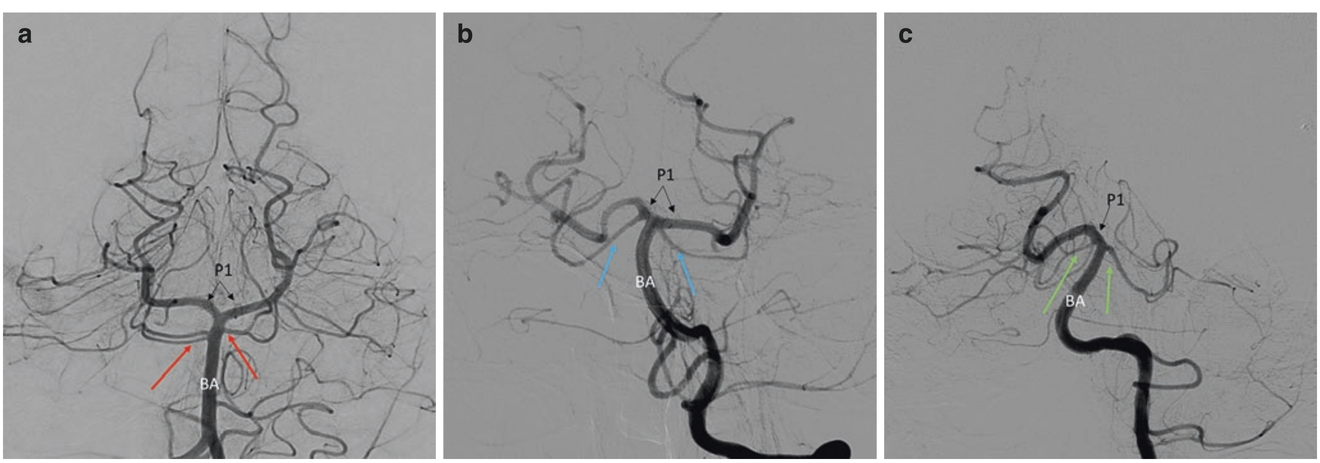


Fig. 1 Basilar tip configuration. Figures (**a**, **b**, and **c**) show respectively cases of symmetrical cranial, symmetrical caudal and asymmetrical configuration of the basilar tip. In figure a, the superior cerebellar artery (SCA, red arrows) originates on both sides from the basilar artery (BA). On the right side, it appears doubled. In figure (**b**), the SCA (blue arrows) arises on both sides from the P1 segment of the posterior cerebral artery. In figure (**c**), the SCA (green arrows) arises on the right side from the P1 segment and on the left side from the BA. There is a concomitant absence of the P1 segment on the left side

studies, 0.3–0.6% in conventional angiography, and 1–2.07% in magnetic resonance angiography series [35, 39–46]. The incidence of BA fenestration has been found as slightly higher (9%) in patient series with subarachnoid hemorrhage (SAH), even if the origin of the hemorrhage was not clearly associated with the aneurysm on the fenestration [40].

The average length of a BA fenestration is variable (between 2 and 15 mm) and few authors also described a fenestration of the entire BA, with a frequency of 0.3% [39, 43, 46–50]. One of these cases, described by Uchino et al., was an incidental finding during MRI investigation of median cleft face syndrome with nasopharyngeal teratoma [49]. This case can be considered as an extreme fenestration of the BA, since a prominent connection in the distal and proximal ends of the BA persists. On the other side, in the case described by Goldstein et al., the two unfused BA course distant to each other and are connected only through small superior and inferior arteries, suggesting a real duplication of the BA [48]. A similar case was also described by Hoh et al., even if the absence of angiographic images does not allow to establish the presence of anastomoses between the two arteries [50].

Sogawa et al. also described three cases of double fenestration, with two cases of adjacent fenestrations and one case with separate fenestrations in the distal and proximal BA segments [46].

The BA fenestration has been classified in literature according to different criteria:

- *Location*: the BA can be classified as proximal (lower third), mid, or distal according to the BA segment in which it occurs. The most common site of occurrence of this variant is its proximal part, near the vertebrobasilar junction, reflecting the craniocaudal direction of LNAs fusion [38, 45, 46, 51]. A middle and distal fenestration was rarely described [36, 38, 44, 46, 51].
- *Morphology*: Uchino et al. described two principal configurations of the fenestration and named it slit-like and convex-lent [44]. The vascular appearance of the slit-like shape is short, and its opening is minimal. On the other side, the convex-lent shape is long, and its opening is wide.
- *Origin of the AICA:* The relationship between the BA fenestration and the origin of the AICA was studied by Gao et al., after the observation of Uchino et al. that the AICA frequently arises from the fenestration [38, 45]. Gao et al. described a four-type classification:
  - In type I, the fenestration is proximal to the AICA origin.
  - In type II, the AICA origins bilaterally from the fenestrated segment.
  - In type III, the AICA origins only unilaterally from the fenestrated segment.
  - In type IV, the fenestration is distal to the AICA origin.

The result of its analysis showed that the AICA is inclined to originate from the BA fenestration, especially in the case of convex-lens configuration [38].

Basilar Artery Segmental Agenesis

Agenesis of the BA could be defined as the absence or regression of a portion of the BA. The absence of a segment of this artery can determine the interruption of the link between the BA trunk, the ICA, and the vertebral arteries. The interruption of the continuity between these systems creates the need for flow compensation into the different BA segments. This compensation can be provided by the carotid system through the PComA, by a persistent carotido-vertebral anastomosis, or by a rete mirabile.

Only few cases of BA agenesis are described in the literature, and most of the cases of rete compensation were described in association with a carotid rete mirabile [10, 50, 52–58]. A clinical case of BA agenesis is also reported in Fig. 2. Because of the limited number of cases described, is it difficult to classify the different forms of BA agenesis, however we identified three forms of compensation in the cases described in the literature:

1. *ICA compensation through the circle of Willis (PComA):* the first case with this type of compensation was described by Lasjaunias et al. in 1979; in this case, the two vertebral arteries gave origin to the two PICAs and terminated in a fine network of vessels [53]. The distal basilar artery received a flow compensation through a fetal left PComA, which also gave origin to both the PCAs. The upper BA also gave origin to the SCAs and the AICAs. Other two cases were described by Burger et al. in 2004 [59]. In their first case, the DSA showed that VAs continued both sides as PICAs, without any connection with the BA. The right PComA supplied the BA with a retrograde opacification of the trunk until to the AICAs origin. The parenchymal territory of the right PCA was however supplied by an enlarged AChoA, without any connection with the PComA and the BA. On the left side, the PCA territory was supplied by a fetal PComA. In the second case, the PCA territory was supplied bilaterally by a fetal PComA. In these two cases, no persistent carotido-vertebral anastomosis was observed.
2. *ICA compensation through persistent carotido-vertebral anastomosis*: Two cases of BA agenesis associated with a persistent TA have been reported [50, 59]. In both cases, the PCA territory is filled by a fetal PComA, without any connection with the BA. At the same time, the VAs terminates bilaterally as PICAs. The BA appears as an isolated trunk and is vascularized only by a persistent TA. The

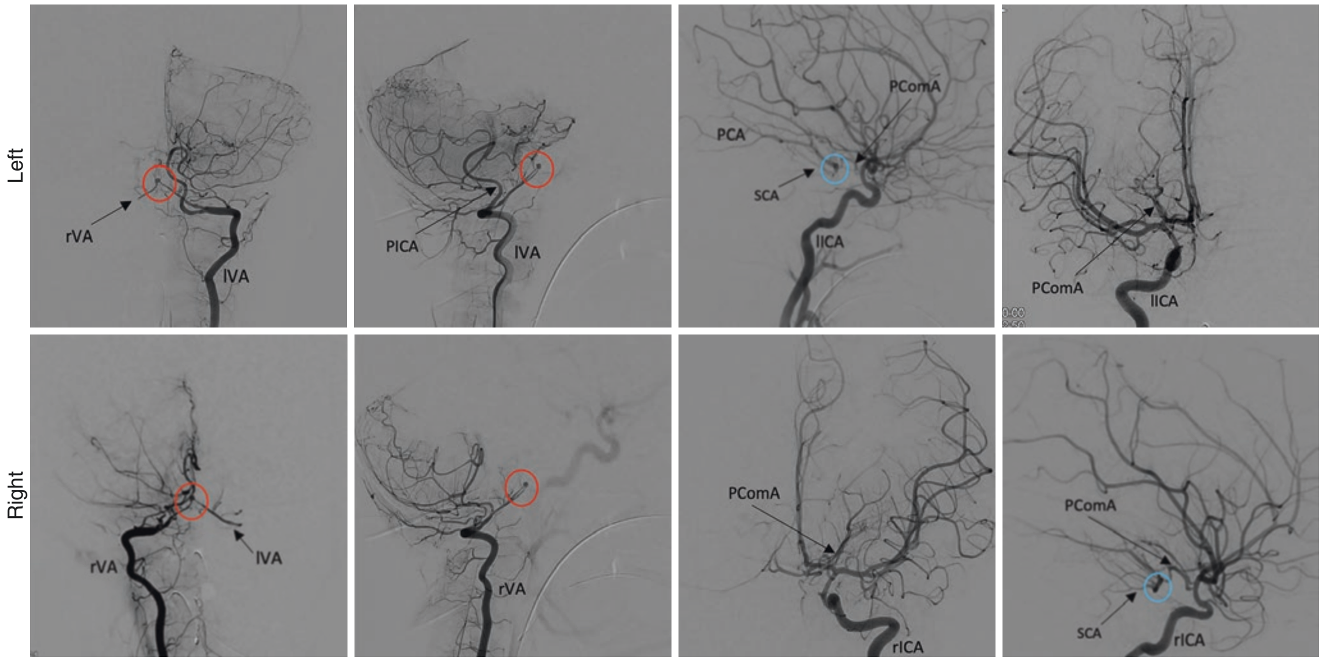


Fig. 2 Case of BA agenesis. The figure shows a 6-projection DSA of a case of basilar artery (BA) agenesis. In the right and left injection of the vertebral artery (VA), both the arteries give origin to the postero-inferior cerebellar arteries (PICAs), which supplies with arterio-arterial anastomoses the vascular territory of the antero-inferior cerebellar arteries (AICAs). The VAs join at the midline to form the proximal part of the BA (red circle). The internal carotid artery (ICA) injections on both sides show that the ICA supplies the proximal part of the BA trunk (blue circle) through the posterior communicating artery (PComA). The superior cerebellar artery (SCA) arises from the proximal trunk of the BA

AICA arose in one case from the isolated BA trunk and in the other one from a common trunk with the PICA (from VA). The SCA origin was identified in both cases on the isolated BA trunk.

3. *Compensation through a rete mirabile:* The vertebral rete mirabile is a very rare anatomical variant and it has been always described in association with a carotid rete mirabile [52, 54–58]. It mostly consists in a vertebral artery segmental agenesis, in which the flow compensation of the vertebro-basilar territory is provided by branches coming from the spinal arteries, dural arteries, and external carotid artery branches. Among all cases described, only two cases can be really referred to a BA agenesis with a rete compensation [52, 56]. The first case was described by Kim et al. and presents a bilateral ICA agenesis with rete compensation, associated with BA absence and flow compensation through VA muscular and meningeal branches [52]. In the second case, the BA is incomplete with only a segmental agenesis of its proximal part, associated with a flow compensation through VA branches [56].

A summary of these types of BA agenesis is presented in Table 3.

Table 3 Classification of the BA agenesis

Type of compensation	Description
ICA compensation through the circle of Willis (PComA)	• VA terminates as PICA • PComA supplies the distal BA, the AICA, and the SCA
ICA compensation through persistent carotido-vertebral anastomosis	• VA terminates as PICA • PComA supplies PCA • BA as isolated trunk supplied by persistent TA
Compensation through a rete mirabile	BA absence and vascular compensation by ECA, VA, and spinal branches

Superior Cerebellar Artery

The SCA will be described in detail in its dedicated chapter. However, because of its embryological affinity with the posterior part of the circle of Willis, we will cite some pertinent peculiarities of this artery.

The SCA is considered as the most constant cerebellar artery, and no case of its absence has been reported [6, 7, 18, 33]. The artery origin in 78–98% of cases from the BA, but in 2–22% of cases it can arise from the P1 segment [7, 18, 33]. The caudal fusion of the basilar tip determines a greater possibility of a P1 origin of the SCA [7].

The artery usually origins as a single trunk, in 70–95% of cases, even if its duplicated origin is not infrequent and has been described in 5–28% of cases [6, 7, 18, 33, 60]. Since the SCA usually divides into a lateral and a medial branch, its duplicated origin can correspond to the different origin of the two branches from the P1 segment or the BA.

Only rarely, an ICA origin of a cerebellar artery has been described in the literature [7, 61–63]. Even rarer, this anomalous origin has been attributed to the SCA. In these cases, it is observed an anastomosis between the cavernous segment of the ICA and the SCA, with or without an anastomosis with the BA. The embryological explication of these cases consists in a partial persistence of the trigeminal artery, which takes in charge the vascular territory of the SCA, combined with the incomplete fusion of the longitudinal neural arteries. The grade of fusion of the LNAs and formation of the BA will influence the presence of an interposing BA in the ICA-SCA anastomosis [61]. Details of this anatomical variation will be described in the dedicated chapter.

Clinical Implications

The anatomical variant of the posterior part of the circle of Willis should be known by neurosurgeons, neurologists, and neuroradiologists, for their implications in the development of aneurysms and stroke.

Association between Circle of Willis Variants and Stroke

The presence of a collateral circulation in the brain is important for maintaining a sufficient level of cerebral blood flow in case of obstructive disease. The circle of Willis has a major role in redistributing the blood in case of diminished supply through the ICA and the BA. Radiological, clinical, and anatomical observations have suggested a relationship between the different possible configuration of the circle of Willis, and the development of cerebrovascular disease.

In case of arterial obstruction, the most rapidly recruited collaterals are the communicating arteries of the circle of Willis. In presence of a fetal PCA, the ICA can supply the PCA territory in case of occlusion in the vertebro-basilar system. On the other side, an hypoplastic PComA cannot provide this flow compensation [64]. The number of collaterals in the circle of Willis has been associated with lower stroke incidence in 2-year follow-up studies in patients with stroke [65].

This correlation has been reinforced observing that the prevalence of vascular variants in the circle of Willis is higher in brains with vascular insults, if compared with normal brains [3, 4].

Associations of Basilar Artery Fenestration with Aneurysm

The association of basilar fenestration with aneurysms is described in the literature [46, 47, 66–68]. The changes related to the high-speed turbulences and wall stress at the fenestration fork are responsible for aneurysm growth. Indeed, the defect of the media of the proximal and distal ends of the fenestrated segment can facilitate the aneurysm formation [35].

The overall incidence of aneurysm associated with basilar fenestration has been described as very variable, between 7% and 66% [35, 69]. However, results suggest that the prevalence of aneurysms at the BA fenestration is higher in symptomatic patients (SAH) than in the case of incidental BA fenestration [46]. This lower prevalence seems to be more similar to that of the general population. A clinical case of BA fenestration associated with aneurysm is shown in Fig. 3.

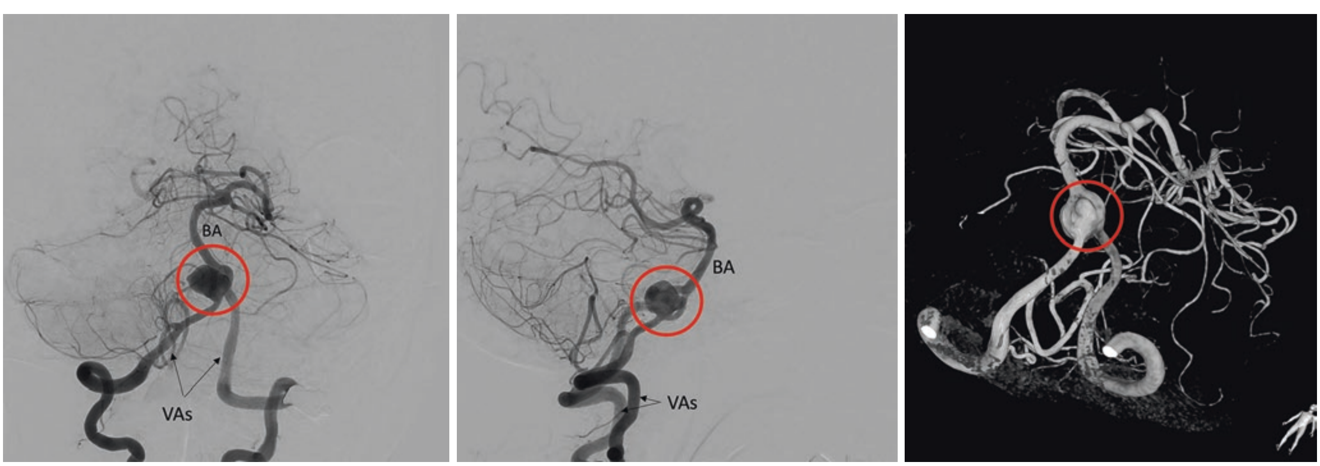


Fig. 3 Clinical case of BA fenestration with aneurysms. The figure shows the antero-posterior, lateral, and 3D reconstruction DSA after injection of the right vertebral artery. The vertebral arteries (Vas) join on the midline to form the basilar artery (BA). In its proximal segment the BA is fenestrated, and a saccular aneurysm arises from the fenestrated segment (red circle)

Association of Basilar Artery Fenestration with Stroke

The high prevalence of AICA arising from the fenestrated segment of the BA has been related to the occurrence of pontine infarction [45]. Since fenestrated segments are usually smaller in caliber than those of normal BA, they are more exposed to thrombosis. This event may cause the consequent occlusion of an AICA determining a pontine infarction [70, 71].

References

1. Willis T. The anatomy of the brain and the nerves Facsimile of the English edition by Samuel Pordage. 1681;2:82.
2. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–62.
3. Alpers BJ, Berry RG. Circle of willis in cerebral vascular disorders. The anatomical structure. Arch Neurol. 1963;8:398–402. https://doi.org/10.1001/archneur.1963.00460040068006.
4. Alpers BJ, Berry RG, Paddison RM. Anatomical studies of the circle of willis in normal brain. AMA Arch Neurol Psychiatry. 1959;81(4):409–18. https://doi.org/10.1001/archneurpsyc.1959.02340160007002.
5. Riggs HE, Rupp C. Variation in form of circle of Willis. The relation of the variations to collateral circulation: anatomic analysis. Arch Neurol. 1963;8:8–14. https://doi.org/10.1001/archneur.1963.00460010024002.
6. Saeki N, Rhoton AL Jr. Microsurgical anatomy of the upper basilar artery and the posterior circle of willis. J Neurosurg. 1977;46(5):563–78. https://doi.org/10.3171/jns.1977.46.5.0563.
7. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer edition; 2001.
8. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
9. Lasjaunias P, Santoyo-Vazquez A. Segmental agenesis of the internal carotid artery: angiographic aspects with embryological discussion. Anat Clin. 1984;6(2):133–41. https://doi.org/10.1007/bf01773165.
10. Gailloud P, Clatterbuck RE, Fasel JH, Tamargo RJ, Murphy KJ. Segmental agenesis of the internal carotid artery distal to the posterior communicating artery leading to the definition of a new embryologic segment. AJNR Am J Neuroradiol. 2004;25(7):1189–93.
11. Namba K. Carotid-vertebrobasilar anastomoses with reference to their segmental property. Neurol Med Chir (Tokyo). 2017;57(6):267–77. https://doi.org/10.2176/nmc.ra.2017-0050.
12. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: a pictorial review. Surg Radiol Anat. 2021;43(6):961–73. https://doi.org/10.1007/s00276-021-02684-y.
13. Padget DH. The circle of willis: its embryology and anatomy. In: Dandy WE, editor. Intracranial arterial aneurysm. New York: Comstock Publishing Company; 1945. p. 67–90.
14. Van Overbeeke JJ, Hillen B, Tulleken CA. A comparative study of the circle of Willis in fetal and adult life. The configuration of the posterior bifurcation of the posterior communicating artery. J Anat. 1991;176:45–54.
15. Brassier G. The posterior part of the arterial circle of the skull base. Caudal division of the embryonic internal carotid artery. Riv Neurochirurgia. 1994;7:85.
16. Kamath S. Observations on the length and diameter of vessels forming the circle of Willis. J Anat. 1981;133(Pt 3):419–23.
17. Caruso G, Vincentelli F, Rabehanta P, Giudicelli G, Grisoli F. Anomalies of the P1 segment of the posterior cerebral artery: early bifurcation or duplication, fenestration, common trunk with the superior cerebellar artery. Acta Neurochir. 1991;109(1–2):66–71. https://doi.org/10.1007/BF01405701.
18. Yasargil MG. Microneurosurgery. Stuttgart: Thieme; 1984.
19. Bisaria KK. Anomalies of the posterior communicating artery and their potential clinical significance. J Neurosurg. 1984;60(3):572–6. https://doi.org/10.3171/jns.1984.60.3.0572.
20. Gunnal SA, Farooqui MS, Wabale RN. Anatomical variability of the posterior communicating artery. Asian J Neurosurg. 2018;13(2):363–9. https://doi.org/10.4103/ajns.AJNS_152_16.
21. Naeini RM, De J, Satow T, Benndorf G. Unilateral agenesis of internal carotid artery with ophthalmic artery arising from posterior communicating artery. AJR Am J Roentgenol. 2005;184(2):571–3. https://doi.org/10.2214/ajr.184.2.01840571.
22. Nakata H, Iwata Y. Agenesis of the left internal carotid artery with an ophthalmic artery arising from the posterior communicating artery. No Shinkei Geka. 1987;15(1):57–62.
23. Priman J, Christie DH. A case of abnormal internal carotid artery and associated vascular anomalies. Anat Rec. 1959;134:87–95. https://doi.org/10.1002/ar.1091340109.
24. Zeal AA, Rhoton AL Jr. Microsurgical anatomy of the posterior cerebral artery. J Neurosurg. 1978;48(4):534–59. https://doi.org/10.3171/jns.1978.48.4.0534.
25. Giannantoni N, Bonasia S, Bertulli L, Staedler C, Robert T. Rare neurovascular conflict between oculomotor nerve and posterior communicating artery. Neuroradiology. 2020;62(12):1717–20. https://doi.org/10.1007/s00234-020-02489-7.
26. Gunnal S, Farooqui M, Wabale R. Anatomical variability in the termination of the basilar artery in the human cadaveric brain. Turk Neurosurg. 2015;25(4):586–94. https://doi.org/10.5137/1019-5149.JTN.12812-14.0.
27. Uz A, Mine EK. A morphological study of the posterior communicating artery. Folia Morphol (Warsz). 2004;63(4):397–9.
28. Tripathi M, Goel V, Padma MV, Jain S, Maheshwari MC, Gaikwad S, et al. Fenestration of the posterior communicating artery. Neurol India. 2003;51(1):75–6.
29. Chen H. Common origin of the anterior choroidal artery and posterior communicating artery with a concomitant aneurysm at the internal carotid artery-posterior communicating artery junction: a case report. Med Int. 2021;1:1–4. https://doi.org/10.3892/mi.2021.12.
30. Krayenbühl HA, Yasargil MG. Die vaskulären Erkrankungen im Gebiet der Arteria vertebralis und Arteria basilaris: eine anatomische und patologische, klinische und neuroradiologische Studie. Stuttgart. 1957.
31. Gordon-Shaw C. Two cases of reduplication of the arteria cerebri posterior. J Anat Physiol. 1910;44(Pt 3):244–8.
32. Windle BC. The arteries forming the circle of willis. J Anat Physiol. 1888;22(Pt 2):289–93.
33. Hardy DG, Peace DA, Rhoton AL Jr. Microsurgical anatomy of the superior cerebellar artery. Neurosurgery. 1980;6(1):10–28. https://doi.org/10.1227/00006123-198001000-00002.
34. Campos C, Churojana A, Rodesch G, Alvarez H, Lasjaunias P. Basilar tip aneurysms and basilar tip anatomy. Interv Neuroradiol. 1998;4(2):121–5. https://doi.org/10.1177/159101999800400203.
35. Sanders WP, Sorek PA, Mehta BA. Fenestration of intracranial arteries with special attention to associated aneurysms and other anomalies. AJNR Am J Neuroradiol. 1993;14(3):675–80.
36. Trivelato FP, Abud DG, Nakiri GS, de Castro Afonso LH, Ulhoa AC, Manzato LB, et al. Basilar artery fenestration aneurysms: endovascular treatment strategies based on 3D morphology. Clin Neuroradiol. 2016;26(1):73–9. https://doi.org/10.1007/s00062-014-0336-0.

37. Bayrak AH, Senturk S, Akay HO, Ozmen CA, Bukte Y, Nazaroglu H. The frequency of intracranial arterial fenestrations: a study with 64-detector CT-angiography. Eur J Radiol. 2011;77(3):392–6. https://doi.org/10.1016/j.ejrad.2009.09.015.
38. Gao LY, Guo X, Zhou JJ, Zhang Q, Fu J, Chen WJ, et al. Basilar artery fenestration detected with CT angiography. Eur Radiol. 2013;23(10):2861–7. https://doi.org/10.1007/s00330-013-2890-2.
39. Takahashi M, Tamakawa Y, Kishikawa T, Kowada M. Fenestration of the basilar artery. Report of three cases and review of the literature. Radiology. 1973;109(1):79–82.
40. van Rooij SB, Bechan RS, Peluso JP, Sluzewski M, van Rooij WJ. Fenestrations of intracranial arteries. AJNR Am J Neuroradiol. 2015;36(6):1167–70. https://doi.org/10.3174/ajnr.A4236.
41. Wollschlaeger G, Wollschlaeger PB, Lucas FV, Lopez VF. Experience and result with postmortem cerebral angiography performed as routine procedure of the autopsy. Am J Roentgenol Radium Therapy, Nucl Med. 1967;101(1):68–87. https://doi.org/10.2214/ajr.101.1.68.
42. Patel MA, Caplan JM, Yang W, Colby GP, Coon AL, Tamargo RJ, et al. Arterial fenestrations and their association with cerebral aneurysms. J Clin Neurosci. 2014;21(12):2184–8. https://doi.org/10.1016/j.jocn.2014.07.005.
43. Tanaka M, Kikuchi Y, Ouchi T. Neuroradiological analysis of 23 cases of basilar artery fenestration based on 2280 cases of MR angiographies. Interv Neuroradiol. 2006;12(Suppl 1):39–44. https://doi.org/10.1177/15910199060120S103.
44. Uchino A, Kato A, Takase Y, Kudo S. Basilar artery fenestrations detected by MR angiography. Radiat Med. 2001;19(2):71–4.
45. Uchino A, Saito N, Okada Y, Kozawa E, Nishi N, Mizukoshi W, et al. Fenestrations of the intracranial vertebrobasilar system diagnosed by MR angiography. Neuroradiology. 2012;54(5):445–50. https://doi.org/10.1007/s00234-011-0903-x.
46. Sogawa K, Kikuchi Y, O'Uchi T, Tanaka M, Inoue T. Fenestrations of the basilar artery demonstrated on magnetic resonance angiograms: an analysis of 212 cases. Interv Neuroradiol. 2013;19(4):461–5. https://doi.org/10.1177/159101991301900409.
47. Campos J, Fox AJ, Vinuela F, Lylyk P, Ferguson GG, Drake CG, et al. Saccular aneurysms in basilar artery fenestration. AJNR Am J Neuroradiol. 1987;8(2):233–6.
48. Goldstein JH, Woodcock R, Do HM, Phillips CD, Dion JE. Complete duplication or extreme fenestration of the basilar artery. AJNR Am J Neuroradiol. 1999;20(1):149–50.
49. Uchino A, Sawada A, Takase Y, Fujita I, Kudo S. Extreme fenestration of the basilar artery associated with cleft palate, nasopharyngeal mature teratoma, and hypophyseal duplication. Eur Radiol. 2002;12(8):2087–90. https://doi.org/10.1007/s00330-001-1194-0.
50. Hoh BL, Rabinov JD, Pryor JC, Hirsch JA, Dooling EC, Ogilvy CS. Persistent nonfused segments of the basilar artery: longitudinal versus axial nonfusion. AJNR Am J Neuroradiol. 2004;25(7):1194–6.
51. Zhang D, Wang H, Feng Y, Xu N. Fenestration deformity of the basilar artery trunk with an aneurysm: a case report. Medicine (Baltimore). 2019;98(28):e16393. https://doi.org/10.1097/MD.0000000000016393.
52. Kim MS, Lee SJ, Lee CH, Park HI. Bilateral segmental absence of the internal carotid artery with rete compensation associated with absence of basilar artery: case report. Surg Neurol. 2006;65(6):615–9; discussion 9; author reply 20. https://doi.org/10.1016/j.surneu.2005.07.072.
53. Lasjaunias P, Manelfe C, Roche A, Rascol A. Segmental aplasia of the basilar artery in man. Report of a case. J Neuroradiol. 1979;6(2):127–36.
54. Mahadevan J, Batista L, Alvarez H, Bravo-Castro E, Lasjaunias P. Bilateral segmental regression of the carotid and vertebral arteries with rete compensation in a Western patient. Neuroradiology. 2004;46(6):444–9. https://doi.org/10.1007/s00234-003-1086-x.
55. Jones RR, Wetzel N. Bilateral carotid vertebrobasilar rete mirabile. Case report. J Neurosurg. 1970;33(5):581–6. https://doi.org/10.3171/jns.1970.33.5.0581.
56. Sahin H, Cinar C, Oran I. Carotid and vertebrobasilar rete mirabile: a case report. Surg Radiol Anat. 2010;32(2):95–8. https://doi.org/10.1007/s00276-009-0531-x.
57. Hong JM, Lee JH, Yong SW. Neurological picture. Asymtomatic rete mirabile in carotid and vertebral circulation systems. J Neurol Neurosurg Psychiatry. 2010;81(9):1022–3. https://doi.org/10.1136/jnnp.2009.178368.
58. Lee SY, Cha SH. Bilateral carotid and vertebral rete mirabile presenting with a prominent anterior spinal artery mimicking a spinal dural AV fistula at MRI. Korean J Radiol. 2011;12(6):740–4. https://doi.org/10.3348/kjr.2011.12.6.740.
59. Burger IM, Siclari F, Gregg L, Gailloud P. Bilateral segmental agenesis of the vertebrobasilar junction: developmental and angiographic anatomy. AJNR Am J Neuroradiol. 2007;28(10):2017–22. https://doi.org/10.3174/ajnr.A0719.
60. Mani RL, Newton TH, Glickman MG. The superior cerebellar artery: an anatomic-roentgenographic correlation. Radiology. 1968;91(6):1102–8. https://doi.org/10.1148/91.6.1102.
61. Haughton VM, Rosenbaum AE, Pearce J. Internal carotid artery origins of the inferior cerebellar arteries. AJR Am J Roentgenol. 1978;130(6):1191–2. https://doi.org/10.2214/ajr.130.6.1191.
62. Teal JS, Rumbaugh CL, Bergeron RT, Scanlan RL, Segall HD. Persistent carotid-superior cerebellar artery anastomosis: a variant of persistent trigeminal artery. Radiology. 1972;103(2):335–41. https://doi.org/10.1148/103.2.335.
63. Scotti G. Anterior inferior cerebellar artery originating from the cavernous portion of the internal carotid artery. Radiology. 1975;116(1):93–4. https://doi.org/10.1148/116.1.93.
64. van Raamt AF, Mali WP, van Laar PJ, van der Graaf Y. The fetal variant of the circle of Willis and its influence on the cerebral collateral circulation. Cerebrovasc Dis. 2006;22(4):217–24. https://doi.org/10.1159/000094007.
65. Henderson RD, Eliasziw M, Fox AJ, Rothwell PM, Barnett HJ. Angiographically defined collateral circulation and risk of stroke in patients with severe carotid artery stenosis. North American symptomatic carotid endarterectomy trial (NASCET) group. Stroke. 2000;31(1):128–32. https://doi.org/10.1161/01.str.31.1.128.
66. Black SP, Ansbacher LE. Saccular aneurysm associated with segmental duplication of the basilar artery. A morphological study. J Neurosurg. 1984;61(6):1005–8. https://doi.org/10.3171/jns.1984.61.6.1005.
67. Andrews BT, Brant-Zawadzki M, Wilson CB. Variant aneurysms of the fenestrated basilar artery. Neurosurgery. 1986;18(2):204–7. https://doi.org/10.1227/00006123-198602000-00017.
68. Matricali B, van Dulken H. Aneurysm of fenestrated basilar artery. Surg Neurol. 1981;15(3):189–91. https://doi.org/10.1016/0090-3019(81)90139-7.
69. Tasker AD, Byrne JV. Basilar artery fenestration in association with aneurysms of the posterior cerebral circulation. Neuroradiology. 1997;39(3):185–9. https://doi.org/10.1007/s002340050389.
70. Scherer A, Siebler M, Aulich A. Virtual arterial endoscopy as a diagnostic aid in a patient with basilar artery fenestration and thromboembolic pontine infarct. AJNR Am J Neuroradiol. 2002;23(7):1237–9.
71. Kloska SP, Schlegel PM, Strater R, Niederstadt TU. Causality of pediatric brainstem infarction and basilar artery fenestration? Pediatr Neurol. 2006;35(6):436–8. https://doi.org/10.1016/j.pediatrneurol.2006.06.013.

Embryology and Anatomy of the Posterior Cerebral Artery

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As stated by Arnold A. Zeal and Albert L. Rhoton in a public conference "*the posterior cerebral artery is one of the most hazardous arteries to approach surgically because of its deep location, its frequent origin in the midline, the complex series of perforating vessels surrounding and arising from it, and its intimate relationship to the extraocular nerves and upper brainstem*" [1]. Therefore, in the present era of microscopic and neuroendoscopic procedures, the surgical anatomy of the posterior skull base vessels and the need for a better understanding of the microanatomy of the posterior cerebral artery (PCA) have gained increased significance [1–4]. Moreover, given the remarkable anatomic complexity of the PCA, it is crucial to have a detailed knowledge of its distribution [5]. Since the illustration of Thomas Willis in 1664, many anatomical variations have been described about the configuration of the posterior part of the "circulus arteriosus cerebri" and several different classifications of the segments of the PCA have been proposed during the last 150 years [2]. In Mammalia, the posterior cerebral artery is said to arise from the basilar artery and its main trunk not only supplies the posterior part of the cerebral hemispheres, as its name implies, but also sends critical branches to the thalamus, midbrain, and other deep structures including the choroid plexus and walls of the lateral and third ventricles [2, 6]. One of the peculiar features of the PCA is growing so far away from its primitive parent, the internal carotid artery, that even the caudal division will not suffice for its requirements.

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The purpose of this chapter is to describe the embryology of the PCA, its branches and anatomical variations, the spatial distribution, and, finally, the pathological clinical syndromes related with this artery.

History

The idea of the rete mirabilis of Herophilus (335–280 BC) and Claudius Galenus (129–201 CE) described in "*De usu partium*" presented the evident limitations of studies based on animal dissection. The illustration of brain vessels by Andreas Vesalius (1514–1564) in the "*Liber Septimus of the De humani corporis fabrica*," even if revolutionary, had the insurmountable errors in the distribution of the arteries due to the practice of freeing the vessel from the specimen for the drawing [7–10]. Thomas Willis (1621–1675), however, was not the first who described the "circulus arteriosus cerebri" and the posterior cerebral circulation anatomy; Gabriel Fallopius (1523–1563), in fact, one century earlier, in his "*Observationes Anatomicae"* (1561), recognized the ramifications of the basilar artery and the posterior communicating artery. Moreover, it seems that Johann Jakob Wepfer von Schaffhausen (1620–1695), from Switzerland, was the real discoverer of the "arterial circle" and in fact, 6 years before Thomas Willis, in his work "*Observationes anatomicae ex cadaveribus eorum quos sustulit apoplexia"* (1658), he stated that "*the posterior branch of the carotid artery travels a little on its tract and then joins the vertebral artery that has divided again into two*" [11, 12].

Willis, however, in the "*De Cerebri Anatome*" in 1664 was the first who described and illustrated the posterior cerebral arteries and their connections. He stated that: "*the Carotid Arteries of one side, in many, are united with the Carotids of the other side; besides the Vertebrals of either side among themselves, and are also inoculated into the posterior branches of the Carotids before united*" [11–13].

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For the following two centuries, no particular contributions have been given to the study of the posterior cerebral artery anatomy. At the beginning of the twentieth century, new interest has raised regarding the variability of the major vessels (Bevor in 1909 [14], Shellshear in 1927 [15] and Foix 1925 [16]) and the PCA in particular (Williams in 1936 [17]). In the fifties, Alpers et al. (1959) [18] studied the incidence of the fetal-type in a series of 837 brains [13] while Padget (1947) [19, 20] and Seydel (1964) [21] distinguished the differences between adult and fetal configurations of the posterior communicating artery in relation with the precommunicating and postcommunicating parts of the PCA. Certainly, a contribution to a better understanding of the embryology of the vessels, after Padget, has been given by Moffat in the sixties [22, 23]. In the second half of the twentieth century, several classifications have been proposed for the different parts of the artery, by Ecker and Riemenschneider (1955) [24, 25], Krayenbuhl and Yasargil (1968) [26], or Margolis (1971) [27]. The latter, for example, renamed the segments in relation with the structures and the cisterns involved in the course of the artery identifying the peduncular, ambient, and quadrigeminal parts of the PCA [27]. Further description and illustrated classifications are encountered in the last thirties of the century thanks to the widespread of the imaging and the new atlas (Salamon in 1971 [28] and Waddington in 1974 [29]). Lasjaunias can be considered the first who correlated the knowledge obtained from the cadaveric dissection with the more and more precise diagnostic tools such as the angiographic studies; thus, his work is definitely considered one of the most important of the modern era [30].

Nowadays, doubtless, the major contribution in the description and comprehension of the microneurosurgery of the PCA belongs to the "Rhoton' legacy." Since 1977, Saeki N and Zeal A. have accompanied Rhoton in the definition of the microsurgical anatomy of the upper basilar artery and the posterior circle of Willis [1, 2, 31]. Their work represents the most complete and precise contribution for better understanding the anatomy of the PCA and its branches.

Embryological Development

Few studies before the comprehensive research conducted by Padget in 1948 have dealt with the development of the PCA. The most likely reason for this, it seems to be the fact that the development of this artery occurs at a relatively late stage, moment in which the serial sections of a too large fetus cannot be feasible [22, 23].

Even if the 22 sectioned embryos from Padget represent the most comprehensive series for the description and illustration of the PCA development during the embryology, it is important to mention the studies on rats performed in the early twentieth century by Greene and those by Moffat in 1962 [22, 32]. In his series, Moffat compared his 196 injected rat embryos plus 5 human specimens with the series of Hoffmann (1900), Abbie (1934) [6], Greene (1935) [32], and Padget herself (1948 and 1956) [20, 22]. Both Hoffmann and Abbie have pointed out the caudal displacement of the origin of the posterior cerebral artery in response to the backward growth of the telencephalon. Moffat confirmed this argument and identified the origin of the PCA from a part of the posterior communicating artery (PComA), the common stem of origin of the posterior choroidal, diencephalic, and mesencephalic arteries plus a new vessel which develops in the plexus on the medial wall of the telencephalic vesicle [6, 22].

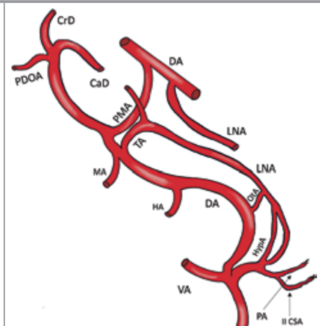
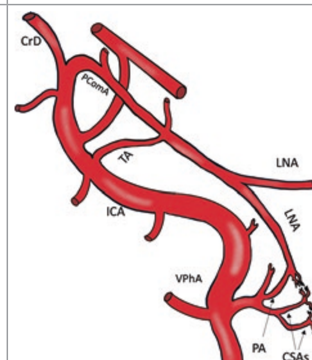
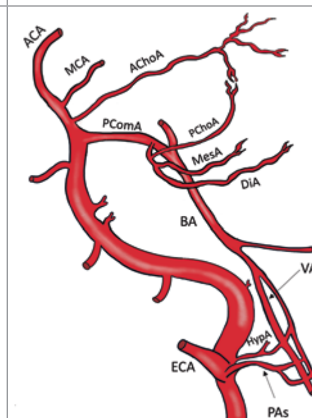
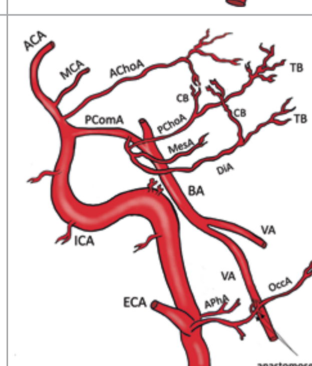
According with Padget, Lazorthes, and Lasjaunias, the PCA is, from the embryological point of view, a diencephalon-mesencephalic vessel: at Padget stage 3 the diencephalic artery and the mesencephalic artery emerge from the caudal end of the PComA. From the common trunk of these branches, the posterior choroidal artery (PChoA) develops and reaches the atrium to anastomose with the anterior choroidal artery (AChoA). This anastomosis forms the choroidal circle with a choroidal supply and a telencephalic branch. This latter is gradually annexed by the PChoA [19, 20, 30, 33, 34].

However, it is at the Padget stage IV and in particular at Padget stage V (Carnegie Stage 19), when the embryo has the crown-rump (CR) length of 16/18 mm, that the PCA starts to develop and to become recognizable. At these stages, in fact, the main trunk of the PChoA with its telencephalic branch starts forming the main posterior cerebral artery stem (P2, P3, P4 segments), supplying most of the cortical territories previously supplied by the cranial division. The choroidal branch, instead, will become the posterior lateral choroidal artery (PLChoA) while the choroidal branch of the diencephalic artery will become the posterior medial choroidal artery (PMChoA) [19, 20, 30, 34].

At Padget stage VII, thanks to the enlargement of the PCA which gathers the telencephalic supply also annexing the telencephalic branch of the diencephalic trunk, and thanks to the definitive position of the PComA, the circle of Willis is closed and recognizable [19, 20, 34–37]. Moreover, already from stage VI, since the dorsal branches of the mesencephalic arteries have extended across the expanded cerebral hemisphere, this new mesencephalic-PCA territory is supplied progressively more by the vertebrobasilar system [30, 34, 37].

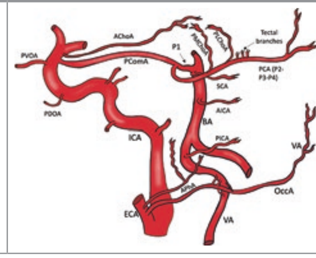
During the remaining fetal period, the PCA enlarges further, and the other smaller arteries become PCA branches, as we can see in the adult configuration [19, 20, 33–35]. The adult configuration, however, in which the P1 segment has a larger diameter than the PComA, is not the only variant the circle can present as regard the relationship between these two vessels [2, 13, 19, 30, 31, 38–41]. Apart from the normal adult configuration, a *transitional* and a *fetal or embryonic* configuration are described, and they are determined during this stage of the fetal development. The transitional configuration is noted when the PComA and P1 have the same diameter while, in the fetal or embryonic one, the P1 is smaller than the PComA and the ICAs are the main blood suppliers to the occipital lobes [13, 39]. These different configurations are described in detail in the following paragraph regarding the variants in number and origin of the artery. The main embryological steps in the formation of the PCA are summarized and illustrated in Table 1.

Table 1 Major embryological changes in the formation of the vertebro-basilar system

Stage	Embryo size (mm)	Major evolutions	Graphic illustration
I	4–5	– Paired longitudinal neural arteries (LNAs) visible – Carotid-vertebral anastomoses visible: Trigeminal (TA), otic (OtA), hypoglossal (HypA), proatlantal (PA), and second cervical segmental artery (II CSA) – Primitive ICA bifurcation visible into caudal and cranial divisions (CaD, CrD) – PComA and BA not formed	
II	5–6	– Formation of the PComA – Regression of otic and hypoglossal arteries – Midline fusion of LNAs to form the basilar artery (BA)	
III	7–12	– Basilar artery (BA) completely formed – PComA completely formed – Origin from the distal PComA of a diencephalon-mesencephalic trunk (DiA-MesA). PChoA arising from the common trunk and anastomoses with the AChoA – Regression of trigeminal and proatlantal arteries – Formation of the vertebral artery VA by transverse anastomoses between the intersegmental arteries	
IV	13–15	– Development of a choroidal (CB) and telencephalic branch (TB) from the PChoA and the diencephalic artery (DiA) – Remodeling of the BA – Migration of OccA origin on the external carotid artery (ECA) – Persistence of the anastomoses between OccA and VA	

(continued)

Table 1 (continued)

Stage	Embryo size (mm)	Major evolutions	Graphic illustration
V	16–18	– The telencephalic branch of the PChoA forms the main PCA trunk – The choroidal branches of the PChoA and of the diencephalic artery form the PLChoA and PMChoA – Complete formation of the VA – Complete formation of the OccA (remnant of the proatlantal artery) – Complete formation of the ascending pharyngeal artery (APhA, remnant of the hypoglossal artery) – SCA, AICA, and PICA are visible	

Number and Origin of the Artery

Very few reports have been published in terms of variability of the anatomy of the PCA in comparison to the anterior and middle cerebral arteries. Nevertheless, among the main vessels, the PCA seems to be the most variable [37, 42]. As stated by Zeal and Rhoton, "embryologically, the PCA arises as a branch of the internal carotid artery, but by birth its most frequent origin is from the basilar artery" [2]. Consequently, the so-called "classic pattern" identifies the origin of the PCA from the basilar artery, while the "embryonic pattern" is described when the PCA originates from the carotid artery. Not uncommonly, it may originate differently on each side [37]. Fisher et al. described in their study that in 71% of cases, the PCA arises from the terminal basilary artery bilaterally, from the internal carotid artery (ICA) unilaterally in 22% of cases and bilaterally in 7% [43]. Therefore, the basilar artery bifurcation can be considered the origin of the PCA. In the series from Rhoton et al., the origin resulted located between 1.3 mm below the pontomesencephalic junction and the mammillary body at the region of the adjacent floor of the third ventricle [2, 4]. The diameter of the PCA, measured just after the division of the basilar artery, is very variable and ranged from 2 to 4 mm, being most often 3.4 mm [44, 45]. Some studies have identified a difference in the average diameter of the vessel between the two sides, being from 2.1 to 2.7 mm on the right and between 1 and 2.5 mm on the left [46]. Regarding the variability on the length, the PCA can range between 6.8 and 7.5 mm according with the different series [38, 46]. Few studies report on the anomalies of PCA, described principally as duplication, triplication, fetal-type, and fenestration. As concerns the duplication, this variant can occur in a range between 0.2% and 2.3% [42, 47]. In case of duplication, the additional branch originates most commonly from P1. However, duplicated vessels arising from PcomA or P2 segment have been also described. When the additional branch arises from the P2 segment, this can present similar to early branching of the PCA [42, 47]. In the balance of territory distribution of perfusion, in case of duplicate PCAs, the anterior choroidal artery supplies the inferior temporal cortex and the PCA supplies the medial temporo-occipital cortex [37]. Few studies have reported cases of triplication of the PCA, being even extremely rarer. Kapoor et al. [48] in 2008 observed the triplication in eight cases (0.8%); in the series the branches were noticed to origin from P1 and supply the temporal and occipital lobes [42]. More common is certainly the variant of the fetal-type configuration of P1. This variation is described in literature with percentages between 10% and 40%, depending on the series [47, 49]. As presented in the discussion about the embryology, this configuration is caused by the uncomplete development of the PCA starting at Padget stage V. As a result, the P1 segment is hypoplastic or absent. The variant is named "fetal" according to the persistence of the early fetal configuration of the PCA in which it originates from the internal carotid artery instead of the vertebrobasilar system [39]. The fetal-type configuration is characterized by a larger size of the PComA compared with the ipsilateral precommunicating segment (P1) [50]. As for clinical interest, the fetal variant seems to be associated with larger neck aneurysms arising from this vessel [50, 51].

Another unusual anomaly of the PCA is the fenestration. According to Lasjaunias, this is defined as a rare congenital abnormality with a common vessel origin after which the artery subsequently splits into two channels that rejoin distally [30]. This configuration can be referred also as a partial or incomplete duplication [30, 42, 52–54]. Cooke et al., in 2014, in their cohort of 10,927 patients, observed the presence of the fenestration in the 1.5% of cases (167 cases), while Milisavljevic et al. (1988) reported a percentage of the 1.4% (1 case in 70 patients) [42, 55, 56]. As for the other anomalies, the fenestration usually involves the P1 segment. Nevertheless, different authors also observed fenestration in the P2 segment or the distal segments of the PCA, although these variants are considered extremely rare [36, 42, 47, 55–58].

Course of the Artery

Many classifications of the segments of the PCA have been published in the literature but the most common and currently adopted is the one proposed by Rhoton et al. [2, 4]. They divided PCA into four segments, from P1 to P4 [2, 5, 16, 26–29, 31, 49].

After being originated at the level of the region between the pontomesencephalic junction and the mammillary body, at the lateral margin of the interpeduncular cistern, before entering the crural cistern, the PCA is joined by the posterior communicating artery. Then, the artery passes through the ambient cistern before terminating in the quadrigeminal cistern for its terminal distribution [2, 4]. The segments and the course of the PCA are illustrated in Fig. 1 and summarized in Table 2.

- The P1 segment is also called precommunicating. Several other names have been proposed for this tract such as "proximal peduncular," "mesencephalic," "circular," or "pars basilaris" [26, 31]. It starts at the basilar artery bifurcation and ends at the anastomosis of the PCA with the posterior communicating artery. Normally, P1 segment is larger than the PComA with the differences between the adult, transitional, and fetal configurations as previously described [2, 4, 31]. Regarding the length, instead, if a fetal pattern is present, the P1 segment is usually longer than the normal one reaching 9 mm compared to the average of 7 mm of the normal configuration (average 3–14 mm) [2, 4].
- The P2 segment (or perimesencephalic or postcommunicating) starts at the anastomosis of the PComA with the PCA and ends at the posterior edge of the lateral surface of the midbrain [4, 5]. Because of its topography and anatomic relationship, the P2 is considered a complex segment to be approached surgically [5]. It is divided into two parts: anterior and posterior. The anterior part, called P2A segment or "crural" or "peduncular" segment, begins at the anastomosis with the PComA and ends at the most lateral aspect of the cerebral peduncle, in the crural cistern. Approximately, the P2A segment has an average length of 23.6 mm and a diameter of 1.7 mm. The course of the P2A is defined medially by the cerebral peduncle, laterally by the uncus and superiorly by the optic tract and the basal vein. The posterior part, instead, called P2P segment or "ambient" or "lateral mesencephalic," begins at the junction between the crural and the ambient cisterns, at the most lateral aspect of the cerebral peduncle, and

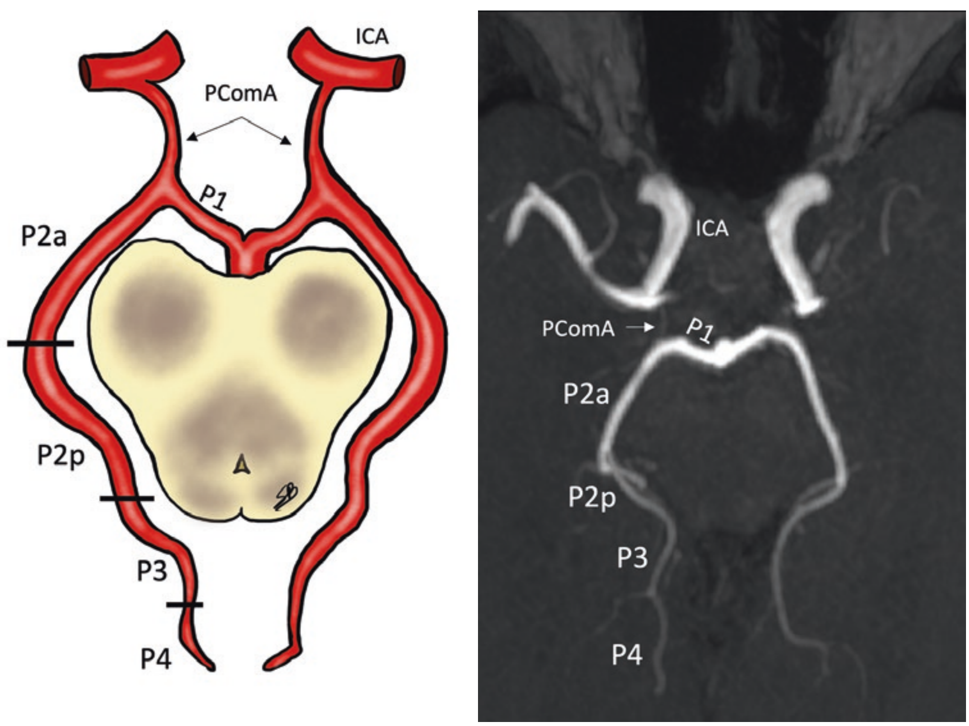


Fig. 1 Segments of the PCA, pictorial and radiologic (MRI, TOF). P1 segment: from the basilar artery bifurcation to the anastomosis of the PCA with the posterior communicating artery (PComA). P2 segment: P2a segment or "crural" or "peduncular" segment, from the anastomosis with the PComA to the most lateral aspect of the cerebral peduncle, in the crural cistern; P2p segment or "ambient" or "lateral mesencephalic," from the junction between the crural and the ambient cisterns, to the posterior edge of the lateral surface of the midbrain. P3 segment or "quadrigeminal": from the posterior edge of the lateral surface of the midbrain to the anterior limit of the calcarine fissure through the lateral portion of the quadrigeminal cistern. The P4 segment represents the terminal segment of the PCA and gives off its terminal cortical branches

Table 2 Course of the artery, segments, and morphometry

Segments	Course	Length average (mm)	Diameter average (mm)
P1 (precommunicating)	From the basilar artery to the anastomosis of the PCA with the posterior communicating artery	7	2.8
P2 (postcommunicating or perimesencephalic)			
P2A (crural or peduncular)	From the anastomosis of the PComA with the PCA to the cerebral peduncle	23.6	1.7
P2P (ambient or lateral mesencephalic)	From the junction between the crural and the ambient cisterns to the posterior edge of the midbrain	16.4	1.4
P3 (quadrigeminal)	From the posterior edge of the midbrain to the calcarine fissure	19.8	1.1
P4 (parieto-occipital and calcarine arteries)	Through the parieto-occipital sulcus and the distal part of the calcarine fissure	Specific for each terminal arteries	

ends at the posterior edge of the lateral surface of the midbrain. The P2P has an average length of 16.4 mm and a diameter of 1.4 mm. In its course in the ambient cistern, the segment is surrounded superiorly by the optic tract, the basal vein, the geniculate bodies, and the pulvinar, medially by the lateral midbrain, and laterally by the parahippocampal and dentate gyrus. The P2P segment is located just above and medially of the tentorial edge and the trochlear nerve [2, 4, 5, 31].

- The P3 segment or "quadrigeminal" courses from the posterior edge of the lateral surface of the midbrain to the anterior limit of the calcarine fissure through the lateral portion of the quadrigeminal cistern where it divides into its major terminal branches, the calcarine and parieto-occipital arteries. The P3 segment has an average length of 19.8 mm, 1.1 mm of diameter and it reaches a point called "collicular" or "quadrigeminal" representing the nearest distance between the two PCAs. The average distance between both P3 segments at the level of their collicular points is quite variable ranging from 3.5 to 17 mm [2, 4, 5, 31].
- The P4 segment corresponds to the part of the PCA that course along or inside both the parieto-occipital sulcus and the distal part of the calcarine fissure: they are named, respectively, the parieto-occipital and calcarine arteries, and includes the branches distributed to the cortical surface [2, 4, 5, 31].

Branches of the Artery

Three types of branches are originated from the PCA [4]:

- Ventricular branches to the choroid plexus and walls of the lateral and third ventricles and adjacent structures (treated in chapter "Embryology and Variations of the Posterior Choroidal Artery")
- Central perforating branches to the diencephalon and midbrain (treated in chapter "Embryology and Anatomy of the Posterior Cerebral Artery")
- Cerebral branches to the cerebral cortex and splenium of the corpus callosum as treated below

Compared to the literature dedicated to the branches of other major arteries, few studies report patterns and variations of the PCA cortical branches such as their absence, duplications, triplications, or abnormal origin [42]. In a recent study, Cilliers et al. selected cases where the PCA cortical branches have origin from the internal carotid artery, including the parieto-occipital artery, the calcarine artery, and the posterior-inferior temporal artery [42]. PCA branches are exposed in surgical approaches to the basilar apex, tentorial notch, lateral and third ventricles, inferior temporal, and medial parieto-occipital areas, in addition to the pineal region; all relatively inaccessible areas [2, 31].

The cerebral branches, which are illustrated in Fig. 2, include:

1. The inferior temporal group of branches, which are divided into:
   (a) The hippocampal artery
   (b) The anterior temporal artery
   (c) The middle temporal artery
   (d) The posterior temporal artery
   (e) The common temporal artery
2. The parieto-occipital artery
3. The calcarine artery
4. The splenial artery
5. The lateral convexity branches

The Inferior Temporal Group Branches

The hippocampal artery. The *hippocampal artery* is the first cortical branch of the PCA but is not always present. It starts in the crural or ambient cistern and enters the uncal sulcus emerging on the surface of the pyriform lobe (anterior hippocampal arteries). The posterior hippocampal arteries run in the superficial course of the hippocampal sulcus terminating in large and small branches. The large branches penetrate the hippocampus and small branches supply the margo-denticulatus and fimbrio-dentate sulcus [59]. An anatomical study by Erdem and Yasargil on the

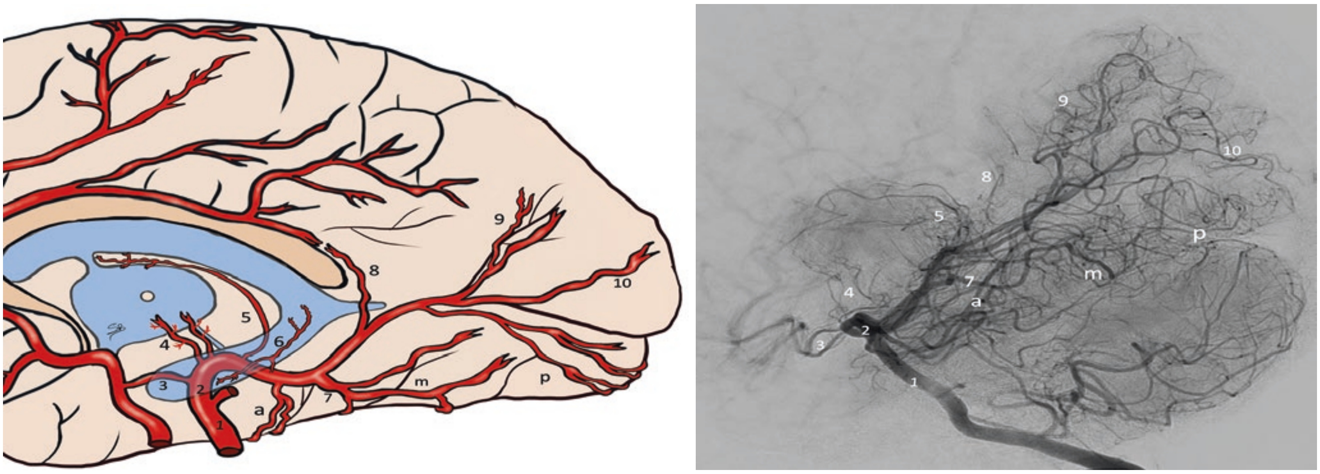


Fig. 2 PCA course and branches, pictorial and angiographic. (*1*) Basilar artery; (*2*) P1 segment; (*3*) posterior communicating artery; (*4*) thalamo-perforating arteries; (*5*) posterior medial choroidal artery; (*6*) posterior-lateral choroidal artery; (*7*) inferior temporal group; a: anterior temporal artery; m: middle temporal artery; p: posterior temporal artery; (*8*) splenial or posterior pericallosal artery; (*9*) parieto-occipital artery; (*10*) calcarine artery

vascularization of the hippocampus (1993) shows that in the 26.6% of the hemispheres examined, one of the hippocampal arteries arose from the lateral posterior choroidal artery [2, 4, 31, 60].

The anterior temporal artery. The *anterior temporal artery* is the first branch when the hippocampal is not present, but usually is the second cortical branch of the PCA. It usually arises from P2A and less frequently from the proximal P2P; it runs the anteroinferior surface of the temporal lobe, occasionally reaching a portion of the temporal pole and the lateral cerebral surface in the region of the middle temporal sulcus and gyrus [2, 4, 31].

The middle temporal artery. The *middle temporal artery* is the smallest and has the fewest branches of the inferior temporal arteries [2]. It arises from P2A in 16% and from P2P in 22%. Few studies reported this branch as absent, inconstant, or uncommon [2, 26, 27].

The posterior temporal artery. The *posterior temporal artery*, almost always present, arises more commonly from P2P and less frequently from P2A and P3. It runs obliquely and postero-laterally reaching the inferior temporal surface but also the occipital surfaces, the occipital pole, and the lingual gyrus.

The common temporal artery. Depending on the pattern, a *common temporal artery* can present as a single inferior PCA branch supplying most of the inferior surface of the temporal and occipital lobes. In the series from Zeal and Rhoton, this configuration was present in 16% of the hemispheres and it arises from P2P in 10% of the cases, and from P2A in the other 6% [2, 4]. When present, this large branch is referred as the “lateral division of the PCA,” the “lateral occipital artery,” or as the common trunk from which the anterior and posterior temporal arteries arise [2, 4, 26–28].

The above arteries are included in the inferior temporal group of arteries originating from the PCA and can present in different patterns with inconstant presence of one or more of them. They balance the supply to the temporal and occipital lobes with the superior temporal group of arteries arising from the MCA.

The Parieto-Occipital Artery

The *parieto-occipital artery* is one of the two terminal branches of the PCA. In the series of Marinkovic et al., it originated from the PCA in either the ambient (in 15% of the brains examined) or quadrigeminal cistern (18.3%), or in the calcarine sulcus (66.7%) [61]. In the series of Zeal and Rhoton, it arises from P2A in 10%, P2P in 40%, and P3 in 46% of hemispheres [2]. Branches to the midbrain, thalamus, pulvinar, lateral geniculate bodies but also to the choroid plexus through the choroidal fissure emerge from the artery with a more proximal origin. After the origin, the parieto-occipital artery (POA) enters the rostral portion of the calcarine sulcus and then continues along the parieto-occipital sulcus. In one of the sulci, the artery divided into two-, three-, or four-terminal stems, the branches of which were distributed to the medial and sometimes the lateral surface of the occipital and parietal lobes [2, 4, 61, 62].

The Calcarine Artery

The *calcarine artery* is the other terminal branch of the PCA; named according to its course in the calcarine fissure, it arises in most of the cases from P3 but also from P2P. In few cases, it is reported to originate as a branch of the parieto-occipital artery. It courses deep in the fissure to reach the occipital pole, giving branches both to the cuneus (above the fissure) and the lingual gyrus (below the fissure) [2, 4].

The Splenial Artery

The last branch described in this chapter is the *splenial artery* for the splenium of the corpus callosum. It arises from the following arteries: parieto-occipital (62%), calcarine (12%), medial posterior choroidal (8%), posterior temporal (6%), P2P (4%), P3 (4%), and the lateral posterior choroidal (4%). As interest, the splenial artery made a loop close to the extra-ventricular part of the hippocampal tail and can give off multiple vessels to this structure [2, 4, 60].

The Lateral Convexity Branches

Finally, it needs to be mentioned the presence of *lateral convexity* branches which originate from one of the cortical arteries described above and passing around the lateral margin of the hemispheres to reach the lateral surface. They have a diameter range between 0.4 and 0.6 mm and they most frequently arise from the posterior temporal artery or the parieto-occipital artery [4].

Possible Anastomoses

Due to its specific vascularization and the different arteries involved in the blood supply, the hippocampal region represents an important anastomosis between anterior and posterior circulation.

- The uncal sulcus was found to be a crucial anastomotic site between the hippocampal branches of the AChA and the hippocampal branches of the PCA [60].
- A second anastomosis is the one between the splenial or posterior pericallosal artery and the anterior pericallosal artery, a branch of the ACA. It usually develops a few centimeters anterior to the tip of the splenium. Retrograde filling of this artery through the pericallosal artery suggests occlusion of the PCA proximal to the origin of the splenial artery [2, 4, 63].

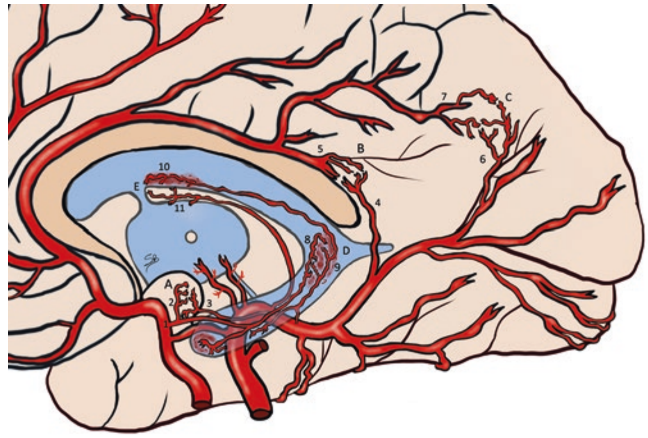


Fig. 3 Major anastomoses of the posterior cerebral artery (PCA). (*A*) Uncal region anastomoses between the hippocampal branch (*2*) of the anterior choroidal artery (*1*) and the hippocampal branch of the PCA (*3*); (*B*) Splenial region anastomoses between the posterior pericallosal artery (*4*) from the PCA and the anterior pericallosal artery (5) from the anterior cerebral artery. (*C*) Pio-pial anastomoses between cortical branches of the PCA (6) and the anterior cerebral artery (*7*) at the watershed region; (*D*) choroidal plexus anastomoses between the anterior choroidal artery (*8*) and the postero-lateral choroidal artery from the PCA (*9*); the choroidal anastomoses at the interventricular foramen (*E*) happen mostly between the postero-medial (*11*) and postero-lateral (*10*) choroidal arteries

- Another important anastomosis is described on the lateral surface of the hemispheres between the lateral convexity branches arising from the cortical branches of the PCA and the superficial temporal arteries or the occipital arteries [4]. Pio-pial or leptomeningeal connections between cortical branches of the PCA, most frequently the parieto-occipital artery, and cortical branches from the ACA are also described at the watershed zone of the two territories [2, 4, 63].

Of mention but related to the choroidal vascularization and therefore treated in detail elsewhere in the book, the medial posterior choroidal artery and lateral posterior choroidal artery, originating from the P1 and P2 anastomose with the branches of the anterior choroidal artery and the anterior cerebral artery with terminal branches of the anterior pericallosal artery [2, 4, 63]. The major anastomotic points of the PCA are illustrated in Fig. 3.

The Accessory Arteries

No "official" accessory posterior cerebral arteries are described in literature. Uchino et al. in 2016 propose the name "accessory PCA" to describe an anterior choroidal artery (AChA) that supplies part of the territory of the PCA or "replaced PCA" to describe the vessel that supplies this territory instead of "hyperplastic AChA" [64]. Two further accessory arteries, more "properly" variations, are described in literature related to the PCA:

- The artery of Percheron
- The Uchimura artery

The Artery of Percheron

The artery of Percheron (AoP), discovered in 1973, is a rare anatomic variant of the paramedian thalamic-mesencephalic arteries. This artery arises as a solitary vessel trunk from the P1 segment of the PCA and supplies the bilateral thalami and rostral midbrain [65–70]. The prevalence of the artery of Percheron is unknown [71]; the scarcity of documented case of AoP occlusion is most likely attributed to the rarity of AoP in the population [69]. Few authors described the ischemic stroke in the territory of AoP: occlusion of Percheron causes a bilateral paramedian thalamic infarction with or without midbrain infarction resulting, from a clinical point of view, in a triad of altered mental status, vertical gaze palsy, and memory impairment [65–70].

The Artery of Uchimura

The second variable rare branch of the PCA is the artery of Uchimura, which originates from P2 or P3. It was described by Yushi Uchimura in 1928 considering a variation in which the posterior cerebral artery gives rise to the hippocampal arteries. Erdem and Yasargil in 1993 described that in the 3% of their cases, the hippocampus was predominantly supplied by arteries originating from a variant of the main trunk of the PCA, the so-called Uchimura artery [60].

Parenchymal Territory

Because of its strategic location, the PCA has a significant role in the blood supply of the brain. The PCA and its branches, in fact, provide blood to a large territory derived from mesencephalic, diencephalic, and telencephalic embryologic structures. Respective territories of each PCA branch are listed in Tables 3 and 4 [72].

In this paragraph, we treat the parenchymal territory of the cortical branches. The blood supply from perforating branches is treated in the appropriate chapter (chapter "Perforating Branches of the Posterior Cerebral Artery"). Similarly, the choroidal and ventricular territory of vascularization are described in the related chapter (chapter "Embryology and Variations of the Posterior Choroidal Artery").

Table 3 Cerebral branches of the PCA with their respective parenchymal territory. For the ventricular branches, see chapter "Embryology and Variations of the Posterior Choroidal Artery" and for the central perforating branches see chapter "Embryology and Anatomy of the Posterior Communicating Artery and Basilar Artery"

PCA branches	Origin from the PCA (segment, most frequent)	Territory
Cerebral branches		
The inferior temporal group		
Hippocampal a.	P2A–P2P	The hippocampus
Anterior temporal a.	P2A	The anteroinferior surface if the temporal lobe and, occasionally, the temporal pole
Middle temporal a.	P2A–P2P	The inferior surface of the temporal lobe
Posterior temporal a.	P2P	The inferior surface of the temporal lobe, the occipital pole, and the lingual gyrus
Common temporal a.	P2P	If present, most of the inferior surface of the temporal and occipital lobes
The parieto-occipital a.	P2P–P3	Posterior parasagittal region, cuneus, precuneus, lateral occipital gyrus, and, rarely, the precentral and superior parietal lobules
The calcarine a.	P3	Primary visual cortex
The splenial a.	Parieto-occipital artery	The splenium of the corpus callosum
The lateral convexity branches	Posterior temporal artery–parieto-occipital artery	The lateral surface of the hemisphere

- Regarding the cerebral branches, Rhoton et al. gave a detailed description of the territory according to his branches classification and the different pattern proposed in particular regarding the inferior temporal group of arteries [2, 4]. This group is responsible for the blood supply of the inferior parts of the temporal lobe. Usually, all the inferior temporal branches (hippocampal and anterior, middle, and posterior temporal arteries) participate in the vascularization. However, in case of a single trunk, named common temporal artery, the entire inferior temporal lobe and part of the occipital lobe are supplied by this large vessel [4].

Table 4 Branches of the PCA with their possible anastomoses and territories involved

PCA branches	Origin from the PCA	Territory	Possible anastomosis
Hippocampal arteries	P2A–P2P	Hippocampus	Hippocampal arteries of the AChA
Splenial or posterior pericallosal artery	Parieto-occipital artery	Splenium of the corpus callosum	Anterior pericallosal artery of the ACA
Lateral convexity branches	Posterior temporal artery–parieto-occipital artery	Lateral surface of the hemispheres	Superficial temporal arteries or the occipital arteries

- Regarding the territory of vascularization of the hippocampal artery, the uncus and the head of the hippocampus are supplied by the anterior hippocampal arteries, while the body and tail of the hippocampus by the posterior hippocampal arteries [59]. An anatomical study by Erdem and Yasargil on the vascularization of the hippocampus (1993) confirms that the PCA, directly or by its branches, contributes much more to the blood supply of the hippocampal formation than the AChA [60]. However, even if rarely, the PCA may leave part of its forebrain territories in case of hypertrophy of the anterior choroidal artery [37].
- The anterior temporal artery supplies the anteroinferior surface of the temporal lobe and, in 6% of the specimens analyzed in the series of Zeal and Rhoton, even a portion of the temporal pole [2].
- A contribution to the blood supply of the inferior surface of the temporal lobe is provided also by the middle temporal artery and the posterior temporal artery. The latter also reaches the occipital pole and the lingual gyrus [2, 4].
- The parieto-occipital artery supplies the posterior parasagittal region, cuneus, precuneus, lateral occipital gyrus, and, rarely, the precentral and superior parietal lobules. Sometimes, also midbrain, thalamus, pulvinar, lateral geniculate bodies, and the choroid plexus can receive a contribution from this artery [2, 4, 61, 62].
- The primary visual cortex is supplied mainly by the calcarine artery but can also receive blood from the parieto-occipital artery [27, 61, 73, 74].
- Several branches of the PCA can give blood supply to the splenium of the corpus callosum including branches from the parieto-occipital, the calcarine, the medial posterior choroidal, the posterior temporal, and the lateral posterior choroidal. The splenial artery, electively, supplies the splenium [2, 4].

Although this commonly used description, some authors such as Zwan et al., studied the variability of the territories and observed one case in which, the PCA, did not even extend to the occipital pole [75]. Interestingly, this variation was already described by Beevor [14, 75]. Of interest, historically, Duret was the first who reported in 1874 that the PCA partly supplies even the head of the caudate nucleus [75].

Clinical Implications

Posterior cerebral arteries supply the midbrain, thalamus, medial temporal area, a part of the parietal lobe, and the occipital lobe. Accidental occlusion or injury of the PCA can cause infarction of the cited regions and lead to different several clinical syndromes according to the location of infarction [76, 77]. In fact, in literature, authors described the stroke as the biggest PCA disease [43, 74, 76–82].

Infarction in the PCA territories is commonly caused by embolism, either cardiogenic or from a more proximal vertebrobasilar intra-arterial clot of unknown source. In contrast, PCA stenosis due to atherosclerosis is an uncommon cause [80].

Historically, the syndrome of infarction in the PCA territory was first described by the French neurologist Charles Foix in 1923 [16, 81]. However, the frequency of PCA territory infarcts in stroke registries is not more than 5–10% and, subsequently, clinical features and causes have not been studied as extensively as in other vascular territories [43, 81].

The PCA syndrome includes more clinical signs varying from headache and ocular disturbance to sensorimotor abnormalities and neuropsychological symptoms [43, 78, 81].

Headache is known to be more frequent with infarcts in the posterior than the anterior circulation but, in the PCA stroke, is described by patients as severe or pulsating. Other initial symptoms of PCA territory stroke may include dizziness, confusion, nausea, and vomiting [43, 81].

Regarding the visual symptoms, the most frequent finding is contralateral hemianopsia; it is in most cases complete, but an upper quadrantanopsia is also found occasionally; isolated lower quadrantanopia is rare. When the central portion of the visual field is spared, it is known as "macular-sparing" visual defect.

A sensory deficit occurred in one-third only of cases and, when present, varied in severity from slight to virtually complete anesthesia of face, arm, and leg [43, 78].

Neuropsychological deficits with PCA ischemia are mostly described in case studies; most common are language-related disorders as dysphasia, dyslexia, dyscalculia, and color dysnomia with PCA infarcts in the dominant hemisphere [81]. Bilateral occlusion of the vessel to the medial temporal area supplied by the hippocampal artery may cause a severe memory loss and a deficit resembling Korsakoff's syndrome [2, 4].

Less frequent are disorders of visual cognitive functions with spatial disorientation, dyschromatopsia, visual agnosia including prosopagnosia, and palinopsia in patients with non-dominant hemisphere infarcts [81]. Depression and obsessive thinking were prominent in several patients with involvement of the dominant hemisphere [43].

Summarizing, unilateral PCA infarct can lead to contralateral homonymous hemianopia with macular sparing if only the occipital lobe is involved. There is association with alexia without agraphia if the splenium of the corpus callosum is also injured. Unilateral PCA infarct of the ventral occipital plus the infra-calcarine cortex leads to achromatopsia plus superior quadrantanopia. The latter alone is caused, instead, by the involvement of the infra-calcarine cortex, the Meyer loop, and the temporal lobe. The inferior quadrantanopia is due to the infarction of the supra-calcarine cortex and the optic radiation [83, 84].

The rare case of bilateral PCA infarct involving both striate and parastriate cortices of the occipital lobes can provoke the classic Anton-Babinski syndrome with impairment of the interpretation of vision. The patient is unaware of his visual loss and denies its presence. Formally, the syndrome includes cortical blindness with denial of blindness, visual hallucinations, and confabulations [73, 84, 85].

PCA and Aneurysms

Aneurysms of the PCA are unusual; the most frequent presentation was with subarachnoid hemorrhage (SAH) in 76% of the series of Rhoton [2, 4]. Their surgical treatment demands great care for the central perforating branches of this vessel irrigate the midbrain and thalamus while the main trunk nourishes the optic radiation and visual cortex [86]. There are very few reports of surgical treatment of posterior cerebral aneurysms except for isolated cases. Although there was no specific clinical picture for the distal aneurysms, each of the proximal saccular aneurysms were associated with a third nerve paresis and controlateral hemiparesis [86–88].

In the Rhoton series, the most frequent focal deficit with a PCA aneurysm was a partial or complete oculomotor nerve deficit in 45% of cases, motor weakness in 28%, a combination of unilateral oculomotor nerve and a contralateral corticospinal deficit (Weber's syndrome) in 23%, and **hemianopsia** in 6% [2]. Other presenting signs included difficulty with upward gaze (Parinaud's syndrome) and paresis of the trochlear, facial, and abducens nerve [2, 4].

References

1. Zeal AA, Rhoton AL. Proceedings of the 33rd annual sessions of the Forum on Fundamental Surgical Problems, 63rd Clinical Congress of the American College of Surgeons, Dallas, Texas, October 1977. In: Surgical Forum. 1977. pp. 1–588.
2. Zeal AA, Rhoton AL. Microsurgical anatomy of the posterior cerebral artery. J Neurosurg. 1978;48:534–59. https://doi.org/10.3171/jns.1978.48.4.0534.
3. Idowu O, Malomo A, Akang E. Surgical anatomy of the Vertebrobasilar territory and posterior circle of Willis. West Afr J Med. 2011;29:230. https://doi.org/10.4314/wajm.v29i4.68234.
4. Rhoton AL Jr. Rhoton's cranial anatomy and surgical approaches. Philadelphia, PA: Lippincott Williams & Wilkins; 2007.
5. Párraga RG, Ribas GC, Andrade SEGL, De Oliveira E. Microsurgical anatomy of the posterior cerebral artery in three-dimensional images. World Neurosurg. 2011;75:233–57. https://doi.org/10.1016/j.wneu.2010.10.053.
6. Abbie AA. The morphology of the forebrain arteries, with especial reference to the evolution of the basal ganglia. J Nerv Ment Dis. 1935;82:325. https://doi.org/10.1097/00005053-193509000-00034.
7. MacMillan M. Evolution and the neurosciences down-under. J Hist Neurosci. 2009;18:150–96. https://doi.org/10.1080/09647040701662377.
8. Hart MN. The origins of neuroscience. A history of explorations into brain function. J Neuropathol Exp Neurol. 1994;53:545. https://doi.org/10.1097/00005072-199409000-00015.
9. Giménez-Roldán S. Andreas Vesalius and the brain: limitations of De Humani Corporis fabrica libri septem and some comments on the matter. Neurosci Hist. 2020;8:76–86.
10. Vesalius A, Saunders JB, O'Malley CD. The illustrations from the works of Andreas Vesalius of Brussels. 248.
11. Dumitrescu AM, Costea CF, Cucu AI, et al. The discovery of the circle of Willis as a result of using the scientific method in anatomical dissection. Romanian J Morphol Embryol. 2020;61:959–65. https://doi.org/10.47162/RJME.61.3.38.
12. Meyer A, Hierons R. Observations on the history of the 'Circle of willis'. Med Hist. 1962;6:119–30. https://doi.org/10.1017/S0025727300027095.
13. Veras TWR, Elhert GW. Variation of the posterior cerebral artery and its embryological explanation: a cadaveric study. Bol Asoc Med P R. 2014;102:55–8.
14. Beevor CE. On the distribution of the different arteries supplying the human brain. Philos Trans R Soc London Ser B. 1909;200:1–55. https://doi.org/10.1098/rstb.1909.0001.
15. Shellshear JL. The basal arteries of the forebrain and their functional significance. J Nerv Ment Dis. 1924;60:296–7. https://doi.org/10.1097/00005053-192409000-00038.
16. Foix CHP. Les arteres de l'axe encephalique jusqu'au diencephale inclusivement. Rev Neurol. 1925;2:705–39.
17. Williams DJ. The origin of the posterior cerebral artery. Brain. 1936;59:175–80. https://doi.org/10.1093/brain/59.2.175.
18. Alpers BJ, Berry RG, Paddison RM. Anatomical studies of the circle of willis in normal brain. AMA Arch Neurol Psychiatry. 1959;81:409–18. https://doi.org/10.1001/ARCHNEURPSYC.1959.02340160007002.
19. Padget DH. The development of the cranial arteries. Contrib Embryol. 1948;32:207–61.
20. Padget DH. The cranial venous system in man in reference to development, adult configuration, and relation to the arteries. Am J Anat. 1956;98:307–55. https://doi.org/10.1002/aja.1000980302.
21. Seydel HG. The diameters of the cerebral arteries of the human fetus. Anat Rec. 1964;150:79–88. https://doi.org/10.1002/ar.1091500108.

22. Moffat DB. The development of the posterior cerebral artery. J Anat. 1961;95:485–94.
23. Moffat DB. The embryology of the arteries of the brain. Ann R Coll Surg Engl. 1962;30:368–82.
24. Riemenschneider PA, Ecker A. Venographic clues to localization of intracranial masses. Am J Roentgenol radium Ther Nucl. 1954;72:740–53.
25. Riemenschneider PA, Ecker A. Angiographic localization of intracranial masses. South Med J. 1955;48:1139–40. https://doi.org/10.1097/00007611-195510000-00048.
26. Krayenbühl H. Cerebral angiography. 2d ed. Philadelphia, PA: Lippincott; 1968.
27. Margolis MT, Newton TH, Hoyt WF. Cortical branches of the posterior cerebral artery. Anat Radiol Correlat Neuroradiol. 1971;2:127–35. https://doi.org/10.1007/BF00335041.
28. Salamon G. Atlas de la Vascularization Arterielle du Cerveau chez l'Homme. Paris: Sandoz; 1973.
29. Waddington M. Atlas of cerebral angiography with anatomic correlation. Boston, MA: Little, Brown; 1974.
30. Lasjaunias P, Berenstein A. Surgical neuroangiography. Berlin, Heidelberg: Springer; 1987.
31. Saeki N, Rhoton AL. Microsurgical anatomy of the upper basilar artery and the posterior circle of Willis. J Neurosurg. 1977;46:563–78. https://doi.org/10.3171/jns.1977.46.5.0563.
32. Greene EC. Anatomy of the rat. JSTOR. 1935
33. Lazorthes G. Vascularisation et circulation cérébrales. Paris: Masson; 1961.
34. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: a pictorial review. Surg Radiol Anat. 2021;43:961–73. https://doi.org/10.1007/S00276-021-02684-Y.
35. Kathuria S, Gregg L, Chen J, Gandhi D. Normal cerebral arterial development and variations. Semin Ultrasound CT MRI. 2011;32:242–51. https://doi.org/10.1053/j.sult.2011.02.002.
36. Okahara M, Kiyosue H, Mori H, et al. Anatomic variations of the cerebral arteries and their embryology: a pictorial review. Eur Radiol. 2002;12:2548–61. https://doi.org/10.1007/s00330-001-1286-x.
37. Raybaud C. Normal and abnormal embryology and development of the intracranial vascular system. Neurosurg Clin N Am. 2010;21:399–426. https://doi.org/10.1016/j.nec.2010.03.011.
38. Kamath S. Observations on the length and diameter of vessels forming the circle of willis. J Anat. 1981;133:419–23.
39. Van Overbeeke JJ, Hillen B, Tulleken CA. A comparative study of the circle of willis in fetal and adult life. The configuration of the posterior bifurcation of the posterior communicating artery. J Anat. 1991;176:45–54.
40. Riggs HE, Rupp C. Variation in form of circle of willis: the relation of the variations to collateral circulation: anatomic analysis. Arch Neurol. 1963;8:8–14. https://doi.org/10.1001/archneur.1963.00460010024002.
41. Tulleken CAF, Luiten MLFB. The basilar artery bifurcation: Microscopical anatomy. Acta Neurochir. 1987;85:50–5. https://doi.org/10.1007/BF01402371.
42. Cilliers K, Page BJ. Variation and anomalies of the posterior cerebral artery: review and pilot study. Turk Neurosurg. 2019;29:1–8. https://doi.org/10.5137/1019-5149.JTN.21443-17.2.
43. Fisher CM. The posterior cerebral artery syndrome. Can J Neurol Sci. 1986;13:232–9. https://doi.org/10.1017/S0317167100036337.
44. Bartosiak P, Borowski S, Golab B. Variation of the origin, the course and branching of posterior cerebral arteries in man. Folia Morphol (Warsz). 1983;42:165–73.
45. Gunnal SA, Farooqui MS, Wabale RN. Study of posterior cerebral artery in human cadaveric brain. Anat Res Int. 2015;2015:1–10. https://doi.org/10.1155/2015/681903.
46. Krishnamurthy A, Nayak SR, Ganesh Kumar C, et al. Morphometry of posterior cerebral artery: embryological and clinical significance. Romanian J Morphol Embryol. 2008;49:43–5.
47. Caruso G, Vincentelli F, Rabehanta P, et al. Anomalies of the P1 segment of the posterior cerebral artery: early bifurcation or duplication, fenestration, common trunk with the superior cerebellar artery. Acta Neurochir. 1991;109:66–71. https://doi.org/10.1007/BF01405701.
48. Kapoor K, Singh B, Dewan LIJ. Variations in the configuration of the circle of willis. Anat Sci Int. 2008;83:96–106. https://doi.org/10.1111/j.1447-073X.2007.00216.x.
49. Yaşargil MG. Microneurosurgery. Stuttgart: Georg Thieme; 1984.
50. Thiarawat P, Jahromi BR, Kozyrev DA, et al. Are fetal-type posterior cerebral arteries associated with an increased risk of posterior communicating artery aneurysms? Clin Neurosurg. 2019;84:1306–12. https://doi.org/10.1093/neuros/nyy186.
51. He Z, Wan Y. Is fetal-type posterior cerebral artery a risk factor for intracranial aneurysm as analyzed by multislice CT angiography. Exp Ther Med. 2018;15:838–46. https://doi.org/10.3892/etm.2017.5504.
52. Bharatha A, Aviv RI, White J, et al. Intracranial arterial fenestrations: frequency on CT angiography and association with other vascular lesions. Surg Radiol Anat. 2008;30:397–401. https://doi.org/10.1007/s00276-008-0340-7.
53. Parmar H, Sitoh YY, Hui F. Normal variants of the intracranial circulation demonstrated by MR angiography at 3 T. Eur J Radiol. 2005;56:220–8. https://doi.org/10.1016/j.ejrad.2005.05.005.
54. Vlajković S, Vasović L, Trandafilović M, et al. Fenestrations of the human posterior cerebral artery. Childs Nerv Syst. 2015;31:381–7. https://doi.org/10.1007/s00381-014-2597-y.
55. Cooke DL, Stout CE, Kim WT, et al. Cerebral arterial fenestrations. Interv Neuroradiol. 2014;20:261–74.
56. Milisavljević M, Marinković S, Marinković Z, Malobabić S. Anatomic basis for surgical approach to the distal segment of the posterior cerebral artery. Surg Radiol Anat. 1988;10:259–66. https://doi.org/10.1007/BF02107896.
57. Bayrak AH, Senturk S, Akay HO, et al. The frequency of intracranial arterial fenestrations: a study with 64-detector CT-angiography. Eur J Radiol. 2011;77:392–6. https://doi.org/10.1016/j.ejrad.2009.09.015.
58. Hacein-Bey L, Muszynski CA, Varelas PN. Saccular aneurysm associated with posterior cerebral artery fenestration manifesting as a subarachnoid hemorrhage in a child. Am J Neuroradiol. 2002;23:1291–4.
59. Isolan GR, Stefani MA, Schneider FL, et al. Hippocampal vascularization: proposal for a new classification. Surg Neurol Int. 2020;11:378. https://doi.org/10.25259/SNI_708_2020.
60. Erdem A, Yasargil MG, Roth P. Microsurgical anatomy of the hippocampal arteries. J Neurosurg. 1993;79:256–65. https://doi.org/10.3171/jns.1993.79.2.0256.
61. Marinković SV, Milisavljević MM, Lol Ić-Draganić V, Kovačević MS. Distribution of the occipital branches of the posterior cerebral artery correlation with occipital lobe infarcts. Stroke. 1987;18:728–32. https://doi.org/10.1161/01.STR.18.4.728.
62. Kalamatianos T, Mavridis IN, Karakosta E, et al. The Parieto-occipital artery revisited: a microsurgical anatomic study. World Neurosurg. 2019;126:e1130–9. https://doi.org/10.1016/J.WNEU.2019.02.215.
63. Bonasia S, Ciccio G, Smajda S, et al. Angiographic analysis of natural anastomoses between the posterior and anterior cerebral arteries in moyamoya disease and syndrome. Am J Neuroradiol. 2019;40:2066–72.
64. Uchino A, Saito N, Takahashi M, et al. Variations of the posterior cerebral artery diagnosed by MR angiography at 3 tesla. Neuroradiology. 2016;58:141–6. https://doi.org/10.1007/s00234-015-1614-5.
65. Anghelescu A. Uncommon association of two anatomical variants of cerebral circulation: a fetal-type posterior cerebral artery and inferred artery of Percheron, complicated with parame-

dian thalamomesencephalic stroke—case presentation and literature review. Case Rep Neurol Med. 2018;2018:1–9. https://doi.org/10.1155/2018/4567206.
66. Lazzaro NA, Wright B, Castillo M, et al. Artery of percheron infarction: imaging patterns and clinical spectrum. Am J Neuroradiol. 2010;31:1283–9. https://doi.org/10.3174/ajnr.A2044.
67. O'Reilly MK, Mogensen MA. Artery of Percheron stroke: imaging and clinical findings. PM R. 2019;11:1135–8. https://doi.org/10.1002/pmrj.12210.
68. Stamm BJ, Lineback CM, Skolarus LE, et al. Artery of percheron infarct: 12 cases and their complex clinical courses. Neurohospitalist. 2018;8:141–5. https://doi.org/10.1177/1941874417748543.
69. Li X, Agarwal N, Hansberry DR, et al. Contemporary therapeutic strategies for occlusion of the artery of Percheron: a review of the literature. J Neurointerv Surg. 2015;7:95–8. https://doi.org/10.1136/neurintsurg-2013-010913.
70. Percheron G. The anatomy of the arterial supply of the human thalamus and its use for the interpretation of the thalamic vascular pathology. Z Neurol. 1973;205:1–13. https://doi.org/10.1007/BF00315956.
71. Lamot U, Ribaric I, Popovic KS. Artery of Percheron infarction: review of literature with a case report. Radiol Oncol. 2015;49:141–6. https://doi.org/10.2478/raon-2014-0037.
72. Cereda C, Carrera E. Posterior cerebral artery territory infarctions. Front Neurol Neurosci. 2012;30:128–31. https://doi.org/10.1159/0003336104.
73. Das JM, Naqvi IA. Bahrain Specialist Hospital, Juffair, Bahrain, Temple University, Anton syndrome. StatPearls; 2019. p. 3–7.
74. Javed K, Reddy V, Das JM. Neuroanatomy, posterior cerebral arteries. StatPearls; 2020. p. 1–8. https://pubmed.ncbi.nlm.nih.gov/?term=Javed+K&cauthor_id=30860709
75. van der Zwan A, Hillen B, Tulleken CAF, Dujovny M. A quantitative investigation of the variability of the major cerebral arterial territories. Stroke. 1993;24:1951–9. https://doi.org/10.1161/01.STR.24.12.1951.
76. Lee E, Kang DW, Kwon SU, Kim JS. Posterior cerebral artery infarction: diffusion-weighted MRI analysis of 205 patients. Cerebrovasc Dis. 2009;28:298–305. https://doi.org/10.1159/000229016.
77. Kim JS, Caplan LR. Clinical stroke syndromes. Front Neurol Neurosci. 2016;40:72–92. https://doi.org/10.1159/000448303.
78. Fisher CM. Unusual vascular events in the territory of the posterior cerebral artery. Can J Neurol Sci. 1986;13:1–7. https://doi.org/10.1017/S031716710003571X.
79. Servan J, Verstichel P, Catala M, et al. Aphasia and infarction of the posterior cerebral artery territory. J Neurol. 1995;242:87–92. https://doi.org/10.1007/BF00887822.
80. Pessin MS, Kwan ES, DeWitt LD, et al. Posterior cerebral artery stenosis. Ann Neurol. 1987;21:85–9. https://doi.org/10.1002/ana.410210115.
81. Brandt T, Steinke W, Thie A, et al. Posterior cerebral artery territory infarcts: clinical features, infarct topography, causes and outcome. Cerebrovasc Dis. 2000;10:170–82. https://doi.org/10.1159/000016053.
82. Strambo D, Bartolini B, Beaud V, et al. Thrombectomy and thrombolysis of isolated posterior cerebral artery occlusion: cognitive, visual, and disability outcomes. Stroke. 2020;51:254–61. https://doi.org/10.1161/STROKEAHA.119.026907.
83. Mark S, Greenberg MD. Handbook of neurosurgery. 7th ed [Reviewed by Edward M. Cohn, MD]. Thieme Medical Publishers; 2010. ISBN: 978-1-60406-326-4.
84. Lindsay KW, Bone I, Fuller G. Neurology and neurosurgery illustrated. 5th edition. 2010
85. Aldrich MS, Alessi AG, Beck RW, Gilman S. Cortical blindness: etiology, diagnosis, and prognosis. Ann Neurol. 1987;21:149–58. https://doi.org/10.1002/ana.410210207.
86. Drake CG, Amacher AL. Aneurysms of the posterior cerebral artery. J Neurosurg. 1969;30:468–74. https://doi.org/10.3171/jns.1969.30.4.0468.
87. Hunt WE, Hess RM. Aneurysm of the posterior cerebral artery with unexpected postoperative neurological deficit. Case report. J Neurosurg. 1967;26:633–5. https://doi.org/10.3171/jns.1967.26.6.0633.
88. Weibel J, Fields WS, Campos RJ. Aneurysms of the posterior cervicocranial circulation: clinical and angiographic considerations. J Neurosurg. 1967;26:223–34. https://doi.org/10.3171/jns.1967.26.2.0223.

Perforating Branches of the Posterior Cerebral Artery

F. Marchi, Sara Bonasia, and Thomas Robert

The perforating vessels are terminal vessels which, normally, represent specific small branches of the main cerebral arteries. The perforating branches of the vertebrobasilar system derive from the posterior-inferior cerebellar artery (PICA), the basilar artery (BA), the anterior-inferior cerebellar artery (AICA), the superior cerebellar artery (SCA), and the posterior cerebral artery (PCA), and they supply the paramedian region of the brainstem, the diencephalon, and the central part of the cerebral hemisphere [1–5]. During neurosurgical procedures, despite the use of the microscopic, these small vessels are at risk of damage both for their dimension and for their deep location [2]. Considering the territories of vascularization of the perforating arteries, their damage or occlusion during the surgical approaches can provoke deep intracerebral infarcts and catastrophic event [6–9].

Consequently, the anatomy of the deep cerebral perforators and their high variability is becoming, more and more, a topic of great interest and debate in the scientific literature [6].

However, regarding the specific topic of this chapter, many authors have described anatomic features of the posterior cerebral arteries and their branches, but only a few reports studied exclusively the perforating branches of the PCA [2, 10, 11].

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History

In the second half of the twentieth century, various attempts have been performed in order to define the precise courses and territories of distribution of the perforating arteries arising from the circle of Willis [6]. Duret and Heubner, almost at the same time, described first the thalamoperforating arteries and named them as "optic arteries" [12]. Lazorthes referred them as inferior thalamic arteries [13], while Percheron as thalamo-subthalamic arteries [14]. Rhoton et al. described, in their anatomical cadaveric series in 1977–1978, the PCA anatomy and the perforators arising from this vessel. Even if other authors, in the course of time, have given their contribution to the description of these vessels, the classification proposed by Rhoton remains the most complete and precise in literature and the one we follow in this chapter [1, 15–18].

Although fundamental aspects on the basic anatomy of these vessels have been achieved in the last decades, the very limited number of dissected hemispheres could not take into account the high variability typical for the deep perforators [6]. According with a recently published meta-analysis by Vogels V. et al. in 2021, of a total of 2715 articles screened, 53 were included of whom 11 only dealing with the posterior cerebral artery perforators [6].

Certainly, a helpful contribution in the better understanding of these vessels has been given by the advent of the operative microscope and the development of the angiography. Nevertheless, because of their small diameters, these tiny branches are inconstantly visualized even at the angiographic studies. New perspectives in the description of the perforators are emerged thanks to the improvement of the precision in the MRI sequences (for example, TOF, time-of-flight sequences) such as those performed with 7 Tesla machines [6, 15].

However, to date, intracranial surgery remains the best technique in order to evaluate and describe more precisely the cerebral anatomy of the perforators and their relationship

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with the surrounding structures. Efforts to improve their visualization pre-operatively should be prioritized in the future [6].

Embryological Development

If the description of the adult configuration of the perforating branches of the PCA remains not deeply known, even harder is the precise embryological development of these variable and tiny vessels.

In general, from an embryological point of view, it is not infrequent that perforators deriving from the same vessel, in the adult configuration, may present a large variability in terms of assignment to a perforator group [6].

The studies from Padget or Moffat did not describe specifically the embryological development of the perforating branches of the PCA [19–21]. However, as mentioned in the previous chapter in the paragraph on the embryology of the PCA, at Padget stage 5, the development of the latter starts as the caudal segment of the posterior communicating artery (PComA). From this stage until the end of the development, the perforators branches originate variably and inconstantly mainly from P1 and P2 [22].

The studies from Lasjaunias have been addressed mainly to the perforators arising from the anterior circulation or the basilar artery and the PICA [22]. However, he noticed that a collicular group of arteries originate from the distal portion of the caudal division of the internal carotid artery (ICA) (Pl-BA junction including the superior cerebellar artery, SCA) [22, 23]. In this context, these collicular or long circumflex arteries can be considered embryologically derived from the mesencephalic artery of Moffat's embryo while the additional branches including the medial choroidal artery from the diencephalic system of Moffat's embryo [7, 8, 20, 22, 23].

It remains unclear how the evolution of the original perforators present in the embryo interact with the neuro-embryogenesis of structures such as the striatum or median ganglionic eminence dictating the eventual anatomy in the adult, being necessary further research on this topic [6].

Number and Origin of the Artery

PCA gave origin to three type of perforator arteries and their branches (Table 1):

1. *Central branches* to the brain stem
   (a) direct perforating arteries
      (i) Thalamoperforating arteries (TPA)
      (ii) Thalamogeniculate arteries (TGA)
      (iii) peduncular perforating arteries
   (b) Circumflex perforating arteries
      (i) Short perforating arteries
      (ii) Long perforating arteries
2. *Ventricular branches* (treated in chapter "Embryology and Variations of the Posterior Choroidal Artery")
3. *Cerebral branches* (treated in chapter "Cortical and Perforating Branches of the Middle Cerebral Artery")

Table 1 Central branches of the PCA. For the ventricular branches see chapter "Embryology and Variations of the Posterior Choroidal Artery" and for the cerebral branches see chapter "Cortical and Perforating Branches of the Middle Cerebral Artery"

PCA branches	Origin from the PCA (segment, most frequent)	Territory
Direct perforating arteries		
Thalamoperforating a. (TPA)	P1	Anterior and part of posterior thalamus, hypothalamus, subthalamus, substantia nigra, red nucleus, oculomotor nerve, mesencephalic reticular formation, pretectum, rostromedial floor of fourth ventricle, and posterior portion of the internal capsule
Thalamogeniculate a. (TGA)	P2	Posterior half of the lateral thalamus, posterior limb of the internal capsule and the optic tract
Peduncular perforating a.	P2A	Corticospinal and corticobulbar pathways as well as the substantia nigra, red nucleus, and other structures of the tegmentum, and the oculomotor nerve
Circumflex perforating arteries		
Short perforating a.	P1	Geniculate bodies and the tegmentum
Long perforating a.	P2A	Quadrigeminal bodies

The Central Branches

The central branches (Fig. 1), treated in this chapter, are divided into *direct* and *circumflex perforating arteries*. The direct perforators include the *thalamoperforating* (TPA), the *peduncular perforating*, and the *thalamogeniculate* (TGA) arteries. The circumflex perforators are divided into *short* and *long* perforating arteries [7–9].

The Direct Perforating Arteries

The PCA perforating branches arise mainly from the P1 and P2 segments. The average number of direct perforating arteries arising from each P1 was 2.7 in Zeal and Rhoton's study but they also observed some cases of absence of

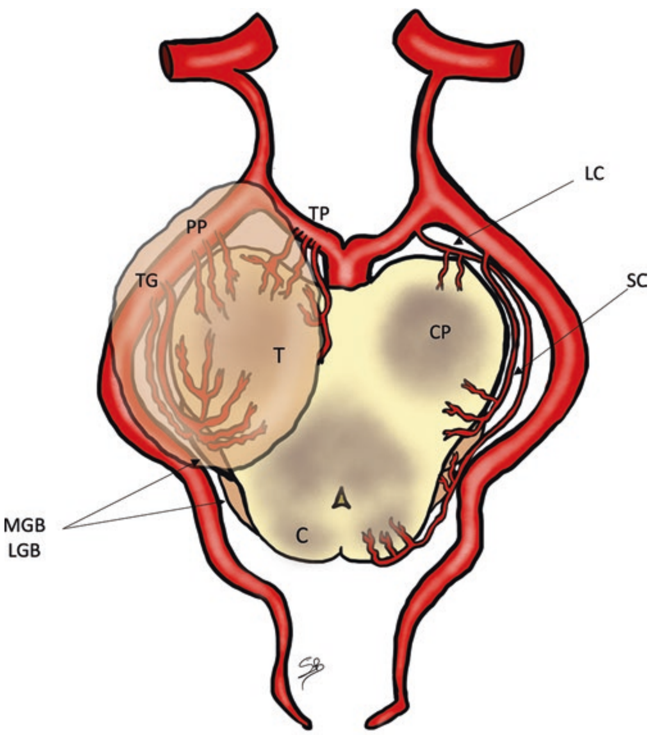

Fig. 1 Illustration of the central branches of the posterior cerebral artery (PCA). Thalamoperforating arteries (TP); peduncular perforating arteries (PP); thalamogeniculate arteries (TG); long circumflex arteries (LC); short circumflex arteries (SC); T: thalamus; CP: cerebral peduncle; C: colliculi; MGB, LGB: medial and lateral geniculate bodies

branches arising from P1 segments (2% of the series) [8, 9]. As noted by Lasjaunias, in that situation the contralateral Pl gives the branches for the ipsilateral and contralateral territories [8, 22].

The Thalamoperforating Arteries (TPA)

Among the direct perforators, the *thalamoperforating* arteries (*TPA*) are the major branches [1] and arise most often between the basilar bifurcation and the origin of the posterior communicating artery, specifically from the middle-third of P1 [2, 7–9]. In fact, Saeki, Zeal, and Rhoton observed in their studies that the TPAs originated on the medial 1 mm of P1 in 8% of the specimens, and on the lateral 1 mm in 5%, but arising, in the majority of cases, on the central part of P1 [2, 7–9]. In one specimen, described by Kaya in 2010, the TPA started out from a fenestrating branch between the right and the left P1 segment [11]. Regarding their number, the thalamoperforating arteries are described in a range from 2 to 5 (mean 3.1) by Dujelic et al. in a study published in 2015, and in a range from 0 to 10 (median 3) in the overview by Vogels et al. in 2021 in which 128 hemispheres were included [2, 6].

The Thalamogeniculate Arteries (TGA)

According with the data in literature, the *thalamogeniculate* arteries mainly originate directly from the P2, beneath the lateral thalamus. They usually arise close to the junction between the crural (P2A) and the ambient (P2P) segments, with an equal number of branches from each segment [7–9]. Nevertheless, some exceptions need to be noted: Djulejic et al. [2], for example, noted that the TGAs originated by the medial posterior choroidal artery (MPChoA) in 59.4% of cases, while Milisavljevic et al. [24] and Vogels et al. [6] founded some cases in which the origin occurred from the P3 segment (in one and two cases, respectively). The number of TGAs can be variable as well: normally in number of two or three, they can range between 2 and 12 with a median of 5 [6, 24].

The Peduncular Arteries

The third group of direct perforators are the *peduncular arteries*. Usually in number of two or three (median 2.8), they have been described in a range from 0 to 6 according to all authors and without particular divergence [6–9, 15, 24]. The peduncular arteries arise, according to Rhoton, from P2 segment and, specifically, from P2A in the 94% of the cases and from P2P in the remnant. Some cases of P1 origin have been described [6–16].

The Circumflex Perforating Arteries

The *central perforating arteries* include the *circumflex group* of arteries which arise from the P1 and the P2 segments. As mentioned above, they are differentiated as short and long circumflex arteries. The former, which reach only the geniculate bodies, are referred to as *short circumflex arteries* (1.2.1); the latter, reaching the culliculi, are called *long circumflex arteries* (1.2.2). The firsts arising mainly from P2 and were present in the 66% of the hemispheres of the Rhoton series [6–8]. Kaya et al., however, described cases in which the origin occurred at P1 [11]. The long circumflex arteries, instead, were present in the 96% of the hemispheres analyzed by Rhoton, usually arising from the P1 segment (80%), but occasionally also from the P2A (20%) [6–8]. Regarding the number, the short circumflex arteries range from 0 to 3, while the long ones result to be variable between 0 and 2 [6].

Course of the Branches

Generally, the main difference in the course between the direct perforating and the circumflex branches can be identified in the fact that the former pass directly from the parent trunk to the brainstem, while the latter encircle the brainstem for a variable distance before entering the diencephalon and mesencephalon. Their specific courses are treated in detail in this paragraph [6–8].

- The *thalamoperforating branches* enter the brain by passing through the posterior perforating substance and terminate predominantly in the interpeduncular fossa [7, 25], posterior perforating substance [7, 26, 27], or the retromamillary area [7, 28]. Their branches also terminated in the posterior hypothalamus and the medial portion of the upper midbrain [7].

- The *thalamogeniculate arteries* arise directly from the P2 and pass through the geniculate bodies and adjacent part of the roof of the ambient cistern. Some branches originating from the circumflex arteries and from the medial posterior choroidal arteries can reach the same region, but they typically encircle the brainstem instead of arising directly from the P2 segment. The TGA meet the TPA near the middle of the thalamus and the premamillary branch of the PComA anteriorly in the lateral nucleus [7–9].
- The *peduncular perforating branches* pass directly from the PCA into the cerebral peduncle.

As suggested by their name, the *circumflex arteries* "encircle" the midbrain parallel and medial to the PCA [7].

- The *short branches* pass medial to P2, the medial posterior choroidal artery and the long circumflex arteries, reaching the posterolateral border of the peduncle in the 76% of the specimens, the tegmental region in the 11%, and the medial geniculate bodies and the tegmentum in the remaining cases [7–9, 11].
- The *long circumflex* arteries encircle the midbrain medial to the PCA and lateral to the short circumflex arteries reaching the quadrigeminal plate. Five or more small branches are sent to the peduncle, the geniculate bodies, and the tegmentum [8, 9].

Possible Anastomoses

Despite the limitations in the precise description of the anatomy of the small vessels treated in this chapter and, consequently, their possible anastomoses, more and more studies support the idea of a collateral circulation with anastomoses between the different perforator groups [2, 6, 10, 15, 24].

In a specific study on the anastomoses among the thalamoperforating arteries, Marinkovic et al. (1986) observed that in the 79% of the cases there were anastomotic channels, that their main diameter was 148 μmm and an average length of 3.3 mm [10]. In their study, they proposed a classification into five groups. These different groups are summarized in Table 2 and illustrated in Fig. 2.

- The first group comprised anastomoses among the branches of single large TPA of the PCA through two collateral perforating branches, or one terminal and one collateral, or between one collateral and one peduncular branch arising from the same thalamoperforating artery.
- Anastomoses within the second group connected individual TPAs of the same PCA.
- The third group included anastomoses among the TPA and peduncular branches, or paramedian mesencephalic or pontine branches of the basilar or superior cerebellar arteries.
- In the fourth group are observed connections between the peduncular branches of the PCA and the mesencephalic or pontine branches of the ipsilateral superior cerebellar and basilar arteries.
- The fifth group comprised the anastomoses connecting the TPA with the contralateral vessels such as the other TPA, the peduncular branches, branches to the mamillary bodies, and the mesencephalic or pontine branches of the basilar and superior cerebellar arteries on the opposite side. The cross anastomoses (right-left) were the most frequent ones, described in the 56.9% of the cases [10, 24].

Regarding the other central perforating branches of the PCA, Djulecic et al. observed that the anastomoses of the thalamogeniculate branches of the PCA were present in the 40.6% of the specimens, while, for Milisavljevic even in the 66.7% of the hemispheres analyzed [2]. For the latter, the anastomoses were most frequent between the TGA vessels

Table 2 Group classification anastomoses of the perforating arteries of the PCA according with Marinkovic et al. (Adapted from [10])

Group	Origin from the PCA perforating	Possible anastomosis
I	Thalamoperforating artery (TPA)	Branches of the same TPA
II	Thalamoperforating artery (TPA)	TPAs of the same PCA
III	Thalamoperforating artery (TPA)	TPA and peduncular arteries or branches of the basilar or the SCA
IV	Peduncular arteries	Peduncular arteries and branches of the ipsilateral basilar artery or SCA
V	Thalamoperforating artery (TPA)	TPA and contralateral vessels: TPA, peduncular arteries, branches to the mammillary bodies, branches of the basilar or the SCA

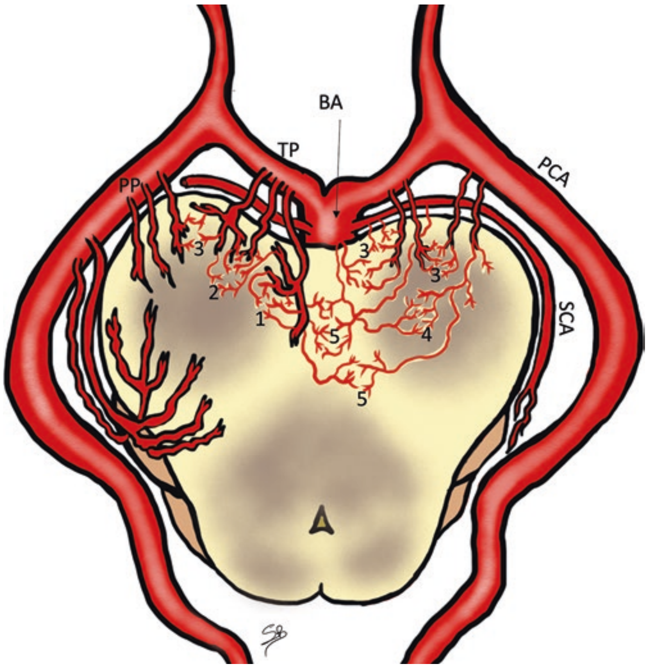

Fig. 2 Group classification anastomoses of the perforating arteries of the PCA according with Marinkovic et al. (Adapted from [10]). *1*: anastomoses between branches of the same thalamoperforating artery (TP); *2*: anastomoses between branches of different TP arteries coming from the same posterior cerebral artery (PCA); *3*: anastomoses between TP arteries and peduncular arteries (PP) or branches of the basilar artery (BA) or the superior cerebellar artery (SCA) on the same side; *4*: anastomoses between PP and branches of the ipsilateral BA or SCA; *5*: anastomoses between TP and contralateral vessels: TPA, PP, branches of the BA or the SCA

and the medial posterior choroidal arteries in 33.3%, or the mesencephalo-thalamic arteries in 26.6% [24].

Parenchymal Territory

The perforators of the PCA supply critical structures such as the interpeduncular fossa, the cerebral peduncles, the posterior perforated substance, the mammillary bodies, the tegmentum, the thalamus, the hypothalamus, the internal capsule, and the deep nuclei of the basal ganglia, brainstem, and quadrigeminal plate. Not surprisingly, an injury to these vessels and the relative territories can provoke devastating effects [8, 11, 17].

- The thalamoperforating arteries supply the anterior and part of posterior thalamus, hypothalamus, subthalamus, substantia nigra, red nucleus, oculomotor nerve, mesencephalic reticular formation, pretectum, rostromedial floor of fourth ventricle, and the posterior portion of the internal capsule [8, 27, 29–32].
- The thalamogeniculate arteries supply the posterior half of the lateral thalamus, posterior limb of the internal capsule, and the optic tract [7–9, 13, 31].
- The peduncular perforating arteries supply the corticospinal and corticobulbar pathways, as well as the substantia nigra, red nucleus, and other structures of the tegmentum, and may send branches to the oculomotor nerve [7–9].

The short circumflex branches supply the geniculate bodies and the tegmentum, while the long circumflex supply the quadrigeminal bodies. Some authors state that the long circumflex arteries supply the superior colliculi, and that the inferior colliculi are supplied by the superior cerebellar artery [7–9, 13, 18].

Clinical Implications

From a clinical perspective, perforators anatomy is essential for understanding the pathogenesis and consequences of ischemic and hemorrhagic stroke. These branches of the PCA are extremely vulnerable to injuries both during endovascular and, particularly, microsurgical procedures, such as the treatment of basilar apex aneurysm [6, 11]. Unfortunately, to date, the injury of a single small vessel tends to be missed by the imaging modalities in use and ischemic events from pathologies such as small vessel disease and lacunar stroke may present at times with negative imaging findings. Even if arterial occlusion is the most common cause of a typical thalamic syndrome, vascular malformations or tumors of the thalamus can be rare but possible causes [6, 8, 33, 34].

- The TPA occlusion may produce a variety of syndromes, depending on the extension of the area of ischemia. The injury to this vessel is characterized by contralateral hemiplegia, cerebellar ataxia, or a “rubral” tremor associated with ipsilateral oculomotor nerve paresis (Nothnagel’s syndrome or Benedikt’s syndrome). Typically, contralateral hemiballismus is caused by lesions affecting the subthalamus [8].
- TGA occlusion results in the thalamic syndrome of Dejerine and Roussy, consisting of a contralateral loss of sensation with an intractable, “hyperpathic” pain with hypersensitivity to mild touch, pain, and temperature stimuli, associated with contralateral hemiplegia, choreoathetoid or dystonic movements, and homonymous hemianopsia [8, 35].
- The injury to the long circumflex branches can provoke infarction of the posterior commissure or of the nuclei of Darkschewitsch or Cajal resulting in the Parinaud’s syndrome with the classical triad of impaired upward gaze, convergence retraction nystagmus, and pupillary hyporeflexia [9, 36].

In conclusion, because of the clinical implications above exposed, extensive knowledge of the microsurgical anatomy of this area is mandatory to prevent catastrophic events and poor post-operative outcomes.

References

1. Lucifero AG, Baldoncini M, Bruno N, et al. Microsurgical neurovascular anatomy of the brain: the posterior circulation (part ii). Acta Biomed. 2021;92:e2021413. https://doi.org/10.23750/abm.v92iS4.12119.
2. Djulejić V, Marinković S, Milić V, et al. Common features of the cerebral perforating arteries and their clinical significance. Acta Neurochir. 2015;157:743–54. https://doi.org/10.1007/s00701-015-2378-8.
3. Marinković S, Gibo H, Milisavljević M. The surgical anatomy of the relationships between the perforating and the leptomeningeal arteries. Neurosurgery. 1996;39:72–83. https://doi.org/10.1097/00006123-199607000-00016.
4. Rosner SS, Rhoton AL, Ono M, Barry M. Microsurgical anatomy of the anterior perforating arteries. J Neurosurg. 1984;61:468–85. https://doi.org/10.3171/jns.1984.61.3.0468.
5. Yaşargil MG. Microneurosurgery. Stuttgart: Georg Thieme; 1984.
6. Vogels V, Dammers R, Van Bilsen M, Volovici V. Deep cerebral perforators: anatomical distribution and clinical symptoms: an overview. Stroke. 2021;52:E660–74. https://doi.org/10.1161/STROKEAHA.120.034096.
7. Saeki N, Rhoton AL. Microsurgical anatomy of the upper basilar artery and the posterior circle of Willis. J Neurosurg. 1977;46:563–78. https://doi.org/10.3171/jns.1977.46.5.0563.
8. Zeal AA, Rhoton AL. Microsurgical anatomy of the posterior cerebral artery. J Neurosurg. 1978;48:534–59. https://doi.org/10.3171/jns.1978.48.4.0534.
9. Rhoton AL Jr. Rhoton's cranial anatomy and surgical approaches. Philadelphia, PA: Lippincott Williams & Wilkins; 2007.
10. Marinkovic SV, Milisavljevic MM, Kovacevic MS. Anastomoses among the thalamoperforating branches of the posterior cerebral artery. Arch Neurol. 1986;43:811–4. https://doi.org/10.1001/archneur.1986.00520080053020.
11. Kaya AH, Dagcinar A, Ulu MO, et al. The perforating branches of the P1 segment of the posterior cerebral artery. J Clin Neurosci. 2010;17:80–4. https://doi.org/10.1016/j.jocn.2009.03.046.
12. Grochowski C, Maciejewski R. Diversity among posterior thalamoperforating branches originated from P1 segment: systematic review. Folia Morphol (Warsz). 2017;76:335–9.
13. Lazorthes G, Salamon G. The arteries of the thalamus: an anatomical and radiological study. J Neurosurg. 1971;34:23–6. https://doi.org/10.3171/jns.1971.34.1.0023.
14. Percheron G. The anatomy of the arterial supply of the human thalamus and its use for the interpretation of the thalamic vascular pathology. Z Neurol. 1973;205:1–13. https://doi.org/10.1007/BF00315956.
15. Marinkovic S, Milisavljevic M, Kovacevic M. Interpeduncular perforating branches of the posterior cerebral artery. Microsurgical anatomy of their extracerebral and intracerebral segments. Surg Neurol. 1986;26:349–59. https://doi.org/10.1016/0090-3019(86)90135-7.
16. Párraga RG, Ribas GC, Andrade SEGL, De Oliveira E. Microsurgical anatomy of the posterior cerebral artery in three-dimensional images. World Neurosurg. 2011;75:233–57. https://doi.org/10.1016/j.wneu.2010.10.053.
17. Caruso G, Vincentelli F, Rabehanta P, et al. Anomalies of the P1 segment of the posterior cerebral artery: early bifurcation or duplication, fenestration, common trunk with the superior cerebellar artery. Acta Neurochir. 1991;109:66–71. https://doi.org/10.1007/BF01405701.
18. Lazorthes G. Vascularisation et circulation cérébrales. Paris: Masson; 1961.
19. Padget DH. The development of the cranial arteries. Contrib Embryol. 1948;32:207–61.
20. Moffat DB. The development of the posterior cerebral artery. J Anat. 1961;95:485–94.
21. Moffat DB. The embryology of the arteries of the brain. Ann R Coll Surg Engl. 1962;30:368–82.
22. Lasjaunias P, Berenstein A. Surgical neuroangiography. Berlin, Heidelberg: Springer; 1987.
23. Mercier PH, Brassier G, Fournier HD, et al. Vascular microanatomy of the pontomedullary junction, posterior inferior cerebellar arteries, and the lateral spinal arteries. Interv Neuroradiol. 2008;14:49–58. https://doi.org/10.1177/159101990801400107.
24. Milisavljevic MM, Marinkovic SV, Gibo H, Puskas LF. The thalamogeniculate perforators of the posterior cerebral artery: the microsurgical anatomy. Neurosurgery. 1991;28:523–30. https://doi.org/10.1227/00006123-199104000-00007.
25. Hara K, Fujino Y. The thalamoperforate artery. Acta Radiol Diagn (Stockh). 1966;5:192–200. https://doi.org/10.1177/02841851660050P122.
26. Gillilan LA. The arterial and venous blood supplies to the forebrain (including the internal capsule) of primates. Neurology. 1968;18:653–70. https://doi.org/10.1212/wnl.18.7.653.
27. Margolis MT, Newton TH, Hoyt WF. Cortical branches of the posterior cerebral artery. Anatomic-radiologic correlation. Neuroradiology. 1971;2:127–35. https://doi.org/10.1007/BF00335041.
28. Westberg G. Arteries of the basal ganglia. Acta Radiol Diagn (Stockh). 1966;5:581–96. https://doi.org/10.1177/02841851660050P163.
29. Perlmutter D, Rhoton AL. Microsurgical anatomy of the distal anterior cerebral artery. J Neurosurg. 1978;49:204–28. https://doi.org/10.3171/jns.1978.49.2.0204.
30. Schwartz HG. Arterial aneurysm of the posterior fossa. J Neurosurg. 1948;5:312–6. https://doi.org/10.3171/jns.1948.5.3.0312.
31. Krayenbühl H. Cerebral angiography. 2d ed. Philadelphia, PA: Lippincott; 1968.
32. Salamon G. Atlas de la Vascularization Arterielle du Cerveau chez l'Homme. Paris: Sandoz; 1973.
33. Kim JS, Caplan LR. Clinical stroke syndromes. Front Neurol Neurosci. 2016;40:72–92. https://doi.org/10.1159/000448303.
34. Caplan LR. Lacunar infarction and small vessel disease: pathology and pathophysiology. J Stroke. 2015;17:2–6.
35. Urits I, Gress K, Charipova K, et al. Diagnosis, treatment, and management of Dejerine–Roussy syndrome: a comprehensive review. Curr Pain Headache Rep. 2020;24:48.
36. Ortiz JF, Eissa-Garces A, Ruxmohan S, et al. Understanding parinaud's syndrome. Brain Sci. 2021;11:1469.

Embryology and Anatomy of the Vertebral Artery

M. Pileggi and F. Di Caterino

The vertebral arteries represent, together with the carotid arteries, the main blood supply channels for the intracranial structures. Endowed with a secondary role during fetal life, during which the structures of the posterior fossa are mostly vascularized by the caudal divisions of the internal carotids (posterior communicating arteries), during postnatal life they become the main feeders of numerous structures of the brainstem, cerebellum, thalami and a variable portion of the temporal and occipital lobes. The knowledge of the anatomy of these arteries is essential for neurosurgeons and neurointerventionalists because of their close proximity to the bony structures of the cervical spine, the importance of the nerve structures vascularized by them and the numerous and complex anastomoses that can be observed with the internal and external carotids and the cervical arteries.

History

The father of modern neuroanatomy Thomas Willis already in 1664 described the macroscopic anatomy of the vertebral artery (VA), going so far as to describe the muscular branches and the medullary branches afferent to the anterior spinal artery (ASA) in his book *Cerebri anatome* [1].

Since the mid-eighteenth century, more detailed descriptions of the artery were published and the course of the VA was divided into segments. The two oldest segmentation patterns were described by Powers (1860) and Barbieri (1867) [2].

A new powerful tool for the study was provided by angiography: the first angiogram of the vertebral arteries was performed by Egas Moniz in 1934, by exposing the subclavian artery (SBA; peripherally compressed) and obtaining unsubtracted images of extracranial and intracranial vertebrobasilar system by injection [3].

In 1938 Sjoquist exposed the VA and injected the contrast medium directly into it for the first time, with the goal of producing good quality angiograms and being successful in six cases out of eight [3].

In 1948 Padget [4] demonstrated that the vertebral arteries are established as longitudinal anastomoses between the segmental arteries from the subclavian artery to the craniocervical junction. This concept was further elaborated in the monumental work of Lasjaunias, Berenstein and TerBrugge [5] as a framework to explain the several variations of origin and course of the artery.

Embryological Development

The embryological development of the vertebral artery (VA) occurs between 33 and 38–40 days during intrauterine life and according to the cerebral artery development described by Padget in 1948 [4] different embryological stage can be identified.

At the initial stage of development, there are two aortic trunks (ventral and dorsal aorta for each side) [6] which will evolve to form the carotid system and two arterial channels ventral to the rhombencephalon, one on each side of the midline: the ventral longitudinal neural arteries (vLNAs). This longitudinal neural system will give origin to the VA, the basilar trunk and the spinal artery.

At about 4–5 mm embryo length (≃30 days, Padget stage 1) in the anterior circulation, the primitive internal carotid artery (ICA) can already be recognized with its division in

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two main branches: the cranial branch (future anterior cerebral artery, ACA) and the caudal branch that will become the posterior communicating artery (PcomA) [4, 7, 8]. Instead, in the posterior cerebral region (hindbrain), the two vLNAs appear in the form of plexiform structures in the midline supplied by multiple arterial bridges with the carotid system: the trigeminal artery (TA) and the otic artery (OtA) cranially and the hypoglossal artery (HA) and the first cervical segmental artery (also called proatlantal artery (PA) type I) caudally [4, 7–10].

At about 5–6 mm embryo length (≃31 days, Padget stage 2) the definitive PcomA is formed. The latter replaces the supply of TA to the two vLNAs, resulting in progressive TA involution. The fusion on the midline of the two vLNAs also begins to form the basilar trunk (BT) [4, 7–10].

Like the trigeminal artery, the hypoglossal artery also undergoes a progressive involution because most of the blood supply to the two vLNAs and to the BT comes from the first cervical segmental artery [11]. The flow towards the posterior circulation, which was from cranial to caudal, following the regression of the carotico-vertebral anastomoses, is now inverted directed from caudal to cranial [12].

At about 7–12 mm embryo length (≃33 days, Padget stage 3) the VA begins its formation by a transverse anastomosis between seven dorsal cervical intersegmental arterials (CIAs) branches arised from each of the paired dorsal aortae [4, 13]. Most authorities believe that the VA originates from the persistent longitudinal anastomosis of CIAs, while the horizontal segments would have a progressive obliteration except for the seventh intersegmental artery. The latter would give rise to the base of the subclavian artery including the origin of the vertebral artery [4, 13–16].

At about 12–14 mm embryo length (≃36 days, Padget stage 4) the definitive conformation of the cranial division of the ICA is well identifiable while it is at about 16–18 mm embryo length (≃40 days, Padget stage 5) that the VA is more regular and straightened [11] acquiring the morphology recognized in the adult.

The major embryological changes of the VA are summarized in Table 1.

Table 1 Major embryological changes in the formation of the vertebral artery

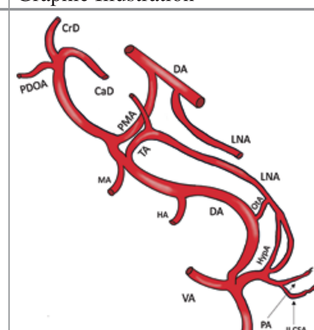
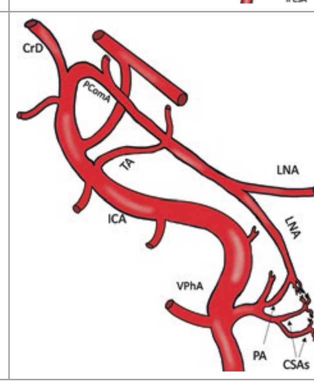
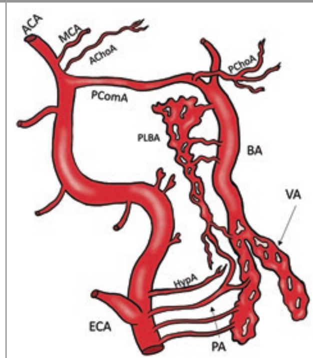
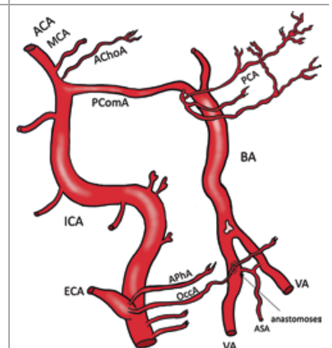
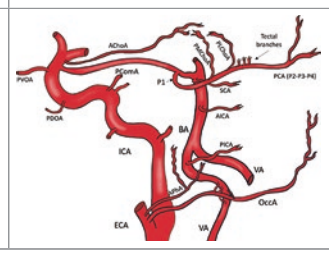
Stage	Embryo size (mm)	Major evolutions	Graphic Illustration
I	4–5	• Paired longitudinal neural arteries (LNAs) visible • Carotido-vertebral anastomoses visible: Trigeminal (TA), otic (OtA), hypoglossal (HypA), proatlantal (PA) and second cervical segmental artery (II CSA) • Primitive ICA bifurcation visible into caudal and cranial divisions (CaD, CrD) • PComA and basilar artery (BA) not formed	
II	5–6	• Formation of the PComA • Regression of otic and hypoglossal arteries • Midline fusion of LNAs to form the basilar artery (BA) • Initial transverse anastomoses between the intersegmental arteries	

Table 1 (continued)

Stage	Embryo size (mm)	Major evolutions	Graphic Illustration
III	7–12	• Basilar artery (BA) completely formed • PComA completely formed • Regression of trigeminal and proatlantal arteries • Formation of the vertebral artery (VA) by transverse anastomoses between the intersegmental arteries (plexiform aspect) • Lateral basilo-vertebral anastomosis (PLBA) visible	
IV	13–15	• Remodeling of the BA and development of the PCA • Migration of occipital artery (OccA) origin on the external carotid artery (ECA) • Persistence of the anastomoses between OccA and VA • Formation of the anterior spinal artery (ASA)	
V	16–18	• Complete formation of the VA • Complete formation of the Occipital Artery (remnant of the proatlantal artery) • Complete formation of the ascending pharyngeal artery (APhA, remnant of the hypoglossal artery) • Potential stem of the anterior inferior cerebellar artery (AICA) visible	

Number and Origin of the Artery

Origin of the Artery

Classically right and left vertebral arteries arise from the posterosuperior aspect of the first part of subclavian artery (SBA) [17] but different angiographic and anatomic post-mortem studies describe multiple cases of anomalous origin of VA with relevant implication during endovascular intervention and neck surgery. The variants in origin of the VA are illustrated in Fig. 1.

Variations of VA origin usually occur on the left side with the commonest reported atypical origin from the aortic arch (AOA) [18, 19]. In a comprehensive review in 2018 [20] including cadaveric and angiographic studies on 14,738 subjects, an atypical VA origin was detected in 4.6%, more often unilaterally. In cases of unilateral aberrancy, the left VA was more frequently involved (4%) than the right VA (0.7%). The rate of atypical vertebral artery origin is summarized in Table 2.

The variations may concern a VA with typical origin from the SBA or a VA with an origin different from that of the subclavian artery.

In case of a typical origin of VA from the SBA, more frequently the level of vertebral origin is between the seventh cervical vertebra and the first thoracic vertebra, but VA can emerge between the first and second thoracic vertebra as well [21].

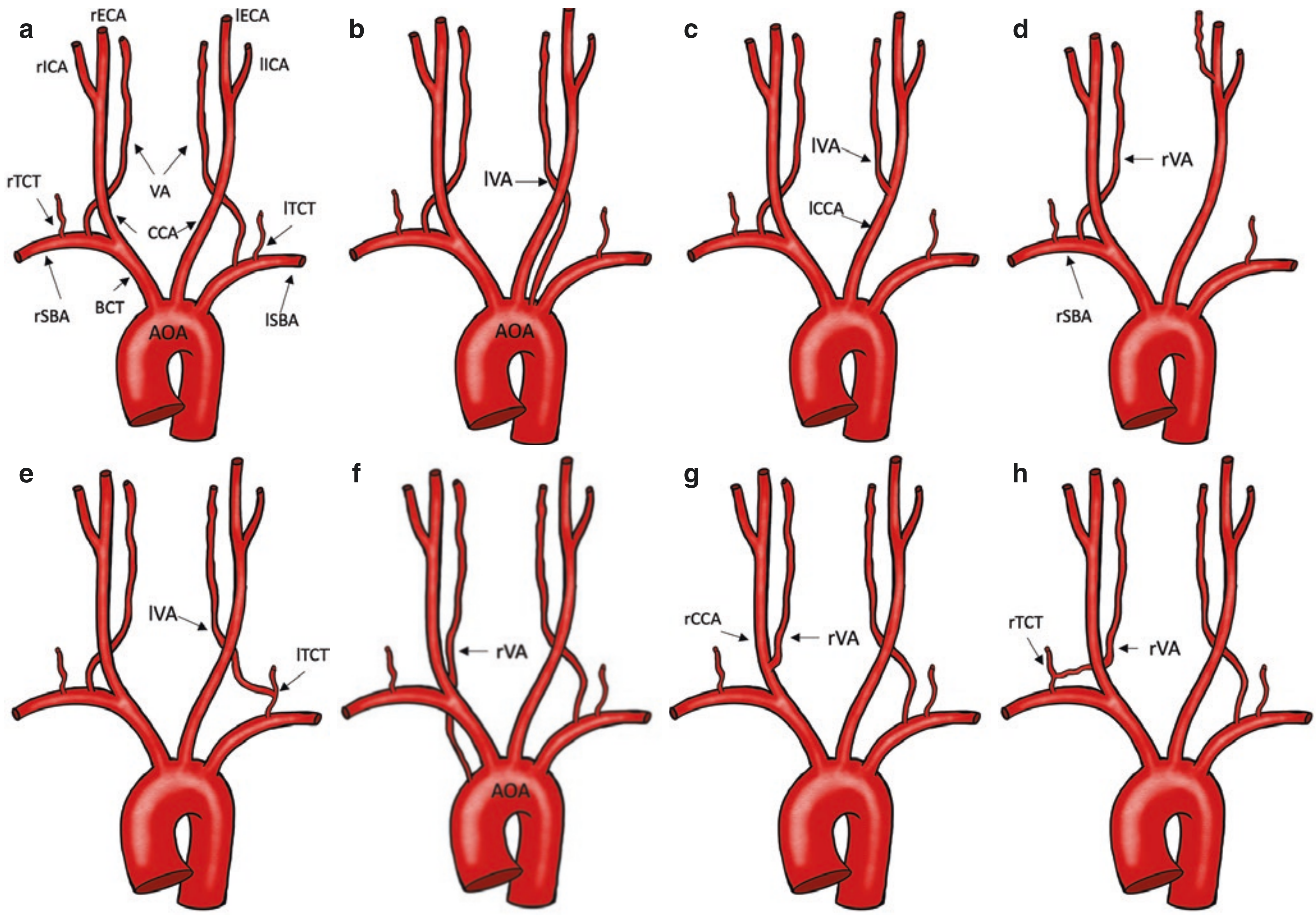


Fig. 1 Different possible origins of the vertebral artery. (**a**) normal configuration of the aortic arch (AOA). *BCT* brachiocephalic trunk, *CCA* common carotid artery, *rSBA* right subclavian artery, *lSBA* left subclavian artery, *rTCT* right thyrocervical trunk, *lTCT* left thyrocervical trunk, *VA* vertebral artery, *lICA* left internal carotid artery, *rICA* right internal carotid artery, *rECA* right external carotid artery, *lECA* left external carotid artery. (**b**) lVA origin from the AOA. (**c**) lVA origin from the lCCA. (**d**) rVA origin from the rSBA. (**e**) lVA origin from the lTCT. (**f**) rVA origin from the AOA. (**g**) rVA origin from the rCCA. (**h**) rVA origin from the rTCT

Table 2 Rate of atypical vertebral artery origin

Lazaridis et al. [20]	14,738 subjects evaluated	Atypical VA origin	676/14,738 (4.6%)
		Unilaterally atypical VA origin	574/14,738 (3.9%)
		Bilaterally atypical VA origin	7/14,738 (0.05%)
		Atypical left VA origin	587/14,738 (4%)
		Atypical right VA origin	101/14,738 (0.69%)

Concerning the atypical origin, we can consider VA originating from the aortic arch (AOA) and those originating neither from the arch nor from the SBA.

VA origin from the AOA is the commonest reported variation (range 0.79–8%) and usually it affects the left VA. The rate of aortic arch origin of the left vertebral artery is summarized in Table 3.

Table 3 Aortic arch origin of the left vertebral artery (adapted from 20 Lazaridis N, Piagkou M, Loukas M, Piperaki E-T, Totlis T, Noussios G, Natsis K (2018). A systematic classification of the vertebral artery variable origin: clinical and surgical implications. Surg Radiol Anat 40:779–797)

Lazaridis et al. [20]	527 subjects with left VA atypical origin	Left VA origin between left common carotid artery (CCA) and left superior cerebellar artery (SCA)	455/527 (86.3%)
		Left VA origin between the common trunk of brachiocephalic trunk (BCT)—left CCA and left SCA	40/527 (7.6%)
		Left VA origin between left CCA and left internal thoracic artery	1/527 (0.19%)
		Left VA between left SCA and ARSCA	1/527 (0.19%)

The most frequent configuration is the left VA emerging between the left common carotid artery (CCA) and left SBA [22, 23]. Lazaridis et al. [10] on 587 cases of left VA atypical origin, VA arised from AOA was detected in 89% of cases with different configuration: between left CCA and left SBA in 86.3% of cases while most rarely VA emanates between the common trunk of brachiocephalic trunk (BCT)—left CCA and left SBA (7.6%) or distal to left SBA (1.3%).

Additionally, unique cases of left VA emersion between left CCA and left internal thoracic artery [20] or between left SBA and aberrant right subclavian artery (ARSBA) [24] have been reported.

The aortic origin of the right VA is rarer. The rate of aortic arch origin of the right vertebral artery is summarized in Table 4 and a clinical case of this rare variant is shown in Fig. 2.

Lazaridis et al. [20] on 101 cases of right VA atypical origin, VA arised from AOA in 6.9% of cases. The most common anomalous origin of the right VA occurs from distal to the origin of the left SBA and in these cases, the right VA took a retro-esophageal course to reach its entry into the vertebral foramen [6]. The VA origin between the left CCA and the left SBA has been documented only twice [23, 25], while the rarest case described only once in the literature is the origin of right VA from the ascending aortic arch; the latter was in coexistence with an aberrant right subclavian artery [26].

Regarding the anomalous extra-aortic arch origin of VA, several configurations have been reported.

For the left VA, extra-aortic arch origin included the left thyrocervical trunk (TCT) in 0.02% [20] and the left SBA near its origin in 0.22% [20]. Additionally, atypical origin of left VA from left CCA [27] and left external carotid artery (ECA) [28] have been reported. The rate of extra-aortic arch origin of the left VA is summarized in Table 5.

Regarding the right VA, the most common anomalous extra-aortic arch origin is from the right CCA, followed by the origin from the right TCT. In the review of Lazaridis

Table 4 Aortic arch origin of the right vertebral artery

Lazaridis et al. [20]	101 subjects with right VA atypical origin	Right VA origin distal to the origin of the left SCA	6/101 (5.9%)
Wasserman et al. [23]		The VA origin between the left CCA and the left SCA	1 case
Albayram et al. [25]		The VA origin between the left CCA and the left SCA	1 case
Akdeniz et al. [26]		Right VA origin from the ascending aortic arch	1 case

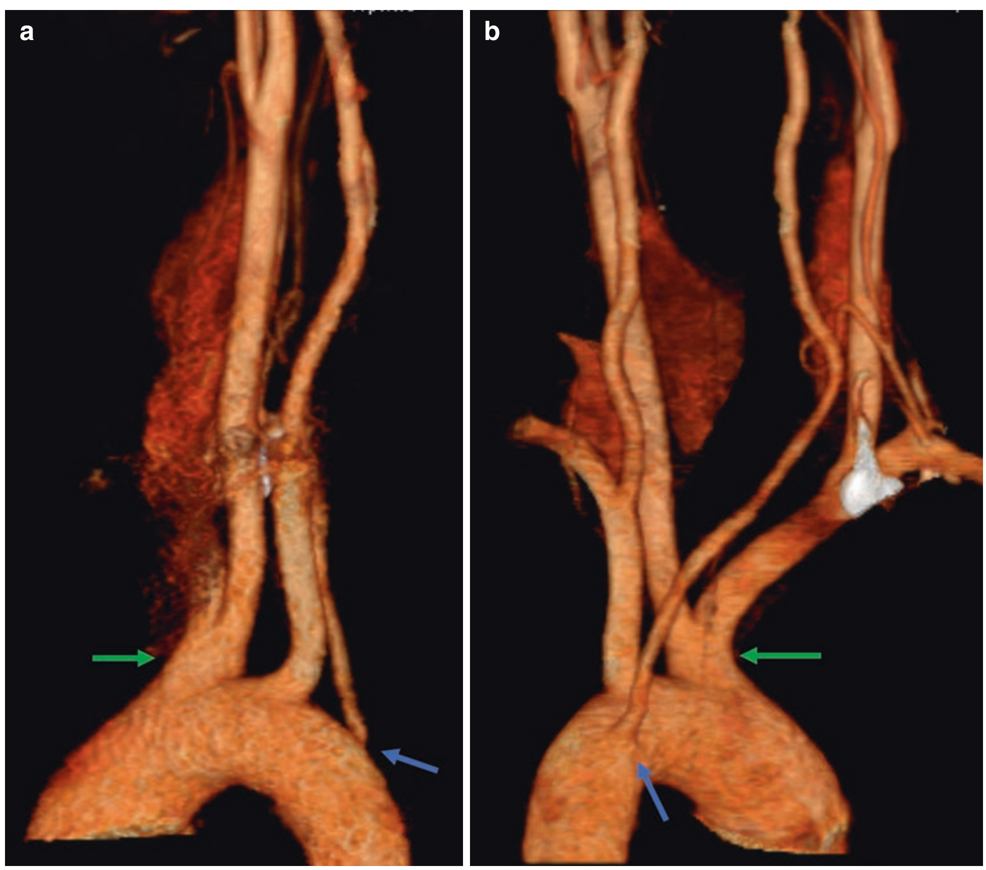


Fig. 2 Clinical case of vertebral artery origin from the aortic arch. 3D volume rendering of a CT scan in a patient with a rare origin of the right vertebral artery from the aortic arch. Anterior oblique (**a**) and posterior view (**b**) show a common origin of the brachiocephalic trunk (green arrows) and the origin of the right vertebral from the proximal descending aorta (blue arrows), distal to the left subclavian artery

Table 5 Extra-aortic arch origin of the left VA

Lazaridis et al. [20]	14,738 subjects evaluated	Left VA origin from the left (TCT)	3/14,738 (0.02%)
		Left VA origin from the left SCA	33/14,738 (0.22%)
Thompson [27]		Left VA origin from the left CCA	1 case
Flynn [28]		Left VA origin from the left ExCA	1 case

Table 6 Extra-aortic arch origin of the right VA

Lazaridis et al. [20]	101 subjects with right VA atypical origin	Right VA origin from the right CCA	53/101 (52.5%)
		Right VA origin from the right TCT	22/101 (21.8%)
		Right VA origin from the right SCA	3/101 (3%)
Shiva Kumar et al. [29]		Right VA origin from the BCT	1 case
Yamaki et al. [30]		Right VA origin from the BCT	1 case
Nasir et al. [31]		Right VA origin from the right ExCA	1 case
Lemke et al. [33]		Right VA origin from the right ICA	1 case
Ulusoy et al. [34]		Right VA origin from the right carotid bulb (common carotid artery trifurcation)	1 case

et al. [20], right VA originated from the right CCA in 52.5% of the cases and from the right TCT in 21.8% of cases. A rarely reported origin includes the BCT [29, 30], right ExCA [31], second portion of the right SBA [32] and the ARSBA [20].

Additionally, unique cases of right VA origin from the right internal carotid artery (ICA) [33] and from ipsilateral carotid bulb (common carotid artery trifurcation) [34] have been reported. The rate of extra-aortic arch origin of the right VA is summarized in Table 6.

Taking into consideration that the embryological development of the VA is connected to the involution of some cervical intersegmental arteries and to the persistence of some of them, variations of VA are thought to be caused by defects in this process of regression and persistence of the cervical intersegmental arteries [35, 36]. The persistence could occur in the upper (first or second) intersegmental arteries and this may be responsible of an abnormal origin of the VA from the internal or external carotid artery. Differently, the absence of regression of a lower (third to sixth) intersegmental arteries could result in an abnormal origin of the VA from the aortic arch or the common carotid artery [37].

Regarding the origin of VA directly from the AOA, for the left VA, some authors suggest that a large absorption of embryonic tissue of the left superior cerebellar artery (SCA) between the origin of the VA and the AOA may cause a direct origin of left VA from the AOA, between the origins of the left SBA and left CCA [38]. Regarding the origin of the right VA as the last branch of the aorta distal to the left SBA, it is thought to be caused from persistence of the right dorsal aorta and the obliteration of the right fourth arch [6].

Duplication, Triplication and Fenestration

The term "Duplication" of VA refers to a condition where the VA has two origins that fuse at different levels of the neck [39].

Right VA can have a dual origin in 0.04 to 0.4% and left VA in 0.09 to 3.3% of the cases [40, 41]. Bilateral dual origin of both VAs is very rare and it has been reported only three times [42].

Regarding the dual origin of left VA, different configurations have been described: Lazaridis et al. [20] on 16 cases of left VA dual origin, reported 12 cases where one leg emanated from the AOA and the other leg from the left SCA, two cases where one leg originated from the left SBA and the other one from the left TCT, one case from the left SBA and the left ECA and one case where both legs emanated from the AOA (between the left SBA and left CCA). Additionally, another configuration where both legs originated from the left SBA has been described [39, 43].

Regarding the dual origin of right VA in most cases, the two legs originate from the right SBA [42]. Lazaridis et al. [20] on four cases of right VA dual origin, in three cases both legs emanated from the right SBA, in one case one leg from the right TCT and the other one from the right SBA. Other possible configurations include cases where one leg originated from the right SCA and the other leg either from the right BCT, right CCA, right TCT or from the AOA [20, 42]. Additionally, a dual origin of the right VA with both legs emanated from the AOA has been sporadically reported [20].

Regarding the triplication, a unique case of a left VA triple origin has been reported [44]. In this case three distinct origins were detected, two of them from the SCA and the third from the inferior thyroid artery.

Despite sometimes erroneously used as a synonym of duplication [39], another different anatomical phenomenon is the "fenestration" of VA. The term "fenestration" describes the single origin of a vessel that splits to form two channels that re-fuse distally [6]. In the literature the incidence of VA fenestration is between 0.23% and 1.95% [45, 46] and even though it can occur either intra- or extracranially, extracranial fenestration at the upper cervical level is more commonly reported [47, 48].

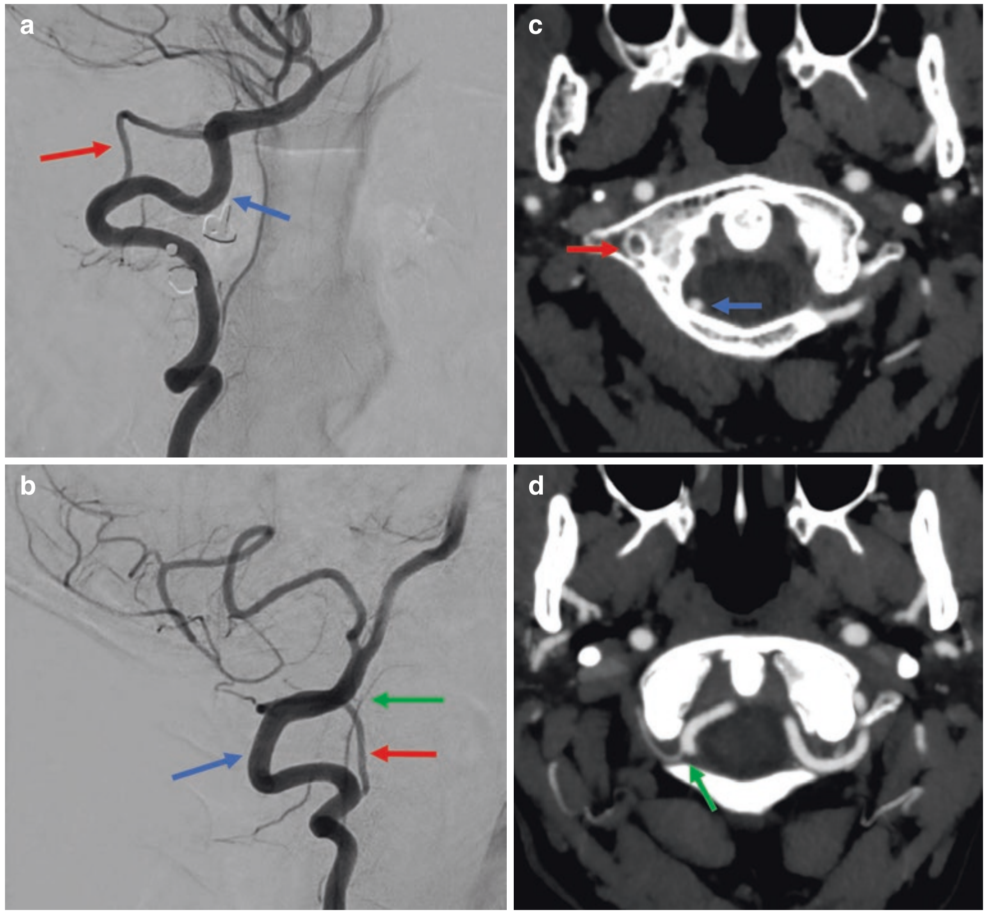


Fig. 3 Clinical case of vertebral artery fenestration. Digital subtraction angiography (DSA) of right vertebral artery on antero-posterior (**a**) and lateral view (**b**) and axial CT scan images (**c**, **d**) in a patient with fenestration of the V3 segment. A larger branch runs medially between the lateral masses of C1 and C2 and superiorly within spinal canal at C1 (blue arrows), while a smaller branch runs in the transverse foramen of C1 (red arrows), rejoining the larger branch before entering the dura (green arrow)

The persistence of more than one intersegmental metameric artery explains probably the duplication of the extracranial VA, while the fenestration could be probably due to the partial persistence of the longitudinal plexiform structure [12]. A clinical case of VA fenestration is shown in Fig. 3.

Course of the Artery

Among many segmentation patterns described in the last 150 years, the most widely adopted in the current medical literature was described by Barbieri in the "Monografia della arteria vertebrale" in 1867, later popularized by Krayenbühl and Yaşargil in 1957 [2, 49]. According to these authors, the VA is divided into four segments, from the subclavian artery to the vertebrobasilar junction. The segments of the VA are illustrated in Fig. 4.

Vertebral arteries are often symmetric, but the relative hypoplasia (defined as diameter ≤2 mm and asymmetry ratio of ≤1:1.7) of one side is relatively common and visible in 15.6% of cases, with 66% of the hypoplastic artery on the right side [50]. The more common dominance of the left side does not appear to be related to hemisphere dominance [51].

The V1 is a short segment that more commonly originates as the first branch of the subclavian artery. It travels superiorly and posteriorly, behind the anterior scalene muscle, to the level of C6, where it enters the foramen in the transverse process in 88–93% of cases [52, 53]; in some cases the artery might enter the transverse foramina at the level of C5 (4–7%), or C7 (1–3%) [53, 54], reflecting a different embryological development with a C5 or C7 segmental artery (future V1 segment) being dominant in supplying the longitudinal anastomoses (future V2 segment).

An even higher entrance in the foramen has been described at the level of C4 (0.2%) and C3 (1%) [52]; but rather than a variation of the course of the artery, it probably reflects the origin of the VA from a cervical artery, with the latter supplying flow to distal VA at C5, C4 or C3 level (so-called C5 to C3 vertebral entry of VA) [5]. The V1 segment provides arterial supply to the stellate ganglion [55].

The V2 segment travels vertically through the foramina transversaria, usually from C6 to C2, surrounded by a venous plexus that drains into the subclavian or internal jugular veins [56]. For some authors the V2 segment ends at C1 rather than C2, following a tradition that dates back to 1860, and this generated confusion in many papers in the current

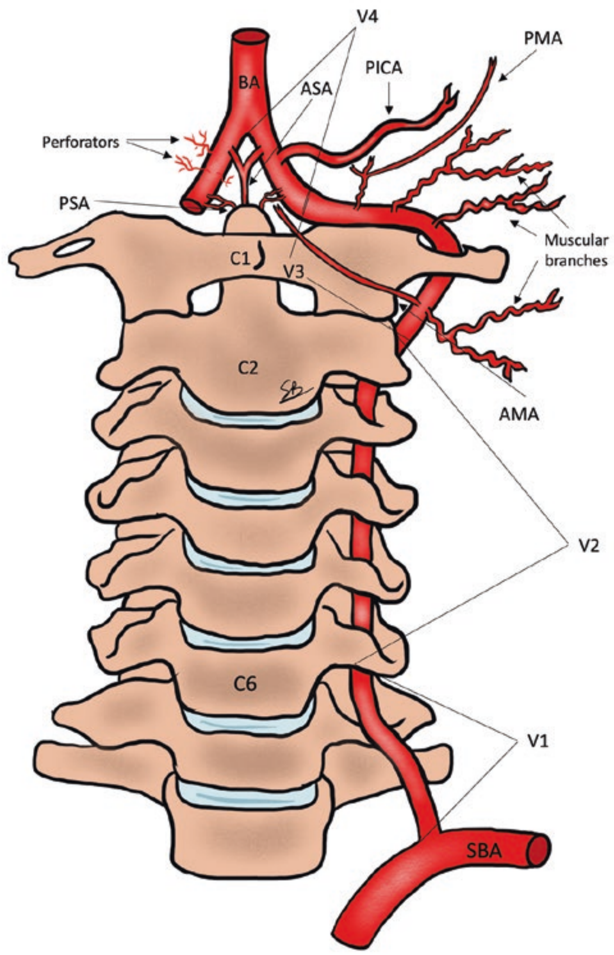


Fig. 4 Normal anatomy and branches of the vertebral artery. The Figure shows the four segments of the vertebral artery (V1-V2-V3-V4) and its main branches: posterior inferior cerebellar artery (PICA), anterior spinal artery (ASA), posterior spinal artery (PSA), posterior meningeal artery (PMA), anterior meningeal artery (AMA), muscular branches and perforators

literature [2]. The average length of the V2 segment of the vertebral artery is 78.2 mm, while the average diameters of the left and right VAs are 4.6 and 4.2 mm, respectively [57].

In a small portion of cases (2.0%) the VA might form a medially directed "loop" while running in the foramina [52, 58]; these loops can be found into an unusually large transverse foramen with the internal border medial to the uncovertebral joint or at the level of the intervertebral disc space, more commonly between C3 and C5 [52].

The V3 segment begins as the VA exits the transverse foramen of C2. Between the foramina of C2 and C1, the artery slightly curves laterally to enter the transverse foramen of C1. The segment between the C1 and C2 transverse foramina sometimes forms a proximal loop, with an average length of the lateral projection of 16 mm; the proximal loop projects posteriorly 48% of cases, directly laterally in 20% of cases, and is absent in 32% of cases, with the artery running in a straight line between C1 and C2 [59]. Exiting from C1 it turns backward and runs in the sulcus of the posterior arch of C1 and then turns again anteriorly and medially to reach the foramen magnum, where the artery penetrates the dura mater. It is widely thought that these curves allow the VA to accommodate the movements of the atlanto-occipital segment.

Multiple variants of fenestrations, duplications and intradural course at the level of C1–C2 have been described, and different embryological explanations provided to explain them [60]. Lasjaunias proposed that a number of variations can be understood as variations in the development of the VA and of one of its branches, the lateral spinal artery, that runs intradurally [61]. Siclari et al. proposed that some variations are rather variations of the posterior spinal arteries [62]. According to Uchino, the intradural VA at C2 is explained as a persistent first intersegmental artery [63]; according to the author, the persistent first intersegmental artery also might explain the extracranial origin of the posterior inferior cerebellar artery (PICA) at C1–C2.

Two important variations of the V3 segment involve the proatlantal arteries types 1 and 2. The former artery is the embryonic form of the first segmental artery, originating from the cervical internal carotid artery. At the level of C1 the VA is no longer derived by the union of intersegmental arteries but it is rather a segment of the proatlantal artery type 1 [5]; a failure in the development of the lower intersegmental arteries might lead to the origin of the VA from the internal carotid artery. The proatlantal artery type 2 corresponds to the second segmental artery; when the embryonic artery regresses, it gives origin to the C2 occipital-vertebral anastomosis [5].

The V4 segment is the intradural, terminal portion of the artery, and extends from its entrance through the dura to the vertebrobasilar junction. At the level of the dural entrance, a small change in the caliber is often identifiable, and the dura forms a fibrous dural ring around the artery [64]. As it enters the skull, the VA wall shows marked change, with a reduction in the thickness of the adventitial and medial layers, and a reduction of elastic fibers in the media and external elastic lamina [65]. In the subarachnoid space, the artery runs superiorly and medially, anterolateral to the medulla oblongata. The vertebrobasilar junction is usually located at the level of the bulbopontine junction in 20% of cases, slightly below in 67% and slightly above in 12%, and the total length of the V4 segment averages 22 mm [66].

As for the lower segments, there is a slightly prevalence of left dominance in the V4 segments, with an average diameter of 2.85 mm on the right side and 3.02 mm on the left [67]. The V4 segment is hypoplastic in about 22% of cases on the right side and in 14% of cases on the left side; in about 5% of cases, both V4 segments might be hypoplastic [67].

Branches

During its course the VA provide a large number of muscular branches, spinal segmental arteries, meningeal branches and intracranial branches to the upper spinal cord, brainstem and cerebellum. The angiographic configuration and the main branches of V3 and V4 segments are shown in Fig. 5. The branches arising from the V2 and V3 segments are shown in Fig. 6.

Muscular and Osseous Branches of V2 and V3

Multiple small branches for the cervical muscles and bones arise from the V2 and V3 segment, with multiple anastomoses with arterial branches of the ascending cervical artery and deep cervical artery and with branches of the external carotid artery.

These small arteries have a mean diameter between 0.5 and 1.1 mm and might branch from anterior, posterior, lateral and medial wall, with the posterior being the most common [68]. Most muscular branches arise posteriorly or posterolaterally, while osseous branches often originate anteromedially. In some cases, muscular, osseous and radicular branches may originate from a single common trunk [68].

A muscular branch from the V3 segments is observed in 20–67% of cases [69, 70] and is commonly known as artery of Salmon. It can be unilateral or, more commonly, bilateral and can originate as a single artery or up to three small branches that run superiorly to supply the suboccipital musculature and related tissues [71]: it can anastomose with branches of the occipital, ascending cervical and deep cervical arteries [72].

Radicular Spinal Arteries

The spinal arteries supply the nerve roots and the dura mater of the cervical spine (radicular arteries) from C6 to C1, together with branches of the ascending cervical artery and the deep cervical artery [5].

At some levels and with an unpredictable pattern, the radicular branches provide contribution to the anterior spinal artery for the spinal cord (radiculomedullary arteries). According to Tubbs et al. [68] and Bruneau et al. [68, 73], no radiculomedullary artery originates from the V3 segment and thus surgical intervention at this level carries minimal risk of complications for the spinal cord.

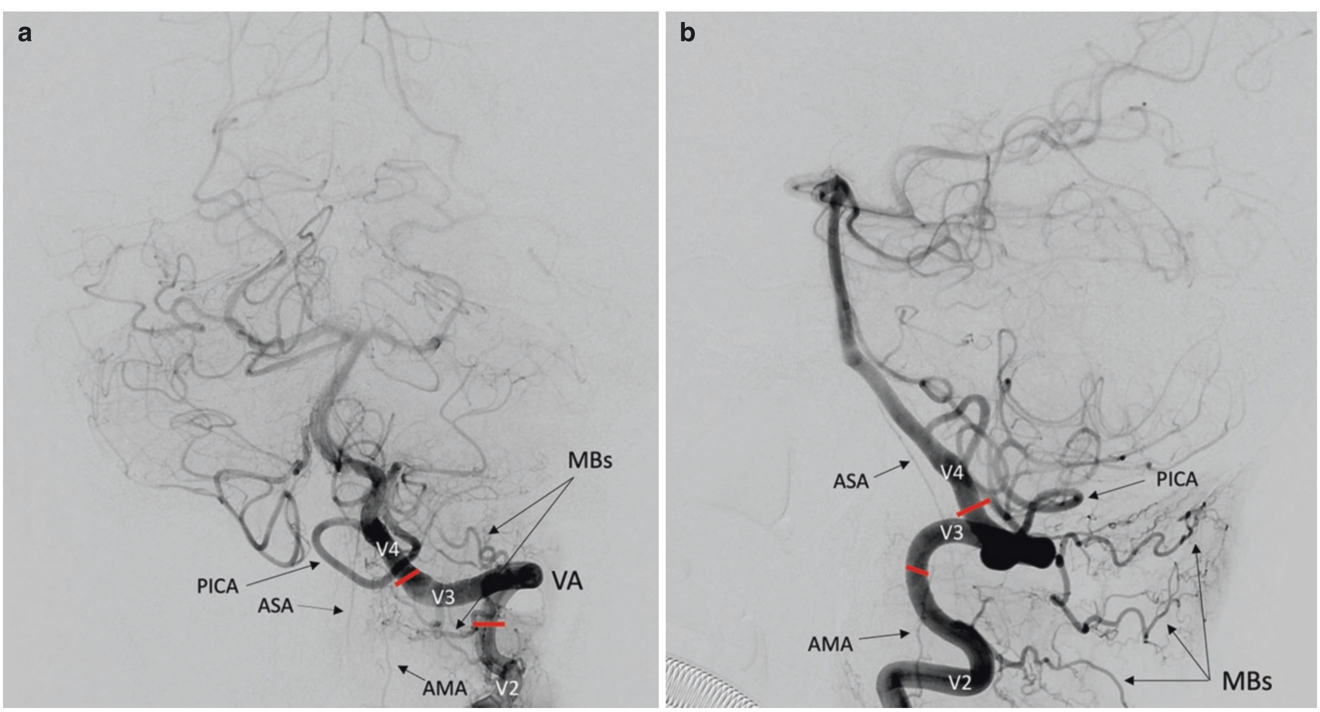


Fig. 5 Angiographic configuration of segments V3 and V4 of the VA. Left vertebral artery angiogram AP view (**a**) and lateral view (**b**). The anterior meningeal artery (AMA) arises from the V2 segment and courses upward to supply the dura of the foramen magnum. It is located laterally (**a**) and anteriorly (**b**) to the anterior spinal artery (ASA). Muscular branches (MBs) arise from the V2 and V3 segments. The posterior inferior cerebellar artery (PICA) and the ASA arise from the V4 segment

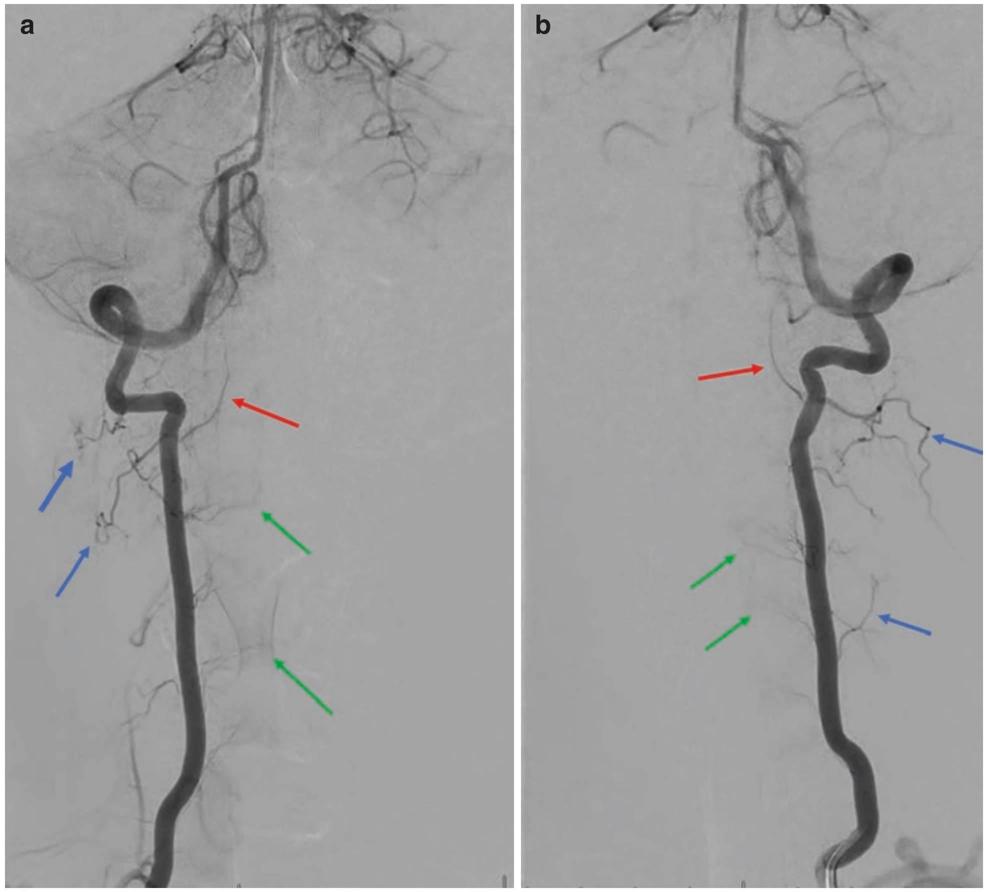


Fig. 6 DSA with branches of the VA. Digital subtraction angiography of left (**a**) and right (**b**) vertebral arteries, antero-posterior view. Multiple branches of the V2 and V3 segments are visible, including muscular branches (blue arrows), radicular and radiculo-medullary arteries (green arrows) and the C3 artery (the so-called anterior meningeal artery of the VA, red arrow) supplying the dura of the anterior foramen magnum and the inferior clivus

Anterior Meningeal Artery

This artery arises from anteromedial surface of the distal V2 segment of the vertebral artery (VA) immediately below its first bend at the level of the axis or at the level of the third vertebral body and it supplies the dura of the anterior foramen magnum and the inferior clivus.

For more details see the chapter "Dural Branches of Vertebral Artery."

Posterior Meningeal Artery

This artery usually arises from the posterior portion of the V3 segment of the VA, above the level of the arch of the atlas just below the foramen magnum.

The posterior meningeal artery (PMA) supplies the medial portions of the dura of the occipital posterior fossa as well as the falx cerebelli. In addition, it may extend above the tentorium to supply the posterior segment of the falx cerebri and adjacent tentorium.

For more details see the chapter "Dural Branches of Vertebral Artery."

Posterior Inferior Cerebellar Artery (PICA)

The PICA commonly originates in the V4 segment approximately 16–17 mm proximal to the vertebrobasilar junction, and 8–9 mm superior to the foramen magnum [74], but an extracranial origin is a frequent variation.

Anterior and Posterior Spinal Arteries

The anterior spinal artery (ASA) is formed by the fusion of two vascular roots originating from the VA. The two roots of the ASA arise from the VA 6.5 mm proximal to the vertebrobasilar junction, and have an average diameter of 523 μm (range 210–1000 μm) [75]. The fusion of the arterial roots can be observed at variable level, caudal to the foramen magnum; sometimes two long unfused cervical anterior spinal arteries of no pathological significance can be detected [5]. In 30–50% of cases a small communicating artery known as the anterior spinal communicating artery connects the left and right anterior spinal arteries [66, 75].

Small branches originating from the posterior aspect of the VA form the most cranial portion of the posterior spinal arteries [9]. These branches commonly originate from the distal V3 segment, proximally to the point of dural penetration, or from the PICA. Some controversies exist on whether these branches could have been inaccurately labeled as feeders of the lateral spinal arteries [9, 62].

Perforator Branches

Perforators are arterial branches from the VA or from the arterial roots of the ASA that supply the lateral medulla, inferior cerebellar peduncle and medullary surface of the cerebellum [66].

The perforating arteries are 6.5 on average (range 1 to 11), with an average diameter of 243 μm (100–520 μm), and they may arise directly from the VA (54.54% of cases) or from the vascular roots of the ASA (over 90% of cases) [75].

In patients with a PICA originating from the anterior inferior cerebellar artery (AICA) (AICA-PICA variant) the VA gives significantly higher numbers of perforating arteries [76].

It is possible to observe anastomoses involving the perforators in about 40% of cases or side branches in over 90% of cases, with an average diameter of 169 μm and 161 respectively [75].

In about 35% of cases one or two branches originating between the vertebrobasilar junction and the ASA, more commonly on the right side, supply nuclei of the floor of the fourth ventricle [66].

Possible Anastomoses

The VA presents multiple anastomoses with the cervical arteries originating from the subclavian artery and with branches of the external carotid artery, the ascending pharyngeal artery and the occipital artery.

The monumental work of Lasjaunias, Berenstein and TerBrugge [5] described the hemodynamic balance between the muscular elements of the VA and the other arteries in the neck, with the segmental branches anastomosing according to embryonic arrangement of metameric levels.

Anastomoses at C1–C2

The C1 and C2 branches of the VA are in balance with the occipital artery and represent the persistent connections of proatlantal arteries when the proximal VA is hypoplastic.

Two types of proatlantal arteries have been described according to the cervical level of occipital-vertebral anastomoses.

The type I proatlantal artery corresponds to the embryonic first segmental artery, arising from the proximal the internal carotid artery, running posteriorly into the occipitocervical space. The vertebral artery itself, at the level of C1, is the distal remnant of the proatlantal I artery.

In the normal anatomy of adults, the proximal remnant of that first segmental artery is the occipital artery and its C1 occipito-vertebral anastomosis.

The type II proatlantal artery corresponds to the second segmental artery, arising from the proximal external carotid artery. After regression of the connection, the C2 occipito-vertebral anastomosis represents the remnant of the embryonic artery.

The persistent proatlantal artery in adults is observed with the persistence of the C1 or C2 segmental artery's anastomosis associated to hypoplasia of the proximal vertebral artery.

Anastomoses at C3–C6

In the adult's vascular system, the deep cervical and ascending cervical arteries can be considered the artery of the C3–C4 segments and the artery of C5–C6 segments respectively, while the proximal VA can be considered the artery of C7 segment.

The anastomoses between the VA and the cervical arteries follow the same pattern, with VA-deep cervical connection more commonly observed at C3–C4 and VA-ascending cervical more frequently at C5–C6. However, different configuration can be observed.

C3 is also at the level of the odontoid arcade, which represents an anastomotic arcade between VA, the occipital artery and the ascending pharyngeal artery.

Parenchymal and/or Dural Territory(ies)

In addition to the ASA for spinal cord vascularization, which is not the aim of this chapter, the VA gives rise to multiple small branches which supply the medulla oblongata. They arise from the posterior surface of the VA [77] and they run on the anterior and lateral surface of the medulla. Anastomoses between these superficial vessels and branches of the PICA and BA are present typically on the surface of the olive [12].

Regarding dural vessels, three meningeal branches arise from the distal extracranial VA: the anterior meningeal artery (AMA), the posterior meningeal artery and the falx cerebelli artery described above.

Clinical Implications

The patients with VA variants are usually asymptomatic and in most cases, the anomalous origin of the VA is an incidental finding. However, in rare cases, a VA variation was found accidentally during a clinical examination of headache or dizziness, but these do not seem to be associated with the anomalous origin [78].

There is no evidence supporting that a VA anatomic variations predispose to cerebrovascular disorders but some authors have suggested that an anomalous origin, caliber and distribution of the VAs might be associated to cerebral disorders due to a cerebral hemodynamic perturbation [23, 33, 79].

Turbulent flow in an aberrant vessel origin may increase the risk of thrombosis, artery occlusion, intracranial aneurysms formation, artery occlusion, dissection and potentially atherosclerosis [77].

A higher incidence of arterial dissection in case of a left VA arising directly from the AOA has been reported [80]. On the other hand, the association between arterial dissection and an aberrant origin of VA from AOA remains to be elucidated [80]. It has been suggested that a left VA originating directly from the AOA could predispose to dissection as it typically enters the C4 or C5 foramen transversarium, resulting in a longer V1 segment in the neck [14, 20].

The origin variations of the VA may coexist with abnormalities in the course of the vessel, bifid origin, vessel fenestration, tortuosity, elongation and kinking, aneurysm formation and associated hereditary connective tissue disorders [81]. An increased incidence of VA variations has also been reported in patients with Klippel–Feil and Down syndrome [34, 82]. Additionally, a higher incidence of VA anomalies in patients presenting with certain congenital cardiovascular variations has been reported. The most common of these congenital cardiovascular variations among patients with anomalous VA origin is the presence of an ARSBA [14]. Tsai et al. [24] found a VA anomaly in 15.7% of patients with ARSBA.

Although the origin variations of the VA remain asymptomatic, their knowledge seems to be mandatory as there is a potential clinical impact in case of endovascular procedure, aortic arch and head and neck surgery.

An anomalous VA origin also represents a potential pitfall in the diagnosis of cerebrovascular injury.

An origin of right VA as the last branch of AOA may be wrongly assumed to be occluded either by eluding catheterization during angiography or because its origin is not included in the acquisition field during noninvasive studies such as computed tomography angiography, magnetic resonance angiography, or Doppler sonography. During imaging, scanning of the entire AOA up to the level of ligamentum arteriosum is mandatory so as to rule out any aberrancy [83]. A possible VA origin emersion from the inferior thyroid artery should be taken into consideration during inferior thyroid artery catheterization and thyroidectomy procedures [20]. Additionally, in case the VA originates from the common or the external carotid artery, a ligation of the common carotid artery could obviously be catastrophic as it deprives the posterior cranial fossa of its arterial supply [20]. Finally, a dual origin of the VA may be erroneously interpreted as dissection [13].

In conclusion, although the anomalous origin of the VA is typically an incidental finding, its identification is required before any surgical/endovascular intervention in order to avoid any misinterpretations or inadvertent injuries.

References

1. Willis T. Cerebri Anatome Cui Accessit Nervorum Descriptio et Usus. London: Flesher, Martyn and Allestry; 1664.
2. Gailloud P. The segmentation of the vertebral artery: an ambiguous anatomical concept. Interv Neuroradiol. 2021;28(6):765–72. https://doi.org/10.1177/15910199211063275.
3. Radner S. Intracranial angiography via the vertebral artery: preliminary report of a new technique. Acta Radiol. 1947;28:838–42.
4. Padget DH. The development of the cranial arteries in the human embryo, Contribution to embryology, vol. 212. Washington: Carnegie Institution; 1948. p. 205–62.
5. Lasjaunias PL, Berenstein A, ter KG B. Surgical neuroangiography. 2nd ed. Berlin/New York: Springer; 2001.
6. Maiti TK, Konar SK, Bir S, Nanda A, Cuellar H. Anomalous origin of the right vertebral artery: incidence and significance. World Neurosurg. 2016;89:601–10.
7. Menshawi K, Mohr JP, Gutierrez J. A functional perspective on the embryology and anatomy of the cerebral blood supply. J Stroke. 2015;17:144.
8. Raybaud C. Normal and abnormal embryology and development of the intracranial vascular system. Neurosurg Clin N Am. 2010;21:399–426.
9. Gregg L, Gailloud P. The role of the primitive lateral Basilovertebral anastomosis of Padget in variations of the Vertebrobasilar arterial system: the primitive lateral basilovertebral anastomosis. Anat Rec. 2017;300:2025–38.
10. Kathuria S, Gregg L, Chen J, Gandhi D. Normal cerebral arterial development and variations. Semin Ultrasound CT MRI. 2011;32:242–51.
11. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: a pictorial review. Surg Radiol Anat. 2021;43:961–73.
12. Bradac GB (2017) Applied cerebral angiography: Normal anatomy and vascular pathology.
13. Thomas AJ, Germanwala AV, Vora N, Prevedello DM, Jovin T, Kassam A, Horowitz M. Dual origin extracranial vertebral artery: case report and embryology. J Neuroimaging. 2008;18:173–6.
14. Yuan S-M. Aberrant origin of vertebral artery and its clinical implications. Braz J Cardiovasc Surg. 2015;31:52–9. https://doi.org/10.5935/1678-9741.20150071.
15. Satti SR, Cerniglia CA, Koenigsberg RA. Cervical vertebral artery variations: an anatomic study. AJNR Am J Neuroradiol. 2007;28:976–80.
16. Newton T, Mani R. The vertebral artery. In: Newton TH, Potts DG, editors. Radiology of skull and brain. St. Louis: Mosby; 1974. p. 1659–72.
17. Rozin L, Rozin R, Koehler SA, Shakir A, Ladham S, Barmada M, Dominick J, Wecht CH. Death during Transforaminal epidural steroid nerve root block (C7) due to perforation of the left vertebral artery. Am J Forensic Med Pathol. 2003;24:351–5.
18. Çavdar S, Arisan E. Variations in the extracranial origin of the human vertebral artery. Cells Tissues Organs. 1989;135:236–8.

19. Stoesslein F, Porstmann W, Schueler F, Schoepke W. Aberrant vertebral artery originating from the descending aorta: a new congenital steal syndrome in coarctation. Eur J Radiol. 1982;2:157–9.
20. Lazaridis N, Piagkou M, Loukas M, Piperaki E-T, Totlis T, Noussios G, Natsis K. A systematic classification of the vertebral artery variable origin: clinical and surgical implications. Surg Radiol Anat. 2018;40:779–97.
21. Shin I-Y, Chung Y-G, Shin W-H, Im S-B, Hwang S-C, Kim B-T. A morphometric study on cadaveric aortic arch and its major branches in 25 Korean adults : the perspective of endovascular surgery. J Korean Neurosurg Soc. 2008;44:78.
22. Magklara EP, Pantelia ET, Solia E, Panagouli E, Piagkou M, Mazarakis A, Skandalakis P, Troupis T, Filippou D. Vertebral artery variations revised: origin, course, branches and embryonic development. Folia Morphol (Warsz). 2021;80(1):1–12. https://doi.org/10.5603/FM.a2020.0022.
23. Wasserman BA, Mikulis DJ, Manzione JV. Origin of the right vertebral artery from the left side of the aortic arch proximal to the origin of the left subclavian artery. AJNR Am J Neuroradiol. 1992;13:355–8.
24. Tsai I-C, Tzeng W-S, Lee T, Jan S-L, Fu Y-C, Chen M-C, Lin P-C, Liao W-C, Chen CC-C. Vertebral and carotid artery anomalies in patients with aberrant right subclavian arteries. Pediatr Radiol. 2007;37:1007–12.
25. Albayram S, Gailloud P, Wasserman BA. Bilateral arch origin of the vertebral arteries. AJNR Am J Neuroradiol. 2002;23:455–8.
26. Akdeniz B, Yilmaz E, Pekel N, Ergul BU. Anomalous origin of the right vertebral artery from the ascending aorta in the presence of an aberrant right subclavian artery. Int J Card Imaging. 2007;23:39–42.
27. Thomson A. Third annual report of committee of collective investigation of anatomical society of great Britain and Ireland for the year 1891-92. J Anat Physiol. 1893;27:183–94.
28. Flynn RE. External carotid origin of the dominant vertebral artery: case report. J Neurosurg. 1968;29:300–1.
29. Shiva Kumar GL, Pamidi N, Somayaji SN, Nayak S, Vollala VR. Anomalous branching pattern of the aortic arch and its clinical applications. Singap Med J. 2010;51:e182–3.
30. Yamaki K, Saga T, Hirata T, Sakaino M, Nohno M, Kobayashi S, Hirao T. Anatomical study of the vertebral artery in Japanese adults. Anat Sci Int. 2006;81:100–6.
31. Nasir S, Hussain M, Khan SA, Mansoor MA, Sharif S. Anomalous origin of right vertebral artery from right external carotid artery. J Coll Physicians Surg Pak. 2010;20:208–10.
32. Daseler EH, Anson BJ. Surgical anatomy of the subclavian artery and its branches. Surg Gynecol Obstet. 1959;108:149–74.
33. Lemke AJ, Benndorf G, Liebig T, Felix R. Anomalous origin of the right vertebral artery: review of the literature and case report of right vertebral artery origin distal to the left subclavian artery. AJNR Am J Neuroradiol. 1999;20:1318–21.
34. Ulusoy OL, Sasani H, Barlas SB, Mutlu A, Sasani M. A case of anomalous origin and course of vertebral artery in a patient with Klippel Feil syndrome. Korean J Radiol. 2016;17:554–7.
35. Uchino A, Kato A, Abe M, Kudo S. Association of cerebral arteriovenous malformation with cerebral arterial fenestration. Eur Radiol. 2001;11:493–6.
36. Tseng Y-C, Hsu H-L, Lee T-H, Wang L-J, Wong Y-C, Chen C-J. Fenestration of the vertebral artery at the lower cervical segment—imaging findings and literature review. Eur J Radiol. 2004;49:37–40.
37. Takagi T, Yamashita K. Right vertebral artery as the last branch of the aortic arch and a coronary sinus opening into the left atrium as found in a cadaver. Kaibogaku Zasshi. 1992;67:49–52.
38. Budhiraja V, Rastogi R, Jain V, Bankwar V, Raghuwanshi S. Anatomical variations in the branching pattern of human aortic arch: a cadaveric study from Central India. ISRN Anat. 2013;2013:828969.
39. Ionete C, Omojola MF. MR angiographic demonstration of bilateral duplication of the extracranial vertebral artery: unusual course and review of the literature. AJNR Am J Neuroradiol. 2006;27:1304–6.
40. Uchino A, Sawada A, Takase Y, Kudo S. Extreme fenestration of the right vertebral artery: magnetic resonance angiographic demonstration. Eur Radiol. 2002;12(Suppl 3):S32–4.
41. Meila D, Tysiac M, Petersen M, Theisen O, Wetter A, Mangold A, Schlunz-Hendann M, Papke K, Brassel F, Berenstein A. Origin and course of the extracranial vertebral artery: CTA findings and embryologic considerations. Clin Neuroradiol. 2012;22:327–33.
42. Rameshbabu C, Gupta OP, Gupta KK, Qasim M. Bilateral asymmetrical duplicated origin of vertebral arteries: multidetector row CT angiographic study. Indian J Radiol Imaging. 2014;24:61–5.
43. Mahmutyazicioğlu K, Saraç K, Bölük A, Kutlu R. Duplicate origin of left vertebral artery with thrombosis at the origin: color Doppler sonography and CT angiography findings. J Clin Ultrasound. 1998;26:323–5.
44. Quain R. The anatomy of the arteries of the human body with its applications to pathology and operative surgery. London: Taylor and Walton; 1844.
45. Rieger P, Huber G. Fenestration and duplicate origin of the left vertebral artery in angiography. Report of three cases. Neuroradiology. 1983;25:45–50.
46. Tran-Dinh HD, Soo YS, Jayasinghe LS. Duplication of the vertebrobasilar system. Australas Radiol. 1991;35:220–4.
47. Sim E, Vaccaro AR, Berzlanovich A, Thaler H, Ullrich CG. Fenestration of the extracranial vertebral artery: review of the literature. Spine (Phila Pa 1976). 2001;26:E139–42.
48. Takahashi M, Kawanami H, Watanabe N, Matsuoka S. Fenestration of the extra-cranial vertebral artery. Radiology. 1970;96:359–60.
49. Krayenbuhl H, Yasargil MG. The vascular diseases in the region of the basilar & vertebral arteries; an anatomopathological, clinical & neuroradiological study. Fortschr Geb Rontgenstr Nuklearmed. 1957;87:1–170.
50. Thierfelder KM, Baumann AB, Sommer WH, Armbruster M, Opherk C, Janssen H, Reiser MF, Straube A, von Baumgarten L. Vertebral artery hypoplasia: frequency and effect on cerebellar blood flow characteristics. Stroke. 2014;45:1363–8.
51. Vural A, Derin Çiçek EE. Is the asymmetry between the vertebral arteries related to cerebral dominance? Turk J Med Sci. 2019;49:1721–6.
52. Bruneau M, Cornelius JF, Marneffe V, Triffaux M, George B. Anatomical variations of the V2 segment of the vertebral artery. Operative. Neurosurgery. 2006;59:ONS-20–4.
53. Kubikova E, Osvaldova M, Mizerakova P, El Falougy H, Benuska J. A variable origin of the vertebral artery. Bratisl Lek Listy. 2008;109:28–30.
54. Shin HY, Park JK, Park SK, Jung GS, Choi YS. Variations in entrance of vertebral artery in Korean cervical spine: MDCT-based analysis. Korean J Pain. 2014;27:266–70.
55. Tubbs RS, Salter G, Wellons JC, Oakes WJ. Blood supply of the human cervical sympathetic chain and ganglia. Eur J Morphol. 2002;40:283–8.
56. Brink B. Approaches to the second segment of the vertebral artery. In: Berguer R, Bauer R, editors. Vertebrobasilar arterial occlusive disease. New York: Raven Press; 1984. p. 257–64.
57. Özgen S, Pait TG, ÇağLar YŞ. The V2 segment of the vertebral artery and its branches. J Neurosurg Spine. 2004;1:299–305.
58. Gkekas N, Georgakoulias N. The medial loop of the V2 segment of the vertebral artery and the importance of this variation in correlation with the resection safe zone and technical characteristics of spinal cages during anterior cervical approaches. Acta Neurochir. 2013;155:1247–8.
59. Ulm AJ, Quiroga M, Russo A, Russo VM, Graziano F, Velasquez A, Albanese E. Normal anatomical variations of the V3 segment of

the vertebral artery: surgical implications: laboratory investigation. J Neurosurg Spine. 2010;13:451–60.
60. Kim MS. Developmental anomalies of the distal vertebral artery and posterior inferior cerebellar artery: diagnosis by CT angiography and literature review. Surg Radiol Anat. 2016;38:997–1006.
61. Lasjaunias P, Vallee B, Person H, Ter Brugge K, Chiu M. The lateral spinal artery of the upper cervical spinal cord: anatomy, normal variations, and angiographic aspects. J Neurosurg. 1985;63:235–41.
62. Siclari F, Burger IM, Fasel JHD, Gailloud P. Developmental anatomy of the distal vertebral artery in relationship to variants of the posterior and lateral spinal arterial systems. Am J Neuroradiol. 2007;28:1185–90.
63. Uchino A, Saito N, Watadani T, Okada Y, Kozawa E, Nishi N, Mizukoshi W, Inoue K, Nakajima R, Takahashi M. Vertebral artery variations at the C1-2 level diagnosed by magnetic resonance angiography. Neuroradiology. 2012;54:19–23.
64. de Oliveira E, Rhoton AL, Peace D. Microsurgical anatomy of the region of the foramen magnum. Surg Neurol. 1985;24:293–352.
65. Wilkinson IMS. The vertebral artery: extracranial and intracranial structure. Arch Neurol. 1972;27:392.
66. Akar ZC, Dujovny M, Slavin KV, Gomez-Tortosa E, Ausman JI. Microsurgical anatomy of the intracranial part of the vertebral artery. Neurol Res. 1994;16:171–80.
67. Songur A, Gonul Y, Ozen OA, Kucuker H, Uzun I, Bas O, Toktas M. Variations in the intracranial vertebrobasilar system. Surg Radiol Anat. 2008;30:257–64.
68. Tubbs RS, Shah NA, Sullivan BP, Marchase ND, Cohen-Gadol AA. Surgical anatomy and quantitation of the branches of the V2 and V3 segments of the vertebral artery. Laboratory investigation. J Neurosurg Spine. 2009;11:84–7.
69. D'Antoni AV, Battaglia F, Dilandro AC, Moore GD. Anatomic study of the suboccipital artery of Salmon with surgical significance. Clin Anat. 2010;23:798–802.
70. Macchi V, Porzionato A, Morra A, D'Antoni AV, Tubbs RS, De Caro R. Anatomico-radiologic study of the distribution of the suboccipital artery of Salmon. Clin Neurol Neurosurg. 2014;117:80–5.
71. George B, Laurian C. Surgical possibilities in the third portion of the vertebral artery (above C2). Anatomical study and report of a case of anastomosis between subclavian artery and vertebral artery at C1-C2 level. Acta Neurochir Suppl (Wien). 1979;28:263–9.
72. Standring S, Gray H. Gray's anatomy: the anatomical basis of clinical practice. Edinburgh: Churchill Livingstone/Elsevier; 2008.
73. Bruneau M, Cornelius JF, George B. Antero-lateral approach to the V3 segment of the vertebral artery. Neurosurgery. 2006;58:ONS29–35. discussion ONS29–35.
74. Lister JR, Rhoton AL, Matsushima T, Peace DA. Microsurgical anatomy of the posterior inferior cerebellar artery. Neurosurgery. 1982;10:170–99.
75. Marinković S, Milisavljević M, Gibo H, Maliković A, Djulejić V. Microsurgical anatomy of the perforating branches of the vertebral artery. Surg Neurol. 2004;61:190–7.
76. Tanoue S, Endo H, Hiramatsu M, et al. Delineability and anatomical variations of perforating arteries from normal vertebral artery on 3D DSA: implications for endovascular treatment of dissecting aneurysms. Neuroradiology. 2021;63:609–17.
77. Gluncic V, Ivkic G, Marin D, Percac S. Anomalous origin of both vertebral arteries. Clin Anat. 1999;12:281–4.
78. Gabrielli R, Rosati MS. Ataxia and vertigo due to anomalous origin of the left vertebral artery. J Vasc Surg. 2013;58:803.
79. Bernardi L, Dettori P. Angiographic study of a rare anomalous origin of the vertebral artery. Neuroradiology. 1975;9:43–7.
80. Komiyama M, Morikawa T, Nakajima H, Nishikawa M, Yasui T. High incidence of arterial dissection associated with left vertebral artery of aortic origin. Neurol Med Chir (Tokyo). 2001;41:8–11. discussion 11–12
81. Lale P, Toprak U, Yagız G, Kaya T, Uyanık SA. Variations in the branching pattern of the aortic arch detected with computerized tomography angiography. Adv Radiol. 2014;2014:1–6.
82. Rathore MH, Sreenivasan VV. Vertebral and right subclavian artery abnormalities in the down syndrome. Am J Cardiol. 1989;63:1528–9.
83. Al-Okaili R, Schwartz ED. Bilateral aortic origins of the vertebral arteries with right vertebral artery arising distal to left subclavian artery: case report. Surg Neurol. 2007;67:174–6. discussion 176.

Anatomy and Variations of the Posterior Inferior Cerebellar Artery

Thomas Robert, Gabriele Cicciò, and Sara Bonasia

Observations concerning the posterior inferior cerebellar artery (PICA) are mostly based on embryological studies, cadaveric dissections, surgical and angiographic evaluation [1–3]. The correlation between vascular occlusion and clinical implications as well as the evaluation of space-occupying brain lesions has encouraged numerous researches, thus exploring a scientific path of considerable interest [4]. This allowed embryologists, anatomists, neurosurgeons, neurologists and ultimately interventional neuroradiologists to carry out an extensive and in-depth research for the PICA. This phylogenic recent artery is one of the most complex and fascinating cerebral arteries with a high number of anatomical variations perhaps due to its late embryological development [5, 6]. In this chapter, we will develop each variation implicating the PICA after a little embryological remind and "normal anatomy" description.

History

Pioneers in the anatomical studies, Gaspard Vieusseux in 1810 followed by Adolph Wallenberg's observations in 1895, undisputedly proposed a remarkable focus on the spino-cerebellar arteries including the PICA [7, 8]. Continuing with this clinico-anatomical correlation method, the studies of Wilson [9], Harris [8], Gowler and Hope [10] represent a bridge of scientific connection with writings of Krayenbuhl and Yasargil in 1957 [11], and of Margolis and Newton 1972 [12]. The amazing and milestone studies of Congdon, [13] and Padget [2] on embryology followed by those of Lasjaunias allowed a solid scientific discussion, providing explanations and formulating concrete hypotheses about anatomical variations of the PICA [1, 14].

Embryological Development

In this section, we will only explain the embryological development of the cranio-cervical vascular system with the aim to understand the different variations of the PICA. In order to simplify this complex embryological development, we first describe the general development of the posterior circulation; after that, we will focus on the cranio-cervical junction. The main steps of the PICA development are summarized and illustrated in Table 1. Our embryological knowledge is based on key publications provided by the studies and dissections of Congdon and Padget in human specimens and by Moffat in rat [2, 6, 13]. In more recent publications based on observations of anatomical variations, authors proposed very interesting but usually contradicting hypotheses [14–16].

General Posterior Circulation Development

Even if the formation of the posterior circulation succeeds early in the embryological life, it is always developed later than the carotid system and the anterior circulation [5, 17].

At stage I of Padget (4–5 mm), the paired longitudinal neural arteries (LNA) appear, fed cranially by the trigeminal artery (TA) and caudally by the proatlantal artery (PA). Other carotido-vertebral anastomosis formed slightly later with the formation of the otic and hypoglossal arteries (OtA and HypA) [2, 13, 17].

At stage II of Padget (5–6 mm), the two LNAs initiate their medial fusion to form the future basilar artery. The pos-

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Table 1 Major embryological changes in the formation of the PICA

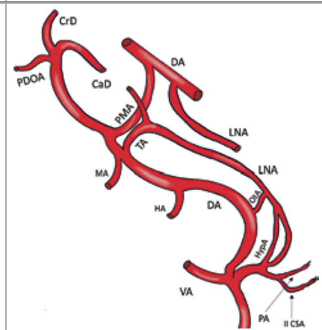
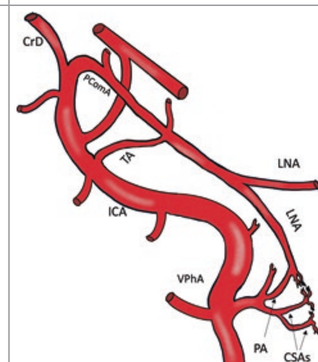
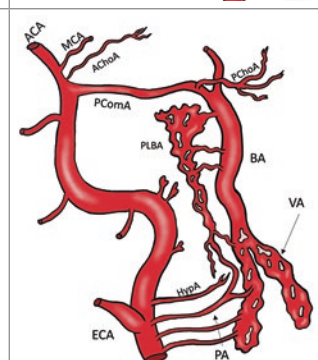
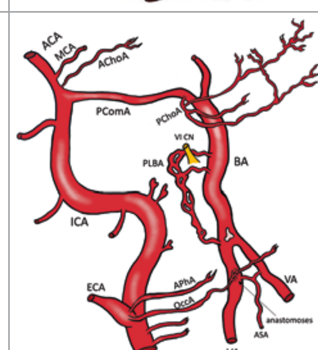
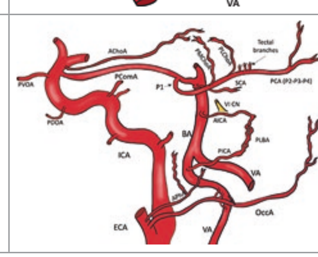
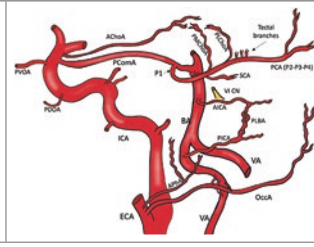
Stage	Embryo size (mm)	Major evolutions	Graphic illustration
I	4–5	• Paired longitudinal neural arteries (LNAs) visible • Carotido-vertebral anastomoses visible: Trigeminal (TA), otic (OtA), hypoglossal (HypA), proatlantal (PA) and second cervical segmental artery (II CSA) • Primitive ICA bifurcation visible into caudal and cranial divisions (CaD, CrD) • Posterior communicating artery (PComA) and BA not formed	
II	5–6	• Formation of the PComA • Regression of otic and hypoglossal arteries • Midline fusion of LNAs to form the basilar artery (BA) • Initial transverse anastomoses between the intersegmental arteries	
III	7–12	• Basilar artery (BA) completely formed • PComA completely formed • Regression of trigeminal and proatlantal arteries • Formation of the vertebral artery (VA) by transverse anastomoses between the intersegmental arteries • Lateral basilo-vertebral anastomosis (PLBA) visible	
IV	13–15	• Remodeling of the BA and development of the PCA • Anastomotic ring around the VI cranial nerve visible • Migration of occipital artery (OccA) origin on the external carotid artery (ECA) • Persistence of the anastomoses between OccA and VA • Formation of the anterior spinal artery (ASA)	
V	16–18	• Complete formation of the VA • Complete formation of the OccA (remnant of the proatlantal artery) • Complete formation of the ascending pharyngeal artery (APhA, remnant of the hypoglossal artery) • Potential stem of the AICA visible	

Table 1 (continued)

Stage	Embryo size (mm)	Major evolutions	Graphic illustration
VI	20–24	• Complete development of the Circle of Willis • Interaction between the primitive stem of the AICA and PICA with the PLBA to determine its definitive configuration	

terior communicating artery is completely formed and, consequently, the TA regresses [2].

At the stage III of Padget (7–12 mm), the vertebral artery (VA) is formed by anastomosis of the different intersegmental cervical arteries. At the cranio-cervical junction, arteries have a more plexiform aspect, and the vertebrobasilar junction is not completely formed [17].

At stage V of Padget (16–18 mm), numerous small arteries develop from the vertebral artery and course cranially to the choroid plexus of the posterior fossa, which could be interpreted as precursor of the proximal stem of the PICA [1, 17]. Some authors arbitrarily named it the artery of the restiform body [1].

At stage VI (20–24 mm), D. Padget noted the difficulty to recognize stems of the anterior inferior cerebellar artery (AICA) and PICA among many arterial branches supplying the posterior part of the hindbrain [2].

Cranio-Cervical Junction Vascular Development

The proatlantal artery or first cervical intersegmental artery plays a cornerstone role in the formation of the arteries of the cranio-cervical junction. This artery is a transverse artery that passes in the first intersegmental space (between the occiput and the atlas) and gives two distinct branches [14]. The anterior branch passes anterior to the medulla and forms with its two rami the vertebrobasilar junction and the future anterior spinal arteries. The posterior branch of the PA also develops two distinct rami: an ascending one and a descending one. These two posterior rami play a role in the formation of the proximal stem of the PICA [1]. D. Padget had already seen in embryos of 7–12 mm (stage III) the development of a lateral channel parallel to the LNA passing posterior to the cranial nerves VI and XII but anterior to cranial nerves VII through XI and named it the *primitive lateral basilovertebral anastomosis* (PLBA) [2]. This could be the cranial part (ascending ramus) of the posterior branch of the PA. On the other hand, Moffat also noted in rat a similar vascular network in the upper cervical region that he named the *lateral longitudinal artery* which could be the extracranial part (descending ramus) of the posterior branch of the PA [6]. In embryos of 16–24 mm (stage V and VI), D. Padget noted the development of arterial branches from the PLBA that could be the precursor of stems of the AICA and PICA but she highlighted that the high number of branches in this region make difficult to recognize which of them are the stems of the two future cerebellar arteries [2].

Even if we do not have any strong embryological argument but only based on observations of anatomical variations, few authors rightly hypothesized that the PLBA participates in the formation of the AICA and PICA in the posterior fossa. For the upper cervical region, the PLBA or more precisely the lateral longitudinal artery of Moffat could be the precursor of a spinal artery named the lateral spinal artery (LSA) by Lasjaunias et al. but interpreted differently by other authors [14–16].

Whatever the name you give to these structures, the PLBA described by Padget in human embryo and the lateral longitudinal artery described by Moffat in rat have a lot of similitudes and seem to be the same entity. On the other hand, the late formation of stems of the AICA and PICA from a plexiform network, together with the formation in the upper cervical spine of the lateral spinal (or postero-lateral spinal artery for other authors), explains most of anatomical variations of arteries of the cranio-cervical junction including the proximal PICA.

Number and Origin of the Artery

The PICA is the cerebellar artery with the highest incidence of variations in origin, course and number. It seems that its late formation in the embryological life and the complexity of the cranio-cervical junction vasculature are the two major causes of this high variability.

"Normal" Origin

In the "normal" anatomical description, the PICA arises from the distal segment of the vertebral artery (VA) [3, 18]. With its lateral course around the medulla oblongata, the

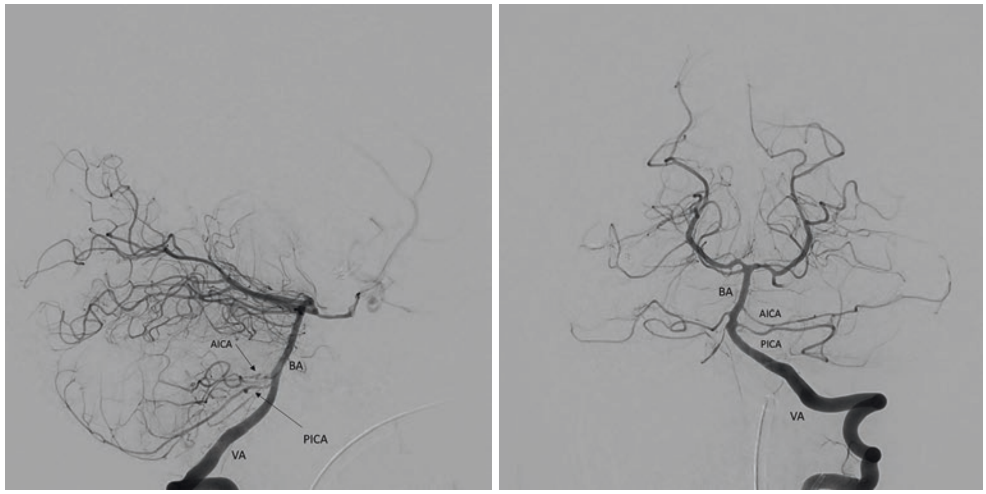


Fig. 1 Clinical case of BA origin of the PICA. The figure shows a Digital subtraction angiography (DSA) obtained after the injection of the left vertebral artery (VA) in lateral and antero-posterior view. On the left side of the basilar artery (BA) are visible the origin of the AICA and of the PICA. On the VA, the PICA origin is not visible

PICA branches off from the lateral or posterior aspect of the vertebral artery [3]. The distance between the point of origin of the artery and the vertebrobasilar junction is 16.8 mm (0–35 mm) [3, 11, 19]. This "normal" anatomy is found in 70–90% with a high variability among different studies depending on the technique of evaluation [19]. A case of PICA origin from the basilar artery (BA) is shown in Fig. 1.

"Absence" of the PICA

Few authors described an "absence" or an "agenesis" of the PICA in relatively high incidence (2–24%) which could be more precisely defined as the absence of the classical origin of the PICA [18–21]. In these cases, the territory of the PICA is taken in care by another cerebellar artery, or its origin is from another artery.

AICA Origin of the PICA (AICA-PICA)

AICA-PICA is the most frequent variation of the PICA origin (2–8%) [22, 23]. In this configuration, the PICA does not originate from the vertebral artery but is a branch of the AICA. A case of this variant is shown in Fig. 2. This variant could be explained by the partial persistence of the PLBA during the embryological life [1]. Lasjaunias et al. proposed a four-type classification as follows:

- Type I: a single trunk arises from proximal segment of basilar artery with distal bifurcation in AICA and PICA.
- Type II: a bifid PICA arises from intradural segment of vertebral artery.

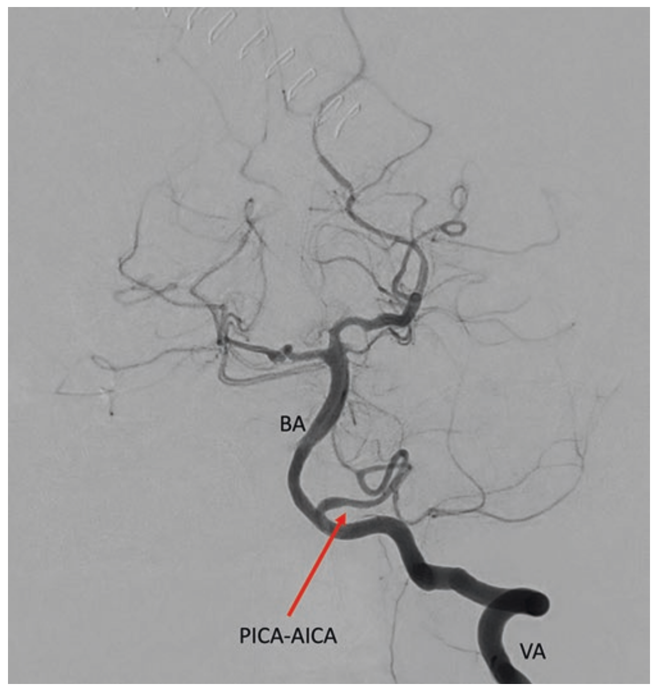


Fig. 2 Clinical case of PICA-AICA variant. The figure shows a DSA obtained after the injection of the left vertebral artery (VA). On the distal part of the VA, a PICA-AICA complex is visible. It is defined as the absence of AICA origin from the basilar artery (BA) with its territory supplied by a superior branch of the PICA

- Type III: a bifid PICA arises from vertebrobasilar junction.
- Type IV: a normal PICA without AICA.

Rare distal aneurysms of an AICA-PICA were described but represent a challenging situation. In this case, major attention must be taken to preserve a large cerebellar territory [24–26].

Extradural Vertebral Artery Origin of the PICA

This variant is clearly correlated with the development of the VA, lateral spinal (LSA), posterior spinal arteries (PSAs) and the proatlantal intersegmental artery (ProA) as described in the section about embryology. Lasjaunias et al. explained this variant by dilatation of a radicular artery at C1, C2 or C3 anastomoses with a dilated lateral spinal artery that continues as the normal PICA [1, 14]. Other authors suggest that these variations could also implicate the posterior spinal artery instead of the lateral spinal artery [16]. Extradural origin of the PICA has been reported in 5–19%, with prevalence on the nondominant VA [27–29] In this configuration, the PICA usually arises from dorsal aspect of the VA and the perforating arteries arise from the distal VA instead of the proximal PICA [30, 31]. It could be associated with a fenestration of the VA between C1 and C2 that highlights the role of the PLBA in these variants. A clinical case of extradural VA origin is shown in Fig. 3.

Occipital Artery Origin of the PICA

This variant represents a partial persistence of the primitive proatlantal artery that explains the anastomosis between the occipital artery and the vertebrobasilar system. Consequently, the PICA could arise from the occipital artery at the level C1 or C2 depending on the type of persistent proatlantal artery (type I or II) [32–34].

Ascending Pharyngeal Artery Origin of the PICA

Few cases of the ascending pharyngeal artery (APhA) origin of the PICA have been described [35–39]. Initially, Lasjaunias et al. interpreted it as a partial persistence of the hypoglossal artery thinking that the anastomotic vessel passes through the hypoglossal canal [36]. More recent publications with high-definition slice images demonstrated that the anastomotic vessel passes through the jugular canal instead of the hypoglossal one [37, 38]. In this situation, the hypothesis would be a persistence of the glosso-pharyngeal artery.

Cavernous Internal Carotid Artery (ICA) Origin of the PICA

This variation in origin of the PICA is not so frequent and only described in a few cases [12, 15, 40–45]. The PICA takes its origin from the pre-cavernous or cavernous portion of the ipsilateral ICA, courses posteriorly in the cavum of Meckel to take in charge the territory of the PICA. The embryological explanation is a partial perstistence of the primitive trigeminal artery (PTA). In fact, the PTA is connected to the cranial part of the PLBA during the embryological life. Its persistence usually gives a direct connection between the ICA and the basilar artery, but various variations have been described by Saltzman [46]. In the type III of Saltzman, the PTA connects posteriorly to a cerebellar artery and, in particular, to the PICA in type IIIc. Perot et al. described a particular case where only the hemisphere trunk of the PICA is connected to the PTA [43]. Another case was published by Siclari et al. where, surprisingly, all the three cerebellar arteries raised from the primitive trigeminal artery [16].

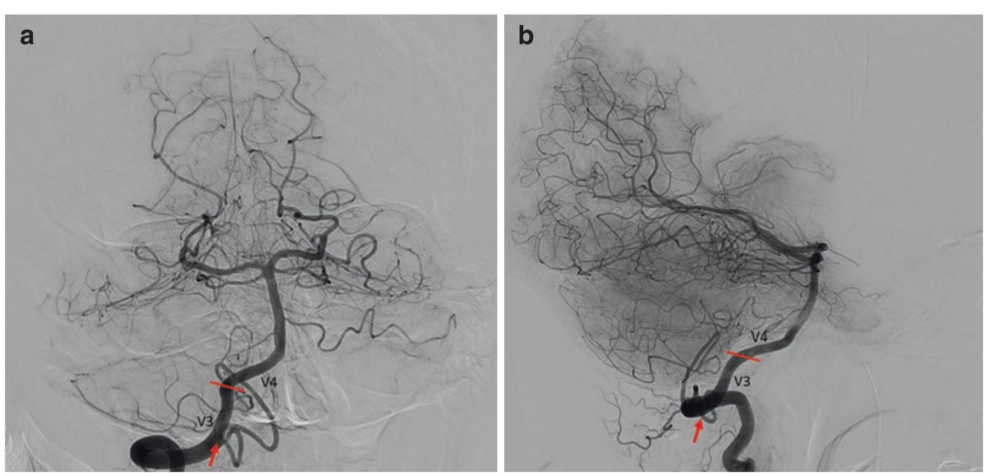


Fig. 3 Clinical case (**a** and **b**) of extradural origin of the PICA. The PICA normally origins from the intradural segment of the vertebral artery (V4). It can also arise from the extradural portion of the vertebral artery. In this case the PICA arises from the VA at the level of C1 (V3 segment) and then courses intradural to supply its usual vascular territory

Cervical ICA Origin of the PICA or Persistent Hypoglossal Artery Variant

Few cases of origin of the PICA from the cervical portion of the ICA have been described and the most frequent hypothesis is a partial persistence of the primitive hypoglossal artery. Recent publications argued this hypothesis showing the course of the artery through the hypoglossal canal [47–50].

Anterior Spinal Artery Origin of the PICA

Only two cases of anterior spinal artery (ASA) origin of the PICA have been described. Authors postulated that the PICA is connected to the ASA through a dilated coronary artery that goes around the medulla [51, 52].

Double Origin of the PICA

This variant could be found in the literature as supernumerary PICA, double origin of PICA, bifid PICA, DOPICA or double trunk of PICA [4, 53–60]. It is defined by a double origin with two separate vessels arising from the same vertebral artery with a distal convergence, usually at the midportion of anterior medullary segment. According to Lasjaunias et al., the double origin of the PICA represents an embryologic remnant of the normal anastomosis between LSA and PICA (remnant of the PLBA) [1, 14, 61]. The cranial origin of the PICA is the PICA proper and the caudal one is a dilated LSA. The caudal PICA could arise from the vertebral artery at C1 or C2 level. It could also be associated with a fenestration of the VA of the same side [62, 63].

Course of the Artery

The PICA has the particularity to be tortuous making different loops around the cerebellar structures [64]. Its median diameter at origin is 2.0 mm (0.5–3.4 mm). According to Rhoton, the PICA can be divided into five segments as follows [3, 18, 65]. The PICA's segments are shown in Fig. 4.

Anterior Medullary Segment

This segment extends from the origin of the artery to the point where the artery crosses the most prominent point of the olive. The presence of this segment is dependent on the course of the vertebral artery and on the level of origin of the PICA. When the PICA has a low origin along the VA, this first segment does not exist. The PICA usually passes below the hypoglossal nerve in 47.5%, above in 37.5% and between the rootlets of the nerve in 12.5% [66].

Lateral Medullary Segment

This segment begins when the PICA passes the most prominent point of the olive and ends at the level of origin of the glossopharyngeal, vagus and accessory nerves. This segment is present in most PICA and its course shows high variability.

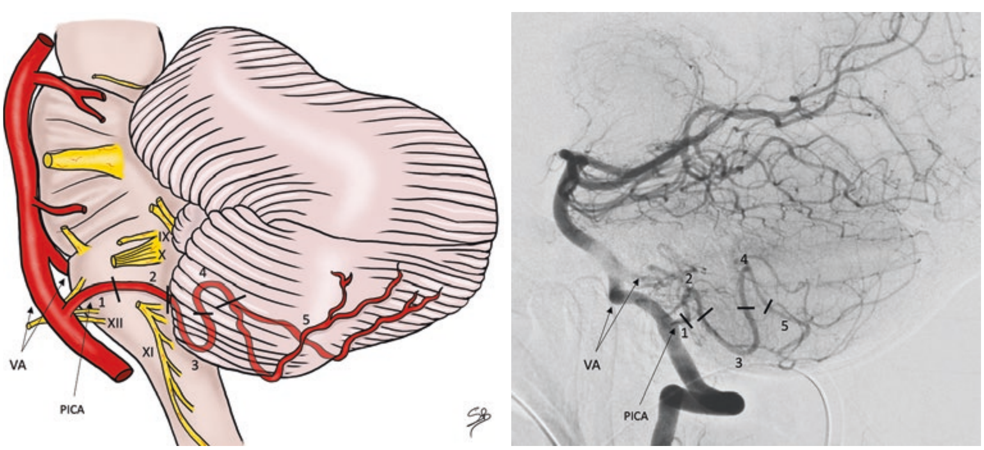


Fig. 4 Artist's drawing and DSA image of PICA segments. The posterior inferior cerebellar artery (PICA) arises from the distal portion of the vertebral artery (VA). The antero-medullary segment (*1*) courses below the hypoglossal nerve (XII) and ends at the most prominent part of the olive. The lateral medullary segment (*2*) courses until the origin of the vagal, glossopharyngeal and accessory nerves (IX, X, XI). The tonsillomedullary segment (*3*) courses from the origin of these nerves and ends on the medial surface of the tonsil. The telovelotonsillar segment (*4*) begins at the medial surface of the tonsil and ends at the exit point from the fissure between the vermis and the cerebellar hemisphere. The cortical segment (*5*) includes the terminal branches of the artery

In most cases, the PICA passes through the rootlets of the accessory nerve (65%) but could also course between the vagus and accessory nerves (in 20% of cases) or between the glossopharyngeal and vagus nerves in 12.5% [66].

Tonsillomedullary Segment

This segment begins where the PICA passes posterior to the glossopharyngeal, vagus and accessory nerves. It ends where the artery ascends to the mid-level of the medial surface of the tonsil. In most cases, along this segment, the artery presents a typical caudal or tonsillar loop passing around the tonsil. This loop could be in straight relation with the caudal point of the tonsil but could also be lower to it. On the contrary, when the PICA has a low origin, the artery passes directly on the medial surface of the tonsil without forming this caudal loop [18].

Telovelotonsillar Segment

This segment is the most complex one with a high variability. It begins at the midportion ascent of the artery along the medial aspect of the tonsil. The end of this segment is where the artery exits from the fissure between the vermis and the cerebellar hemisphere. Where the artery passes on the inferior medullary velum, it forms another typical loop called the cranial loop where the artery gives its branches to the tela choroidea and to the choroid plexus.

Cortical Segment

This segment extends from the point where PICA exits from the fissure between the vermis and the hemisphere, to the terminal branches. The bifurcation of the artery in medial and lateral trunk is usually on the proximal portion of this segment. The lateral or hemispheric trunk is the largest one reaching the hemispheric and tonsillar surfaces. The medial trunk is smaller and courses on the posterior surface of the vermis. In only 5% of cases, no bifurcation of the PICA could be seen [18].

Branches

P. Lasjaunias compared the PICA to a hypertrophied radiculopial artery that allows to understand the embryological development of the artery and also its different type of branches: perforating, choroidal and cortical [1, 67].

Perforating Branches

Perforating branches origin from the first three segments of PICA and supply the medulla oblongata. Two types of these branches have been described: direct and circumflex [3, 18, 65]. The direct perforating branches have a course around the brainstem of less than 90 ° before their perforating point. On the contrary, circumflex perforating branches have a course of more than 90 ° around the brainstem before entering in it. All these perforating arteries together with perforating arteries from the vertebral artery form an arterial plexus that supply the lateral part of the medulla oblongata [1, 21, 68]. The anterior segment of the PICA has between 0 and 2 branches, its lateral medullary segment between 0 and 5 and the tonsillomedullary segment 0–11 branches. For ectopic origin of the PICA or low-lying PICA, perforating branches arise from the vertebral artery instead of the PICA [27, 61].

Choroidal Branches

PICA usually supplies most of the choroid plexus of the fourth ventricle and the tela choroidea. Choroidal branches arise mostly from tonsillomedullary and telovelotonsillar segments but little branches from the lateral medullary segment of the PICA also supply the lateral part of the choroid plexus passing through the foramen of Luschka [12, 18].

Cortical Branches

The cortical arteries arise from the distal segment of PICA and are divided into vermian, hemispheric and tonsillar branches, with frequent overlap in the areas supplied [3, 18].

Vermian branches usually arise from the medial trunk and usually supply the inferior vermis and the adjacent part of the hemisphere. Two vermian branches (0–3) are identified in most hemispheres.

Hemispheric branches usually arise from the lateral trunk and show variability in number and extension. Three hemispheric branches (0–9) supply the suboccipital portion of the hemisphere lateral to the vermis.

Tonsillar branches also arise from the lateral trunk and have a high variability in number.

Inconstant Branches

Lasjaunias et al., in a didactic article, described in 1985 the lateral spinal artery postulating a strong hypothesis about its embryological development [14]. They showed that this artery arises from the lateral segment of the PICA in 73% of cases. This data has been confirmed later in cadaveric studies [61].

Rare cases of PICA origin of the posterior meningeal artery (PMA) have been described in the literature [49, 69]. The PMA is normally a branch of the vertebral artery but only in rare case it could arise from the tonsillomedullary of the PICA highlighting a possible embryological anastomosis between dural and parenchymal territories.

PICA-AICA

On the contrary to the AICA-PICA variants described previously, the PICA-AICA is defined as the absence of AICA with its territory supplied by a superior branch of the PICA. In this configuration that highlights one more time the role of the PLBA in the development of cerebellar arteries, the AICA is considered as a branch of the PICA [1, 21].

Bihemispheric PICA

In case of absence of the PICA, the contralateral PICA could supply both cerebellar hemispheres, this is the definition of the bihemispheric PICA [70–72]. Few authors named the branch of the PICA that crosses the midline the "PICA communicating artery" [73, 74]. The PICA on the dominant vertebral artery is more frequently involved but the true incidence of this anatomical variation is not known (between 0.1 to 3.6%) [1]. Two types of bihemispheric PICA have been described:

- Type I or "true" bihemispheric PICA when the PICA completely supplies the contralateral PICA territory.
- Type II or vermian variant when only the contralateral vermis is supplied by the PICA. The hemisphere is consequently supplied by the AICA or the superior cerebellar artery (SCA).

Few cases of aneurysm located at the origin of a bihemispheric PICA have been described highlighting the importance of the hemodynamic effect on the formation of such aneurysms. The treatment of these aneurysms is also more challenging because an ischemic complication could involve both cerebellar hemispheres [73, 75]. A case of bihemispheric PICA is shown in Fig. 5.

Possible Anastomoses

The PICA presents few anastomoses with other arteries that could be classified in two types: pio-pial anastomosis and persistence of embryological anastomosis [76]. The cerebellum is entirely supplied by its three cerebellar arteries that divide between them this large territory. A high variability of cerebellar territory among these three arteries is noted and a lot of terminal anastomoses are presented between their branches. Consequently, PICA anastomoses with branches of the AICA and the SCA that are important in case of proximal occlusion of one of these three arteries to limit the extent of a cerebellar infarct [8].

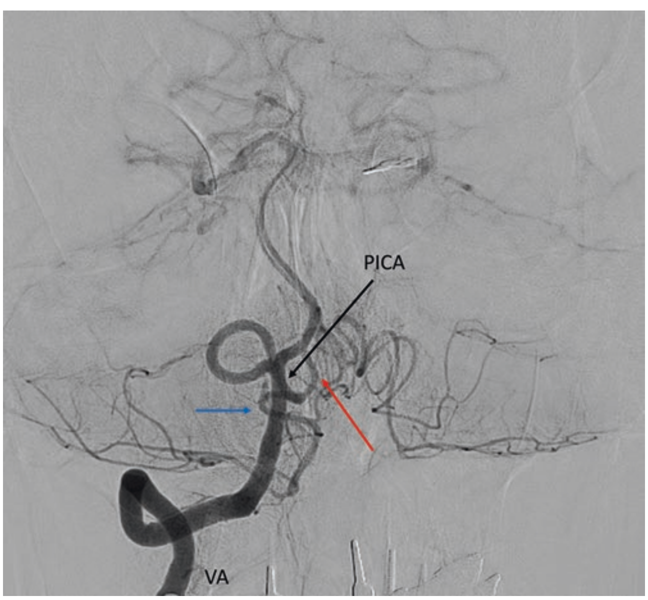


Fig. 5 Clinical case of bihemispheric PICA. The figure shows a DSA obtained after the injection of the right vertebral artery (VA). The PICA arises from the distal VA and gives a branch to the right (blue arrow) and left hemisphere (red arrow) to supply both cerebellar hemispheres

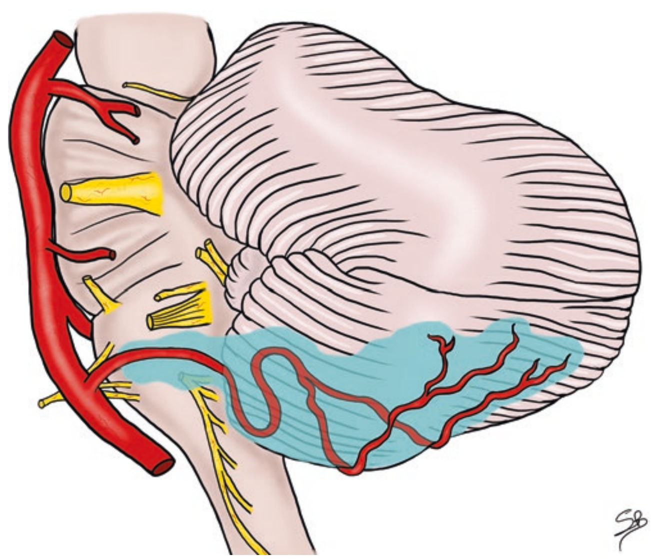


Fig. 6 Illustration of the parenchymal territory of the PICA (light blue area). The territory supplied by the PICA includes the lateral portion of the medulla, the inferior part of the vermis, the uvula, the pyramids, the inferior part of the fourth ventricle and the tonsils. Also the biventral lobule is usually supplied by the PICA

Other anastomoses could be explained by the persistence of an embryological vessel. This is the case of a partial persistence of the proatlantal artery that leaves an occipital artery originating from an extra-dural PICA [14, 33]. Another example is the anastomosis between the proximal segment of the PICA and the anterior spinal artery [51, 77].

Parenchymal and/or Dural Territories

The territory of PICA is the most variable of cerebellar arteries [3, 18, 65]. Its parenchymal territory is illustrated in Fig. 6 and could be summarized as follows:

- Lateral part of the medulla oblongata
- Nucleus gracilus
- Inferior part of the vermis: uvula, pyramid
- Nodule and flocculus
- Inferior part of the fourth ventricle
- Tonsils
- Biventral lobule
- Semilunar lobules (inconstant)
- Emboliform and globose nuclei
- Dentate nucleus (not accepted by all authors)

Variants

Most of the variants concerning the PICA have been discussed in the preceding paragraphs. Here, we describe other two anatomical variations that could not be incorporated in another section.

VA-PICA

VA-PICA is characterized by a vertebral artery (usually hypoplastic) that does not connect with contralateral vertebral artery or with basilar artery to form the usual vertebrobasilar junction and terminates in PICA [78]. This vascular configuration could be considered as a variant of the vertebral artery and not exactly of the PICA. Considered as a relatively common variant, its real incidence is not really known (0.4–18.7%) [1, 79].

Fenestration of PICA

The definition of a fenestration of an artery is a luminal division of a single vessel with two distinct endothelia but in only one adventitia that differentiates it from a duplication [1]. The incidence of PICA fenestration is very low, not comparable to the more common vertebral or basilar fenestrations. It could be explained by a partial regression of double-PICA or by a cranial nerve entrapment [80, 81]. The fenestration always involved the proximal part of the PICA where the artery crosses the cranial nerves. Few cases of fenestration on a double PICA have been published that emphasize the role of the lateral spinal artery in the development of the proximal segment of the PICA [53, 62].

Clinical Implications

PICA is the most important cerebellar artery and is involved in ischemic and hemorrhagic pathologies [82].

Ischemic stroke of the PICA is frequent and leads to a typical lateral medullary syndrome or Wallenberg's syndrome. It is always the consequence of atherosclerosis, cardiac embols or a vertebral artery dissection. Most of clinical signs of the Wallenberg's syndrome are the clinical expression of the perforating arteries of the PICA. The variability in origin of these little arteries that could arise from the PICA or from the distal VA depending on the origin of the PICA itself allows to understand the different clinical situations of this syndrome.

Aneurysms of the PICA represents only 1–4% of all intracranial aneurysms but have a high tendency to bleed. Most of anatomical variations in origin of the PICA have been described in the presence of a proximal aneurysm on the artery that highlights the hemodynamic factor in the development of such aneurysms [83]. The treatment of a PICA aneurysm in the presence of a variation could be more challenging and a lack of knowledge or analysis of the variants could lead to catastrophic complications [84, 85].

The PICA has a straight relation with lower cranial nerves and could have a conflict with the glosso pharyngeal nerve. Even if this pathology is rare, the knowledge of the anatomy of the PICA with its perforating branches is mandatory to treat surgically such pathology.

References

1. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical Neuroangiography. Berlin: Springer edition; 2001.
2. Padget DH. The development of cranial arteries in the human embryo, Contribution to embryology, vol. 212. Washingtion: Carnegie Institution; 1948. p. 205–62.
3. Rhoton AL Jr. The cerebellar arteries. Neurosurgery. 2000;47(3 Suppl):S29–68. https://doi.org/10.1097/00006123-200009001-00010.
4. Singh R, Kumar R, Kumar A. Vascular anomalies of posterior fossa and their implications. J Craniofac Surg. 2017;28(8):2145–50. https://doi.org/10.1097/SCS.0000000000003867.
5. Abbie AA. The morphology of the fore-brain arteries, with especial reference to the evolution of the basal ganglia. J Anat. 1934;68(Pt 4):433–70.
6. Moffat DB. The embryology of the arteries of the brain. Ann R Coll Surg Engl. 1962;30:368–82.

7. Marcet. History of a singular nervous or paralytic affection attended with anomalous morbid sensations. Med Chir Trans. 1811;MCT-2:217–35.
8. Harris W. A case of thrombosis of the left posterior inferior cerebellar artery followed by severe trigeminal neuralgia in the analgesic facial area, vol. 3; 1910. p. 81.
9. Wilson SA. A case of thrombosis of the left posterior inferior cerebellar artery. Proc R Soc Med. 1909;2(Neurol Sect):52–76.
10. Gowler DR, Hope BM. A Case of occlusion of the right posterior inferior cerebellar artery. J Neurol Psychopathol. 1923;4(13):43–9. https://doi.org/10.1136/jnnp.s1-4.13.43.
11. Yasargil MG. Microneurosurgery. New York: Thieme Medical Publisher; 1984.
12. Margolis MT, Newton TH. Borderlands of the normal and abnormal posterior inferior cerebellar artery. Acta Radiol Diagn (Stockh). 1972;13(1):163–76. https://doi.org/10.1177/02841851720130p122.
13. Congdon ED. Transformation of the aortic-arch system during the development of the human embryo. Contrib Embryol Carneg Instn. 1922;68:47–110.
14. Lasjaunias P, Vallee B, Person H, Ter Brugge K, Chiu M. The lateral spinal artery of the upper cervical spinal cord. Anatomy, normal variations, and angiographic aspects. J Neurosurg. 1985;63(2):235–41. https://doi.org/10.3171/jns.1985.63.2.0235.
15. Gregg L, Gailloud P. The role of the primitive lateral Basilovertebral anastomosis of Padget in variations of the Vertebrobasilar arterial system. Anat Rec (Hoboken). 2017;300(11):2025–38. https://doi.org/10.1002/ar.23633.
16. Siclari F, Burger IM, Fasel JH, Gailloud P. Developmental anatomy of the distal vertebral artery in relationship to variants of the posterior and lateral spinal arterial systems. AJNR Am J Neuroradiol. 2007;28(6):1185–90. https://doi.org/10.3174/ajnr.A0498.
17. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: a pictorial review. Surg Radiol Anat. 2021;43(6):961–73. https://doi.org/10.1007/s00276-021-02684-y.
18. Lister JR, Rhoton AL Jr, Matsushima T, Peace DA. Microsurgical anatomy of the posterior inferior cerebellar artery. Neurosurgery. 1982;10(2):170–99.
19. Saylam C, Ucerler H, Orhan M, Cagli S, Zileli M. The relationship of the posterior inferior cerebellar artery to cranial nerves VII-XII. Clin Anat. 2007;20(8):886–91. https://doi.org/10.1002/ca.20541.
20. Akgun V, Battal B, Bozkurt Y, Oz O, Hamcan S, Sari S, et al. Normal anatomical features and variations of the vertebrobasilar circulation and its branches: an analysis with 64-detector row CT and 3T MR angiographies. ScientificWorldJournal. 2013;2013:620162. https://doi.org/10.1155/2013/620162.
21. Margolis MT, Newton TH. The posterior inferior cerebellar artery. In: Newton TH, Potts DG, editors. Radiology of the skull and brain. St Louis: Mosby; 1974. p. 1710–74.
22. Brinjikji W, Cloft H, Kallmes DF. Anatomy of the posterior inferior cerebellar artery: relevance for C1-C2 puncture procedures. Clin Anat. 2009;22(3):319–23. https://doi.org/10.1002/ca.20785.
23. Sharifi M, Ciszek B. Bilaterally absent posterior inferior cerebellar artery: case report. Surg Radiol Anat. 2013;35(7):623–5. https://doi.org/10.1007/s00276-013-1073-9.
24. Akhtar S, Azeem A, Jiwani A, Javed G. Aneurysm in the anterior inferior cerebellar artery-posterior inferior cerebellar artery variant: Case report and review of literature. Int J Surg Case Rep. 2016;22:23–7. https://doi.org/10.1016/j.ijscr.2016.03.006.
25. Ansari KA, Menon G, Nair S, Abraham M. Aneurysm of Anterior Inferior Cerebellar Artery-posterior Inferior Cerebellar Artery Variant. Asian J Neurosurg. 2018;13(1):53–5. https://doi.org/10.4103/1793-5482.180935.
26. Gopalakrishnan CV, Menon G, Nair S. Aneurysm of the anterior inferior cerebellar artery-posterior inferior cerebellar artery variant: two case reports and review of literature. Br J Neurosurg. 2009;23(5):554–6. https://doi.org/10.1080/02688690902987778.
27. Isaji T, Yasuda M, Kawaguchi R, Aoyama M, Niwa A, Nakura T, et al. Posterior inferior cerebellar artery with an extradural origin from the V3 segment: higher incidence on the nondominant vertebral artery. J Neurosurg Spine. 2018;28(2):154–9. https://doi.org/10.3171/2017.5.SPINE161286.
28. Wang X, Tang G, Li M. Bilateral extradural posterior inferior cerebellar artery origins where vertebral artery ascends between transverse foramina of C-2 and C-1, with simultaneous right double origin PICA: rare Case report and literature review. World Neurosurg. 2019;125:234–9. https://doi.org/10.1016/j.wneu.2019.01.233.
29. Demartini ZJ, de Oliveira TVH, Guimaraes R, Lohr A Jr, Koppe GL, Gatto LAM. Posterior inferior cerebellar artery origin over the C2 posterior arch. Pediatr Neurosurg. 2018;53(5):364–6. https://doi.org/10.1159/000489814.
30. Bhat DI, Somanna S, Kovoor J, Chandramoul BA. Aneurysms from extracranial, extradurally originating posterior inferior cerebellar arteries: a rare case report. Surg Neurol. 2009;72(4):406–8. https://doi.org/10.1016/j.surneu.2008.09.021.
31. Fine AD, Cardoso A, Rhoton AL Jr. Microsurgical anatomy of the extracranial-extradural origin of the posterior inferior cerebellar artery. J Neurosurg. 1999;91(4):645–52. https://doi.org/10.3171/jns.1999.91.4.0645.
32. Katsuno M, Tanikawa R, Ota N, Izumi N, Hashimoto M. Subarachnoid hemorrhage caused by ruptured posterior inferior cerebellar artery aneurysm arising from the extracranial portion of an anomalous vertebral artery. Neurol Med Chir (Tokyo). 2012;52(7):499–501. https://doi.org/10.2176/nmc.52.499.
33. Tayebi Meybodi A, Lawton MT, Benet A. Dual origin of extradural posterior inferior cerebellar artery from vertebral and occipital arteries: anatomic Case report. Oper Neurosurg (Hagerstown). 2015;11(4):564–8. https://doi.org/10.1227/NEU.0000000000000923.
34. Huynh-Le P, Matsushima T, Muratani H, Hikita T, Hirokawa E. Persistent primitive hypoglossal artery associated with proximal posterior inferior cerebellar artery aneurysm. Surg Neurol. 2004;62(6):546–51; discussion 51. https://doi.org/10.1016/j.surneu.2004.03.018.
35. Fujihara F, Takahara M, Katsuta T, Takemoto K, Higashi T, Inoue T. Posterior inferior cerebellar artery originating from the jugular branch of the ascending pharyngeal artery. NMC Case Rep J. 2019;6(1):21–4. https://doi.org/10.2176/nmccrj.cr.2018-0171.
36. Lasjaunias P, Guibert-Tranier F, Braun JP. The pharyngo-cerebellar artery or ascending pharyngeal artery origin of the posterior inferior cerebellar artery. J Neuroradiol. 1981;8(4):317–25.
37. Uchino A, Ohno H, Kondo R, Ishihara S. Ascending pharyngeal artery-posterior inferior cerebellar artery anastomosis via the jugular foramen: a case report and literature review. Surg Radiol Anat. 2021;43(6):1019–22. https://doi.org/10.1007/s00276-020-02667-5.
38. Uchino A, Suzuki C. Posterior inferior cerebellar artery supplied by the jugular branch of the ascending pharyngeal artery diagnosed by MR angiography: report of two cases. Cerebellum. 2011;10(2):204–7. https://doi.org/10.1007/s12311-011-0267-0.
39. Kim JT, Heo SH, Lee SH, Choi SM, Park MS, Kim BC, et al. An uncommon anastomosis of the posterior inferior cerebellar artery and the external carotid artery with the patent vertebrobasilar system. Br J Radiol. 2009;82(981):e171–4. https://doi.org/10.1259/bjr/73518980.
40. Ali S, Radaideh MM, Shaibani A, Russell EJ, Walker MT. Persistent trigeminal artery terminating in the posterior inferior cerebellar artery: case report. Neurosurgery. 2008;62(3):E746–8.; discussion E–8. https://doi.org/10.1227/01.neu.0000317327.17225.f8.

41. Chambers AA, Lukin R. Trigeminal artery connection to the posterior inferior cerebellar arteries. Neuroradiology. 1975;9(3):121–3. https://doi.org/10.1007/BF00332958.
42. Lee GY, Heo YJ, Jung HS, Choo HJ, Cho YJ, Jeong HW, et al. Persistent trigeminal artery variant terminating in the posterior inferior cerebellar artery: a case report. Surg Radiol Anat. 2018;40(2):237–40. https://doi.org/10.1007/s00276-017-1933-9.
43. Perot G, Clarencon F, Di Maria F, Sourour N, Biondi A, Cornu P, et al. Persistent trigeminal artery feeding a hemispheric branch of the posterior inferior cerebellar artery: a rare anatomic variant. J Neuroradiol. 2011;38(4):251–4. https://doi.org/10.1016/j.neurad.2011.02.006.
44. Raphaeli G, Bandeira A, Mine B, Brisbois D, Lubicz B. A rare variant of persistent trigeminal artery: cavernous carotid-cerebellar artery anastomosis--a case report and a systematic review. Cerebellum. 2009;8(4):445–7. https://doi.org/10.1007/s12311-009-0120-x.
45. Ahuja A, Graves VB, Crosby DL, Strother CM. Anomalous origin of the posterior inferior cerebellar artery from the internal carotid artery. AJNR Am J Neuroradiol. 1992;13(6):1625–6.
46. Saltzman GF. Patent primitive trigeminal artery studied by cerebral angiography. Acta Radiol. 1959;51(5):329–36. https://doi.org/10.3109/00016925909171103.
47. Andoh K, Tanohata K, Moriya N, Hagiwara H, Lee J, Sato M, et al. The posterior inferior cerebellar artery arising from the extracranial segment of the internal carotid artery via the hypoglossal canal without an interposed segment of the basilar artery: a persistent primitive hypoglossal artery variant. Clin Imaging. 2001;25(2):86–9. https://doi.org/10.1016/s0899-7071(01)00265-0.
48. Murayama Y, Fujimoto N, Matsumoto K. Bilateral persistent primitive hypoglossal arteries associated with a large ruptured aneurysm on one side. Surg Neurol. 1985;24(5):498–502. https://doi.org/10.1016/0090-3019(85)90263-0.
49. Ogawa T, Fujita H, Inugami A, Shishido F, Higano S, Uemura K. Anomalous origin of the posterior inferior cerebellar artery from the posterior meningeal artery. AJNR Am J Neuroradiol. 1991;12(1):186.
50. Uchino A, Suzuki C. Variant of a persistent hypoglossal artery supplying only the posterior inferior cerebellar artery diagnosed by magnetic resonance angiography: a case report. Surg Radiol Anat. 2018;40(7):807–10. https://doi.org/10.1007/s00276-018-1970-z.
51. Gabrieli J, Sourour NA, Chauvet D, Di Maria F, Chiras J, Clarencon F. Anterior spinal and bulbar artery supply to the posterior inferior cerebellar artery revealed by a ruptured aneurysm: case report. J Neurosurg. 2017;126(2):596–9. https://doi.org/10.3171/2016.1.JNS152099.
52. Mizutani K, Akiyama T, Kamamoto D, Nagashima H, Yoshida K. A ruptured aneurysm in the vasa corona at the craniocervical junction with dysgenesis of the posterior inferior cerebellar artery. BJR Case Rep. 2016;2(3):20160004. https://doi.org/10.1259/bjrcr.20160004.
53. Cho YD, Han MH, Lee JY. Double origin of the posterior inferior cerebellar artery with juxta-proximal fenestration of caudal component. Surg Radiol Anat. 2011;33(3):271–3. https://doi.org/10.1007/s00276-010-0747-9.
54. Lesley WS, Rajab MH, Case RS. Double origin of the posterior inferior cerebellar artery: association with intracranial aneurysm on catheter angiography. AJR Am J Roentgenol. 2007;189(4):893–7. https://doi.org/10.2214/AJR.07.2453.
55. Meybodi AT, Moreira LB, Zhao X, Lawton MT, Preul MC. Double origin of the posterior inferior cerebellar artery: anatomic Case report. World Neurosurg. 2019;124:110. https://doi.org/10.1016/j.wneu.2018.12.127.
56. Pasco A, Thouveny F, Papon X, Tanguy JY, Mercier P, Caron-Poitreau C, et al. Ruptured aneurysm on a double origin of the posterior inferior cerebellar artery: a pathological entity in an anatomical variation. Report of two cases and review of the literature. J Neurosurg. 2002;96(1):127–31. https://doi.org/10.3171/jns.2002.96.1.0127.
57. Plumb AA, Herwadkar A, Laitt R. Double origin of the posterior inferior cerebellar artery with findings on conventional and CT angiography. Surg Radiol Anat. 2009;31(5):393–5. https://doi.org/10.1007/s00276-008-0448-9.
58. Sharifi M, Ungier E, Ciszek B. Double posterior inferior cerebellar artery. Surg Radiol Anat. 2010;32(1):87–9. https://doi.org/10.1007/s00276-009-0512-0.
59. Silva MA, See AP, Aziz-Sultan MA, Patel NJ. Surgical treatment of a double origin posterior inferior cerebellar artery aneurysm and insights from embryology: Case report and literature review. Oper Neurosurg (Hagerstown). 2017;13(3):E8–E12. https://doi.org/10.1093/ons/opx002.
60. Tatit RT, Pacheco C, Aguiar P, Isolan GR, Campero A, Barbo M, et al. The posterior inferior cerebellar artery (PICA): an anatomical and clinical analysis. Turk Neurosurg. 2022;32(2):244–50. https://doi.org/10.5137/1019-5149.JTN.33441-20.3.
61. Mercier PH, Brassier G, Fournier HD, Picquet J, Papon X, Lasjaunias P. Vascular microanatomy of the pontomedullary junction, posterior inferior cerebellar arteries, and the lateral spinal arteries. Interv Neuroradiol. 2008;14(1):49–58. https://doi.org/10.1177/159101990801400107.
62. Lee SH, Koh JS, Ryu CW, Bang JS. Fenestration of the double origin of the posterior inferior cerebellar artery associated with a contralateral vertebral artery dissection. Cerebellum. 2009;8(3):382–4. https://doi.org/10.1007/s12311-009-0108-6.
63. Giragani S, Pavunesan SK, Balasubramaniam A, Agrawal V, Alwala S, Kumar A. Rete mirabile of posterior inferior cerebellar artery: a rare cause of subarachnoid hemorrhage. Interv Neuroradiol. 2018;24(6):662–5. https://doi.org/10.1177/1591019918782147.
64. Margolis MT, Newton TH. An angiographic sign of cerebellar tonsillar herniation. Neuroradiology. 1971;2(1):3–8. https://doi.org/10.1007/BF00345864.
65. Rodriguez-Hernandez A, Rhoton AL Jr, Lawton MT. Segmental anatomy of cerebellar arteries: a proposed nomenclature. Laboratory investigation J Neurosurg. 2011;115(2):387–97. https://doi.org/10.3171/2011.3.JNS101413.
66. Macchi V, Porzionato A, Parenti A, De Caro R. The course of the posterior inferior cerebellar artery may be related to its level of origin. Surg Radiol Anat. 2004;26(1):60–5. https://doi.org/10.1007/s00276-003-0190-2.
67. Ucerler H, Saylam C, Cagli S, Orhan M, Zileli M. The posterior inferior cerebellar artery and its branches in relation to the cerebellomedullary fissure. Clin Anat. 2008;21(2):119–26. https://doi.org/10.1002/ca.20581.
68. Giotta Lucifero A, Baldoncini M, Bruno N, Tartaglia N, Ambrosi A, Marseglia GL, et al. Microsurgical Neurovascular Anatomy of the Brain: The Posterior Circulation (Part II). Acta Biomed. 2021;92(S4):e2021413. https://doi.org/10.23750/abm.v92iS4.12119.
69. Tanohata K, Maehara T, Noda M, Katoh H, Sugiyama S, Okazaki A. Anomalous origin of the posterior meningeal artery from the lateral medullary segment of the posterior inferior cerebellar artery. Neuroradiology. 1987;29(1):89–92. https://doi.org/10.1007/BF00341048.
70. Cullen SP, Ozanne A, Alvarez H, Lasjaunias P. The bihemispheric posterior inferior cerebellar artery. Neuroradiology. 2005;47(11):809–12. https://doi.org/10.1007/s00234-005-1427-z.
71. Ogasawara Y, Kashimura H, Aso K, Saura H. Subarachnoid hemorrhage due to ruptured intracranial aneurysm arising from a vertebral artery-Bihemispheric posterior inferior cerebellar artery bifurcation. J Neurosci Rural Pract. 2017;8(4):654–6. https://doi.org/10.4103/jnrp.jnrp_285_17.
72. Carlson AP, Alaraj A, Dashti R, Aletich VA. The bihemispheric posterior interior cerebellar artery: anatomic variations and clinical

relevance in 11 cases. J Neurointerv Surg. 2013;5(6):601–4. https://doi.org/10.1136/neurintsurg-2012-010527.
73. Satoh Y, Matsuzaki K, Haboshi T, Shimada K, Niki H, Hanaoka M, et al. Ruptured aneurysm originating from a posterior inferior cerebellar artery communicating artery: a case report and review of the literature. J Med Investig. 2020;67(1.2):197–9. https://doi.org/10.2152/jmi.67.197.
74. Hlavin ML, Takaoka Y, Smith AS. A "PICA communicating artery" aneurysm: case report. Neurosurgery. 1991;29(6):926–9. https://doi.org/10.1097/00006123-199112000-00024.
75. Sugiyama S, Fujimura M, Inoue T, Shimizu H, Watanabe M, Tominaga T. Ruptured aneurysm of a posterior inferior cerebellar artery communicating artery. Case report and histological findings. Neurol Med Chir (Tokyo). 2012;52(2):81–3. https://doi.org/10.2176/nmc.52.81.
76. Ota T, Dofuku S, Sato M. Persistence of primitive lateral Basilovertebral anastomosis with a ruptured posterior inferior cerebellar artery aneurysm: a Case report. NMC Case Rep J. 2022;9:69–72. https://doi.org/10.2176/jns-nmc.2021-0386.
77. Yamakawa H, Yoshimura S, Iwama T. Anterior spinal artery as a collateral channel in patients with acute bilateral vertebral artery occlusions. Two case reports. Neurol Med Chir (Tokyo). 2009;49(8):354–8. https://doi.org/10.2176/nmc.49.354.
78. Aoyama T, Obara N. Vertebral artery terminating posterior inferior cerebellar artery (PICA-VA) as a potential risk factor in cervical spine surgery. World Neurosurg. 2020;143:e535–e40. https://doi.org/10.1016/j.wneu.2020.08.003.
79. Liu IW, Ho BL, Chen CF, Han K, Lin CJ, Sheng WY, et al. Vertebral artery terminating in posterior inferior cerebellar artery: a normal variation with clinical significance. PLoS One. 2017;12(4):e0175264. https://doi.org/10.1371/journal.pone.0175264.
80. Theodosopoulos PV, Lawton MT. Fenestration of the posteroinferior cerebellar artery: case report. Neurosurgery. 2000;47(2):463–5. https://doi.org/10.1097/00006123-200008000-00041.
81. Lesley WS. Fenestration of the posterior inferior cerebellar artery. Cerebellum. 2008;7(3):240–1. https://doi.org/10.1007/s12311-008-0002-7.
82. Miao HL, Zhang DY, Wang T, Jiao XT, Jiao LQ. Clinical importance of the posterior inferior cerebellar artery: a review of the literature. Int J Med Sci. 2020;17(18):3005–19. https://doi.org/10.7150/ijms.49137.
83. Lewis SB, Chang DJ, Peace DA, Lafrentz PJ, Day AL. Distal posterior inferior cerebellar artery aneurysms: clinical features and management. J Neurosurg. 2002;97(4):756–66. https://doi.org/10.3171/jns.2002.97.4.0756.
84. Drake CG, Peerless SJ, Hernesniemi J. Surgery of Vertebrobasilar aneurysms: London, Ontario experience on 1767 patients. Wien: Springer; 1996.
85. Kubota H, Suehiro E, Yoneda H, Nomura S, Kajiwara K, Fujii M, et al. Lateral spinal artery aneurysm associated with a posterior inferior cerebellar artery main trunk occlusion. Case illustration J Neurosurg Spine. 2006;4(4):347. https://doi.org/10.3171/spi.2006.4.4.347.

Embryology and Variations of the Anterior Inferior Cerebellar Artery

L. Valci, Sara Bonasia, and Thomas Robert

The anatomical complexity of the vascularization of the posterior cranial fossa finds its greatest expression in the variations in number, origin, morphology and course of the anterior inferior cerebellar artery (AICA).

This concerns the main artery of the ponto-cerebellar angle, sometimes referred to as the 'cerebellum-labyrinthine artery' to emphasize its exclusive role in supplying the inner ear [1, 2], or as it has been called, improperly according to Kielbasinski, the 'middle cerebellar artery' [3].

It constitutes the broadest cerebellar branch of the basilar artery (BA), provides the arterial supply to the anterior (petrous) portion of the inferior surface of the cerebellar hemispheres and is firmly anchored by its branches to the petrous bone.

It is often considered a terminal artery, or one with poor collateralization, and therefore its acute occlusion can prove fatal.

This chapter discusses the peculiarities of this vessel in embryological and anatomical terms, as well as the clinical implications related to its impairment.

History

'Ramus centralis arteriae radicularis nervi faciei': Wallenberg already defined the anterior inferior cerebellar artery in these terms in 1901 [4]. However, the tortuous course and numerous variations have meant that a comprehensive study of this artery only became possible after the advent and routine use of digital subtraction angiography (DSA) techniques. Until then, descriptions were based on studies of cadavers and thus it often happened that the definition of syndromes resulting from occlusion of the AICA, or its branches, preceded the embryological, or anatomical treatment of the vessel: an example of this is the work of Adams RD on occlusion of the AICA (1943), which preceded the embryological and anatomical treatments of Padget DH (1948) and Atkinson WJ (1949) respectively [5–7]. In the years to follow, important contributions to the understanding of anatomical details will come from surgery thanks to many authors, including Lazorthes G et al. (1950) and Rhoton AL et al. (1968), just to name a few in purely chronological order [8, 9].

Embryological Development

From the coalescence of the vessels of a primitive vascular plexus, called the neuronal longitudinal system, originates the basilar artery at week 5 of the embryonic stage.

Many arterial outgrowths with a horizontal course emerge from it and each of them has the potential to evolve into AICA, gradually taking on the characteristics of a dominant transverse artery.

The specific needs for arterial supply during cerebral development of the embryo may result in different modes of development of the AICA, hence the enormous variability in number, morphology of this artery and its anastomotic network [10].

From Padget's illustrations a first outgrowth of the AICA from the basilar artery is already visible in this stage, and in the 16–18 mm embryo (48 days gestation, Padget stage V) the AICA shows a clear course. In stage V, the primitive stem of the AICA arises in front of the vestibulocochlear nerve and terminates into the choroidal plexus of the fourth ventricle. Once the original pedicle of the AICA is created, the pontine branches are formed [6].

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At the same time, a kind of vascular circle is also created around the VI cranial nerve: the manner and timing of the regression of certain parts of this circle will cause the internal auditory artery to arise separately from the basilar artery, or to originate as a branch of the AICA [11].

A detailed description of the embryological steps involving the AICA is furnished in Table 1.

Number and Origin of the Artery

Like all arteries of the posterior cranial fossa, the AICA can present, from its origin, a wide spectrum of variants, ranging from absence and hypoplasia to duplication or triplication, and knowledge of the precise anatomical conformation of the artery's origin has important implications in safeguarding neurological function during both tumor and purely vascular surgery.

The left and right AICA do not necessarily originate at the same level. The AICA arises at right angles from the lateral wall of the basilar artery and, if we consider its origin as a function of the latter, we would say that it departs from the caudal half of the basilar artery, or, according to McDermott AL et al. [12], from its lower third (52%), middle third (46%) and upper third (2%).

In the Hou K angiographic classification, the origin of AICA is divided into four types: AICA with origin from the upper half of the basilar artery (high origin), from the lower half (normal), from the basilar artery-vertebral artery (VA) junction (junctional) and from the vertebral artery (low origin) [10].

However, it is also possible to refer the origin of the vessel to proximal structures, and so we can say that the AICA generally originates at the junction of the medulla oblongata and the pons.

The AICA may originate from the ipsilateral vertebral artery, particularly when the ipsilateral posterior inferior cerebellar artery (PICA) is absent, or arises directly from the PICA, even from the contralateral PICA, or as a common trunk with the PICA, which in turn originates from the basilar artery, or from the vertebral artery: in this specific case, often one of the two branches is hypoplastic, while the other compensates with a larger diameter and wider vascularization zone [13].

As an example of the variability of origin, Rajasekhar SN et al. describe a duplication of the AICA with one of the origins from the PICA and the other from the distal hypoplastic tract of the vertebral artery [14].

Generally, the diameter of the AICA varies between 0.5 mm and 1 mm, but it is largely dependent on the caliber of other vessels, e.g. it is larger when it has to supplant a small PICA [15].

Fig. 1 Clinical case of AICA duplication. The figure shows a DSA in antero-posterior view after left vertebral artery (VA) injection. The AICA appears duplicated (red arrows). The PICA origin on the right side is visible on the VA. *BA* basilar artery

In most cases it is single, with numerical variants duplication and triplication accounting for 20% and 1% of the occurrences respectively.

In the case of duplication, one of the vessels has a larger diameter, while the second is referred to as 'accessory', either larger or smaller than the first [16]. A case of AICA duplication is shown in Fig. 1.

On the other hand, it is absent in sporadic cases and situations have been described in which the AICA is absent; however, the territory of its supply is supplemented by an aberrant artery of the cavernous, or pre-cavernous, tract of the ipsilateral internal carotid artery [17–19].

According to Brzegowy K et al., AICA is the most frequent variant of the primitive trigeminal artery (TA), which in turn represents the main anastomosis between the carotid and basilar arteries [20].

In relation to the state of development, Hou K et al. distinguish four grades: absence, hypoplasia, normal AICA and hyperplasia [10].

Course of the Artery

When describing the course of the AICA, it is easy to take the pons as a reference point and then consider the position of the artery in relation to it; the AICA can therefore be divided into the anterior pontine, lateral pontine and

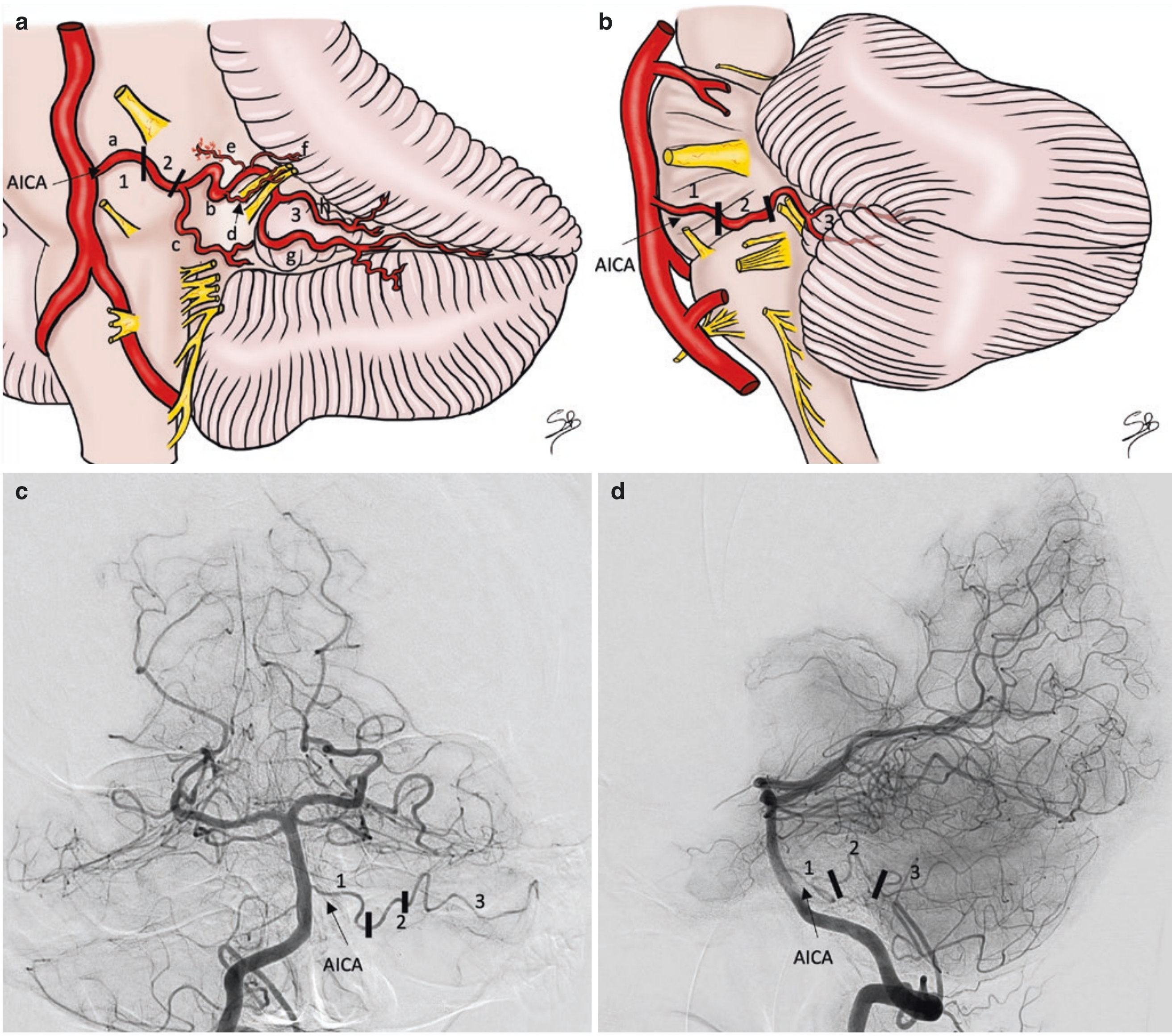


Fig. 2 Illustration and DSA of segments and branches of the anterior inferior cerebellar artery (AICA). Figures (**a** and **b**) show a graphic illustration of the segments and branches of the AICA. Figures (**c** and **d**) show an antero-posterior and lateral DSA view after right vertebral artery injection. The AICA can be divided into three segments: anterior pontine (*1*), lateral pontine (*2*), and cerebello-pontine segment (*3*). The main AICA trunk (*a*) usually divides into a rostro-lateral (*b*) and a medio-caudal (*c*) ramus. Usually from the former some branches arise: the internal auditory or labyrinthine artery (*d*), the recurrent perforating pontine branch (*e*), and the subarcuate artery (*f*). Above the flocculus, the rostro-lateral artery divides into an ascending (*h*) and a descending branch (*g*)

cerebello-pontine sections [21]. In Fig. 2, this segmental division is graphically and radiologically represented.

Another possibility is to define the course of the AICA in relation to the internal acoustic meatus and, in this case, there will be the segments: pre-meatal, meatal and post-meatal.

Finally, its course can be divided into: anterior pontine segment, lateral pontine segment, flocculo-peduncular segment and cortical segment [22].

In the following we will use the first subdivision, which is of more use in a didactic sense.

Anterior Pontine Segment

The AICA originates from the basilar artery in the manner described above and leads posteriorly, inferiorly and laterally to the belly of the pons.

Lateral Pontine Segment

The AICA heads to the radicles of emergence of the abducens nerve (VI), with which it comes into contact, usually either ventrally or dorsally, and in rare cases crosses it [23]. A few millimeters lateral to the VI nerve, the artery (main trunk) bifurcates into two branches, namely the rostro-lateral artery and the caudo-medial artery.

Cerebello-Pontine Segment (Consisting of the Rostro-Lateral and Caudo-Medial Arteries)

The rostro-lateral artery enters into a variable relationship with nerves VII and VIII anterior to the flocculus: in most cases, its course is ventral to the neural pedicle, but it may also cross its radicles, or it may run dorsal to these nerves; generally, the artery runs in a plane between the facial/intermediate nerve and the cochlear and vestibular nerves [24].

This is the segment where the artery constantly forms a loop, which can be in correspondence with the acoustic pore, or enter it, as well as being distant from it and not contracting any relationship with the petrous bone [21].

Sometimes the artery describes a second loop, the apex of which may reach the postero-inferior portion of the trigeminal sensory origin (V). Both loops, however, remain located anterior to the flocculus.

The artery then continues posteriorly on the brachium pontis entering the supra-floccular portion of the horizontal fissure. At the postero-superior angle of the flocculus, the rostro-lateral artery gives rise to an ascending branch to the horizontal fissure and a descending branch, directed to the postero-lateral fissure.

The ascending branch in turn leads to the petrous surface of the cerebellum but can also be broad and run for a longer distance to the occipital side of the cerebellum.

The descending branch, on the other hand, runs between the flocculus anteriorly and the biventral lobule posteriorly: it provides small branches for the lower semilunar and biventral lobules. From this segment may originate a small artery, or an arterial plexus, which runs in the depth of the postero-lateral fissure and is called the lateral recess artery [16].

The caudo-medial artery passes through the ponto-medial groove and describes a caudal loop on the lateral surface of the pons and medulla. Sometimes, particularly when it is of substantial caliber, it runs lateral to the supra-olivary dimple and forms a laterally directed loop, which reaches the radicles of the IX, X and XI cranial nerves, the foramen of Luschka and the emergence of the choroid plexus, which is supplied by arterioles from its side.

The caudo-medial artery then terminates on the lateral side, or at the margin of the biventral lobule, or reaches the posterior surface of the cerebellum through the cerebello-medial fissure [16].

It must be said that a bifurcation of the main trunk, which is proximal to the emergence of the VII and VIII cranial nerves, can cause both branches to come into contact with the two nerves.

Branches

The main branches of the AICA, particularly when the artery starts as an individual vessel, originate at the emergence of the VI cranial nerve, and result from the bifurcation of the main trunk into rostro-lateral and caudo-medial arteries.

The rostro-lateral artery forms a loop on the lateral wall of the pons and then reaches cranial nerves VII and VIII anterior to the flocculus; at this level it has another loop, which may be single or double: in the first case we speak of a meatal loop because of its correspondence with the acoustic pore; anterior to the VII cranial nerve this branch gives rise to the internal auditory artery, also called labyrinthine artery, and then descends again and provides recurrent perforating pontine branches, as well as the subarcuate artery; this in turn passes through the subarcuate fossa and reaches the subarcuate canal, in some cases forming a second loop itself, so as to create an M-shaped configuration of the arterial tract, which is related to cranial nerves VII and VIII; above the flocculus the rostro-lateral artery divides into an ascending and a descending branch.

Mazzoni et al. describe a cerebello-subarcuate artery, usually proximal to the meatal loop, as a small branch of the AICA, from which originates one branch directed to the subarcuate fossa and another to the cerebellum [25].

The internal auditory artery terminates with the vestibular, cochlear and vestibulocochlear arteries, which can also be single, double or triple.

The distal branches of the rostro-lateral artery are those that most frequently have relationships with cranial nerves VII and VIII, and, precisely on the basis of their relationship with these and the internal auditory meatus. Richard G et al. distinguish them into three segments: pre-meatal, meatal and post-meatal. However, the terminal branches of the AICA tract in relation to cranial nerves VII and VIII can be highly variable, by site, in origin and number [26].

The caudo-medial artery, which varies in caliber, also describes a loop at the level of the lateral wall of the pons and stops at this level when short, or, if long, reaches the lateral portion of the flocculus and the biventral lobule [16]. Figure 2 shows the main branches of the AICA.

Possible Anastomoses

In the context of the complexity and variability of the arterial network of the posterior cranial fossa, it is often difficult to conceptually distinguish anastomoses from anomalies of AICA origin, and therefore, for the sake of simplicity, we will consider anastomoses proper, those that arise in the more peripheral vascularization regions: in this case, connections will be relatively frequent between the AICA and the superior cerebellar artery, e.g. at the level of the flocculus, or the horizontal cerebellar fissure, and again with the postero-inferior cerebellar artery at the level of the tonsil, the biventral lobule and inferior semilunar artery, in some cases also contralaterally [3]. The subarcuate artery can anastomose with the middle meningeal artery [27].

Parenchymal and Dural Territories

The vascularization territories reached by the branches of the AICA are relatively constant and the clinical differences resulting from their impairment, especially in the case of infarct lesions, are almost always attributable to the presence of a wide spectrum of anastomotic variants with the other cerebellar arteries.

The portion of cerebellar parenchyma pertaining to the AICA is represented by the petrous surface, although the artery is often of greater caliber, particularly when it supplants the vascularization of the PICA, supplying a large part of the latter's territory. It almost constantly supplies the flocculus, at the level of which it terminates, although it can reach, with its terminal branches, the anterior, superior semilunar, inferior semilunar, gracile and biventral lobules [28].

The pontine distribution was described in detail by Duvernoy in 1978 [29]. A group of descending branches enters the pons at the level of the bulbo-pontine sulcus and supplies the lateral area of the medulla and, in particular, the fibers of the IX and X cranial nerves and the upper part of the nucleus ambiguus. Another group crosses the lower boundary of the pontine tegmentum to reach the lateral part of the pons, and, more specifically, the superior olivary nucleus, the facial nucleus, the superior, lateral and medial vestibular nuclei, the trapezoid body and the cochlear nucleus, and sometimes the lateral part of the abducens nerve nucleus; the supplied fasciculi are instead: the lateral leminiscus, the spino-thalamic tract, the spino-trigeminal tract; the same branches supply the middle cerebellar peduncle. The ascending branches can reach the trigeminal roots.

The AICA constantly supplies the middle cerebellar peduncle, often the lower third of the lateral portion of the pons, frequently its middle third, and in some cases the upper and lateral portions of the bulb [30]. Through the internal auditory artery, it supplies the cochlea and vestibular labyrinth. The vascular territory supplied by the AICA is illustrated in Fig. 3.

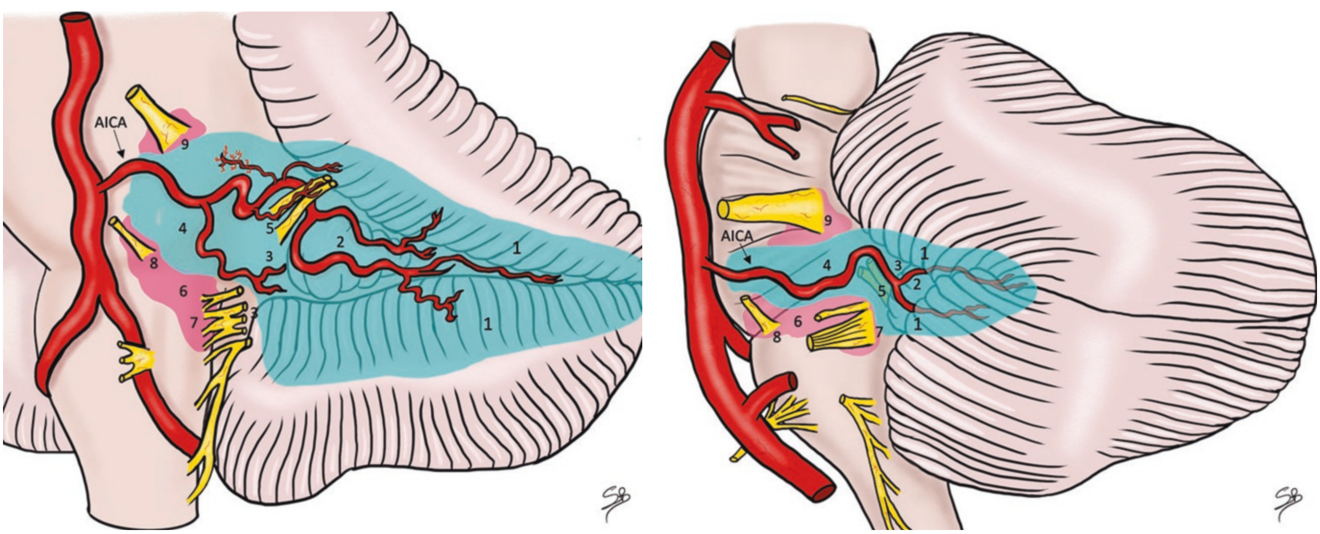


Fig. 3 Illustration of the vascular territory supplied by the AICA. The area constantly supplied by the AICA is represented by the blue area in the figure. The AICA constantly supplies the petrous surface of the cerebellum (*1*), the flocculus (*2*) and the middle cerebellar peduncle (*3*). It also supplies the lower third of the anterolateral portion of the pons (*4*). Through the internal auditory artery, it supplies the VII and VIII cranial nerves, the cochlea and the vestibular labyrinth (5). The rosa area indicates the area that the AICA can occasionally supply. In some cases, it supplies the upper and lateral portions of the bulb (6). In this case it can contribute to the supply of the X and XI nerve (*7*). The AICA can also contribute to the supply of the VI nerve, together with the basilar artery, and the ponto-medullary artery (*8*). Cranially, its fibers can reach the V nerve origin (*9*)

Variants

In order to understand the main anatomical variants of the AICA, it is necessary to relook at embryology, and in particular at the fact that, at the time the basilar artery originates from the plexiform structure of the longitudinal neural system (week 5, Padget Stage 4), the vascularization of the posterior fossa is still dependent on the anterior circulation through the vertebrobasilar anastomoses; this is where the discussion of persistent trigeminal arteries comes in.

The persistent trigeminal artery is the most frequent and cephalic anomaly in anastomoses between the internal carotid and basilar circuits; its name derives from the fact that it runs ventro-medially to the trigeminal ganglion [31]. The trigeminal artery originates as the second of two branches of the first aortic arch, the first branch being the primitive internal carotid artery [32]. The variant of the persistent trigeminal artery is an anastomosis between the internal carotid artery and a cerebellar artery with or without interposition of the basilar artery: it usually originates from the cavernous, or pre-cavernous, tract of the internal carotid artery, runs along the nerve and enters the posterior fossa through Meckel's cave, or through a separate dural foramen to directly supply the cerebellum.

In physiological development, both the primitive longitudinal neural system and the trigeminal arteries regress: the persistence of a trigeminal artery and a basilar artery normally originating from the coalescence of the primitive neural arteries will result in a carotid-basilar anastomosis; the persistence of a trigeminal artery, without a complete fusion of the primitive neuronal arteries in the formation of the basilar artery, will give rise to arteries which directly supply the cerebellum and, depending on their position, will vascularize the territories of the superior cerebellar artery, or the AICA [17, 18]; cerebellar arteries which originate directly from the internal carotid artery are found in 0.18% of cases on angiography [26]. It was Quain in 1844 and, much later, Teal et al. in 1972, who first described the possible origin of the cerebellar arteries from the internal carotid artery [33, 34].

According to Padget, the primitive trigeminal artery is already recognizable as an anastomosis between carotid and neural longitudinal arteries in the 4 mm embryo. With the fusion of the longitudinal neural arteries (LNAs), the primitive trigeminal artery regresses; its regression coincides temporally with the formation of the basilar artery (between 5 and 5.5 weeks) [6]. The lack of fusion of the longitudinal neural arteries means that the trigeminal artery persists to support the territory, which would have been of the fused artery, and its appearance will be that of a cerebellar artery originating from the internal carotid artery [35].

During the development of the structures of the posterior fossa, transverse arteries, in varying numbers, originate from the longitudinal system and gradually flow into the cerebellum and trunk. Some of the transverse arteries gradually become dominant in caliber, forming the superior cerebellar arteries (SCA), the anterior inferior cerebellar arteries (AICA) and the posterior inferior cerebellar arteries (PICA): it is at this embryological stage that, mainly depending on the blood supply requirements of the different developing structures, variants of the AICA can be created, particularly in number, but also in type of origin, e.g. the formation of the AICA-PICA complex, in which the PICA is absent and the AICA is particularly developed and supplies the territories of both arteries [27]. A clinical case of the AICA-PICA complex is shown in Fig. 4. Agenesis of the AICA also accounts for 29% of the posterior circulation variants [36].

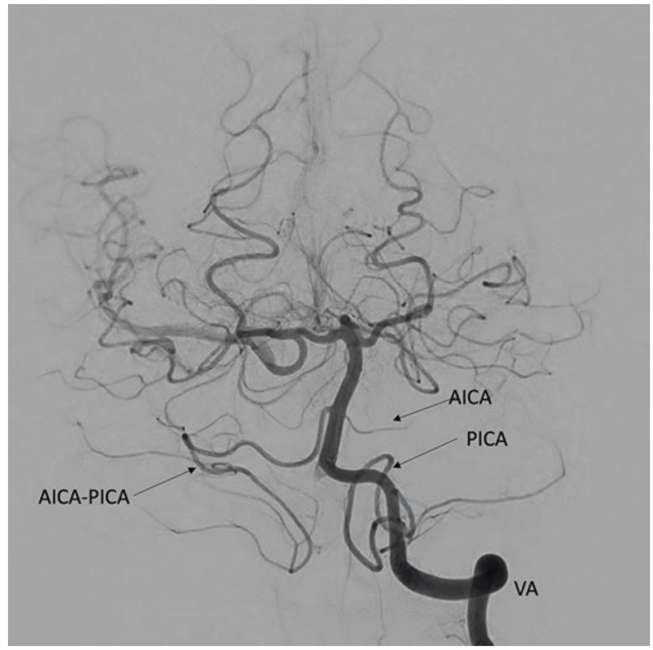


Fig. 4 Clinical case of AICA-PICA variant. The figure shows a DSA after left vertebral artery (VA) injection. It shows on the left side a separate origin of the AICA and the PICA. On the right side the two arteries arise as a single trunk from the typical origin point of the AICA, and supply both vascular territories, forming the so-called AICA-PICA complex

Clinical Implications

The anatomical variability of the AICA, particularly with regard to possible anastomotic combinations with other arteries of the vertebrobasilar and, in some cases, the carotid circulation, results in clinical variability.

The predominant pathology is ischaemic on an atherosclerotic and, more rarely, cardiogenic embolization, basis, and may result from variable involvement of the inferolateral portion of the pons, the middle cerebellar pedicle, and the anterior portion of the cerebellar hemisphere.

The AICA occlusion usually produces a ponto-cerebellar infarct, the constants of which are the involvement of the lateral portion (middle and inferior region) of the pons, the anterolateral part of the cerebellum, the middle cerebellar pedicle, the flocculus, and the anterior part of the cerebellar lobules [30].

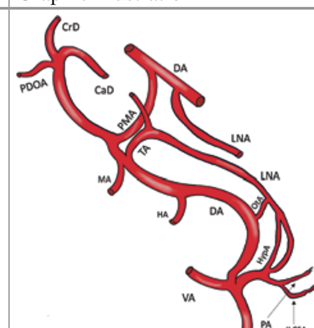
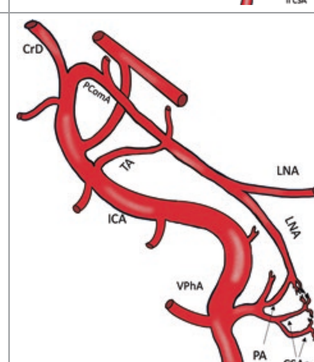
The multiplicity of symptoms resulting from a lack of blood supply to specific territories constitutes a syndrome, in the context of which, however, individual symptoms may combine in various ways, be absent, mild or extremely severe.

The AICA occlusion syndrome has already been comprehensively described by Adams in 1943, and thus includes symptoms and signs: the patient in fact may experience reduction/loss of hearing, vertigo, diplopia, nausea and shows signs of nystagmus, paresis of the gaze, dysphagia, dysarthria, dysmetria, trunk and gait ataxia, ipsilateral facial paresis, ipsilateral facial sensitivity deficit, reduction of contralateral brachio-crural sensitivity from involvement of the spino-thalamic tract or ipsilateral Horner's syndrome due to involvement of the pupillary-dilator fibers of the lateral portion of the pons. Involvement of cranial nerves V, VII and VIII, IX may result from nuclear, or intra-axial fiber, damage [5].

Hearing impairment is present in 30–100% of cases, and the preservation of this function depends on the presence of collateral circuits, thanks to which the middle and occipital meningeal arteries connect with the labyrinthine artery via the subarcuate artery [27]. Acute impairment of auditory and vestibular functions (labyrinthine, or inner ear, infarction) results from a lack of blood supply to the cochlea and inner labyrinth, which are supplied by the internal auditory artery, and may represent a very early event of AICA infarction with prevention implications in the extension of the infarction region [37]. Hearing reduction may, however, also depend on a lesion of the cochlear nuclei at the ponto-bulbar junction, at the level of the pontine tegmentum or of the intra-axial acoustic fibers in the lateral portion of the pons.

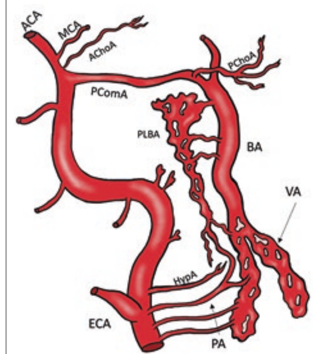
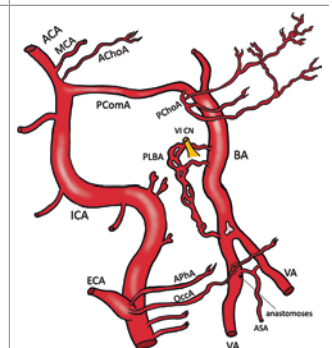
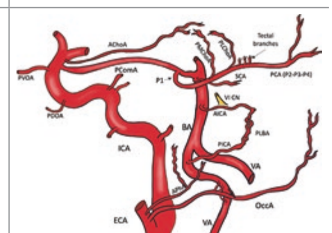
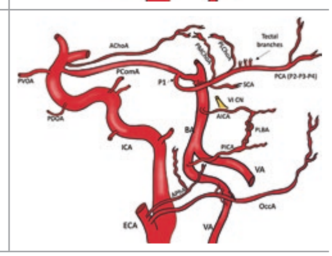
A small part of AICA pathology is concerned with neurovascular conflicts, a topic still being debated due to the fact that the presence of vascular loops, even pronounced in contact with cranial nerves, are in most cases asymptomatic; McDermott AL et al. report a statistically significant association between the presence of a loop, extended in the internal acoustic meatus for more than 50% of its length, and unilateral hearing impairment, while there is no correlation with tinnitus Table 1 [12].

Table 1 Major embryological changes in the formation of the AICA

Stage	Embryo size (mm)	Major evolutions	Graphic illustration
I	4–5	• Paired longitudinal neural arteries (LNAs) visible • Carotido-vertebral anastomoses visible: Trigeminal (TA), otic (OtA), hypoglossal (HypA), proatlantal (PA) and second cervical segmental artery (II CSA) • Primitive internal carotid artery (ICA) bifurcation visible into caudal and cranial divisions (CaD, CrD) • Posterior communicating artery (PComA) and basilar artery (BA) not formed	
II	5–6	• Formation of the PComA • Regression of otic and hypoglossal arteries • Midline fusion of LNAs to form the basilar artery (BA) • Initial transverse anastomoses between the intersegmental arteries	

(continued)

Table 1 (continued)

Stage	Embryo size (mm)	Major evolutions	Graphic illustration
III	7–12	• Basilar artery (BA) completely formed • PComA completely formed • Regression of trigeminal and proatlantal arteries • Formation of the vertebral artery (VA) by transverse anastomoses between the intersegmental arteries • Lateral basilo-vertebral anastomosis (PLBA) visible	
IV	13–15	• Remodeling of the BA and development of the PCA • Anastomotic ring around the VI cranial nerve visible • Migration of occipital artery (OccA) origin on the external carotid artery (ECA) • Persistence of the anastomoses between OccA and VA • Formation of the anterior spinal artery (ASA)	
V	16–18	• Complete formation of the VA • Complete formation of the OccA (remnant of the proatlantal artery) • Complete formation of the ascending pharyngeal artery (APhA, remnant of the hypoglossal artery) • Potential stem of the AICA visible	
VI	20–24	• Complete development of the circle of Willis • Interaction between the primitive stem of the AICA and PICA with the PLBA to determine its definitive configuration	

At stage I of Padget (4–5 mm), the paired longitudinal neural arteries (LNA) appear fed cranially by the trigeminal artery (TA) and caudally by the proatlantal artery (PA). Other carotid-vertebral anastomosis formed slightly later with the formation of the otic and hypoglossal arteries [6]. The proatlantal (PA) and other cervical segmental arteries (II CSA) are visible.

At stage II of Padget (5–6 mm), the two LNAs initiate their medial fusion to form the future basilar artery. The posterior communicating artery is completely formed and, consequently, the carotid-vertebral anastomoses start their regression, starting from the otic and hypoglossal arteries [6]. The cervical segmental arteries develop transverse anastomotic channels to each other.

At the stage III of Padget (7–12 mm), the vertebral artery (VA) is formed by transverse anastomoses between the segmental cervical arteries. The VAs and the cranio-cervical junction have still a plexiform aspect, and the vertebrobasilar junction is not completely formed. In this phase is visible a lateral plexiform channel, parallel to the LNAs. This channel represents an accessory basilo-vertebral anastomosis and passes posterior to the cranial nerves VI and XII but anterior to cranial nerves VII and XI. It was called by Padget primitive lateral basilo-vertebral anastomosis (PLBA) and can play a role in the definitive formation of the cerebellar arteries [6]. This channel could origin from the ascending branch of the posterior stem of the proatlantal artery and is supposed to play a role in the development of the arteries of the cranial-cervical junction as well as for the definitive configuration of the AICA and the PICA [6, 38].

The stage IV of Padget (embryos of 12–14 mm) is characterized by the definitive remodeling of the vertebrobasilar system in its definitive configuration. This determines the definitive change from the exclusive carotid supply of all cerebral arteries to the vertebral predominance in the hindbrain region [6]. In this stage the evolution of some branches arising from the BA can determine the future relationship between the AICA and the VI cranial nerve, as well as the definitive origin of the internal auditory artery. In this phase the VI cranial nerve is surrounded by a vascular anastomotic ring, composed medially by the basilar artery, cranially by a branch of the basilar artery (the precursor of the internal auditory artery), and caudally by a second BA branch (the precursor of the AICA). The lateral part of the ring is formed by anastomoses between the two BA branches, that can be interpreted as a part of the PLBA visible in stage III. In case of regression of the lateral anastomotic channel, the AICA and the internal auditory artery will origin separately from the BA. In case of regression of the dorsal artery, the AICA will course under the VI cranial nerve and will give origin to the internal auditory artery. In case of regression of the caudal artery, the AICA will course above the VI cranial nerve and will give origin to the internal auditory artery. The evolution of the proatlantal artery (PA) brings to the formation of the occipital artery (OccA) and to the initial development of the anterior spinal artery (ASA).

At the same time the posterior choroidal artery (PChoA) progresses with its evolution.

During the stage V of Padget (embryos of 16–18 mm) the branches arising from the BA continue their evolution. The potential stem of the AICA is visible and still interact with the PLBA and with the PICA. At this stage the evolution of the initial stem of the PChoA bring to the formation of the posterior cerebral artery, with its branches (PMChoA, PLChoA) and segments (P1, P2, P3, P4). The ascending pharyngeal artery (APhA) and the occipital artery (OccA) complete their development.

The stage VI of Padget (embryos of 20–24 mm) is characterized by the complete formation of the circle of Willis, which is completed after the development of the anterior communicating artery, when the embryo is approximatively 24 mm [6]. At this stage D. Padget noted a difficulty to recognize stems of the AICA and PICA among many other arterial branches supplying the posterior part of the hindbrain. In this contest, the PLBA previously described persists considering that the cerebellar lobes have not yet formed. The relationship between this plexus and the original stems of these arteries can be the origin of many variations, such as the duplicated origin of the AICA, the AICA-PICA complex and the AICA origin from the ICA.

References

1. Delion M, Dinomais M, Mercier P. Arteries and veins of the cerebellum. Cerebellum. 2017;16(5–6):880–912. https://doi.org/10.1007/s12311-016-0828-3.
2. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical Neuroangiography. Berlin: Springer edition; 2001.
3. Kielbasinski G. Course and area supplied by the anterior inferior cerebellar arteries in man. Folia Morphol (Warsz). 1977;36(3):181–90.
4. Perneczky A, Perneczky G, Tschabitscher M, Samec P. The relationship between the caudolateral pontine syndrome and the anterior inferior cerebellar artery. Acta Neurochir. 1981;58(3–4):245–57. https://doi.org/10.1007/BF01407131.
5. Adams RD. Occlusion of the anterior inferior cerebellar artery. Arch Neurol Psychiatr (Chicago). 1943;49:765–70.
6. Padget DH. The development of cranial arteries in the human embryo, Contribution to embryology, vol. 212. Washingtion: Carnegie Institution; 1948. p. 205–62.
7. Atkinson WJ. The anterior inferior cerebellar artery; its variations, pontine distribution, and significance in the surgery of cerebello-pontine angle tumours. J Neurol Neurosurg Psychiatry. 1949;12(2):137–51. https://doi.org/10.1136/jnnp.12.2.137.
8. Lazorthes G, Poulhes J, Espagno J. Les artères du cervelet. Cr Ass Anat. 1950;37:279–86.
9. Rhoton AL Jr, Kobayashi S, Hollinshead WH. Nervus intermedius. J Neurosurg. 1968;29(6):609–18. https://doi.org/10.3171/jns.1968.29.6.0609.
10. Hou K, Li G, Luan T, Xu K, Xu B, Yu J. Anatomical study of anterior inferior cerebellar artery and its reciprocal relationship with posterior inferior cerebellar artery based on angiographic data. World Neurosurg. 2020;133:e459–e72. https://doi.org/10.1016/j.wneu.2019.09.047.
11. Wende S, Nakayama N, Schwerdtfeger P. The internal auditory artery: (embryology, anatomy, angiography, pathology). J Neurol. 1975;210(1):21–31. https://doi.org/10.1007/BF00314321.
12. McDermott AL, Dutt SN, Irving RM, Pahor AL, Chavda SV. Anterior inferior cerebellar artery syndrome: fact or fiction. Clin Otolaryngol Allied Sci. 2003;28(2):75–80. https://doi.org/10.1046/j.1365-2273.2003.00662.x.
13. Gerald B, Wolpert SM, Haimovici H. Angiographic anatomy of the anterior inferior cerebellar artery. Am J Roentgenol Radium Therapy, Nucl Med. 1973;118(3):617–21. https://doi.org/10.2214/ajr.118.3.617.
14. Rajasekhar S, Aravindhan K, Tamgire DW. Duplicated and Hypoplastic V4 segment of vertebral artery along with duplication of anterior inferior cerebellar artery: a case report. J Clin Diagn Res. 2017;11(8):AD03–AD5. https://doi.org/10.7860/JCDR/2017/29157.10366.
15. Pekcevik Y, Pekcevik R. Variations of the cerebellar arteries at CT angiography. Surg Radiol Anat. 2014;36(5):455–61. https://doi.org/10.1007/s00276-013-1208-z.
16. Naidich TP, Kricheff II, George AE, Lin JP. The normal anterior inferior cerebellar artery. Anatomic-radiographic correlation with emphasis on the lateral projection. Radiology. 1976;119(2):355–73. https://doi.org/10.1148/119.2.355.
17. Ito J, Takeda N, Suzuki Y, Tekeuchi S, Osugi S, Yoshida Y. Anomalous origin of the anterior inferior cerebellar arteries from the internal carotid artery. Neuroradiology. 1980;19(2):105–9. https://doi.org/10.1007/BF00342605.

18. Scotti G. Anterior inferior cerebellar artery originating from the cavernous portion of the internal carotid artery. Radiology. 1975;116(1):93–4. https://doi.org/10.1148/116.1.93.
19. Blackburn JW. Anomalies of the encephalic arteries among the insane. J Comp Neurol Psychol. 1907;17:493–517.
20. Brzegowy K, Pekala PA, Zarzecki MP, Pekala JR, Roy J, Aziz HM, et al. Prevalence and clinical implications of the primitive trigeminal artery and its variants: a meta-analysis. World Neurosurg. 2020;133:e401–e11. https://doi.org/10.1016/j.wneu.2019.09.042.
21. Jimenez-Castellanos J Jr, Carmona A, Catalina CJ, Jimenez-Castellanos J Sr. Gross (mesoscopic) and applied anatomy of the anterior inferior cerebellar artery in man with special reference to its course through the cerebellopontine angle region. Acta Anat (Basel). 1992;143(3):182–7. https://doi.org/10.1159/000147245.
22. Rhoton AL Jr. The cerebellar arteries. Neurosurgery. 2000;47(3 Suppl):S29–68. https://doi.org/10.1097/00006123-200009001-00010.
23. Tschabitscher M, Perneczky A, Sinzinger H, Doskar K, Hoyer-Volavsek C. Vascularization of the cerebellum in man. Verh Anat Ges. 1975;69:559–61.
24. Alonso F, Kassem MW, Iwanaga J, Oskouian RJ, Loukas M, Demerdash A, et al. Anterior inferior cerebellar arteries juxtaposed with the internal acoustic meatus and their relationship to the cranial nerve VII/VIII complex. Cureus. 2017;9(8):e1570. https://doi.org/10.7759/cureus.1570.
25. Mazzoni A. Internal auditory canal arterial relations at the Porus acusticus. Ann Otol Rhinol Laryngol. 1969;78(4):797–814. https://doi.org/10.1177/000348946907800413.
26. Rhee SJ, Kim MS, Lee CH, Lee GJ. Persistent trigeminal artery variant detected by conventional angiography and magnetic resonance angiography-incidence and clinical significance. J Korean Neurosurg Soc. 2007;42(6):446–9. https://doi.org/10.3340/jkns.2007.42.6.446.
27. Chen MM, Chen SR, Diaz-Marchan P, Schomer D, Kumar VA. Anterior inferior cerebellar artery strokes based on variant vascular anatomy of the posterior circulation: clinical deficits and imaging territories. J Stroke Cerebrovasc Dis. 2018;27(4):e59–64. https://doi.org/10.1016/j.jstrokecerebrovasdis.2017.10.007.
28. Lazorthes G. Vascularisation et circulation cérébrale. Paris: Masson; 1961.
29. Duvernoy HM. Human brainstem vessels. Berlin: Springer; 1978.
30. Amarenco P, Hauw JJ. Cerebellar infarction in the territory of the superior cerebellar artery: a clinicopathologic study of 33 cases. Neurology. 1990;40(9):1383–90. https://doi.org/10.1212/wnl.40.9.1383.
31. Suttner N, Mura J, Tedeschi H, Ferreira MA, Wen HT, de Oliveira E, et al. Persistent trigeminal artery: a unique anatomic specimen--analysis and therapeutic implications. Neurosurgery. 2000;47(2):428–33.; discussion 33–4. https://doi.org/10.1097/00006123-200008000-00030.
32. Samaniego EA, Dabus G, Andreone V, Linfante I. Rare anatomical variations of persistent trigeminal artery in two patients with non-aneurysmal subarachnoid hemorrhage. J Neurointerv Surg. 2011;3(3):282–4. https://doi.org/10.1136/jnis.2010.003665.
33. Quain R. The anatomy of the arteries of the human body and its applications to pathology and operative surgery with a series of lithographic drawings. London: Taylor and Walton; 1844.
34. Teal JS, Rumbaugh CL, Bergeron RT, Scanlan RL, Segall HD. Persistent carotid-superior cerebellar artery anastomosis: a variant of persistent trigeminal artery. Radiology. 1972;103(2):335–41. https://doi.org/10.1148/103.2.335.
35. Shoja MM, Loukas M, Tubbs RS, D'Antoni A, DiLandro A, Cure JK. An aberrant cerebellar artery originating from the internal carotid artery. Surg Radiol Anat. 2012;34(3):285–8. https://doi.org/10.1007/s00276-011-0891-x.
36. Akgun V, Battal B, Bozkurt Y, Oz O, Hamcan S, Sari S, et al. Normal anatomical features and variations of the vertebrobasilar circulation and its branches: an analysis with 64-detector row CT and 3T MR angiographies. ScientificWorldJournal. 2013;2013:620162. https://doi.org/10.1155/2013/620162.
37. Lee H. Audiovestibular loss in anterior inferior cerebellar artery territory infarction: a window to early detection? J Neurol Sci. 2012;313(1–2):153–9. https://doi.org/10.1016/j.jns.2011.08.039.
38. Moffat DB. The embryology of the arteries of the brain. Ann R Coll Surg Engl. 1962;30:368–82.

Embryology and Variations of Superior Cerebellar Artery

A. Venier and G. Lanzino

The superior cerebellar artery (SCA) is one of three pair of arteries supplying the cerebellum and it is the most consistent in terms of presence and supply area. Reports of its absence are rare, but there are a lot of reports about variations in its number and origin.

Embryologically, the SCA arises from the caudal division of internal carotid artery (ICA) and it is inextricably linked to its type of fusion and to the reciprocal position with the basilar tip.

Clinical consequences of its occlusion may vary from asymptomatic to fatal, depending on the degree of anastomosis with the contralateral cerebellar arteries and the posterior inferior cerebellar artery (PICA). Clinically, the SCA has an important role not only in posterior circulation infarction but also in posterior fossa tumors, vascular malformations and neurovascular compression syndromes.

History

The modern understanding of the SCA begins in 1968 with Mani et al. and their anatomic-roentgenographic correlation study, which led the basis for the landmark description by Rhoton and Hardy in 1980, which still represents one of the most exhaustive report about SCA anatomy [1, 2]. In 1984 Yasargil detailed ten different variations of SCA in terms of number and origin and in 1987 Lasjaunias et al. clarified the embryological origin and development of the artery [3, 4].

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Embryological Development

Most of our knowledge about SCA embryology comes from the milestone work of Lasjaunias et al. who identified seven stages of arterial development, from the prechoroidal to the adult disposition [1, 4, 5].

Embryologically, the SCA originates from the caudal division of ICA. According to Lasjaunias, it is already visible in the prechoroidal stage (corresponding to Padget's stage 3) as arising from the distal unfused portion of the caudal division of the ICA [4, 6]. In the adult configuration, the SCA will originate typically from the distal basilar artery (BA) (telencephalic primitive stage, corresponding to Padget stage 5). However, its origin can vary according to the fusion pattern of the BA.

The main embryological steps of the SCA are illustrated in Tables 1 and 2.

Lasjaunias et al. described different types of basilar-tip dispositions based on the type of midline caudal ICA fusion and using the SCA origin as angiographic landmark. They identified three types, which are illustrated in Fig. 1 [4, 7]:

- Type 1: symmetrical cranial fusion if SCAs originate from BA (30.4%).
- Type 2: symmetrical caudal fusion if SCAs originate from the first segment of posterior cerebral artery (PCA), P1 (26.1%).
- Type 3: asymmetrical or partial fusion if one SCA originates from BA and the other one from P1 (43.5%).

Few years later, Brassier et al., starting from Lasjaunias observations and analyzing 25 cadaveric brains, described five types of arrangement of the superior basilar complex [5]:

- Type A: symmetric cranial fusion of both P1 (24%)
- Type B: symmetric caudal fusion (8%)
- Type C: symmetric caudal fusion with unilateral duplication of SCA arising from P1 (8%)

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Table 1 SCA embryological development according to the seven stages artery development of Lasjaunias et al. (Adapted from [4]: Lasjaunias P, Berenstein A, Ter Brugge KG. Surgical Neuroangiography—*Clinical Vascular Anatomy and Variations*: Springer; 1987)

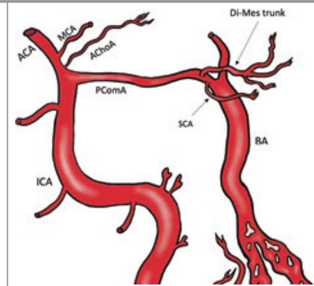
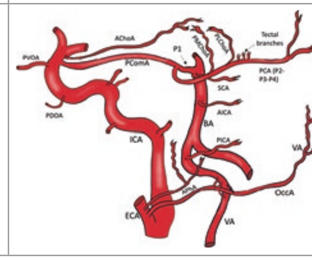
Lasjaunias stage	Events	Illustrations
I—Prechoroidal	From caudal division of ICA arise the diencephalon-mesencephalic trunk (Di-Mes trunk) and the superior cerebellar artery (SCA)	
III—Telencephalic primitive	In the adult configuration, the SCA arises from the distal basilar artery (BA)	

Table 2 Major embryological changes of the SCA according to Padget. Adapted from [6]: Padget DH. "The development of the cranial arteries in the human embryo." Contrib Embryol. 1948(32):205–62

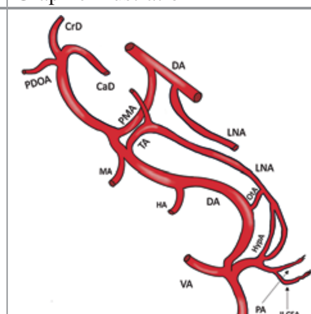
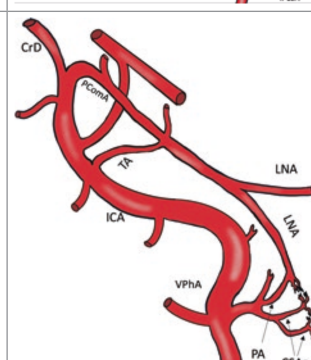
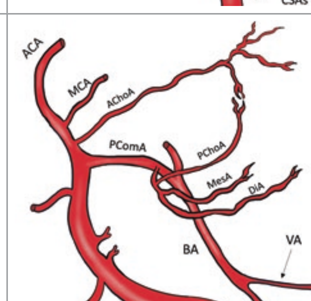
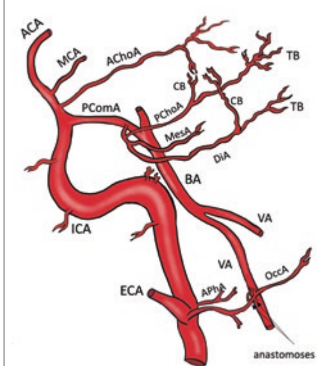
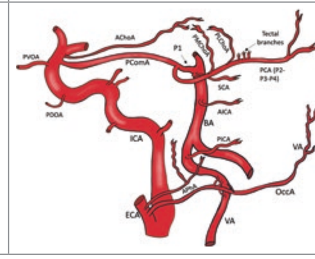
Stage	Embryo size (mm)	Major evolutions	Graphic illustration
I	4–5	• Paired longitudinal neural arteries (LNAs) visible • Carotido-vertebral anastomoses visible: Trigeminal (TA), otic (OtA), hypoglossal (HypA), proatlantal (PA) and second cervical segmental artery (II CSA) • Primitive ICA bifurcation visible into caudal and cranial divisions (CaD, CrD) • Posterior communicating artery (PComA) and BA not formed	
II	5–6	• Formation of the PComA • Regression of otic and hypoglossal arteries • Midline fusion of LNAs to form the basilar artery (BA)	
III	7–12	• Basilar artery (BA) completely formed • PComA completely formed • Origin from the distal PComA of a diencephalon-mesencephalic trunk (DiA-MesA). PChoA arising from the common trunk and anastomoses with the AChoA • Regression of trigeminal and proatlantal arteries • Formation of the vertebral artery VA by transverse anastomoses between the intersegmental arteries	

Table 2 (continued)

Stage	Embryo size (mm)	Major evolutions	Graphic illustration
IV	13–15	• Development of a choroidal (CB) and telencephalic branch (TB) from the PChoA and the diencephalic artery (DiA) • Remodeling of the BA • Migration of OccA origin on the external carotid artery (ECA) • Persistence of the anastomoses between OccA and VA	
V	16–18	• The telencephalic branch of the PChoA forms the main PCA trunk • The choroidal branches of the PChoA and of the diencephalic artery form the PLChoA and PMChoA • Complete formation of the VA • Complete formation of the OccA (remnant of the proatlantal artery) • Complete formation of the ascending pharyngeal artery (APhA, remnant of the hypoglossal artery) • SCA, AICA and PICA are visible	

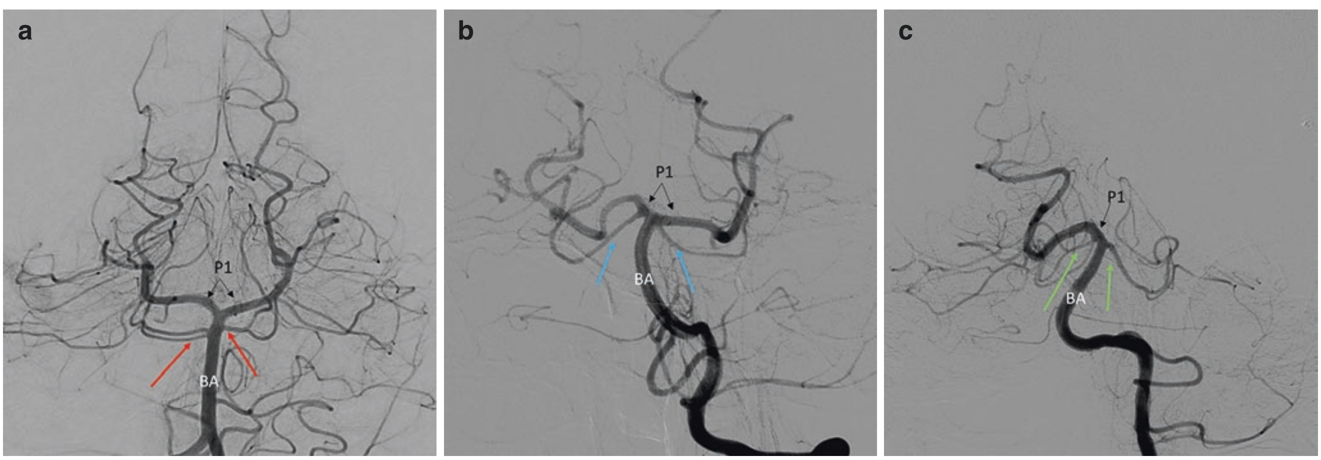


Fig. 1 Basilar-tip dispositions. The figures **a**, **b** and **c** show respectively cases of symmetrical cranial, symmetrical caudal and asymmetrical configuration of the basilar tip. In figure **a**, the superior cerebellar artery (SCA, red arrows) originates on both sides from the basilar artery (BA). On the right side it appears doubled. In figure **b**, the SCA (blue arrows) arises on both sides from the P1 segment of the posterior cerebral artery. In figure **c**, the SCA (green arrows) arises on the right side from the P1 segment and on the left side from the BA. There is a concomitant absence of the P1 segment on the left side

- Type D: asymmetric or partial caudal fusion (56%)
- Type E: asymmetric or partial caudal fusion with duplication of SCA arising from P1 (4%)

These considerations are of utmost importance to predict the localization of P1 perforators: bilaterally if the fusion is symmetric, unilaterally if it is asymmetric [4].

More details can be found in the chapter on the BA.

Number and Origin of the Artery

The SCA is the most constant cerebellar artery: while the anterior inferior cerebellar artery (AICA) and the posterior inferior cerebellar artery (PICA) sometimes may be absent, absence of the SCA is exceptional [3, 4, 8–10].

In most cases the SCA arises as a single trunk (70–97%), sometimes can have a duplicated (3–28%), and rarely a triplicated origin (2%) (Fig. 2) [1–3, 9, 11–22]. Duplication was

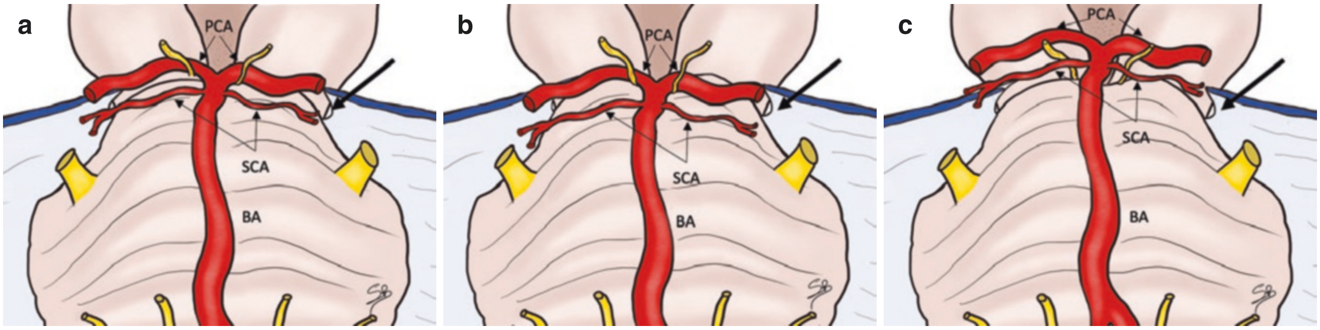


Fig. 2 SCA origin variants according to tentorium's free edge: normal, high and low. In the normal configuration (**a**) the basilar artery (BA) bifurcation into the two posterior cerebral arteries (PCA) occurs at the ponto-mesencephalic junction. In this case the origin of the superior cerebellar artery (SCA) is at the level of the tentorial incisura (black arrow). In case of BA bifurcation in the interpeduncular fossa (**b**), the origin of the SCA is above the tentorial incisura. If the BA bifurcates in the prepontine cistern (**c**), the origin of the SCA will be below the tentorial incisura

observed to be unilateral in 3–28% and bilateral in 1–8% [1, 8, 11, 24].

In most cases it arises from the BA (78–98%) between P1 and 7 mm below it (the average distance between P1 and the SCA origin being 2.5 mm), sometimes from the PCA (2–22%). It is almost never absent or at least there are few reports about it [1–4, 6, 9, 10, 12–14, 16, 17, 25].

Embryologically, the SCA arises from the BA when a symmetrical cranial fusion has occurred; otherwise from the P1 when a symmetrical caudal fusion has occurred; sometimes from both when an asymmetrical fusion has occurred (Fig. 1) [4]. When basilar-tip fusion is caudal, origin from P1 is more likely, but also a duplicate origin may be present [4]. A duplicated origin of the SCA corresponds to separate origins of medial and lateral divisions from BA or P1; while a triplicated origin corresponds to separate origins of medial and lateral branches from the medial division [4]. According to Lasjaunias et al., the duplicated origin and/or the P1's origin of SCA is always associated with a caudal (late) fusion of P1 but it also corresponds to a very early division of the main trunk into two branches [4, 5]. More details are reported in the dedicated BA's chapter.

The origins of SCAs and PCAs have frequently a cruciate configuration, crossing at the BA's apex [2, 26]. The height of BA's bifurcation is fundamental to define the position of SCA's origin in relationship to the tentorium's free edge. If BA's bifurcation occurs at the ponto-mesencephalic junction (normal), the SCA's origin will be medial to the tentorium's free edge; otherwise if the bifurcation is anterior to the mesencephalon in the interpeduncular cistern (high) or to the pons in the prepontine cistern (low), the origin of the SCA will be above or below the free edge of tentorium, respectively [2, 26]. These three possible configurations are shown in Fig. 2. Rhoton and coworkers, analyzing 25 cadaveric brains, found a normal height BA's bifurcation in 18 cases, high in 6 and low in 1 [2, 8].

Course of the Artery

The course of SCA can be divided into four segments according to Rhoton: anterior ponto-mesencephalic, lateral ponto-mesencephalic, cerebello-mesencephalic and cortical. The segments, branches and course of the SCA are illustrated in Fig. 3 [26].

s1—Anterior Ponto-Mesencephalic Segment

From its origin at the ponto-mesencephalic junction, to the antero-lateral brainstem margin, into the interpecuncular (78%) or prepontine cistern (22%), between the dorsum sellae and the upper brainstem [2, 18]. The SCA runs laterally passing below the oculomotor nerve if it arises from BA and above if it arises from P1. It is medial to the free edge of tentorium.

s2—Lateral Ponto-Mesencephalic Segment

From the antero-lateral brainstem margin to the cerebello-mesencephalic fissure, into the ambient cistern. It runs caudally, encompassing the brainstem, below the trochlear nerve, passing from above to below the tentorium. It ends at the anterior margin of the cerebello-mesencephalic fissure with the basal vein of Rosenthal and the PCA above and parallel to it.

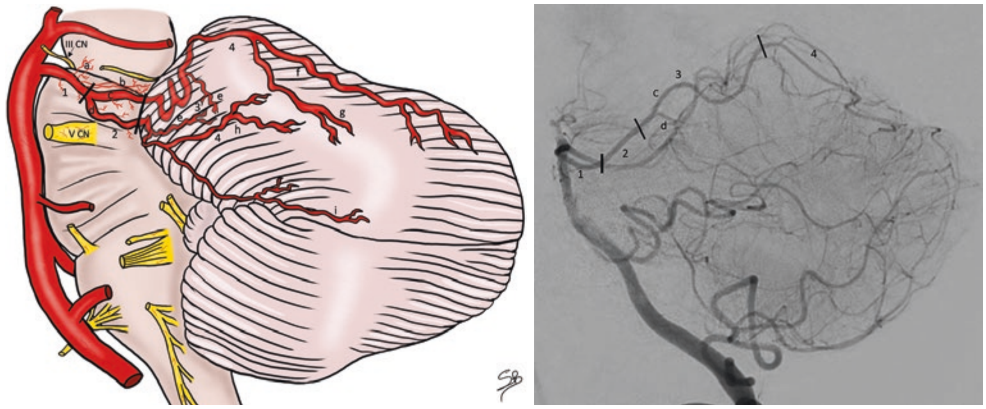


Fig. 3 SCA course, segments, and branches. (1) Anterior ponto-mesencephalic segment; (2) lateral ponto-mesencephalic segment; (3) cerebello-mesencephalic segment; (4) cortical segment. (a) direct perforating arteries; (b) circumflex perforating arteries; (c) lateral trunk; (d) medial trunk; (e) vermian arteries; (f) medial branch; (g) intermediate branch; (h) lateral brunch; (i) marginal artery; III CN: oculomotor nerve; V CN: trigeminal nerve

s3—Cerebello-Mesencephalic Segment

From the cerebello-mesencephalic fissure to the antero-superior cerebellar surface, into the quadrigeminal cistern. It deepens medially, above the trigeminal nerve, pointing to the midline, behind the superior medullary velum. After some hairpin-like curves, it loops deeply into the fissure and goes upward to the anterior edge of tentorium.

s4—Cortical Segment

From the antero-superior cerebellar surface to the terminal branches into the superior cerebellar cistern, below the tentorium.

Several studies reported on the length and diameter of the SCAs [2, 8, 10, 18–20, 27]. For example, according to Hardy et al. the mean diameter of a single origin SCA is 1.9 mm (1.2–2.8 mm) and after bifurcation the medial and the lateral trunks have a mean diameter of 1.4 mm (range 0.9–1.9 mm) and 1.2 mm (range 0.6–1.8 mm), respectively [2]. If the origin is duplicated, the medial and lateral trunks have a mean diameter of 1.3 mm (range 1.1–1.5 mm) and 1.1 mm (range 0.8–1.5 mm), respectively. For Garcia-Gonzales et al. the mean length of both SCAs was 14.4 ± 7.9 mm and the mean distance between SCA and PCA origins was 1.19 ± 0.8 mm (range 0–3.4 mm) [18]. Right and left SCA may have equal size in 33% of cases, right may be larger in 31%, left may be larger in 38% according to Stopford et al. [10].

Cranial Nerve Relationships

The SCA passes near the oculomotor, the trochlear and the trigeminal nerves and frequently has some points of contact with them.

The oculomotor nerve courses between the PCA above and the SCA below. Sunderland has suggested that occasionally the nerve could be constricted between these two arteries [28].

About two-thirds of SCAs, especially the main trunk or the medial trunk, have a point of contact with the III nerve usually on the nerve's inferior surface. Only when the SCA arises from the PCA the contact could be on the nerve' superior surface. In case of duplicate origin, low origin or fetal PCA, there is less likely a neurovascular contact [2, 26]. Different anatomical variations such as common SCA-PCA trunk or duplicated SCA may predispose to oculomotor nerve compression [21].

The trochlear nerve courses forward in the cerebello-mesencephalic fissure from the medial to the lateral side of the SCA's lateral and medial trunks, then runs between the lower surface of tentorium above and the SCA below. In the vast majority of cases (92%) there is a point of contact with the main trunk (34%), medial (52%) or lateral trunks (4%), or both (2%) usually on its inferior surface [2, 26].

The trigeminal nerve arises from the lateral surface of the pons and runs obliquely upward to exit the posterior fossa passing beneath the tentorium. The SCA traverses the brainstem just above the nerve making a shallow caudal loop on the pons' lateral surface [9]. About half of SCAs, usually the main trunk (24%), medial (4%) or lateral trunks (22%), both (8%), or marginal branch (2%), have a point of contact with the superior or superomedial surface of the V nerve at its root entry zone, generally because there is a prominent caudal loop [2, 26].

Tentorium Relationship

The proximal SCA runs medially to the anterior third of tentorium free edge and about 20% of SCAs have a point of contact with it in the anterior half. Then, the SCA loops caudally and normally passes beneath the tentorium free edge but sometimes can be in contact with its middle third. The distal SCA branches course medially to the posterior third of the free edge and finally passing below it to reach cortical surface of the cerebellum [2, 26].

Branches and Possible Anastomosis

Several different nomenclatures have been used to describe SCA branches. In particular, concerning the two SCA main trunks, many authors used different names to describe them, therefore generating a lot a confusion: medial and lateral (also marginal), rostral and caudal, or also superior and inferior [2, 3, 8, 26, 29–32]. In this chapter we refer to the two main trunks as the medial and the lateral one.

Yasargil described in detail the course of these two main trunks [3]. The lateral one courses antero-laterally along the quadrangular lobule, above the trigeminal nerve, and then postero-laterally in the horizontal fissure region to supply the supero-lateral part of cerebellar hemispheres and deep nuclei. The medial one encircles the ponto-mesencephalic junction to enter the quadrigeminal cistern in the inferior colliculus area giving off several hemispheric branches and perforators to the brachium conjunctivum area and inferior colliculus. Then it approximates with the contralateral branch turning inferiorly over the vermis as the superior vermian artery. Several anastomoses occur also between the medial branch of SCA and branches of PCA in the quadrigeminal cistern [2, 3, 23]. Critchley and Schuster called them *plexus pedunculi* but Duvernoy demonstrated that they were only ipsilateral [29, 31].

Garcia-Gonzales et al. demonstrated that the SCA bifurcates into lateral and medial branches at the ambient cistern in 84.1% of cases [18]. They also observed that the mean diameter of the lateral branch is 1.07 ± 0.37 mm (range 0.5–1.4 mm), and that of the medial one is 1.11 ± 0.27 mm (range 0.5–1.5 mm). After the bifurcation the medial branch is usually the dominant vessel. Both branches course caudally in the infratentorial part of the ambient cistern, near P2-P3 segments, the lateral posterior choroidal artery, the quadrigeminal artery, the lateral ponto-mesencephalic vein, the basal vein of Rosenthal and the trochlear nerve.

The Rhoton's group observed that SCAs arising as a single vessel bifurcate into a lateral and a medial trunk on average at 18.5 mm from the origin (range 0.6–34 mm), at the point of maximal caudal descent along brainstem's lateral side [2, 8, 26]. Generally, the medial trunk courses superior to the caudal and they have a nearly equal diameter (1.2 mm on average), but if one is smaller, more frequently the lateral trunk, the medial will supply a larger area. These two trunks could be the result of a duplicated origin or a true bifurcation of the parent artery.

The SCA and its lateral and medial trunks give off multiple branches along their course: perforating, precerebellar and cortical branches (Table 2) [2, 26].

Perforating arteries are classified according to the system devised by Hardy et al. in direct and circumflex types which both enter the brainstem [2]. The first ones have a straight and short course, while the second ones encompass the brainstem before entering it. The circumflex arteries in turn are subdivided in short and long types in relation to their travel distance, less or more than 90° around the brainstem [2, 26].

Usually perforating arteries come from the proximal SCA (within 1 mm 33%, within 2 mm 50%), from the main trunk or its divisions in a number from 2 to 5, rarely to 10 or more (range 1–12) [2, 8, 18, 26]. They are generally less than 1 mm in diameter (mean 0.5 mm, range 0.1–1 mm) [2, 18]. The most common type of perforating arteries coming from the main trunk are the long circumflex (61%) and they terminate in the tegmentum, interpeduncular fossa, cerebral peduncle, collicular region. On the other hand, from lateral and medial trunks, perforators are mainly circumflex and terminate in tegmentum, inferior colliculus, cerebral peduncle, interpeduncular fossa [2, 26].

Garcia-Gonzales et al. identified 59% of direct, 28% short circumflex and 13% long circumflex perforators [18]. The first perforator was at a median distance of 2.2 mm (range 0.2–15 mm) from the origin of the right SCA and 1.75 mm (range 0.1–12 mm) on the left side.

Perforators from the SCA enter the brainstem in two main different zones, identified and named by Yasargil: interpeduncular, superior and inferior medial pontine, lateral pontine and basal cerebellar groups enter the basal perforator zone, whereas the lemniscal trigone group enters the dorsal perforator zone [3]. The direct SCA perforators belonged to the interpeduncular group in 85% of cases, the reminder belonged to superior medial pontine group. The short circumflex belonged to the lateral pontine and superior medial pontine groups, while the long circumflex belonged to the basal cerebellar and lemniscal trigone groups.

However, there are some perforator-free zones: the BA between SCA and AICA is described as a variable perforator-free zone and some studies suggest also that this area may extend to the proximal SCA [4, 33–36]. This kind of information during both microsurgical and endovascular procedures is of utmost importance.

Precerebellar arteries arise from the distal part of the trunks and from the proximal part of cortical branches within the cerebello-mesencephalic fissure. They have a sharp course in the fissure that make difficult their dissection and identification. They are divided in a medial and a lateral group. The first one is represented by small branches passing between the superior medullary velum and the central lobule, the second one by larger branches passing between the superior and middle cerebellar peduncles and the central lobule's wings. Precerebellar branches coming from cortical arteries supplying the hemispheric surface are directed to the dentate and deep cerebellar nuclei (average 4, range 2–8), while those coming from cortical arteries supplying the vermis are directed to the inferior colliculi and the superior medullary velum (average 2, range 1–6) [2, 26]. The mean diameter was 0.3 mm (range 0.1–0.7 mm), according to Hardy et al. [2].

Cortical arteries are divided into hemispheric and vermian groups. In turn, the hemispheric group is subdivided into medial, intermediate and lateral branches that supply the tentorial surface lateral to the vermis and the vermian group into medial and paramedian branches [2, 26].

The hemispheric arteries arise from the lateral and medial trunks in the cerebello-mesencephalic fissure and course upward sharply to reach the superior surface, running parallel, and fan out in a radial pattern, extending to the horizontal fissure supplying the supero-medial cerebellar hemisphere and the dentate nucleus [1–3, 26, 30]. They give rise to the precerebellar arteries. The most common configuration is represented by three arteries, medial, intermediate and lateral, but they can be sometimes up to five, and are in a reciprocal relationship in terms of dimensions and territory of supply. The medial and lateral branches generally arise from the medial and lateral trunks, respectively. Once they reach the tentorial surface, hemispheric arteries split into one to seven branches (three on average) that arborize progressively and disappear in the cerebellar folia [2, 26]. The mean diameter is 0.9 mm (range 0.3–1.8 mm), according to Hardy et al. [2].

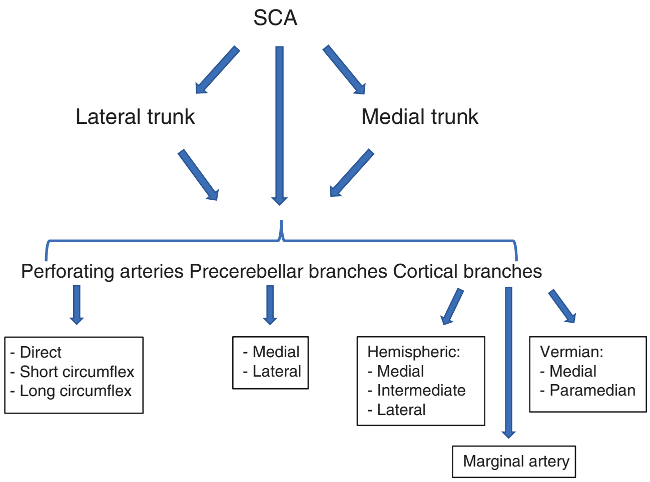
The vermian arteries arise mainly from the medial trunk in the cerebello-mesencephalic fissure and the most common configuration is represented by two arteries, medial and paramedian, but they can be sometimes up to four, and are in a reciprocal relationship in terms of dimensions and territory of supply, several anastomoses existing between the two sides, especially near the apex of the tentorial surface [2, 26]. The artery of the superior vermis is the terminal branch of the SCA originating from the third segment. The right and left side arteries anastomose with each other within the quadrigeminal cistern and then course posteriorly over the vermis close to the midline [17]. The distal part of these arteries anastomoses with the arteries of the inferior vermis which are branches of PICA [1, 3]. The mean diameter is 0.5 mm (range 0.1–1.2 mm), according to Hardy et al. [2].

The marginal artery, when present (in 52% of cases according to Hardy et al.), represents the first and largest cortical branch of the SCA and its size varies and it is inversely related to AICA [1, 2, 26]. Generally, it arises from the medial trunk but could also arise from the main trunk, from the lateral one or from the BA. From the lateral pontomesencephalic segment, in the ambient cistern, it points directly to the petrosal surface adjoining the tentorial one in the horizontal fissure [26]. The distal part of the artery represents a landmark, that demarcates superior and inferior cerebellar lobes when dipped in the horizontal fissure [1]. The most constant supply is to the anterior part of the lips of the petrosal fissure, at its largest it can supply the full extent of both lips of the petrous fissure and the superior part of the petrosal surface, always being in a reciprocal balance with AICA: if the marginal artery is absent or small, most of the supply is given by AICA and its branches [2]. Anastomoses between these two arteries are frequent and most prominent if there is a large marginal branch [26].

Within the fissure, the artery may anastomose with hemispheric branches of AICA or PICA [2, 3]. Perforating arteries arising from this branch terminate in the region of the middle cerebellar peduncle [26]. The mean diameter is 0.8 mm (range 0.4–1.3 mm), according to Hardy et al. [2].

A meningeal branch of the SCA was first observed by Wollschlaeger et al. in only one out of ten barium-injected cadaver brains they studied and was considered as a small arterial anastomosis between SCA and the artery of Davidoff and Schechter (ADS) [37]. Later they reported that although they believe that ADS was constant, the meningeal branch of SCA was present only occasionally, running rostrally to the quadrigeminal plate [27]. Ono et al. identified a meningeal branch of SCA in 28% of 25 cadaver specimens describing that it arises from the medial trunk and crosses the middle incisural space under the free edge of the tentorium [38]. However, the presence of a meningeal branch of SCA is not reported in the masterpiece study of dural arteries anatomy of the same group [39]. Some years later, Umeoka et al. first identified surgically this branch arising from s2 in 25.6% of patients operated for trigeminal neuralgia [40]. They stated that this branch can be cut without eliciting neurological deficits. This branch can become hypertrophic and supply tentorial dural arteriovenous fistulas. Table 3 summarizes the main branches of the SCA.

Table 3 SCA branches



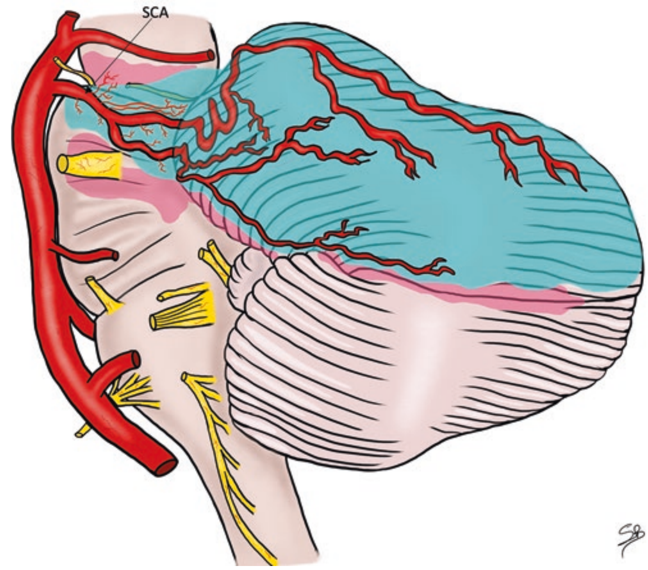


Fig. 4 SCA parenchymal territories. The blue area shows the territories constantly supplied by the superior cerebellar artery (SCA), which corresponds to the antero-lateral part of the ponto-mesencephalic junction, and to the tentorial and petrosal surfaces of the cerebellum. Its territory extends to the cerebello-mesencephalic groove, to the vermian and paravermian areas. Sometimes its territory can include the region of the horizontal fissure, the area of the trigeminal nerve and the mesencephalon region (pink area)

Parenchymal Territories

The cortical territory of the SCA is more constant than those of AICA and PICA but is reciprocal with them and is illustrated in Fig. 4. The most constant cortical supply is to the tentorial and petrosal surfaces: the SCA usually supplies most of the tentorial surface and frequently the adjacent upper part of the petrosal surface, within the cerebello-mesencephalic groove and above the petrosal fissure. The maximal field of supply includes a full half of the tentorial surface with overlap onto the opposite half of the vermis, the superior part of the suboccipital surface, and the upper two-thirds of the petrosal surface, including both lips of the petrosal fissure. The smallest field of supply includes only the part of the tentorial surface that lies anterior to the tentorial fissure [2, 26].

The territories distribution of the two main trunks are generally constant: the medial one supplies vermian and paravermian areas and the lateral one the cerebellar hemisphere on the suboccipital surface. Sometimes PICA or AICA or both may join SCA on the superior part of the cerebellar hemisphere, according to Lang et al. in 33% of cases [13]. On the contrary, the SCA may supply the inferior part of cerebellar hemispheres together with PICA in 3% of cases [13]. The region of the horizontal fissure in the majority of cases is supplied by AICA (48%), but frequently also by SCA (40%), both in 12% of cases [32]. Moreover, the lateral trunk supplies almost exclusively the deep cerebellar nuclei, with a medial branch to emboliform, globose and fastigial nuclei and a lateral one to the dentate nucleus [12]. Additional contributions come from transcerebellar arteries, including PICA [30].

Sometimes the tentorial surface lateral to the vermis may be supplied by only two hemispheric branches or by adjacent hemispheric branches coming from AICA and PICA or by some vermian branches or marginal artery that could overlap the medial and lateral hemispheric branches, respectively [26]. For example, Rhoton found in one cadaveric brain that the whole tentorial surface was supplied by a single branch arising from the lateral trunk on one side and by branches arising from the medial trunk on the other one.

Lazorthes et al. described in 15% of cases a bilateral vermian supply that was even greater (53%) according to Scialfa et al. [12, 16].

Handa et al. was the only one to find an SCA supply to the internal auditory canal [41].

The SCA contributes not only to parenchymal territories supply but also to dural supply: the true vascular supply of the tentorium is represented by an anastomotic network between PCA on the superior surface and SCA on the inferior one [42].

Variants

Cadaveric studies, radiological ones and case reports have detailed variations of the branching pattern, origin and course of the SCAs [1, 2, 5, 8, 18, 19, 25, 33, 43–49].

Number and Origin

Garcia-Gonzales et al., after analyzing 20 cadaveric brains, proposed a classification of the SCA origin based on the spectrum variation reported by Yasargil [3, 18]:

- Type A: as single vessel from BA trunk, clearly separated from PCA (35%)
- Type B: from BA bifurcation (35%)
- Type C: as separate vessel from PCA (5%)
- Type D: one SCA from BA, the other from PCA (5%)
- Type E: one from BA bifurcation, the other from PCA (5%)
- Type F: bilateral duplication from BA (5%)
- Type G: double SCA on one side of BA, single one on the other side (10%)

The most common patterns of origin were A and B (both 35%). Most SCA (50%) arise from the BA with a normal-lying bifurcation (type B 50%), the reminder 45% had a high bifurcation (type A 67%).

Krzyzewski et al. proposed another classification of the SCA origin but reviewing 200 cerebral tomography (CT) [45]:

- Type 1: typical configuration (both SCA from BA)
- Type 2: aplastic SCA
  - 2a: bilaterally
  - 2b: unilaterally
- Type 3: duplicated
  - 3a: bilaterally
  - 3b: unilaterally
- Type 4: common origin of PCA and SCA from BA
  - 4a: bilaterally
  - 4b: unilaterally
- Type 5: SCA arises from PCA
  - 5a: bilaterally
  - 5b: unilaterally

They also observed that the diameter of SCA differs significantly depending on the origin: the largest being from BA. Smaller vessels are more likely to predispose to ischemic events especially after surgical manipulation and a common PCA-SCA trunk increases the risk of accidental closing of this branch during clipping PCA or SCA aneurysms.

Mani et al. dissected 30 cadaveric brains and found that in 8% of patients with SCA duplication, the upper trunk arose from the PCA, while in the reminder both trunks arose from the BA [1]. In all patients with duplication, the marginal artery arose from the lower trunk and the superior vermis artery from the upper trunk.

Caruso et al., studying 100 fixed human brains, identified a rare variation: a bilateral common trunk between PCA and SCA of 4 mm in diameter and 5.3 mm in length, with an angle P1-SCA of 180° [43]. This anomaly may be due to a missing fusion of the neural primitive arteries at the level of the basilar rostral limit.

Uchino et al. reviewed 136 magnetic resonance imaging (MRI) looking for proximal SCA variations [25]. Several one may be due to a missing fusion of the neural primitive arteries at the level of the basilar rostral limit during early embryonic stage. They identified in 9.6% of cases a duplicated SCA, in 4.4% a common PCA-SCA trunk, in 3% an early bifurcating SCA.

A bilateral origin of the SCA from the PCA is very rarely reported [25, 43, 44] and presents an usual radiological appearance, according to Aydin et al. Embryologically, this anomaly is probably associated with an unusual development of the distal basilar region during embryogenesis and in particular may be explained as a lack of normal fusion of the BA at the origin of SCA during BA development from the primitive neural arteries or PCA anastomoses with BA more caudally than usual [44].

Porzionato et al. reported a double origin of the left SCA from two small arteries arising from BA at a reciprocal distance of 0.5 mm and joining after 9 mm in a larger vessel [47]. It was associated with a subacute ischemic lesion with hemorrhagic evolution in the SCA territory, maybe to the hemodynamic instability, and also a bilateral xanthogranuloma of the choroid plexuses of the lateral ventricles coexisted. The abnormal persistence of an embryological vessel may be hypothesized but the very close relationship between the two vessels and the rarity of this anomaly couldn't exclude other rarer mechanism, for example the division of a single vessel.

Rarely the SCA originates from the ICA [4, 46, 50–52]. The communication between the ICA siphon and SCA represents a partial trigeminal artery persistence which supplies SCA territories, combined with an incomplete fusion of the longitudinal neural arteries (LNAs). The presence of an interposing BA between ICA and SCA is defined by the grade of fusion of longitudinal neural arteries and the BA [46]. In such cases, an anastomosis between the cavernous segment of the ICA and the SCA is observed, with or without an anastomosis with the BA. The embryological explanation of these cases resides in a partial persistence of the trigeminal artery, that takes charge of the vascular territory of the SCA, combined to the incomplete fusion of the longitudinal neural arteries (LNAs) [6]. The grade of fusion of the LNAs and formation of the BA will influence the presence of an interposing BA in the ICA-SCA anastomosis.

Teal et al. described a case of ICA and SCA anastomosis without an interposed segment of BA [51]. The embryological sequence of events leading to this anomaly is unknown but it is likely that the primary anastomosis was between the ICA and an ipsilateral island of LNA (primordial SCA) and the main source of blood supply of the hindbrain was the primitive hypoglossal artery (HypA), the primitive otic artery (OtA), the first cervical segmental artery (ICSA) or any combination of them.

Course

The junction between s1 and s2 is frequently asymmetrical (56%): the downward convex curve can be higher on one side compared to the contralateral one [33].

Uchino et al. reported a case of an extremely tortuous SCA in its s1 segment that mimicked an AVM, potentially a pure arterial malformation, which remained stable over the years [49]. Similarly to the carotid *rete mirabile*, a segmental interruption of the SCA and consequent collateral network formation at an early embryonic stage may be the embryological explanation of this anomaly [53].

Fenestration of the SCA has been rarely reported [54, 55].

Clinical Implication

The SCA is important in both ischemic and hemorrhagic cerebrovascular disease of the posterior circulation. In particular, the dentate nucleus is the most common site of spontaneous cerebellar hemorrhage and it is supplied by precerebellar and penetrating cortical branches of the SCA [30].

The SCA and its branches are exposed during surgery of tumors involving the cerebellum, posterior cavernous sinus, tentorial incisura, pineal region and cerebellopontine angle; in dealing with aneurysms arising at the basilar apex, the origin of the SCA and PCA; with arteriovenous malformations; but also during vascular decompression of the trigeminal nerve in trigeminal neuralgia and during a revascularization bypass procedure for posterior fossa ischemia.

SCA Infarction

Isolated SCA occlusion is uncommon, and its effect could range from clinical silence to brainstem and cerebellar infarction, swelling, hemorrhage and death [2, 9]. Since cerebellar arteries display a wide array of potential and extensive anastomoses, collateral circulation and variations in arterial distribution will influence patient's recovery and survival after occlusion of the SCA.

The territories supplied by the SCA are the most vulnerable to ischemic damage secondary to decreased flow in the posterior circulation because they are localized at the distal end of the vertebral artery (VA) and BA axes [30]. Infarct may occur in the SCA territories in the absence of SCA occlusion but instead in case of VA or BA's occlusions.

SCA occlusion may result in infarction, both ischemic and hemorrhagic, of the cerebellum, dentate nucleus, brachium conjunctivum, and long sensory pathways in the tegmentum of the rostral pons and, in typical cases, its clinical presentation is quite characteristic. At the onset, vomiting, dizziness and inability to stand or walk may be present [56]. The SCA syndrome is represented by:

- cerebellar dysfunction (cerebellum and deep nuclei and peduncles),
- ipsilateral intention tremor (dentate nucleus and superior cerebellar peduncle),
- ipsilateral Horner's syndrome (descending oculosympathetic fibers),
- contralateral loss of pain and temperature sensation (lateral spinothalamic and trigeminothalamic tracts),
- nystagmus (medial longitudinal fasciculus and cerebellar pathways),
- contralateral disturbance of hearing (crossed fibers of the lateral lemniscus),
- loss of emotional expression on the analgesic side (involuntary mimetic pathways in the upper brainstem).

Mills first described the clinical manifestation of an SCA stroke characterized by ipsilateral cerebellar and brainstem signs and contralateral dissociated sensory impairment secondary to SCA occlusion at its origin [57, 58]. Terao et al. noted that the cerebellar symptoms were due to the interruption of the distal flow of lateral or medial branches, determining an anterior rostral cerebellar infarction, whereas if the proximal SCA is interrupted with its perforators, it produces a rostral brainstem infarction [59].

Posterior circulation infarction is quite infrequent in children and vertebral artery dissection even more. A case was reported by Lin et al. of a 12-year-old boy with occlusion of the left vertebral artery determining an artery-to-artery embolism of SCA [60]. Other possible mechanisms can be a cardiac source of emboli, dissection or fibromuscular dysplasia [61–67].

Trigeminal Neuralgia

Trigeminal neuralgia as the result of arterial compression and distortion was first postulated by Dandy and then confirmed by Jannetta [68–70]. Dandy described in 30.7% of his 215 cases of trigeminal neuralgia neural compression from the SCA [68]. Hardy and Jannetta agreed that the SCA is the most common cause of trigeminal neuralgia [9, 70]. Sindou et al. reported that in 66.5–88% of cases the cause of trigeminal compression was the SCA. When there is a prominent caudal loop of the SCA, fascicles of trigeminal nerve may be distorted at the root entry zone. Less frequently, trigeminal neuralgia may be caused by AICA, PICA or superior petrosal veins such as transverse pontine veins [71].

Tentorial Dural Arterio-Venous Fistulas (TDAVF)

TDAVFs represent a specific subgroup of DAVFs (<4%), located in the reflected dura of tentorium and its attachments, that usually drain into cortical veins, thus definitive treatment is recommended [72–75]. The arterial supply of midline TDAVFs is mostly represented by the artery of Davidoff and Schechter, a tentorial branch of the PC, and the medial dural-tentorial branch of the SCA [37]. In particular, the latter supplies midline TDAVFs posterior to the incisura. In physiologic condition these two arteries are too small to be seen in anatomic studies and digital subtraction angiography (DSA) [2, 38, 39]. Generally, they are visible during surgery and in DSA only when pathologically enlarged [48, 76]. Lawton et al. demonstrated the importance of pial arteries contribution to TDAVFs: PCA and SCA branches together supply 26% of the TDAVFs analyzed, especially galenic ones [74].

SCA Aneurysms

SCA aneurysms are uncommon; they account for 1.5–4.5% of all intracranial aneurysms and 10–13.5% of posterior circulation ones [77, 78]. They have a higher risk of rupture at a smaller size (5 mm) and typically they have a wide neck with a dome/neck ratio < 2 [77].

SCA aneurysms may be found incidentally but can present clinically with the classical subarachnoidal hemorrhage (SAH), cerebellar signs or III, IV, V cranial nerve palsies, generally affected by direction or growth pattern rather than size [79].

Generally, SCA aneurysms are grouped with posterior circulation ones which are considered, as a group, challenging from a surgical perspective. However, SCA aneurysms have some peculiar characteristics which make them easier (compared to basilar bifurcation aneurysms) to access from a surgical point of view, given their lateral orientation from the basilar trunk and often small neck [80, 81]. As a matter of fact, SCA aneurysms can be reached either with a conventional pterional approach or with a subtemporal one with or without their modifications. Moreover, aneurysms of the SCA origin do not pose particular risks of perforating arteries injury because there are no perforating arteries in the shoulder of the SCA where generally the aneurysm's neck is dissected [82].

Hzuka et al. treated 69 patients with BA-SCA aneurysms and identified three patterns of neck position according to SCA involvement degree [83]:

- Type A (no involvement)—SCA originates directly from BA and the aneurysm neck is on the junction (35%).
- Type B (half involvement)—SCA originates from the lateral side of the aneurysm neck (45%).
- Type C (complete involvement)—all the neck mounts on the first segment (20%).

They observed some peculiar details: SCA involved types (B + C) represented 76% of ruptured aneurysms and 53% of unruptured ones, in particular type C accounted for 47% of ruptured and type A for 46% unruptured; type B and C had a higher tendency to incomplete treatment and higher risk of SCA occlusion; in 90.5% of cases aneurysms formed on the inclined side of BA trunk.

Jin et al. analyzed 33 patients with SCA aneurysms at the junction BA-SCA and categorized them according to the main direction of the sac and the angle between BA and SCA aneurysm center [79]. Most aneurysms are lateral-superior (69%), lateral-horizontal (20.7%), lateral-inferior (6.9%) and posterior (3.4%). They don't differ significantly according to the treatment modality.

If treatment of the aneurysm is indicated, endovascular treatment is preferred in small neck aneurysm or with a dome/neck ratio > 2, type A aneurysm and calcifications. On the other hand, surgical treatment is preferred in wide neck aneurysms or with a dome/neck ratio < 2, type B or C aneurysms and intraluminal thrombus. Before surgery, it is of utmost importance to consider the relationship between the SCA, the dorsum sellae and the free tentorial edge. If the SCA origin is high, a pterional craniotomy with an eventual orbitozygomatic extension may be appropriate. Otherwise, if the origin is low, an antero-lateral approach with transcavernous extension, or a lateral approach with anterior petrosectomy or a retrosigmoid approach may be needed [77].

Neoplasms

The SCA and its branches may be stretched against the tentorial edge by expanding lesions of the posterior fossa protruding rostrally through the tentorial incisura [2, 26].

Elevation of the SCA over the PCA visible in DSA lateral projection may indicate the presence of an expanding mass in posterior fossa [1].

The upper vermian branch is elevated when the culmen is displaced upward into the tentorial incisura by quadrigeminal plate regions' masses such as arachnoid cysts, suprasellar tumors [33]. Brainstem lesions are rarely large enough to displace the SCA and its branches but pontine hematomas, for example, may anteriorly displace the interpeduncular branches. Extracerebellar tumors expanding in the retrosellar space may displace the pontine segment backward and downward while large acoustic tumors and petrous apex meningiomas medially and posteriorly. Tentorial meningiomas may separate PCA from SCA [33].

Surgery

The SCA and its branches may be exposed in surgical approaches to the tentorial notch, pineal region, clivus, BA apex, trigeminal nerve, cerebello-pontine angle and the superior surface of cerebellum [2].

The only approach that gives a satisfactory exposure of SCA origin and its segments is a subtemporal craniotomy with elevation of temporal lobe combined with division and retraction of the tentorium [2]. To expose the distal cortical branch, a supracerebellar approach may be needed. For an adequate visualization of the course of the vessel in the depth of the cerebellomesencephalic fissure, a posterior interhemispheric transtentorial approach may be indicated. To expose the trigeminal region a unilateral suboccipital craniotomy is needed [2].

For revascularization procedures, lateral and medial trunks of SCA can be used and may be exposed with a combined petrosal, lateral supracerebellar-infratentorial or subtemporal approach [19].

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References

1. Mani RL, Newton TH, Glickman MG. The superior cerebellar artery: an anatomic-roentgenographic correlation. Radiology. 1968;91(6):1102–8.
2. Hardy DG, Peace DA, Rhoton AL Jr. Microsurgical anatomy of the superior cerebellar artery. Neurosurgery. 1980;6(1):10–28.
3. Yasargil MG. Microsurgical anatomy of the basal cisterns and vessels of the brain. Diagnostic studies, general operative techniques and pathological considerations of the intracranial aneurysms. In: Microneurosurgery. I. Stuttgart: Thieme; 1984.
4. Lasjaunias P, Berenstein A, Ter Brugge KG. Surgical neuroangiography—clinical vascular anatomy and variations. Springer; 1987.
5. Brassier G, Morandi X, Mercier P, Velut S. The posterior part of the arterial circle of the skull base. Caudal division of the embryonic internal carotid artery. Riv Neurol. 1994;7:85–9.
6. Padget DH. The development of the cranial arteries in the human embryo, Contribution to embryology, vol. 32. Washingtion: Carnegie Institution; 1948. p. 205–62.
7. Campos C, Churojana A, Rodesch G, Alvarez H, Lasjaunias P. Basilar tip aneurysms and basilar tip anatomy. Interv Neuroradiol. 1998;4(2):121–5.
8. Saeki N, Rhoton AL Jr. Microsurgical anatomy of the upper basilar artery and the posterior circle of Willis. J Neurosurg. 1977;46(5):563–78.
9. Hardy DG, Rhoton AL Jr. Microsurgical relationships of the superior cerebellar artery and the trigeminal nerve. J Neurosurg. 1978;49(5):669–78.
10. Stopford JSB. The arteries of the pons and medulla oblongata. J Anat. 1916;50:131–64.
11. Blackburn IW. Anomalies of the encephalic arteries among the insane. J Comp Neurol. 1907;17:493–517.
12. Lazorthes G, Gouaze A, G. S. Vascularisation et circulation de l'encéphale. Anatomie descriptive et fonctionnelle. Paris: Masson; 1976.
13. Lang J. Clinical anatomy of the head. Neurocranium, orbit, craniocervical regions. Berlin, Heidelberg, New York: Springer; 1983.
14. Marini de Ajauro M. Vascularisation artérielle du cervelet. Folia Anatomica Universitatis Conimbrigensis. 1970;50:3–55.
15. Mitterwallner FV. Variationsstatistische Untersuchungen an den basalen HirngefaBen. Acta Anat (Basel). 1955;24:51–88.
16. Scialfa G, Bank W, Meget M, Corbaz. The posterior fossa arteries: 1. The morphology and variation of the anterior inferior cerebellar artery. 2. The arteriographic localisation of the fourth ventricle. In: Salamon G, editor. Advances in cerebral angiography. Berlin, Heidelberg, New York: Springer; 1975. p. 55–61.
17. Salamon G, Huang YP. Radiologic anatomy of the brain. Berlin: Springer-Verlag; 1976.
18. Garcia-Gonzalez U, Cavalcanti DD, Agrawal A, Spetzler RF, Preul MC. Anatomical study on the "perforator-free zone": reconsidering the proximal superior cerebellar artery and basilar artery perforators. Neurosurgery. 2012;70(3):764–72. discussion 71–2.
19. Dodevski A, Tosovska Lazarova D, Zhivadinovik J, Lazareska M, Stojovska-Jovanovska E. Morphological characteristics of the superior cerebellar artery. Pril (Makedon Akad Nauk Umet Odd Med Nauki). 2015;36(1):79–83.
20. Akgun V, Battal B, Bozkurt Y, Oz O, Hamcan S, Sari S, et al. Normal anatomical features and variations of the vertebrobasilar circulation and its branches: an analysis with 64-detector row CT and 3T MR angiographies. Scientific World Journal. 2013;2013:620162.
21. Pai BS, Varma RG, Kulkarni RN, Nirmala S, Manjunath LC, Rakshith S. Microsurgical anatomy of the posterior circulation. Neurol India. 2007;55(1):31–41.
22. Zenteno M, Moscote-Salazaar LR, Alvis-Miranda HR, Rojas A, Lee A. Unilateral triplication of superior cerebellar artery associated with fetal posterior cerebral artery: case report. Acta Medica Int. 2015;2:158–9.
23. Marinkovic SV, Gibo H. The surgical anatomy of the perforating branches of the basilar artery. Neurosurgery. 1993;33(1):80–7.
24. Schwartz HG. Arterial aneurysm of the posterior fossa. J Neurosurg. 1948;5(3):312–6.
25. Uchino A, Sawada A, Takase Y, Kudo S. Variations of the superior cerebellar artery: MR angiographic demonstration. Radiat Med. 2003;21(6):235–8.
26. Rhoton AL Jr. The cerebellar arteries. Neurosurgery. 2000;47(3 Suppl):S29–68.
27. Wollschlaeger G, Wollschlaeger PB, Lucas FV, Lopez VF. Experience and result with postmortem cerebral angiography performed as routine procedure of the autopsy. Am J Roentgenol Radium Therapy, Nucl Med. 1967;101(1):68–87.
28. Sunderland S. Neurovascular relations and anomalies at the base of the brain. J Neurol Neurosurg Psychiatry. 1948;2:243–57.

29. Critchley M, Schuster P. Beiträge zur Anatomie und Pathologie der Arteria cerebelli superior. Zeitschrift für die gesamte Neurologie und Psychiatrie. 1933;144:681–741.
30. Stephens RB, Stilwell DL. Arteries and veins of the human brain. Springfield: Charles C Thomas; 1969.
31. Duvernoy HM. Human brain stem vessels. Berlin, Heidelberg, New York: Springer; 1978.
32. Hoffman HB, Margolis MT, Newton TH. The superior cerebellar artery, section I. Normal gross and radiologic anatomy. In: Newton THPP, editor. Radiology of the skull and brain. 2nd ed. St. Louis: Mosby; 1974. p. 1809–30.
33. Huber P. Cerebral arteries—superior cerebellar artery. Cerebral angiography. New York: Thieme Medical Publishers; 1982. p. 158–63.
34. Chaloupka JC, Putman CM, Awad IA. Endovascular therapeutic approach to peripheral aneurysms of the superior cerebellar artery. AJNR Am J Neuroradiol. 1996;17(7):1338–42.
35. Guglielmi G, Vinuela F, Guidetti G, Dazzi M. The Guglielmi detachable coil "crescent" in the endovascular treatment of peripheral brain aneurysms: technical case report. Neurosurgery. 2007;61(5 Suppl 2):E295–6. discussion E6.
36. Senveli ME, Aciduman A, Belen D. Giant distal superior cerebellar artery aneurysm: case report and review of the literature. Turk Neurosurg. 2008;18(2):177–82.
37. Wollschlaeger PB, Wollschlaeger G. An infratentorial meningeal artery. Radiologe. 1965;5(11):451–2.
38. Ono M, Ono M, Rhoton AL Jr, Barry M. Microsurgical anatomy of the region of the tentorial incisura. J Neurosurg. 1984;60(2):365–99.
39. Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A Jr. Microsurgical anatomy of the dural arteries. Neurosurgery. 2005;56(2 Suppl):211–51. discussion −51.
40. Umeoka K, Takusakawa Y, Kominami S, Kobayashi S, Morita A. The meningeal branches of the superior cerebellar artery: a surgical observation study. J Neurosurg. 2016;124(1):244–7.
41. Handa J, Matsuda I, Nakasu S, Nakano Y. Agenesis of an internal carotid artery: angiographic, tomographic and computed tomographic correlation. Neuroradiology. 1980;19(4):207–11.
42. Merland JJ, Bories J, Djindjian R. The blood supply of the falx cerebri, the falx cerebelli and the tentorium cerebelli. J Neuroradiol. 1977;4(2):175–202.
43. Caruso G, Vincentelli F, Rabehanta P, Giudicelli G, Grisoli F. Anomalies of the P1 segment of the posterior cerebral artery: early bifurcation or duplication, fenestration, common trunk with the superior cerebellar artery. Acta Neurochir. 1991;109(1–2):66–71.
44. Aydin ME, Kaya AH, Kopuz C, Demir MT, Corumlu U, Dagcinar A. Bilateral origin of superior cerebellar arteries from the posterior cerebral arteries, and clues to its embryologic basis. Anat Cell Biol. 2011;44(2):164–7.
45. Krzyzewski RM, Stachura MK, Stachura AM, Rybus J, Tomaszewski KA, Klimek-Piotrowska W, et al. Variations and morphometric analysis of the proximal segment of the superior cerebellar artery. Neurol Neurochir Pol. 2014;48(4):229–35.
46. Haughton VM, Rosenbaum AE, Pearce J. Internal carotid artery origins of the inferior cerebellar arteries. AJR Am J Roentgenol. 1978;130(6):1191–2.
47. Porzionato A, Macchi V, Massaro L, Morra A, Sarasin G, Rambaldo A, et al. Double origin of the superior cerebellar artery associated with homolateral haemorrhagic infarction of cerebellum. Folia Neuropathol. 2016;54(4):410–7.
48. Weil AG, McLaughlin N, Denis D, Bojanowski MW. Tentorial branch of the superior cerebellar artery. Surg Neurol Int. 2011;2:71.
49. Uchino A, Abe M, Sawada A, Takase Y, Kudo S. Extremely tortuous superior cerebellar artery. Eur Radiol. 2003;13(Suppl 4):L237–8.
50. Scotti G. Anterior inferior cerebellar artery originating from the cavernous portion of the internal carotid artery. Radiology. 1975;116(1):93–4.
51. Teal JS, Rumbaugh CL, Bergeron RT, Scanlan RL, Segall HD. Persistent carotid-superior cerebellar artery anastomosis: a variant of persistent trigeminal artery. Radiology. 1972;103(2):335–41.
52. Lasjaunias P, Manelfe C, Roche A, Rascol A. Segmental aplasia of the basilar artery in man. Report of a case. J Neuroradiol. 1979;6(2):127–36.
53. Hyogo T, Nakagawara J, Nakamura J, Suematsu K. Multiple segmental agenesis of the cerebral arteries: case report. Neuroradiology. 1996;38(5):433–6.
54. Tolabin I, Bertona CA, Bertona JJ, Gribaudo N. Anatomical variations of the circle of Willis in magnetic resonance angiography. Imagenes. 2014;3:17–27.
55. Uchino A. Cerebral arterial variation and anomalies diagnosed by MR angiography. Neurovascular imaging. London: Springer-Verlag; 2010.
56. Luhan JA, Pollock SL. Occlusion of the superior cerebellar artery. Arch Neurol Psychiatr. 1950;63(5):836–7.
57. Mills C. Hemianesthesia to pain and temperature and loss of emotional expression on the right side, with ataxia of the upper limb on the left: the symptoms probably due to a lesion of the thalamus or superior peduncles. J Nerv Ment Dis. 1908;35:331–2.
58. Mills C. Preliminary note on a new symptom complex due to lesion of the cerebellum and cerebello-rubro-thalamic system, the main symptoms being ataxia of the upper and lower extremities of one side, and on the other side deafness, paralysis of emotional expression in the face, and loss of sense of pain, heat and cold over the entire half of the body. J Nerv Ment Dis. 1912;39(2):73–6.
59. Terao S, Sobue G, Izumi M, Miura N, Takeda A, Mitsuma T. Infarction of superior cerebellar artery presenting as cerebellar symptoms. Stroke. 1996;27(9):1679–81.
60. Lin JJ, Lin KL, Chou ML, Wong AM, Wang HS. Cerebellar infarction in the territory of the superior cerebellar artery in children. Pediatr Neurol. 2007;37(6):435–7.
61. Sohn SI, Lee H, Lee SR, Baloh RW. Cerebellar infarction in the territory of the medial branch of the superior cerebellar artery. Neurology. 2006;66(1):115–7.
62. Kumral E, Kisabay A, Atac C. Lesion patterns and etiology of ischemia in superior cerebellar artery territory infarcts. Cerebrovasc Dis. 2005;19(5):283–90.
63. Erdemoglu AK, Duman T. Superior cerebellar artery territory stroke. Acta Neurol Scand. 1998;98(4):283–7.
64. Levine SR, Welch KM. Superior cerebellar artery infarction and vertebral artery dissection. Stroke. 1988;19(11):1431–4.
65. Barinagarrementeria F, Amaya LE, Cantu C. Causes and mechanisms of cerebellar infarction in young patients. Stroke. 1997;28(12):2400–4.
66. Gotoh H, Takahashi T, Shimizu H, Ezura M, Tominaga T. Dissection of the superior cerebellar artery: a report of two cases and review of the literature. J Clin Neurosci. 2004;11(2):196–9.
67. Kalyan-Raman UP, Kowalski RV, Lee RH, Fierer JA. Dissecting aneurysm of superior cerebellar artery. Its association with fibromuscular dysplasia. Arch Neurol. 1983;40(2):120–2.
68. Dandy WE. Concerning the cause of trigeminal neuralgia. Am J Surg. 1934;24:447–55.
69. Jannetta PJ. Arterial compression of the trigeminal nerve at the pons in patients with trigeminal neuralgia. J Neurosurg. 1967;26:159–62.
70. Jannetta PJ. Vascular decompression in trigeminal neuralgia. The cranial nerves: anatomy, pathology, pathophysiology, diagnosis, treatment. New York: Springer-Verlag; 1981. p. 331–40.

71. Rhoton AL Jr. The cerebellopontine angle and posterior fossa cranial nerves by the retrosigmoid approach. Neurosurgery. 2000;47(3 Suppl):S93–129.
72. Picard L, Bracard S, Islak C, Roy D, Moreno A, Marchal JC, et al. Dural fistulae of the tentorium cerebelli. Radioanatomical, clinical and therapeutic considerations. J Neuroradiol. 1990;17(3):161–81.
73. Tomak PR, Cloft HJ, Kaga A, Cawley CM, Dion J, Barrow DL. Evolution of the management of tentorial dural arteriovenous malformations. Neurosurgery. 2003;52(4):750–60. discussion 60–2.
74. Lawton MT, Sanchez-Mejia RO, Pham D, Tan J, Halbach VV. Tentorial dural arteriovenous fistulae: operative strategies and microsurgical results for six types. Neurosurgery. 2008;62(3 Suppl 1):110–24. discussion 24–5.
75. Davies MA, Ter Brugge K, Willinsky R, Wallace MC. The natural history and management of intracranial dural arteriovenous fistulae. Part 2: aggressive lesions. Interv Neuroradiol. 1997;3(4):303–11.
76. Byrne JV, Garcia M. Tentorial dural fistulas: endovascular management and description of the medial dural-tentorial branch of the superior cerebellar artery. AJNR Am J Neuroradiol. 2013;34(9):1798–804.
77. Russel J, Tymianski M. Superior cerebellar artery aneurysms. Decision making in neurovascular disease. Thieme; 2018. p. 277–82.
78. Papo I, Caruselli G, Salvolini U. Aneurysm of the superior cerebellar artery. Surg Neurol. 1977;7(1):15–7.
79. Jin SC, Park ES, Kwon DH, Ahn JS, Kwun BD, Kim CJ, et al. Endovascular and microsurgical treatment of superior cerebellar artery aneurysms. J Cerebrovasc Endovasc Neurosurg. 2012;14(1):29–36.
80. Haw C, Willinsky R, Agid R, TerBrugge K. The endovascular management of superior cerebellar artery aneurysms. Can J Neurol Sci. 2004;31(1):53–7.
81. Peluso JP, van Rooij WJ, Sluzewski M, Beute GN. Superior cerebellar artery aneurysms: incidence, clinical presentation and midterm outcome of endovascular treatment. Neuroradiology. 2007;49(9):747–51.
82. Sanai N, Tarapore P, Lee AC, Lawton MT. The current role of microsurgery for posterior circulation aneurysms: a selective approach in the endovascular era. Neurosurgery. 2008;62(6):1236–49. discussion 49–53.
83. Hzuka H, Miyachi S, Oshima T, Izumi T, Tsurumi A, Yoshida J. Morphological study of aneurysms at the junction of the superior cerebellar artery. Interv Neuroradiol. 2008;14:259–66.

Perforating Branches of the Basilar and Posterior Communicating Arteries

Sara Bonasia and Thomas Robert

The basilar artery (BA) and the posterior communicating artery (PComA) are described in detail in chapter "Embryology and Anatomy of the Posterior Communicating Artery and Basilar Artery." In the present chapter, we will focus on the perforating branches of these two arteries, while the superior cerebellar artery (SCA) and the antero-interior cerebellar arteries will be described in their respective chapter. Although the embryological affinity with the P1 segment of the posterior cerebral artery, the perforating branches of this latter will be described in the chapter dedicated to the posterior cerebral artery.

Embryology

The embryological development of perforators arising from the BA and the PComA is not known in detail. Our embryological knowledge is mostly based on works of Padget and Lasjaunias et al. [1, 2]. The development of these branches relates to that of the PComA and the BA, and consequently to the caudal division of the internal carotid artery (ICA) and to the longitudinal neural arteries. Considering the lack of specific information concerning the embryological development of these perforators, we suggest referring to chapter "Embryology and Anatomy of the Posterior Communicating Artery and Basilar Artery." for detailed information about the development of the PComA and the BA.

Branches of the Basilar Artery

The BA gives off along its course three types of branches:

1. *Cerebellar arteries*: these are the superior and antero-inferior cerebellar arteries (SCA, AICA), which are described in their respective chapter. The postero-inferior cerebellar artery (PICA) can arise from the BA in 25% of cases, while normally arises from the vertebral artery [3].
2. *Large pontine vessels*: these vessels arise directly from the BA or from one of its collaterals. They comprise the pontomedullary artery, the posterolateral artery, and the long lateral pontine arteries. The pontomedullary artery most often originated from the proximal part of the BA and terminated in the retro-olivary fossa of the medulla. The posterolateral artery arises from the distal part of the BA, just caudal to the SCA. Finally, the long pontine arteries arise from the distal half of the BA and terminated close to the trigeminal nerve. Commonly, two of them are present on each side of the brainstem: the superolateral and the inferolateral long pontine arteries [3, 4].

   Although these vessels are quite always present, their origin and their vascular territory is very variable and depend on their possible direct origin from the BA, from the BA major branches (SCA, AICA) or directly from the perforators.
3. *Perforating branches*: these branches are usually tiny vessels that penetrate the brainstem and will be described in the following paragraph.

Perforating Branches of the Basilar Artery

The perforating branches of the BA arise from the posterior (50%) and posterolateral wall of the artery (25% from each side) [5, 6]. No branches arise from the anterior surface of the BA [3, 5]. Their average number is 10 (range 3–17), with a diameter between 0.08–0.94 mm (average 0.6 mm) [3, 5].

These branches have been classified according to different criteria:

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1. *Entry point into the brainstem*:
   (a) *Median or paramedian arteries*: they are numerous tiny branches that originate from the posterior aspect of the BA [4, 5, 7–10]. They enter the brainstem close to the midline, along the anterior median fissure of the medulla and the basilar sulcus of the pons [11]. They have a median length of 6 mm (range 4.5–7.05 mm) and have a high concentration around the foramen caecum near the pontomedullary junction [9–11].
   (b) *Lateral, transverse, or circumferential arteries*: they are less numerous but larger branches, usually symmetric, that originate from the posterolateral aspect of the BA [4, 5, 7–9, 12]. They enter the lateral pons, cerebellar peduncles, and posterior perforated substance. Their median length is estimated as 17 mm (range 14.7–17.6 mm) [10].
2. *Point of origin from the BA*:
   (a) *Caudal group*: perforators arise from the proximal part of the BA, between the junctional site of the vertebral arteries and the origin sites of the AICA. These branches range in number from 2–5 and in diameter from 0.08 to 0.6 mm [3]. However they can be absent on one side (8.3% of cases); in this case, a large common trunk of perforators was noted on the opposite side [3]. These branches mostly contribute to the vascularization of the area of the foramen cecum at the junctional point of the pontomedullary sulcus and the anterior median sulcus [3, 11]. The caudal perforators can also give rise to collateral branches in 58.3% of cases [3]. Most commonly they give off the pontomedullary artery, and rarer the posterolateral branches; Marinkovic et al. also described the possible origin from these perforators of three kinds of branches over the ventral surface of the medulla: the pyramidal branches, the branches to the rostral part of the anterior median sulcus of the medulla and large branches to the hypoglossal nerve [3]. Mahmood et al. described for the caudal group four branching patterns: type A was the most common and corresponds to a single trunk dividing into many branches; in type B, C and D, there were respectively two, three and four trunks arising from the BA [11]. The branching pattern seems also to be correlated to the location of the vertebro-basilar junction. In fact, when the junction was above the foramen caecum, the predominant pattern was the type A. On the other side, in case of junction below the foramen caecum, there was a predominance of types D, B or C in order of frequency [11].
   (b) *Middle group*: perforators arise from the middle part of the BA, between the origin of the AICA and the posterolateral artery [3]. These branches are relatively constant, in a number between 5 and 9 and with a diameter of 0.2–0.9 mm [3]. The perforators can origin as common trunks or as individual vessels. In both cases, their branching pattern can be summarized into two types according to Mahmood et al.: in type A, there are two trunks that descend along the basilar sulcus on both sides; in type B, there is only one trunk that descend posterior or lateral to the basilar artery [11]. These perforators can also arise from other branches of the BA. They can arise from the AICA in 16.6%, from the pontomedullary artery in 16.6% of cases, and in 25% of cases from a long pontine artery [3]. It was also observed a possible common origin with the posterolateral artery or with a perforator from the caudal group [3]. On the other side, also the other collateral branches can arise from the perforators of the middle group. In detail, the long pontine arteries originated from the perforators in 25% of the hemispheres in the study of Marinkovic et al. [3]. On the other hand, one or more perforators gave rise to the anterolateral branches in many of the cases examined, where these latter gave off branches to the abducens nerve. The terminal branches of the middle perforators penetrated the edges of the basilar sulcus. They divide into the long and short intrapontine branches, which course close to the raphe of the pons.
   (c) *Rostral group*: they arise from the distal part of the BA, included between the posterolateral artery and the BA bifurcation, and enter the interpeduncular fossa. They range in number from one to five (mean 3) and have an average diameter of 0.25 mm [3, 5, 13, 14]. Like in the other groups, they can arise as individual vessels, as common stems, or from other collateral arteries (from the SCA or from the posterolateral artery). The higher concentration of these branches has been identified distal to the SCA and in proximity of the BA tip [3, 14]. These perforators enter the most caudal part of the interpeduncular fossa, just caudal to the penetration sites of the mesencephalic perforators of the posterior cerebral artery [3].

The configuration of the perforators of the BA is illustrated in Fig. 1.

Anastomoses Among Perforators

The three groups of perforators tend to form an intricate network of vessels along the anterior surface of the brainstem. These anastomoses among perforators allow a precise and dense vascularization of these high functional areas.

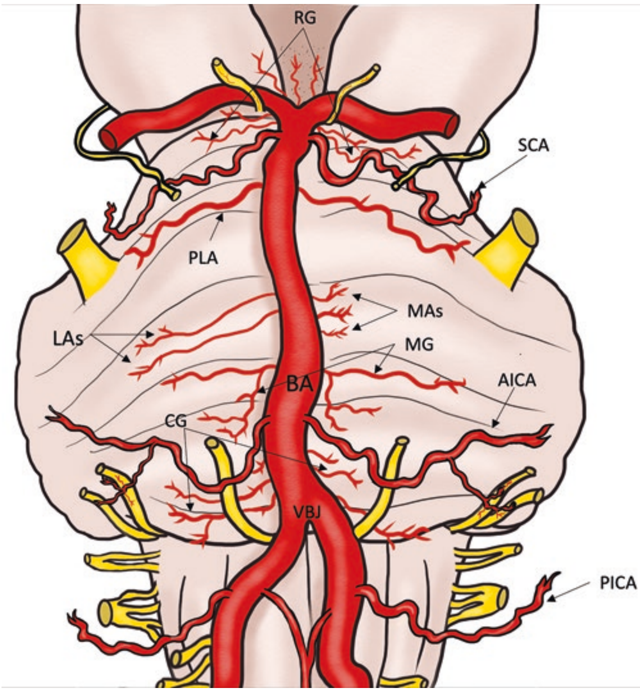

Fig. 1 Illustrative classification of the basilar artery's perforators. *BA* basilar artery, *VBJ* vertebro-basilar junction, *SCA* superior cerebellar artery, *AICA* anterior inferior cerebellar artery, *PICA* posterior inferior cerebellar artery, *PLA* posterolateral artery, *LAs* lateral arteries, *Mas* medial arteries, *RG* rostral group, *MG* middle group, *CG* caudal group

The anastomoses among the perforators of the BA themselves and/or with branches of the vertebral artery can be divided into the following:

- Major anastomoses, when the main trunks from the vertebro-basilar system join to form an arch and perforators arise from this arch.
- Minor anastomoses, when the anastomoses are between perforators themselves [11].

To simplify the comprehension of these complex networks, we will describe in the next paragraphs the anastomoses of each group of perforators.

Caudal Group

This group of perforators forms anastomoses most often with the middle group (in 33.3% of cases according to Marinkovic et al.) [3]. Rarer are the connections between the caudal perforators and perforators from the vertebral artery and anterior spinal arteries (16.6% of cases) [3]. The anastomoses were also noted among the caudal perforators themselves (8.3% of cases) [3].

Middle Group

According to Marinkovic et al., this group of perforators forms anastomoses more frequently (in 66.6% of cases), but in lower number [3]. They were most often found between the middle and caudal perforating arteries (33.3%), while anastomoses between middle perforators themselves were seen in 25% of the hemispheres. Vascular connections between the rostral and middle perforators were rarely identified (16.6%) [3].

Rostral Group

This group of perforators form anastomoses in 41.6% of brains according to Marinkovic et al. [3]. Most of them are unilateral, but right-left anastomoses are also seen occasionally. The anastomoses interconnect the rostral perforators themselves, and with the interpeduncular branches of the posterior cerebral artery, and finally the rostral perforators and the main stem of the SCA [3].

Vascular Supply

There is generally a lack of precise data concerning the exact vascular supply from the perforators of the BA, especially in relation to their high anatomical variability. However, in this paragraph we describe the most common vascular territory of each group of perforators.

The caudal perforators entering at the foramen caecum as median branches penetrate the brainstem to reach the tegmentum and supply the cortico-spinal and cortico-bulbar tracts, the pontine nuclei, the medial longitudinal fasciculus, the medial lemniscus, the abducens nucleus and the cranial end of the hypoglossal nucleus [10, 11, 15]. Those that penetrate the brainstem more laterally supply the lateral part of the pons, the trigeminal and facial nuclei, the middle cerebellar peduncles and the lateral parts of the cortico-spinal and cortico-bulbar tracts, as well as the medial lemniscus [10, 11].

The medial group of perforators is usually in balance with the other two groups. In fact, the most caudal branches of this group descend along the basilar sulcus, while the most cranial ones ascend forming anastomoses with the other two groups. They mostly supply the paramedian pontine reticular formation and the cranial and caudal portion of the structures supplied by the other two groups [3, 10].

The rostral group of perforators is composed by branches that enter the interpeduncular fossa. They usually enter the postero-inferior region of the posterior perforated substance and can give off branches to the cisternal part of the oculomotor nerve and to the medial surface of the cerebral peduncles [3, 13, 16, 17].

Branches of the Posterior Communicating Artery

Along its course, the PComA gives off an average of seven branches (range 4–12), with a diameter from 0.1 to 0.6 mm. These branches arise from the superior and lateral aspects of the PComA, even if rarely some perforators have been identified emerging from its medial side [5, 18]. Moreover, the number of perforators origin from the PComA does not depend on the PComA size [5, 6].

According to the perforators' origin, the PComA can be divided into three segments. The perforators will be consequently classified into the following [5]:

- *Anterior group*: branches arise from this anterior part of the PcomA in 54% of cases. These branches usually supply the hypothalamus, the ventral thalamus, the anterior third of the optic tract and the posterior limb of the internal capsule.
- *Middle group*: perforators origin from the middle third of the PComA in 21% of cases.
- *Posterior group*: the perforators origin is prevalent from this part of the artery in 25% of cases. This group supplies the posterior perforated substance and the subthalamic nucleus.

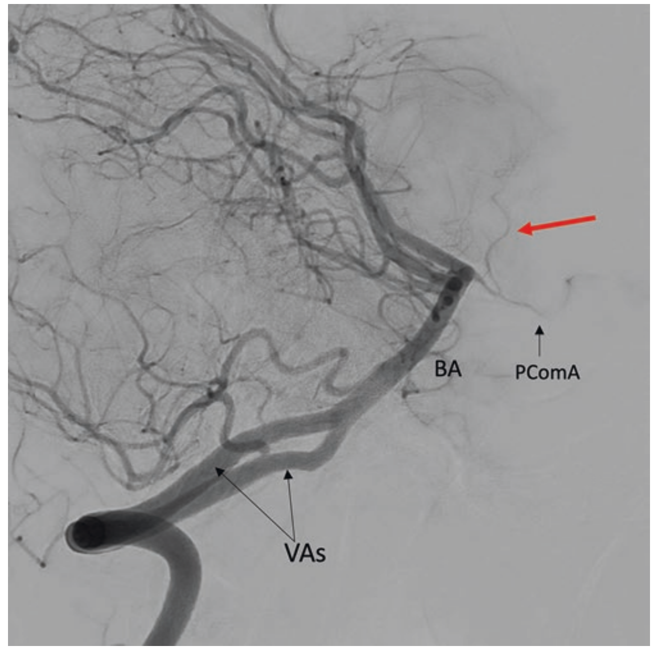

Fig. 2 DSA showing the premamillary artery. Lateral angiogram of the vertebral artery (VA) showing the posterior communicating artery (PComA) giving birth to a prominent premamillary artery (red arrow). Basilar artery (BA)

The Premamillary Artery

Independently from the site of origin from the PComA, its largest branch ends in 80% of cases in the so called premamillary area, between the mammillary bodies and the optic tract. This artery was thus called premamillary artery [5, 19]. Other denominations of this artery, that will not be used in this chapter, are anterior thalamo-perforating artery, thalamo-tuberal artery or thalamic-polar artery [5, 20]. The anatomical area supplied by the premamillary artery was later more precisely described by Percheron et al. [20] and defined as the paramedian perforated substance [20, 21]. This latter is a triangle-shaped area, limited by the mamillary body and tuber cinereum medially, by the optic tract anterolaterally and by the cerebral peduncle posterolaterally [21, 22]. The premamillary artery is a constant branch of the PComA, even if Pedroza et al. described two cases of its origin from the posterior cerebral artery (PCA) and Gibo et al. reported its possible origin at the PComA-PCA junction [21, 23]. This artery arises from the anterior part of the PComA in 8–17% of cases, from the posterior part in 8–13% of cases, and from the middle part of the PComA in 35–50% of cases [5, 21]. Its average diameter is 0.6 mm, with a median length of 13 mm [21].

Even if the premamillary artery is usually a single branch, it can also be represented by two or three branches, without the presence of a dominant artery in 20% of cases [5, 21, 23]. In this case, this arterial group is called "premamillary arterial complex" [5]. In their detailed anatomical study, Pedroza et al. identified in 63% of cases multiple branches arising from the premamillary artery and directed to the posterior perforated substance and to the cerebral peduncles [21, 23]. These branches vary in number from 1 to 9 and in diameter from 0.09 to 0.4 mm [23]. In 37% of cases, no branches arise from this artery [23]. The premamillary artery and its branches form in 35% of cases an anastomotic network with other adjacent perforator vessels [23]. The vascular territory supplied by this artery is represented by the cerebral peduncles (ventro-medial part of the rostral crus cerebri), the ventro-medial part of the optic tracts, the paramedian perforated substance, the lateral and anterior portion of the thalamus and the lateral and anterior portion of the hypothalamus [5, 18, 21, 23]. A clinical case showing the premamillary artery is presented in Fig. 2.

Clinical Implications

The knowledge of the perforators arising from the BA and the PComA represents a mandatory skill for neurologists, neurosurgeons and neuroradiologists. These tiny branches are in fact implicated in many ischemic syndromes that involve the brainstem. Their clinical importance has also acquired even more importance with the development of endovascular techniques for the treatment of aneurysms of the posterior circulation. Here we resume the most important pathologies in which the perforators arising from the BA and PComA are involved.

Ischemic Stroke of the Basilar Artery

The clinical importance of the BA becomes even more evident in case of its ischemic stroke. The origin of the stroke is usually different depending on the portion of BA involved and is usually thrombotic in its proximal third, and embolic in its distal portion [10, 11]. The symptoms and signs of BA occlusion vary from confusional status to coma, associated with dysarthria, ocular palsy, hemi or quadriplegia. In case of perforators occlusion and consequent brain stem lesions, the symptoms developed are usually included in the so-called alternating syndromes [10, 15]. The same symptoms and syndromes can occur in case of iatrogenic occlusion of perforating vessels while approaching aneurysms arising from the basilar artery.

Aneurysms of the Basilar Artery

The vertebro-basilar aneurysms represent between 2.5 and 15% of all intracranial aneurysms [9, 24, 25]. Among them, aneurysms arising from the BA represent 59–77% of the total with a predominant origin at its bifurcation. The localization of perforators and their site of origin from the BA must be carefully considered in the choice of treatment for these aneurysms. For BA bifurcation aneurysms, neurosurgeons and neuroradiologists should consider their prevalent origin from the posterior and lateral wall of the artery. The perforators of the middle and caudal group should be considered in case of endovascular treatment of saccular and/or fusiform aneurysms involving the basilar trunk, considering that one of the most frequent complications described after stenting or coiling of these aneurysms consist in ischemic stroke in the perforator's vascular territory.

Aneurysms of the Perforators of the Basilar Artery

Aneurysms arising from a perforator branch represent a minor part of the intracranial aneurysms and have a general prevalence <1% with fewer than 70 cases reported in literature. Among them, about 50 cases concern perforating artery aneurysm of the anterior circulation, and only 19 cases of perforating artery's aneurysm of the BA have been reported [26, 27]. The first case of BA perforator's aneurysm was described by Ghogawala et al. in 1996 [28]. After its initial report, other 18 cases were reported; all of them were cases of ruptured aneurysms, and mostly arising from the middle and distal portion of the BA [29–31]. These aneurysms are rare entity and represent a real diagnostic and treatment challenge. In fact, they can be initially angiographically occult because of their small size and the possible formation of thrombi within the aneurysmal sac [26]. Moreover, their treatment deserves some difficulties because of their location, that makes the surgical approach challenging, and because of the possible catastrophic clinical consequence in case of perforators occlusion after stent treatment [26, 28, 29].

Aneurysms of the PComA

Perforators arising from the PComA should be considered while approaching aneurysms of the PComA. Particular attention should be paid to the premamillary artery. Although the clinical consequences of premamillary artery occlusion are still matter of debate, a clinical syndrome characterized by memory loss, personality changes, loss of endocrine function, anorexia and disorders of temperature regulation and consciousness was described by Hayman et al. [20, 32].

References

1. Padget DH. The development of cranial arteries in the human embryo, vol. 212; 1948. p. 205–62.
2. Lasjaunias P, Berenstein A, Ter Brugge KG. Surgical Neuroangiography. Berlin: Springer Edition; 2001.
3. Marinkovic SV, Gibo H. The surgical anatomy of the perforating branches of the basilar artery. Neurosurgery. 1993;33(1):80–7. https://doi.org/10.1227/00006123-199307000-00012.
4. Kaplan HA, Rabiner AM, Browder J. Anatomical study of the arteries of the midbrain, pons and medulla. Trans Am Neurol Assoc. 1953;3(78th Meeting):54–5. discussion, 5–6.
5. Saeki N, Rhoton AL Jr. Microsurgical anatomy of the upper basilar artery and the posterior circle of Willis. J Neurosurg. 1977;46(5):563–78. https://doi.org/10.3171/jns.1977.46.5.0563.
6. Rhoton AL Jr. Aneurysms. Neurosurgery. 2002;51(4 Suppl):S121–58.
7. Gabrielsen TO, Amundsen P. The pontine arteries in vertebral angiography. Am J Roentgenol Radium Ther Nucl Med. 1969;106(2):296–302. https://doi.org/10.2214/ajr.106.2.296.
8. Stopford JS. The arteries of the pons and medulla oblongata. J Anat Physiol. 1916;50(Pt 2):131–64.
9. Yasargil MG. Microneurosurgery. Thieme; 1984.
10. Torche M, Mahmood A, Araujo R, Dujovny M, Dragovic L, Ausman JI. Microsurgical anatomy of the lower basilar artery. Neurol Res. 1992;14(3):259–62. https://doi.org/10.1080/01616412.1992.11740066.
11. Mahmood A, Dujovny M, Torche M, Dragovic L, Ausman JI. Microvascular anatomy of foramen caecum medullae oblongatae. J Neurosurg. 1991;75(2):299–304. https://doi.org/10.3171/jns.1991.75.2.0299.
12. Hassler O. Arterial pattern of human brainstem. Normal appearance and deformation in expanding supratentorial conditions. Neurology. 1967;17(4):368–75 passim. https://doi.org/10.1212/wnl.17.4.368.
13. Brassier G, et al. The posterior part of the arterial circle of the skull base. Caudal division of the embryonic internal carotid artery. Rivista di Neurochirurgia. 1994;7:85.

14. Grand W, Hopkins LN. The microsurgical anatomy of the basilar artery bifurcation. Neurosurgery. 1977;1(2):128–31. https://doi.org/10.1227/00006123-197709000-00008.
15. Gillilan LA. The correlation of the blood supply to the human brain stem with clinical brain stem lesions. J Neuropathol Exp Neurol. 1964;23:78–108. https://doi.org/10.1097/00005072-196401000-00006.
16. Brassier G, Morandi X, Riffaud L, Mercier P. Basilar artery anatomy. J Neurosurg. 2000;93(2):368–9. https://doi.org/10.3171/jns.2000.93.2.0368.
17. Hendrix P, Griessenauer CJ, Foreman P, Shoja MM, Loukas M, Tubbs RS. Arterial supply of the upper cranial nerves: a comprehensive review. Clin Anat. 2014;27(8):1159–66. https://doi.org/10.1002/ca.22415.
18. Vincentelli F, Caruso G, Grisoli F, Rabehanta P, Andriamamonjy C, Gouaze A. Microsurgical anatomy of the cisternal course of the perforating branches of the posterior communicating artery. Neurosurgery. 1990;26(5):824–31. https://doi.org/10.1097/00006123-199005000-00015.
19. Foix C. Les arteres de l'axe encephalique jusqu'au diencephale inclusivement. Rev Neurol. 1925;2:160–79.
20. Percheron G. Arteries of the human thalamus. II. Arteries and paramedian thalamic territory of the communicating basilar artery. Rev Neurol (Paris). 1976;132(5):309–24.
21. Pedroza A, Dujovny M, Cabezudo-Artero J, Umansky F, Berman SK, Diaz FG, et al. Microanatomy of the premamillary artery. Acta Neurochir. 1987;86(1–2):50–5. https://doi.org/10.1007/BF01419504.
22. Pedroza A, Dujovny M, Artero JC, Umansky F, Berman SK, Diaz FG, et al. Microanatomy of the posterior communicating artery. Neurosurgery. 1987;20(2):228–35. https://doi.org/10.1227/00006123-198702000-00005.
23. Gibo H, Marinkovic S, Brigante L. The microsurgical anatomy of the premamillary artery. J Clin Neurosci. 2001;8(3):256–60. https://doi.org/10.1054/jocn.2000.0822.
24. Drake CG. Further experience with surgical treatment of aneurysm of the basilar artery. J Neurosurg. 1968;29(4):372–92. https://doi.org/10.3171/jns.1968.29.4.0372.
25. Drake CG. Surgical treatment of ruptured aneurysms of the basilar artery. Experience with 14 cases. J Neurosurg. 1965;23(5):457–73. https://doi.org/10.3171/jns.1965.23.5.0457.
26. Satti SR, Vance AZ, Fowler D, Farmah AV, Sivapatham T. Basilar artery perforator aneurysms (BAPAs): review of the literature and classification. J Neurointerv Surg. 2017;9(7):669–73. https://doi.org/10.1136/neurintsurg-2016-012407.
27. Vargas J, Walsh K, Turner R, Chaudry I, Turk A, Spiotta A. Lenticulostriate aneurysms: a case series and review of the literature. J Neurointerv Surg. 2015;7(3):194–201. https://doi.org/10.1136/neurintsurg-2013-010969.
28. Ghogawala Z, Shumacher JM, Ogilvy CS. Distal basilar perforator artery aneurysm: case report. Neurosurgery. 1996;39(2):393–6. https://doi.org/10.1097/00006123-199608000-00034.
29. Gross BA, Puri AS, Du R. Basilar trunk perforator artery aneurysms. Case report and literature review. Neurosurg Rev. 2013;36(1):163–8; discussion 8. https://doi.org/10.1007/s10143-012-0422-1.
30. Nyberg EM, Chaudry MI, Turk AS, Spiotta AM, Fiorella D, Turner RD. Report of two cases of a rare cause of subarachnoid hemorrhage including unusual presentation and an emerging and effective treatment option. J Neurointerv Surg. 2013;5(5):e30. https://doi.org/10.1136/neurintsurg-2012-010387.
31. Ding D, Starke RM, Jensen ME, Evans AJ, Kassell NF, Liu KC. Perforator aneurysms of the posterior circulation: case series and review of the literature. J Neurointerv Surg. 2013;5(6):546–51. https://doi.org/10.1136/neurintsurg-2012-010557.
32. Hayman LA, Berman SA, Hinck VC. Correlation of CT cerebral vascular territories with function: II. Posterior cerebral artery. AJR Am J Roentgenol. 1981;137(1):13–9. https://doi.org/10.2214/ajr.137.1.13.

Part III

Arterial System of the Dura and Supply of the Cranial Nerves

Anatomy, Embryology and Variations of the Middle Meningeal Artery

Thomas Robert, Sara Bonasia, S. Smajda, G. Ciccio', and Michel W. Bojanowski

The middle meningeal artery (MMA) is one of the largest branches of the external carotid artery (ECA) and the most important dural artery, since it supplies more than two-thirds of the cranial dura [1]. However, the most interesting aspects of the MMA are not its size or its clinical importance, but its embryological development and numerous anatomical variations. The understanding of the anatomy and variants of the MMA imposes a good knowledge of the hyostapedial system and of the vascular anatomy of the middle ear. In this chapter, we will summarize the most important steps of the MMA embryological development and will explain the possible anatomical variations of the MMA. The knowledge of these variants is important especially for neuroradiologists to treat dural pathologies as well as for surgeons that approach the middle ear pathologies.

History

First anatomists interested about the MMA and about the persistence of the stapedial artery (SA) have published the results of their dissection during the first half of the nineteenth century. The first cadaveric case of persistent stapedial artery was described by Hyrtl in 1836 [2]. After him, a lot of authors published results of comparative anatomy with animals that bear a stapedial artery and this literature helps a lot in the understanding of vascular variations in humans. Successive isolated cases of persistent stapedial artery are described by ENT surgeons that found this artery passing through the window of the stapes during middle ear surgeries. In the first half of the twentieth century, the phenomenal publication of Dorcas Padget, based on the dissections of 22 human embryos of the Carnegie collection, gave lot of information about the embryological development of the cranio-facial arteries and in particular of the hyostapedial system [3]. In the same period, brilliant authors as Altmann (1947) paid a particular attention to furnish a comprehensive explanation in the development of the embryologic aortic arches and in variations of the carotid system [4]. However, only in the 1960s, we find the first publication etc. of angiographic demonstration of MMA variations and in particular, the persistence of a stapedial artery [5, 6]. Pioneers of the interventional neuroradiology also furnished a fantastic work in the understanding of all anatomical variations of the cranio-facial arteries [6, 7]. At the end of the 1970s, J. Moret published a series of articles, based on its medical thesis, describing angiographic anatomy and branches of the MMA with a particular attention to explain variants of them [8–10]. The other important contributor in the understanding of all anatomical variations of cranio-facial arteries is P. Lasjaunias who described almost all variants concerning the MMA and was able to give a comprehensive explanation of all variations implicating the stapedial artery. Its more famous articles were published between 1975 and 1990 and are summarized in its textbook [8–15]. Diamond in the 1990s also published a series of articles based on the comparative anatomy principally with the great apes that increase our understanding in the development of the MMA and the stapedial system [16–18]. In this last three decades, few case reports of interesting ana-

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tomical variations are published but the most important contribution was given by Martins et al. who published in 2005 a review about all dural arteries illustrated by high quality cadaveric photographs [1].

Embryology

It is necessary to know the embryology of the hyostapedial system to understand the embryological development of the middle meningeal artery but, also the knowledge of the embryology of the ophthalmic artery (OA) and carotid system are cornerstones in the explanation of all variations of the MMA. In this chapter, we will successively summarize the embryology of the carotid system, the hyostapedial system, the trigeminal artery (TA) and the inferolateral trunk (ILT). The embryology of the ophthalmic artery is widely treated in chapter “Embryology and Variations of the Ophthalmic Artery.”

The Carotid System

Precedent chapters concerning the development and variations of the internal carotid artery (ICA) describe more in detail the embryology of the carotid system. In the present chapter, you will find only crucial information to understand the formation of an “aberrant flow of the carotid artery” which consists in an intratympanic course of the pseudo-ICA and that could be associated with a variation in origin of the MMA.

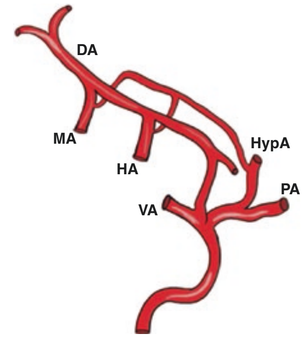
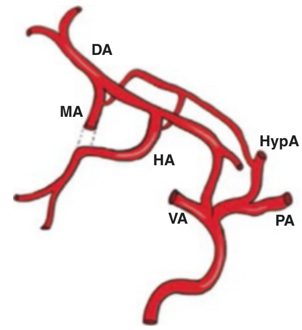
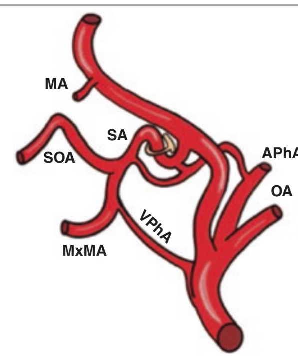
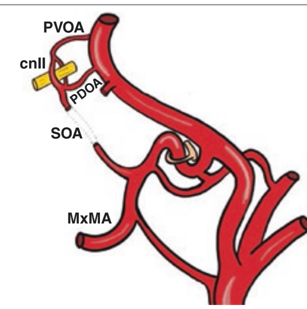
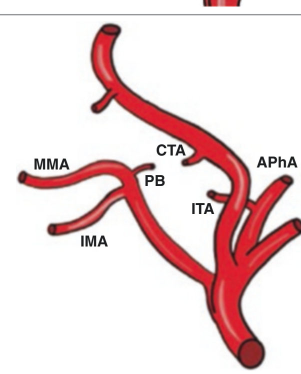
Early in the development (first stage of Padget, embryos of 4–5 mm), the two first aortic arches initiate their natural regression allowing the internal carotid artery to be individualized. The embryological segments of the ICA are derived from the third aortic arch and from the dorsal aorta cranial to the third aortic arch [3, 4, 15]. The dorsal aorta also regresses at the same time between the third and fourth aortic arches. Embryologically, we could divide the internal carotid artery in seven different segments. The first one corresponds to the third aortic arch from the origin of the ventral pharyngeal artery (future external carotid artery) to the junction between the third aortic arch and the dorsal aorta. The second segment is the dorsal aorta between the second and third aortic arches. The third segment is the dorsal aorta between the first and second aortic arches. The fourth segment is the dorsal aorta between the origin of the trigeminal artery (and the primitive maxillary artery) and the first aortic arch. The fifth segment is the dorsal aorta between the origin of the primitive dorsal ophthalmic artery (PDOA, future inferolateral trunk (ILT)) and the origin of the trigeminal artery (and the primitive maxillary artery). The sixth segment is between the origin of the primitive ventral ophthalmic artery (PVOA) and the origin of the primitive dorsal ophthalmic artery (PDOA, future inferolateral trunk (ILT)). The seventh segment is between the PVOA and the primitive carotid bifurcation [3, 12, 13, 15]. It is important to note that the carotid bulb has not the same embryological origin than the other segments of the ICA. It originates from the pharyngo-occipital system that easily explains variations in origin of the ascending pharyngeal and occipital arteries. Agenesis or abnormal regression of one or more segments of the carotid artery explains an intratympanic course of the ICA (by pharyngo-carotid anastomosis) and also the different type of “reperfusion” in case of ICA agenesis [4, 19]. All anatomical variations in the course of the ICA are described in detail in chapter “Segmental Agenesis of the Internal Carotid Artery.” The intratympanic course of the ICA also named as “aberrant flow of the ICA” is the consequence of the abnormal regression of the first and second segments with anastomosis between the inferior tympanic artery (from the ascending pharyngeal artery) and the caroticotympanic artery (from the carotid artery) that infuses distally the carotid artery. The pseudo-ICA has consequently an intratympanic course without passing through the stapes [15, 19, 20].

The Hyostapedial System

“Hyostapedial artery” is the term used to describe a part or the complete embryological development that derivate from the second aortic arch. The stapedial artery, that develops from the hyoid artery and takes its name after passing through the stapes, is an embryological artery presented between stages III and VI of Padget (9 mm–24 mm). This is an important embryological system from which numerous dural, orbital and facial arteries develop. All steps of the stapedial artery development are summarized in Table 1.

The hyoid artery is the dorsal remnant of the second aortic arch which regresses early in the embryological development (4–5 mm, stage I of Padget) [19, 21]. After this arch regression, approximately at the 9 mm stage (stage III of Padget), the hyoid artery presents a rapid lateral elongation between the origins of the seventh and nineteenth weeks of gestation and gives an anastomosis to the mandibular artery (remnant of the first aortic arch) [3]. At the 16 mm stage (stage V of Padget), the stapedial artery (SA) continues its elongation to the gasserian region passing into the future tympanic cavity and particularly through the crus of the stapes reaching its maximal development and giving two distinct branches [3, 4, 12]. The supraorbital artery follows the ophthalmic root of the trigeminal nerve and allows the development of orbital branches (supraorbital, lacrimal, ethmoids and frontal arteries) and also of the middle meningeal artery [15]. The maxillomandibular artery gets out of the cranial cavity through the foramen spinosum and gives extracrani-

Table 1 Major embryological changes in the formation of the stapedial artery

Stage	Embryo size (mm)	Major evolutions	Graphic illustration
I	4–5	• Regression of the ventral part of the first and second aortic arches • Hyoid artery (HA) formation (dorsal remnant of the second aortic arch)	
II	5–6	• Elongation of the hyoid artery (HA) • Annexation of the mandibular artery (MA) territory (first aortic arch) by the hyoid artery (second aortic arch)	
III/ IV	7–12 12–14	• Cranial growing of the hyoid artery (stapedial artery) passing into the middle ear (crus of the stapes) • Extension of the two branches of the stapedial artery: Supraorbital (SOA) and Maxillomandibular (MxMA)	
V	16–18	• Maximal development of the stapedial artery • Annexation of the supraorbital branch (SOA) by the ophthalmic artery (PDOA, PVOA) • Regression of the trans-osseous (superior orbital fissure) segment of the supraorbital branch	
VI	20–24	• Annexation of the maxillomandibular branch by the ventral pharyngeal artery • Regression of the intratympanic segment of the stapedial artery	

ally its two branches: infraorbital and mandibular artery (future infraorbital and inferior alveolar arteries). After this maximal development of the SA, two annexations and two regressions occur to give the adult configuration of the MMA. Intracranially, the orbital branches are annexed by the primitive ophthalmic artery and the trans-sphenoidal segment of the supraorbital branch regresses leaving an anastomotic artery between the anterior branch of the MMA and the lacrimal artery (the sphenoidal artery) that penetrates the orbit through the superior orbital fissure. Extracranially, the ventral pharyngeal artery annexes the maxillomandibular artery of the SA forming the proximal stem of the MMA and becoming the external carotid artery. Consequently, by flow reversal into the SA, its tympanic portion regresses and leaves as remnants the carotico-tympanic artery (from the ICA) and the superior tympanic artery (from the petrous branch of the MMA). These annexations and regressions concerning the SA happen during the stage VI of Padget (embryos of 24 mm) [3, 15].

The Trigeminal Artery

The trigeminal artery (TA) is one of the carotid-basilar anastomoses and is an embryological artery. It appears at the 4–5 mm embryos (stage I of Padget) and disappears at the 12 mm embryos (stage III of Padget). Its origin is on the basilar artery (BA) between the superior and the anterior inferior cerebellar arteries, passes medial to the gasserian ganglion and follows the trigeminal nerves to the primitive carotid artery (junction of the fourth and fifth segments of the carotid artery). The TA has a common origin on the carotid artery with the primitive maxillary artery. The carotid remnant of these two primitive arteries will be the future meningo-hypophyseal trunk (lateral clival, marginal tentorial and inferior hypophyseal arteries) [3, 4].

Formation of the Inferolateral Trunk

As supported by the theory of P. Lasjaunias concerning the embryology of the ophthalmic artery, the inferolateral trunk (ILT) is the carotid remnant of the primitive dorsal ophthalmic artery (PDOA) [12, 13]. The PDOA develops from the cavernous segment of the primitive internal carotid artery and penetrates the orbit through the superior orbital fissure. In the 40 mm embryos, the proximal part of the PDOA regresses and its remnant becomes the inferolateral trunk of the primitive carotid artery. At the adult configuration, the ILT is composed of four branches: (1) superior branch that supply the roof of the cavernous sinus, (2) anteromedial branch which passes into the superior orbital fissure, (3) anterolateral branch that runs into the foramen rotundum and (4) posterior branch passing medial to the gasserian ganglion. These branches present a lot of anastomosis in the cavernous region that correspond to remnants of primitive trigeminal, stapedial and maxillary arteries [15].

Origin of the Artery

In almost all cases, the MMA arises from the internal maxillary artery (IMA), but it could also take its origin from the ICA or more surprisingly from the basilar artery (BA). Possible origins of the MMA are listed in Table 2 with their respective embryological explanation.

Internal Maxillary Artery Origin

The normal origin of the MMA is usually described on the first segment of the internal maxillary artery (IMA) into the infratemporal fossa, just behind the condylar process of the mandible [1, 9]. The MMA is the largest ascending branch of the IMA [9]. Usually, the MMA is the first ascending branch of the IMA but could also have a common trunk with the accessory meningeal artery (AccMA); it depends on the position of the IMA course regarding the external pterygoid muscle [15]. When the IMA passes superficially to the muscle, the MMA and AccMA have a common origin from the IMA and the inferior dental and posterior deep temporal arteries have a separate origin. On the contrary, when the IMA passes deep to the external pterygoid muscle, the MMA and AccMA have distinct origins from the IMA and the inferior dental and the posterior deep temporal arteries share a common trunk [9].

Low (1946) described an interesting cadaveric case of distal (third segment) IMA origin of the MMA [22]. In his study, he inspected the osseous grooves of the skull and noted that, associated to the absence of the foramen spinosum, the osseous groove of the MMA converges to the superior orbital fissure. He concluded that the MMA takes its origin in the pterygoid fossa from the distal IMA and passes through the inferior and superior orbital fissures. Probably, in his description, he misinterpreted an ophthalmic artery (OA) origin of the MMA, not already know upon its publication [15].

Basilar Artery Origin

Altmann (1947) was the first author to describe a case of basilar artery (BA) origin of the MMA in its monumental paper about anomalies of the carotid system but failed to give clear embryological explanation of the anatomical variation [4]. Surprisingly, he described the origin of the artery between the anterior inferior and posterior inferior cerebel-

Table 2 Different origin of the MMA with modifications associated and embryological explanation

Variations in origin of the MMA		Embryological implications	
Type	Associated changes	Embryological explanation	Embryos size (mm)
IMA origin	Normal anatomy	Normal embryology	
Basilar artery origin	Absence foramen spinosum	• Anastomosis between SA and trigeminal artery • Anastomosis between SA and lateral pontine artery	12
Cavernous ICA origin	Absence foramen spinosum	• Anastomosis between ILT and SA	16
Partial persistent SA	Absence foramen spinosum Enlargement of the facial canal	• Regression of the proximal part of the maxillomandibular branch • Persistence of the intratympanic segment of the SA	24
Complete persistent SA	Enlargement of the facial canal	• Lack of annexation of the maxillomandibular branch by the ventral pharyngeal artery • Persistence of the intratympanic segment of the SA	24
Pseudo-petrous ICA origin	Absence foramen spinosum Enlargement of the facial canal Absence of the exocranial opening of the carotid canal	• Agenesis of the first and second segments of the ICA • Intratympanic anastomosis between inferior tympanic and caroticotympanic arteries • Persistence of the intratympanic segment of the SA	4–5 24
Cervical ICA origin	Absence foramen spinosum Enlargement of the facial canal	• Intratympanic anastomosis between inferior and superior tympanic arteries • Regression of the proximal part of the maxillomandibular branch • Persistence of the intratympanic segment of the SA	16 24
Occipital artery origin	Absence foramen spinosum Enlargement of the facial canal	No clear explanation	
Distal petrous ICA origin	Absence foramen spinosum	• Lack of annexation of the mandibular artery (first aortic arch) by the SA (second aortic arch)	9

lar arteries (AICA and PICA) and its course as "accompanying the acoustic-facial nerve" passing through the internal acoustic canal to reach the superior branch of the SA. After this initial description, less than ten cases of BA origin of the MMA were successively published [11, 12, 23–27]. Usually, the MMA takes its origin from the distal third of the BA between the SCA and the AICA. It courses anteriorly along the trigeminal nerve to reach the gasserian region where it anastomoses with the petrosal branch (Pb) of the MMA. Usually, only the posterior (parieto-occipital) branch of the MMA arises from the BA and the anterior (frontal) branch keeps its normal origin from the IMA but in few cases, the complete territory of the MMA has a BA origin [11, 23–28]. Two distinct embryological explanations are postulated by authors to explain this anatomical variation. Seeger and Lasjaunias explained it by an anastomosis in the gasserian region between the basilar remnant of the trigeminal artery and the persistent stapedial artery [11, 12, 15]. Consequently, after regression of the proximal stem of the SA at the level of the stapes, the MMA takes its origin from the BA. Other authors postulated that an enlarged lateral pontine artery develops during the embryologic life and anastomoses with the SA. A rare case of MMA origin from an enlarged pontine artery is presented in Fig. 1 [19, 23, 24]. Kuruvilla et al. (2011) described a particular case where the MMA arises directly from the PICA and not from the BA. No embryological explanation was found to explain such an origin [25].

Ophthalmic Artery Origin

The MMA could also originate from the OA instead of the IMA. The incidence of this vascular variation is estimated to 0.5% by Dilenge et al. (1980) based on a large angiographic series [29]. Few cases of MMA arising from the OA have been described in the literature [5, 8, 12, 17, 30–35]. The first case was presented by Curnow (1873) and in the same period Meyer (1887) also cited four cadaveric cases originally described by Zuckerkandl in 1876 during a congress presentation [36, 37]. This vascular anomaly is considered as the consequence of two different embryologic processes [12]. The first one is the failure of the supraorbital branch (stapedial artery) to regress. The second one is the absence of anastomosis between the maxillomandibular branch of the SA and the IMA [13]. Consequently, the MMA origins from the OA passing through the lateral part of the superior orbital fissure and the foramen spinosum are usually absent. Maiuri et al. (1998) proposed three different types of this vascular variation as highlighted in Table 3 [38]. The first type is the complete MMA territory that takes in charge by the OA through the superficial recurrent OA. The second type is only the anterior branch of the MMA with OA origin; the poste-

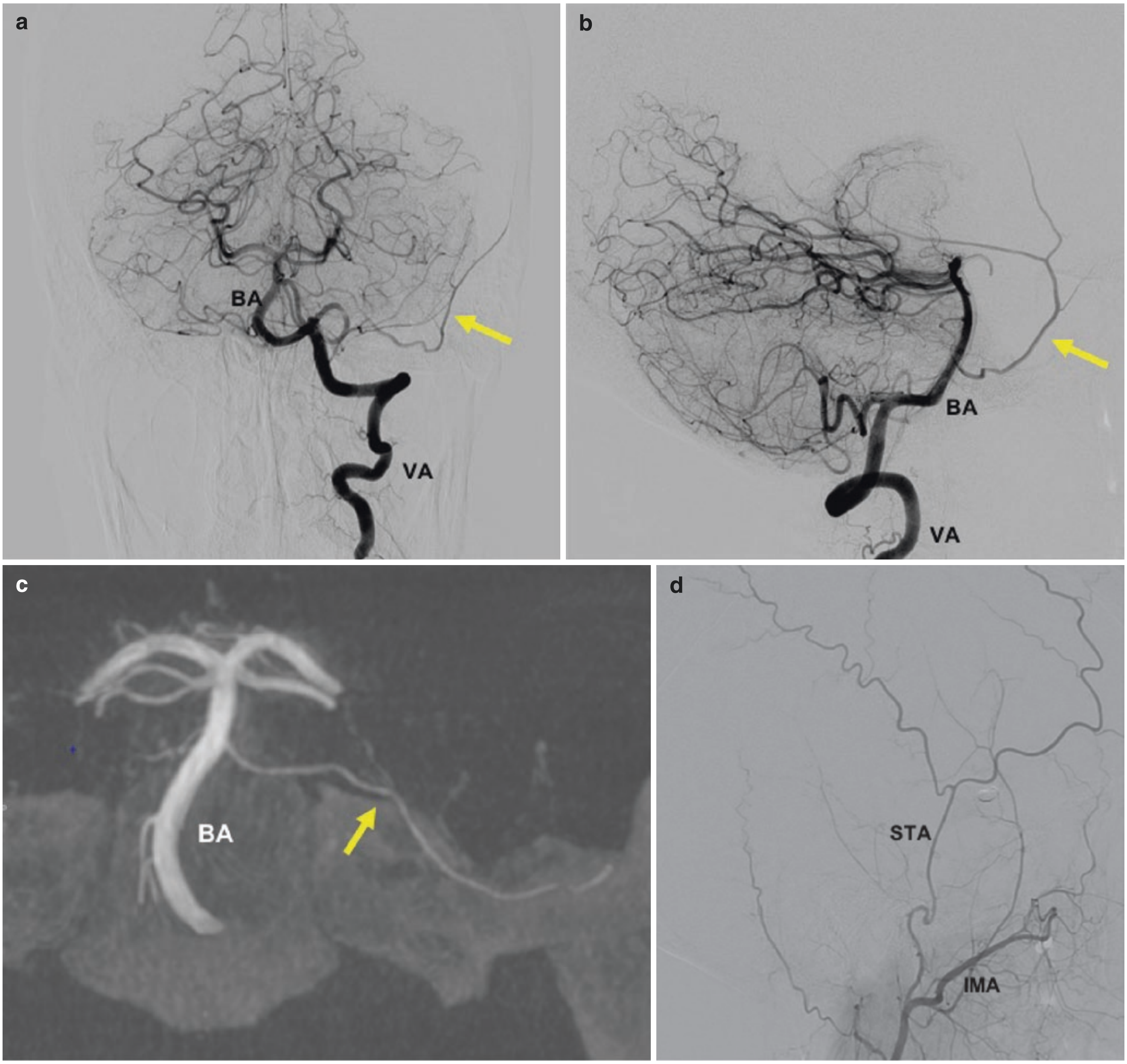


Fig. 1 Case of MMA origin from BA pontine perforating branch. In this case, the MMA origins from a pontine branch of the basilar artery (BA), as indicated by the yellow arrow in figure (**a**–**c**). The figures a and b show respectively a frontal and lateral view of left vertebral artery (VA) injection. Figure c shows a frontal xperCT reconstruction with the same MMA origin. Figure (**d**) shows a distal external carotid artery injection, where the superficial temporal artery (STA) and the internal maxillary artery (IMA) are visible, without the typical MMA origin from the IMA

Table 3 Different types of OA origin of the MMA by Maiuri et al. [38]

Type	Vascular anatomy	Foramen spinosum
I	Complete OA origin of the MMA	Absence
II	Partial OA origin of the MMA Anterior division from the OA Posterior division from the IMA	Reduced in size
III	OA origin of the accessory meningeal artery	Normal

rior branch of the MMA keeps its origin from the internal maxillary artery. The third type is not really an OA origin of the MMA but an anastomosis between the OA and the accessory meningeal artery (through the deep recurrent OA) and consequently, the anterior meningeal territory is supplied by both the MMA and the OA without any communication. Two cases of complete and partial origin of the MMA from the OA are reported in Fig. 2. It is still a matter of debate if the MMA originates from the OA directly or from the proximal part of the lacrimal artery [15, 17].

Cavernous ICA Origin

The lonely case of cavernous ICA origin of the MMA was described by Lasjaunias (1977) in one of its most famous articles summarizing distinct vascular anomalies encountered at the base of the skull [12]. He published another time the same case in its textbook to illustrate this very rare anatomical variant [15]. In this case, all branches of the MMA were filled during ICA injection and its origin arose from the horizontal portion of the cavernous ICA. Lasjaunias, ele-

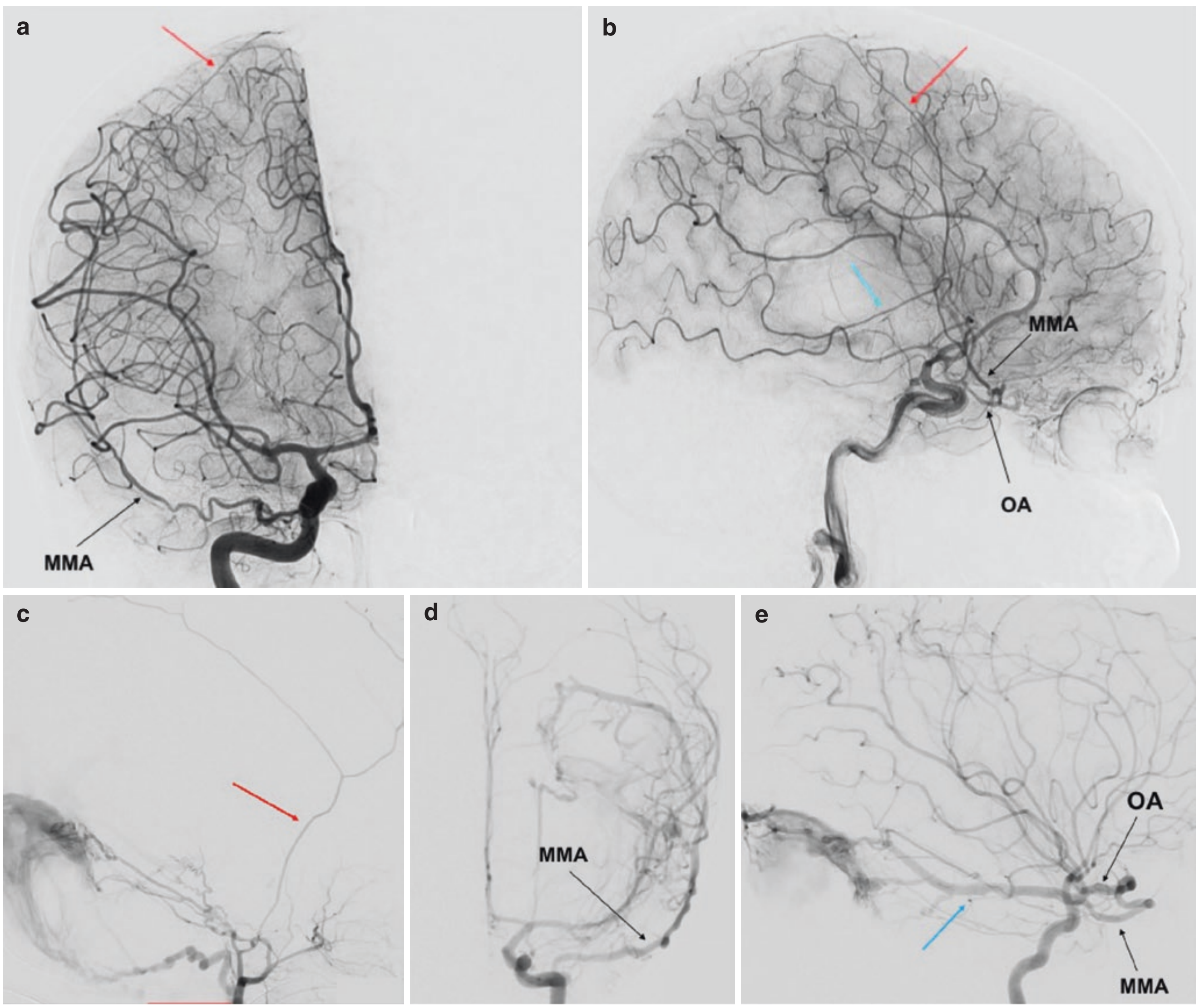


Fig. 2 Complete and partial MMA origin from OA. The antero-posterior and lateral view angiograms (**a** and **b**) show a rare case of complete MMA origin from the OA. The OA, through the superficial recurrent OA, gives birth to the MMA, that passes through the lateral part of the superior orbital fissure, and gives its anterior (red arrow) and posterior division (blue arrow). In the angiograms (**c**–**e**) a rare case of partial origin of the MMA from the OA is shown. The angiograms d and e show a left ICA injection in frontal and lateral view, where the posterior branch of the MMA (blue arrow) originates from the OA and feeds a tentorial arterio-venous fistula. After the ECA injection (**c**), only the anterior branch of the MMA is enhanced (red arrow)

gantly, explained this variation by the anastomosis during the embryological life between the posterior branch of the ILT and the SA; consequently the proximal stem of the MMA regressed (or was not formed) and the foramen spinosum was absent [12].

Petrous ICA Origin (Partial or Complete Persistence of the Stapedial Artery)

The complete persistence of the stapedial artery is a very rare variant, only two cases were published in a context of ICA aneurysm or PHACE syndrome. In these cases, the SA keeps its embryonic origin from the petrous ICA, passes through the middle ear and gives its two branches: one intracranial that corresponds to the MMA and the other extracranial leaving the cranial cavity through the foramen spinosum [14, 15]. Consequently, the foramen spinosum is enlarged, the cochlear promontory is eroded and the IMA arises from the SA instead of the ECA. Such an anatomical variant could easily be explained by the absence of embryological annexation of the maxillomandibular branch (of the SA) by the ventral pharyngeal artery. Consequently, in the absence of reversion of the arterial flow into the stapedial artery, its proximal (intratympanic) stem could not regress [15].

The partial persistence of the stapedial artery is more frequent and, in this case, only the intracranial branch of the SA keeps its origin from the petrous ICA [1, 4, 12, 13, 39–44]. The foramen spinosum is absent (an orifice does not exist without its contents) or reduced in size and the MMA arises from the SA instead of the IMA. This variant is explained by the regression of the proximal part of the maxillomandibular artery instead of the proximal part of the SA [15].

Pseudo-Petrous ICA (Persistence of the SA Associated with Aberrant Flow of the ICA)

In rare cases, the persistence of the SA, and the consequent origin of the MMA from the petrous ICA, is associated with an intratympanic course of the ICA (also known as "aberrant flow of the ICA") [12, 13, 19, 40, 45, 46]. This association of vascular variants concerning both carotid and hyostapedial systems was described anatomically and angiographically [4, 15]. In these cases, the ICA enters the skull base through an enlarged inferior tympanic canal (narrowing of the vessel on digital subtraction angiography (DSA)), passes into the tympanic cavity to bend anteriorly and reaches its normal carotid canal. The MMA arises from the ICA in its tympanic segment and passes through the stapes to have the same course described in the previous paragraph. The extracranial orifice of the carotid canal is therefore absent in these cases [12, 13].

The intratympanic course of the ICA is explained by the agenesis of the two first segments of the primitive carotid artery, with a hypertrophied inferior tympanic artery that maintains the anastomosis between the ascendant pharyngeal artery and the carotico-tympanic artery (branch of the ICA) into the tympanic cavity. Therefore, the cervical and intratympanic segments of this artery do not derivate from the carotid system but from the pharyngo-occipital and hyostapedial systems (pseudo-ICA) [12, 13, 15]. The agenesis of the first two segments of the ICA could be partial and the ICA appears duplicated [45].

Cervical ICA Origin (Pharyngo-tympano-stapedial Artery)

The MMA could also arise from the cervical segment of the ICA. This very rare variant was first described by Lasjaunias (1977) in its original publication [12]. The same case served as illustration in the textbook *Surgical Angiography* and only one similar case was published by Baltsavias et al. (2012) [15, 47]. The MMA arises from the cervical portion of the ICA, ascends along the cervical ICA, enters into the tympanic cavity through the inferior tympanic canal and follows the usual course of the SA. The two cases described were presented as "partial" persistence of the SA with only the MMA arising from the SA and the absence of the foramen spinosum. In this variant, an annexation of the SA by the inferior tympanic artery (branch of the ascending pharyngeal artery) with regression of the proximal part of the SA explains this vascular configuration. Therefore, the SA arises from the cervical instead of the petrous segment of the ICA.

Occipital Artery Origin

Diamond (1987) described a remarkable case of partial persistent SA found during a temporal bone dissection [16]. The particularity of this case was the origin of the SA that arose from the occipital artery instead of the petrous ICA passing through a "special foramen" to enter the petrous part of the temporal bone between the styloid process and the carotid canal. After passing through the posterior wall of the tympanic cavity and through the stapes, the SA entered the facial canal to reach the petrous apex and to give all branches of the MMA. In this article, Diamond (1987) failed to give a clear embryonic explanation or a hypothesis of the variant he found [16].

Distal Petrous ICA Origin (Mandibular Artery Origin)

Lasjaunias et al. (2001) was the lonely author to show a case of distal petrous ICA origin of the MMA near the normal origin of the Vidian artery [15]. In this case, the MMA does not follow an intratympanic course. The author explains this variant by the absence of annexation of the first aortic arch by the second aortic arch (stapedial artery). Therefore, the mandibular artery retains its primary territory which is the middle meningeal artery territory [15].

Course of the Artery

The first extracranial segment of the MMA is from its origin to its entry into the foramen spinosum. More anterior the origin of the MMA is, the more oblique backward is the extracranial segment. At the level of the foramen spinosum, the artery bends anteriorly and laterally to follow the temporal fossa. This bend is responsible for the characteristic aspect of the MMA on DSA. After its entry into the cranial cavity, the MMA has a lateral course grooving the greater sphenoid wing. Merland et al. (1977) describe three intracranial segments of the MMA [9]. The first one is the temporo-basal segment where the artery follows the temporal fossa and curves upward where it becomes the second or temporo-pterional segment. After passing the pterional region, the artery enters in its coronal segment where the artery follows the coronal suture to end at the region of the bregma. Martins et al. (2005) considered the course of the artery shorter and simpler [1]. They described the termination of the MMA where it divides in anterior and posterior divisions at the pterional region. The anterior division of the MMA is classically the coronal segment previously described by Merland et al. (1977) [9].

Branches of the Artery

The two most precise and complete publications that describe the branches of the MMA were published by Merland et al. (1977) and Martins et al. (2005) [1, 9]. One description is based on cerebral angiographies, the other one on cadaveric dissections. Before them, Salamon et al. (1967) paid particular attention to correlate the anatomy with the angiographic images [6, 7]. As noted in the previous paragraph, these two authors used a different naming of the branches, even if the terminology used by Martins et al. seems to be the most comprehensive one. Table 4 shows different branches of the MMA with the dural territory associated and possible anastomosis with other dural arteries [7].

The course and branches of MMA are indicated on the DSA shown in Fig. 3. The MMA divides at the pterional region in two divisions: anterior and posterior. Before its bifurcation, the MMA has two branches supplying the dura of the temporal fossa [8, 9, 13]. The first one is the petrosal branch which courses on the petrous apex and supplies the dura of this region (including the gasserian ganglion) and also the superior part of the tympanic cavity via the superior tympanic artery passing through the facial canal. The second basal branch of the MMA is the cavernous branch which

Table 4 Different branches of the MMA with their respective anastomosis

MMA branches	Origin from the MMA	Territory (dural and neural)	Possible anastomosis
Petrosal branch	Foramen spinosum	Trigeminal ganglion and nerves Posteromedial floor of the middle fossa Insertion of tentorium (medial half) Superior petrosal sinus	Ascending pharyngeal artery (carotid branch) Medial and lateral tentorial arteries internal carotid artery (ICA)
Superior tympanic artery	Petrosal branch	Greater superficial petrosal nerve Geniculate ganglion Tympanic cavity (superior part)	
Cavernous branch	Petrosal branch	Lateral wall of the cavernous sinus	Accessory meningeal artery Inferolateral trunk (ICA)
Anterior division	Pterional region	Frontal and anterior parietal convexity Superior sagittal sinus Anterior and middle fossa (lateral part)	Anterior and posterior ethmoidal arteries (OA) Contralateral MMA
Falcine arteries	Anterior and posterior division	Falx cerebri	Anterior falcine artery (OA) Anterior cerebral artery Posterior meningeal artery (vertebral artery)
Medial branch	Anterior division	Lesser sphenoid wing Superior orbital fissure Peri-orbita (lateral)	Recurrent meningeal branches (OA) Inferolateral trunk (ICA)
Petro-squamosal branch	Posterior division	Posterolateral floor of the middle fossa Insertion of tentorium (lateral half) Superior petrosal sinus Transverse and sigmoid sinuses Dura of the posterior fossa (superior part)	Ascending pharyngeal artery (jugular branch) Lateral tentorial artery (ICA) Occipital artery (mastoid branch)
Parieto-occipital branch	Posterior division	Temporo-squamous dura Parieto-occipital convexity Superior sagittal sinus	Posterior meningeal artery (vertebral artery)

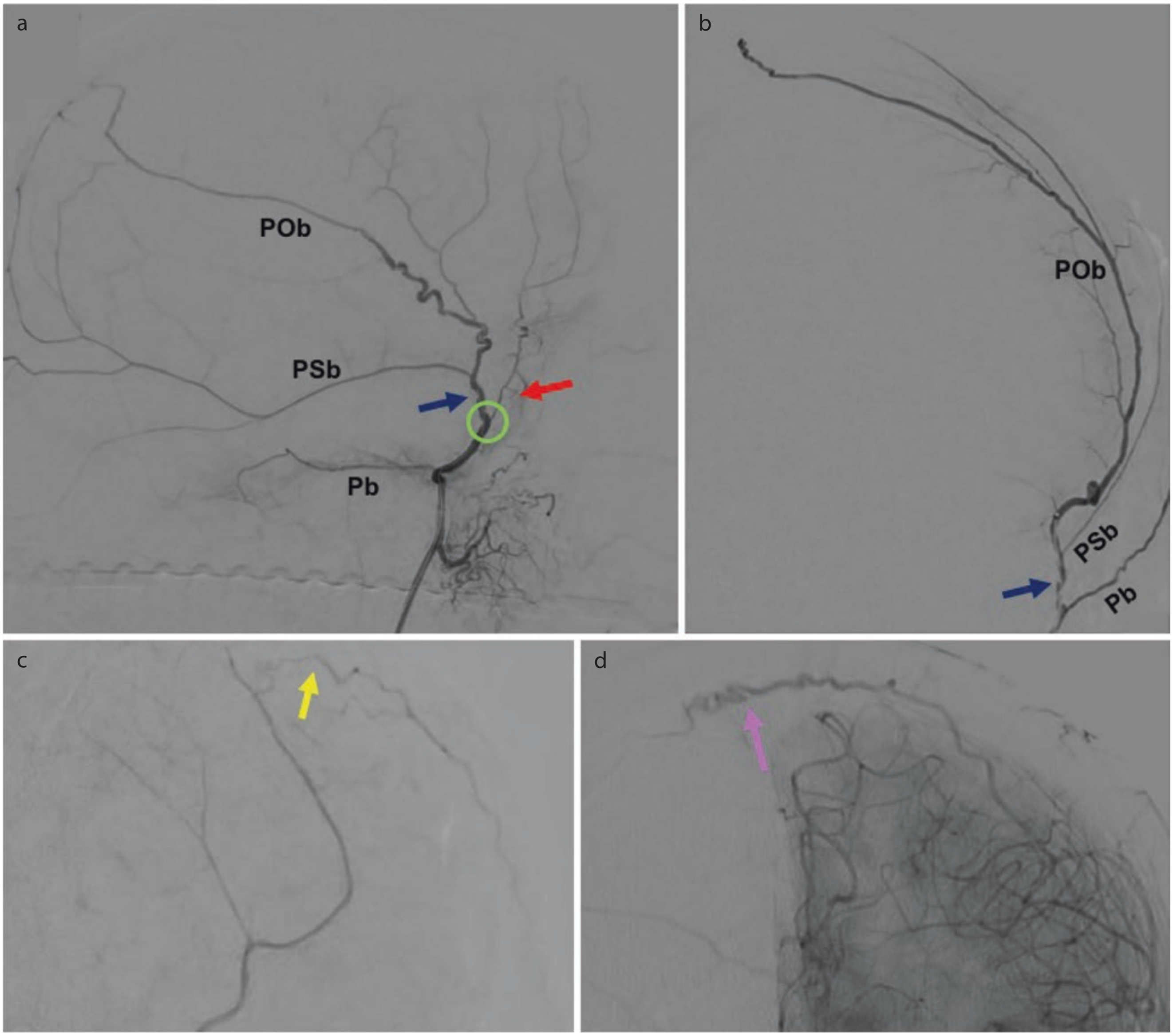


Fig. 3 DSA showing segments and branches of the MMA. The figures a and b show selective MMA injection from a lateral (**a**) and frontal (**b**) view. The MMA bifurcates at the pterional region (green circle) in two, an anterior (red arrow) and posterior (blue arrow) division. Before its bifurcation the MMA gives the petrosal branch (Pb) which courses on the petrous apex. The posterior division gives two principal branches: the petro-squamosal branch (PSb) and the parieto-occipital branch (POb). The anterior division ends with two kinds of terminal branches, visible after common carotid artery injection: the falcine arteries (yellow arrow, figure (**c**)), that anastomose with branches of the anterior falcine artery from the ophthalmic artery, and contralateral branches (purple arrow, figure (**d**)) that cross the midline to anastomose with contralateral MMA

supplies the lateral wall of the cavernous sinus and anastomoses with branches of the ILT [1].

The anterior division of the MMA is in the dura of the convexity following the coronal suture until the bregma. This anterior division of the MMA has two types of terminal branches: (1) falcine arteries which anastomose with branches of the anterior falcine artery and (2) contralateral branches that cross the midline to anastomose with branches of the contralateral MMA. Near the pterional region, the anterior division gives a medial branch that runs under the lesser sphenoid wing that supplies the dura of anterior part of the temporal fossa, the superior orbital fissure and could anastomose with the recurrent meningeal branches of the OA [1, 9].

The posterior division of the MMA also supplies the dura of the convexity and gives two principal branches: the petro-squamosal branch (PSb) and the parieto-occipital branch (POb) [1]. These two branches participate in the vascularization of the parieto-temporo-occipital dura, the transverse sinuses and also the posterior two-thirds of the tentorium [10]. Branches of the posterior division of the MMA anastomose with dural branches of the occipital, ascending pharyngeal, subarcuate and vertebral arteries [7, 10].

Dural Territory

The MMA supplies most of the dura of the cranial convexity via its anterior and posterior divisions and usually participates in the vascularization of the superficial half of the falx cerebri. The fronto-medial dura and the occipito-medial dura are on the contrary usually respectively supplied by the anterior meningeal artery (AMA) from the OA and by posterior meningeal artery (PMA) from the vertebral artery [1]. Obviously, the supra-tentorial dural territory of the convexity is in balance between these three arteries and therefore, the territory of the MMA could be variable [9].

The dura of the superior sagittal sinus is also supplied by branches of the MMA but also branches of the anterior meningeal artery (anterior part) and branches of the posterior meningeal artery (posterior part) [12]. The MMA could also supply the torcular region via its posterior division [1].

At the skull base, the MMA supplies the middle cranial fossa and lateral part of the anterior cranial fossa, and also the inferior part of the lateral wall of the cavernous sinus via its cavernous branch [7]. Its participation in the vascularization of the tentorium is limited to the insertion of the tentorium and to the superior sagittal sinus [48].

The anterior division of the MMA through its medial branch supplies the dura of the lesser sphenoid wing and the superior orbital fissure region [9]. These branches also participate in the vascularization of the lateral wall of the periorbita and could also participate in the vascularization of orbital branches by anastomoses with the lacrimal artery (through the deep recurrent meningeal artery of the OA) [8].

Through its posterior division, the MMA also gives branches destined to the dura of the supero-lateral part of the cerebellar fossa. Moret et al. (1978) showed the importance of the MMA in the vascularization of the posterior fossa dura, always in balance with branches of the ascending pharyngeal and occipital arteries [10].

The MMA also supplies partially the trigeminal and facial nerves [1, 9, 15]. Indeed, the petrosal branch gives branches to the gasserian ganglion and to the maxillary and mandibular divisions of the trigeminal nerve in their cavernous portion. The greater superficial petrosal nerve and the geniculate ganglion of the facial nerve also receive branches from the petrosal branch of the MMA [1].

Finally, the superior tympanic artery, which is the petrosal remnant of the SA and therefore, a branch of the petrosal branch supplies the superior part of the tympanic cavity [6].

Possible Anastomoses

The dural vascular territory of the MMA is in balance between numerous arteries coming from ICA, ECA as well as from the vertebro-basilar system. Therefore, the MMA presents a lot of anastomoses with branches arising from the other arteries, most of which are represented in Fig. 4. The three major anatomical regions of vascular anastomoses are:

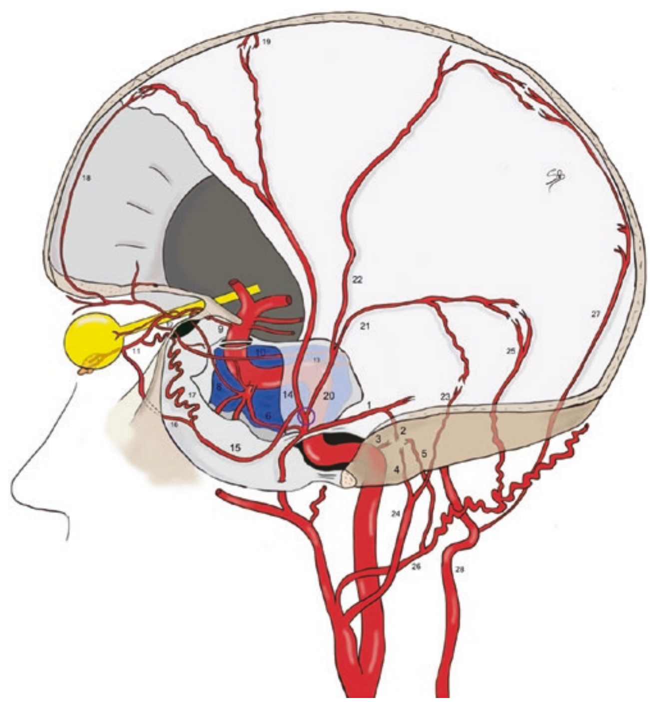


Fig. 4 Anastomoses of the MMA. Before the MMA bifurcation (purple circle), the petrous branch (*1*), from which the superior tympanic artery (*2*) origins, anastomoses into the middle ear with the carotico-tympanic artery (*3*, from ICA), with the inferior tympanic artery (*4*, from ascending pharyngeal artery), with the posterior tympanic artery (*5*, from occipital artery). The cavernous branch of the MMA (*6*) on the other side anastomoses with the ILT (*7*), which is itself connected to the OA (*9*) through the deep recurrent OA (*8*). The ILT, the MMA and the OA are also linked to each other through the marginal tentorial artery (*10*), whose origin can vary from the lacrimal artery (*11*), via superficial recurrent OA (*12*), to the MHT (*13*). After the MMA bifurcation at the pterion, its frontal division (*14*) gives a medial branch (*15*) that can bifurcate intracranially into a lateral meningo-lacrimal artery (16) and a medial sphenoidal artery (*17*). Both the branches reach the lacrimal artery, even if the former distally and the latter proximally. The anastomoses with the OA and the ILT represent the most dangerous connections in case of MMA transarterial embolization, because of the risk of particles embolism into these arteries. The frontal division of the MMA reaches the convexity following the coronal suture and anastomoses with the anterior falcine artery (*18*, OA-anterior ethmoidal artery) and with branches of the contralateral MMA (*19*) [1, 8]. The posterior division of the MMA (*20*) divides into a petro-squamosal branch (*21*) and a parieto-occipital branch (*22*). The former anastomoses with the jugular branch (*23*) of the ascending pharyngeal artery (*24*) and with the mastoid branch (*25*) of the occipital artery (*26*). The latter is linked to the posterior meningeal artery (*27*), from the vertebral artery (*28*), at the border areas [10]

the peri- and cavernous region, the fronto-medial region and the posterior fossa [1].

On the skull base, branches of the MMA, principally its petrosal branch anastomose with the accessory meningeal artery, the ascending pharyngeal artery (via its carotid branch), the ILT, the meningo-hypophyseal trunk (MHT) and the recurrent meningeal artery of the OA [7, 8, 20].

At the frontal region, the anterior division of the MMA gives anastomoses principally with dural branches of the OA: the anterior and posterior ethmoidal arteries and also the anterior falcine artery [7, 29].

In the posterior fossa, it anastomoses principally with the posterior meningeal artery and with the mastoid branch of the occipital artery [10].

The last natural anastomosis to be noted is between the MMAs of both sides on the midline [1].

Clinical Implications

The understanding of the vascular anatomy of the dura mater has a major importance both for neuroradiologists and for neurosurgeons.

Chronic Subdural Hematomas

During the last 5 years, MMA has been of interests of neuroradiologists and neurosurgeons in the treatment of recurrent chronic subdural hematomas (cSDH). Mostly in patients older than 65 years, under anticoagulant and antiplatelet therapy, mild head trauma can cause bridging veins injury leading to chronic subdural hematoma [49]. Traditional treatment consists of burr holes evacuation, with a high rate of recurrence (2–37%) [49, 50]. This trend is attributed to the inflammatory response caused by residual blood that causes the formation of membranes around the hematoma. Membranes are also stimulated by angiogenic factors to neoangiogenesis, resulting in the formation of fragile microcapillaries, with high rebleeding risk. In this context, some authors like Link et al. 2018, proposed the management of recurrent cSDH through endovascular MMA embolization [49]. MMA represents the principal blood supply of the bleeding membranes; thus, its occlusion allows the collection to be resorbed [49–51]. These authors described a recurrence rate similar to the surgical evacuation, but with a less invasive procedure. Thus, this technique could represent an alternative treatment option, especially for elderly patients with recurrent cSDH.

Meningeal Tumors

Meningiomas are the most common benign intracranial tumors, located mostly at the skull vault or at the skull base. Since MMA is the major dural artery, most cranial meningiomas receive its supply. Surgery represents the first line treatment for symptomatic meningiomas, but preoperative MMA embolization could be used to reduce the blood supply of the meningioma to limit the blood loss during resection. Depending on their location, meningioma can be supplied by ICA dural branches, ECA dural branches or a combination of them. Richter et al. classified meningiomas into four types depending on their vascular supply: type I, with exclusive ECA vascularization; type II, with mixed ICA/ECA blood supply with ECA prevalence; type III, with mixed supply with ICA prevalence and type IV, with exclusive ICA supply [52]. Usually, anterior cranial fossa meningiomas are supplied by MMA and anterior falcine artery from the OA, middle cranial fossa meningiomas are fed by MMA and dural branches of petrous and cavernous ICA. On the other hand, posterior fossa meningiomas are rarely supplied by MMA, but from posterior meningeal artery from the vertebral artery and from other ECA branches. For these reasons, MMA embolization should be reserved for meningiomas type I or II of Richter classification, with typical DSA blush, for middle cranial fossa and selective cases of anterior cranial fossa lesions [53]. During the procedure the neuroradiologist should keep in mind the possible anastomoses between MMA and ICA branches to avoid complications. After MMA embolization, surgery can be performed with a very variable timing (from 0 to 30 days after the procedure) [54]. Of course also the neurosurgeon has to keep in mind the general organization of the dural vascularization which gives an important help for meningioma's surgery and other meningeal tumors [29].

Dural Arteriovenous Fistula

The principal dural pathology treated by neuroradiologists is the dural arteriovenous fistula (dAVF). Also for these procedures, a good knowledge of the precise anatomy of the MMA, of its variations and its vascular anastomoses helps in the avoidance of complications [15]. Transarterial embolization through MMA has been described as a successful option for dAVF treatment [55–57]. According to Griessenauer et al., a robust MMA supply is the best predictor for successful embolization [55]. Other factors that make the MMA a favorable way to perform transarterial embolization are its long straight course, which facilitates the penetration of Onyx, its quite large diameter, that allows the introduction of catheters, and its large dural territory, that often reaches dAVFs in various locations.

As already noted, the presence of an SA has also an important impact on the technical difficulty for performing a stapedectomy [2, 52]. The middle ear surgeon could avoid excessive blood loss knowing the presence of such a vascular variation during the surgical planning.

References

1. Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A, Jr. Microsurgical anatomy of the dural arteries. Neurosurgery 2005;56(2 Suppl):211–251; discussion 211−51.
2. Maran AG. Persistent stapedial artery. J Laryngol Otol. 1965;79(11):971–5.
3. Padget DH. The development of cranial arteries in the human embryo, Contribution to embryology, vol. 212. Washingtion: Carnegie Institution; 1948. p. 205–62.
4. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
5. Gabriele OF, Bell D. Ophthalmic origin of the middle meningeal artery. Radiology. 1967;89(5):841–4. https://doi.org/10.1148/89.5.841.
6. Salamon G, Guerinel G, Demard F. Radioanatomical study of the external carotid artery. Ann Radiol (Paris). 1968;11(3):199–215.
7. Salamon G, Grisoli J, Paillas JE, Faure J, Giudicelli G. Arteriographic study of the meningeal arteries. (role of selective injections, of subtraction and of radio-anatomical correlations). Neurochirurgia (Stuttg). 1967;10(1):1–19. https://doi.org/10.1055/s-0028-1095333.
8. Moret J, Lasjaunias P, Theron J, Merland JJ. The middle meningeal artery. Its contribution to the vascularisation of the orbit. J Neuroradiol. 1977;4(2):225–48.
9. Merland JJ, Theron J, Lasjaunias P, Moret J. Meningeal blood supply of the convexity. J Neuroradiol. 1977;4(2):129–74.
10. Moret J, Lasjaunias P, Vignaud J, Doyon D. The middle meningeal blood supply to the posterior fossa (author's transl). Neuroradiology. 1978;16:306–7.
11. Seeger JF, Hemmer JF. Persistent basilar/middle meningeal artery anastomosis. Radiology. 1976;118(2):367–70. https://doi.org/10.1148/118.2.367.
12. Lasjaunias P, Moret J, Manelfe C, Theron J, Hasso T, Seeger J. Arterial anomalies at the base of the skull. Neuroradiology. 1977;13(5):267–72.
13. Lasjaunias P, Moret J. Normal and non-pathological variations in the angiographic aspects of the arteries of the middle ear. Neuroradiology. 1978;15(4):213–9.
14. Rodesch G, Choi IS, Lasjaunias P. Complete persistence of the hyoido-stapedial artery in man. Case report. Intra-petrous origin of the maxillary artery from ICA. Surg Radiol Anat. 1991;13(1):63–5.
15. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical Neuroangiography. Berlin: Springer edition; 2001.
16. Diamond MK. Unusual example of a persistent stapedial artery in a human. Anat Rec. 1987;218(3):345–54. https://doi.org/10.1002/ar.1092180316.
17. Diamond MK. Homologies of the meningeal-orbital arteries of humans: a reappraisal. J Anat. 1991;178:223–41.
18. Diamond MK. Homologies of the stapedial artery in humans, with a reconstruction of the primitive stapedial artery configuration of Euprimates. Am J Phys Anthropol. 1991;84(4):433–62. https://doi.org/10.1002/ajpa.1330840408.
19. Steffen TN. Vascular anomalites of the middle ear. Laryngoscope. 1968;78(2):171–97. https://doi.org/10.1288/00005537-196802000-00001.
20. Margolis MT, Newton TH. Collateral pathways between the cavernous portion of the internal carotid and external carotid arteries. Radiology. 1969;93(4):834–6. https://doi.org/10.1148/93.4.834.
21. Kelemen G. Arteria stapedia, in bilateral persistence. AMA Arch Otolaryngol. 1958;67(6):668–77.
22. Low FN. An anomalous middle meningeal artery. Anat Rec. 1946;95:347–51.
23. Katz M, Wisoff HS, Zimmerman RD. Basilar-middle meningeal artery anastomoses associated with a cerebral aneurysm. Case report. J Neurosurg. 1981;54(5):677–80. https://doi.org/10.3171/jns.1981.54.5.0677.
24. Shah QA, Hurst RW. Anomalous origin of the middle meningeal artery from the basilar artery: a case report. J Neuroimaging. 2007;17(3):261–3. https://doi.org/10.1111/j.1552-6569.2007.00108.x.
25. Kuruvilla A, Aguwa AN, Lee AW, Xavier AR. Anomalous origin of the middle meningeal artery from the posterior inferior cerebellar artery. J Neuroimaging. 2011;21(3):269–72. https://doi.org/10.1111/j.1552-6569.2010.00475.x.
26. Kumar S, Mishra NK. Middle meningeal artery arising from the basilar artery: report of a case and its probable embryological mechanism. J Neurointerv Surg. 2012;4(1):43–4. https://doi.org/10.1136/jnis.2010.004465.
27. Salem MM, Fusco MR, Dolati P, Reddy AS, Gross BA, Ogilvy CS, et al. Middle meningeal artery arising from the basilar artery. J Cerebrovasc Endovasc Neurosurg. 2014;16(4):364–7. https://doi.org/10.7461/jcen.2014.16.4.364.
28. Waga S, Okada M, Yamamoto Y. Basilar-middle meningeal arterial anastomosis. Case report J Neurosurg. 1978;49(3):450–2. https://doi.org/10.3171/jns.1978.49.3.0450.
29. Dilenge D, Ascherl GF Jr. Variations of the ophthalmic and middle meningeal arteries: relation to the embryonic stapedial artery. AJNR Am J Neuroradiol. 1980;1(1):45–54.
30. Brucher J. Origin of the ophthalmic artery from the middle meningeal artery. Radiology. 1969;93(1):51–2. https://doi.org/10.1148/93.1.51.
31. Royle G, Motson R. An anomalous origin of the middle meningeal artery. J Neurol Neurosurg Psychiatry. 1973;36(5):874–6. https://doi.org/10.1136/jnnp.36.5.874.
32. Liu Q, Rhoton AL Jr. Middle meningeal origin of the ophthalmic artery. Neurosurgery. 2001;49(2):401–6. discussion 6–7.
33. Plas B, Bonneville F, Dupuy M, Sol JC, Chaynes P. Bilateral ophthalmic origin of the middle meningeal artery. Neurochirurgie. 2013;59(4–5):183–6. https://doi.org/10.1016/j.neuchi.2013.08.005.
34. Cvetko E, Bosnjak R. Unilateral absence of foramen spinosum with bilateral ophthalmic origin of the middle meningeal artery: case report and review of the literature. Folia Morphol (Warsz). 2014;73(1):87–91. https://doi.org/10.5603/FM.2014.0013.
35. Kimball D, Kimball H, Tubbs RS, Loukas M. Variant middle meningeal artery origin from the ophthalmic artery: a case report. Surg Radiol Anat. 2015;37(1):105–8. https://doi.org/10.1007/s00276-014-1272-z.
36. Meyer F. Zur anatomie der Orbitalarteien Morph Jahr 1887;12:414–458.
37. Curnow J. Two instances of irregular ophthalmic and middle meningeal arteries. J Anat Physiol. 1873;8(Pt 1):155–6.
38. Maiuri F, Donzelli R, de Divitiis O, Fusco M, Briganti F. Anomalous meningeal branches of the ophthalmic artery feeding meningiomas of the brain convexity. Surg Radiol Anat. 1998;20(4):279–84.
39. Fisher AG. A case of complete absence of both internal carotid arteries, with a preliminary note on the developmental history of the Stapedial artery. J Anat Physiol. 1913;48(Pt 1):37–46.
40. Guinto FC Jr, Garrabrant EC, Radcliffe WB. Radiology of the persistent stapedial artery. Radiology. 1972;105(2):365–9. https://doi.org/10.1148/105.2.365.
41. Teal JS, Rumbaugh CL, Bergeron RT, Segall HD. Congenital absence of the internal carotid artery associated with cerebral hemiatrophy, absence of the external carotid artery, and persistence of the stapedial artery. Am J Roentgenol Radium Therapy, Nucl Med. 1973;118(3):534–45.
42. McLennan JE, Rosenbaum AE, Haughton VM. Internal carotid origins of the middle meningeal artery. The ophthalmic-middle meningeal and stapedial-middle meningeal arteries. Neuroradiology. 1974;7(5):265–75.
43. Sheikh BY, Coates R, Siqueira EB. Stapedial artery supplying sphenoid wing meningioma: case report. Neuroradiology. 1993;35(7):537–8.

44. Kawai K, Yoshinaga K, Koizumi M, Honma S, Tokiyoshi A, Kodama K. A middle meningeal artery which arises from the internal carotid artery in which the first branchial artery participates. Ann Anat. 2006;188(1):33–8. https://doi.org/10.1016/j.aanat.2005.08.013.
45. Koenigsberg RA, Zito JL, Patel M, Swartz JD, Goldofsky E, Zahtz G. Fenestration of the internal carotid artery: a rare mass of the hypotympanum associated with persistence of the stapedial artery. AJNR Am J Neuroradiol. 1995;16(4 Suppl):908–10.
46. Silbergleit R, Quint DJ, Mehta BA, Patel SC, Metes JJ, Noujaim SE. The persistent stapedial artery. AJNR Am J Neuroradiol. 2000;21(3):572–7.
47. Baltsavias G, Kumar R, Valavanis A. The pharyngo-tympano-stapedial variant of the middle meningeal artery. A case report. Interv Neuroradiol. 2012;18(3):255–8. https://doi.org/10.1177/159101991201800302.
48. Silvela J, Zamarron MA. Tentorial arteries arising from the external carotid artery. Neuroradiology. 1978;14(5):267–9.
49. Link TW, Boddu S, Marcus J, Rapoport BI, Lavi E, Knopman J. Middle meningeal artery embolization as treatment for chronic subdural hematoma: a case series. Oper Neurosurg (Hagerstown). 2018;14(5):556–62. https://doi.org/10.1093/ons/opx154.
50. Fiorella D, Arthur AS. Middle meningeal artery embolization for the management of chronic subdural hematoma. J Neurointerv Surg. 2019;11(9):912–5. https://doi.org/10.1136/neurintsurg-2019-014730.
51. Haldrup M, Ketharanathan B, Debrabant B, Schwartz OS, Mikkelsen R, Fugleholm K, et al. Embolization of the middle meningeal artery in patients with chronic subdural hematoma-a systematic review and meta-analysis. Acta Neurochir. 2020;162(4):777–84. https://doi.org/10.1007/s00701-020-04266-0.
52. Richter HP, Schachenmayr W. Preoperative embolization of intracranial meningiomas. Neurosurgery. 1983;13(3):261–8. https://doi.org/10.1227/00006123-198309000-00008.
53. Dubel GJ, Ahn SH, Soares GM. Contemporary endovascular embolotherapy for meningioma. Semin Intervent Radiol. 2013;30(3):263–77. https://doi.org/10.1055/s-0033-1353479.
54. Shah A, Choudhri O, Jung H, Li G. Preoperative endovascular embolization of meningiomas: update on therapeutic options. Neurosurg Focus. 2015;38(3):E7. https://doi.org/10.3171/2014.12.FOCUS14728.
55. Griessenauer CJ, He L, Salem M, Chua MH, Ogilvy CS, Thomas AJ. Middle meningeal artery: gateway for effective transarterial onyx embolization of dural arteriovenous fistulas. Clin Anat. 2016;29(6):718–28. https://doi.org/10.1002/ca.22733.
56. Hayashi K, Ohmoro Y, Kaku Y, Takemoto Y, Nakagawa T, Yano S, et al. Non-sinus-type Dural arteriovenous fistula cured by Transarterial embolization from middle meningeal artery: two case reports. J Neuroendovascular Ther. 2017;12:542. https://doi.org/10.5797/jnet.cr.2017-0131.
57. Hlavica M, Ineichen BV, Fathi AR, Remonda L, Diepers M. Parasagittal Dural arteriovenous fistula treated with Embozene microspheres. J Endovasc Ther. 2015;22(6):952–5. https://doi.org/10.1177/1526602815604464.

Dural Branches of the Internal Maxillary Artery

Thomas Robert and Sara Bonasia

The internal maxillary artery (IMA) is the branch of the external carotid artery with the most important meningeal territory [1, 2]. Most of it is the function of the middle meningeal artery (MMA), but two other less known branches of the IMA have a little meningeal supply [3]. In this chapter, we will not develop the anatomy and variations of the MMA, which is presented in the chapter "Anatomy, Embryology and Variations of the Middle Meningeal Artery." The anatomy of the IMA and its other branches are described in chapter "Embryology and Anatomy of the Internal Maxillary Artery." Origin, branches, territory, and anastomoses of the accessory meningeal artery (AccMA) and artery of the foramen rotundum are described in this chapter and summarized in Table 1.

Table 1 Origin, course, territory, and anastomoses of dural branches of the internal maxillary artery (IMA) (For the middle meningeal artery, see chapter "Anatomy, Embryology and Variations of the Middle Meningeal Artery")

Dural branches IMA	Origin from the IMA	Foramen	Meningeal territory	Meningeal anastomoses
Accessory meningeal artery	First segment	Ovale or Vesalius	Medial third of the anterior skull base	Cavernous branch (MMA) Posterior branch (ILT) Recurrent branch (OA)
Artery of the foramen rotundum	Third segment	Rotundum	Middle cranial fossa dura	Anterolateral branch (ILT)

Accessory Meningeal Artery

The first description of the accessory meningeal artery (AccMA) was offered in 1888 by Sappey [4]. After him, few anatomists and, more recently, neuroradiologists dedicated their works to this little artery [1, 3–7]. The AccMA is not a purely meningeal artery, and its extracranial supply is most extended than its meningeal one. For this reason, few authors proposed another nomenclature as the *pterygomeningeal artery* [4].

The AccMA is present in almost all cases; Baumel and Beard found it in 96% of their 76 cadaveric dissections [4]. The diameter of the AccMA is usually one third the diameter of the MMA [2].

The origin of the AccMA could be directly from the IMA or from the MMA depending on the course of the IMA [7]. In case of superficial course of the IMA, the AccMA arises directly from the IMA (65–85% of cases). In case of deep course of the IMA, the AccMA is a collateral branch of the MMA (15–35% of cases) [7–10]. The artery is unique in 70% but could be represented as multiple little arteries in 30% of cases [4, 5].

The foramen through which the AccMA penetrates the skull base also depends on the course of the IMA. The AccMA usually enters in the skull base through the foramen ovale (when the IMA has a superficial course) but penetrates through the foramen of Vesalius or sphenoidal emissary foramen in 15–35% of cases (when the IMA has a deep course) [4, 5, 7, 11].

Extracranially, the AccMA supplies the Eustachian tube, external acoustic meatus, lateral pharyngeal wall, medial pterygoid muscle, sphenoid periosteum, otic ganglion, and the mandibular division (V3) of the trigeminal nerve [4, 7]. Intracranially, the AccMA supplies in part the Gasserian ganglion, the cavernous sinus wall, and a part of the middle cranial fossa dura [5].

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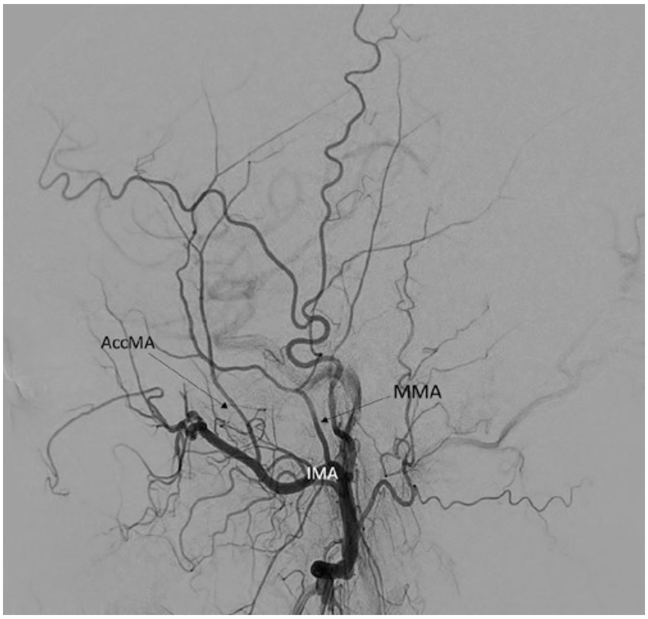

Fig. 1 digital subtraction angiography (DSA) showing the accessory meningeal artery. Lateral angiogram obtained after injection of the external carotid artery. The internal maxillary artery (IMA) gives off the middle meningeal artery (MMA) and the accessory meningeal artery (AccMA)

Extracranially, the AccMA anastomoses with branches of the ascending pharyngeal artery and pterygopalatine artery [4]. The AccMA shares its dural territory with the carotid branch of the MMA, the posterior branch of the inferolateral trunk (ICA), and the recurrent branch of the ophthalmic artery and has intradural anastomoses with these arteries [1, 12]. A clinical case showing this artery is represented in Fig. 1.

Artery of the Foramen Rotundum

The artery of the foramen rotundum is a little branch of the third segment of the IMA [1, 2, 12, 13]. It is not well-known and studied. In the non-pathological anatomy, this artery is difficult to see angiographically [14]. It has a posterosuperior course until the foramen rotundum where it supplies the dura of the middle cranial fossa around the foramen [2]. Its territory is in balance with territories of the inferolateral trunk with which it anastomoses (anterolateral branch) [1]. The angiographic configuration of this artery is shown in Fig. 2.

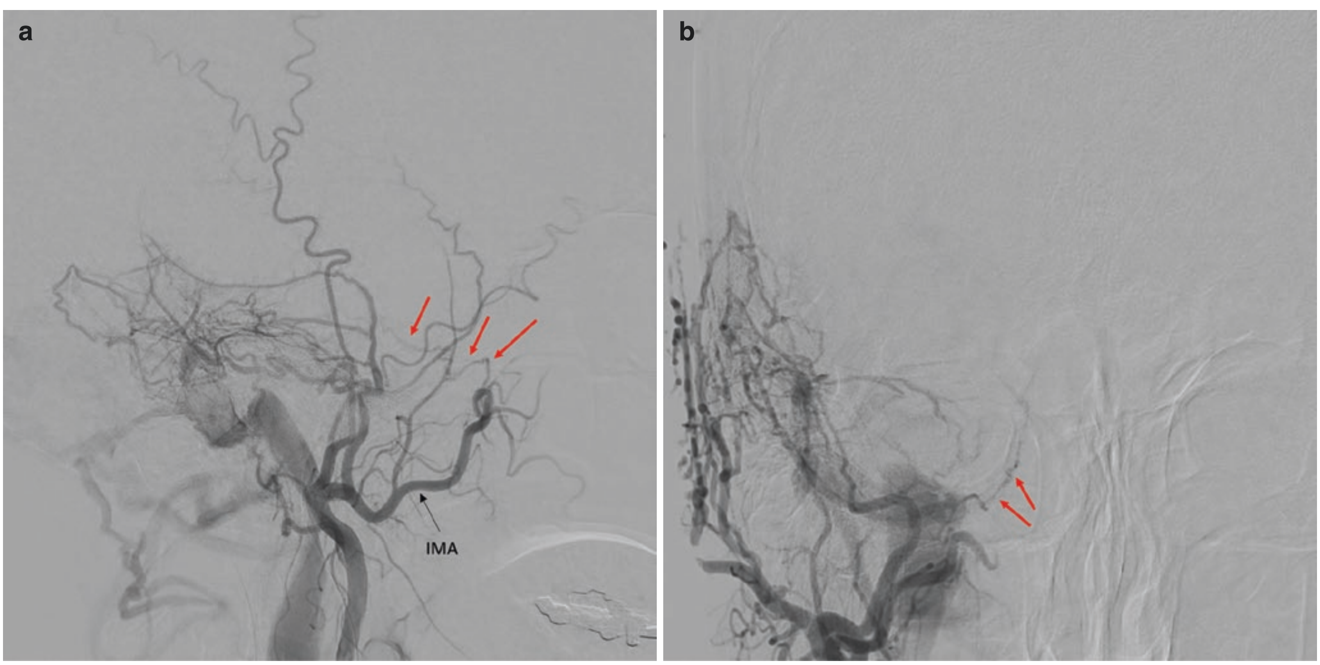

Fig. 2 DSA showing the artery of the foramen rotundum. Lateral (**a**) and anteroposterior view (**b**) obtained after injection of the right external carotid artery. The internal maxillary artery (IMA) gives origin to the artery of the foramen rotundum (red arrows) with its posterosuperior course

References

1. Geibprasert S, Pongpech S, Armstrong D, Krings T. Dangerous extracranial-intracranial anastomoses and supply to the cranial nerves: Vessels the neurointerventionalist needs to know. AJNR Am J Neuroradiol. 2009;30(8):1459–68. https://doi.org/10.3174/ajnr.A1500.
2. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer edition; 2001.
3. Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A Jr. Microsurgical anatomy of the dural arteries. Neurosurgery. 2005;56(2 Suppl):211–51.; discussion 211−251. https://doi.org/10.1227/01.neu.0000144823.94402.3d.
4. Baumel JJ, Beard DY. The accessory meningeal artery of man. J Anat. 1961;95:386–402.
5. Dilenge D, Geraud G. Accessory meningeal artery. Acta Radiol Suppl. 1976;347:63–9. https://doi.org/10.1177/0284185175016s34709.
6. Doyon D, Markovits P, Aaron C, Simon J, Richard J, Gasquet V, et al. Radio-anatomic study of the external carotid. J Radiol Electrol Med Nucl. 1967;48(5):301–6.
7. Lasjaunias P, Theron J. Radiographic anatomy of the accessory meningeal artery. Radiology. 1976;121(1):99–104. https://doi.org/10.1148/121.1.99.
8. Gulses A, Oren C, Altug HA, Ilica T, Sencimen M. Radiologic assessment of the relationship between the maxillary artery and the lateral pterygoid muscle. J Craniofac Surg. 2012;23(5):1465–7. https://doi.org/10.1097/SCS.0b013e3182575793.
9. Navarro JA, Filho JL, Zorzetto NL. Anatomy of the maxillary artery into the pterygomaxillopalatine fossa. Anat Anz. 1982;152(5):413–33.
10. Orbay H, Kerem M, Unlu RE, Comert A, Tuccar E, Sensoz O. Maxillary artery: Anatomical landmarks and relationship with the mandibular subcondyle. Plast Reconstr Surg. 2007;120(7):1865–70. https://doi.org/10.1097/01.prs.0000287137.72674.3c.
11. Lasjaunias P, Berges O, Doyon D. Collateral circulation of the internal maxillary artery. J Neuroradiol. 1979;6(3):197–205.
12. Rinaldo L, Brinjikji W. Dangerous extracranial-intracranial anastomoses: What the interventionalist must know. Semin Interv Radiol. 2020;37(2):140–9. https://doi.org/10.1055/s-0040-1709155.
13. Margolis MT, Newton TH. Collateral pathways between the cavernous portion of the internal carotid and external carotid arteries. Radiology. 1969;93(4):834–6. https://doi.org/10.1148/93.4.834.
14. Salamon G, Guerinel G, Demard F. Radioanatomical study of the external carotid artery. Ann Radiol (Paris). 1968;11(3):199–215.

Dural Branches of the Internal Carotid Artery

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The internal carotid artery (ICA) is principally known to be one of the most important arteries to supply the brain parenchyma; its dural territory is less known. Almost all the dural branches of the ICA originate from the cavernous segment of the ICA but not only from this segment. The dural territory of the ICA, which is the parasellar region, is in balance with other dural arteries [1]. This dural region presents a lot of arterio-arterial anastomoses and consequently numerous anatomical variations [2].

History

Even if Luschka already described in 1860 the presence of intracavernous branches of the internal carotid artery (ICA), their origin, course, and function remained unknown before the anatomic works of McConnell (1953), Stattin (1961), and Parkinson (1964) [3–5]. In 1956, Bernasconi and Cassinari were the first to describe the tentorial artery in seven cases of meningiomas but thought that the artery originates from the external carotid artery (ECA) [5]. After them, other authors showed that the tentorial artery is not specific for tentorial meningiomas but could also be enlarged in case of arterio-venous malformation (AVM) [6] or glioblastoma [6]. Stattin (1961), analyzing ten cases of meningiomas, showed that the tentorial artery arises from the cavernous portion of the ICA and not from the ECA [7]. The most complete anatomic works on dural branches of the ICA were proposed by Parkinson in 1964 (200 cadaveric dissections) and Rhoton in 2005 [5, 8]. Manelfe (1972 and 1974) proposed an interesting angiographic analysis of these branches [9, 10]. Finally, as for other craniofacial arteries, P. Lasjaunias furnished important knowledge and understanding in the embryological origin and also in the anastomosis of these dural branches [1, 2, 11–13].

Embryology

Dural branches of the ICA arise principally from its cavernous segment and are almost all the remnants of an embryonic artery that partially regressed during the embryologic life. Other than the general development of the ICA, the knowledge of the primitive maxillary artery (PMA), the trigeminal artery (TA), and the primitive dorsal ophthalmic artery (PDOA) is important to understand different dural branches of the ICA, their possible anastomoses, and variants [14].

Primitive Maxillary Artery

The PMA is one of the first embryonic branches of the ICA noted by Padget [15]. She thought that this branch was one of the precursors of the orbital supply. It appears before the first stage of Padget (2–3 mm) and disappears during the first stage of Padget (4–5 mm). It originates from the medial surface of the carotid siphon and could have a common origin with the TA. The PMA is the precursor of the inferior hypophyseal artery [16, 17].

Trigeminal Artery

The TA is one of the carotid-basilar anastomoses and is an embryological artery. It appears at the 4–5 mm embryos (Stage I of Padget) and disappears at the 12 mm embryos (Stage III of Padget) [15]. Its origin on the basilar artery is between the superior and the anteroinferior cerebellar arteries, passes medial to

Expert comment by S. Smajda

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the gasserian ganglion, and follows the trigeminal nerves to the primitive carotid artery (junction of the fourth and fifth segments of the carotid artery). The carotid remnant of the TA will be the lateral clival artery and the tentorial artery [1].

Ophthalmic Artery

The embryogenesis of the PDOA depends on two different arteries during 4–18 mm stages, the PDOA and the primitive ventral ophthalmic artery (PVOA) [15]. The PDOA develops from the cavernous segment of the primitive ICA, and the PVOA develops from the anterior division of the primitive ICA. The PDOA penetrates the orbit through the superior orbital fissure (SOF), and the PVOA penetrates through the optic canal [15].

Then, two major anastomoses between these two arteries are formed. The first one is an intraorbital plexiform anastomosis supplied by the two arteries around the optic nerve (future second segment of the ophthalmic artery). The second anastomosis is intradural between the PVOA and the primitive carotid artery to form the definitive supraclinoidal origin of the ophthalmic artery (OA). The proximal portion of the PVOA (between its origin on the anterior cerebral artery (ACA) and its anastomosis with the ICA) then regresses to give the adult stem of the OA [11].

In a following step, the proximal part of the PDOA regresses, and its remnant becomes the inferolateral trunk (ILT) of the primitive carotid artery [11].

Dural Branches

The dural branches of the ICA, which are described below, are illustrated in Fig. 1.

Meningohypophyseal Trunk

The meningohypophyseal trunk (MHT) is the largest cavernous branch of the ICA [8]. Its origin is on the posteromedial surface of the ICA at the level of the posterior genu in its cavernous segment [8, 18]. Table 1 summarizes branches and anastomosis of the MHT. Parkinson noted its presence in 100% of its 200 cadaveric dissections with a common origin with the ILT in 6% [5]. The MHT is composed of three arteries: (1) the tentorial artery, (2) the lateral clival artery, and (3) the inferior hypophyseal artery. Lasjaunias highlighted the different embryologic origins of these arteries, differentiating the inferior hypophyseal artery as remnant of the PMA and the two other arteries as remnants of the TA [1, 13]. For this reason, Lasjaunias disagrees with Parkinson who noted the common origin of these three arteries in almost all cases and only in 10% for Lasjaunias (1978) [2]. The majority of authors who presented parasellar region dissections highlighted moreover the presence of side-by-side anastomosis of all branches of the MHT [9, 10].

1. Tentorial artery

   The tentorial artery is the carotid remnant of the TA and is composed of two branches: the medial and the lateral tentorial arteries [8].

   (a) The medial tentorial artery, also called the artery of Bernasconi and Cassinari or the marginal tentorial artery, courses on the tentorial incisura until the falcotentorial region. This artery supplies the third and fourth cranial nerves, the posterior wall of the cavernous sinus, and the medial third of the tentorium [8]. Even if the MHT is the most frequent origin, the medial tentorial artery could also arise from the ILT, the accessory meningeal artery, the OA, or the middle meningeal artery (MMA) [2]. The medial tentorial artery presents a lot of anastomoses with other dural branches: its opposite side, the jugular branch of the ascending pharyngeal artery, the medial branch of the vertebral artery, and, possibly, dural branches of the OA [9, 10, 19].

   (b) The lateral tentorial artery, also called the basal tentorial artery, courses on the lateral part of the tentorium with the superior petrosal sinus. It supplies the lateral third of the tentorium and presents mainly anastomoses with the petrosal and petrosquamosal branches of the MMA [8].

2. Lateral clival artery

   The lateral clival artery is also known as the dorsal meningeal artery and is also considered as a carotid remnant of the TA. Its origin is from the MHT in 90% and directly from the ICA in 6% [5]. This artery principally supplies the dura of the dorsum sellae and clivus with abundant anastomoses with its mate from the other side, with the jugular branch of the ascending pharyngeal artery and with dural branches of the vertebral artery [5, 8]. The lateral clival artery has two main branches: the medial one, which also supplies the dura of the Dorello's canal and the sixth cranial nerve, and the lateral one, which supplies the trigeminal nerve and the dura of the Meckel's cave.

3. Inferior hypophyseal artery

   The inferior hypophyseal artery is considered as the remnant of the PMA [8]. Its origin could be from the MHT or directly from the posterior genu of the ICA. The two branches (superior and inferior) supply the dura of the sella and posterior clinoid process and also the posterior hypophysis. It also presents anastomoses with the contralateral inferior hypophyseal artery [5].

Even if the MHT is the largest dural branch arising from the ICA, its visualization remains difficult in normal digital subtraction angiography (DSA). A case of dural arteriovenous fistula (dAVF) in which the MHT is enlarged and visible with its branches is shown in Fig. 2.

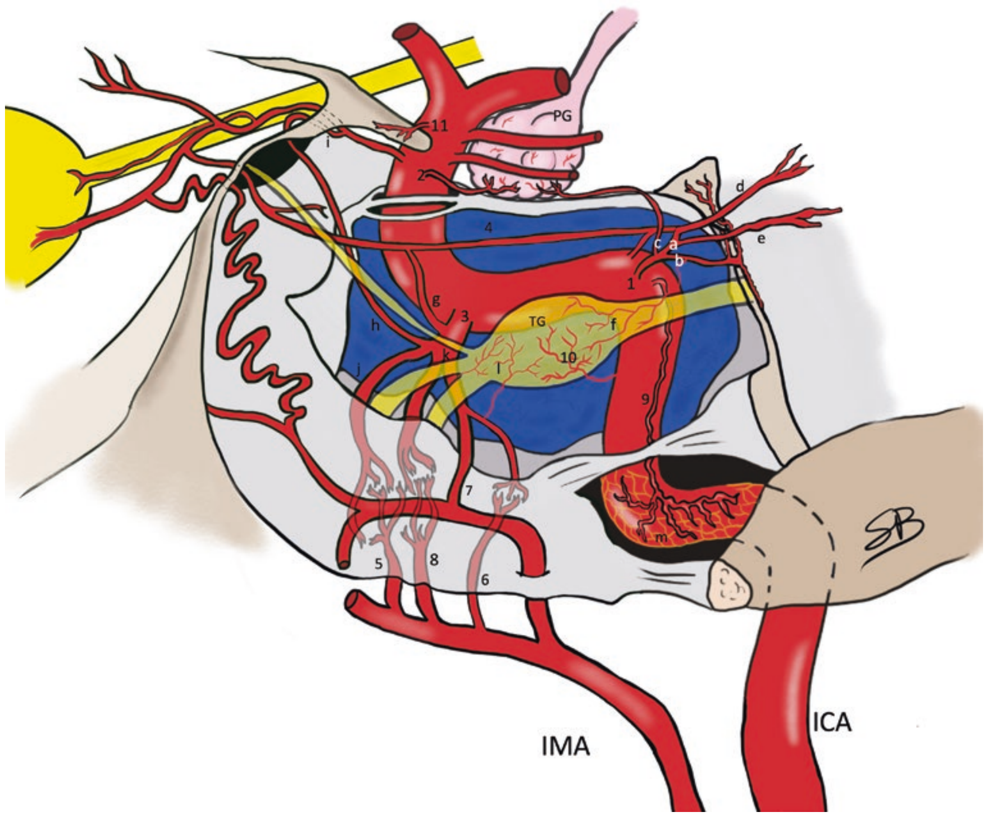

Fig. 1 Dural branches of the internal carotid artery (ICA). The meningohypophyseal trunk (MHT, 1) is the major dural trunk origin from the cavernous ICA. It gives three main branches: the tentorial (a), lateral clival (b), and inferior hypophyseal artery (c). The tentorial artery divides into two: medial (d) and lateral tentorial arteries (e). The lateral clival artery supplies the dura of the dorsum sellae and clivus and gives branches (f) to the lateral third of the trigeminal ganglion (TG). The inferior hypophyseal supplies the pituitary gland (PG), together with the superior hypophyseal artery (2). The inferolateral trunk (ILT, 3) originates from the inferolateral aspect of the cavernous ICA. It gives a superior branch (g) that supplies the roof of the cavernous sinus and can be the origin of the marginal tentorial artery (4); an anteromedial branch (h), which is the remnant of the primitive dorsal ophthalmic artery (PDOA) and anastomoses with the deep recurrent ophthalmic artery (i); an anterolateral branch (j) that anastomoses with the artery of the foramen rotundum (5), which is a branch of the internal maxillary artery (5); and a posterior branch (k), which is composed of two ramus (medial and lateral), which give branches (l) that supply the medial third and the mid-third of the TG. The posterior branch of the ILT has anastomoses with the accessory meningeal artery (6), the cavernous branch of the middle meningeal artery (MMA) (7), and the branch of the MMA that reaches the foramen ovale (8). The recurrent artery of the foramen lacerum (9) arises from the cavernous segment of the ICA proximal to its posterior genu and proximal to the origin of the MHT. It descends until the foramen lacerum and supplies the pericarotid autonomic plexus (m). The lateral TA (10) originates from the vertical portion of the cavernous ICA and supplies the lateral part of the TG. The artery of the clinoid process (11) originates from the sovraclinoid ICA and supplies the anterior clinoid process and the adjacent dura

Table 1 Dural branches of the meningohypophyseal trunk (MHT) with their respective territory and anastomosis

ICA trunk	ICA branch	Origin from the ICA	Territory (dural and neural)	Possible anastomosis
Meningohypophyseal trunk	Medial tentorial artery	Cavernous segment Posterior genu	Cranial nerves: III and IV Roof cavernous sinus Medial third tentorium	Opposite side Deep recurrent OA Ascending pharyngeal A Vertebral artery
	Lateral tentorial artery		Lateral third tentorium Superior petrosal sinus	Petrosal branch MMA Petro-squamous branch MMA
	Dorsal meningeal artery		Cranial nerve: V and VI Dorello's canal and Meckel's cave Dorsum sellae and upper clivus	Opposite side Ascending pharyngeal A Vertebral artery
	Inferior hypophyseal artery		Posterior hypophysis Sella and posterior clinoid process	Opposite side Capsular arteries

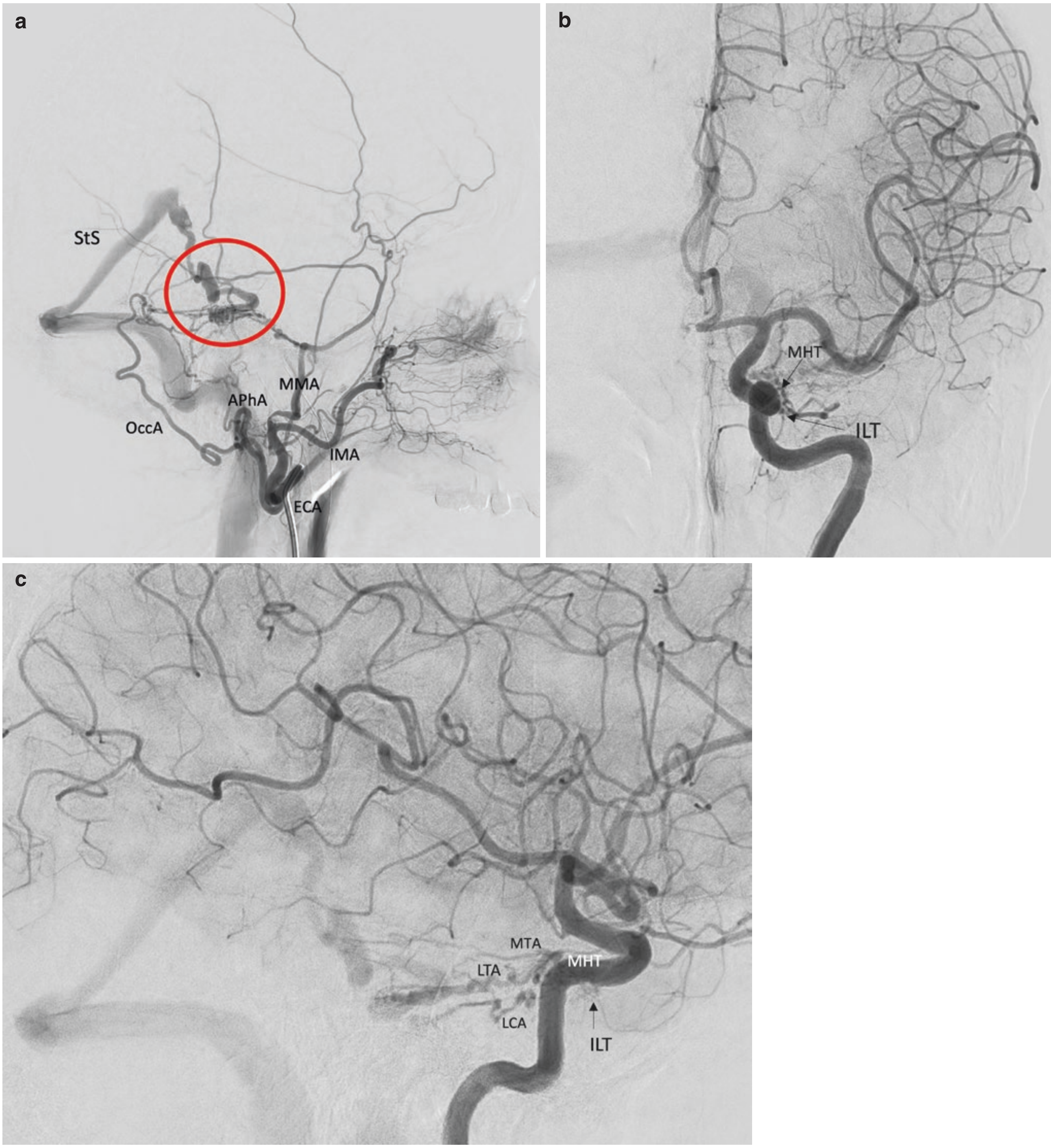

Fig. 2 Case of dural arteriovenous fistula (dAVF) supplied by branches of meningohypophyseal trunk (MHT). The MHT and the inferolateral trunk (ILT) are usually difficult to see in a normal DSA. However, they can be enlarged and consequently visible in the case of dAVFs. The figure (**a**) shows a case of dAVF (red circle) located at the petrous apex on the left side. The major feeders come from the external carotid artery (ECA), especially from dural branches of the occipital artery (OccA), the ascendent pharyngeal artery (APhA), and the middle meningeal artery (MMA), which originates from the internal maxillary artery (IMA), which is also visible. The venous drainage is from the superior petrous vein, the transverse pontine vein, and later into the Galen vein and the straight sinus (StS). Figures (**b**) and (**c**) show, respectively, an anteroposterior and lateral view after a left internal carotid artery (ICA) injection. In both the projections, the MHT and ILT are visible. The MHT divides into the marginal tentorial artery (MTA), the lateral tentorial artery (LTA), and the lateral clival artery (LCA). The inferior hypophyseal artery is not visible. The ILT is visible at its origin from the ICA on its inferolateral aspect

Inferolateral Trunk

The ILT or artery of the inferior cavernous sinus is present in 80% of cases [5, 18]. The ILT is considered to be the carotid remnant of the PDOA, and its function is limited to a dural and neural supply [2]. The ILT arises from the lateral surface of the horizontal portion of the cavernous ICA, directly from the ICA in 94%, and shares a common origin with the MHT in 6% [5]. The ILT courses above the sixth cranial nerve in 96% and below in 4% [8]. This is an important source of anastomoses with other dural arteries of the parasellar region and is composed by four different branches (Table 2).

1. The superior branch supplies the roof of the cavernous sinus and the third and fourth cranial nerves and could be the origin of the marginal tentorial artery [1].
2. The anteromedial branch, which is the remnant of the PDOA, has an anterior course until the SOF where it anastomoses with the deep recurrent OA. It principally supplies the dura of the SOF, the third, fourth, and sixth cranial nerves into the SOF [13].
3. The anterolateral branch courses laterally until the foramen rotundum where it anastomoses with the artery of the foramen rotundum (branch of the internal maxillary artery). This branch supplies the dura of the temporal fossa and the maxillary ramus of the trigeminal nerve (V2). Other than its anastomosis with the artery of the foramen rotundum, it anastomoses with the temporal branch of the MMA [1].
4. The posterior branch is composed of two ramus (medial and lateral), which, respectively, supply the medial third and the mid-third of the gasserian ganglion (the lateral third is supplied by the lateral clival artery) [2, 11]. The medial ramus also supplies the sixth cranial nerve at its entrance in the cavernous sinus. The posterior branch of the ILT has anastomoses with the accessory meningeal artery and the cavernous branch of the MMA.

Capsular Arteries

The capsular arteries (McConnell's arteries) are little branches that arise from the medial wall of the cavernous ICA distal to the origin of the ILT (Table 3) [8]. These arteries are presented in 8–50% of the cavernous sinus and are divided in two groups: inferior and anterior [8, 18]. Inferior arteries are proximal and supply the medial wall of the cavernous sinus and the sella, whereas anterior arteries are more distal and supply the anterior wall of the sella. These branches present anastomoses with contralateral capsular arteries and branches of the inferior hypophyseal artery [1].

Table 3 Other dural branches of the internal carotid artery (ICA) with their respective territory and anastomosis

ICA branch	Origin from the ICA	Territory (dural and neural)	Possible anastomosis
Capsular arteries	Cavernous segment Horizontal segment	Sella turcica Anterior part dorsum sellae Medial wall cavernous sinus	Opposite side Inferior hypophyseal artery
Recurrent artery of the foramen lacerum	Cavernous segment Vertical segment	Pericarotid autonomic nervous system	Ascending pharyngeal A (carotid branch) ILT (posterior branch) MMA (cavernous branch)
Lateral artery of the trigeminal ganglion	Cavernous segment Vertical segment	Trigeminal ganglion Meckel's cave	MHT (lateral clival artery)
Artery of the anterior clinoid process	Supraclinoid segment	Anterior clinoid process	Posterior ethmoidal artery Middle meningeal artery

Table 2 Different dural branches of the inferolateral trunk (ILT) with their respective territory and anastomosis

ICA Trunk	ICA branch	Origin from the ICA	Territory (dural and neural)	Possible anastomosis
Inferolateral trunk	Superior branch	Cavernous segment Horizontal segment	Cranial nerves: III and IV Roof cavernous sinus	Superficial recurrent OA Marginal tentorial artery
	Anteromedial branch		Cranial nerves: III, IV and VI Superior orbital fissure dura	Deep recurrent OA
	Anterolateral branch		Cranial nerves: V2 Temporal fossa dura	Artery of the foramen rotundum (IMA) Temporal branch (MMA)
	Posterior branch		Cranial nerves: V and VI Meckel's cave	Accessory meningeal artery (IMA) Cavernous branch (MMA)

Recurrent Artery of the Foramen Lacerum

The recurrent artery of the foramen lacerum arises from the cavernous segment of the ICA proximal to its posterior genu and proximal to the origin of the MHT (Table 3) [1]. This little dural branch has a recurrent course until the foramen lacerum to supply pericarotid autonomic nervous system. This branch has anastomosis with the carotid branch of the ascending pharyngeal artery, the posterior branch of the ILT, and the cavernous ramus of the MMA [8].

Lateral Artery of the Trigeminal Ganglion

This is a little branch that arises from the vertical portion of the cavernous segment and participates in the supply of the trigeminal ganglion (with the ILT) and the adjacent dura mater (Table 3) [1].

Artery of the Anterior Clinoid Process

The artery of the anterior clinoid process is the lonely dural branch of the ICA that does not arise from its cavernous segment (Table 3). Its origin is on the supraclinoid segment of the ICA in majority of cases but could also arise from the ICA bifurcation [20]. This artery is directed retrogradely through the external membrane of the arachnoid to supply the anterior clinoid process where its function is in balance with territories of posterior ethmoidal and middle meningeal arteries [20]. The embryologic origin of this artery remains unknown, but one can wonder if it could be the remnant of the PVOA proximal to its anastomosis with the ICA.

Dural Supply

With the three principal meningeal branches described before, the dural territory of the ICA corresponds to the dura of the sella, the cavernous sinus, the anterior clinoid process, the temporal dura, the upper part of the clivus, and the majority of the tentorium [8]. Figure 3 illustrates this dural territory with respective ICA branches. This vascular territory is very variable, and the ICA is in balance with other meningeal arteries of this region that will be discussed in their respective chapter. These arteries are principally the MMA (cavernous ramus), the accessory meningeal artery, the OA, the ascending pharyngeal artery, and the vertebral artery [2, 11, 13].

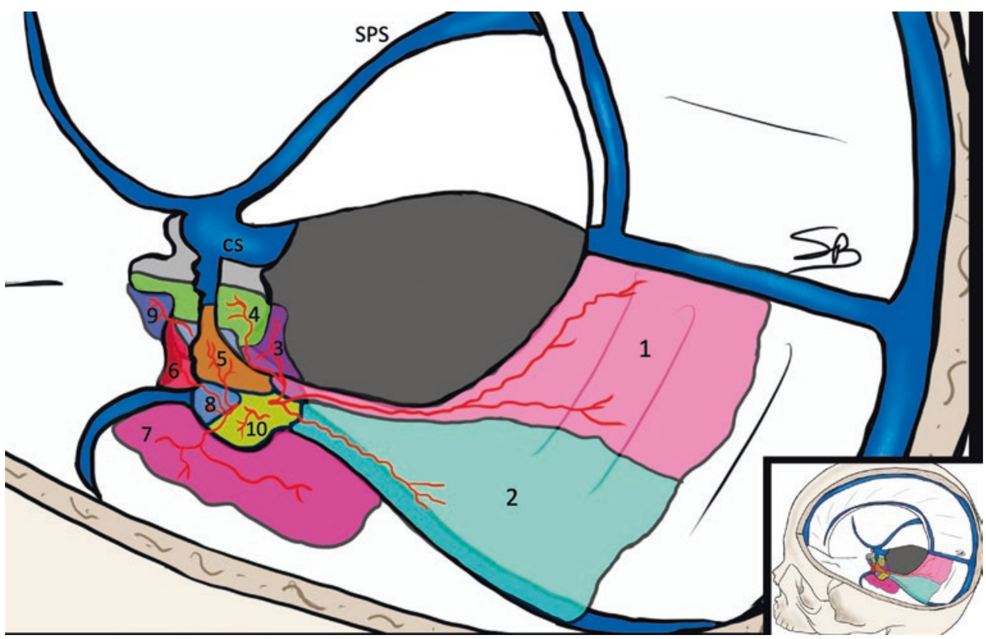

Fig. 3 Dural territory supplied by internal carotid artery (ICA) dural branches. The illustration shows the dural territory of the main ICA dural branches. The marginal tentorial artery courses along the free margin of the tentorium and supplies the posterior wall of the cavernous sinus (CS) and the medial third of the tentorium (1); the lateral tentorial artery supplies the lateral part of the tentorium (2), coursing along the superior petrosal sinus (SPS). The lateral clival artery supplies the dorsum sellae and the upper clival region (3). The inferior hypophyseal artery supplies the dura of the sellae, including the posterior clinoid process (4). The superior branch of the inferolateral trunk supplies the roof of the cavernous sinus (5); the anteromedial branch usually reaches the superior orbital fissure and the adjacent dura (6); and the anterolateral branch supplies the dura of the temporal fossa (7). The capsular branches (8) supply the dura of the anteroinferior part of the cavernous sinus and the medial part of the sella. The lateral trigeminal artery contributes not only to the vascularization of the trigeminal ganglion but also to the lateral wall of the cavernous sinus (10). The artery of the clinoid process is the only dural branch that does not arise from the cavernous tract of the internal carotid artery and usually supplies the dura of the anterior clinoid process (9)

Variations in Dural Branches of the ICA

ICA Origin of the Marginal Tentorial Artery

The marginal tentorial artery (or artery of the free margin of the tentorium cerebelli) normally is a branch of the meningohypophyseal trunk, but its origin is variable. This artery supplies the medial third of the tentorium, partially the walls of the cavernous sinus, and also the transdural segment of the oculomotor and trochlear nerves [1]. Marginal tentorial arteries arising from the MMA, the accessory meningeal artery, the ICA, or the ILT were also described [21, 22]. Figure 4 shows a case of OA origin of the marginal tentorial artery.

Intracavernous Origin of the Ophthalmic Artery

The intracavernous origin of the OA is a rare variation, and its incidence is estimated to be about 0.4% [23]. The first author who described this variation was Dilenge in 1965, and after him, few case reports have been published [24–28]. Lasjaunias et al. (1977) described a case of intracavernous origin and explained this anatomical variation by the embryological regression of the PVOA instead of the PDOA, supporting his theory about the embryogenesis of the OA [13, 21, 25, 27, 29, 30]. On the other hand, authors that support the theory of Padget explain this anatomical variation by the persistence of an anastomosis between the first segment of the OA and the ILT (vestiges of the PMA) with consequent regression of stems from both PDOA and PVOA [23, 26, 31–33]. This anastomotic vessel is so-called deep recurrent OA [26]. Whatever theory you support and consequently how you call this anatomic variation (persistent PDOA or cavernous origin of the OA), this artery always arises from the lateral aspect of the horizontal segment of the ICA and penetrates the orbit through the medial part of the SOF. A rare case of intracavernous origin of the OA is shown in Fig. 5.

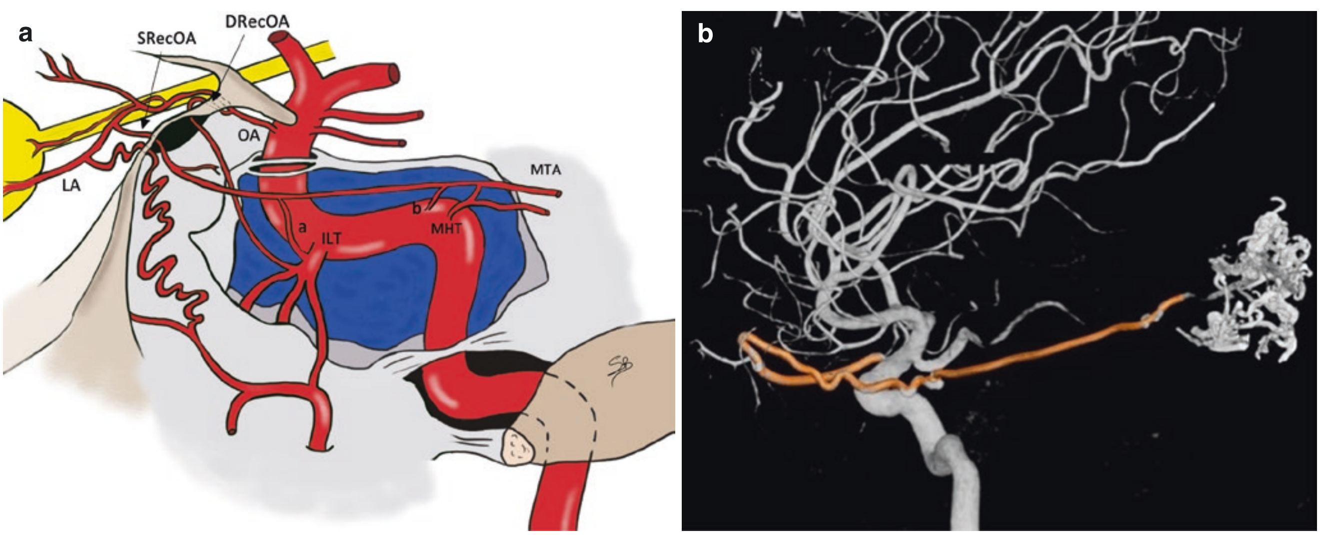

Fig. 4 Case of marginal tentorial artery (MTA) origin from the ophthalmic artery (OA). The MTA, also called artery of the free margin of the tentorium or artery of Bernasconi-Cassinari, may have different origins, which are shown in the graphic representation. It can arise from the lacrimal artery (LA) within the orbit, through the superficial recurrent ophthalmic artery (SRecOA), from the inferolateral trunk (ILT) through its superior branch (**a**), directly from the internal carotid artery (**b**) and from the meningohypophyseal trunk (MHT). The artery courses posterolaterally along the free margin of the tentorium. The figure also shows a 3D DSA reconstruction of a rare case of MTA (highlighted in red) origin from the OA. In this case, the MTA exits the orbit through the superior orbital fissure and directs posteriorly to feed an arteriovenous malformation

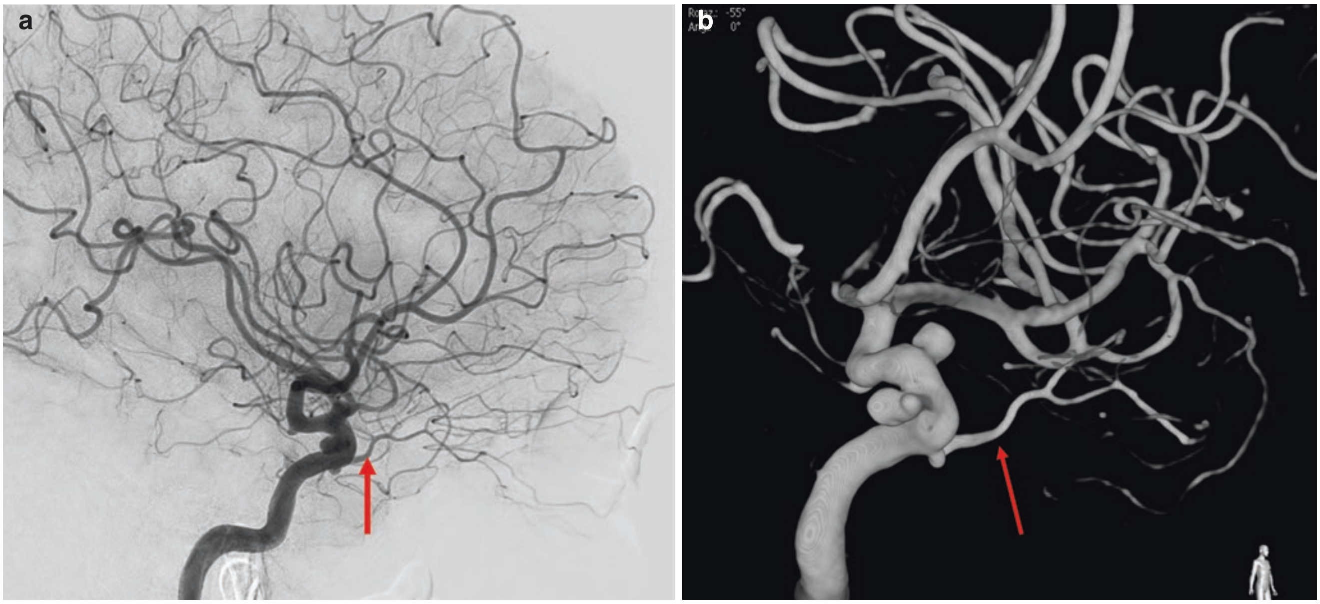

Fig. 5 Case of intracavernous origin of the ophthalmic artery (OA). The figure shows the case of a patient who underwent DSA to study the cavernous internal carotid artery (ICA) aneurysms occasionally discovered (**a**) during an MRI. The lateral and 3D view show the origin of the OA (red arrows) from the cavernous segment of the ICA (**b**). This variant is explained as the persistency of the primitive dorsal ophthalmic artery instead of the primitive ventral ophthalmic artery as definitive ophthalmic artery. In this case, the OA enters the orbit through the superior orbital fissure instead of the optic canal

Clinical Implications

Even if surgical approaches of the cavernous sinus have been largely described during the second half of the twentieth century, their indications are today very limited or completely vanished thanks to results of endovascular therapy for vascular pathologies and to radiosurgery for oncologic pathologies. The knowledge of dural branches of the ICA and their possible anastomoses remains important for physicians who treat the following pathologies.

Carotid-Cavernous Fistulas

Carotid-cavernous fistulas are generally treated by venous approach of the cavernous sinus through the inferior petrosal sinus [8]. Even if its uncommon, arterial embolization could be necessary through the MMA or branches of the ascending pharyngeal artery [8]. In these cases, a detailed knowledge of arterio-arterial anastomosis is mandatory to avoid postoperative cranial nerve deficit or liquid embolic agent migration into the ICA.

Lateral Skull Base and Tentorial Meningiomas

Meningioma of the lateral skull base is principally supplied by an enlarged MMA, but the surgeon has to remember the possible anastomosis of the MMA with branches of the ICA to prevent important blood loss [8]. Meningiomas of the anterior tentorium could have an important supply by the ICA through the tentorial artery. This is important information that could influence the surgical strategy (lateral approach instead of posterior one).

Dural Arteriovenous Fistulas

Subtemporal and tentorial dAVF are generally supplied by dural branches of the ICA. As for the treatment of carotid-cavernous fistulas, a detailed knowledge of these branches, their anastomosis, and function (in particular in the cranial nerve supply) is mandatory to avoid complications and inadvertent liquid agent embolism [8].

Expert Comment

In this chapter, the authors describe and discuss a little known part of the anatomy of the ICA: its dural branches. Knowledge of these anatomical particularities is crucial in the management of numerous pathologies, both neurovascular and craniofacial. As highlighted by the authors, the fact that nearly all of these branches are the remnant of embryonic arteries that regress (at least partially) during growth warns us of their functional importance especially as their identification in non-pathological conditions is difficult due to their small size and their location at the level of the cranial base.

The synthetic, clear, and practical description provided by the authors will help the reader to navigate through this complex region where the overlap in vascular territories is the rule. Their careful anatomic description based on a critical review of the current published literature provides to the clinicians the knowledge needed to understand the different anatomical variants and to anticipate the potential technical problems when treating various pathologies in the involved regions.

We congratulate the authors for their logical presentation, cogent discussion of anatomical variants, and superb illustrations with this complex vascular anatomy.

S. Smajda.

References

1. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical Neuroangiography. Berlin: Springer edition; 2001.
2. Lasjaunias P, Moret J, Doyon D, Vignaud J. C5 collaterals of the internal carotid siphon: Embryology, angiographic anatomical correlations, pathological radio-anatomy (author's transl). Neuroradiology. 1978;16:304–5. https://doi.org/10.1007/bf00395282.
3. Margolis MT, Newton TH. Collateral pathways between the cavernous portion of the internal carotid and external carotid arteries. Radiology. 1969;93(4):834–6. https://doi.org/10.1148/93.4.834.
4. McConnell EM. The arterial blood supply of the human hypophysis cerebri. Anat Rec. 1953;115(2):175–203. https://doi.org/10.1002/ar.1091150204.
5. Parkinson D. Collateral circulation of cavernous carotid artery: Anatomy. Can J Surg. 1964;7:251–68.
6. Cortes O, Chase NE, Leeds N. Visualization of tentorial branches of the internal carotid artery in intracranial lesions other than meningiomas. Radiology. 1964;82:1024–8. https://doi.org/10.1148/82.6.1024.
7. Faure J, Binnert D, Michotey P, Salamon G. Etude radio-anatomique des branches collatéralles de l'artère carotide interne. Neuro-Chirurgie. 1971;18(7):561–79.
8. Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A Jr. Microsurgical anatomy of the dural arteries. Neurosurgery. 2005;56(2 Suppl):211–51.; discussion 211–251. https://doi.org/10.1227/01.neu.0000144823.94402.3d.
9. Manelfe C, Tremoulet M, Roulleau J. Arteriographic study of the intracavernous branches of the internal carotid. Neurochirurgie. 1972;18(7):581–98.
10. Manelfe C, Tremoulet M, Roulleau J. Intracavernous collaterals of the internal carotid artery. 2. Angiographic study. Ann Radiol (Paris). 1974;17(3):268–70.
11. Lasjaunias P, Moret J, Mink J. The anatomy of the inferolateral trunk (ILT) of the internal carotid artery. Neuroradiology. 1977;13(4):215–20. https://doi.org/10.1007/bf00344216.
12. Lasjaunias P, Santoyo-Vazquez A. Segmental agenesis of the internal carotid artery: Angiographic aspects with embryological discussion. Anat Clin. 1984;6(2):133–41.
13. Willinsky R, Lasjaunias P, Berenstein A. Intracavernous branches of the internal carotid artery (ICA). Comprehensive review of their variations. Surg Radiol Anat. 1987;9(3):201–15.
14. Dungan DH, Heiserman JE. The carotid artery: Embryology, normal anatomy, and physiology. Neuroimaging Clin N Am. 1996;6(4):789–99.
15. Padget DH. The development of cranial arteries in the human embryo, Contribution to embryology, vol. 212. Washington: Carnegie Institution; 1948. p. 205–62.
16. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
17. Barry A. The aortic arch derivatives in human adult. Anat Rec. 1951;111(2):221–38.
18. Vasovic L, Trandafilovic M, Vlajkovic S, Radenkovic G. Congenital absence of the bilateral internal carotid artery: A review of the associated (ab)normalities from a newborn status to the eighth decade of life. Childs Nerv Syst. 2018;34(1):35–49. https://doi.org/10.1007/s00381-017-3629-1.
19. Santini JJ, Laffont J, Gouaze A. Intracavernous collaterals of the internal carotid artery. 1. Anatomical study. Ann Radiol (Paris). 1974;17(3):265–6.
20. Salunke P, Singh A, Rekhapalli R. Dural artery from supraclinoid internal carotid artery to anterior clinoid process: Origin, course, and clinical implications. World Neurosurg. 2018;124:e222. https://doi.org/10.1016/j.wneu.2018.12.071.
21. Lasjaunias P, Moret J, Manelfe C, Theron J, Hasso T, Seeger J. Arterial anomalies at the base of the skull. Neuroradiology. 1977;13(5):267–72.
22. Lasjaunias P, Moret J. Normal and non-pathological variations in the angiographic aspects of the arteries of the middle ear. Neuroradiology. 1978;15(4):213–9.
23. Bertelli E, Regoli M, Bracco S. An update on the variations of the orbital blood supply and hemodynamic. Surg Radiol Anat. 2017;39(5):485–96. https://doi.org/10.1007/s00276-016-1776-9.
24. Fiore DL, Pardatscher K, Fiore D, Zuccarello M, Iraci G. Persistent dorsal ophthalmic artery. Report of a case with associated fibromuscular hyperplasia of the extracranial internal carotid artery and multiple cerebral aneurysms. Neurochirurgia (Stuttg). 1981;24(3):106–8.
25. Pretterklieber ML, Schindler A, Krammer EB. Unilateral persistence of the dorsal ophthalmic artery in man. Acta Anat (Basel). 1994;149(4):300–5.
26. Gailloud P, Gregg L, Ruiz DS. Developmental anatomy, angiography, and clinical implications of orbital arterial variations involving the stapedial artery. Neuroimaging Clin N Am. 2009;19(2):169–79. https://doi.org/10.1016/j.nic.2009.02.001.
27. Agarwal N, Singh PL, Karimi RJ, Gandhi CD, Prestigiacomo CJ. Persistent vestige of dorsal ophthalmic artery: A case report. J Neurointerv Surg. 2013;5(4):e25. https://doi.org/10.1136/neurintsurg-2011-010196.
28. Lombardi G. Ophthalmic artery anomalies. Ophthalmologica. 1969;157(5):321–7. https://doi.org/10.1159/000305676.
29. Louw L. Different ophthalmic artery origins: Embryology and clinical significance. Clin Anat. 2015;28(5):576–83. https://doi.org/10.1002/ca.22470.
30. Uchino A, Saito N, Takahashi M, Kozawa E, Mizukoshi W, Nakajima R, et al. Persistent dorsal ophthalmic artery and ophthalmic artery arising from the middle meningeal artery diagnosed by MR angiography at 3 T. Surg Radiol Anat. 2013;35(9):775–82. https://doi.org/10.1007/s00276-013-1085-5.
31. Parlato C, di Nuzzo G, Luongo M, Tortora F, Briganti F. Anatomical variant of origin of ophthalmic artery: Case report. Surg Radiol Anat. 2011;33(3):275–8. https://doi.org/10.1007/s00276-010-0745-y.
32. Toma N. Anatomy of the ophthalmic artery: Embryological consideration. Neurol Med Chir (Tokyo). 2016;56(10):585–91. https://doi.org/10.2176/nmc.ra.2015-0324.
33. Gregg L, San Millan D, Orru E, Tamargo RJ, Gailloud P. Ventral and dorsal persistent primitive ophthalmic arteries. Oper Neurosurg (Hagerstown). 2016;12(2):141–52. https://doi.org/10.1227/NEU.0000000000001066.

Dural Branches of the Anterior and Posterior Cerebral Arteries

Thomas Robert and Sara Bonasia

Dural branches of the anterior and posterior cerebral arteries are not well described in the literature because of their limited clinical implications and because of the difficulty to be highlighted with a digital subtraction angiography [1]. Anatomy and variations of both anterior and posterior cerebral arteries are described in respective chapters, and in the present chapter, their dural branches will only be discussed. Figure 1 illustrates the dural territory supplied by branches of these two arteries.

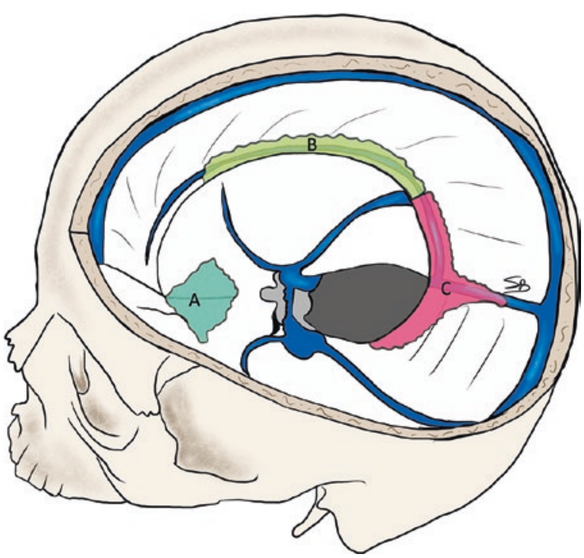

Fig. 1 Dural territory of the anterior cerebral artery (ACA) and posterior cerebral artery (PCA). The Area A shows the dural territory supplied the olfactory branches of the anterior cerebral artery, which corresponds to the middle third of the anterior cranial fossa. The Area B, in correspondence with the free edge of the falx cerebri, is supplied by pericallosal branches of the anterior cerebral artery. The posterior part of the falx cerebri, the falcotentorial junction, and the medial part of the tentorium (Area C) are supplied by the so-called artery of Davidoff and Schechter from the posterior cerebral artery

Dural Branches of the Anterior Cerebral Artery

Dural branches of the anterior cerebral artery (ACA) are two different types: olfactory and pericallosal [1–3]. Table 1 summarizes the origin, course, territory, and anastomoses of these branches.

Olfactory Branches

These dural branches arise from the horizontal segment of the ACA or from the orbitofrontal branch and could be considered as the remnant of the embryonic primitive olfactory artery [4, 5]. The detailed embryology of the primitive olfactory artery is described in chapter "Embryology, Anatomy, and Variations of the Anterior Cerebral Artery." Signorelli et al. (2010) noted surgically the increased in diameter of this olfactory branch in case of meningioma of the planum sphenoidale [5]. The dural territory of this branch is the medial third of the anterior cranial fossa and is in balance with territories of the ethmoidal arteries (from the ophthalmic artery) and of the middle meningeal artery [1]. Possible anastomoses with posterior and anterior ethmoidal artery could be visible angiographically [4].

Pericallosal Branches

These branches of the ACA arise from the distal part of the ACA (pericallosal artery) and course into the epicallosal sulcus to enter into the falx cerebri [1, 3, 4, 6]. The dural territory of these little branches is the free edge of the falx cerebri and is in balance with territories of the anterior fal-

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Table 1 Origin, course, territory, and anastomoses of dural branches of the anterior cerebral artery (ACA)

ACA branches	Origin from the ACA	Course	Territory	Arterio-arterial anastomoses
Olfactory branches	A1 Orbitofrontal A	Cistern of the lamina terminalis	Medial third of the anterior skull base	Anterior ethmoidal artery Posterior ethmoidal artery Middle meningeal artery
Pericallosal branches	A2–A5	Epicallosal sulcus	Free edge of the falx cerebri Inferior sagittal sinus	Anterior falcine artery Dural branch of the PCA Medial tentorial artery

cine artery (from the anterior ethmoidal artery) of the medial tentorial artery (from the internal carotid artery) and of the dural branch of the posterior cerebral artery (PCA) [1].

Dural Branches of the Posterior Cerebral Artery

The principal role of the PCA is the supply of the posterior part of the cerebral hemispheres, but, as the ACA, it could participate in the supply of a little part of the dura mater [4]. Dural branches of the PCA could be summarized in only one artery, which is the artery of Davidoff and Schechter. This artery bears its eponym in the memory of the mentors of the two first physicians who described it in 1965: Wollschlaeger and Wollschlaeger [7]. They dissected ten cadaveric brains and found this dural branch of the PCA in nine of them [3, 7].

The artery of Davidoff and Schechter arises from the pedicular (P1) or ambient (P2) segment of the PCA and turns around the mesencephalon following other PCA branches in the perimesencephalic cisterns [8]. It passes under the free border of the tentorium toward the falcotentorial angle where it pierces the tentorium [8–10]. At this point, the artery of Davidoff and Schechter gives two distinct branches: one anterior and the other posterior [8]. Griessenauer et al. provide the largest cadaveric series about this artery and saw that it is an inconstant artery with an incidence of 25% of sides [11]. It is rarely bilateral (only one case on 20 brains in their series) and is more frequent on the left side. Its mean diameter is 0.8 mm, and its length is 12 mm [11, 12].

On normal digital subtraction angiography, the artery of Davidoff and Schechter is not visible because it is too thin and is superimposed with other PCA branches [4]. It could only be seen in the case of dural arteriovenous fistula or falcotentorial meningioma [1, 9, 12–14]. This artery, when present, is a branch of the PCA, but few reports showed it as a branch of the superior cerebellar artery [15]. The dural territory of this artery is the medial part of the tentorium and the posterior part of the falx cerebri and is in balance with territories of dural branches of the ACA, the middle meningeal artery, and the occipital artery [1, 4, 12].

References

1. Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A Jr. Microsurgical anatomy of the dural arteries. Neurosurgery. 2005;56(2 Suppl):211–51.; discussion 211−251. https://doi.org/10.1227/01.neu.0000144823.94402.3d.
2. Gomes F, Dujovny M, Umansky F, Ausman JI, Diaz FG, Ray WJ, et al. Microsurgical anatomy of the recurrent artery of Heubner. J Neurosurg. 1984;60(1):130–9. https://doi.org/10.3171/jns.1984.60.1.0130.
3. Yasargil MG. Microneurosurgery. New York: Thieme Medical Publisher; 1984.
4. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer edition; 2001.
5. Signorelli F, Scholtes F, Kauffmann C, McLaughlin N, Bojanowski MW. Dural artery from the anterior cerebral artery to the planum sphenoidale. Acta Neurochir. 2010;152(10):1807–8. https://doi.org/10.1007/s00701-010-0716-4.
6. Perlmutter D, Rhoton AL Jr. Microsurgical anatomy of the distal anterior cerebral artery. J Neurosurg. 1978;49(2):204–28. https://doi.org/10.3171/jns.1978.49.2.0204.
7. Wollschlaeger PB, Wollschlaeger G. An infratentorial meningeal artery. Radiologe. 1965;5(11):451–2.
8. Weinstein M, Stein R, Pollock J, Stucker TB, Newton TH. Meningeal branch of the posterior cerebral artery. Neuroradiology. 1974;7(3):129–31. https://doi.org/10.1007/BF00344317.
9. Roman NIS, Rodriguez P, Nasser H, Cox M, Ramchand P, Choudhri O, et al. Artery of Davidoff and Schechter: A large angiographic case series of dural AV fistulas. Neurohospitalist. 2022;12(1):155–61. https://doi.org/10.1177/19418744211022217.
10. Tayebi Meybodi A, Vigo V, Lawton MT, Benet A. The artery of Wollschlaeger and Wollschlaeger: An anatomical-clinical illustration. Br J Neurosurg. 2017;31(5):593–5.
11. Griessenauer CJ, Loukas M, Scott JA, Tubbs RS, Cohen-Gadol AA. The artery of Davidoff and Schechter: An anatomical study with neurosurgical case correlates. Br J Neurosurg. 2013;27(6):815–8. https://doi.org/10.3109/02688697.2013.798856.
12. Hart JL, Davagnanam I, Chandrashekar HS, Brew S. Angiography and selective microcatheter embolization of a falcine meningioma supplied by the artery of Davidoff and Schechter. Case report. J Neurosurg. 2011;114(3):710–3. https://doi.org/10.3171/2010.4.JNS10218.
13. Bhatia KD, Kortman H, Walchli T, Radovanovic I, Pereira VM, Krings T. Artery of Davidoff and Schechter supply in dural arteriovenous fistulas. AJNR Am J Neuroradiol. 2020;41(2):300–4. https://doi.org/10.3174/ajnr.A6380.
14. Puri AS. Dural arteriovenous fistula supplied by the artery of Davidoff and Schechter. Radiol Case Rep. 2010;5(2):375. https://doi.org/10.2484/rcr.v5i2.375.
15. D'Andrea M, Mongardi L, Fuschillo D, Musio A, Commodaro C, Quilis-Quesada V, et al. Vermian subtentorial arteriovenous malformation supplied by the artery of Wollschlaeger and Wollschlaeger. Clin Neurol Neurosurg. 2021;206:106670. https://doi.org/10.1016/j.clineuro.2021.106670.

Dural Branches of the Ophthalmic Artery

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The ophthalmic artery (OA), which will be widely discussed in chapter "Embryology and Variations of the Ophthalmic Artery," is a very fascinating artery for its complex embryological development as well as for the numerous vascular anastomoses developed with branches of the external carotid artery. The role of the OA in the dural supply is not well-known, but the understanding of the dural function of the OA and of its possible variations is a cornerstone for surgical and endovascular treatment of dural pathologies (dural arteriovenous fistulas, skull base meningiomas, and chronic subdural hematoma embolization). In this chapter, we will focus on the dural branches of the OA with special attention to their rare variations, like the OA origin of the middle meningeal artery (MMA) and the OA origin of the marginal tentorial artery (MTA).

History

Meyer (1887), who was considered as pioneer in the orbital vascular anatomy, was the first to precisely describe all branches of the ophthalmic artery including its dural territory [1]. Few years before, Curnow (1873) had already described three cadaveric cases of variations in the origin of the OA [2]. One of these three cases was the first description of an OA origin of the middle meningeal artery (MMA). With the advent of digital subtraction angiography (DSA), Kuru (1967) gave a detailed description of the OA meningeal branches, and after him, few authors focused on the variants of the OA and of the MMA [3–10]. Lasjaunias gave his crucial contribution to the comprehension of the orbital and meningeal vascular supply, combining his knowledge of embryology with an accurate angiographic analysis [7, 8, 11]. The last author that gave a comprehensive description of dural vascularization was Rhoton et al. (2005) based on his large cadaveric dissection experience [12, 13].

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Embryology

A detailed description and different hypotheses concerning the embryology of the OA and orbital vascularization are explained in chapter "Embryology and Variations of the Ophthalmic Artery." In this chapter, we will focus on a few points on the embryological development necessary to understand the variants of the OA dural branches.

The OA starts its development when the embryo is about 4 mm and reaches its adult configuration at about 40 mm. Its development is strictly connected with the arterial embryology of the primitive internal carotid artery (ICA), the stapedial artery (SA), and the pharyngeal artery system. In the last centuries, two authors were interested in the comprehension of the complex events of the OA formation: Padget that formulated her theory after the dissection of 22 embryos and Lasjaunias, who added an accurate angiographic evaluation to Padget's knowledge [11, 14]. Both authors agree that the definitive features of the OA depend mostly on two embryonic arteries: the primitive dorsal ophthalmic artery (PDOA) and the primitive ventral ophthalmic artery (PVOA).

According to Padget's theory, the embryological development of the OA could be divided into six stages, which are summarized in Table 1 [14]. The PDOA appears when the embryo is about 4–5 mm (Stage I), originating from the bifurcation of the primitive ICA. In Stage II (9 mm embryos), while the PDOA enlarges through the optic cup as plexiform channels, the PVOA arises from the cranial division of the primitive ICA. The PDOA and PVOA are then destined to elongate following the ventral shifting of the optic cup and the dorsal

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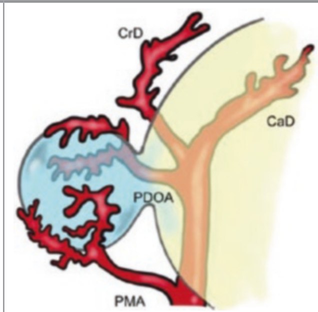
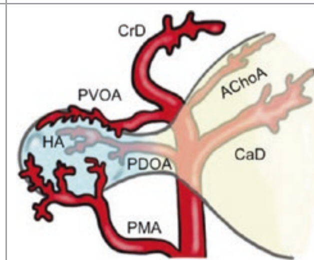
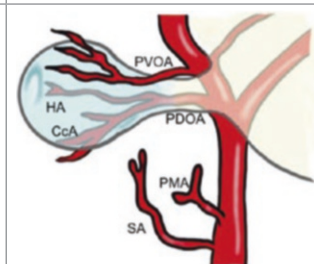
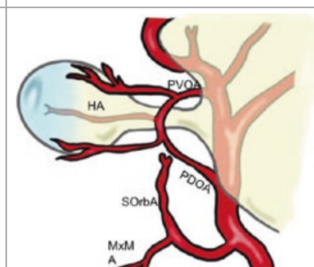
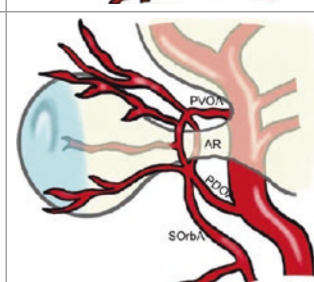
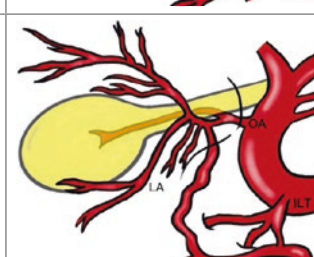
Table 1 Stages of embryological development of the ophthalmic artery (OA) (Padget's concept). (Adapted from [14]. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–262)

Stage	Embryo size (mm)	Events	Graphic representation
I	4–5	• Primitive maxillary artery (PMA) as temporary branch • PDOA appearance	
II	9	• Primitive hyaloid artery (HA) as plexiform channels • PVOA appearance	
III	14	• Formation of primitive hyaloid and common ciliary arteries • Stapedial artery (SA) development	
IV	18	• Migration of the PDOA origin • Formation of the supraorbital branch (SOrbA) of the SA • Regression of the PVOA	
V	20	• Maximal development of the SA • Formation of the anastomotic ring (AR)	
VI	40	• Ventral interruption of the AR • Regression of the SOrbA	

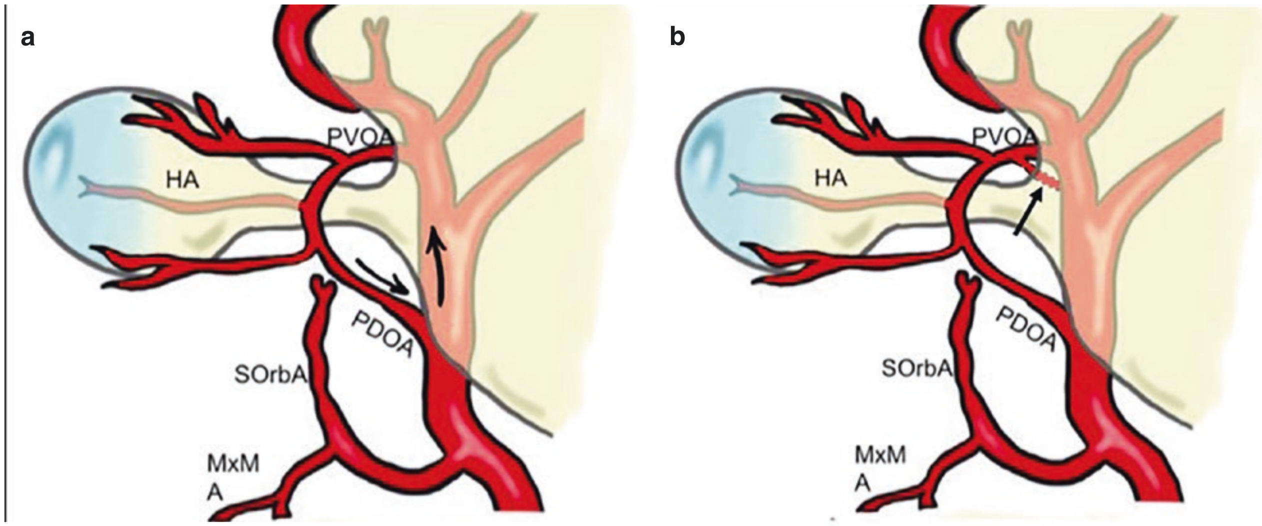


Fig. 1 Padget's and Lasjaunias' theories about ophthalmic artery (OA) origin migration. When the embryo is about 18 mm, the OA reaches its definitive origin on the supraclinoid internal carotid artery (ICA). This phenomenon is explicated by Padget by the cranial elongation of the ICA during this stage with the consequent movement of the primitive dorsal ophthalmic artery (PDOA) origin (black arrows in figure (**a**)). On the other hand, Lasjaunias hypothesized the presence of an intradural anastomosis between the primitive ventral ophthalmic artery (PVOA) and the primitive ICA (black arrow in figure (**b**)) in correspondence of the future origin with successive regression of the original stem

shifting of the cerebral hemispheres. In embryos of 14–17 mm, it is possible to note the appearance of two branches from the PDOA artery: the primitive hyaloid artery (HA) and the common temporal ciliary artery (future lateral posterior ciliary artery). The PVOA gives off at the same time the common nasal ciliary artery (future medial posterior ciliary artery).

In an embryo of about 18 mm (Stage IV), the OA is invested by the process that will bring to the migration of its origin on the supraclinoid ICA. This phenomenon is explained by Padget by the cranial elongation of the ICA during this stage with the consequent movement of the PDOA. On the other hand, Lasjaunias explained this migration through the presence of an intradural anastomosis between the PVOA and the primitive carotid artery in correspondence of the future origin with consequent regression of the original stem [8, 11, 15]. The two theories about OA origin migration are illustrated in Fig. 1.

From Stage I to IV, another artery grows at the same time and contributes to the adult configuration of the orbital arteries: the SA. In the first stages, the optic cup is supplied in its ventral side by the primitive maxillary artery (PMA). However, it starts to regress at the end of Stage II, which is to be substituted by the SA in its orbital territory. This latter gives off two branches that follow the three divisions of the trigeminal nerve: the maxillomandibular artery and the supraorbital artery. The supraorbital artery enters the orbit through the superior orbital fissure and gives two branches: the ethmoido-nasal and the lacrimal arteries.

The relationship between the PDOA, the PVOA, and the SA reaches the highest importance in the last two Stages (V: embryo of 20 mm; VI: embryo of 40 mm). During Stage V, an anastomotic ring appears around the optic nerve, formed by the anastomosis between the PVOA, the PDOA, and the supraorbital artery (through the ethmoido-nasal artery). However, in Stage VI, this ring is ventrally interrupted to give the definitive configuration of the OA. The part of the anastomotic ring that regresses is crucial to determine which of the two primitive OAs persist to form the adult OA. According to Padget, it is the PDOA that persists, with consequent PVOA proximal regression. On the other hand, Lasjaunias wrote that the distal portion of the PDOA regresses, and its proximal part is destined to form the future inferolateral trunk [8, 11, 15]. Thus, in his opinion, it is the PVOA that mostly contributes to the formation of the definitive OA.

At the same time, the extraorbital part of the supraorbital artery regresses, to let the lacrimal artery be annexed by the OA.

Dural Branches of the OA

Different dural branches of the OA and their possible anastomoses with other dural arteries are listed in Table 2; their respective dural territories are illustrated in Fig. 2.

Table 2 Origin of the dural branches of the ophthalmic artery (OA) with their respective supply and anastomoses

OA branches	Origin from the OA	Foramen	Dural territory	Possible anastomosis
Deep recurrent OA	First segment	Superior orbital fissure	Superior orbital fissure (lateral part) Sphenoid wing	Inferolateral trunk (ICA)
Superficial recurrent OA	Second segment	Superior orbital fissure	Anterior clinoid process Lesser sphenoid wing Middle fossa (anteromedial portion)	Posterior ethmoidal artery MMA (anterior division) Medial tentorial artery (ICA)
Anterior ethmoidal artery	Third segment	Anterior ethmoidal canal	Anterior convexity (anterior meningeal artery) Anterior cranial fossa (medial third) Anterior falx cerebri (anterior falcine artery)	Contralateral anterior ethmoidal artery Bilateral MMAs Posterior ethmoidal artery Olfactory branch (ACA)
Posterior ethmoidal artery	Third segment	Posterior ethmoidal canal	Anterior cranial fossa (medial third) Anterior clinoid process Chiasmatic groove	Contralateral posterior ethmoidal artery Anterior ethmoidal artery MMA (anterior division)

Deep Recurrent Ophthalmic Artery

The deep recurrent ophthalmic artery arises from the first segment of the OA and has a recurrent course through the medial part of the superior orbital fissure. This artery supplies the dura of the lateral wall of the cavernous sinus. It consistently anastomoses with the anteromedial branch of the inferolateral trunk and often with the cavernous branch of the MMA and with the accessory meningeal artery. It is considered as the remnant of the PDOA [8].

Superficial Recurrent Ophthalmic Artery

The superficial recurrent ophthalmic artery is a meningeal branch that takes its origin from the proximal part of the lacrimal or directly from the second segment of the OA [8, 11, 16]. This artery passes through the lateral part of the superior orbital fissure to reach the dura over the anterior clinoid process and of the cavernous sinus roof [10, 13]. The superficial recurrent ophthalmic artery also supplies the intradural part of the third and fourth cranial nerves. This artery is the orbital remnant of the supraorbital branch of the SA [11].

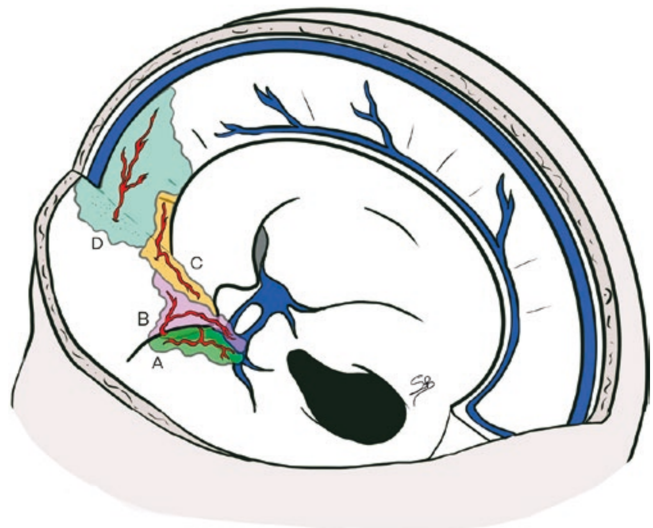


Fig. 2 Dural territories of ophthalmic artery (OA) branches. A (green): territory of the deep recurrent ophthalmic artery, which exits from the medial part of the superior orbital fissure and supplies the dura of the lateral wall of the cavernous sinus. B (pink): dural territory of the superficial recurrent ophthalmic artery, which passes through the lateral part of the superior orbital fissure to reach the dura over the anterior clinoid process and of the cavernous sinus roof; C (orange): the posterior ethmoidal artery passes through the posterior ethmoidal canal to reach the dura of the planum sphenoidale, the posterior cribriform plate, and the anterior clinoid process. D (light blue): the anterior ethmoidal artery passes through the anterior ethmoidal canal, and its meningeal territory consists of the anterior part of the cribriform plate, the medial part of the orbital roofs, and the anterior third of the falx cerebri

Posterior Ethmoidal Artery

The posterior ethmoidal artery is a small meningeal branch that arises from the third segment of the OA and exits the orbit through the posterior ethmoidal canal [16]. Its average diameter is 0.4 mm and is usually in balance with the diameter of the anterior ethmoidal artery [17]. This artery supplies the dura of the planum sphenoidale, the posterior cribriform plate, and the anterior clinoid process [11]. Martins et al. (2005) showed that the posterior ethmoidal artery often anastomoses with dural branches of the ICA, MMA, and anterior ethmoidal artery [13]. When absent (approximately 20% of cases), its meningeal territory is supplied by these three other arteries.

Anterior Ethmoidal Artery

The anterior ethmoidal artery is a more constant artery, which has been found in more than 90% of orbits if the OA crosses over the optic nerve and in 80% of cases when the OA crosses under the nerve. It arises from the distal part of the OA and could give from one to five little branches that pass through the anterior ethmoidal canal. Other than its mucosal supply on the nasal septum and nasal fossa, its meningeal territory is limited to the anterior part of the cribriform plate, the medial part of the orbital roofs, and the anterior third of the falx cerebri. The anterior ethmoidal artery gives a branch, well described angiographically by Kuru (1965), along the falx cerebri that is named the anterior falcine artery or the artery of the falx cerebri [10]. This anterior falcine artery could be present bilaterally, but usually one side is predominant. If the anterior ethmoidal artery is well-developed, it can give some branches called "anterior meningeal arteries" that differ from the anterior falcine artery because of their paramedial course and can supply the dura of the anterior convexity.

Dural Supply of the OA

The four meningeal branches previously described supply the dura of the cribriform plate, the planum, the anterior clinoid process, the superior orbital region, the roof and the lateral part of the cavernous sinus, the medial part of the orbital roof, and the anterior part of the falx cerebri. This vascular territory is very variable, and the OA is in balance with other meningeal arteries of this region like the MMA (cavernous ramus), the accessory meningeal artery, and the inferolateral trunk.

Variations of Dural Branches of the Ophthalmic Artery

Ophthalmic Artery Origin of the Middle Meningeal Artery

In a rare case, the MMA could originate from the OA instead of the internal maxillary artery. The incidence of this vascular variation is estimated to 0.5% by Dilenge et al. (1980) based on a large angiographic series [9]. Few cases of the MMA arising from the OA have been described in the literature. The first case was presented by Curnow (1873), and in the same period, Meyer (1887) also cited four cadaveric cases originally described by Zuckerkandl in 1876 during a congress presentation [1, 2]. Two rare cases of this variation are shown in Fig. 3. This vascular anomaly is considered as the consequence of two different embryologic processes. The first one is the failure of the supraorbital branch (SA) regression. The second one is the absence of anastomosis between the maxillomandibular branch of the SA and the internal maxillary artery. Consequently, the MMA originates from the OA and passes through the lateral part of the superior orbital fissure; thus, the foramen spinosum is usually absent. Maiuri et al. (1998) proposed three different types of this vascular variation as highlighted in Table 3 [18]. The first type is the complete MMA territory supplied by the OA through the superficial recurrent OA. In the second type, only the anterior branch of the MMA originates from the OA, and the posterior branch of the MMA keeps its origin from the internal maxillary artery. The third type is not really an OA origin of the MMA but an anastomosis between the OA and the accessory meningeal artery (through the deep recurrent OA). The consequence is that the anterior meningeal territory is supplied by both the MMA and the OA without any communication. It is still matter of debate if the MMA originates from the OA directly or from the proximal part of the lacrimal artery.

Ophthalmic Artery Origin of the Marginal Tentorial Artery

The marginal tentorial artery (MTA) (or artery of the free margin of the tentorium cerebelli) normally arises from the meningohypophyseal trunk, but its origin is variable, as illustrated in Fig. 4. This artery supplies the medial third of the tentorium, partially the walls of the cavernous sinus, and also the transdural segment of the oculomotor and trochlear nerves [13]. An OA origin of this artery has been described by Lasjaunias (2001), distinguishing two different types [11]. The first one is when the MTA arises from the lacrimal artery. The second one is when the MTA arises directly from the OA and the lacrimal artery originates from the MMA (meningolacrimal type).

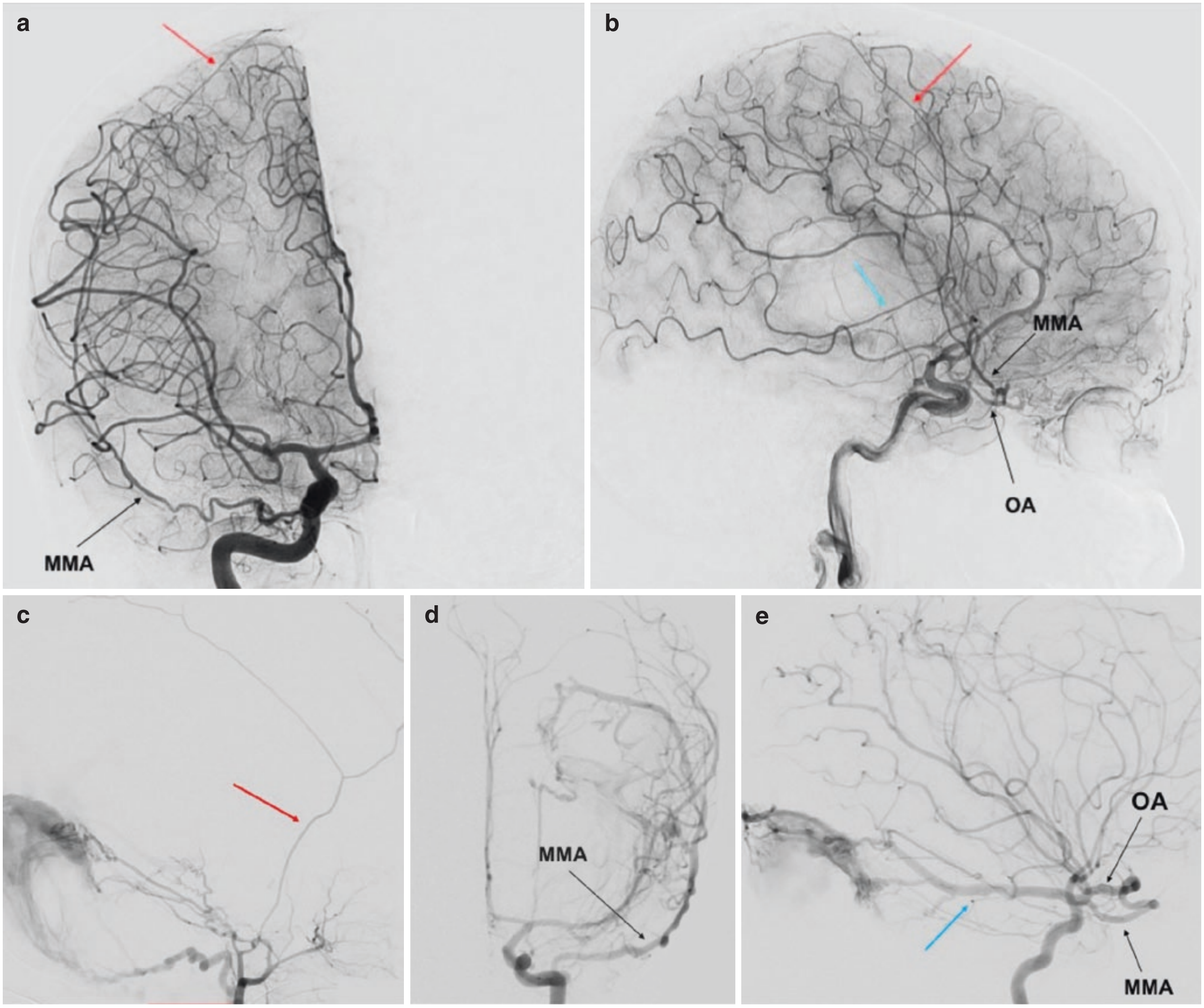


Fig. 3 Middle meningeal artery (MMA) origin from ophthalmic artery (OA). The anteroposterior and lateral view angiograms ((**a**) and (**b**)) show a rare case of complete MMA origin from the OA. The OA, through the superficial recurrent OA, gives birth to the MMA, which passes through the lateral part of the superior orbital fissure and gives its anterior (red arrow) and posterior division (blue arrow). In the angiograms (**c**–**e**), a rare case of partial origin of the MMA from the OA is shown. The angiograms d and e show a left internal carotid artery (ICA) injection in frontal and lateral view, where the posterior branch of the MMA (blue arrow) originates from the OA and feeds a tentorial arteriovenous fistula. After the ECA injection (**c**), only the anterior branch of the MMA is enhanced (red arrow)

Table 3 Different types of ophthalmic artery (OA) origin of the middle meningeal artery (MMA) by Maiuri et al. (Adapted from [18])

Type	Vascular anatomy	Foramen spinosum
I	Complete OA origin of the MMA	Absence
II	Partial OA origin of the MMA Anterior division from the OA Posterior division from the IMA	Reduced in size
III	OA origin of the accessory meningeal artery	Normal

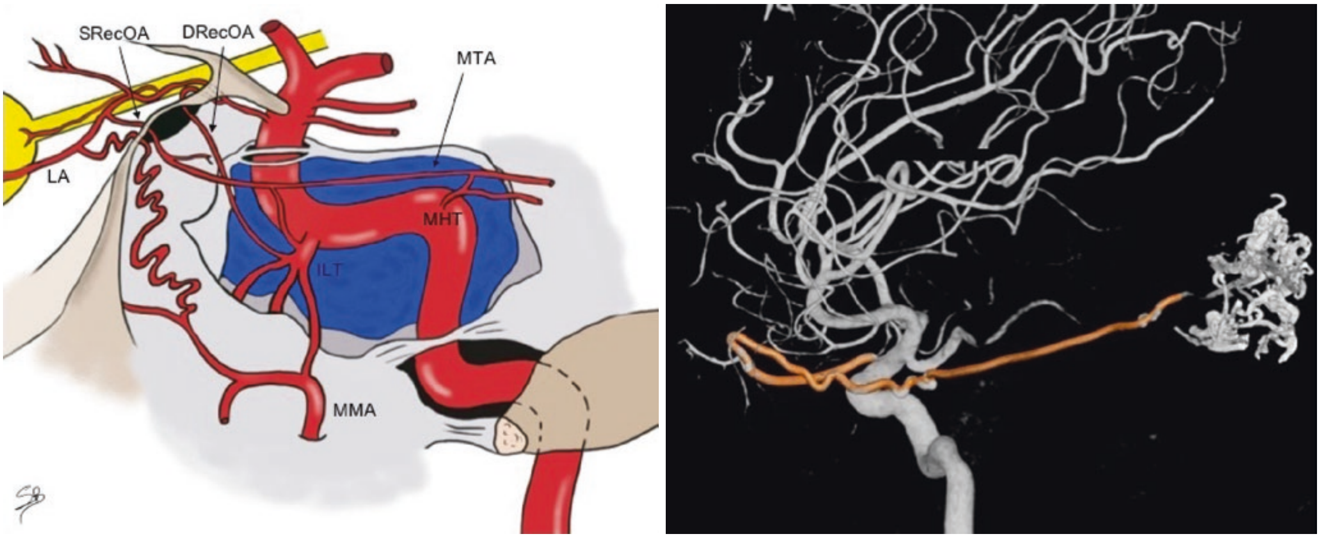


Fig. 4 Marginal tentorial artery (MTA) origin and course. The marginal tentorial artery, also called artery of the free margin of the tentorium or artery of Bernasconi-Cassinari, may have different origins, which are shown in graphic representation. It can arise from the lacrimal artery (LA) within the orbit, through the superficial recurrent ophthalmic artery (SRecOA), from the inferolateral trunk (ILT), and from the meningohypophyseal trunk (MHT). The artery courses posterolaterally along the free margin of the tentorium. The figure also shows a 3D DSA reconstruction of a rare case of MTA (highlighted in red) origin from the ophthalmic artery (OA). The MTA exits the orbit through the superior orbital fissure (SOF) and directs posteriorly to feed an arteriovenous malformation

Clinical Implications

Knowledge of the dural branches arising from the OA and their variations represents the cornerstone for interventional neuroradiologists and neurosurgeons who approaches anterior and middle cranial fossa pathologies. Two critical examples are the cribriform plate dural arteriovenous fistulas (dAVFs) and anterior and middle skull base meningiomas.

Cribriform Plate Dural Artero-Venous Fistulas

Cribriform plate dAVFs are usually mostly supplied by the anterior ethmoidal artery and the MMA. A bilateral supply of the dAVF, found in approximately 10% of cases, is well-explained by the anastomoses between the two anterior ethmoidal arteries within the dural or ethmoidal sinuses. Endovascular treatment of such pathologies consists of embolization, usually through branches of the MMA. The neuroradiologists must consider the presence of dural MMA–OA anastomoses during the injection of the liquid agent to avoid retrograde flow into ocular branches of the OA. In case of direct embolization of the dAVF through the ophthalmic artery, attention should be paid to the possible retrograde flow of the embolic agent into ocular branches. Since the central retinal artery usually arises from the second segment of the OA, the injection should be performed as distal as possible in order to limit the eventual damage caused by the reflux.

The surgical exclusion of a cribriform plate dAVF also necessitates a precise knowledge of dural branches of the OA. The aim of the treatment is to exclude the cortical venous drainage of the dAVF, clipping or coagulating the draining vein at its exit point from the dura. A case of a cribriform plate dAVF treated surgically is shown in Fig. 5. Knowledge of the arterio-arterial anastomoses between anterior ethmoidal, posterior ethmoidal, and middle meningeal arteries is necessary to understand the dAVF and the technical difficulties of the treatment. Another case of dAVF fed by dural branches of the OA is shown in Fig. 6, with also a contribution from the MTA.

Knowledge of the dural branches of the OA and MMA origin from the OA also well explains the possible participation of OA branches in the supply of carotid-cavernous fistulas or tentorial pathologies (Figs. 3 and 4).

Anterior and Middle Cranial Fossa Meningioma

The surgical removal of a cribriform plate or sphenoid wing meningiomas requires a detailed knowledge of vascular normal anatomy and tumor vascular supply. Meningeal tumors of the anterior and middle skull base are usually supplied by dural branches of the MMA, ICA, and by the ophthalmic artery. It is of paramount importance for interventional neuroradiologists who plan an embolization, usually performed through the MMA, to consider the possible variations in the supply of the skull base dura to avoid involuntary OA reflux of embolic liquid agent.

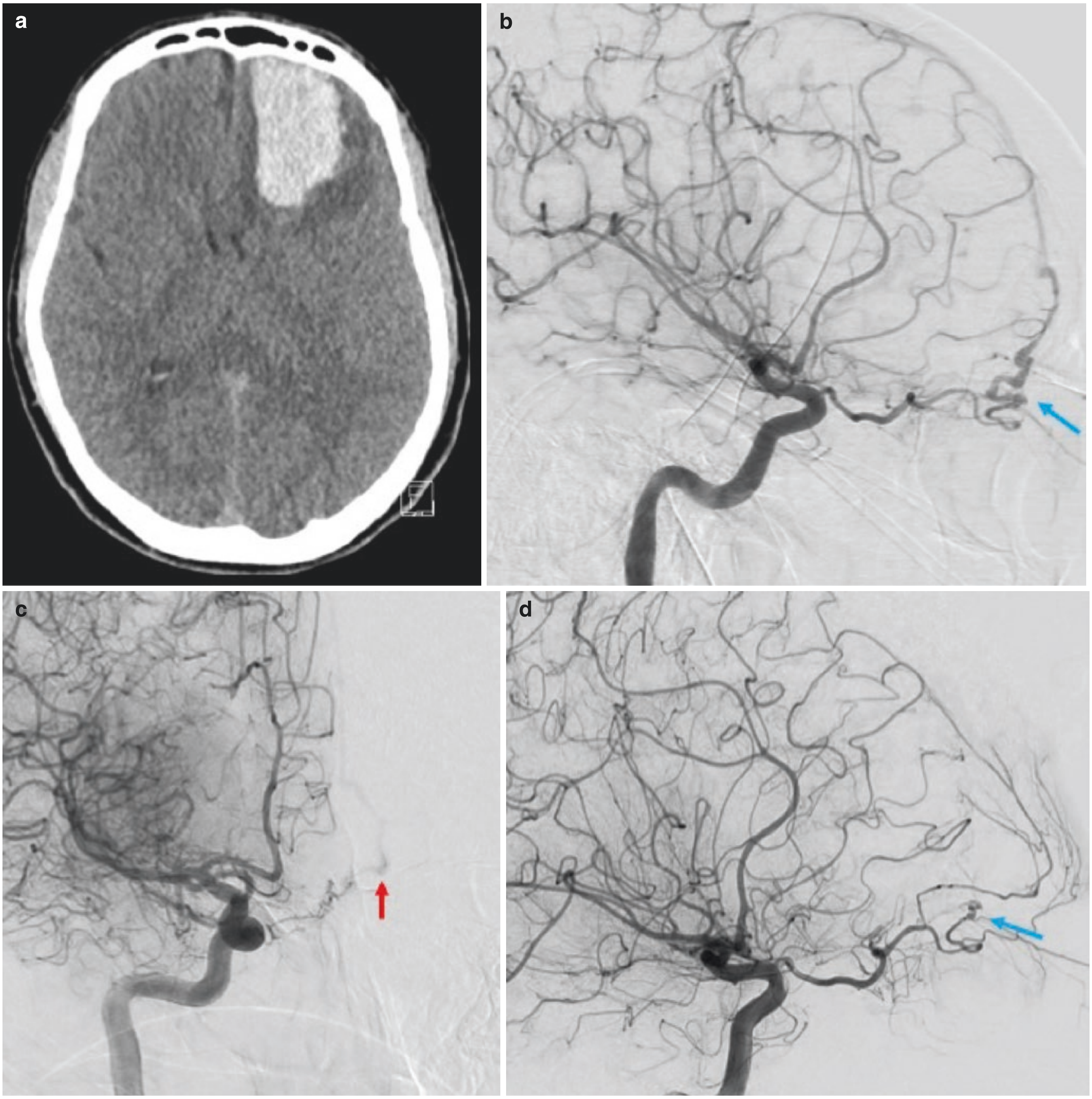


Fig. 5 Clinical case of ruptured cribriform plate dural arteriovenous fistula (dAVF). The figure shows the case of a 49-year-old man, taken in charge for sudden onset of unusual headache with nausea and vomiting. The CT scan performed in ER (**a**) showed a left frontal basis intraparenchymal hematoma. The DSA highlighted a cribriform plate dAVF with major feeders represented by the left anterior ethmoidal artery from left ophthalmic artery (OA) (blue arrow in figure (**b**)). The right internal carotid artery (ICA) injection showed also a contribution from the contralateral OA through its ethmoidal branches (red arrow in figure (**c**)). The venous drainage was represented by a single cortical vein directed into the superior sagittal sinus (Type III according to Cognard-Lariboisière classification [25]). The patient underwent successfully left supraorbital craniotomy and clipping of the dAVF (figure (**d**)), with no enhancement of the dAVF in the postoperative DSA (blue arrow) and clinical complete recovery

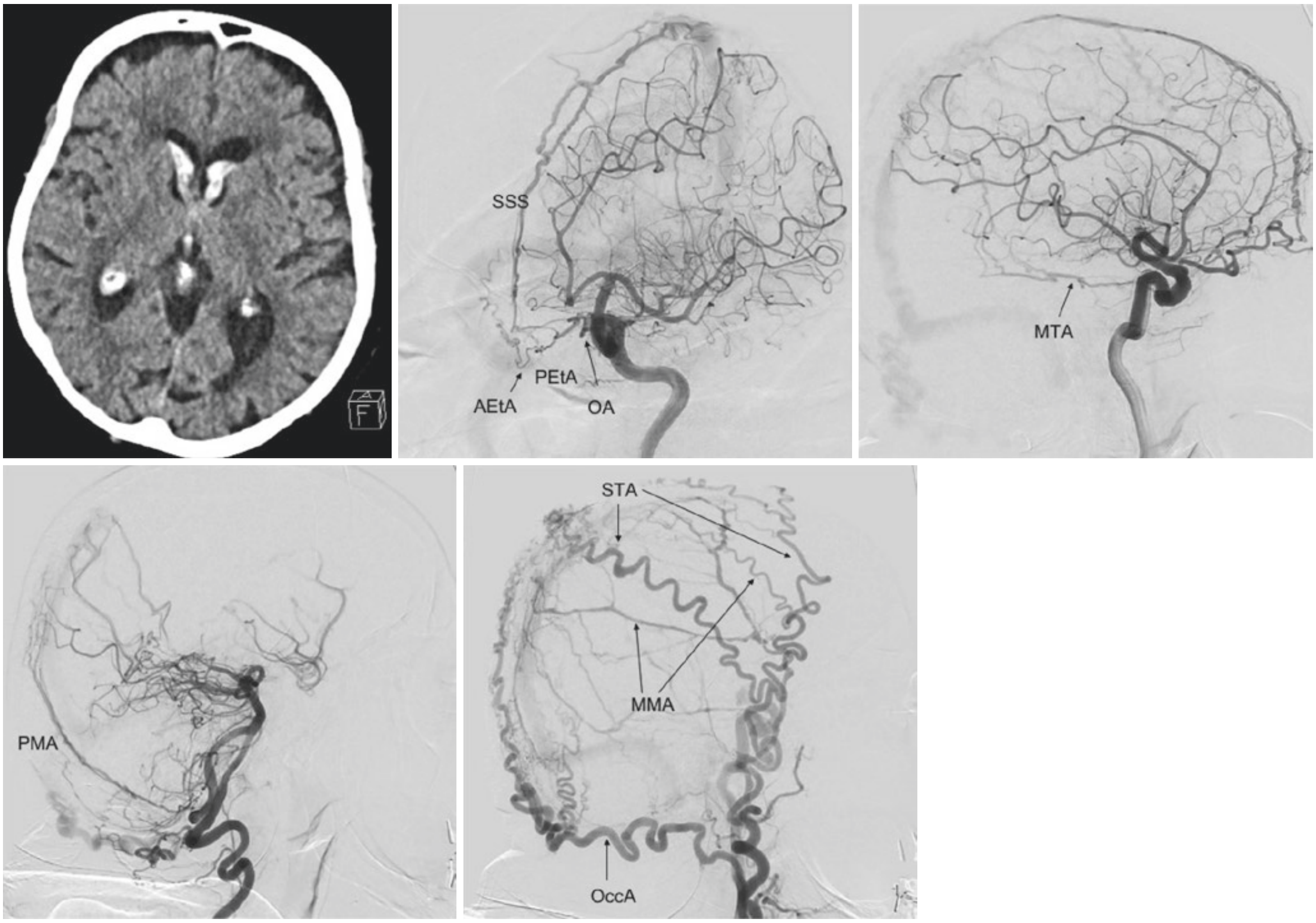


Fig. 6 Clinical case of dural arteriovenous fistula (dAVF) fed by multiple ophthalmic artery (OA) dural branches. The figure shows the case of an 89-year-old woman, previously operated for pituitary adenoma, taken in charge for unusual headache associated with vomiting. The CT scan showed an intraventricular hemorrhage with mild hydrocephalus. The diagnostic DSA showed a complex dAVF (Cognard-Lariboisière Grade IIa + b [25]) supplied by the OA through the anterior and posterior ethmoidal arteries (AEtA and PEtA), with both direct and indirect shunt with the superior sagittal sinus (SSS). Another point of shunt with the SSS is reached by the marginal tentorial artery (MTA) and by the posterior meningeal and middle meningeal artery (PMA and MMA). Also, other branches from the ECA contribute to the shunt, like the occipital artery (OccA) and the superficial temporal artery (STA). Because of age, the complexity of the dAVF, the high risks associated with every treatment option, and the absence of alteration of consciousness, we managed the dAVF conservatively

In case of surgery of middle cranial fossa meningioma, the devascularization of the tumor as first step could be helpful to better understand the arterial supply of the lesion and to limit drastically the blood loss.

Intra-Arterial Injection of Chemotherapy for Retinoblastoma

The classical technique used to inject chemotherapeutic agents into the OA for the treatment of retinoblastoma requires the super-selective catheterization of the OA [19]. However, knowledge of OA dural branches acquires a more important role when direct catheterization of the OA is not possible, like it can happen in children for its reduced size. In these cases, alternative ways to indirectly reach the OA have been described, especially through the catheterization of the MMA [20]. In this way, the pharmacologic agents can be injected through the anterior division of the MMA, incannulating its meningo-lacrimal branch. The reflux into the OA could be provided from anastomoses between MMA orbital branches and the recurrent branches of the OA, granted by the lacrimal artery or sometimes from the direct origin of the OA from the MMA.

Surgical and Endovascular Treatment of Refractory Epistaxis

Refractory epistaxis may be caused by a lot of clinical conditions and occurs in about 60% of the adult population, and most of them are considered idiopathic. Among them, about

6% of the epistaxis is refractory to conservative management and requires a surgical or interventional treatment [21].

The best way to understand which is the source of bleeding in case of refractory epistaxis is to perform a diagnostic DSA including ICA and external carotid artery (ECA). The DSA allows to identify the so-called "vascular blush," an anastomotic plexus located in the nasal septum, considered as the source of 90% of epistaxis. The sphenopalatine artery represents its main blood supply, and it's most commonly responsible for refractory epistaxis, even if in rare cases also the ethmoidal arteries could be involved [22]. If these latter are involved in the bleeding, they can be ligated through a surgical approach. On the other hand, if the sphenopalatine artery is responsible for bleeding, it can be occluded through an endonasal approach, or it can be embolized [22]. The diagnostic DSA allows to identify possible dangerous anastomoses between branches of the ECA and the OA, which can result in post-embolization visual or central deficits. The occurrence of cerebrovascular accident and obstruction of the central retinal artery has been described to occur in about 0–2% of cases [23]. The most important anastomoses to consider during such procedures are those between the sphenopalatine and anterior ethmoidal arteries via the turbinate and infraorbital arteries and those between the lacrimal artery and the MMA through the recurrent meningeal artery [22, 23]. The relevance of these anastomoses and the periprocedural risk can be estimated analyzing the "choroidal blush." This blush is commonly visualized after contrast injection into the ICA. However, if the anastomoses between the posterior ciliary arteries, the lacrimal artery, and the MMA are very consistent or if the lacrimal artery and the OA branch directly from the MMA, the choroidal blush can be seen after the ECA injection [23].

Embolization of Facial Tumors

Even if epistaxes are mostly idiopathic, some cases can be due to neoplastic erosion of vascular structures or as result of tumor necrosis after treatment. In these cases, the symptoms can also cause hemoptysis due to the frequent nasopharyngeal localization of these tumors. Endovascular treatment should be considered in these cases to treat uncontrollable epistaxis or hemoptysis. Also benign tumors, like paragangliomas and nasopharyngeal angiofibromas, can benefit from endovascular embolization, a preoperative procedure to reduce intraoperative blood loss. In these cases, the embolization of the sphenopalatine artery could be insufficient, and devascularization requires embolization of the facial artery and ascending pharyngeal artery [22]. In these cases, neuroradiologists should pay attention to the known anastomoses between the facial artery and the dorsal nasal artery (through the angular artery) [24]. For these pathologies, the neuroradiologists should observe the same rules previously described to avoid complications due to ICA–ECA anastomoses [22].

In conclusion, knowledge of embryology and anatomy of the dural branches of the OA is mandatory for treating pathology of the dura mater located in the anterior and middle cranial fossa. These arteries show high variability and supply territories in competition with the MMA and ICA branches.

References

1. Meyer F. Zur anatomie der Orbitalarteien Morph Jahr. 1887;12:414–458.
2. Curnow J. Two instances of irregular ophthalmic and middle meningeal arteries. J Anat Physiol. 1873;8(Pt 1):155–6.
3. Gabriele OF, Bell D. Ophthalmic origin of the middle meningeal artery. Radiology 1967;89(5):841–84844. doi: https://doi.org/10.1148/89.5.841.
4. Royle G, Motson R. An anomalous origin of the middle meningeal artery. J Neurol Neurosurg Psychiatry. 1973;36(5):874–6. https://doi.org/10.1136/jnnp.36.5.874.
5. McLennan JE, Rosenbaum AE, Haughton VM. Internal carotid origins of the middle meningeal artery. The ophthalmic-middle meningeal and stapedial-middle meningeal arteries. Neuroradiology. 1974;7(5):265–75. https://doi.org/10.1007/BF00344246.
6. Vignaud J, Hasso AN, Lasjaunias P, Clay C. Orbital vascular anatomy and embryology. Radiology. 1974;111(3):617–26. https://doi.org/10.1148/111.3.617.
7. Lasjaunias P, Moret J, Manelfe C, Theron J, Hasso T, Seeger J. Arterial anomalies at the base of the skull. Neuroradiology. 1977;13(5):267–72. https://doi.org/10.1007/BF00347072.
8. Lasjaunias P, Brismar J, Moret J, Theron J. Recurrent cavernous branches of the ophthalmic artery. Acta Radiol Diagn (Stockh). 1978;19(4):553–60. https://doi.org/10.1177/028418517801900402.
9. Dilenge D, Ascherl GF Jr. Variations of the ophthalmic and middle meningeal arteries: Relation to the embryonic stapedial artery. AJNR Am J Neuroradiol. 1980;1(1):45–54.
10. Kuru Y. Meningeal branches of the ophthalmic artery. Acta Radiol Diagn (Stockh). 1967;6(3):241–51. https://doi.org/10.1177/028418516700600304.
11. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer edition; 2001.
12. Rhoton AL Jr. The orbit. Neurosurgery. 2002;51(4 Suppl):S303–34.
13. Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A Jr. Microsurgical anatomy of the dural arteries. Neurosurgery. 2005;56(2 Suppl):211–51.; discussion 211−251. https://doi.org/10.1227/01.neu.0000144823.94402.3d.
14. Padget DH. The development of cranial arteries in the human embryo. Contribution to embryology. Carnegie Institution: Washington, 1948;212:205–2262.
15. Lasjaunias P, Moret J, Mink J. The anatomy of the inferolateral trunk (ILT) of the internal carotid artery. Neuroradiology. 1977;13(4):215–20.
16. Hayreh SS. The ophthalmic artery: iii Branches. Br J Ophthalmol. 1962;46(4):212–47. https://doi.org/10.1136/bjo.46.4.212.
17. Lang J, Kageyama I. The ophthalmic artery and its branches, measurements and clinical importance. Surg Radiol Anat. 1990;12(2):83–90. https://doi.org/10.1007/BF01623328.
18. Maiuri F, Donzelli R, de Divitiis O, Fusco M, Briganti F. Anomalous meningeal branches of the ophthalmic artery feeding meningio-

mas of the brain convexity. Surg Radiol Anat. 1998;20(4):279–84. https://doi.org/10.1007/BF01628491.
19. Yamane T, Kaneko A, Mohri M. The technique of ophthalmic arterial infusion therapy for patients with intraocular retinoblastoma. Int J Clin Oncol. 2004;9(2):69–73. https://doi.org/10.1007/s10147-004-0392-6.
20. Klufas MA, Gobin YP, Marr B, Brodie SE, Dunkel IJ, Abramson DH. Intra-arterial chemotherapy as a treatment for intraocular retinoblastoma: alternatives to direct ophthalmic artery catheterization. AJNR Am J Neuroradiol. 2012;33(8):1608–14. https://doi.org/10.3174/ajnr.A3019.
21. Christensen NP, Smith DS, Barnwell SL, Wax MK. Arterial embolization in the management of posterior epistaxis. Otolaryngol Head Neck Surg. 2005;133(5):748–53. https://doi.org/10.1016/j.otohns.2005.07.041.
22. Reyre A, Michel J, Santini L, Dessi P, Vidal V, Bartoli JM, et al. Epistaxis: The role of arterial embolization. Diagn Interv Imaging. 2015;96(7–8):757–73. https://doi.org/10.1016/j.diii.2015.06.006.
23. Mames RN, Snady-McCoy L, Guy J. Central retinal and posterior ciliary artery occlusion after particle embolization of the external carotid artery system. Ophthalmology. 1991;98(4):527–31. https://doi.org/10.1016/s0161-6420(91)32261-9.
24. Bertelli E, Regoli M, Bracco S. An update on the variations of the orbital blood supply and hemodynamic. Surg Radiol Anat. 2017;39(5):485–96. https://doi.org/10.1007/s00276-016-1776-9.
25. Cognard C, Gobin YP, Pierot L, Bailly AL, Houdart E, Casasco A, et al. Cerebral dural arteriovenous fistulas: Clinical and angiographic correlation with a revised classification of venous drainage. Radiology 1995;194(3):671–6680. doi: https://doi.org/10.1148/radiology.194.3.7862961.

Dural Branches of the Cerebellar Arteries

Thomas Robert and Sara Bonasia

The dura mater of the posterior fossa is predominantly supplied by purely dural branches (without parenchymal territory associated). The most important of them are the occipital artery, the ascending pharyngeal artery, and the posterior meningeal artery [1, 2]. The middle meningeal artery also plays a role in the supply of the dura of the posterior fossa [1, 3]. The territory of each dural artery is in balance with each other [2]. Cerebellar arteries have a very little role in the supply of the dura. Most of their dural territories are inconstant and depend on anatomical variations or arterio-arterial anastomoses.

Dural Branches of the Superior Cerebellar Artery

In 1965, in their original article, Wollschlaeger and Wollschlaeger described a little dural branch of the posterior cerebral artery (PCA) and named it the "artery of Davidoff and Schechter" (ADS) [4, 5]. In their cadaveric study on ten cadavers, they find the presence of the ADS in 90% of cases, but in one of them, they noted that this artery arose from the superior cerebellar artery (SCA) instead of from the PCA [4]. The SCA has no other dural branch than this tentorial artery that inconstantly arises from the SCA and not from the PCA [5–7].

After its origin, this meningeal branch enters in the inferior surface of the tentorium to supply the posterior part of the tentorial incisura. It has the same course and territory than the ADS originating from the PCA [8, 9].

Ono et al. noted the presence of this meningeal branch from the SCA in 28% of their 25 cadaveric dissections [10]. Umeoka et al., during trigeminal decompression surgery, saw this artery intraoperatively in 25% of cases [11]. In the non-pathological state, this artery is too thin to be seen in DSA or MRI, but in the case of dural tentorial arteriovenous fistula (dAVF), it could be dilated and visible [1–3].

Dural Branches of the Anterior Inferior Cerebellar Artery

The anterior inferior cerebellar artery (AICA) is the lonely cerebellar artery that possesses a constant dural territory [2]. Its dural branch is named the "subarcuate artery" (SAA) that could arise from the lateral pontine segment of the AICA (70%) or from the labyrinthine artery (30%) [12]. After a short cisternal segment, it penetrates the dura at the subarcuate fossa before penetrating the subarcuate canal [2, 12, 13]. The dural territory of the AICA is limited to the posterior surface of the petrous bone and to the superolateral surface of the internal auditory canal [2, 14]. After it, the SAA has a bony course in the petrous bone where it participates in the internal ear supply.

The SAA presents a high variability in origin, and the most detailed publication about this aspect is of Mazzoni et al. after a dissection of 100 petrous bones [13]. The SAA arises from the AICA in 44%, from a cerebellar branch of the AICA in 38%, from the labyrinthine artery in 16%, and interestingly from a branch of the PICA in 3%. Rasmussen et al. also proposed interesting work describing an anatomical classification of the AICA in five grades depending on its course and on the origin of the SAA [15]. Considering its dural supply, the SAA can be involved in the supply of dAVF.

Dural Branches of the Posterior Inferior Cerebellar Artery

In the normal anatomical description, the posterior inferior cerebellar artery (PICA) has no meningeal branch, and the posterior meningeal artery (PMA) arises from the extracra-

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nial (V3) segment of the vertebral artery [2]. Inconsistently, the PMA could arise intracranially and share its origin with the PICA. P. Lasjaunias called the meningeal artery of the PICA "the artery of the falx cerebelli," but it could be the same artery as the PMA with PICA origin that explains its different angiographic course [1].

Ohshima et al. proposed a classification of the PMA depending on the origin of the artery [16]. In their analysis of 300 cases, they noted that the PMA arises intracranially in only 1% of cases.

Only few cases of PICA origin of the PMA are described in the literature [17–19]. In these cases, the PMA arises from the lateral medullary segment of the PICA to enter the dura of the cerebellar convexity. Its dural territory constitutes the cerebellar convexity, the falx cerebelli, and the posteromedial part of the tentorium. A case of this variant is shown in Fig. 1.

Ogawa et al. described an interesting case of retrograde opacification of the PICA through the PMA, highlighting the presence of more distal arterio-arterial anastomoses between the PICA and the PMA [20]. Tsutsumi et al. also showed a case of distal anastomosis between the two arteries in the presence of distinct origin from the vertebral artery, highlighting one more time the presence of distal PICA–PMA anastomoses [18].

This anatomical variation has few clinical implications. In fact, the formation of aneurysm located at the origin of the PMA from the PICA has been described and could be challenging [16]. Posterior-located tentorial dAVF could also be supplied by PMA arising from the PICA that could complicate an eventual endovascular embolization. Table 1 summarizes the branches of the cerebellar arteries.

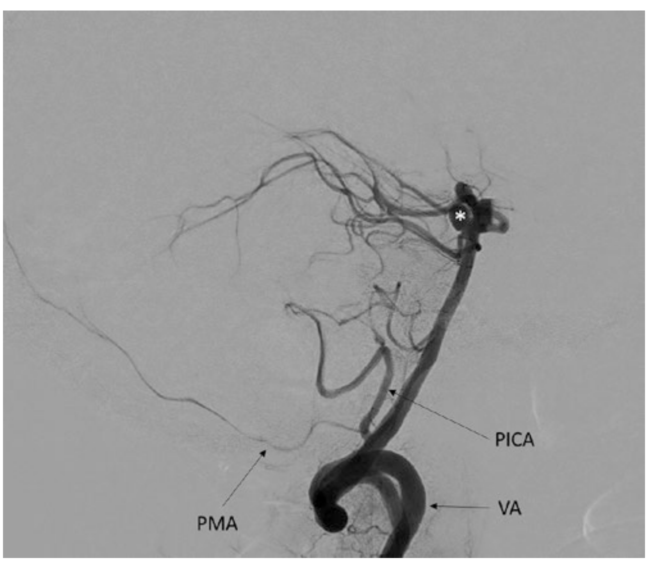

Fig. 1 Posterior meningeal artery (PMA) origin from the posterior inferior cerebellar artery (PICA). The figure shows an anatomical variant with the PMA origin from the PICA instead of from the vertebral artery (VA). After its origin, the PMA enters the dura of the cerebellar convexity with its typical angiographic aspect. The asterisk indicates a concomitant basilar apex aneurysm

Table 1 Meningeal branches of the cerebellar arteries

Cerebellar artery	Meningeal branch	Dural territory
SCA	Tentorial branch or artery of Davidoff and Schechter	Posterior part of the tentorial incisura
AICA	Subarcuate artery	Petrous ridge Subarcuate fossa
PICA	Posterior meningeal artery or artery of the falx cerebelli	Cerebellar convexity Posteromedial part of the tentorium Falx cerebelli

Conclusion

Meningeal branches of the cerebellar arteries are not well-known branches because they are not seen in the normal anatomy in most neuroimaging studies. Clinical implications of these little arteries are not less important, and their knowledge is important to improve treatment quality of dural vascular pathology of the posterior fossa.

References

1. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer edition; 2001.
2. Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A Jr. Microsurgical anatomy of the dural arteries. Neurosurgery. 2005;56(2 Suppl):211–51.; discussion 211−251. https://doi.org/10.1227/01.neu.0000144823.94402.3d.
3. Merland JJ, Bories J, Djindjian R. The blood supply of the falx cerebri, the falx cerebelli and the tentorium cerebelli. J Neuroradiol. 1977;4(2):175–202.
4. Wollschlaeger PB, Wollschlaeger G. An infratentorial meningeal artery. Radiologe. 1965;5(11):451–2.
5. Tayebi Meybodi A, Vigo V, Lawton MT, Benet A. The artery of Wollschlaeger and Wollschlaeger: An anatomical-clinical illustration. Br J Neurosurg. 2017;31(5):593–5. https://doi.org/10.1080/02688697.2017.1333573.
6. Kvint S, Ramchand P, Cox M, Sedora-Roman NI, Bagley L, O'Rourke DM, et al. Type V Dural arteriovenous fistula supplied by the artery of Wollschlaeger and Wollschlaeger causing cervical myelopathy and quadriparesis. World Neurosurg. 2020;137:55–61. https://doi.org/10.1016/j.wneu.2020.01.122.
7. Weil AG, McLaughlin N, Denis D, Bojanowski MW. Tentorial branch of the superior cerebellar artery. Surg Neurol Int. 2011;2:71. https://doi.org/10.4103/2152-7806.81733.
8. D'Andrea M, Mongardi L, Fuschillo D, Musio A, Commodaro C, Quilis-Quesada V, et al. Vermian subtentorial arteriovenous malformation supplied by the artery of Wollschlaeger and Wollschlaeger. Clin Neurol Neurosurg. 2021;206:106670. https://doi.org/10.1016/j.clineuro.2021.106670.
9. Krafft PR, Liu SS, Agarwalla PK, Van Loveren HR. The meningeal branch of the superior cerebellar artery. Br J Neurosurg.

2018;32(3):273–5. https://doi.org/10.1080/02688697.2017.1416066.
10. Ono M, Ono M, Rhoton AL Jr, Barry M. Microsurgical anatomy of the region of the tentorial incisura. J Neurosurg. 1984;60(2):365–99. https://doi.org/10.3171/jns.1984.60.2.0365.
11. Umeoka K, Takusakawa Y, Kominami S, Kobayashi S, Morita A. The meningeal branches of the superior cerebellar artery: A surgical observation study. J Neurosurg. 2016;124(1):244–7. https://doi.org/10.3171/2014.12.JNS141190.
12. Akyol Y, Galheigo D, Massimore M, Fatterpekar G. Subarcuate artery and canal: An important anatomic variant. J Comput Assist Tomogr. 2011;35(6):688–9. https://doi.org/10.1097/RCT.0b013e318234232a.
13. Mazzoni A. The subarcuate artery in man. Laryngoscope. 1970;80(1):69–79. https://doi.org/10.1288/00005537-197001000-00006.
14. Salgado-Lopez L, Leonel LCP, Aydin SO, Peris-Celda M. Surgical anatomy of the labyrinthine and subarcuate arteries and clinical implications. World Neurosurg. 2020;141:e880–7. https://doi.org/10.1016/j.wneu.2020.06.083.
15. Rasmussen J, Plou P, Campero A, Ajler P. A classification for the anterior inferior cerebellar artery-subarcuate artery complex based on the embryological development. J Neurol Surg B Skull Base. 2020;81(5):536–45. https://doi.org/10.1055/s-0039-1692474.
16. Ohshima T, Handa T, Ishikawa K, Miyachi S, Matsuo N, Kawaguchi R, et al. Posterior meningeal artery origin patterns among 300 cases and their clinical importance. J Stroke Cerebrovasc Dis. 2018;27(7):2032–4. https://doi.org/10.1016/j.jstrokecerebrovasdis.2018.02.059.
17. Tanohata K, Maehara T, Noda M, Katoh H, Sugiyama S, Okazaki A. Anomalous origin of the posterior meningeal artery from the lateral medullary segment of the posterior inferior cerebellar artery. Neuroradiology. 1987;29(1):89–92. https://doi.org/10.1007/BF00341048.
18. Tsutsumi M, Kazekawa K, Aikawa H, Iko M, Kodama T, Nii K, et al. Development of unusual collateral channel from the posterior meningeal artery after endovascular proximal occlusion of the posterior inferior cerebellar artery. Neurol Med Chir (Tokyo). 2007;47(11):503–5. https://doi.org/10.2176/nmc.47.503.
19. Newton TH, Mani RL. The vertebral artery. Radiology of the skull and brain. St Louis: CV Mosby; 1974. p. 1673–4.
20. Ogawa T, Fujita H, Inugami A, Shishido F, Higano S, Uemura K. Anomalous origin of the posterior inferior cerebellar artery from the posterior meningeal artery. AJNR Am J Neuroradiol. 1991;12(1):186.

Dural Branches of the Vertebral Artery

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Many collateral branches arise from the vertebral artery (VA) and supply intracranial and extracranial structures (see also Chap. 16). In addition to the several spinal arteries, from C6 to C1 involved in the dura mater vascularization of the cervical spine, which is not the aim of this chapter, the VA gives rise to two meningeal branches, which supply the dura of the inferior posterior fossa: the anterior meningeal artery and the posterior meningeal artery.

Anterior Meningeal Artery

The anterior meningeal artery (AMA) arises from the anteromedial surface of the distal V2 segment of the vertebral artery (VA) immediately below its first bend at the level of the axis [1] or at the level of the third vertebral body [2].

The AMA on normal angiography is tiny, usually less than 0.5 mm in diameter, and the incidence of its angiographic visualization is approximately 48% [3].

The artery has been first described by Greitz and Lauren in 1968 [1], and it had been thought that its territory was purely meningeal in early radiological reports [1, 3], but subsequent anatomical studies showed that the AMA supplies the odontoid process and ligamentous structures as well [4, 5]. Lasjaunias et al. [6] evaluated the embryology of the artery, and they remarked that if the occipital artery represents the arterial pedicle of the C2 somite [7], the AMA could be considered as a branch of the pedicle of the C3 somite, and therefore the AMA should be called the C3 artery of the cervical VA.

After its origin, the AMA passes medially through the intravertebral foramen anterior to the dural sheath of the third cervical nerve root and its surrounding intravertebral veins [1]. Then, it runs upward slightly medially in the spinal canal, dorsal to the deep layer of the posterior longitudinal ligament (PLL) and the ventral surface of the anterior internal vertebral venous plexus [8]. Cranially, it penetrates the tectorial membrane, the upper extent of the PLL, and it forms an arcade above the apex of the odontoid process (Fig. 1) with the contralateral artery [5], supplying the dura of the anterior foramen magnum and the inferior clivus. On the vertebral angiogram, the artery is visible as a fine branch located anteriorly and laterally to the anterior spinal artery (ASA) (Fig. 2).

Before penetrating the tectorial membrane, the AMA gives off several tiny branches to the deep layer of the PLL and vertebral bodies [5].

At the level of the odontoid process, in addition to the anastomosis with the contralateral branch, the AMA is connected with the hypoglossal artery, which is a branch of the ascending pharyngeal artery. These connections form the so-called odontoid arch. The latter has been reported to cause palsy of the lower cranial nerve in the case of endovascular embolization [6].

Enlargement of the AMA has been observed in different pathologies: glomus jugular tumor, meningioma, hemangioblastoma, metastatic tumor, plasmacytoma, chordoma, schwannoma of the vagus nerve, dural arteriovenous fistula, and traumatic arteriovenous fistula [3, 9–13].

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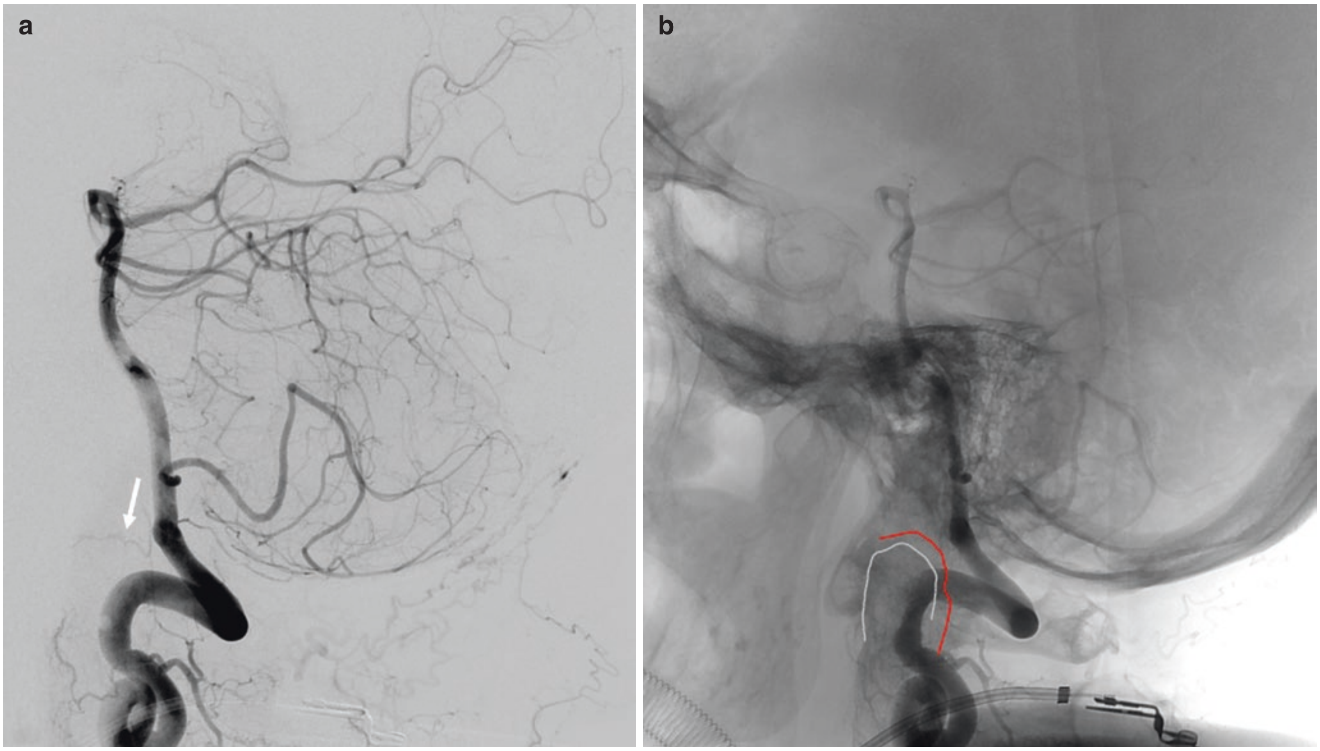

Fig. 1 The anterior meningeal artery and the odontoid arch. Left vertebral artery angiogram lateral view with (**a**) and without substraction (**b**). The anterior meningeal artery (white arrow in (**a**) and red line in (**b**)) forms an arcade above the apex of the odontoid process (white line in (**b**))

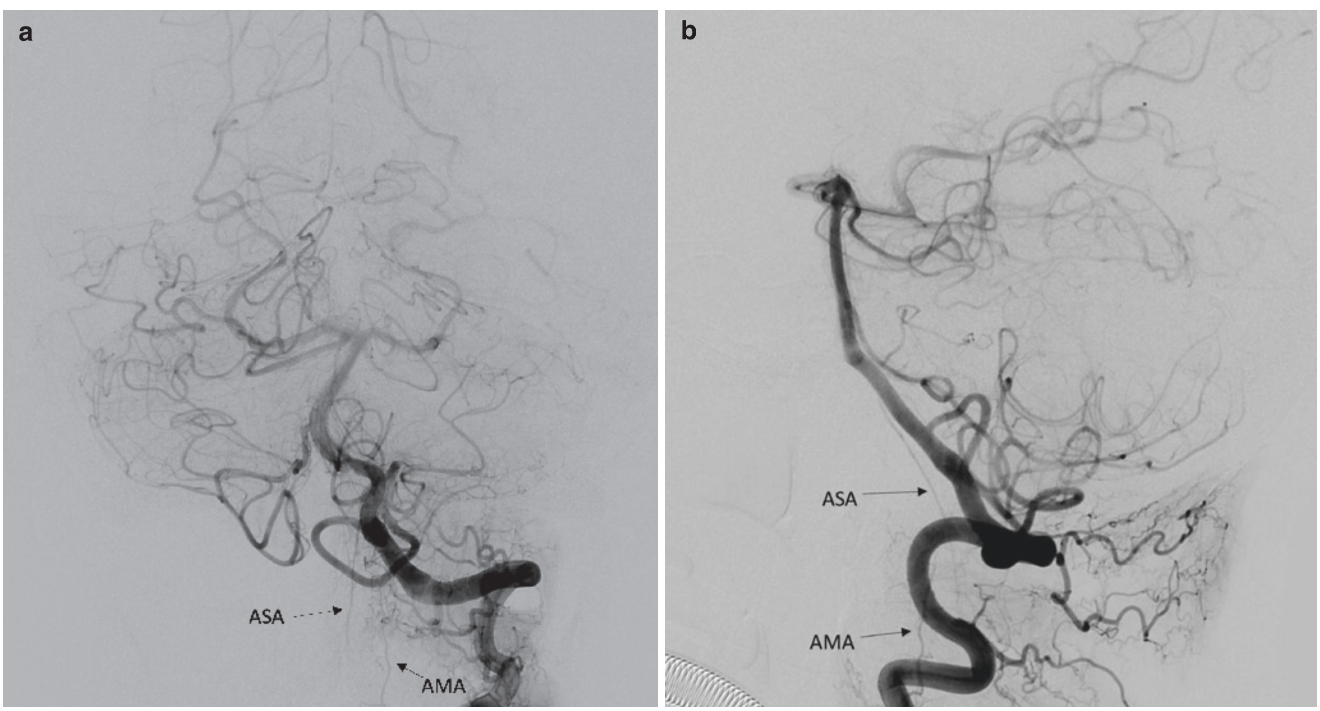

Fig. 2 Angiographic configuration of the anterior meningeal and anterior spinal arteries. Left vertebral artery angiogram AP view (**a**) and lateral view (**b**). The anterior meningeal artery (AMA) is located laterally (**a**) and anteriorly (**b**) to the anterior spinal artery (ASA)

Posterior Meningeal Artery

The posterior meningeal artery (PMA), previously identified as the artery of the falx cerebelli [14], usually arises from the posterior portion of the V3 segment of the VA [15] above the level of the arch of the atlas and just below the foramen magnum [3]. More rarely, it can arise from the V4 segment of the VA or from the occipital artery [16], ascending pharyngeal artery [17], cervical internal carotid artery [18], or posterior inferior cerebellar artery [19].

This artery supplies the medial portions of the dura of the occipital posterior fossa as well as the falx cerebelli. In addition, it may extend above the tentorium to supply the posterior segment of the falx cerebri and adjacent tentorium [3].

The PMA on normal angiography is tiny, usually less than 0.5 mm in diameter, and the incidence of its angiographic visualization is approximately 35% [3]. It is more frequently unilateral (84.7% of cases) than bilateral, and when it is found to be unilateral, right dominance is more common (77.4%) than left dominance [20]. Regarding its course, an extracranial and an intracranial segment can be identified. In the extracranial portion, shorter than the intracranial one, the artery is tortuous to allow cranio-cervical junction motility, whereas the intracranial segment is straight. There are two branches of this artery, which ramify within the dura (Fig. 3) [21]. The medial branch, also called "falx cerebelli artery" (FCA) [2], courses superiorly in the falx cerebelli parallel to the occipital bone and frequently enters the posterior portion of the falx cerebri [21]. It anastomoses superiorly with the paramedian branches of the middle meningeal artery. On the lateral angiogram, the FCA runs slightly away from the inner table of the skull, and it is near the midline on the anteroposterior projection (AP) view (Fig. 4). The lateral branch supplies the medial aspect of the dura in the posterior fossa [21]. It runs close to the inner table of the skull on the lateral angiogram (Fig. 5). Enlargement of the PMA has been observed in different pathologies: meningioma, hemangioblastoma, and dural arteriovenous fistula [3] (Fig. 6).

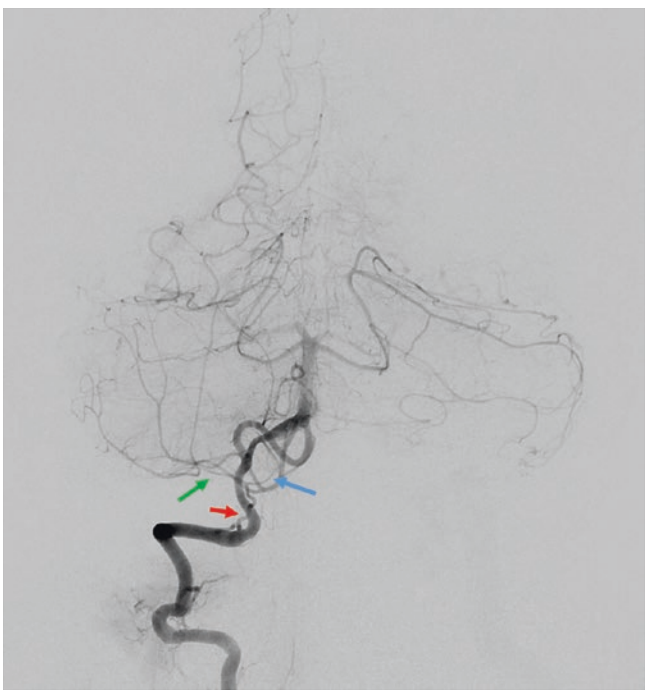

Fig. 3 The posterior meningeal artery and its branches. Right vertebral artery angiogram AP view. Posterior meningeal artery (red arrow) with division in its medial branch (blue arrow), also known as "falx cerebelli artery," and its lateral branch (green arrow)

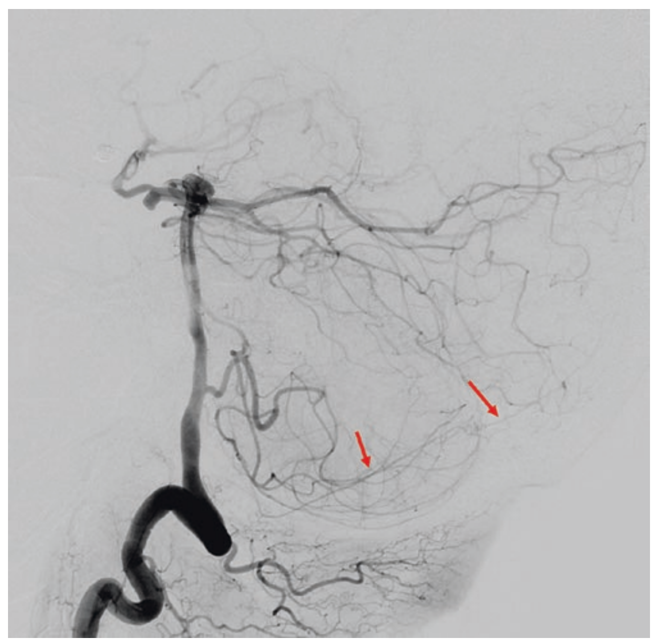

Fig. 4 Angiographic configuration of the medial branch of the posterior meningeal artery. Left vertebral artery angiogram lateral view. Falx cerebelly artery (red arrows) running slightly away from the inner table of the skull

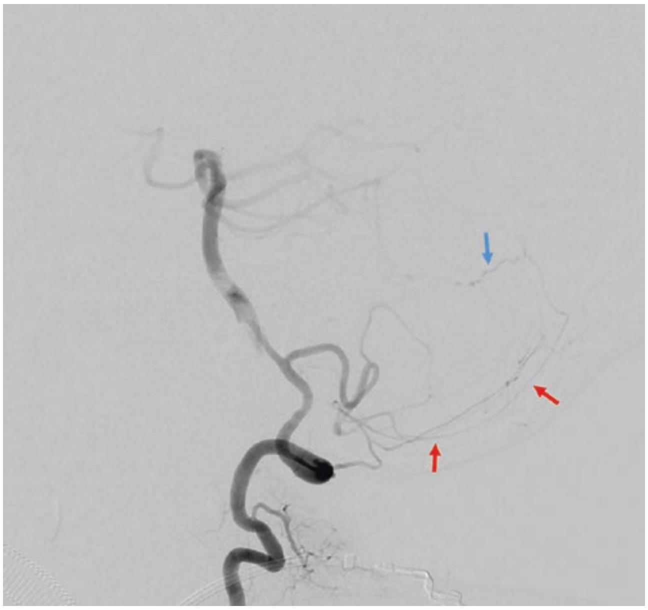

Fig. 5 Angiographic configuration of the lateral branch of the posterior meningeal artery. Right vertebral artery angiogram later view. Lateral branch of the posterior meningeal artery (red arrows) running close to the inner table of the skull. Note a small dural shunt (blue arrow) supplied by the lateral division branch of the posterior meningeal artery

Fig. 6 Hypertrophic posterior meningeal artery. Right vertebral artery angiogram lateral view. Hypertrophic posterior meningeal artery (red arrow) supplying a dural arteriovenous fistula of the superior sagittal sinus

References

1. Greitz T, Laurén T. Anterior meningeal branch of the vertebral artery. Acta Radiologica Diagnosis. 1968;7:219–24.
2. Bradac GB (2017) Applied cerebral angiography: Normal anatomy and vascular pathology.
3. Newton TH. The anterior and posterior meningeal branches of the vertebral artery. Radiology. 1968;91:271–9.
4. Schiff DC, Parke WW. The arterial supply of the odontoid process. J Bone Joint Surg Am. 1973;55:1450–6.
5. Shimizu S, Garcia AS, Tanriover N, Fujii K. The so-called anterior meningeal artery: An anatomic study for treatment modalities. Interv Neuroradiol. 2004;10:293–9.
6. Lasjaunias P, Moret J, Theron J. The so-called anterior meningeal artery of the cervical vertebral artery: Normal radioanatomy and anastomoses. Neuroradiology. 1978;17:51–5.
7. Lasjaunias P, Théron J, Moret J. The occipital artery. Anatomy – Normal arteriographic aspects – Embryological significance. Neuroradiology. 1978;15:31–7.
8. Kubo Y, Waga S, Kojima T, Matsubara T, Kuga Y, Nakagawa Y. Microsurgical anatomy of the lower cervical spine and cord. Neurosurgery. 1994;34:895–0. discussion 901–902.
9. Alford BR, Guilford FR. A comprehensive study of tumors of the glomus jugulare. Laryngoscope. 1962;72:765–805.
10. Castellano F, Ruggiero G. Meningiomas of the posterior fossa. Acta Radiol Suppl. 1953;104:1–177.
11. Hooper RS. The glomus jugulare tumour; clinical and radiological features. J Fac Radiol. 1955;7:77–89.
12. Melmon KL, Rosen SW. Lindau's disease. Review of the literature and study of a large kindred. Am J Med. 1964;36:595–617.
13. Morris L. The anterior meningeal branch of the vertebral artery. Br J Radiol. 1969;42:308–10.
14. Hawkins TD, Melcher DH. A meningeal artery in the falx cerebelli. Clin Radiol. 1966;17:377–83.
15. Wang G, Yu J, Hou K, Guo Y, Yu J. Clinical importance of the posterior meningeal artery: A review of the literature. Neuroradiol J. 2019;32:158–65.
16. Théron J, Lasjaunias P, Moret J, Merland JJ. Vascularization of the posterior fossa dura mater. J Neuroradiol. 1977;4:203–24.
17. Hsu CY, Sheu JJ, Huang KM, Li YW. Bilateral anomalous origins of the posterior meningeal artery from the ascending pharyngeal arteries. J Neuroradiol. 2001;28:123–5.
18. Kwak S, Nagashima T, Kobayashi S. Anomalous origin of the posterior meningeal artery from the internal carotid artery. Neuroradiology. 1980;19:103–4.
19. Bojanowski WM, Rigamonti D, Spetzler RF, Flom R. Angiographic demonstration of the meningeal branch of the posterior cerebral artery. AJNR Am J Neuroradiol. 1988;9:808.
20. Ohshima T, Handa T, Ishikawa K, Miyachi S, Matsuo N, Kawaguchi R, Takayasu M. Posterior meningeal artery origin patterns among 300 cases and their clinical importance. J Stroke Cerebrovasc Dis. 2018;27:2032–4.
21. Takahashi M. Atlas of vertebral angiography. 1st ed. Baltimore: University Park Press; 1974.

Dural Branches of the Occipital Artery

Thomas Robert and Sara Bonasia

The occipital artery is one of the major external carotid artery collaterals and represents a fascinating artery concerning its complex territory of supply (muscular, neural, and dural) and its high number of anastomoses with the vertebrobasilar system [1, 2]. This artery also participates in the complex anastomotic odontoid arch [3]. The anatomy of the occipital artery and its variations are addressed in chapter "Embryology and Variations of the Occipital Artery." Branches from the occipital artery that participate in the odontoid arch are summarized in chapter "Anastomotic network of the Cranio-Cervical Junction." In the present chapter, we will discuss only dural branches of the occipital artery with their territory and their possible anastomoses with other dural branches.

Embryology

In this chapter, only notions about embryological development that allow to understand the pharyngo-occipital system will be developed (Table 1). For more detailed embryological development about the vertebral artery, the carotid system, and the carotid-vertebral anastomoses, please refer to the respective chapters.

Early in embryological life (Stage I of Padget, embryos of 4–5 mm), the formation of the vertebrobasilar system initiates with the presence of the paired longitudinal artery named "longitudinal neural arteries (LNAs)." These LNAs are supplied by the carotid system through different carotid-vertebral anastomoses, and the vertebral arteries are not developed yet. LNAs are supplied by the trigeminal artery, the otic artery, the hypoglossal artery, and the proatlantal artery from cranial to caudal [4–7].

During Stage II of Padget (embryos of 5–6 mm), the caudal division of the primitive internal carotid artery anastomoses with the cranial part of the LNA (formation of the posterior communicating artery) and consequently the carotid-vertebral anastomoses initiate their involution. The first two arteries that regress are the otic and the hypoglossal arteries, and the LNAs are supplied principally by the trigeminal artery cranially and the proatlantal artery caudally. At the same time, the two LNAs come closer to the midline to fuse together [5, 7].

At Stage III of Padget (embryos of 7–12 mm), the basilar artery is completely formed by fusion of the two LNAs as well as the posterior communicating artery, which is anastomosed with the upper part of the basilar artery. Trigeminal and proatlantal arteries progressively regress, and the primitive vertebral artery is formed by transverse anastomoses between the six upper cervical intersegmental arteries [5, 7].

The occipital artery is considered as the remnant of the first (proatlantal intersegmental artery) and second cervical segmental arteries. Between Stages III and V, the origin of the occipital artery shifts from the primitive internal carotid artery to the external carotid artery, and the distal parts of the two first intersegmental cervical arteries leave only thin anastomosis between the occipital artery and the vertebral artery. At Stage V (embryos of 16–18 mm), the definite occipital artery could be clearly individualized [2].

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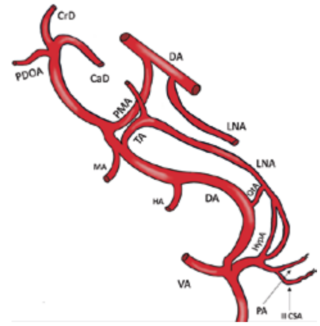
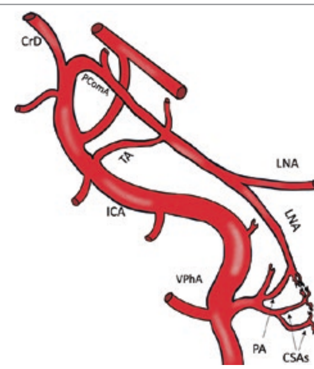
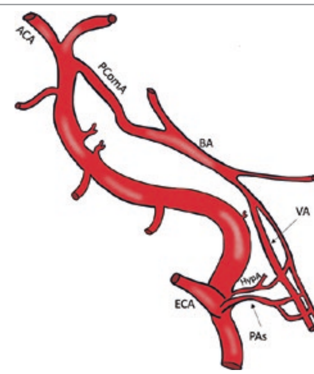
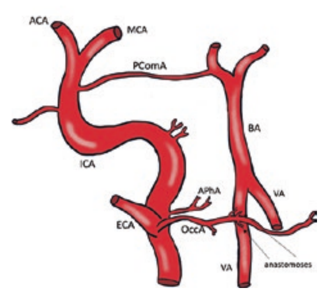
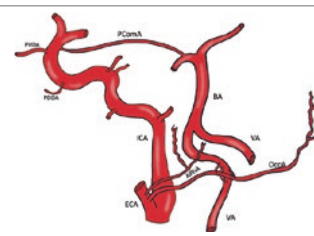
Table 1 Major embryological changes in the formation of the occipito-pharyngeal system

Stage	Embryo size (mm)	Major evolutions	Graphic illustration
I	4–5	• Paired longitudinal neural arteries (LNA) • Trigeminal (TA), otic (OtA), hypoglossal (HypA), proatlantal (PA), and second cervical segmental artery (II CSA) • PComA not formed	
II	5–6	• Formation of the PComA • Regression of otic and hypoglossal arteries • Fusion of LNAs in basilar artery (initiation) • Initial transverse anastomoses between the intersegmental arteries	
III	7–12	• Basilar artery (BA) completely formed • PComA completely formed • Regression of trigeminal and proatlantal arteries • Formation of the vertebral artery VA (initiation)	
IV	13–15	• Migration of OccA origin on the external carotid artery (ECA) • Persistence of the anastomoses between OccA and VA	
V	16–18	• Complete formation of the VA • Complete formation of the OccA (remnant of the proatlantal artery) • Complete formation of the ascending pharyngeal artery (APhA, remnant of the hypoglossal artery)	

Dural Arteries

The occipital artery (OccA) is a particular artery because it supplies muscles of the neck, the dura of the posterior fossa, and the peripheral cranial and cervical nerves. In this paragraph, only dural branches of the occipital will be described (Table 2).

Dural branches of the OccA principally originate from the second segment of the artery. The largest dural branch is the mastoid artery that passes through the mastoid foramen [8]. This artery supplies the lateral part of the posterior fossa dura and gives off three distinct branches:

- The mastoid branch that courses superiorly after its entrance into the posterior fossa
- The hypoglossal branch, which has a descending course toward the hypoglossal canal
- The jugular branch that is oriented to the jugular foramen

Few authors also described dural branches that are transosseous branches from the second segment of the occipital artery. These thin branches pierces the occipital bone along the course of the artery to supply the dura of the cerebellar fossa [9]. Other dural branches from the OccA follow the radicular nerves of the first and second intervertebral spaces. Among these "radicular" arteries, the dural artery of the first intervertebral space could be larger and could give off the artery of the falx cerebelli (in 50% of cases). In other cases, this artery could originate from the posteroinferior cerebellar artery or directly from the vertebral artery [8, 10]. Among variations of the dural territory of the OccA, the posterior meningeal artery, which is normally a dural branch of the vertebral artery, could take its origin from the occipital artery and represents a partial persistence of the proatlantal artery Type I [2].

Another inconstant dural branch originates from the terminal part of the OccA and passes through the parietal foramen; it is consequently named the parietal branch or the supratentorial ramus [11]. A clinical case showing the dural branches of the OccA is presented in Fig. 1.

Table 2 Different dural branches of the occipital artery with their respective anastomosis

Occipital artery branches	Segment of the OccA	Territory (dural and neural)	Possible anastomosis
Mastoid branch (superior ramus)	First	Petrosal dura (lateral part) Wall of sigmoid sinus Endolymphatic sac and duct	Petrosquamosal branch (MMA) MHT (internal carotid artery) Subarcuate artery (AICA)
Mastoid branch (jugular ramus)	First	Wall of jugular bulb Posterior part foramen magnum	Jugular branch (AscPhaA)
Mastoid branch (hypoglossal ramus)	First	Wall of inferior petrosal sinus	Hypoglossal branch (AscPhaA)
Artery of the falx cerebelli	Second	Falx cerebelli	Posterior meningeal artery (Vertebral artery) Artery of the falx cerebri (MMA)
Parietal branch	Third	Parietal convexity dura	Parietal branch (MMA)

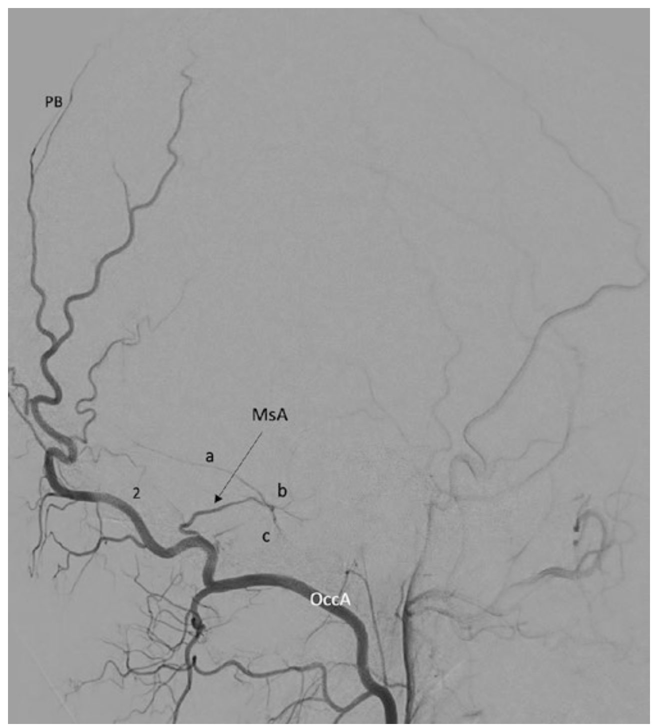


Fig. 1 Dural branches occipital artery (OccA). The lateral view digital subtraction angiography (DSA) shows an injection of the external carotid artery with visualization of the OccA. Among the dural branches, the largest is the mastoid artery (MsA). It passes through the mastoid foramen and gives off three branches: the mastoid (*a*), hypoglossal (*b*), and jugular branches (*c*). The terminal segment gives an inconstant terminal dural branch, which enters the parietal foramen, called parietal branch (PB)

Anastomoses With Other Dural Branches

The occipital artery is particular for its important number and dangerous anastomosis especially with the vertebrobasilar system. Even if arterio-arterial anastomoses between dural arteries do not represent the most important danger for embolization, their comprehensive knowledge helps in understanding pathologies such as dural arteriovenous fistulas or meningiomas [7].

The occipital artery has an important network with other dural arteries; its dural territory is always in balance with these other dural branches. The most important anastomotic network is formed with dural branches of the vertebral artery in the region of the foramen magnum and in the dura of the cerebellar convexity [11]. Posterior meningeal artery and the artery of the falx cerebelli, which could be branches of the OccA or of the vertebral artery, represent an important zone of anastomosis between OccA and vertebral artery [10]. The artery of the falx cerebelli also anastomoses at its upper end with the equivalent branch from the middle meningeal artery [8, 11]. The occipital artery presents anastomoses through its mastoid branch with the ascending pharyngeal artery (jugular and hypoglossal branches), with the anteroinferior cerebellar artery (subarcuate artery) and with the internal carotid artery (through the meningohypophyseal and inferolateral trunks) [12, 13]. Another anastomosis is present between the OccA and the middle meningeal artery through the parietal branch of the OccA (also called the supratentorial ramus), which enters the cranial cavity through the parietal foramen [1, 14].

Dural Territory

The complete dural territory of the occipital artery is in the posterior fossa, and variations are very frequent due to the sharing of this supply with several other dural arteries [11]. The dural territory of the occipital is the lateral part of the cerebellopontine angle (petrosal dura) and the cerebellar fossa [15]. Branches of the mastoid artery also supply the wall of the inferior petrosal sinus, the wall of the sigmoid sinus, and the jugular bulb [11]. The cerebellar fossa and the posterior edge of the foramen magnum are supplied by the occipital artery, which is in balance with dural branches of the vertebral artery. Also, the terminal part of the occipital artery could have a dural supply on the parietal convexity (parietal branch) [1].

Clinical Implications

A comprehensive knowledge in the anatomy of the occipital artery is mandatory for neck surgeons but also for interventional neuroradiologists and in a few situations for neurosurgeons. First, it is interesting to note that a few isolated cases of pulsatile tinnitus in cases of OccA origin of the vertebral artery or persistence of the proatlantal artery are described in the literature. This symptom, often difficult to explain, could be correlated in these situations with a high flow of the occipital artery.

In these last decades, the use of double-lumen catheter gives the possibility to use the occipital artery for liquid agent embolization in the case of dural arteriovenous fistulas. Knowledge of all dangerous anastomoses with the vertebrobasilar system and the “rule” for secure embolization are mandatory for these treatments. A clinical case of dural arteriovenous fistula supplied by dural branches of the OccA is presented in Fig. 2. The occipital artery is used as a donor for extracranial–intracranial bypasses. The OccA could be anastomosed to the posteroinferior cerebellar artery or with the posterior cerebral artery in cases of complex aneurysm trapping. In few reports, a variation in the origin of the OccA was described in the situation of a carotid endarterectomy. It is of course important to know the different possible origins of the OccA and to modify the surgical management according to the anatomical variation.

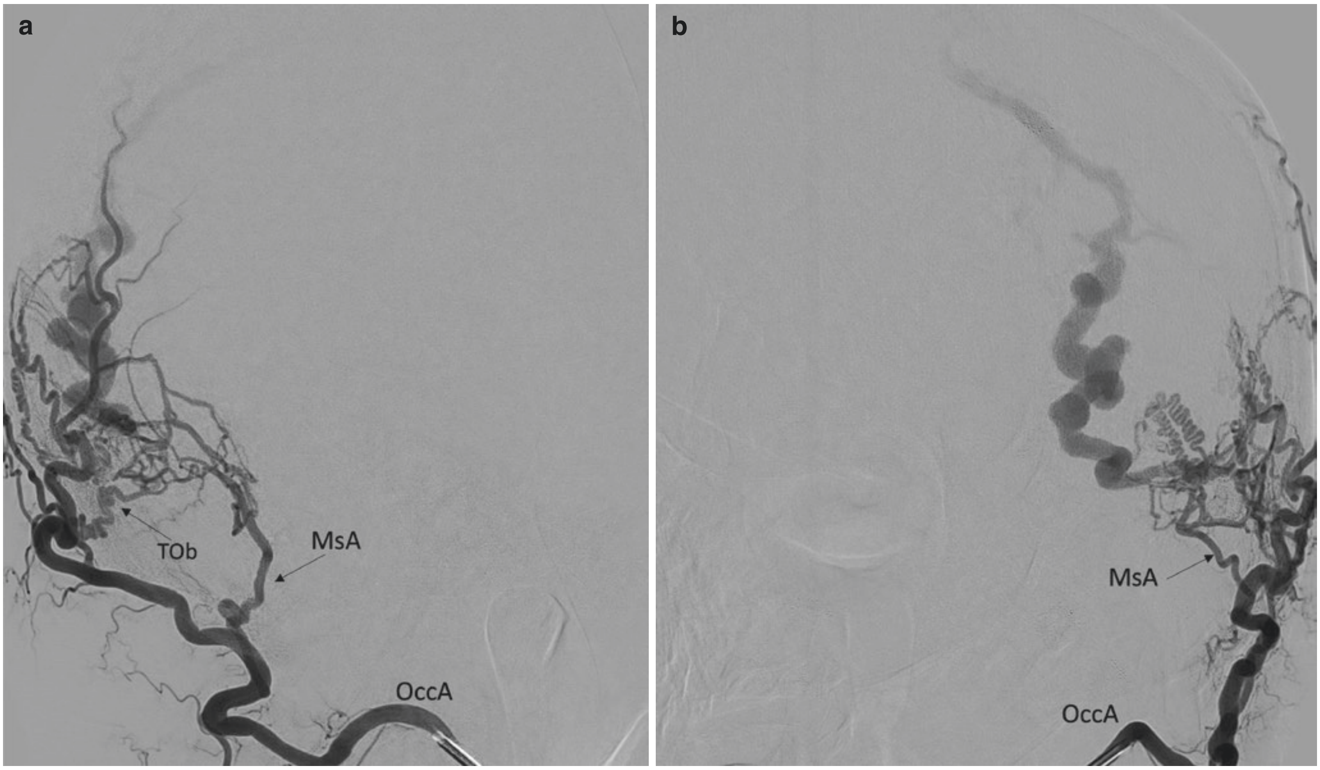


Fig. 2 Clinical case of dural arteriovenous fistula (dAVF) and enlargement of dural branches of the occipital artery (OccA). Figures (**a**) and (**b**), respectively, show a lateral and anteroposterior angiogram of the OccA. In this case, the mastoid artery (MsA) and transosseous branches (Tob) of the OccA are enlarged because of the presence of a dural arteriovenous fistula

References

1. Lasjaunias P, Theron J, Moret J. The occipital artery. Anatomy – Normal arteriographic aspects – Embryological significance. Neuroradiology. 1978;15(1):31–7. https://doi.org/10.1007/BF00327443.
2. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer edition; 2001.
3. Lasjaunias P, Moret J, Theron J. The so-called anterior meningeal artery of the cervical vertebral artery. Normal radioanatomy and anastomoses. Neuroradiology. 1978;17(1):51–5. https://doi.org/10.1007/BF00345270.
4. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
5. Padget DH. The development of cranial arteries in the human embryo, Contribution to Embryology, vol. 212. Washington: Carnegie Institution; 1948. p. 205–62.
6. Moffat DB, Morris ED. Abnormal origin of the basilar artery from the cervical part of the internal carotid and its embryological significance. Anat Rec. 1956;125(4):701–11. https://doi.org/10.1002/ar.1091250404.
7. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: A pictorial review. Surg Radiol Anat. 2021;43(6):961–73. https://doi.org/10.1007/s00276-021-02684-y.
8. Alvernia JE, Fraser K, Lanzino G. The occipital artery: A microanatomical study. Neurosurgery. 2006;58(1 Suppl):ONS114–22.; discussion ONS–22. https://doi.org/10.1227/01.NEU.0000193519.00443.34.
9. Parkinson D, Reddy V, Ross RT. Congenital anastomosis between the vertebral artery and internal carotid artery in the neck. Case report. J Neurosurg. 1979;51(5):697–9. https://doi.org/10.3171/jns.1979.51.5.0697.
10. Lasjaunias P, Manelfe C. Arterial supply for the upper cervical nerves and the cervicocarotid anastomotic channels: Systematization of radiological anatomy. Neuroradiology. 1979;18(3):125–31. https://doi.org/10.1007/BF00346866.
11. Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A Jr. Microsurgical anatomy of the dural arteries. Neurosurgery. 2005;56(2 Suppl):211–51.; discussion −251. https://doi.org/10.1227/01.neu.0000144823.94402.3d.
12. Aggarwal NR, Krishnamoorthy T, Devasia B, Menon G, Chandrasekhar K. Variant origin of superior thyroid artery, occipital artery and ascending pharyngeal artery from a common trunk from the cervical segment of internal carotid artery. Surg Radiol Anat. 2006;28(6):650–3. https://doi.org/10.1007/s00276-006-0145-5.
13. Graham RB, Sugrue PA, Rahme RJ, Batjer HH, Bendok BR. Pharyngo-occipital artery variant arising from the internal carotid artery impacting surgical technique during carotid endarterectomy. J Neurointerv Surg. 2013;5(3):e14. https://doi.org/10.1136/neurintsurg-2011-010215.
14. Geibprasert S, Pongpech S, Armstrong D, Krings T. Dangerous extracranial-intracranial anastomoses and supply to the cranial nerves: Vessels the neurointerventionalist needs to know. AJNR Am J Neuroradiol. 2009;30(8):1459–68. https://doi.org/10.3174/ajnr.A1500.
15. Salamon G, Guerinel G, Demard F. Radioanatomical study of the external carotid artery. Ann Radiol (Paris). 1968;11(3):199–215.

Dural Branches of the Ascending Pharyngeal Artery

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The dural territory of the ascending pharyngeal artery (APhA) is very important since it is involved in the majority of dural arteriovenous fistulas [1]. The APhA was known as a dangerous artery for the endovascular treatment of such pathologies due to its participation in the cranial nerves' vascularization and due to its numerous anastomoses with the vertebral artery [2]. But, with the amount of more navigable and thin micro-catheters, the APhA became an option these last few years for embolization [3]. For this reason, knowledge of dural branches of the APhA is mandatory before treating such pathologies. In this chapter, we will focus on the description of the dural branches of the APhA. This artery will also be described in detail in chapter "Embryology and Variations of the Ascending Pharyngeal Artery."

Embryological Development

In this chapter, only notions about embryological development that allow to understand the pharyngo-occipital system will be developed (Table 1). For more detailed embryological development about the vertebral artery, the carotid system, and the carotid-vertebral anastomoses, we give you to the respective chapters.

Early in embryological life (Stage I of Padget, embryos of 4–5 mm), the formation of the vertebrobasilar system initiates with the presence of the paired longitudinal artery named "longitudinal neural arteries (LNAs)" [4]. These LNAs are supplied by the carotid system through different carotid-vertebral anastomoses, and the vertebral arteries are not developed yet. LNAs are supplied by the trigeminal artery, the otic artery, the hypoglossal artery, and the proatlantal artery from cranial to caudal [1, 5].

During the Stage II of Padget (embryos of 5–6 mm), the caudal division of the primitive internal carotid artery (ICA) anastomoses with the cranial part of the LNA (formation of the posterior communicating artery) and consequently the carotid-vertebral anastomoses initiate their involution. The first two arteries that regress are the otic and the hypoglossal arteries, and the LNAs are supplied principally by the trigeminal artery cranially and the proatlantal artery caudally. At the same time, the two LNAs come closer to the midline to fuse together [4, 5].

At Stage III of Padget (embryos of 7–12 mm), the basilar artery is completely formed by fusion of the two LNAs as well as the posterior communicating artery, which is anastomosed with the upper part of the basilar artery. Trigeminal and proatlantal arteries progressively regress, and the primitive vertebral artery is formed by transverse anastomoses between the six upper cervical intersegmental arteries [1, 4, 6].

The ascending pharyngeal artery (APhA) is considered as the remnant of the hypoglossal artery. This is the reason why P. Lasjaunias named it the "artery of the first cervical somite" [7, 8]. Between Stages III and V, the origin of the APhA shifts from the primitive internal carotid artery to the external carotid artery, and the distal part of the primitive hypoglossal artery leaves only the territory of the hypoglossal branch of the APhA. At Stage V (embryos of 16–18 mm), the definite ascending pharyngeal artery could be clearly individualized [1, 9–11].

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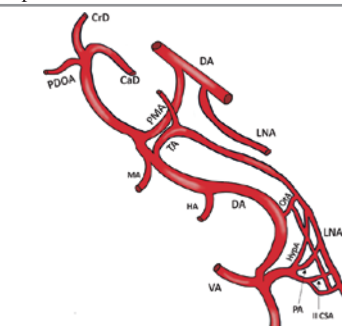
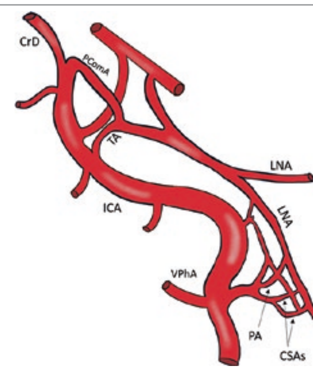
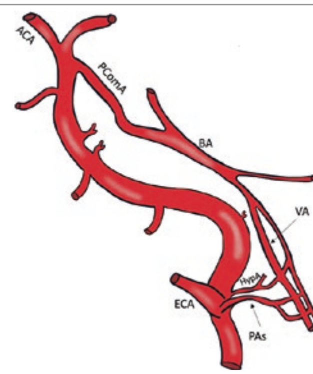
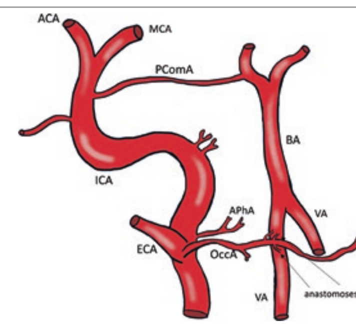
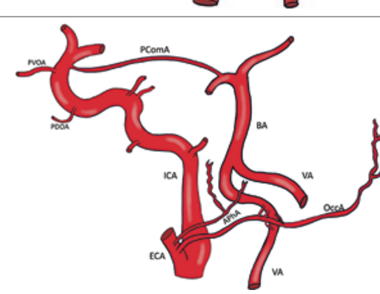
Table 1 Major embryological changes in the formation of the occipito-pharyngeal system

Stage	Embryo size (mm)	Major evolutions	Graphic illustration
I	4–5	• Paired longitudinal neural arteries (LNA) • Trigeminal (TA), otic (OtA), hypoglossal (HypA), proatlantal (PA), and second cervical segmental artery (II CSA) • PComA not formed	
II	5–6	• Formation of the PComA • Regression of otic and hypoglossal arteries • Fusion of LNAs in basilar artery (initiation)	
III	7–12	• Basilar artery (BA) completely formed • PComA completely formed • Regression of trigeminal and proatlantal arteries • Formation of the vertebral artery VA (initiation)	
IV	13–15	• Migration of OccA origin on the external carotid artery (ECA) • Persistence of the anastomoses between OccA and VA	
V	16–18	• Complete formation of the VA • Complete formation of the OccA (remnant of the proatlantal artery) • Complete formation of the ascending pharyngeal artery (APhA, remnant of the hypoglossal artery)	

Dural Branches

APhA presents three distinct dural branches, one from its anterior trunk and two from its posterior one. Table 2 summarizes these dural branches with their respective territory and arterio-arterial anastomoses.

Branch of the Foramen Lacerum or Carotid Ramus

This little dural branch named the "branch of the foramen lacerum or carotid ramus" depends on the anterior trunk of the APhA and particularly the superior pharyngeal ramus. It enters the skull base by the foramen of the same name [12–14]. This APhA branch is inconstant and found in 85% of cases [15].

The carotid branch of the APhA has a little dural territory in balance with dural branches of the ICA [12]. It is limited to the periosteum of the foramen lacerum and of the carotid canal. This artery also gives little vasa vasorum to the wall of the ICA in its petrous segment [1].

It gives anastomoses to the recurrent artery of the foramen lacerum, which is a dural branch of the internal carotid artery [1, 15–17]. Martins et al. (2005) also described inconstant anastomoses with the inferolateral trunk of the ICA, the cavernous branch of the MMA, and the accessory meningeal artery [12].

Jugular Branch

The jugular branch of the APhA enters the skull base through the jugular foramen. After it, this little dural branch gives off a medial branch that courses along the inferior petrosal sinus and a lateral branch that courses on the petrosal surface.

The jugular branch of the APhA supplies the dura of the jugular foramen; the petrosal surface (inferior part); and the wall of the jugular bulb, the inferior petrosal sinus, and the sigmoid sinus (inferior part) [12].

The jugular branch of the APhA has duro-dural anastomosis with the hypoglossal branch of the APhA because of the proximity of their respective territories [12]. It also anastomoses with meningeal branches of the occipital artery with the subarcuate artery (anteroinferior cerebellar artery), with the petro-squamosal branch of the MMA, and with the lateral clival artery (meningohypophyseal trunk of the ICA) [18]. Effendi et al. (2015) also described a possible anastomosis between the jugular branch of the APhA and the posteroinferior cerebellar artery (PICA) [19].

Hypoglossal Branch

The hypoglossal branch enters the skull through the hypoglossal canal (anterior condyloid canal) and is considered as the remnant of the primitive hypoglossal artery. It is composed by an ascending branch that follows the clivus and a descending branch that participates in the odontoid arterial arch [12, 15].

The hypoglossal branch of the APhA supplies the inferior part of the clival dura, the anterolateral part of the foramen magnum, and the inferior part of the cerebellar fossa [15, 20].

It has a more complex anastomotic network organizing as follows. Through its descending branch, it participates in the odontoid arterial arch and mainly anastomoses with the anterior meningeal artery (vertebral artery branch) at the C3 level [10]. It presents also anastomoses with the posterior meningeal artery (vertebral artery), with meningeal branches of the occipital artery, and with the medial clival artery (bilateral meningohypophyseal trunk) [15].

Two clinical cases of dural arteriovenous fistula showing the jugular and hypoglossal branches of the APhA are presented in Figs. 1 and 2.

Table 2 Different dural branches of the ascending pharyngeal artery (APhA) with their respective anastomosis

APhA branches	Trunk	Dural territory	Possible anastomosis
Branch of the foramen lacerum (carotid ramus)	Anterior	Foramen lacerum, carotid canal, and carotid artery wall	Recurrent artery of the foramen lacerum (cavernous ICA) Inferolateral trunk (cavernous ICA) Cavernous branch (MMA) Accessory meningeal artery
Jugular branch	Posterior	Jugular foramen, wall inferior petrosal sinus, wall jugular bulb, wall sigmoid sinus, and dura petrosal surface	Petrosquamous branch (MMA) Subarcuate artery (AICA) Lateral clival artery (ICA) Meningeal branch (occipital artery)
Hypoglossal branch	Posterior	Anterolateral part foramen magnum, clivus, and cerebellar fossa	Anterior meningeal artery (vertebral artery) Medial clival artery (ICA) Meningeal branch (occipital artery)

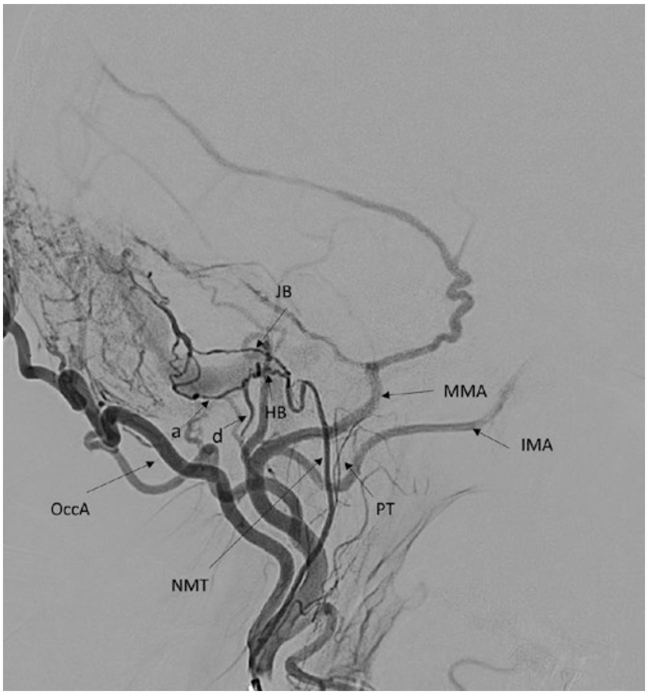


Fig. 1 Clinical case of dural arteriovenous fistula (dAVF) with neuromeningeal trunk. The figure shows the case of a dural arteriovenous fistula supplied by the occipital artery (OccA), the middle meningeal artery (MMA), and the neuromeningeal trunk (NMT) of the ascendent pharyngeal artery. The pharyngeal trunk (PT) is visible but smaller and not pathologic. The NMT gives origin to a jugular branch (JB) and a hypoglossal branch (HB). This latter divides into an ascendent (a) and a descendent (b) trunk. The internal maxillary artery (IMA) is also visible

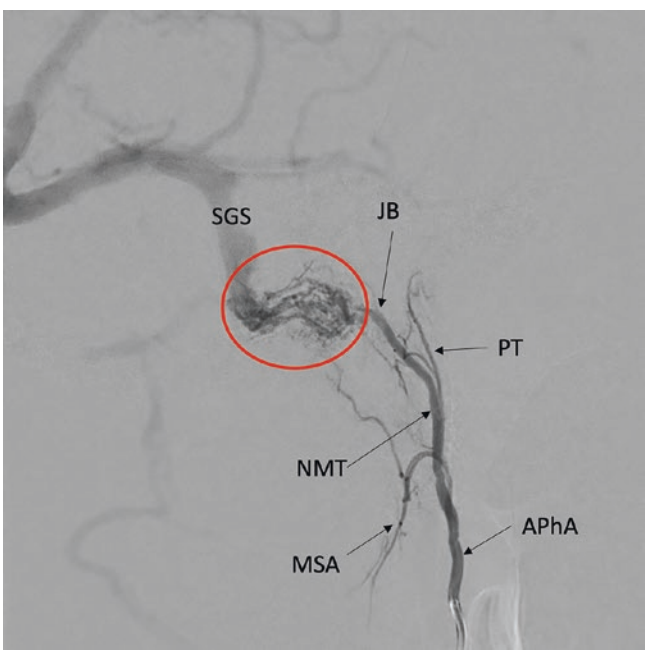


Fig. 2 Clinical case of dural arteriovenous fistula (dAVF). The patient presents a dural arteriovenous fistula at the level of the junction between the sigmoid sinus (SGS) and the jugular vein (red circle). The ascending pharyngeal artery (APhA) contributes to the fistula with the neuromeningeal trunk (NMT) and its jugular (JB). The pharyngeal trunk (PT) and the muscolo-spinal artery are also visible

Variations Implicating the Dural Branches of the APhA

Ascending Pharyngeal Artery Origin of the Posteroinferior Cerebellar Artery (PICA) (Pharyngo-Cerebellar Artery)

In 1973, Teal et al. described a case of "PICA that origins from the cervical ICA" without more explanation [21]. After them, Lasjaunias et al. in 1981 showed a case of APhA origin of the PICA illustrated with angiographic images without CT scan [22]. They interpreted the case as a partial persistent of the hypoglossal artery and spoke about an anastomosis between the hypoglossal branch of the APhA and the PICA. Unfortunately, they did not prove it with osseous CT scan and named this variation the "pharyngo-cerebellar artery." This last decade, eight isolated case reports of APhA origin of the PICA were successively described [19, 23–28]. All these cases are similar with the Lasjaunias' case with the anatomical difference that the "aberrant" artery does not pass through the hypoglossal canal but through the jugular canal. This important detail raised the matter that this variation is not a partial persistent hypoglossal artery but could be the consequence of another embryonic carotid-vertebral anastomosis not already known passing through the jugular foramen. Ryi et al. (2016), observing that the anastomotic vessel passes through the pars vascularis of the foramen, named this variant a "persistent primitive glossopharyngeal artery" [25].

Ascending Pharyngeal Artery Origin of the Posterior Meningeal Artery

Salamon et al. (1967), in their description of the posterior fossa dura vascularization, cited that the posterior meningeal artery could sometimes arise from the ascending pharyngeal artery [13]. After their original description, no other case of this variation was described in the literature.

References

1. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer edition; 2001.
2. Lapresle J, Lasjaunias P. Cranial nerve ischaemic arterial syndromes. A review. Brain. 1986;109(Pt 1):207–16. https://doi.org/10.1093/brain/109.1.207.
3. Geibprasert S, Pongpech S, Armstrong D, Krings T. Dangerous extracranial-intracranial anastomoses and supply to the cranial nerves: Vessels the neurointerventionalist needs to know. AJNR Am J Neuroradiol. 2009;30(8):1459–68. https://doi.org/10.3174/ajnr.A1500.
4. Padget DH. The development of cranial arteries in the human embryo, Contribution to Embryology, vol. 212. Washington: Carnegie Institution; 1948. p. 205–62.

5. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: A pictorial review. Surg Radiol Anat. 2021;43(6):961–73. https://doi.org/10.1007/s00276-021-02684-y.
6. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
7. Lasjaunias P, Moret J. The ascending pharyngeal artery: Normal and pathological radioanatomy. Neuroradiology. 1976;11(2):77–82. https://doi.org/10.1007/BF00345017.
8. Lasjaunias P, Doyon D. The ascending pharyngeal artery and the blood supply of the lower cranial nerves. J Neuroradiol. 1978;5(4):287–301.
9. Lasjaunias P, Theron J, Moret J. The occipital artery. Anatomy – Normal arteriographic aspects – Embryological significance. Neuroradiology. 1978;15(1):31–7. https://doi.org/10.1007/BF00327443.
10. Lasjaunias P, Moret J, Theron J. The so-called anterior meningeal artery of the cervical vertebral artery. Normal radioanatomy and anastomoses. Neuroradiology. 1978;17(1):51–5. https://doi.org/10.1007/BF00345270.
11. Lasjaunias P, Manelfe C. Arterial supply for the upper cervical nerves and the cervicocarotid anastomotic channels: Systematization of radiological anatomy. Neuroradiology. 1979;18(3):125–31. https://doi.org/10.1007/BF00346866.
12. Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A Jr. Microsurgical anatomy of the dural arteries. Neurosurgery. 2005;56(2 Suppl):211–51.; discussion −251. https://doi.org/10.1227/01.neu.0000144823.94402.3d.
13. Salamon G, Grisoli J, Paillas JE, Faure J, Giudicelli G. Arteriographic study of the meningeal arteries. (Role of selective injections, of subtraction and of radio-anatomical correlations). Neurochirurgia (Stuttg). 1967;10(1):1–19. https://doi.org/10.1055/s-0028-1095333.
14. Salamon G, Guerinel G, Demard F. Radioanatomical study of the external carotid artery. Ann Radiol (Paris). 1968;11(3):199–215.
15. Hacein-Bey L, Daniels DL, Ulmer JL, Mark LP, Smith MM, Strottmann JM, et al. The ascending pharyngeal artery: Branches, anastomoses, and clinical significance. AJNR Am J Neuroradiol. 2002;23(7):1246–56.
16. Cavalcanti DD, Reis CV, Hanel R, Safavi-Abbasi S, Deshmukh P, Spetzler RF, et al. The ascending pharyngeal artery and its relevance for neurosurgical and endovascular procedures. Neurosurgery. 2009;65(6 Suppl):114–20.; discussion 20. https://doi.org/10.1227/01.NEU.0000339172.78949.5B.
17. Quisling RG, Seeger JF. Ascending pharyngeal artery collateral circulation simulating internal carotid artery hypoplasia. Neuroradiology. 1979;18(5):277–80. https://doi.org/10.1007/BF00327693.
18. Manelfe C, Roulleau J, Combrelles R, Grisoli F. The vascularization of the dura mater of the occipital foramen. Neurochirurgie. 1971;17(6):469–80.
19. Effendi K, Magro E, Gentric JC, Darsaut TE, Raymond J, Seizeur R, et al. Anastomosis between the ascending pharyngeal artery and the posterior inferior cerebellar artery through the jugular foramen: A cadaveric observation. Oper Neurosurg (Hagerstown). 2016;12(2):163–7. https://doi.org/10.1227/NEU.0000000000001098.
20. Al-Rafiah A, AA EL-H, Aal IH, Zaki AI. Anatomical study of the carotid bifurcation and origin variations of the ascending pharyngeal and superior thyroid arteries. Folia Morphol (Warsz). 2011;70(1):47–55.
21. Teal JS, Rumbaugh CL, Segall HD, Bergeron RT. Anomalous branches of the internal cartoid artery. Radiology. 1973;106(3):567–73. https://doi.org/10.1148/106.3.567.
22. Lasjaunias P, Guibert-Tranier F, Braun JP. The pharyngo-cerebellar artery or ascending pharyngeal artery origin of the posterior inferior cerebellar artery. J Neuroradiol. 1981;8(4):317–25.
23. Fujihara F, Takahara M, Katsuta T, Takemoto K, Higashi T, Inoue T. Posterior inferior cerebellar artery originating from the jugular branch of the ascending pharyngeal artery. NMC Case Rep J. 2019;6(1):21–4. https://doi.org/10.2176/nmccrj.cr.2018-0171.
24. Iihoshi S, Kohyama S. Anomalous origin of the posterior inferior cerebellar artery from the jugular branch of the ascending pharyngeal artery: A case report. Radiol Case Rep. 2020;15(9):1697–700. https://doi.org/10.1016/j.radcr.2020.06.051.
25. Ryu B, Anami H, Ishikawa T, Inoue T, Sugiura M, Kawamata T. Extremely rare persistent primitive artery passing through the jugular foramen with symptomatic ipsilateral carotid artery stenosis. Acta Neurochir. 2016;158(10):1925–9. https://doi.org/10.1007/s00701-016-2896-z.
26. Thomas KL, Hughes MA, Frederickson AM, Branstetter BFt, Vilensky JA, Sekula RF. Hemifacial spasm caused by an aberrant jugular branch of the ascending pharyngeal artery. Br J Neurosurg. 2015;29(1):97–9. https://doi.org/10.3109/02688697.2014.957158.
27. Uchino A, Ohno H, Kondo R, Ishihara S. Ascending pharyngeal artery-posterior inferior cerebellar artery anastomosis via the jugular foramen: A case report and literature review. Surg Radiol Anat. 2021;43(6):1019–22. https://doi.org/10.1007/s00276-020-02667-5.
28. Uchino A, Suzuki C. Posterior inferior cerebellar artery supplied by the jugular branch of the ascending pharyngeal artery diagnosed by MR angiography: Report of two cases. Cerebellum. 2011;10(2):204–7. https://doi.org/10.1007/s12311-011-0267-0.

Arterial Supply of the Cranial Nerves

Thomas Robert and Sara Bonasia

The blood supply of cranial nerves represents only a very specific part of craniofacial vascularization. Even if the number of publications about this topic is not very important, few authors dedicated their works on it and gave an important contribution in the understanding of different vascular systems that participate in cranial nerves supply [1–3]. Among them, Rhoton, Gibo, and Marinkovic published tremendous anatomic works concentrated on vascular supply of the cisternal part of few cranial nerves [4–8]. On the other hand, Lapresle and Lasjaunias also published fantastic reflections correlating vascular anatomy, embryological knowledge, and neurological syndromes [9–17]. Knowledge of the blood supply of cranial nerves takes an important place these last decades to avoid neurological complications during external carotid artery embolization [13, 18, 19]. In this chapter, after a little correlation between embryological development and cranial nerves supply, we will summarize the vascular supply of each nerve. Finally, we will present different vascular syndromes of cranial nerves deficit.

Embryological Development

Cranial nerves could not be considered equally from an embryological point of view. Most of them are segmentary nerves as spinal nerves, but some of them are particular [9]. The first two cranial nerves (olfactory and optic nerves) are not true nerves but are prolongations of the central nervous system [20]. The 12 cranial nerves (hypoglossal nerve) are not cranial nerves but spinal nerves that are incorporated in the cranial cavity. Finally, the eleventh (accessory) nerve has also some particularity because it represents the fusion of two different nerves: the first one is cranial and the second one is cervical [1].

Embryological development of cranial nerves is mainly done during the second month of the embryological life, after the development of the primitive vascular system, and one could consider that the primitive arterial craniofacial system serves as a tutor to the cranial nerves development [9].

We could associate each cranial nerve to a primitive vascular system or a carotid-vertebral anastomosis as follows (Table 1):

- Olfactory nerve (I) with the primitive olfactory artery
- Optic nerve (II) with the ventral primitive ophthalmic artery
- Oculomotor (III), trochlear (IV), and abducens (VI) nerves with the dorsal primitive ophthalmic artery
- Trigeminal nerve (V) with the primitive trigeminal artery
- Facial (VII) and cochlea-vestibular nerve (VIII) with the stapedial artery
- Glossopharyngeal (IX), vagus (X), and accessory (XI) nerves with the primitive glossopharyngeal artery
- Hypoglossal nerve (XII) with the primitive hypoglossal artery

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Table 1 Development of cranial nerves depending on primitive embryological arteries

Cranial nerves	Branchial arch	Primitive arterial system	Main blood supply
Olfactory nerve (I)	Pre-branchial	Primitive olfactory artery	Olfactory artery
Optic nerve (II)	Pre-branchial	Ventral primitive ophthalmic artery	Ophthalmic artery
Oculomotor nerve (III) Trochlear nerve (IV) Abducens nerve (VI)	First branchial	Dorsal primitive ophthalmic artery	Inferolateral trunk
Trigeminal nerve (V)	First branchial	Primitive trigeminal artery	Meningohypophyseal trunk
Facial nerve (VII) Cochleovestibular nerve (VIII)	Second branchial	Stapedial artery	Middle meningeal artery
Glossopharyngeal nerve (IX) Vagus nerve (X) Accessory nerve (XI)	Third branchial	Primitive glossopharyngeal artery	Jugular branch of the ascending pharyngeal artery
Hypoglossal nerve (XII)	Fourth branchial	Primitive hypoglossal artery	Hypoglossal branch of the ascending pharyngeal artery

Blood Supply of Each Cranial Nerve

Olfactory Nerve

Most of the blood supply of the olfactory nerve comes from the olfactory artery [3]. This is a little branch of the anterior cerebral artery that branches off from the lateral aspect of the A2 segment directly in 54% or from the medial frontobasal artery in 46% [21]. The olfactory artery is the remnant of the primitive olfactory artery and courses in the olfactory cistern, deep to the nerve [21, 22]. It gives branches to all the tract and the bulb of the olfactory nerve [3].

The olfactory bulb and the bipolar neurons have another vascular supply from the pterygopalatine artery and from the posterior and anterior ethmoidal arteries. In few cases, a larger branch from the posterior ethmoidal artery supplies the olfactory bulb and is named the "accessory olfactory artery" [3, 23].

Cases of anosmia after clipping or coiling of anterior communicating artery aneurysms was published, and one of the hypotheses is a stripping or thrombosis of the olfactory artery that led to an olfactory tract necrosis [24–27].

The arterial supply of the olfactory nerve is illustrated in Fig. 1.

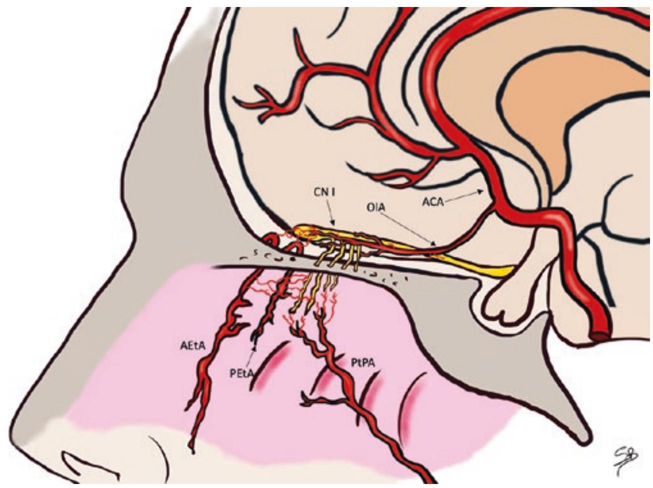

Fig. 1 Illustration of the vascular supply of the olfactory nerve. The olfactory nerve (CN I) is supplied by the olfactory artery (OlA), which arises from the A2 segment of the anterior cerebral artery (ACA), from branches of the anterior and posterior ethmoidal arteries (AEtA, PEtA), and from terminal branches of the pterygopalatine artery (PtPA)

Optic Nerve

The optic nerve has long intracisternal and intraorbital courses with a complex blood supply that depends predominantly on the ventral primitive ophthalmic artery. The vascular supply of this nerve is illustrated in Fig. 2.

The blood supply of the optic tract is ensured by perforating arteries from the anterior choroidal artery (posterior part) and from the internal carotid artery and posterior communicating artery (anterior part) [21].

The optic chiasm benefits on a rich arterial network that could be divided in three distinct parts [28, 29]:

- The superior part is composed by perforating arteries from the anterior cerebral artery and from the anterior communicating artery [28, 30].
- The inferior network is the most developed one and is mainly supplied by the recurrent branch of the superior hypophyseal artery. The other perforating arteries come from the internal carotid artery, the posterior communicating artery, and the basilar artery [3].
- The lateral network is composed of branches from the internal carotid artery [31].

The cisternal part of the optic nerve is also supplied by the recurrent branch of the superior hypophyseal artery that

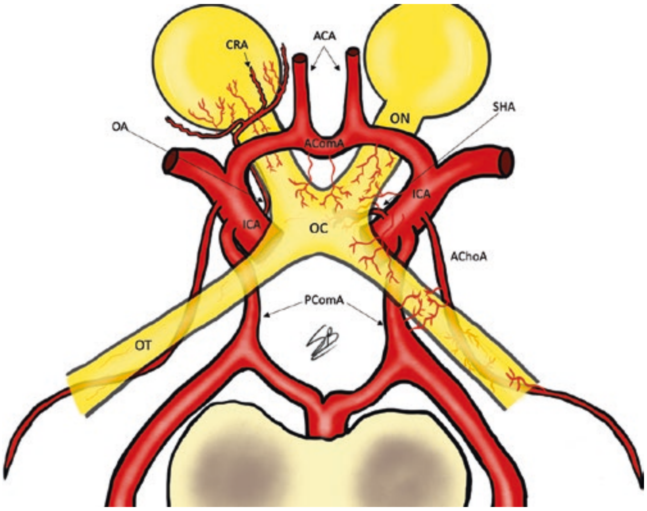

Fig. 2 Vascular supply of the optic nerve, chiasm, and tract. *ACA* anterior cerebral artery, *AChoA* anterior choroidal artery, *CRA* central retinal artery, *ICA* internal carotid artery; *OA* ophthalmic artery, *OC* optic chiasm, *ON* optic nerve, *OT* optic tract, *PComA* posterior communicating artery, *SHA* superior hypophyseal artery

courses inferiorly to the optic nerve from posterior to anterior and finishes at the optic canal region [32].

The intracanalicular portion of the nerve is followed by the ophthalmic artery that courses under the optic nerve and gives one to three distinct branches destined to the nerve [33].

Into the orbit, the optic nerve is always followed by the ophthalmic artery and by the central retinal artery that ensure most of its blood supply [33–36]. The central retinal artery penetrates the optic nerve posterior to the ocular globe to finish into the retina. Inside the optic nerve, the central retinal artery gives recurrent branches that constitute a centrifugal arterial system to the nerve. On the other hand, short and long ciliary arteries also give branches that penetrate the optic nerve and represent a centripetal blood supply system (arterial circle of Zinn) [34–38].

Oculomotor Nerve

The oculomotor nerve is one of the nerves that depends on the first branchial arch, the mandibular artery (dorsal primitive ophthalmic artery). Consequently, most of its blood supply is ensured by the inferolateral trunk (ILT) of the internal carotid artery [1, 39–41].

The cisternal part of the nerve is supplied by perforating arteries from the basilar and posterior cerebral arteries [3, 42]. In the majority of cases, these perforating branches are mesencephalic arteries (88.9%) or diencephalic branches (40.7%) but could also be branches from the collicular artery (long circumflex artery) in 51.8% from the P1 segment of the posterior cerebral artery [4]. In few cases, both oculomotor nerves in their cisternal part could be supplied by only one bilateral trunk from the basilar artery [9]. The most anterior part of the cisternal portion of the nerve also received a vascular supply by perforating arteries from the posterior communicating artery and from the tentorial artery (branch of the meningohypophyseal trunk) [3].

In the cavernous sinus, the ILT is the main supply of the oculomotor nerve [16, 40]. Its superior or tentorial branch follows the nerve and gives the majority of branches for its supply, while its anteromedial branch (artery of the superior orbital fissure, SOF) supplies the oculomotor nerve only in the anterior part of the cavernous sinus and into the SOF [43].

Into the orbit, the oculomotor nerve is supplied by branches of the ophthalmic artery, mainly muscular branches [40, 44, 45].

Trochlear Nerve

As the oculomotor nerve described before, the development of the trochlear nerve depends on the dorsal primitive ophthalmic artery and future ILT of the internal carotid artery [9, 39, 40].

During its long intracisternal course around the mesencephalon, the trochlear nerve is supplied by branches of the superior cerebellar artery (vermian, paravermian, and terminal arteries), the posterior cerebral artery, and the collicular artery (circumflex artery) [3, 42, 46].

Following the inferior border of the tentorium before entering the cavernous sinus, the nerve receives blood from the medial tentorial artery (or free margin artery of the tentorium) that is a branch of the meningohypophyseal trunk (MHT) or the ILT [16, 39]. Embryologically, this is the superior branch of the ILT that could originate from the MHT by anastomosis [9, 40].

In its cavernous course, the trochlear nerve is mainly supplied by the superior branch of the ILT, and in its anterior part, the artery of the SOF (anteromedial branch of the ILT) supplies the nerve [44, 47].

The ophthalmic artery or more precisely the posterior ethmoidal artery is the main supply of the nerve into the orbit [33].

Trigeminal Nerve

The development of the trigeminal nerve depends on a carotid-vertebral anastomosis during the embryological life: the trigeminal artery [9, 20, 48]. Considering that this temporary artery regresses early in the development, the blood supply of the trigeminal nerve is complex, also including the middle meningeal artery (MMA) and the internal maxillary artery [2, 49]. Carotid and basilar remnants of the trigeminal artery participate in the blood supply of the nerve but are helped by other vascular systems thanks to different arterio-arterial anastomoses [39].

The cisternal part of the nerve is supplied by posterior remnants of the trigeminal artery that are thin branches from the anteroinferior cerebellar artery (AICA) (88%), the superior cerebellar artery (SCA) (12%), or directly from the basilar artery [18]. These branches form a proximal ring around the nerve with a centripetal supply and are named by some authors as superolateral pontine, inferolateral pontine, and posterolateral pontine arteries [2, 49].

In the Meckel's cave, the Gasserian ganglion has a complex blood supply that is organized as follows [15, 40, 44, 50, 51]:

- The medial third of the ganglion is supplied by the tentorial artery (branch of the MHT or of the ILT).
- The middle third is supplied by the superior branch of the ILT and by the carotid branch of the ascending pharyngeal artery (APhA).
- The lateral third of the ganglion is supplied by the cavernous and petrous branches of the MMA.

In the temporal fossa, each branch of the trigeminal nerve has a different supply that mainly depends on the ILT and MMA [9, 13].

The first division of the trigeminal nerve (V1) receives blood from the anteromedial branch of the ILT from its emergence to its entrance in the SOF [40].

The second division of the nerve (V2) is supplied by the anterolateral branch of the ILT, which is in balance with the artery of the foramen rotundum (branch of the internal maxillary artery) near the entrance of the nerve in the foramen of the same name [9, 39, 40].

The third division of the nerve (V3) and its motor nerve (Vm) receive supply from the posterior branch of the ILT, which is in balance with the accessory meningeal artery near the foramen ovale and with the cavernous branch of the MMA [50].

The participation of the ILT in the vascularization of the trigeminal nerve explains the possible involvement of V1 and V2 in case of ILT syndrome (see below) [15, 16].

Into the orbit, different branches of the V1 nerve are supplied by respective arteries: the lacrimal nerve by the lacrimal artery, the frontal nerve by the supraorbital artery, and the nasociliary nerve by ethmoidal arteries [2, 33, 52]. In the pterygopalatine fossa, the V2 nerve is supplied by the infraorbital artery (branch of the internal maxillary artery). The accessory meningeal artery remains the main blood supply of the V3 and Vm nerves in the infratemporal fossa [2].

Abducens Nerve

The abducens nerve, as the oculomotor and the trochlear nerves, is developed along the dorsal primitive ophthalmic artery [40, 41].

After its exit from the brain stem, the cisternal portion of the nerve is supplied by perforating arteries from the basilar artery directly, the AICA, and the ponto-medullary artery [3, 18].

In the dorsum sellae region and in the Dorello's canal, the abducens nerve received blood from the lateral clival artery (branch of the MHT) and from the jugular branch of the APhA [10, 42, 44, 45, 53].

During its intracavernous course, the main supply of the nerve is the anteromedial branch of the ILT that follows the nerve until its entrance into the SOF. Into the orbit, the nerve has a lateral course along the lacrimal artery that gives thin branches to the nerve [9, 16, 40].

Facial Nerve

The facial nerve depends on the stapedial system; consequently, even if its vascularization is multiple and complex, the MMA is the main blood supply of the facial nerve [9, 54–56].

The cisternal and meatal parts of the facial nerve are principally supplied by the AICA and its branches [2, 18]. The most important branch that supplies these parts of the nerve is the labyrinthine artery (or internal auditory artery) [13, 57, 58]. This artery is double (64%) or unique (36%) and gives off three distinct branches inside of the internal auditory canal: the anterior vestibular artery, the vestibulocochlear artery, and the cochlear artery [59].

In its intratemporal course, the facial nerve has a dual vascularization. The petrous branch of the MMA supplies the second segment of the nerve, and the stylomastoid artery (branch of the occipital artery or posterior auricular artery) supplies the third segment [57, 60, 61]. Anastomosis between these two arterial systems is known as the facial arcade [8, 11]. The blood supply of the intratemporal nerve is dual (MMA and stylomastoid artery) in 70–75%; the petrosal artery supply is dominant in 10%, and the stylomastoid artery is predominant in 15–20% [9, 11, 12]. In almost all cases, the labyrinthine artery has no anastomosis with the facial arcade, but Lasjaunias et al. described a case of anastomosis between the stylomastoid artery and the subarcuate artery (branch of the AICA) [9].

The geniculate ganglion and the great superficial petrosal nerve are supplied by the petrous branch of the MMA instead of the chorda tympani that depends on the stylomastoid artery [2, 61–64].

Distal to its intratemporal course, the facial nerve passes in the parotid gland where it receives branches from the internal maxillary artery. The superior branch of the facial nerve is supplied by the superior temporal artery and the transverse facial artery. The inferior branch of the nerve is supplied by the internal maxillary artery and the facial artery [2].

Cochleovestibular Nerve

The cochleovestibular nerve shares most of its vascularization with the facial nerve [2]. In the pontocerebellar cistern, the nerve is supplied by the AICA and the labyrinthine artery [57]. Direct branches from the basilar artery could also supply the nerve at its entrance in the brain stem [58, 65, 66].

In a case where there are two labyrinthine arteries (64%), the superior one becomes the vestibulocochlear artery in the internal auditory canal, and the inferior one gives two branches: the cochlear and the anterior vestibular arteries. If the labyrinthine artery is unique (36%), it gives the three distinct branches in the internal auditory canal [2].

The cisternal supply of the oculomotor, trochlear, trigeminal, abducens, facial, and vestibulocochlear nerves are illustrated in Fig. 3. The intracavernous supply of the oculomotor, trochlear, abducens, and trigeminal nerves is shown in Fig. 4.

Glossopharyngeal Nerve

The cisternal part of the glossopharyngeal nerve is supplied by a thin branch that arises from the vertebral artery (VA) and is named the artery of the glossopharyngeal nerve or artery of the lateral fossula of the medulla oblongata. This artery also supplies the vagus nerve in its cisternal portion [2].

In the jugular foramen, the jugular branch of the APhA supplies the three nerves that pass through the foramen: glossopharyngeal, vagus, and accessory nerves [9, 10, 14, 16, 17, 67, 68].

Extracranially, the nerve is also supplied by branches of the APhA in the retropharyngeal space instead of being supplied by the descending palatine artery (IMA branch), ascending palatine artery (facial artery branch), and sphenopalatine artery (IMA branch) in the tonsillar region. The dorsal lingual artery (branch of the lingual artery) supplies the terminal part of the nerve [2].

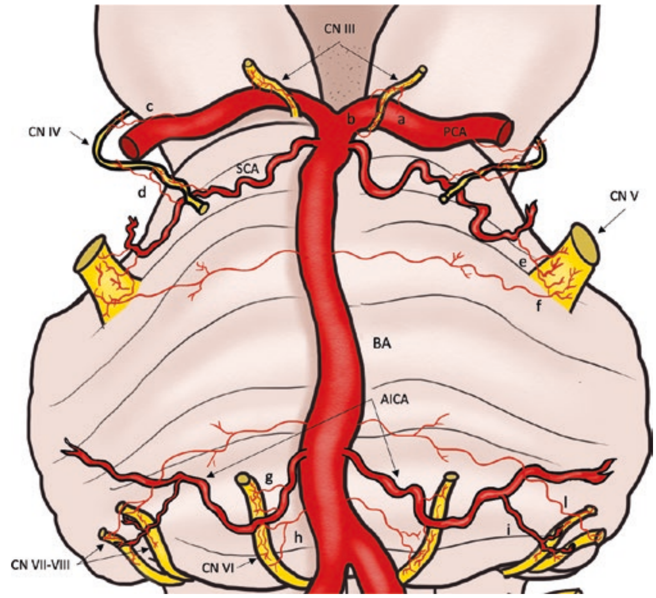

Fig. 3 Cisternal vascularization of the III, IV, V, VI, and VII–VIII cranial nerves. The oculomotor nerve (CN III) is supplied by branches (*a*) from the posterior cerebral artery (PCA) and the basilar artery (*b*, BA). The trochlear nerve (CN IV) is supplied by branches from the BA (*c*) from the superior cerebellar artery (*d*, SCA). The trigeminal nerve (CN V) receives its supply from the SCA (*e*) and direct branches from the BA (*f*). The abducens nerve (CN VI) is supplied by branches from the labyrinthine artery (*i*), from the anterosuperior cerebellar artery (AICA), and by direct branches from the BA (*l*)

Vagus Nerve

The artery of the lateral fossula of the medulla oblongata, branch of the vertebral artery, supplies the vagus nerve in its cisternal portion [2]. In the jugular foramen, the jugular branch of the APhA supplies the three nerves that pass through the foramen: glossopharyngeal, vagus, and accessory nerves [9, 10, 14, 16, 67]. In the laryngeal space, direct branches from the ICA supply the vagus nerve. In the cervical part, the vagus nerve receives branches from the ICA and the common carotid artery (CCA) but also from the inferior thyroid artery (named the vagal artery). In its thoracic part, the nerve is supplied by the vertebral artery and the internal thoracic artery [69].

Accessory Nerve

The accessory nerve is a particular nerve with its cranial and spinal divisions that receive a dual vascular supply [9, 10, 14, 16, 17, 67]. The spinal division of the nerve receives blood supply from the anterior spinal artery and posterior spinal

Fig. 4 Cavernous sinus vascularization of the III, IV, V, and VI cranial nerves. In this area the vascular supply is provided by the following arteries: (1) *Oculomotor nerve* (CN III): superior or tentorial branch (*a*) of the inferolateral trunk (ILT) and anteromedial branch (*b*) of the ILT. (2) *Trochlear nerve* (CN IV): superior or tentorial branch (*a*) of the ILT and anteromedial branch (*b*) of the ILT. (3) *Abducens nerve* (CN VI): anteromedial branch (*b*) of the ILT. (4) *Trigeminal nerve* (CN V): The first division of the trigeminal nerve (V1) receives blood from the anteromedial branch (*b*) of the ILT. The second division of the nerve (V2) is supplied by the anterolateral branch (*c*) of the ILT, which is in balance with the artery of the foramen rotundum (*m*, branch of the internal maxillary artery). The third division of the nerve (V3) receives supply from the posterior branch (*d*) of the ILT, which is in balance with the accessory meningeal artery (*n*) and with the cavernous branch of the middle meningeal artery (MMA) (*f*). The trigeminal ganglion (TG) is mostly supplied by the tentorial artery (*h*), a branch of the meningohypophyseal trunk (MHT); additional supply comes from the cavernous branch of the MMA (*f*), from the lateral trigeminal artery (*g*), from the internal carotid artery (ICA), and from the lateral clival artery (*l*) from the MHT

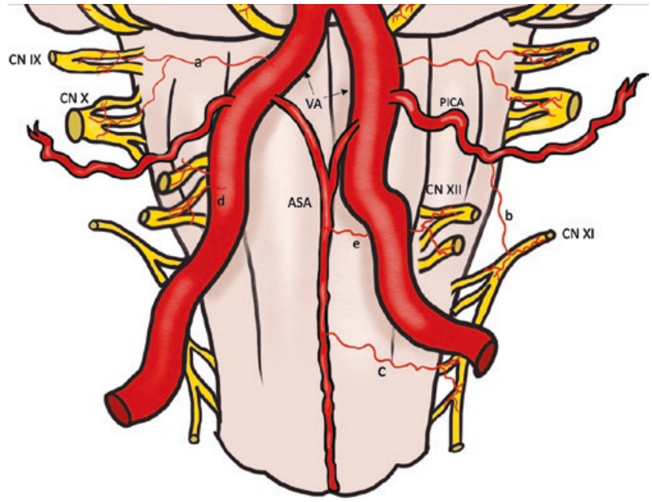

Fig. 5 Vascularization of the cisternal portion of the IX-X-XI-XII cranial nerves. The glossopharyngeal nerve (CN IX) is supplied by a branch that arises from the vertebral artery (VA) called artery of the glossopharyngeal nerve or artery of the lateral fossula of the medulla oblongata (*a*). The same artery also supplies the vagal nerve (CN X). The accessory nerve (CN XI) is supplied by a branch (*b*) of the posteroinferior cerebellar artery (PICA) in its cranial component and by branches (*c*) of the anterior spinal artery (ASA) in its spinal component. The hypoglossal nerve (CN XII) is supplied by the anterolateral and the lateral medullary arteries that can arise from the VA (*d*) or the ASA (*e*)

artery [2]. The musculospinal branch of the APhA penetrates the spinal canal at the third cervical space and also supplies the spinal part of the nerve (odontoid arch system) [9, 10, 67]. The cranial division of the accessory nerve receives a supply from the branches of the posteroinferior cerebellar artery (PICA) [2]. In the jugular foramen, the accessory nerve is supplied by the jugular branch of the APhA [9].

Hypoglossal Nerve

The cisternal part of the hypoglossal nerve is supplied by the anterolateral and the lateral medullary arteries [2]. These two branches have variable origin and could arise from the anterior spinal artery, the PICA, the VA, or the basilar artery [7]. The hypoglossal nerve is then supplied by the hypoglossal branch of the APhA [10, 67]. Extracranially, the nerve received blood supply from branches of the occipital artery, the facial artery, and the lingual artery [2, 7].

The cisternal supply of the glossopharyngeal, vagal, accessory, and hypoglossal nerves is shown in Fig. 5.

Vascular Syndrome Involving Cranial Nerves (Table 2)

Syndrome of the Inferolateral Trunk

The ILT of the cavernous ICA supplies all oculomotor nerves and a part of the trigeminal nerve [70]. Consequently, a thromboembolic event or an accidental embolization of the ILT results in a complete extraocular muscle paresis associated with a hypesthesia in the territory of the V1 [3, 16, 71–73].

Syndrome of the Middle Meningeal Artery

The MMA participates in the blood supply of cranial nerves mainly by its petrous branch that arises from the principal trunk of the MMA near the foramen spinosum. The syndrome of the MMA includes a paresis of the facial nerve associated with involvement of the third branch of the trigeminal nerve (V3) [11, 12, 15, 18, 55, 57, 62, 74, 75].

Syndrome of the Ascending Pharyngeal Artery

The ascending pharyngeal artery is the main arterial supply for the last four cranial nerves. Cranial nerves IX, X, and XI receive branches from the jugular branch of the APhA and

Table 2 Clinical vascular syndrome involving cranial nerves

Syndrome	Cranial nerves involved	Motor deficit	Sensitive deficit
Inferolateral trunk	Oculomotor Trochlear Abducens Trigeminal (V1)	Ophthalmoplegia	Periorbital pain (V1) ± maxillary (V2)
Middle meningeal artery	Facial Trigeminal (V2–V3)	Peripheric facial paresis	Maxillary (V2) and mandibular (V3) Auricular pain
Ascending pharyngeal artery	Glossopharyngeal Vagus Accessory Hypoglossal	Pharyngeal and laryngeal paresis Lingual paresis	Pharynx and larynx Sparing of XI function if isolated to the neuromeningeal trunk

hypoglossal nerve from the hypoglossal branch of the APhA [9]. Of these last cranial nerves, the accessory nerve is the only one to receive a dual vascularization; its spinal division is supplied by the musculospinal branch of the APhA [67]. This anatomical organization explained the different clinical situations described in the literature. In the case of proximal thrombosis or an embolic event of the neuromeningeal trunk, all the last four nerves are involved. In the case of jugular branch closure, the hypoglossal is spared as well as the accessory nerves, thanks to its dual vascularization [9, 14, 16, 17, 76].

Cranial nerve blood supply is a very specific but important topic. Numerous neurologic pathologies that involve the cranial nerves such as zona, diabetes, cervical ICA dissection, or idiopathic facial paresis could be explained by neural necrosis [14–17, 55, 57, 59, 62, 71, 72, 75, 77–80]. These last decades, the increasing amount of embolization in the terminal branches of the external carotid artery branches led to a better knowledge of cranial nerve arterial supply and of its cornerstone importance [9, 11, 12, 54, 55].

References

1. Abbie AA. The morphology of the fore-brain arteries, with especial reference to the evolution of the basal ganglia. J Anat. 1934;68(Pt 4):433–70.
2. Hendrix P, Griessenauer CJ, Foreman P, Loukas M, Fisher WS 3rd, Rizk E, et al. Arterial supply of the lower cranial nerves: A comprehensive review. Clin Anat. 2014;27(1):108–17. https://doi.org/10.1002/ca.22318.
3. Hendrix P, Griessenauer CJ, Foreman P, Shoja MM, Loukas M, Tubbs RS. Arterial supply of the upper cranial nerves: A comprehensive review. Clin Anat. 2014;27(8):1159–66. https://doi.org/10.1002/ca.22415.
4. Marinkovic S, Gibo H. The neurovascular relationships and the blood supply of the oculomotor nerve: The microsurgical anatomy of its cisternal segment. Surg Neurol. 1994;42(6):505–16. https://doi.org/10.1016/0090-3019(94)90081-7.
5. Marinkovic S, Gibo H, Zelic O, Nikodijevic I. The neurovascular relationships and the blood supply of the trochlear nerve: Surgical anatomy of its cisternal segment. Neurosurgery. 1996;38(1):161–9. https://doi.org/10.1097/00006123-199601000-00036.
6. Marinkovic SV, Gibo H. The blood supply of the trigeminal nerve root, with special reference to the trigeminocerebellar artery. Neurosurgery. 1995;37(2):309–17. https://doi.org/10.1227/00006123-199508000-00017.
7. Gibo H, Marinkovic S, Nikodijevic I, Stimec B, Erden A. The blood supply of the hypoglossal nerve: The microsurgical anatomy of its cisternal segment. Surg Neurol. 1997;48(1):85–91. https://doi.org/10.1016/s0090-3019(96)00475-2.
8. El-Khouly H, Fernandez-Miranda J, Rhoton AL Jr. Blood supply of the facial nerve in the middle fossa: The petrosal artery. Neurosurgery. 2008;62(5 Suppl 2):ONS297-303:discussion ONS-4. https://doi.org/10.1227/01.neu.0000326010.53821.a3.
9. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical Neuroangiography. Berlin: Springer edition; 2001.
10. Lasjaunias P, Doyon D. The ascending pharyngeal artery and the blood supply of the lower cranial nerves. J Neuroradiol. 1978;5(4):287–301.
11. Lasjaunias P, Doyon D, Edouard A. Post-embolisation lower motor neurone facial paralysis. Report of one case, discussion, prevention (author's transl). Ann Otolaryngol Chir Cervicofac. 1978;95(9):595–602.
12. Lasjaunias P, Moret J, Doyon D, Vignaud J. Arteriographic exploration of the intrapetrous facial nerve: Embryology and normal radiologic anatomy (author's transl). Neuroradiology. 1978;16:246–8. https://doi.org/10.1007/BF00395262.
13. Ozanne A, Pereira V, Krings T, Toulgoat F, Lasjaunias P. Arterial vascularization of the cranial nerves. Neuroimaging Clin N Am. 2008;18(2):431–9., xii. https://doi.org/10.1016/j.nic.2007.12.010.
14. Lapresle J, Faux N. Unilateral involvement of IX, X, Xi and XII in cervical zoster. Cranial nerve contribution to vascular pathology. J Neurol Sci. 1981;52(2–3):351–7. https://doi.org/10.1016/0022-510x(81)90016-2.
15. Lapresle J, Fernandez Manchola I, Lasjaunias P. Trigeminal sensory involvement in Bell's palsy (author's transl). Nouv Press Med. 1980;9(5):291–3.
16. Lapresle J, Lasjaunias P. Cranial nerve ischaemic arterial syndromes. A review. Brain. 1986;109(Pt 1):207–16. https://doi.org/10.1093/brain/109.1.207.
17. Lapresle J, Lasjaunias P, Thevenier D. Transitory paralysis of cranial nerves IX, X and XII as well as the left VII after angiography. Contribution to the ischemic pathology of the cranial nerves. Rev Neurol (Paris). 1980;136(11):787–91.
18. Geibprasert S, Pongpech S, Armstrong D, Krings T. Dangerous extracranial-intracranial anastomoses and supply to the cranial nerves: Vessels the neurointerventionalist needs to know. AJNR Am J Neuroradiol. 2009;30(8):1459–68. https://doi.org/10.3174/ajnr.A1500.
19. Rinaldo L, Brinjikji W. Dangerous Extracranial-intracranial anastomoses: What the Interventionalist must know. Semin Interv Radiol. 2020;37(2):140–9. https://doi.org/10.1055/s-0040-1709155.

20. Padget DH. The development of cranial arteries in the human embryo, Contribution to Embryology, vol. 212. Washington: Carnegie Institution; 1948. p. 205–62.
21. Bonasia S, Smajda S, Ciccio G, Bojanowski MW, Robert T. Embryology of the anterior communicating artery complex: Implications on possible adult variants. Surg Radiol Anat. 2022;44(5):737–48. https://doi.org/10.1007/s00276-022-02928-5.
22. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: A pictorial review. Surg Radiol Anat. 2021;43(6):961–73. https://doi.org/10.1007/s00276-021-02684-y.
23. Favre JJ, Chaffanjon P, Passagia JG, Chirossel JP. Blood supply of the olfactory nerve. Meningeal relationships and surgical relevance. Surg Radiol Anat. 1995;17(2):133–8., 12–14. https://doi.org/10.1007/BF01627573.
24. Eriksen KD, Boge-Rasmussen T, Kruse-Larsen C. Anosmia following operation for cerebral aneurysms in the anterior circulation. J Neurosurg. 1990;72(6):864–5. https://doi.org/10.3171/jns.1990.72.6.0864.
25. Aydin IH, Kadioglu HH, Tuzun Y, Kayaoglu CR, Takci E, Ozturk M. Postoperative anosmia after anterior communicating artery aneurysms surgery by the pterional approach. Minim Invasive Neurosurg. 1996;39(3):71–3. https://doi.org/10.1055/s-2008-1052220.
26. Wermer MJ, Donswijk M, Greebe P, Verweij BH, Rinkel GJ. Anosmia after aneurysmal subarachnoid hemorrhage. Neurosurgery. 2007;61(5):918–22.; discussion 22–23. https://doi.org/10.1227/01.neu.0000303187.34308.7b.
27. Moman MR, Verweij BH, Buwalda J, Rinkel GJ. Anosmia after endovascular and open surgical treatment of intracranial aneurysms. J Neurosurg. 2009;110(3):482–6. https://doi.org/10.3171/2008.8.JNS08761.
28. Blunt MJ, Steele EJ. The blood supply of the optic nerve and chiasma in man. J Anat. 1956;90(4):486–93.
29. Blunt MJ. Implications of the vascular anatomy of the optic nerve and chiasma. Proc R Soc Med. 1956;49(7):433–9.
30. Dawson BH. The blood vessels of the human optic chiasma and their relation to those of the hypophysis and hypothalamus. Brain. 1958;81(2):207–17. https://doi.org/10.1093/brain/81.2.207.
31. Baykal D, Yilmazlar S, Fedakar R. A neurosurgical assessment of the blood supply in the optochiasmatic system: A cadaveric-anatomic study. Anat Sci Int. 2021;96(2):294–300. https://doi.org/10.1007/s12565-020-00595-3.
32. Francois J, Neetens A, Collette JM. Vascular supply of the optic pathway. II. Further studies by micro-arteriography of the optic nerve. Br J Ophthalmol. 1955;39(4):220–32. https://doi.org/10.1136/bjo.39.4.220.
33. Bonasia S, Bojanowski M, Robert T. Embryology and anatomical variations of the ophthalmic artery. Neuroradiology. 2020;62(2):139–52. https://doi.org/10.1007/s00234-019-02336-4.
34. Hayreh SS. The central artery of the retina. Its role in the blood supply of the optic nerve. Br J Ophthalmol. 1963;47:651–63. https://doi.org/10.1136/bjo.47.11.651.
35. Francois J, Fryczkowski A. The blood supply of the optic nerve. Adv Ophthalmol. 1978;36:164–73.
36. Hayreh SS. Blood supply of the optic nerve head. Ophthalmologica. 1996;210(5):285–95. https://doi.org/10.1159/000310727.
37. Hayreh SS. The blood supply of the optic nerve head and the evaluation of it – Myth and reality. Prog Retin Eye Res. 2001;20(5):563–93. https://doi.org/10.1016/s1350-9462(01)00004-0.
38. Hughes B. Blood supply of the optic nerves and chiasma and its clinical significance. Br J Ophthalmol. 1958;42(2):106–25. https://doi.org/10.1136/bjo.42.2.106.
39. Lasjaunias P, Moret J, Doyon D, Vignaud J. C5 collaterals of the internal carotid siphon: Embryology, angiographic anatomical correlations, pathological radio-anatomy (author's transl). Neuroradiology. 1978;16:304–5. https://doi.org/10.1007/BF00395282.
40. Lasjaunias P, Moret J, Mink J. The anatomy of the inferolateral trunk (ILT) of the internal carotid artery. Neuroradiology. 1977;13(4):215–20. https://doi.org/10.1007/BF00344216.
41. Willinsky R, Lasjaunias P, Berenstein A. Intracavernous branches of the internal carotid artery (ICA). Comprehensive review of their variations. Surg Radiol Anat. 1987;9(3):201–15. https://doi.org/10.1007/BF02109631.
42. Milisavljevic M, Marinkovic S, Lolic-Draganic V, Kovacevic M. Oculomotor, trochlear, and abducens nerves penetrated by cerebral vessels. Microanatomy and possible clinical significance. Arch Neurol. 1986;43(1):58–61. https://doi.org/10.1001/archneur.1986.00520010054022.
43. Cahill M, Bannigan J, Eustace P. Anatomy of the extraneural blood supply to the intracranial oculomotor nerve. Br J Ophthalmol. 1996;80(2):177–81. https://doi.org/10.1136/bjo.80.2.177.
44. Krisht A, Barnett DW, Barrow DL, Bonner G. The blood supply of the intracavernous cranial nerves: An anatomic study. Neurosurgery. 1994;34(2):275–9.;discussion 9. https://doi.org/10.1227/00006123-199402000-00011.
45. Najera E, Ibrahim B, Muhsen BA, Ali A, Sanchez C, Obrzut M, et al. Blood supply of cranial nerves passing through the cavernous sinus: An anatomical study and its implications for microsurgical and endoscopic cavernous sinus surgery. Front Oncol. 2021;11:702574. https://doi.org/10.3389/fonc.2021.702574.
46. Girard PF, Bonamour G, Durand DG. Paralysis of the trochlear nerve in the pons varolii syndrome of the superior cerebellar artery. Sem Hop. 1952;28(55–56):2311–7.
47. d'Avella E, Tschabitscher M, Santoro A, Delfini R. Blood supply to the intracavernous cranial nerves: Comparison of the endoscopic and microsurgical perspectives. Neurosurgery. 2008;62(5 Suppl 2):ONS305-10:discussion ONS10-1. https://doi.org/10.1227/01.neu.0000326011.53821.ea.
48. Sunderland S. Neurovascular relations and anomalies at the base of the brain. J Neurol Neurosurg Psychiatry. 1948;11(4):243–57. https://doi.org/10.1136/jnnp.11.4.243.
49. Cetkovic M, Antunovic V, Marinkovic S, Todorovic V, Vitosevic Z, Milisavljevic M. Vasculature and neurovascular relationships of the trigeminal nerve root. Acta Neurochir. 2011;153(5):1051–7.; discussion 7. https://doi.org/10.1007/s00701-010-0913-1.
50. Lasjaunias P, Theron J. Radiographic anatomy of the accessory meningeal artery. Radiology. 1976;121(1):99–104. https://doi.org/10.1148/121.1.99.
51. Tubbs RS, Hansasuta A, Loukas M, Louis RG Jr, Shoja MM, Salter EG, et al. Branches of the petrous and cavernous segments of the internal carotid artery. Clin Anat. 2007;20(6):596–601. https://doi.org/10.1002/ca.20434.
52. Bonasia S, Smajda S, Ciccio G, Robert T. Anatomic and embryologic analysis of the dural branches of the ophthalmic artery. AJNR Am J Neuroradiol. 2021;42(3):414–21. https://doi.org/10.3174/ajnr.A6939.
53. Nuza AB, Taner D. Anatomical variations of the intracavernous branches of the internal carotid artery with reference to the relationship of the internal carotid artery and sixth cranial nerve. A microsurgical study. Acta Anat (Basel). 1990;138(3):238–45. https://doi.org/10.1159/000146946.
54. Moret J, Delvert JC, Bretonneau CH, Lasjaunias P, de Bicetre CH. Vascularization of the ear: Normal-variations-glomus tumors. J Neuroradiol. 1982;9(3):209–60.
55. Moret J, Lasjaunias P, Doyon D, Vignaud J. Angiographic study of facial paralyses (author's transl). Neuroradiology. 1978;16:308–10. https://doi.org/10.1007/BF00395284.
56. Guerrier Y. Arteries supplying the facial nerve. Montp Med. 1951;39–40(4):366–78.
57. Balkany T, Fradis M, Jafek BW, Rucker NC. Intrinsic vasculature of the labyrinthine segment of the facial

nerve – Implications for site of lesion in Bell's palsy. Otolaryngol Head Neck Surg. 1991;104(1):20–3. https://doi.org/10.1177/019459989110400105.
58. Sando I, Ogawa A, Jafek BW. Inner ear pathology following injury to the eighth cranial nerve and the labyrinthine artery. Ann Otol Rhinol Laryngol. 1982;91(2 Pt 1):136–41. https://doi.org/10.1177/000348948209100202.
59. Brunsteins DB, Ferreri AJ. Microsurgical anatomy of arteries related to the internal acoustic meatus. Acta Anat (Basel). 1995;152(2):143–50. https://doi.org/10.1159/000147693.
60. Ishibe K, Tamatsu Y, Miura M, Shimada K. Morphological study of the vasa nervorum in the peripheral branch of human facial nerve. Okajimas Folia Anat Jpn. 2011;88(3):111–9. https://doi.org/10.2535/ofaj.88.111.
61. Minatogawa T, Kumoi T, Hosomi H, Kokan T. The blood supply of the facial nerve in the human temporal bone. Auris Nasus Larynx. 1980;7(1):7–18. https://doi.org/10.1016/s0385-8146(80)80009-5.
62. Michelucci R, Forti A, de Maria R, Amore M, Tassinari CA. Trigeminal and facial nerve involvement resulting from ischaemia of the petrosal branch of the middle meningeal artery. J Neurol Neurosurg Psychiatry. 1983;46(5):460–1. https://doi.org/10.1136/jnnp.46.5.460-a.
63. Moreau S, Salame E, Delmas P. Arterial anatomy of the facial nerve at the stylo-mastoid foramen. Morphologie. 2001;85(269):19–22.
64. Chabot AB, Alimi Y, Iwanaga J, Kikuta S, Loukas M, Dumont AS, et al. Blood supply to the chorda tympani: A review and clinical applications. Ann Anat. 2020;232:151561. https://doi.org/10.1016/j.aanat.2020.151561.
65. Nager GT, Nager M. The arteries of the human middle ear, with particular regard to the blood supply of the auditory ossicles. Ann Otol Rhinol Laryngol. 1953;62(4):923–49. https://doi.org/10.1177/000348945306200401.
66. Hausler R, Levine RA. Auditory dysfunction in stroke. Acta Otolaryngol. 2000;120(6):689–703. https://doi.org/10.1080/000164800750000207.
67. Lasjaunias P, Moret J. The ascending pharyngeal artery: normal and pathological radioanatomy. Neuroradiology. 1976;11(2):77–82. https://doi.org/10.1007/BF00345017.
68. Hacein-Bey L, Daniels DL, Ulmer JL, Mark LP, Smith MM, Strottmann JM, et al. The ascending pharyngeal artery: Branches, anastomoses, and clinical significance. AJNR Am J Neuroradiol. 2002;23(7):1246–56.
69. Mohr PD. The blood supply of the vagus nerve. Acta Anat (Basel). 1969;73(1):19–26. https://doi.org/10.1159/000143278.
70. Blunt MJ. The blood supply of the facial nerve. J Anat. 1954;88(4):520–6.
71. Campos CR, Massaro AR, Scaff M. Isolated oculomotor nerve palsy in spontaneous internal carotid artery dissection: Case report. Arq Neuropsiquiatr. 2003;61(3A):668–70. https://doi.org/10.1590/s0004-282x2003000400027.
72. Teuscher AU, Meienberg O. Ischaemic oculomotor nerve palsy. Clinical features and vascular risk factors in 23 patients. J Neurol. 1985;232(3):144–9. https://doi.org/10.1007/BF00313889.
73. Capo H, Kupersmith MJ, Berenstein A, Choi IS, Diamond GA. The clinical importance of the inferolateral trunk of the internal carotid artery. Neurosurgery. 1991;28(5):733–7.; discussion 7–8. https://doi.org/10.1097/00006123-199105000-00018.
74. Bentson J, Rand R, Calcaterra T, Lasjaunias P. Unexpected complications following therapeutic embolization. Neuroradiology. 1978;16:420–3. https://doi.org/10.1007/BF00395321.
75. Calcaterra TC, Rand RW, Bentson JR. Ischemic paralysis of the facial nerve: A possible etiologic factor in Bell's palsy. Laryngoscope. 1976;86(1):92–7. https://doi.org/10.1288/00005537-197601000-00019.
76. Lasjaunias P, Theron J, Moret J. The occipital artery. Anatomy – Normal arteriographic aspects – Embryological significance. Neuroradiology. 1978;15(1):31–7. https://doi.org/10.1007/BF00327443.
77. Mokri B, Schievink WI, Olsen KD, Piepgras DG. Spontaneous dissection of the cervical internal carotid artery. Presentation with lower cranial nerve palsies. Arch Otolaryngol Head Neck Surg. 1992;118(4):431–5. https://doi.org/10.1001/archotol.1992.01880040097016.
78. Mokri B, Silbert PL, Schievink WI, Piepgras DG. Cranial nerve palsy in spontaneous dissection of the extracranial internal carotid artery. Neurology. 1996;46(2):356–9. https://doi.org/10.1212/wnl.46.2.356.
79. Schievink WI, Mokri B, Garrity JA, Nichols DA, Piepgras DG. Ocular motor nerve palsies in spontaneous dissections of the cervical internal carotid artery. Neurology. 1993;43(10):1938–41. https://doi.org/10.1212/wnl.43.10.1938.
80. Vargas ME, Desrouleaux JR, Kupersmith MJ. Ophthalmoplegia as a presenting manifestation of internal carotid artery dissection. J Clin Neuroophthalmol. 1992;12(4):268–71.

Part IV

Arterial Systems of the Skull Base

Arterial Supply of the Middle Ear

Sara Bonasia and Thomas Robert

The middle ear, located into the temporal bone, has one of the most complex arterial supply that depends on numerous little branches of the external carotid artery (ECA). This arterial supply is not well-studied because of the non-opacification of these branches on digital subtraction angiography (DSA) under physiological conditions. Even if the detailed anatomy of these little arteries is not well known, there are fascinating anatomical variations involving these tympanic branches [1]. The knowledge of these anatomical variations represents a keystone to avoid dramatic complications for neurosurgeons, neuroradiologists and mainly otologists. In this chapter, the anatomy of each tympanic artery with the related anatomical variations will be developed. Vascular supply of the external ear and of the internal ear will be explained in other chapters.

History

Only few anatomists or otologists published articles about the normal arterial supply of the middle ear. The most complete description of this vascularization was proposed by Nager and Nager (1953) who published a masterpiece work with very detailed description of these little arteries based on the dissection of 100 prepared temporal bones [2]. The other author that gave a particular attention to the arterial supply of the middle ear describing the normal anatomy, the anatomical variations and the vascularization in case of highly vascularized tumors is J. Moret (1982). In his article, he proposed a clear description of these arteries based on his huge angiographic experience [1].

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Each anatomical variation involving middle ear arteries was well described by P. Lasjaunias in numerous articles, as always based on its angiographic experience and on his phenomenal knowledge of the embryologic development of cranio-facial arteries [3–6].

Embryology

In the present chapter, you will find only information to understand the formation of each artery that participates in the supply of the middle ear and to understand different vascular variations that involve tympanic arteries. A reminder of the different embryological systems involved will be presented in the following paragraphs.

Aortic Arches

The embryological vascular development initiates early in the embryologic life [3]. The primitive heart is already recognizable at approximately 19 days (embryos of 2 mm) and paired ventral and dorsal aortas are formed and already have a cranial continuity, which is the first cranial arch [6, 7]. From 21 to 32 days (embryos of 3–12 mm), the other aortic arches are successively formed from front to back. At the same time, the dorsal aortas give a cranial extension to the first aortic arch. At the 28 days (embryos of 4–5 mm), the first aortic arch initiates to involute, followed by the second aortic arch. Consequently, the six pairs of aortic arches are not all present in the same time [6]. Each aortic arch has a different function, the two first one involute completely, the third arch persists and will become a part of the internal carotid artery (ICA), the fourth one persists and will give the adult aortic arch on the left and the brachiocephalic artery on the right. The fifth aortic arch will completely involute and the sixth one will become the pulmonary arteries [7, 8].

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Table 1 and Fig. 1 summarize the development and regression of different aortic arches and of the primitive ICA.

The first aortic arch, also known as mandibular artery, is a temporary artery that involutes very early in the embryological development. Its real function is not so clear. D. Padget (1948) evoked its contribution in the formation of the ophthalmic artery. Other authors, as P. Lasjaunias (2001) gave to the mandibular artery the role of precursor of the Vidian artery arising from the petrous ICA in the adult configuration [3, 6].

Table 1 Summary of the different aortic arches

Aortic arches	Stage of development	Stage of regression	Adult remnant
I (Mandibular artery)	Embryos of 2 mm	Stage I (4–5 mm)	Vidian artery
II (Hyo-stapedial artery)	Embryos of 2 mm	Stage VI (24 mm)	Carotico-tympanic artery Superior tympanic artery
III	Stage I (4–5 mm)	No regression	Proximal part of the ICA
IV	Stage I (4–5 mm)	No regression	Adult aortic arch
V	Stage II (5–7 mm)	Not known	Not known
VI	Stage II (5–7 mm)	No regression	Pulmonary arteries

Hyostapedial Artery

"Hyostapedial artery" is the term used to describe the complete embryological development of the second aortic arch. The stapedial artery (SA), that develops from the hyoid artery and takes its name after passing through the crus of the stapes, is an embryonic artery present between the stages III and VI of Padget (9–24 mm) [6, 7]. This is an important embryological system from which numerous dural, orbital and facial arteries develop. The steps of SA development are summarized in Table 2.

The hyoid artery is the dorsal remnant of the second aortic arch which regresses early in the embryological development (4–5 mm, stage I of Padget) [6]. After this

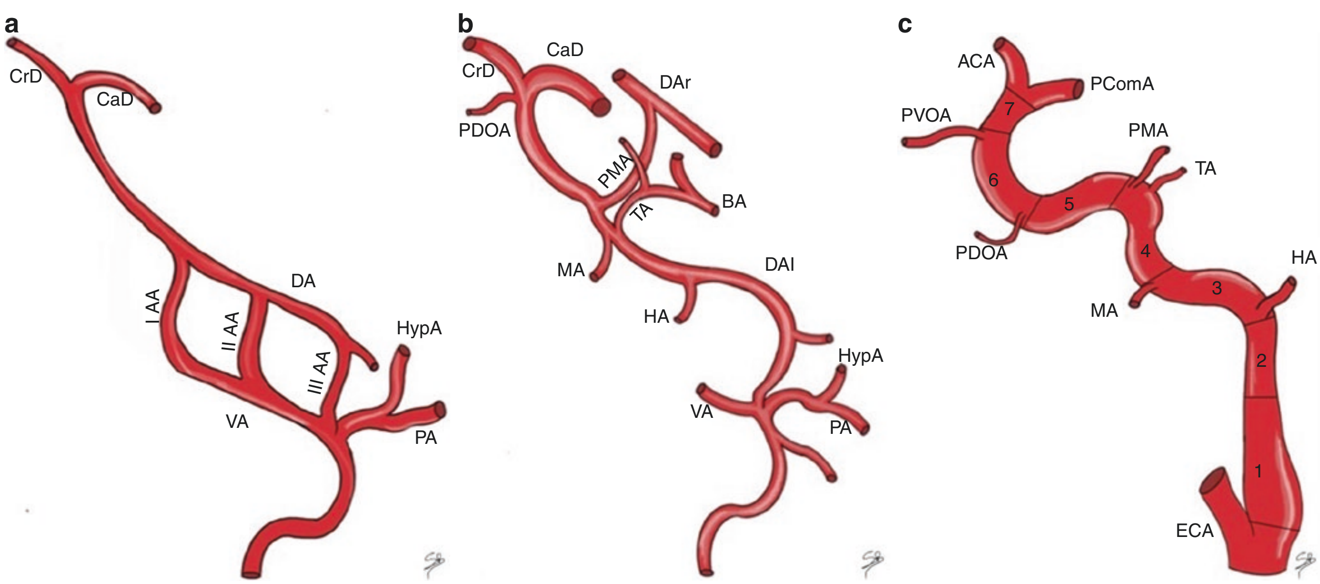
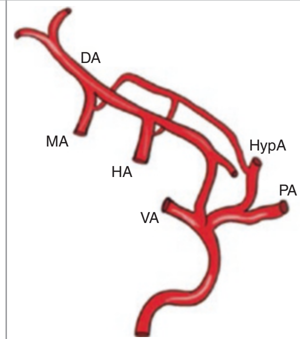
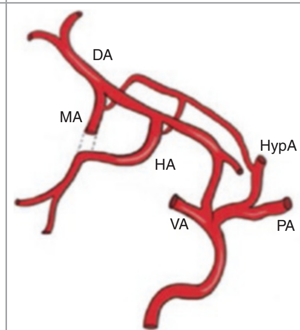
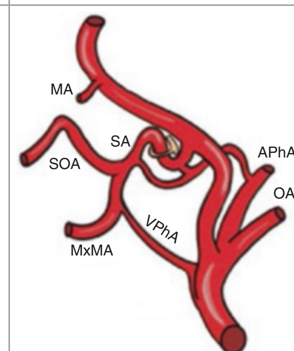
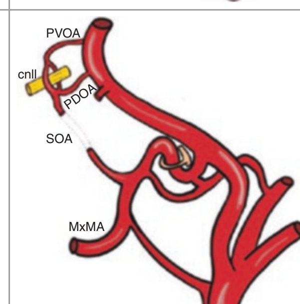
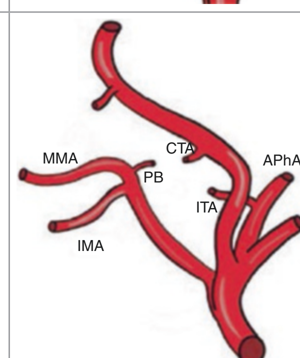


Fig. 1 Embryology and embryological segments of the Internal carotid artery. The illustrations (**a**–**c**) show consecutive stages of ICA embryological development. The first stages of development (**a**) are characterized by the presence of three aortic arches that link the ventral and dorsal aorta (VA, DA). The VA regresses together with the ventral part of the aortic arches. The dorsal remnants of the aortic arches persist as embryonic arteries. These embryonic arteries divide the ICA into seven embryological segments: (1) The cervical segment: it derives from the remnant of the third aortic arch (III AA). (2) The ascending intrapetrous segment: it is the remnant of the dorsal aorta (DA) between the second (II AA) and third aortic arches. The division point between the segment 2 and 3 is at the point of origin of the Hyoid artery (HA), that is the dorsal remnant of the second aortic arch. (3) The horizontal intrapetrous segment: it is the remnant of the DA between the first (I AA) and second aortic arches. The division point is at the point of origin of the mandibular artery (MA), that corresponds to the dorsal remnant of the first aortic arch. (4) The intracavernous ascending segment: it originates from the DA between the first aortic arch and the primitive maxillary artery (PMA), that connects the DA of the two sides (DAl: dorsal aorta left; DAr: dorsal aorta right). At the junction between segments 4 and 5 also the trigeminal artery (TA) takes its origin. The latter represents a primitive connection between the cavernous ICA and the basilar artery (BA). (5) The horizontal intracavernous segment: it derives from the DA between the PMA and the primitive dorsal ophthalmic artery (PDOA). (6) The clinoid segment: corresponds to the DA between the PDOA and the primitive ventral ophthalmic artery (PVOA). (7) The terminal segment: the terminal ICA between (PVOA) and the primitive ICA bifurcation into the future anterior cerebral artery (ACA) and future posterior communicating artery (PComA). The figure shows also the hypoglossal artery (HypA) and the proatlantal artery (PA), that origin proximal to the third aortic arch and will contribute to the formation of external carotid artery (ECA) branches

Table 2 Major embryological changes in the formation of the stapedial artery

Stage	Embryo size (mm)	Major evolutions	Graphic illustration
I	4–5	– Regression of the ventral part of the first and second aortic arches – Hyoid artery (HA) formation (dorsal remnant of the second aortic arch)	
II	9	– Elongation of the hyoid artery (HA) – Annexation of the mandibular artery (MA) territory (first aortic arch) by the hyoid artery (second aortic arch)	
III	16	– Cranial growing of the hyoid artery (stapedial artery) passing into the middle ear (crus of the stapes) – Maximal development of the stapedial artery – Extension of the two branches of the stapedial artery: Supraorbital (SOA) and Maxillomandibular (MxMA)	
IV	24	– Annexation of the supraorbital branch (SOA) by the ophthalmic artery (PDOA, PVOA) – Regression of the trans-osseous (superior orbital fissure) segment of the supraorbital branch	
V	24	– Annexation of the maxillomandibular branch by the ventral pharyngeal artery – Regression of the intratympanic segment of the stapedial artery	

arch regression, approximately at the 9 mm stage (stage III of Padget), the hyoid artery presents a rapid lateral elongation between the beginning of the seventh and nineteenth weeks of gestation, when it gives an anastomosis to the mandibular artery (remnant of the first aortic arch) [6, 7]. At the 16 mm stage (stage V of Padget), the SA continues its elongation to the gasserian region passing into the future tympanic cavity and particularly through the crus of the stapes reaching its maximal development and giving two distinct branches: the supraorbital and the maxillo-mandibular artery [3]. The supraorbital artery follows the ophthalmic root of the trigeminal nerve and allows the development of orbital branches (supraorbital, lacrimal, ethmoidal and frontal arteries) and also of the middle meningeal artery (MMA) [3]. The maxillomandibular artery gets out of the cranial cavity through the foramen spinosum and gives extracranially its two branches: infraorbital and mandibular arteries (future infraorbital and inferior alveolar arteries). After this maximal development of the SA, two annexations and two regressions occur to give the adult configuration of the MMA. Intracranially, the orbital branches are annexed by the primitive OA and the trans-sphenoidal segment of the supraorbital branch regresses leaving an anastomotic artery between the anterior branch of the MMA and the lacrimal artery (the sphenoidal artery) that penetrates the orbit through the superior orbital fissure (SOF). Extracranially, the ventral pharyngeal artery annexes the maxillomandibular artery of the SA forming the proximal stem of the MMA and becoming the ECA. Consequently, by flow reversal into the SA, its tympanic portion regresses and leaves as remnants the carotico-tympanic artery (from the ICA) and the superior tympanic artery (from the petrous branch of the MMA). These annexations and regressions concerning the SA happen during the stage VI of Padget (embryos of 24 mm) [3, 9].

Pharyngo-Occipital System

Between the first and third stages of Padget (embryos between 4 and 12 mm), the posterior circulation, which is not already formed, is represented by two longitudinal neural arteries (LNAs) which are connected with the primitive carotid artery through four anastomotic arteries from cranial to caudal: the trigeminal, otic, hypoglossal and proatlantal arteries (HypA and PA) [6, 10]. These carotido-vertebral anastomoses regress at the same time as the formation of the vertebral arteries by transverse fusion of the cervical inter-segmental arteries. The pharyngo-occipital is considered as the remnant of the hypoglossal (for the ascending pharyngeal artery) and of the proatlantal (for the occipital artery) arteries [3, 5, 11–13].

Normal Vascular Anatomy of the Middle Ear

Arteries that supply the tympanic cavity are principally branches of the external carotid artery and are numbered as five different branches. These arteries are summarized in Table 3 and are illustrated in Fig. 2.

Table 3 Principal tympanic arteries with their respective territory

Tympanic branches	Origin	Foramen	Territory	Anastomosis
Carotico-tympanic artery	ICA (petrous segment)	Petrous bone	Anterior wall Medial wall (anterior part)	Inferior tympanic artery
Superior tympanic artery	MMA (petrosal branch)	Canalicus tympanicus superior	Superior wall Epitympanic recess Tensor tympani muscle Staples	Anterior tympanic artery Stylomastoid artery
Inferior tympanic artery	Ascending pharyngeal artery	Jacobson (inferior tympanic) foramen	Inferior wall Medial wall (inferior part) Anterior wall (inferior part) Promontory Staples	Carotico-tympanic artery Stylomastoid artery MMA (petrosal branch)
Anterior tympanic artery	Internal maxillary artery (45–78%)	Petro-tympanic fissure	Anterior wall Medial wall (anterior part) Tegmen tympani Chorda tympani Malleus and incus	Stylomastoid artery
Stylomastoid artery	Posterior auricular artery	Stylomastoid foramen	Posterior wall Chorda tympani Facial nerve	Anterior tympanic artery MMA (petrosal branch) AICA (subarcuate branch)

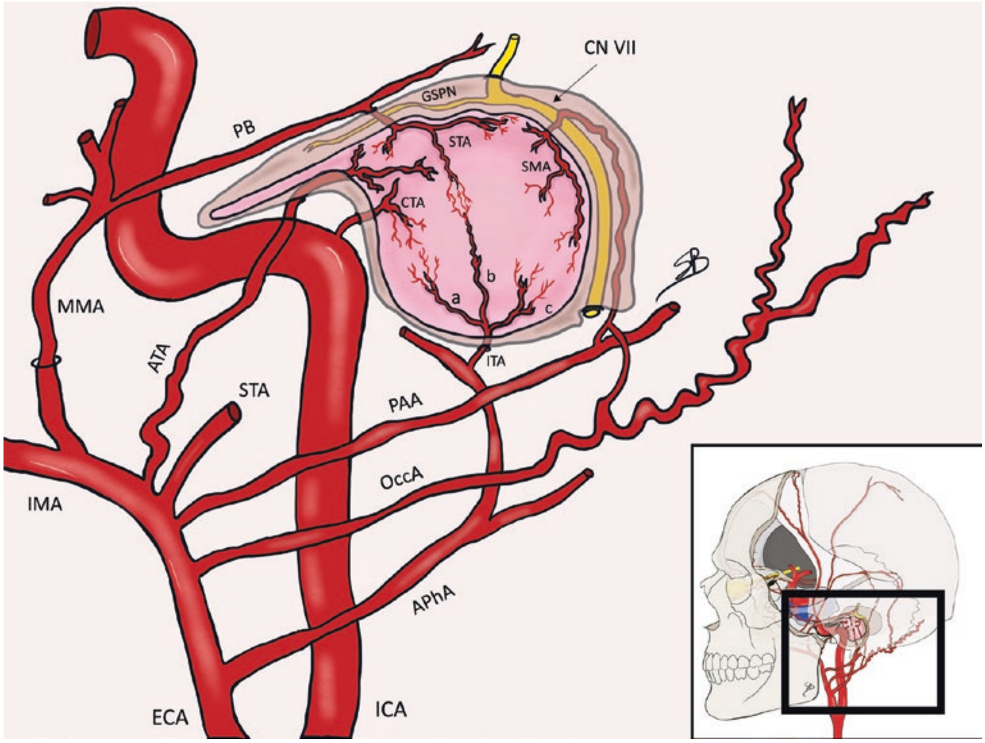


Fig. 2 Artist's illustration of the vascularization of the middle ear. The middle ear is supplied by the carotico-tympanic artery (CTA) which originates from the petrous internal carotid artery (ICA); by the anterior tympanic artery (ATA), that is the first branch of the internal maxillary artery (IMA) after its bifurcation from the superficial temporal artery (STA); by the superior tympanic artery (STA) that originates from the petrous branch (PB) of the middle meningeal artery (MMA) after coursing together with the greater superficial petrosal nerve (GSPN); by the inferior tympanic artery (ITA) which originates from the anterior division of the ascending pharyngeal artery (APhA) and enters the cavity through the Jacobson's canal; it divides into an anterior (**a**), middle (**b**) and posterior (**c**) stems which anastomoses with branches of the CTA, STA and SMA; by the stylomastoid artery (SMA) that can originate from the posterior auricular artery (PAA) or from the occipital artery (OccA) and enters the facial canal together with the facial nerve (CN VII) and contributes also to its vascularization

Carotico-Tympanic Artery

The carotico-tympanic artery arises from the vertical part of the petrous segment of the ICA and is considered as the proximal remnant of the stapedial artery [1, 3]. In almost all cases, this artery is unique but in 2% of cases, two distinct little arteries could be identified [2]. The carotico-tympanic artery penetrates into the tympanic cavity through a little osseous canal and supplies the anterior wall of the tympanic cavity and also the anterior part of its medial wall [14, 15]. Branches of the carotico-tympanic artery could have arterial anastomosis with the inferior tympanic artery [2, 16].

Superior Tympanic Artery

The superior tympanic artery is a little branch of the petrosal branch of the MMA. As the carotico-tympanic artery, it is considered as a remnant of the stapedial artery. The origin of this artery is near the foramen spinosum, it courses posteriorly along the greater superior petrosal nerve (GSPN) and then enters into the tympanic cavity through the canalicus tympanicus superior [2]. The vascular territory of the superior tympanic artery is the superior wall of the tympanic cavity, the medial wall of the epitympanic recess, the tensor tympani muscle and a part of the staples [1–3, 17]. The superior tympanic artery is known to have a rich anastomotic network with the anterior tympanic artery and the stylomastoid artery [2]. This last anastomosis between the superior tympanic artery and the stylomastoid artery is located into the facial canal where these two arteries participate in the blood supply of the facial nerve [1]. It constitutes the "facial arcade" on lateral angiograms that represents an anatomic landmark of the geniculate ganglion (petrous ridge) [3].

Inferior Tympanic Artery

The inferior tympanic artery is a branch of the ascending pharyngeal artery [1–3, 5, 18]. It enters into the tympanic cavity passing through the Jacobson canal (inferior tympanic canal) with the Jacobson nerve (branch of the glosso-pharyngeal nerve) [1–3]. The inferior tympanic artery has three different groups of branches. The anterior group follows the glossopharyngeal nerve and anastomoses with the carotico-tympanic artery. The medial group follows the greater and lesser superficial petrosal nerves and anastomoses with the petrosal branch of the MMA. The posterior group anastomoses with the stylomastoid artery [1, 3]. The vascular territory of the inferior tympanic artery is principally the inferior wall of the tympanic cavity, inferior part of its anterior and medial walls, the promontory and the staples [1, 2].

Anterior Tympanic Artery

The anterior tympanic artery is the first branch of the internal maxillary artery in 45–78% and could rarely have a double origin [19, 20]. In other cases, it arises from the superficial temporal artery (20–40%) or directly from the ECA (2–4%) [1, 19]. The anterior tympanic artery penetrates into the tympanic cavity through an osseous canal located into the temporo-mandibular joint (petro-tympanic fissure) [2, 19]. This artery supplies the anterior wall and the anterior part of the medial wall of the tympanic cavity, the superior wall of the tegmen tympani and also the malleus and the incus by its ossicular branch [1–3, 19]. The anterior tympanic artery is also the principal artery of the chorda tympani [20]. The anterior tympanic artery has anastomoses with branches of the stylomastoid artery [15, 19].

Stylomastoid Artery

The stylomastoid artery is the temporal branch of the posterior auricular artery that arises from the external carotid artery (50%) or from the occipital artery (50%) [1, 3]. After passing through the stylomastoid foramen, branches of the stylomastoid artery participate in the vascular supply of the lateral, posterior and inferior walls of the tympanic cavity [11, 15, 20]. It also supplies the chorda tympani by its posterior tympanic artery and the facial nerve [3]. It anastomoses with the subarcuate branch of the antero-inferior cerebellar artery into the internal ear and with the petrosal branch of the MMA (facial arcade) [1, 2].

Anatomical Vascular Variations of the Middle Ear

Complete Persistence of the Stapedial Artery

The complete persistence of the stapedial artery is a very rare variant, only two cases were published in a context of ICA aneurysm or PHACE syndrome [3, 9]. In these cases, the stapedial artery could be seen as in the embryo taking its origin from the petrous ICA, passing through the middle ear and giving its two branches: one intracranial that corresponds to the MMA and the other extracranial leaving the cranial cavity through the foramen spinosum. Consequently, the foramen spinosum is enlarged, the cochlear promontory is eroded and the internal maxillary artery arises from the stapedial artery instead of the external carotid artery. Such an anatomical variant could easily be explained by embryology and particularly by the absence of annexation of the maxillomandibular branch (of the stapedial artery) by the ventral pharyngeal artery. Consequently, in the absence of reversion of the arterial flow into the stapedial artery, its proximal (intratympanic) stem could not regress [3].

Partial Persistence of the Stapedial Artery

The partial persistence of the stapedial artery is more frequent and in this case, only the intracranial branch of the stapedial artery keeps its origin from the petrous ICA [4, 5, 7, 11, 21–26]. This anatomical variation is illustrated in Fig. 3. The foramen spinosum is absent (an orifice does not exist without its content) or reduced in size and the MMA arises from the stapedial artery instead of the IMA. This variant is explained by the regression of the proximal part of the maxillomandibular artery instead of the proximal part of the stapedial artery [3]. We must note that it is surprising that the complete persistence of the stapedial artery is even much rarer than its partial persistence. This could be the illustration of a lack of embryological knowledge of the SA or a misunderstanding, particularly in the annexation of the maxillomandibular branch by the ventral pharyngeal artery.

Intra-tympanic Flow of the ICA

Aberrant ICA or intra-tympanic flow of the ICA is a rare but well-known anatomical variation of the internal carotid artery. It was described in different circumstances. An aberrant ICA could remain asymptomatic or could be responsible for non-specific symptoms: pulsatile tinnitus, recurrent otitis media, conductive hearing loss, vertigo or otosclerosis [27–57]. In numerous cases, the aberrant ICA was misdiagnosed for a glomus tympanicum and bore the physician in uncontrollable bleeding loss during a middle ear surgery [1, 28, 33, 38, 51]. The intra-tympanic course of the ICA is explained by the agenesis of the two first segments of the primitive carotid artery. The cervical segment is in fact the ascending pharyngeal artery with a hypertrophied inferior tympanic artery (branch of the ascending pharyngeal artery) that maintains its anastomosis with the carotico-tympanic artery (branch of the ICA) into the tympanic cavity (Fig. 4) [3, 8]. The correct term of this aberrant flow of the ICA is actually tympano-carotico-tympanic variant [3, 18]. It bypasses the

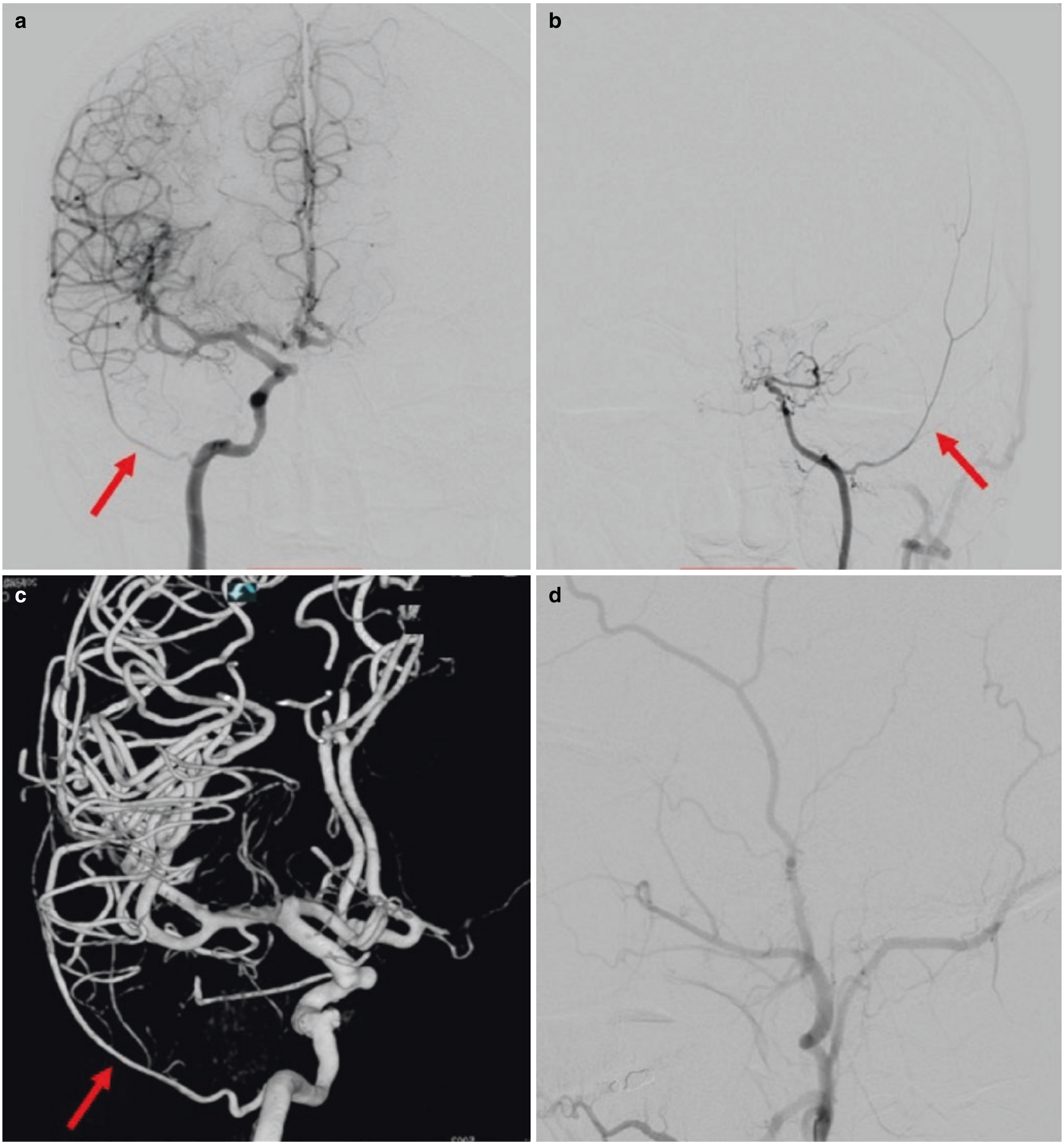


Fig. 3 DSA with bilateral partial persistent Stapedial Artery in patient with moyamoya disease. (**a**) and (**b**) Shows respectively right and left ICA injection with the MMA (red arrow) arising from the petrous ICA. This phenomenon is possible because of bilateral stapedial artery persistency. In (**b**) the ICA is narrowed and stopped at the level of the ophthalmic artery due to moyamoya disease. (**c**) Shows a 3D reconstruction of the right ICA from which the MMA originates (red arrow). (**d**) Shows the left ECA injection without the MMA enhancement, because of its absence

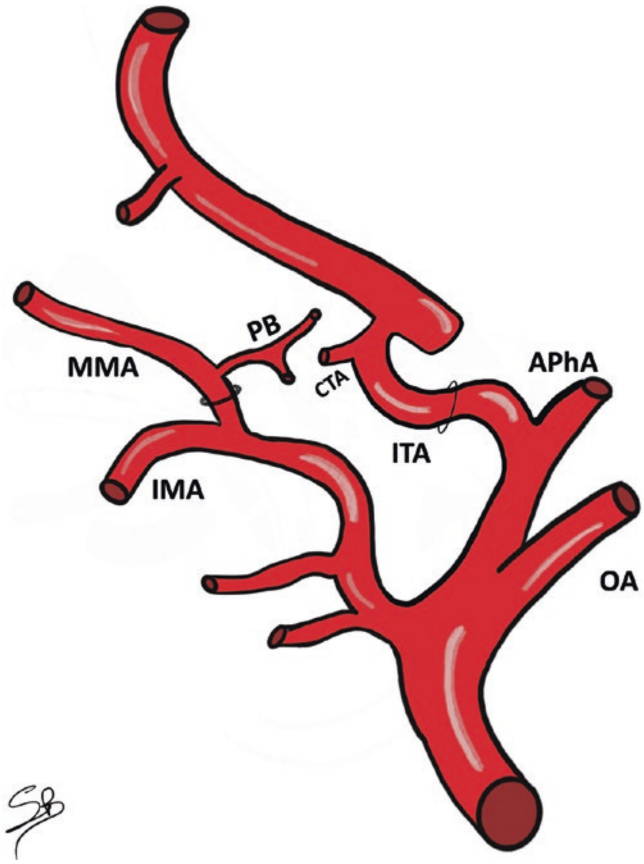


Fig. 4 Pseudo-petrous or aberrant intratympanic ICA. This variant is due to the agenesis of the cervical ICA (third aortic arch). The cervical ICA agenesis is bypassed through a hypertrophic inferior tympanic artery (ITA), that courses from the ascendant pharyngeal artery (APhA), through the inferior tympanic canal and reaches the carotico-tympanic artery (CTA) to supply the ICA distal flow. The intratympanic ICA flow is called "aberrant" since the artery does not course into its usual canal, but into an enlarged inferior tympanic canal

absence of the cervical ICA and provides a collateral circulation to the remainder of the ICA. Therefore, the cervical and intra-tympanic segments of this artery do not derivate from the carotid system but from the pharyngo-occipital and hyostapedial systems (pseudo-ICA) [3, 8].

Lapayowker (1971) defined the angiographic findings in case of aberrant ICA with in particular the lateral displacement of the petrous ICA, lateral to the "vestibular line" [58]. During the 1980s, few authors described the radiological criteria of aberrant ICA in CT scan and could be summarized as follows: (1) posterior displacement of the petrous ICA, (2) enlargement of the Jacobson's canal (inferior tympanic canal), (3) aplasia or hypoplasia of the vertical segment of the ICA into the petrous bone, (4) enhancing mass into the hypotympanum, (5) absence of the bone plate between the ICA and the middle ear and (6) absence of the extracranial orifice of the carotid canal (an orifice does not exist without its contents) [29, 52, 59, 60].

Intra-tympanic Flow of the ICA with Persistent Stapedial Artery

In rare cases, an intra-tympanic course of the ICA is associated with the persistence of the stapedial artery (Fig. 5) [17, 21, 22, 31, 46, 61]. This association of vascular variants concerning both carotid and hyostapedial systems was described anatomically and angiographically [5, 34, 38, 52, 62]. In these cases, the ICA enters the skull base through an enlarged inferior tympanic canal (narrowing of the vessel on DSA), passes into the tympanic cavity to bend anteriorly and reaches its normal carotid canal. The MMA arises from the ICA in its tympanic segment and passes through the stapes to enter the skull cavity through the cochlear promontory [3, 8].

Duplication of the ICA

The agenesis of the first two segments of the ICA could be complete or partial. In some cases, the agenesis is complete and the rerouting flow through the tympanic cavity allows maintaining a normal flow into the distal ICA. In rare cases, the agenesis of the cervical and petrous ICA is only partial

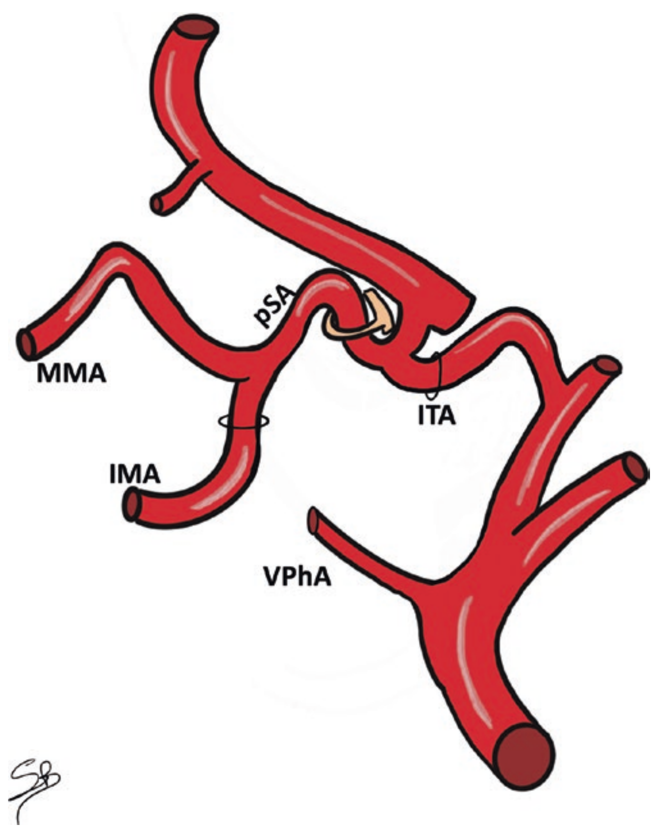


Fig. 5 Pseudo-petrous ICA + SA persistency. This variant is due to two phenomena: the agenesis of the cervical ICA, and stapedial artery persistency. In this case the inferior tympanic artery (ITA) does not reach the carotico-tympanic artery (CTA) but the persistent SA (pSA)

and consequently, the cervical and petrous ICA seem duplicated. The normal course of the ICA is generally hypoplastic in these cases [46, 63–69].

Pharyngo-Tympano-Stapedial Artery

This very rare variant was first described by Lasjaunias (1977) in its original publication [5]. The same case served as illustration in the textbook *Surgical Angiography* and only one similar case was published by Baltsavias et al. (2012) [3, 18]. The MMA arises from the cervical portion of the ICA, ascends along the cervical ICA, enters into the tympanic cavity through the inferior tympanic canal and follows the usual course of the stapedial artery. The two cases described were presented as "partial" persistence of the SA with only the MMA arising from the SA and the absence of the foramen spinosum. In this variant, an annexation of the SA by the inferior tympanic artery (branch of the ascending pharyngeal artery) with regression of the proximal part of the SA explains this vascular configuration. Therefore, the SA arises from the cervical instead of the petrous segment of the ICA.

Particular Variations

Almost all cases of aberrant flow of the ICA are sporadic and unilateral. Only three cases of bilateral aberrant flow of the ICA were presented in the English literature by Roll (2003), Chang (2010) and Toros (2010) [31, 36, 53].

Koenisberg et al. (1995) described a rare case of duplicated ICA associated with the persistence of the stapedial artery on the same side [46]. This is an interesting case that highlights the embryological link between the carotid and hyostapedial systems in the formation of the middle ear vascularization.

The most surprising case was presented by Willinsky et al. (1990) and consists of an anastomosis between the inferior tympanic artery (branch of the ascending pharyngeal artery) and the superior tympanic artery (stapedial artery) [60]. Consequently, the patient presented a duplicated ICA with anastomosis between the two ICAs in the horizontal petrous segment instead of the vertical petrous segment. Additionally, the middle meningeal artery arose from the ophthalmic artery instead of the external carotid artery. This case is similar to the two cases of a pharyngo-tympano-stapedial variant presented by P. Lasjaunias (1977) and by Baltsavias (2012) that presented a middle meningeal artery origin from the ascending pharyngeal artery [5, 18]. The difference between the case of Willinsky and these two cases is the presence or not of an aberrant flow of the ICA [60].

We could also find in the literature a few cases of aberrant course of the ICA associated with a cervical ICA origin of the occipital artery [70–73]. In fact, knowing that in case of aberrant course of the ICA, the cervical segment of the ICA does not exist; the occipital artery arises from the ascending pharyngeal system. This anatomical variation is easy to understand because the occipital and ascending pharyngeal arteries have the same embryological development [3].

Clinical Implications

A lot of cases of aberrant ICA were discovered during a surgery of the middle ear. The lack of knowledge of this anatomical variation leads in catastrophic hemorrhagic complication [74–76].

This anatomical variation must be differentiated from other diagnosis of vascular mass in the middle ear (high jugular bulb or glomus tympanicum) [49, 51, 77].

Almost all authors agree in the absence of surgical treatment of aberrant flow of the ICA [33, 44]. Only Rugg and Reed (1972) were favorable for an aggressive and surgical treatment in case of symptomatology [78]. They described a surgical technique coverage of the promontory defect by temporal muscle fascia graft and argue the indication of the surgery by "avoiding cerebrovascular accident." Today, it is accepted by almost all experts that intra-tympanic course of the ICA is an anatomical variation but not a pathology. Only few cases necessitate a surgical treatment of a pathology in relation to an aberrant ICA [44, 55]. Winfuhr (2004) proposed a management protocol and advocated to consider a surgical or endovascular treatment only in case of intra-tympanic aneurysmal formation, injury during a middle ear surgery or recurrent otitis media [33]. In case of pseudoaneurysm, the options are the trapping of the vessel with surgical bypass, the stenting of carotid artery or the endovascular occlusion of the vessel [33, 44, 55].

References

1. Moret J, Delvert JC, Bretonneau CH, Lasjaunias P, de Bicetre CH. Vascularization of the ear: normal-variations-glomus tumors. J Neuroradiol. 1982;9(3):209–60.
2. Nager GT, Nager M. The arteries of the human middle ear, with particular regard to the blood supply of the auditory ossicles. Ann Otol Rhinol Laryngol. 1953;62(4):923–49. https://doi.org/10.1177/000348945306200401.
3. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer; 2001.
4. Lasjaunias P, Moret J. Normal and non-pathological variations in the angiographic aspects of the arteries of the middle ear. Neuroradiology. 1978;15(4):213–9. https://doi.org/10.1007/bf00327529.
5. Lasjaunias P, Moret J, Manelfe C, Theron J, Hasso T, Seeger J. Arterial anomalies at the base of the skull. Neuroradiology. 1977;13(5):267–72. https://doi.org/10.1007/bf00347072.
6. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol. 1948;32:205–62.
7. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical signifi-

cance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
8. Lasjaunias P, Santoyo-Vazquez A. Segmental agenesis of the internal carotid artery: angiographic aspects with embryological discussion. Anat Clin. 1984;6(2):133–41. https://doi.org/10.1007/bf01773165.
9. Rodesch G, Choi IS, Lasjaunias P. Complete persistence of the hyoido-stapedial artery in man. Case report. Intra-petrous origin of the maxillary artery from ICA. Surg Radiol Anat. 1991;13(1):63–5.
10. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: a pictorial review. Surg Radiol Anat. 2021;43(6):961–73. https://doi.org/10.1007/s00276-021-02684-y.
11. Fisher AG. A case of complete absence of both internal carotid arteries, with a preliminary note on the developmental history of the stapedial artery. J Anat Physiol. 1913;48(Pt 1):37–46.
12. Lasjaunias P, Theron J, Moret J. The occipital artery. Anatomy–normal arteriographic aspects–embryological significance. Neuroradiology. 1978;15(1):31–7. https://doi.org/10.1007/BF00327443.
13. Lasjaunias P, Doyon D. The ascending pharyngeal artery and the blood supply of the lower cranial nerves. J Neuroradiol. 1978;5(4):287–301.
14. Andreo JC, Caldas Navarro JA, Toledo Filho JL, de Araujo JC. Caroticotympanic artery: anatomically a normal branch or a variation of the internal carotid artery? Cells Tissues Organs. 2000;166(3):319–22. https://doi.org/10.1159/000016745.
15. Alicandri-Ciufelli M, Guarino P, Fabbri F, Cunsolo EM, Presutti L, Anschuetz L. Middle ear microvascularization: an “in vivo” endoscopic anatomical study. Eur Arch Otorhinolaryngol. 2018;275(4):889–94. https://doi.org/10.1007/s00405-018-4905-8.
16. Salamon G, Guerinel G, Demard F. Radioanatomical study of the external carotid artery. Ann Radiol (Paris). 1968;11(3):199–215.
17. Silbergleit R, Quint DJ, Mehta BA, Patel SC, Metes JJ, Noujaim SE. The persistent stapedial artery. AJNR Am J Neuroradiol. 2000;21(3):572–7.
18. Baltsavias G, Kumar R, Valavanis A. The pharyngo-tympano-stapedial variant of the middle meningeal artery. A case report. Interv Neuroradiol. 2012;18(3):255–8. https://doi.org/10.1177/159101991201800302.
19. Merida Velasco JR, Rodriguez Vazquez JF, Jimenez CJ. Anterior tympanic artery: course, ramification and relationship with the temporomandibular joint. Acta Anat (Basel). 1997;158(3):222–6. https://doi.org/10.1159/000147933.
20. Chabot AB, Alimi Y, Iwanaga J, Kikuta S, Loukas M, Dumont AS, et al. Blood supply to the chorda tympani: a review and clinical applications. Ann Anat. 2020;232:151561. https://doi.org/10.1016/j.aanat.2020.151561.
21. Guinto FC Jr, Garrabrant EC, Radcliffe WB. Radiology of the persistent stapedial artery. Radiology. 1972;105(2):365–9. https://doi.org/10.1148/105.2.365.
22. Teal JS, Rumbaugh CL, Bergeron RT, Segall HD. Congenital absence of the internal carotid artery associated with cerebral hemiatrophy, absence of the external carotid artery, and persistence of the stapedial artery. Am J Roentgenol Radium Ther Nucl Med. 1973;118(3):534–45. https://doi.org/10.2214/ajr.118.3.534.
23. McLennan JE, Rosenbaum AE, Haughton VM. Internal carotid origins of the middle meningeal artery. The ophthalmic-middle meningeal and stapedial-middle meningeal arteries. Neuroradiology. 1974;7(5):265–75.
24. Sheikh BY, Coates R, Siqueira EB. Stapedial artery supplying sphenoid wing meningioma: case report. Neuroradiology. 1993;35(7):537–8.
25. Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A Jr. Microsurgical anatomy of the dural arteries. Neurosurgery. 2005;56(2 Suppl):211–51; discussion 51. https://doi.org/10.1227/01.neu.0000144823.94402.3d.
26. Kawai K, Yoshinaga K, Koizumi M, Honma S, Tokiyoshi A, Kodama K. A middle meningeal artery which arises from the internal carotid artery in which the first branchial artery participates. Ann Anat. 2006;188(1):33–8. https://doi.org/10.1016/j.aanat.2005.08.013.
27. Lo WW, Solti-Bohman LG, McElveen JT Jr. Aberrant carotid artery: radiologic diagnosis with emphasis on high-resolution computed tomography. Radiographics. 1985;5(6):985–93. https://doi.org/10.1148/radiographics.5.6.3880011.
28. Swartz JD, Bazarnic ML, Naidich TP, Lowry LD, Doan HT. Aberrant internal carotid artery lying within the middle ear. High resolution CT diagnosis and differential diagnosis. Neuroradiology. 1985;27(4):322–6. https://doi.org/10.1007/bf00339565.
29. McElveen JT Jr, Lo WW, el Gabri TH, Nigri P. Aberrant internal carotid artery: classic findings on computed tomography. Otolaryngol Head Neck Surg. 1986;94(5):616–21. https://doi.org/10.1177/019459988609400515.
30. Selesnick SH, Lessow AS. Aberrant internal carotid artery. Am J Otolaryngol. 1999;20(3):403–4.
31. Roll JD, Urban MA, Larson TC 3rd, Gailloud P, Jacob P, Harnsberger HR. Bilateral aberrant internal carotid arteries with bilateral persistent stapedial arteries and bilateral duplicated internal carotid arteries. AJNR Am J Neuroradiol. 2003;24(4):762–5.
32. Wei CJ, Chang FC, Chiou SY, Teng MM, Hu HH, Chung CP, et al. Aberrant ascending pharyngeal artery mimicking a partially occluded internal carotid artery. J Neuroimaging. 2004;14(1):67–70.
33. Windfuhr JP. Aberrant internal carotid artery in the middle ear. Ann Otol Rhinol Laryngol Suppl. 2004;192:1–16. https://doi.org/10.1177/00034894041130s301.
34. Saini J, Kesavadas C, Thomas B, Arvinda HR. Aberrant petrous internal carotid artery with cochlear anomaly-an unusual association. Surg Radiol Anat. 2008;30(5):453–7. https://doi.org/10.1007/s00276-008-0341-6.
35. Shimizu S, Sasahara G, Iida Y, Shibuya M, Numata T. Aberrant internal carotid artery in the middle ear with a deficiency in the origin of the anterior cerebral artery: a case report. Auris Nasus Larynx. 2009;36(3):359–62. https://doi.org/10.1016/j.anl.2008.08.008.
36. Chang CY. Bilateral aberrant internal carotid artery. Ear Nose Throat J. 2010;89(3):114–5.
37. Picel AC, Davidson TM. An aberrant internal carotid artery discovered during evaluation of obstructive sleep apnea: a report of 2 cases with consideration of a possible association. Ear Nose Throat J. 2011;90(1):29–31. https://doi.org/10.1177/014556131109000109.
38. Celebi I, Oz A, Yildirim H, Bankeroglu H, Basak M. A case of an aberrant internal carotid artery with a persistent stapedial artery: association of hypoplasia of the A1 segment of the anterior cerebral artery. Surg Radiol Anat. 2012;34(7):665–70. https://doi.org/10.1007/s00276-011-0912-9.
39. Nicolay S, De Foer B, Bernaerts A, Van Dinther J, Parizel PM. Aberrant internal carotid artery presenting as a retrotympanic vascular mass. Acta Radiol Short Rep. 2014;10:2047981614553695.
40. Yanmaz R, Okuyucu S, Burakgazi G, Bayarogullari H. Aberrant internal carotid artery in the tympanic cavity. J Craniofac Surg. 2016;27(8):2001–3. https://doi.org/10.1097/scs.0000000000003080.
41. Koizuka I, Hattori K, Tsutsumi K, Sakuma A, Katsumi N, Kikuchi H, et al. Objective tinnitus caused by an aberrant internal carotid artery. Auris Nasus Larynx. 1998;25(3):323–7. https://doi.org/10.1016/s0385-8146(98)00016-9.
42. Endo K, Maruyama Y, Tsukatani T, Furukawa M. Aberrant internal carotid artery as a cause of objective pulsatile tinnitus. Auris Nasus Larynx. 2006;33(4):447–50. https://doi.org/10.1016/j.anl.2006.03.007.
43. Park CW, Jeon EJ. Pulsatile tinnitus arising from aberrant internal carotid artery at nasopharynx. Korean J Audiol. 2012;16(1):35–8. https://doi.org/10.7874/kja.2012.16.1.35.
44. Song YS, Yuan YY, Wang GJ, Dai P, Han DY. Aberrant internal carotid artery causing objective pulsatile tinnitus and conductive hearing loss. Acta Otolaryngol. 2012;132(10):1126–30. https://doi.org/10.3109/00016489.2012.684400.

45. Honkura Y, Hidaka H, Ohta J, Gorai S, Katori Y, Kobayashi T. Surgical treatment for the aberrant internal carotid artery in the middle ear with pulsatile tinnitus. Auris Nasus Larynx. 2014;41(2):215–8. https://doi.org/10.1016/j.anl.2013.10.002.
46. Koenigsberg RA, Zito JL, Patel M, Swartz JD, Goldofsky E, Zahtz G. Fenestration of the internal carotid artery: a rare mass of the hypotympanum associated with persistence of the stapedial artery. AJNR Am J Neuroradiol. 1995;16(4 Suppl):908–10.
47. Botma M, Kell RA, Bhattacharya J, Crowther JA. Aberrant internal carotid artery in the middle-ear space. J Laryngol Otol. 2000;114(10):784–7. https://doi.org/10.1258/0022215001903924.
48. Cohen JE, Gomori JM, Leker RR. Internal carotid artery agenesis: diagnosis, clinical spectrum, associated conditions and its importance in the era of stroke interventions. Neurol Res. 2010;32(10):1027–32. https://doi.org/10.1179/016164110x12767786356273.
49. Lin YY, Wang CH, Liu SC, Chen HC. Aberrant internal carotid artery in the middle ear with dehiscent high jugular bulb. J Laryngol Otol. 2012;126(6):645–7. https://doi.org/10.1017/s0022215112000564.
50. Okawa M, Higashi T, Komiyama M, Fukuda K, Abe H, Inoue T. Left internal carotid artery agenesis with trans-sellar collateral and a right aortic arch: case report. Interv Neuroradiol. 2015;21(6):759–64. https://doi.org/10.1177/1591019915609130.
51. Anagiotos A, Kazantzi M, Tapis M. Aberrant internal carotid artery in the middle ear: the duplication variant. BMJ Case Rep. 2019;12(4):e228865. https://doi.org/10.1136/bcr-2018-228865.
52. Sauvaget E, Paris J, Kici S, Kania R, Guichard JP, Chapot R, et al. Aberrant internal carotid artery in the temporal bone: imaging findings and management. Arch Otolaryngol Head Neck Surg. 2006;132(1):86–91. https://doi.org/10.1001/archotol.132.1.86.
53. Toros SZ, Karaca CT, Noseri H, Naiboglu B, Kalaycik C, Egeli E. Bilateral aberrant internal carotid arteries: a case report presenting with pulsatile middle ear discharge. Int J Pediatr Otorhinolaryngol. 2010;74(1):97–8. https://doi.org/10.1016/j.ijporl.2009.10.015.
54. Stott CE, Kuroiwa MA, Carrasco FJ, Delano PH. Recurrent acute otitis media as the manifestation of an aberrant internal carotid artery. Otol Neurotol. 2013;34(8):e117–8. https://doi.org/10.1097/MAO.0b013e3182923eeb.
55. Kawamura Y, Sayama T, Maehara N, Nishimura A, Iihara K. Ruptured aneurysm of an aberrant internal carotid artery successfully treated with simultaneous intervention and surgery in a hybrid operating room. World Neurosurg. 2017;102:695.e1–5. https://doi.org/10.1016/j.wneu.2017.03.133.
56. Ottaviano G, Calzolari F, Martini A. Goldenhar syndrome in association with agenesia of the internal carotid artery. Int J Pediatr Otorhinolaryngol. 2007;71(3):509–12. https://doi.org/10.1016/j.ijporl.2006.11.003.
57. Weinberg A, Albers AE. Injury of an aberrant internal carotid artery after myringotomy. Pan Afr Med J. 2017;27:237. https://doi.org/10.11604/pamj.2017.27.237.9092.
58. Lapayowker MS, Liebman EP, Ronis ML, Safer JN. Presentation of the internal carotid artery as a tumor of the middle ear. Radiology. 1971;98(2):293–7. https://doi.org/10.1148/98.2.293.
59. Shankar L, Mehta AL, Hawke M, Rutka J. High-resolution computed tomography of an aberrant internal carotid artery. J Otolaryngol. 1992;21(5):373–5.
60. Willinsky RA, Ter Brugge K, Lawson V, Mirich D, Lasjaunias P. Aberrant carotid artery: recognition on high resolution computed tomography. J Otolaryngol. 1990;19(4):249–51.
61. Lau CC, Oghalai JS, Jackler RK. Combination of aberrant internal carotid artery and persistent stapedial artery. Otol Neurotol. 2004;25(5):850–1. https://doi.org/10.1097/00129492-200409000-00033.
62. Hatipoglu HG, Cetin MA, Yuksel E, Dere H. A case of a coexisting aberrant internal carotid artery and persistent stapedial artery: the role of MR angiography in the diagnosis. Ear Nose Throat J. 2011;90(5):E17–20. https://doi.org/10.1177/014556131109000513.
63. Hasegawa T, Kashihara K, Ito H, Yamamoto S. Fenestration of the internal carotid artery. Surg Neurol. 1985;23(4):391–5. https://doi.org/10.1016/0090-3019(85)90214-9.
64. Chess MA, Barsotti JB, Chang JK, Ketonen LM, Westesson PL. Duplication of the extracranial internal carotid artery. AJNR Am J Neuroradiol. 1995;16(7):1545–7.
65. Fujimura M, Seki H, Sugawara T, Tomichi N, Oku T, Higuchi H. Anomalous internal carotid artery-anterior cerebral artery anastomosis associated with fenestration and cerebral aneurysm. Neurol Med Chir (Tokyo). 1996;36(4):229–33. https://doi.org/10.2176/nmc.36.229.
66. Mangla S, Teitelbaum GP. Multichannel fenestrations of the petrous segment of the internal carotid artery. AJNR Am J Neuroradiol. 1999;20(4):590–2.
67. Yim NY, Ha HI, Park JH, Moon YJ, Yoon W, Kim JK. Agenesis of bilateral internal carotid artery associated with basilar artery fenestration mimicking intra-arterial thrombus: a case report. Vasc Endovasc Surg. 2010;44(1):69–74. https://doi.org/10.1177/1538574409347393.
68. Hacein-Bey L, Raghavan N, Mukundan G, Sekhon AK, Dodrill LK, Park TC. Fenestration of the petrous internal carotid artery with short segment duplication mimicking an arterial dissection. Clin Radiol. 2013;68(9):972–5. https://doi.org/10.1016/j.crad.2013.04.015.
69. Uchino A, Ehara T, Kurita H. Hypoplasia of the internal carotid artery with associated fenestration and extremely long P1 segment of the ipsilateral posterior cerebral artery diagnosed by MR angiography. Surg Radiol Anat. 2019;41(6):707–11. https://doi.org/10.1007/s00276-019-02212-z.
70. Hachem K, Slaba S, Nassar J, Kanso H, Ashoush R, Ghossain M. Imaging of an aberrant occipital artery arising from the extracranial segment of the internal carotid artery. J Mal Vasc. 2004;29(4):205–9. https://doi.org/10.1016/s0398-0499(04)96749-2.
71. Aggarwal NR, Krishnamoorthy T, Devasia B, Menon G, Chandrasekhar K. Variant origin of superior thyroid artery, occipital artery and ascending pharyngeal artery from a common trunk from the cervical segment of internal carotid artery. Surg Radiol Anat. 2006;28(6):650–3. https://doi.org/10.1007/s00276-006-0145-5.
72. Sundick SA, Weaver M, Faries PL, Marin M. Aberrant origin of occipital artery proximal to internal carotid artery stenosis. J Vasc Surg. 2014;59(1):244. https://doi.org/10.1016/j.jvs.2013.06.089.
73. Uchino A, Saito N, Okano N, Kakehi Y. Aberrant internal carotid artery associated with occipital artery arising from the internal carotid artery. Surg Radiol Anat. 2015;37(9):1137–40. https://doi.org/10.1007/s00276-015-1451-6.
74. Ide M, Jimbo M, Yamamoto M, Hagiwara S. Posterior cerebral artery aneurysm associated with unilateral internal carotid artery agenesis–case report. Neurol Med Chir (Tokyo). 1995;35(11):825–9. https://doi.org/10.2176/nmc.35.825.
75. Prokopakis EP, Bourolias CA, Bizaki AJ, Karampekios SK, Velegrakis GA, Bizakis JG. Ectopic internal carotid artery presenting as an oropharyngeal mass. Head Face Med. 2008;4:20. https://doi.org/10.1186/1746-160x-4-20.
76. Muderris T, Bercin S, Sevil E, Cetin H, Kiris M. A potentially catastrophic anatomical variation: aberrant internal carotid artery in the middle ear cavity. Case Rep Otolaryngol. 2013;2013:743021. https://doi.org/10.1155/2013/743021.
77. Blazic S, Milojevic M, Grgurevic U, Djeric D. Anatomical variations and relations of large blood vessels to the tympanic cavity. Surg Radiol Anat. 2015;37(4):327–31. https://doi.org/10.1007/s00276-014-1370-y.
78. Ruggles RL, Reed RC. Treatment of aberrant carotid arteries in the middle ear: a report of two cases. Laryngoscope. 1972;82(7):1199–205. https://doi.org/10.1288/00005537-197207000-00008.

Embryological Development of the Hyostapedial System

Sara Bonasia, Michel W. Bojanowski, and Thomas Robert

The stapedial artery (SA) is an embryological artery that allows the development of orbital and dural arteries as well as maxillary branches. Its complex embryological development explains numerous anatomical variations of the middle meningeal artery (MMA) and orbital arteries. A few anatomists dissected a human middle ear that bore a persistent SA describing the origin and course of this artery [1–7]. ENT surgeons reported cases discovered during middle ear surgeries explaining the technical difficulties of the surgery in the presence of a persistent SA [8–20]. More recently, neuroradiologists paid particular attention to this anatomical variant [21–26]. These studies, associated with the comparative anatomy, allow to understand the persistence of the SA. In this chapter, after an embryological refresh concerning hyostapedial, carotid and ophthalmic systems, the anatomical variations including the SA will be explained [27–29].

History

The first cadaveric case of persistent SA was described by Hyrtl in 1836, who called attention to an artery running across the obturator of the stapes in a human cadaver, with some similarities with a vessel found in hibernating animals [30]. In the first half of the twentieth century, the phenomenal publication of Dorcas Padget, based on the dissections of 22 human embryos of the Carnegie collection, gave lot of information about the embryological development of the cranio-facial arteries and in particular of the hyostapedial system [31]. In the same period, Altmann (1947) furnished a comprehensive explanation of the development of the aortic arches and of the carotid system variants [32]. However, only in the 1960s we find the first angiographic demonstration of MMA variations and in particular, the persistence of a SA [33, 34]. At the end of the 1970s, P. Lasjaunias was able to give a comprehensive explanation of all variations implicating the SA. Its more famous articles were published between 1975 and 1990 and are summarized in its textbook [23–25, 35–39]. Diamond in the 1990s also published some articles based on the comparative anatomy principally with the great apes that increased our understanding of the stapedial system [5, 27, 28]. In this last three decades, few case reports of interesting anatomical variations implicating the SA were published [17, 40–47].

Comparative Anatomy

The studies of comparative anatomy were fundamental in the understanding of the embryological development of the SA. At the beginning of the twentieth century, Tandler, studying the SA development in rats, explained the SA regression and the role of the external carotid artery (ECA) in the formation of the MMA [31]. Rats differed from human embryos, because the supraorbital branch of the SA is primarily dependent on the first aortic arch (mandibular branch). Fuchs (1905), in the rabbit, also showed that the first two first aortic arches participate in the formation of the ECA as in human embryos [20]. More recently, Diamond (1991) and Falk (1993) compared the development of the SA between human and great apes' embryos and highlighted some analogies [28, 29].

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Embryology

The development and regression of the SA is strictly related to some embryonic systems. In this part of the chapter, we will summarize the main steps of the internal carotid artery (ICA) and ophthalmic artery (OA) development. We will also present a detailed description of the hyostapedial system, in order to understand the different possible adult variations related to this artery.

The Carotid System

Since the detailed description of the ICA embryological development has been widely treated in the chapter "Embryological Development of the Internal Carotid Artery," we will focus only on the understanding of the "aberrant flow of the ICA." It consists in an intratympanic course of the ICA and could be associated with a persistent SA and consequent variation in origin of the MMA. The principal steps of ICA development are resumed in Fig. 1.

In the first stage of Padget (4–5 mm embryos), the two first aortic arches initiate their natural regression allowing the ICA to be individualized. The embryological segments of the ICA are derived from the third aortic arch and from the dorsal aorta cranial to the third aortic arch [31, 32, 39]. The dorsal aorta also regresses at the same time between the third and fourth aortic arches. Embryologically, the ICA has been divided by Lasjaunias et al. in seven different segments [48]. The first one corresponds to the third aortic arch from the origin of the ventral pharyngeal artery (future ECA) to the junction between the third aortic arch and the dorsal aorta. The second segment is the dorsal aorta between the third and second aortic arches. The third segment is the dorsal aorta between the second and first aortic arches. The fourth segment is the dorsal aorta between the first aortic arch and the origin of the trigeminal artery (TA) (and the primitive maxillary artery (PMA)). The fifth segment is the dorsal aorta between the origin of the TA (and the primitive maxillary artery) and the origin of the primitive dorsal ophthalmic artery (PDOA, future inferolateral trunk (ILT)). The sixth segment is between the origin of the PDOA and the origin of

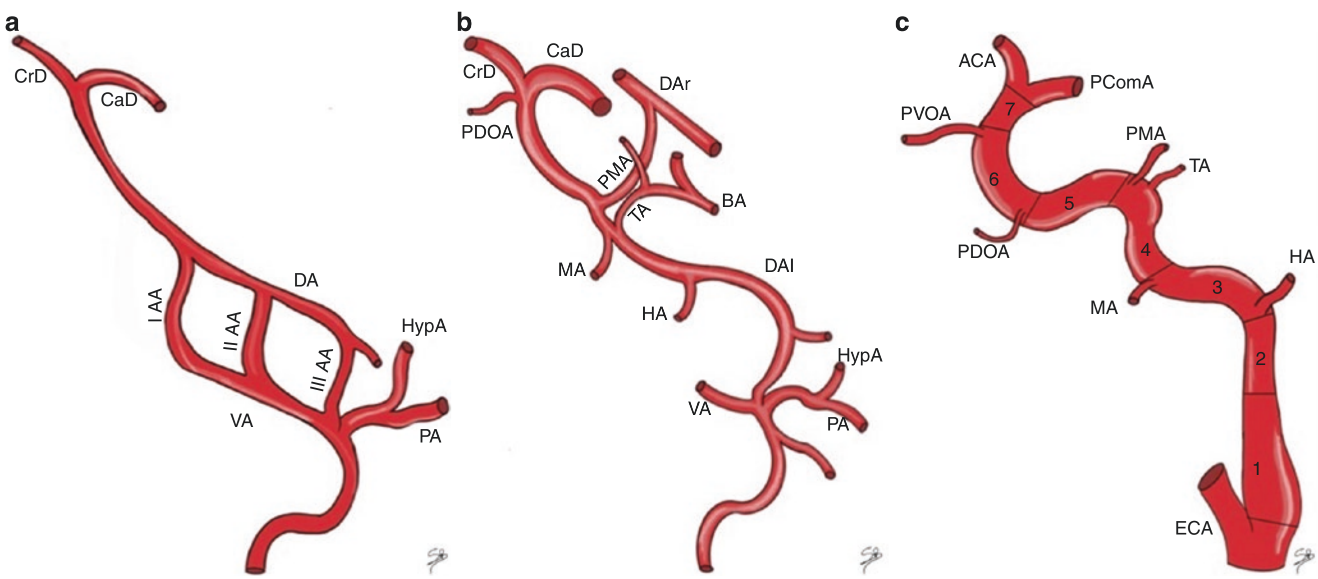


Fig. 1 Embryological segments of the internal carotid artery. The illustrations (**a**–**c**) show consecutive stages of ICA embryological development. The first stages of development (**a**) are characterized by the presence of three aortic arches that link the ventral and dorsal aorta (VA, DA). The VA regresses together with the ventral part of the aortic arches. The dorsal remnants of the aortic arches persist as embryonic arteries. These embryonic arteries divide the ICA into seven embryological segments: (1) The cervical segment: it derives from the remnant of the third aortic arch (III AA). (2) The ascending intrapetrous segment: it is the remnant of the dorsal aorta (DA) between the second (II AA) and third aortic arches. The division point between segments 2 and 3 is at the point of origin of the Hyoid artery (HA), that is the dorsal remnant of the second aortic arch. (3) The horizontal intrapetrous segment: it is the remnant of the DA between the first (I AA) and second aortic arches. The division point is at the point of origin of the mandibular artery (MA), that corresponds to the dorsal remnant of the first aortic arch. (4) The intracavernous ascending segment: it originates from the DA between the first aortic arch and the primitive maxillary artery (PMA), that connects the DA of the two sides (DAl: dorsal aorta left; DAr: dorsal aorta right). At the junction between segments 4 and 5 also the trigeminal artery (TA) takes its origin. This latter represents a primitive connection between the cavernous ICA and the basilar artery (BA). (5) The horizontal intracavernous segment: it derives from the DA between the PMA and the primitive dorsal ophthalmic artery (PDOA). (6) The clinoid segment: corresponds to the DA between the PDOA and the primitive ventral ophthalmic artery (PVOA). (7) The terminal segment: the terminal ICA between (PVOA) and the primitive ICA bifurcation into the future anterior cerebral artery (ACA) and future posterior communicating artery (PComA). The figure shows also the hypoglossal artery (HypA) and the proatlantal artery (PA), which have their origin proximal to the third aortic arch and will contribute to the formation of external carotid artery (ECA) branches

the primitive ventral ophthalmic artery (PVOA). The seventh segment is between the PVOA and the primitive carotid bifurcation [23, 24, 31, 39].

It is important to note that the carotid bulb has not the same embryological origin than the other segments of the ICA. It originates from the pharyngo-occipital system that easily explains variations in origin of the ascending pharyngeal and occipital arteries. Agenesis or abnormal regression of one or more segments of the ICA explains an intratympanic course of the ICA (by pharyngo-carotid anastomosis) and also the different type of "reperfusion" in case of ICA agenesis [12, 32]. The intratympanic course of the ICA also named as "aberrant flow of the ICA" is the consequence of the abnormal regression of the first and second segments with anastomosis between the inferior tympanic artery (from the ascending pharyngeal artery) and the caroticotympanic artery (from the carotid artery) that infuses distally the ICA. The pseudo-ICA has consequently an intratympanic course without passing through the stapes [12, 39, 49].

The Hyostapedial System

"Hyostapedial artery" is the term used to describe the complete embryological development of the second aortic arch. The SA, that develops from the hyoid artery and takes its name after passing through the crus of the stapes, is an embryonic artery present between the stages III and VI of Padget (9–24 mm). This is an important embryological system from which numerous dural, orbital and facial arteries develop. The steps of SA development are summarized in Table 1.

The hyoid artery is the dorsal remnant of the second aortic arch which regresses early in the embryological development (4–5 mm, stage I of Padget) [2, 12]. After this arch regression, approximately at the 5–6 mm stage (stage II of Padget), the hyoid artery presents a rapid lateral elongation between the beginning of the 7th and 19th weeks of gestation, when it gives an anastomosis to the mandibular artery (remnant of the first aortic arch) [31]. During the stage III of Padget (7–12 mm embryos) the hyoid artery is prominent and grows cranially, passing, as stapedial artery, through the crus of stapes. In contrast, the mandibular artery is short and difficult to identify. During this stage the initial formation of the two main branches of the SA (supraorbital and maxillomandibular divisions) is visible, even if they are only completely established in the stage IV of Padget (12–14 mm). At the 16–18 mm stage (stage V of Padget), the SA continues its elongation to the gasserian region passing into the future tympanic cavity and particularly through the crus of the stapes reaching its maximal development [23, 31, 32]. The supraorbital artery, which follows the ophthalmic root of the trigeminal nerve, allows the development of orbital branches (supraorbital, lacrimal, ethmoids and frontal arteries) and

Table 1 Major embryological changes in the formation of the stapedial artery

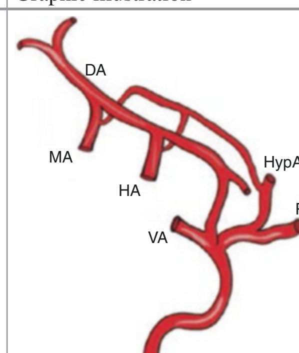
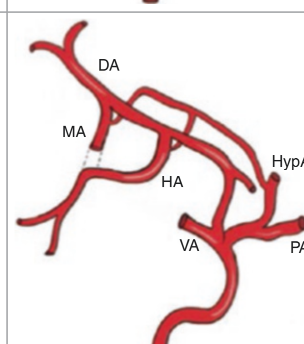
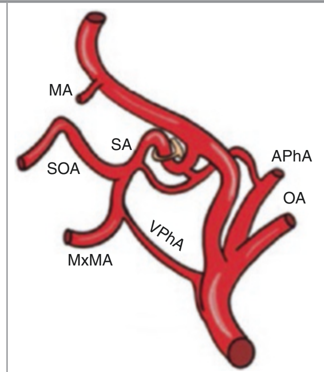
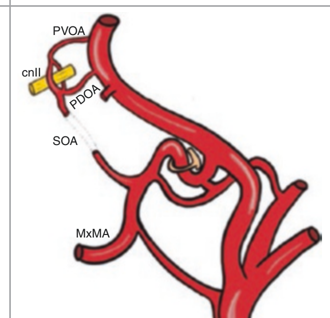
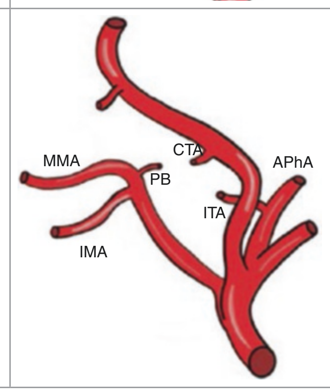
Stage	Embryo size (mm)	Major evolutions	Graphic illustration
I	4–5	– Regression of the ventral part of the first and second aortic arches – Hyoid artery (HA) formation (dorsal remnant of the second aortic arch)	
II	5–6	– Elongation of the hyoid artery (HA) – Annexation of the mandibular artery (MA) territory (first aortic arch) by the hyoid artery (second aortic arch)	

Table 1 (continued)

Stage	Embryo size (mm)	Major evolutions	Graphic illustration
III/IV	7–12 12–14	– Cranial growing of the hyoid artery (stapedial artery) passing into the middle ear (crus of the stapes) – Extension of the two branches of the stapedial artery: Supraorbital (SOA) and Maxillomandibular (MxMA)	
V	16–18	– Maximal development of the stapedial artery – Annexation of the supraorbital branch (SOA) by the ophthalmic artery (PDOA, PVOA) – Regression of the trans-osseous (superior orbital fissure) segment of the supraorbital branch	
VI	20–24	– Annexation of the maxillomandibular branch by the ventral pharyngeal artery – Regression of the intratympanic segment of the stapedial artery	

also of the MMA [39]. The maxillomandibular artery gets out of the cranial cavity through the foramen spinosum and gives extracranially its two branches: infraorbital and mandibular arteries (future infraorbital and inferior alveolar arteries). After this maximal development of the SA, two annexations and two regressions occur to give the adult configuration of the MMA. Intracranially, the orbital branches are annexed by the primitive OA and the trans-sphenoidal segment of the supraorbital branch regresses leaving an anastomotic artery between the anterior branch of the MMA and the lacrimal artery (the sphenoidal artery) that penetrates the orbit through the superior orbital fissure (SOF). Extracranially, the ventral pharyngeal artery annexes the maxillomandibular artery of the SA, forming the proximal stem of the MMA and becoming the ECA. Consequently, by flow reversal into the SA, its tympanic portion regresses and leaves as remnants the caroticotympanic artery (from the ICA) and the superior tympanic artery (from the petrous branch of the MMA). These annexations and regressions concerning the SA happen during the stage VI of Padget (20–24 mm embryos) [31, 39].

Ophthalmic Artery

The embryogenesis of the primitive OA is related in some phases to the hyostapedial systems. These phases are resumed and related to the concomitant SA steps of development in Table 2.

The formation of the OA depends on two different arteries during 4–18 mm stages, the PDOA and the PVOA. The PDOA develops from the cavernous segment of the primitive

Table 2 Summary of embryological steps of stapedial and ophthalmic systems

Stapedial system	Embryo size (mm)	Ophthalmic system
Regression of the second aortic arch Hyoid artery formation (dorsal remnant of the second aortic arch)	4–5	Primitive maxillary artery as temporary branch PDOA appearance
Elongation of the hyoid artery Annexation of the mandibular artery (first aortic arch) by the hyoid artery (second aortic arch)	5–6	Primitive hyaloid artery = plexiform channels PVOA appearance
Cranial growing of the hyoid artery (stapedial artery) passing into the middle ear (crus of the stapes)	7–14	Formation of primitive hyaloid and common ciliary arteries
Maximal development of the stapedial artery Extension of the two branches of the stapedial artery: Supraorbital and Maxillomandibular	16–19	Migration in origin of the PVOA Regression of the PDOA Formation of the anastomotic ring
Regression of the trans-osseous (superior orbital fissure) segment of the supraorbital branch	20–24	Ventral interruption of the anastomotic ring Annexation of the supraorbital branch by the OA

internal carotid artery and the PVOA from the anterior division of the primitive ICA. The PDOA penetrates the orbit through the superior orbital fissure and the PVOA through the optic canal.

Then, two major anastomoses between these two arteries are formed. The first one is an intraorbital plexiform anastomosis supplied by the two arteries around the optic nerve (future second segment of the OA). The second anastomosis is intradural between the PVOA and the primitive ICA to form the definitive supraclinoidal origin of the OA. The proximal portion of the PVOA (between its origin on the ACA and its anastomosis with the ICA) then regresses to give the adult stem of the OA.

In a following step, the proximal part of the PDOA regresses and its remnant becomes the ILT of the primitive ICA.

Concomitantly, in 7 and 24 mm embryos (stages III–VI), the SA arises from the primitive hyoid arch of the petrous ICA. As described above, its supraorbital artery enters the orbit to give orbital branches. It divides into two different branches: the ethmoido-nasal and the lacrimal arteries. The ethmoido-nasal artery anastomoses with the primitive OA at the arterial ring around the optic nerve described before. Then, the trans-sphenoidal part of the supraorbital artery regresses and its orbital branches are annexed by the primitive OA to give the adult conformation.

The Trigeminal Artery

The trigeminal artery (TA) represents one of the carotid-basilar anastomoses, that appears in the 4–5 mm embryos (stage I of Padget) and disappears in the 12 mm embryos (stage III of Padget). It originates from the basilar artery (BA), between the superior and the anteroinferior cerebellar arteries, passes medial to the gasserian ganglion and follows the trigeminal nerve to the primitive ICA, at the level of the junction between the fourth and fifth segments of the ICA. The TA has a common origin on the ICA with the primitive maxillary artery. The carotid remnant of these two primitive arteries will be the future meningo-hypophyseal trunk, from which the lateral clival, marginal tentorial and inferior hypophyseal arteries will originate [31, 32]. On the other hand, the basilar remnant of the TA could be considered responsible for MMA possible origin from the BA in adult, thanks to its anastomosis in the gasserian region with a persistent stapedial artery. The details of this variant are explained in the chapter "Anatomy, Embryology and Variations of the Middle Meningeal Artery."

Formation of the Inferolateral Trunk

As supported by the theory of P. Lasjaunias concerning the embryology of the ophthalmic artery, the ILT is the carotid remnant of the PDOA. The PDOA develops from the cavernous segment of the primitive ICA and penetrates the orbit through the SOF. In the 40 mm embryos, the proximal part of the PDOA regresses and its remnant becomes the ILT. At the adult configuration, the ILT is composed of four branches: (1) superior branch that supplies the roof of the cavernous sinus; (2) anteromedial branch which passes into the SOF; (3) anterolateral branch that runs into the foramen rotundum and (4) posterior branch passing medial to the gasserian ganglion. These branches present a lot of anastomoses in the cavernous region that correspond to remnants of primitive trigeminal, ophthalmic, stapedial and maxillary arteries.

Persistence of the Stapedial Artery

Changes in the events previously described can determine different adult configurations of the arteries related to the hyostapedial system, like the persistency of the SA. The incidence of this variant is estimated to 0.48% after a series of more than 1000 temporal bone dissections [6]. The possible variants related to the persistency of the SA are illustrated in Fig. 2, including the rarer association of SA persistency with the so called pseudo-petrous ICA.

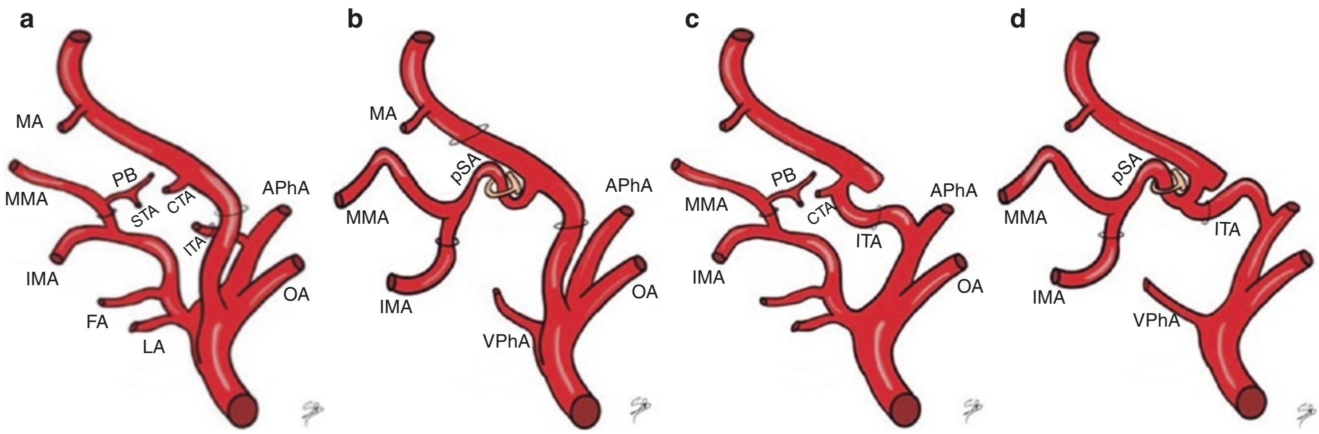


Fig. 2 Persistent stapedial artery and intratympanic flow of the ICA. (**a**) *Normal regression process of the hyostapedial system*. After the stapedial artery proximal regression, its proximal remnant originates from the ICA as carotico-tympanic artery (CTA). Its distal part is annexed by the ventral pharingeal artery, future external carotid artery, to give the internal maxillary artery (IMA) and its first and major branch, the middle meningeal artery (MMA). The MMA side of the stapedial artery persists as petrosal branch (PB), that gives birth to the superior tympanic artery (STA). The anastomotic branch to the hyostapedial system from the ascending pharyngeal artery (APhA) persists as inferior tympanic artery (ITA). The CTA, STA and ITA participate to the adult vascularization of the middle ear. (**b**) *Persistent stapedial artery*. In case of complete SA persistency, this artery originates from the petrous ICA, and gives intracranially the MMA and the IMA exits the middle fosse skull base through the foramen spinosum as extracranial branch. This variant is due to the lack of annexation of the maxillo-mandibular branch by the ventral pharyngeal artery. (**c**) *Pseudo-petrous or aberrant intratympanic ICA*. This variant is due to the agenesis of the cervical ICA (third aortic arch). The cervical ICA agenesis is bypassed through a hypertrophic inferior tympanic artery that courses from the APhA, through the inferior tympanic canal and reaches the CTA. The intratympanic ICA flow is called "aberrant" since the artery does not course into its usual canal, but into an enlarged inferior tympanic canal. (**d**) *Pseudo-petrous ICA + SA persistency*. This variant is due to two phenomena: the agenesis of the cervical ICA, and stapedial artery persistency. In this case the ITA does not reach the CTA but the persistent SA (pSA)

Complete Persistence of the Stapedial Artery

The complete persistence of the SA is a very rare variant, only two cases were published in a context of ICA aneurysm or PHACE syndrome [25, 39]. In these cases, the SA could be seen as in the embryo taking its origin from the petrous ICA, passing through the middle ear and giving its two branches: one intracranial that corresponds to the MMA and the other extracranial leaving the cranial cavity through the foramen spinosum. Consequently, the foramen spinosum is enlarged, the cochlear promontory is eroded and the IMA arises from the SA instead of the ECA. Such an anatomical variant could easily be explained by the embryology and particularly by the absence of annexation of the maxillomandibular branch (of the SA) by the ventral pharyngeal artery. Consequently, in absence of reversion of the arterial flow into the SA, its proximal (intratympanic) stem could not regress [39].

Partial Persistence of the Stapedial Artery

The partial persistence of the SA is more frequent and in this case, only the intracranial branch of the SA keeps its origin from the petrous ICA [1, 7, 21–24, 26, 32, 50, 51]. The foramen spinosum is absent (an orifice does not exist without its contents) or reduced in size and the MMA arises from the SA instead of the IMA. This variant is explained by the regression of the proximal part of the maxillomandibular artery instead of the proximal part of the SA [39]. A rare case of bilateral partial persistent SA is illustrated in Fig. 3. We must note that it is surprising that the complete persistence of the SA is even much rarer than its partial persistence. This could be the illustration of a lack of embryological knowledge of the SA or a misunderstanding, particularly in the annexation of the maxillomandibular branch by the ventral pharyngeal artery.

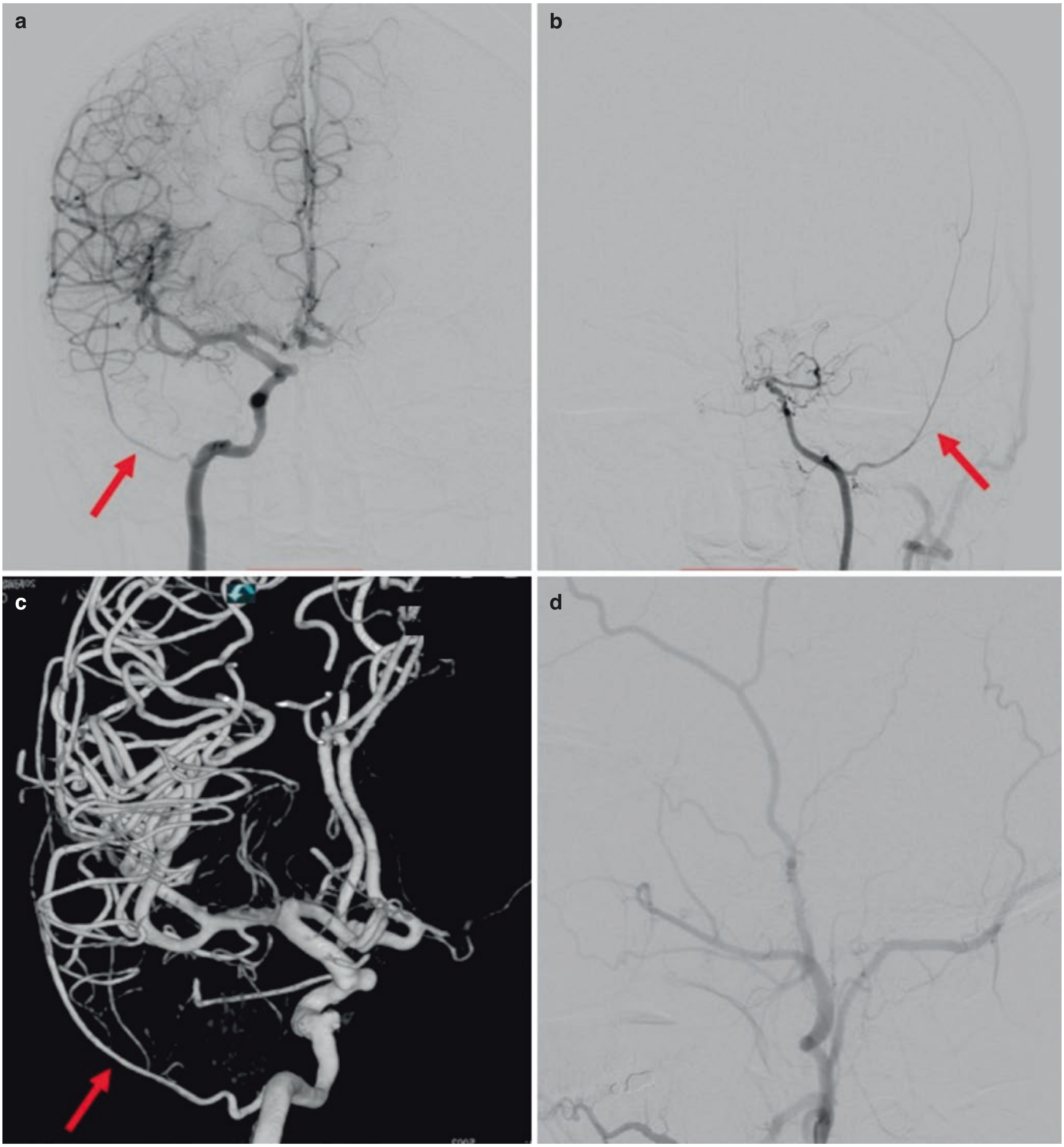


Fig. 3 DSA with bilateral partial persistent Stapedial Artery in patient with moyamoya disease. (**a**) and (**b**) Show respectively right and left ICA injection with the MMA (red arrow) arising from the petrous ICA. This phenomenon is possible because of bilateral stapedial artery persistency. In (**b**) the ICA is narrowed and stopped at the level of the ophthalmic artery due to moyamoya disease. (**c**) Shows a 3D reconstruction of the right ICA from which the MMA originates (red arrow). (**d**) Shows the left ECA injection without the MMA enhancement, because of its absence

Persistence of the SA Associated with Aberrant Flow of the ICA (Pseudo-Petrous ICA)

In rare cases, the persistence of the SA is associated with an intratympanic course of the ICA (also known as "aberrant flow of the ICA") [12, 21, 23, 24, 52, 53]. This association of vascular variants concerning both carotid and hyostapedial systems was described anatomically and angiographically [32, 39]. In these cases, the ICA enters the skull base through an enlarged inferior tympanic canal (narrowing of the vessel on digital subtraction angiography (DSA)), passes into the tympanic cavity to bend anteriorly and reaches its normal carotid canal. The MMA arises from the ICA in its tympanic segment and passes through the stapes to have the same course described in the previous paragraph. The exocranial orifice of the carotid canal is therefore absent in these cases [23, 24]. The intratympanic course of the ICA is explained by the agenesis of the two first segments of the primitive ICA. The cervical segment is in fact the ascending pharyngeal artery with a hypertrophied inferior tympanic artery that maintains its anastomosis with the caroticotympanic artery (branch of the ICA) into the tympanic cavity. The correct term of this aberrant flow of the ICA is, in reality, tympano-caroticotympanic variant [39]. It bypasses the absence of the cervical ICA and provides a collateral circulation to the remainder of the ICA. Therefore, the cervical and intratympanic segments of this artery do not derivate from the carotid system but from the pharyngo-occipital and hyostapedial systems (pseudo-ICA) [23, 24, 39]. The agenesis of the first two segments of the ICA could be partial and the ICA appears duplicated [52].

Pharyngo-Tympano-Stapedial Artery

This very rare variant was first described by Lasjaunias (1977) in its original publication [23]. The same case served as illustration in the textbook *Surgical Angiography* and only one similar case was published by Baltsavias et al. (2012) [39, 42]. The MMA arises from the cervical portion of the ICA, ascends along the cervical ICA, enters the tympanic cavity through the inferior tympanic canal and follows the usual course of the SA. The two cases described were presented as "partial" persistence of the SA with only the MMA arising from the SA and the absence of the foramen spinosum. In this variant, an annexation of the SA by the inferior tympanic artery (branch of the ascending pharyngeal artery) with regression of the proximal part of the SA explains this vascular configuration. Therefore, the SA arises from the cervical instead of the petrous segment of the ICA.

MMA Origin of the OA

The first description of orbital branches arising from the MMA was published in 1872 by Curnow [54]. He already noted in its case dissection that all orbital branches were supplied by the MMA except the central retinal artery which kept its origin from the supracavernous ICA. Twenty years after this first description, Musgrove (1893) presented another case of MMA origin of the OA where also the central retinal artery was supplied by the MMA and the ICA did not give any orbital branch [55]. After other isolated cases found during cerebral or orbital dissections, Hayreh (1962) reported 6 cases among its 170 orbital dissections whom the MMA participated completely (2 cases) or partially (4 cases) in the orbital supply [1, 27, 56–58]. Then, Moret (1977) and Lasjaunias (1977) described with precision the contribution of the MMA to the orbital vascularization and gave us a more comprehensive explanation of this anatomical variation [23, 36].

The incidence of OA that arises from the MMA and penetrates the orbit through the SOF is estimated between 1.4% and 2.5% [56]. In this anatomical variation, orbital arteries are supplied by the anterior division of the MMA passing through the SOF or through the spheno-orbital foramen. The central retinal artery generally keeps its vascular supply from the supracavernous ICA but in few cases, also the central retinal artery is supplied by the MMA without ICA participation [59]. A case of complete supply of the orbital arteries by the MMA is illustrated in Fig. 4.

As explained before, during the embryogenesis, the SA gives the supraorbital branch which allows the development of the lacrimal artery and anastomoses with the OA around the optic nerve (participation of the peri-optic circle). The proximal part of the supraorbital branch then normally regresses and the lacrimal artery is annexed by the OA [39]. The persistence of MMA branches (or, in extremis, MMA origin of the OA) could be explained by two different phenomena: the absence of anastomosis between the supraorbital branch and the OA, with consequent persistence of a dual supply of the orbital arteries, or the persistence of the proximal stem of the supraorbital branch of the SA with regression of the primitive OA (complete MMA origin of the OA). Although the supraorbital branch gives off its bifurcation (lacrimal artery laterally and ethmoido-nasal medially) usually inside the orbit, in 30% of cases it can divide outside the orbit in the middle cranial fossa. In such cases, the ethmoido-nasal artery passes through the SOF, but the lacrimal artery penetrates the orbit through its own canal: the spheno-orbital foramen (canal of Hyrtl, lacrimal foramen, sinus canal). The medial branch, passing through

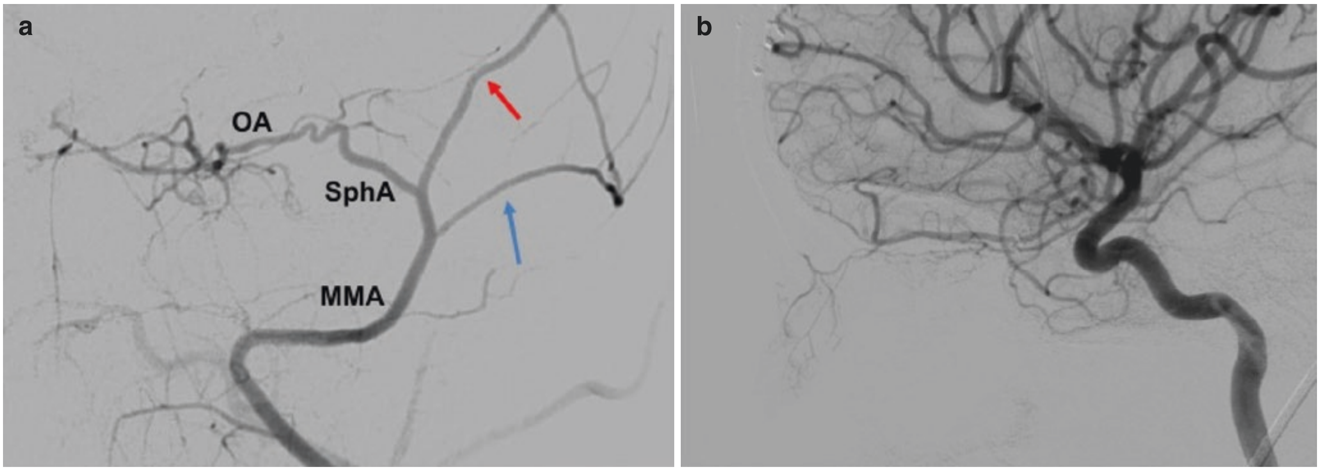


Fig. 4 MMA origin of the OA. The figure shows a case of complete OA origin from the MMA. The selective injection of the MMA in antero-posterior projection (**a**) shows that the sphenoidal artery (SphA) arises from the MMA after its bifurcation into the anterior (red arrow) and posterior division (blue arrow). It courses along the inner surface of the temporal and sphenoidal bone and enters the orbit through the SOF to give rise to the OA. (**b**) Shows an oblique ICA injection highlighting the absence of the OA

the SOF, is named recurrent meningeal artery in its intra-orbital segment and sphenoidal artery in its intracranial segment [23, 31, 39, 56].

Conclusion

All the anatomical variations linked to the development of the SA, resumed in Table 3, could have a clinical impact. Since each vascular segment that regresses during the embryological life, usually persists as anastomosis between two arteries, the knowledge of the embryology and presence of these anastomoses is of paramount importance in case of external carotid branches embolization. Each residual anastomosis represents a potential undesired leak of liquid embolic agent, with possible clinical implications. The knowledge of the embryological variants described in this chapter, represents the basis to understand the different possible adult configurations of the MMA and their clinical implications, which will be described in the chapter "Anatomy, Embryology and Variations of the Middle Meningeal Artery." The most common pathologies, whose treatment requires a detailed knowledge of MMA embryology and variants, are dural arterio-venous fistulas, meningeal tumors and chronic subdural hematomas.

Table 3 Anatomical variations implicating the stapedial artery

Anatomical variations			Embryological implications	
Type	Incidence	Direct and indirect signs	Embryological explanation	Embryos size (mm)
Complete persistence of the SA	2 cases	– Petrous ICA origin of the MMA – Petrous ICA origin of the IMA – Enlarged foramen spinosum – Eroded cochlear promontory	– Lack of annexation of the maxillomandibular branch by the ventral pharyngeal artery – Persistence of the tympanic segment of the SA	24
Partial persistence of the SA	0.4%	– Petrous ICA origin of the MMA – Absent foramen spinosum – Enlarged facial canal	– Regression of the proximal segment of the maxillomandibular branch – Persistence of the tympanic segment of the SA	24
Tympano-carotico-tympanic variant Aberrant flow of the ICA + persistent SA	5 cases	– Intratympanic course of the ICA – Petrous ICA origin of the MMA – Enlarged inferior tympanic canal – Absent exocranial carotid canal	– Regression of the proximal segment of the maxillomandibular branch – Persistence of the tympanic segment of the SA – Agenesis of the first two segments of the ICA – Anastomosis between inferior tympanic artery and caroticotympanic artery	24 4–5
Pharyngo-tympano-stapedial variant	2 cases	– Cervical ICA origin of the MMA – Absent foramen spinosum	– Regression of the proximal segment of the maxillomandibular branch – Persistence of the tympanic segment of the SA – Anastomosis between inferior tympanic artery and caroticotympanic artery	24
MMA origin of the OA	1.4–2.5%	– MMA origin of the OA – No OA from the ICA or only the central retinal artery	– Lack of annexation of the supraorbital branch by the ophthalmic artery – Persistence of the sphenoidal part of the supraorbital branch	24

References

1. Fisher AG. A case of complete absence of both internal carotid arteries, with a preliminary note on the developmental history of the Stapedial artery. J Anat Physiol. 1913;48(Pt 1):37–46.
2. Kelemen G. Arteria stapedia, in bilateral persistence. AMA Arch Otolaryngol. 1958;67(6):668–77.
3. Sando I, Baker B, Black FO, Hemenway WG. Persistence of stapedial artery in trisomy 13–15 syndrome. Arch Otolaryngol. 1972;96(5):441–7.
4. Marano GD, Horton JA, Gabriele OF. Persistent embryologic vascular loop of the internal carotid, middle meningeal, and ophthalmic arteries. Radiology. 1981;141(2):409–10. https://doi.org/10.1148/radiology.141.2.7291564.
5. Diamond MK. Unusual example of a persistent stapedial artery in a human. Anat Rec. 1987;218(3):345–54. https://doi.org/10.1002/ar.1092180316.
6. Moreano EH, Paparella MM, Zelterman D, Goycoolea MV. Prevalence of facial canal dehiscence and of persistent stapedial artery in the human middle ear: a report of 1000 temporal bones. Laryngoscope. 1994;104(3 Pt 1):309–20. https://doi.org/10.1288/00005537-199403000-00012.
7. Kawai K, Yoshinaga K, Koizumi M, Honma S, Tokiyoshi A, Kodama K. A middle meningeal artery which arises from the internal carotid artery in which the first branchial artery participates. Ann Anat. 2006;188(1):33–8. https://doi.org/10.1016/j.aanat.2005.08.013.
8. Baron SH. Persistent stapedial artery, necrosis of the incus, and other problems which have influenced the choice of technique in stapes replacement surgery in otosclerosis. Laryngoscope. 1963;73:769–82. https://doi.org/10.1288/00005537-196306000-00014.
9. House HP, Patterson ME. Persistent Stapedial artery: report of two cases. Trans Am Acad Ophthalmol Otolaryngol. 1964;68:644–6.
10. Maran AG. Persistent stapedial artery. J Laryngol Otol. 1965;79(11):971–5.
11. Davies DG. Persistent stapedial artery: a temporal bone report. J Laryngol Otol. 1967;81(6):649–60.
12. Steffen TN. Vascular anomalites of the middle ear. Laryngoscope. 1968;78(2):171–97. https://doi.org/10.1288/00005537-196802000-00001.
13. Marion M, Hinojosa R, Khan AA. Persistence of the stapedial artery: a histopathologic study. Otolaryngol Head Neck Surg. 1985;93(3):298–312. https://doi.org/10.1177/019459988509300304.
14. Boscia R, Knox RD, Adkins WY, Holgate RC. Persistent stapedial artery supplying a glomus tympanicum tumor. Arch Otolaryngol Head Neck Surg. 1990;116(7):852–4.
15. Pahor AL, Hussain SS. Persistent stapedial artery. J Laryngol Otol. 1992;106(3):254–7.
16. Govaerts PJ, Marquet TF, Cremers WR, Offeciers FE. Persistent stapedial artery: does it prevent successful surgery? Ann Otol Rhinol Laryngol. 1993;102(9):724–8. https://doi.org/10.1177/000348949310200914.
17. Wardrop P, Kerr AI, Moussa SA. Persistent stapedial artery preventing successful cochlear implantation: a case report. Ann Otol Rhinol Laryngol Suppl. 1995;166:443–5.
18. Jain R, Gandhi D, Gujar S, Mukherji SK. Case 67: persistent stapedial artery. Radiology. 2004;230(2):413–6. https://doi.org/10.1148/radiol.2302021108.
19. Yamamoto E, Hirono Y. Persistent stapedial artery associated with otosclerosis. ORL J Otorhinolaryngol Relat Spec. 1988;50(6):382–4. https://doi.org/10.1159/000276017.
20. Hogg ID, Stephens CB, Arnold GE. Theoretical anomalies of the stapedial artery. Ann Otol Rhinol Laryngol. 1972;81(6):860–70.
21. Guinto FC Jr, Garrabrant EC, Radcliffe WB. Radiology of the persistent stapedial artery. Radiology. 1972;105(2):365–9. https://doi.org/10.1148/105.2.365.
22. Teal JS, Rumbaugh CL, Bergeron RT, Segall HD. Congenital absence of the internal carotid artery associated with cerebral hemiatrophy, absence of the external carotid artery, and persistence of

the stapedial artery. Am J Roentgenol Radium Ther Nucl Med. 1973;118(3):534–45.
23. Lasjaunias P, Moret J, Manelfe C, Theron J, Hasso T, Seeger J. Arterial anomalies at the base of the skull. Neuroradiology. 1977;13(5):267–72.
24. Lasjaunias P, Moret J. Normal and non-pathological variations in the angiographic aspects of the arteries of the middle ear. Neuroradiology. 1978;15(4):213–9.
25. Rodesch G, Choi IS, Lasjaunias P. Complete persistence of the hyoido-stapedial artery in man. Case report. Intra-petrous origin of the maxillary artery from ICA. Surg Radiol Anat. 1991;13(1):63–5.
26. Sheikh BY, Coates R, Siqueira EB. Stapedial artery supplying sphenoid wing meningioma: case report. Neuroradiology. 1993;35(7):537–8.
27. Diamond MK. Homologies of the meningeal-orbital arteries of humans: a reappraisal. J Anat. 1991;178:223–41.
28. Diamond MK. Homologies of the stapedial artery in humans, with a reconstruction of the primitive stapedial artery configuration of Euprimates. Am J Phys Anthropol. 1991;84(4):433–62. https://doi.org/10.1002/ajpa.1330840408.
29. Falk D. Meningeal arterial patterns in great apes: implications for hominid vascular evolution. Am J Phys Anthropol. 1993;92(1):81–97. https://doi.org/10.1002/ajpa.1330920107.
30. Hyrtl J. Über mehrere im Menschen vorkommende analogieen derjenigen arterie welche Otto bei mehreren Winterschläfern durch den Steigbügel verlaufend entdeckte. Neue beobachtungen aus dem gebieteder menschlichen und vergleichenden anatomie. Med Jahrb d Oesterreich Staates. 1836;10:457–66.
31. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol. 1948;212:205–62.
32. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
33. Gabriele OF, Bell D. Ophthalmic origin of the middle meningeal artery. Radiology. 1967;89(5):841–4. https://doi.org/10.1148/89.5.841.
34. Salamon G, Guerinel G, Demard F. Radioanatomical study of the external carotid artery. Ann Radiol (Paris). 1968;11(3):199–215.
35. Seeger JF, Hemmer JF. Persistent basilar/middle meningeal artery anastomosis. Radiology. 1976;118(2):367–70. https://doi.org/10.1148/118.2.367.
36. Moret J, Lasjaunias P, Theron J, Merland JJ. The middle meningeal artery. Its contribution to the vascularisation of the orbit. J Neuroradiol. 1977;4(2):225–48.
37. Merland JJ, Theron J, Lasjaunias P, Moret J. Meningeal blood supply of the convexity. J Neuroradiol. 1977;4(2):129–74.
38. Moret J, Lasjaunias P, Vignaud J, Doyon D. The middle meningeal blood supply to the posterior fossa (author's transl). Neuroradiology. 1978;16:306–7.
39. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer; 2001.
40. Maiuri F, Donzelli R, de Divitiis O, Fusco M, Briganti F. Anomalous meningeal branches of the ophthalmic artery feeding meningiomas of the brain convexity. Surg Radiol Anat. 1998;20(4):279–84.
41. Shah QA, Hurst RW. Anomalous origin of the middle meningeal artery from the basilar artery: a case report. J Neuroimaging. 2007;17(3):261–3. https://doi.org/10.1111/j.1552-6569.2007.00108.x.
42. Baltsavias G, Kumar R, Valavanis A. The pharyngo-tympano-stapedial variant of the middle meningeal artery. A case report. Interv Neuroradiol. 2012;18(3):255–8. https://doi.org/10.1177/159101991201800302.
43. Kumar S, Mishra NK. Middle meningeal artery arising from the basilar artery: report of a case and its probable embryological mechanism. J Neurointerv Surg. 2012;4(1):43–4. https://doi.org/10.1136/jnis.2010.004465.
44. Plas B, Bonneville F, Dupuy M, Sol JC, Chaynes P. Bilateral ophthalmic origin of the middle meningeal artery. Neurochirurgie. 2013;59(4–5):183–6. https://doi.org/10.1016/j.neuchi.2013.08.005.
45. Cvetko E, Bosnjak R. Unilateral absence of foramen spinosum with bilateral ophthalmic origin of the middle meningeal artery: case report and review of the literature. Folia Morphol (Warsz). 2014;73(1):87–91. https://doi.org/10.5603/FM.2014.0013.
46. Salem MM, Fusco MR, Dolati P, Reddy AS, Gross BA, Ogilvy CS, et al. Middle meningeal artery arising from the basilar artery. J Cerebrovasc Endovasc Neurosurg. 2014;16(4):364–7. https://doi.org/10.7461/jcen.2014.16.4.364.
47. Kimball D, Kimball H, Tubbs RS, Loukas M. Variant middle meningeal artery origin from the ophthalmic artery: a case report. Surg Radiol Anat. 2015;37(1):105–8. https://doi.org/10.1007/s00276-014-1272-z.
48. Lasjaunias P, Santoyo-Vazquez A. Segmental agenesis of the internal carotid artery: angiographic aspects with embryological discussion. Anat Clin. 1984;6(2):133–41. https://doi.org/10.1007/bf01773165.
49. Margolis MT, Newton TH. Collateral pathways between the cavernous portion of the internal carotid and external carotid arteries. Radiology. 1969;93(4):834–6. https://doi.org/10.1148/93.4.834.
50. McLennan JE, Rosenbaum AE, Haughton VM. Internal carotid origins of the middle meningeal artery. The ophthalmic-middle meningeal and stapedial-middle meningeal arteries. Neuroradiology. 1974;7(5):265–75.
51. Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A Jr. Microsurgical anatomy of the dural arteries. Neurosurgery. 2005;56(2 Suppl):211–51; discussion 51.
52. Koenigsberg RA, Zito JL, Patel M, Swartz JD, Goldofsky E, Zahtz G. Fenestration of the internal carotid artery: a rare mass of the hypotympanum associated with persistence of the stapedial artery. AJNR Am J Neuroradiol. 1995;16(4 Suppl):908–10.
53. Silbergleit R, Quint DJ, Mehta BA, Patel SC, Metes JJ, Noujaim SE. The persistent stapedial artery. AJNR Am J Neuroradiol. 2000;21(3):572–7.
54. Curnow J. Two instances of irregular ophthalmic and middle meningeal arteries. J Anat Physiol. 1873;8(Pt 1):155–6.
55. Musgrove J. Origin of ophthalmic artery from middle meningeal. J Anat Physiol. 1893;27(Pt 2):279–81.
56. Hayreh SS, Dass R. The ophthalmic artery: I. origin and intra-cranial and intra-canalicular course. Br J Ophthalmol. 1962;46(2):65–98.
57. Chanmugan PK. Note on an unusual ophthalmic artery associated with other abnormalities. J Anat. 1936;70:580–2.
58. Bossi R, Pisani C. Collateral cerebral circulation through the ophthalmic artery and its efficiency in internal carotid occlusion. Br J Radiol. 1955;28(333):462–9. https://doi.org/10.1259/0007-1285-28-333-462.
59. Louw L. Different ophthalmic artery origins: embryology and clinical significance. Clin Anat. 2015;28(5):576–83. https://doi.org/10.1002/ca.22470.

Intratympanic Flow of the Internal Carotid Artery

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The term "aberrant flow of the internal carotid artery", even if it is the most used in the literature, is erroneous and does not highlight the pathophysiology of this disease [1–5]. The most precise term to describe this condition was proposed by P. Lasjaunias and is the inferior tympano-carotico-tympanic variant of the internal carotid artery (ICA) [5–7]. This term, even if complex, well illustrates the type of anastomosis encountered in this vascular variation. Another synonym also found in the literature is intratympanic flow of the internal carotid artery [8]. This chapter will focus on this specific vascular variation of the ICA. Other forms of segmental agenesis of the ICA are developed in the chapter "Segmental Agenesis of the Internal Carotid Artery."

History

The first case report of aberrant ICA was reported by Max, an Austrian otologist, in 1899 [9, 10]. He described a case of a young female with pulsatile mass of the middle ear without describing the surgical details. After him, a lot of isolated cases or little case series, approximately 100 cases in total, were published principally by otologist to highlight the importance of this diagnosis, the risk of massive bleeding in case of wrong diagnosis and the radiologic characteristics of aberrant ICA [1, 9, 11–22].

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The works in embryology of the cerebro-facial arteries of Congdon, Padget and Altman remain the cornerstones in the understanding of vascular variation of the cerebro-facial arteries [23, 24]. Steffen (1968) published a text that gives initial explanation of different vascular anomalies of the middle ear based on the embryological knowledge [25]. Few years after, Lapayowker (1971) was the first to describe the angiographic findings of an aberrant ICA and defined the "vestibular line" which is a radiological criterion cited by a lot of authors after him [16, 26–30]. From 1977 to 1984, P. Lasjaunias published masterpiece articles that summarized, always based on angiographic findings and embryological knowledge, the development of cerebro-facial arteries [5, 7, 31]. In 1984, he published an article that remains the reference to explain all segmental agenesis of the internal carotid artery and in particular the aberrant flow of the ICA [31]. J. Moret (1982) also gave an important contribution in the understanding of vascular anomalies of the middle ear [32].

Embryology of the Carotid System

In the present chapter, you will find only information to understand the formation of an "aberrant flow of the carotid artery" which is an intratympanic course of the pseudo-ICA. Table 1 summarizes each segment origin and Fig. 1 illustrates the transformation of the internal carotid artery from embryologic to adult life.

Early in the development (first stage of Padget, embryos of 4–5 mm), the two first aortic arches initiate their natural regression allowing the internal carotid artery to be individualized. The embryological segments of the ICA are derived from the third aortic arch and from the dorsal aorta cranial to the third aortic arch. The dorsal aorta also regresses at the same time between the third and fourth aortic arches [23, 24, 33, 34]. Embryologically, we could divide the internal carotid artery in seven different segments [6, 31]. The first one corresponds to the third aortic arch from the origin of the

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Table 1 Embryological development of the ICA

Embryological segment	Embryological origin	Adult segment	Proximal limit	Distal limit
First segment	Third aortic arch	Cervical ICA	Carotid bulb	Junction cervical-petrous ICA
Second segment	Dorsal aorta between second and third aortic arches	Ascending petrous segment	Junction cervical-petrous ICA	Caroticotympanic artery
Third segment	Dorsal aorta between first and second aortic arches	Horizontal petrous segment	Caroticotympanic artery	Vidian artery
Fourth segment	Dorsal aorta between first aortic arch and the primitive maxillary artery	Ascending lacerum segment	Vidian artery	Meningo-hypophysary trunk
Fifth segment	Dorsal aorta between the primitive maxillary artery and the PDOA	Horizontal cavernous segment	Meningo-hypophysary trunk	Infero-lateral trunk
Sixth segment	Dorsal aorta between the PDOA and the PVOA	Clinoidal segment	Infero-lateral trunk	Ophthalmic artery
Seventh segment	Dorsal aorta Distal to the PVOA	Ophthalmic segment	Ophthalmic artery	Posterior communicating artery

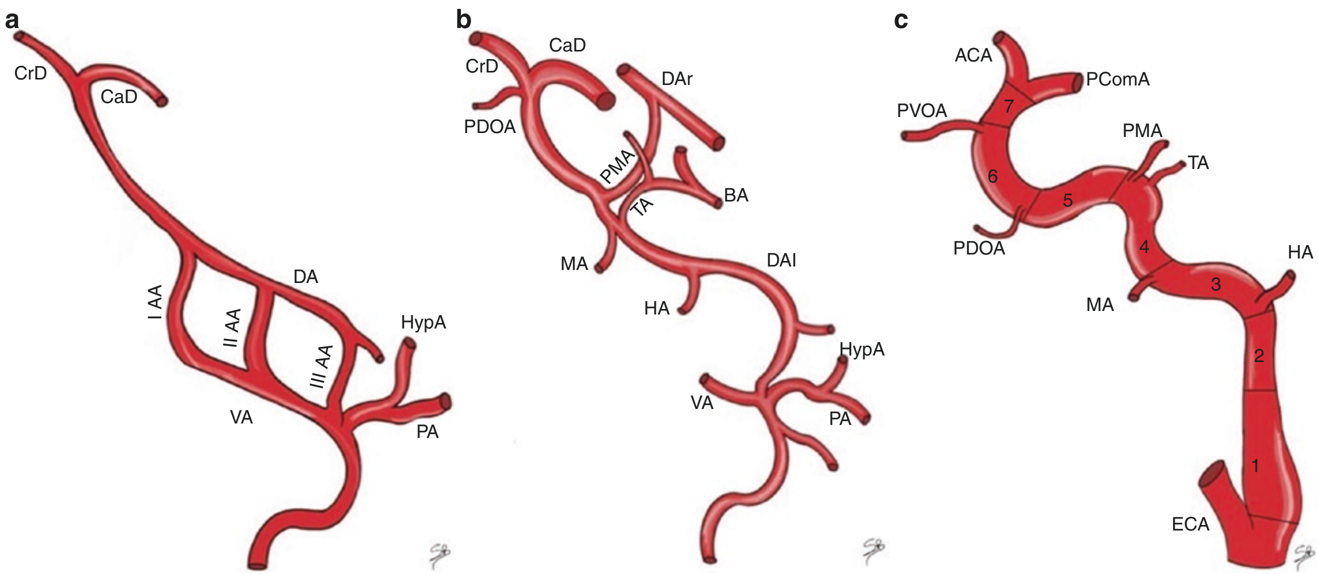

Fig. 1 Embryology and embryological segments of the internal carotid artery. The illustrations (**a**–**c**) show consecutive stages of ICA embryological development. The first stages of development (**a**) are characterized by the presence of three aortic arches that link the ventral and dorsal aorta (VA, DA). The VA regresses together with the ventral part of the aortic arches. The dorsal remnants of the aortic arches persist as embryonic arteries. These embryonic arteries divide the ICA into seven embryological segments: (1) The cervical segment: it derives from the remnant of the third aortic arch (III AA). (2) The ascending intrapetrous segment: it is the remnant of the dorsal aorta (DA) between the second (II AA) and third aortic arches. The division point between the segment 2 and 3 is at the point of origin of the Hyoid artery (HA), that is the dorsal remnant of the second aortic arch. (3) The horizontal intrapetrous segment: it is the remnant of the DA between the first (I AA) and second aortic arches. The division point is at the point of origin of the mandibular artery (MA), that corresponds to the dorsal remnant of the first aortic arch. (4) The intracavernous ascending segment: it origins from the DA between the first aortic arch and the primitive maxillary artery (PMA), that connects the DA of the two sides (*DAl* dorsal aorta left, *DAr* dorsal aorta right). At the junction between segments 4 and 5 also the trigeminal artery (TA) takes its origin. This latter represents a primitive connection between the cavernous ICA and the basilar artery (BA). (5) The horizontal intracavernous segment: it derives from the DA between the PMA and the primitive dorsal ophthalmic artery (PDOA). (6) The clinoid segment: corresponds to the DA between the PDOA and the primitive ventral ophthalmic artery (PVOA). (7) The terminal segment: the terminal ICA between (PVOA) and the primitive ICA bifurcation into the future anterior cerebral artery (ACA) and future posterior communicating artery (PComA). The figure shows also the hypoglossal artery (HypA) and the proatlantal artery (PA), that origin proximal to the third aortic arch and will contribute to the formation of external carotid artery (ECA) branches

ventral pharyngeal artery (future external carotid artery) to the junction between the third aortic arch and the dorsal aorta. The second segment is the dorsal aorta between the second and third aortic arches. The third segment is the dorsal aorta between the first and second aortic arch. The fourth segment is the dorsal aorta between the origin of the trigeminal artery (and the primitive maxillary artery) and the first aortic arch. The fifth segment is the dorsal aorta between the origin of the primitive dorsal ophthalmic artery (PDOA, future inferolateral trunk-ILT) and the origin of the trigeminal artery (and the primitive maxillary artery). The sixth segment is between the origin of the primitive ventral ophthalmic artery (PVOA) and the origin of the primitive dorsal ophthalmic artery (PDOA, future inferolateral trunk-ILT). The seventh segment is between the PVOA and the primitive carotid bifurcation.

It is important to note that the carotid bulb has not the same embryological origin than the other segments of the ICA. It originates from the pharyngo-occipital system that easily explains variations in origin of the ascending pharyngeal and occipital arteries. Agenesis or abnormal regression of one or more segments of the carotid artery explains an intratympanic course of the ICA (by pharyngo-carotid anastomosis) and also the different type of "reperfusion" in case of ICA agenesis.

The intratympanic course of the ICA, also named as "aberrant flow of the ICA", is the consequence of the abnormal regression of the first and second segments with anastomosis between the inferior tympanic artery (from the ascending pharyngeal artery) and the carotico-tympanic artery (from the carotid artery) that infuses distally the carotid artery [6, 31]. The understanding of an anastomosis between these branches well explains why the term "inferior tympano-carotico-tympanic variant of the internal carotid artery" is the most appropriate [6, 31, 35, 36]. The pseudo-ICA has consequently an intratympanic course without passing through the stapes [6, 7, 31].

Intratympanic Flow of the ICA

Aberrant ICA or intratympanic flow of the ICA is a rare but well-known anatomical variation of the internal carotid artery. An aberrant ICA could remain asymptomatic or could be responsible for nonspecific symptoms: pulsatile tinnitus, recurrent otitis media, conductive hearing loss, vertigo, or otosclerosis [1–3, 8–11, 15, 17–19, 21, 22, 37–54]. In numerous cases, the aberrant ICA was misdiagnosed for a glomus tympanicum and bored the physician in uncontrollable bleeding loss during a middle ear surgery [1, 10, 32, 38, 43]. The intratympanic course

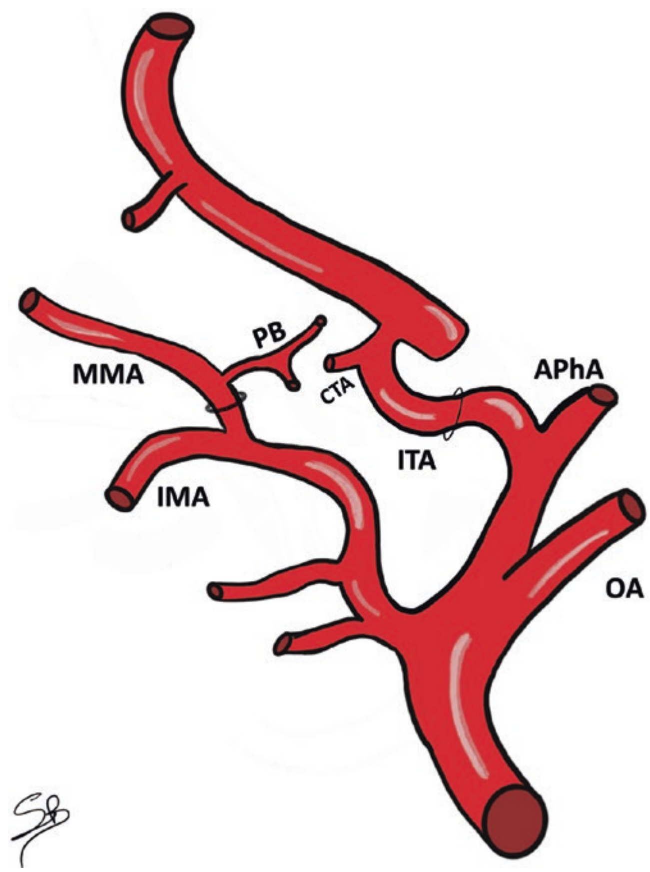

Fig. 2 Pseudo-petrous or aberrant intratympanic ICA. This variant is due to the agenesis of the cervical ICA (third aortic arch). The cervical ICA agenesis is bypassed through a hypertrophic inferior tympanic artery (ITA), that courses from the ascendent pharyngeal artery (APhA), through the inferior tympanic canal and reaches the carotico-tympanic artery (CTA) to supply the ICA distal flow. The intratympanic ICA flow is called "aberrant" since the artery does not course into its usual canal but into an enlarged inferior tympanic canal

of the ICA is explained by the agenesis of the two first segments of the primitive carotid artery. The cervical segment is composed in this case by the ascending pharyngeal artery with a hypertrophied inferior tympanic artery (branch of the ascending pharyngeal artery) that maintains its anastomosis with the carotico-tympanic artery (branch of the ICA) into the tympanic cavity (Fig. 2) [6, 31]. The correct term for this variant is thus tympano-carotico-tympanic variant [6, 35]. It bypasses the absence of the cervical ICA and provides a collateral circulation to the remainder of the ICA. Therefore, the cervical and intratympanic segments of this artery do not derivate from the carotid system but from the pharyngo-occipital and hyostapedial systems (pseudo-ICA) [6, 31].

Lapayowker (1971) defined the angiographic findings in case of aberrant ICA, with in particular the lateral displacement of the petrous ICA, lateral to the "vestibular line" [26].

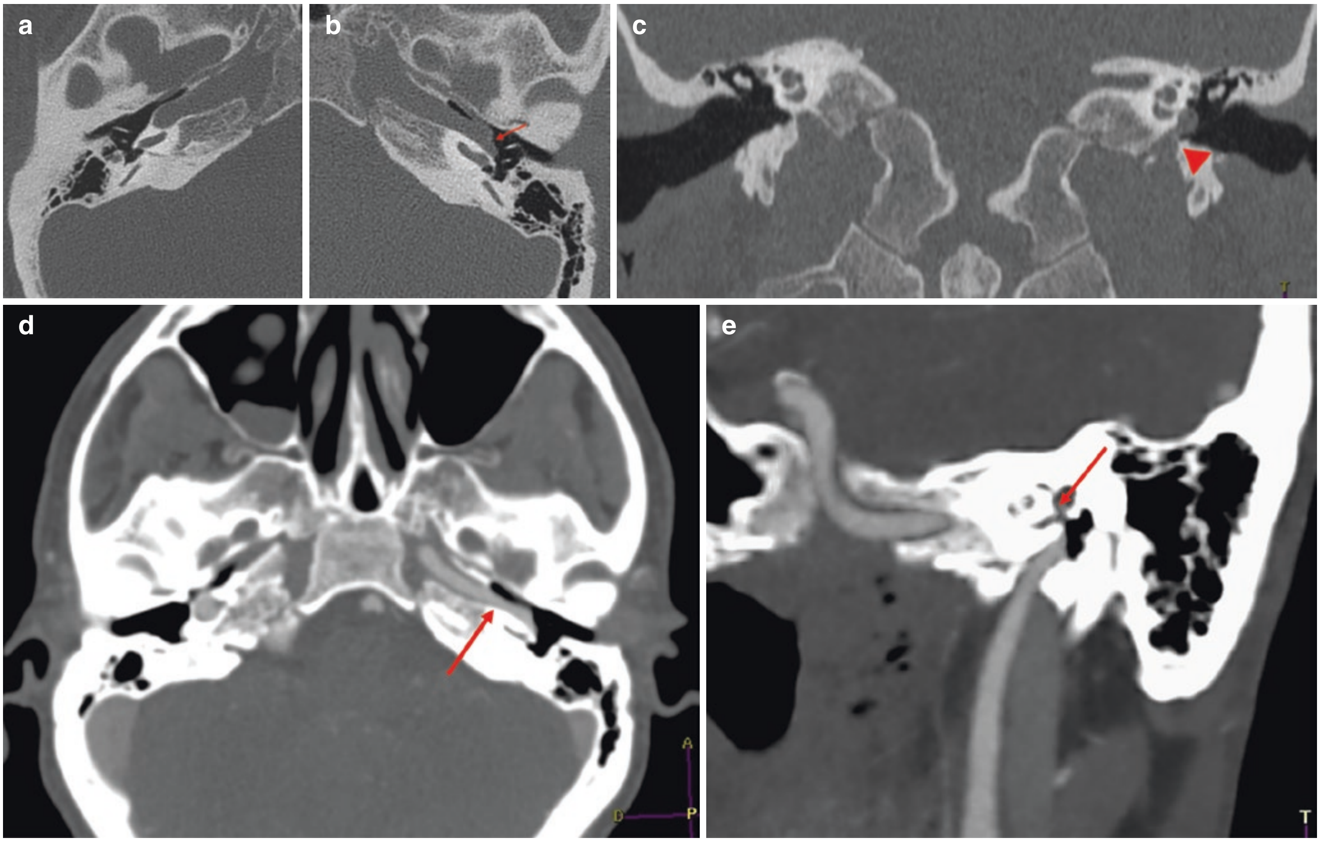

Fig. 3 Clinical case of pseudo-petrous or aberrant ICA. Compared to normal side (**a**), axial image (**b**) and coronal (**c**) projection reveal on the left side a tubular structure crossing the middle ear cavity close to the cochlear promontory. Note the caliber change of the aberrant vessel rejoining the horizontal petrous ICA (red arrow in **b**) and the enlargement of the left inferior tympanic canaliculus (red arrowhead in **c**). CTA axial (**d**) and oblique coronal reconstruction (**e**) depicts the left aberrant ICA abutting the cochlear promontory within the middle ear (red arrow)

During the 1980s, few authors described the radiologic criteria of aberrant ICA in CT scan and could be summarized as follow [8, 9, 27, 55]:

1. Posterior displacement of the petrous ICA
2. Enlargement of the Jacobson's canal (inferior tympanic canal)
3. Aplasia or hypoplasia of the vertical segment of the ICA into the petrous bone
4. Enhancing mass into the hypotympanum
5. Absence of the bone plate between the ICA and the middle ear and
6. Absence of the exocranial orifice of the carotid canal (an orifice does not exist without its contents)

Some of these signs are visible in the clinical case shown in Fig. 3

Intratympanic Flow of the ICA with Persistent Stapedial Artery

In rare cases, an intratympanic course of the ICA is associated with the persistence of the stapedial artery [40, 49, 56–59]. This association of vascular variants concerning both carotid and hyostapedial systems was described anatomically and angiographically [7, 9, 16, 42, 43]. In these cases, the ICA enters the skull base through an enlarged inferior tympanic canal (narrowing of the vessel on DSA), passes into the tympanic cavity to bend anteriorly and reaches its

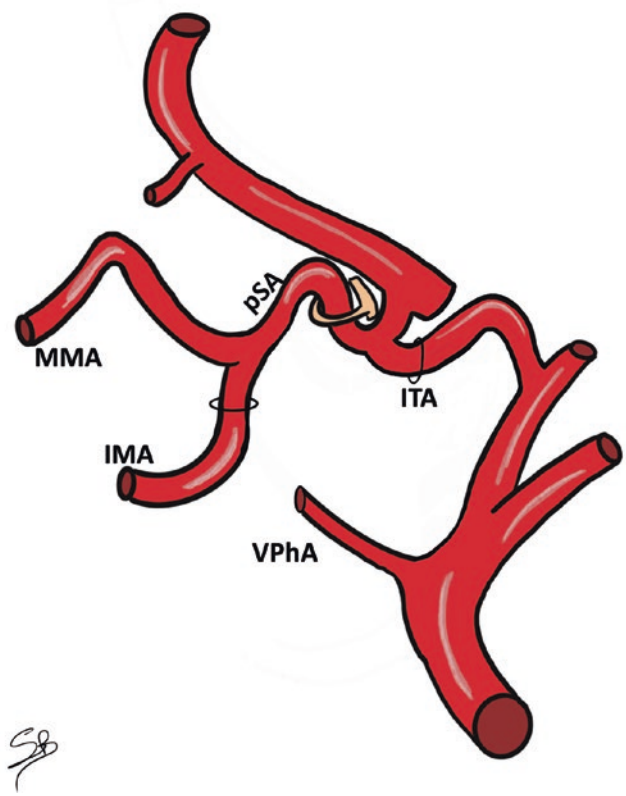

Fig. 4 Pseudo-petrous ICA + SA persistency. This variant is due to two phenomena: the agenesis of the cervical ICA and stapedial artery persistency. In this case the inferior tympanic artery (ITA) does not reach the carotico-tympanic artery (CTA) but the persistent SA (pSA)

normal carotid canal. The middle meningeal artery arises from the ICA in its tympanic segment and passes through the stapes to enter the skull cavity through the cochlear promontory [6, 31]. This variant is represented in Fig. 4.

Duplication (Fenestration) of the ICA

The agenesis of the first two segments of the ICA could be complete or partial. In some cases, the agenesis is complete and the rerouting flow through the tympanic cavity allows to maintain a normal flow into the distal ICA. In rare cases, the agenesis of the cervical and petrous ICA is only partial and consequently, the cervical and petrous ICA seems duplicated. The normal course of the ICA is generally hypoplastic in these cases [49, 60–66].

Table 2 Different vascular variations including an intratympanic flow of the ICA

Vascular variations	Number of cases	Clinical signs	Radiological signs
Intratympanic flow of the ICA	More than 100 2 bilateral cases	Pulsatile tinnitus Conductive hearing loss Vertigo Recurrent otitis media Pseudo-aneurysm	Posterior position ICA Enlarged Jacobson's canal Aplasia cervical and vertical petrous segments ICA Enhancing mass in the hypotympanum Absence of the exocranial carotid canal Absence bony plate ICA-middle ear
Intratympanic flow of the ICA + persistence of the stapedial artery	5–10	Pulsatile tinnitus Conductive hearing loss Vertigo Recurrent otitis media Pseudo-aneurysm	Radiological signs of aberrant flow of the ICA Absence or reduced size of the foramen spinosum
Duplication of cervical and petrous ICA	4	Asymptomatic	Duplicated ICA Enlarged Jacobson's canal Hypoplasia cervical and vertical petrous segments ICA
Duplication of cervical and petrous ICA + persistence of the stapedial artery	2	Recurrent otitis media Otosclerosis	Duplicated ICA Absence of the foramen spinosum
Duplication of cervical and petrous ICA with inferior tympanic-superior tympanic anastomosis	1	Pulsatile tinnitus	Duplicated ICA Absence of the foramen spinosum Ophthalmic artery origin of the middle meningeal artery

Particular Variations

Almost all cases of aberrant flow of the ICA are sporadic and unilateral (Table 2). Only three cases of bilateral aberrant flow of the ICA were presented in the English literature by Roll (2003), Chang (2010), and Toros (2010) [3, 40, 52].

Koenisberg et al. (1995) described a rare case of duplicated ICA associated with the persistence of the stapedial artery on the same side [49]. This is an interesting case that highlights the embryological link between the carotid and hyostapedial systems in the formation of the middle ear vascularization.

The most surprising case was presented by Willinsky et al. (1990) and consists of an anastomosis between the inferior tympanic artery (branch of the ascending pharyngeal artery) and the superior tympanic artery (stapedial artery) [55]. Consequently, the patient presented a duplicated ICA with anastomosis between the two ICAs in the horizontal petrous segment instead of the vertical petrous segment. Additionally, the middle meningeal artery arose from the ophthalmic artery instead of the external carotid artery. This case is similar to the two cases of a pharyngo-tympano-stapedial variant presented by P.Lasjaunias (1977) and by Baltsavias (2012) that presented a middle meningeal artery origin from the ascending pharyngeal artery [7, 35]. The difference between the case of Willinsky and these two cases is the presence or not of an aberrant flow of the ICA [55].

We could also find in the literature few cases of aberrant course of the ICA associated with a cervical ICA origin of the occipital artery [14, 28, 67, 68]. In fact, knowing that in case of aberrant course of the ICA, the cervical segment of the ICA does not exist, the occipital artery arises from the ascending pharyngeal system. This anatomical variation is easy to understand because the occipital and ascending pharyngeal arteries have the same embryological development [6].

Clinical Implications

A lot of cases of aberrant ICA were discovered during a surgery of the middle ear. The lack of knowledge of this anatomical variation leads to catastrophic hemorrhagic complication [20, 69, 70].

This anatomical variation must be differentiated from other diagnosis of vascular mass in the middle ear (high jugular bulb or glomus tympanicum) [1, 18, 71].

Almost all authors agree in the absence of surgical treatment of aberrant flow of the ICA [10, 47]. Only Rugg and Reed (1972) were favorable for an aggressive and surgical treatment in case of symptomatology [72]. They described a surgical technique coverage of the promontory defect by temporal muscle fascia graft and argued the indication for the surgery by "avoiding cerebrovascular accident." Today, it is accepted by almost all experts that intratympanic course of the ICA is an anatomical variation but not a pathology. Only few cases necessitate a surgical treatment of a pathology in relation to an aberrant ICA [47, 53]. Winfuhr (2004) proposed a management protocol and advocated to consider a surgical or endovascular treatment only in case of intratympanic aneurysmal formation, injury during a middle ear surgery, or recurrent otitis media [10]. In case of pseudoaneurysm, the options are the trapping of the vessel with surgical bypass, the stenting of carotid artery, or the endovascular occlusion of the vessel [10, 47, 53].

References

1. Anagiotos A, Kazantzi M, Tapis M. Aberrant internal carotid artery in the middle ear: the duplication variant. BMJ Case Rep. 2019;12(4):e228865. https://doi.org/10.1136/bcr-2018-228865.
2. Botma M, Kell RA, Bhattacharya J, Crowther JA. Aberrant internal carotid artery in the middle-ear space. J Laryngol Otol. 2000;114(10):784–7. https://doi.org/10.1258/0022215001903924.
3. Chang CY. Bilateral aberrant internal carotid artery. Ear Nose Throat J. 2010;89(3):114–5.
4. Desal HA, Freund P, Auffray-Calvier E, Guillon B, Martin S, Esselimani L, et al. Current imaging of an "aberrant course" of the internal carotid artery. J Neuroradiol. 1998;25(2):147–50.
5. Lasjaunias P, Moret J. Normal and non-pathological variations in the angiographic aspects of the arteries of the middle ear. Neuroradiology. 1978;15(4):213–9. https://doi.org/10.1007/bf00327529.
6. Lasjaunias P, Berenstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer; 2001.
7. Lasjaunias P, Moret J, Manelfe C, Theron J, Hasso T, Seeger J. Arterial anomalies at the base of the skull. Neuroradiology. 1977;13(5):267–72. https://doi.org/10.1007/bf00347072.
8. McElveen JT Jr, Lo WW, el Gabri TH, Nigri P. Aberrant internal carotid artery: classic findings on computed tomography. Otolaryngol Head Neck Surg. 1986;94(5):616–21. https://doi.org/10.1177/019459988609400515.
9. Sauvaget E, Paris J, Kici S, Kania R, Guichard JP, Chapot R, et al. Aberrant internal carotid artery in the temporal bone: imaging findings and management. Arch Otolaryngol Head Neck Surg. 2006;132(1):86–91. https://doi.org/10.1001/archotol.132.1.86.
10. Windfuhr JP. Aberrant internal carotid artery in the middle ear. Ann Otol Rhinol Laryngol Suppl. 2004;192:1–16. https://doi.org/10.1177/00034894041130s301.
11. Weinberg A, Albers AE. Injury of an aberrant internal carotid artery after myringotomy. Pan Afr Med J. 2017;27:237. https://doi.org/10.11604/pamj.2017.27.237.9092.
12. Lahiani R, Mahfoudhi M. Aberrant course of the internal carotid artery. Pan Afr Med J. 2015;22:257. https://doi.org/10.11604/pamj.2015.22.257.8102.
13. Perla FM, Carbotta G, Di Nardo D, D'Avanzo M, Colaiacomo MC, Di Biasi C, et al. Agenesis of the internal carotid artery: a family pathology? G Chir. 2017;38(1):46–9. https://doi.org/10.11138/gchir/2017.38.1.046.
14. Sundick SA, Weaver M, Faries PL, Marin M. Aberrant origin of occipital artery proximal to internal carotid artery stenosis. J Vasc Surg. 2014;59(1):244. https://doi.org/10.1016/j.jvs.2013.06.089.
15. Shimizu S, Sasahara G, Iida Y, Shibuya M, Numata T. Aberrant internal carotid artery in the middle ear with a deficiency in the origin of the anterior cerebral artery: a case report. Auris Nasus Larynx. 2009;36(3):359–62. https://doi.org/10.1016/j.anl.2008.08.008.
16. Hatipoglu HG, Cetin MA, Yuksel E, Dere H. A case of a coexisting aberrant internal carotid artery and persistent stapedial artery: the role of MR angiography in the diagnosis. Ear Nose Throat J. 2011;90(5):E17–20. https://doi.org/10.1177/014556131109000513.
17. Picel AC, Davidson TM. An aberrant internal carotid artery discovered during evaluation of obstructive sleep apnea: a report of 2 cases with consideration of a possible association. Ear Nose Throat J. 2011;90(1):29–31. https://doi.org/10.1177/014556131109000109.

18. Lin YY, Wang CH, Liu SC, Chen HC. Aberrant internal carotid artery in the middle ear with dehiscent high jugular bulb. J Laryngol Otol. 2012;126(6):645–7. https://doi.org/10.1017/s0022215112000564.
19. Park CW, Jeon EJ. Pulsatile tinnitus arising from aberrant internal carotid artery at nasopharynx. Korean J Audiol. 2012;16(1):35–8. https://doi.org/10.7874/kja.2012.16.1.35.
20. Muderris T, Bercin S, Sevil E, Cetin H, Kiris M. A potentially catastrophic anatomical variation: aberrant internal carotid artery in the middle ear cavity. Case Rep Otolaryngol. 2013;2013:743021. https://doi.org/10.1155/2013/743021.
21. Stott CE, Kuroiwa MA, Carrasco FJ, Delano PH. Recurrent acute otitis media as the manifestation of an aberrant internal carotid artery. Otol Neurotol. 2013;34(8):e117–8. https://doi.org/10.1097/MAO.0b013e3182923eeb.
22. Nicolay S, De Foer B, Bernaerts A, Van Dinther J, Parizel PM. Aberrant internal carotid artery presenting as a retrotympanic vascular mass. Acta Radiol Short Rep. 2014;10:2047981614553695.
23. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol. 1948;212:205–62.
24. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
25. Steffen TN. Vascular anomalites of the middle ear. Laryngoscope. 1968;78(2):171–97. https://doi.org/10.1288/00005537-196802000-00001.
26. Lapayowker MS, Liebman EP, Ronis ML, Safer JN. Presentation of the internal carotid artery as a tumor of the middle ear. Radiology. 1971;98(2):293–7. https://doi.org/10.1148/98.2.293.
27. Shankar L, Mehta AL, Hawke M, Rutka J. High-resolution computed tomography of an aberrant internal carotid artery. J Otolaryngol. 1992;21(5):373–5.
28. Hachem K, Slaba S, Nassar J, Kanso H, Ashoush R, Ghossain M. Imaging of an aberrant occipital artery arising from the extracranial segment of the internal carotid artery. J Mal Vasc. 2004;29(4):205–9. https://doi.org/10.1016/s0398-0499(04)96749-2.
29. Glastonbury CM, Harnsberger HR, Hudgins PA, Salzman KL. Lateralized petrous internal carotid artery: imaging features and distinction from the aberrant internal carotid artery. Neuroradiology. 2012;54(9):1007–13. https://doi.org/10.1007/s00234-012-1034-8.
30. Schaumberg J, Tomandl B, Brunner F. Ultrasonographic, CTI and MRI of aberrant internal carotid artery: infrequent but important to know. Int J Angiol. 2016;5:e142–e4.
31. Lasjaunias P, Santoyo-Vazquez A. Segmental agenesis of the internal carotid artery: angiographic aspects with embryological discussion. Anat Clin. 1984;6(2):133–41. https://doi.org/10.1007/bf01773165.
32. Moret J, Delvert JC, Bretonneau CH, Lasjaunias P, de Bicetre CH. Vascularization of the ear: normal-variations-glomus tumors. J Neuroradiol. 1982;9(3):209–60.
33. Barry A. The aortic arch derivatives in human adult. Anat Rec. 1951;111(2):221–38. https://doi.org/10.1002/ar.1091110207.
34. Legait E. Le réseau admirable carotidien [The admirable carotid network]. Biol Med (Paris). 1947;36(9–10):139–65.
35. Baltsavias G, Kumar R, Valavanis A. The pharyngo-tympano-stapedial variant of the middle meningeal artery. A case report. Interv Neuroradiol. 2012;18(3):255–8. https://doi.org/10.1177/159101991201800302.
36. Gailloud P, Clatterbuck RE, Fasel JH, Tamargo RJ, Murphy KJ. Segmental agenesis of the internal carotid artery distal to the posterior communicating artery leading to the definition of a new embryologic segment. AJNR Am J Neuroradiol. 2004;25(7):1189–93.
37. Lo WW, Solti-Bohman LG, McElveen JT Jr. Aberrant carotid artery: radiologic diagnosis with emphasis on high-resolution computed tomography. Radiographics. 1985;5(6):985–93. https://doi.org/10.1148/radiographics.5.6.3880011.
38. Swartz JD, Bazarnic ML, Naidich TP, Lowry LD, Doan HT. Aberrant internal carotid artery lying within the middle ear. High resolution CT diagnosis and differential diagnosis. Neuroradiology. 1985;27(4):322–6. https://doi.org/10.1007/bf00339565.
39. Selesnick SH, Lessow AS. Aberrant internal carotid artery. Am J Otolaryngol. 1999;20(3):403–4.
40. Roll JD, Urban MA, Larson TC 3rd, Gailloud P, Jacob P, Harnsberger HR. Bilateral aberrant internal carotid arteries with bilateral persistent stapedial arteries and bilateral duplicated internal carotid arteries. AJNR Am J Neuroradiol. 2003;24(4):762–5.
41. Wei CJ, Chang FC, Chiou SY, Teng MM, Hu HH, Chung CP, et al. Aberrant ascending pharyngeal artery mimicking a partially occluded internal carotid artery. J Neuroimaging. 2004;14(1):67–70.
42. Saini J, Kesavadas C, Thomas B, Arvinda HR. Aberrant petrous internal carotid artery with cochlear anomaly-an unusual association. Surg Radiol Anat. 2008;30(5):453–7. https://doi.org/10.1007/s00276-008-0341-6.
43. Celebi I, Oz A, Yildirim H, Bankeroglu H, Basak M. A case of an aberrant internal carotid artery with a persistent stapedial artery: association of hypoplasia of the A1 segment of the anterior cerebral artery. Surg Radiol Anat. 2012;34(7):665–70. https://doi.org/10.1007/s00276-011-0912-9.
44. Yanmaz R, Okuyucu S, Burakgazi G, Bayarogullari H. Aberrant internal carotid artery in the tympanic cavity. J Craniofac Surg. 2016;27(8):2001–3. https://doi.org/10.1097/scs.0000000000003080.
45. Koizuka I, Hattori K, Tsutsumi K, Sakuma A, Katsumi N, Kikuchi H, et al. Objective tinnitus caused by an aberrant internal carotid artery. Auris Nasus Larynx. 1998;25(3):323–7. https://doi.org/10.1016/s0385-8146(98)00016-9.
46. Endo K, Maruyama Y, Tsukatani T, Furukawa M. Aberrant internal carotid artery as a cause of objective pulsatile tinnitus. Auris Nasus Larynx. 2006;33(4):447–50. https://doi.org/10.1016/j.anl.2006.03.007.
47. Song YS, Yuan YY, Wang GJ, Dai P, Han DY. Aberrant internal carotid artery causing objective pulsatile tinnitus and conductive hearing loss. Acta Otolaryngol. 2012;132(10):1126–30. https://doi.org/10.3109/00016489.2012.684400.
48. Honkura Y, Hidaka H, Ohta J, Gorai S, Katori Y, Kobayashi T. Surgical treatment for the aberrant internal carotid artery in the middle ear with pulsatile tinnitus. Auris Nasus Larynx. 2014;41(2):215–8. https://doi.org/10.1016/j.anl.2013.10.002.
49. Koenigsberg RA, Zito JL, Patel M, Swartz JD, Goldofsky E, Zahtz G. Fenestration of the internal carotid artery: a rare mass of the hypotympanum associated with persistence of the stapedial artery. AJNR Am J Neuroradiol. 1995;16(4 Suppl):908–10.
50. Cohen JE, Gomori JM, Leker RR. Internal carotid artery agenesis: diagnosis, clinical spectrum, associated conditions and its importance in the era of stroke interventions. Neurol Res. 2010;32(10):1027–32. https://doi.org/10.1179/016164110x12767786356273.
51. Okawa M, Higashi T, Komiyama M, Fukuda K, Abe H, Inoue T. Left internal carotid artery agenesis with transsellar collateral and a right aortic arch: case report. Interv Neuroradiol. 2015;21(6):759–64. https://doi.org/10.1177/1591019915609130.
52. Toros SZ, Karaca CT, Noseri H, Naiboglu B, Kalaycik C, Egeli E. Bilateral aberrant internal carotid arteries: a case report presenting with pulsatile middle ear discharge. Int J Pediatr Otorhinolaryngol. 2010;74(1):97–8. https://doi.org/10.1016/j.ijporl.2009.10.015.
53. Kawamura Y, Sayama T, Maehara N, Nishimura A, Iihara K. Ruptured aneurysm of an aberrant internal carotid artery suc-

cessfully treated with simultaneous intervention and surgery in a hybrid operating room. World Neurosurg. 2017;102:695.e1–5. https://doi.org/10.1016/j.wneu.2017.03.133.
54. Ottaviano G, Calzolari F, Martini A. Goldenhar syndrome in association with agenesia of the internal carotid artery. Int J Pediatr Otorhinolaryngol. 2007;71(3):509–12. https://doi.org/10.1016/j.ijporl.2006.11.003.
55. Willinsky RA, Ter Brugge K, Lawson V, Mirich D, Lasjaunias P. Aberrant carotid artery: recognition on high resolution computed tomography. J Otolaryngol. 1990;19(4):249–51.
56. Guinto FC Jr, Garrabrant EC, Radcliffe WB. Radiology of the persistent stapedial artery. Radiology. 1972;105(2):365–9. https://doi.org/10.1148/105.2.365.
57. Teal JS, Rumbaugh CL, Bergeron RT, Segall HD. Congenital absence of the internal carotid artery associated with cerebral hemiatrophy, absence of the external carotid artery, and persistence of the stapedial artery. Am J Roentgenol Radium Ther Nucl Med. 1973;118(3):534–45. https://doi.org/10.2214/ajr.118.3.534.
58. Silbergleit R, Quint DJ, Mehta BA, Patel SC, Metes JJ, Noujaim SE. The persistent stapedial artery. AJNR Am J Neuroradiol. 2000;21(3):572–7.
59. Lau CC, Oghalai JS, Jackler RK. Combination of aberrant internal carotid artery and persistent stapedial artery. Otol Neurotol. 2004;25(5):850–1. https://doi.org/10.1097/00129492-200409000-00033.
60. Hasegawa T, Kashihara K, Ito H, Yamamoto S. Fenestration of the internal carotid artery. Surg Neurol. 1985;23(4):391–5. https://doi.org/10.1016/0090-3019(85)90214-9.
61. Chess MA, Barsotti JB, Chang JK, Ketonen LM, Westesson PL. Duplication of the extracranial internal carotid artery. AJNR Am J Neuroradiol. 1995;16(7):1545–7.
62. Fujimura M, Seki H, Sugawara T, Tomichi N, Oku T, Higuchi H. Anomalous internal carotid artery-anterior cerebral artery anastomosis associated with fenestration and cerebral aneurysm. Neurol Med Chir (Tokyo). 1996;36(4):229–33. https://doi.org/10.2176/nmc.36.229.
63. Mangla S, Teitelbaum GP. Multichannel fenestrations of the petrous segment of the internal carotid artery. AJNR Am J Neuroradiol. 1999;20(4):590–2.
64. Yim NY, Ha HI, Park JH, Moon YJ, Yoon W, Kim JK. Agenesis of bilateral internal carotid artery associated with basilar artery fenestration mimicking intra-arterial thrombus: a case report. Vasc Endovasc Surg. 2010;44(1):69–74. https://doi.org/10.1177/1538574409347393.
65. Hacein-Bey L, Raghavan N, Mukundan G, Sekhon AK, Dodrill LK, Park TC. Fenestration of the petrous internal carotid artery with short segment duplication mimicking an arterial dissection. Clin Radiol. 2013;68(9):972–5. https://doi.org/10.1016/j.crad.2013.04.015.
66. Uchino A, Ehara T, Kurita H. Hypoplasia of the internal carotid artery with associated fenestration and extremely long P1 segment of the ipsilateral posterior cerebral artery diagnosed by MR angiography. Surg Radiol Anat. 2019;41(6):707–11. https://doi.org/10.1007/s00276-019-02212-z.
67. Aggarwal NR, Krishnamoorthy T, Devasia B, Menon G, Chandrasekhar K. Variant origin of superior thyroid artery, occipital artery and ascending pharyngeal artery from a common trunk from the cervical segment of internal carotid artery. Surg Radiol Anat. 2006;28(6):650–3. https://doi.org/10.1007/s00276-006-0145-5.
68. Uchino A, Saito N, Okano N, Kakehi Y. Aberrant internal carotid artery associated with occipital artery arising from the internal carotid artery. Surg Radiol Anat. 2015;37(9):1137–40. https://doi.org/10.1007/s00276-015-1451-6.
69. Ide M, Jimbo M, Yamamoto M, Hagiwara S. Posterior cerebral artery aneurysm associated with unilateral internal carotid artery agenesis–case report. Neurol Med Chir (Tokyo). 1995;35(11):825–9. https://doi.org/10.2176/nmc.35.825.
70. Prokopakis EP, Bourolias CA, Bizaki AJ, Karampekios SK, Velegrakis GA, Bizakis JG. Ectopic internal carotid artery presenting as an oropharyngeal mass. Head Face Med. 2008;4:20. https://doi.org/10.1186/1746-160x-4-20.
71. Blazic S, Milojevic M, Grgurevic U, Djeric D. Anatomical variations and relations of large blood vessels to the tympanic cavity. Surg Radiol Anat. 2015;37(4):327–31. https://doi.org/10.1007/s00276-014-1370-y.
72. Ruggles RL, Reed RC. Treatment of aberrant carotid arteries in the middle ear: a report of two cases. Laryngoscope. 1972;82(7):1199–205. https://doi.org/10.1288/00005537-197207000-00008.

Embryology and Anatomy of the Internal Maxillary Artery

Thomas Robert, Sara Bonasia, and Gabriele Cicciò'

The internal maxillary artery (IMA), also known as maxillary artery, is one of the two terminal branches of the external carotid artery (ECA) with the superficial temporal artery (STA) [1]. This is the largest branch of the ECA that has an important role in the supply of the facial structures. The first description of the IMA was given by Haller in 1745, and numerous authors described it as an artery with a high branching pattern variation [2–5]. The classical description of the IMA gives it not less than 14 branches that supply skin, mucosa, muscles, bones, and dura mater [3, 6, 7]. In this chapter, we will describe the anatomy of the internal maxillary artery with its variations.

Embryological Development

The detailed embryological formation of the IMA is not completely elucidated, but few elements described by Padget are important to understand the general development of the IMA [8]. In this chapter, we will limit to the important phases in the development of the IMA. The most important phases of the IMA development are summarized in Table 1.

Early Development of the IMA from the Internal Carotid Artery (ICA)

During the pre-padget embryological period (embryos <4–5 mm), the mandibular artery which represents the first aortic arch is visible and regresses rapidly during the stage I of Padget (4–5 mm) [8]. Even if all the development of this aortic arch is not known, the mandibular artery is the precursor of the Vidian artery, branch of the IMA [9].

Between the stage II and V of Padget (6–18 mm), the second aortic arch has an important development with the hyo-stapedial system [8, 9]. This artery presents two distinct branches: the supraorbital and the maxillomandibular. The first one is the precursor of most of the ophthalmic artery branches and middle meningeal artery (MMA), while the second one is the precursor of the IMA branches [8].

Formation of the Ventral Pharyngeal Artery

During the stage II of Padget (5–6 mm), the ventral pharyngeal artery initiates its development from the aortic bulb and progresses until the stage VI of Padget. This artery is the precursor of most of the ECA branches.

Annexation of the Maxillomandibular Artery

During the stage VI of Padget, the ventral pharyngeal artery annexes the mandibulomaxillary branch of the stapedial artery to form the external carotid artery. Consequently, branches of the IMA are considered as vestiges of the stapedial artery and not of the ventral pharyngeal artery [9].

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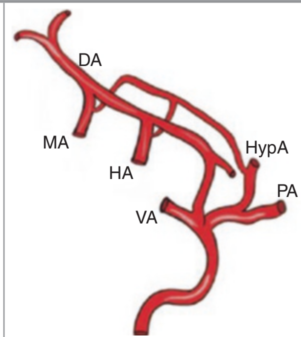
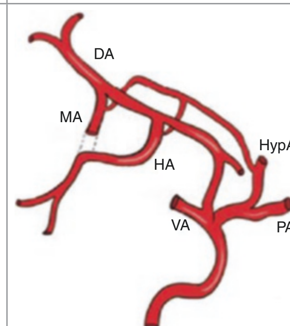
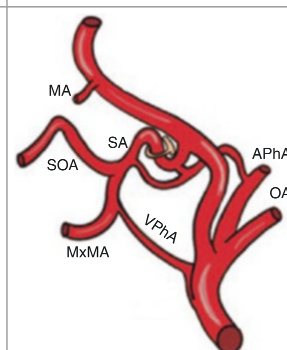
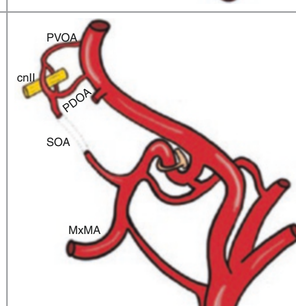
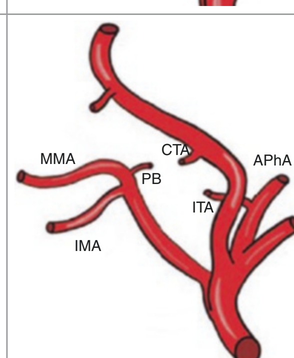
Table 1 Embryological development of the internal maxillary artery

Stage	Embryo size (mm)	Major evolutions	Graphic illustration
I	4–5	• Regression of the ventral part of the first and second aortic arches • Hyoid artery (HA) formation (dorsal remnant of the second aortic arch)	
II	5–6	• Elongation of the hyoid artery (HA) • Annexation of the mandibular artery (MA) territory (first aortic arch) by the hyoid artery (second aortic arch)	
III/IV	7–12 12–14	• Cranial growing of the hyoid artery (stapedial artery) passing into the middle ear (crus of the stapes) • Extension of the two branches of the stapedial artery: Supraorbital (SOA) and Maxillomandibular (MxMA)	
V	16–18	• Maximal development of the stapedial artery • Annexation of the supraorbital branch (SOA) by the ophthalmic artery (PDOA, PVOA) • Regression of the trans-osseous (superior orbital fissure) segment of the supraorbital branch	
VI	20–24	• Annexation of the maxillomandibular branch by the ventral pharyngeal artery • Regression of the intratympanic segment of the stapedial artery	

Regression of the Proximal Part of the Stapedial Artery

At stage VI of Padget (20–24 mm), the proximal portion of the stapedial artery regresses living its two branches supplied by the ophthalmic artery (for the supraorbital branch) and the external carotid artery (for the maxillomandibular branch) [9, 10]. The MMA becomes a branch of the IMA, and the connection between the two branches of the stapedial artery partially regresses to become the recurrent meningeal artery. The maxillomandibular branch passing through the foramen spinosum, it forms the middle meningeal artery and all branches of the internal maxillary artery [8].

Number and Origin of the Artery

The origin and the proximal trunk of the IMA do not present a lot of variations. As one of the terminal branches of the external carotid artery, it arises from the ECA in almost all cases [5, 7].

In case of complete persistence of the stapedial artery, the IMA arises from the petrous segment of the internal carotid artery reproducing the configuration of embryos at stage V of Padget (16–18 mm). Only two of such cases were described in the literature, and this variation is far rarer than the partial persistence of the stapedial artery [9, 11].

At its origin, the internal maxillary artery has an average diameter of 3.8 mm, its length is 42.8 mm [5, 7]. Duplication of the IMA at its origin was not described in the literature but an anatomical variation named "divided" IMA was published and consisted in the early division of the IMA in two distinct trunks, one superficial and the other deep along the second segment of the artery [12–14]. Different configurations were noted with the deep trunk larger or on the contrary, the superficial one larger. One of this case described by Claire et al. is particular by the reunion of the two trunks giving the anatomy of a fenestration of the second segment of the IMA encircling the pterygoid muscles [13].

Course of the Artery

The internal maxillary artery takes its origin in the retromandibular region where the ECA bifurcates in IMA and superficial temporal artery [6]. Three different segments of the IMA are described depending on the three anatomic regions crossed by the artery [5, 7]. The first one is the mandibular segment that extends from the origin of the artery to the lateral edge of the lateral pterygoid muscle. The second segment is called pterygoid, the artery is in the infratemporal fossa and extends from the lateral edge of the lateral pterygoid muscle to its entrance in the spheno-maxillary fissure. The distal segment is the pterygo-palatine one, the artery is in the fossa of the same name, and it corresponds to the part of the IMA distal to its entrance in the spheno-maxillary fissure [5, 7, 15, 16].

The major anatomical variation in the course of the IMA is its superficial or deep course in relation to the pterygoid muscles [2, 10, 17–22]. Even if a high ethnical variability is described, the superficial type of IMA is the most frequent with an incidence between 65% and 85% of cases [6, 23–27]. This major anatomical variation could be explained by the embryological development of the IMA. At an early stage, the internal maxillary artery is represented by an arterial plexus. The superficial or deep position of the IMA depends on which part of the plexus regresses during the embryological evolution. With the aim to include other minor variations of the IMA, few authors described a classification of the course of the IMA in six different types [23].

Branches of the Artery

The internal maxillary artery presents a high number of collateral branches usually described as 14 distinct branches, but a high variability of these branches makes difficult to identify each one of them [5, 7, 9, 10]. With the aim to give you a general organization of these branches, we will separate them by the IMA segment from where they arise.

Branches of the First Segment (Table 2)

- The **deep auricular artery** is the first branch of the IMA, its average diameter is 1.0 mm. It could have a common origin with the anterior tympanic artery and supplies the external acoustic meatus.
- The **anterior tympanic artery** has a diameter of 0.7 mm and enters the middle ear to participate to its arterial supply with branches of the ascending pharyngeal artery and internal carotid artery.
- The **middle meningeal artery** (MMA) is the largest branch of the IMA. It passes through the foramen spinosum to supply the dura of the convexity. Chapter "Anatomy, Embryology and Variations of the Middle Meningeal Artery" is entirely consecrated to this artery.
- The **accessory meningeal artery** (AccMA) arises from the IMA with a diameter of 1.9 mm. It has a vertical and cranial course to the skull base passing through the foramen ovale [28–31]. The accessory meningeal artery presents an important extracranial supply to the Eustachian tube, pterygoid muscles, and to the external acoustic meatus but also a dural supply in the parasellar region. The course of the AccMA depends on the course of the IMA and penetrates the cranial cavity through the canal of

Table 2 Branches of the first (mandibular) segment of the IMA

IMA branch	Mean diameter (mm)	Variations	Supply	Anastomoses
Deep auricular artery	1.0	Common origin with the anterior tympanic artery	External acoustic meatus	
Anterior tympanic artery	0.7	Common origin with the deep auricular artery	Middle ear (anterior part)	Stylomastoid artery (OccA) Inferior tympanic artery (APhA) Caroticotympanic artery (ICA)
Middle meningeal artery	2.5	Origin from the OA (most frequent)	Convexity dura Falx cerebri Temporal dura	Lacrimal artery (OA) Infero-lateral trunk (ICA) Anterior falcine artery (OA)
Accessory meningeal artery	1.7	Origin from the MMA	Eustachian tube Pterygoid muscles External acoustic meatus Parasellar dura Mandibular division (V3)	Superior pharyngeal artery (APhA) Antero-lateral branch (ILT) Cavernous branch (MMA) Recurrent branch (OA)
Inferior alveolar artery	1.3	Double origin	Inferior alveolar rims Dental roots of mandibula	Submental artery (facial artery)

Vesalius (sphenoidal emissary foramen) when the IMA has a deep course [9, 29]. This artery is described in detail in the chapter "Dural Branches of the Internal Maxillary Artery."

- The **inferior alveolar artery** (or inferior dental artery) has an antero-inferior course along the mandibula to enter in the mandibular canal. The mean diameter of this branch is 1.3 mm, and it supplies the dental roots and the mandibular bone [5, 7]. It could have a double origin from the IMA or arise directly from the ECA in case of deep course of the IMA. The distal part of the inferior alveolar artery anastomoses with the submental artery (branch of the facial artery).

Branches of the Second Segment (Table 3)

- The **anterior deep temporal artery** supplies the deep part of the temporal muscle. It has a mean diameter of 1.4 mm and could share its origin with the inferior alveolar artery. Its distal part anastomoses with the lacrimal artery (branch of the ophthalmic artery) [7].
- The **posterior deep temporal artery** is a second branch to the deep part of the temporal muscle, its mean diameter is also 1.4 mm and anastomoses with muscular branches of the STA.
- The **pterygoid arteries** could have a number between 1 and 5 and are small branches to the pterygoid muscles [5].
- The **masseteric artery** is also a muscular branch with a diameter of 1 mm.
- The **buccal artery**, with a mean diameter of 1 mm, supplies the buccinator muscle and could share its origin with the infraorbital artery. It anastomoses distally with the jugal branch of the facial artery and with the superior masseteric artery (branch of the transverse facial artery).

Table 3 Branches of the second (pterygoid) segment of the IMA

IMA branch	Mean diameter (mm)	Variations	Supply	Anastomoses
Anterior deep temporal artery	1.4	Common origin with inferior alveolar artery	Temporal muscle (deep part)	Lacrimal artery (OA) Superficial temporal artery
Posterior deep temporal artery	1.4		Temporal muscle (deep part)	Superficial temporal artery
Pterygoid arteries		Number: 1–5	Pterygoid muscles	
Masseteric artery	1.0		Masseteric muscle	Jugal artery (facial artery)
Buccal artery	1.0	Rotundum	Buccinator muscles	Superior masseteric artery (transverse facial artery)

Branches of the Third Segment (Table 4)

- The **infraorbital artery** (diameter of 1.3 mm) has an anterior course in the inferior orbital fissure to enter in the infraorbital canal. It gives two branches: the superficial palpebral and the naso-orbital branches. Distally, it anastomoses with branches of the ophthalmic artery and of the facial artery [9, 10].

Table 4 Branches of the third (pterygo-palatine) segment of the IMA

IMA branch	Mean diameter (mm)	Variations	Supply	Anastomoses
Infraorbital artery	1.3		Maxilla Orbital contents (inferior part)	Lacrimal artery (OA) Facial artery
Superior alveolar artery	1.3	Common origin with infraorbital artery	Upper alveolar rim Dental roots of maxilla	
Descending palatine artery	0.6		Upper teeth	Posterior ethmoidal artery (OA) Ascending palatine artery (APhA)
Vidian artery	0.7		Pterygoid canal Vidian nerve	Petrous ICA
Pharyngeal branches	NA		Pharynx mucosa	Superior pharyngeal artery (APhA)
Artery of the foramen rotundum	NA		Middle cranial fossa dura	Antero-lateral branch (ILT)
Pterygo-vaginal artery	NA		Eustachian tube	Accessory meningeal artery Ascending pharyngeal artery
Spheno-palatine artery	1.7		Hard palate Nasal septum Turbinates Nasal mucosa	Posterior ethmoidal artery (OA) Contralateral counterpart

- The **superior alveolar artery** supplies the upper alveolar rim and the dental roots of the maxilla. It has a mean diameter of 1.3 mm and could have a common origin with the infraorbital artery.
- The **descending palatine artery** is a little branch (0.6 mm) also called the greater palatine artery. It follows the upper alveolar rim until the incisive canal and anastomoses with the major artery of the soft palate (branch of the posterior ethmoidal artery).
- The **artery of the pterygoid canal or Vidian artery** is a little but important artery (0.7 mm) because it represents a direct anastomosis between the distal IMA and the petrous ICA. It has a posterior course to the pterygoid canal to anastomose with the Vidian artery of the ICA (vestige of the first aortic arch or mandibular artery) [32, 33].
- **Pharyngeal branches** of the IMA are little branches that supply the mucosa of the pharynx and anastomose with branches of the ascending pharyngeal artery.
- The **artery of the foramen rotundum** is a little dural branch of the IMA that enters the cranial cavity through the foramen of the same name, supplies the dura of the temporal fossa, and anastomoses with the antero-lateral branch of the infero-lateral trunk (cavernous ICA).
- The **pterygo-vaginal artery** supplies the Eustachian tube and anastomoses with the AccMA and with branches of the ascending pharyngeal artery.
- The **spheno-palatine artery** is considered as the terminal branch of the IMA and is the largest one (mean diameter of 1.7 mm). It presents two distinct branches, a medial one to the septum and a lateral one to the turbinates [5, 7]. It anastomoses with numerous branches of the facial and ethmoidal arteries. The septal branches also anastomose with their contralateral counterpart. Lasjaunias et al. described a “theorical” anastomosis with the embryologic olfactory artery [10].

The branches of the IMA are illustrated in Fig. 1.

Fig. 1 Branches of the internal maxillary artery. (1) Middle meningeal artery; (2) accessory meningeal artery; (3) middle deep temporal artery; (4) inferior alveolar artery; (5) pterygoid artery; (6) masseteric artery; (7) buccal artery; (8) artery of foramen rotundum; (9) sphenopalatine artery; (10) posterior superior (dental) alveolar artery; (11) Vidian artery; (12) anterior deep temporal artery; (13) infraorbital artery

Possible Anastomoses

The IMA is one of the principal natural collaterals between ECA and ICA that has a positive effect for example in case of proximal ICA stenosis, but it also allows the migration of embolic agent from the IMA to the ICA branches [34, 35]. We could divide the different IMA anastomoses in two different zones: the proximal region (Eustachian tube region) and the distal one (pterygo-maxillary region) [9, 10, 36]. Other than arterio-arterial anastomosis with branches of ICA and ECA branches, medial branches of the IMA (septal, pharyngeal, and infraorbital) have anastomoses with their contralateral counterpart [7].

Eustachian Tube Region

Major anastomoses of the IMA in this region are by the AccMA and the descending palatine artery that anastomose respectively to the superior pharyngeal artery and to the ascending palatine artery (APhA) [5, 7]. The anterior tympanic artery also presents in the middle ear anastomoses with the stylomastoid artery (occipital artery), the inferior tympanic artery (branch of the ascending pharyngeal artery), and with the carotico-tympanic artery (from the ICA) [9, 22].

Pterygo-maxillary Region

Most of anastomoses of the IMA are from its branches of the third segment with branches of the internal and external carotid arteries but also through intra-muscular anastomosis [7]. The infraorbital artery anastomoses with branches of the lacrimal artery (ophthalmic artery) and with the facial artery [10]. The anterior deep temporal artery also anastomoses with the lacrimal artery and with branches of the superficial temporal artery [29]. From its muscular branches (buccal and masseteric arteries), the IMA anastomoses with the transverse facial (superior masseteric branch) and facial (jugal branch) arteries [5]. The descending palatine artery and the sphenopalatine artery present anastomosis to the posterior ethmoidal artery [7]. The most direct anastomosis between the IMA and the ICA is the Vidian artery which connects the distal IMA with the petrous segment of the ICA [32, 33]. In the pharyngeal mucosa, pharyngeal branches from the IMA anastomose with the superior pharyngeal branch of the ascending pharyngeal artery. The inferior alveolar artery anastomoses with the submental artery (facial artery). The pterygo-vaginal artery anastomoses with branches of the AccMA and with the ascending pharyngeal artery [5, 7, 17].

Meningeal Anastomoses

The IMA presents three distinct meningeal branches: The middle meningeal artery, the accessory meningeal artery, and the artery of the foramen rotundum [28–30]. Meningeal anastomoses with other dural branches are numerous and could be found in detail in the dedicated chapters "Anatomy, Embryology and Variations of the Middle Meningeal Artery" and "Dural Branches of the Internal Maxillary Artery." The MMA is the most prominent dural artery. Its major anastomoses are with branches of the meningo-hypophyseal trunk, infero-lateral trunk, ascending pharyngeal artery, ophthalmic artery, and anterior falcine artery [9]. In the chapter "Anatomy, Embryology and Variations of the Middle Meningeal Artery," these anastomoses are described in detail. The AccMA has anastomoses with the cavernous branch of the MMA, with the posterior branch of the infero-lateral trunk (ICA) and with the recurrent branch of the ophthalmic artery [28, 29, 37]. The artery of the foramen rotundum anastomoses with the antero-lateral branch of the infero-lateral trunk (ICA) [10].

Supplied Territory

The IMA has an important role in the supply of face and is interested in the supply of all tissue types [7, 16]. With the aim to give you a comprehensive overview, we classify the territory of supply by tissue.

Mucosal Supply

This is the most important role of the IMA. It supplies the lateral part of the pharyngeal mucosa, the Eustachian tube, the mucosa of the middle ear (anterior part), the external acoustic meatus, the soft palate, and the mucosa of the cheek. Branches involved in this function are the Vidian artery, the anterior tympanic artery, the deep auricular artery, the AccMA, and the buccal artery [7].

Muscular Supply

Muscular branches of the IMA arise from its second segment and supply the pterygoid muscles (pterygoid arteries), the temporal muscle (deep temporal arteries), the masseteric muscle (masseteric branch), and the buccinator muscles (buccal artery) [16].

Teeth and Bone Supply

The inferior and superior alveolar arteries respectively supply the inferior and superior teeth with the surrounding bone (mandibula and maxilla) [5, 7]. The infraorbital artery also participate in the supply of the maxilla. The hard palate, nasal septum, and turbinates are supplied by the sphenopalatine and superior alveolar arteries [9].

Meningeal Supply

Considering that the MMA is the largest meningeal artery, the IMA presents an important role in the dural supply. The MMA supplies the dura of the convexity, a part of the falx cerebri, the temporal fossa, and the lateral part of the tentorium [9]. The AccMA has a most limited territory to the lateral wall of the cavernous sinus and to the foramen ovale region. The artery of the foramen rotundum also has a very narrow dural territory around the foramen rotundum [28–30].

Peripheric Cranial Nerve Supply

The IMA participates in the supply of the trigeminal nerve, the AccMA gives little branches to the mandibular division (V3) extracranially and in the foramen ovale, and to the Gasserian ganglion in the cavum of Meckel [28]. The artery of the foramen rotundum gives similar branches to the maxillary division (V2). The inferior and superior alveolar arteries also supply peripheric branches of the trigeminal nerve [9].

Clinical Implications

A lot of physicians deal with the IMA during endovascular procedures or surgery, and the importance of this artery increased these last two decades with the evolution of the endoscopic ENT surgery and of the endovascular procedures [9, 34, 35, 38].

As we have seen before, the IMA presents a lot of arterio-arterial anastomoses with other branches of the ECA and with branches of the ICA. These anastomoses could be useful in case of proximal ICA stenosis for example [10]. On the other hand, they represent a dangerous source of complication in case of liquid embolic agents use in naso-pharyngeal pathologies [34]. Liquid agents' embolization is indicated for naso-pharyngeal tumors, facial AVM, or massive epistaxis. Anastomoses between the distal IMA and branches of the ICA could have catastrophic complication with retrograde migration of the liquid agent in the parenchymal branches of the ICA.

Endonasal endoscopic approach practically became the gold standard in the treatment of naso-pharyngal tumors or pituitary tumors. This approach, even if the mucosal resection is limited, has as complication the bleeding of the spheno-palatine artery and more precisely of its septal branch [20, 22, 39, 40]. A strong knowledge of the vascular anatomy of the IMA is important to limit such complications. The use of pediculated mucosal flap for the reconstruction also necessitates a good knowledge of IMA branches.

This last decade, few authors published rare cases of surgical bypass between the IMA and the middle cerebral artery. Due to its surgical technical difficulty, such surgeries are limited to expert hands and by its limited indication (giant cavernous internal carotid artery aneurysm or cavernous region tumors) [7, 41, 42].

References

1. Salamon G, Guerinel G, Demard F. Radioanatomical study of the external carotid artery. Ann Radiol (Paris). 1968;11(3):199–215.
2. Lasker GW, Opdyke DL, Miller H. The position of the internal maxillary artery and its questionable relation to the cephalic index. Anat Rec. 1951;109(1):119–26. https://doi.org/10.1002/ar.1091090109.
3. Doyon D, Markovits P, Aaron C, Simon J, Richard J, Gasquet V, et al. Radio-anatomic study of the external carotid. J Radiol Electrol Med Nucl. 1967;48(5):301–6.
4. Salamon G, Grisoli J, Paillas JE, Faure J, Giudicelli G. Arteriographic study of the meningeal arteries. (Role of selective injections, of subtraction and of radio-anatomical correlations). Neurochirurgia (Stuttg). 1967;10(1):1–19. https://doi.org/10.1055/s-0028-1095333.
5. Allen WE 3rd, Kier EL, Rothman SL. The maxillary artery: normal arteriographic anatomy. Am J Roentgenol Radium Ther Nucl Med. 1973;118(3):517–27. https://doi.org/10.2214/ajr.118.3.517.
6. Turvey TA, Fonseca RJ. The anatomy of the internal maxillary artery in the pterygopalatine fossa: its relationship to maxillary surgery. J Oral Surg. 1980;38(2):92–5.
7. Alvernia JE, Hidalgo J, Sindou MP, Washington C, Luzardo G, Perkins E, et al. The maxillary artery and its variants: an anatomical study with neurosurgical applications. Acta Neurochir. 2017;159(4):655–64. https://doi.org/10.1007/s00701-017-3092-5.
8. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol. 1948;212:205–62.
9. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer; 2001.
10. Lasjaunias P, Berges O, Doyon D. Collateral circulation of the internal maxillary artery. J Neuroradiol. 1979;6(3):197–205.
11. Rodesch G, Choi IS, Lasjaunias P. Complete persistence of the hyoido-stapedial artery in man. Case report. Intra-petrous origin of the maxillary artery from ICA. Surg Radiol Anat. 1991;13(1):63–5.
12. Aland RC, Shaw V. Divided maxillary artery in relation to the lateral pterygoid muscle. Anat Sci Int. 2016;91(2):207–10. https://doi.org/10.1007/s12565-015-0289-7.
13. Claire PG, Gibbs K, Hwang SH, Hill RV. Divided and reunited maxillary artery: developmental and clinical considerations. Anat Sci Int. 2011;86(4):232–6. https://doi.org/10.1007/s12565-011-0106-x.
14. Tadokoro O, Umemura Y, Utsuno H, Inoue K. A case of a divided maxillary artery in the infratemporal fossa. Okajimas Folia Anat Jpn. 2008;85(3):97–101. https://doi.org/10.2535/ofaj.85.97.
15. Abul-Hassan HS, von Drasek AG, Acland RD. Surgical anatomy and blood supply of the fascial layers of the temporal region. Plast Reconstr Surg. 1986;77(1):17–28.
16. Correa MB, Wafae GC, Pereira LA, Wafae N, Ruiz CR. Arterial branches to the temporal muscle. Ital J Anat Embryol. 2008;113(2):109–15.
17. Apinhasmit W, Methathrathip D, Ploytubtim S, Chompoopong S, Ariyawatkul T, Lertsirithong A. Anatomical study of the maxillary artery at the pterygomaxillary fissure in a Thai population: its relationship to maxillary osteotomy. J Med Assoc Thail. 2004;87(10):1212–7.
18. Hussain A, Binahmed A, Karim A, Sandor GK. Relationship of the maxillary artery and lateral pterygoid muscle in a caucasian sample. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2008;105(1):32–6. https://doi.org/10.1016/j.tripleo.2007.04.010.
19. Maeda S, Aizawa Y, Kumaki K, Kageyama I. Variations in the course of the maxillary artery in Japanese adults. Anat Sci Int. 2012;87(4):187–94. https://doi.org/10.1007/s12565-012-0146-x.
20. Nakajima H, Imanishi N, Minabe T. The arterial anatomy of the temporal region and the vascular basis of various temporal flaps. Br J Plast Surg. 1995;48(7):439–50. https://doi.org/10.1016/0007-1226(95)90118-3.
21. Navarro JA, Filho JL, Zorzetto NL. Anatomy of the maxillary artery into the pterygomaxillopalatine fossa. Anat Anz. 1982;152(5):413–33.
22. Orbay H, Kerem M, Unlu RE, Comert A, Tuccar E, Sensoz O. Maxillary artery: anatomical landmarks and relationship with the mandibular subcondyle. Plast Reconstr Surg. 2007;120(7):1865–70. https://doi.org/10.1097/01.prs.0000287137.72674.3c.
23. Rischmuller B, Meiring JH. Course and relationships of the arteria maxillaris. Acta Anat (Basel). 1991;142(1):77–80. https://doi.org/10.1159/000147164.
24. Sashi R, Tomura N, Hashimoto M, Kobayashi M, Watarai J. Angiographic anatomy of the first and second segments of the maxillary artery. Radiat Med. 1996;14(3):133–8.
25. Suwa F, Takemura A, Ehara Y, Takeda N, Masu M. On the arteria maxillaris which passes medial to the pterygoideus lateralis muscle of the Japanese–patterns of origin of the inferior alveolar, the masseteric and the posterior temporal arteries. Okajimas Folia Anat Jpn. 1990;67(5):303–8. https://doi.org/10.2535/ofaj1936.67.5_303.
26. Toure G. Distribution of the maxillary artery in the deep regions of the face and the maxilla: clinical applications. J Plast Reconstr Aesthet Surg. 2019;72(6):1020–4. https://doi.org/10.1016/j.bjps.2019.02.008.
27. Gulses A, Oren C, Altug HA, Ilica T, Sencimen M. Radiologic assessment of the relationship between the maxillary artery and the lateral pterygoid muscle. J Craniofac Surg. 2012;23(5):1465–7. https://doi.org/10.1097/SCS.0b013e3182575793.
28. Baumel JJ, Beard DY. The accessory meningeal artery of man. J Anat. 1961;95:386–402.
29. Dilenge D, Geraud G. Accessory meningeal artery. Acta Radiol Suppl. 1976;347:63–9. https://doi.org/10.1177/0284185175016s34709.
30. Lasjaunias P, Theron J. Radiographic anatomy of the accessory meningeal artery. Radiology. 1976;121(1):99–104. https://doi.org/10.1148/121.1.99.
31. Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A Jr. Microsurgical anatomy of the dural arteries. Neurosurgery. 2005;56(2 Suppl):211–51; discussion 51. https://doi.org/10.1227/01.neu.0000144823.94402.3d.
32. Osborn AG. The vidian artery: normal and pathologic anatomy. Radiology. 1980;136(2):373–8. https://doi.org/10.1148/radiology.136.2.7403513.
33. Takeuchi M, Kuwayama N, Kubo M, Umemura K, Hirashima Y, Endo S. Vidian artery as a collateral channel between the external and occluded internal carotid arteries–case report. Neurol Med Chir (Tokyo). 2005;45(9):470–1. https://doi.org/10.2176/nmc.45.470.
34. Geibprasert S, Pongpech S, Armstrong D, Krings T. Dangerous extracranial-intracranial anastomoses and supply to the cranial nerves: vessels the neurointerventionalist needs to know. AJNR Am J Neuroradiol. 2009;30(8):1459–68. https://doi.org/10.3174/ajnr.A1500.

35. Rinaldo L, Brinjikji W. Dangerous extracranial-intracranial anastomoses: what the Interventionalist must know. Semin Interv Radiol. 2020;37(2):140–9. https://doi.org/10.1055/s-0040-1709155.
36. Margolis MT, Newton TH. Collateral pathways between the cavernous portion of the internal carotid and external carotid arteries. Radiology. 1969;93(4):834–6. https://doi.org/10.1148/93.4.834.
37. Parkinson D. Collateral circulation of cavernous carotid artery: anatomy. Can J Surg. 1964;7:251–68.
38. Otake I, Kageyama I, Mataga I. Clinical anatomy of the maxillary artery. Okajimas Folia Anat Jpn. 2011;87(4):155–64. https://doi.org/10.2535/ofaj.87.155.
39. Morton AL, Khan A. Internal maxillary artery variability in the pterygopalatine fossa. Otolaryngol Head Neck Surg. 1991;104(2):204–9. https://doi.org/10.1177/019459989110400208.
40. Ottone NE, Sandoval C, Cid-Gutierrez P, Vasquez-Balboa ML, Tubbs RS, Fuentes R. Systematic review and meta-analysis of the anatomy of the maxillary artery using the anatomical quality assurance (AQUA) checklist. Surg Radiol Anat. 2021;43(11):1875–86. https://doi.org/10.1007/s00276-021-02825-3.
41. Akiyama O, Gungor A, Middlebrooks EH, Kondo A, Arai H. Microsurgical anatomy of the maxillary artery for extracranial-intracranial bypass in the pterygopalatine segment of the maxillary artery. Clin Anat. 2018;31(5):724–33. https://doi.org/10.1002/ca.22926.
42. Wang L, Cai L, Lu S, Qian H, Lawton MT, Shi X. The history and evolution of internal maxillary artery bypass. World Neurosurg. 2018;113:320–32. https://doi.org/10.1016/j.wneu.2018.02.158.

Embryology and Variations of the Ophthalmic Artery

Sara Bonasia, Michel W. Bojanowski, and Thomas Robert

The ophthalmic artery (OA) is a very fascinating artery that has been studied by various ophthalmologists, neurosurgeons, and more recently by interventional neuroradiologists. The complexity of its embryological development, which is not completely discovered, directly involved the primitive internal carotid artery (ICA) as well as the stapedial artery systems [1]. This complex embryological development explains the numerous variations of this artery in its origin, course, and branching. During the last decades, neuroradiologists have a particular attention to anastomoses between the OA and external carotid artery (ECA) branches because of their importance in case of liquid embolization of pathologies in the ECA territory [2].

History

The first orbital dissection was described at the end of the nineteenth century, in particular with the works of Meyer (1887) [3] who was the first anatomist who paid attention to the accurate description of the orbital vascularization. Based on macroscopic cadaveric dissection, this German anatomist already defined the "normal" anatomy and branching of the ophthalmic artery. Eighty years after this first generation of anatomists and ophthalmologists, Sohan Sing Hayreh, another ophthalmologist, realized in 1962 a fantastic work describing with high precision the origin [4], course [5], and branches [6] of the OA. His findings were based on dissections of 170 human orbits and were dispatched in three different publications [4–6]. In the same period, Kuru (1967) [7] proposed a comprehensive description of few variations of the orbital vascularization based on 100 cerebral angiographies. From the 1970s till today, neuroradiologists showed increased interests to the OA for its numerous anastomoses with branches of the ECA. In particular, Moret (1977) [8] described in detail the orbital supply from the middle meningeal artery in his medical thesis. Others authors proposed, as Vignaud (1974) [9], Lasjaunias (1978) [10], or Shimada (1995) [11], fantastic articles that are today considered as landmarks on this topic. The knowledge and understanding of the embryology of the OA are based, as for other craniofacial arteries, on the work of Padget (1948) [1] and on brilliant hypotheses formulated by Lasjaunias [10, 12] that explain possible anatomical and angiographic variations.

Embryology of the Orbital Arteries

The OA can be considered as the cranio-facial artery with the most fascinating and complex embryological development. This complexity is due to several factors. The first one, and the most important, is the lack of knowledge of various embryological steps, the second one is the implication of three different embryological systems: the primitive internal carotid artery, the stapedial artery, and the pharyngeal artery systems. The last but not least factor that complicates the understanding of the embryological development of the orbital arteries, is the high number of hypotheses formulated by various authors on this topic without scientific basis that could be misinterpreted as evidence-based information. Our knowledge about the embryological development of the cerebral vasculature is principally based on the collection of embryos at the Carnegie institute and on the writings of Streeter (1918) [13] and Padget (1948) [1]. Later, Lasjaunias

Expert comment by Torstein R. Meling.

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(2001) [12] proposed other theories regarding few changes in the development of the OA based on different angiographic variations of this artery. In this chapter, we will present first these two theories about the embryologic development of the OA. Then, divergent points of view will be addressed [14, 15].

Padget's Concept [1] (Table 1)

The embryological development of the ophthalmic artery is a series of development and regression of primitive arteries and could be divided in six stages. These six stages described by Padget [1] differ from the stages of the general embryological development also elaborated by the same author.

Stage I: In the early embryologic life (4–5 mm), the optic vesicles begin their differentiation in optic stalk and optic vesicle. The primitive maxillary artery, which originated from the future cavernous segment of the primitive carotid artery, sends a temporary branch to the base of the optic vesicle. The other major event of this stage is the formation of the first primitive ophthalmic artery from the primitive carotid artery: the primitive dorsal ophthalmic artery (PDOA) which takes its origin at the bifurcation of the primitive carotid artery in its caudal and cranial division (future posterior communicating segment).

Stage II: In the 9 mm embryos, the PDOA now invests the optic cup by a large plexus without clear direct supply to the lens. The primitive hyaloid artery (future central retinal artery) is also developing through plexiform channels. The

Table 1 Stages of embryological development of the OA (Padget's concept)

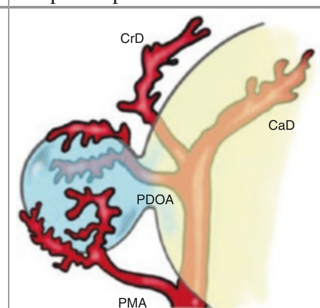
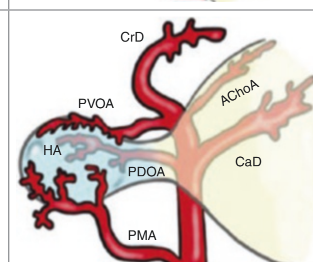
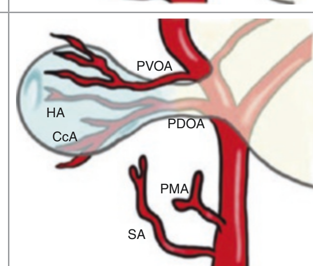
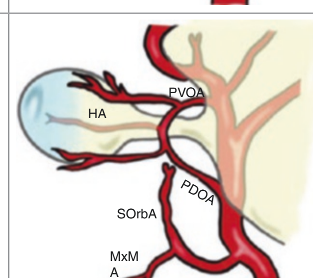
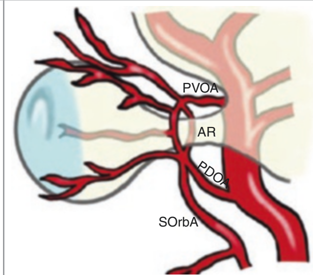
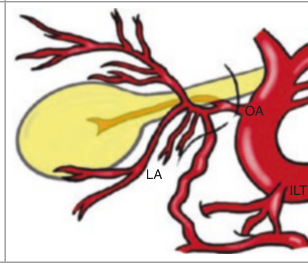
Stage	Embryo size (mm)	Events	Graphic representation
I	4–5	– Primitive maxillary artery (PMA) as temporary branch – PDOA appearance	
II	9	– Primitive hyaloid artery (HA) as plexiform channels – PVOA appearance	
III	14	– Formation of primitive hyaloid and common ciliary arteries – Stapedial artery (SA) development	
IV	18	– Migration of the PDOA origin – Formation of the supraorbital branch (SOrbA) of the SA – Regression of the PVOA	

Table 1 (continued)

Stage	Embryo size (mm)	Events	Graphic representation
V	20	– Maximal development of the SA – Formation of the anastomotic ring (AR)	
VI	40	– Ventral interruption of the AR – Regression of the SOrbA	

Adapted from Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–62

major change at this stage is the formation of another primitive ophthalmic artery from the primitive carotid artery: the primitive ventral ophthalmic artery (PVOA). This second primitive ophthalmic artery originates from the cranial division of the primitive carotid artery at the level of the anterior choroidal artery. At this stage, the PVOA supplies the cranial and mesial part of the plexus investing the optic cup.

Stage III: In the 14 mm embryos, both PDOA and PVOA are elongated, probably due to the ventral shifting of the optic cup and dorsal shifting of cerebral hemispheres. The PDOA develops two branches to the lens, which are the primitive hyaloid artery and the common temporal ciliary artery (future lateral posterior ciliary artery). The PVOA gives the common nasal ciliary artery (future medial posterior ciliary artery). On the other hand, the stapedial artery begins its development, and one of its collateral branches is captured by the ventral pharyngeal artery (future stem of the middle meningeal artery).

Stage IV: At approximately 18 mm, the elongated stem of the PDOA is gradually pulled along the primitive internal carotid artery, and its origin migrates to its adult position. Streeter (1918) [13] called this phenomenon "anastomotic progression" in reference to vessels of other organs. The hyaloid artery (branch of the PDOA) develops an anastomosis with the PVOA, which starts regressing. Concomitantly, the stapedial artery continues its progression and gives off two branches, the maxillomandibular artery that follows the second and third branches of the trigeminal nerve and anastomoses with the future external carotid artery, and the supraorbital artery. This supraorbital branch passes into the orbital cavity through the superior orbital fissure (following the first branch of the trigeminal nerve).

Stage V: In the 20 mm embryos, the supraorbital branch of the stapedial artery develops several orbital branches (ocular arteries are already formed from primitive ophthalmic arteries) and anastomoses with the primitive ophthalmic artery. An anastomotic ring is formed around the optic nerve by anastomosis between the supraorbital artery, the PDOA, and the PVOA.

Stage VI: At approximately 40 mm, the adult conformation of the ophthalmic artery could be recognizable after the ventral interruption of the anastomotic ring that forms the future second segment of the ophthalmic artery. The extraorbital part of the supraorbital artery also regresses and the lacrimal artery is naturally annexed by the primitive ophthalmic artery.

Lasjaunias' Concept [10, 12, 16, 17]

The theory of P. Lasjaunias is based on the knowledge of the tremendous work of Padget and on different angiographic variations he observed.

The embryogenesis of the primitive ophthalmic artery depends on two different arteries during the 4–18 mm stages: the PDOA and the PVOA. The PDOA develops from the cavernous segment of the primitive internal carotid artery and the PVOA from the anterior division of the primitive internal carotid artery. The PDOA penetrates the orbit through the superior orbital fissure and the PVOA through the optic canal.

Then, two major anastomoses between these two arteries are formed. The first one is an intraorbital plexiform anastomosis supplied by the two arteries around the optic nerve (future second segment of the ophthalmic artery). The second anastomosis is intradural between the PVOA and the primitive carotid artery to form the definitive supraclinoidal origin of the ophthalmic artery. The proximal portion of the PVOA (between its origin on the ACA and its anastomosis with the ICA) then regresses to give the adult stem of the ophthalmic artery.

In a following step, the proximal part of the PDOA regresses, and its remnant becomes the inferolateral trunk of the primitive carotid artery. Distally to the anastomotic ring around the optic nerve, the hyaloid artery and the lateral and medial posterior ciliary arteries develop as described before.

Concomitantly, between 18 and 40 mm stages, the stapedial artery arises from the primitive hyoid arch of the petrous carotid artery, giving rise to the maxillomandibular and to the supraorbital branches. The maxillomandibular artery leaves the cranial cavity through the foramen spinosum to anastomoses with the ventral pharyngeal artery. The supraorbital artery enters the orbit to give orbital branches. It divides into two different branches: the ethmoidonasal and the lacrimal arteries. The ethmoidonasal artery anastomoses with the primitive ophthalmic artery at the arterial ring around the optic nerve described before. Then, the transsphenoidal part of the supraorbital artery regresses, and its orbital branches are annexed by the primitive ophthalmic artery to give the adult conformation.

Divergence Between the Two Concepts (Table 2)

These major differences between the two different concepts could be summarized as follow.

The major divergence between the two concepts is the name of the primitive ophthalmic artery that persists and gives the definitive OA: this artery is named PDOA by Padget and PVOA by Lasjaunias. After giving a particular attention to the publication of Padget with analysis of the references she cited for a comparative anatomy between human embryos and rabbit anatomy, and in particular the publication of Mann (1928) [18], it seems that she misinterpreted Mann's writings. Indeed, because of a different angle that the eye is viewed, she named PDOA the PVOA [19]. Whatever is the name given to the artery, all authors agree that this artery enters the orbit through the optic canal and gives off the central retina artery.

The other important divergence between the two concepts concerns the embryogenesis of the inferolateral trunk. For Padget, it is the remnant of the primitive maxillary artery (premandibulary arch) after its partial regression. For Lasjaunias, the ILT is the remnant of the PDOA.

Another small difference is the mechanism to explain the "migration" of the definitive origin of the OA to the supraclinoidal segment of the ICA. Padget elegantly described the migration of the POA origin due to its elongation and to the cranial growing of the primitive carotid artery. On the other hand, Lasjaunias postulated for an intradural anastomosis between the POA and the primitive carotid artery, probably due to the osseous formation of the optic canal that brings the OA near to the primitive carotid artery. This different theory is represented in Fig. 1.

Table 2 Major divergences between the two embryological concepts

Differences	D. Padget	P. Lasjaunias
Definitive primitive ophthalmic artery	Primitive dorsal ophthalmic artery	Primitive ventral ophthalmic artery
Formation of the infero-lateral trunk	Primitive maxillary artery	Primitive dorsal ophthalmic artery
Formation of the proximal part of the definitive OA	Anastomotic progression	Intradural anastomosis PVOA-ICA

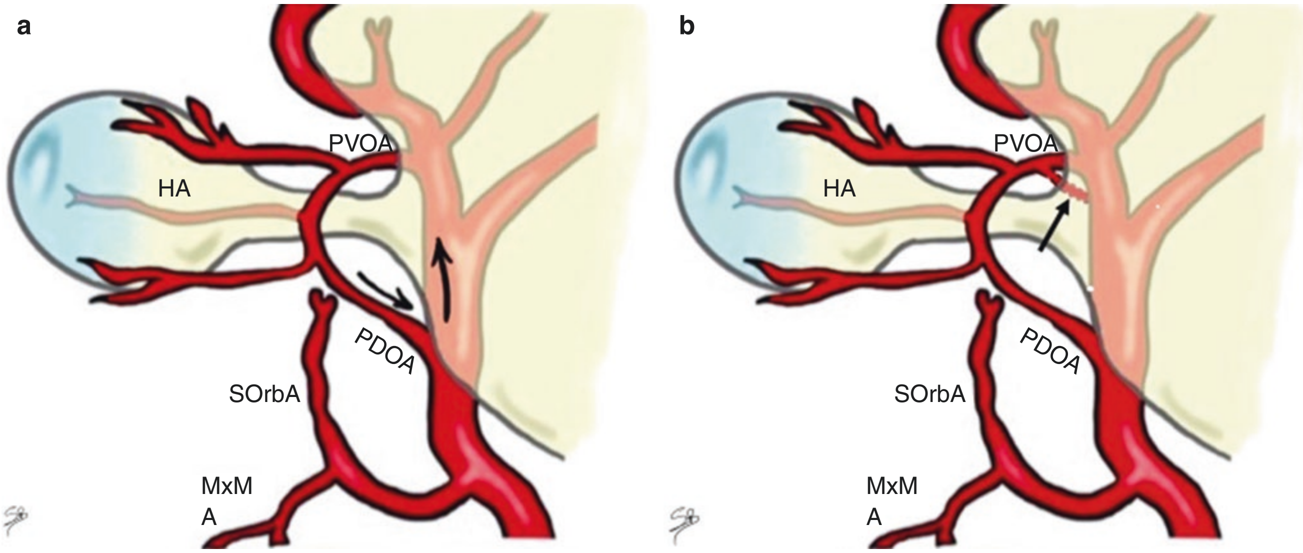


Fig. 1 Padget's and Lasjaunias' theories about OA origin migration. When the embryo is about 18 mm, the OA reaches its definitive origin on the supraclinoid ICA. This phenomenon is explained by Padget [1] by the cranial elongation of the ICA during this stage with the consequent movement of the PDOA origin (black arrows in **a**). On the other hand, Lasjaunias [12] hypothesized the presence of an intradural anastomosis between the PVOA and the primitive ICA (black arrow in **b**) in correspondence with the future origin with successive regression of the original stem

Atypical Origins of the Ophthalmic Artery (Table 3)

Normal Origin Variations

In more than 90% of cases [20, 21], the OA is the first branch of the intradural (C6 segment) internal carotid artery. Hayreh (1962) [4–6] furnished fantastic precisions about the different origins of the OA based on the dissection of 170 human orbits. He noted that the OA origin could vary from 3 mm proximal to 4.8 mm distal to the distal dural ring. Matsumura et al. (1999) [22] also paid a particular attention to the exact location of the origin of the OA and described a four-type classification (Fig. 2); type A when the OA origin is distal intradural over the cavernous sinus roof (4.7%); type B if the OA origin is always intradural but inside the cavernous sinus roof (54.7%); type C if the OA originates beneath the CS roof (26.6%); type D if the OA origin is intracavernous (14.1%). In few cases of intracavernous origin (3%), the OA borrows a separate bony canal to reach the optic canal (duplicate optic canal) [23]. Erdogmus et al. (2006) [24] confirmed that the intradural origin of the OA is the most frequent origin and found an extradural origin of the OA in only 5% of the orbits he dissected. Hayreh (1962) [4] described that the OA arose from the antero-medial aspect of the ICA anterior genu in most of cases (57%). The other possible positions of OA origin are the supero-medial (35%), the medial (7%) or the antero-superior (1%) aspects of the ICA.

Intracavernous Origin

In the cadaveric series of Hayreh (1962) [4], no case of intracavernous origin of the OA was observed. The intracavernous origin of the OA is a rare variation, and its incidence is estimated to 0.4% [25]. The first author who described this variation was Dilenge [26] in 1965 and after him, few case reports have been published [27–31]. Lasjaunias et al. (1977) [16] described a case of intracavernous origin and explained this anatomical variation by the embryological regression of the PVOA instead of the PDOA, supporting its theory about the embryogenesis of the OA [28, 30, 32–34]. On the other hand, authors that support the theory of Padget explain this anatomical variation by the persistence of an anastomosis between the first segment of the OA and the infero-lateral trunk (vestiges of the primitive maxillary artery) with consequent regression of stems from both PDOA and PVOA [25, 29, 35–37]. This anastomotic vessel is so-called deep recurrent OA [29]. Whatever theory you support and consequently what you call this anatomic variation (persistent PDOA or cavernous origin of the OA), this artery always arises from the lateral aspect of the horizontal segment of ICA (C3) and

Table 3 Correlation between embryological development and variations in origin of the OA

Variations in origin of the OA		Embryological implications	
Type	Incidence	Embryological explanation (Lasjaunias' concept) (Padget's concept)	Embryos size (mm)
Intracavernous origin	0.4%	Persistence of PDOA instead of PVOA Anastomosis ILT-recurrent meningeal artery	18
Double origin (from the ICA)	0.2%	Absence of anastomosis between the two primitive OA Persistence of primitive maxillary artery	20
MMA origin	1.4–2.5%	Persistence of the supraorbital branch of the stapedial artery	40
Choroidal and communicating segments of the ICA origin	5 cases	Incomplete intradural anastomosis PVOA-ICA	18
Anterior cerebral artery origin	6 cases	Lack of intradural anastomosis PVOA-ICA	18
Middle cerebral artery origin	2 cases	Lack of intradural anastomosis PVOA-ICA	18
Basilar artery origin	3 cases	Persistence of the supraorbital branch of the stapedial artery Persistent trigeminal artery	40
Infraoptic course of the A1 segment	0.1%	Persistence of proximal segment of the PVOA instead of the A1 segment	18

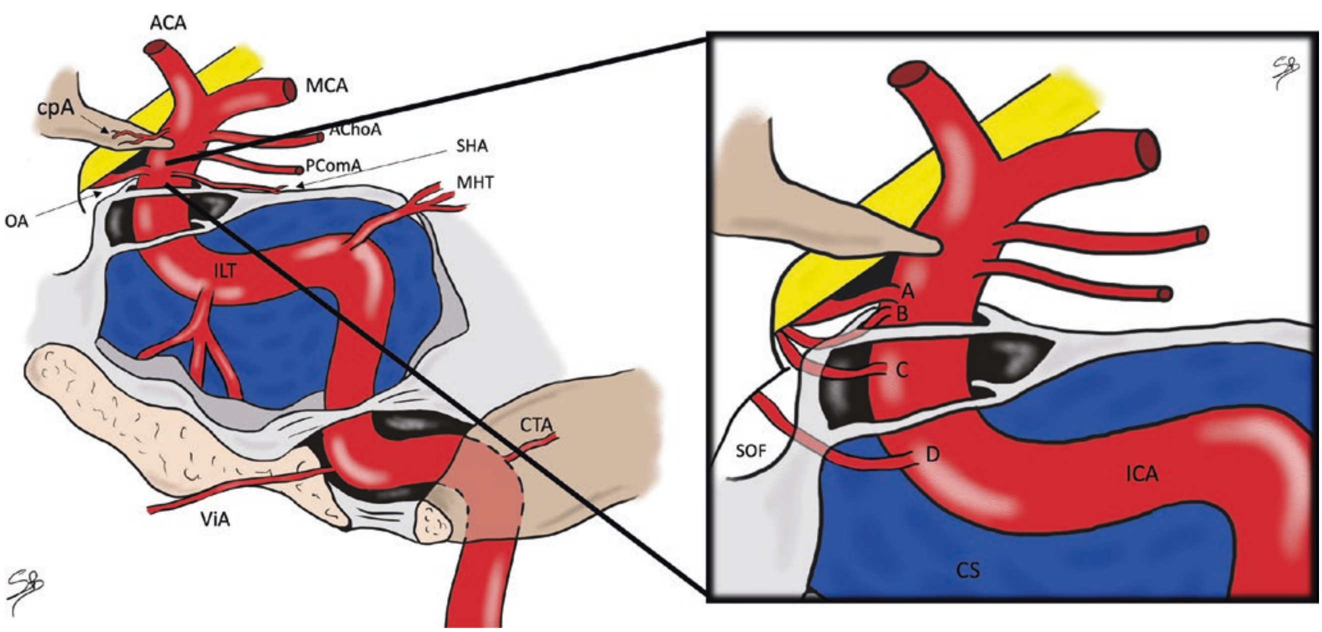


Fig. 2 OA origin from the clinoidal segment of the ICA and Matsumura's classification. (A) Distal intradural origin, (B) intradural origin but inside the cavernous sinus roof, (C) OA origin beneath the CS roof, (D) intracavernous origin. *ACA* anterior cerebral artery, *AchoA* anterior choroidal artery, *CS* cavernous sinus, *ICA* internal carotid artery, *MCA* middle cerebral artery, *OA* ophthalmic artery, *PComA* posterior communicating artery, *ILT* infero-lateral trunk, *ViA* vidian artery, *CTA* carotico-tympanic artery

penetrates the orbit through the medial part of the superior orbital fissure. A case of intracavernous origin of the OA is shown in Fig. 3.

Double Origin from the Internal Carotid Artery (Fig. 4a)

A double origin of the OA is a very rare variant, its incidence is estimated around 0.2% [25], and only few cases are reported in the literature [16, 25, 32, 37, 44–48]. The first case was described by Lasjaunias (1977) [16] who explained this anatomical variation by the lack of anastomotic ring around the optic nerve between the PDOA and the PVOA. Consequently, the PDOA does not regress. Based on Padget's concept, it is more difficult to explain this anatomical variant, but some authors postulated that the persistence of the primitive maxillary artery could explain the intracavernous branch followed by a normal development of the primitive OA [25, 37, 49]. In most of these rare cases, one of the two arteries arises from the supracavernous segment of the ICA, penetrates the orbit through the optic canal, and

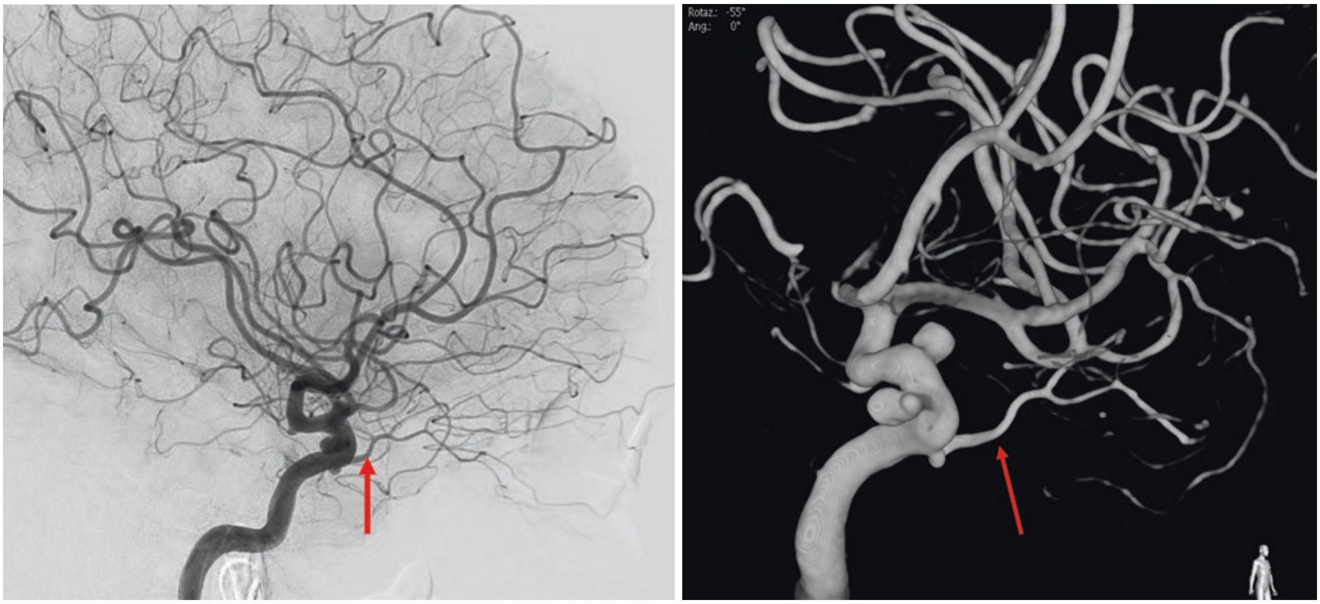


Fig. 3 Case of intracavernous origin of the OA. The figure shows the case of a patient who underwent DSA to study the cavernous ICA aneurysms incidentally discovered during an MRI. The lateral and 3D view show the origin of the OA (red arrows) from the cavernous segment of the ICA. This variant is explained by the persistency of the primitive dorsal ophthalmic artery instead of the primitive ventral OA as definitive ophthalmic artery. In this case the ophthalmic artery enters the orbit through the superior orbital fissure instead of the optic canal

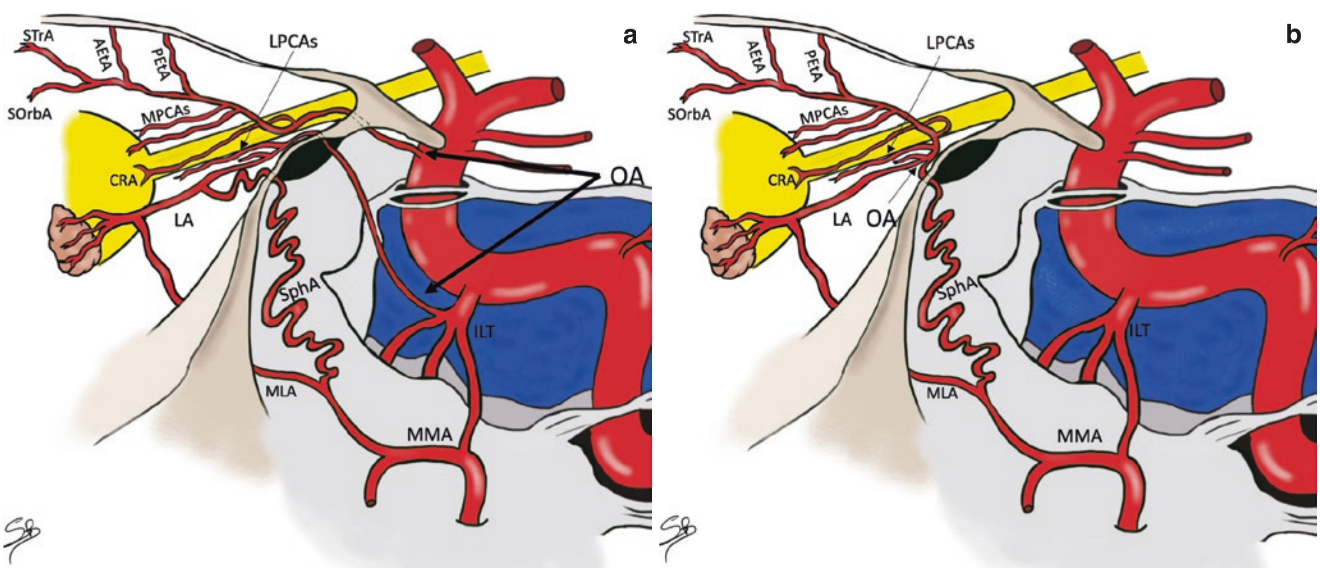


Fig. 4 Different possible origin of the OA. (**a**) Double origin from ICA of the OA. The first of the two arteries arise from the supracavernous segment of the ICA, penetrates the orbit through the optic canal and gives the posterior medial ciliary (PMCAs) and the central retinal artery (CRA). The second OA arises from the infero-lateral aspect of the cavernous segment of the ICA, passes through the superior orbital fissure (SOF) and gives off all the other orbital branches including the posterior lateral ciliary artery (PLCAs). (**b**) MMA origin of the OA. Orbital arteries origin from the anterior division of the MMA passing through the SOF or through the spheno-orbital foramen [26, 38–43]. The CRA can keep its origin from the supracavernous ICA but in few cases, also the central retinal artery is supplied by the MMA without ICA participation

gives the posterior medial ciliary and the central retinal arteries. The "second" OA arises from the infero-lateral aspect of the cavernous segment of the ICA, passes through the SOF, and gives off all the other orbital branches including the posterior lateral ciliary artery [12]. Only one case described by Uchino et al. (2013) [47] was different than others, because the intracavernous origin of one of the OAs arose from the posterior genu of the cavernous segment of the ICA (meningo-hypophyseal trunk rather than infero-lateral trunk). Authors postulated that other than the first anatomical variation (nonanastomosis between PDOA and PVOA), another one with an anastomosis between the infero-lateral and meningo-hypophyseal trunks, which consequently could explain a posterior origin of the intracavernous OA [47].

Middle Meningeal Artery Origin (Fig. 4b)

The first description of orbital branches arising from the middle meningeal artery (MMA) was published in 1872 by Curnow [50]. He noted in his case dissection that all orbital branches were supplied by the MMA except the central retinal artery, which kept its origin from the supracavernous ICA. Twenty years after this first description, Musgrove (1893) [51] presented another case of MMA origin of the OA where also the central retinal artery was supplied by the MMA, and the ICA did not give any orbital branch. After another isolated case found during cerebral or orbital dissections [52–55], Hayreh (1962) [4] reported 6 cases among his 170 orbital dissections where the MMA participated completely (2 cases) or partially (4 cases) in the orbital supply. Later, Moret (1977) [8] and Lasjaunias (1977) [16] described with precision the contribution of the MMA to the orbital vascularization and gave us a more comprehensive explanation of this anatomical variation.

The incidence of OA that arises from the MMA and penetrates the orbit through the SOF is estimated between 1.4 and 2.5% [25, 32, 36, 56, 57]. In this anatomical variation, orbital arteries are supplied by the anterior division of the MMA passing through the SOF or through the spheno-orbital foramen [26, 38–43]. The central retinal artery generally keeps its vascular supply from the supracavernous ICA, but in few cases, the central retinal artery is supplied by the MMA without ICA participation [8, 9, 58].

As explained before, during the embryogenesis, the stapedial artery gives the supraorbital branch which allows the development of the lacrimal artery and anastomoses with the OA around the optic nerve (participation of the peri-optic circle) [1, 12]. The proximal part of the supraorbital branch then normally regresses, and the lacrimal artery is annexed by the OA. The persistence of MMA branches (or, in extremis, MMA origin of the OA) could be explained by two different phenomena: the absence of anastomosis between the supraorbital branch and the OA, with consequent double supply of the orbital arteries, or the persistence of the proximal stem of the supraorbital branch of the stapedial artery with regression of the primitive OA (complete MMA origin of the OA) [8, 10, 12, 16, 17]. Although the supraorbital branch gives off its bifurcation (lacrimal artery laterally and ethmoido-nasal medially) usually inside the orbit, in 30% of cases it can divide outside the orbit in the middle cranial fossa. In such cases, the ethmoido-nasal artery passes through the SOF, but the lacrimal artery penetrates the orbit through its own canal: the spheno-orbital foramen [29] (canal of Hyrtl, lacrimal foramen, sinus canal) [12]. Depending on the author, these branches could be named differently. The lateral branch, passing through the canal of Hyrtl, is named meningolacrimal artery. The medial branch, passing through the SOF, is named recurrent meningeal artery or sphenoidal artery [25, 36, 37].

Atypical Origins from the Internal Carotid Artery

In rare cases, the migration of the origin of the primitive OA along the primitive ICA is not complete or the anastomosis between the PVOA and the ICA happens above the ophthalmic segment of the ICA [12, 32]. Consequently, the definitive OA arises from the supraclinoid segment of the ICA but from its choroidal [35, 48, 59] or communicating portions [36, 60].

Other Possible Origins of the OA

Our limited knowledge in embryogenesis of the OA does not allow to explain all other possible origins of the OA. Usually, these anatomical variations are associated with segmental anomalies: ICA agenesis [61] or persistence of other embryonic vessels [23]. Few cases of ACA [20, 48, 62–66] origin of the OA were described and are always explained by the nonmigration of the PVOA.

Two cases of MCA origin of the OA have been described and angiographically illustrated [32, 36, 52]. This rare variation could also be explained by the nonmigration of the PVOA remembering that the MCA is embryologically a branch of the anterior division of the primitive ICA appearing later than the normal migration of the PVOA.

Two other cases [32, 61] of posterior communicating artery (PComA) origin of the OA have been described but could not be explained by our knowledge of the embryogenesis and were concomitant with an ICA agenesis. It is not clear if the OA really arose from the PComA or from the supraclinoid segment of the ICA only perfused by the PComA.

Another three interesting cases of basilar artery origin of the OA have been angiographically demonstrated [67–69]. Even if it is difficult to explain this variant with the embryogenesis, authors postulated two interesting hypotheses. The first one is the concomitant persistence of the PDOA with a persistent trigeminal artery, consequently by multiple anastomoses the OA arises from the BA. The second hypothesis is the persistence of the stapedial artery with anastomosis between this latter and the BA and regression of the proximal segment of the stapedial artery.

Another anatomical variation that interests the embryogenesis of the OA is the intra optic course of the A1 segment that will be described in details in the chapter about the anterior cerebral artery [64], a variant which could be found when the A1 segment regresses instead of the proximal part of the PVOA [12].

In rare cases of agenesis of the ICA, two cases are described with the OA arising from the clinoidal segment of the contralateral ICA [70, 71].

Variations in the Course of the Ophthalmic Artery

The course of the OA could be divided in three different parts: intracranial, intracanalicular, and intraorbital portions. Meyer (1887) [3], as pioneer in the description of the OA presented it in three segments as illustrated in Fig. 5b.

After its origin from the ICA, the OA has a short intracranial course where the artery is in the subdural space in 85% of cases, between the two dura layers in 10% and completely extradural in 5% [4]. The OA generally stays on the inferior aspect of the optic nerve (infero-lateral: 70%, infero-central: 15%, and infero-medial: 15%) [24] to reach the optic canal.

In its intracanalicular course, the OA usually lies in the inferior part of the optic canal under the optic nerve. Hayreh and Dass (1962) [4] showed that the OA have an infero-lateral relationship to the optic nerve all along the optic canal in 64%. In other cases, the OA could have a more medial course, but in their cadaveric series, they noted that the OA was always inferior to the nerve.

The OA then penetrates the orbit, and its intraorbital part can also be separated in three segments [5, 72, 73]. The first one extends from its point of entrance in the orbit to the point where the artery bends to become the second part. In the first part, the OA runs along the inferolateral aspect of the optic nerve. The second part crosses over or under the optic nerve in a medial direction to reach the supero-medial aspect of the nerve. It corresponds to the second segment of the OA. The third part extends from the point where the artery bends another time to its terminal point.

The second segment of the artery is the most exposed to anatomical variations. Usually, the OA crosses cranial to the optic nerve in approximately 85% and caudal to the nerve in about 15% [4, 23]. This anatomical variation depends on which part of the primitive arterial ring persisted during the embryologic life [1, 12]. Hayreh (2006) described in details this embryonic arterial ring and the processes of partial regression, dividing it into four different segments among the four primitive arteries (stapedial artery, PVOA, PDOA, and the distal OA) [23]. Rare cases of complete persistence of the peri-optic ring have also been described [74, 75].

In its third segment, the OA runs medial to the optic nerve to reach the supero-medial angle of the orbital opening. After its bend (end of the second segment), the artery runs between the medial rectus and the superior oblique muscles to reach the medial wall of the orbit close to the anterior ethmoidal foramen. At the supero-medial angle of the orbit, the OA ter-

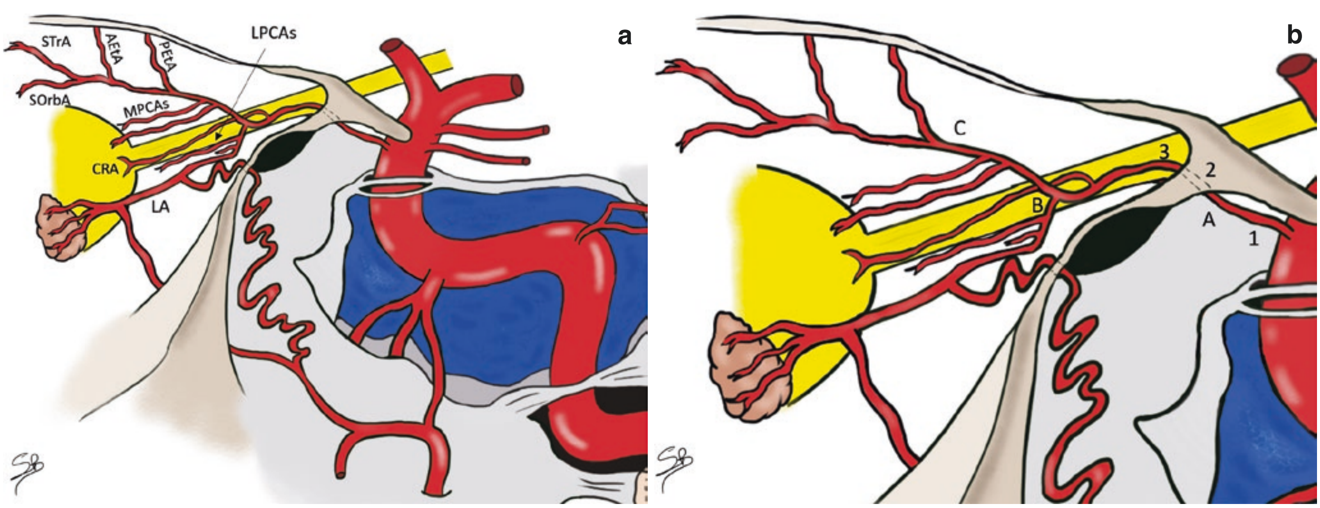


Fig. 5 (**a**) Course and branches of the OA. The central retinal artery (CRA) and the medial and lateral posterior ciliary arteries (MPCAs and LPCAs) are the first branches of the OA, which vascularize the ocular globe and its choroid. The lacrimal artery (LA) supplies the lacrimal gland (LG), the lateral part of the eyelid, the lateral rectus muscle, and inconstantly part of the dura of the middle cranial fossa. The anterior and posterior ethmoidal artery (AEtA and PEtA) supply the dura of the planum sphenoidale [6, 72, 73]. The supratrochlear (STrA) and supraorbital (SOrbA) arteries feed the peri-orbita, eyelids, and skin of the orbital region. *AccMA* accessory meningeal artery, *FRA* foramen rotundum artery, *ILT* inferolateral trunk, *MLA* meningolacrimal artery, *MMA* middle meningeal artery, *SphA* sphenoidal artery [12]. (**b**) OA segments classifications: intracranial (1), intracanalicular (2), and intraorbital (3). (A) From origin of the artery to the point where the artery bends around the nerve; (B) from the point where the OA crosses over or under the optic nerve in a medial direction to the supero-medial aspect of the nerve; (C) from the point where the artery bends another time to its terminal point

minates dividing into supra-trochlear and dorsal nasal arteries in 83% of cases [5]. In the other cases, the OA terminates at the level of the anterior ethmoidal foramen, dividing into anterior ethmoidal artery and another small branch that reach the supero-medial angle of the orbit [23].

Variations of Branches of the Ophthalmic Artery

Branches of the OA can be divided into ocular (central retinal and ciliary arteries), orbital (muscular and lacrimal arteries), and extraorbital groups and are summarized in Table 4 and in Fig. 5a. All branches arise from the second and the third segments of the OA. Branches destined to the ocular globe and also to the retina originate from the second segment of the artery [23]. Few variations of origin of these branches have been described. We will just cite the most common since this aspect goes beyond the purpose of this review.

The central retinal artery and the medial posterior ciliary artery, which are the first two branches of the OA, could have a common origin or distinct origins [6].

Whether the OA crosses cranial or caudal to the optic nerve in its second segment makes an evident difference in the order of its branches [6]. When it crosses under the optic nerve, the first is invariably the lateral posterior ciliary artery, the second one the central retinal artery, and the third is the medial posterior ciliary artery followed by the other branches. When the OA crosses over the optic nerve, the first branch is the central retinal artery, arising from a common trunk with the medial posterior ciliary artery, and the second one is the lateral posterior ciliary artery [23, 76].

The ocular globe and its choroid is vascularized by the central retinal artery and by the posterior ciliary arteries [6, 12]. The lacrimal artery supplies the lacrimal gland, the lateral part of the eyelid, the lateral rectus muscle and inconstantly gives off meningeal branches to supply the dura of the middle cranial fossa [6, 77]. The two main muscular branches (lateral and medial) supply the lateral and superior rectus muscles (lateral artery) and the inferior and medial rectus muscles (medial artery) [78]. The dura of the planum sphenoidale is supplied by the anterior and posterior ethmoidal artery, and the anterior third of the falx cerebri is also supplied by the anterior ethmoidal artery through the anterior falcine artery [6, 72, 73]. Other branches of the OA have

Table 4 Branches of the OA with corresponding territory

OA branches	Origin from the OA (segment)	Course	Branches	Territory
Central retinal artery	2nd	Central	2	Optic nerve Macula
Medial posterior ciliary artery	2nd	Central	1	Retina (medial part)
Lateral posterior ciliary artery	2nd	Central	1	Retina (lateral part)
Lacrimal artery	2nd	Lateral	3–4	Lacrimal gland Lateral rectus Dura of the SOF Eyelids (lateral part)
Lateral muscular artery	2nd	Lateral	3–6	Lateral rectus Superior rectus Superior oblique Levator palpebrae
Supraorbital artery	3rd	Superior	3–6	Levator palpebrae Orbital periosteum Upper eyelids
Posterior ethmoidal artery	3rd	Medial	1–4	Superior oblique Ethmoid air cells Dura (planum)
Medial muscular artery	3rd	Medial	3–6	Medial rectus Inferior rectus Inferior oblique
Anterior ethmoidal artery	3rd	Medial	2–5	Ethmoid air cells Medial rectus Dura (lamina cribrosa, falx)
Medial palpebral artery	3rd	Medial	4	Eyelids Lacrimal sac Naso-lacrimal duct
Frontal (supratrochlear) artery	3rd	Superior	1	Scalp (supraorbital)
Dorsal nasal artery	3rd	Superior	2	Scalp Forehead

principally a role in the vascularization of the peri-orbita, eyelids, and skin of the orbital region [12]. This superficial vascular supply is in balance with various branches of the external carotid artery [23].

Possible Anastomoses Between the Ophthalmic Artery and Branches of the External Carotid Artery

The first author who described an arterial anastomosis between the OA and branches of the ECA was Bossi (1955) [54] presenting angiographic cases of ICA occlusion.

The OA, that belongs to the internal carotid system, is a source of numerous anastomoses with branches of the external carotid artery [2, 79, 80]. These ECA-ICA natural anastomoses are important to know and are summarized in the Table 5.

The first one is already cited and well explained by the embryology. This is the anastomosis between the lacrimal artery and the anterior branch of the middle meningeal artery passing through the SOF or through the canal of Hyrtl [8, 81]. More distally, the lacrimal artery gives other anastomosis to deep temporal, transverse facial, orbitozygomatic, and infraorbital arteries [25].

The dorsal nasal artery also presents anastomoses with the facial artery via the angular artery or with the infraorbital artery [25, 54]. An anastomosis between the dorsal nasal artery of each side could be present in the midline [12].

The supraorbital, and more rarely the supratrochlear branches, anastomose with frontal branches of the superficial temporal artery [54].

Table 5 ECA-OA anastomoses

OA branch	ECA branch	Name of the anastomosis	Foramen
Lacrimal artery	Middle meningeal artery	Deep recurrent OA	SOF
Lacrimal artery	Middle meningeal artery	Meningo-ophthalmic artery	Hyrtl canal
Lacrimal artery	Anterior deep temporal artery	Inferior branch	Osseous canals
Lacrimal artery	Infraorbital artery	Inferior branch	Orbital opening
Dorsal nasal artery	Facial artery	Angular artery	Orbital opening
Supraorbital artery	Superficial temporal artery		Supraorbital foramen
Ethmoidal arteries	Sphenopalatine artery		Ethmoidal foramen

Clinical Implications

The knowledge of the anatomical variations and possible origins of the OA could be very important for both neuroradiologists and neurosurgeons in the management of the following pathologies.

Cribriform Plate dAVF

Usually, cribriform plate dAVFs are principally supplied by ethmoidal branches of the OA but also by MMA and IMA branches [82]. The anastomoses between the OA and the ECA previously described, well explain the possibility to visualize the fistula after an ECA injection even if there are not always feeders from the MMA or the IMA. An anastomosis between dorsal nasal artery of both sides could explain the frequent bilateral supply of the fistula. In case of surgical exclusion, the anatomy of the OA, including eventual variants should be known in order to identify the AV shunt and to avoid complications [83]. A few studies also show the possibility to manage this pathology with an endovascular approach [82, 84]. Knowledge of OA anatomy is mandatory to choose the best injection point (MMA or OA) and to limit the risk of arterial reflux into ocular branches [82].

Sphenoid Wing dAVF

For sphenoid wing dAVF, branches of the ILT, MMA, and of the IMA are usually the main arterial feeders [85, 86]. The OA, through its dural recurrent branches, could also play an important role [85]. A deep knowledge of the OA dural territories and of possible anastomoses between OA and MMA is necessary to choose the best approach (microsurgical exclusion or endovascular occlusion) and avoid complications. In case of transarterial embolization through the MMA, physicians should prevent the embolic agent reflux into OA branches, using coils, acrylics, or temporary balloon occlusion [85].

Carotid-Ophthalmic Aneurysm

The treatment of these aneurysms includes many possibilities, as surgical clipping and endovascular treatment with simple coiling or flow-diverter stenting [87]. In case of stenting, OA occlusion or embolism can occur [88]. Some studies [87–89] compared aneurysms occlusion rate, recurrence, and visual complications and reported no significant difference of amaurosis fugax and retinal embolism between simple coiling and flow-diverter stenting (FDS). However, it seems that flow diversion achieved a more stable sac thrombosis compared to other techniques [87]. The patho-mechanisms of visual field deficits could be related to small emboli outgoing from stent, to a modified blood flow in the OA after FDS placement, or to insufficient blood compensation from the ECA collaterals [89].

On the other hand, in case of surgical clipping of carotid-ophthalmic aneurysm, major attention has to be paid to possible variations in OA origin and course during the anterior clinoid process drilling. Before performing the anterior clinoidectomy, OA origin in relationship to the superficial and deep dural rings, eventual double OA, and other variations should be considered.

Olfactory Groove Meningioma

The blood supply of olfactory groove meningiomas mainly arises from ethmoidal branches of ophthalmic artery [90, 91]. Preoperative embolization to this supply has the risk of affecting the ocular branches of the OA and is therefore generally not performed. A preoperative embolization is usually performed through the MMA because of the lower risk of complications, even if some reports describe the possibility to work through superselective catheterization of the OA [91]. In this latter case, the most important rule is obviously to place the microcatheter tip as distal as possible [90]. The analysis of OA anatomy, origins, and eventual anatomic variations is also mandatory. In case of surgical approach, the first step of the resection is to coagulate the feeding vessels at the skull base in order to limit blood loss [91].

Refractory Epistaxis

Refractory or severe epistaxis is a good indication for embolization through branches of the internal maxillary artery [92]. Before performing this treatment, full analysis and knowledge of the anastomotic connections between the sphenopalatine artery and other branches (MMA, ethmoidal branches) is important to avoid uncontrolled embolization. Depending on the series, these complications occur in 0–2% of cases, and must be considered before the procedure [93]. Only in rare cases, ethmoidal arteries are the source of bleeding and the treatment of choice is usually surgical ligation [92].

Intra-arterial Chemotherapy for Retinoblastoma

The classical technique consists in the superselective catheterization of the OA with a microcatheter and subsequent chemotherapy injection [94]. However, direct catheterization of the OA is not always possible because of the small size of this artery in young children or anatomic variations, making it impossible to catheterize with the standard technique. Klufas et al. [95] described two alternative ways to reach the orbita: catheterization through the MMA (possible for its anastomosis with the OA, or because it gives sometimes the whole orbital vascularization) and balloon assisted injection.

In conclusion, knowledge of OA anatomy and the possible variations is mandatory for both neurosurgeons and neuroradiologists who approach the pathologies previously described. The analysis of the embryological steps that led to OA development is also very helpful to understand the possible variants in the adult configuration.

Appendix: Expert Comment by Torstein R. Meling

In this chapter, the authors make a *tour de force* presentation of the ophthalmic artery (OA), which is both an important and an intriguing artery.

The OA is an important artery because it contributes to the blood supply of the optic apparatus, i.e. the optic nerve and the retina, via its ocular branches, and to the optic adnexae, e.g. the extra-ocular muscles, lacrimal glands, and eyelids, via its orbital and extra-orbital branches. Secondly, the OA is intriguing because of the considerable variations in its anatomy, such as anomalous origins of the OA, as well as various anastomoses between the internal carotid artery (ICA) and external carotid artery (ECA) systems via the OA. The authors present the complex OA embryology that can explain these anatomical variations.

From our microsurgical anatomy books, we know that the OA is the first major branch of the ICA, that it usually originates just above the distal dural ring, and that enters the orbit via the optic canal, running infer-lateral to the optic nerve (ON). However, several important anomalous origins of the OA have been reported in the literature. The most important variant is an MMA origin, in which the OA originates from the MMA to reach the orbit via the superior orbital fissure (SOF) or a foramen in the greater wing of the sphenoid bone. In most such cases, the OA typically has double origins, i.e. with one branch arising from the MMA, which is an ECA branch, and the second branch from the ICA. However, a variant with the OA being supplied solely by the MMA has also been described. The second most common variant is an OA arising from the cavernous segment of the ICA to reach the orbit via the SOF.

The central retinal artery, lateral posterior ciliary artery, and medial posterior ciliary artery are the three ocular branches of the OA that emerge from the intraorbital segment of the OA and contribute to the blood supply of the optic apparatus. Thus, injury to the OA, whether traumatic or caused by iatrogenic factors, may have serious neuro-ophthalmological repercussions.

Furthermore, the OA has several orbital and extra-orbital branches, with no regular pattern because of significant inter-individual variations. These include the lacrimal artery, muscular arteries, the posterior and anterior ethmoidal arteries, the supraorbital artery, the medial palpebral artery, the dorsal nasal artery, and the frontal artery. There are numerous potential EC-IC anastomoses between the distal orbital and extra-orbital branches of the OA and the branches of the maxillary artery of the ECA. Examples include: (1) between medial and lateral muscular branches of the OA and the infraorbital artery at the level of the orbital floor; (2) between the anterior and posterior ethmoidal arteries and the septal branch of the spheno-palatine artery, (3) between the lacrimal artery branch the of the OA and the anterior division of the MMA, and lastly, (4) between cutaneous branches of the OA and cutaneous ECA branches from the superficial temporal artery, the transverse facial artery, and the facial artery.

Why is this relevant? First, anatomical variations carry a potential risk of procedural complications of skull base surgery, such as an inadvertent sacrifice of a middle meningeal artery (MMA) that supplies the OA; of microsurgical clipping of OA aneurysms, where the OA origin in relationship to the distal dural ring, the possible presence of a double OA or other variations should be considered before performing an anterior clinoidectomy; of endovascular treatment of dural arteriovenous fistulas (dAVFs), where migration of embolization material can compromise flow in OA branches through EC-IC anastomoses, and of endovascular treatment of OA aneurysms, where flow-diverter stenting may lead to OA occlusion. Second, knowledge of anatomical variations help us better understand complex dAVFs of the cribriform plate or the sphenoid wing.

References

1. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Inst. 1948;212:205–62.
2. Geibprasert S, Pongpech S, Armstrong D, Krings T. Dangerous extracranial-intracranial anastomoses and supply to the cranial nerves: vessels the neurointerventionalist needs to know. AJNR Am J Neuroradiol. 2009;30(8):1459–68. https://doi.org/10.3174/ajnr.A1500.
3. Meyer F. Zur anatomie der Orbitalarteien. Morph Jahr. 1887;12:414–58.
4. Hayreh SS, Dass R. The ophthalmic artery: I. origin and intra-cranial and intra-Canalicular course. Br J Ophthalmol. 1962;46(2):65–98.
5. Hayreh SS, Dass R. The ophthalmic artery: II. Intra-Orbital Course. Br J Ophthalmol. 1962;46(3):165–85.
6. Hayreh SS. The ophthalmic artery: iii. Branches Br J Ophthalmol. 1962;46(4):212–47.
7. Kuru Y. Meningeal branches of the ophthalmic artery. Acta Radiol Diagn (Stockh). 1967;6(3):241–51.
8. Moret J, Lasjaunias P, Theron J, Merland JJ. The middle meningeal artery. Its contribution to the vascularisation of the orbit. J Neuroradiol. 1977;4(2):225–48.
9. Vignaud J, Hasso AN, Lasjaunias P, Clay C. Orbital vascular anatomy and embryology. Radiology. 1974;111(3):617–26. https://doi.org/10.1148/111.3.617.
10. Lasjaunias P, Brismar J, Moret J, Theron J. Recurrent cavernous branches of the ophthalmic artery. Acta Radiol Diagn (Stockh). 1978;19(4):553–60.
11. Shimada K, Kaneko Y, Sato I, Ezure H, Murakami G. Classification of the ophthalmic artery that arises from the middle meningeal artery in Japanese adults. Okajimas Folia Anat Jpn. 1995;72(2–3):163–76.
12. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer; 2001.
13. Streeter GL. The developmental alterations in the vascular system of the brain of the human embryo. Contrib Embryol Carneg Inst. 1918;8(271):5–38.
14. Byrne JV. Tutorials in endovascular neurosurgery and interventional neuroradiology. New York: Springer; 2017.
15. Bradac GB. Applied cerebral angiography. New York: Springer; 2017.
16. Lasjaunias P, Moret J, Manelfe C, Theron J, Hasso T, Seeger J. Arterial anomalies at the base of the skull. Neuroradiology. 1977;13(5):267–72.
17. Lasjaunias P, Moret J, Mink J. The anatomy of the inferolateral trunk (ILT) of the internal carotid artery. Neuroradiology. 1977;13(4):215–20.
18. Mann IC. The relations of the Hyaloid Canal in the Foetus and in the adult. J Anat. 1928;62(Pt 3):290–6.
19. Komiyama M. Letter to the editor—Embryology of the ophthalmic artery: a revived concept. Interv Neuroradiol. 2009;15(3):363–8. https://doi.org/10.1177/159101990901500317.
20. Picard L, Vignaud J, Lombardi G, Roland J. Radiological anatomy of the origin of the ophthalmic artery. In: Second international symposium on orbital disorders. Amsterdam: S. Karger; 1973.
21. Kyoshima K, Oikawa S, Kobayashi S. Interdural origin of the ophthalmic artery at the dural ring of the internal carotid artery. Report of two cases. J Neurosurg. 2000;92(3):488–9. https://doi.org/10.3171/jns.2000.92.3.0488.
22. Matsumura Y, Nagashima M. Anatomical variations in the origin of the human ophthalmic artery with special reference to the cavernous sinus and surrounding meninges. Cells Tissues Organs. 1999;164(2):112–21. https://doi.org/10.1159/000016648.
23. Hayreh SS. Orbital vascular anatomy. Eye (Lond). 2006;20(10):1130–44. https://doi.org/10.1038/sj.eye.6702377.
24. Erdogmus S, Govsa F. Anatomic features of the intracranial and intracanalicular portions of ophthalmic artery: for the surgical procedures. Neurosurg Rev. 2006;29(3):213–8. https://doi.org/10.1007/s10143-006-0028-6.
25. Bertelli E, Regoli M, Bracco S. An update on the variations of the orbital blood supply and hemodynamic. Surg Radiol Anat. 2017;39(5):485–96. https://doi.org/10.1007/s00276-016-1776-9.
26. Dilenge D, Ascherl GF Jr. Variations of the ophthalmic and middle meningeal arteries: relation to the embryonic stapedial artery. AJNR Am J Neuroradiol. 1980;1(1):45–54.

27. Fiore DL, Pardatscher K, Fiore D, Zuccarello M, Iraci G. Persistent dorsal ophthalmic artery. Report of a case with associated fibromuscular hyperplasia of the extracranial internal carotid artery and multiple cerebral aneurysms. Neurochirurgia (Stuttg). 1981;24(3):106–8.
28. Pretterklieber ML, Schindler A, Krammer EB. Unilateral persistence of the dorsal ophthalmic artery in man. Acta Anat (Basel). 1994;149(4):300–5.
29. Gailloud P, Gregg L, Ruiz DS. Developmental anatomy, angiography, and clinical implications of orbital arterial variations involving the stapedial artery. Neuroimaging Clin N Am. 2009;19(2):169–79. https://doi.org/10.1016/j.nic.2009.02.001.
30. Agarwal N, Singh PL, Karimi RJ, Gandhi CD, Prestigiacomo CJ. Persistent vestige of dorsal ophthalmic artery: a case report. J Neurointerv Surg. 2013;5(4):e25. https://doi.org/10.1136/neurintsurg-2011-010196.
31. Lombardi G. Ophthalmic artery anomalies. Ophthalmologica. 1969;157(5):321–7. https://doi.org/10.1159/000305676.
32. Louw L. Different ophthalmic artery origins: embryology and clinical significance. Clin Anat. 2015;28(5):576–83. https://doi.org/10.1002/ca.22470.
33. Uchino A, Saito N, Takahashi M, Kozawa E, Mizukoshi W, Nakajima R, et al. Persistent dorsal ophthalmic artery and ophthalmic artery arising from the middle meningeal artery diagnosed by MR angiography at 3 T. Surg Radiol Anat. 2013;35(9):775–82. https://doi.org/10.1007/s00276-013-1085-5.
34. Willinsky R, Lasjaunias P, Berenstein A. Intracavernous branches of the internal carotid artery (ICA). Comprehensive review of their variations. Surg Radiol Anat. 1987;9(3):201–15.
35. Parlato C, di Nuzzo G, Luongo M, Tortora F, Briganti F. Anatomical variant of origin of ophthalmic artery: case report. Surg Radiol Anat. 2011;33(3):275–8. https://doi.org/10.1007/s00276-010-0745-y.
36. Toma N. Anatomy of the ophthalmic artery: embryological consideration. Neurol Med Chir (Tokyo). 2016;56(10):585–91. https://doi.org/10.2176/nmc.ra.2015-0324.
37. Gregg L, San Millan D, Orru E, Tamargo RJ, Gailloud P. Ventral and dorsal persistent primitive ophthalmic arteries. Oper Neurosurg (Hagerstown). 2016;12(2):141–52. https://doi.org/10.1227/NEU.0000000000001066.
38. Hiura A. An anomalous ophthalmic artery arising from the middle meningeal artery. Anat Anz. 1980;147(5):473–6.
39. Ducasse A, Segal A, Delattre JF, Burette A, Flament JB. Participation of the external carotid artery in orbital vascularization. J Fr Ophtalmol. 1985;8(4):333–9.
40. Liu Q, Rhoton AL Jr. Middle meningeal origin of the ophthalmic artery. Neurosurgery. 2001;49(2):401–6; discussion 6–7.
41. Kimball D, Kimball H, Tubbs RS, Loukas M. Variant middle meningeal artery origin from the ophthalmic artery: a case report. Surg Radiol Anat. 2015;37(1):105–8. https://doi.org/10.1007/s00276-014-1272-z.
42. McLennan JE, Rosenbaum AE, Haughton VM. Internal carotid origins of the middle meningeal artery. The ophthalmic-middle meningeal and stapedial-middle meningeal arteries. Neuroradiology. 1974;7(5):265–75.
43. Georgiou C, Cassell MD. The foramen meningo-orbitale and its relationship to the development of the ophthalmic artery. J Anat. 1992;180(Pt 1):119–25.
44. Ogawa T, Miyauchi T, Kato T, Tamakawa Y. Internal carotid origin of double ophthalmic arteries. Neuroradiology. 1990;32(6):508–10.
45. Kam CK, Alvarez H, Lasjaunias P. Double internal carotid origin of the ophthalmic artery with ruptured aneurysm of the posterior communicating artery. A case report. Interv Neuroradiol. 2003;9(4):383–8. https://doi.org/10.1177/159101990300900409.
46. Namba K, Nemoto S. Double ophthalmic artery visualized with new technology. Neuroradiol J. 2013;26(4):371–2. https://doi.org/10.1177/197140091302600401.
47. Uchino A, Saito N, Kurita H, Ishihara S. Double ophthalmic arteries arising from the internal carotid artery. Surg Radiol Anat. 2013;35(2):173–5. https://doi.org/10.1007/s00276-012-1005-0.
48. Islak C, Ogut G, Numan F, Cokyuksel O, Kuday C. Persistent nonmigrated ventral primitive ophthalmic artery. Report on one case. J Neuroradiol. 1994;21(1):46–9.
49. Bracco S, Gennari P, Vallone IM, Tassi R, Acampa M, Martini G, et al. Double ophthalmic arteries arising from the internal carotid artery: a case report of a hidden second ophthalmic artery. Surg Radiol Anat. 2016;38(10):1233–7. https://doi.org/10.1007/s00276-016-1672-3.
50. Curnow J. Two instances of irregular ophthalmic and middle meningeal arteries. J Anat Physiol. 1873;8(Pt 1):155–6.
51. Musgrove J. Origin of ophthalmic artery from middle meningeal. J Anat Physiol. 1893;27(Pt 2):279–81.
52. Fisher AG. A case of complete absence of both internal carotid arteries, with a preliminary note on the developmental history of the Stapedial artery. J Anat Physiol. 1913;48(Pt 1):37–46.
53. Chanmugan PK. Note on an unusual ophthalmic artery associated with other abnormalities. J Anat. 1936;70:580–2.
54. Bossi R, Pisani C. Collateral cerebral circulation through the ophthalmic artery and its efficiency in internal carotid occlusion. Br J Radiol. 1955;28(333):462–9. https://doi.org/10.1259/0007-1285-28-333-462.
55. Diamond MK. Homologies of the meningeal-orbital arteries of humans: a reappraisal. J Anat. 1991;178:223–41.
56. Gabriele OF, Bell D. Ophthalmic origin of the middle meningeal artery. Radiology. 1967;89(5):841–4. https://doi.org/10.1148/89.5.841.
57. Brucher J. Origin of the ophthalmic artery from the middle meningeal artery. Radiology. 1969;93(1):51–2. https://doi.org/10.1148/93.1.51.
58. Morandi X, Le Bourdon E, Darnault P, Brassier G, Duval JM. Unusual origin of the ophthalmic artery and occlusion of the central retinal artery. Surg Radiol Anat. 1998;20(1):69–71.
59. Hamada J, Kitamura I, Kurino M, Sueyoshi N, Uemura S, Ushio Y. Abnormal origin of bilateral ophthalmic arteries. Case report. J Neurosurg. 1991;74(2):287–9. https://doi.org/10.3171/jns.1991.74.2.0287.
60. Baltsavias G, Turk Y, Valavanis A. Persistent ventral ophthalmic artery associated with supraclinoid internal carotid artery aneurysm: case report and review of the literature. J Neuroradiol. 2012;39(3):186–9. https://doi.org/10.1016/j.neurad.2011.08.002.
61. Naeini RM, De J, Satow T, Benndorf G. Unilateral agenesis of internal carotid artery with ophthalmic artery arising from posterior communicating artery. AJR Am J Roentgenol. 2005;184(2):571–3. https://doi.org/10.2214/ajr.184.2.01840571.
62. Hassler W, Zentner J, Voigt K. Abnormal origin of the ophthalmic artery from the anterior cerebral artery: neuroradiological and intraoperative findings. Neuroradiology. 1989;31(1):85–7.
63. Li Y, Horiuchi T, Yako T, Ishizaka S, Hongo K. Anomalous origin of the ophthalmic artery from the anterior cerebral artery. Neurol Med Chir (Tokyo). 2011;51(8):579–81.
64. Hannequin P, Peltier J, Destrieux C, Velut S, Havet E, Le Gars D. The inter-optic course of a unique precommunicating anterior cerebral artery with aberrant origin of an ophthalmic artery: an anatomic case report. Surg Radiol Anat. 2013;35(3):269–71. https://doi.org/10.1007/s00276-012-1028-6.
65. Honma Y, Ogawa T, Nagao S. Angiographically occult anomalous ophthalmic artery arising from the anterior cerebral artery. Acta Neurochir. 1997;139(5):480–1.
66. Belotti F, Ferrari M, Doglietto F, Cocchi MA, Lancini D, Buffoli B, et al. Ophthalmic artery originating from the anterior cerebral artery: anatomo-radiological study, histological analysis, and literature review. Neurosurg Rev. 2016;39(3):483–93. https://doi.org/10.1007/s10143-016-0715-x.

67. Schumacher M, Wakhloo AK. An orbital arteriovenous malformation in a patient with origin of the ophthalmic artery from the basilar artery. AJNR Am J Neuroradiol. 1994;15(3):550–3.
68. Sade B, Tampieri D, Mohr G. Ophthalmic artery originating from basilar artery: a rare variant. AJNR Am J Neuroradiol. 2004;25(10):1730–1.
69. Rivera R, Choi IS, Sordo JG, Giacaman P, Badilla L, Bravo E, et al. Unusual origin of the left ophthalmic artery from the basilar trunk. Surg Radiol Anat. 2015;37(4):399–401. https://doi.org/10.1007/s00276-014-1327-1.
70. Zhou WZ, Zhao LB, Liu S, Shi HB. Teaching NeuroImages: unilateral agenesis of internal carotid artery with ophthalmic artery from opposite side. Neurology. 2015;84(9):e65–6. https://doi.org/10.1212/WNL.0000000000001317.
71. Zink WE, Komotar RJ, Meyers PM. Internal carotid aplasia/hypoplasia and intracranial saccular aneurysms: series of three new cases and systematic review of the literature. J Neuroimaging. 2007;17(2):141–7. https://doi.org/10.1111/j.1552-6569.2007.00092.x.
72. Rhoton AL Jr. The orbit. Neurosurgery. 2002;51(4 Suppl):S303–34.
73. Rhoton AL Jr. The supratentorial arteries. Neurosurgery. 2002;51(4 Suppl):S53–120.
74. Marano GD, Horton JA, Gabriele OF. Persistent embryologic vascular loop of the internal carotid, middle meningeal, and ophthalmic arteries. Radiology. 1981;141(2):409–10. https://doi.org/10.1148/radiology.141.2.7291564.
75. Roland J, Georges JL, Bracard S, Picard L. Persistence of the perioptic ring of the ophthalmic artery. Anatomical and embryological observations. Bull Assoc Anat (Nancy). 1984;68(200):65–70.
76. Lang J, Kageyama I. The ophthalmic artery and its branches, measurements and clinical importance. Surg Radiol Anat. 1990;12(2):83–90.
77. Cordier J, Rauber G, Raspiller A, George JL. Arterial vascularization of the musculi rectus verticalis and obliquus oculi (author's transl). J Fr Ophtalmol. 1981;4(5):397–404.
78. Maiuri F, Donzelli R, de Divitiis O, Fusco M, Briganti F. Anomalous meningeal branches of the ophthalmic artery feeding meningiomas of the brain convexity. Surg Radiol Anat. 1998;20(4):279–84.
79. Alvarez H, Rodesch G, Garcia-Monaco R, Lasjaunias P. Embolisation of the ophthalmic artery branches distal to its visual supply. Surg Radiol Anat. 1990;12(4):293–7.
80. Sabatier A, Robert PY, Sauvage JP, Bessede JP, Adenis JP, Chapot R. Ophthalmic arteries originating from external carotid arteries. J Fr Ophtalmol. 2006;29(8):e20.
81. Perrini P, Cardia A, Fraser K, Lanzino G. A microsurgical study of the anatomy and course of the ophthalmic artery and its possibly dangerous anastomoses. J Neurosurg. 2007;106(1):142–50. https://doi.org/10.3171/jns.2007.106.1.142.
82. Robert T, Blanc R, Smajda S, Ciccio G, Redjem H, Bartolini B, et al. Endovascular treatment of cribriform plate dural arteriovenous fistulas: technical difficulties and complications avoidance. J Neurointerv Surg. 2016;8(9):954–8. https://doi.org/10.1136/neurintsurg-2015-011956.
83. Kakarla UK, Deshmukh VR, Zabramski JM, Albuquerque FC, McDougall CG, Spetzler RF. Surgical treatment of high-risk intracranial dural arteriovenous fistulae: clinical outcomes and avoidance of complications. Neurosurgery. 2007;61(3):447–57; discussion 57–9. https://doi.org/10.1227/01.NEU.0000290889.62201.7F.
84. Agid R, Terbrugge K, Rodesch G, Andersson T, Soderman M. Management strategies for anterior cranial fossa (ethmoidal) dural arteriovenous fistulas with an emphasis on endovascular treatment. J Neurosurg. 2009;110(1):79–84. https://doi.org/10.3171/2008.6.17601.
85. Shi ZS, Ziegler J, Feng L, Gonzalez NR, Tateshima S, Jahan R, et al. Middle cranial fossa sphenoidal region dural arteriovenous fistulas: anatomic and treatment considerations. AJNR Am J Neuroradiol. 2013;34(2):373–80. https://doi.org/10.3174/ajnr.A3193.
86. Kandyba DV, Babichev KN, Stanishevskiy AV, Abramyan AA, Svistov DV. Dural arteriovenous fistula in the sphenoid bone lesser wing region: endovascular adjuvant techniques of treatment and literature review. Interv Neuroradiol. 2018;24(5):559–66. https://doi.org/10.1177/1591019918777233.
87. Di Maria F, Pistocchi S, Clarencon F, Bartolini B, Blanc R, Biondi A, et al. Flow diversion versus standard endovascular techniques for the treatment of Unruptured carotid-ophthalmic aneurysms. AJNR Am J Neuroradiol. 2015;36(12):2325–30. https://doi.org/10.3174/ajnr.A4437.
88. Zhou G, Zhu YQ, Su M, Gao KD, Li MH. Flow-diverting devices versus coil embolization for intracranial aneurysms: a systematic literature review and meta-analysis. World Neurosurg. 2016;88:640–5. https://doi.org/10.1016/j.wneu.2015.11.007.
89. Touze R, Gravellier B, Rolla-Bigliani C, Touitou V, Shotar E, Lenck S, et al. Occlusion rate and visual complications with flow-diverter stent placed across the ophthalmic artery's origin for carotid-ophthalmic aneurysms: a meta-analysis. Neurosurgery. 2019;86:455. https://doi.org/10.1093/neuros/nyz202.
90. Trivelatto F, Nakiri GS, Manisor M, Riva R, Al-Khawaldeh M, Kessler I, et al. Preoperative onyx embolization of meningiomas fed by the ophthalmic artery: a case series. AJNR Am J Neuroradiol. 2011;32(9):1762–6. https://doi.org/10.3174/ajnr.A2591.
91. Shah A, Choudhri O, Jung H, Li G. Preoperative endovascular embolization of meningiomas: update on therapeutic options. Neurosurg Focus. 2015;38(3):E7. https://doi.org/10.3171/2014.12.FOCUS14728.
92. Reyre A, Michel J, Santini L, Dessi P, Vidal V, Bartoli JM, et al. Epistaxis: the role of arterial embolization. Diagn Interv Imaging. 2015;96(7–8):757–73. https://doi.org/10.1016/j.diii.2015.06.006.
93. Mames RN, Snady-McCoy L, Guy J. Central retinal and posterior ciliary artery occlusion after particle embolization of the external carotid artery system. Ophthalmology. 1991;98(4):527–31. https://doi.org/10.1016/s0161-6420(91)32261-9.
94. Yamane T, Kaneko A, Mohri M. The technique of ophthalmic arterial infusion therapy for patients with intraocular retinoblastoma. Int J Clin Oncol. 2004;9(2):69–73. https://doi.org/10.1007/s10147-004-0392-6.
95. Klufas MA, Gobin YP, Marr B, Brodie SE, Dunkel IJ, Abramson DH. Intra-arterial chemotherapy as a treatment for intraocular retinoblastoma: alternatives to direct ophthalmic artery catheterization. AJNR Am J Neuroradiol. 2012;33(8):1608–14. https://doi.org/10.3174/ajnr.A3019.

Anatomy and Variations of the Superficial Temporal Artery

Thomas Robert and Sara Bonasia

The superficial temporal artery (STA) is considered as one of the terminal branches of the external carotid artery (ECA) together with the internal maxillary artery (IMA) [1]. This is the largest among the scalp vessels. Its cutaneous territory is in balance with that of the occipital and posterior auricular arteries [2]. The STA is easily recognized on DSA by its characteristic tortuosity [1, 3]. Even if the STA is not involved in most of the cranial vascular pathologies, the knowledge of its accurate anatomy is important for pediculated cutaneous flap, extracranial-intracranial vascular anastomosis, or to avoid liquid embolic agent in its natural anastomoses with arteries of the orbital region [4–8].

History

The STA is a well-known artery since the beginning of the twentieth century. The first and most famous author who described this artery is Eutathianos, who proposed a comprehensive description of its anatomy [9, 10]. After him, during the 1960s, plastic surgeons and anatomists paid attention to its anatomy and described most of the pediculated cutaneous flap based on the STA supply [11–13]. From the 1970s, neurosurgeons also showed a particular interest in the anatomy of the STA particularly in the position and diameter of its parietal branch for extracranial-intracranial vascular anastomosis [2].

Embryological Development

Details about the embryological development of the STA are not known, but given that the STA is a distal branch of the external carotid artery, its embryological development depends on those of the ventral pharyngeal artery (VPA), which appears at the third stage of Padget (7–12 mm) [14]. The STA seems to arise directly from the primitive ventral pharyngeal artery and not to be part of another embryological system annexed by the VPA [15].

Number and Origin of the Artery

The STA arises from the distal part of the ECA together with the IMA inside of the parotid gland, at a depth of 25 mm approximately [1]. The STA has a vertical course, while the IMA curves medially and anteriorly to enter in the infratemporal and successively pterygo-palatine fossae [12, 13, 16]. A duplicated origin of the STA is not described but its main (terminal) bifurcation could be lower than the zygomatic arch in 7–11% or over the posterior part of the zygomatic arch in 3–26% giving to the artery a pseudo-duplication appearance [17]. In most cases, the bifurcation of the STA in frontal (anterior) and parietal (posterior) branches is above the zygomatic arch [10, 12, 13, 18].

Course of the Artery

The STA has a vertical course from the parotid gland, passing over the posterior part of the zygomatic arch and anteriorly to the bony external auditory canal [19, 20]. The mean diameter of the STA at its origin is 2.5 and 2.2 mm at the level of the zygomatic arch [21]. The average length of the principal trunk of the STA is 31.7 mm [20]. Ricbourg et al. described three distinct segments of the STA [12, 13]:

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- I: Intraparotid with a length of 15 mm
- II: Deep in the subcutaneous plan
- III: Superficial in the supra-fascial plan

Between the second and third segments, Ricbourg et al. described the typical temporal siphon of the artery [12, 13].

Depending on the branching pattern of the STA, different configuration could be individualized and are summarized in Table 1. We could see a classical disposition in bifurcation (frontal and parietal branches), a double parietal branch, a double frontal branch, or a trifurcation if the zygomatico-orbital branch is dominant [21–25]. The caliber and domi-

Table 1 Different branching pattern of the superficial temporal artery

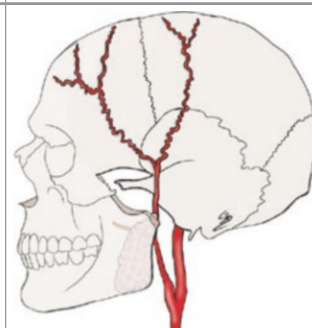
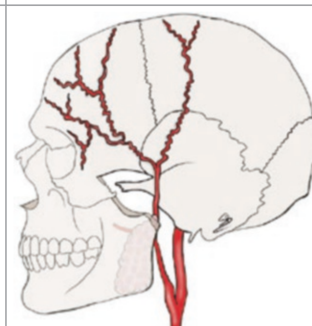
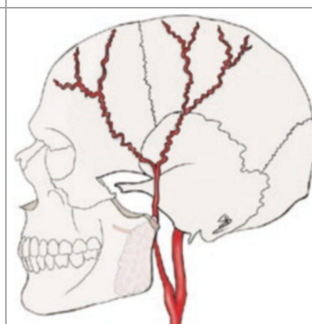
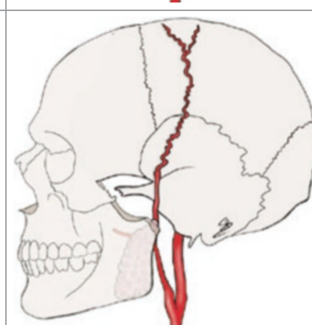
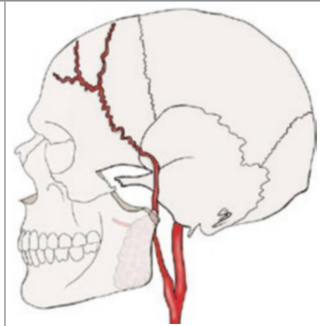
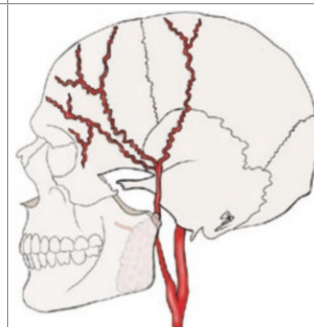
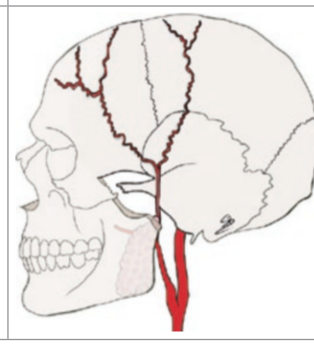
Branching pattern	Incidence (%)	Configuration
"Classic" bifurcation	66	
Double frontal branch	8	
Double parietal branch	4	
Parietal branch only	6	

Table 1 (continued)

Branching pattern	Incidence (%)	Configuration
Frontal branch only	2	
Pseudo-trifurcation	7	
Hypoplastic STA	7	

nance of different branches of the STA have an important variability and Marano et al. described not less than 10 distinct branching patterns of this artery [21].

Branches of the Artery

Table 2 and Fig. 1 summarize different branches of the STA with their respective arterio-arterial anastomoses.

Close to its origin, the STA gives few parotid branches and also 2–3 little branches to the temporo-mandibular joint [12, 13, 18, 21].

After that, one of its first collateral branches is the transverse facial artery which has an antero-inferior orientation with a mean diameter of 0.7 mm [5]. It could also arise directly from the ECA in 7% or from the anterior branch of the STA [12, 13]. This artery has a double origin in 4% and gives two distinct branches, the first one is muscular and the second one is tegmental [22].

Table 2 Different branches of the superficial temporal artery with their respective anastomosis

Superficial temporal artery branches	Blood supply	Possible anastomosis
Parotid branches	Deep half of the parotid gland	Posterior auricular artery
Temporo-mandibular joint branches	Temporo-mandibular joint	
Anterior auricular artery	External ear	Posterior auricular artery
Transverse facial artery	Infra-temporal fossa	Facial artery
Zygomatico-orbital artery	Lateral angle of the orbit	Lacrimal artery (OA) Superior and inferior palpebral arteries (IMA)
Posterior deep temporal artery	Lateral	Middle and anterior deep temporal artery (IMA)
Frontal branch	Forehead Anterior part of the scalp	Superior orbital artery (OA) Supratrochlear artery (OA)
Parietal branch	Middle part of the scalp	Occipital artery Posterior auricular artery

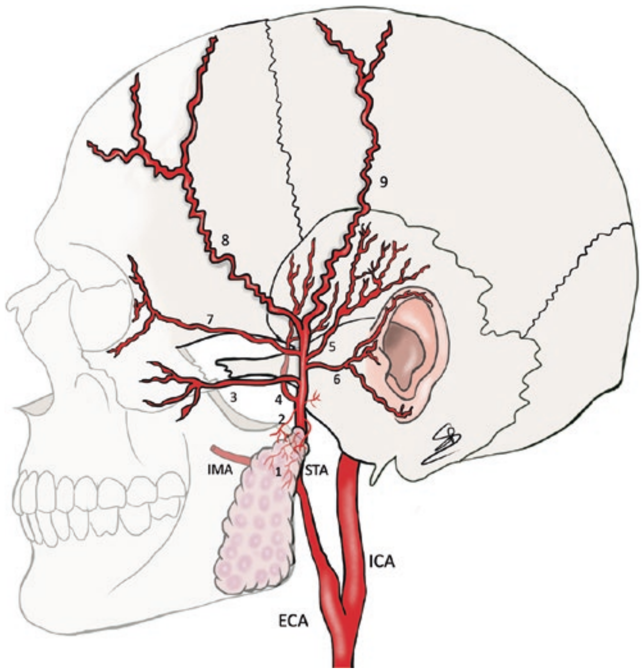


Fig. 1 Branches of the STA. *ICA* internal carotid artery, *ECA* external carotid artery; (1) parotid branches; (2) branches for the temporo-mandibular joint; (3) transverse facial artery; (4) middle temporal artery; (5) posterior deep temporal artery; (6) anterior auricular artery; (7) zygomatic-orbital branch; (8) frontal division; (9) parietal division

The second collateral branch of the STA is the zygomatico-orbital branch that has an anterior orientation to the lateral part of the orbit. It could be absent in case of low-lying bifurcation of the STA, but it could also be dominant (trifurcation appearance of the STA) [6, 21, 26]. Ricbourg et al. described two types depending on its origin: the type I when arises from the STA (80%) and the type II when arises from its anterior branch (20%) [13].

The anterior auricular artery is a posterior collateral of the STA that gives three distinct little branches, one for the tragus, one for the lobule of the ear, and the last one for the helix [27].

The STA also participates in the supply of the temporal muscle with two different arteries [28–30]. The first one is the middle temporal artery that passes over the zygomatic arch to follow the intermediate temporal fascia [13, 28]. The other temporal artery from the STA is the posterior deep temporal artery which passes below the zygomatic arch and courses in the deep temporal fascia with the middle and anterior deep temporal arteries (branches of the IMA) [2].

After giving all its collateral branches, the STA bifurcates in frontal (anterior) and parietal (posterior) branches that are constant branches; their respective diameter is 1.14 and 1.2 mm, and their respective length is 114 and 115 mm [9, 31].

Possible Anastomoses

The STA presents a lot of anastomoses, and we classified them in subcutaneous, facial, and cutaneo-meningeal anastomosis to avoid confusion.

Subcutaneous Anastomoses

The subcutaneous territory of the STA is shared with other subcutaneous arteries, which contribute to the formation of a rich anastomotic network [2, 7, 8]. The posterior branch of the STA anastomoses with branches of the posterior auricular artery and with branches of the occipital artery. Both terminal branches of the STA also have anastomoses in the midline region with their contralateral counterpart [1, 3, 12, 13].

Cutaneo-meningeal Anastomoses

This represents only a little part of the anastomoses of the STA but clinically important for its risk of accidental embolic risk during endovascular procedures [7, 8]. The STA presents some anastomoses with the middle meningeal artery through the foramina of emissary veins in the midline region.

Facial Anastomoses

The anterior auricular artery through its helical branch anastomoses with branches of the posterior auricular artery [12, 13]. The transverse facial artery has anastomosis with the facial artery (branch of the ECA). In the deep temporal muscle fascia, the posterior deep temporal artery anastomoses with branches of the middle and anterior deep temporal arteries (collaterals of the IMA) [32, 33].

The most clinically relevant anastomoses of the STA are anastomoses with branches of the ophthalmic artery (OA) because they constitute a potential communication between the external carotid and internal carotid systems and, consequently a dangerous route for liquid embolic procedures of branches of the ECA [7, 8]. The anterior branch of the STA anastomoses with the supraorbital and supratrochlear arteries of the OA. The zygomatico-orbital branch of the STA has anastomoses with the lacrimal artery (OA) and with the superior and inferior palpebral arteries (branches of the IMA) [2].

Blood Supply

The principal role of the STA is the vascularization of the anterior two-third of the scalp and forehead [11, 26, 31, 32]. Other than this principal function, the STA participates in the tegmental and muscular supply of the preauricular region and partially supplies the following structures [10, 12, 13, 18]:

- The lateral angle of the orbit (zygomatico-orbital branch)
- The temporal muscle (deep temporal artery)
- The anterior part of the external ear (anterior auricular artery)
- The deep half of the parotid gland
- The posterior part of the temporo-mandibular joint
- The masseter muscles

Clinical Implications

Even if the STA is "only" a cutaneous and an extracranial artery, it is the branch of the ECA with the most important clinical implication.

Neurosurgeons pay an important attention for the STA for the treatment of chronic ischemic disease as Moyamoya disease for its utility in extracranial-intracranial vascular anastomosis. For this sort of treatment, a precise knowledge in the anatomy of the STA is important. Different anatomical variations need to be known to decide the surgical strategy [4].

The STA is also a useful artery for pedicular facial cutaneous flap used by plastic surgeons. A lot of surgical technics are based on the anatomy and territory of supply of the STA. In case of free-flap reconstruction, the STA is one more time the first-choice arterial pedicle used [17, 20, 21, 24, 26, 27].

Knowledge of the presence of anastomosis between the STA and branches of the ophthalmic artery is also crucial in the treatment and understanding of ECA vascular pathology. Indeed, the treatment of anterior skull base pathologies by STA liquid embolization has the risk of accidental migration of the liquid agent in the OA and potentially in the ICA [2, 7].

References

1. Salamon G, Guerinel G, Demard F. Radioanatomical study of the external carotid artery. Ann Radiol (Paris). 1968;11(3):199–215.
2. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springe; 2001.
3. Dilenge D. The superficial temporal artery. Presse Med. 1967;75(14):717–20.
4. Lee S, Kim SK, Phi JH. Anatomic variation of the superficial temporal artery and posterior auricular artery in a pediatric Moyamoya disease population. AJNR Am J Neuroradiol. 2021;42(6):1157–62. https://doi.org/10.3174/ajnr.A7059.
5. Manoli T, Medesan R, Held M, Schaller HE, Ernemann U, Korn A, et al. Bilateral comparison of the vascular pattern of the superficial temporal artery based on digital subtraction angiography. Surg Radiol Anat. 2016;38(2):179–86. https://doi.org/10.1007/s00276-015-1538-0.
6. Imran FH, Yong CK, Das S, Huei YL. Anatomical variants of the superficial temporal artery in patients with microtia: a pilot descriptive study. Anat Cell Biol. 2016;49(4):273–80. https://doi.org/10.5115/acb.2016.49.4.273.
7. Geibprasert S, Pongpech S, Armstrong D, Krings T. Dangerous extracranial-intracranial anastomoses and supply to the cranial nerves: vessels the neurointerventionalist needs to know. AJNR Am J Neuroradiol. 2009;30(8):1459–68. https://doi.org/10.3174/ajnr.A1500.
8. Rinaldo L, Brinjikji W. Dangerous extracranial-intracranial anastomoses: what the Interventionalist must know. Semin Interv Radiol. 2020;37(2):140–9. https://doi.org/10.1055/s-0040-1709155.
9. Koziej M, Wnuk J, Polak J, Trybus M, Pekala P, Pekala J, et al. The superficial temporal artery: a meta-analysis of its prevalence and morphology. Clin Anat. 2020;33(8):1130–7. https://doi.org/10.1002/ca.23550.
10. Rusu MC, Jianu AM, Radoi PM. Anatomic variations of the superficial temporal artery. Surg Radiol Anat. 2021;43(3):445–50. https://doi.org/10.1007/s00276-020-02629-x.
11. Cormia FE. Vasculature of the Normal scalp. Arch Dermatol. 1963;88:692–701. https://doi.org/10.1001/archderm.1963.01590240016004.
12. Ricbourg B, Mitz V, Lassau JP. superficial temporal artery. Study of 50 cases. Regional topography. Arch Anat Pathol (Paris). 1973;21(4):381–8.
13. Ricbourg B, Mitz V, Lassau JP. Superficial temporal artery, anatomic study and practical deductions. Ann Chir Plast. 1975;20(2):197–213.
14. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: a pictorial review. Surg Radiol Anat. 2021;43(6):961–73. https://doi.org/10.1007/s00276-021-02684-y.
15. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–62.
16. Stock AL, Collins HP, Davidson TM. Anatomy of the superficial temporal artery. Head Neck Surg. 1980;2(6):466–9. https://doi.org/10.1002/hed.2890020604.
17. Tayfur V, Edizer M, Magden O. Anatomic bases of superficial temporal artery and temporal branch of facial nerve. J Craniofac Surg. 2010;21(6):1945–7. https://doi.org/10.1097/SCS.0b013e3181f4ee6a.
18. Pinar YA, Govsa F. Anatomy of the superficial temporal artery and its branches: its importance for surgery. Surg Radiol Anat. 2006;28(3):248–53. https://doi.org/10.1007/s00276-006-0094-z.
19. Correa MB, Wafae GC, Pereira LA, Wafae N, Ruiz CR. Arterial branches to the temporal muscle. Ital J Anat Embryol. 2008;113(2):109–15.
20. Czerwinski F. Variability in the course of the superficial temporal artery in man. Folia Morphol (Warsz). 1992;51(1):49–54.
21. Marano SR, Fischer DW, Gaines C, Sonntag VK. Anatomical study of the superficial temporal artery. Neurosurgery. 1985;16(6):786–90. https://doi.org/10.1227/00006123-198506000-00008.
22. Medved F, Manoli T, Medesan R, Janghorban Esfahani B, Stahl S, Schaller HE, et al. In vivo analysis of the vascular pattern of the superficial temporal artery based on digital subtraction angiography. Microsurgery. 2015;35(5):380–6. https://doi.org/10.1002/micr.22348.
23. Mwachaka P, Sinkeet S, Ogeng'o J. Superficial temporal artery among Kenyans: pattern of branching and its relation to pericranial structures. Folia Morphol (Warsz). 2010;69(1):51–3.
24. Ndiaye A, Toure S, Diop FM, Ndiaye AS, Diallo BK, Sow ML. The superficial temporal artery: anatomical type and clini-

cal application to the flap of the fascia temporal superficialis. Morphologie. 2004;88(282):155–9. https://doi.org/10.1016/s1286-0115(04)98140-4.

25. Yonenaga K, Tohnai I, Mitsudo K, Mori Y, Saijo H, Iwai T, et al. Anatomical study of the external carotid artery and its branches for administration of superselective intra-arterial chemotherapy via the superficial temporal artery. Int J Clin Oncol. 2011;16(6):654–9. https://doi.org/10.1007/s10147-011-0238-y.
26. Kim BS, Jung YJ, Chang CH, Choi BY. The anatomy of the superficial temporal artery in adult koreans using 3-dimensional computed tomographic angiogram: clinical research. J Cerebrovasc Endovasc Neurosurg. 2013;15(3):145–51. https://doi.org/10.7461/jcen.2013.15.3.145.
27. Chen TH, Chen CH, Shyu JF, Wu CW, Lui WY, Liu JC. Distribution of the superficial temporal artery in the Chinese adult. Plast Reconstr Surg. 1999;104(5):1276–9. https://doi.org/10.1097/00006534-199910000-00006.
28. Abul-Hassan HS, von Drasek AG, Acland RD. Surgical anatomy and blood supply of the fascial layers of the temporal region. Plast Reconstr Surg. 1986;77(1):17–28.
29. Lee JG, Yang HM, Hu KS, Lee YI, Lee HJ, Choi YJ, et al. Frontal branch of the superficial temporal artery: anatomical study and clinical implications regarding injectable treatments. Surg Radiol Anat. 2015;37(1):61–8. https://doi.org/10.1007/s00276-014-1306-6.
30. Shin KJ, Shin HJ, Lee SH, Koh KS, Song WC. Surgical anatomy of the superficial temporal artery to prevent facial nerve injury during arterial biopsy. Clin Anat. 2018;31(4):608–13. https://doi.org/10.1002/ca.23033.
31. Har-Shai Y, Fukuta K, Collares MV, Stefanovic PD, Filipovic BR, Herschman BR, et al. The vascular anatomy of the galeal flap in the interparietal and midline regions. Plast Reconstr Surg. 1992;89(1):64–9.
32. Kuruoglu E, Cokluk C, Marangoz AH, Aydin K. The evaluation of three-dimensional anatomy of the superficial temporal artery using the volume rendering technique. Turk Neurosurg. 2015;25(2):285–8. https://doi.org/10.5137/1019-5149.JTN.10630-14.1.
33. Nakajima H, Imanishi N, Minabe T. The arterial anatomy of the temporal region and the vascular basis of various temporal flaps. Br J Plast Surg. 1995;48(7):439–50. https://doi.org/10.1016/0007-1226(95)90118-3.

Embryology and Variations of the Occipital Artery

Thomas Robert and Sara Bonasia

The occipital artery (OccA) is one of the major branches of the external carotid artery (ECA) [1, 2]. During the last 20 years, neuroradiologists and neurosurgeons demonstrated an increasing interest on the OccA for different reasons. For example, for the treatment of dural arteriovenous fistula since the OccA is often concerned or for the use of this artery in case of intracranial-extracranial arterial anastomosis [3, 4]. The occipital artery is also fascinating for its complex embryological development that is not already thoroughly known [5]. In this chapter, we will summarize the anatomy and variations concerning the OccA after a comprehensive description of the embryological development of the pharyngo-occipital system.

History

The first cadaveric reports about the OccA were published during the nineteenth century principally by case reports of neck dissections. We must wait for the beginning of the twentieth century to have different key papers about the embryology and about the phylogeny to understand better the complexity of the occipital artery. As for others cranio-facial arteries, the masterpiece work of D. Padget (1948) allows to light up our actual knowledge about the complex embryology of this artery [5]. Adams (1957) furnished an excellent work concerning the homologies of the occipital artery among different animals [6]. With the increase amount of interest due to the emergence of external carotid artery branches embolization, the number of publications concerning the occipital artery rises after the 1970s. Lasjaunias was a pioneer during this period and gave important information about the embryology and variations of all cranio-facial arteries [7–10]. After him, a lot of case reports describing variations of the occipital artery were based on his publications. Among them, we need to cite Martins (2005) who gave a comprehensive description of all the dural arteries including the occipital artery and Alvernia (2006) for its anatomical report of the same artery [2, 11].

Embryological Development

From a phylogenic point of view, the occipital artery is very fascinating because of its large disparity between animals [6, 12, 13]. In fishes, no true occipital artery or equivalent could be visualized. In batrachians, an occipital ramus could be individualized but arises from the vertebral artery [6]. On the other hand, birds present a ventral subclavian artery that supply the two first cervical segments, and for whom Adams (1957) found a lot of similarities with the human occipital artery [6]. In mammals, a true occipital artery could be seen but with little differences among species [1]. For example, in horses and dogs, the occipital artery also gives jugular and hypoglossal branches (normal territory of the ascending pharyngeal artery in humans). Another interesting example is that of sheep which present an occipital artery that passes through the hypoglossal canal (anterior condyloid canal) and turns backwards in the cervical column to anastomose with collaterals of the arteries of the spine [9].

In this chapter, only notions about the embryological development that allow to understand the pharyngo-occipital system will be developed (Table 1). For more detailed embryological development about the vertebral artery, the carotid system, and the carotido-vertebral anastomoses, we send you to the respective chapters.

Early in the embryological life (stage I of Padget, embryos of 4–5 mm), the formation of the vertebro-basilar system ini-

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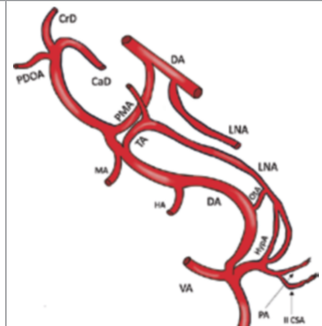
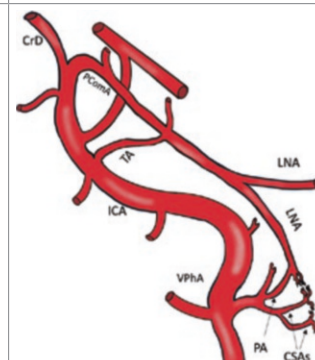
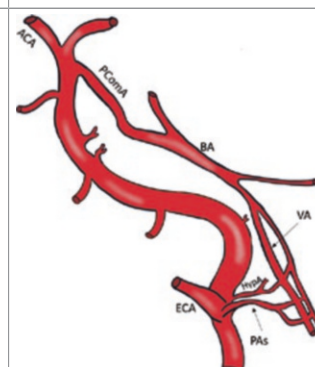
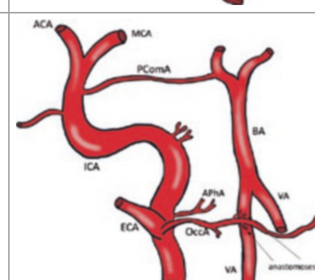
Table 1 Major embryological changes in the formation of the occipito-pharyngeal system

Stage	Embryo size (mm)	Major evolutions	Graphic illustration
I	4–5	– Paired longitudinal neural arteries (LNA) – Trigeminal (TA), otic (OtA), hypoglossal (HypA), proatlantal (PA), and second cervical segmental artery (II CSA) – PComA not formed	
II	5–6	– Formation of the PComA – Regression of otic and hypoglossal arteries – Fusion of LNAs in basilar artery (initiation) – Initial transverse anastomoses between the intersegmental arteries	
III	7–12	– Basilar artery (BA) completely formed – PComA completely formed – Regression of trigeminal and proatlantal arteries – Formation of the vertebral artery VA (initiation)	
IV	13–15	– Migration of OccA origin on the external carotid artery (ECA) – Persistence of the anastomoses between OccA and VA	
V	16–18	– Complete formation of the VA – Complete formation of the OccA (remnant of the proatlantal artery) – Complete formation of the ascending pharyngeal artery (APhA, remnant of the hypoglossal artery)	

tiates with the presence of paired longitudinal artery named “longitudinal neural arteries (LNA)” [5]. These LNAs are supplied by the carotid system through different carotido-vertebral anastomosis, and the vertebral arteries are not developed yet. LNAs are supplied by the trigeminal artery, the otic artery, the hypoglossal artery, and the proatlantal artery from cranial to caudal [1, 14].

During the stage II of Padget (embryos of 5–6 mm), the caudal division of the primitive internal carotid artery anastomoses with the cranial part of the LNA (formation of the posterior communicating artery), and consequently the carotido-vertebral anastomoses initiate their involution. The first two arteries that regress are the otic and the hypoglossal arteries, and the LNAs are supplied principally by the trigeminal artery cranially and the proatlantal artery caudally. At the same time, the two LNAs come closer to the midline to fuse together [5, 14].

At stage III of Padget (embryos of 7–12 mm), the basilar artery is completely formed by fusion of the two LNAs as well as the posterior communicating artery which is anastomosed with the upper part of the basilar artery. Trigeminal and proatlantal arteries progressively regress, and the primitive vertebral artery is formed by transverse anastomoses between the six upper cervical intersegmental arteries [1, 5, 15].

The occipital artery is considered as the remnant of the first (proatlantal intersegmental artery) and second cervical segmental arteries. Between stages III and V, the origin of the occipital artery shifts from the primitive internal carotid artery to the external carotid artery, and the distal parts of the two first intersegmental cervical arteries leave only thin anastomosis between the occipital artery and the vertebral artery. At stage V (embryos of 16–18 mm), the definite occipital artery could be clearly individualized [1, 8, 10, 16].

Origin of the Artery

The most common origin of the artery is the posterior aspect of the external carotid artery [2]. The distance between the common carotid artery bifurcation and the origin of the OccA is 7–28 mm (median 17 mm), and its overall diameter at origin is 2.3 mm [2]. The origin of the OccA is superior to the facial artery in 57%, between the facial and the lingual arteries in 32% and inferior to the lingual artery in only 11% [17].

The most frequent variation in the origin of the occipital artery is named the pharyngo-occipital artery [1, 8, 18]. It consists in a common trunk from the external carotid artery shared by the occipital artery and the ascending pharyngeal artery. Embryologically, it is not a surprising variation given that these two arteries have the same embryological origin from the third aortic arch and not from the ventral pharyngeal artery [1]. The incidence of this pharyngo-occipital artery is estimated at 6% by radiological studies [17]. The occipital artery could also share its origin with other branches of the external carotid artery: the posterior auricular artery or the superior thyroidal artery. These variations are rarer, and only few case reports were published [19]. The pharyngo-occipital artery variant almost all the time originates from the external carotid artery, but in few cases, it was described as an unusual branch of the internal carotid artery (whom two cases were bilateral) [19–22].

Another interesting variation in the origin of the occipital artery is the ICA origin of the occipital artery. The first anatomic case was reported by Hyrtl (1841) and the first angiographic demonstration in 1965 by Seidel [17]. Uchino et al. (2011) estimated its incidence at 0.21% based on magnetic resonance angiography studies [23]. We have found 16 cases of ICA origin of the occipital artery in the literature [4, 17, 24–30]. Two of them were bilateral, the others were unilateral without predominance of side [28, 31]. One of these cases published by Uchino et al. (2015) was very interesting because it associated an “aberrant” ICA with a pharyngo-occipital artery that gave off from the pseudo-ICA [32]. Embryologically, it is not surprising given that the cervical pseudo-ICA is the equivalent of a dilated proximal ascending pharyngeal artery [1]. An ICA origin of the occipital artery could be assimilated as a partial persistence of the proatlantal artery (lack in migration of the proatlantal artery from the ICA to the ECA) [8].

Common carotid artery (CCA) origin of the occipital artery was also described but is very rare [1, 8, 33]. Commonly, the occipital artery arises from the CCA near to its terminal bifurcation and could also be named a CCA trifurcation by few authors [33].

Course of the Artery

The occipital artery has a course under the lateral skull base to end in the posterior part of the scalp [2]. Its course is divided in three distinct segments:

- The digastric segment from its origin to its passage into the occipital groove [2, 11]. The occipital artery takes a postero-superior direction between the posterior belly of the digastric muscle (laterally) and the great vessels and lower cranial nerves (medially). The diameter of the OccA in its proximal segment is comprised between 2.2 and 2.9 mm before it passes medial to the mastoid and enters into its occipital groove or canal (in 12% of cases) [2].
- The cervico-occipital or suboccipital segment is the second segment of the OccA from its exit from the occipital groove to its intersection with the superior nuchal line

where it pierces the insertion of the splenius capitis muscle [8, 12]. The orientation of this segment is superomedial and the diameter of the artery is between 1.4 and 1.9 mm. The entire length of this segment is under or between the nuchal muscles [2].

- The subgaleal or terminal segment of the artery is the artery and its branches after it distal to the superior nuchal line. The OccA is less than 1.4 mm in diameter, and its average length is 35 mm. The artery courses over the occipital muscles surface, have a near vertical paramedian orientation (2–4 cm from the midline) [2].

The segments and main branches of the OccA are summarized in Fig. 1.

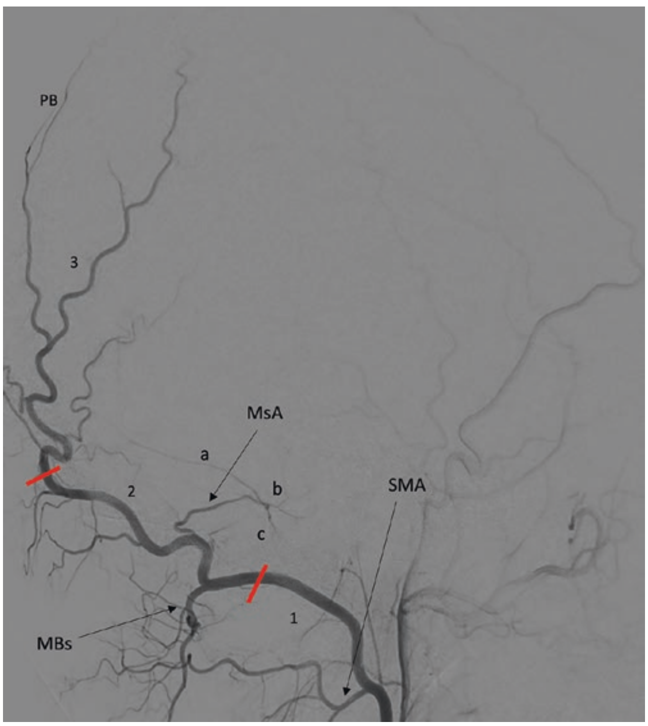


Fig. 1 OccA course and segments. The lateral view DSA shows an injection of the external carotid artery with visualization of the Occipital artery (OccA). The digastric segment (1) extends from the origin of the OccA to the occipital groove. It gives usually muscular branches that can share a common origin. Like in this case, the common stem is called sterno-mastoid artery (SMA). The cervico-occipital or suboccipital segment (2) extends from the occipital groove to its intersection with the superior nuchal line. It gives usually muscular branches (MBs) that can share a common origin and dural branches, the largest of which is the mastoid artery (MsA). It passes through the mastoid foramen and gives off three branches: the mastoid (a), hypoglossal (b), and jugular branches (c). The subgaleal or terminal segment (3) gives subgaleal and muscular branches and can give an inconstant terminal dural branch that enters the parietal foramen, called parietal branch (PB)

Branches of the Artery

The OccA is particular because it supplies the muscles of the neck, the dura of the posterior fossa, and also the peripheral cranial and cervical nerves [1, 8–10, 16]. With the aim to make the reading easier, this paragraph will be divided in muscular branches, dural branches, and neural branches.

Muscular Branches

Muscular branches of the OccA are present all along its course from the three segments (Table 2).

From the first segment, the OccA gives off 2–8 thin muscular branches to the sterno-cleido-mastoid, digastric, rectus

Table 2 Different muscular branches of the occipital artery with their respective anastomosis

Muscular branches	Segment of the OccA	Muscles supplied	Possible anastomosis
Superior sterno-cleido-mastoid artery	First	Sterno-cleido-mastoid muscle Digastric muscle (posterior belly) Rectus lateral M Inferior/superior oblique M	
Superficial descending branch	Second	Sterno-cleido-mastoid muscle Splenius capitis M	Ascending cervical artery
Deep descending branch	Second	Deep cervical muscles	Vertebral artery (C1–C2–C3) Vertebral artery (muscular branches)
"Horizontal" branches	Second	Splenius capitis muscle	Anterior and posterior ethmoidal arteries (OA) Contralateral MMA
Terminal branches	Third	Occipito-frontal fascia	Contralateral OccA Superficial temporal artery Posterior auricular artery

lateralis, inferior oblique, superior oblique, longissimus capitis muscles. When these branches share a common origin, it is named the superior sterno-mastoid artery [1, 2, 34].

From the second segment of the OccA, the most important muscular branches are the deep and superficial descending branches which participate in the supply of the cervical muscles [2]. In 75% of cases, these two arteries share a common trunk with a diameter of 1–1.3 mm. Other "horizontal" muscular branches from the second segment supply the splenius capitis and the sterno-cleido-mastoid muscles [2]. Thin osteo-articular branches of the second segment of the OccA supply the occipital condyle and the occipito-cervical junction [2].

From the third segment of the OccA, a large number (15–30) of thin muscular branches are dedicated to the occipito-frontal muscles.

Dural Branches

These dural branches of the OccA and their respective territory are described in detail in a dedicated chapter due to their clinical importance for dural arterio-venous fistulas and other dural pathologies. Dural branches of the OccA principally arise from the second segment of the artery. The largest dural branch is the mastoid artery that passes through the mastoid foramen [11]. This artery supplies the lateral part of the posterior fossa dura and gives off three distinct branches:

- The mastoid branch that courses superiorly after its entrance into the posterior fossa [1].
- The hypoglossal branch which has a descending course toward the hypoglossal canal [11].
- The jugular branch that is oriented to the jugular foramen [11].

Few authors also described dural branches that are transosseous branches from the second segment of the occipital artery [3, 11, 35]. Other dural branches from the OccA follow the radicular nerves of the first and second intervertebral spaces. The dural artery of the first intervertebral space could be larger and could give off the artery of the falx cerebelli (in 50% of cases) [1, 8]. In other cases, this artery could originate from the postero-inferior cerebellar artery or directly from the vertebral artery [1]. The posterior meningeal artery, normally a dural branch of the vertebral artery, could take its origin from the occipital artery and represents a partial persistence of the proatlantal artery of type I [8]. A case of posterior meningeal artery origin from the OccA is shown in Fig. 2.

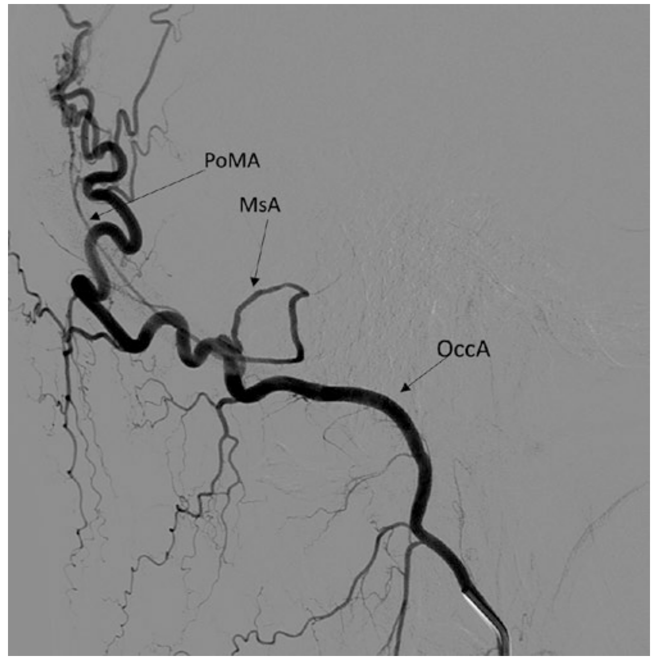


Fig. 2 Posterior meningeal artery origin from the OccA. Selective DSA injection of the OccA in lateral view. The mastoid artery (MsA) is visible arising from the second segment of the occipital artery (OccA) and entering the mastoid foramen. At the end of its course it gives origin to the posterior meningeal artery (PoMA)

Another inconstant dural branch originates from the terminal part of the OccA and passes through the parietal foramen, it is consequently named the parietal branch [11].

Neural Branches

During all its course, the occipital artery gives off thin branches for supplying peripheral lower cranial and upper cervical nerves [1, 10, 16]. The most important neural branch is the stylo-mastoid artery that enters the middle ear cavity through the stylo-mastoid foramen [8]. During its intracanalar course, this artery gives off little branches to the facial nerve that courses in the same canal. In 50% of cases, this artery can arise from the posterior auricular artery. A case of enlarged stylo-mastoid artery in the contest of a dural arterio-venous fistula is shown in Fig. 3. Other neural branches are organized as follow [16]:

- From the first segment of the OccA to the spinal accessory and hypoglossal nerves
- From the second segment of the OccA to the C1 and C2 nerves
- From the third segment of the OccA to the greater occipital nerve

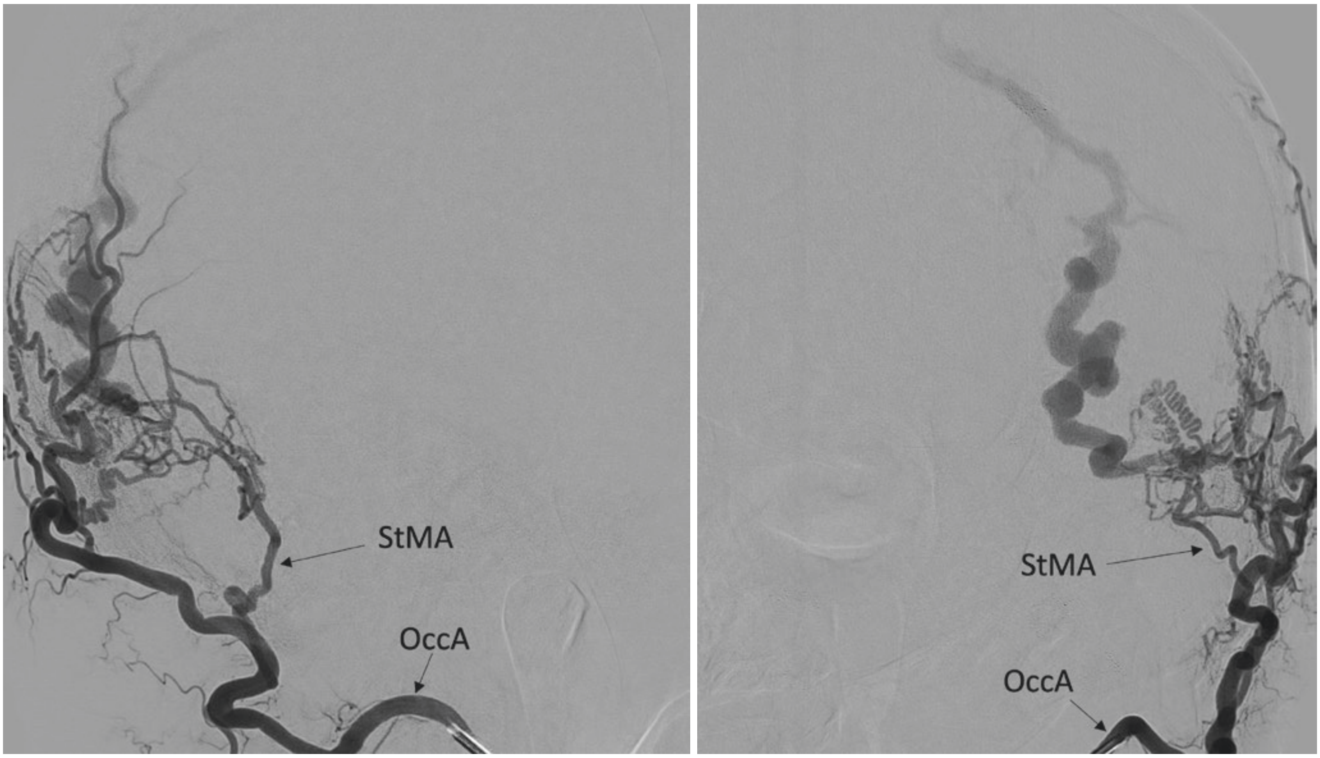


Fig. 3 Stylomastoid origin from the OccA. Selective OccA DSA injection in lateral and antero-posterior view. The stylo-mastoid artery (StMA) arises from the occipital artery (OccA) and has an enlarged configuration since it contributes to the arterial supply of a tentorial dural arterio-venous fistula

Possible Anastomoses

The occipital artery is particular for its important number and dangerous anastomosis especially with the vertebro-basilar system. The anastomoses formed between the OccA and other arteries are summarized in Table 3. Even if the presence of occipito-vertebral anastomosis can be seen in only 0.8% of DSA, they are almost always present in post-mortem analysis [6, 36–38]. With the aim to make these anastomoses more understandable, we classified them in extradural and dural anastomoses.

Extradural Anastomoses

The first artery of the OccA that gives anastomoses with other branches is the stylomastoid artery. These anastomoses are described in the chapter concerning the supply of the middle ear [1]. The stylomastoid artery penetrates the middle ear through the stylomastoid foramen and anastomoses with branches of the posterior auricular artery, with the petrosal branch of the middle meningeal artery and with the ascending pharyngeal artery (inferior tympanic artery) [8].

The occipital artery has an important network of anastomosis through its deep descending branches principally with the vertebral artery and with the ascending cervical artery [39–42]. Theses anastomoses represent the remnants of the distal part of the embryonic proatlantal artery. The most important anastomoses with the vertebral artery are at the first, second, and third intervertebral spaces. These anastomoses could be direct between the OccA and the vertebral artery or indirect through muscular branches of both principal trunks [8, 35, 43, 44]. Alvernia et al. (2005) classified these anastomoses in three distinct types based on their cadaveric study [2]:

- Type I are anastomoses between the deep descending branch of the OccA and the VA at the first intervertebral space (found in 18%)
- Type II between the superficial descending branch of the OccA and the VA at the second intervertebral space (found in 72% of cases)
- Type III between the superficial descending branch of the OccA and the VA at the third intervertebral space (found in only 10% of cases)

Clinical examples of type I and II anastomoses are shown in Fig. 4.

As extradural dural anastomosis, we also need to mention the subcutaneous anastomoses of the OccA during its termi-

Table 3 Different dural and neural branches of the occipital artery with their respective anastomosis

Occipital artery branches	Segment of the OccA	Territory (dural and neural)	Possible anastomosis
Stylomastoid artery	First	No dural territory VII	Posterior auricular artery Petrosquamosal branch (MMA) Inferior tympanic artery (AscPhaA)
Little branches	First	Lower cranial nerves (spinal XI, XII)	
Mastoid branch (superior ramus)	First	Petrosal dura (lateral part) Wall of sigmoid sinus Endolymphatic sac and duct	Petrosquamosal branch (MMA) MHT (internal carotid artery) Subarcuate artery (AICA)
Mastoid branch (inferior ramus)	First	Wall of jugular bulb Posterior part foramen magnum Wall of inferior petrosal sinus	Jugular branch (AscPhaA) Hypoglossal branch (AscPhaA)
Artery of the falx cerebelli	Second	Falx cerebelli	Posterior meningeal artery (vertebral artery) Artery of the falx cerebri (MMA)
Deep descending branch	Second	Posterior rami C1, C2, C3	Radicular branches vertebral artery
Parietal branch	Third	Parietal convexity dura	Parietal branch (MMA)
Little branches	Third	Greater occipital nerve	

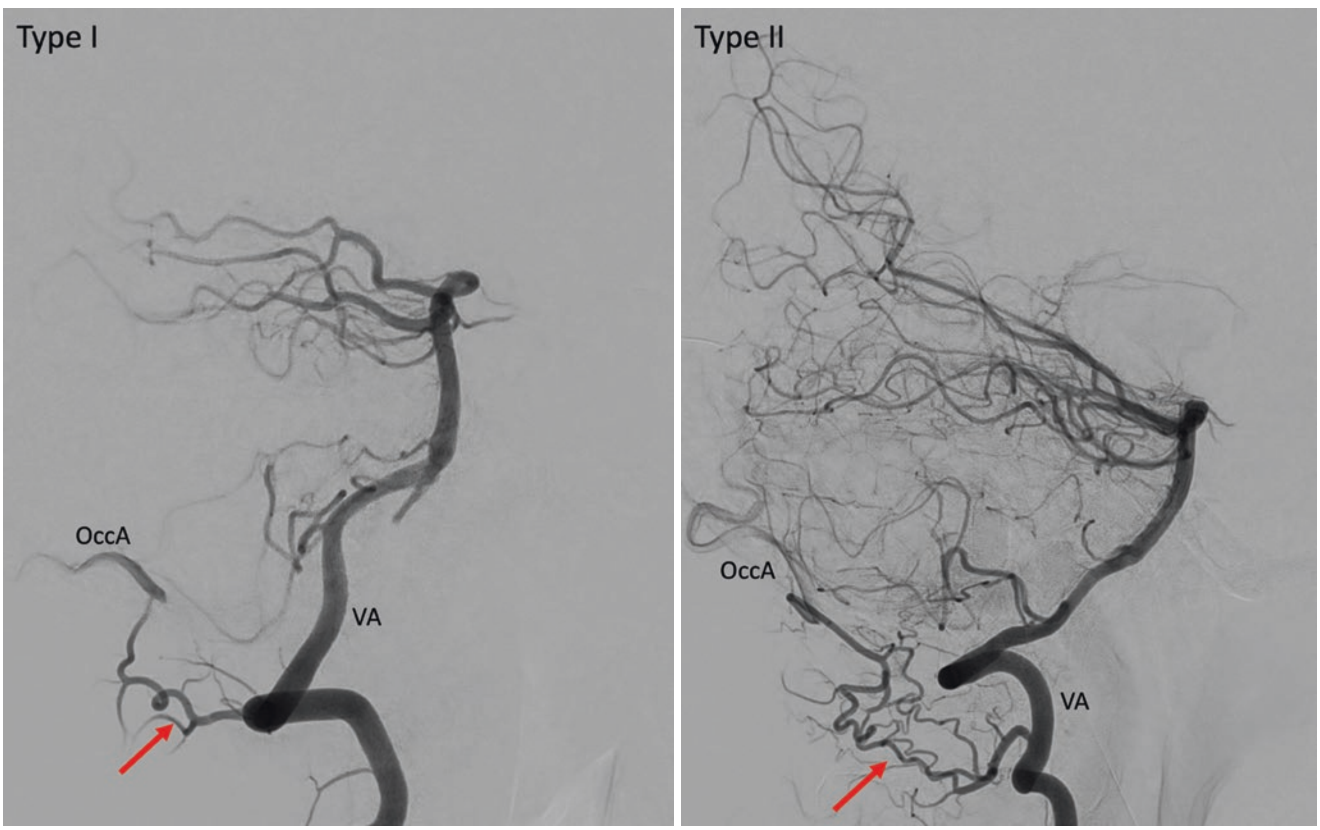


Fig. 4 Clinical cases of OccA-VA anastomoses type I and II. Lateral DSA after VA injection. The red arrows show the anastomoses between the occipital artery (OccA) and vertebral artery (VA). These anastomoses are located at the first intervertebral space in type I and at the second intervertebral space in type II

nal course (third segment) [2]. These anastomoses are with other subcutaneous arteries as the contralateral occipital artery, the posterior auricular artery, and the superficial temporal artery.

Dural Anastomoses

The occipital artery also has an important network with other dural arteries, its dural territory is always in balance with these other dural branches [3, 8, 11]. The most important anastomotic network is formed with dural branches of the vertebral artery in the region of the foramen magnum and in the dura of the cerebellar convexity [8, 40, 45]. Posterior meningeal artery and the artery of the falx cerebelli which could be branches of the OccA or of the vertebral artery represent an important zone of anastomosis between OccA and vertebral artery. The artery of the falx cerebelli also anastomoses at its upper end with the equivalent branch from the middle meningeal artery [1, 8–10, 16].

The OccA presents anastomoses through its mastoid branch with the ascending pharyngeal artery (jugular and hypoglossal branches), with the antero-inferior cerebellar artery (subarcuate artery) and with the internal carotid artery (through the meningo-hypophysary and infero-lateral trunks) [11].

Another anastomosis is present between the OccA and the middle meningeal artery through the parietal branch of the OccA (also called the supratentorial ramus) which enters the cranial cavity through the parietal foramen [11].

Supply Territory

The occipital artery has no parenchymal territory but presents an important supply for the upper cervical muscles, for the dura of the posterior fossa and gives some branches to peripheral cervical and cranial nerves.

Muscular Supply

During all its course, the occipital artery gives off branches provided for cervical and nuchal muscles. From the first segment of the OccA, the sterno-cleido-mastoid muscle, posterior belly of the digastric muscle, inferior oblique muscle, superior oblique muscle, and the longissimus capitis muscle are all supplied by the OccA [2]. The second segment of the OccA supply the posterior part of the sterno-cleido-mastoid muscle and the splenius capitis muscle. The third segment has only few thin branches to the posterior part of the occipito-frontal fascia [1, 8].

Dural Territory

The complete dural territory of the occipital artery is in the posterior fossa and variations are very frequent due to the sharing of this supply with different dural arteries [8]. The dural territory of the OccA is the lateral part of the cerebellopontine angle and the cerebellar fossa. Branches of the mastoid artery also supply the wall of the inferior petrosal sinus, the wall of the sigmoid sinus and jugular bulb [11]. The cerebellar fossa and the posterior edge of the foramen magnum are supplied by the occipital artery which is in balance with dural branches of the vertebral artery. Also, the terminal part of the occipital artery could have a dural supply on the parietal convexity (parietal branch) [8].

Peripheral Nerves Supply

A minor part of the occipital artery is dedicated in the supply of upper cervical nerves and of lower cranial nerves in their peripheral course. Indeed, branches from the first segment of the OccA supply the spinal accessory nerve and the hypoglossal nerve [1]. The stylomastoid artery supply the facial nerve into the stylomastoid canal. The occipital artery supplies posterior ramus of the upper cervical nerves of the first, second, and third intervertebral spaces. Finally, branches for supplying the greater occipital nerve come from the OccA in its terminal segment [2].

Variations Implicating the Occipital Artery

Few of these variations could be explained by the embryological development of the OccA and are summarized in Table 4.

Table 4 Different variations of the occipital artery with embryological explanation

Type	Embryological explanation
Persistence proatlantal artery type I	Complete persistence PA I
Persistence proatlantal artery type II	Complete persistence PA II
OccA-VA anastomosis at C1	Incomplete PPA I
OccA-VA anastomosis at C2	Incomplete PPA II
ICA origin OccA	Proximal part PA I or PA II
C1 VA origin of the OccA	Incomplete PPA I
C2 VA origin of the OccA	Incomplete PPA II
Common pharyngo-occipital trunk	Third aortic arch Hypoglossal artery and PA
OccA origin of PICA at C1	Incomplete PPA I
OccA origin of PICA at C2	Incomplete PPA II
Segmental cervical ICA agenesis + “cervical ICA” origin of OccA	Nonformation of the dorsal aorta (between third and second AA) Common pharyngo-occipital trunk

Persistence of the Proatlantal Artery

The complete persistence of the proatlantal artery (PPA) is a very rare persistent embryonic carotido-vertebral anastomosis, and its true incidence is not known, only 55 isolated cases were described. This interesting anatomical variation is exhaustively described in the Chapter “Carotido-Vertebral Anastomoses” and here is only a summary about PPA [1].

PPA corresponds to a carotid-vertebral anastomosis with the following criteria:

- Originates from the cervical ICA, the ECA or the CCA
- Joins the vertebral artery at the first or second cervical space
- PPA enters the skull through the foramen magnum and not through the hypoglossal canal that is the most reliable criteria
- Agenesis of the vertebral artery in 50% of cases

The majority of PPA cases are unilateral but not less than nine cases of bilateral PPA are described that represents 16% of cases and a proportion higher than for other carotido-vertebral anastomoses [46–54].

Almost all cases of PPA are asymptomatic, and occasional findings but two of them presented a neurological sign directly correlated to the PPA (one case of PPA aneurysm and one case of cochleo-vestibular nerve compression). The presence of a PPA could be associated with a lot of other pathology or anatomical variants as a bilateral ICA agenesis, a proximal ICA stenosis, vein of Galen malformation, AVM, dAVF, cerebral aneurysm, PTA, or duplicated MCA [25, 44, 52, 55–62].

Vertebral Artery Origin of the Occipital Artery (Fig. 5)

This anatomical variation could be assimilated to a partial persistence of the proatlantal artery. In this case, the distal (third segment) part of the OccA arises from the vertebral artery instead of from the ECA [36]. It can originate from a direct anastomosis (OccA-VA) at the first cervical space (remnant of the PPA type I) or at the second cervical space (remnant of the PPA type II).

Occipital Artery Origin of the Posterior Inferior Cerebellar Artery

This is a very rare incomplete persistence of the proatlantal artery type II. When the posterior inferior cerebellar artery (PICA) has an extradural origin at the level of C2, it could take its origin from the OccA instead of from the VA. Only two cases are described in the literature; one with angiographic demonstration and the other showed by cadaveric photographs [1, 63].

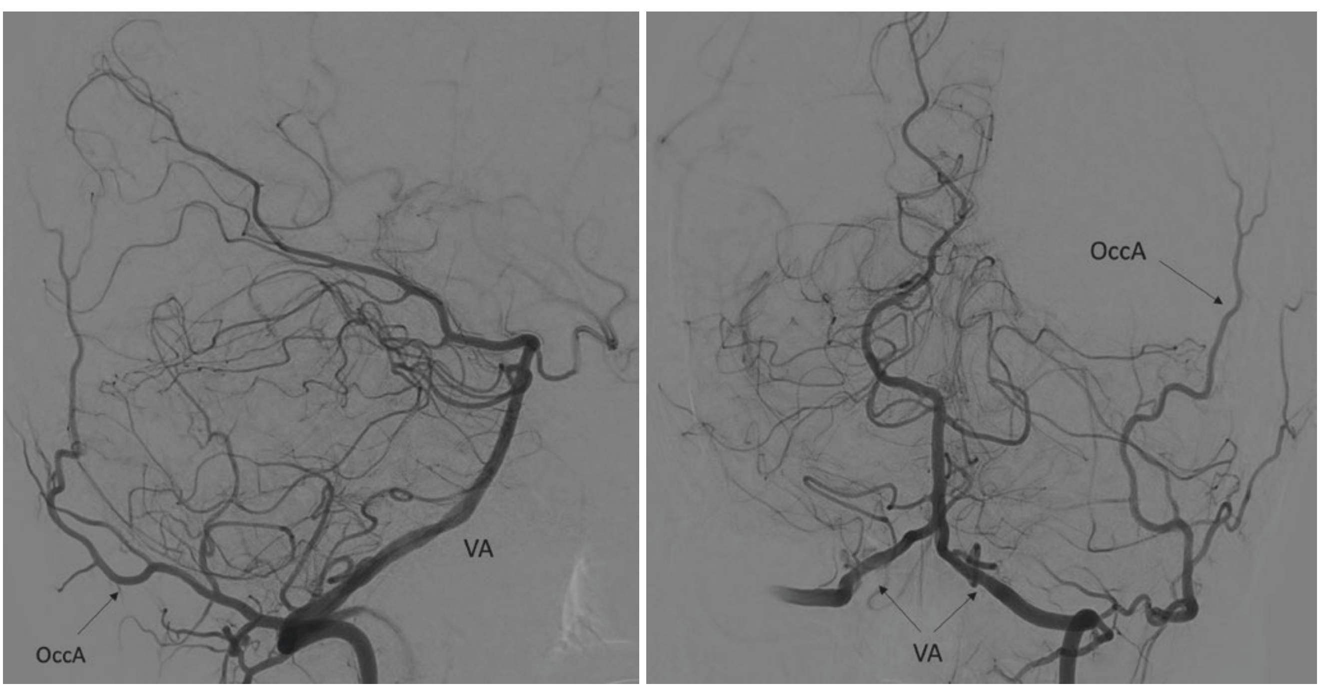


Fig. 5 Clinical case of OccA origin from the vertebral artery. Lateral and anterior-posterior DSA after VA injections. On the right side, the occipital artery (OccA) arises from the vertebral artery (VA) at the level of the first intervertebral space

Clinical Implications

A comprehensive knowledge in the anatomy of the occipital artery is mandatory for neck surgeons but also for interventional neuroradiologists and in few situations for neurosurgeons.

First, it is interesting to note that few isolated cases of pulsative tinnitus in cases of OccA origin of the vertebral artery or persistence of the proatlantal artery are described in the literature. This symptom, often difficult to explain, could be correlated in these situations with a high flow of the occipital artery.

In this last decade, the use of double-lumen catheter gives the possibility to use the occipital artery for liquid agent embolization in case of dural arterio-venous fistulas. Knowledge of all dangerous anastomoses with the vertebro-basilar system and "rules" for secure embolization through the OccA are mandatory for these treatments.

The occipital artery is used as donor for extracranial-intracranial bypasses. The OccA could be anastomosed to the postero-inferior cerebellar artery or with the posterior cerebral artery in cases of complex aneurysm trapping.

In few reports, a variation in origin of the OccA was described in situation of a carotid endarterectomy. It is of course important to know the different possible origins of the OccA and to modify the surgical management according to the anatomical variation.

References

1. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer; 2001.
2. Alvernia JE, Fraser K, Lanzino G. The occipital artery: a microanatomical study. Neurosurgery. 2006;58(1 Suppl):ONS114–22; discussion ONS-22. https://doi.org/10.1227/01.NEU.0000193519.00443.34.
3. Geibprasert S, Pongpech S, Armstrong D, Krings T. Dangerous extracranial-intracranial anastomoses and supply to the cranial nerves: vessels the neurointerventionalist needs to know. AJNR Am J Neuroradiol. 2009;30(8):1459–68. https://doi.org/10.3174/ajnr.A1500.
4. Guo Y, Chen H, Chen X, Yu J. Clinical importance of the occipital artery in vascular lesions: a review of the literature. Neuroradiol J. 2019;32(5):366–75. https://doi.org/10.1177/1971400919857245.
5. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–62.
6. Adams WE. On the possible homologies of the occipital artery in mammals, with some remarks on the phylogeny and certain anomalies of the subclavian and carotid arteries. Acta Anat (Basel). 1957;29(1–2):90–113. https://doi.org/10.1159/000141162.
7. Theron J, Lasjaunias P, Moret J, Merland JJ. Vascularization of the posterior fossa dura mater. J Neuroradiol. 1977;4(2):203–24.
8. Lasjaunias P, Theron J, Moret J. The occipital artery. Anatomy–normal arteriographic aspects–embryological significance. Neuroradiology. 1978;15(1):31–7. https://doi.org/10.1007/BF00327443.
9. Lasjaunias P, Doyon D. The ascending pharyngeal artery and the blood supply of the lower cranial nerves. J Neuroradiol. 1978;5(4):287–301.
10. Lasjaunias P, Moret J, Theron J. The so-called anterior meningeal artery of the cervical vertebral artery. Normal radioanatomy and anastomoses. Neuroradiology. 1978;17(1):51–5. https://doi.org/10.1007/BF00345270.
11. Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A Jr. Microsurgical anatomy of the dural arteries. Neurosurgery. 2005;56(2 Suppl):211–51; discussion 51. https://doi.org/10.1227/01.neu.0000144823.94402.3d.
12. Iyer AA. Some anomalies of origin of the vertebral artery. J Anat. 1927;62(Pt 1):121–2.
13. Moffat DB, Morris ED. Abnormal origin of the basilar artery from the cervical part of the internal carotid and its embryological significance. Anat Rec. 1956;125(4):701–11. https://doi.org/10.1002/ar.1091250404.
14. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: a pictorial review. Surg Radiol Anat. 2021;43(6):961–73. https://doi.org/10.1007/s00276-021-02684-y.
15. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
16. Lasjaunias P, Manelfe C. Arterial supply for the upper cervical nerves and the cervicocarotid anastomotic channels: systematization of radiological anatomy. Neuroradiology. 1979;18(3):125–31. https://doi.org/10.1007/BF00346866.
17. Keser N, Kula O, Is M, Yardimcioglu I. Prevalence of anomalously originating occipital artery in a group of Turkish individuals: a retrospective study using angiography. Turk Neurosurg. 2019;29(3):335–9. https://doi.org/10.5137/1019-5149.JTN.23232-18.1.
18. Lasjaunias P, Moret J. The ascending pharyngeal artery: normal and pathological radioanatomy. Neuroradiology. 1976;11(2):77–82. https://doi.org/10.1007/BF00345017.
19. Aggarwal NR, Krishnamoorthy T, Devasia B, Menon G, Chandrasekhar K. Variant origin of superior thyroid artery, occipital artery and ascending pharyngeal artery from a common trunk from the cervical segment of internal carotid artery. Surg Radiol Anat. 2006;28(6):650–3. https://doi.org/10.1007/s00276-006-0145-5.
20. Cohen JE, Leker RR, Moshe Gomori J, Itshayek E. Pharyngo-occipital artery variant arising proximal to occluded internal carotid artery: the risk of an unnecessary endarterectomy. J Clin Neurosci. 2014;21(3):529–31. https://doi.org/10.1016/j.jocn.2013.06.007.
21. Graham RB, Sugrue PA, Rahme RJ, Batjer HH, Bendok BR. Pharyngo-occipital artery variant arising from the internal carotid artery impacting surgical technique during carotid endarterectomy. J Neurointerv Surg. 2013;5(3):e14. https://doi.org/10.1136/neurintsurg-2011-010215.
22. Ustunsoz B, Gumus B, Koksal A, Koroglu M, Akhan O. Missed total occlusion due to the occipital artery arising from the internal carotid artery. Cardiovasc Intervent Radiol. 2007;30(1):116–7. https://doi.org/10.1007/s00270-006-0060-5.
23. Uchino A, Saito N, Mizukoshi W, Okada Y. Anomalous origin of the occipital artery diagnosed by magnetic resonance angiography. Neuroradiology. 2011;53(11):853–7. https://doi.org/10.1007/s00234-010-0825-z.
24. Arraez-Aybar LA, Navia-Alvarez P, Mendez-Cendon JC. A case of a type II proatlantal artery with arteriovenous malformation. Surg Radiol Anat. 2011;33(1):85–9. https://doi.org/10.1007/s00276-010-0685-6.
25. Berger L, Gouicem D, Lebied el D, Felisaz A, Coffin O, Maiza D. Internal carotid and ipsilateral type II proatlantal artery stenoses causing simultaneous hemispheric and vertebrobasilar transient ischemia. J Vasc Surg. 2011;53(2):475–7. https://doi.org/10.1016/j.jvs.2010.08.017.
26. Iwai T, Izumi T, Inoue T, Maegawa J, Fuwa N, Mitsudo K, et al. Occipital artery arising from the anterior aspect of the internal carotid artery identified by three-dimensional computed tomog-

raphy angiography. Iran J Radiol. 2012;9(2):103–5. https://doi.org/10.5812/iranjradiol.7809.
27. Sundick SA, Weaver M, Faries PL, Marin M. Aberrant origin of occipital artery proximal to internal carotid artery stenosis. J Vasc Surg. 2014;59(1):244. https://doi.org/10.1016/j.jvs.2013.06.089.
28. Cetin N, Akkan K, Ucar M, Onal B, Ilgit E. Bilateral occipital arteries of internal carotid origin: report of a case and review of the literature. J Belg Soc Radiol. 2015;99(1):69–71. https://doi.org/10.5334/jbr-btr.828.
29. Ozgur O, Sindel M, Hizay A, Ozturk S, Aytac G, Sindel T. Occipital artery arising from the internal carotid artery: a case report. Surg Radiol Anat. 2017;39(2):219–22. https://doi.org/10.1007/s00276-016-1710-1.
30. Uchino A, Saito N. Occipital artery arising from the cervical internal carotid artery at the level of the C2 vertebral body: three cases detected utilizing magnetic resonance angiography. Surg Radiol Anat. 2020;42(7):831–4. https://doi.org/10.1007/s00276-020-02460-4.
31. Matsuda I, Handa J, Handa H, Mizuno H. Bilateral anomalous occipital artery of internal carotid origin: case report. Nihon Geka Hokan. 1977;46(1):57–61.
32. Uchino A, Saito N, Okano N, Kakehi Y. Aberrant internal carotid artery associated with occipital artery arising from the internal carotid artery. Surg Radiol Anat. 2015;37(9):1137–40. https://doi.org/10.1007/s00276-015-1451-6.
33. Gurbuz J, Cavdar S, Ozdogmus O. Trifurcation of the left common carotid artery: a case report. Clin Anat. 2001;14(1):58–61. https://doi.org/10.1002/1098-2353(200101)14:1<58::AID-CA1011>3.0.CO;2-M.
34. Keser N, Avci E, Soylemez B, Karatas D, Baskaya MK. Occipital artery and its segments in vertebral artery revascularization surgery: a microsurgical anatomic study. World Neurosurg. 2018;112:e534–e9. https://doi.org/10.1016/j.wneu.2018.01.073.
35. Alexander MD, English J, Hetts SW. Occipital artery anastomosis to vertebral artery causing pulsatile tinnitus. BMJ Case Rep. 2013;2013:bcr2012010632. https://doi.org/10.1136/bcr-2012-010632.
36. Richter HR. Collaterals between the external carotid artery and the vertebral artery in cases of thrombosis of the internal carotid artery. Acta Radiol. 1953;40(2–3):108–12. https://doi.org/10.3109/00016925309176574.
37. Salamon G, Grisoli J, Paillas JE, Faure J, Giudicelli G. Arteriographic study of the meningeal arteries. (Role of selective injections, of subtraction and of radio-anatomical correlations). Neurochirurgia (Stuttg). 1967;10(1):1–19. https://doi.org/10.1055/s-0028-1095333.
38. Salamon G, Guerinel G, Demard F. Radioanatomical study of the external carotid artery. Ann Radiol (Paris). 1968;11(3):199–215.
39. Schechter MM. The occipital-vertebral anastomosis. J Neurosurg. 1964;21:758–62. https://doi.org/10.3171/jns.1964.21.9.0758.
40. Conforti P, Armenise B, Galligioni F. Anomalous carotid-vertebral anastomosis: primitive cervical segmental artery. Neurochirurgia (Stuttg). 1966;9(3):99–106. https://doi.org/10.1055/s-0028-1095361.
41. Hackett ER, Wilson CB. Congenital external carotid-vertebral anastomosis. A case report. Am J Roentgenol Radium Ther Nucl Med. 1968;104(1):86–9. https://doi.org/10.2214/ajr.104.1.86.
42. Tsai FY, Mahon J, Woodruff JV, Roach JF. Congenital absence of bilateral vertebral arteries with occipital-basilar anastomosis. Am J Roentgenol Radium Ther Nucl Med. 1975;124(2):281–6. https://doi.org/10.2214/ajr.124.2.281.
43. Pasco A, Papon X, Bracard S, Tanguy JY, Ter Minassian A, Mercier P. Persistent carotid-vertebrobasilar anastomoses: how and why differentiating them? J Neuroradiol. 2004;31(5):391–6. https://doi.org/10.1016/s0150-9861(04)97022-8.
44. Briganti F, Tortora F, Elefante A, Volpe A, Maiuri F. Persistent carotid-vertebral anastomosis associated with contralateral accessory middle cerebral artery. Surg Radiol Anat. 2005;27(5):450–4. https://doi.org/10.1007/s00276-005-0002-y.
45. Legre J, Tapias PL, Nardin JY, Philip E, Perier E. Embryonic intersegmental anastomosis between the external carotid and vertebral arteries. Classification problems of the proatlantal artery. J Neuroradiol. 1980;7(2):97–104.
46. Gumus T, Onal B, Ilgit ET. Bilateral persistence of type 1 proatlantal arteries: report of a case and review of the literature. AJNR Am J Neuroradiol. 2004;25(9):1622–4.
47. Lode I. Persistence of both proatlantal arteries with occlusion of the internal carotid arteries. A case report. Acta Radiol. 2000;41(5):410–1. https://doi.org/10.1080/028418500127345758.
48. Lui CC, Liu YH, Wai YY, Tsai CC. Persistence of both proatlantal arteries with absence of vertebral arteries. Neuroradiology. 1987;29(3):304–5. https://doi.org/10.1007/BF00451775.
49. Okumura A, Lee T, Shimojima K, Hisata K, Shoji H, Takanashi J, et al. Brainstem disconnection associated with nodular heterotopia and proatlantal arteries. Am J Med Genet A. 2009;149A(11):2479–83. https://doi.org/10.1002/ajmg.a.33044.
50. Pelz D, Menon RK, Swartz RH, Aviv RI, Symons SP. Bilateral type II persistent Proatlantal intersegmental arteries. Can J Neurol Sci. 2013;40(6):873–4. https://doi.org/10.1017/s031716710001605x.
51. Pulli R, Gatti M, Pratesi C. Carotid endarterectomy in rare bilateral carotid-vertebral anastomosis. J Vasc Surg. 2001;33(5):1122–4. https://doi.org/10.1067/mva.2001.113975.
52. Purkayastha S, Gupta AK, Varma R, Kapilamoorthy TR. Proatlantal intersegmental arteries of external carotid artery origin associated with Galen's vein malformation. AJNR Am J Neuroradiol. 2005;26(9):2378–83.
53. Woodcock RJ, Cloft HJ, Dion JE. Bilateral type 1 proatlantal arteries with absence of vertebral arteries. AJNR Am J Neuroradiol. 2001;22(2):418–20.
54. Zarghouni M, Marichal D. Persistent bilateral proatlantal type II artery. Proc (Baylor Univ Med Cent). 2013;26(1):50–1. https://doi.org/10.1080/08998280.2013.11928918.
55. Tanaka Y, Hara H, Momose G, Kobayashi S, Kobayashi S, Sugita K. Proatlantal intersegmental artery and trigeminal artery associated with an aneurysm. Case report. J Neurosurg. 1983;59(3):520–3. https://doi.org/10.3171/jns.1983.59.3.0520.
56. Capel C, Peltier J, Lefranc M, Desenclos C, Chivot C, Deramond H, et al. Type II proatlantal artery with unusual origin of the occipital artery feeding of a type IV dural arteriovenous fistula: case report. Neurochirurgie. 2013;59(6):214–7. https://doi.org/10.1016/j.neuchi.2013.08.007.
57. Berman IG, Stuckey SL. Anomalous communication of external carotid and vertebral arteries with associated intracranial parenchymal arteriovenous malformation: magnetic resonance angiography and angiographic findings. Australas Radiol. 2003;47(4):472–4. https://doi.org/10.1046/j.1440-1673.2003.01225.x.
58. Parkinson D, Reddy V, Ross RT. Congenital anastomosis between the vertebral artery and internal carotid artery in the neck. Case report. J Neurosurg. 1979;51(5):697–9. https://doi.org/10.3171/jns.1979.51.5.0697.
59. Bour P, Bracard S, Frisch N, Frisch R, Fieve G. Persistent proatlantal artery associated with carotid artery stenosis treatment by percutaneous transluminal balloon angioplasty. Ann Vasc Surg. 1991;5(1):38–40. https://doi.org/10.1007/BF02021775.
60. Grego F, Stramana R, Lepidi S, Antonello M, Bonvini S, Zaramella M, et al. Primitive proatlantal intersegmental artery and carotid end-

arterectomy. J Vasc Surg. 2004;39(3):691. https://doi.org/10.1016/s0741-5214(03)00712-2.

61. Ouriel K, Green RM, DeWeese JA. Anomalous carotid-basilar anastomoses in cerebrovascular surgery. J Vasc Surg. 1988;7(6):774–7. https://doi.org/10.1067/mva.1988.avs0070774.

62. Basekim CC, Silit E, Mutlu H, Pekkafali MZ, Ozturk E, Kizilkaya E. Type I proatlantal artery with bilateral absence of the external carotid arteries. AJNR Am J Neuroradiol. 2004;25(9):1619–21.

63. Tayebi Meybodi A, Lawton MT, Benet A. Dual origin of extradural posterior inferior cerebellar artery from vertebral and occipital arteries: anatomic case report. Oper Neurosurg (Hagerstown). 2015;11(4):564–8. https://doi.org/10.1227/NEU.0000000000000923.

Embryology and Variations of the Ascending Pharyngeal Artery

Thomas Robert and Sara Bonasia

The ascending pharyngeal artery (APhA) is one of the most complex and interesting branches of the external carotid artery (ECA) [1]. The complexity of the APhA could be explained by three distinct reasons. The first one is the discrepancy between its small diameter and its important function [2]. The second reason is the variety of its territories of supply that include dura mater, peripheral nerves, bones, mucosa, and muscles. The only compartment that is not supplied by the APhA is the skin [3]. The third interesting particularity of the APhA is its numerous anastomoses with all other vascular systems: cervical, vertebral, and carotid [4]. In this chapter, we propose a comprehensive anatomical description of the APhA and of its variations after a reminder of the pharyngo-occipital system embryology.

History

Due to its little size and to its course along the internal carotid artery (ICA) under the skull base, the APhA was not well studied until the 1970s. The first authors that described the APhA were Manelfe et al. (1971) and Salamon et al. (1967) with their important angiographic works [5–7]. On the other hand, different key papers about the embryology and about the phylogeny allows to understand the complexity of the APhA [8–11]. As for other cranio-facial arteries, the masterpiece work of D. Padget (1948) allows to light up our actual knowledge about the complex embryology of the APhA [11]. With the increased amount of interest due to the emergence of ECA branches embolization, the number of publications concerning the APhA raised after the 1980s [1, 4, 12, 13]. Lasjaunias, as for other cranio-facial arteries, was a pioneer during this period and gave important information about the embryology and variations of all cranio-facial arteries [3, 10]. After him, a lot of case reports describing variations of the APhA were based on his publications. These last 20 years, a lot of fascinating papers were published by neuroradiologists as well as by anatomists: on one hand for the attention to the dangerous anastomoses between the APhA and other arteries for arterial embolization; on the other hand with the aim to prevent catastrophic complications for neck surgeries [4, 14–22].

Embryological Development

In this chapter, only notions about the embryological development that allow to understand the pharyngo-occipital system will be developed (Table 1). For more detailed embryological development about the vertebral artery, the carotid system, and the carotido-vertebral anastomoses, we send you to the respective chapters.

Early in the embryological life (stage I of Padget, embryos of 4–5 mm), the formation of the vertebro-basilar system starts with the presence of paired longitunal arteries named “longitudinal neural arteries (LNA)” [11]. These LNAs are supplied by the carotid system through different carotido-vertebral anastomosis and the vertebral arteries are not developed yet. LNAs are supplied by the trigeminal artery, the otic artery, the hypoglossal artery, and the proatlantal artery from cranial to caudal [9, 10].

During the stage II of Padget (embryos of 5–6 mm), the caudal division of the primitive internal carotid artery anastomoses with the cranial part of the LNA (formation of the posterior communicating artery), and consequently the carotido-vertebral anastomoses initiate their involution. The first two arteries that regress, are the otic and the hypoglossal arteries, and the LNAs are supplied principally by the trigeminal artery cranially and the proatlantal artery caudally.

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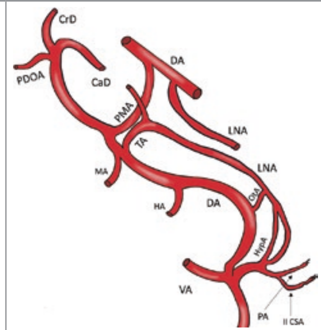
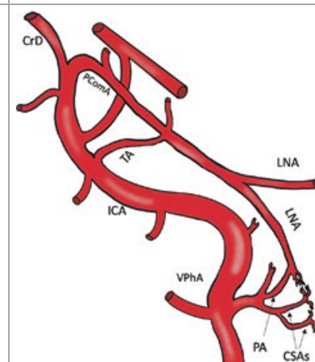
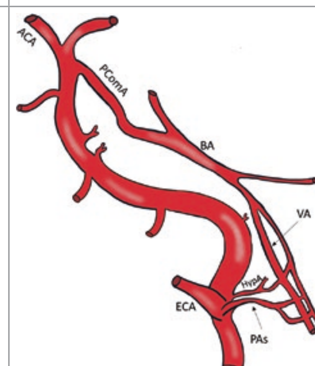
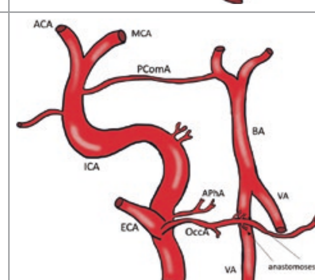
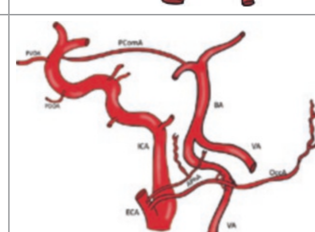
Table 1 Major embryological changes in the formation of the occipito-pharyngeal system

Stage	Embryo size (mm)	Major evolutions	Graphic illustration
I	4–5	– Paired longitudinal neural arteries (LNA) – Trigeminal (TA), otic (OtA), hypoglossal (HypA), proatlantal (PA), and second cervical segmental artery (II CSA) – PComA not formed	
II	5–6	– Formation of the PComA – Regression of otic and hypoglossal arteries – Fusion of LNAs in basilar artery (initiation) – Initial transverse anastomoses between the intersegmental arteries	
III	7–12	– Basilar artery (BA) completely formed – PComA completely formed – Regression of trigeminal and proatlantal arteries – Formation of the vertebral artery VA (initiation)	
IV	13–15	– Migration of OccA origin on the external carotid artery (ECA) – Persistence of the anastomoses between OccA and VA	
V	16–18	– Complete formation of the VA – Complete formation of the OccA (remnant of the proatlantal artery) – Complete formation of the ascending pharyngeal artery (APhA, remnant of the hypoglossal artery)	

At the same time, the two LNAs come closer to the midline to fuse together [9, 11].

At stage III of Padget (embryos of 7–12 mm), the basilar artery is completely formed by fusion of the two LNAs as well as the posterior communicating artery which is anastomosed with the upper part of the basilar artery. Trigeminal and proatlantal arteries progressively regress, and the primitive vertebral artery is formed by transverse anastomoses between the six upper cervical intersegmental arteries [8, 10, 11].

The APhA is considered as the remnant of the hypoglossal artery. This is the reason why P. Lasjaunias named it the *artery of the first cervical somite* [3, 23]. Between stages III and V, the origin of the APhA shifts from the primitive ICA to the ECA, and the distal part of the primitive hypoglossal artery leaves only the territory of the hypoglossal branch of the APhA. At the stage V (embryos of 16–18 mm), the definitive ascending pharyngeal artery could be clearly individualized [10, 24–26].

Origin of the Artery

The APhA normally arises from the postero-lateral aspect of the ECA, but its variability is very high, and it is not infrequent to see a variation in origin of the APhA [2, 3]. In literature, the rate of "normal origin" of the APhA varies from 65% to 95% and essentially depends on the technique of the study (cadaveric or angiographic observations) [2, 3, 27]. The other possible origins of the APhA are the occipital artery, internal carotid artery, the facial artery, the lingual artery, or directly from the common carotid artery (CCA) [14, 18, 28].

The most frequent variation is the ICA origin of the APhA that is largely described in the neuroradiological and surgical literature. The frequency of this variation is comprised between 4% and 8% but most of these studies are of small number of cases [14]. The most reliable study is based on the review of 2500 CT scans, and the authors found a rate of ICA origin of the APhA at 6.25% [20]. In this study, the mean distance between the CCA bifurcation and the origin of the APhA is 4 mm [20]. This variation is explained embryologically by the absence of shifting of the APhA origin from the ICA to the ECA between stages III and IV [10]. A clinical case of this variant is presented in Fig. 1.

The APhA could also share its origin with one or more other branches of the ECA. A common origin between the APhA and the occipital artery (OccA) is the most frequent and is not surprising since they develop from the same embryological system [21, 29]. The incidence of this common origin APhA-OccA is between 0.2% and 9% [18, 29]. The common trunk could branch off from the ECA or usually from the ICA [17, 30].

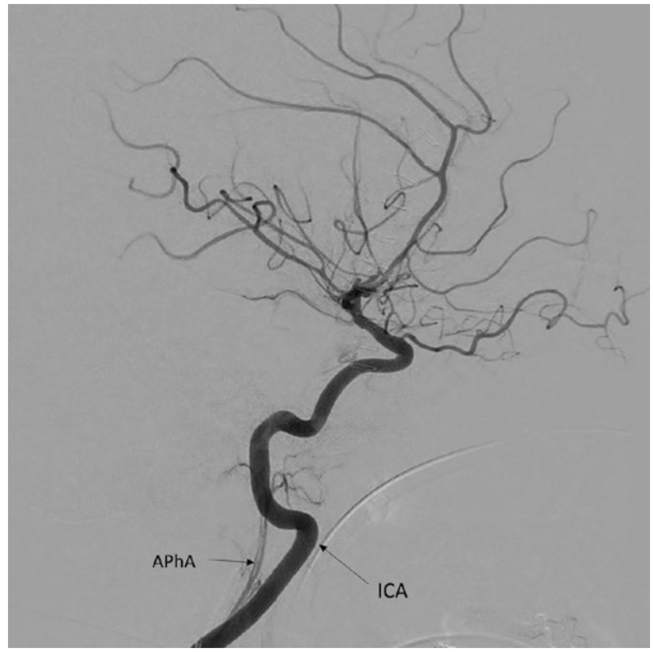


Fig. 1 Clinical case of ascendent pharyngeal artery origin from the internal carotid artery. Lateral DSA obtained after internal carotid artery (ICA) injection. The ascendent pharyngeal artery (APhA) arises from the ICA instead of the external carotid artery

Other different configurations in the branching from the ECA were described but are less frequent and correspond to case reports or little series. A common origin between facial artery, lingual artery, and APhA from the ECA was described in the series of Al-Rafiah (2011) [27]. Aggarwal (2006) showed another interesting disposition with an APhA that shares its origin form the ICA with the OccA and the superior thyroid artery [31]. On the other hand, Geraci (2009) described a case of common origin APhA-superior thyroid artery from the CCA [32].

The APhA could also have a double origin from the ECA, its anterior and posterior trunks have two distinct origins. These cases were described by Nitescu (1977), Devadas (2018) and Al-Rafiah (2011) [27, 33, 34].

P.Lasjaunias (2001), in its textbook, also showed a very interesting case of ascending cervical artery origin of the APhA and takes it as example to illustrate the independence of embryological development between the pharyngo-occipital system and the ventral pharyngeal artery (future ECA) [10].

Course of the Artery

The APhA is one of the littlest branches of the ECA, its mean diameter at its origin from the ECA is 1.54 mm (1.1–2.1 mm) [1, 2, 27]. The APhA almost always divides in two distinct branches, the pharyngeal trunk (anterior branch) and the neuromeningeal trunk (posterior branch) [1, 3]. The mean length of the APhA from its origin to its bifurcation is 17.8 mm (8–30 mm) [1]. The neuromeningeal trunk has a mean diameter of 0.93 mm (0.5–1.6 mm) [2, 27, 33].

From its origin, the APhA has a superior course medial to the ICA and postero-lateral to the wall of the pharynx. Medially to the APhA, the glossopharyngeal nerve and the superior cervical ganglion are in strait relation with the artery where it gives its bifurcation [2].

Branches of the Artery

In this chapter, all branches of the APhA are cited and described but a more detailed description of its dural branches and territory will be find in the chapter "Dural Branches of the Ascending Pharyngeal Artery."

No consensus in the name of the APhA branches and in its branching could be found in literature. With the aim to limit confusion in this chapter, we will describe the branches of the APhA in a two-trunk configuration: the anterior or pharyngeal trunk and the posterior or neuromeningeal trunk. The main branches of the APhA are illustrated in Fig. 2. Figure 3 also shows a clinical case with the main trunks of the APhA.

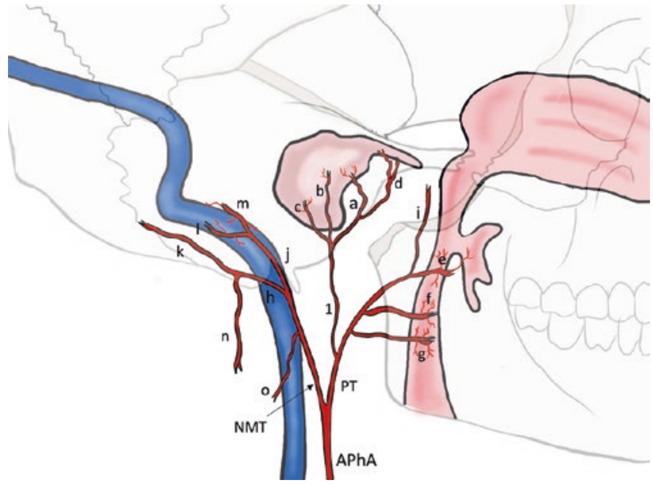


Fig. 2 Illustration of the branches of the ascendent pharyngeal artery (APhA). The main trunk of the APhA divides into an anterior pharyngeal trunk (PT) and a posterior neuro-meningeal trunk (NMT). The PT gives off the inferior tympanic artery (1), which branches into anterior (a), ascendent (b) and posterior trunks (c). The anterior trunk gives origin to a branch for the Eustachian tube (d). The PT also gives origin to the superior (e) I, middle (f), and inferior (g) pharyngeal branches. The artery of the foramen lacerum (i) arises from the superior pharyngeal trunk. The NMT divides into a hypoglossal trunk (h) and a jugular trunk (j). The former gives origin to an ascendent (k) and a descendent (n) ramus. The latter branches into a medial (m) and a lateral (l) artery. The musculo-spinal artery (o) also usually arises from the NMT

Pharyngeal Trunk (Anterior)

The anterior or pharyngeal trunk presents three distinct rami for the pharyngeal mucosa supply: inferior, middle, and superior [3, 10]. Other than these three rami, the anterior trunk of the APhA gives off a dural branch (usually from the superior pharyngeal ramus) named the *branch of the foramen lacerum* or *carotid ramus* that enters the skull base by the foramen of the same name [5, 6, 35]. This APhA branch is inconstant and found in 85% of cases [1].

The inferior tympanic artery (ITA) can arise as a branch of the anterior trunk or as a middle trunk directly from the APhA [36]. The ITA has a superior course and accompanies the tympanic branch of the glosso-pharyngeal nerve into the Jacobson's canal (inferior tympanic canal) [16, 37]. Moret et al. (1982) has angiographically analyzed all branches of the middle ear and described three branches from the ITA: anterior, ascending, and posterior [36]. The anterior trunk also gives off from its superior branch a little ramus to the Eustachian tube supply in 75% of cases and is consequently named the *anterior ramus for the Eustachian tube* [1, 35].

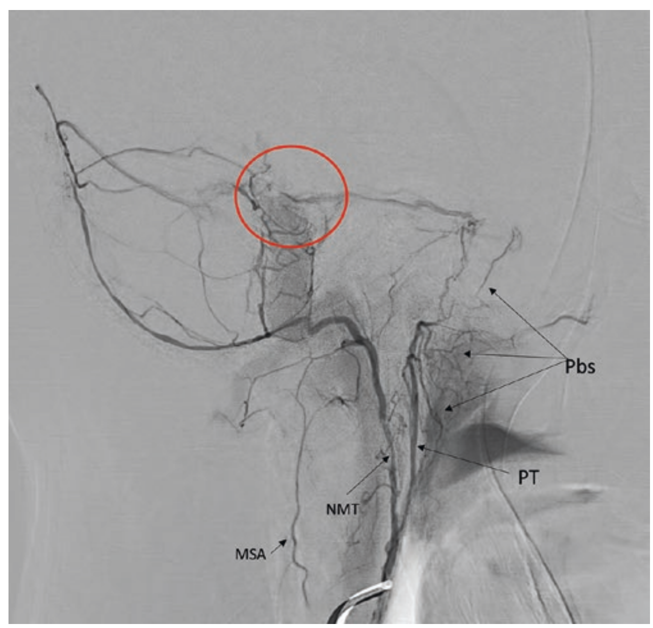


Fig. 3 Clinical case of dAVF with main trunks of the APhA. In the pathologic contest of a dural arterio-venous fistula (red circle), the main trunks of the ascendent pharyngeal artery are well visible. The pharyngeal trunk (PT) courses anteriorly and gives rise to pharyngeal branches (Pb). The neuromeningeal trunk (NMT) courses posteriorly and gives origin to the muscolo-spinal artery (MSA)

Neuromeningeal Trunk (Posterior)

The posterior or neuromeningeal trunk arises from the APhA in 85% but could also be a branch of the occipital artery (10%) or of the posterior auricular artery (5%) [1, 2]. Its two branches are the jugular branch and the hypoglossal branch [35]. The former enters the skull through the jugular foramen and the latter one through the hypoglossal canal (anterior condyloid foramen). The jugular branch gives off a medial branch that courses along the inferior petrosal sinus and a lateral branch for the petrosal surface. The hypoglossal branch is composed by an ascending branch that follows the clivus and a descending branch that participates in the odontoid arterial arch [1, 35].

The musculo-spinal artery, also known as the prevertebral branch or as the posterior muscular branch, is another branch of the APhA that can arise from the posterior trunk or as an independent branch. This artery usually originates from the posterior part of the APhA and has a descending course along the anterior part of the cervical column ending at the C4 level. It is also considered as one of the odontoid arch arteries [3, 37].

Possible Anastomoses

One of the characteristics of the APhA is its complex and high number of arterio-arterial anastomoses with the vertebral, cervical, and carotid systems [4]. With the aim of a higher clarity of this paragraph, anastomoses will be described as extradural or dural ones. All anastomoses of the APhA are also summarized in the Tables 2, 3 and 4.

Extradural Anastomosis (Table 2)

- In the pharyngeal mucosa, the three rami of the APhA are anastomosed with their respective counterpart [3, 10]. The superior pharyngeal ramus also anastomoses with the prevertebral artery and indirectly with the vertebral artery. The middle pharyngeal ramus has anastomosis with the ascending palatine artery (facial artery branch) and the inferior pharyngeal ramus with the superior laryngeal artery (superior thyroid artery branch) [1].
- The inferior tympanic artery, after entering the middle ear cavity through the Jacobson's canal, has anastomoses with the carotico-tympanic artery (petrous ICA branch), the superior tympanic artery (middle meningeal artery), and the stylomastoid artery (posterior auricular artery) [36].
- The anterior ramus for the Eustachian tube also has anastomoses with the pterygo-palatine artery (internal maxillary artery) and with the accessory meningeal artery (branch of the IMA in 65% or of the MMA in 35%) [2, 33].
- The musculo-spinal artery is the APhA's branch that presents the most complex and dangerous anastomotic network with other artery due to its participation in the odontoid arterial arch [25, 38]. This branch anastomoses with the vertebral artery through radicular arteries at C2 and C3, with the occipital artery at C1 and C2 foramina, with the ascending cervical artery at C4 and with the deep cervical artery [26]. This complex anastomotic odontoid arch will be developed in another chapter.

Table 2 Different extradural branches of the APhA with their respective anastomosis

Extradural branches	Trunk	Territory	Possible anastomosis
Superior pharyngeal branch	Anterior	Pharyngeal mucosa, naso-pharynx	Contralateral APhA Odontoid arch (vertebral artery)
Middle pharyngeal branch	Anterior	Pharyngeal mucosa, oro-pharynx	Contralateral APhA Ascending palatine artery (facial artery)
Inferior pharyngeal branch	Anterior	Pharyngeal mucosa, fossa of Rosenmüller	Contralateral APhA Superior laryngeal artery (superior thyroid artery)
Inferior tympanic artery	Anterior	Middle ear, staples, promontory	Carotico-tympanic artery (petrous ICA) Superior tympanic artery (MMA) Stylo-mastoid artery (posterior auricular artery)
Anterior ramus for the Eustachian tube	Anterior	Eustachian tube (anterior part)	Pterygo-palatine artery (internal maxillary artery) Accessory meningeal artery
Musculo-spinal artery	Posterior	Prevertebral muscles	Radicular branch (vertebral artery) Descending branch (occipital artery) Ascending cervical artery Deep cervical artery

Table 3 Different dural branches of the APhA with their respective anastomosis

APhA branches	Trunk	Dural territory	Possible anastomosis
Branch of the foramen lacerum (carotid ramus)	Anterior	Foramen lacerum, carotid canal, carotid artery wall	Recurrent artery of the foramen lacerum (cavernous ICA) Infero-lateral trunk (cavernous ICA) Cavernous branch (MMA) Accessory meningeal artery
Jugular branch	Posterior	Jugular foramen, wall inferior petrosal sinus, wall jugular bulb, wall sigmoid sinus, dura petrosal surface	Petro-squamous branch (MMA) Subarcuate artery (AICA) Lateral clival artery (ICA) Meningeal branch (occipital artery)
Hypoglossal branch	Posterior	Antero-lateral part foramen magnum, clivus, cerebellar fossa	Anterior meningeal artery (vertebral artery) Medial clival artery (ICA) Meningeal branch (occipital artery)

Table 4 Peripheral nerves supply by the APhA

APhA branches	Trunk	Neural supply
Branch of the foramen lacerum (carotid ramus)	Anterior	Inferior part of the Gasserian ganglion Pericarotid sympathetic plexus
Jugular branch	Posterior	IX, X, XI (transcranial portion)
Hypoglossal branch	Posterior	XII (transcranial portion) VI (retroclival portion)
Musculo-spinal artery	Posterior	XI (spinal part) Third and fourth cervical nerves (motor part) Superior cervical ganglion

Dural Anastomosis (Table 3)

- The branch of the foramen lacerum of the APhA gives anastomosis to the recurrent artery of the foramen lacerum, which is a dural branch of the internal carotid artery [1, 2, 10, 39]. Martins et al. (2005) also described inconstant anastomoses with the infero-lateral trunk of the ICA, cavernous branch of the MMA and with the accessory meningeal artery [35].
- The jugular branch of the APhA has duro-dural anastomosis with the hypoglossal branch of the APhA because of the vicinity of their respective territories [35]. It also anastomoses with meningeal branches of the occipital artery, with the subarcuate artery (antero-inferior cerebellar artery), with the petro-squamosal branch of the MMA, and with the lateral clival artery (meningo-hypophysary trunk of the ICA) [7]. Effendi et al. (2015) also described a possible anastomosis between the jugular branch of the APhA and the postero-inferior cerebellar artery (PICA) [40].
- The hypoglossal branch has a more complex anastomotic network. Through its descending branch, it participates in the odontoid arterial arch and mainly anastomoses with the anterior meningeal artery (vertebral artery branch) at the C3 level [25]. It also presents anastomoses with the posterior meningeal artery (vertebral artery), with meningeal branches of the occipital artery, and with the medial clival artery (bilateral meningo-hypophysary trunk) [1].

Supplied Territory

The APhA has a complex and important supply for neck muscles, bone, mucosa, dura mater of the posterior fossa, and for peripheral nerves. Tables 2, 3, and 4 summarize all territories supplied by branches of the APhA. In this paragraph, we divide it in three distinct parts: muscular and mucosa supply, dural supply, and peripheral nerve supply.

Muscular and Mucosal Supplies

The anterior trunk of the APhA supplies the pharyngeal submucosal spaces and the submucosal space of the fossa of Rosenmüller [3, 10]. Pharyngeal, nasal, and palatine mucosa are in part supplied by the APhA in competition with branches of the facial artery [10]. A part of the Eustachian tube is also supplied by the APhA (in competition with branches of the pterygo-palatine artery) [26].

Muscular territory is limited to the prevertebral muscles and longus colli muscles from the clivus to C4 and is principally supplied by the musculo-spinal artery [23].

Dural Territory

The dural territory of the APhA is supplied by its three distinct branches: carotid (or branch of the foramen lacerum), jugular, and hypoglossal.

- The carotid branch of the APhA has a little dural territory in balance with dural branches of the ICA [35]. It is limited to the periosteum of the foramen lacerum and of the carotid canal. This artery also gives little vaso-vasorum to the wall of the ICA in its petrous segment [10].
- The jugular branch of the APhA supplies the dura of the jugular foramen, of the petrosal surface (inferior part), the wall of the jugular bulb, inferior petrosal sinus, and of the sigmoid sinus (inferior part) [35].
- The hypoglossal branch of the APhA supplies the inferior part of the clival dura, the antero-lateral part of the foramen magnum, and the inferior part of the cerebellar fossa [1, 27].

Peripheral Nerves Supply

The APhA presents an important participation in the vascularization of the last four cranial nerves [41]. Passing in their respective foramen, the jugular and hypoglossal branches give off branches to the nerves they accompany [10]. Consequently, the jugular branch supplies the IXth, Xth, and XIth cranial nerves, and the hypoglossal branch supplies the XIIth cranial nerve. The spinal part of the accessory nerve (XI) is also supplied more peripherally by the musculo-spinal branch of the APhA [26, 41].

The hypoglossal branch of the APhA could also give off branches to the retro-clival part of the abducens nerve (VI) [10, 35].

Inconsistently, the carotid branch of the APhA could supply the inferior part of the gasserian ganglion (V) [35].

The APhA also participates in the vascularization of the cervical nerves, especially for the anterior ramus (motor part) of the third and fourth cervical nerves by branches of the musculo-spinal artery [41].

To conclude, APhA also presents a minor supply in the autonomic sympathetic system by giving off branches to the superior cervical ganglion (musculo-spinal artery) and to the pericarotid sympathetic plexus (carotid branch) [3, 10].

Variations Implicating the Ascending Pharyngeal Artery

The ascending pharyngeal artery could be implicated in a lot of anatomical variations. We arbitrarily classified these different variations in three distinct groups: the first one is the group of the intra-tympanic flow of the ICA (or aberrant ICA), the second group is composed of variations of the persistent hypoglossal artery, and the last one is the APhA origin of the PICA.

Intratympanic Flow of the Internal Carotid Artery and Associated Variations

The aberrant ICA or intratympanic flow of the ICA is well described in the chapter "Intra-tympanic Flow of the Internal Carotid Artery." This is explained by the agenesis of the two first segments of the primitive carotid artery [10, 37]. The cervical segment is consequently composed by the ascending pharyngeal artery with a hypertrophied inferior tympanic artery that maintains its anastomosis with the caroticotympanic artery (branch of the ICA) into the tympanic cavity that allows a rerouting of the ICA [8]. This vascular variation could be associated to another vascular variation and the different possibilities are the following:

- **Pharyngo-hyo-stapedial artery.** This is an aberrant ICA with persistence of the normal first two segments of the ICA (also known as duplicated ICA) that involved the APhA by its inferior tympanic branch, associated to the persistence of the stapedial artery (SA). The SA arises from the intratympanic part of the ICA. Only few isolated cases of this anatomical variation were described [16, 42, 43].
- **Pharyngo-tympano-stapedial artery**. This very rare variant was first described by Lasjaunias (1977) in its original publication [38]. The same case served as illustration in its textbook "Surgical Angiography," and only two similar cases were published by Baltsavias et al. (2012) and by Jehl et al. (2006) [10, 44, 45]. The middle meningeal artery (MMA) arises from the cervical portion of the ICA, ascends along the cervical ICA, enters the tympanic cavity through the inferior tympanic canal, and follows the usual course of the SA. In this variant, an annexation of the SA by the inferior tympanic artery (branch of the APhA) with regression of the proximal part of the SA explains this vascular configuration. Therefore, the SA arises from the cervical instead of from the petrous segment of the ICA.
- **Intratympanic flow of the internal carotid artery with persistent stapedial artery**. In rare cases, the ICA enters the skull base through an enlarged inferior tympanic

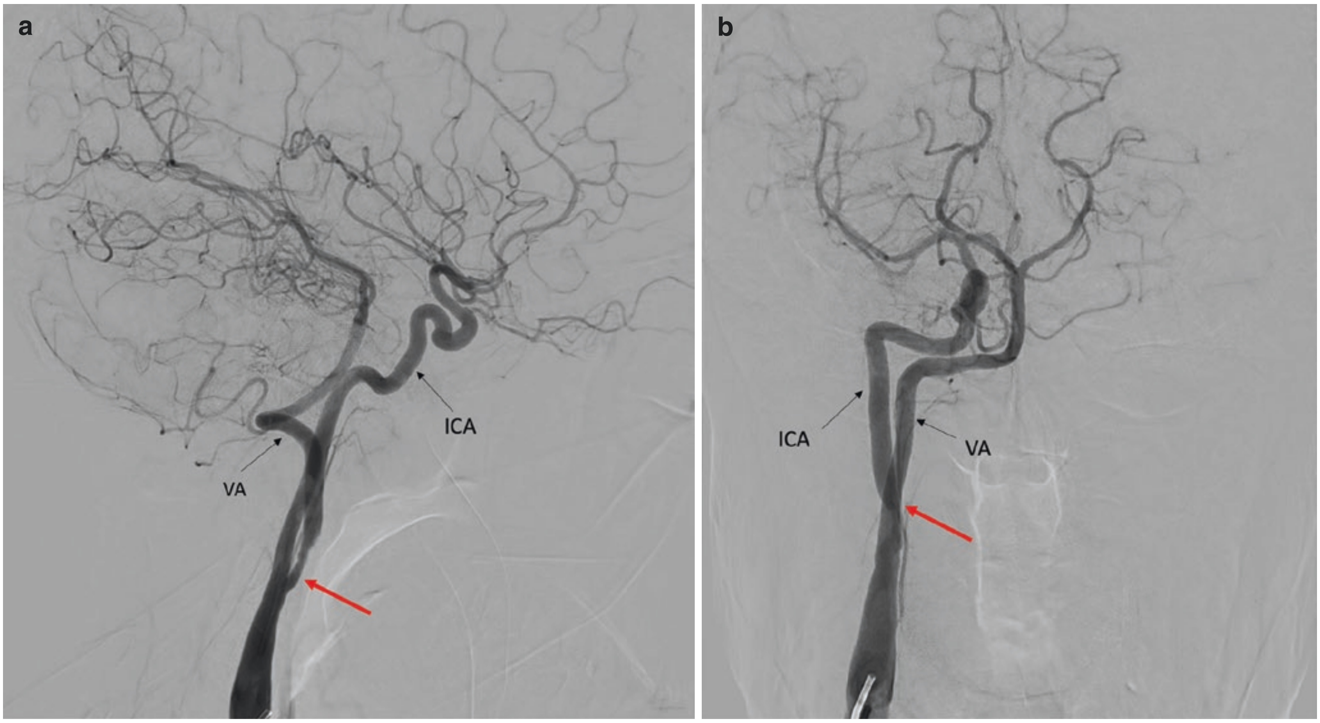


Fig. 4 Clinical case of persistent hypoglossal artery. Lateral (**a**) and antero-posterior (**b**) angiogram of the internal carotid artery (ICA) showing a persistent hypoglossal artery (red arrow). This artery represents a persistent anastomosis between the ICA and the vertebral artery (VA). It arises from the cervical segment of the ICA at the level C1–C3 and enters the skull through the hypoglossal canal

canal, passes into the tympanic cavity to bend anteriorly, and reaches its normal carotid canal [38, 46, 47]. The MMA arises from the ICA in its tympanic segment and passes through the stapes [37, 42].

- **Intratympanic flow of the ICA with pseudo-ICA origin of the occipital artery**. In this case, the aberrant ICA is associated with a common origin between the APhA and the OccA. Consequently, the occipital artery arises from the pseudo-cervical ICA that corresponds to the proximal part of the APhA.

Persistence of the Hypoglossal Artery

The complete persistence of the hypoglossal artery (PHA) and its variants are largely described in the chapter "Carotido-Vertebral Anastomoses." This is the second most frequent persistence of embryonic carotido-vertebral anastomosis after the trigeminal artery and its incidence is between 0.027% and 0.26% [10, 48, 49]. More than 130 isolated cases are described in the English literature [50, 51]. The definition of the PHA was proposed in 1968 by Lie and corresponds to the four following criteria [52]:

- The PHA is a robust artery from the cervical segment of the internal carotid artery (ICA) at the level C1–C3.
- The PHA enters the skull through the hypoglossal canal.
- The basilar artery is filled only distally from the PHA.
- Posterior communicating arteries (PComA) are absent and vertebral arteries are absent or hypoplastic.

PHA could be considered as an anatomical predisposition for basilar artery aneurysm since more than 20% of cases are associated with this type of aneurysm [49, 51, 53–55].

Incomplete persistence of the hypoglossal artery could also explain cases of cervical ICA origin of the PICA passing through the hypoglossal canal. This anatomical variation was first described by P. Lasjaunias who named it the *pharyngo-cerebellar artery* [10, 56]. The same author, in its textbook, showed cases of ascending pharyngeal artery-AICA anastomosis or of ascending pharyngeal artery-basilar artery anastomosis and cited them as PHA variants. A clinical case of PHA is shown in Fig. 4.

Ascending Pharyngeal Artery Origin of the Postero-inferior Cerebellar Artery (PICA) (Pharyngo-cerebellar Artery)

In 1973, Teal et al. described a case of "PICA that arises from the cervical ICA" without more explanation [57]. After them, Lasjaunias et al. in 1981, showed a case of APhA origin of the PICA illustrated with angiographic images without CT scan [56]. They interpreted the case as a partial persistent of the hypoglossal artery and spoke about an anastomosis between the hypoglossal branch of the APhA and the PICA. Unfortunately, they did not prove it with osseous CT scan and named this variation the *pharyngo-cerebellar artery*. This last decade, eight isolated case reports of APhA origin of the PICA were successively described [15, 40, 58–62]. All these cases are similar than Lasjaunias' case with the anatomical difference that the "aberrant" artery does not pass through the hypoglossal canal but through the jugular canal. This important detail raised the matter that this variation is not a partial persistent hypoglossal artery but could be the consequence of another embryonic carotido-vertebral anastomosis not already known passing through the jugular foramen. Ryi et al. (2016), observing that the anastomotic vessel passes through the pars vascularis of the foramen, named this variant a *persistent primitive glosso-pharyngeal artery* [60].

Ascending Pharyngeal Artery Origin of the Posterior Meningeal Artery

Salamon et al. (1967), in their description of the posterior fossa dura vascularization, cited that the posterior meningeal artery could sometimes arise from the ascending pharyngeal artery [5]. After their original description, no other case of this variation was described in the literature.

Clinical Implications

Even if the ascending pharyngeal artery is not a well-studied artery, comprehensive knowledge of anatomy and variations of this artery is important and allows to limit surgical or endovascular complications [15, 16, 18, 20, 22].

The first situation in which is mandatory to know the anatomy and the numerous anastomoses of the APhA is the arterial embolization by liquid embolic agent [4]. Indeed, during the endovascular treatment of a dural arteriovenous fistula, the liquid agent could enter into the vertebro-basilar system through numerous anastomoses. A similar situation is represented by the treatment of naso-pharyngeal tumor or of epistaxis by liquid agent embolization [1, 10].

Variations in the origin of the APhA are important for surgeons to avoid also surgical complications as can happen during carotid endarterectomy in case of a variation in origin of the pharyngo-occipital system [14, 20, 33, 63]. Thanks to its anastomoses with the vertebral artery, the APhA could also be a natural way of distal ICA perfusion in case of severe carotid stenosis [13].

Another important but not well-known clinical implication of the APhA anatomy is the cranial nerves palsies due to its ischemia [41]. Indeed, the APhA participates in the vascularization of the last cranial nerves, and in case of stenosis, surgical ligature, or involuntary embolization, a Vernet or Villaret syndrome could be observed.

References

1. Hacein-Bey L, Daniels DL, Ulmer JL, Mark LP, Smith MM, Strottmann JM, et al. The ascending pharyngeal artery: branches, anastomoses, and clinical significance. AJNR Am J Neuroradiol. 2002;23(7):1246–56.
2. Cavalcanti DD, Reis CV, Hanel R, Safavi-Abbasi S, Deshmukh P, Spetzler RF, et al. The ascending pharyngeal artery and its relevance for neurosurgical and endovascular procedures. Neurosurgery. 2009;65(6 Suppl):114–20; discussion 20. https://doi.org/10.1227/01.NEU.0000339172.78949.5B.
3. Lasjaunias P, Moret J. The ascending pharyngeal artery: normal and pathological radioanatomy. Neuroradiology. 1976;11(2):77–82. https://doi.org/10.1007/BF00345017.
4. Geibprasert S, Pongpech S, Armstrong D, Krings T. Dangerous extracranial-intracranial anastomoses and supply to the cranial nerves: vessels the neurointerventionalist needs to know. AJNR Am J Neuroradiol. 2009;30(8):1459–68. https://doi.org/10.3174/ajnr.A1500.
5. Salamon G, Grisoli J, Paillas JE, Faure J, Giudicelli G. Arteriographic study of the meningeal arteries. (role of selective injections, of subtraction and of radio-anatomical correlations). Neurochirurgia (Stuttg). 1967;10(1):1–19. https://doi.org/10.1055/s-0028-1095333.
6. Salamon G, Guerinel G, Demard F. Radioanatomical study of the external carotid artery. Ann Radiol (Paris). 1968;11(3):199–215.
7. Manelfe C, Roulleau J, Combrelles R, Grisoli F. The vascularization of the dura mater of the occipital foramen. Neurochirurgie. 1971;17(6):469–80.
8. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
9. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: a pictorial review. Surg Radiol Anat. 2021;43(6):961–73. https://doi.org/10.1007/s00276-021-02684-y.
10. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer; 2001.
11. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–62.
12. Alvernia JE, Fraser K, Lanzino G. The occipital artery: a microanatomical study. Neurosurgery. 2006;58(1 Suppl):ONS114–22; discussion ONS-22. https://doi.org/10.1227/01.NEU.0000193519.00443.34.
13. Sato Y, Kashiwagi N, Nakanishi K, Yoshino K, Tomiyama N. Ascending pharyngeal-vertebral anastomosis demonstrated by computed tomography angiography of the ascending pharyngeal artery: a case report. Acta Radiol. 2011;52(9):951–3. https://doi.org/10.1258/ar.2011.100308.
14. Cortes-Franco S, Munoz AL, Franco TC, Ruiz T. Anomalous ascending pharyngeal artery arising from the internal carotid artery: report of three cases. Ann Vasc Surg. 2013;27(2):240 e1–4. https://doi.org/10.1016/j.avsg.2012.10.003.
15. Thomas KL, Hughes MA, Frederickson AM, Branstetter BF, Vilensky JA, Sekula RF. Hemifacial spasm caused by an aberrant jugular branch of the ascending pharyngeal artery. Br J Neurosurg. 2015;29(1):97–9. https://doi.org/10.3109/02688697.2014.957158.
16. Cagnazzo F, Zannoni R, Lefevre PH, Dargazanli C, Derraz I, Gascou G, et al. A symptomatic atherosclerotic persistent pharyngo-

hyo-stapedial artery: treatment management and embryological considerations. Interv Neuroradiol. 2020;26(5):675–80. https://doi.org/10.1177/1591019920949095.
17. Graham RB, Sugrue PA, Rahme RJ, Batjer HH, Bendok BR. Pharyngo-occipital artery variant arising from the internal carotid artery impacting surgical technique during carotid endarterectomy. J Neurointerv Surg. 2013;5(3):e14. https://doi.org/10.1136/neurintsurg-2011-010215.
18. Cohen JE, Leker RR, Moshe Gomori J, Itshayek E. Pharyngo-occipital artery variant arising proximal to occluded internal carotid artery: the risk of an unnecessary endarterectomy. J Clin Neurosci. 2014;21(3):529–31. https://doi.org/10.1016/j.jocn.2013.06.007.
19. Liechty JM, Weddle RJ, Shutze WP, Smith BL. Occurrence of a type 2 proatlantal intersegmental artery during carotid endarterectomy for symptomatic stenosis. J Vasc Surg. 2016;64(3):807–8. https://doi.org/10.1016/j.jvs.2014.10.100.
20. De Freitas S, Malas MB. Ectopic origin of the ascending pharyngeal artery: implications for carotid surgery. Surg Radiol Anat. 2018;40(10):1181–3. https://doi.org/10.1007/s00276-018-2088-z.
21. Keser N, Kula O, Is M, Yardimcioglu I. Prevalence of anomalously originating occipital artery in a Group of Turkish Individuals: a retrospective study using angiography. Turk Neurosurg. 2019;29(3):335–9. https://doi.org/10.5137/1019-5149.JTN.23232-18.1.
22. Babic S, Mitrasinovic A, Gajin P, Ilijevski N, Radak D. Missed dissection as a result of the ascending pharyngeal artery arising from the internal carotid artery. Ann Vasc Surg. 2011;25(5):701.e5–8. https://doi.org/10.1016/j.avsg.2011.02.009.
23. Lasjaunias P, Doyon D. The ascending pharyngeal artery and the blood supply of the lower cranial nerves. J Neuroradiol. 1978;5(4):287–301.
24. Lasjaunias P, Theron J, Moret J. The occipital artery. Anatomy–normal arteriographic aspects–embryological significance. Neuroradiology. 1978;15(1):31–7. https://doi.org/10.1007/BF00327443.
25. Lasjaunias P, Moret J, Theron J. The so-called anterior meningeal artery of the cervical vertebral artery. Normal radioanatomy and anastomoses. Neuroradiology. 1978;17(1):51–5. https://doi.org/10.1007/BF00345270.
26. Lasjaunias P, Manelfe C. Arterial supply for the upper cervical nerves and the cervicocarotid anastomotic channels: systematization of radiological anatomy. Neuroradiology. 1979;18(3):125–31. https://doi.org/10.1007/BF00346866.
27. Al-Rafiah A, EL-Haggagy AA, Aal IH, Zaki AI. Anatomical study of the carotid bifurcation and origin variations of the ascending pharyngeal and superior thyroid arteries. Folia Morphol (Warsz). 2011;70(1):47–55.
28. Gluncic V, Lukic IK, Kopljar M, Sabljar-Matovinovic M, Hebrang A, Marusic A. Persistent cervical intersegmental artery and aortic arch coarctation. Surg Radiol Anat. 2002;24(3–4):226–30. https://doi.org/10.1007/s00276-002-0030-9.
29. Woodruff WW 3rd, Brown NJ. Duplex sonographic detection of ascending pharyngeal artery arising from the internal carotid artery and supplying a dural arteriovenous malformation. J Ultrasound Med. 1993;12(4):227–9. https://doi.org/10.7863/jum.1993.12.4.227.
30. Ustunsoz B, Gumus B, Koksal A, Koroglu M, Akhan O. Missed total occlusion due to the occipital artery arising from the internal carotid artery. Cardiovasc Intervent Radiol. 2007;30(1):116–7. https://doi.org/10.1007/s00270-006-0060-5.
31. Aggarwal NR, Krishnamoorthy T, Devasia B, Menon G, Chandrasekhar K. Variant origin of superior thyroid artery, occipital artery and ascending pharyngeal artery from a common trunk from the cervical segment of internal carotid artery. Surg Radiol Anat. 2006;28(6):650–3. https://doi.org/10.1007/s00276-006-0145-5.
32. Geraci C, Mule G, Mogavero M, Geraci G, Foraci AC, Cerasola G. Aberrant origin of the superior thyroid artery and ascending pharyngeal artery from a common trunk arising from the common carotid artery in a hypertensive patient with dizziness. Minerva Cardioangiol. 2009;57(5):684–6.
33. Devadas D, Pillay M, Sukumaran TT. A cadaveric study on variations in branching pattern of external carotid artery. Anat Cell Biol. 2018;51(4):225–31. https://doi.org/10.5115/acb.2018.51.4.225.
34. Nitescu V. The ascending pharyngeal artery. Morphol Embryol (Bucur). 1977;23(1):35–9.
35. Martins C, Yasuda A, Campero A, Ulm AJ, Tanriover N, Rhoton A Jr. Microsurgical anatomy of the dural arteries. Neurosurgery. 2005;56(2 Suppl):211–51; discussion 51. https://doi.org/10.1227/01.neu.0000144823.94402.3d.
36. Moret J, Delvert JC, Bretonneau CH, Lasjaunias P, de Bicetre CH. Vascularization of the ear: normal-variations-glomus tumors. J Neuroradiol. 1982;9(3):209–60.
37. Lasjaunias P, Moret J. Normal and non-pathological variations in the angiographic aspects of the arteries of the middle ear. Neuroradiology. 1978;15(4):213–9. https://doi.org/10.1007/bf00327529.
38. Lasjaunias P, Moret J, Manelfe C, Theron J, Hasso T, Seeger J. Arterial anomalies at the base of the skull. Neuroradiology. 1977;13(5):267–72. https://doi.org/10.1007/bf00347072.
39. Quisling RG, Seeger JF. Ascending pharyngeal artery collateral circulation simulating internal carotid artery hypoplasia. Neuroradiology. 1979;18(5):277–80. https://doi.org/10.1007/BF00327693.
40. Effendi K, Magro E, Gentric JC, Darsaut TE, Raymond J, Seizeur R, et al. Anastomosis between the ascending pharyngeal artery and the posterior inferior cerebellar artery through the jugular foramen: a cadaveric observation. Oper Neurosurg (Hagerstown). 2016;12(2):163–7. https://doi.org/10.1227/NEU.0000000000001098.
41. Lapresle J, Lasjaunias P. Cranial nerve ischaemic arterial syndromes. A review. Brain. 1986;109(Pt 1):207–16. https://doi.org/10.1093/brain/109.1.207.
42. Koenigsberg RA, Zito JL, Patel M, Swartz JD, Goldofsky E, Zahtz G. Fenestration of the internal carotid artery: a rare mass of the hypotympanum associated with persistence of the stapedial artery. AJNR Am J Neuroradiol. 1995;16(4 Suppl):908–10.
43. Lefournier V, Vasdev A, Bessou P, Boubagra K. A persistent pharyngohyostapedial artery: embryologic implications. AJNR Am J Neuroradiol. 1999;20(2):271–4.
44. Jehl J, Jeunet L, Berraiah M, Bonneville JF. Bilateral persistent Pharyngo-Stapedial arteries revealed during evaluation of a carotid-cavernous fistula. 3 tesla MR angiographic aspects. Interv Neuroradiol. 2006;12(4):327–34. https://doi.org/10.1177/159101990601200406.
45. Baltsavias G, Kumar R, Valavanis A. The pharyngo-tympano-stapedial variant of the middle meningeal artery. A case report. Interv Neuroradiol. 2012;18(3):255–8. https://doi.org/10.1177/159101991201800302.
46. Guinto FC Jr, Garrabrant EC, Radcliffe WB. Radiology of the persistent stapedial artery. Radiology. 1972;105(2):365–9. https://doi.org/10.1148/105.2.365.
47. Steffen TN. Vascular anomalites of the middle ear. Laryngoscope. 1968;78(2):171–97. https://doi.org/10.1288/00005537-196802000-00001.
48. Luh GY, Dean BL, Tomsick TA, Wallace RC. The persistent fetal carotid-vertebrobasilar anastomoses. AJR Am J Roentgenol. 1999;172(5):1427–32. https://doi.org/10.2214/ajr.172.5.10227532.
49. Pasco A, Papon X, Bracard S, Tanguy JY, Ter Minassian A, Mercier P. Persistent carotid-vertebrobasilar anastomoses: how and why differentiating them? J Neuroradiol. 2004;31(5):391–6. https://doi.org/10.1016/s0150-9861(04)97022-8.
50. Yamamoto R, Mori N, Nakae Y, Tanaka F, Johkura K. Anomalous anastomosis between the external carotid artery and vertebro-

basilar artery via the hypoglossal canal: a case report and review of literature. Surg Radiol Anat. 2019;41(7):849–52. https://doi.org/10.1007/s00276-019-02205-y.
51. Uchino A. Carotid-vertebrobasilar anastomosis: magnetic resonance and computed tomographic angiographic demonstration. Jpn J Radiol. 2019;37(8):565–78. https://doi.org/10.1007/s11604-019-00847-x.
52. Lie TA. The congenital anomalies of the carotid arteries. Amsterdam: Excerpta Medica Foundation; 1968.
53. Udvarhelyi GB, Lai M. Subarachnoid haemorrhage due to rupture of an aneurysm on a persistent left hypoglossal artery. Br J Radiol. 1963;36:843–7. https://doi.org/10.1259/0007-1285-36-431-843.
54. Waga S, Morooka Y, Kojima T. Aneurysm on a persistent hypoglossal artery. Acta Neurochir. 1981;59(1–2):71–8. https://doi.org/10.1007/BF01411193.
55. Yamamoto S, Sunada I, Matsuoka Y, Hakuba A, Nishimura S. Persistent primitive hypoglossal artery aneurysms–report of two cases. Neurol Med Chir (Tokyo). 1991;31(4):199–202. https://doi.org/10.2176/nmc.31.199.
56. Lasjaunias P, Guibert-Tranier F, Braun JP. The pharyngo-cerebellar artery or ascending pharyngeal artery origin of the posterior inferior cerebellar artery. J Neuroradiol. 1981;8(4):317–25.
57. Teal JS, Rumbaugh CL, Segall HD, Bergeron RT. Anomalous branches of the internal cartoid artery. Radiology. 1973;106(3):567–73. https://doi.org/10.1148/106.3.567.
58. Fujihara F, Takahara M, Katsuta T, Takemoto K, Higashi T, Inoue T. Posterior inferior cerebellar artery originating from the jugular branch of the ascending pharyngeal artery. NMC Case Rep J. 2019;6(1):21–4. https://doi.org/10.2176/nmccrj.cr.2018-0171.
59. Iihoshi S, Kohyama S. Anomalous origin of the posterior inferior cerebellar artery from the jugular branch of the ascending pharyngeal artery: a case report. Radiol Case Rep. 2020;15(9):1697–700. https://doi.org/10.1016/j.radcr.2020.06.051.
60. Ryu B, Anami H, Ishikawa T, Inoue T, Sugiura M, Kawamata T. Extremely rare persistent primitive artery passing through the jugular foramen with symptomatic ipsilateral carotid artery stenosis. Acta Neurochir. 2016;158(10):1925–9. https://doi.org/10.1007/s00701-016-2896-z.
61. Uchino A, Ohno H, Kondo R, Ishihara S. Ascending pharyngeal artery-posterior inferior cerebellar artery anastomosis via the jugular foramen: a case report and literature review. Surg Radiol Anat. 2021;43(6):1019–22. https://doi.org/10.1007/s00276-020-02667-5.
62. Uchino A, Suzuki C. Posterior inferior cerebellar artery supplied by the jugular branch of the ascending pharyngeal artery diagnosed by MR angiography: report of two cases. Cerebellum. 2011;10(2):204–7. https://doi.org/10.1007/s12311-011-0267-0.
63. Ranchod AI, Gora S, Swartz RN, Andronikou S, Mngomezulu V. A rare carotid-basilar anastomosis traversing the jugular foramen: origin and clinical implications. Interv Neuroradiol. 2011;17(3):347–50. https://doi.org/10.1177/159101991101700311.

Anatomy and Variations of the Posterior Auricular Artery

Thomas Robert and Sara Bonasia

The posterior auricular artery (PAA) is a little but constant branch of the external carotid artery (ECA) [1]. Its principal territory is cutaneous, but it also sends some branches to the middle ear and often to the facial nerve [2]. The cutaneous territory of the PAA is in hemodynamic balance with that of the occipital artery (OccA) [3–5]. The literature about this artery is relatively poor because of its little clinical implication. In this chapter, we will describe the anatomy of the PAA and its possible variations.

Embryological Development

Details about the embryological development of the PAA are not known, but given that the PAA is a distal branch of the ECA, its embryological development depends on that of the ventral pharyngeal artery (VPA) which appears at the third stage of Padget (7–12 mm) [6]. The PAA seems to be formed directly by the primitive ventral pharyngeal artery and does not be part of another embryological system annexed by the VPA [7].

Number and Origin of the Artery

The normal origin of the PAA is the distal part of the ECA just proximal to the emergence of the superficial temporal artery [8]. In more than 95% of cases, the PAA has a simple and normal origin, but it could also share its origin with the OccA [2, 9, 10]. In case of common occipito-auricular trunk, two different possibilities can be seen:

- Proximal common occipito-auricular trunk. In this case, the PAA arises from the occipital artery, its normal origin is consequently not visible [3].
- Distal common occipito-auricular trunk. This is the case of a complete regression of the metameric part of the occipital artery. The PAA is dominant and takes most of the cutaneous territory of the occipital artery. If both branches are present, it seems as a pseudo-duplication of the occipital artery [3].

Course of the Artery

The PAA arises from the ECA in the retro-mandibular space medial to the parotid gland [11]. First, the artery has a posterior course to enter in the auriculo-mastoid sulcus. It passes posterior to the pinna and anterior to the mastoid tip (overall distance: 12.9 mm) [11, 12]. After that, the PAA has a superior course in the subcutaneous space where it gives it distal branches.

Tokugawa et al. proposed a four-type classification of the PAA depending on its course or more precisely on its importance as follows [10]:

- A: small and slender PAA or absent
- B: larger but always slender PAA
- C: PAA easy to identify on DSA but smaller than the superficial temporal artery (STA)
- D: PAA as large as STA

Branches of the Artery

Table 1 and Fig. 1 summarize different branches of the PAA with their respective arterio-arterial anastomoses. The most proximal branches of the PAA, but inconstant, are little branches directed to the parotid gland [2]. Another inconstant but important branch of the PAA is the stylomastoid branch which arises from the

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Table 1 Different branches of the posterior auricular artery with their respective anastomosis

Posterior auricular artery branches	Blood supply	Possible anastomosis
Parotid branches	Deep part of the parotid gland	
Stylomastoid artery (50%)	Facial nerve	Petrosal branch (MMA) Subarcuate artery (AICA) Inferior tympanic artery (APhA)
Auricular branch	External ear	Anterior auricular artery (STA)
Descending branch	Occipitalis muscle Sterno-cleido-mastoid muscle	
Cutaneous branch	Lateral third scalp Postero-inferior part scalp	Occipital artery Parietal branch (STA)

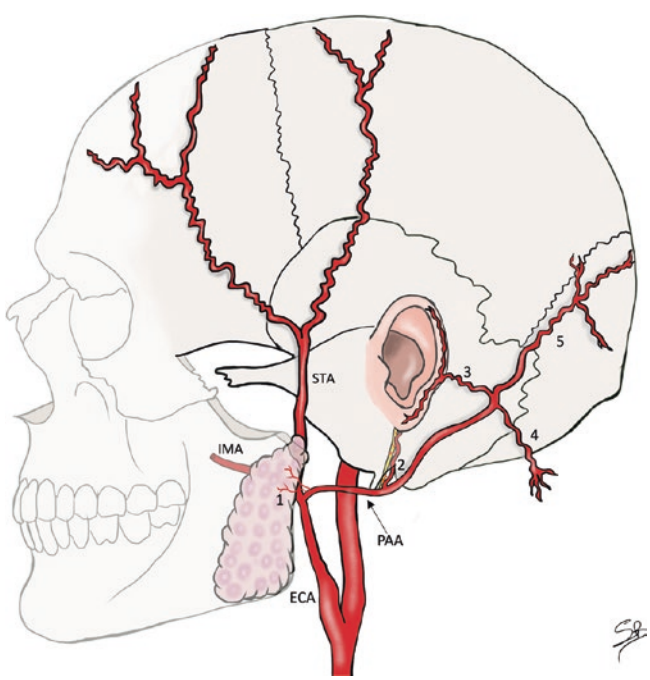

Fig. 1 Branches of the PAA. *ECA* external carotid artery, *IMA* internal maxillary artery, *STA* superficial temporal artery; (1) parotid branches; (2) stylo-mastoid artery; (3) branches to the external ear; (4) descending branches to the nuchal muscles; (5) cutaneous branches for the occipito-parietal scalp

PAA in approximately 50% of cases [3, 5, 13, 14]. The other possible origin of the stylomastoid artery is the occipital artery [13]. This branch is important because it enters the stylomastoid canal to give branches to the facial nerve and to the middle ear [5, 11, 14–16].

Then, the PAA gives its three terminal branches which are:

- The auricular branch destined to the external ear [17].
- The descending branch for the major rotating nuchal muscles [8].
- The cutaneous branch for the occipito-parietal scalp. One of its cutaneous branches, named the transverse nuchal artery by Touré et al., is recognizable by its anastomosis with its contralateral counterpart [9, 12, 18].

Possible Anastomoses

The PAA presents two types of arterial anastomoses, the first one is a cutaneous anastomosis and the second one is an auricular anastomosis [8, 18].

The auricular branch of the PAA has anastomoses with the anterior auricular artery (branch of the superficial temporal artery) [17].

The cutaneous branches of the auricular artery have mainly connections with branches of the occipital artery but also with the parietal branch of the superficial temporal artery [12, 18].

Kumar et al. noted that the PAA has only few anastomoses with its contralateral counterpart [18].

The stylomastoid branch of the PAA could also rarely present an anastomosis with the subarcuate artery (branch of the anterior inferior cerebellar artery) [3, 19].

Blood Supply

The PAA is predominantly a cutaneous artery that supplies the lateral third and postero-inferior part of the scalp [1, 8, 18]. Its cutaneous territory is always in balance with territories of the occipital and superficial temporal arteries. The PAA perfuses the majority of the external ear with the help of the anterior auricular artery [3, 17].

The PAA also contributes to the supply of the posterior auricular muscles, the occipitalis muscle, the temporalis muscle, and the superior part of the sterno-cleido-mastoid muscle [3, 8].

In case of PAA origin of the stylomastoid artery, it also contributes to the blood supply of the facial nerve in its third segment and to the blood supply of the lateral part of the posterior fossa dura mater [5, 13, 14, 16].

Clinical Implications

The PAA has not a lot of clinical implications but is important in two distinct situations. The first one is for plastic surgeons for pedicular flap of the scalp or for reconstruction of the external ear [2, 8, 12, 18]. The second situation where it is important to know the anatomy of the PAA is the extracranial-intracranial anastomosis if the superficial temporal artery is not a suitable option [9, 12]. In this case, the PAA could be used for anastomosis with a branch of the middle cerebral artery (MCA). The

PAA presents some limitations for this surgery, the first one is its little diameter usually inferior to 1 mm; the second one is its posterior localization that limits its anterior mobilization. In their large study, Lee et al. conclude that the PAA is a good candidate for PAA-MCA in only 2.6% [9].

References

1. Trost O, Kadlub N, Cheynel N, Benkhadra M, Malka G, Trouilloud P. Contribution of the posterior auricular artery in the vascularization of the facial nerve in prestylian space: anatomical study and surgical applications. Morphologie. 2008;92(299):171–5. https://doi.org/10.1016/j.morpho.2008.07.016.
2. McKinnon BJ, Wall MP, Karakla DW. The vascular anatomy and angiosome of the posterior auricular artery. A cadaver study. Arch Facial Plast Surg. 1999;1(2):101–4. https://doi.org/10.1001/archfaci.1.2.101.
3. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer; 2001.
4. Lasjaunias P, Moret J, Doyon D, Vignaud J. Arteriographic exploration of the intrapetrous facial nerve: embryology and normal radiologic anatomy (author's transl). Neuroradiology. 1978;16:246–8. https://doi.org/10.1007/BF00395262.
5. Lasjaunias P, Theron J, Moret J. The occipital artery. Anatomy–normal arteriographic aspects–embryological significance. Neuroradiology. 1978;15(1):31–7. https://doi.org/10.1007/BF00327443.
6. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: a pictorial review. Surg Radiol Anat. 2021;43(6):961–73. https://doi.org/10.1007/s00276-021-02684-y.
7. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol. 1948;212:205–62.
8. Nguyen JD, Duong H. Head and neck, posterior auricular artery. In: StatPearls. Treasure Island, FL: StatPearls Publishing; 2022.
9. Lee S, Kim SK, Phi JH. Anatomic variation of the superficial temporal artery and posterior auricular artery in a pediatric Moyamoya disease population. AJNR Am J Neuroradiol. 2021;42(6):1157–62. https://doi.org/10.3174/ajnr.A7059.
10. Tokugawa J, Cho N, Suzuki H, Sugiyama N, Akiyama O, Nakao Y, et al. Novel classification of the posterior auricular artery based on angiographical appearance. PLoS One. 2015;10(6):e0128723. https://doi.org/10.1371/journal.pone.0128723.
11. Liu M, Wang SJ, Benet A, Meybodi AT, Tabani H, Ei-Sayed IH. Posterior auricular artery as a novel anatomic landmark for identification of the facial nerve: a cadaveric study. Head Neck. 2018;40(7):1461–5. https://doi.org/10.1002/hed.25127.
12. Toure G, Meningaud JP, Vacher C. Arterial vascularization of occipital scalp: mapping of vascular cutaneous territories and surgical applications. Surg Radiol Anat. 2010;32(8):739–43. https://doi.org/10.1007/s00276-010-0673-x.
13. Alvernia JE, Fraser K, Lanzino G. The occipital artery: a microanatomical study. Neurosurgery. 2006;58(1 Suppl):ONS114–22; discussion ONS-22. https://doi.org/10.1227/01.NEU.0000193519.00443.34.
14. Moreau S, Salame E, Delmas P. Arterial anatomy of the facial nerve at the stylo-mastoid foramen. Morphologie. 2001;85(269):19–22.
15. Elvan O, Bobus A, Erdogan S, Aktekin M, Olgunus ZK. Fetal anatomy of the facial nerve trunk and its relationship with posterior auricular artery. Surg Radiol Anat. 2019;41(2):153–9. https://doi.org/10.1007/s00276-018-2126-x.
16. Upile T, Jerjes W, Nouraei SA, Singh SU, Kafas P, Sandison A, et al. The stylomastoid artery as an anatomical landmark to the facial nerve during parotid surgery: a clinico-anatomic study. World J Surg Oncol. 2009;7:71. https://doi.org/10.1186/1477-7819-7-71.
17. Zilinsky I, Erdmann D, Weissman O, Hammer N, Sora MC, Schenck TL, et al. Reevaluation of the arterial blood supply of the auricle. J Anat. 2017;230(2):315–24. https://doi.org/10.1111/joa.12550.
18. Sharma RK, Kobayashi K, Jackson IT, Carls FR. Vascular anatomy of the galeal occipitalis flap: a cadaver study. Plast Reconstr Surg. 1996;97(1):25–31; discussion 2. https://doi.org/10.1097/00006534-199601000-00003.
19. Geibprasert S, Pongpech S, Armstrong D, Krings T. Dangerous extracranial-intracranial anastomoses and supply to the cranial nerves: vessels the neurointerventionalist needs to know. AJNR Am J Neuroradiol. 2009;30(8):1459–68. https://doi.org/10.3174/ajnr.A1500.

Part V

Arterial Systems of the Cranio-Cervical Junction

Anastomotic Network of the Cranio-Cervical Junction

Thomas Robert and Sara Bonasia

The cranio-cervical junction (CCJ) is a very particular region from all point of view and its arterial supply is one of its most fascinating particularities. The first specificity of this region is the osseous anatomy of the atlanto-axial complex allowing the head rotation [1]. The second one is the complexity of its arterial supply with a vertical system (represented by the vertebral artery) and a horizontal one (represented by branches of the external carotid artery), also called the suboccipital knot for the multidirectional possible flow between different arterial systems [2]. Even if this is complex, the arterial supply of the suboccipital area is well-studied because of its important anastomotic network between the external carotid artery and the vertebral artery representing a pitfall for arterial embolization of vascular or tumoral pathologies [3]. The scope of this chapter is to summarize all arterial systems that participate in the vascularization of the CCJ and their different anastomotic routes. Figure 1 illustrates the complex vascular network of this area.

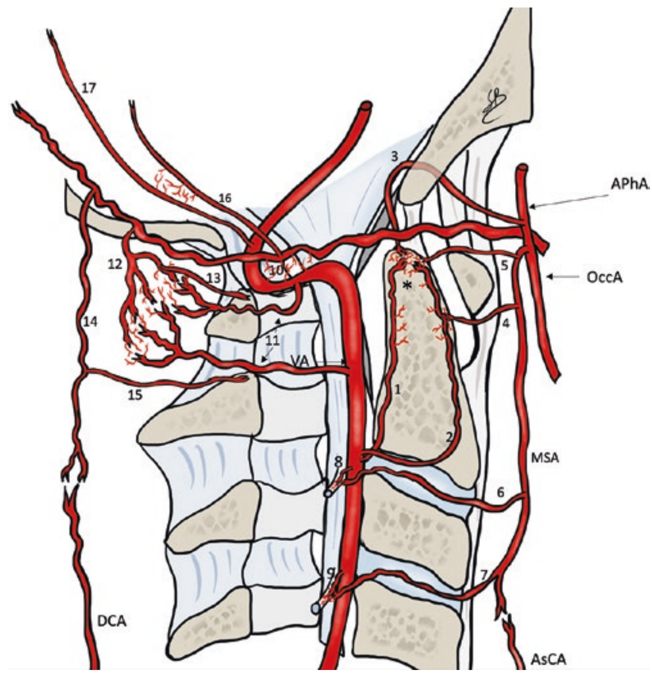

Fig. 1 Artist's illustration of the cranio-cervical junction arterial network. The vertebral artery (VA) gives off the posterior ascendent artery (1) and the anterior ascendent artery (2) whose anastomosis constitutes the odontoid arch (asterisk). To this arch contribute also the hypoglossal branch (3) of the ascendent pharyngeal artery (APhA) and the first (4) and second (5) transverse artery from the musculo-spinal branch (MSA) of the APhA. The APhA gives also branches for the third and fourth cervical nerve roots (6, 7), that anastomose with the corresponding radicular arteries from the VA (8, 9). The terminal portion of the MSA anastomoses with the ascendent cervical artery (AsCA). The VA contracts direct anastomosis with the occipital artery (OccA) at the first and second intervertebral space (10). Muscular anastomoses between them are seen between muscular branches of the VA (11) and the deep descendent branch (12) of the OccA. This latter also gives off a radicular artery for the first nerve root (13). The superficial descendent branch (14) of the OccA gives the radicular artery for the second nerve root (15) and anastomoses with the deep cervical artery (DCA). Dural anastomoses between the VA and the OccA are seen between the artery of the falx cerebelli (16) and the posterior meningeal artery (17)

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History

First studies about the vascularization of the upper cervical spine were proposed during the 1960s by orthopedics and anatomists with the hypothesis that the high rate of nonunion of odontoid process fracture was explained by the poor arterial supply of the region [4–6]. After them, angiographic reports gave a comprehensive description of this anatomy with a different preoccupation for neuroradiogists that was the presence of important arterio-arterial anastomosis between the various arterial systems [3, 7, 8]. To cite only one of them, P. Lasjaunias furnished an impressive work during the 1970s with different key papers that described the occipital artery, the ascending pharyngeal artery, and the arterial supply of the upper cervical spine [1, 9–11].

Vertebral Artery Branches

The vertebral artery is the most represented artery in the sub-occipital region. Branches destined to the CCJ take their origin from the second and the third segment of the VA and are organized as follows:

- The posterior ascending artery (PAA), also known as the *anterior meningeal artery* or the *C3 artery*, is a branch of the VA that originates from the second segment of the artery between the second and third cervical vertebrae. Initially, the PAA follows the third cervical nerve to enter the vertebral canal and the dura mater at this level [5, 6, 12]. This artery courses on the postero-lateral aspect of the axis and odontoid process, crosses the posterior surface of the transverse ligament. The PAA gives transversal branches to the posterior part of the axis, odontoid process, transverse ligament, and tectorial membrane. This artery also has a dural territory that corresponds to the anterior dura of the upper cervical spine until the anterior aspect of the foramen magnum [13]. The PAA ends at the apex of the odontoid to anastomose with its contralateral counterpart and with the two anterior ascending arteries. This anastomosis between the two anterior and the two posterior ascending arteries is called the *apical arcade* or *odontoid arch*. A clinical case showing this arch is shown in Fig. 2.
- The anterior ascending artery (AAA) also arises from the second segment of the VA at the level of the C3 foramen [8, 9, 12]. This artery courses anterior to the axis and posterior to the anterior arch of the atlas deep into the longus colli muscle. Along its course, the AAA gives branches that anastomoses side-by-side and osseous perforators for the anterior arch of the atlas, and for the anterior part of the axis [4]. This artery ends at the anterior surface of the alar ligament where it anastomoses with its contralateral counterpart and to the posterior ascending arteries to form the *apical arcade* [1, 14].
- The posterior meningeal artery is a branch of the third segment of the VA that arises posteriorly to the occipito-atlantal space. This is a dural branch that gives anastomosis near its origin with the occipital artery. Its dural territory is the cerebellar fossa and is in balance with territories of branches of the MMA and occipital artery.

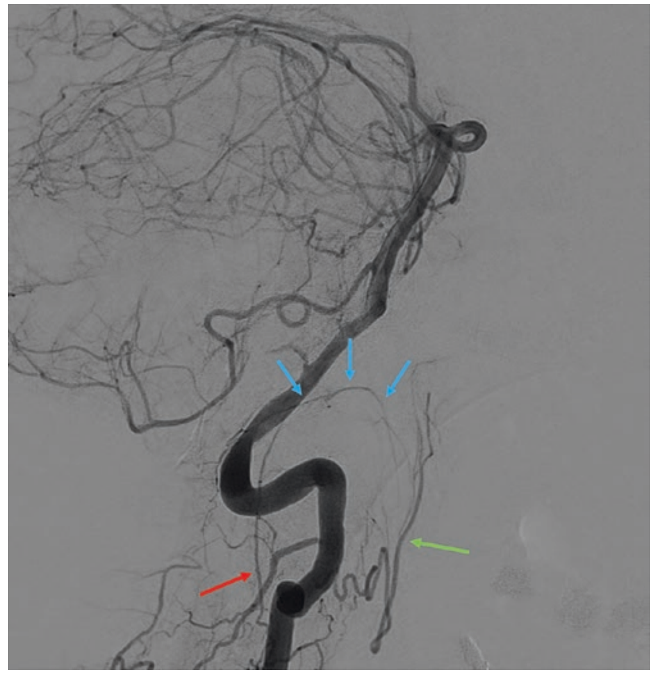

Fig. 2 Clinical case showing the odontoid arch. DSA lateral view after injection of the vertebral artery. The red arrow indicates the anterior meningeal artery or posterior ascendent artery that courses postero-laterally to the odontoid process and anastomoses with the anterior ascendent artery (green arrow) to form the odontoid arch (blue arrows)

Ascending Cervical Artery and Ascending Pharyngeal Artery

The ascending pharyngeal artery (APhA), remnant of the hypoglossal artery, is considered as the arterial supply of the motor (anterior) ramus of the upper cervical nerves and anastomoses with the ascending cervical artery at the level of the fourth cervical vertebrae [7, 15–18]. The two first cervical nerves are purely sensitive; consequently, the APhA does not give branches to these two nerves but only to the third and fourth cervical nerves [1]. These neural branches come from the musculo-spinal artery of the APhA that courses in the prevertebral muscles. The branches from the musculo-spinal artery anastomose with the anterior ascending artery and were called by few authors *cleft perforators* [8, 15].

The APhA participates in the arterial supply of the CCJ also through the hypoglossal branch of the neuro-meningeal trunk [11]. This hypoglossal branch after passing through the hypoglossal canal gives a descending branch that courses in the antero-lateral dura of the foramen magnum until the apex of the odontoid process where it anastomoses with the apical arcade [4, 5].

Deep Cervical Artery and Occipital Artery

The occipital artery (OccA), remnant of the proatlantal artery, is considered as the arterial supply of the posterior (sensitive ramus) of the upper cervical nerves [17, 18]. Superficial and deep descending branches of the OccA give the radicular arteries for the first two cervical nerves (C1 and C2) whose sensitive function is predominant [1]. The superficial descending branch anastomoses with the deep cervical artery at the level of the fourth cervical vertebrae in the paravertebral muscles [2]. Along the two first cervical nerves, the OccA has direct anastomoses with the vertebral artery that are the true remnants of the proatlantal artery I (at C0-C1) and the proatlantal artery II (at C1–C2) [8, 14, 18].

The occipital artery has also possible indirect anastomosis with the vertebral artery at the origin of the posterior meningeal artery [8]. This artery could also in rare case be a branch of the OccA instead of the vertebral artery.

Anastomotic Networks

Even if each anastomosis of the suboccipital area is described in the preceding paragraphs, a summary is necessary to understand the complexity of this arterial network.

Vertebral Artery-Ascending Pharyngeal Artery Anastomoses

The musculo-spinal branch of the APhA that courses in the prevertebral muscles gives little branches called cleft perforators that anastomose with the anterior ascending artery of the vertebral artery [1, 2, 11, 14]. These perforators are divided in *first transverse artery* that lies under the inferior edge of the transverse ligament and in *second transverse artery* at the level of C0–C1 that anastomoses at the level of the apical arch [3].

The musculo-spinal branch of the APhA presents anastomoses with radicular arteries (from the vertebral artery) around the third and fourth cervical nerves before its terminal anastomosis with the ascending cervical artery [15, 16].

Another important anastomosis is also noted from the hypoglossal branch of the APhA that gives a descending branch that anastomoses with the apical arcade (union of the anterior and posterior ascending arteries of the VA) [8, 11].

Vertebral Artery-Occipital Artery Anastomoses

Anastomoses between these two arteries are of three types: direct, muscular, and dural [10].

Direct anastomoses between the OccA and the VA are situated at the level of the first and second cervical spaces. These anastomoses could be as developed as the distal OccA origins from the VA or in other hand, the ICA could be retrogradely supplied in case of proximal ICA stenosis [19, 20].

Muscular anastomoses course between the deep descending branch (OccA) and muscular branches (VA) and are generally seen posterior to the cervical column between the occiput and the third cervical vertebrae [10].

Dural anastomosis are between the posterior meningeal artery (from the VA) and the artery of the falx cerebelli (from the OccA) that share the dural territory of the foramen magnum [8].

Clinical Implications

The presence of this complex anastomotic network at the cranio-cervical junction is of paramount importance especially for the treatment of dural arteriovenous fistulas and tumors of the foramen magnum region. In case of proximal ICA stenosis, the anastomotic network of the CCJ could allow the perfusion of the carotid territory, its knowledge is also important to avoid dramatic complication.

External Carotid Artery Embolization

These last 40 years, endovascular treatment of dural arteriovenous fistulas changed with the possibility of catheterization of ECA branches [3, 8]. The majority of dural arteriovenous fistulas located posteriorly have an arterial supply from the occipital and/or the ascending pharyngeal artery. Catheterization and embolization of the pathology through the occipital artery could be the best option. In this case, rules of embolization need to be known to avoid liquid embolic agent leaks into the vertebro-basilar system (through arterio-arterial anastomosis) [3, 16, 21]. These anastomotic channels are not always seen on the DSA before the treatment, and the physician must consider them as opened even if they are not visualizable. The catheterization of the OccA needs to be as distal as possible to avoid embolic agent reflux into the horizontal portion of the artery [3].

Internal Carotid Artery Stenosis

An important number of cases of carotido-vertebral anastomosis were described in a context of a proximal ICA stenosis. In such cases, the particularity is the flow-reversion from the vertebral system to the carotid system to compensate the hypo-perfusion due to the stenosis. The knowledge of such natural compensation is of paramount importance to plan a surgical or endovascular treatment. Indeed, lack of knowledge could bring the physician in catastrophic complication if he does not respect the anastomotic vessel [19, 20].

References

1. Lasjaunias P, Manelfe C. Arterial supply for the upper cervical nerves and the cervicocarotid anastomotic channels: systematization of radiological anatomy. Neuroradiology. 1979;18(3):125–31. https://doi.org/10.1007/BF00346866.
2. Haffajee MR. A contribution by the ascending pharyngeal artery to the arterial supply of the odontoid process of the axis vertebra. Clin Anat. 1997;10(1):14–8. https://doi.org/10.1002/(SICI)1098-2353(1997)10:1<14::AID-CA2>3.0.CO;2-T.
3. Geibprasert S, Pongpech S, Armstrong D, Krings T. Dangerous extracranial-intracranial anastomoses and supply to the cranial nerves: vessels the neurointerventionalist needs to know. AJNR Am J Neuroradiol. 2009;30(8):1459–68. https://doi.org/10.3174/ajnr.A1500.
4. Schiff DC, Parke WW. The arterial supply of the odontoid process. J Bone Joint Surg Am. 1973;55(7):1450–6.
5. Fischer LP, Carret JP, Gonon GP, Sayfi Y. Arterial vascularization of the axis. Bull Assoc Anat (Nancy). 1976;60(169):335–46.
6. Althoff B, Goldie IF. The arterial supply of the odontoid process of the axis. Acta Orthop Scand. 1977;48(6):622–9. https://doi.org/10.3109/17453677708994808.
7. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
8. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer; 2001.
9. Lasjaunias P, Moret J, Theron J. The so-called anterior meningeal artery of the cervical vertebral artery. Normal radioanatomy and anastomoses. Neuroradiology. 1978;17(1):51–5. https://doi.org/10.1007/BF00345270.
10. Lasjaunias P, Theron J, Moret J. The occipital artery. Anatomy–normal arteriographic aspects--embryological significance. Neuroradiology. 1978;15(1):31–7. https://doi.org/10.1007/BF00327443.
11. Lasjaunias P, Doyon D. The ascending pharyngeal artery and the blood supply of the lower cranial nerves. J Neuroradiol. 1978;5(4):287–301.
12. Greitz T, Lauren T. Anterior meningeal branch of the vertebral artery. Acta Radiol Diagn (Stockh). 1968;7(3):219–24. https://doi.org/10.1177/028418516800700303.
13. Manelfe C, Roulleau J, Combrelles R, Grisoli F. The vascularization of the dura mater of the occipital foramen. Neurochirurgie. 1971;17(6):469–80.
14. Parke WW. The vascular relations of the upper cervical vertebrae. Orthop Clin North Am. 1978;9(4):879–89.
15. Hacein-Bey L, Daniels DL, Ulmer JL, Mark LP, Smith MM, Strottmann JM, et al. The ascending pharyngeal artery: branches, anastomoses, and clinical significance. AJNR Am J Neuroradiol. 2002;23(7):1246–56.
16. Cavalcanti DD, Reis CV, Hanel R, Safavi-Abbasi S, Deshmukh P, Spetzler RF, et al. The ascending pharyngeal artery and its relevance for neurosurgical and endovascular procedures. Neurosurgery. 2009;65(6 Suppl):114–20; discussion 20. https://doi.org/10.1227/01.NEU.0000339172.78949.5B.
17. Raybaud C. Normal and abnormal embryology and development of the intracranial vascular system. Neurosurg Clin N Am. 2010;21(3):399–426. https://doi.org/10.1016/j.nec.2010.03.011.
18. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: a pictorial review. Surg Radiol Anat. 2021;43(6):961–73. https://doi.org/10.1007/s00276-021-02684-y.
19. Schechter MM. The Occipital-vertebral anastomosis. J Neurosurg. 1964;21:758–62. https://doi.org/10.3171/jns.1964.21.9.0758.
20. Nierling DA, Wollschlaeger PB, Wollschlaeger G. Ascending pharyngeal-vertebral anastomosis. Am J Roentgenol Radium Ther Nucl Med. 1966;98(3):599–601. https://doi.org/10.2214/ajr.98.3.599.
21. Okahara M, Kiyosue H, Mori H, Tanoue S, Sainou M, Nagatomi H. Anatomic variations of the cerebral arteries and their embryology: a pictorial review. Eur Radiol. 2002;12(10):2548–61. https://doi.org/10.1007/s00330-001-1286-x.

Carotido-Vertebral Anastomoses

Thomas Robert and L. Bertulli

During the early embryological life, the posterior circulation of the brain is temporarily supplied by ephemeral arteries coming from the carotid system [1, 2]. Even if these embryonic arteries are present during a very short period, they could rarely persist in adult life. Some of them, as the persistent trigeminal artery (TA), are well-known and recognized entities [3]. On the contrary, the persistence of the otic artery is very rare and not recognized by all authors [3]. Another particularity is the persistent glosso-pharyngeal artery that was clearly described few times in adults, but its embryonic precursor was not seen in embryo's studies. In this chapter, we will focus on each carotido-vertebral anastomosis described in the adult after a reminder of embryological knowledge.

Embryological Development

In this paragraph, we describe the development of the posterior circulation with the development and regression of various carotido-vertebral anastomoses following the stages of Padget [2, 4]. Table 1 summarizes our embryological knowledge for each carotido-vertebral anastomosis.

Early in the embryologic life (stage I of Padget, embryos of 4–5 mm), the formation of the vertebro-basilar system starts with the presence of paired longitudinal arteries named "longitudinal neural arteries (LNA)" [2]. These LNAs are supplied by the carotid system through different carotido-vertebral anastomosis and the vertebral arteries are not developed yet. LNAs are supplied by the trigeminal artery (TA), the otic artery (OtA), the hypoglossal artery (HypA), and the proatlantal artery (PA) from cranial to caudal [3, 4] (Figs. 1 and 2).

Table 1 Embryological development and regression of carotido-vertebral anastomoses

CVA	Stage of development	Stage of regression	Remnants in adult
Posterior communicating artery	II (56 mm)	No regression	Posterior communicating artery
Trigeminal artery	I (4–5 mm)	II (5–6 mm)	Meningo-hypophyseal trunk
Otic artery	I (4–5 mm)	II (5–6 mm)	Not known
Glosso-pharyngeal artery	Not known	Not known	Ascending pharyngeal artery (jugular branch)
Hypoglossal artery	I (4–5 mm)	II (5–6 mm)	Ascending pharyngeal artery (hypoglossal branch)
Proatlantal artery	I (4–5 mm)	III (7–12 mm)	Occipital artery

During the stage II of Padget (embryos of 5–6 mm), the caudal division of the primitive internal carotid artery anastomoses with the cranial part of the LNA (formation of the posterior communicating artery) and consequently the carotido-vertebral anastomoses initiate their involution. The first two arteries that regress are the otic and the hypoglossal arteries, and the LNAs are supplied principally by the trigeminal artery cranially and the proatlantal artery caudally. At the same time, the two LNAs come closer to the midline to fuse together [2, 4].

At stage III of Padget (embryos of 7–12 mm), the basilar artery is completely formed by fusion of the two LNAs as well as the posterior communicating artery which is anastomosed with the upper part of the basilar artery. Trigeminal and proatlantal arteries progressively regress and the primitive vertebral artery is formed by transverse anastomoses between the six upper cervical intersegmental arteries

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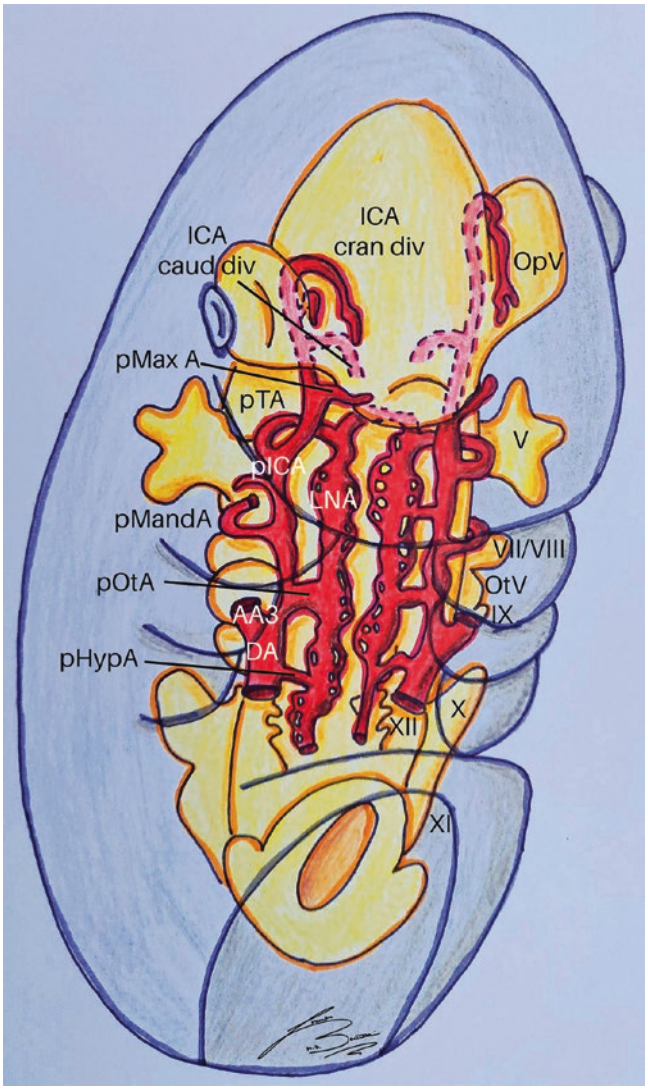

Fig. 1 Padget stage 1; 3/4 view of a 4–5 mm, ≃30 days old embryo. In gray, the outer structure of the embryo is outlined; in yellow, the developing nervous system is represented. Developing cranial nerves are numbered from V to XII. *AA3* third aortic arch, *caud* caudal, *cran* cranial, *DA* dorsal aorta, *div* division, *ICA* internal carotid artery, *LNA* longitudinal neural artery, *MandA* mandibular artery, *MaxA* maxillary artery, *OpV* optic vesicle, *OtA* otic artery, *OtV* otic vesicle, *p* primitive, *TA* trigeminal artery, *HypA* hypoglossal artery

acquiring the adult configuration of the posterior circulation [1–3].

Fig. 2 Possible persistent carotid-basilar anastomoses in the adult life. Asterisk, persistent trigeminal artery; single arrow: persistent otic artery; double arrow: persistent hypoglossal artery; broad arrow: persistent proatlantal artery

Persistent Trigeminal Artery

The persistent trigeminal artery (PTA) was first described by Quain in his original drawings in 1844, and Sutton described it angiographically in 1950 [5, 6]. Among the primitive carotid-basilar anastomoses that persist into adulthood, PTA is the most common, with an estimated incidence of 0.6–1.0% on cerebral angiograms [7–13]. Several cases of PTA and its variants documented at autopsy, on cerebral angiograms, MRIs, and MRA scans provide clear evidence of the anatomy of this primitive anastomosis and its links to various vascular diseases [7, 9, 11, 12, 14–19].

Moreover, Ohshiro et al. provided an autopsy report on an adult whose PTA was found to pass through a dural foramen located immediately medial to Meckel's cave and superolateral to Dorello's canal to enter the posterior fossa [20].

Before, at, and after the dural foramen, the measured PTA diameter was 4.0 mm, 1.0 mm, and 2.0 mm, respectively. The stenosis of the PTA at the foramen led them to speculate that the dural foramen might contribute to the regression of the PTA during the fetal stage of development. According to some authors, failure of regression of the trigeminal artery may be due to obstruction of the proximal portion of the internal carotid artery in the fetus. This results in the trigeminal artery being forced to maintain adequate blood supply to the forebrain by bringing blood retrogradely from the basilar to the carotid arteries. Variations of the PTA originate directly from the precavernous portion of the ICA but terminate in cerebellar arteries rather than anastomosing with the basilar artery. In case of the contemporary presence of the PTA along with an incomplete fusion of the longitudinal neural arteries, either an inferior or a superior cerebellar artery may arise atypically from the cavernous ICA. As a result, the trigeminal artery is not directly connected to the BA and thus terminates as a cerebellar artery [21–29].

A PTA typically originates from the posterior bend of the cavernous carotid artery [7, 9, 11, 30, 31]. Some reports, however, mention a PTA emerging from the medial wall of the cavernous ICA [32, 33]. In 2000, Suttner et al. have described a PTA originating from the superomedial portion of the distal horizontal segment of the cavernous ICA. This PTA then coursed medially and posteroinferiorly and continued between the posterior bend of the carotid artery (laterally) and the pituitary gland (medially), and exiting the posterior wall of the cavernous sinus [9].

Around 50–59% of all cases of PTA from the dorsum sellae penetrate the sella turcica, follow their groove, perforate the dura near the clivus, and join the basilar artery [7, 11, 16, 34]. Thinned sellar floors and abnormalities of the dorsum sellae are common findings. In the other 41–50% of cases, the PTA runs lateral to the sella turcica [7, 9, 13]. Ohshiro et al. classified the PTA in two different types [20]:

- A medial type in which the artery courses through the dorsum sellae and penetrates the dura mater directly adjacent to the clivus.
- A lateral type in which the artery runs between the sensory root of the trigeminal nerve and the lateral side of the sella to reach the dura mater medially to Meckel's cave.

A case that these authors described was also classified as lateral type, where the vessel passed through a dural foramen located immediately medial to Meckel's cave; the foramen was located superolateral to Dorello's canal and lateral to the petroclinoid ligament [20]. Inoue et al. found that both the abducens and trigeminal nerves ran laterally to the PTA on one specimen [35]. One case is reported by Tulsi and Locket in 1985, where the trigeminal artery crosses below the pituitary fossa and penetrates the dorsum sellae; in two other cases, the PTA crosses lateral to the abducens nerve and pierces the dura of the posterior fossa just medial to the sensory root of the trigeminal nerve [36].

The PTA usually joins the basilar artery (BA) between the superior cerebellar artery (SCA) and antero-inferior cerebellar artery (AICA) (Fig. 3) [36]. In one anatomical report, the vessel was reported to enter the basilar artery 2 mm inferiorly to the superior cerebellar artery. From the trigeminal ring to the basilar artery, the artery was 22 mm long, and the overall length was 33 mm. The average diameter was 3.5 mm after the dural ring [18].

Two main classifications of the PTA, one surgical and one neuroradiological, were proposed.

Salas et al. distinguished between lateral (petrosal) and medial (sphenoidal) variations of the PTA according to their relation to the abducens nerve [18]. When the trigeminal artery passes lateral to the sixth cranial nerve, the vessel arises from the posterolateral aspect of the C4 segment of the cavernous carotid (petrosal variation); it then crosses underneath the nerve, which may be displaced superiorly by the PTA. In the petrosal variation of the PTA, the artery pierces the dura just medial to the sensory root of the trigeminal nerve. When the PTA courses medial to the abducens nerve, it arises from the posteromedial aspect of the cavernous carotid artery and pierces the dura of the dorsum sellae (sphenoidal variation). In clinical terms, the lateral variant may present with brainstem ischemia, ophthalmoplegia, and trigeminal neuralgia. In some cases, the medial variant can lead to posterior fossa symptoms because of a steal phenomenon. During transsphenoidal surgery, a medial PTA should also be recognized to avoid severe bleeding.

Saltzman proposed the first angiographic classification for the PTA in 1959 based on eight cases [37]. In Saltzman Type 1, the PTA enters the basilar artery distally to the AICA and proximally to the SCA (Fig. 4). It is possible that the BA proximal to the entering of the PTA is hypoplastic, and the posterior communicating artery (PComA) is absent. In this type of arrangement, the PTA acts as the main supply to the distal BA, PCA, and SCA territories, supplying the entire basilar artery system distal to the anastomosis. Like Saltzman Type 1, in Saltzman Type 2 the PTA joins the BA below the

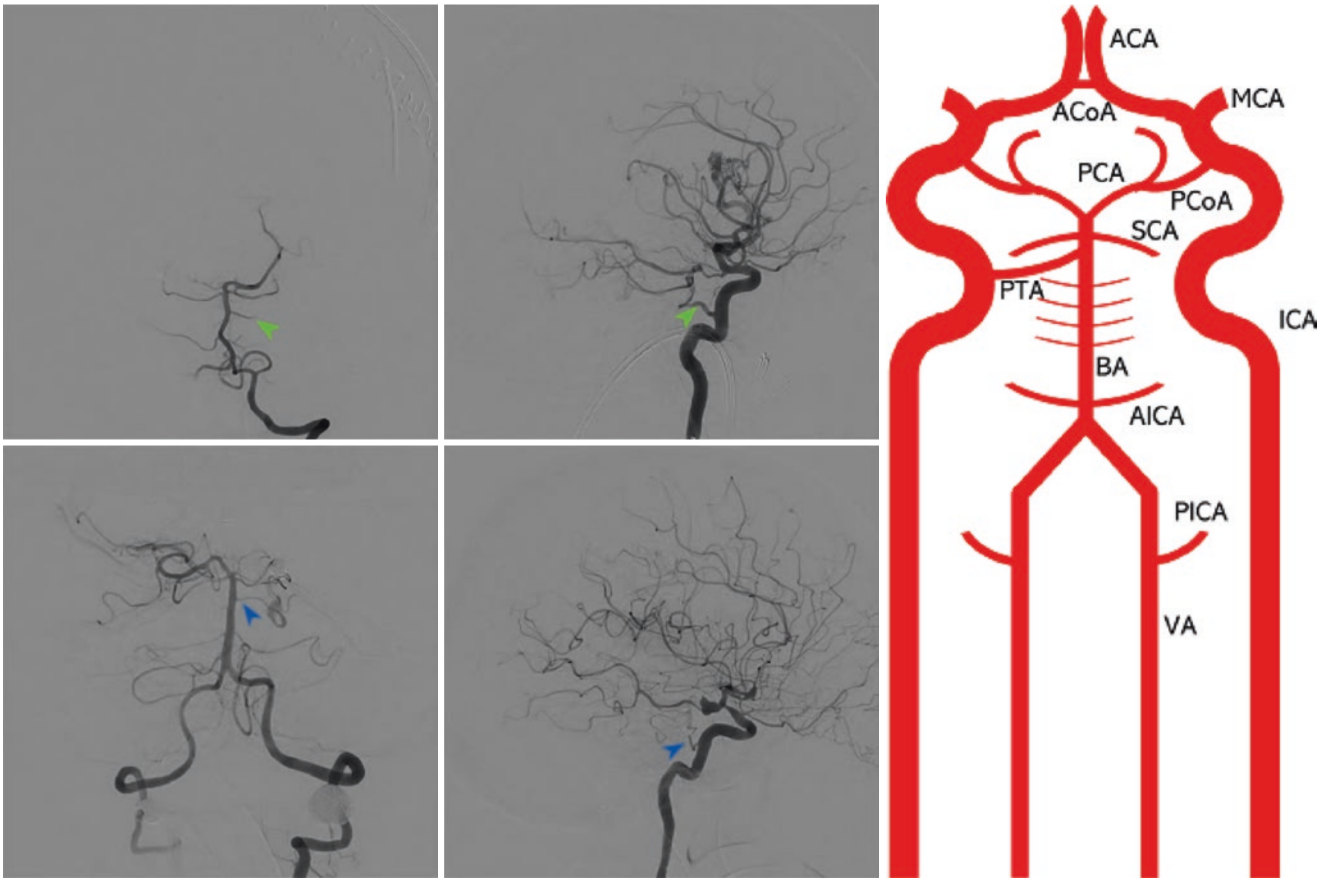

Fig. 3 Clinical cases of persistent trigeminal artery. On the top row on the left, left VA and left ICA injections of a patient with a left frontal AVM showing a PTA (green arrow) originating from the cavernous ICA to end in the BA between SCA and AICA. On the bottom row on the left, left ICA and VA injections in a case of a PComA aneurysm in a patient with a PTA (blue arrow) with similar origin and course as in the other case. On the right, a schematic view of a typical PTA

origin of the SCAs; however, it supplies mainly the SCAs bilaterally (Fig. 5). Consequently, the distal end of the BA is poorly visible angiographically, as the PCAs are supplied predominantly by patent PComAs. It has been reported that the relative incidence of Saltzman type 1 and type 2 is about the same [7]. On the other hand, in a recent review that used 3.0 T MRA to analyze 25 cases of PTA, the incidence of type I was 24%, whereas type II was only 16% [38]. Saltzman classified one case as a combination of the two types, the PComA supplying the PCA on the same side as the PTA, and the PTA providing both the SCAs and the PCA on the other side (Fig. 6) [37]. A similar report was published in 1974 by Parkinson & Shields, who found both angiographic and anatomic evidence of a PTA supplying both SCAs and the contralateral PCA, with a fetal PComA feeding the ipsilateral PCA on the same side of the PTA [39]; this describes a combination of the Saltzman Types 1 and 2. Several Saltzman variants have been described, sometimes defined as Saltzman type 3 [8, 12, 16]. These variants consist of the PTA's direct entering into a cerebellar artery. SCA (Saltzman Type 3a, Fig. 7), AICA (Saltzman Type 3b, Fig. 8), or PICA (Saltzman Type 3c, Fig. 9) arise directly from the ICA and do not anastomose with the BA. PTA variants mainly represent Type 3b with termination at the AICA; types 3a and 3c are exceptionally rare in the literature [12, 27, 32, 40]. According to this theory, anterior vessels are more likely to originate anomalously from the carotid artery than posterior ones since the PTA is closer to the origin of the AICA than the PICA during embryological development [21, 24, 32]. PTA variants are rare, with a reported incidence of 0.18% [19, 22, 25].

More recently, Weon et al. proposed a new classification based on MRA: [8] they identified five types of persistent PTA. In type 1, the PPTA supplies the distal basilar artery (BA), posterior cerebral artery (PCA), and superior cerebellar artery (SCA) territories with bilateral adult PCA configuration. In type 2, the PPTA supplies the SCA territories, and

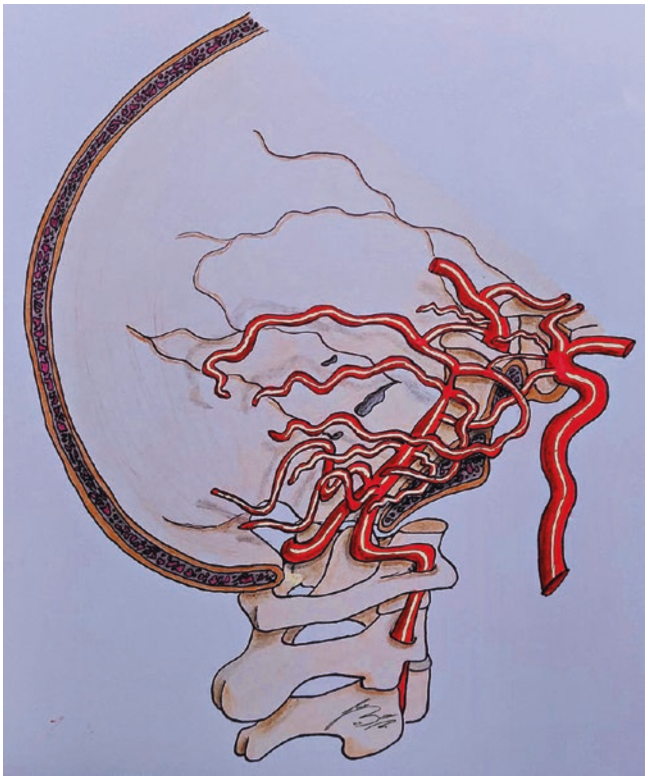

Fig. 4 Saltzman Type 1 PTA. The PTA enters the basilar artery distally to the AICA and proximally to the SCA. It is possible that the BA proximal to the entering of the PTA is hypoplastic, and the posterior communicating artery (PComA) is absent

Fig. 5 Saltzman Type 2 PTA. The PTA joins the BA always below the origin of the SCAs, but it supplies mainly the SCAs bilaterally. Consequently, the distal end of the BA is poorly visible angiographically, as the PCAs are supplied predominantly by patent PComAs

Fig. 6 Combination of Saltzman Type 1 and 2. The PComA supplies the PCA on the same side as the PTA, and the PTA provides both the SCAs and the PCA on the other side

Fig. 7 Saltzman Type 3a. The PTA ends as a SCA, which consequently originates directly from the ICA

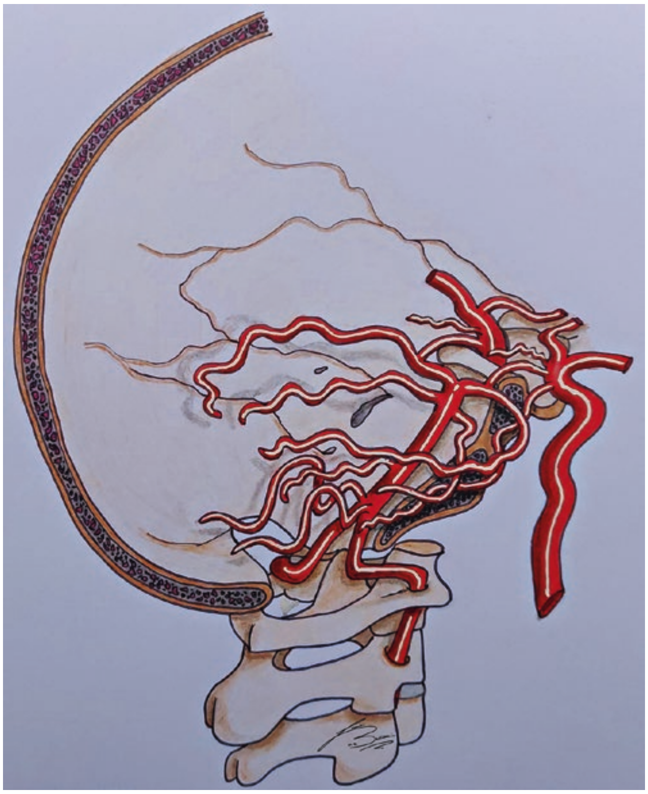

Fig. 8 Saltzman Type 3b. The PTA ends as an AICA, which consequently originates directly from the ICA

Fig. 9 Saltzman Type 3c. The PTA ends as a PICA, which consequently originates directly from the ICA

the PCAs are primarily supplied by the PComA. In type 3, the contralateral PCA is supplied by the PPTA while the ipsilateral PCA receives its blood supply from anterior circulation. In type 4 it's the opposite, as the ipsilateral PCA is supplied by the PPTA and contralateral PCA is supplied by the PComA. Lastly, type 5 consists in a PPTA without the interposition of the BA and can be subdivided into types 5 a, b, and c according to the terminative pattern of the PPTA: 5a, superior cerebellar artery (SCA); 5b, anterior inferior cerebellar artery (AICA); and 5c, posterior inferior cerebellar artery (PICA).

PTA Branches

PTA branches have been observed in adult anatomic specimens. They include pontine perforators as well as branches feeding the trigeminal ganglion, the meningohypophyseal trunk and its branches, and the cerebellar arteries [7, 9, 12, 41].

Pontine Perforators and Branches to the Trigeminal Ganglion

Khodadad in 1976 showed pontine branches emerging from the PTA trunk in foetal brains at 4, 6, and 8 months. According to him, if the PTAs persist, they could be functional and clinically significant [42]. Ohshiro et al. in 1993 reported the presence of two branches of the PTA's cisternal portion. There was one branch that fed the trigeminal nerve root on the left side and sent a pontine perforator. A second branch penetrated directly into the pons [20]. According to Salas et al., there are four pontine perforating branches arising from the intradural segment of the artery originating 7, 10, 11, and 17 mm distal to the dural ring in Meckel's cave. Even in adults, these branches are likely functioning vessels, with occlusion of the PTA causing ischemic lesions to the brainstem [18].

Cerebellar Arteries

Arakawa et al. described a case in which the AICA originates from the internal carotid artery and travels medial to the abducens nerve [43]. From this artery, a small branch communicates with the basilar artery, runs laterally along the trigeminal nerve root, and continues backward to the cerebellum's dorsal surface; thus, AICA can be considered a part of PTA. From this artery, four twigs distribute at the ventral surface of the brainstem: two to the medial surface, one to the lateral surface, and the fourth directly to the basilar artery. After passing lateral to the trigeminal nerve root, the main trunk of the artery ended up on the anteroinferior surface of the cerebellum. The authors also observed that the PTA gives off medial and lateral arterial branches that open into the basilar artery. The first one, located medially, was a pontine branch supplying the basal pons. The lateral branch leaves a twig on the lateral surface of the trigeminal nerve root as it passes caudally. After passing medial to the trigeminal nerve root, this arterial branch splits into two lateral and medial branches. The lateral twig is connected with the AICA. The medial twig, which passed medially, gave off a nutrient artery on the ventral surface of the pons near the facial nerve root. This medial branch also provided some pontine perforators and eventually anastomosed with the basilar artery.

Meningohypophyseal Trunk and Its Branches

The meningohypophyseal trunk (MHT) branches off at the origin of the PTA on cerebral angiograms, according to Lie [44]. Various anatomic descriptions indicated the PTA and MHT had common or separate origins, and the PTA might originate vessels normally arising from the MHT [12, 18, 32]. Among the specimens described by Inoue et al., the dorsal meningeal, tentorial, and inferior hypophyseal arteries were found to arise from the PTA as separate branches [35]. Ohshiro et al. report that the MHT and SCA split at the origin of the PTA [20]. According to their findings, the PTA, MHT, and SCA may have a close link in the embryo. In a case described by Suttner et al., [9] PTA was found to branch out into two branches: the inferior hypophyseal artery medially and the dorsal meningeal artery inferolaterally. Originating from the cavernous carotid artery, the McConnell capsular artery crosses the PTA below the pituitary capsule [9]. Tentorial arteries emerge directly from the cavernous carotid artery at its posterior bend [9, 18]. Salas et al.'s medial (sphenoidal) variation, as well as Parkinson and Shields's superior variation, may represent another carotid-basilar anastomosis from the PTA and the MHT might be a remnant of this vessel [9, 18, 39]. As for the lateral (petrosal) variation, it was considered to be the true PTA, as it can supply blood to both the brainstem and the trigeminal ganglion [9, 45]. Parkinson suggested that the MHT may be a remnant of a carotid basilar connection above the sixth nerve that differs from the actual PTA below the sixth nerve. In analyzing the differences between the two vessels, Parkinson and Shields stressed the fact that the MHT has three branches, whereas the PTA does not have one [39].

Variations and Associated Conditions to the PTA

Associated Variations/Anomalies

Anatomical variations and anomalies associated with PTA include ipsilateral fetal PCAs, bilateral fetal PCAs (Saltzman Type 2), hypoplastic BAs proximal to the junction with the PTA, as well as hypoplastic VAs below the junction with the PTA [7, 12, 46]. Hypoplasia of the BA near the PTA anastomosis occurs frequently with this type of vascular anomaly and is likely due to poor flow stimulating further development [7–9]. Several other anomalies have also been reported, such as a duplicated SCA and hypoplastic AICA, bilateral absence of PICAs, and a fenestrated oculomotor nerve around the PCA [7, 11, 12]. One publication reports a PTA that passes over the clivus in a dedicated canal, then becomes intradural and anastomoses with the BA [39]. The cerebral vessels however were not found to have any other anomalies in some reports [18].

The persistence of the PTA could also be associated with the simultaneous presence of other primitive carotido-basilar anastomoses, such as the primitive hypoglossal or the primitive otic artery [11].

Vascular Anomalies and Syndromes

Trigeminal arteries are the last and most cranial segmental arteries, located at the point of reversal of flow between the connectors of the developing BA; they are located proximally to the VA system and distally to the caudal ICA division [12]. This persistence may inhibit the development of the BA proximal to the anastomosis due to reduced flow-induced stimulations [11, 12, 32]. There may be an explanation for its frequent association with BA hypoplasia, which was found in 75% of cases (moderate/severe degrees) in a large case series [30–32]. It has also been described that the PTA may provide vital blood supply to an isolated midbasilar trunk in rare cases of segmental agenesis of the BA [12]. However, the persistence of multiple embryonic arteries together with cardiovascular anomalies and structural brain malformations represent the same embryologic error in arterial development and not just a hemodynamic effect [47–49]. One example of such an association is the PHACE syndrome (an acronym for **P**osterior fossa brain malformations, large facial **H**emangiomas, **A**rterial anomalies, **C**ardiac anomalies, and **E**ye abnormalities) [11, 12, 48]. This may include arterial agenesis or hypoplasia, aneurysmal dilatation, the PTA and proatlantal arteries, arterial stenoses and occlusions (e.g., Moyamoya phenomenon), aneurysms, angiomatous changes, and arteriovenous malformations (AVMs) [31, 50]. In children with PHACE syndrome, the PTA occurs much more frequently (12–16%) than in other large cerebral angiographic series (0.1–0.2%) [11]. An extensive series of cases found PTA (5 of 17 cases) and ICA or VA agenesis to be the most common anomalies [12, 51, 52]. PTA and arterial anomalies such as carotid and VA agenesis as well as aortic arch anomalies are also described in case reports and small series [12, 22, 25]. A rare coincidence of PTA with other persistent carotido-basilar anastomoses, such as proatlantal intersegmental artery, has been documented [47]. Recently, large-scale series have reported that PTA frequently occurs in conjunction with other cerebrovascular anomalies, such as fenestrations and duplications, illustrating the possibility of common underlying errors during arterial development [12].

PTA and Aneurysms

The frequency of intracranial aneurysms in other locations coexisting with a PTA/PTA variant was 4.2%/4.0% in a recent analysis of a large number of MRA studies (>16,000 patients), similar to that of the general population (3.7 ± 0.7%) [8, 30, 53]. Hence, other intracranial aneurysms do not appear to be associated with PTA or its variants, unlike previously reported [54]. It is likely that the previously reported higher rates of cerebral aneurysms in patients with PTA (14–32%), reflect a selection bias towards patients who underwent cerebral angiography for symptomatic aneurysms [12]. Although the PTA is a bifurcation, it is not more likely to form aneurysms than any other bifurcation [8, 13]. A screening for cerebral aneurysms when an incidental diagnosis of PTA or PTA variant is made is therefore not recommended. There are three possible locations for PTA aneurysms: at the bifurcation between the cavernous ICA segment and the PTA, on the trunk of the PTA, or at its junction with the BA [7, 11, 32]. A PTA aneurysm may cause oculomotor or abducens nerve palsy due to mass effect in the cavernous sinus [11, 30, 47]. Depending on whether they are intracavernous or extracavernous portions of the PTA, they can present with acute subarachnoid hemorrhage accompanied by trigeminal nerve involvement or trigeminal cavernous fistulas [7, 32, 47]. The surgical treatment of PTA aneurysms frequently involves extradural dissection and manipulation in the region of Meckel's cave and/or the cavernous sinus, which can cause cranial nerve palsies [12, 47]. PTA aneurysms can be managed endovascularly by using balloon-assisted and stent-assisted coiling techniques [41, 47]. In planning parent artery sacrifice for a giant cavernous ICA aneurysm, retrograde aneurysm filling should be considered with a PTA concurrently present [12]. In the case of hypoplastic VAs, the PTA may also be used for coiling procedures in the vertebrobasilar circulation [7].

Trigeminal Cavernous Fistulas

A trigeminal cavernous fistula is an abnormal communication between a PTA and the cavernous sinus. The first such fistula reported by Enomoto et al. developed after rupture of a PTA aneurysm [55]. A recent literature review of trigeminal cavernous fistulas found 17 out of 18 fistulas to be Saltzman type II, while one patient presented with a PTA variant [12]. Additionally, the majority of fistulas presented with ocular symptoms such as pain, ophthalmoplegia, pulsatile exophthalmos, and chemosis, or by producing pulse-synchronized bruit [12, 41]. When venous hypertension is present, these fistulas can also cause intracranial hemorrhage [56]. Fistulas are more likely to develop following trauma than spontaneously, with aneurysms suspected to be the most common cause in 5 of 18 cases [12, 41]. In the presence of a patent fistula, it may be difficult to distinguish an underlying aneurysm from a pseudoaneurysm or a dilated venous compartment [12, 41, 56]. The PTA may also be hard to visualize directly in the presence of a fistula since that high-contrast flow may obscure the imaging. The anomalous vessel may only be visible following ICA injection after successful fistula occlusion. Therefore, an angiographic evaluation that includes a vertebrobasilar study, which frequently reveals a retrograde fistula filled with PTA, is essential [12, 41]. Due to the extremely small connections, it may however be impossible to visualize the fistula or the complete trigeminal artery via retrograde visualization from the posterior circulation, especially in cases of PTA variants [11, 12, 53]. As a result, these fistulas are frequently diagnosed as direct CCFs at first. In order to treat trigeminal cavernous fistulas, it is imperative to occlude the fistula while preserving the parent artery, which is accomplished by occluding the first venous pouch [12, 47]. Although ICA trapping alone may be effective, it is generally avoided because the fistula may still receive inflow via the posterior circulation [12]. Various endovascular strategies, including transarterial balloon or coil embolization, have been successful in treating fistulas [12, 41, 47]. Transvenous fistula occlusion with fibred or GDC coils or glue embolization may be an option when there is no underlying aneurysm [41]. Whether the PTA must be preserved depends on the variation type [32]. In Saltzman type I, sacrificing the PTA will result in a posterior circulation infarction [41, 47]. Saltzman type II does not require preservation of the PTA [41, 47]. When dealing with PTA variants, it is imperative to understand the exact anatomy of the communicating vascular channels [8, 41, 57]. Neurosurgical treatment, such as surgical packing, bypass surgery, or even carotid ligation after careful analysis of hemodynamic conditions, may still be an option after failed embolization, despite being no longer the first choice [9, 41, 47].

Other Associated Clinical Conditions

Usually, the PTA or a variant of it is an incidental finding with no clinical significance. However, there are well-known associations with cerebral vascular disease, such as vertebrobasilar embolic ischemia, and with vascular nerve compression syndromes, such as trigeminal neuralgia [11, 12, 32, 45, 53, 58]. According to a recent MRI/MRA study, 2.2% of patients with trigeminal neuralgia experience PTA as a result of direct contact with the nerve [12, 16, 32, 53]. Nevertheless, a study by Morita et al. indicates that a variant of the PTA originating from the ICA may be more prone to causing trigeminal neuralgia than the classic-type PTA [59]. It was explained by differences in the anatomical course, since the PTA variant usually runs very close to the trigeminal nerve root entry zone. From both a developmental and anatomical standpoint, the PTA could involve the cranial nerves III, IV, V, and VI as it passes through the cavernous sinus and the prepontine cistern [11–13, 48, 60]. It is, however, difficult to establish the correct clinical-pathological correlation between cranial nerve lesions and a demonstrable PTA as the causative factor. PTA has been demonstrated to compress adjacent cranial nerves on pathological specimens and intraoperatively; improvement in clinical symptoms has been noted following successful decompression surgery [12]. The association between a PTA and vascular diseases such as AVM, Moyamoya disease, and Sturge–Weber syndrome was previously described based on case reports or small case series [11, 31, 32]. In the absence of other arterial vascular anomalies or syndromes, these are more likely to be coincidental findings rather than true associations. Furthermore, there is no evidence linking a PTA to higher incidences of mental retardation, unlike previously reported [28, 60, 61]. Multiple case reports describe vertebrobasilar ischemia, including brainstem TIAs, cortical infarctions with cortical blindness, associated with a thromboembolic source originating from the IAC (e.g., cervical ICA stenosis or dissection) or even thrombosis of the PTA itself [10, 11, 32, 53]. Hence, noting the presence of PTA before performing Wada testing (injecting barbiturate selectively into the ICA circulation to predict perioperative risk of language delay) is essential in order to avoid perfusion of barbiturate into the posterior circulation [11, 12].

NF-1, Klippel-Feil syndrome, as well as infratentorial and supratentorial AVMs have been also associated with PTA [7, 11, 34, 50].

Persistent Otic Artery

Nowadays, we could find in the English literature 12 cases named or interpreted as a persistent otic (or acoustic) artery (POtA) [31, 49, 62–71]. Lie (1968) described the POtA as a carotido-basilar anastomosis with three distinct criteria [72]:

- The carotido-basilar anastomosis origins from the lateral portion of the petrous segment of the ICA.
- The anastomotic vessel passes through the internal acoustic meatus (IAM).
- The POtA joins the posterior circulation at the caudal part of the basilar artery.

Other minor criteria were also proposed by other authors as the aplasia of the homolateral vertebral artery and the hypoplasia of the contralateral VA [48, 69]. Among the different isolated cases described, the lonely case that responds to all criteria of the POtA is the case of Kempe et al [66]. This is a cadaveric dissection of a patient that presented a geniculate neuralgia explained in postmortem by the presence of a carotido-basilar anastomosis that passes through the IAM. All other cases presented are radiological and principally angiographic cases without clear demonstration of the passage of the anastomotic vessel through the IAM. Lee et al. (2016) published a very interesting article that showed the possible misinterpretation of a low-lying persistent trigeminal artery as a POtA [31]. Another possible misinterpretation is the partial persistent of a stapedial artery into the middle ear that could mimic a POtA [73, 74].

For these reasons, the existence of the persistence otic artery is of important matter of debate. In his masterpiece textbook, P. Lasjaunias declared that the persistent otic artery does not exist without clear arguments, but other authors gives some reasons that make us thinking that the POtA does not exist [3]. The first reason is that the otic structures are not segmental structures but develops completely from the otic placode. The second reason is the lake of evidence of the otic artery in the lower animals. Padget named it as primitive otic artery because it was near the cochlear nerve, but it was a highly transitory presegmental branch seen in only two own specimens of 4–5 mm.

Persistent Glosso-Pharyngeal Artery

In the embryos, no carotido-vertebral anastomoses have been described following the cranial nerves into the jugular foramen, but recent surprising observations raise the question of a possible embryonic artery passing through the jugular foramen [1, 2].

In 1973, Teal et al. described a case of "PICA that origins from the cervical ICA" without more explanation [75]. After them, Lasjaunias et al. in 1981, showed a case of APhA origin of the PICA illustrated with angiographic images without CT scan [76]. They interpreted the case as a partial persistency of the hypoglossal artery and spoke about an anastomosis between the hypoglossal branch of the APhA and the PICA. Unfortunately, they did not prove it with osseous CT scan and named this variation the *pharyngo-cerebellar artery*. This last decade, eight isolated case reports of APhA origin of the PICA were successively described [77–83]. All these cases are similar to the Lasjaunias case with the anatomical difference that the "aberrant" artery does not pass through the hypoglossal canal but through the jugular canal. This important detail raised the matter that this variation is not a partial persistent hypoglossal artery but could be the consequence of another embryonic carotido-vertebral anastomosis not already known passing through the jugular foramen. Ryi et al. (2016), observing that the anastomotic vessel passes through the pars vascularis of the foramen, named this variant a *persistent primitive glosso-pharyngeal artery* [80].

Persistent Hypoglossal Artery

The first case of persistent hypoglossal artery (PHA) was discovered in a cadaveric specimen by Batujeff in 1889 [84–87]. After him, Oertel in 1922 made the same postmortem observation and introduced the name of *hypoglossal artery* [1, 88]. In 1950, Lindgren showed the first angiographic case of PHA followed by other angiographic description in the same period [85]. Morris and Moffat (1956) described a case of PHA postulating that the adult PHA could be embryologically divided in three different portions: the proximal part corresponds to the embryonic HA; the intermediate part is the origin of the postero-inferior cerebellar artery (PICA), and the distal part is primitive vertebro-basilar junction [84]. Until now, 138 cases of PHA were described in the English literature and the PHA is considered as the second most frequent carotido-vertebral anastomosis by persistence of an embryonic artery (Fig. 10) [89–92].

Most cases described in the literature are case reports and the true incidence of PHA is not really known. We could only make an approximation with few large series of CT or MRI observations. The largest series is whom of Uchino et al. (2013), who reviewed more than 2000 CT scans and found 11 cases of PHA [93]. They concluded that the incidence of PHA in their series was 0.53%. Other authors postulated that the incidence of PHA is comprised between 0.027% and 0.26% and this data is taken over by numerous authors without true scientific basis [94, 95]. We just have to remember that the incidence of PHA is largely lower than 1% but not exactly known.

Lie, in 1968, described in his monograph the PHA with four distinct criteria as follows [72]:

- The PHA is a robust artery from the cervical segment of the internal carotid artery (ICA) at the level C1-C3.
- The PHA enters the skull through the hypoglossal canal.
- The basilar artery is filled distally only from the PHA.
- Posterior communicating arteries are absent and vertebral arteries are absent or hypoplastic.

Due to the important number of cases of PHA that did not present the four Lie's criteria, Brismar in 2009 proposed to modify these criteria enhancing that only the first two criteria of Lie are mandatory to describe a PHA [96].

More than 130 cases are described in the English literature and almost all cases are isolated case reports [81, 90–92, 97, 98]. Among them, the majority of PHA origin from the internal carotid artery but few cases of PHA take their origin from the external carotid artery or from the common carotid artery [91, 99–106]. For this reason, Uchino et al. (2013) proposed a classification for PHA with type I corresponding to PHA originating from the ICA and type II from the ECA, but no consensus was found for this classification [93]. Almost all cases of PHA are unilateral but two cases of bilateral PHA are described and represented only 1.4% of cases [95]. In all cases of PHA, PComA and vertebral artery of the same side are always absent, their contralateral counterparts are absent in 30% of cases [95]. The PICA often takes its origin from the PHA when present and Pasco et al. found it in half of cases [95]. Few cases of PHA described are particular and catch our attention. The first one is the case showed by Elhammady et al. who described a case of retrograde flow in the PHA due to a proximal ICA stenosis [107]. In this case, the presence of the PHA allows to compensate the decrease in flow in the carotid territory due to the proxi-

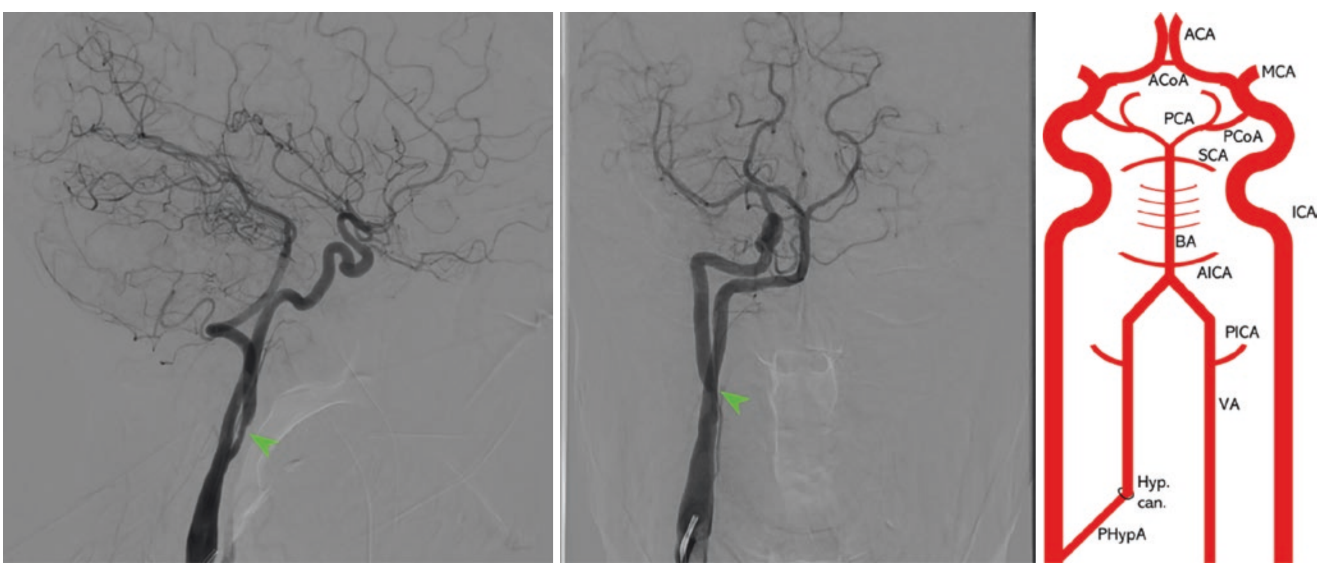

Fig. 10 Clinical case of a persistent hypoglossal artery. Lateral (left) and a-p (middle) views of a right ICA injection showing a persistent hypoglossal artery (green arrow) originating from the cervical ICA and giving the ipsilateral vertebral artery, entering from the hypoglossal canal. On the right a schematic view of a persistent hypoglossal artery is shown

mal ICA stenosis. Suzuki et al. also described another particular case of PHA from which originates the distal part of the occipital artery [108]. Nishida et al. presented another very interesting case of PHA associated with bilateral vertebral artery hypoplasia and contralateral ICA hypoplasia [109]. Consequently, in this case, the whole cerebral perfusion depends on the ICA that bears the PHA. Another very interesting case was presented by Kirkland et al. (2016) and consisted in a case of "transclival artery" [110]. They showed a particular case of carotido-vertebral anastomosis that looks like a persistent hypoglossal artery but with an important difference: the artery does not pass through the hypoglossal canal but through its own canal, located antero-medial to the hypoglossal canal.

PHA is in almost all cases considered as an incidental finding without correlation with any neurological disorder. It is consequently often described in association with another pathology (with or without correlation). The most frequent associated pathology is the cerebral aneurysm with an incidence of 26.9% [91, 93, 105]. We need to differentiate two different types of cerebral aneurysms. The first type is a cerebral aneurysm directly of the PHA and is located at the junction between the PHA and the basilar artery [66, 94, 111–116]. In this situation, the majority of cases are cases of ruptured aneurysm and that highlights the importance of the hemodynamic factor in the formation of cerebral aneurysms. The second type of cerebral aneurysm is an aneurysm of other locations without any relation to the PHA [91, 93, 105, 117, 118]. Other pathologies encountered in patients with PHA are cervical ICA stenosis (20.9%), cerebral AVMs (3%), Moyamoya disease, basilar impression, ischemic stroke, basilar artery fenestration, and Arnold-Chiari malformation [86, 89, 94, 98, 107, 109, 119–134]. Even if it is rare, the presence of a PHA could explain the neurological symptom or sign. Isolated cases of hypoglossal nerve palsy, glosso-pharyngeal neuralgia and posterior fossa ischemic disease due to PHA atherosclerosis are described [66, 117, 130, 134].

Even if the presence of PHA is rarely responsible for the clinical problem, its presence has some important clinical implications and need to be known in particular situations.

- In case of carotid artery stenosis, the presence of PHA has an important impact on the treatment strategy for surgical endarterectomy and carotid artery stenting [89, 98, 107, 122, 130, 135–142]. The knowledge of this anatomical variation dramatically helps in the complication avoidance. Silva et al. also proposed in these particular cases an interesting technique of double distal embolic protection for carotid artery stenting [132].
- In case of carotid artery stenosis associated with PHA, ischemic events could be in the vertebro-basilar territory. In such cases, the presence of the PHA need to be known to understand the correlation between the stenosis and the ischemic event. A misdiagnosis of vertebral arteries stenosis could implicate dramatic therapeutic decision.
- The treatment of true PHA aneurysm is also challenging for surgical clipping as for endovascular coiling [97, 111–113, 119, 121, 143–149]. The localization of the aneurysm at the antero-lateral aspect of the medulla oblongata needs a far lateral approach to see the aneurysmal neck and sac. In the last years, the majority of these aneurysms are treated by endovascular coiling, but no stenting or other devices are used.
- Cases of posterior fossa stroke were correlated to atherosclerosis of the persisting hypoglossal artery itself treated with success by angioplasty or by stent deployment [130, 134].

Other than the complete persistence of the hypoglossal artery, an incomplete variant could explain an ICA origin of the PICA (passing through the hypoglossal canal) or other cases of ascending pharyngeal artery-basilar artery anastomosis [3, 76, 90, 150, 151].

Persistent Proatlantal Artery

In 1885, Gottschau described after a cadaveric dissection the first case of persistent proatlantal artery (PPA) and nowadays, 55 cases of carotido-vertebral anastomosis that correspond to criteria of PPA were described in the English literature. Among them, Sutton in 1962 proposed the first angiographic description of a PPA [95, 152, 153]. This carotido-vertebral anastomosis is one of the most complex for different reason. The first reason is the different point-of-view of authors regarding its embryological origin [3]. The second reason is the absence of consensus in naming this artery and the last one is the confusing classifications to describe different PPAs [3, 154].

PPA is a very rare persistent embryonic carotido-vertebral anastomosis, and its true incidence is not known, only 55 isolated cases were described. The only study that gives an idea about the incidence of PPA is whom of Yilmaz et al. (1995) who analyzed a series of 5500 cerebral CT scans and found only one case of PPA. They concluded that the incidence of PPA is 0.023% [155].

Different authors proposed their own classification depending on the embryological artery that persists or on the origin of the PPA, but no clear criteria were described as for the PHA [156]. P. Lasjaunias proposed the most comprehensive classification based on the embryological development in two different types [3]:

- The type I is the persistence of the *first cervical intersegmental artery*, takes its origin from the cervical ICA and

joins the vertebral artery at the first cervical space to enter the skull through the foramen magnum.
- The type II is the persistence of the *second cervical intersegmental artery*, takes its origin from the ECA and joins the vertebral artery at the second cervical space to pass into the vertebral foramen of C1 before entering the foramen magnum.

Li et al. proposed another classification that is followed by a lot of authors and is most simple [92, 154]. They described as type I, a PPA that origins from the ICA and as type II, a PPA that branches from the ECA whatever where it joins the vertebral artery. This classification, even if is simpler than the first one, created confusing description about PPA and is not based on embryological knowledge.

To summarize and to include all cases of PPA described in the literature, we could propose the criteria of PPA as follow:

- Carotido-vertebral anastomosis.
- Origins from the cervical ICA, the ECA, or the CCA.
- Joins the vertebral artery at the first or second cervical space.
- PPA enters the skull through the foramen magnum and not through the hypoglossal canal that is the most reliable criteria.
- Agenesis of the vertebral artery in 50% of cases.

In the literature, we found 55 cases of PPA including 49 cases (89%) of PPA that joins the vertebral artery at the first cervical space and only 6 cases (11%) at the second cervical space. Among cases of PPA I, 24 cases origin from the ICA, 23 cases from the ECA, and 2 cases from the CCA instead of PPA II takes its origin from the ECA in 5 cases or from the ICA in 1 case [83, 90, 92, 139, 154, 155, 157–193]. Table 2 summarizes different cases of PPA described in the literature.

The majority of PPA cases are unilateral but not less than 9 cases of bilateral PPA are described that represents 16% of cases and a proportion higher than for other carotido-vertebral anastomoses [181, 184, 188, 192, 194–198].

Almost all cases of PPA are asymptomatic and occasional findings, but two of them presented a neurological sign directly correlated to the PPA. The first one is a case of ruptured aneurysm located at the junction between the PPA and the vertebral artery published in the series of Yilmaz et al. The second one is a case of objective pulsatile tinnitus due to cochleo-vestibular nerve compression by a PPA [155, 199]. Interesting cases of retrograde flow from the vertebral artery to the carotid system through the PPA were described in case of proximal ICA stenosis [193, 200]. The presence of a PPA could be associated with a lot of other pathology or anatomical variants as a bilateral ICA agenesis, a proximal ICA stenosis, vein of Galen malformation, AVM, dAVF, cerebral aneurysm, PTA, or duplicated MCA [139, 163–166, 178, 180, 187, 197, 201, 202].

Incomplete persistence of a PPA could also explain other anatomical variations as follows:

- Occipital-vertebral arteries anastomosis at C1 or C2 [203].
- Vertebral artery origin of the occipital artery at C1 or C2.
- Low origin of the PICA at C1 or C2.
- Occipital artery origin of the PICA.
- Cervical ICA origin of the posterior meningeal artery [204].

Carotido-vertebral anastomoses are anatomical variations that illustrate well the important vascular modifications during the fetal life. Knowledge of the different type of carotido-vertebral anastomosis is of paramount importance in the clinical practice for neurosurgeons, neuroradiologists to avoid misinterpretation with possible catastrophic complications. Indeed, the persistence of a carotido-vertebral anastomosis is a highly functional and precious vessel that always need to be spared in any situation.

Table 2 Different type of PPA described in the literature

	Passing through C0–C1	Passing through C1–C2	Total
From ICA	24	1	25 (45.5%)
From ECA	23	5	28 (51%)
From CCA	2	0	2 (3.5%)
Total	49 (89%)	6 (11%)	55

References

1. Altmann F. Anomalies of the internal carotid artery and its branches; their embryological and comparative anatomical significance; report of a new case of persistent stapedial artery in man. Laryngoscope. 1947;57(5):313–39.
2. Padget DH. The development of cranial arteries in the human embryo. Contrib Embryol Carneg Instn. 1948;212:205–62.
3. Lasjaunias P, Bereinstein A, Ter Brugge KG. Surgical neuroangiography. Berlin: Springer; 2001.
4. Bertulli L, Robert T. Embryological development of the human cranio-facial arterial system: a pictorial review. Surg Radiol Anat. 2021;43(6):961–73. https://doi.org/10.1007/s00276-021-02684-y.
5. Quain R. The anatomy of the arteries of the human body and its applications to pathology and operative surgery with a series of lithographic drawings. London: Taylor and Walton; 1844.
6. Sutton D. Anomalous carotid-basilar anastomosis. Br J Radiol. 1950;23(274):617–9. https://doi.org/10.1259/0007-1285-23-274-617.
7. Azab W, Delashaw J, Mohammed M. Persistent primitive trigeminal artery: a review. Turk Neurosurg. 2012;22(4):399–406. https://doi.org/10.5137/1019-5149.JTN.4427-11.1.

8. Weon YC, Choi SH, Hwang JC, Shin SH, Kwon WJ, Kang BS. Classification of persistent primitive trigeminal artery (PPTA): a reconsideration based on MRA. Acta Radiol. 2011;52(9):1043–51. https://doi.org/10.1258/ar.2011.110191.
9. Suttner N, Mura J, Tedeschi H, Ferreira MA, Wen HT, de Oliveira E, et al. Persistent trigeminal artery: a unique anatomic specimen–analysis and therapeutic implications. Neurosurgery. 2000;47(2):428–33; discussion 33–4. https://doi.org/10.1097/00006123-200008000-00030.
10. Xu Y, Kong Y, Xu Y, Wang P. The protective effect of persistent trigeminal artery in patients with ischemic stroke. BMC Neurol. 2019;19(1):158. https://doi.org/10.1186/s12883-019-1374-7.
11. Vasovic L, Jovanovic I, Ugrenovic S, Vlajkovic S, Jovanovic P, Stojanovic V. Trigeminal artery: a review of normal and pathological features. Childs Nerv Syst. 2012;28(1):33–46. https://doi.org/10.1007/s00381-011-1622-7.
12. Meckel S, Spittau B, McAuliffe W. The persistent trigeminal artery: development, imaging anatomy, variants, and associated vascular pathologies. Neuroradiology. 2013;55(1):5–16. https://doi.org/10.1007/s00234-011-0995-3.
13. Brzegowy K, Pekala PA, Zarzecki MP, Pekala JR, Roy J, Aziz HM, et al. Prevalence and clinical implications of the primitive trigeminal artery and its variants: a meta-analysis. World Neurosurg. 2020;133:e401–e11. https://doi.org/10.1016/j.wneu.2019.09.042.
14. O'Uchi E, O'Uchi T. Persistent primitive trigeminal arteries (PTA) and its variant (PTAV): analysis of 103 cases detected in 16,415 cases of MRA over 3 years. Neuroradiology. 2010;52(12):1111–9. https://doi.org/10.1007/s00234-010-0669-6.
15. Komatsu F, Komatsu M, Di Ieva A, Tschabitscher M. Endoscopic anatomy of persistent trigeminal artery: a cadaveric study. Minim Invasive Neurosurg. 2011;54(5-6):223–7. https://doi.org/10.1055/s-0031-1287831.
16. Pereira LP, Nepomuceno LA, Coimbra PP, Oliveira Neto SR, Natal MR. Persistent trigeminal artery: angio-tomography and angio-magnetic resonance finding. Arq Neuropsiquiatr. 2009;67(3B):882–5. https://doi.org/10.1590/s0004-282x2009000500019.
17. Rath S, Mathai KV, Chandy J. Persistent trigeminal artery. Arch Neurol. 1968;19(1):121–2. https://doi.org/10.1001/archneur.1968.00480010139012.
18. Salas E, Ziyal IM, Sekhar LN, Wright DC. Persistent trigeminal artery: an anatomic study. Neurosurgery. 1998;43(3):557–61; discussion 61–2. https://doi.org/10.1097/00006123-199809000-00082.
19. Uchino A, Saito N, Okada Y, Kozawa E, Mizukoshi W, Inoue K, et al. Persistent trigeminal artery and its variants on MR angiography. Surg Radiol Anat. 2012;34(3):271–6. https://doi.org/10.1007/s00276-011-0848-0.
20. Ohshiro S, Inoue T, Hamada Y, Matsuno H. Branches of the persistent primitive trigeminal artery–an autopsy case. Neurosurgery. 1993;32(1):144–8. https://doi.org/10.1227/00006123-199301000-00025.
21. Ahuja A, Graves VB, Crosby DL, Strother CM. Anomalous origin of the posterior inferior cerebellar artery from the internal carotid artery. AJNR Am J Neuroradiol. 1992;13(6):1625–6.
22. Ali S, Radaideh MM, Shaibani A, Russell EJ, Walker MT. Persistent trigeminal artery terminating in the posterior inferior cerebellar artery: case report. Neurosurgery. 2008;62(3):E746–8; discussion 8. https://doi.org/10.1227/01.neu.0000317327.17225.f8.
23. Cobb SR, Hieshima GB, Mehringer CM, Grinnell VS, Pribram HW. Persistent trigeminal artery variant. Carotid-anterior inferior cerebellar artery anastomosis. Surg Neurol. 1983;19(3):263–6. https://doi.org/10.1016/s0090-3019(83)80013-5.
24. Haughton VM, Rosenbaum AE, Pearce J. Internal carotid artery origins of the inferior cerebellar arteries. AJR Am J Roentgenol. 1978;130(6):1191–2. https://doi.org/10.2214/ajr.130.6.1191.
25. Lee GY, Heo YJ, Jung HS, Choo HJ, Cho YJ, Jeong HW, et al. Persistent trigeminal artery variant terminating in the posterior inferior cerebellar artery: a case report. Surg Radiol Anat. 2018;40(2):237–40. https://doi.org/10.1007/s00276-017-1933-9.
26. Lochner P, Zuccoli G, Brigo F, Tezzon F, Trinka E, Nardone R. Persistent trigeminal artery: an unusual rescue vessel collateralization? Can J Neurol Sci. 2013;40(3):429–30. https://doi.org/10.1017/s0317167100014438.
27. Raphaeli G, Bandeira A, Mine B, Brisbois D, Lubicz B. A rare variant of persistent trigeminal artery: cavernous carotid-cerebellar artery anastomosis--a case report and a systematic review. Cerebellum. 2009;8(4):445–7. https://doi.org/10.1007/s12311-009-0120-x.
28. Teal JS, Rumbaugh CL, Bergeron RT, Scanlan RL, Segall HD. Persistent carotid-superior cerebellar artery anastomosis: a variant of persistent trigeminal artery. Radiology. 1972;103(2):335–41. https://doi.org/10.1148/103.2.335.
29. Siqueira M, Piske R, Ono M, Marino JR. Cerebellar arteries originating from the internal carotid artery. AJNR Am J Neuroradiol. 1993;14(5):1229–35.
30. Sulima K, Chojdak-Lukasiewicz J, Paradowski B, Guzinski M. Persistent trigeminal artery as a rare cause of vertebrobasilar insufficiency. Folia Morphol (Warsz). 2021;81(3):785–90. https://doi.org/10.5603/FM.a2021.0067.
31. Lee K, Park H, Park I, Han J. Persistent primitive trigeminal artery that mimics persistent primitive otic artery on cerebral angiography. J Cerebrovasc Endovasc Neurosurg. 2016;18(2):120–3. https://doi.org/10.7461/jcen.2016.18.2.120.
32. Tyagi G, Sadashiva N, Konar S, Aravinda HR, Saini J, Shukla D, et al. Persistent trigeminal artery: neuroanatomic and clinical relevance. World Neurosurg. 2020;134:e214–e23. https://doi.org/10.1016/j.wneu.2019.10.025.
33. Schmid AH. Presistent trigeminal artery.An autopsy report. Neuroradiology. 1974;7(3):173–5. https://doi.org/10.1007/BF00344324.
34. Paksoy Y, SEker M, Kalkan E. Klippel-Feil syndrome associated with persistent trigeminal artery. Spine (Phila Pa 1976). 2004;29(9):E193–6. https://doi.org/10.1097/00007632-200405010-00024.
35. Inoue T, Rhoton AL Jr, Theele D, Barry ME. Surgical approaches to the cavernous sinus: a microsurgical study. Neurosurgery. 1990;26(6):903–32. https://doi.org/10.1097/00006123-199006000-00001.
36. Tulsi RS, Locket NA. Persistent trigeminal artery: an anatomical study. Aust N Z J Surg. 1985;55(4):397–402. https://doi.org/10.1111/j.1445-2197.1985.tb00908.x.
37. Saltzman GF. Patent primitive trigeminal artery studied by cerebral angiography. Acta Radiol. 1959;51(5):329–36. https://doi.org/10.3109/00016925909171103.
38. Chen YC, Li MH, Chen SW, Hu DJ, Qiao RH. Incidental findings of persistent primitive trigeminal artery on 3-dimensional time-of-flight magnetic resonance angiography at 3.0 T: an analysis of 25 cases. J Neuroimaging. 2011;21(2):152–8. https://doi.org/10.1111/j.1552-6569.2010.00472.x.
39. Parkinson D, Shields CB. Persistent trigeminal artery: its relationship to the normal branches of the cavernous carotid. J Neurosurg. 1974;40(2):244–8. https://doi.org/10.3171/jns.1974.40.2.0244.
40. Fu C, Zhao C, Li D, Sun L, Hu Z, Yu W. Primitive trigeminal artery as a rare cause of trigeminal neuralgia. Vasa. 2015;44(2):140–4. https://doi.org/10.1024/0301-1526/a000420.
41. Diana F, Mangiafico S, Valente V, Wlderk A, Grillea G, Colonnese C, et al. Persistent trigeminal artery aneurysms: case report and systematic review. J Neurointerv Surg. 2019;11(12):1261–5. https://doi.org/10.1136/neurintsurg-2019-015046.
42. Khodadad G. Persistent trigeminal artery in the fetus. Radiology. 1976;121(3 Pt. 1):653–6. https://doi.org/10.1148/121.3.653.

43. Arakawa T, Koizumi M, Terashima T, Honma S, Kawai K, Kodama K, et al. Two anatomical autopsy cases of direct communication between a persistent primitive trigeminal artery and an anterior inferior cerebellar artery. Ann Anat. 2007;189(5):489–98. https://doi.org/10.1016/j.aanat.2006.11.012.
44. Lie TA. Congenital anomalies of the carotid arteries: including the carotid-basilar and carotid-vertebral anastomoses. An angiographic study and a review of the literature. An Excerpta Medica Monograph. Amsterdam: Excerpta Medica; 1968.
45. Engelhardt J, El Hage G, Bojanowski MW. Persistent trigeminal artery as collateral circulation in ischemic stroke. World Neurosurg. 2021;148:67–9. https://doi.org/10.1016/j.wneu.2021.01.034.
46. Chiang FL, Zafar AM, Bazan C 3rd. Persistent trigeminal artery communicating with a fetal posterior communicating artery: a case report. Neuroradiol J. 2017;30(2):172–4. https://doi.org/10.1177/1971400916684668.
47. Lam JJH, Shah M, Chung SL, Ho CL. Persistent primitive trigeminal artery associated with a cavernous carotid aneurysm. Case report and literature review. J Radiol Case Rep. 2018;12(11):1–11. https://doi.org/10.3941/jrcr.v12i11.3500.
48. Namba K. Carotid-vertebrobasilar anastomoses with reference to their segmental property. Neurol Med Chir (Tokyo). 2017;57(6):267–77. https://doi.org/10.2176/nmc.ra.2017-0050.
49. Tomsick TA, Lukin RR, Chambers AA. Persistent trigeminal artery: unusual associated abnormalities. Neuroradiology. 1979;17(5):253–7. https://doi.org/10.1007/BF00337535.
50. Eluvathingal Muttikkal TJ, Varghese SP, Chavan VN. Persistent trigeminal artery and associated vascular variations. Australas Radiol. 2007;51:B31–3. https://doi.org/10.1111/j.1440-1673.2007.01828.x.
51. Kim D, Baek JW, Heo YJ, Jeong HW. Congenital absence of the internal carotid artery with a persistent primitive trigeminal artery as a collateral pathway: a report of an anatomic variation. Surg Radiol Anat. 2018;40(7):811–3. https://doi.org/10.1007/s00276-018-2025-1.
52. Rivera R, Choi IS, Sordo JG, Giacaman P, Badilla L, Bravo E, et al. Unusual origin of the left ophthalmic artery from the basilar trunk. Surg Radiol Anat. 2015;37(4):399–401. https://doi.org/10.1007/s00276-014-1327-1.
53. Ling MM, Gupta M, Acharya J. Trigeminal neuralgia associated with a variant of persistent trigeminal artery. Radiol Case Rep. 2020;15(11):2225–8. https://doi.org/10.1016/j.radcr.2020.08.057.
54. Freitas PE, Aquini MG, Chemale I. Persistent primitive trigeminal artery aneurysm. Surg Neurol. 1986;26(4):373–4. https://doi.org/10.1016/0090-3019(86)90139-4.
55. Enomoto T, Sato A, Maki Y. Carotid-cavernous sinus fistula caused by rupture of a primitive trigeminal artery aneurysm. Case report. J Neurosurg. 1977;46(3):373–6. https://doi.org/10.3171/jns.1977.46.3.0373.
56. Tokunaga K, Sugiu K, Kameda M, Sakai K, Terasaka K, Higashi T, et al. Persistent primitive trigeminal artery-cavernous sinus fistula with intracerebral hemorrhage: endovascular treatment using detachable coils in a transarterial double-catheter technique. Case report and review of the literature. J Neurosurg. 2004;101(4):697–9. https://doi.org/10.3171/jns.2004.101.4.0697.
57. Panda A, Arora A, Jana M. Persistent primitive trigeminal artery: an unusual cause of vascular tinnitus. Case Rep Otolaryngol. 2013;2013:275820. https://doi.org/10.1155/2013/275820.
58. Battista RA, Kwartler JA, Martinez DM. Persistent trigeminal artery as a cause of dizziness. Ear Nose Throat J. 1997;76(1):43–5.
59. Morita A, Fukushima T, Miyazaki S, Shimizu T, Atsuchi M. Tic douloureux caused by primitive trigeminal artery or its variant. J Neurosurg. 1989;70(3):415–9. https://doi.org/10.3171/jns.1989.70.3.0415.
60. Campbell RL, Dyken ML. Four cases of carotid-basilar anastomosis associated with central nervous system dysfunction. J Neurol Neurosurg Psychiatry. 1961;24:250–3. https://doi.org/10.1136/jnnp.24.3.250.
61. Wise BL, Palubinskas AJ. Persistent trigeminal artery (carotid-basilar anastomosis). J Neurosurg. 1964;21:199–206. https://doi.org/10.3171/jns.1964.21.3.0199.
62. Carella A, Cavone L, Lamberti P. Vestigial carotido-basilar anastomosis. Presentation of 8 cases. Acta Neurol (Napoli). 1969;24(5):687–700.
63. Franz M, Berlit P, Tornow K. General dysplasia of the cerebral arteries with persistent primitive acoustic artery and giant aneurysm. Eur Arch Psychiatry Neurol Sci. 1989;238(4):196–8. https://doi.org/10.1007/BF00381464.
64. Kaido T, Nakase H, Uranishi R, Sakaki T. Anomalous internal carotid artery in a patient with pituitary adenoma. Case illustration. J Neurosurg. 1999;91(4):710. https://doi.org/10.3171/jns.1999.91.4.0710.
65. Karasawa J, Kikuchi H, Furuse S, Sakaki T, Yoshida Y, Ohnishi H. Bilateral persistent carotid-basilar anastomoses. AJR Am J Roentgenol. 1976;127(6):1053–6. https://doi.org/10.2214/ajr.127.6.1053.
66. Kempe LG, Smith DR. Trigeminal neuralgia, facial spasm, intermedius and glossopharyngeal neuralgia with persistent carotid basilar anastomosis. J Neurosurg. 1969;31(4):445–51. https://doi.org/10.3171/jns.1969.31.4.0445.
67. Patel AB, Gandhi CD, Bederson JB. Angiographic documentation of a persistent otic artery. AJNR Am J Neuroradiol. 2003;24(1):124–6.
68. Petik B, Colak D, Sirik M, Erturk SM. Rare variants of carotid-vertebrobasilar anastomoses. J Belg Soc Radiol. 2016;100(1):74. https://doi.org/10.5334/jbr-btr.1167.
69. Reynolds AF Jr, Stovring J, Turner PT. Persistent otic artery. Surg Neurol. 1980;13(2):115–7.
70. Tubbs RS, Mortazavi MM, Denardo AJ, Cohen-Gadol AA. Persistence of the otic artery with neurological sequelae: case report. Surg Radiol Anat. 2012;34(2):191–3. https://doi.org/10.1007/s00276-011-0853-3.
71. Zhang CW, Xie XD, Yang ZG, Wang CH, You C, Mao BY, et al. Giant cavernous aneurysm associated with a persistent trigeminal artery and persistent otic artery. Korean J Radiol. 2009;10(5):519–22. https://doi.org/10.3348/kjr.2009.10.5.519.
72. Lie TA. The congenital anomalies of the carotid arteries. Amsterdam, NY: Excerpta Medica Foundation; 1968.
73. Bhattacharya JJ, Lamin S, Thammaroj J. Otic or mythic? AJNR Am J Neuroradiol. 2004;25(1):160–2; author reply 2.
74. Croft HJ. Persistent otic artery. AJNR Am J Neuroradiol. 2004;25(1):162; author reply.
75. Teal JS, Rumbaugh CL, Segall HD, Bergeron RT. Anomalous branches of the internal cartoid artery. Radiology. 1973;106(3):567–73. https://doi.org/10.1148/106.3.567.
76. Lasjaunias P, Guibert-Tranier F, Braun JP. The pharyngo-cerebellar artery or ascending pharyngeal artery origin of the posterior inferior cerebellar artery. J Neuroradiol. 1981;8(4):317–25.
77. Effendi K, Magro E, Gentric JC, Darsaut TE, Raymond J, Seizeur R, et al. Anastomosis between the ascending pharyngeal artery and the posterior inferior cerebellar artery through the jugular foramen: a cadaveric observation. Oper Neurosurg (Hagerstown). 2016;12(2):163–7. https://doi.org/10.1227/NEU.0000000000001098.
78. Fujihara F, Takahara M, Katsuta T, Takemoto K, Higashi T, Inoue T. Posterior inferior cerebellar artery originating from the jugular branch of the ascending pharyngeal artery. NMC Case Rep J. 2019;6(1):21–4. https://doi.org/10.2176/nmccrj.cr.2018-0171.
79. Iihoshi S, Kohyama S. Anomalous origin of the posterior inferior cerebellar artery from the jugular branch of the ascending pharyngeal artery: a case report. Radiol Case Rep. 2020;15(9):1697–700. https://doi.org/10.1016/j.radcr.2020.06.051.
80. Ryu B, Anami H, Ishikawa T, Inoue T, Sugiura M, Kawamata T. Extremely rare persistent primitive artery passing through the jugular foramen with symptomatic ipsilateral carotid artery

stenosis. Acta Neurochir. 2016;158(10):1925–9. https://doi.org/10.1007/s00701-016-2896-z.
81. Thomas KL, Hughes MA, Frederickson AM, Branstetter BFt, Vilensky JA, Sekula RF. Hemifacial spasm caused by an aberrant jugular branch of the ascending pharyngeal artery. Br J Neurosurg. 2015;29(1):97–9. https://doi.org/10.3109/02688697.2014.957158.
82. Uchino A, Ohno H, Kondo R, Ishihara S. Ascending pharyngeal artery-posterior inferior cerebellar artery anastomosis via the jugular foramen: a case report and literature review. Surg Radiol Anat. 2021;43(6):1019–22. https://doi.org/10.1007/s00276-020-02667-5.
83. Uchino A, Suzuki C. Posterior inferior cerebellar artery supplied by the jugular branch of the ascending pharyngeal artery diagnosed by MR angiography: report of two cases. Cerebellum. 2011;10(2):204–7. https://doi.org/10.1007/s12311-011-0267-0.
84. Moffat DB, Morris ED. Abnormal origin of the basilar artery from the cervical part of the internal carotid and its embryological significance. Anat Rec. 1956;125(4):701–11. https://doi.org/10.1002/ar.1091250404.
85. Begg AC. Radiographic demonstration of the "hypoglossal artery". A rare type of persistent anomalous carotidbasilar anastomosis. Clin Radiol. 1961;12:187–9. https://doi.org/10.1016/s0009-9260(61)80007-x.
86. Gilmartin D. Hypoglossal artery associated with internal carotid stenosis. Br J Radiol. 1963;36:849–51. https://doi.org/10.1259/0007-1285-36-431-849.
87. Udvarhelyi GB, Lai M. Subarachnoid haemorrhage due to rupture of an aneurysm on a persistent left hypoglossal artery. Br J Radiol. 1963;36:843–7. https://doi.org/10.1259/0007-1285-36-431-843.
88. Blain JG, Logothetis J. The persistent hypoglossal artery. J Neurol Neurosurg Psychiatry. 1966;29(4):346–9. https://doi.org/10.1136/jnnp.29.4.346.
89. Zhang L, Song G, Chen L, Jiao L, Chen Y, Wang Y. Concomitant asymptomatic internal carotid artery and persistent primitive hypoglossal artery stenosis treated by endovascular stenting with proximal embolic protection. J Vasc Surg. 2016;63(1):237–40. https://doi.org/10.1016/j.jvs.2014.04.066.
90. Uchino A, Suzuki C. Variant of a persistent hypoglossal artery supplying only the posterior inferior cerebellar artery diagnosed by magnetic resonance angiography: a case report. Surg Radiol Anat. 2018;40(7):807–10. https://doi.org/10.1007/s00276-018-1970-z.
91. Yamamoto R, Mori N, Nakae Y, Tanaka F, Johkura K. Anomalous anastomosis between the external carotid artery and vertebrobasilar artery via the hypoglossal canal: a case report and review of literature. Surg Radiol Anat. 2019;41(7):849–52. https://doi.org/10.1007/s00276-019-02205-y.
92. Uchino A. Carotid-vertebrobasilar anastomosis: magnetic resonance and computed tomographic angiographic demonstration. Jpn J Radiol. 2019;37(8):565–78. https://doi.org/10.1007/s11604-019-00847-x.
93. Uchino A, Saito N, Okada Y, Kozawa E, Nishi N, Mizukoshi W, et al. Persistent hypoglossal artery and its variants diagnosed by CT and MR angiography. Neuroradiology. 2013;55(1):17–23. https://doi.org/10.1007/s00234-012-1074-0.
94. Matsumura M, Nojiri K, Yumoto Y. Persistent primitive hypoglossal artery associated with Arnold-Chiari type I malformation. Surg Neurol. 1985;24(3):241–4. https://doi.org/10.1016/0090-3019(85)90029-1.
95. Pasco A, Papon X, Bracard S, Tanguy JY, Ter Minassian A, Mercier P. Persistent carotid-vertebrobasilar anastomoses: how and why differentiating them? J Neuroradiol. 2004;31(5):391–6. https://doi.org/10.1016/s0150-9861(04)97022-8.
96. Brismar J. Persistent hypoglossal artery, diagnostic criteria. Report of a case. Acta Radiol Diagn (Stockh). 1976;17(2):160–6. https://doi.org/10.1177/028418517601700204.
97. Garge S, Moses V, Keshava S, Ahmed M, Moorthy R. Persistent hypoglossal arteries with aneurysmal dilation of left hypoglossal artery: a rare case report and review of the literature. BJR Case Rep. 2016;2(2):20150301. https://doi.org/10.1259/bjrcr.20150301.
98. Murai S, Kusaka N, Umakoshi M, Itami H, Otsuka S, Nishiura T, et al. Stenting for internal carotid artery stenosis associated with persistent primitive hypoglossal artery using proximal flow blockade and distal protection system: a technical case report and literature review. J Stroke Cerebrovasc Dis. 2016;25(6):e98–e102. https://doi.org/10.1016/j.jstrokecerebrovasdis.2016.03.026.
99. Lee EJ, Chang HW, Cho CH, Kim E, Lee SK, Kwon JH. Rare variant of persistent primitive hypoglossal artery in magnetic resonance angiography. Surg Radiol Anat. 2010;32(8):801–4. https://doi.org/10.1007/s00276-010-0664-y.
100. Meguro T, Terada K, Hirotsune N, Nishino S, Asano T. Unusual variant of persistent primitive hypoglossal artery. Br J Radiol. 2007;80(960):e314–6. https://doi.org/10.1259/bjr/17918337.
101. Nakamura M, Kobayashi S, Yoshida T, Kamagata M, Sasaki T. Persistent external carotid-vertebrobasilar anastomosis via the hypoglossal canal. Neuroradiology. 2000;42(11):821–3. https://doi.org/10.1007/s002340000337.
102. Nanto M, Takado M, Ohbuchi H, Mandai A, Osaka Y, Nakahara Y, et al. Rare variant of persistent primitive hypoglossal artery, arising from the external carotid artery. Neurol Med Chir (Tokyo). 2012;52(7):513–5. https://doi.org/10.2176/nmc.52.513.
103. Sabouri S, Ebrahimzadeh SA, Rahimian N. Unusual variant of persistent primitive hypoglossal artery diagnosed by CT angiography: a case report and literature review. Clin Neuroradiol. 2014;24(1):59–63. https://doi.org/10.1007/s00062-013-0201-6.
104. Welten RJ, Eikelboom BC, Ackerstaff RG, Ludwig JW. A persistent hypoglossal artery arising from the external carotid artery. Eur J Vasc Surg. 1988;2(4):269–72. https://doi.org/10.1016/s0950-821x(88)80038-0.
105. Yamamoto S, Sunada I, Matsuoka Y, Hakuba A, Nishimura S. Persistent primitive hypoglossal artery aneurysms–report of two cases. Neurol Med Chir (Tokyo). 1991;31(4):199–202. https://doi.org/10.2176/nmc.31.199.
106. Stern J, Correll JW, Bryan N. Persistent hypoglossal artery and persistent trigeminal artery presenting with posterior fossa transient ischemic attacks. Report of two cases. J Neurosurg. 1978;49(4):614–9. https://doi.org/10.3171/jns.1978.49.4.0614.
107. Elhammady MS, Baskaya MK, Sonmez OF, Morcos JJ. Persistent primitive hypoglossal artery with retrograde flow from the vertebrobasilar system: a case report. Neurosurg Rev. 2007;30(4):345–9; discussion 9. https://doi.org/10.1007/s10143-007-0092-6.
108. Suzuki S, Nobechi T, Itoh I, Yakura M, Iwashita K. Persistent proatlantal intersegmental artery and occipital artery originating from internal carotid artery. Neuroradiology. 1979;17(2):105–9. https://doi.org/10.1007/BF00556026.
109. Nishida C, Ashikaga R, Araki Y, Nakamatsu K, Ono Y, Fujii K, et al. Persistent hypoglossal artery associated with arteriovenous malformation: a case report. Eur J Radiol. 2000;33(1):59–62. https://doi.org/10.1016/s0720-048x(99)00058-3.
110. Kirkland JD, Dahlin BC, O'Brien WT. The transclival artery: a variant persistent carotid-basilar arterial anastomosis not previously reported. J Neurointerv Surg. 2017;9(3):e11. https://doi.org/10.1136/neurintsurg-2016-012464.rep.
111. Bongartz EB, Nau HE, Grote W. Successful operative treatment of an aneurysm on a left persistent hypoglossal artery. Neurochirurgia (Stuttg). 1977;20(5):169–73. https://doi.org/10.1055/s-0028-1090373.
112. Huber P, Rivoir R. Aneurysm on a persistent left hypoglossal artery. Neuroradiology. 1974;6(5):277–8. https://doi.org/10.1007/BF00345788.
113. Kanai H, Nagai H, Wakabayashi S, Hashimoto N. A large aneurysm of the persistent primitive hypoglossal artery. Neurosurgery. 1992;30(5):794–7.

114. Kobayashi M, Akaji K, Tanizaki Y, Mihara B, Ohira T, Kawase T. Posterior inferior cerebellar artery aneurysm associated with persistent primitive hypoglossal artery. Neurol Med Chir (Tokyo). 2008;48(6):259–61. https://doi.org/10.2176/nmc.48.259.
115. Waga S, Morooka Y, Kojima T. Aneurysm on a persistent hypoglossal artery. Acta Neurochir. 1981;59(1–2):71–8. https://doi.org/10.1007/BF01411193.
116. Yokota N, Yokoyama T, Ryu H. Aneurysm of persistent primitive hypoglossal artery. Br J Neurosurg. 1999;13(6):608–10. https://doi.org/10.1080/02688699943178.
117. Al-Memar A, Thrush D. Unilateral hypoglossal nerve palsy due to aneurysm of the stump of persistent hypoglossal artery. J Neurol Neurosurg Psychiatry. 1998;64(3):405. https://doi.org/10.1136/jnnp.64.3.405.
118. Yeh H, Heiss JD, Tew JM Jr. Persistent hypoglossal artery associated with basilar artery aneurysm. Neurochirurgia (Stuttg). 1987;30(5):158–9. https://doi.org/10.1055/s-2008-1054087.
119. Kanematsu M, Satoh K, Nakajima N, Hamazaki F, Nagahiro S. Ruptured aneurysm arising from a basilar artery fenestration and associated with a persistent primitive hypoglossal artery. Case report and review of the literature. J Neurosurg. 2004;101(3):532–5. https://doi.org/10.3171/jns.2004.101.3.0532.
120. De Caro R, Parenti A, Munari PF. The persistent primitive hypoglossal artery: a rare anatomic variation with frequent clinical implications. Ann Anat. 1995;177(2):193–8. https://doi.org/10.1016/S0940-9602(11)80073-7.
121. Hatayama T, Yamane K, Shima T, Okada Y, Nishida M. Persistent primitive hypoglossal artery associated with cerebral aneurysm and cervical internal carotid artery stenosis–case report. Neurol Med Chir (Tokyo). 1999;39(5):372–5. https://doi.org/10.2176/nmc.39.372.
122. Katoh M, Kamiyama H, Kobayashi N, Makino K, Takano K, Tokumitsu N, et al. Severe stenosis of the internal carotid artery presenting as loss of consciousness due to the presence of a primitive hypoglossal artery: a case report. Surg Neurol. 1999;51(3):310–2. https://doi.org/10.1016/s0090-3019(98)00053-6.
123. Warot P, Lambert P, Gozet G. A case of primary hypoglossal artery. Rev Neurol (Paris). 1970;123(1):53–6.
124. Katayama W, Enomoto T, Yanaka K, Nose T. Moyamoya disease associated with persistent primitive hypoglossal artery: report of a case. Pediatr Neurosurg. 2001;35(5):262–5. https://doi.org/10.1159/000050433.
125. Komiyama M, Nakajima H, Nishikawa M, Yasui T, Kitano S, Sakamoto H, et al. High incidence of persistent primitive arteries in moyamoya and quasi-moyamoya diseases. Neurol Med Chir (Tokyo). 1999;39(6):416–20; discussion 20–2. https://doi.org/10.2176/nmc.39.416.
126. Garza-Mercado R, Cavazos E, Urrutia G. Persistent hypoglossal artery in combination with multifocal arteriovenous malformations of the brain: case report. Neurosurgery. 1990;26(5):871–6. https://doi.org/10.1097/00006123-199005000-00024.
127. Gupta AK. Cerebral arteriovenous malformation embolized through persistent primitive hypoglossal artery: a case report. Interv Neuroradiol. 2005;11(3):241–6. https://doi.org/10.1177/159101990501100307.
128. Shibata Y, Hyodo A, Saito A, Yoshii Y, Nose T. Large arteriovenous malformation associated with persistent primitive hypoglossal artery–case report. Neurol Med Chir (Tokyo). 1991;31(12):804–8. https://doi.org/10.2176/nmc.31.804.
129. Yamanaka K, Noguchi K, Hayasaki K, Matsuoka Y. Persistent primitive hypoglossal artery associated with arteriovenous malformation–case report. Neurol Med Chir (Tokyo). 1990;30(12):949–55. https://doi.org/10.2176/nmc.30.949.
130. Eller JL, Jahshan S, Dumont TM, Kan P, Siddiqui AH. Tandem symptomatic internal carotid artery and persistent hypoglossal artery stenosis treated by endovascular stenting and flow reversal. BMJ Case Rep. 2013;2013:bcr2012010578. https://doi.org/10.1136/bcr-2012-010578.
131. Pyun HW, Lee DH, Kwon SU, Lee JH, Choi CG, Kim SJ, et al. Internal carotid artery stenosis with ipsilateral persistent hypoglossal artery presenting as a multiterritorial embolic infarction: a case report. Acta Radiol. 2007;48(1):116–8. https://doi.org/10.1080/02841850601026443.
132. Silva CF, Hou SY, Kuhn AL, Whitten RH, Wakhloo AK. Double embolic protection during carotid artery stenting with persistent hypoglossal artery. J Neurointerv Surg. 2014;6(3):e23. https://doi.org/10.1136/neurintsurg-2013-010709.rep.
133. Sunada I, Yamamoto S, Matsuoka Y, Nishimura S. Endarterectomy for persistent primitive hypoglossal artery–case report. Neurol Med Chir (Tokyo). 1991;31(2):104–8. https://doi.org/10.2176/nmc.31.104.
134. Touho H, Ohnishi H, Seno M, Furuoka N, Komatsu T, Karasawa J. Percutaneous transluminal angioplasty of stenotic primitive hypoglossal artery–case report. Neurol Med Chir (Tokyo). 1994;34(6):371–4. https://doi.org/10.2176/nmc.34.371.
135. Cartier R. Combined internal carotid and hypoglossal artery endarterectomies in a symptom-free patient with contralateral internal carotid artery occlusion. J Vasc Surg. 1995;22(1):120–2. https://doi.org/10.1016/s0741-5214(95)70096-x.
136. Kawabori M, Kuroda S, Yasuda H, Hokari M, Nakayama N, Saito H, et al. Carotid endarterectomy for internal carotid artery stenosis associated with persistent primitive hypoglossal artery: efficacy of intraoperative multi-modality monitoring. Minim Invasive Neurosurg. 2009;52(5–6):263–6. https://doi.org/10.1055/s-0029-1243243.
137. Koleilat I, Hanover T. Carotid endarterectomy in the face of a persistent hypoglossal artery. Ann Vasc Surg. 2015;29(8):1660 e1–4. https://doi.org/10.1016/j.avsg.2015.05.032.
138. Osgood CP, Montanez JA, Karunaratne ER, Vandevander DJ. Endarterectomy for atheromatous hypoglossal artery. Case report. J Neurosurg. 1983;58(6):935–6. https://doi.org/10.3171/jns.1983.58.6.0935.
139. Ouriel K, Green RM, DeWeese JA. Anomalous carotid-basilar anastomoses in cerebrovascular surgery. J Vasc Surg. 1988;7(6):774–7. https://doi.org/10.1067/mva.1988.avs0070774.
140. Kanazawa R, Ishihara S, Okawara M, Ishihara H, Kohyama S, Yamane F. A successful treatment with carotid arterial stenting for symptomatic internal carotid artery severe stenosis with ipsilateral persistent primitive hypoglossal artery: case report and review of the literature. Minim Invasive Neurosurg. 2008;51(5):298–302. https://doi.org/10.1055/s-0028-1082299.
141. Nii K, Aikawa H, Tsutsumi M, Onizuka M, Nakau H, Inoue R, et al. Carotid artery stenting in a patient with internal carotid artery stenosis and ipsilateral persistent primitive hypoglossal artery presenting with transient ischemia of the vertebrobasilar system: case report. Neurol Med Chir (Tokyo). 2010;50(10):921–4. https://doi.org/10.2176/nmc.50.921.
142. Ryu B, Ishikawa T, Hashimoto K, Shimizu M, Yagi S, Shimizu T, et al. Internal carotid artery stenosis with persistent primitive hypoglossal artery treated with carotid artery stenting: a case report and literature review. Neuroradiol J. 2016;29(2):115–21. https://doi.org/10.1177/1971400915626427.
143. Baldi S, Zander T, Rabellino M, Maynar M. Stent-assisted coil embolization of a wide-neck aneurysm of a persistent primitive hypoglossal artery. Cardiovasc Intervent Radiol. 2009;32(2):352–5. https://doi.org/10.1007/s00270-008-9415-4.
144. Baltsavias GM, Chourmouzi D, Tasianas N, Drevelengas A, Damianovski D, Jovkovski S. Ruptured aneurysm of a persistent primitive hypoglossal artery treated by endovascular approach–case report and literature review. Surg Neurol. 2007;68(3):338–43; discussion 43. https://doi.org/10.1016/j.surneu.2006.10.053.
145. Bapuraj JR, Ojili V, Khandelwal N, Shanbhogue AK, Gupta SK. Basilar artery aneurysm treated with coil

embolization via persistent primitive hypoglossal artery. Australas Radiol. 2007;51(Suppl):B340–3. https://doi.org/10.1111/j.1440-1673.2007.01759.x.
146. Bohmfalk GL, Story JL. Aneurysms of the persistent hypoglossal artery. Neurosurgery. 1977;1(3):291–6. https://doi.org/10.1227/00006123-197711000-00014.
147. De Blasi R, Medicamento N, Chiumarullo L, Salvati A, Maghenzani M, Dicuonzo F, et al. A case of aneurysm on a persistent hypoglossal artery treated by endovascular coiling. Interv Neuroradiol. 2009;15(2):175–8. https://doi.org/10.1177/159101990901500206.
148. Duffill J, Lang DA, Dwyer GN. Subarachnoid haemorrhage in a child from an aneurysm of a persistent primitive hypoglossal artery. Br J Neurosurg. 1996;10(6):607–10. https://doi.org/10.1080/02688699646952.
149. Hui FK, Schuette AJ, Cawley CM. Endovascular treatment of an aneurysm of a persistent primitive hypoglossal artery with complete resolution of brainstem compressive symptoms: case report. Neurosurgery. 2011;68(3):E854–7; discussion E7. https://doi.org/10.1227/NEU.0b013e3182077d75.
150. Kim T, Chung S, Lanzino G. Carotid artery-hypoglossal nerve relationships in the neck: an anatomical work. Neurol Res. 2009;31(9):895–9. https://doi.org/10.1179/174313209X385590.
151. Andoh K, Tanohata K, Moriya N, Hagiwara H, Lee J, Sato M, et al. The posterior inferior cerebellar artery arising from the extracranial segment of the internal carotid artery via the hypoglossal canal without an interposed segment of the basilar artery: a persistent primitive hypoglossal artery variant. Clin Imaging. 2001;25(2):86–9. https://doi.org/10.1016/s0899-7071(01)00265-0.
152. Luh GY, Dean BL, Tomsick TA, Wallace RC. The persistent fetal carotid-vertebrobasilar anastomoses. AJR Am J Roentgenol. 1999;172(5):1427–32. https://doi.org/10.2214/ajr.172.5.10227532.
153. Okahara M, Kiyosue H, Mori H, Tanoue S, Sainou M, Nagatomi H. Anatomic variations of the cerebral arteries and their embryology: a pictorial review. Eur Radiol. 2002;12(10):2548–61. https://doi.org/10.1007/s00330-001-1286-x.
154. Li TH, Lan MY, Liu JS, Tseng YL, Wu HS, Chang YY. Type II proatlantal intersegmental artery associated with objective pulsatile tinnitus. Neurology. 2008;71(4):295–6. https://doi.org/10.1212/01.wnl.0000318276.07527.70.
155. Yilmaz E, Ilgit E, Taner D. Primitive persistent carotid-basilar and carotid-vertebral anastomoses: a report of seven cases and a review of the literature. Clin Anat. 1995;8(1):36–43. https://doi.org/10.1002/ca.980080107.
156. Vasovic L, Mojsilovic M, Andelkovic Z, Jovanovic I, Arsic S, Vlajkovic S, et al. Proatlantal intersegmental artery: a review of normal and pathological features. Childs Nerv Syst. 2009;25(4):411–21. https://doi.org/10.1007/s00381-008-0765-7.
157. Horowitz M, Bansal S, Dastur K. Aortic arch origin of the left external carotid artery and type II proatlantal fetal anastomosis. AJNR Am J Neuroradiol. 2003;24(3):323–5.
158. Kurose K, Kishi H, Nishijima Y. Type 2 proatlantal artery associated with a ruptured aneurysm–case report. Neurol Med Chir (Tokyo). 1990;30(3):191–3. https://doi.org/10.2176/nmc.30.191.
159. Nakashima K, Itokawa H, Oishi A, Imaizumi Y, Izumiyama H. Persistent primitive first cervical intersegmental artery (proatlantal artery II) associated with subarachnoid hemorrhage of unknown origin. Clin Neurol Neurosurg. 2012;114(1):90–2. https://doi.org/10.1016/j.clineuro.2011.09.006.
160. Pinstein ML, Gerald B. Anomalous communication of the external carotid and vertebral arteries. Persistence of the proatlantal artery. Radiology. 1976;118(3):626. https://doi.org/10.1148/118.3.626.
161. Palmer FJ, Philips RL. Persistent proatlantal artery arising from the common carotid bifurcation. Australas Radiol. 1978;22(3):226–8. https://doi.org/10.1111/j.1440-1673.1978.tb02063.x.
162. Arraez-Aybar LA, Navia-Alvarez P, Mendez-Cendon JC. A case of a type II proatlantal artery with arteriovenous malformation. Surg Radiol Anat. 2011;33(1):85–9. https://doi.org/10.1007/s00276-010-0685-6.
163. Basekim CC, Silit E, Mutlu H, Pekkafali MZ, Ozturk E, Kizilkaya E. Type I proatlantal artery with bilateral absence of the external carotid arteries. AJNR Am J Neuroradiol. 2004;25(9):1619–21.
164. Berger L, Gouicem D, Lebied el D, Felisaz A, Coffin O, Maiza D. Internal carotid and ipsilateral type II proatlantal artery stenoses causing simultaneous hemispheric and vertebrobasilar transient ischemia. J Vasc Surg. 2011;53(2):475–7. https://doi.org/10.1016/j.jvs.2010.08.017.
165. Berman IG, Stuckey SL. Anomalous communication of external carotid and vertebral arteries with associated intracranial parenchymal arteriovenous malformation: magnetic resonance angiography and angiographic findings. Australas Radiol. 2003;47(4):472–4. https://doi.org/10.1046/j.1440-1673.2003.01225.x.
166. Capel C, Peltier J, Lefranc M, Desenclos C, Chivot C, Deramond H, et al. Type II proatlantal artery with unusual origin of the occipital artery feeding of a type IV dural arteriovenous fistula: case report. Neurochirurgie. 2013;59(6):214–7. https://doi.org/10.1016/j.neuchi.2013.08.007.
167. Choudhary G, Adhikari N, Chokr J, Gupta N. Type 2 persistent primitive proatlantal intersegmental artery, a rare variant of persistent carotid-vertebrobasilar anastomoses. Proc (Baylor Univ Med Cent). 2019;32(1):101–4. https://doi.org/10.1080/08998280.2018.1533312.
168. Legre J, Tapias PL, Nardin JY, Philip E, Perier E. Embryonic intersegmental anastomosis between the external carotid and vertebral arteries. Classification problems of the proatlantal artery. J Neuroradiol. 1980;7(2):97–104.
169. Liechty JM, Weddle RJ, Shutze WP, Smith BL. Occurrence of a type 2 proatlantal intersegmental artery during carotid endarterectomy for symptomatic stenosis. J Vasc Surg. 2016;64(3):807–8. https://doi.org/10.1016/j.jvs.2014.10.100.
170. Obayashi T, Furuse M. The proatlantal intersegmental artery: a case report and review of the literature. Arch Neurol. 1980;37(6):387–9. https://doi.org/10.1001/archneur.1980.00500550089018.
171. Rao TS, Sethi PK. Persistent proatlantal artery with carotid-vertebral anastomosis. Case report. J Neurosurg. 1975;43(4):499–501. https://doi.org/10.3171/jns.1975.43.4.0499.
172. Tsai FY, Mahon J, Woodruff JV, Roach JF. Congenital absence of bilateral vertebral arteries with occipital-basilar anastomosis. Am J Roentgenol Radium Ther Nucl Med. 1975;124(2):281–6. https://doi.org/10.2214/ajr.124.2.281.
173. Hackett ER, Wilson CB. Congenital external carotid-vertebral anastomosis. A case report. Am J Roentgenol Radium Ther Nucl Med. 1968;104(1):86–9. https://doi.org/10.2214/ajr.104.1.86.
174. Oner Z, Oner S, Kahraman AS. The right vertebral artery originating from the right occipital artery and the absence of the transverse foramen: a rare anatomical variation. Surg Radiol Anat. 2017;39(12):1397–400. https://doi.org/10.1007/s00276-017-1882-3.
175. Bahsi YZ, Uysal H, Peker S, Yurdakul M. Persistent primitive proatlantal intersegmental artery (proatlantal artery I) results in 'top of the basilar' syndrome. Stroke. 1993;24(12):2114–7. https://doi.org/10.1161/01.str.24.12.2114.
176. Bailey MA, Holroyd HR, Patel JV, Lansbury AJ, Scott DJ. The right vertebral artery arising as a branch of the right internal carotid artery: report of a rare case. Surg Radiol Anat. 2009;31(10):819–21. https://doi.org/10.1007/s00276-009-0523-x.
177. Bloch S, Danziger J. Proatlantal intersegmental artery. Neuroradiology. 1974;7(1):5–8. https://doi.org/10.1007/BF00344667.
178. Briganti F, Tortora F, Elefante A, Volpe A, Maiuri F. Persistent carotid-vertebral anastomosis associated with contralateral acces-

sory middle cerebral artery. Surg Radiol Anat. 2005;27(5):450–4. https://doi.org/10.1007/s00276-005-0002-y.
179. Conforti P, Armenise B, Galligioni F. Anomalous carotid-vertebral anastomosis: primitive cervical segmental artery. Neurochirurgia (Stuttg). 1966;9(3):99–106. https://doi.org/10.1055/s-0028-1095361.
180. Grego F, Stramana R, Lepidi S, Antonello M, Bonvini S, Zaramella M, et al. Primitive proatlantal intersegmental artery and carotid endarterectomy. J Vasc Surg. 2004;39(3):691. https://doi.org/10.1016/s0741-5214(03)00712-2.
181. Gumus T, Onal B, Ilgit ET. Bilateral persistence of type 1 proatlantal arteries: report of a case and review of the literature. AJNR Am J Neuroradiol. 2004;25(9):1622–4.
182. Hutchinson NA, Miller JD. Persistent proatlantal artery. J Neurol Neurosurg Psychiatry. 1970;33(4):524–7. https://doi.org/10.1136/jnnp.33.4.524.
183. Kolbinger R, Heindel W, Pawlik G, Erasmi-Korber H. Right proatlantal artery type I, right internal carotid occlusion, and left internal carotid stenosis: case report and review of the literature. J Neurol Sci. 1993;117(1–2):232–9. https://doi.org/10.1016/0022-510x(93)90178-2.
184. Lode I. Persistence of both proatlantal arteries with occlusion of the internal carotid arteries. A case report. Acta Radiol. 2000;41(5):410–1. https://doi.org/10.1080/028418500127345758.
185. Montechiari M, Iadanza A, Falini A, Politi LS. Monolateral type I proatlantal artery with bilateral absence of vertebral arteries: description of a case and review of the literature. Surg Radiol Anat. 2013;35(9):863–5. https://doi.org/10.1007/s00276-013-1086-4.
186. Morales M, Anacleto A, Berbert M, Mello M, Centola C, Piccinato CE, et al. Endovascular treatment for recurrent carotid stenosis in a patient with proatlantal intersegmental artery. J Vasc Interv Radiol. 2006;17(2 Pt 1):399–400. https://doi.org/10.1097/01.RVI.0000196319.48140.D7.
187. Parkinson D, Reddy V, Ross RT. Congenital anastomosis between the vertebral artery and internal carotid artery in the neck. Case report. J Neurosurg. 1979;51(5):697–9. https://doi.org/10.3171/jns.1979.51.5.0697.
188. Pulli R, Gatti M, Pratesi C. Carotid endarterectomy in rare bilateral carotid-vertebral anastomosis. J Vasc Surg. 2001;33(5):1122–4. https://doi.org/10.1067/mva.2001.113975.
189. Saito N, Uchino A, Ishihara S. Complex anomalies of type 1 proatlantal intersegmental artery and aortic arch variations. Surg Radiol Anat. 2013;35(2):177–80. https://doi.org/10.1007/s00276-012-1017-9.
190. Schoof J, Skalej M, Halloul Z, Wunderlich MT. Carotid endarterectomy in a patient with persistent proatlantal artery. Cerebrovasc Dis. 2007;23(5–6):458–9. https://doi.org/10.1159/000101748.
191. Tsukamoto S, Hori Y, Utsumi S, Tanigake T, Horiike N, Otani R. Proatlantal intersegmental artery with absence of bilateral vertebral arteries. Case report. J Neurosurg. 1981;54(1):122–4. https://doi.org/10.3171/jns.1981.54.1.0122.
192. Woodcock RJ, Cloft HJ, Dion JE. Bilateral type 1 proatlantal arteries with absence of vertebral arteries. AJNR Am J Neuroradiol. 2001;22(2):418–20.
193. Chan PN, Yu SC, Boet R, Poon WS, Ahuja AT. A rare congenital anastomosis between the vertebral artery and internal carotid artery. AJNR Am J Neuroradiol. 2003;24(9):1885–6.
194. Lui CC, Liu YH, Wai YY, Tsai CC. Persistence of both proatlantal arteries with absence of vertebral arteries. Neuroradiology. 1987;29(3):304–5. https://doi.org/10.1007/BF00451775.
195. Okumura A, Lee T, Shimojima K, Hisata K, Shoji H, Takanashi J, et al. Brainstem disconnection associated with nodular heterotopia and proatlantal arteries. Am J Med Genet A. 2009;149A(11):2479–83. https://doi.org/10.1002/ajmg.a.33044.
196. Pelz D, Menon RK, Swartz RH, Aviv RI, Symons SP. Bilateral Type II persistent proatlantal intersegmental arteries. Can J Neurol Sci. 2013;40(6):873–4. https://doi.org/10.1017/s031716710001605x.
197. Purkayastha S, Gupta AK, Varma R, Kapilamoorthy TR. Proatlantal intersegmental arteries of external carotid artery origin associated with Galen's vein malformation. AJNR Am J Neuroradiol. 2005;26(9):2378–83.
198. Zarghouni M, Marichal D. Persistent bilateral proatlantal type II artery. Proc (Baylor Univ Med Cent). 2013;26(1):50–1. https://doi.org/10.1080/08998280.2013.11928918.
199. Alexander MD, English J, Hetts SW. Occipital artery anastomosis to vertebral artery causing pulsatile tinnitus. J Neurointerv Surg. 2014;6(2):e15. https://doi.org/10.1136/neurintsurg-2012-010632.rep.
200. Flynn RE. External carotid origin of the dominant vertebral artery. Case report. J Neurosurg. 1968;29(3):300–1. https://doi.org/10.3171/jns.1968.29.3.0300.
201. Tanaka Y, Hara H, Momose G, Kobayashi S, Kobayashi S, Sugita K. Proatlantal intersegmental artery and trigeminal artery associated with an aneurysm. Case report. J Neurosurg. 1983;59(3):520–3. https://doi.org/10.3171/jns.1983.59.3.0520.
202. Bour P, Bracard S, Frisch N, Frisch R, Fieve G. Persistent proatlantal artery associated with carotid artery stenosis treatment by percutaneous transluminal balloon angioplasty. Ann Vasc Surg. 1991;5(1):38–40. https://doi.org/10.1007/BF02021775.
203. Richter HR. Collaterals between the external carotid artery and the vertebral artery in cases of thrombosis of the internal carotid artery. Acta Radiol. 1953;40(2–3):108–12. https://doi.org/10.3109/00016925309176574.
204. Kwak S, Nagashima T, Kobayashi S. Anomalous origin of the posterior meningeal artery from the internal carotid artery. Neuroradiology. 1980;19(2):103–4. https://doi.org/10.1007/BF00342604.