Nivedita Agarwal
John D. Port
Editors

Neuroimaging: Anatomy Meets Function

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Editors
Nivedita Agarwal
S. Maria del Carmine Hospital
Azienda Provinciale per i Servizi Sanitari
Rovereto (TN)
Italy

Center for Mind/Brain Sciences (CIMeC)
University of Trento
Rovereto (TN)
Italy

Department of Radiology
Section of Neuroradiology
University of Utah
Salt Lake City (UT)
USA

John D. Port
Department of Radiology
Mayo Clinic
Rochester
Minnesota
USA

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This book is dedicated to my dear friend and extraordinary colleague, Dr. Andrea Poretti, who unexpectedly left this Earth long before he should have. Andrea has been a wonderful teacher, a brilliant neuroscientist, and an awesome friend. His boundless passion for neuroscience lives on as his legacy.

To all those who are in constant search of truth

—Nivedita

Foreword by Anne G. Osborn

Brain anatomy, pathology, and function go hand in hand with clinical findings. Correlating a given pattern of imaging findings with the patient's symptomatology is essential to providing optimal clinical care. While there are a number of texts that focus on one or the other of these essential components, I'm not aware of any that attempt to integrate both of them. This book does just that. It is divided into two main sections covering the supratentorial brain structures and the infratentorial brain stem and the cerebellum. Within each section, extremely detailed anatomical structures have been labeled on the anatomical MR slices as well as diffusion tensor imaging which is now routinely used in most clinical practices. In the chapter(s) that follow, detailed functional descriptions of these labeled gray matter structures and major white matter tracts are provided.

Simple and concise, each section provides detailed information including the embryology, anatomical course, and function of the normal structures as well as pathology and major neurological syndromes. Of special note is the surgical chapter with exquisite hand-drawn diagrams that illustrate common surgical approaches.

I have known Dr. Nivedita Agarwal for almost a decade. She is a double-boarded neurologist and neuroradiologist. She is adept at bringing brain anatomy and brain function together. She is a busy clinical neuroradiologist as well as an excellent teacher at several courses including a course of her own (on which this book is based).

I can see the immediate practicality of this book as an essential reference in radiology departments and neurology clinics and among medical students, residents, and fellows. This unique book is a wonderful addition to the neuroscientific community. It should be immediately at hand in all neuroradiology reading rooms!

Anne G. Osborn, M.D
University Distinguished Professor
William H. and Patricia W. Child Presidential Endowed Chair
Department of Radiology and Imaging Sciences
University of Utah School of Medicine
Salt Lake City, UT, USA

Foreword by E. Turgut Tali

I am delighted to write the Foreword to this book prepared by two outstanding neuroradiologists, Dr. Agarwal and Dr. Port. Functional neuroradiology and MR tractography have opened important new avenues of research, but they are increasingly being used for patient care as opposed to their more traditional role evaluating brain anatomy. Anatomy is always a "must know," and dazzling developments in cross-sectional imaging have changed everything by providing detailed 2-D, 3-D, and even 4-D images in vivo. On the way to explaining the "why" of "what" as Dr. Agarwal states, this book will be an important reference with excellent images obtained using the latest technology, superbly crafted illustrations, and well-written, easily referenced text. Drs. Agarwal and Port, together with several other world-renowned neuroradiologists, have combined their knowledge and experience with extensive coverage of anatomy and function that is critical for the daily practice of neuroradiology. Dr. Agarwal and Dr. Port should be congratulated on their excellent work. I have complete confidence in this work and wish great success for this remarkable book.

E. Turgut Tali, M.D.
Professor of Radiology & Neuroradiology
Director of Section of Neuroradiology,
Gazi University School of Medicine
Ankara, Turkey

Preface by Nivedita Agarwal

"You see what you know, and you search for what you know. If you don't know what to see, you will not search for it."

—me

This work would not have been possible without the several people who have believed in me from even before I started to believe in myself and in the path I had undertaken. As a neurologist, I found myself struggling to delineate between the "what" and the "why" of the diagnosis. I needed the skills to look at the brain and specifically into the areas of the brain that I thought were impacted. As I embarked on my own path toward neuroradiology, I discovered that to know the "why" of "what" you *see*, my search for the "how" to diagnose and become a more effective neuroradiologist.

I am specially thankful to my many mentors who saw in me the potential and pushed me to reach places of personal success especially Prof. Perry R. Renshaw, for his astounding promise of support, teaching, and mentorship, and Prof. Anne G. Osborn, our world-renowned neuroradiologist, for her profound influence on my academic course and continuing to remain a model teacher and inspirational woman. A million thanks to my coeditor John for helping me realize this book. He is an extraordinary friend, colleague, and mentor.

As a neuroradiologist in the hospital of Santa Maria del Carmine, in the beautiful city of Rovereto in the northeast of Italy, it is a privilege to be challenged by my colleagues. I'd like to thank my patients and their families, who look up to us to make the right diagnosis, while for us they remain a motivator to continue to learn and excel. I am particularly indebted to Dr. Luciano Flor and Dr. Giorgio Rossi for their invaluable guidance, encouragement, and unfailing trust, text which helped me progress through several tough endeavors as a neuroradiologist.

I am blessed to have undying support from several friends near and far such as Mindy Harris, Sarah J. Strong, Nichole Shepard, Sabina Vennarini, Ilaria Cominotti, and Stefania Cappellupo, my wonderful students and colleagues at the University of Trento, and, finally, my wonderful parents Radhey Shyam and Santosh, my sister Nalini, and my brother Tarun who continue to be my backbone. Last but not the least, I would like to express my heartfelt gratitude for the love and affection I received from Loris, Danila, and Nicola. Thank you.

Rovereto (TN), Italy — Nivedita Agarwal

Preface by John D. Port

I have been exceptionally fortunate to have a career that has taken me around the world, giving me the opportunity to see places and know people in a way that most will never experience. So when my friend and neuroradiology colleague from Rovereto, Italy, called me and asked me to serve as a coeditor on the book that she was writing, my immediate response was not "should I" but rather "how are we going to do it?" Editing this book has been quite the journey as we are separated by 5000 miles or so, but thanks to modern technology such as WhatsApp, Google Drive, Skype, etc., the process has been relatively painless. We have indeed become one world. I hope that you enjoy the fruits of a diverse multinational multidisciplinary collaboration.

This book is dedicated to the many mentors who knew me better than I knew myself, saw a path for me that I could not envision, and helped me reach farther and higher than I ever thought possible. While there are too many to list them all, a few particularly influential mentors need to be honored: first and foremost, my father Dr. Curtis Port who taught me the value of exploring my dreams; Dr. Vernon Mountcastle and Dr. Michael Steinmetz who showed me the world of neuroscience; Dr. Nick Petridis, Dr. William Tito, and Dr. Gustavo Espinosa who helped me find my way through medical training; Dr. Nick Bryan, Dr. Marty Pomper, and Dr. Norman Beauchamp who were pillars of support during my neuroradiology fellowship; and Dr. Cliff Jack, Dr. John Huston, and Dr. David Mrazek who set the stage for my career a few years ago.

I would like to thank Nivedita for inviting me to participate in making her dream a reality; I hope I did a good job for you. Also many thanks to Dr. Bob Watson, neuroradiologist and neuroanatomist extraordinaire, for his expert review of the anatomical chapters. Many thanks to our contributing authors; their expertise was essential for the creation of this book, and I have learned much from their chapters. Finally, and most importantly, I'd like to thank my wife Dolores and my daughters Cristina and Jenna for their love and support over the years. Couldn't do what I do without you.

Rochester, Minnesota, USA — John D. Port

Introduction

Since the first clinical MRI scan was performed by John Mallard and his team at the University of Aberdeen in 1980, magnetic resonance imaging (MRI) technology has had an enormous impact on how disease is diagnosed and managed. While such technology traditionally has been placed within radiology departments and managed by radiologists, a small but growing number of MRI machines are being installed and managed by psychiatry, neurology, orthopedic surgery, and neurosurgery departments. In these non-radiology departments, patients are being diagnosed and treated by physicians who may be experts in their particular field but who lack the proper training to address pathology outside their focal area of expertise.

In contrast, radiologists receive training in image interpretation that extends to all body areas. As such, they are best qualified to evaluate not only for lesions that may explain the current clinical symptomatology but also additional relevant information as well as incidental findings not related to the patient's condition. They can provide a comprehensive assessment of the imaging, often integrating disparate findings into a cohesive diagnosis that explains all of the findings.

Despite this training, some areas of radiology are so complex that additional training is necessary. Radiology has created a number of fellowship programs that provide an additional 1–2 years of focused subspecialty training in these areas. Neuroradiology is one such subspecialty that is widely recognized by hospitals all over the world. Some countries actually offer and require 1 year or more of specific training in the field of neuroradiology. This is followed by an examination that documents their competence in the field, obtaining a "board certification." Neuroradiologists thus can provide added value to patients and referring clinicians by not only providing the best possible imaging protocols tailored to examine specific clinical questions in individual patients but also the highest-quality image interpretations with the most robust differential diagnoses that guide patients and clinicians toward the best possible clinical care.

So if neuroradiology training is so good, then why write this book? In this era of precision medicine, now more than ever, it is important to integrate all of the available information in order to properly diagnose and treat patients. To do so, general radiologists and neuroradiologists will require considerable understanding of what the referring clinician sees at the bedside. Neuroradiology training is designed primarily to focus on detecting and characterizing disease, with reduced emphasis on the underlying functions of

various brain structures or the clinical symptoms that result when they are damaged. In contrast, neurology, psychiatry, and neurosurgery training tends to focus on detecting and characterizing a patient's clinical symptoms and functional deficits, with less emphasis on the detailed anatomical correlates that generate a given set of symptoms.

This book attempts to marry structure and function into a single clinically useful reference guide that cross-references structure with function. It was assembled from Dr. Agarwal's lecture series of the same name, which she has presented countless times over the past few years. The book is intended primarily for general radiologists and neuroradiologists who wish to provide added value to their patients by not only detecting disease in particular brain structures but also predicting which symptoms should be present due to damage of specific brain structures. Neurologists, psychiatrists, and neurosurgeons can also benefit by looking up specific symptoms and then determining which brain structures are involved in generating those symptoms.

The hope for this book is that clinicians who read it will gain the additional knowledge necessary to provide more comprehensive and integrated care to their patients, thereby reducing the overdiagnosis of "incidental" findings, eliminating unnecessary scans and procedures, and ultimately saving time and money while improving diagnosis and clinical outcomes. We hope that you enjoy the additional perspectives presented within these pages, where anatomy meets function.

Nivedita Agarwal
John D. Port

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List of Authors

Nivedita Agarwal, M.D. S. Maria del Carmine Hospital, Azienda Provinciale per i Servizi Sanitari, Rovereto (TN), Italy

Center for Mind/Brain Sciences (CIMeC), University of Trento, Rovereto (TN), Italy

Department of Radiology, Section of Neuroradiology, University of Utah, Salt Lake City (UT), USA

Isabella M. Björkman-Burtscher Department of Medical Imaging and Physiology, Skåne University Hospital, Lund, Sweden

Department of Diagnostic Radiology, Lund University Bioimaging Center, Lund University, Lund, Sweden

Ari M. Blitz, M.D. Department of Neuroradiology, Russell H. Morgan Department of Radiology and Radiological Sciences, Johns Hopkins University School of Medicine, Baltimore, MD, USA

Karin Markenroth Bloch, Ph.D. Lund University Bioimaging Center, Lund University, Lund, Sweden

Karen S. Chen, M.D. Department of Neuroradiology, Russell H. Morgan Department of Radiology and Radiological Sciences, Johns Hopkins University School of Medicine, Baltimore, MD, USA

Arturo Consoli, M.D. Diagnostic and Interventional Neuroradiology Service, Hopital Foch, Suresnes, France

Interventional Neurovascular Unit, Careggi University Hospital, Florence, Italy

Laura B. Eisenmenger, M.D. Department of Radiology and Imaging Sciences, University of Utah Health Sciences Center, Salt Lake City, UT, USA

Craig Johnson, M.D. Department of Radiology and Imaging Sciences, University of Utah Health Sciences Center, Salt Lake City, UT, USA

Salvatore Mangiafico, M.D. Interventional Neurovascular Unit, Careggi University Hospital, Florence, Italy

Miriam E. Peckham, M.D. Department of Radiology and Imaging Sciences, University of Utah Health Sciences Center, Salt Lake City, UT, USA

Andrea Poretti, M.D. Section of Pediatric Neuroradiology, Division of Pediatric Radiology, Russell H. Morgan Department of Radiology and Radiological Science, Johns Hopkins University School of Medicine, Baltimore, MD, USA

John D. Port, M.D., Ph.D. Department of Radiology, Mayo Clinic, Rochester, MN, USA

Andrea Rosi, M.D. Residency Program in Radiology, University of Florence, Careggi University Hospital, Florence, Italy

Luisella Sibilla, M.D. Section of Neuroradiology, Department of Radiology, Sahlgrenska University Hospital, Gothenburg, Sweden

Pia C. Maly Sundgren, M.D., Ph.D. Department of Diagnostic Radiology, Clinical Sciences Lund, Lund University, Lund, Sweden

Department of Medical Imaging and Physiology, Skåne University Hospital, Lund, Sweden

Vinicio M.F. Valente, M.D. Section of Neurosurgery, Hospital of Cosenza (Azienda Ospedaliera di Cosenza), Cosenza, Italy

Richard H. Wiggins III, M.D. Departments of Radiology and Imaging Sciences, Otolaryngology, Head and Neck Surgery, and Biomedical Informatics, University of Utah Health Sciences Center, Salt Lake City, UT, USA

Part I

Cerebrum

Interpreting neuroimages require a thorough knowledge of the anatomy, not only to accurately describe the location of any lesions, but also to correlate them with the patient's specific symptoms or constellation of symptoms. The cerebral brain structures derived from the embryologic telencephalic and diencephalic parts are described in the next six chapters. We present selected clinical-quality axial, coronal and sagittal MRI brain images (Chaps. 1 and 2) labeled with the most clinically relevant brain structures, images intended to serve as an atlas for clinicians and neuroradiologists. Since sulcal and gyral anatomy changes rapidly from one slice to the other, structures that are easy to identify on one slice may not be so obvious on another one. As such, we chose to provide rigorous detail in the labeling of the structures at the cost of being repetitive. We also provide high-resolution 7T MRI images (Chap. 4) that label the small structures not routinely seen on clinical 3T MRI scans. The fine vascular anatomy of the cerebral circulation is illustrated using selective catheter digital subtraction angiographic images (Chap. 3). Finally, the function of the labeled anatomical structures is described in two separate chapters (Chaps. 5 and 6). In these functional chapters, most symptoms and major clinical syndromes are also listed with brief descriptions for easy referencing during reporting.

Structural Anatomy

1

Nivedita Agarwal and John D. Port

The images presented within this chapter are from a single healthy 25-year-old female subject. Images were obtained from a Siemens Skyra 3 tesla MRI scanner using a 32-channel head coil and a volumetric MP-RAGE sequence acquired in the true sagittal plane (TR = 2000 ms, TE 1.85 ms, 1 mm isotropic voxels). Voxel dimensions are 1 mm in all dimensions (isotropic), with reconstructions created in the true axial and true coronal planes. Selected images were chosen for labeling that best represented the local anatomy in a given region. Images are adjusted to accentuate difference between the gray and white matter so that labeling is more obvious. Note that we chose to reconstruct images in the planes most commonly viewed by radiologists; as such, our labels are more relevant to routine clinical MRI than the labels presented in traditional histological atlases. Labels were generated by referencing a number of excellent atlases [1–10].

Each page contains the labeled images on the left-hand side. A small image on the top right of the page documents the locations of the slices, and a key in the lower right-hand side of the page lists the individual structures.

N. Agarwal, M.D. (✉)
S. Maria del Carmine Hospital, Azienda Provinciale per i Servizi Sanitari, Rovereto (TN), Italy

Center for Mind/Brain Sciences (CIMeC), University of Trento, Rovereto (TN), Italy

Department of Radiology, Section of Neuroradiology, University of Utah, Salt Lake City (UT), USA
e-mail: Nivedita.agarwal@apss.tn.it

J.D. Port, M.D., Ph.D.
Department of Radiology, Mayo Clinic, Rochester, MN, USA

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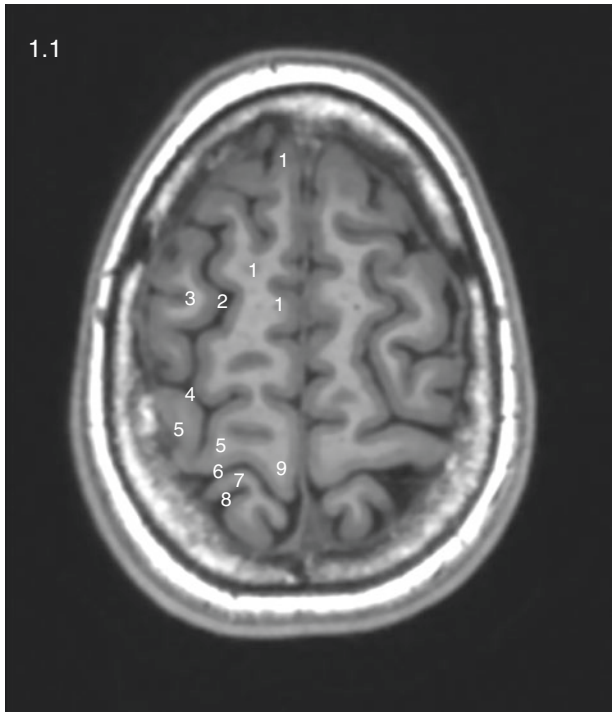
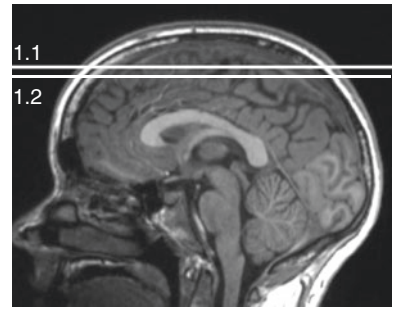
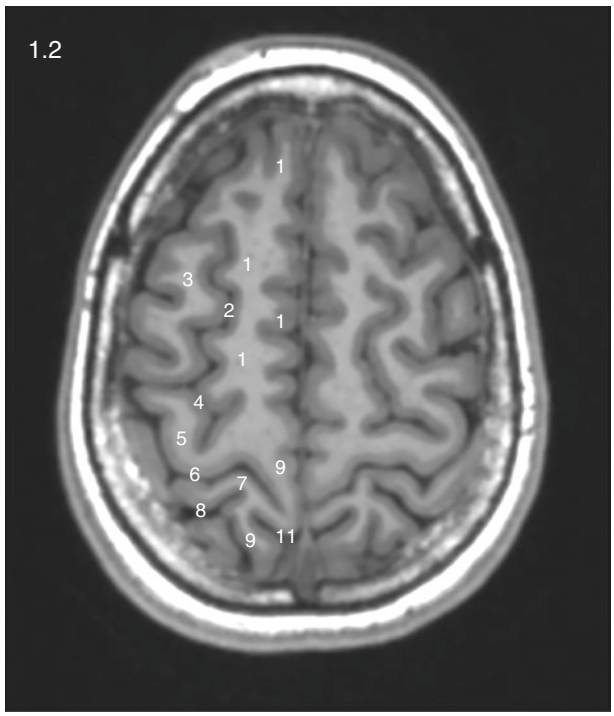
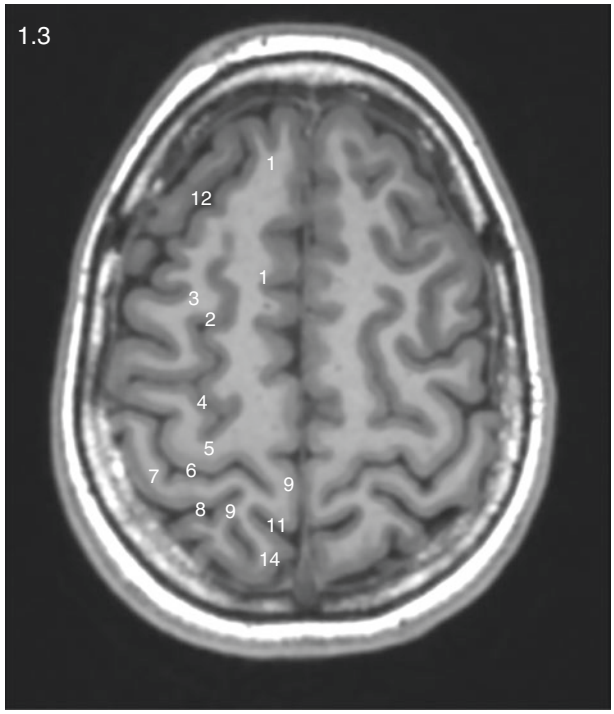
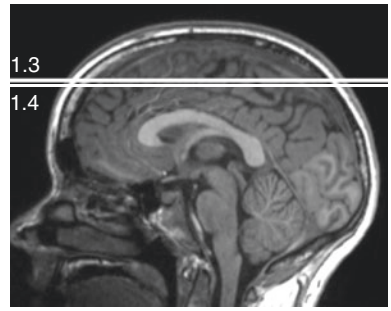




1	Superior frontal gyrus
2	Sup frontal sulcus
3	Middle frontal gyrus
4	Precentral sulcus
5	Precentral gyrus
6	Central sulcus
7	Postcentral gyrus
8	Postcentral sulcus
9	Superior parietal lobe
11	Marginal sulcus (marginal branch of the cingulate sulcus)







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8	Postcentral sulcus
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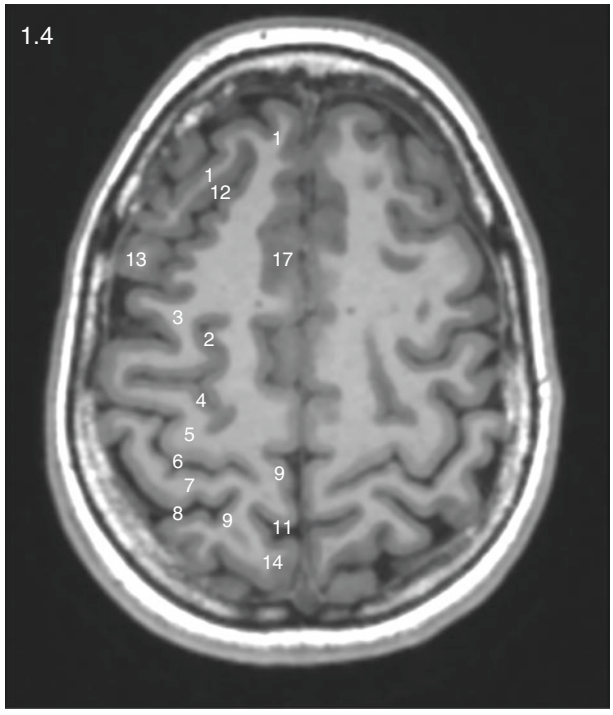
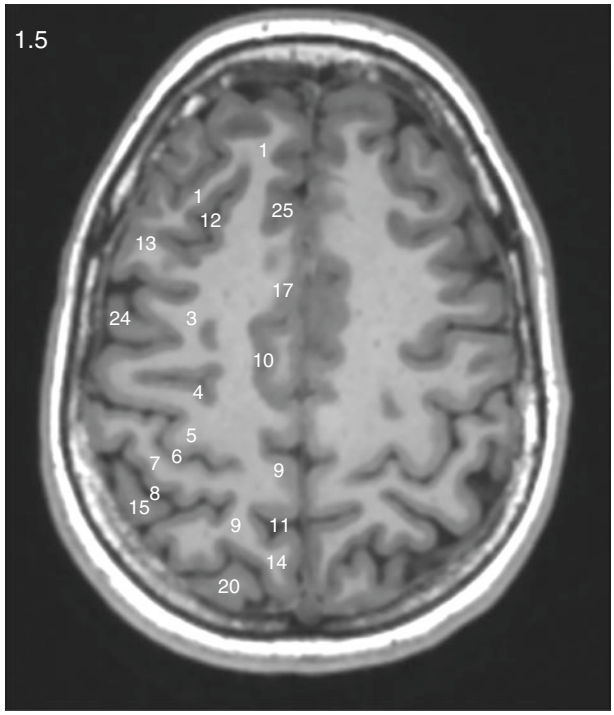
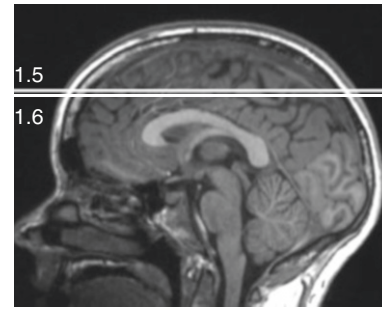
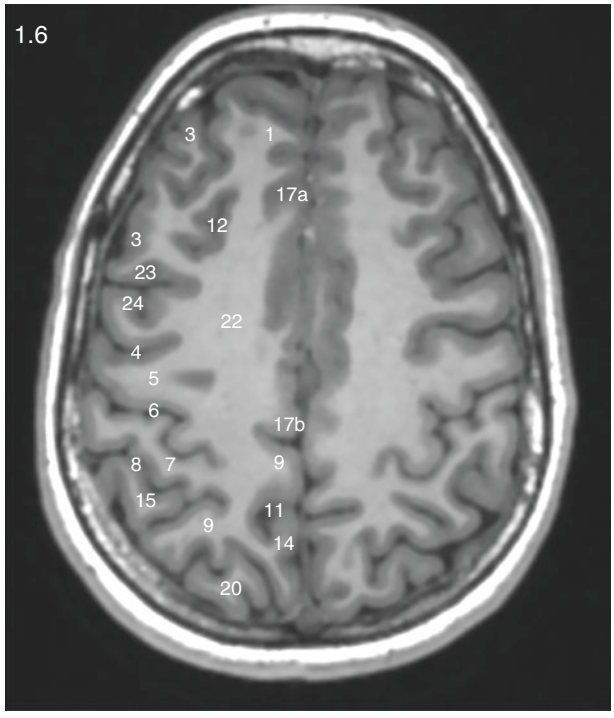
12	Middle frontal sulcus
13	Inferior frontal gyrus
14	Precuneus
17	Cingulate gyrus









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8	Postcentral sulcus
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10	Paracentral lobule
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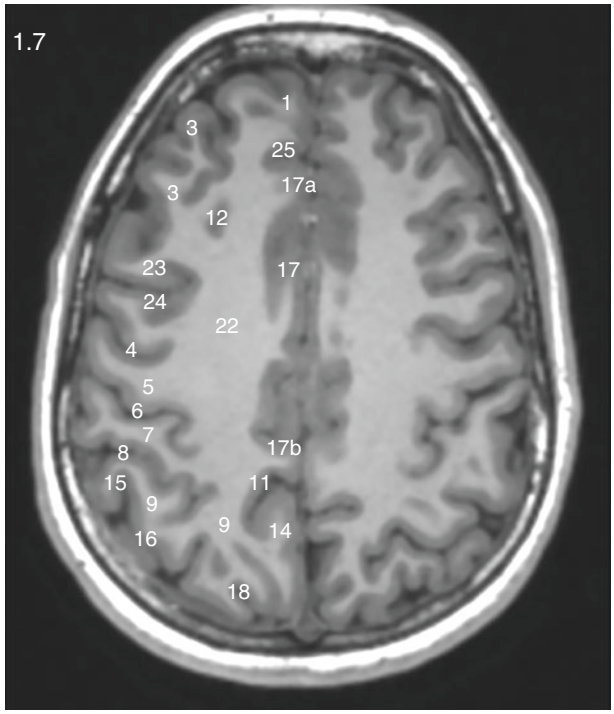
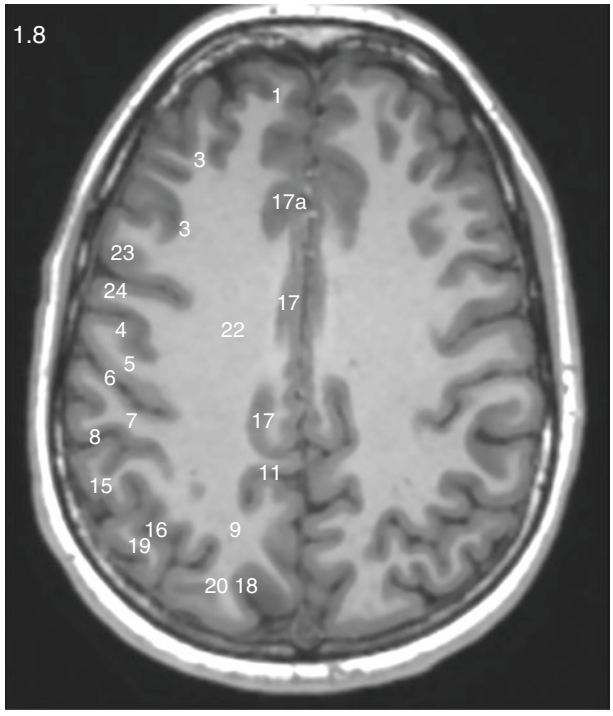
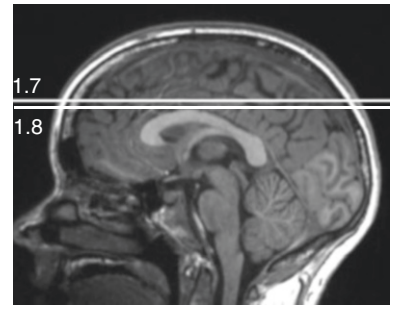
12	Middle frontal sulcus
13	Inferior frontal gyrus
14	Precuneus
15	Supramarginal gyrus (inferior parietal lobule)
17	Cingulate gyrus
17a	Cingulate gyrus, anterior
17b	Cingulate gyrus, posterior
20	Occipital gyri
22	Centrum semiovale
23	Inferior frontal gyrus, pars triangularis
24	Inferior frontal gyrus, pars opercularis







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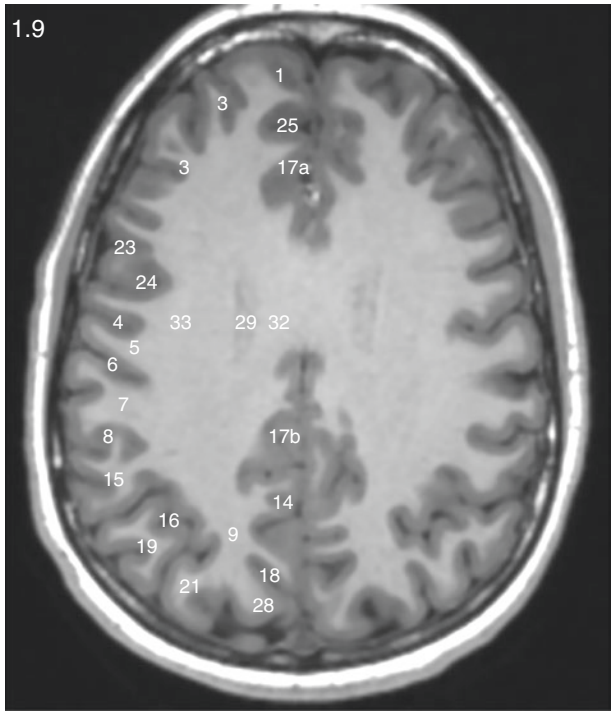

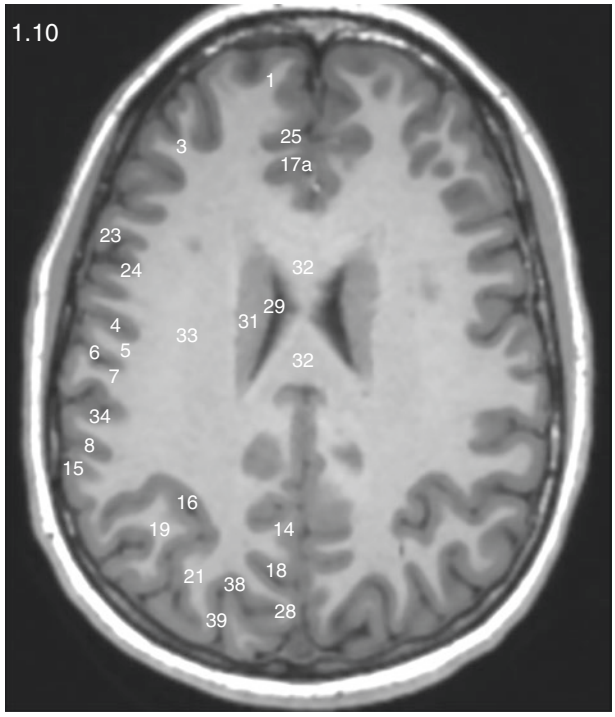

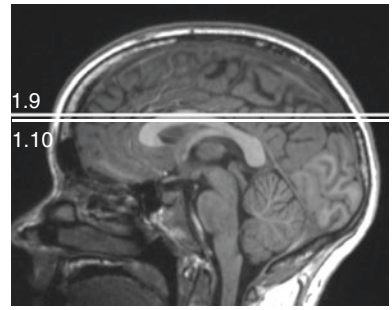

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24	Inferior frontal gyrus, pars opercularis
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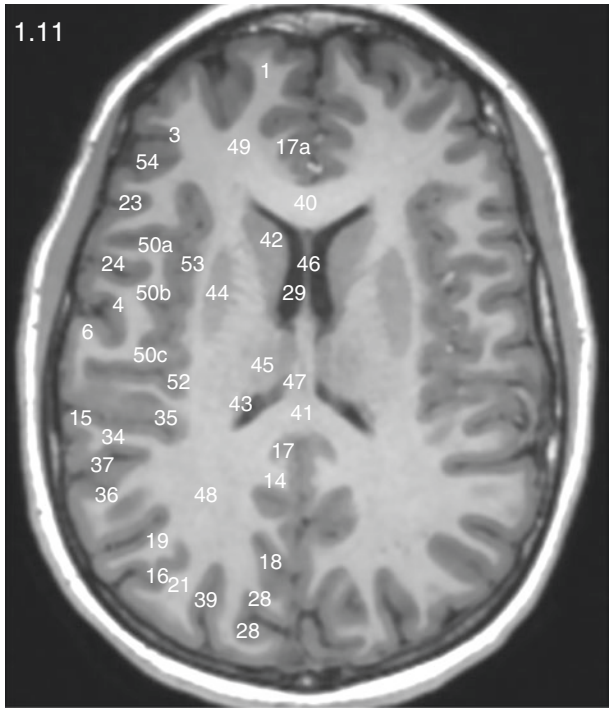
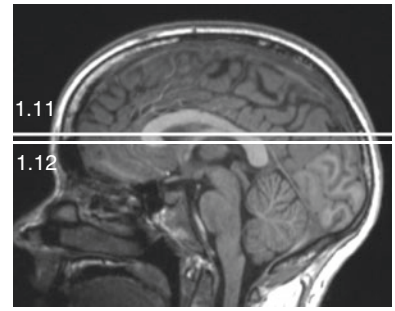
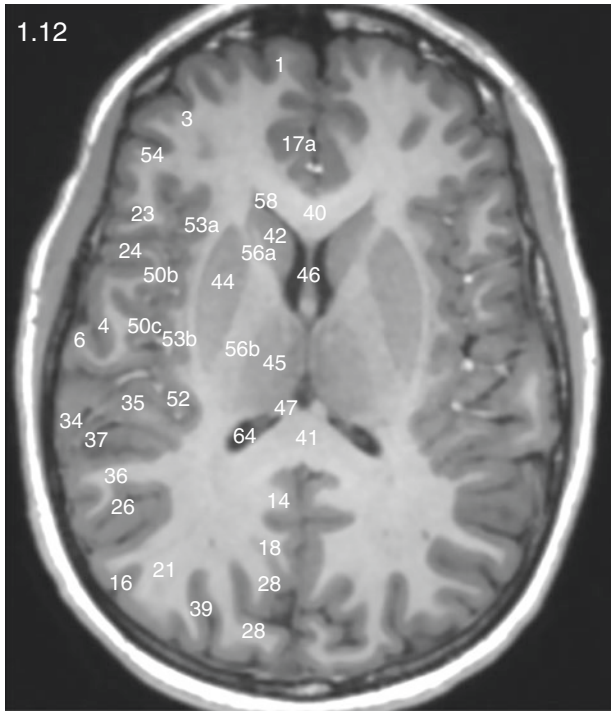
31	Body of the caudate nucleus
32	Body of the corpus callosum
33	Corona radiata
34	Superior temporal gyrus
38	Transverse occipital sulcus
39	Intraoccipital sulcus







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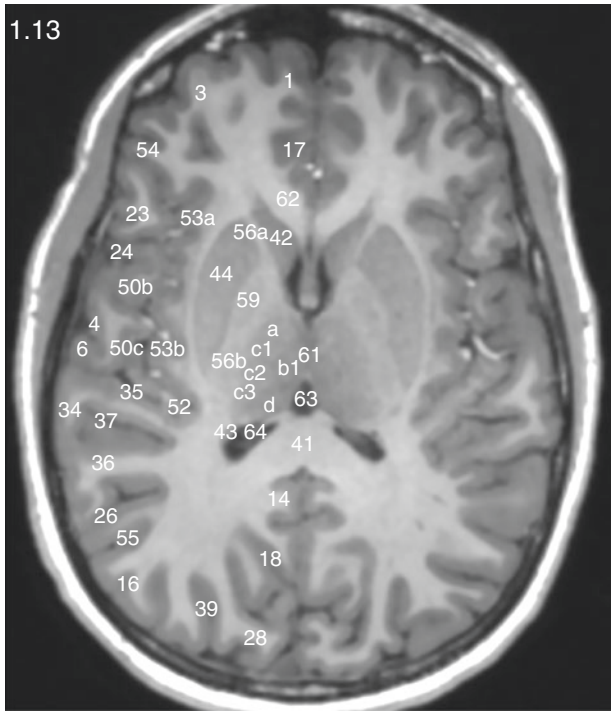
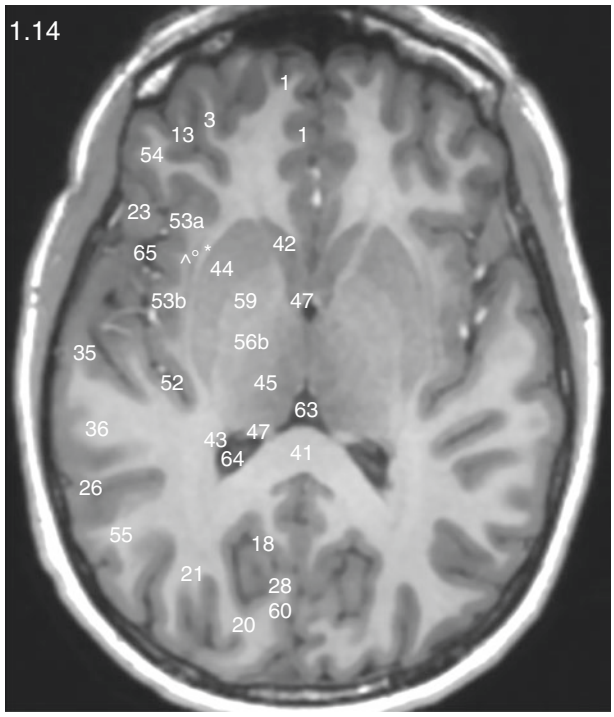
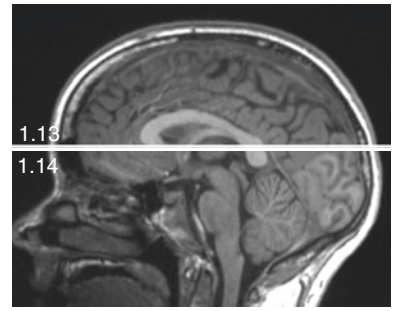
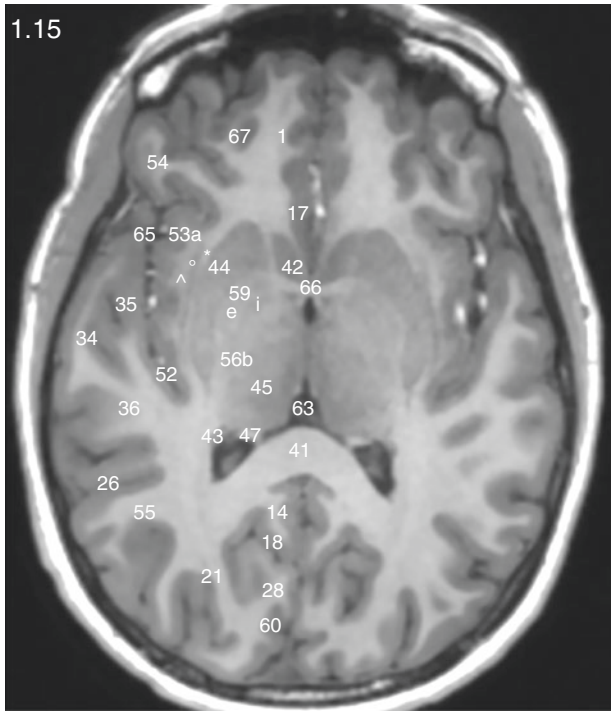
28	Striate gyrus
29	Body of lateral ventricle
34	Superior temporal gyrus
35	Planum temporale
36	Middle temporal gyrus
37	Superior temporal sulcus
39	Intraoccipital sulcus
40	Genu of corpus callosum
41	Splenium of corpus callosum
42	Head of caudate nucleus
43	Tail of caudate nucleus
44	Putamen
45	Thalamus
46	Septum pellucidum
47	Fornix
48	Forceps major
49	Forceps minor
50a	Operculum, frontal
50b	Operculum, parietal
50c	Operculum, temporal
52	Transverse temporal gyrus (Heschl's gyrus)
53	Insula
53a	Insula, short gyri
53b	Insula, long gyri
54	Inferior frontal gyrus, pars orbitalis
56a	Internal capsule, anterior limb
56b	Internal capsule, posterior limb
58	Frontal horn of lateral ventricle
64	Atrium of lateral ventricle

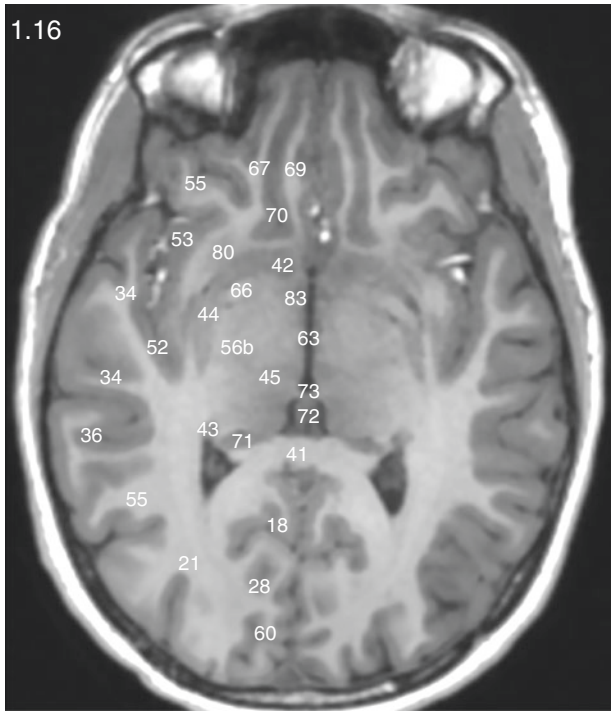


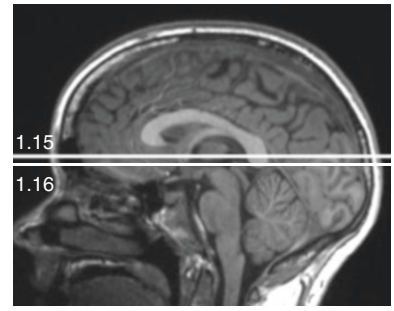




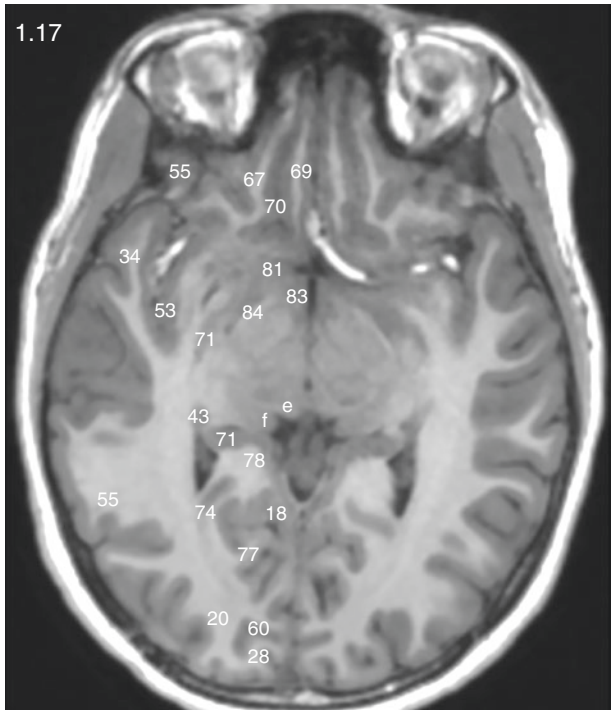
1	Superior frontal gyrus
3	Middle frontal gyrus
4	Precentral sulcus
6	Central sulcus
13	Inferior frontal gyrus
14	Precuneus
16	Intraparietal sulcus
17	Cingulate gyrus
18	Parieto-occipital sulcus
20	Occipital gyri
21	Middle occipital gyrus
23	Inferior frontal gyrus, pars triangularis
24	Inferior frontal gyrus, pars opercularis
26	Inferior temporal sulcus
28	Striate gyrus
34	Superior temporal gyrus
35	Planum temporale
36	Middle temporal gyrus
37	Superior temporal sulcus
39	Intraoccipital sulcus
41	Splenium of corpus callosum
42	Head of caudate nucleus
43	Tail of caudate nucleus
44	Putamen
45	Thalamus
47	Fornix
50b	Operculum, parietal
50c	Operculum, temporal
52	Transverse temporal gyrus (Heschl's gyrus)
53	Insula
53a	Insula, short gyri
53b	Insula, long gyri
54	Inferior frontal gyrus, pars orbitalis
55	Inferior temporal gyrus
56a	Internal capsule, anterior limb
56b	Internal capsule, posterior limb
59	Globus pallidus
60	Calcarine sulcus
61	Intrathalamic adhesion
62	Rostrum of the corpus callosum
63	Third ventricle
64	Atrium of lateral ventricle
65	Sylvian fissure
^	Extreme capsule
*	External capsule
o	Claustrum
a	Anterior nuclei
b1	Dorsomedial nuclei
c1	Ventroanterior nuclei
c2	Ventromedial nuclei
c3	Ventroposteriorlateral nuclei
d	Pulvinar

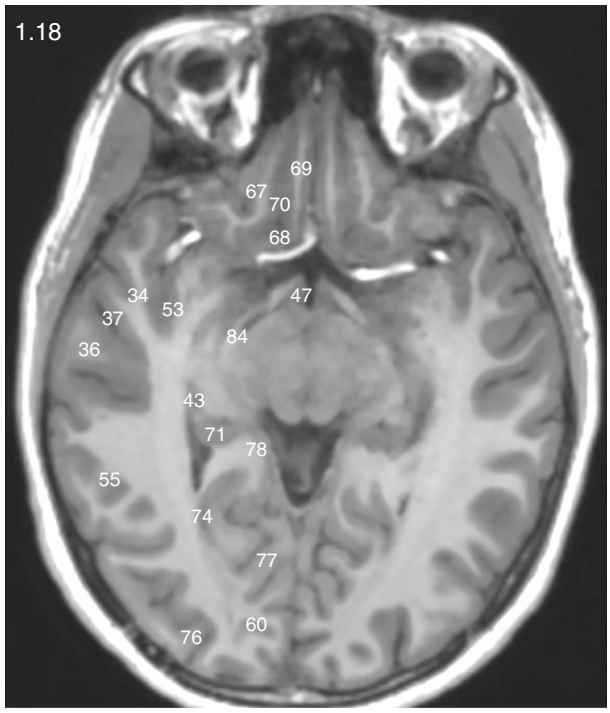


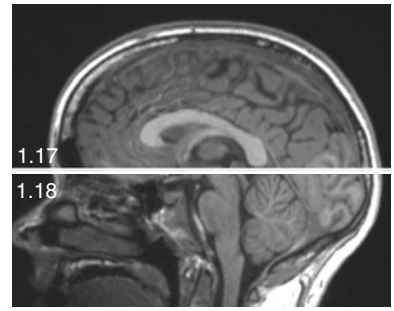




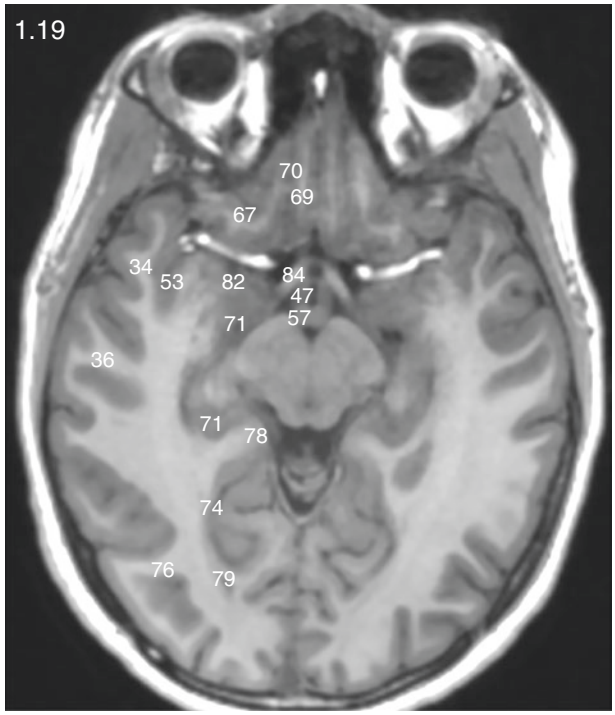
1	Superior frontal gyrus
14	Precuneus
17	Cingulate gyrus
18	Parieto-occipital sulcus
21	Middle occipital gyrus
26	Inferior temporal sulcus
28	Striate gyrus
34	Superior temporal gyrus
35	Planum temporale
36	Middle temporal gyrus
41	Splenium of corpus callosum
42	Head of caudate nucleus
43	Tail of caudate nucleus
44	Putamen
45	Thalamus
47	Fornix
52	Transverse temporal gyrus (Heschl's gyrus)
53	Insula
53a	Insula, short gyri
54	Inferior frontal gyrus, pars orbitalis
55	Inferior temporal gyrus
56b	Internal capsule, posterior limb
59	Globus pallidus
i	Globus pallidus interna
e	Globus pallidus externa
60	Calcarine sulcus
63	Third ventricle
65	Sylvian fissure
66	Anterior commissure
67	Orbital gyrus
69	Straight gyrus
70	Olfactory sulcus
71	Hippocampus
72	Pineal gland
73	Posterior commissure
80	Anterior perforated substance
83	Hypothalamus
^	Extreme capsule
*	External capsule
o	Claustrum

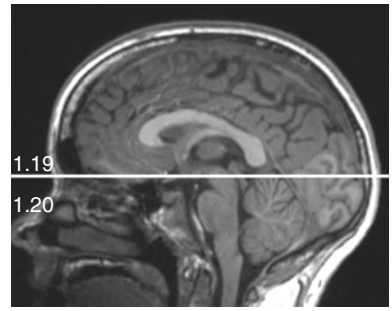


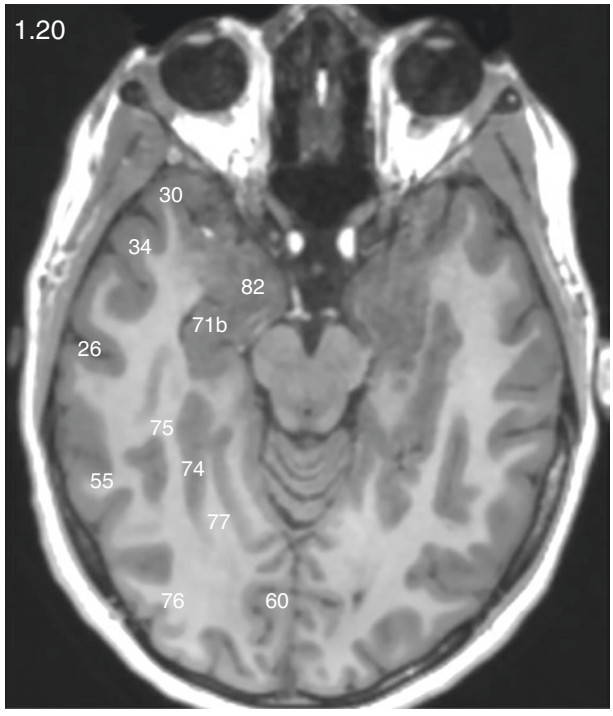




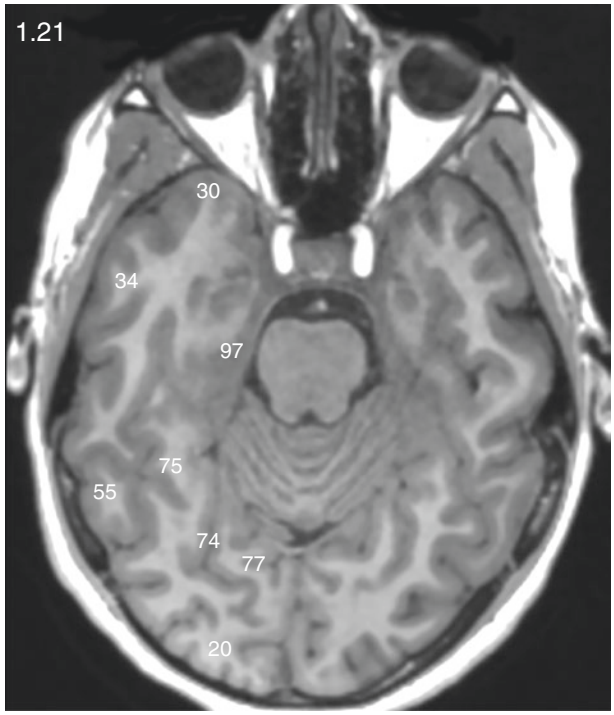
18	Parieto-occipital sulcus
20	Occipital gyri
28	Striate gyrus
34	Superior temporal gyrus
36	Middle temporal gyrus
37	Superior temporal sulcus
43	Tail of caudate nucleus
53	Insula
55	Inferior temporal gyrus
60	Calcarine sulcus
67	Orbital gyrus
68	Paraterminal gyrus
69	Straight gyrus
70	Olfactory sulcus
71	Hippocampus
74	Collateral sulcus
76	Inferior occipital gyrus
77	Lingual gyrus
78	Parahippocampal gyrus
80	Anterior perforated substance
81	Nucleus accumbens
83	Hypothalamus
84	Mammillary body
e	Medial geniculate nucleus
f	Lateral geniculate nucleus

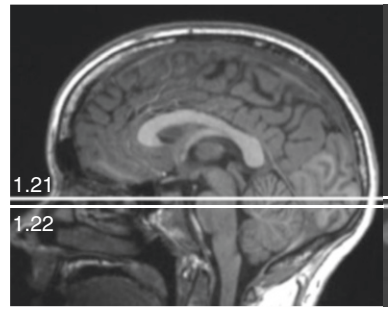




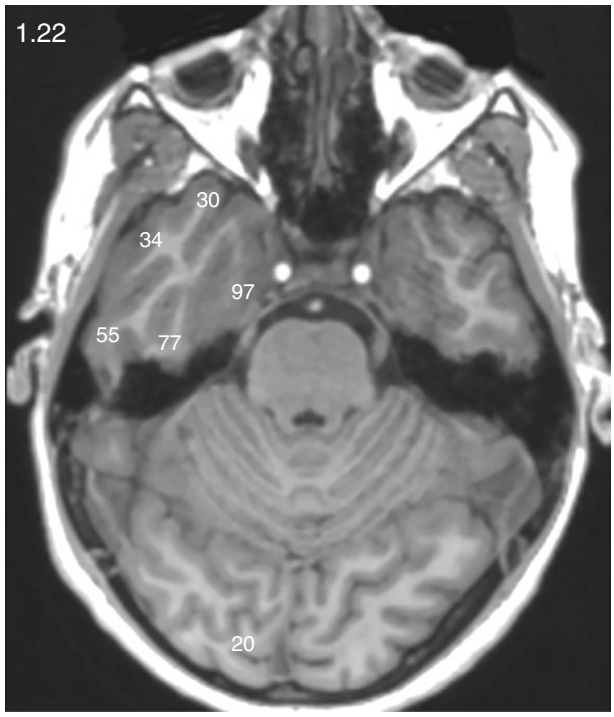


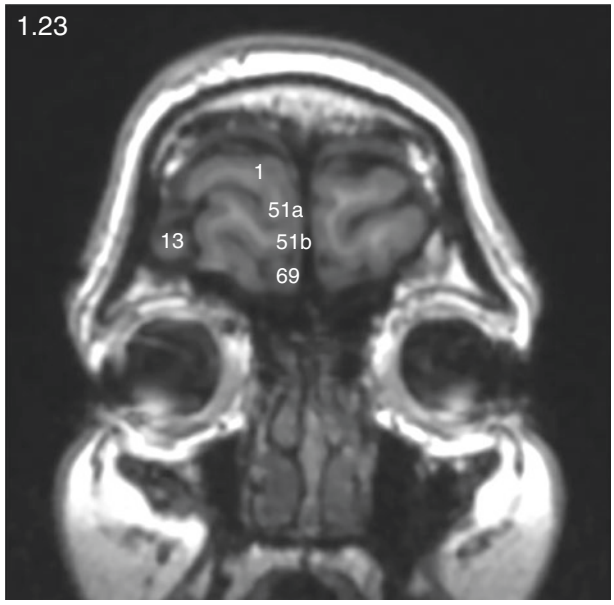
26	Inferior temporal sulcus
30	Temporal pole
34	Superior temporal gyrus
36	Middle temporal gyrus
47	Fornix
53	Insula
55	Inferior temporal gyrus
57	Mammillary body
60	Calcarine sulcus
67	Orbital gyrus
69	Straight gyrus
70	Olfactory sulcus
71	Hippocampus
71b	Hippocampus, fimbria
74	Collateral sulcus
75	Fusiform gyrus
76	Inferior occipital gyrus
77	Lingual gyrus
78	Parahippocampal gyrus
79	Occipital horn, lateral ventricle
82	Amygdala
84	Optic tract

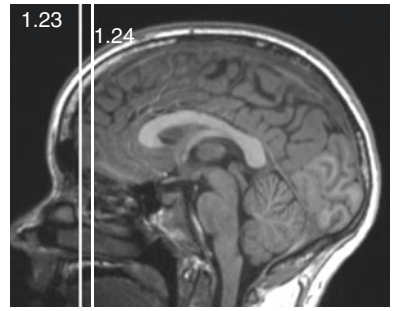




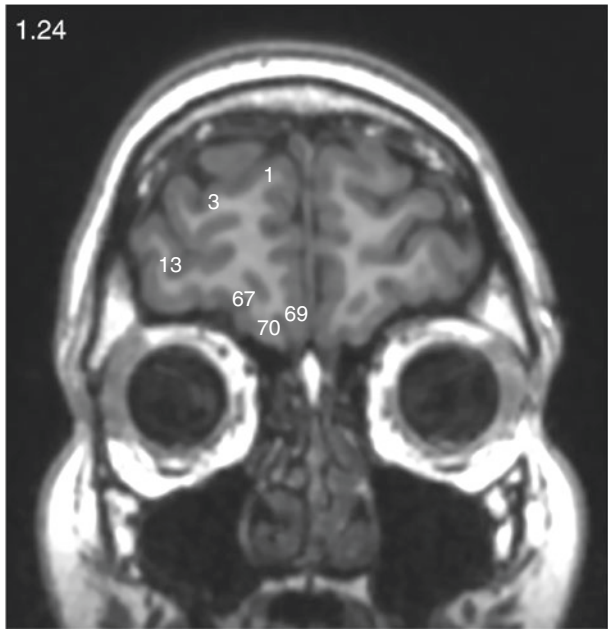
20	Occipital gyri
30	Temporal pole
34	Superior temporal gyrus
55	Inferior temporal gyrus
74	Collateral sulcus
75	Fusiform gyrus
77	Lingual gyrus
97	Entorhinal cortex

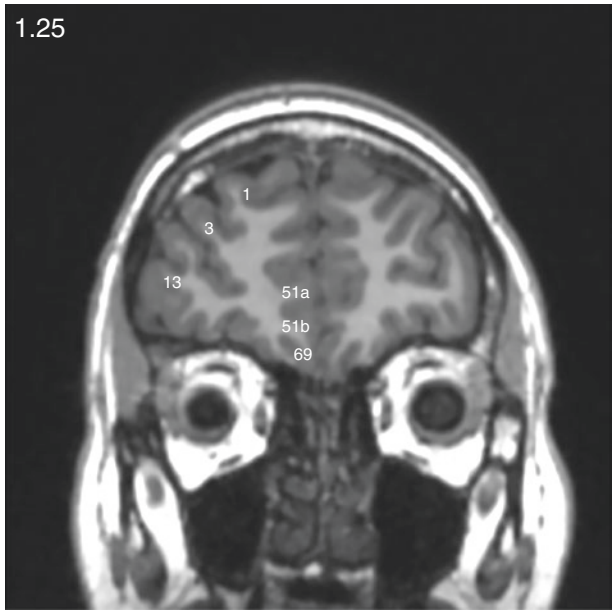


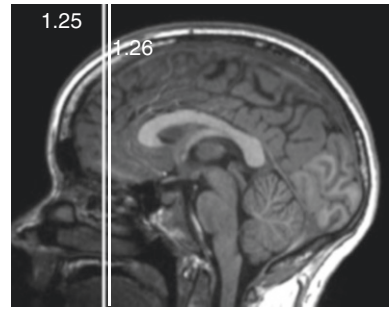




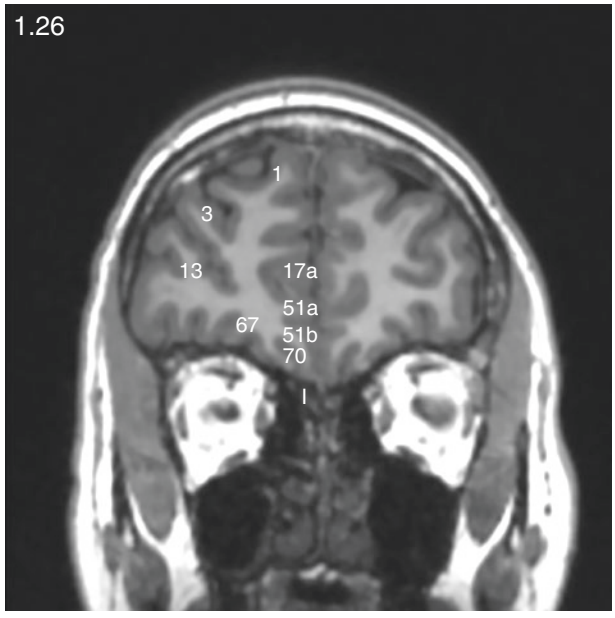
1	Superior frontal gyrus
3	Middle frontal gyrus
13	Inferior frontal gyrus
51a	Superior rostral gyrus
51b	Inferior rostral gyrus
67	Orbital gyrus
69	Straight gyrus
70	Olfactory sulcus

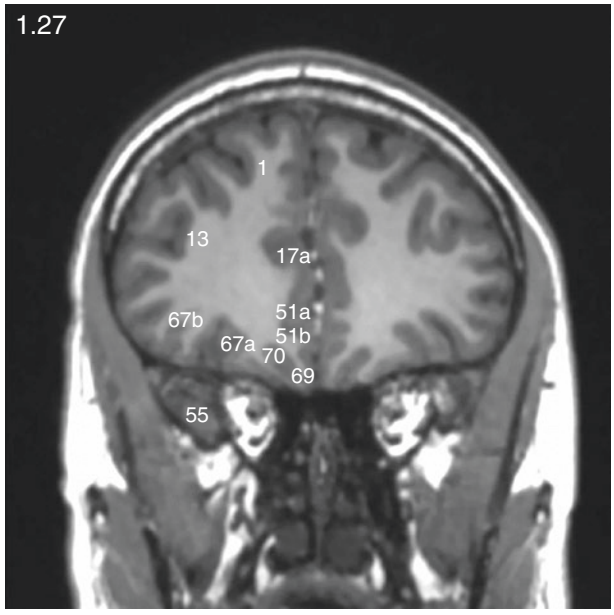


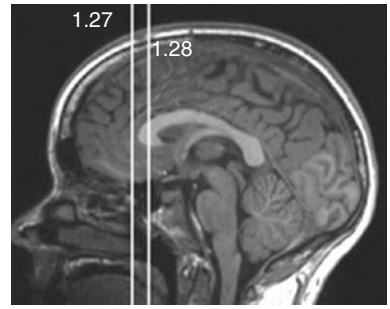


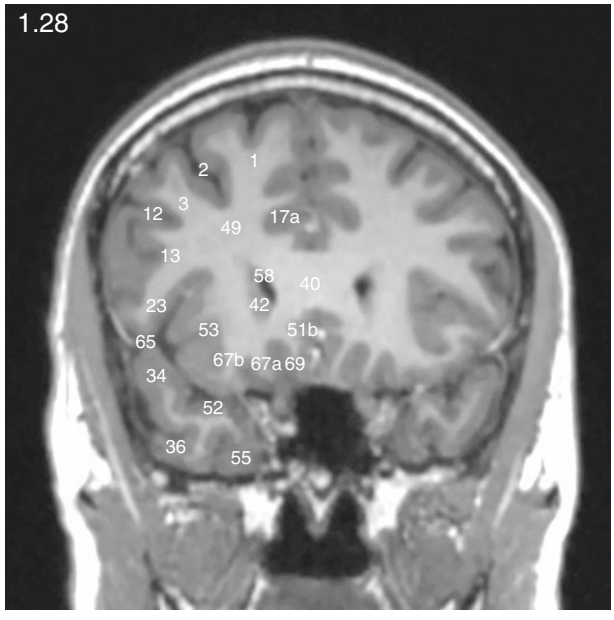


1	Superior frontal gyrus
3	Middle frontal gyrus
13	Inferior frontal gyrus
17a	Cingulate gyrus, anterior
51a	Superior rostral gyrus
51b	Inferior rostral gyrus
67	Orbital gyrus
69	Straight gyrus
70	Olfactory sulcus
I	Olfactory bulb/nerve

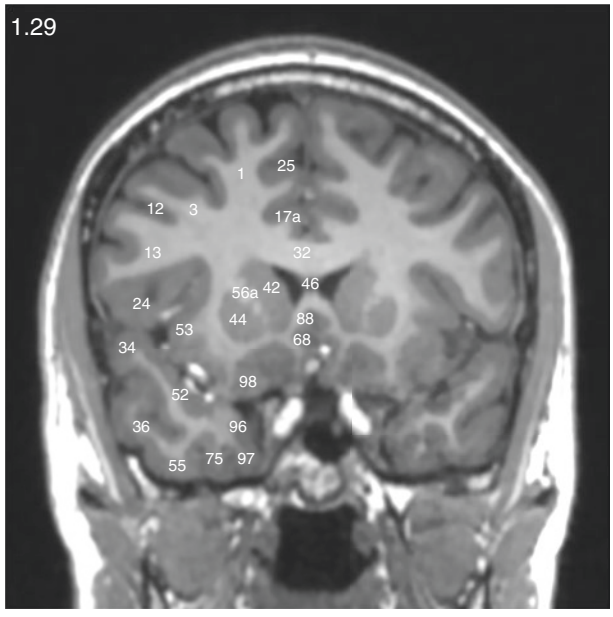


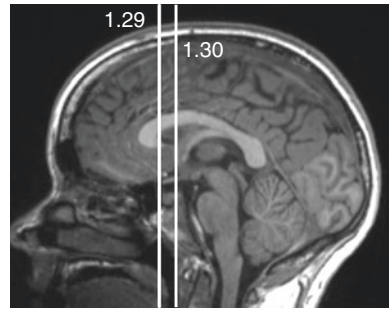


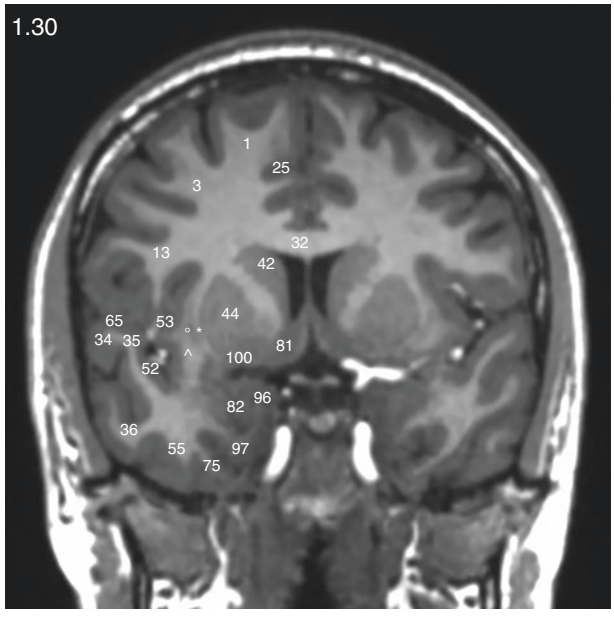




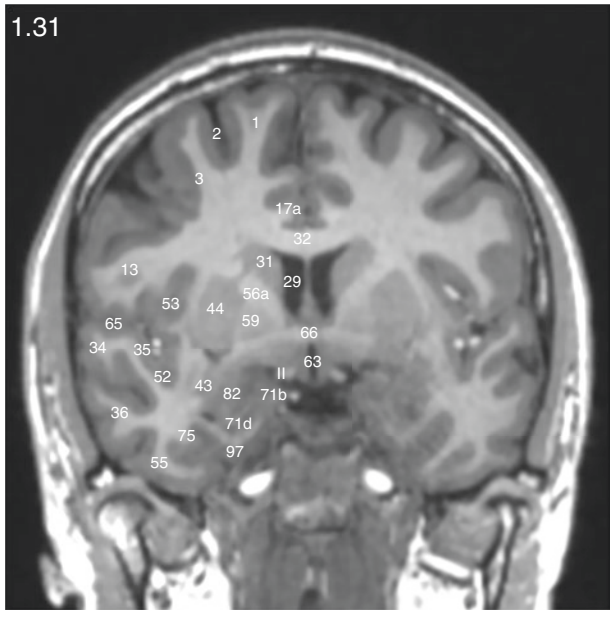
1	Superior frontal gyrus
2	Superior frontal sulcus
3	Middle frontal gyrus
12	Middle frontal sulcus
13	Inferior frontal gyrus
17a	Cingulate gyrus, Anterior
23	Inferior frontal gyrus, pars triangularis
34	Superior temporal gyrus
36	Middle temporal gyrus
40	Genu of corpus callosum
42	Head of caudate nucleus
49	Forceps minor
51a	Superior rostral gyrus
51b	Inferior rostral gyrus
52	Transverse temporal gyrus (Heschl's gyrus)
53	Insula
55	Inferior temporal gyrus
58	Frontal horn of lateral ventricle
65	Sylvian fissure
67a	Medial orbital gyrus
67b	Lateral orbital gyrus
69	Straight gyrus
70	Olfactory sulcus

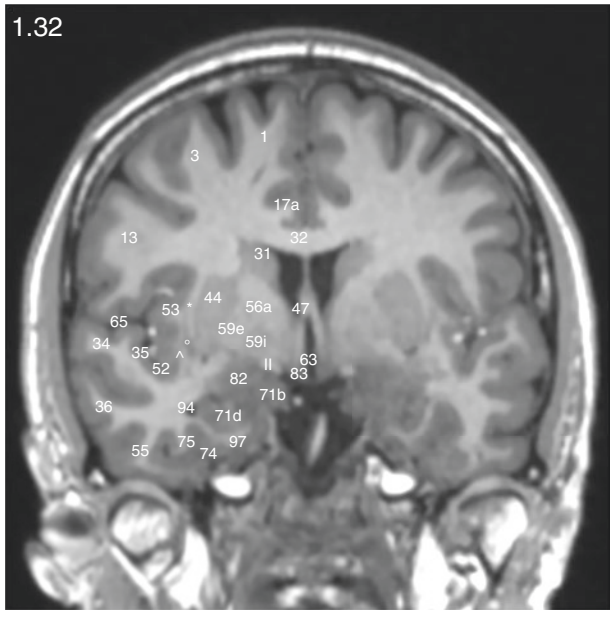


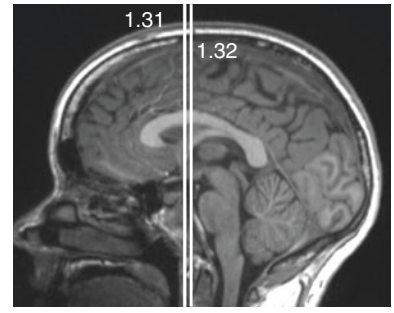




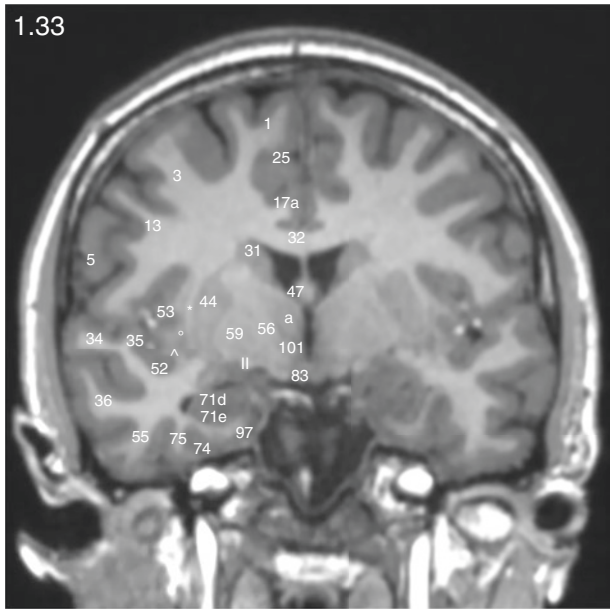
1	Superior frontal gyrus
3	Middle frontal gyrus
12	Middle frontal sulcus
13	Inferior frontal gyrus
17a	Cingulate gyrus, anterior
24	Inferior frontal gyrus, pars opercularis
25	Cingulate sulcus
32	Body of the corpus callosum
34	Superior temporal gyrus
35	Planum temporale
36	Middle temporal gyrus
42	Head of caudate nucleus
44	Putamen
46	Septum pellucidum
52	Transverse temporal gyrus (Heschl's gyrus)
53	Insula
55	Inferior temporal gyrus
56a	Internal capsule, anterior limb
68	Paraterminal gyrus
75	Fusiform gyrus
81	Nucleus accumbens
82	Amygdala
88	Subcallosal gyrus
96	Ambient gyrus
97	Entorhinal cortex
98	Piriform cortex
100	Olfactory tubercle
^	Extreme capsule
*	External capsule
o	Claustrum

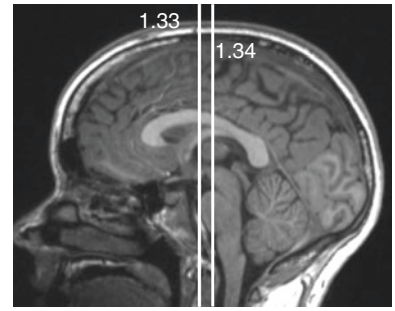


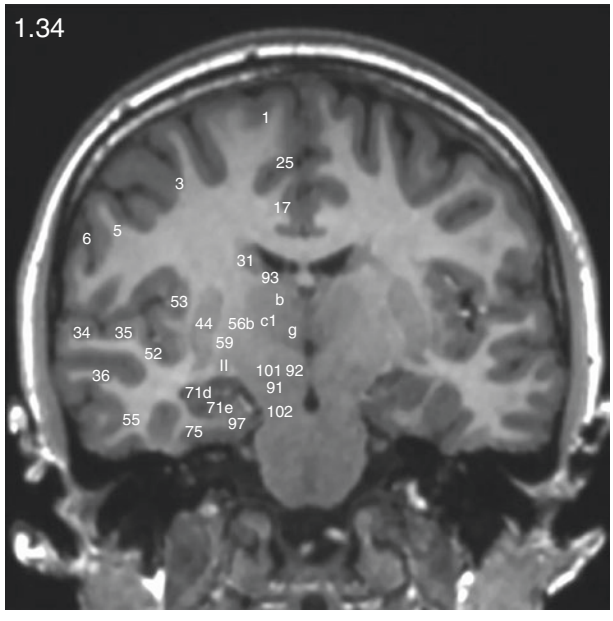




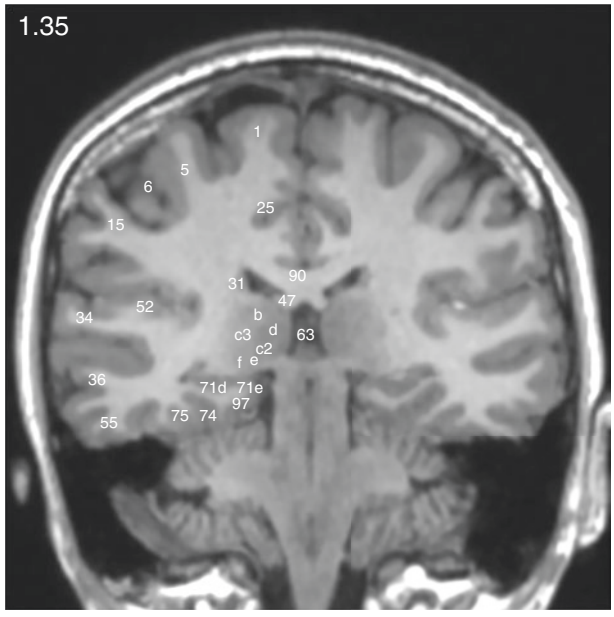
1	Superior frontal gyrus
2	Superior frontal sulcus
3	Middle frontal gyrus
13	Inferior frontal gyrus
17a	Cingulate gyrus, anterior
29	Body of lateral ventricle
31	Body of caudate nucleus
32	Body of corpus callosum
34	Superior temporal gyrus
35	Planum temporale
36	Middle temporal gyrus
43	Tail of caudate nucleus
44	Putamen
47	Fornix
52	Transverse temporal gyrus (Heschl's gyrus)
53	Insula
55	Inferior temporal gyrus
56a	Internal capsule, anterior limb
59	Globus pallidus
59i	Globus pallidus interna
59e	Globus pallidus externa
63	Third ventricle
65	Sylvian fissure
66	Anterior commissure
71b	Hippocampus, uncus
71d	Hippocampus proper
74	Collateral sulcus
75	Fusiform gyrus
82	Amygdala
83	Mammillary body
94	Temporal horn of lateral ventricle
97	Entorhinal cortex
II	Cranial Nerve II
^	Extreme capsule
*	External capsule
o	Claustrum

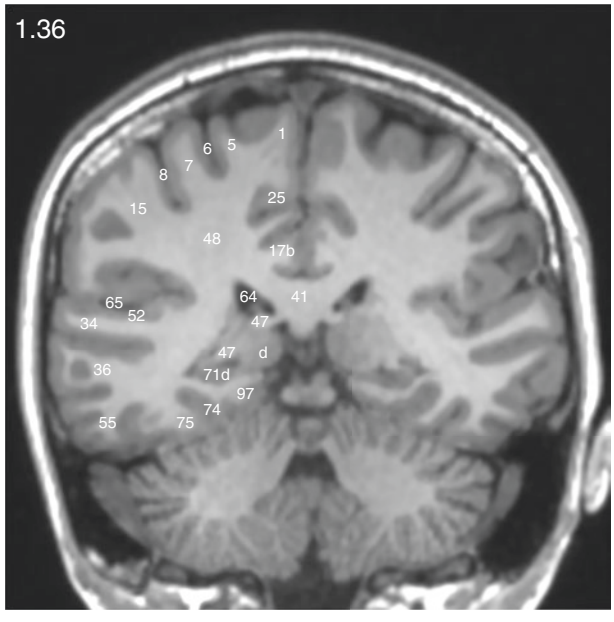


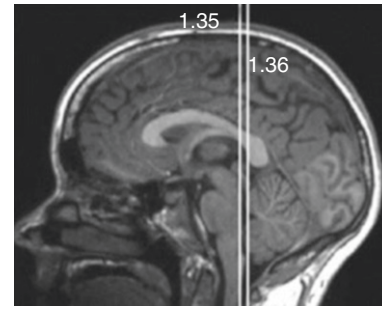




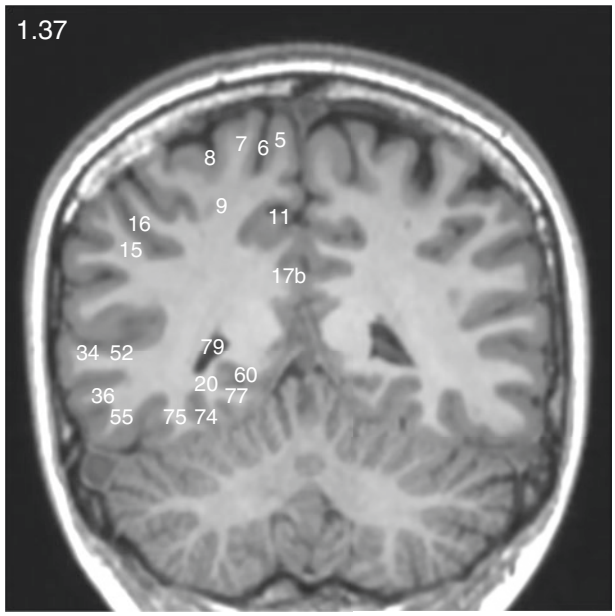
1	Superior frontal gyrus
3	Middle frontal gyrus
5	Precentral gyrus
6	Central sulcus
7	Postcentral gyrus
13	Inferior frontal gyrus
15	Supramarginal gyrus
16	Intraparietal sulcus
17a	Cingulate gyrus, anterior
25	Cingulate sulcus
31	Body of caudate nucleus
32	Body of corpus callosum
34	Superior temporal gyrus
35	Planum temporale
36	Middle temporal gyrus
44	Putamen
47	Fornix
52	Transverse temporal gyrus (Heschl's gyrus)
53	Insula
55	Inferior temporal gyrus
56	Internal capsule
56b	Internal capsule, posterior limb
59	Globus pallidus
71d	Hippocampus proper
71e	Hippocampus, subiculum
74	Collateral sulcus
75	Fusiform gyrus
82	Amygdala
83	Mammillary body
91	Substantia nigra
92	Red nucleus
93	Stria terminalis
97	Entorhinal cortex
101	Subthalamic nucleus
102	Cerebral peduncle
a	Anterior nuclei
b	Dorsomedial nuclei
c1	Ventroanterior nuclei
g	Centromedian nuclei
II	Cranial nerve II
^	Extreme capsule
*	External capsule
o	Claustrum

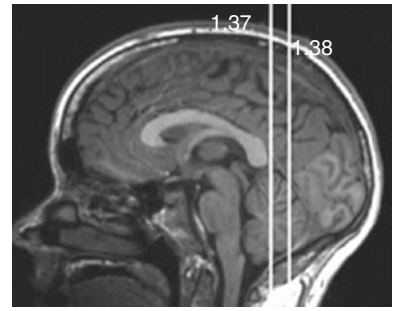


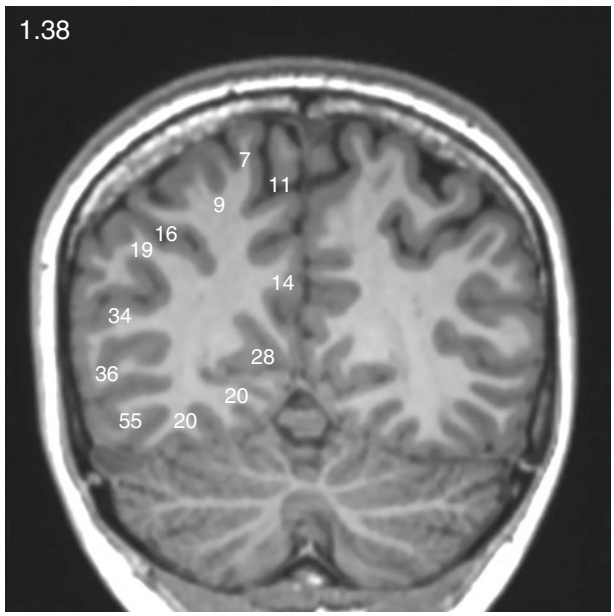




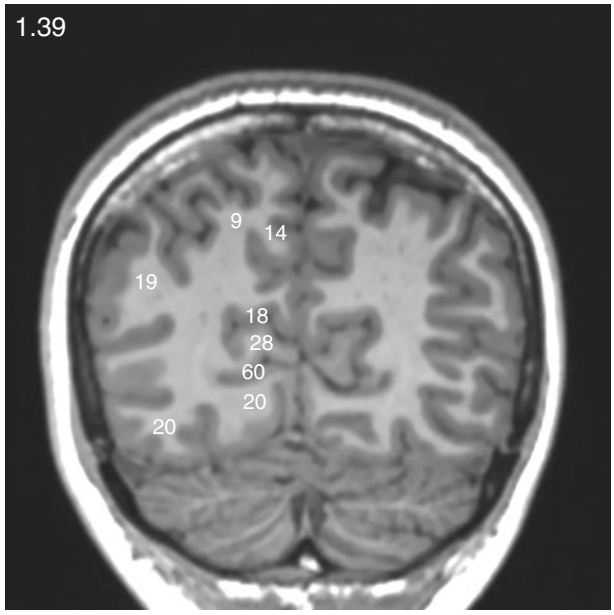
1	Superior frontal gyrus
5	Precentral gyrus
6	Central sulcus
7	Postcentral gyrus
8	Postcentral sulcus
13	Inferior frontal gyrus
15	Precuneus
16	Intraparietal sulcus
17b	Cingulate gyrus, posterior
24	Inferior frontal gyrus, pars opercularis
25	Cingulate sulcus
31	Body of caudate nucleus
34	Superior temporal Gyrus
35	Planum temporale
36	Middle temporal gyrus
41	Splenium of corpus callosum
47	Fornix
48	Forceps major
52	Transverse temporal gyrus (Heschl's gyrus)
53	Insula
55	Inferior temporal gyrus
63	Third ventricle
64	Atrium of lateral ventricle
65	Sylvian fissure
71d	Hippocampus proper
71e	Hippocampus, subiculum
74	Collateral sulcus
75	Fusiform gyrus
90	Isthmus of corpus callosum
97	Entorhinal cortex
b	Dorsomedial nuclei
c2	Ventrolateral nuclei
c3	Ventroposteriorlateral Nuclei
d	Pulvinar
e	Medial geniculate nucleus
f	Lateral geniculate nucleus

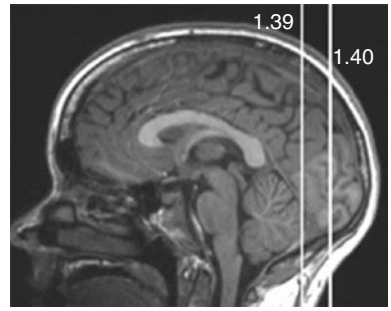




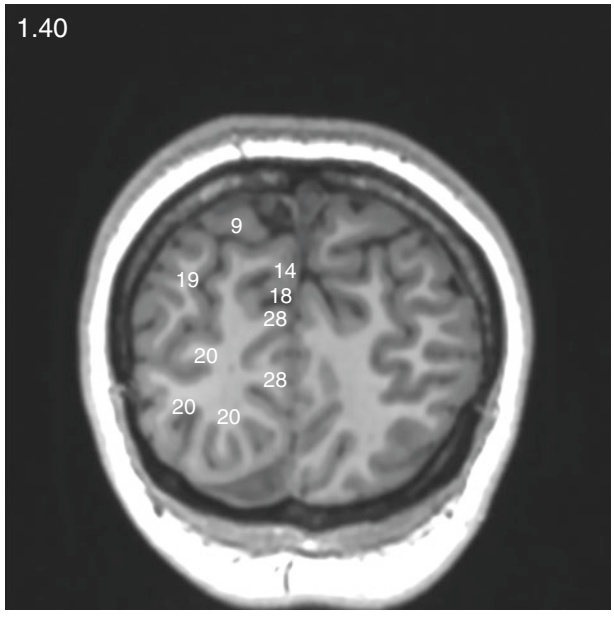


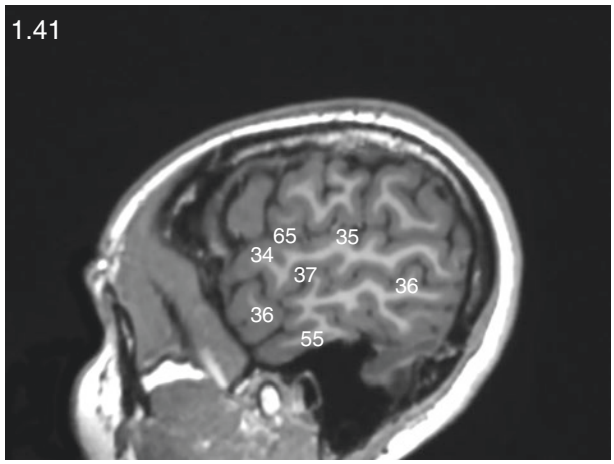
5	Precentral gyrus
6	Central sulcus
7	Postcentral gyrus
8	Postcentral sulcus
9	Superior parietal lobe
11	Marginal sulcus
14	Precuneus
15	Supramarginal gyrus
16	Intraparietal sulcus
17b	Cingulate gyrus, posterior
19	Angular gyrus
20	Occipital gyri
28	Striate gyrus
34	Superior temporal Gyrus
36	Middle temporal gyrus
52	Transverse temporal Gyrus (Heschl's gyrus)
55	Inferior temporal gyrus
60	Straight gyrus
74	Collateral sulcus
75	Fusiform gyrus
77	Lingual gyrus
79	Occipital horn, lateral ventricle

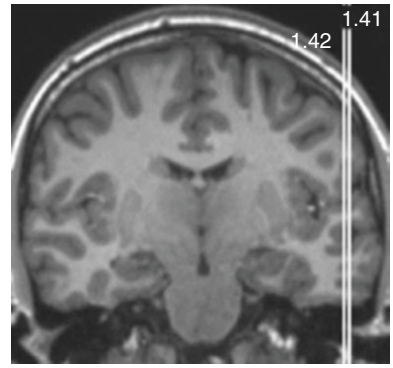




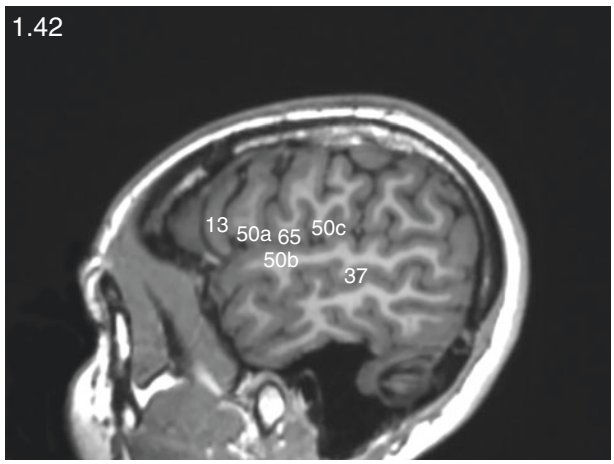
9	Superior parietal lobe
14	Precuneus
15	Supramarginal gyrus
16	Intraparietal sulcus
18	Parieto-occipital sulcus
19	Angular gyrus
20	Occipital gyri
28	Striate gyrus
60	Calcarine sulcus

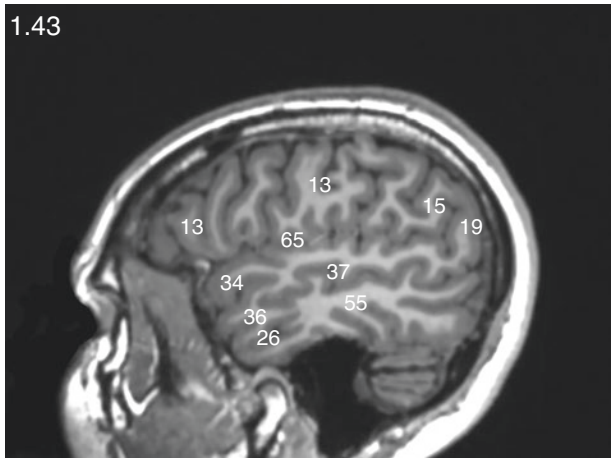


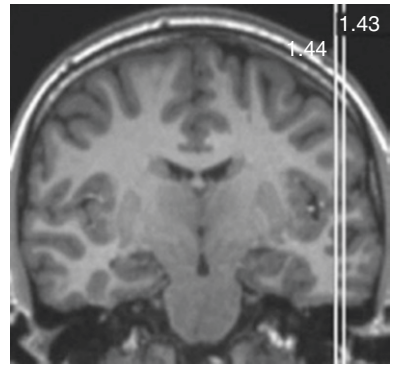




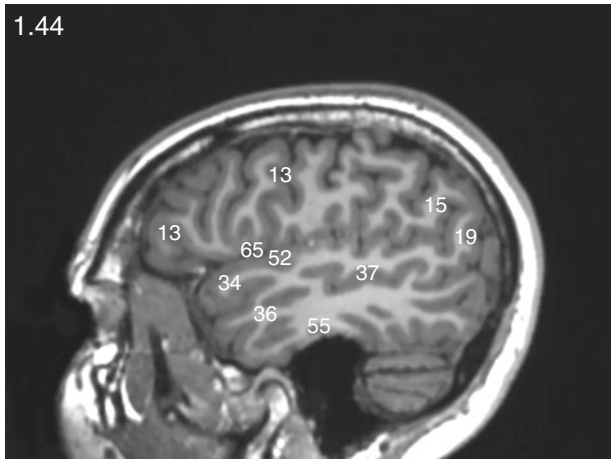
13	Inferior frontal gyrus
34	Superior temporal gyrus
35	Planum temporale
36	Middle temporal gyrus
37	Superior temporal sulcus
50a	Operculum, frontal
50b	Operculum, temporal
50c	Operculum, parietal
55	Inferior temporal gyrus
65	Sylvian fissure

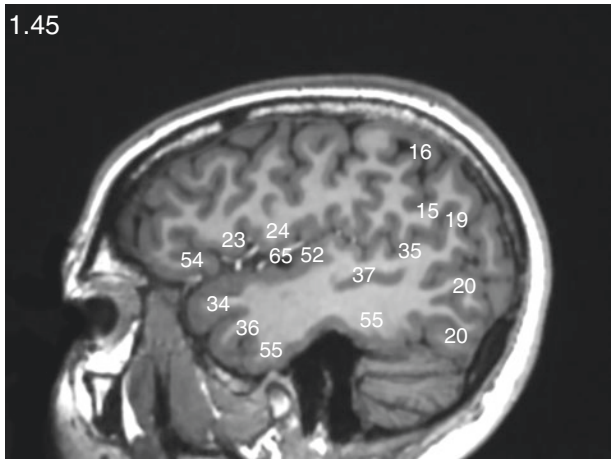


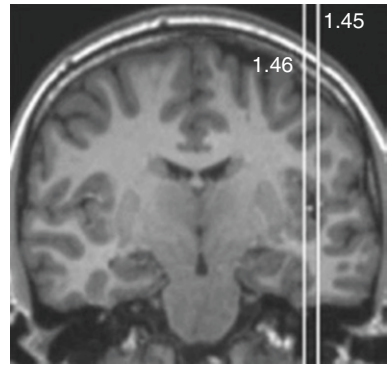


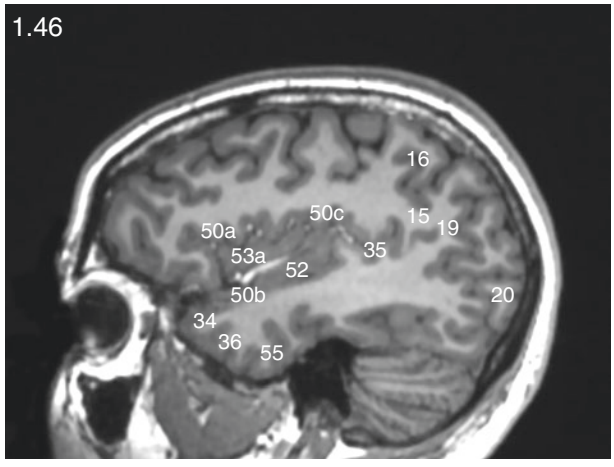


13	Inferior frontal gyrus
15	Supramarginal gyrus
19	Angular gyrus
26	Inferior temporal sulcus
34	Superior temporal gyrus
36	Middle temporal gyrus
37	Superior temporal sulcus
50b	Operculum, temporal
52	Transverse temporal gyrus (Heschl's gyrus)
55	Inferior temporal gyrus
65	Sylvian fissure

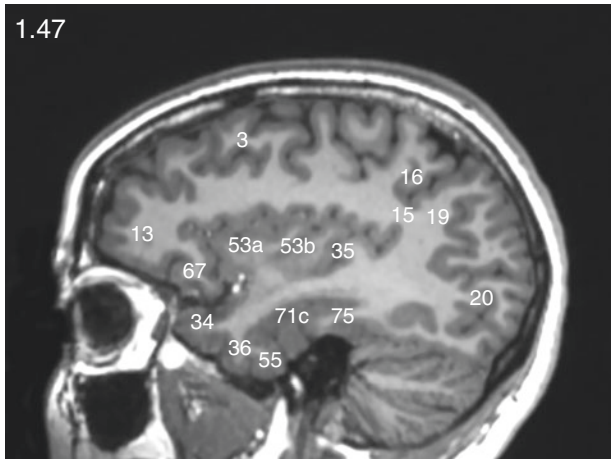


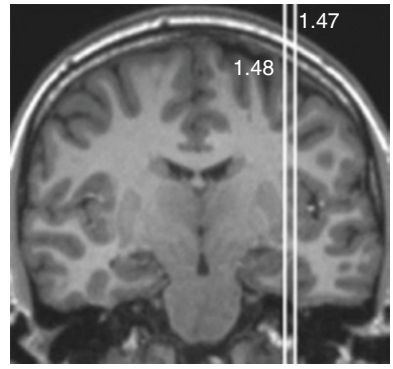




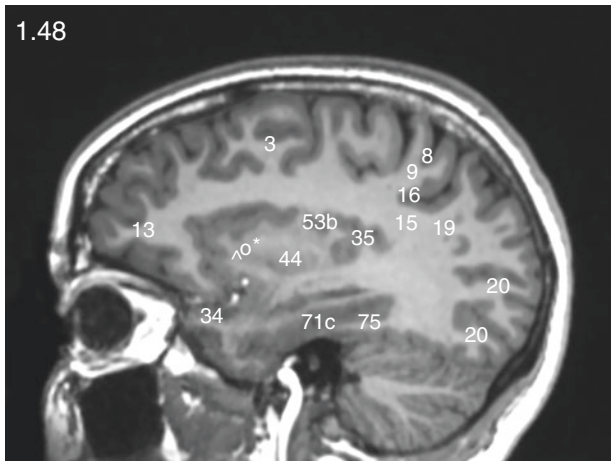


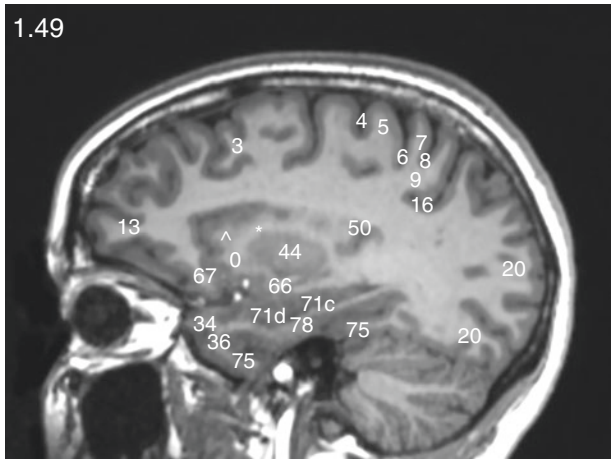
15	Supramarginal gyrus
16	Intraparietal sulcus
19	Angular gyrus
20	Occipital gyri
23	Inferior frontal gyrus, pars triangularis
24	Inferior frontal gyrus, pars opercularis
34	Superior temporal gyrus
35	Planum temporale
36	Middle temporal gyrus
37	Superior temporal sulcus
50a	Operculum, frontal
50b	Operculum, temporal
50c	Operculum, parietal
52	Transverse temporal gyrus (Heschl's gyrus)
53a	Insula, short gyri
54	Inferior frontal gyrus, pars orbitalis
55	Inferior temporal gyrus
65	Sylvian fissure

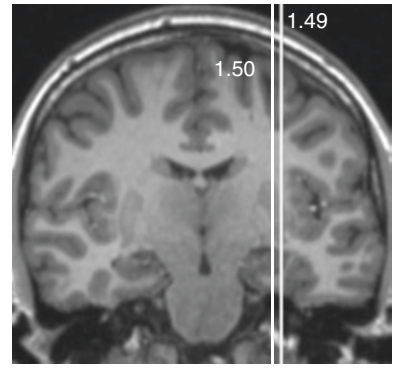


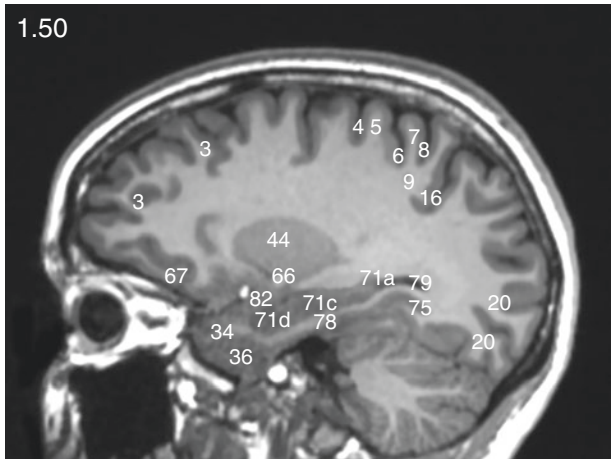


3	Middle frontal gyrus
8	Postcentral sulcus
9	Superior parietal lobe
13	Inferior frontal gyrus
15	Supramarginal gyrus
16	Intraparietal sulcus
19	Angular gyrus
20	Occipital gyri
34	Superior temporal gyrus
35	Planum temporale
36	Middle temporal gyrus
44	Putamen
52	Transverse temporal gyrus (Heschl's gyrus)
53a	Insula, short gyri
53b	Insula, long gyri
55	Inferior temporal gyrus
67	Orbital gyrus
71c	Hippocampus, dentate gyrus
75	Fusiform gyrus
^	Extreme capsule
*	External capsule
o	Claustrum

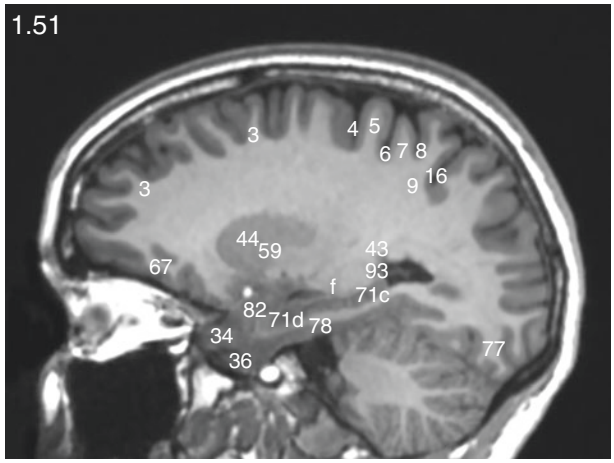


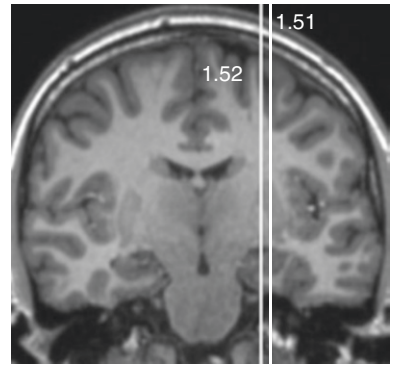


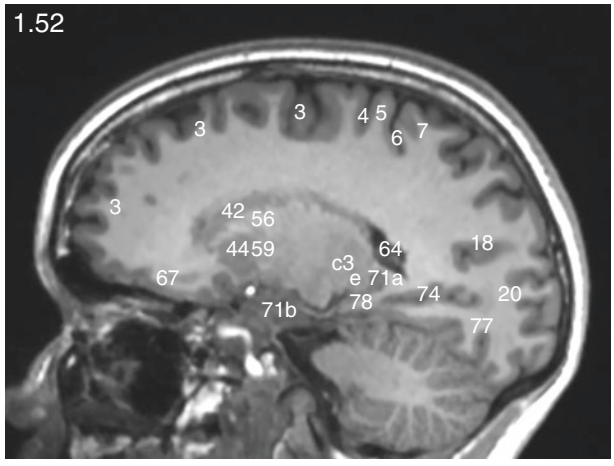




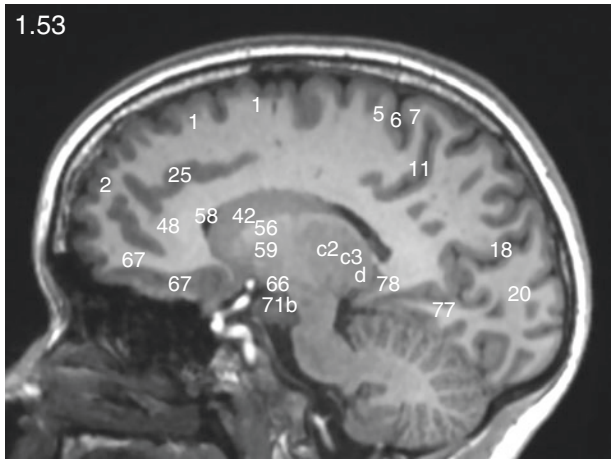
3	Middle frontal gyrus
4	Precentral sulcus
5	Precentral gyrus
6	Central sulcus
7	Postcentral gyrus
8	Postcentral sulcus
9	Superior parietal lobe
13	Inferior frontal gyrus
16	Intraparietal sulcus
19	Angular gyrus
20	Occipital gyri
34	Superior temporal gyrus
36	Middle temporal gyrus
44	Putamen
50	Operculum
51	Rostral gyrus
52	Transverse temporal gyrus (Heschl's gyrus)
53	Insula
66	Anterior commissure
67	Orbital gyrus
71a	Hippocampus, fimbria
71c	Hippocampus, dentate gyrus
71d	Hippocampus proper
75	Fusiform gyrus
78	Parahippocampal gyrus
79	Occipital horn, lateral ventricle
82	Amygdala
^	Extreme capsule
*	External capsule
o	Claustrum

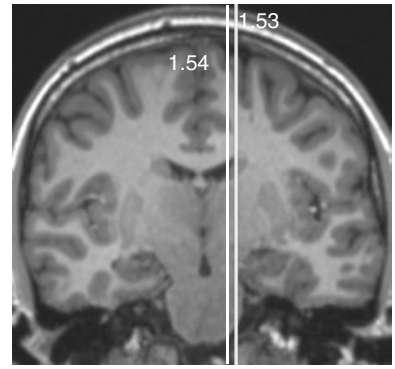


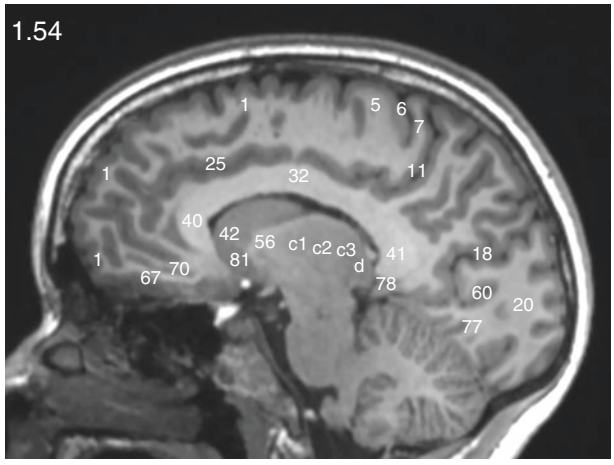




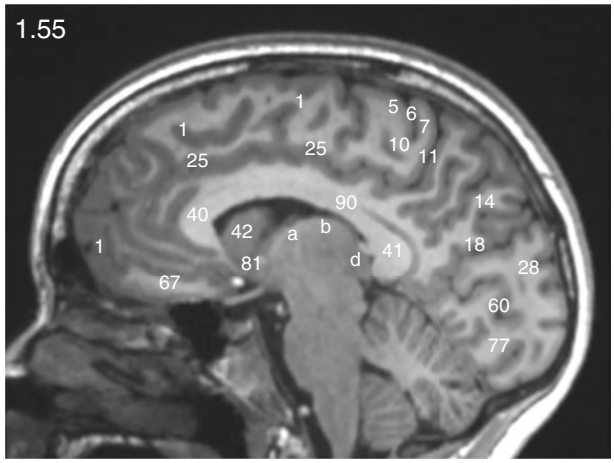
3	Middle frontal gyrus
4	Precentral sulcus
5	Precentral gyrus
6	Central sulcus
7	Postcentral gyrus
8	Postcentral sulcus
9	Superior parietal lobe
16	Intraparietal sulcus
18	Pariteo-occipital sulcus
20	Occipital gyri
34	Superior temporal gyrus
36	Middle temporal gyrus
42	Head of caudate nucleus
43	Tail of caudate nucleus
44	Putamen
56	Internal capsule
59	Globus pallidus
64	Atrium of lateral ventricle
67	Orbital gyrus
71a	Hippocampus, fimbria
71b	Hippocampus, uncus
71c	Hippocampus, dentate gyrus
71d	Hippocampus proper
74	Collateral sulcus
77	Lingual gyrus
78	Parahippocampal gyrus
79	Occipital horn, lateral ventricle
82	Amygdala
93	Stria terminalis
c3	Ventroposteriorlateral nuclei
e	Medial geniculate nucleus
f	Lateral geniculate nucleus

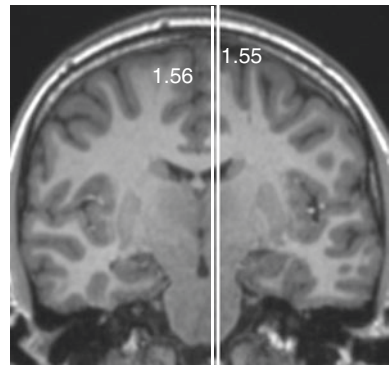


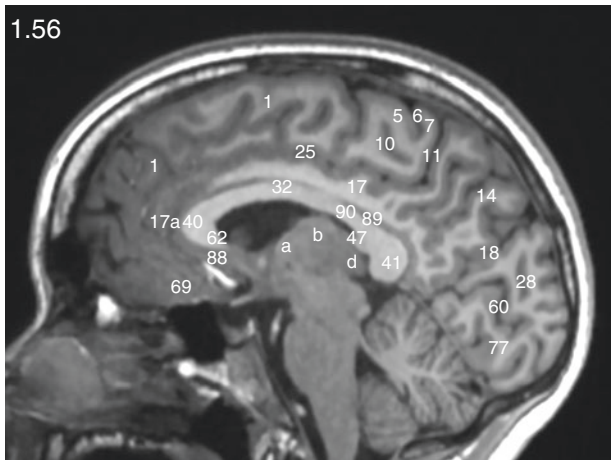




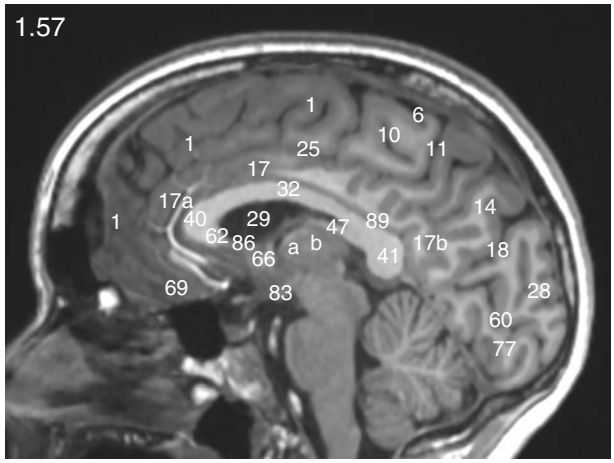
1	Superior frontal gyrus
2	Superior frontal sulcus
5	Precentral gyrus
6	Central sulcus
7	Postcentral gyrus
10	Paracentral lobule
11	Marginal sulcus
14	Precuneus
18	Pariteo-occipital sulcus
20	Occipital gyri
25	Cingulate sulcus
32	Body of corpus callosum
40	Genu of corpus callosum
41	Splenium of corpus callosum
42	Head of caudate nucleus
48	Forceps major
56	Internal capsule
58	Frontal horn of lateral ventricle
59	Globus pallidus
60	Calcarine sulcus
66	Anterior commissure
67	Orbital gyrus
70	Olfactory sulcus
71b	Hippocampus, uncus
77	Lingual gyrus
78	Parahippocampal gyrus
81	Nucleus accumbens
89	Callosal sulcus
c1	Ventral anterior nuclei
c2	Ventrolateral nuclei
c3	Ventroposterolateral nuclei
d	pulvinar

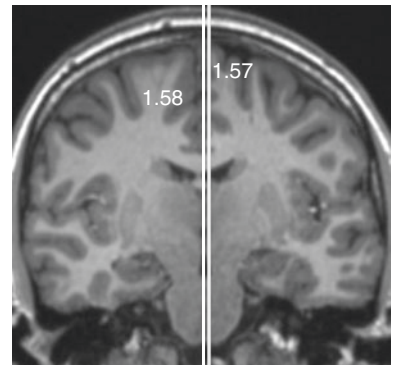


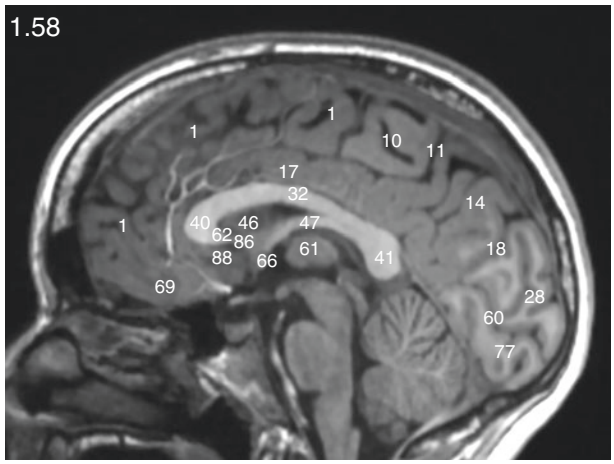




1	Superior frontal gyrus
5	Precentral gyrus
6	Central sulcus
7	Postcentral gyrus
10	Paracentral lobule
11	Marginal sulcus
14	Precuneus
17	Cingulate gyrus
17a	Cingulate gyrus, anterior
18	Parieto-occipital sulcus
25	Cingulate sulcus
28	Striate gyrus
32	Body of corpus callosum
40	Genu of corpus callosum
41	Splenium of corpus callosum
42	Head of caudate nucleus
47	Fornix
60	Calcarine sulcus
62	Rostrum of corpus callosum
67	Orbital gyrus
69	Straight gyrus
77	Lingual gyrus
81	Nucleus accumbens
88	Subcallosal gyrus
89	Callosal sulcus
90	Isthmus of corpus callosum
a	Anterior nuclei
b	Dorsomedial nuclei
d	Pulvinar







1	Superior frontal gyrus
5	Precentral gyrus
6	Central sulcus
7	Postcentral gyrus
8	Postcentral sulcus
10	Paracentral lobule
11	Marginal sulcus
14	Precuneus
17	Cingulate gyrus
17a	Cingulate gyrus, anterior
17b	Cingulate gyrus, posterior
18	Parieto-occipital sulcus
25	Cingulate sulcus
28	Striate gyrus
29	Body of lateral ventricle
32	Body of corpus callosum
40	Genu of corpus callosum
41	Splenium of corpus callosum
46	Septum pellucidum
47	Fornix
60	Calcarine sulcus
61	Intrathalamic adhesion
62	Rostrum of corpus callosum
66	Anterior commissure
69	Straight gyrus
77	Lingual gyrus
83	Hypothalamus
86	Lamina terminalis
88	Subcallosal gyrus
89	Callosal sulcus
a	Anterior nuclei
b	Dorsomedial nuclei

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White Matter Anatomy

2

Andrea Poretti

Diffusion tensor imaging (DTI) is an advanced MRI technique that allows the qualitative or quantitative study of the white matter microscopic structural organization in vivo. Within the brain, diffusion differs between various structures and depends mostly on the microstructural architecture [1, 2]. DTI analyzes the three-dimensional shape and magnitude of diffusion or diffusion tensor. The diffusion tensor is a three-dimensional structure with three principal diffusivities (eigenvalues, λ1, λ2, and λ3) associated with three mutually perpendicular principal directions (eigenvectors). Diffusion that is predominantly along the white matter tracts is called anisotropic, while isotropic diffusion represents diffusion that is equal in all directions. The degree of anisotropy may be quantified by calculating fractional anisotropy (FA). FA is defined as the ratio of the anisotropic component of the diffusion tensor to the whole diffusion tensor and varies between zero and one. Zero represents maximal isotropic diffusion (like in the ventricular system), while one represents maximal anisotropic diffusion (like in highly packed white matter tracts such as the corpus callous of corticospinal tracts). By international convention, the FA maps usually are color coded with red indicating a predominant left-right, green an anterior-posterior, and blue a superior-inferior anisotropic diffusion. This color coding facilitates recognition of major normal and aberrant white matter tracts.

DTI and fiber tractography allows us to study the normal and abnormal connectivity and development of neuronal circuits and centers within the human brain. This information helps us to better understand the normally developing brain, revealing the microstructural anatomy of complex brain malformations, and finally gives insight into the mechanism of compensatory neuronal plasticity after brain injury [3, 4]. Finally, DTI and fiber tractography may also guide neurosurgical interventions by identifying functionally important fiber tracts that may be displaced by adjacent brain tumors.

The images presented within this chapter are from a single healthy 25-year-old male subject. Images were obtained from a Siemens Skyra 3T MRI scanner using a 32-channel head coil. The DTI protocol used to obtain these images is DTI protocol that includes a single-shot spin echo, echo planar axial DTI sequence with diffusion gradients along 20 noncollinear directions (effective high *b*-value of 1000 s/mm^2), and an additional measurement without diffusion weighting (b = 0 s/mm^2). For the DTI acquisition, we typically use the following parameters: repetition time

Andrea Poretti was deceased at the time of publication.

A. Poretti, M.D.
Section of Pediatric Neuroradiology, Division of Pediatric Radiology, Russell H. Morgan Department of Radiology and Radiological Science, Johns Hopkins University School of Medicine, Baltimore, MD 21287-0842, USA
e-mail: nivedita.agarwal@apss.tn.it

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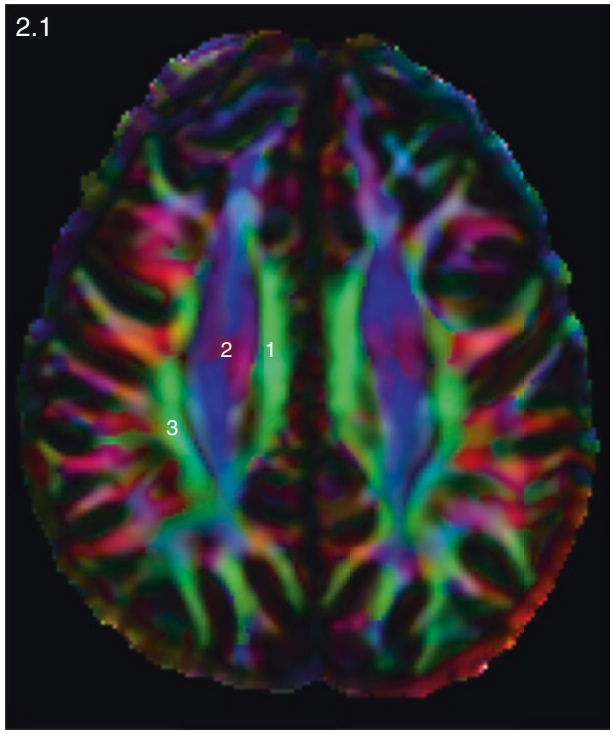
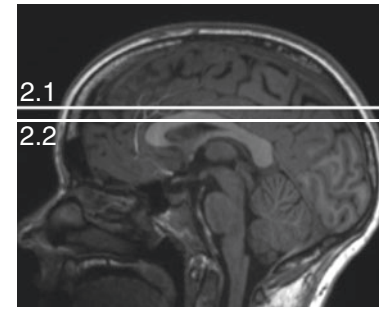
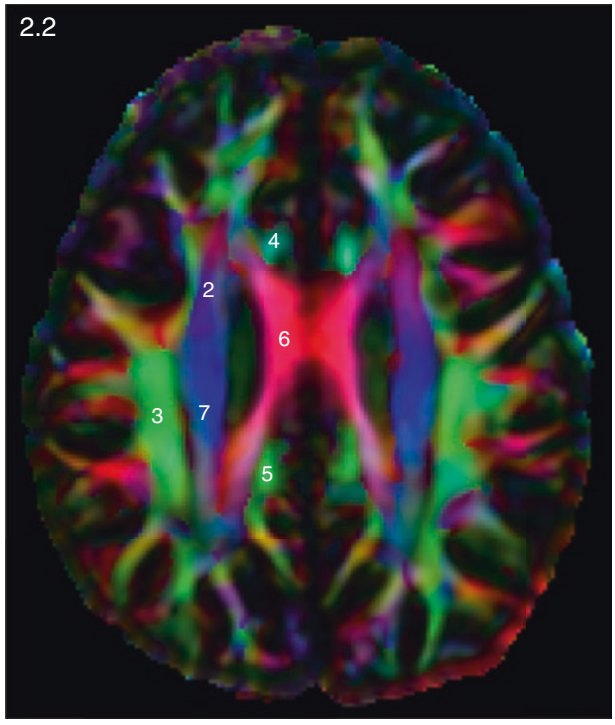
(TR) = 7100 ms, echo time (TE) = 92 ms, slice thickness = 2.5 mm, field of view (FOV) = 240 × 240 mm, and matrix size = 192 × 192. Images were acquired at a voxel resolution of dimensions are 1.25 mm × 1.25 mm in-plane × 2.5 mm thick. Selected images were chosen, resampled at a resolution of 1.0 mm × 1.0 mm × 1.0 mm for labeling that best represented the local anatomy in a given region. Note that we chose to reconstruct images in the planes most commonly viewed by radiologists; as such, our labels are more relevant to routine clinical MRI than the labels presented in traditional histological atlases.

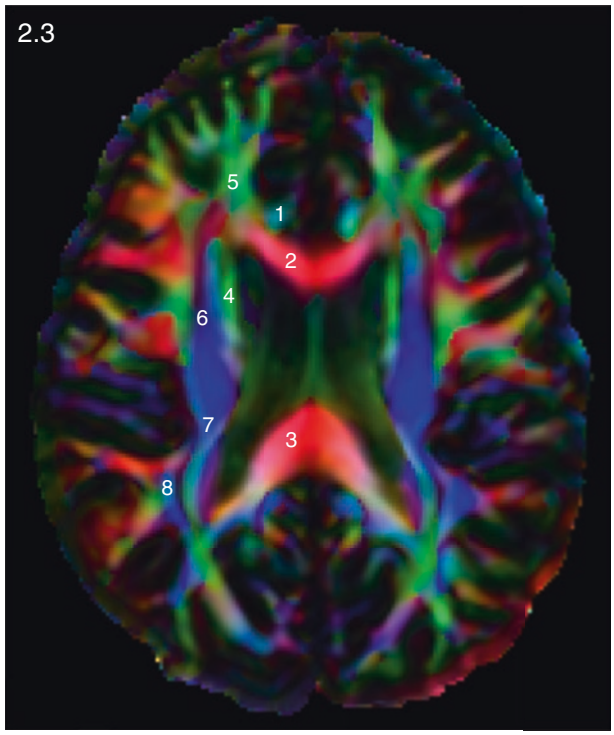
Each page contains the labeled images on the left-hand side. A small image on the top right of the page documents the locations of the slices, and a key in the lower right-hand side of the page lists the individual structures.

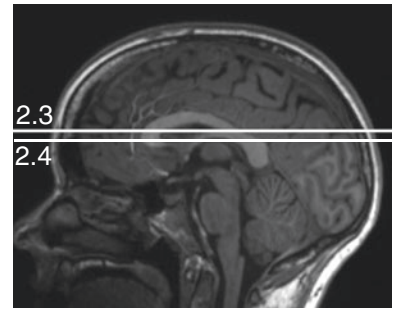




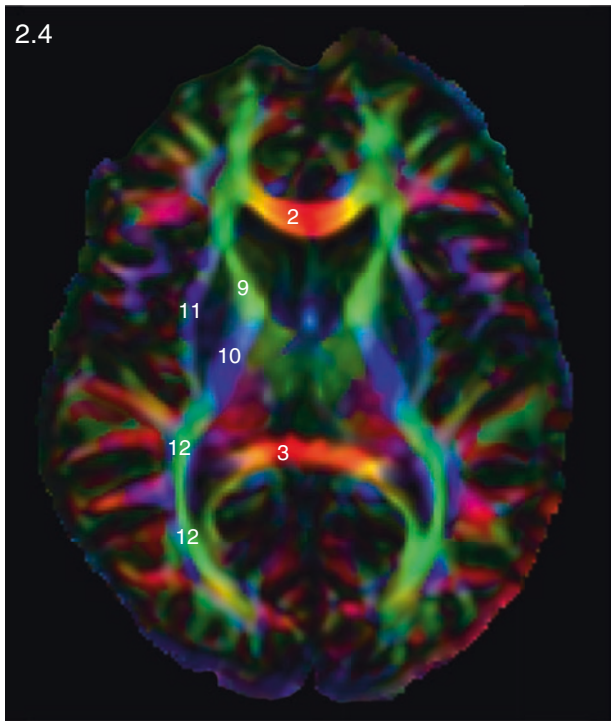
1	Cingulum, middle
2	Superior corona radiata
3	Superior longitudinal fasciculus
4	Cingulum, anterior
5	Cingulum, posterior
6	Corpus callosum, body
7	Posterior corona radiata

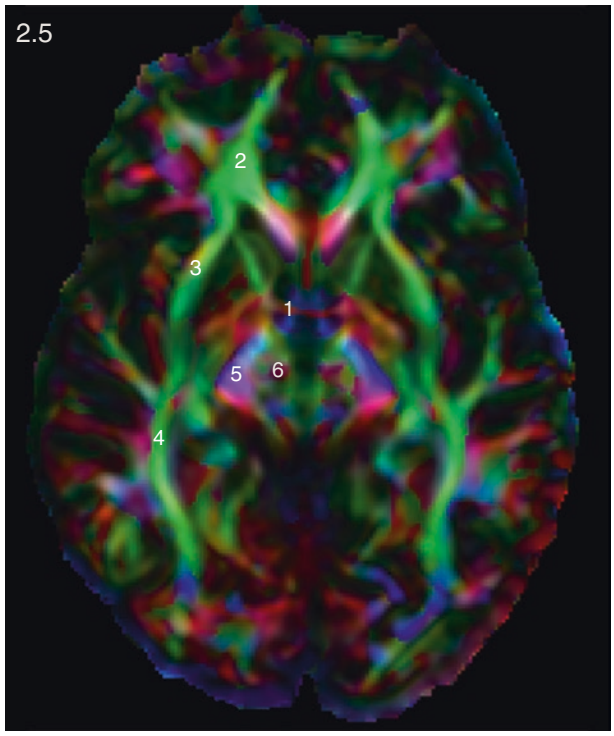


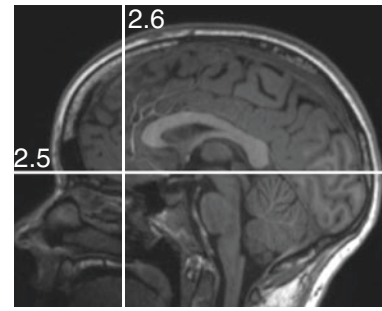




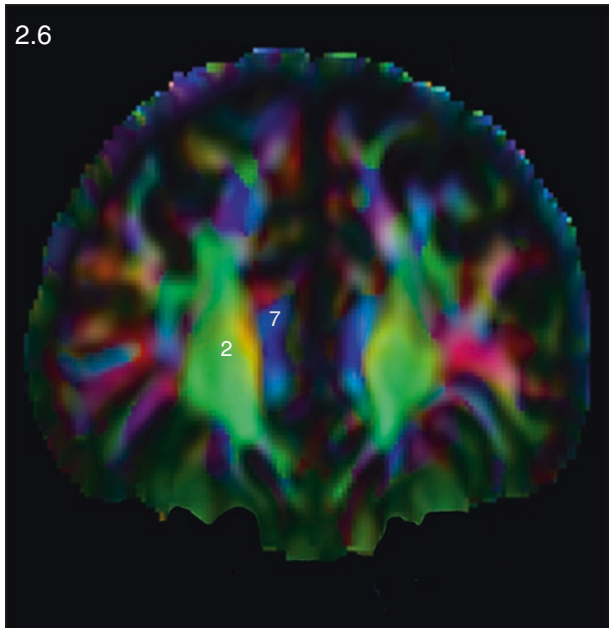
1	Cingulum, anterior
2	Corpus callosum, genu
3	Corpus callosum, splenium
4	Superior fronto-occipital fasciculus
5	Anterior corona radiata
6	Superior corona radiata
7	Posterior corona radiata
8	Superior longitudinal fasciculus
9	Anterior limb internal capsule
10	Posterior limb internal capsule
11	External capsule
12	Posterior thalamic radiation

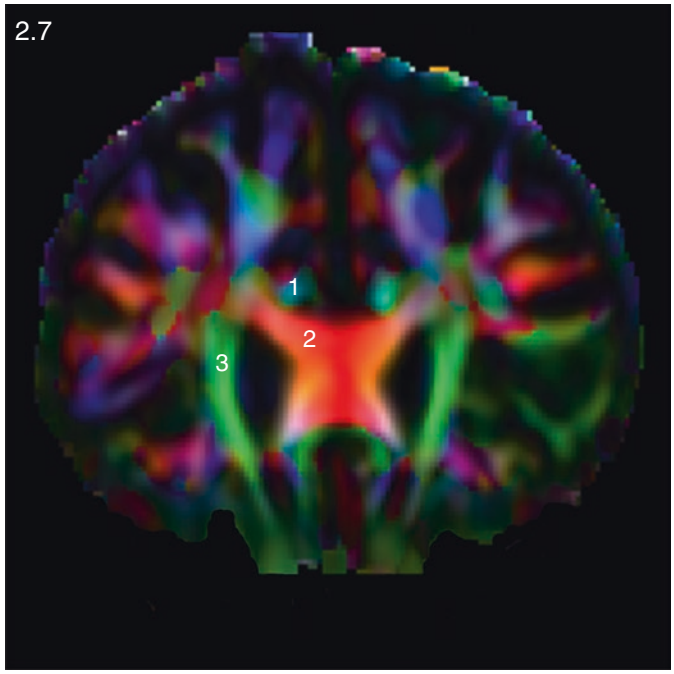


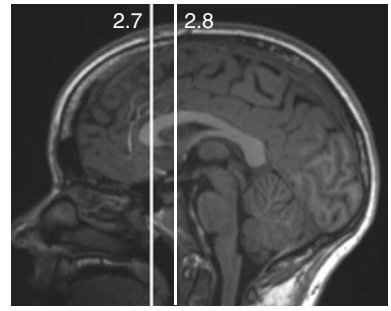




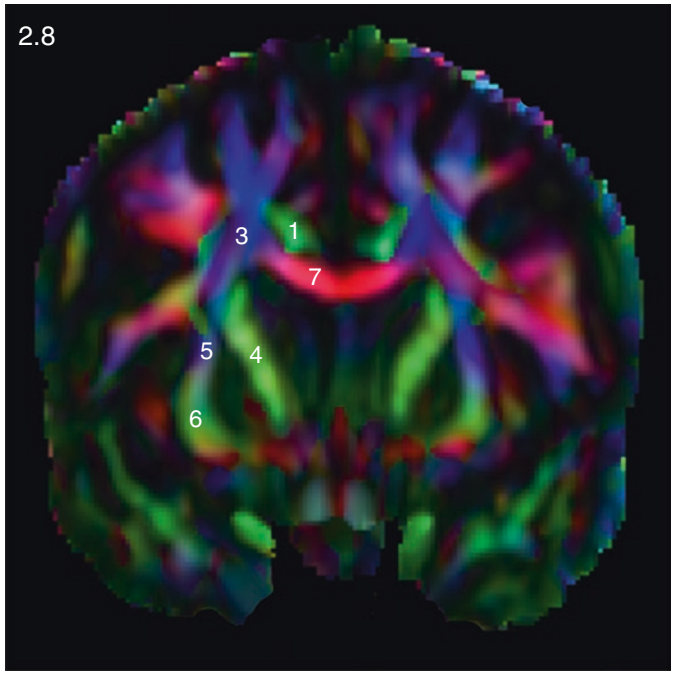
1	Anterior commissure
2	Anterior corona radiata
3	Inferior fronto-occipital fasciculus
4	Sagittal striatum (includes inferior longitudinal fasciculus and inferior fronto-occipital fasciculus)
5	Cerebral peduncle
6	Red nucleus
7	Cingulum

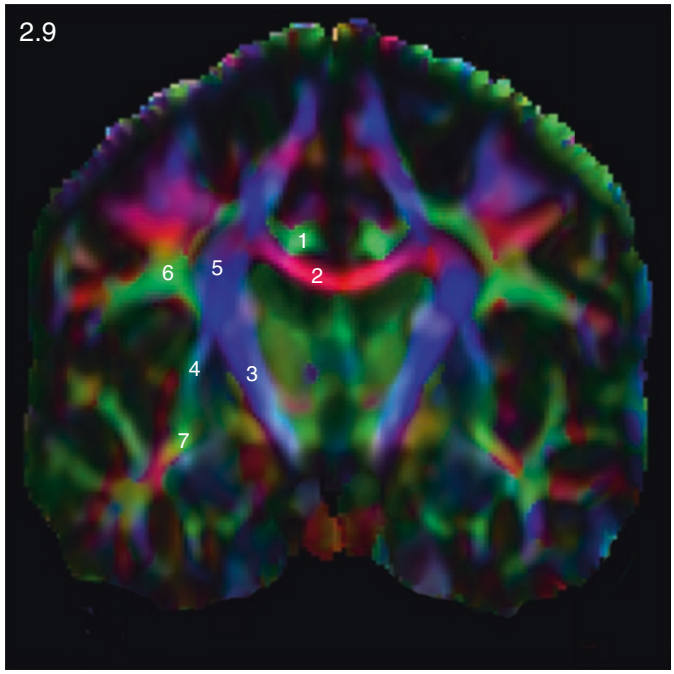


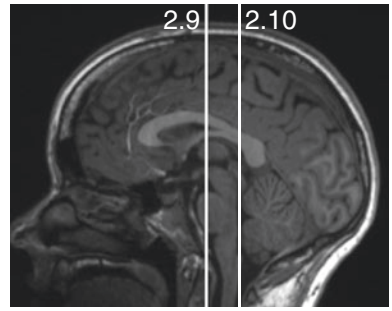




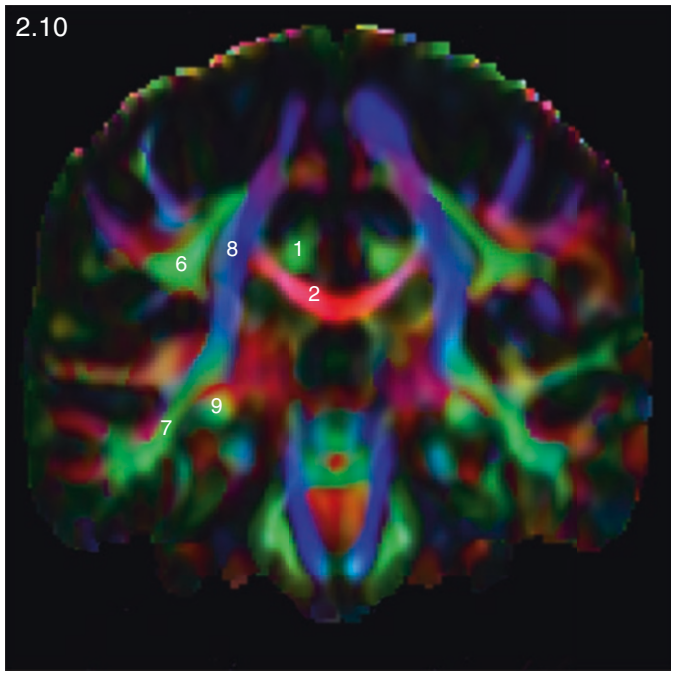
1	Cingulum
2	Corpus callosum, genu
3	Anterior corona radiata
4	Anterior limb internal capsule
5	External capsule
6	Inferior fronto-occipital fasciculus
7	Corpus callosum, body

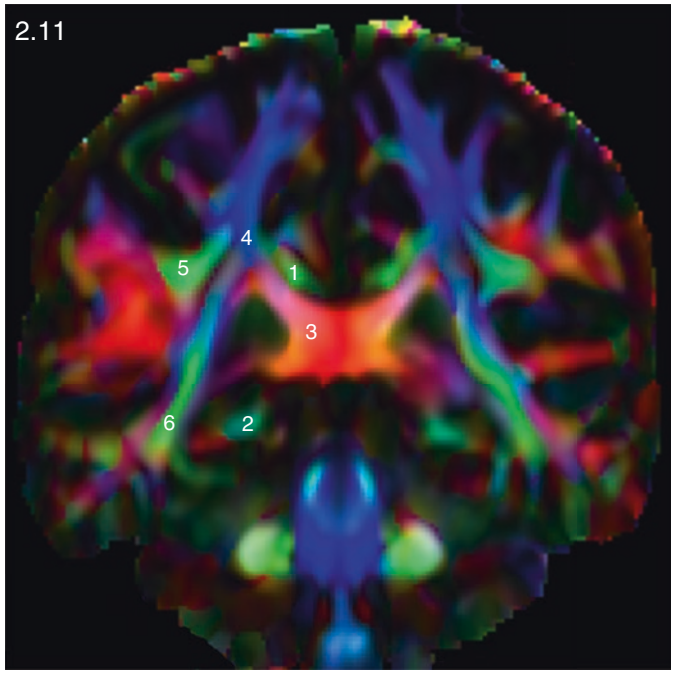


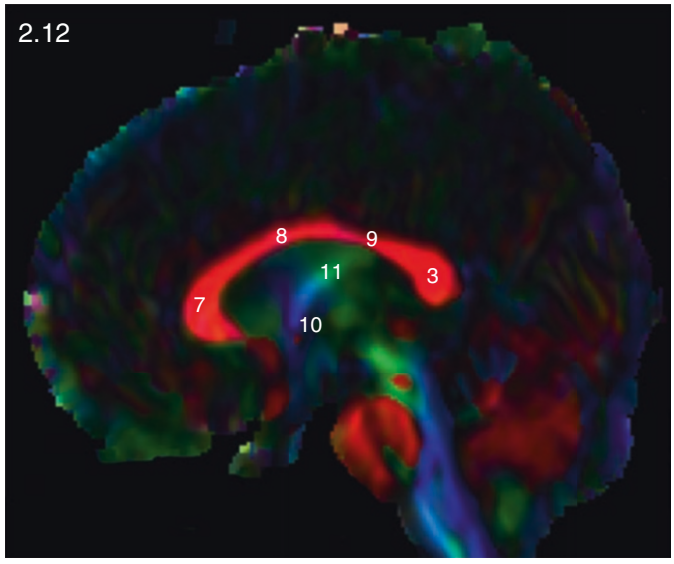


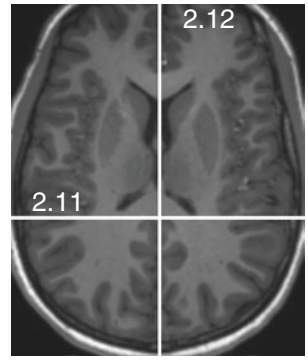


1	Cingulum
2	Corpus callosum, body
3	Posterior limb internal Capsule
4	External capsule
5	Superior corona radiata
6	Superior longitudinal fasciculus
7	Sagittal striatum (includes inferior longitudinal fasciculus and inferior fronto-occipital fasciculus)
8	Posterior corona radiata
9	Fornix

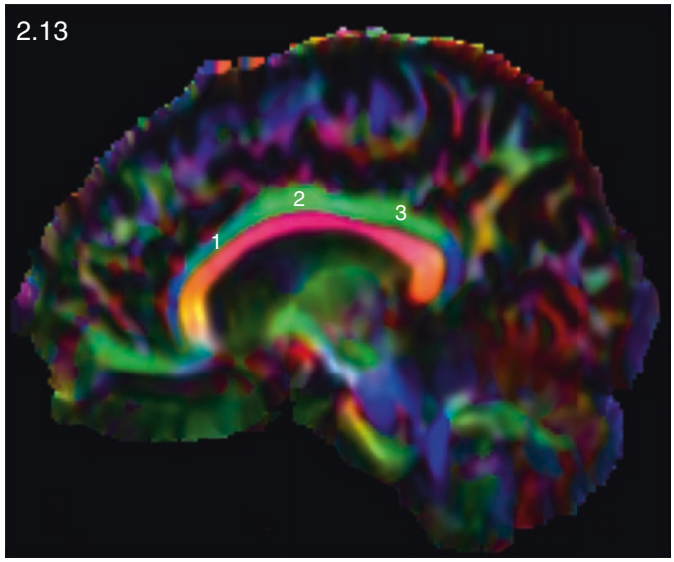


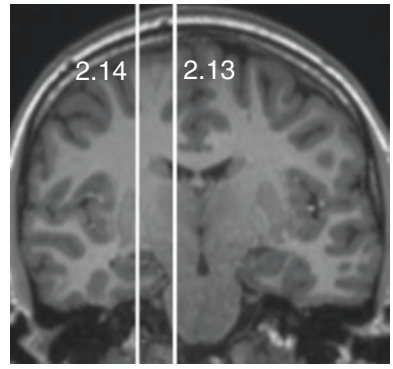




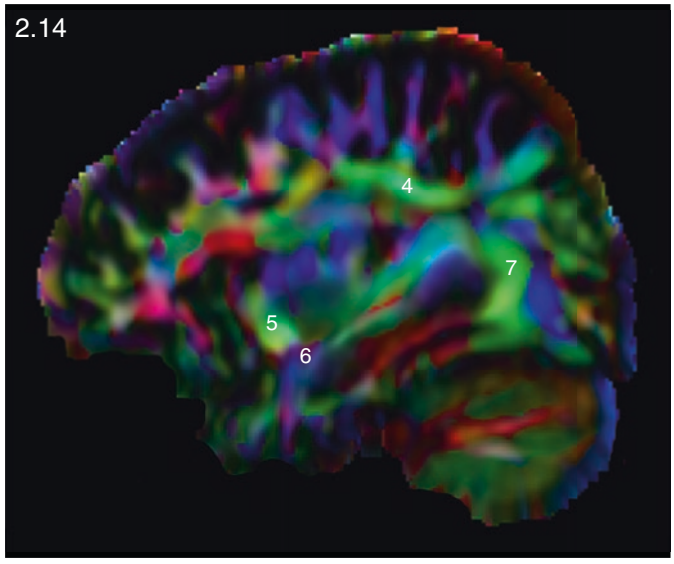


1	Cingulum, superior
2	Cingulum, inferior
3	Corpus callosum, splenium
4	Posterior corona radiata
5	Superior longitudinal fasciculus
6	Sagittal striatum (includes inferior longitudinal fasciculus and inferior fronto-occipital fasciculus)
7	Corpus callosum, genu
8	Corpus callosum, body
9	Corpus callosum, isthmus
10	Anterior commissure
11	Fornix





1	Cingulum, anterior
2	Cingulum, middle
3	Cingulum, posterior
4	Superior longitudinal fasciculus
5	Inferior fronto-occipital fasciculus
6	Uncinate fasciculus
7	Posterior thalamic radiation including optic radiation



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Supratentorial Vascular Anatomy

3

Salvatore Mangiafico, Andrea Rosi, and Arturo Consoli

Digital subtraction angiography (DSA) is a technique that allows imaging of cerebral vasculature and represents the gold standard for high-resolution vascular imaging. It allows visualization of small diameter vascular structures and the evaluation of vascular dynamics by imaging the arterial, capillary, and venous phases. A major advantage is the possibility of therapeutic intervention during the diagnostic study.

In the following chapter, we first describe the major cerebral arteries and their branches, briefly discussing their course and distribution territorial patterns. We then describe the major supratentorial venous drainage. We have included the posterior cerebral arteries in this chapter even though they arise from the infratentorial circulation (see Chap. 9) since they supply the parietal and occipital lobes [1–6].

S. Mangiafico, M.D. (✉)
Interventional Neurovascular Unit, Careggi University Hospital, Florence, Italy
e-mail: mangiax@libero.it

A. Rosi, M.D.
Residency Program in Radiology, University of Florence, Careggi University Hospital, Florence, Italy
e-mail: andrea.rosi87@gmail.com

A. Consoli, M.D.
Diagnostic and Interventional Neuroradiology Service, Hopital Foch, Suresnes, France

Interventional Neurovascular Unit, Careggi University Hospital, Florence, Italy
e-mail: onemed21@gmail.com

The goal of this chapter is to provide readers with an understanding of major arteries and veins and their distribution on typical DSA images. Major symptoms due to specific vessel occlusions are also briefly mentioned. Descriptions of vascular variants are beyond the scope of the book.

Images were acquired by selective injection of the arteries to illustrate the most significant arteries and veins in the human brain. The images are presented in the posterior-anterior, lateral and occipito-frontal, or oblique projections.

3.1 Major Supratentorial Arteries

3.1.1 Internal Carotid Artery

The internal carotid artery (ICA) can be further subdivided into eight segments from the jugular foramen to the bifurcation of the ICA (Figs. 3.1 and 3.2).

1. Cervical segment (C1)
2. Intrapetrous and intracavernous segments (C2-C3-C4)
   (a) Intrapetrous segment (C2)
      - Vertical segment
      - Horizontal segment
        Tympanic artery
        Mandibular-vidian or vidian artery
   (b) Lacerus segment (C3)

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(c) Intracavernous segment (C4)
- Meningohypophyseal trunk: arises from the posterior curve of the siphon and provides vascular supply to the dura mater of the posterior cranial fossa (tentorium and the clivus) and the adenohypophysis.
  - Bernasconi-Cassinari artery or marginal artery of the tentorium
  - Arteries for the clivus (these might occasionally originate from the Bernasconi-Cassinari artery)
  - Inferior hypophyseal artery
- Common inferolateral trunk: supplies the pavement of the middle cranial fossa and the cranial nerves of the cavernous sinus. It arises from the horizontal segment of the intracavernous segment of the carotid anteriorly with respect to meningohypophyseal trunk.
  - Meningeal artery
  - Artery of the Gasser ganglion (not visible on regular DSA)

3. Paraclinoid, supraclinoid, and the carotid apex (C5-C6-C7-C8)
   (a) Clinoid segment (C5): gives rise to the superior hypophyseal artery.
   (b) Supraclinoid segment (C6-C8).
   - Ophthalmic segment (C6): starts from the origin of the ophthalmic artery, which distributes to the eye and the orbital tissues and widely anastomoses with branches of the external carotid artery such as the lacrimal artery, supraorbital arteries, and the ethmoidal arteries.
   - Communicating segment (C7): starts from posterior communicating artery (PComA) origin and ends with the emergence of the anterior choroidal artery.
   - Choroidal segment (C8): between the emergence of the anterior choroidal artery (AchoA) and the internal carotid bifurcation in MCA and ACA.

   (c) ICA terminates into two branches: a more robust middle cerebral artery (M1 segment) and a smaller anterior cerebral artery (A1 segment). From the ICA bifurcation, a variable number of perforators may arise that pierce the anterior perforated substance to reach the genu of the internal capsule.

3.1.1.1 Anterior Choroidal Artery (AChoA)

The AChoA arises from the posterior wall of C8 distal to the origin of the PComA. It divides into (Fig. 3.3):
- Lateral branch (temporal horn and atrium of the lateral ventricle)
- Medial branch (thalamo-pulvinar branch), to end in a number of thalamo-pulvinar arteries that supply the thalamus and anastomose with the thalamo-pulvinaric arteries of the PCA.

Thus during its course, the AChoA gives rise to:
- Anterior group of arteries from the cisternal segment: these provide branches to the optic tract, the temporal uncus, amygdala, cerebral peduncle, anterior group of perforators of the AChoA that reach the medial portion of the globus pallidus through the anterior perforated substance.
- Posterior group of arteries (plexal or scissural segment): cortical arteries to the lateral geniculate body, hippocampus, dentate, gyrus and the fornix. A posterior group of perforators reach the 2/3 posterior of the internal capsule (retrolenticular region, posterior arm) and the optic radiations.

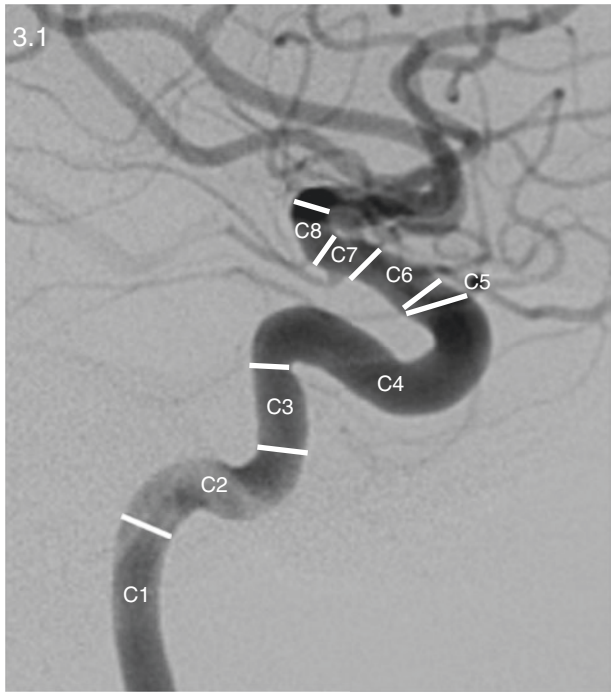


Fig. 3.1 Segments of the internal carotid artery, lateral projection

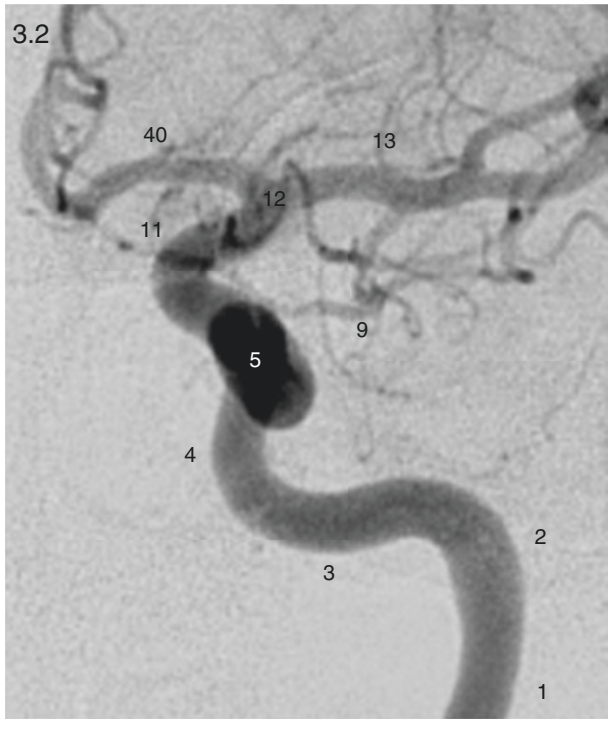


Fig. 3.2 Internal carotid artery, anteroposterior projection

1	Cervical segment (C1) of the internal carotid artery (ICA)
2	Vertical portion of petrous segment (C2) of the internal carotid artery (ICA)
3	Horizontal portion of petrous segment (C2) of the internal carotid artery (ICA)
4	Lacerum segment (C3) of the internal carotid artery (ICA)
5	Posterior bend of cavernous segment (C4) of the internal carotid artery (ICA)
9	Ophthalmic artery
11	Anterior choroidal artery
12	Internal carotid artery bifurcation
13	Alar or sphenoidal segment (M1) of middle cerebral artery (MCA)
40	Horizontal or sovraoptic segment (A1) of the anterior cerebral artery (ACA)

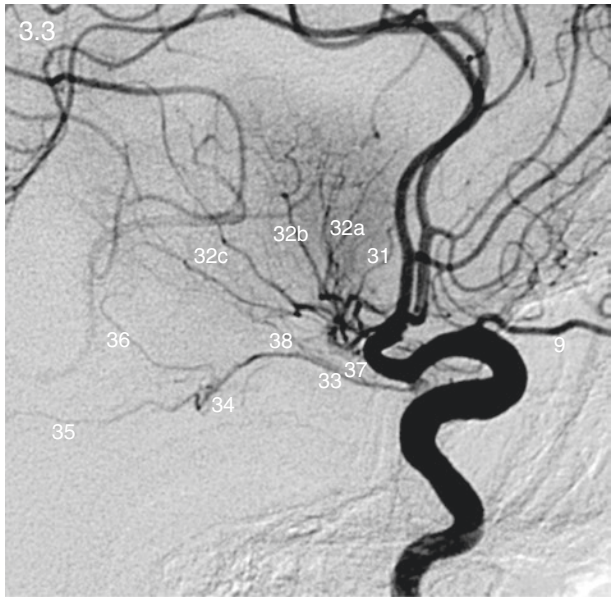


Fig. 3.3 Anterior choroidal artery and lenticulostriate arteries. These are best seen here without overlap from branches of the middle cerebral artery in a patient with complete occlusion of the MCA bifurcation. Lateral projection

9	Ophthalmic artery
31	Medial lenticulostriate arteries and carotid bifurcation's perforating arteries
32a	Lateral lenticulostriate arteries, medial group
32b	Lateral lenticulostriate arteries, intermediate group
32c	Lateral lenticulostriate arteries, lateral group
33	Cisternal segment of the anterior choroidal artery
34	Inferior choroidal point of the anterior choroidal artery
35	Choroidal trunk of plexal segment of the anterior choroidal artery
36	Thalamo-pulvinaric trunk of plexal segment of the anterior choroidal artery
37	Perforating arteries of the anterior choroidal artery, anterior group
38	Perforating arteries of the anterior choroidal artery, posterior group

3.1.1.2 Internal Carotid Artery Pathology

Occlusion syndromes of ICA

- Contralateral hemiplegia and hemianesthesia
- Deviation of the gaze toward of the lesion.
- Homonymous hemianopsia (superior 1/2 of the optic radiation)
- Carotid dissections may cause Bernard-Horner syndrome. The syndrome is characterized by damage to the carotid sympathetic plexus causing a prevalence of parasympathetic innervation resulting in ptosis, miosis, enophthalmos, and rarely facial anhydrosis of the same side of the dissection.
- Dominant hemispheric lesion: Broca's aphasia if the superior division of the MCA is thrombosed; Wernicke's aphasia if the inferior division of the MCA is occluded.
- Nondominant hemispheric lesion: contralateral hemineglect, hemiinattention, tactile extinction, visual extinction, anosognosia, and apraxia.

Specific artery occlusions:

- Lenticulostriate arteries → pure motor hemiparesis.
- Artery of the angular gyrus → agnosia, acalculia, agraphia, disorientation.
- Ophthalmic artery → amaurosis.
- In case of absent collateral compensation from the anterior communicating artery, symptoms arising from anterior cerebral artery occlusions may concur.
- Occlusion of the carotid siphon, with valid MCA collateral flow, neurologic deficit due to occlusion of the anterior choroidal artery may be present together with amaurosis.
- Anterior choroidal artery → hemiparesis (posterior limb of the internal capsule), hemianesthesia (posterolateral nuclei of the thalamus), and hemianopsia (lateral geniculate bodies). In case of bilateral occlusions (rare), get pseudobulbar mutism, diplegia, lethargy, and neglect.

3.1.2 Anterior Cerebral Artery

3.1.2.1 Cortical Territory

The ACA provides the medial surface of the frontal and parietal lobes including the precuneus posteriorly. Cortical branches reach the outer surface of the brain to reach cortical branches of the MCA (pial anastomosis). Five segments can be identified on DSA imaging (Figs. 3.4 and 3.5).

I. A1 segment (supraoptic): It gives rise to deep perforating arteries (see below).
II. A2 segment (infracallosal).
   (a) Fronto-orbital artery: medial region of the orbital surface of the frontal lobe
   (b) Frontopolar artery: for the frontal pole
III. A3 segment (precallosal): At the level of the genu of the corpus callosum ACA. It presents the pericallosal/callosomarginal bifurcation.
   (a) Pericallosal artery.
      i. Artery for the paracentral lobule
      ii. Anterior internal parietal artery
      iii. Anterior posterior internal parietal artery
   (b) Callosomarginal artery: It arises from the A2 to A3 segment at the level of the genu of the corpus callosum. It supplies the frontal cortical territory in front of the paracentral lobule on the medial surface of the frontal lobe. Here it gives rise to three internal frontal arteries (anterior, middle, and posterior).
IV. A4 and A5 segments (supracallosal):
   (a) Superior internal parietal artery
   (b) Inferior internal parietal artery

The ACA might end with an anastomosis with the PCA through the splenial artery.

3.1.2.2 Deep Territory of A1

From the A1 segment of ACA originate, a number of perforating arteries that supply the diencephalon and the subcallosal region by penetrating the anterior perforated substance, reaching the globus pallidus and the head of the caudate nucleus. The main branches are:

- Midline perforators or diencephalic perforators: provide vascular supply to the septum pellucidum, anterior hypothalamus, fornix, lamina terminalis, anterior commissure, rostrum, and genu of the corpus callosum.
- Perforating arteries of the anterior perforated substance or the medial lenticulostriate arteries: provides vascular supply to the anteroinferior region of the lenticular nucleus, the

anteroinferior portion of the caudate nucleus, and the anterior limb of the internal capsule.
- Recurrent artery of Heubner: is a perforating artery that originates at the junction of A1–A2 segments of the anterior cerebral artery and provides arterial supply to the head of the caudate nucleus, the inferior portion of the internal capsule (jointly with the medial lenticulostriate arteries), and the hypothalamus (jointly with the medial lenticulostriate arteries and the subcallosal perforators of A1).

3.1.2.3 Anterior Cerebral Artery Pathology

Occlusion syndromes of ACA
- Hemiparesis and hemianesthesia of the contralateral lower limb
- Abulia with bilateral lesions of the cingulum
- Akinetic mutism (bilateral fronto-mesial lesions)
- Memory loss
- Apathy
- Transcortical motor aphasia
- Head and gaze deviation toward the side of the lesion
- Paratonia
- Loss of capacity of discriminative touch and proprioception
- Urinary incontinence and other vegetative symptoms (e.g., diabetes insipidus)

Specific artery occlusions:
- Pericallosal artery → apraxia, agraphia, and tactile anomia of the left hand
- ACA azygos → paraparesis with or without loss of sensation
- Subcallosal artery → anterograde amnesia, anxiety, and psychomotor agitation
- Recurrent artery of Heubner → facial hemiparesis and loss of tactile sensation

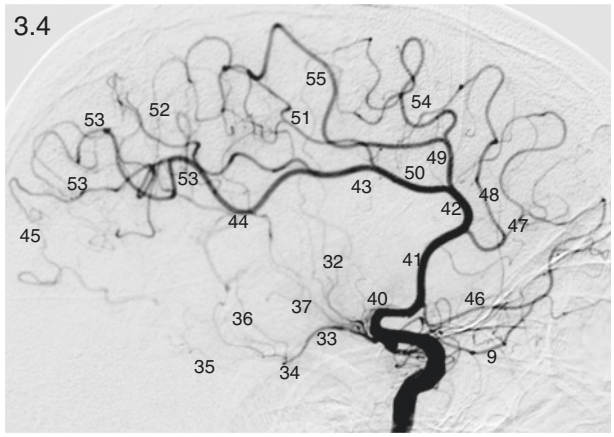


Fig. 3.4 Anterior cerebral artery and anterior choroidal artery, best seen in the absence of contrast medium in the middle cerebral artery territory due to occlusion of the distal portion of the M1 segment. Lateral projection

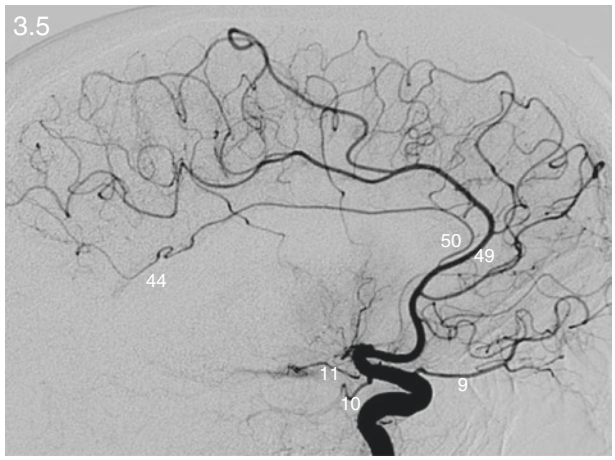


Fig. 3.5 Anatomic variant with dominant callosomarginal artery, best seen in the absence of contrast medium in the middle cerebral artery territory due to occlusion of the distal portion of the M1 segment. Lateral projection

9	Ophthalmic artery
10	Posteriorior communicating artery
11	Anterior choroidal artery
32	Lateral lenticulostriate arteries
33	Cisternal segment of the anterior choroidal artery
34	Inferior choroidal point of the anterior choroidal artery
35	Choroidal trunk of plexal segment of the anterior choroidal artery
36	Thalamo-pulvinaric trunk of plexal segment of the anterior choroidal artery
37	Perforating arteries of the anterior choroidal artery, anterior group
40	Horizontal or sovraoptic segment (A1) of the anterior cerebral artery (ACA)
41	Infracallosal segment (A2) of the anterior cerebral artery (ACA)
42	Precallosal segment (A3) of the anterior cerebral artery (ACA)
43	Supracallosal segment (A4) of the anterior cerebral artery (ACA)
44	Splenial segment (A5) of the anterior cerebral artery (ACA)
45	ACA-MCA pial anastomoses
46	Fronto-orbital artery
47	Frontopolar artery
48	Anterior internal frontal artery
49	Callosomarginal artery
50	Pericallosal artery
51	Paracentral artery
52	superior internal parietal artery
53	Inferior internal parietal artery

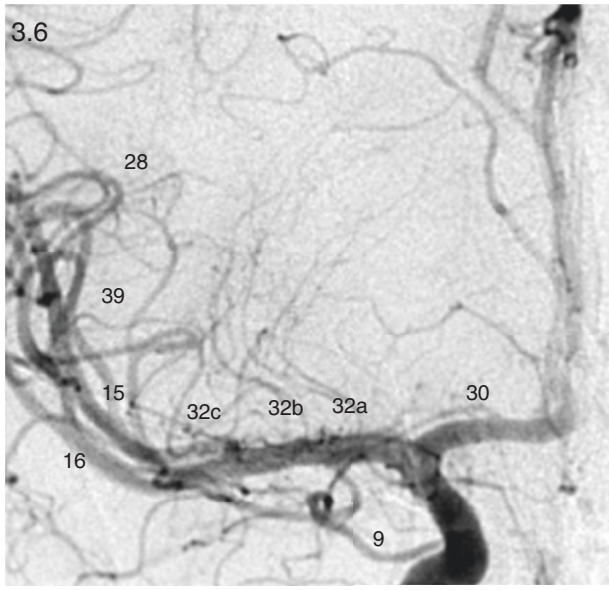


Fig. 3.6 Recurrent artery of Heubner, lenticulostriate perforators, and perforators of the insular segment of MCA. Anteroposterior projection

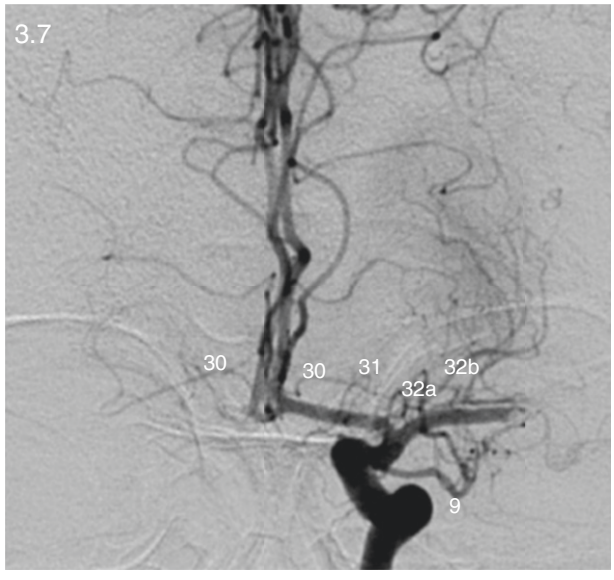


Fig. 3.7 Lenticulostriate arteries and bilateral recurrent artery of Heubner, better seen due to the complete occlusion of the M1 segment of the MCA. Anteroposterior projection

9	Ophthalmic artery
15	Superior or frontal trunk of the middle cerebral artery
16	Inferior or temporal trunk of the middle cerebral artery
28	Sylvian point of middle cerebral artery's trunks
30	Recurrent artery of Heubner
31	Medial lenticulostriate arteries and carotid bifurcation's perforating arteries
32a	Lateral lenticulostriate arteries, medial group
32b	Lateral lenticulostriate arteries, intermediate group
32c	Lateral lenticulostriate arteries, lateral group
39	Insular perforating arteries

3.1.3 Middle Cerebral Artery

3.1.3.1 Cortical Territory

The middle cerebral artery (MCA) provides the temporal lobe, insular cortex, lateral aspects of the frontal lobe and the basal ganglia. It can be subdivided into four different segments (Figs. 3.8, 3.9 and 3.10).

I. M1 segment (sphenoidal or horizontal):
   (a) Lateral lenticulostriate arteries: supply the anterior deep gray matter nuclei (pallidum, putamen, caudate nuclei), internal capsule, adjacent fibers of the corona radiata
   (b) Anterior temporal artery

II. M2 segment (insular): it lies on the insular surface from the genu to the circular sulcus. Between M1 and M2 segment the MCA bifurcates (and sometimes trifurcates) in its main branches:
   (a) Superior branch (frontal opercular branch): this gives rise to prefrontal artery, precentral artery, and central artery.
   (b) Inferior branch (temporal opercular branch): this artery gives rise to the anterior, the middle and the posterior temporal arteries, temporo-occipital artery, and the angular gyrus artery.
   (c) Also, insular medullary perforating arteries (vascular supply to the external regions of the putamen, external capsule, and the claustrum) arise from M2 segment.

III. M3 segment (opercular): it extends from the turn at the level of the circular sulcus to the turn at the Sylvian fissure.

IV. M4 segment (cortical) starts from the superficial part of the Sylvian fissure and its branches are identified by the name of the sulci they lie in.

3.1.3.2 Deep Territory

From the M1 segment of the MCA originate a number of perforating arteries that supply the putamen, the lateral portion of the globus pallidus, the body of caudate nucleus, the superior part of the internal capsule, the lateral part of the anterior commissure, and the substantia innominata. The main branches are (Figs. 3.6, 3.7 and 3.11):

- Lateral lenticulostriate arteries may be further grouped into a medial group (in the proximity of the origin of M1), an intermediate group that arise from the middle third of M1, and a more lateral group that arises from the M1–M2 junction (Fig. 3.7).
- Insular medullary perforating arteries arise from M3, and they typically distribute similarly to the cortical medullary arteries in the depths of the insular cortex to provide vascular supply to the external regions of the putamen, the external capsule, and the claustrum.

3.1.3.3 Middle Cerebral Artery Pathology

Occlusion syndromes of the MCA

- Contralateral hemiplegia and hemianesthesia
- Deviation of gaze toward the lesion
- Homonymous hemianopsia (superior one-half of the optic radiation)
- Dominant hemisphere MCA occlusion
  – Superior division: Broca's aphasia
  – Inferior division: Wernicke's aphasia
- Nondominant hemisphere: hemineglect, hemiinattention, tactile extinction, visual extinction, anosognosia, and apraxia
- Lenticulostriate arteries: pure motor hemiparesis
- Angular gyrus artery: agnosia, acalculia, agraphia, and spatial disorientation

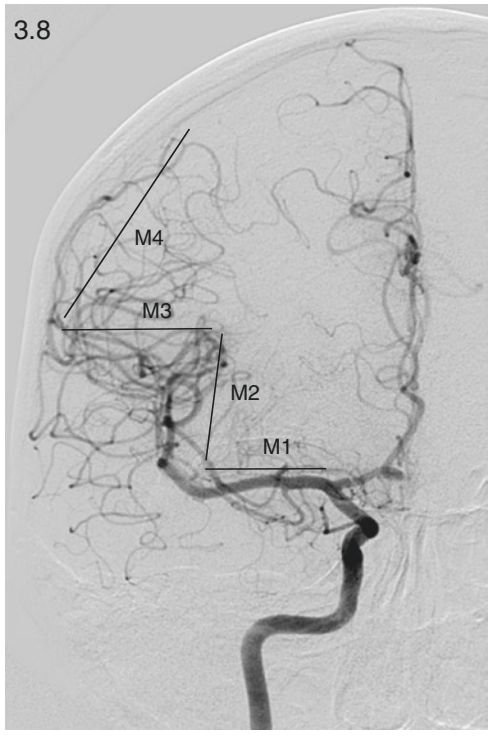


Fig. 3.8 Segments of the middle cerebral artery. Anteroposterior projection

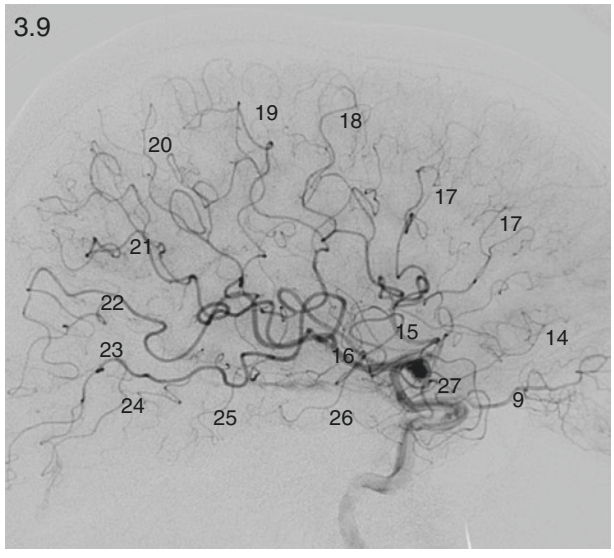


Fig. 3.9 Cortical branches of the middle cerebral artery. Best seen in this patient with congenital agenesis of A1 segment of the ipsilateral anterior cerebral artery. Lateral projection

9	Ophthalmic artery
14	Orbitofrontal artery
15	Superior or frontal trunk of the middle cerebral artery
16	Inferior or temporal trunk of the middle cerebral artery
17	Prefrontal arteries
18	Precentral artery
19	Central artery
20	Anterior parietal artery
21	Posterior parietal artery
22	Angular artery
23	Temporo-occipital artery
24	Posterior temporal artery
25	Middle temporal artery
26	Anterior temporal artery
27	Temporal polar artery

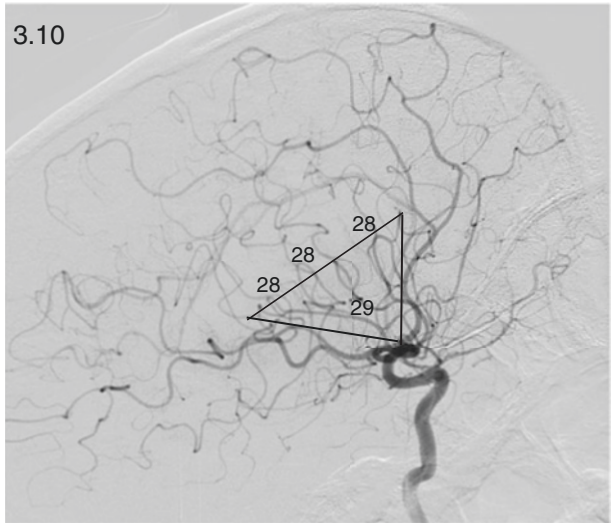
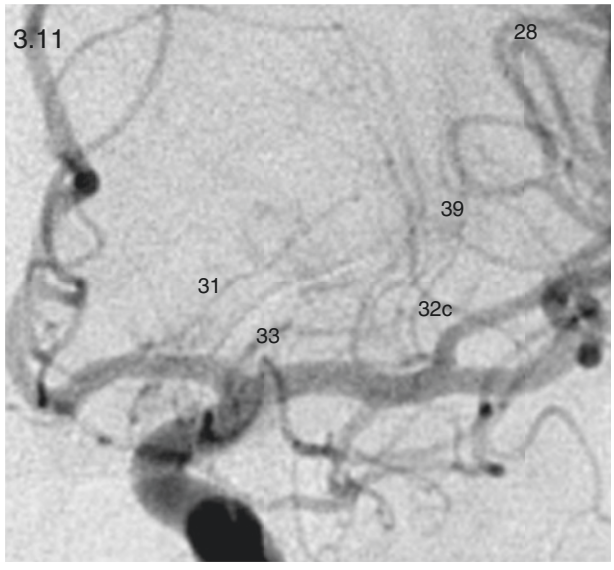


Fig. 3.10 Insular triangle. This is a point of reference in angiography with the superior border represented by "Sylvian points," where the MCA branches bend from the insula surface to the inferior surface of the frontal lobe. Lateral projection

Fig. 3.11 Lenticulostriate arteries. Anteroposterior projection

28	Sylvian point of middle cerebral artery's trunks
29	Insular triangle
31	Medial lenticulostriate arteries and carotid bifurcation's perforating arteries
32c	Lateral lenticulostriate arteries, lateral group
33	Cisternal segment of the anterior choroidal artery
39	Insular perforating arteries



3.1.4 Posterior Cerebral Artery

3.1.4.1 Cortical Territory

Basilar artery bifurcates into the left and right posterior cerebral artery (PCA). However, not infrequently PCA may directly arise as a continuation of the posterior communicating artery originating from the carotid siphon (10% of the cases) and is considered of fetal origin. PCA can be divided into four segments: P1, P2, P3, and P4 segments (Figs. 3.12, 3.13 and 3.14).

I. P1 segment (interpeduncular cisternal). Short segment between the origin of PCA and the conjunction with the PComA. Its branches include:
   (a) Medial thalamic perforating arteries
   (b) Posterior thalamic perforating arteries
   (c) Anterior thalamic perforating arteries

II. P2 segment (perimesencephalic). It can be further subdivided into two segments by the origin of the posterolateral choroidal artery
   (a) Anterior P2 (P2A): from the origin of PComA to the origin of the posterolateral choroidal artery (posterior margin of the cerebral peduncle)
   (b) Posterior P2 (P2P): posterior perimesencephalic from lateral mesencephalic sulcus to the lateral geniculate body

III. P3 (quadrigeminal) segment. Extends from the lateral geniculate body to the anterior margin of the calcarine sulcus; at this level it divides into its main branches
   (a) Calcarine artery: supplies the inferomedial and posterior surface of the occipital lobe)
   (b) Parieto-occipital artery: supplies the mesial surface of the parietal lobe and the precuneus

IV. P4 (cortical) segment: the part of the final branches that spreads along the cortical surfaces
   (a) Anteroinferior temporal artery: is the first cortical branch of the PCA. It arises from the P2A and supplies the inferior and medial portions of the temporal lobe. Its caliber depends mostly on the development of the posteroinferior temporal artery.
   (b) Postero-medial choroidal artery: arises at the junction of the P1 and P2A segments.
   (c) Posterolateral choroidal artery: arises at the junction of the P2 and the P3 segment.
   (d) Thalamogeniculate artery: it supplies the thalamus and the lateral geniculate body.
   (e) Posteroinferior temporal artery: originates from P3 and supplies the lateral portion of the inferior surface of the temporal lobe.
   (f) Internal parieto-occipital artery: it supplies the medial parieto-occipital surface of the brain including the inferior region of the precuneus and the calcarine area.
   (g) Calcarine artery is headed caudal and medial and supplies the calcarine gyrus, to the pole and the basal surface of the occipital lobe.

3.1.4.2 Deep Territory

The PCA has a number of perforator arteries that supply the thalamus (except for the anterior pole, which is a territory of the thalamotuberal arteries, branches of the posterior communicating artery), the midbrain, the pineal gland, the posterior commissure, the fornix, the splenium of the corpus callosum, the hippocampus, the mammillary bodies, and the choroid plexus of the third and the lateral ventricles. The main branches are:

- Medial thalamic perforators or thalamoperforate arteries (originate from the basilar apex).
- Posterior thalamic arteries or thalamogeniculate arteries.
- Mesencephalic perforators.
- Postero-medial choroidal artery: arises from either P1 or P2A and divides into two branches. The main branch reaches the choroidal plexus of the third ventricle and the pulvinar. The other branch supplies the choroidal plexus of the lateral ventricle.

- Posterolateral choroidal artery:
  - Lateral branch that reaches the choroidal plexus in the temporal horn of the lateral ventricle.
  - Medial branch surrounds the pulvinar and terminates at the level of the ventricular trigone to anastomose with the anterior choroidal artery.
- Superior collicular arteries: supply the quadrigeminal plate and may anastomose with the inferior collicular arteries that branch out from the SCA.

3.1.4.3 Posterior Cerebral Artery Pathology

If cortical territories are affected then:

- Contralateral homonymous hemianopsia
- Cortical blindness due to lesions in bilateral occipital lobes
- Visual agnosia
- Prosopagnosia, palinopsia, and micropsia
- Alexia, aphasia anomica, and dyschromatopsia
- Memory loss
- Spatial and temporal disorientation

Occlusion of perforating arteries may give rise to different clinical syndromes:

- Weber's syndrome → contralateral hemiplegia of the face, upper and lower limb (both corticospinal and corticobulbar tracts are involved), and ipsilateral ophthalmoplegia.
- Foville syndrome → it is similar to Weber's syndrome with conjugated gaze paralysis contralateral to the side of the lesion.
- Benedikt's syndrome → bilateral ophthalmoplegia, midriasis, intentional tremor, hemichorea, and hemiathetosis (lesion in the ventral tegmentum of the mesencephalon).
- Claude's syndrome → similar to Benedikt's syndrome with additional involvement of the cerebellar signs without involuntary movements (lesion is in the dorsal mesencephalon).
- Nothnagel's syndrome → ipsilateral oculomotor paralysis with contralateral cerebellar ataxia.
- Parinaud's syndrome → paralysis of vertical gaze, nystagmus, loss of convergence, mydriatic pupil, and incapacity to accommodation.

3.1.5 Deep Gray Matter

The arterial blood supply to the caudate, putamen, globus pallidus, and the thalamus is provided via perforating arteries that arise from the arteries in the basal cisterns. These arteries reach deep into these nuclei through the anterior and posterior perforated substance. The anterior perforated substance is anteriorly delimited by the gyrus rectus and orbital gyrus, laterally by the olfactory stria, medially by the optic tracts, posteriorly by the temporal pole, and laterally by the limen insulae (Figs. 3.6, 3.7, 3.11, 3.13 and 3.15).

Two groups of perforating arteries supply the basal ganglia:

- Perforating arteries of the carotid apex, medial and lateral lenticulostriate arteries, and Heubner's recurrent artery reach the deep nuclei by penetrating through the anterior perforated substance. The anterior and the medial perforating thalamic branches also enter the anterior perforated substance.
- Perforating arteries arising from the anterior choroidal artery in the deep anterior choroidal scissure or arising from the artery in the ambiens cistern that embraces the brainstem (posteromedial choroidal artery, posterolateral choroidal artery, and the thalamogeniculate artery).

The basal ganglia are subdivided in the axial and the sagittal plane in two vascular territories: anterior and posterior. The anterior vascular territory (caudate, putamen, globus pallidus, internal capsule) is supplied by the perforating arteries of the anterior circulation. The posterior territory

(thalamus, geniculate bodies) is supplied by perforating arteries of the posterior circulation. The posterior limb of the internal capsule defines the boundary between the two territories.

Caudate nucleus: The head is supplied by the recurrent artery of Heubner. The body is supplied by the lateral lenticulostriate arteries. An anterior group of arteries supplies the anterior part, the middle group supplies the middle part of the body, and the lateral group supplies the posterior end of the body. The tail is supplied by a posterior group of anterior choroidal artery that provides vascular supply near the temporal horn.

Lentiform nucleus (putamen and globus pallidus): The anterior and inferior portion is supplied laterally by the medial lenticulostriate arteries and medially by the anterior perforators of the anterior choroidal artery. The posterior portion of the globus pallidus to the posterior arm of the internal capsule is supplied by the lateral lenticulostriate arteries.

Thalamus: The thalamus is supplied from the front to the back by the anterior thalamic perforators (arise from the PCom), medial thalamus from the basilar artery (or perforators arising from the P1), lateral thalamus from the thalamogeniculate artery, posterior thalamus from the thalamic arteries of the posteromedial choroidal artery, and posterolateral thalamus from the thalamopulvarinic arteries originating from the P3.

Claustrum, external, and extreme capsule: Supplied by insular branches from the M2.

Subthalamic nuclei, red nucleus, and substantia nigra: Supplied via medial perforators from the anterior choroidal artery, midbrain perforators from P2 to P3, and middle thalamoperforators from the middle group.

Hypothalamus and the third ventricle: The anterior hypothalamus is supplied by the perforating arteries of the anterior communicating artery, the subcallosal artery, and the more medial group of lateral lenticulostriate arteries. The posterior part of the hypothalamus is supplied by the posteromedial choroidal arteries. The basal part of the infundibulum and the tuber cinereum is supplied by the posterior communicating artery. The premammillary artery (also known as the anterior thalamoperforator artery) is the largest artery and supplies the floor of the third ventricle. These arteries also provide arterial supply to the posterior portion of the hypothalamus, the posterior arm of the internal capsule, and the subthalamus. The anterior group of arteries arising from the posterior communicating artery supplies the hypothalamus, the ventral thalamus, the anterior third of the optic nerve, the posterior arm of the internal capsule, and the subthalamic nuclei.

Internal capsule: Genu—perforating arteries from the carotid apex. Anterior limb: medial lenticulostriate arteries arising from A1 and Heubner's recurrent artery (inferior surface). Posterior limb: posterior group of perforating arteries of the anterior choroidal artery and anterior thalamoperforators (premammillary arteries of the posterior communicating artery).

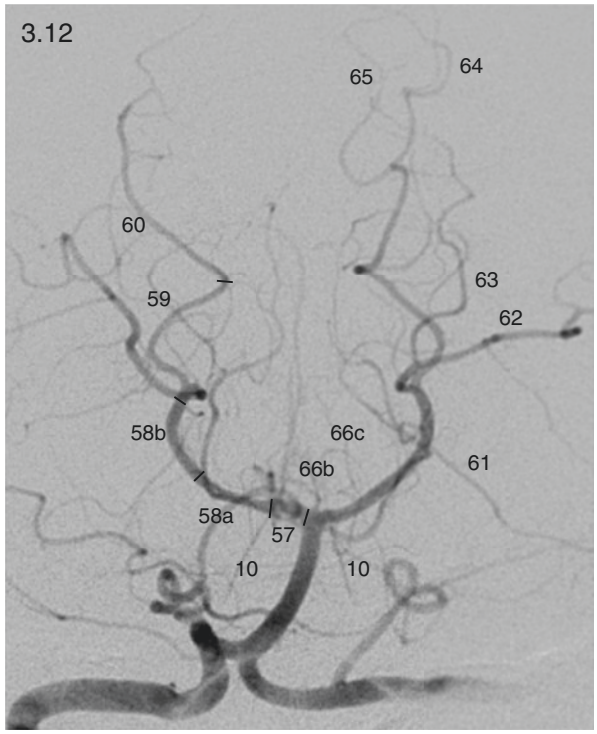


Fig. 3.12 Posterior cerebral arteries with their segments. Occipito-frontal projection

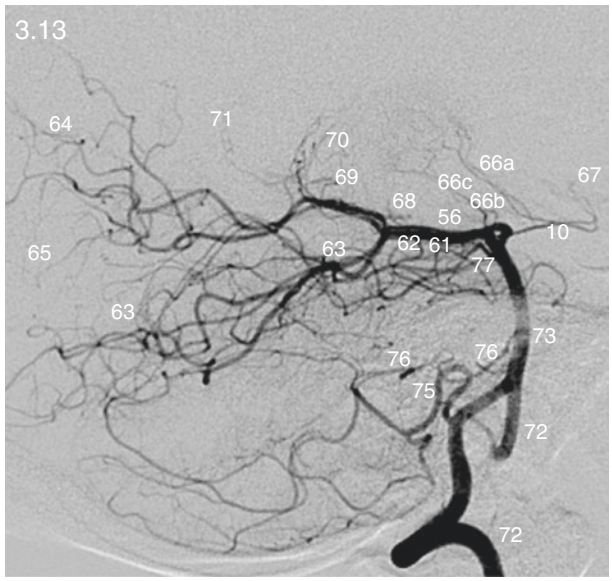


Fig. 3.13 Arteries of the posterior circulation and perforator arteries. Lateral projection

10	Posterior communicating artery
56	Posterior cerebral artery (PCA)
57	Precommunicating or interpeduncular segment (P1) of posterior cerebral artery
58a	Circumpeduncular or ambient segment (P2) of posterior cerebral artery, anterior segment
58b	Circumpeduncular or ambient segment (P2) of posterior cerebral artery, posterior segment
59	Quadrigemina segment (P3) of posterior cerebral artery
60	Cortical segment (P4) of poster or cerebral artery
61	Anterior inferior temporal artery
62	Middle inferior temporal artery
63	Posterior inferior temporal artery
64	Parieto-occipital artery
65	Calcarine artery
66a	Perforating thalamic arteries, anterior thalamic or thalamotuberal artery
66b	Perforating thalamic arteries, middle thalamic or thalamoperforating arteries
66c	Perforating thalamic arteries, posterior thalamic or thalamoperforating arteries
67	Tubero-infundibular artery
68	Thalamogeniculate artery
69	Medial posterior choroidal artery
70	Lateral posterior choroidal artery
71	Splenial or posterior pericallosal artery
72	Vertebral artery (VA)
73	Anterior spinal artery (ASA)
75	Posterior inferior cerebellar artery (PICA)
76	Anterior inferior cerebellar artery (AICA)
77	Superior cerebellar artery (SCA)

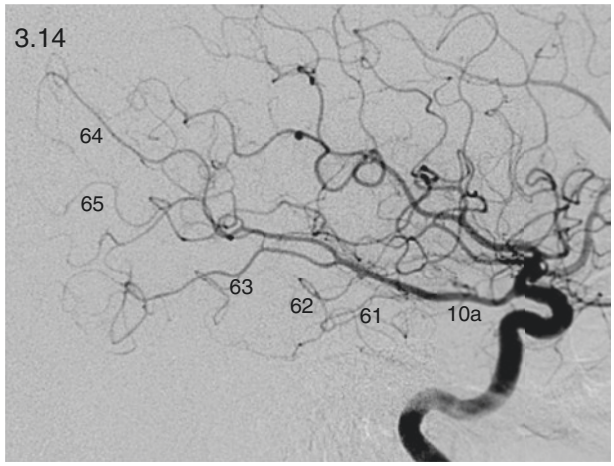


Fig. 3.14 Cortical branches of the posterior cerebral artery, best seen in the absence of contrast medium in the infratentorial arteries in this patient with a fetal variant of the origin of the carotid siphon. Lateral projection

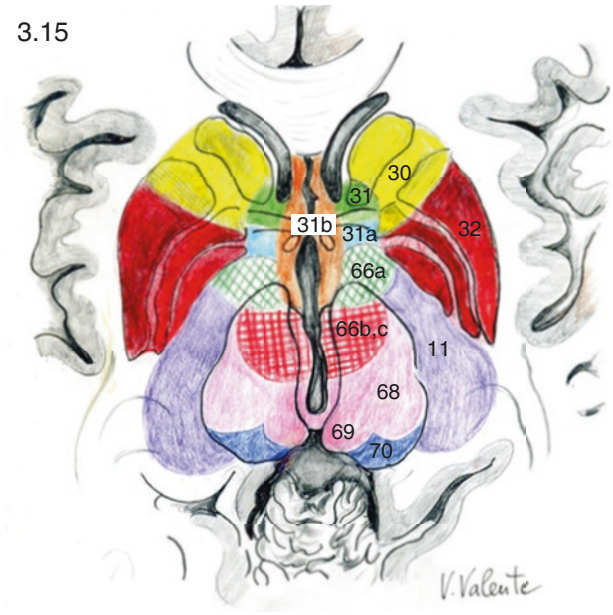


Fig. 3.15 Sketch highlighting the main arterial territories supplying the basal ganglia and thalami

10a	Fetal type posterior cerebral artery (PCA)
11	Anterior choroidal artery
30	Recurrent artery of Heubner
31	Medial lenticulostriate arteries and carotid bifurcation's perforating arteries
31a	Carotid bifurcation's perforating arteries
31b	Midline perforators or diencephalic perforators from the AComA
32	Lateral lenticulostriate arteries
61	Anterior inferior temporal artery
62	Middle inferior temporal artery
63	Posterior inferior temporal artery
64	Parieto-occipital artery
65	Calcarine artery
66a	Perforating thalamic arteries, anterior thalamic or thalamotuberal artery
66b,c	Perforating thalamic arteries, middle and posterior thalamic or thalamoperforating arteries
68	Thalamogeniculate artery
69	Medial posterior choroidal artery
70	Lateral posterior choroidal artery

3.2 Major Supratentorial Veins

The cerebral venous circulation is highly variable among individuals, allowing for only a partial categorization of this system. The differences are due to the variable courses of the veins, the presence or absence of anastomoses with other venous territories, and different levels of dural sinus development and/or their agenesis.

There are two major venous systems: superficial and deep. The superficial venous system refers to the drainage of the short external medullary veins of the cerebral cortex and the subcortical white matter of the gyri. The superficial venous system uses dural sinuses as their common drainage pathway. The deep venous system drains basal ganglia by internal medullary veins that converge toward the ventricles and lie over the subependymal surface of the cerebral ventricles draining into the internal cerebral veins. Basal veins (of Rosenthal) are also considered part of the deep venous system and drain the cortical scissure and the structures of the diencephalic gray matter. Internal cerebral veins and basal veins drain into the vein of Galen, straight sinus, and confluence of sinuses (or torcular herophili) where the superficial and deep systems join and then into the transverse and sigmoid sinuses and finally to the internal jugular veins. Another venous drainage route is at the level of the cavernous sinus. Both the superior and inferior petrosal sinus and the sphenoparietal sinus drain venous blood from the brain structures lying over the petrous surface of the posterior cranial fossa and the cortical structures surrounding the Sylvian fissure.

Superficial venous anastomoses differ at the level of the cerebral convexity. Cortical veins such as Trolard's and Labbè's ensure efficient collateral flow in cases of increased flow, stenosis, or thrombosis of a venous sinus, mediated by the superficial Sylvian veins. However, in the deep venous system, anastomotic veins are scarce and hemodynamic compensation is almost exclusively mediated through venous reflux from the straight sinus.

For the posterior cranial fossa, alternative routes of compensatory flow are through deep veins that allow anastomotic flow between superior petrous, precentral-cerebellar, lateral mesencephalic, and the basal vein. Anastomoses between vermian veins and the cerebellar convexity ensure direct collateral circulation to the basal dural sinuses (Figs. 3.16, 3.17, 3.18, 3.19 and 3.20).

Supratentorial superficial veins. These veins drain the cortex of the external surface of the brain.

I. Lateral group of veins over the convexity drain into the superior sagittal sinus. The principle collector vein in this region is the Trolard vein.
   (a) Frontal veins
   (b) Rolandic veins
   (c) Parieto-occipital veins
II. Medial group of veins that drain the medial surface of the cerebral hemispheres. In lateral projection of DSA images, veins in the median surface can be distinguished from the veins of the external surface by their linear course and smaller length.
   (a) Frontomedial veins
   (b) Veins of the paracentral lobule
   (c) Parieto-occipital medial veins
III. Dural sinus of the basal convexity. Superficial veins that drain into the tentorial sinus (transverse and sigmoid sinuses). The principal collector of this district is the vein of Labbè or inferior anastomotic vein that connects the transverse sinus with the superficial Sylvian vein. The vein of Labbè is classically considered to be more variable and may be missing in almost 50% of the cases.
   (a) Occipital veins
   (b) Occipitotemporal basal veins
   (c) Medial occipital veins
IV. Veins of the Sylvian group (anterior draining veins, anterior group, superficial Sylvian vein). The veins of the Sylvian group commonly flow into the superficial Sylvian vein, and then into the sphenoparietal sinus, and eventually into the cavernous sinus. This ensures a collateral anterior route for the veins of the convexity and the cavernous sinus.
   (a) Fronto-opercular veins
   (b) Temporo-opercular veins
   (c) Parieto-insular veins

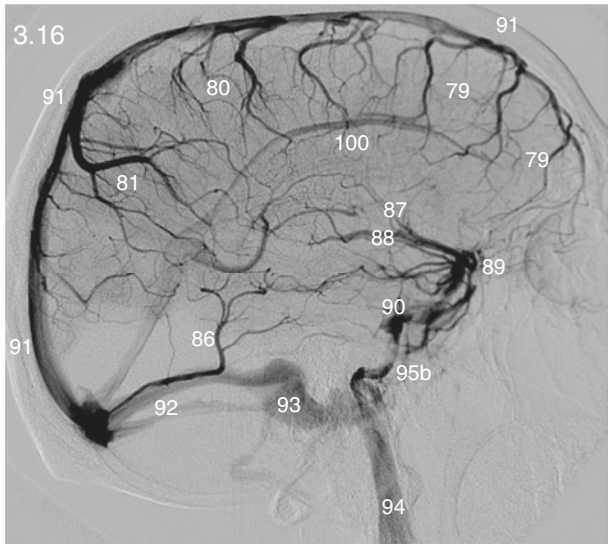


Fig. 3.16 Supratentorial venous system with balanced vascular territories without any predominance of the cortical drainage system. Lateral projection

79	Frontal veins
80	Central veins
81	Parietal veins
86	Temporo-occipital veins
87	Sylvian veins
88	Superficial Sylvian vein or superficial middle cerebral vein
89	Sphenoparietal sinus
90	Cavernous sinus
91	Superior sagittal sinus
92	Transverse sinus
93	Sigmoid sinus
94	Internal jugular vein
95b	Inferior petrosal sinus
100	Inferior sagittal sinus

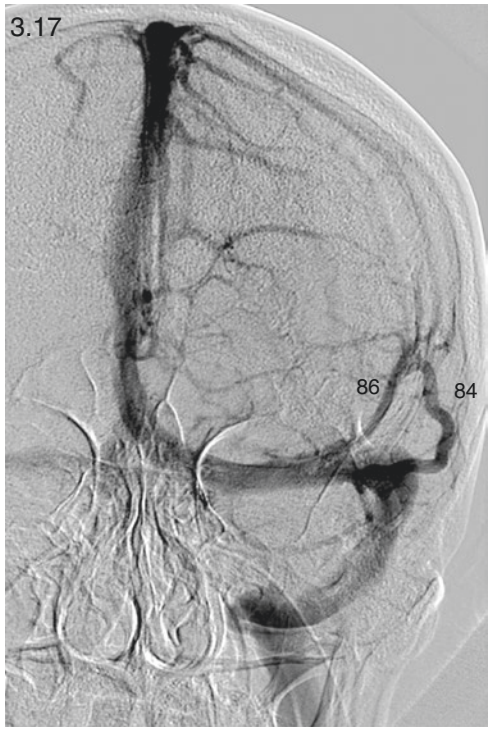


Fig. 3.17 Vein of Labbè. Anteroposterior projection

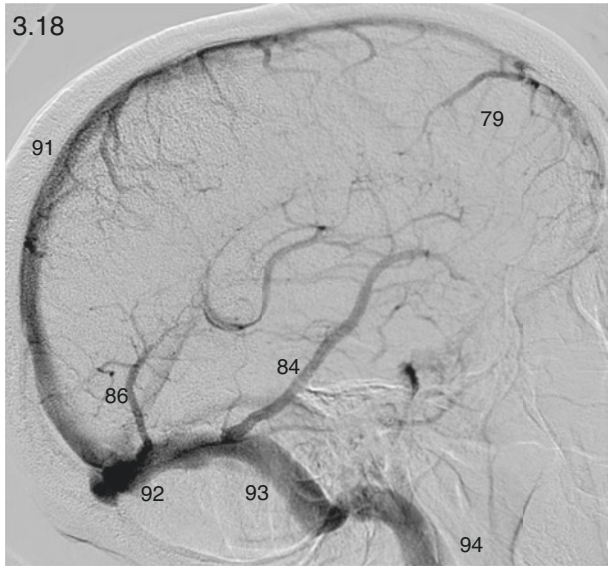


Fig. 3.18 Vein of Labbè. Lateral projection

79	Frontal veins
84	Vein of Labbe or inferior anastomotic vein
86	Temporo-occipital veins
91	Superior sagittal sinus
92	Transverse sinus
93	Sigmoid sinus
94	Internal jugular vein

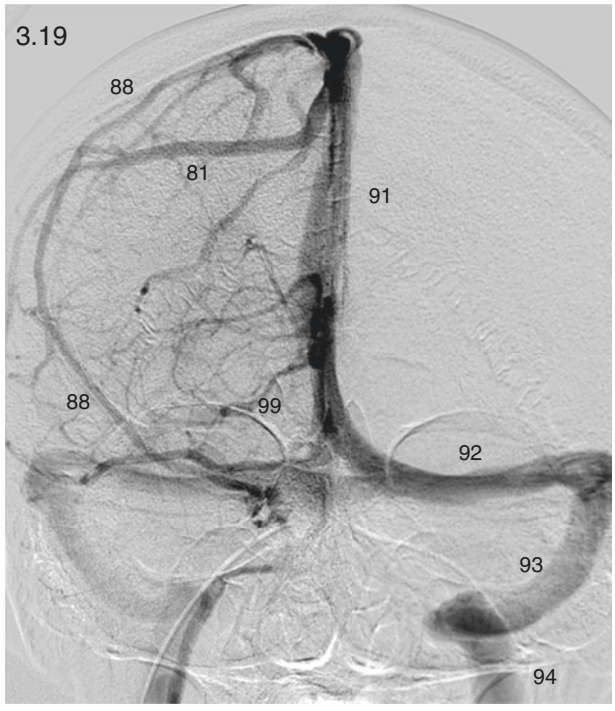


Fig. 3.19 Vein of Trolard and Sylvian vein. Anteroposterior projection

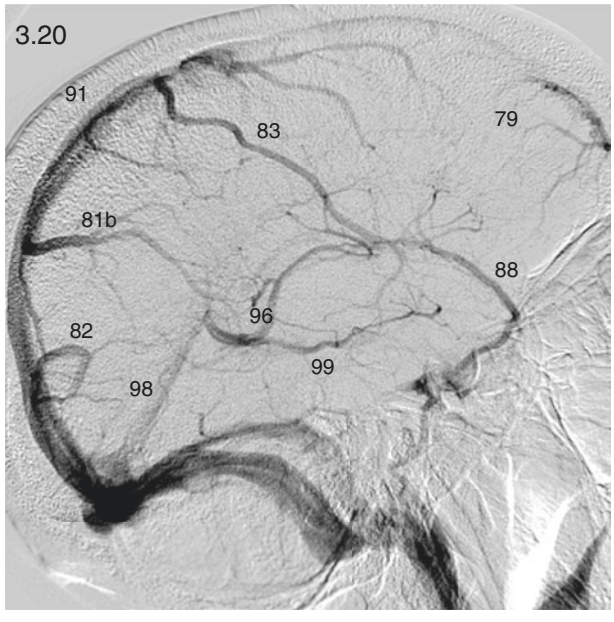


Fig. 3.20 Vein of Trolard and Sylvian vein. Lateral projection

79	Frontal veins
81	Parietal veins
81b	Parietal veins, parietal veins of the lateral convexity
82	Occipital veins
83	Vein of Trolard or superior anastomotic vein
88	Superficial Sylvian vein or superficial middle cerebral vein
91	Superior sagittal sinus
92	Transverse sinus
93	Sigmoid sinus
94	Internal jugular vein
96	Internal cerebral vein
98	Straight sinus
99	Basal vein of Rosenthal

Supratentorial deep veins. These deep veins drain the periventricular white matter, deep gray matter, deep cortical territories, and the diencephalic structures (Figs. 3.21 and 3.22).

The internal cerebral vein system and the vein of Galen are fed from tributaries that course inside the ventricular system and hence are termed ventricular veins. They are further subdivided into medial and lateral tributary veins. Not all of these are visible on DSA imaging because of their very small diameters.

The basal vein originates from the confluence of the anterior cerebral vein with the deep middle cerebral vein. The main territory it drains is the orbitofrontal cortex, the insula, the temporal cortex, the mesial temporal structures, the mesencephalon, the hippocampal structures, the hypothalamus, the inferior part of the striatum, and the thalamus.

3.2.1 Tributaries of the Lateral Ventricular Veins

- Anterior caudate vein (connects with the thalamostriate veins)
- Longitudinal caudate vein
- Thalamostriate vein (drains the frontal posterior group of internal medullary veins, parietal medullary veins, and the internal capsule)
- Posterior caudate vein
- Superior thalamic vein
- Lateral atrial vein or direct lateral vein
- Inferior ventricular vein
- Amygdalar veins in the temporal horn.

3.2.2 Tributaries of the Medial Ventricular Veins

- Anterior septal vein (drain the deep frontal lobe structures, the septum, and the fornix)
- Posterior septal veins in the lateral ventricular body
- Medial atrial vein (courses at the level of the atrium posterior to the direct lateral vein)
- Transverse hippocampal veins
- Superior choroidal veins
- Inferior choroidal vein
- Inferior ventricular vein

3.2.3 Tributaries of the Anterior Segment of the Basal Vein

- Deep middle cerebral vein
- Anterior cerebral veins
- Orbitofrontal vein
- Olfactory vein
- Uncal vein
- Peduncular vein
- Inferior striate veins

3.2.4 Tributaries of the Middle Segment of the Basal Vein

- Inferior ventricular vein for the roof of the temporal horn
- Anterior longitudinal hippocampal vein
- Lateral mesencephalic
- The temporal cortical veins from the posterior two-thirds of the uncus
- Medial temporal veins from the medial surface of the temporal lobe (parahippocampal) and the occipitotemporal gyrus
- Hypothalamic veins

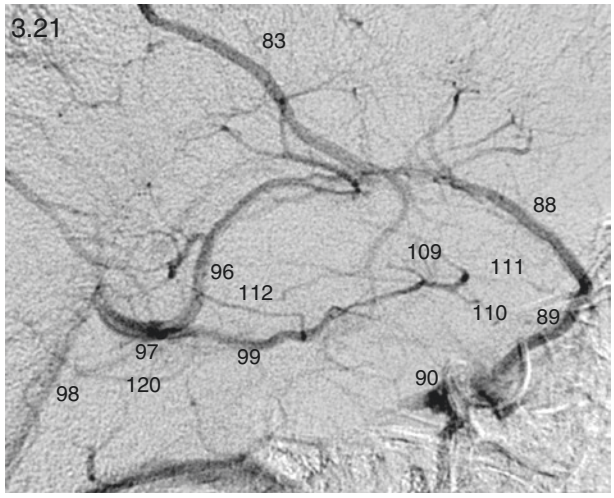


Fig. 3.21 Deep cerebral venous system. Lateral projection

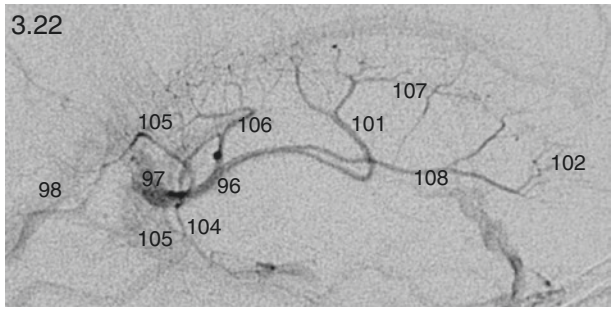


Fig. 3.22 Internal cerebral vein. Lateral projection

83	Vein of Trolard or superior anastomotic vein
88	Superficial Sylvian vein or superficial middle cerebral vein
89	Sphenoparietal sinus
90	Cavernous sinus
96	Internal cerebral vein
97	Vein of Galen
98	Straight sinus
99	Basal vein of Rosenthal
101	Thalamostriate vein
102	Anterior caudate vein
104	Inferior ventricular vein
105	Medial atrial vein
106	Direct lateral vein
107	Longitudinal caudate vein
108	Septal vein
109	Inferior striate veins
110	Olfactory vein
111	Orbitofrontal vein
112	Inferior ventricular vein
120	Internal occipital vein

3.2.5 Tributaries of the Posterior Segment of the Basal Vein

These may connect to the basal vein but also to the terminal end of the internal cerebral veins or directly onto the ampulla of Galen.

- Lateral atrial veins
- Inferior ventricular veins
- Posterior longitudinal hippocampal (posterior portion of the dentate gyrus)
- Posterior pericallosal vein
- Superior vermian vein
- Lateral mesencephalic tectal veins
- Epithalamic veins
- Internal occipital veins, from the calcarine to the parieto-occipital sulci
- Posterior thalamic veins

3.2.6 Supratentorial Venous Pathology

A diagnosis of cerebral venous thrombosis (CVT) is made on the basis of clinical suspicion and neuroimaging findings.

Neurologic deficit due to CVT is related to an increase in intracranial pressure due to altered venous drainage and due to focal cerebral damage resulting from hemorrhage. Headache is the most frequent symptom and is present in 90% of patients with CVT. A small percentage of the patients may present with a more aggressive and sudden onset of headache similar to subarachnoid hemorrhage. Headaches are generally diffuse, constant, and progressively worsening over weeks.

Focal neurologic symptoms may complicate a CVT if there is a resulting retrograde increase in pressure that leads to parenchymal hemorrhage. Symptoms and signs will depend upon the anatomical region involved and include hemiparesis, aphasia, VIII cranial nerve palsy, pulsating tinnitus, nystagmus, monolateral hypoacusia, double vision, and vision loss.

Different clinical characteristics distinguish CVT from other etiologies causing brain damage. In CVT, 40% of the patients may present with seizures and bilateral damage is more frequent. Such is the case with bilateral thalamic infarct in deep venous thrombosis leading to altered levels of consciousness without focal neurologic deficit.

Occlusion of the superior sagittal sinus will result in headache, papilledema, seizures, and variable degree of motor deficit.

Occlusion of the transverse sinus results in headache, fever, nausea, vomiting, hearing disturbances, and pain in the mastoid region. If there is an associated cortical infarct, then there may be associated hemianopsia, contralateral hemiparesis and aphasia depending on the anatomical region involved.

Occlusion of the superficial cerebral veins is rare. Clinical symptoms may be subtle, and usually manifest due to associated hemorrhage, as is seen with the temporal lobe hemorrhage that occurs following thrombosis of the vein of Labbè.

Occlusion of the deep venous system typically causes infarction of the thalami and basal ganglia bilaterally. Headache, nausea, vomit, and altered levels of consciousness (including coma) are the presenting symptoms.

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Detailed Anatomy at 7T

4

Isabella M. Björkman-Burtscher,
Karin Markenroth Bloch, and Pia C. Maly Sundgren

The images presented in this chapter were acquired using an actively shielded Philips 7T Achieva (Best, the Netherlands) MR scanner with a dual-channel transmit and 32-channel receive head coil (Nova Medical, Wilmington, USA). For increased field homogeneity, dielectric pads were used during image acquisition. Axial T2-weighted images were obtained with a turbo spin echo (TSE) sequence, repetition time (TR) 3500 ms, echo time (TE) 60 ms and voxel dimensions of 0.5 × 0.5 × 1 mm for Figs. 4.1–4.9 and 4.13–4.18 and 0.5 × 0.5 × 0.75 mm for Figs. 4.10–4.12. Scan times of approximately 10 min do not aim at maximum achievable image resolution or quality, but are chosen to reflect scan times that are suitable for patients.

This 7T anatomy chapter on the cerebrum focuses on deep brain structures. 7T imaging not only delineates the outer boarders of these better than clinical scanners but also reveals their internal structures in greater detail. The large white matter tracts that cross between hemispheres (e.g. corpus callosum, Fig. 4.1) are well visualized. However, the benefits of 7T are better appreciated when focusing on smaller tracts such as the fornix (Figs. 4.1, 4.3), the anterior and posterior commissure (Figs. 4.4 and 4.5) or tiny white matter bundles such as the mammillothalamic fasciculus or the fasciculus retroflexus (Figs. 4.6 and 4.7). The white matter of the visual pathway is another structure that can be easily followed: the optic tract (Fig. 4.9) passes through the deep brain structures lateral to the cerebral peduncle (Fig. 4.6) to arrive at the lateral geniculate nucleus (Fig. 4.7), after which the optic radiation (Fig. 4.1) extends to primary visual cortex, easily identified by the line of Gennari [1–3] (Fig. 4.2).

7T MR scanners usually rely on transmit/receive coils and do not transmit with a body coil. As such, this may cause problems regarding the field of view accessible for scanning and signal drop at the edge of a coil. Areas prone to artefacts include the temporal lobe of the brain and the caudal structures in the posterior fossa. However, excellent temporal lobe

I.M. Björkman-Burtscher, M.D., Ph.D. (✉)
Department of Medical Imaging and Physiology, Skåne University Hospital, Lund, Sweden

Department of Diagnostic Radiology, Lund University Bioimaging Center, Lund University, Lund, Sweden
e-mail: isabella.bjorkman-burtscher@med.lu.se

K.M. Bloch, Ph.D.
Lund University Bioimaging Center, Lund University, Lund, Sweden
e-mail: Karin.markenroth_bloch@med.lu.se

P.C. Maly Sundgren, M.D., Ph.D.
Department of Diagnostic Radiology, Clinical Sciences Lund, Lund University, Lund, Sweden

Department of Medical Imaging and Physiology, Skåne University Hospital, SE.221 85 Lund, Sweden
e-mail: pia.sundgren@med.lu.se

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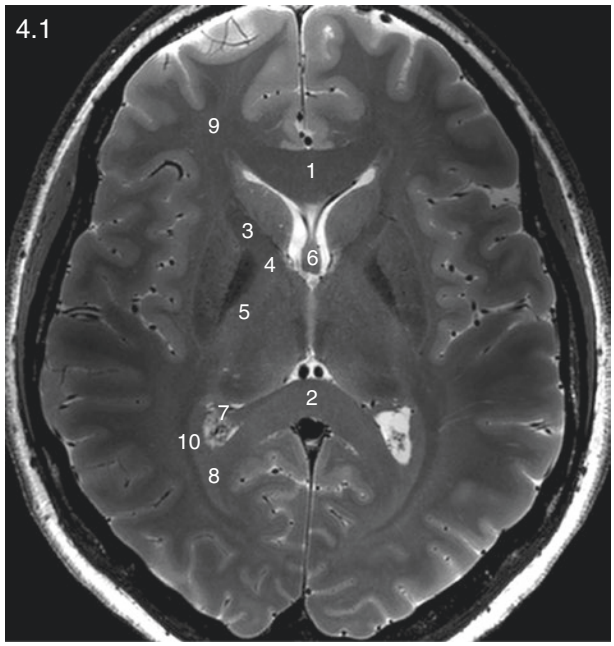
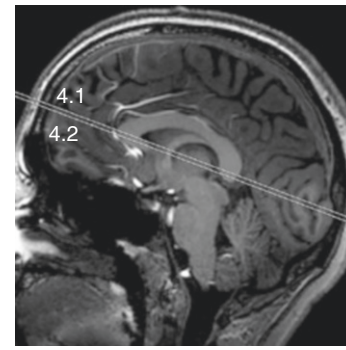
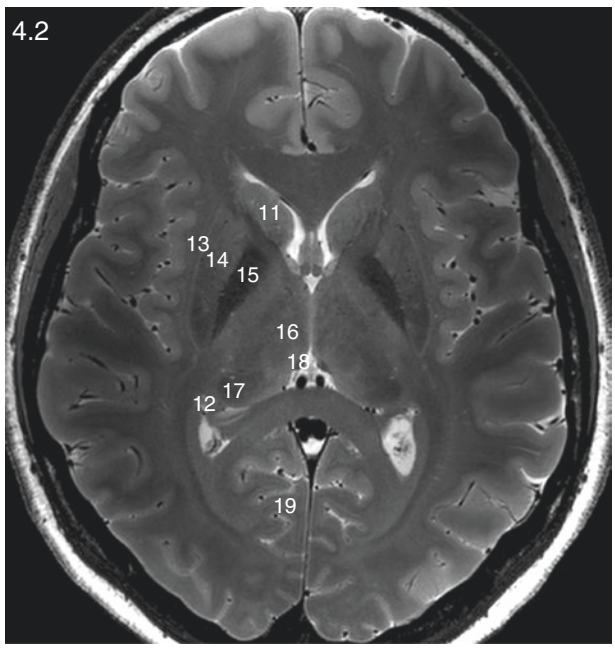
and hippocampal imaging is possible with 7T (Figs. 4.10–4.18). As the right hippocampus and left hippocampus are mirrored anatomically, Figs. 4.10–4.18 only present the right hippocampus.

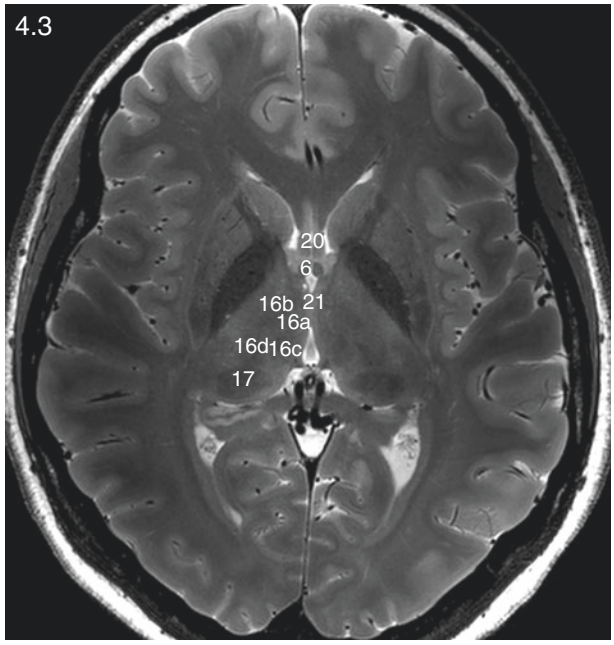
Each page contains the labelled images on the left-hand side. In order to keep the labels small, label numbers are specific to each brain region. Scout images document the locations of the slices, and a key lists the individual structures.

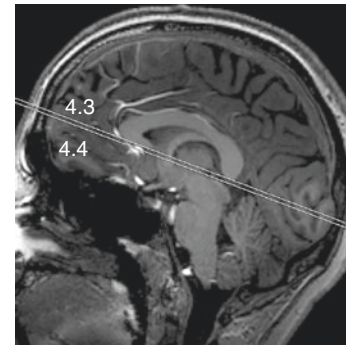


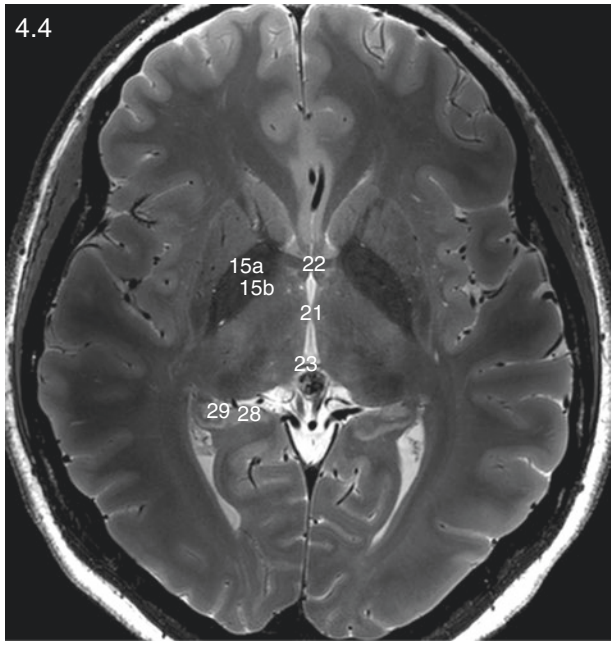




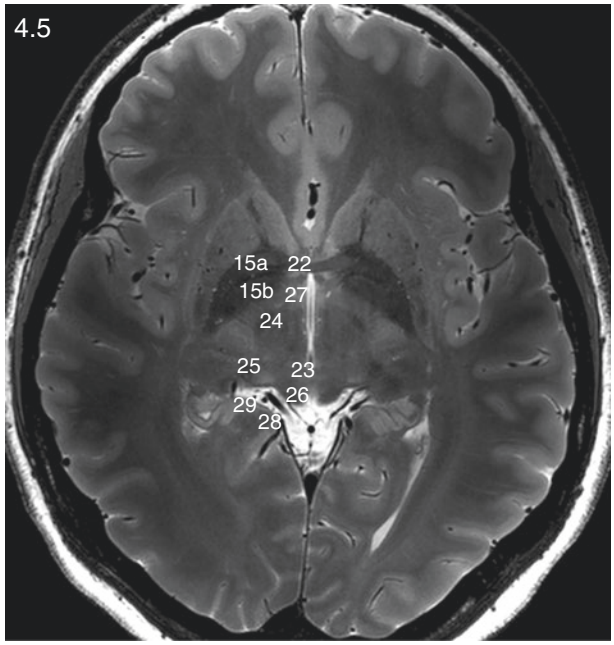
1	Genu of corpus callosum
2	Splenium of corpus callosum
3	Internal capsule, anterior limb
4	Internal capsule, genu
5	Internal capsule, posterior limb
6	Column of the fornix
7	Cauda of the fornix
8	Forceps major
9	Forceps minor
10	Optic radiation
11	Head of caudate nucleus
12	Tail of caudate nucleus
13	Claustrum
14	Putamen
15	Globus pallidus
16	Thalamus
17	Pulvinar nucleus
18	Habenula
19	Visual cortex/stria of Gennari

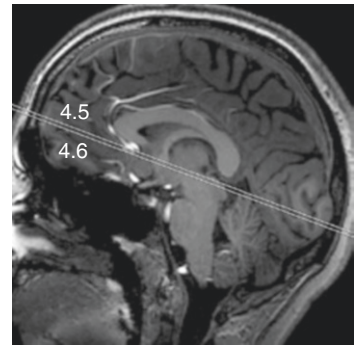


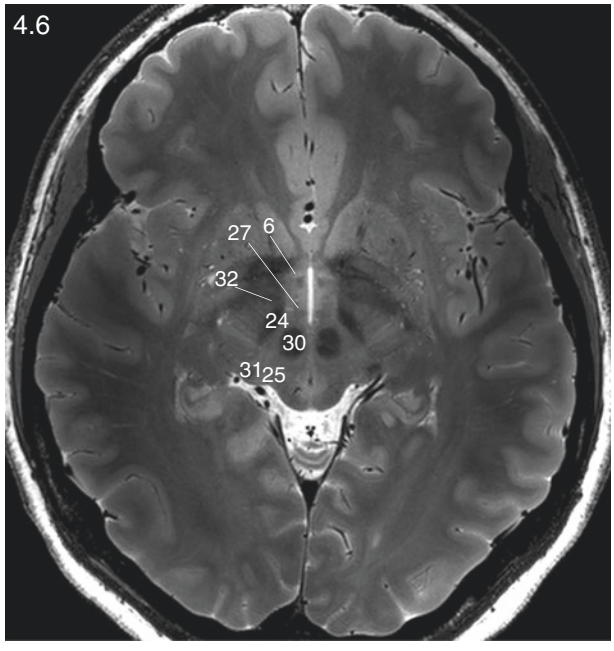




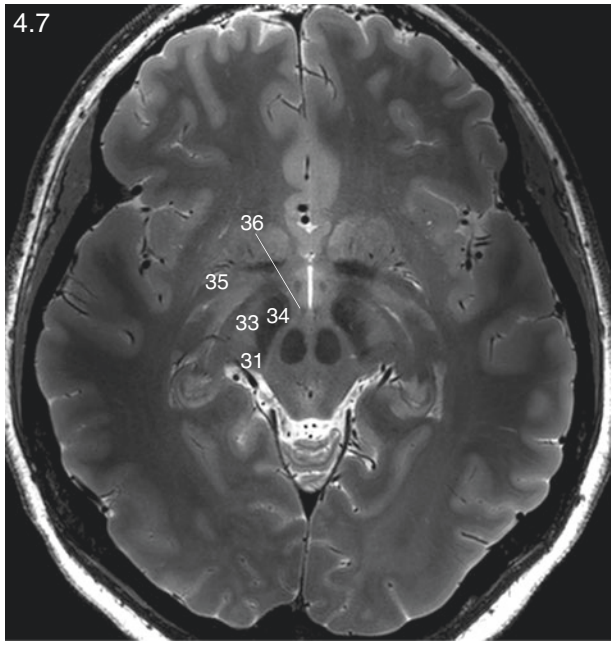
6	Column of the fornix
15a	Globus pallidus externa
15b	Globus pallidus interna
16a	Dorsomedial nucleus
16b	Ventral lateral nucleus
16c	Centromedian nucleus
16d	Ventral posterior nucleus
17	Pulvinar nucleus
20	Subcallosal gyrus
21	Interthalamic adhesion
22	Anterior commissure
23	Posterior commissure
28	Hippocampus
29	Fimbria of the hippocampus

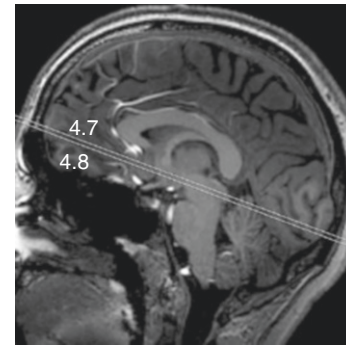




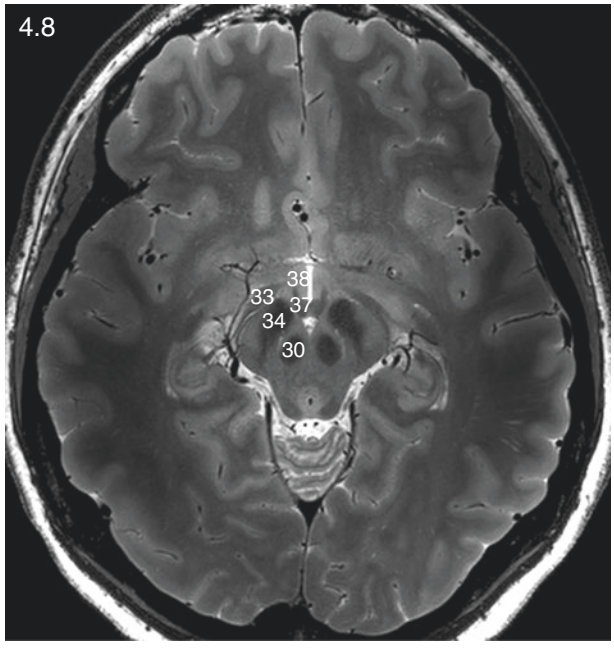


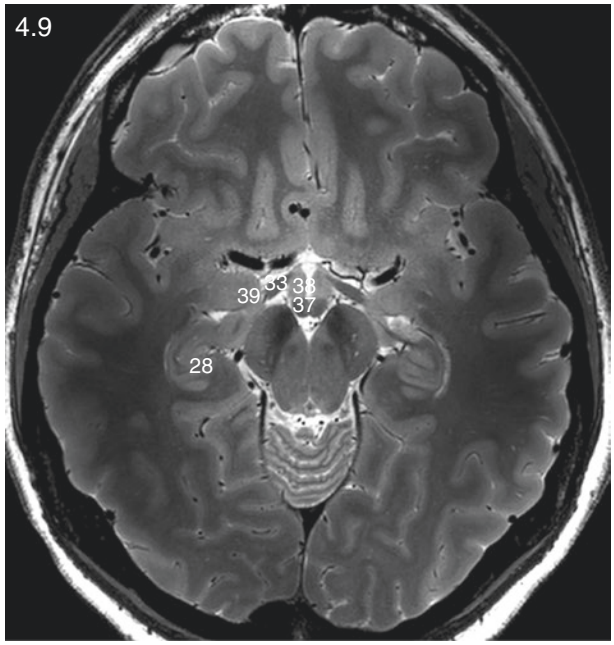
6	Column of the fornix
15a	Globus pallidus externa
15b	Globus pallidus interna
22	Anterior commissure
23	Posterior commissure
24	Subthalamic nucleus
25	Medial geniculate nucleus
26	Superior colliculus
27	Mammillothalamic fasciculus
28	Tail of the hippocampus
29	Fimbria of the hippocampus
30	Red nucleus
31	Lateral geniculate nucleus
32	Ansa lenticularis

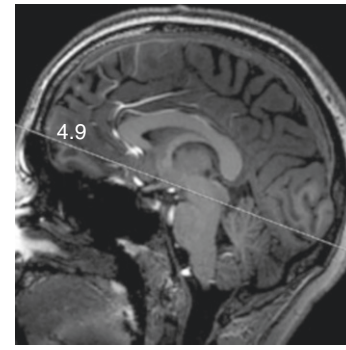




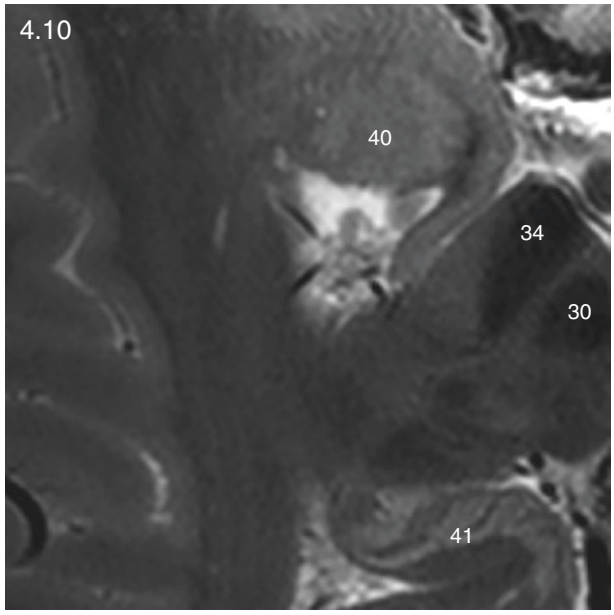
30	Red nucleus
31	Lateral geniculate nucleus
33	Optic tract
34	Substantia nigra
35	Continuation of the anterior commissure
36	Fasciculus retroflexus
37	Mammillary body
38	Hypothalamus

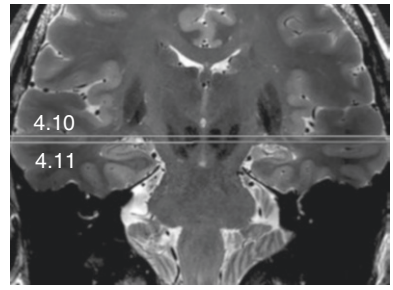




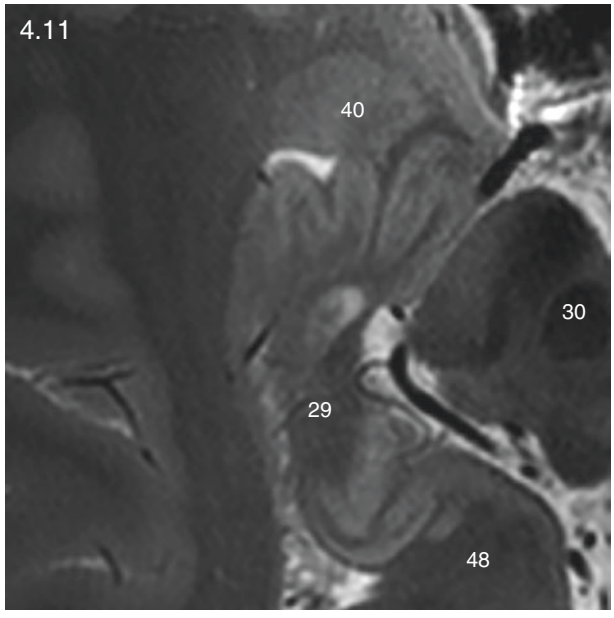


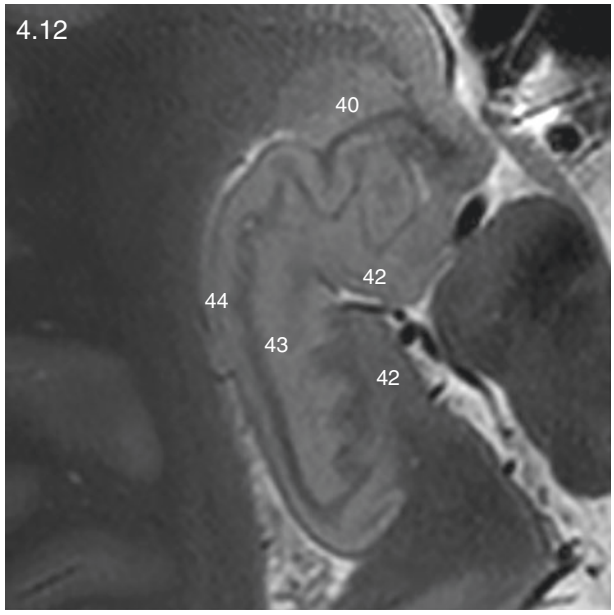
28	Hippocampus
33	Optic tract
37	Mammillary body
38	Hypothalamus
39	Uncus

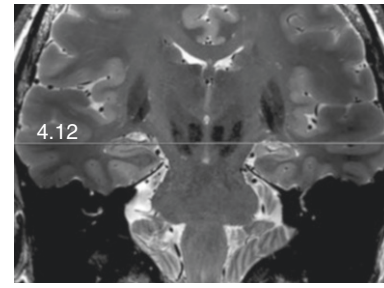




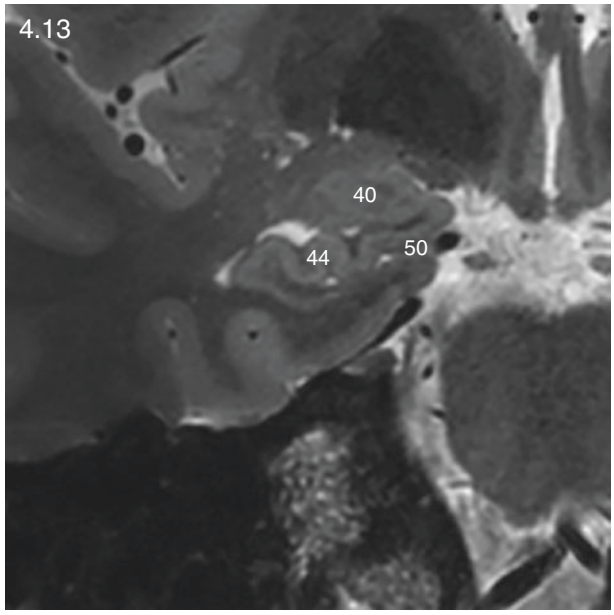
29	Fimbria of the hippocampus
30	Red nucleus
34	Substantia nigra
40	Amygdaloid body of hippocampus
41	Stria and indusium griseum
48	Internal digitations of the hippocampal head

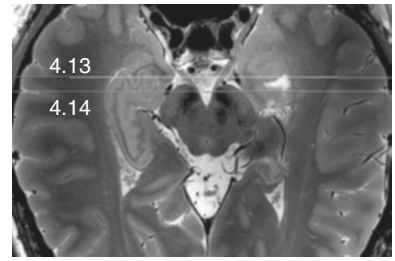




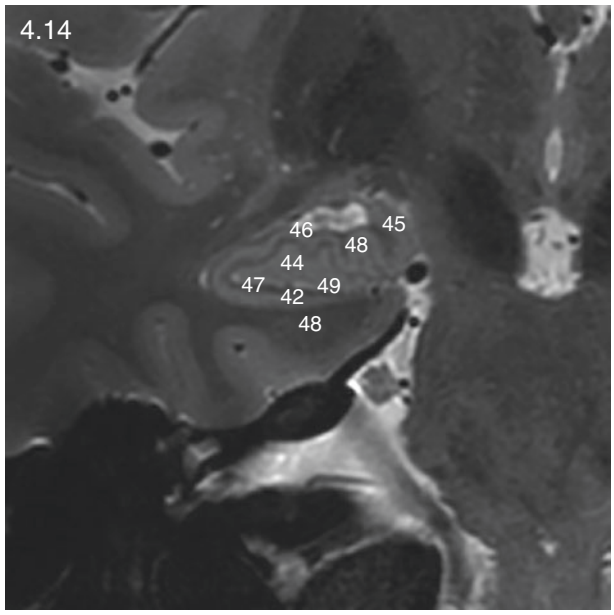


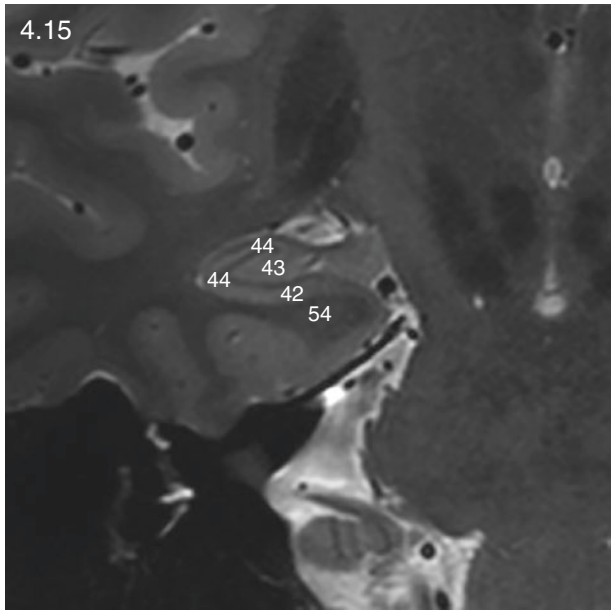
40	Amygdaloid body of hippocampus
42	Subiculum
43	Dentate gyrus
44	Ammon's horn

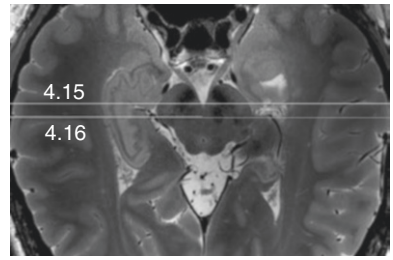




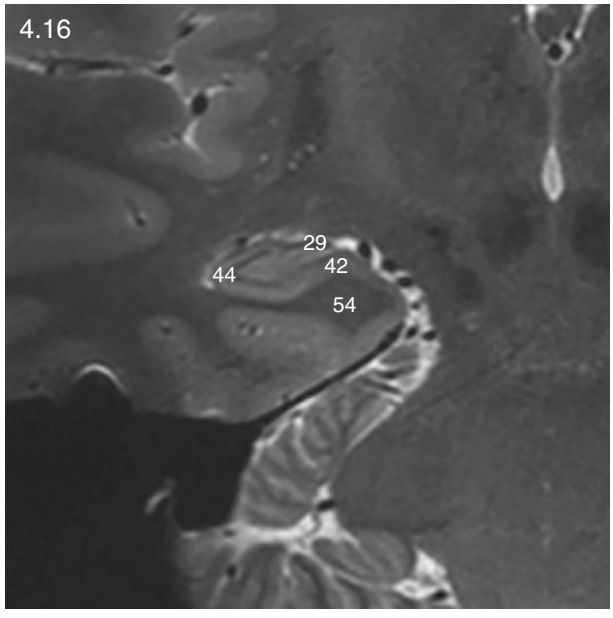
40	Amygdaloid body of hippocampus
42	Subiculum
44	Ammon's horn
45	Band of Giacomini
46	Alveus
47	Composite of strata radiatum, lacunosum, moleculare and vestigial hippocampal sulcus
48	Internal digitations of the hippocampal head
49	Hippocampal sulcus
50	Uncal sulcus

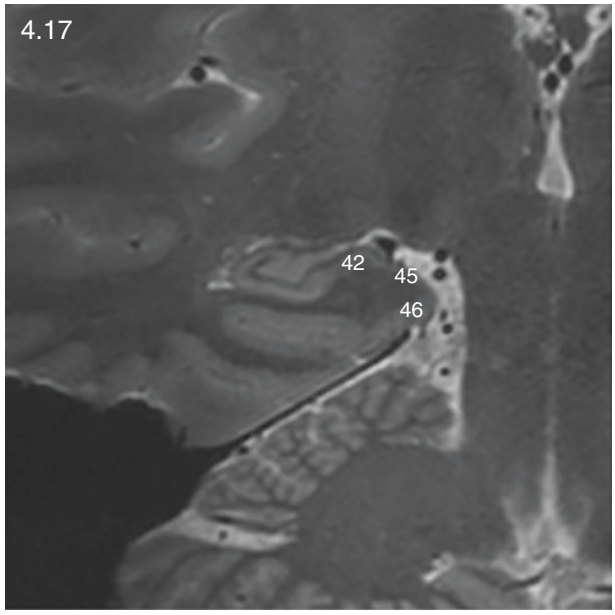


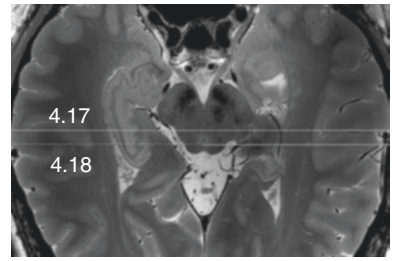




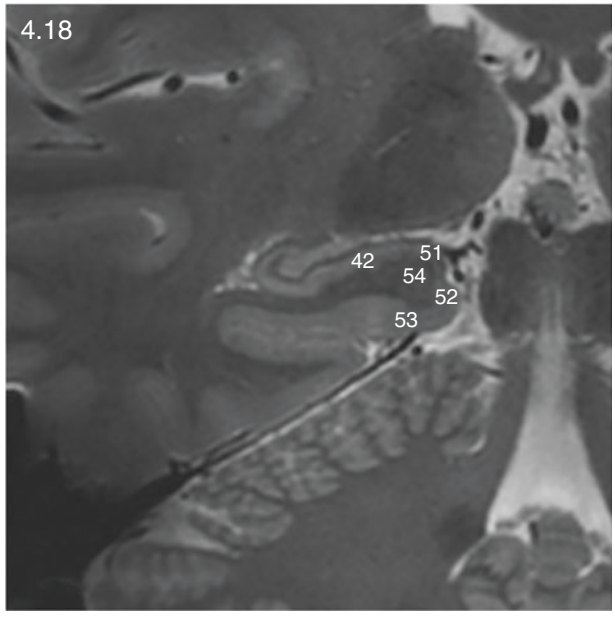
29	Fimbria of the hippocampus
42	Subiculum
43	Dentate gyrus
44	Ammon's horn
54	Parahippocampal gyrus







42	Subiculum
45	Band of Giacomini
46	Alveus
51	Presubiculum
52	Parasubiculum
53	Entorhinal cortex
54	Parahippocampal gyrus



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Functional Anatomy of the Major Lobes

5

Luisella Sibilla

In this chapter we will briefly review the functional anatomy of the major lobes and deep gray structures (cross referencing Chap. 1 with references to relevant figures). We will then describe the major functions of each area and identify specific deficits that can localize the disease in a specific part of the brain region. Where possible, important syndromes have been outlined. These descriptions are not intended to be comprehensive, but rather to present a sample of function and pathology. For more comprehensive descriptions, the reader is referred to standard texts of neurology.

Occasionally we have placed references to the different Brodmann's areas (BA) into the text. These cytoarchitecturally distinct cortical regions were originally described by Korbinian Brodmann in 1909 [1, 2]. Space limitations prohibit a full description of the Brodmann's areas, but we present them such that the reader can cross-reference this text with other texts that use these areas.

L. Sibilla, M.D.
Section of Neuroradiology, Department of Radiology, Sahlgrenska University Hospital, Gröna stråket 2 plan 2, 413 45 Gothenburg, Sweden
e-mail: luisella.sibilla@vgregion.se

5.1 Frontal Lobe

The frontal lobes are primarily involved in voluntary motor movements and higher cognitive functions. Motor functions are located in the primary, secondary, and supplementary motor areas (BA #4, 6, 8, 44, 45). Cognitive functions are located in the more anterior frontal areas known collectively as prefrontal cortex (BA #9, 10, 11, 12, 32, 44, 45, 46) [3].

5.1.1 Motor Functions

5.1.1.1 Primary Motor Cortex (BA #4)

- Precentral gyrus located between the central sulcus and the inferior and frontal sulci on the lateral aspects of frontal lobe; medially it is contiguous with the paracentral lobule.
- Paracentral lobule (anterior part) on the medial aspects of the hemisphere. It is the continuation of the precentral and postcentral gyri. The anterior portion is part of the frontal lobe and contains the supplementary motor area.

The precentral gyrus controls the execution, regulation, and coordination of movements on the opposite side of the body. The gyrus has a somatotopic organization, a "cortical motor homunculus" originally described by Penfield [4–6] that succinctly portrays the functional representation of the parts of the body on the primary motor cortex.

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Functional Anatomy of the Frontal Lobe

Major landmarks:

- Central sulcus (sulcus of Rolando; Figs. 1.5, 1.46) separates it from the parietal lobe.
- Lateral fissure (Sylvian fissure; Figs. 1.45, 1.31) which separates it from temporal lobe. It is the most important and constant of the cerebral sulci. It is made of three parts: the first separates the lateral orbital gyrus and the temporal pole, the second is horizontal segment, and the third is the segment limited anteriorly by the transverse supratemporal sulcus separating Heschl's gyri from the temporal planum and cutting into the superior temporal gyrus.
- Cingulate sulcus (Figs. 1.54, 1.35) lies medially and separates it from cingulate gyrus.

Sulci on the superior and lateral surface:

- Superior frontal sulcus (Figs. 1.4, 1.28, 1.31)
- Inferior frontal sulcus
- Precentral sulcus (Figs. 1.1–1.3, 1.44)

Sulci on the inferior surface:

- Lateral orbital sulcus
- Medial orbital sulcus
- Olfactory sulcus (Figs. 1.16, 1.26)

The gyri on the lateral side:

- Precentral gyrus (perpendicular to all the others) (Figs. 1.2–1.5, 1.57, 1.58)
- Superior frontal gyrus (Figs. 1.2, 1.27)
- Middle frontal gyrus (Figs. 1.2, 1.26)
- Inferior frontal gyrus (Figs. 1.12, 1.14)
  - Pars opercularis (BA#44)
  - Pars orbitalis
  - Pars triangularis (BA#45)

The gyri on the medial side:

- Straight gyrus (Figs. 1.16, 1.27)
- Orbital gyrus (Figs. 1.26, 1.54)
- Lateral orbital gyrus (Fig. 1.28)
- Superior rostral gyrus (Fig. 1.26)
- Inferior rostral gyrus (Fig. 1.26)

The representations of the hand and face are disproportionately large compared to the representation of the trunk and leg because of the relatively more fine movements that the upper body is able to perform.

- Arm, trunk, and hip are represented on the upper lateral surface.
- Lips, tongue, face, and hands are represented on the lower lateral surface.
- Foot, leg, and pelvic region are represented in the paracentral lobule on the medial surface.

5.1.1.2 Premotor Cortex or Secondary Motor Area (BA #6; Parts of Area #8, 44, 45)

Premotor cortex lies between the primary motor cortex and prefrontal cortex. It is the anterior part of precentral gyrus and posterior part of superior, middle, and inferior frontal gyri and extends onto the medial surface. Premotor cortex is involved in preparing and executing limb movements and coordinates with other regions to select appropriate movements. The premotor cortex is also important for learning (imitation) and social cognition (empathy), and it has been considered a site for mirror neurons [7].

5.1.1.3 Supplementary Motor Area (SMA) (BA#6)

The supplementary motor area is located primarily on the medial aspect of the superior frontal gyrus, anterior to the premotor cortex of the lower extremity, and above the cingulate sulcus. On the lateral aspect of the superior frontal gyrus, it is located superior to the premotor area. It is connected with the contralateral SMA through the corpus callosum. The SMA is involved in the planning of complex movements as well as coordinating two-handed movements.

The portion of the SMA located on the medial surface of the superior frontal gyrus in the upper part of the paracentral sulcus is called the supplementary eye field. The supplementary eye field is involved in the generation and control of eye movement together with the frontal eye field and the superior colliculus [8, 9].

5.1.2 Motor Function Pathology

The main causes of motor cortex damage are stroke (middle cerebral artery territory) and/or tumor. Primary symptoms include:

- Contralateral hemiplegia: flaccidity of the muscles on the contralateral side of the body and face; all reflex activity on the affected side is abolished. In contrast, control of trunk muscles is usually preserved.
- Damage in the premotor area causes spasticity (increased muscle tone) and ideomotor apraxia which is the inability to translate an idea into movement.

5.1.3 Cognitive Functions

5.1.3.1 Prefrontal Cortex (BA #9, 10, 11, 12, 32, 46 and Parts of 44 and 45)

Prefrontal cortex is a large multimodal association area that regulates the higher cognitive functions such as attention, working, prospective and temporal memory, planning of behavior which involve decision making and problem solving abilities, programming of movements, language, and self-control [10]. Because of its large size, prefrontal cortex can be further subdivided into lateral and medial portions, known as lateral prefrontal and orbitomedial prefrontal cortex.

Lateral prefrontal cortex is divided into dorsolateral prefrontal cortex (DLPFC) (BA #9, 46) and ventrolateral prefrontal cortex (VLPFC) (BA #47, 44, 45).

The *DLPFC* is the more dorsal component of the lateral prefrontal cortex, located in the anterior portion of the superior and middle frontal gyrus. It is connected with cortical areas that control the somatosensory and visuospatial information. Its primary functions include working memory maintenance, attention, set-shifting (change in behavior related to the rules), reward evaluation, and motor planning. Furthermore, the left DLPFC is important for elaborating verbal and spatial knowledge in working memory, while the right DLPFC is important for verbal and spatial reasoning and arithmetic reasoning.

The *VLPFC* is the more ventral component of the lateral prefrontal cortex, located in the rostral portion of the inferior frontal gyrus, laterally to the gyrus rectus, and above the medial orbitofrontal cortex. The VLPFC is delimited superiorly by the inferior frontal sulcus and inferiorly by the lateral sulcus. It is active during motor inhibition and during orienting of attention. The left VLPFC is involved cognitive control to access information from semantic memory, while the right VLPFC is important for inhibiting motor responses and reflexive reorienting to abrupt perceptual onsets.

The *frontal operculum* is a portion of the VLPFC located inferior to the DLPFC and the ventral portion of the premotor cortex. The frontal operculum is further subdivided into the precentral operculum, the opercular part (pars opercularis) of the inferior frontal gyrus, the triangular part of the inferior frontal gyrus (pars triangularis), and the orbital part of the inferior frontal gyrus. *Broca's area* (BA #44, 45) is located in the pars opercularis and pars triangularis. Broca's area is responsible for speech production, facial neuron control, and language processing. For right-handed people, Broca's area is usually located in the left hemisphere and controls the motor movements for speech production. The corresponding area in the right hemisphere is activated when people try to make sense of ambiguous emotional expression in face images and is responsible for controlling the emotional overtone of the spoken words. Both right and left areas 44 and 45 are active in the detection of errors in musical syntax. Finally, Broca's area could contain mirror neurons and have an important role in imitation [11].

Orbitomedial prefrontal cortex is divided into medial prefrontal cortex (MPFC) (BA #25, 32) and orbital prefrontal cortex (OPFC) (BA #11, 12, 13, and 14).

The *medial prefrontal cortex* is located in the anterior cingulate gyrus and in the subcallosal area on the medial surface of the frontal lobe. It plays a major role in emotional behavior and the control of basic drives [12].

The *orbital prefrontal cortex* lies on the surface of the anterior cranial fossa and forms the inferolateral surface of the frontal lobe. It has direct connections with the cerebral structures of the limbic lobe. It plays an important role in sensory integration, modulation of autonomic reactions, learning, decision making for emotional and reward-related behavior, and pleasantness of foods.

5.1.4 Cognitive Function Pathology

The main causes of prefrontal cortex damage are stroke (middle artery territory) tumor and trauma.

In general, the magnitude of cognitive defects corresponds with the size of the damaged area. Damage to the prefrontal cortex produces numerous cognitive and behavioral symptoms including distractibility, lack of foresight or insight, inability to switch from one task to another (perseveration), lack of ambition (apathy), lack of sense of responsibility, and lack of self-monitoring. These deficits can cause bizarre behaviors such as sexual disinhibition, impulsiveness, and increased gambling/risk-taking behavior. Smaller lesions cause more specific cognitive deficits.

Dysexecutive syndrome (or frontal lobe syndrome) is characterized by deficits falling into three broad categories: cognitive, emotional, and behavioral [13]. Cognitive symptoms include impairment of attention and judgment and reduction in verbal fluency. Emotional symptoms include anger, frustration, and aggressiveness as patients cannot inhibit their emotions. Behavioral symptoms include lack of motor flexibility, perseveration, and utilization behavior problems (using an object in the appropriate way but at an inappropriate time). There is not a specific pattern of damage that causes dysexecutive syndrome, as many different brain structures at different locations have led to the classic symptoms.

More focal damage to the VLPFC causes impaired performance in making decisions based on learned behavioral strategies as well as deficits in motor and syntactic processing of words. Specifically, lesions in Broca's area gives rise to nonfluent aphasia (*Broca's aphasia*), characterized by difficulty formulating sentences and speaking them aloud with good comprehension of verbal and written communication. It may or may not be accompanied by articulation disorders (dysarthria) [14].

Anterior opercular syndrome (Foix-Chavany-Marie syndrome) is also known as facio-labio-pharyngo-glosso-masticatory paralysis with automatic-voluntary dissociation. It consists of anarthria, bilateral volitional paresis of the facial, lingual, pharyngeal, and masticatory muscles with preservation of the reflexive, emotional, and automatic innervations of the same muscles. Lesions are localized in the anterior operculum and are usually bilateral [15].

Anterior cingulate cortex syndrome. Cingulate cortex is involved in the regulation of affect and in the ability to control and manage uncomfortable emotions. Lesions of the anterior cingulate cortex and the medial frontal lobe result in different degrees of spatial neglect, difficulties directing attention to discrete locations in visual space, and akinetic mutism [12].

Medial frontal apathetic syndrome occurs if a lesion is located in the medial motor cortex. This syndrome is characterized by severe impairment of motivation and interest in the environment, and reduction of motor activity [16].

Orbitofrontal disinhibition syndrome is characterized by aggressive and asocial behavior including euphoria and emotional lability, lack of decision making control, judgment impairment, and distractibility [17].

Many of the syndromes described above may occur together in a more complex manner and constellations of these findings may give rise to dementia and psychiatric disorders.

5.2 Temporal Lobe

The temporal lobes are primarily involved in auditory processing including language comprehension, higher-level cross-modal associative

functions, and learning and memory. Auditory and speech functions are localized in the superolateral in the temporal lobe, associative functions localized in the inferolateral temporal lobe, and learning and memory in the medial aspect of the temporal lobe.

5.2.1 Auditory Functions

5.2.1.1 Superior Temporal Gyrus (BA #41, 42)

The transverse gyrus, or Heschl's gyrus, is located dorsally and posteriorly in the superior temporal gyrus. It contains the primary auditory cortex which processes auditory stimuli. It has a topographical map of the cochlea and a tonotopic map which processes the speech sound and music.

5.2.2 Auditory Function Pathology

Central presbycusis, also known as central hearing loss, is very rare. Central presbycusis refers to age-related change in the auditory portions of the central nervous system that negatively affect auditory perception, communication performance, or both. It can be very difficult to differentiate central presbycusis from the more common peripheral presbycusis [18]. Central presbycusis is an important diagnostic consideration in patients presenting with tumors or vascular insults that impact the auditory portions of CNS.

Pure word deafness. Loss of auditory comprehension and preservation of visual comprehension. Speech is fluent, understanding spoken language is very difficult, while understanding writing is normal [19].

Primary progressive aphasia is associated with central hearing deafness and with some neurodegenerative disease such as frontotemporal dementia [20].

Auditory agnosia is the inability to recognize nonverbal sounds. A special type is amusia, the impossibility to process music [21].

Functional Anatomy of the Temporal Lobe

Major landmarks:

- Lateral fissure or Sylvian fissure (Figs. 1.32, 1.36, 1.41, 1.43, 1.45): the boundary with the frontal lobe is the stem of lateral sulcus, and the boundary with the parietal lobe is the posterior ramus of lateral sulcus.
- The occipitotemporal sulcus separates the medial border of the inferior temporal gyrus from the lateral border of the fusiform or inferior occipitotemporal gyrus.
- The preoccipital notch.

Sulci on the lateral surface:

- Superior temporal sulcus (Figs. 1.13, 1.18)
- Inferior temporal sulcus (Fig. 1.15)

Sulci on the inferior surface:

- Collateral sulcus (Figs. 1.19, 1.52) separates the parahippocampal gyrus and the lingual gyrus (also known as the medial occipitotemporal gyrus) from the fusiform gyrus.
- Occipitotemporal sulcus separates the inferior temporal gyrus from the fusiform gyrus (also known as the medial occipitotemporal gyrus).
- The inferior temporal sulcus (Fig. 1.12) separates the middle and the inferior temporal gyri.

Gyri on the lateral aspect:

- Superior temporal gyrus (BA41, BA42, BA22) (Figs. 1.12, 1.13, 1.28)
- Middle temporal gyrus (BA21) (Figs. 1.15, 1.29)
- Inferior temporal gyrus (BA20) (Figs. 1.16, 1.30)

Gyri on the basal surface:

- Parahippocampal gyrus (Figs. 1.18, 1.19)
- Fusiform gyrus (BA37) (Figs. 1.36, 1.50)
- Inferior temporal gyrus (BA20) (Figs. 1.16, 1.17, 1.36)

The *mesial temporal region* is characterized by two major gyri and two major sulci.

- Gyri:
  - Uncus (Figs. 1.31, 1.52): It includes part of the amygdala, the hippocampus, and the piriform cortex. It is the most anterior part of the parahippocampal gyrus, and it is delimited dorsally by the uncal sulcus, medially by the optic tract. The posterior part contains the head of the hippocampus.
  - Parahippocampal gyrus: The anterior border is the perirhinal and the entorhinal cortex, and the posterior boundary is the calcarine sulcus. The collateral sulcus separates it from the fusiform gyrus.
- Sulci:
  - The hippocampal fissure, located superior to the parahippocampal gyrus
  - The collateral sulcus

Temporal pole (BA38) (Fig. 1.21)

5.2.3 Speech Functions

5.2.3.1 Superior Temporal Gyrus (BA #22, Parts of BA #39, 40)

Wernicke's area is located dorsally in the superior temporal gyrus surrounding auditory cortex. Wernicke's area is responsible for the comprehension of speech. In general Wernicke's area is located in the left superior temporal gyrus for right-handed people, and in the right superior temporal gyrus for left-handed people, but there is considerable individual variability. It is connected to Broca's area through the arcuate fasciculus. Wernicke's area is involved in language comprehension, semantic processing, language recognition, and language interpretation.

5.2.4 Speech Function Pathology

Wernicke's aphasia is also called fluent aphasia or receptive aphasia. The patients can produce many words with grammatically correct sentences and normal prosody, but what they say doesn't make a lot of sense. They also have an associated speech comprehension deficit [14].

Prosopagnosia is also known as face blindness. It is a deficit in recognizing familiar faces. It can be acquired following stroke or head trauma and congenital. It is described also in autism spectrum disorder.

Synesthesia is a condition in which a stimulus is perceived simultaneously by more senses. There are different types, but the most frequent is the grapheme-color synesthesia. It consists in the perception of a particular color when seeing letters or numbers [22].

Dyslexia is a difficulty in word reading and fluency.

Charles Bonnet syndrome is a common condition characterized by visual hallucinations in people with impaired vision [23].

5.2.5 Higher-Level Associative Functions

5.2.5.1 Middle Temporal Gyrus (BA #21)

It is located between the superior and the inferior temporal gyrus. Its dorsal boundary is with the angular gyrus and the occipital lobe. The middle temporal gyrus is involved in higher-level cognitive and language processes such as ascertaining distance, recognizing known faces, and comprehending the meaning of words while reading.

5.2.5.2 Inferior Temporal Gyrus (BA #20, 37)

It lies under the middle temporal gyrus and posteriorly is bounded by the inferior occipital lobe. The inferior temporal gyrus is involved in higher-level associative cognitive functions such as semantic memory, language, visual perception, and sensory integration.

5.2.5.3 Fusiform Gyrus (BA #37)

Also known as occipitotemporal gyrus, the fusiform gyrus is located between the lingual gyrus and the parahippocampal gyrus. The posterior end is located in the occipital lobe. It is involved in the

higher visuo-auditory processing functions such as facial recognition, color processing, word and number recognition, and category processing.

5.2.5.4 Temporal Pole (BA #38)

The temporal pole occupies the most rostral part of the temporal lobe. It is the point where the superior, middle, and inferior temporal gyri meet. Ventromedially it blends with the perirhinal area. It has a dorsal, lateral, and mesial surface. The temporal pole is involved in social and sexual behavior as well as cognitive visual functions. It plays a role in autobiographical memory, face and visual pattern recognition, mnemonic matching and learning tasks, linguistic integration, and the processing of emotional language. It is a place for the representation of unique entities such as proper names of peoples and places [23]. The left anterior temporal pole is the area responsible for mapping meaning onto sound, determined from tasks such as object naming [24, 25].

5.2.6 Higher-Level Associative Function Pathology

Damage to the association areas can result in different and specific symptoms that are part of a great variety of diseases, such as stroke, trauma, encephalitis, and some types of dementia.

- Agnosia: inability to access the semantic knowledge of an object.
- Prosopagnosia: inability to recognize familiar faces if a lesion occurs in the fusiform gyrus.
- Visual agnosia: inability to access semantic information by sights.
- Associative agnosia: inability to name objects that can be perceived and drawn in case of an anterior left temporal lobe lesion.
- Aperceptive agnosia: inability to recognize by sights known objects that can be described if bilateral lesions occur in the occipitotemporal associative areas.

Alexia is the inability to understand written language. It can be pure, without agraphia if a lesion occurs in the occipital lobe and splenium of the corpus callosum.

Achromatopsia is the loss of ability to perceive colors and results from a lesion in the medial occipitotemporal region, and in the fusiform gyrus.

Apraxia is the inability to perform skilled movements. It can be:

- Ideational if a damage occurs to the conceptual system, with deficit in gesture comprehension and production
- Ideomotor with impairment to product hand gestures and to imitate the use of tools
  - Left parietal or prefrontal lesions can cause dyspraxia.
  - Insular and left inferior frontal lesions can cause the orobuccal apraxia.
  - Another form of apraxia is the inability to intentionally move arms and legs.
  - Apraxia of the speech is the inability to perform the movements necessary to produce speech.

5.2.7 Memory and Emotion Functions

The medial surface of the temporal lobe comprises the majority of the *limbic lobe* [26]. It is part of the limbic system, also named the "emotional system," a network of cortical and subcortical structures that include, besides the limbic lobe, the cingulate gyrus, amygdala, anterior thalamic nuclei, and olfactory cortex. The limbic lobe consists of two "layers" of brain tissue surrounding the corpus callosum, one inside another. Broca [27] named the outer one the limbic gyrus and the inner one the intralimbic gyrus.

The limbic gyrus includes:

- The parahippocampal gyrus (BA #27, 28, 35, 36, 38) including the entorhinal cortex (BA# 28)
- The cingulate gyrus (BA #23, 24, 25, 26, 29–33)
- The subcallosal area (BA #24, 25, 32)

The intralimbic gyrus includes:

- The hippocampus (horn of Ammon)
- The dentate gyrus
- The supracallosal gyrus (indusium griseum)

The limbic lobe supports low- and high-level functions such as emotion, behavior, short-term memory, and olfaction [28]. It is part of the "circuit of Papez," the neural circuitry thought to serve as the basis of emotion, the oldest part which is firmly rooted in the sense of smell arising from the rhinencephalon.

The medial surface of the temporal lobe proper contains the *hippocampal formation* [29], consisting of the hippocampus, the dentate gyrus, the entorhinal area, the subiculum, the fasciolar gyrus, and the indusium griseum.

The *amygdala* is a group of several nuclei located in the medial part of the temporal pole, anterior to and partly overlapping the hippocampal head, and within the uncus. It lies dorsally to the hippocampal formation and rostrally to the temporal horn of the lateral ventricle. It is caudal to the claustrum, and it is separated from the putamen and the pallidus by fibers of the capsula externa. The amygdala is divided into three large subnuclei: the basolateral, corticomedial, and central groups.

The *uncus* is a brain landmark that includes part of the amygdala, the hippocampus, and the piriform cortex. It is the most anterior part of the parahippocampal gyrus, and it is delimited dorsally by the uncal sulcus and medially by the optic tract. The posterior part contains the head of the hippocampus.

The limbic lobe is involved in functions related to the emotional aspects of memory and recalling:

- The *hippocampus* is involved in long-term memory. Specifically, it is critical for the formation of new long-term memories and is involved in the voluntary recall of past knowledge and experiences.
- The *dentate gyrus* is involved in spatial memory, stress, and depression management and spatial behavior.
- The *parahippocampal gyrus* is involved in spatial memory processes.
- The *entorhinal cortex* plays a role in processing olfactory sensation and olfactory memory. It mediates learning and memory, particularly conscious memory.
- The *amygdala* plays a role in coordinating behavioral response to environmental stimuli, especially responses with emotional content. It is associated with emotional memory, visual recognition of emotionally relevant events, motivation, autonomic responses and hormonal secretions related to emotions, and fear. The left amygdala is involved in recognizing faces that express fear.
- The *uncus* is functionally divided into anterior and posterior parts. The anterior part is associated with the amygdala and its function, while the posterior part is involved in processing scenes and objects, and in autobiographical memory [30].
- The *anterior cingulate cortex* and *subcallosal area* are involved in affect regulation with connections to both the limbic system and the prefrontal cortex.

5.2.8 Memory and Emotion Function Pathology

Epilepsy is commonly caused by hippocampal sclerosis. When hippocampal sclerosis also involves the amygdala and the parahippocampal gyrus, it is named *mesial temporal sclerosis* [31].

Limbic encephalitis is a paraneoplastic autoimmune disease that can involve the hippocampus, amygdala, cingulated gyrus, insula, and orbitofrontal cortex. Symptoms are headache, irritability, mental confusion, memory impairment, personality changes, and sleep disturbances.

In diseases that have *dementia* as a first symptom, such as Alzheimer's disease, frontotemporal dementia, etc., the hippocampus is the first region affected.

Kluver-Bucy syndrome is extremely rare in humans. It occurs with bilateral lesions of the amygdala and is characterized by visual agnosia, placidity, bulimia, hypersexuality, hyperorality, and memory impairment. Kluver-Bucy can occur with head trauma, Alzheimer's disease, Pick's disease, and following herpes encephalitis as part of more wide behavioral symptoms related to bilateral temporal lobe pathology [32].

5.3 The Parietal Lobe

The parietal lobes can be divided into three functional areas. The anterior area includes primary somatosensory cortex and the parietal operculum—regions that are primarily involved in sensation and perception. The superior parietal cortex is responsible for integrating visual and sensory information to generate the perception of self, and the inferior parietal cortex is involved in speech comprehension.

5.3.1 Sensory Functions

5.3.1.1 Postcentral Gyrus (BA #1, 2, 3)

The postcentral gyrus lies dorsal to the central sulcus, bounded caudally by the Sylvian fissure and posteriorly by the inferior and superior postcentral sulcus. This region functions as *primary somatosensory cortex* (a.k.a. S1), receiving and processing sensations from the body obtained through touch and proprioceptive stimuli. Specifically, discriminative touch includes the sensations of touch, pressure, and vibration, while proprioceptive stimuli include sensations of joint positional movement. Primary somatosensory cortex also has a role also in monitoring body temperature and has information about pain, itch, and tickling stimuli.

Functional Anatomy of the Parietal Lobe

Major landmarks:

- Central sulcus (a.k.a. sulcus of Rolando; Figs. 1.1–1.8) anteriorly separates it from frontal lobe.
- Lateral fissure (a.k.a. Sylvian fissure; Figs. 1.32, 1.36, 1.41, 1.43, 1.45), at the inferior aspect, which separates it from temporal lobe.
- Parieto-occipital sulcus (Fig. 1.57) and posterior ramus of the Sylvian fissure posteriorly, which separates it from occipital lobe.

Sulci:

- Postcentral sulcus (Figs. 1.45, 1.48) separates the supramarginal gyrus from the postcentral gyrus.
- Cingulate sulcus (Fig. 1.55) which separates the paracentral lobule and the precuneus.
- Subparietal sulcus separates the precuneus and the cingulate gyrus.
- Calcarine fissure (Figs. 1.39, 1.57, 1.58) between the striate and lingual gyri and the striate area and the occipital gyri.

Gyri on the lateral aspects:

- The postcentral gyrus (BA1, 2, 3) (Figs. 1.33–1.37, 1.57)
- The superior parietal lobule (BA5, 7)
- The inferior parietal lobule:
  - Supramarginal gyrus (BA40) (Figs. 1.7–1.9, 1.34, 1.35)
  - Angular gyrus (Wernicke's area) (BA39) (Figs. 1.9. 1.10, 1.38, 1.39)
- Parietal operculum (BA43) (1.11, 1.47)

Gyri on the medial surface:

- The paracentral lobule (Figs. 1.5, 1.38)
- The precuneus (Figs. 1.6, 1.37)

Somatosensory cortex is organized somatotopically like the primary motor cortex; specifically, there is a strict relation between the location of neurons in the postcentral gyrus and the position of the receptive area in the skin. The body's representation (homunculus) on primary somatosensory cortex is distorted, with the most sensitive parts of our bodies (e.g., fingers, lips, genitalia) covering a larger amount of somatosensory cortex than the least sensitive parts (e.g., back of our necks, lower back). The distortion is related to discriminative properties which are more prominent in some territories than others.

The parietal operculum is part of the frontoparietal operculum formed by the inferior parts of the precentral and postcentral gyri as well as the anterior inferior part of the inferior parietal lobe. It is the so-called *secondary somatosensory*

cortex (SII) and is characterized by a homunculus with the head represented anteriorly, the leg muscles posteriorly, the back inferiorly, and the hands and feet superiorly. SII is involved in the perception of touch, pain, and temperature. The parietal operculum also contains BA #43 located just inferior to precentral gyrus (BA #1, 2, 3). BA #43 is known as *primary gustatory cortex*.

5.3.2 Sensory Function Pathology

Lesions in the postcentral gyrus are associated with changes in the somatosensory thresholds, impairment of the position sense, and deficits in stereognosis (tactile perception). Lesions in the parietal lobe, secondary to stroke, TIA, trauma, tumors, inflammatory/demyelinating disorders, can cause sensory deficits such as sensory loss in half of the body, or various sensations of numbness, tingling, and prickling.

Parietal lobe epilepsy is sometimes difficult to diagnose. It presents contralateral symptoms that may include painful dysesthesias, vertigo, aphasia, tingling and numbness, and pain that can spread in a Jacksonian manner.

Posterior operculum syndrome occurs due to occlusion of the posterior branch of the central sulcus artery with infarction of the parietal operculum. It is characterized of isolated facial-oral sensory loss [33].

5.3.3 Association/Integration Functions

The *superior parietal lobule* (BA #5, 7) is bounded by the upper part of the postcentral sulcus ventrally, the intraparietal sulcus laterally and caudally, and the parieto-occipital sulcus dorsally. It contains the somatosensory association cortex that lies directly dorsal to the primary somatosensory cortex.

The superior parietal lobule performs the sensory integration necessary to generate spatial awareness, including information about somesthesis (tactile perception), visuospatial and attentional processing, perception of position, working memory, and long-term memory.

5.3.4 Association/Integration Function Pathology

Ideomotor apraxia (left superior parietal lobe lesion) is the inability to manipulate and use common objects and to translate an idea into motion.

Astereognosia is the inability to identify objects by touch.

Impairment of sense of position (nondominant hemisphere superior parietal lobe lesions) is the inability to determine the body position in space.

Unilateral neglect is the inability to process and perceive stimuli on the one side of the body without sensory loss [34].

5.3.5 Speech and Cognitive Functions

The *inferior parietal lobule* lies between the lateral fissure inferiorly and the horizontal segment of the intraparietal sulcus superiorly. The anterior boundary is the intraparietal sulcus which separates it from the superior parietal lobule. It is divided into the *supramarginal gyrus* which is located dorsal to the postcentral gyrus and separated posteriorly from the superior parietal lobe by the intraparietal sulcus, and the *angular gyrus*, separated from the supramarginal gyrus by the primary intermediate sulcus. Caudally the angular gyrus is separated from the occipital lobe by the parieto-occipital sulcus.

The *supramarginal gyrus* is involved in language perception and processing, processing of the phonological aspects of words, and speech motor planning.

The *angular gyrus* is involved with the visual guidance of hand and limb movements, semantic processes, recognition of visual symbols,

mathematic and spatial cognition, and attention. The angular gyrus translates written words into a form accessible by Wernicke's area. The angular gyrus is also thought to be involved with the theory of the mind, such as the ability to infer and reason about another person's state of mind [35, 36].

The *precuneus* is bounded anteriorly by the marginal ramus of the cingulate sulcus, posteriorly by the parieto-occipital sulcus, and caudally by the subparietal sulcus. The precuneus is involved in episodic memory, visuospatial processing, self-awareness and introspection, maintaining a state of consciousness, and theory of the mind [37].

5.3.6 Speech and Cognitive Function Pathology

The supramarginal gyrus of the left hemisphere supports semantic processes and verbal creativity. Damage causes alexia, agraphia, and anomia.

Angular gyrus syndrome/Gerstmann's syndrome is associated with lesions in the dominant hemisphere characterized by dyscalculia/acalculia, right-left confusion, dysgraphia, and finger agnosia [38].

Damage to the precuneus has been associated with psychiatric symptoms such as anorexia, borderline personality disorder, dissociative identity disorders, and dissociative amnesia.

5.3.7 Motor Functions and Motor Function Pathology

The *paracentral lobule* is limited anteriorly by the paracentral sulcus and posteriorly by the marginal sulcus, a ramus of the cingulate sulcus. It plays a role in motor and sensory innervations of the contralateral lower extremity, and regulation of physiological function such as defecation and micturition. Damage to the paracentral lobule is associated with contralateral lower limb weakness and urinary incontinence.

5.4 Occipital Lobe

Functional Anatomy of the Occipital Lobe

Major landmarks:

- Parieto-occipital sulcus (Fig. 1.57), located on the medial surface, separates the occipital lobe from parietal lobe.
- On the lateral surface, an imaginary line divides the occipital lobe from the temporal lobe extending from the preoccipital notch to parieto-occipital sulcus.
- Collateral sulcus (Fig. 1.52) which separates it from the limbic lobe.

Sulci:

- Transverse occipital sulcus (Fig. 1.38).
- Lateral or inferior occipital sulcus. It divides the lateral aspect of the occipital lobe in two parts horizontally.
- Intra-occipital sulcus (Figs. 1.10–1.12), divided in superior occipital sulcus and inferior occipital sulcus.
- Calcarine fissure (Figs. 1.39, 1.57, 1.58).

The gyri on the superolateral aspect:

- Superior occipital gyrus
- Middle occipital gyrus (Figs. 1.14–1.16)
- Inferior occipital gyrus (Figs. 1.18–1.20)

The gyri on the medial aspect:

- Striate gyrus (Figs. 1.56–1.58)
- Lingual gyrus (Figs. 1.37, 1.56–1.58)

The gyri on the basal aspect:

- Fusiform gyrus (Figs. 1.36, 1.50)
- Inferior occipital gyrus (Figs. 1.18–1.20)

The occipital lobes are involved in the representation and higher-order processing of visual information, including abstraction of orientation, motion, color, and depth information from visual stimuli. The sulci and gyri of the occipital lobe have great anatomical variation [39–42].

5.4.1 Primary Visual Cortex (BA #17)

Primary visual cortex (V1) lies on the banks of the calcarine sulcus in the cuneus and lingual gyrus. It receives inputs from the lateral geniculate body of the thalamus. V1 is also called the striate cortex because of the very peculiar "stria-like" myelin staining of cortical layers (also termed stria of Gennari). The extrastriate areas consist of visual areas V2, V3, V4 and V5.

Similar to the sensory homunculus, V1 has a perfect retinotopic map (representation) of the entire visual field from the fovea through the periphery. Visual input from the fovea is represented on the occipital pole, while information from the more peripheral visual fields is represented more anteriorly along the calcarine fissure. Microscopically, V1 is comprised of a grid of hypercolumns. Each hypercolumn analyzes information from a small area of the retina, where adjacent hypercolumns analyze information from adjacent areas of the retina. Each hypercolumn abstracts visual information about stereopsis (the visual sense of depth), color recognition, and the orientations of line segments [43].

5.4.2 Associative Visual Cortex (BA #18, 19)

Associative visual cortex is comprised of the superior part of the cuneus, the inferior part of the lingual gyrus, the lateral occipital gyrus, and the superior occipital gyrus of the occipital lobe.

The *cuneus* lies above calcarine fissure on the medial surface of the occipital lobe. It is bounded anteriorly by the parieto-occipital sulcus and often continues onto the posterior pole.

The *lingual gyrus* lies below the calcarine fissure on the medial surface of the occipital lobe between the calcarine sulcus and the posterior part of the collateral sulcus. It continues on the occipital pole posteriorly and anteriorly it joins the parahippocampal gyrus.

In association visual cortex, visual information from V1 divides into two separate visual pathways:

- The ventral stream or the "what" pathway: this pathway links the occipital lobe to the temporal lobe by the inferior longitudinal fasciculus (ILF). The ventral stream carries out information about perceptual features, allowing the creation of long-term representations necessary to identify and recognize objects and form.
- The dorsal stream or the "where" pathway: this pathway connects V1 to the temporal lobe via the inferior longitudinal fasciculus and allows for recognition of objects in the space. The dorsal stream processes information about objects and their locations in a moment-to-moment way and mediates the visual control of skilled actions.

Association visual cortex functions include detecting the intensity of light, color perception, visual pattern detection (distance, depth, size, number), object recognition (shape, orientation), word and face encoding, and encoding visual-spatial information. It helps to assign meaning to what a person is seeing. Association visual cortex may also play a role in generating or recalling dreams [44].

5.4.3 Occipital Lobe Pathology

Damage to V1 due to stroke, trauma, or tumor can cause *cortical blindness.*

Anton's syndrome (visual anosognosia) is a condition in which patients deny their loss of vision.

Damage to V2–V5 can cause a variety of visual deficits. The *visual agnosias* include the loss of the ability to recognize and identify familiar objects. Patients cannot understand the meaning of the previous known objects by sight despite normal visual perception and alertness. They cannot properly process what they see, they are not able to recognize pictures of the objects, and are unable to copy an object, e.g., draw a picture. If higher-level associative areas are also damaged, then patients cannot recall the functions of the objects when they see them.

5.5 Basal Ganglia

The term basal ganglia formally refer to the caudate nuclei, nuclei accumbens, globus pallidi, and putamina together with the amygdalae and the claustra [45]. As defined, the basal ganglia arise from the telencephalon during development. However, from a functional point of view, the substantia nigra and the subthalamic nuclei are also considered part of the basal ganglia, even though the subthalamic nuclei originate from the prosencephalon (part of the diencephalon) and the substantia nigra and ventral tegmentum develop from mesencephalon.

The basal ganglia are important for motor function (especially the storage and replay of complex motor programs such as driving) as well as cognitive and emotional functions.

Functional Anatomy of the Basal Ganglia

- Corpus striatum
  - Dorsal striatum: caudate and putamen (Figs. 1.12, 1.30)
  - Ventral striatum: nucleus accumbens (Fig. 1.54)
- Globus pallidus (Figs. 1.15, 1.32)
- Claustrum (Figs. 1.14, 1.33)
- Subthalamic nucleus (Figs. 1.34)
- Substantia nigra (Figs. 1.34)
- Amygdala: see temporal lobe

The *caudate nucleus* is closely associated with the lateral wall of the lateral ventricle. It has a C-shape with a large portion anteriorly named caput (head) that bulges into the frontal horn; dorsally it narrows and give rise to a body that terminates with a tail at the amygdaloid nucleus region.

The *putamen* is a large gray matter structure separated from the caudate nucleus by the anterior limb of the internal capsule. Fine striations however link the putamen to the head of the caudate nucleus hence the term corpus striatum. It lies laterally of globus pallidus and medially of external capsule. Anteroposterior and superior lays the corona radiata.

The *nucleus accumbens* is part of the ventral striatum. It is a symmetric structure, parallel to the midline, lying anteriorly to the anterior commissure, caudal to the anterior limb of internal capsule, and medial to the claustrum and piriform cortex.

The *globus pallidus* lies medial to the putamen and is subdivided into two parts: a lateral external part also called the pars externa and a more medial part called the pars interna. The globus pallidus is separated from the putamen by the external medullary lamina.

The *claustrum* is a linear thin gray matter band lying between the external and extreme capsules. The dorsal portion lies superior to the collateral sulcus and is caudal to the insular cortex; the ventral portion continues below the collateral sulcus caudally to the olfactory region.

The *subthalamic nucleus* is located under the thalamus on the medial aspect of the internal capsule; it is superior and anterolateral to the red nucleus and dorsal to the substantia nigra.

The *substantia nigra* is located in the midbrain between the tegmentum and the crus cerebri, lateral to the red nuclei, and medial to the crus cerebri. It is divided into pars reticularis, or SNR, superficial with striatonigral fibers, and pars compacta, or SNC, deeply containing melanin pigments.

The *amygdala* is described in the paragraph on the temporal lobe.

5.5.1 Basal Ganglia Connections

The basal ganglia exert their influence on thalamocortical connections via two different pathways: the direct and indirect pathways.

The *direct pathway* excites the corpus striatum (caudate, putamen, nucleus accumbens) through glutamatergic cortico-striatal pathways that cause inhibition of the globus pallidus. The globus pallidus is an inhibitory nucleus that

causes inhibition of the thalamocortical excitatory pathway. Thus inhibition of an inhibitory nucleus leads to disinhibition, thereby releasing the thalamus and allowing for excitation of the cortex. This leads to movement.

The *indirect pathway* inhibits movement through the excitation of the subthalamic nuclei that causes excitation of the globus pallidus. Thus globus pallidus now actively inhibits the thalamocortical pathway leading to no movement.

It is important to realize that similar pathways exist for cognitive behavior [46].

For actual movement to occur, the substantia nigra projects to the striatum and provides dopaminergic tonic and phasic stimulation through specific D1 and D2 receptors on the cells of the neostriatum. This striatonigral pathway is crucial in the facilitation of movement.

The basal ganglia also provide for important dense projections to the several brainstem nuclei including the superior colliculus and oculomotor nuclei to allow for coordinated eye movement.

The claustrum has a widespread connectivity with cortex in a pattern as widespread as that of the thalamus. Functionally it is divided into three parts, the anterior-dorsal compartment connected with the somatosensory and motor cortices, a posterior dorsal connected with visual cortex, and a ventral area connected with auditory cortex [47].

The claustrum has connections with frontal, premotor, ventral anterior cingulate, ventral temporal, visual, motor, somatosensory, and olfactory cortices, but most strongly with the entorhinal cortex. It also has connections with some subcortical structures including the putamen, globus pallidus, and lateral amygdala. It integrates information received from sensory and motor cortices. It plays a role in processing information by correlating the separate activity in the different sensory cortex [48].

A large spectrum of disease with cognitive disturbances, such as dementia with Lewy bodies and Parkinson-dementia complex, involve degeneration of claustrum. Other diseases include schizophrenia, bipolar disorders, depression, and substance abuse [49].

5.5.2 Functions of the Basal Ganglia

Motor functions of the basal ganglia include preparing for movement, the proper initiation of movement, the control of movement including automatic execution of a learned motor plan, and modulating eye movements to coordinate gaze [50].

Cognitive and emotion functions of the basal ganglia include retrieval of episodic and semantic input for explicit memory related to the preparation for a movement, implicit learning of automated responses, and mood regulation related to the dopamine system.

The *corpus striatum* (caudate, putamen, nucleus accumbens) receives input from all cortical areas and projects via thalamus to frontal areas (premotor, prefrontal, and supplementary motor areas) that are involved in motor planning. Motor functions include regulating cortex in response to automatic and voluntary motor information, predicting events, and focusing attention during processes of movement initiation. Right side lesions of putamen are involved in the spatial neglect. Cognitive functions include predicting and potentiating the right-social behavior in a given situation and mediating motivational and emotional processes (nucleus accumbens).

The *subthalamic nucleus* is the main excitatory regulator of motor function related to the basal ganglia. It is involved in the cortico-striato-thalamocortical motor loop [51], exerting an excitatory influence on the globus pallidus. Specific lesions can cause increased involuntary movement. This nucleus is an important site for deep brain stimulation for in the treatment of tremor [52]. Furthermore, the subthalamic nucleus mediates limbic function, implicated in impulse control, self-awareness, and introspective perception of one's own consciousness [53].

The *substantia nigra* (black substance) was named due to high levels of neuromelanin in dopaminergic neurons. Its main functions are

mediated through the striatum. The pars compacta is mainly involved in the motor control, including eye movements, motor planning, reward-seeking, and learning.

5.5.3 Pathology Resulting from Basal Ganglia Lesions

Parkinson's disease is a chronic and progressive movement disorder characterized by tremor, bradykinesia, rigidity, and postural instability.

Huntington's disease is an inherited disease that causes movement, cognitive and psychiatric disorders such as chorea and dystonia, slow or abnormal eye movements, motor incoordination), cognitive difficulties, and emotional difficulties, including depression, apathy, and irritability.

Tourette syndrome is an inherited neuropsychiatric disease characterized by tics, repetitive and involuntary movements, and vocalizations.

Obsessive-compulsive disorder is a chronic condition characterized by recurrent intrusive thoughts and ritualistic behaviors.

Hemiballism is an extremely rare condition related to lesions in the subthalamic nucleus. It is characterized by sudden, large involuntary movements of the proximal limb contralateral to the side of the lesion. The symptoms disappear during sleep and worsen with emotional distress.

5.6 Thalamus

The thalami are two large ovoid masses of gray matter located lateral to third ventricle. Embryologically the thalamus develops from the diencephalon. Besides the thalamus, other related structures arising from the diencephalon include the epithalamus, hypothalamus, and the subthalamus.

The thalamus bounded anteriorly by the lamina terminalis, caudally by the midbrain at level of a plane including the posterior commissure and the mammillary bodies, and laterally by the posterior limb of the internal capsule. Medially the thalami are lined by ependyma and form the lateral wall of the third ventricle. They are connected to each other through the interthalamic adhesion. Superiorly they are related to the fornix, stria terminalis, and nucleus caudatus, and inferiorly lies the hypothalamus ventrally and the subthalamus dorsally.

The thalamus itself is divided by a thin strip of myelinated fibers, the *internal medullary lamina* that runs along the anterior-posterior axis of the structure. The internal medullary lamina splits into two branches that delimit the anterior nucleus. From the anterior nucleus progressing posteriorly, the internal medullary lamina defines:

Medially
- The medial dorsal nucleus (MD)

Laterally, cranially
- The lateral dorsal nucleus (LD)
- The lateral posterior nucleus (LP)

Laterally caudally
- The ventral anterior nucleus (VA)
- The ventral lateral nucleus (VL)
- The ventral intermedial nucleus (VI)
- The ventral posterolateral nucleus (VPL)
- The ventral posteromedial nucleus (VPM)

In the midline
- The intralaminar nuclei
- The centromedian nucleus (CM)

Posteriorly
- The pulvinar nucleus (P)

Finally, caudal to the pulvinar nucleus can be found the lateral and medial geniculate bodies.

5.6.1 Functions of the Thalamic Nuclei

Functionally the thalamic nuclei can be divided into relay nuclei, association nuclei, and nonspecific nuclei. Relay nuclei receive well-defined impulses and project to the cortex. Association nuclei are interconnected with the other diencephalic nuclei and project to the association cortical areas. They correspond to the "high-order" relays, playing an important role in thalamocortical and the cortico-cortical relationships.

Motor relay nuclei

Substantia nigra, pars reticulata	Ventral anterior nucleus (VA)	Area #8	Initiation and planning of movements
Globus pallidum	Ventral anterior nucleus (VA)	Area #6	Initiation and planning of movements
Globus pallidum	Ventral lateral nucleus (VL, pars oralis)	Primary motor cortex	Modulation and coordination of movements
Deep cerebellar nuclei	Ventral lateral nucleus (VL, pars caudalis)	Premotor area	Planning of movements
Cerebellum	Ventrointermedial nucleus (VI)	Primary motor cortex	Coordination of movements

Sensory relay nuclei

Spinothalamic tract Lemniscus medialis	Ventroposterolateral nucleus (VPL)	Primary sensory cortex	Somatic sensation of the contralateral part of the body
Trigemino-thalamic tract	Ventroposteromedial nucleus (VPM)	Primary sensory cortex	Somatic sensations for face
Ascending gustatory fibers	Ventroposteromedial nucleus (VPM)	Parietal operculum area #43	Taste sensations
Retinal input	Lateral geniculate body (LG)	primary visual cortex	High visual acuity Colors
Inferior colliculus	Medial geniculate body (MG)	Primary auditory cortex	Tonal frequencies

Association thalamic nuclei

Olfactory cortex Pallidum Amygdala Hypothalamus	Mediodorsal nucleus (MD)	Frontal eye field Anterior cingulated cortex	Eye movements Emotional meaning of visual stimuli
Visual cortex	Laterodorsal nucleus (LD)	Limbic cortex Orbitofrontal cortex	Spatial learning Memory
Lateral geniculate body Medial geniculate body Superior colliculus Inferior colliculus	Lateral posterior nucleus (LP)	Visual association cortex	Visual discrimination Interpreting symbols
Lateral geniculate body Medial geniculate body Superior colliculus Inferior colliculus	Pulvinar (P)	Visual association cortex	Interpreting symbols Generating language Speech
Hippocampus via mammillary bodies	Anterior nucleus	Posterior cingulate cortex	Emotional learning

Non-specific thalamic nuclei and the midline and intralaminar nuclei receive input from brainstem reticular formation, cortex, and have an inhibitor effect to the thalamic nuclei. They are involved in arousal, alertness, gaze control, nociception, and some visceral functions.

The thalamus is a part of the network that regulates pain information. Nociceptive inputs are transmitted from the dorsal horn of the spinal cord through the spinothalamic tract to the dorsal thalamus and to the postcentral gyrus of the cortex through the capsula interna and corona radiata. Within the tract, thalamic nuclei and up to the cortex the different parts of the body have a somatotopic organization. The thalamus elaborates the different components of the pain, sensory discriminative, and affective motivational.

5.6.2 Pathology Involving the Thalamic Nuclei

Dejerine-Roussy syndrome or *thalamic pain syndrome* is characterized by numbness in the affected side followed by burning and tingling sensations, allodynia, and pain without external stimuli [54].

Thalamic *stroke syndromes* are usually rare because of multiple anastomoses from perforators of the anterior and the posterior circulation. Primary intraparenchymal thalamic hemorrhage occurs in the thalamus in 10–20% of cases. Secondary thalamic hemorrhage is often the result of chronic hypertension [55]. The artery of Percheron is a solitary arterial trunk that arises from one of the proximal segments of a posterior cerebral artery and supplies the paramedian thalami and the rostral midbrain bilaterally. Occlusion of this artery causes bilateral infarction in the paramedian portion of the thalamus, also known as an *artery of Percheron infarct* [56]. Occlusion of the *posterior choroidal artery* causes an infarction of the posterior thalamus as well as infarction of the lateral geniculate body, pulvinar, hippocampus, and parahippocampal gyrus, without involvement of the midbrain and the anterior nucleus of thalamus [57]. Finally, *deep venous sinus (straight sinus) thrombosis* can cause bilateral thalamic infarctions. Symptoms are acute of headache, conscious disturbances, and memory impairment [55].

A number of *metabolic diseases* are associated with basal ganglia lesions. *Fabry disease* is a multisystem X-linked disorder characterized by defect of lysosomal storage due to alpha-galactosidase A gene mutation. Hyperintensity on the pulvinar on T1-weighted images is a pathognomonic sign of the disease; it reflects the presence of calcification and other mineralizing alterations. *Wilson disease, Leigh disease, Krabbe disease, maple syrup urine disease, Canavan disease, Alexander disease*, and *gangliosidosis* can affect the thalamus [58].

Osmotic demyelinating syndrome is associated with electrolyte alterations, usually as complication of the rapid correction of hyponatremia. It has been associated with a variety of other conditions such as chronic alcoholism and malnutrition. Symptoms are quadriparesis and neurocognitive changes [59].

Wernicke's encephalopathy is a pathological condition caused by deficiency of vitamin B1. It is characterized by consciousness alterations, ophthalmoplegia, and ataxia. Lesions are localized in the hypothalamus, dorsomedial nucleus of the thalamus, and mammillary bodies [60].

Creutzfeldt-Jakob disease is a fatal neurodegenerative disorder caused by prions (self-replicating proteinaceous infectious particles) and characterized by progressive dementia, myoclonus, coma, and death. It involves the basal ganglia, cerebral cortex, and thalamus and typically presents with the "pulvinar sign" on MRI (restricted diffusion in the medial pulvinar nuclei) [61].

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Functional Anatomy of the Major Tracts

6

Nivedita Agarwal

The white matter tracts in the brain can be subdivided in three types according to the directions of their connections: (a) *association tracts* are intrahemispheric fibers that interconnect different variably distant cortical regions within a cerebral hemisphere (shown in green in Chap. 2), (b) *projection tracts* interconnect cortical regions within a cerebral hemisphere in a cranio-caudal direction or vice versa (shown as blue in Chap. 2), and (c) *commissural tracts* are fibers that interconnect homologous or heterologous cortical regions between the two hemispheres (shown as red in Chap. 2). The corona radiata represents the bulk of white matter seen around the bodies of the lateral ventricles that contain both association fibers and projection fibers. The tracts discussed here are the most frequently seen and reported in clinical neuroradiology practice. For imaging correlates, please refer to chapter 2.

N. Agarwal, M.D.
S. Maria del Carmine Hospital, Azienda Provinciale per i Servizi Sanitari, Rovereto (TN), Italy

Center for Mind/Brain Sciences (CIMeC), University of Trento, Rovereto (TN), Italy

Department of Radiology, Section of Neuroradiology, University of Utah, Salt Lake City (UT), USA
e-mail: Nivedita.agarwal@apss.tn.it

6.1 Association Tracts

Association tracts can be further divided into short and long association tracts. Short association tracts, otherwise known as subcortical "U"-fibers, are relatively short (1–3 cm) fibers that run immediately beneath the cortex and connect cortical areas on adjacent gyri. Long association tracts span longer distances within a hemisphere. There are seven major and two minor bundles of long association fibers, described in the following sections.

6.1.1 Superior Longitudinal Fasciculus (SLF) (Figs. 2.1, 2.2, 2.9–2.11, and 2.14)

The SLF is a large bundle of white matter in each cerebral hemisphere connecting the parietal, occipital, and temporal lobes with ipsilateral frontal cortices. Four subcomponents have been described. The superior horizontal fibers connect the superior parietal lobe to the frontal and opercular areas (SLF-I), the angular gyrus (SLF-II), the supramarginal gyrus (SLF-III), and the superior temporal gyrus (SLF-IV). SLF-IV is also called the *arcuate fasciculus*, the bundle that connects the superior temporal gyrus and the ventrolateral prefrontal cortex together [1]. A fifth SLF may connect the insular gyri and parietal lobes (a.k.a. temporoparietal SLF). The separate components are not completely identified in

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routine DTI acquisitions. Higher field strengths and high spatial resolution-based tractography is required to identify different components [2].

The SLF facilitates the formation of a bidirectional neural network that is necessary for cognitive processes such as attention, memory, emotions, and language. It connects higher- and lower-order auditory processing with frontal brain areas involved in the control of brain functions such as attention and working memory [3].

Functional deficits due to SLF damage depend on which part of the SLF is damaged. Damage to the left SLF causes language disorders such as impaired repetition, fluent aphasia, anomia, and speech arrest in neurosurgical procedures. Damage to the right SLF results in spatial-attention network deficits such as left hemi-spatial neglect [4]. Ideational apraxia—the inability to execute a sequence of actions in a complex learned motor acts despite understanding verbal commands—is thought to occur due to SLF damage in the region beneath the supramarginal gyrus of either parietal lobe [5]. Depressive disorder can result from SLF lesions in the insular region [6].

6.1.2 Inferior Longitudinal Fasciculus (ILF) (Figs. 2.5, 2.9, and 2.10)

The ILF connects the temporal and occipital lobes. It is the more lateral of the white matter tracts in the temporoparietal region. The long fibers from the superior temporal, middle temporal, inferior frontal gyri, and fusiform gyri project to the cuneus, lingual gyrus, and lateral part of the occipital lobe forming the occipitotemporal connection. The short fibers connect parahippocampal gyrus and the uncus to the more posterior visual association areas. Short fibers also connect the primary, secondary, and association visual areas to the inferior parietal lobe and the intraparietal sulcus.

The right ILF has been implicated in the analysis of faces and plays an important role in visual memory and visually evoked emotions. The left ILF is important for the analysis of colors, forms, and shapes that allowing for object, word, and color recognition [7]. The ILF also plays an important role in language processing via the ventral language pathways including IFOF, UF, and EC [8].

Lesions in the ILF will disrupt information between visual areas and limbic and memory regions and may cause one or more of the following symptoms: prosopagnosia (disorder of face recognition, right greater than left hemisphere); visual object agnosia (left greater than right hemisphere); alexia (difficulty recognizing written words, seen in conjunction with additional splenial lesions); contralateral hemiachromatopsia (disorder of color recognition); impairment of recent visual memory; and deficits in visually evoked emotions (hypoemotionality). The ILF provides an indirect alternative route to ventral semantic processing and may be altered in patients with semantic dementia [9].

6.1.3 Middle Longitudinal Fasciculus (MdLF)

The middle longitudinal fasciculus is abbreviated MdLF to distinguish it from the medial longitudinal fasciculus which is in the brainstem (see Chaps. 7, 8, and 11). The MdLF connects the superior temporal gyrus to the parietal lobe. Dorsally the tract divides into two: one courses ventrolaterally toward the angular gyrus (MdLF-AG) and the other dorsomedially toward the superior parietal lobule (MdLF-SPL) [10]. These two fascicles are difficult to resolve using routine DTI, but it is important to know about the presence of this tract in correlating symptoms in patients with lesions in the anterior limb of the internal capsule.

The MdLF-AG plays a role in attention and language, while the MdLF-SPL is involved in visuospatial and integrative audiovisual functions. These tracts may be involved in neurodegenerative disorders such as primary progressive aphasia, posterior cortical atrophy, frontotemporal dementia, and Alzheimer's disease [11].

6.1.4 Superior Fronto-Occipital Fasciculus (SFOF) (Fig. 2.3)

The SFOF runs lateral to the ventricular ependyma below the corpus callosum. It is also known as the *subcallosal fasciculus*. It is not clear whether this is a cortico-cortical tract or a cortical-subcortical short bundle of fibers. The fibers likely contain corticostriate fibers connecting the cingulate gyrus, supplementary motor area, and the caudate nucleus [12]. It is of note that recent deterministic tractography and connectome studies performed on data from healthy subjects in the Human Connectome Project have challenged the existence of SFOF in humans and remain so at the time of the writing of this book [13].

The SFOF is responsible for the initiation and preparation of speech movement as well as the limbic aspects of speech. Damage to the SFOF leads to akinetic mutism (especially with lesions in the language dominant hemisphere) and transcortical motor aphasia. With the possible nonexistence of this tract, its functional implications require further investigation. Meola et al. [13] suggest that previous functions attributed to SFOF might be subserved by the more dominant IFOF as part of the ventral linguistic pathway.

6.1.5 Inferior Fronto-Occipital Fasciculus (IFOF) (Figs. 2.5, 2.8–2.11, and 2.14)

The IFOF connects ventrolateral and dorsolateral prefrontal cortex (VLPFC and DLPFC, including the frontal eye fields) with posterior temporal cortex (middle and inferior gyri) and the occipital lobe (fusiform gyrus). It runs medial to the inferior longitudinal fasciculus. Both the frontal and parieto-occipital connections of the IFOF are complex as they fan out to reach different areas, and some authors further divide IFOF in five subcomponents (I, II, III, IV, and V), the descriptions of which are beyond the scope of this book [14]. Temporo-parieto-occipital cortex (TPO) is a multimodal region that includes portions of the parieto-occipital junction, intraparietal sulcus, angular gyrus, Wernicke's area on the caudal superior temporal gyrus, superior temporal sulcus, and middle temporo-occipital gyrus. TPO cortex is connected to prefrontal cortex through the IFOF, providing reciprocal connections with multimodal association cortex, the frontal eye fields, and the PFC.

The IFOF facilitates higher visual processing via the ventral processing stream which connects the more lateral and ventral occipitotemporal areas with the frontal areas to facilitate recognition of objects, places, colors, faces, and words. It is also associated with ventral language semantic pathways [15].

Isolated lesions of the IFOF are rare. Associated lesions in the occipitoparietal (dorsal stream) and the occipitotemporal lobe (ventral stream) may cause deficits in visuospatial processing including oculomotor apraxia (loss of volitional saccades, usually accompanied by lesions in the posterior parietal lobe); optic ataxia (inaccurate reaching to objects using vision input, e.g., Balint syndrome); impaired spatial relations related to difficulty in the perception of depth, size, orientation, and shape; and akinetopsia, the inability to recognize motion. Visual agnosia and poor visual memory are usually associated with ventral occipitotemporal stream lesions, including topographagnosia, the inability to remember places and previous routes. Lesions in the right IFOF are also associated with nonverbal semantic deficits, although the integrity of both right and left IFOF is important in the ventral semantic processing [16]. Left IFOF damage has been reported in aphasia [17].

6.1.6 Uncinate Fasciculus (UF) (Fig. 2.14)

The UF connects the anterior tip of the temporal lobes with orbitofrontal cortex. In the inferolateral frontal lobe, it is located inferior to the IFOF and "hooks" around and into the anterior pole of the temporal lobe providing fibers to the parahippocampal gyrus, the uncus, and the amygdala [18, 19].

The UF is involved with retrieval of past information, both semantic and episodic memory. It is also important in encoding and storage of social and emotional concepts [18]. Damage to the right UF results in impaired retrieval of episodic memory including autobiographical and event-related memories, while damage to the left UF results in impaired retrieval of semantic memory including knowledge of concepts and facts. Right UF damage also disrupts emotional empathy making patients apathic and indifferent to how other people feel [20].

6.1.7 **Cingulum** (Figs. 2.1–2.3, 2.6–2.11, and 2.13)

The cingulum bundle is a "C"-shaped fiber bundle that lies immediately beneath the cingulate gyrus, draping over the corpus callosum. It connects the septal area to the uncus. The cingulum is distinguished histologically into an anterior and a posterior cingulum. The anterior cingulum is agranular and links motor cortex with strong connections to parts of the limbic system (amygdala, medial dorsal thalamus) and DLPFC. The posterior cingulum is granular, relaying sensory information to the largely interconnecting multimodal TPO cortex. Recent DTI studies have identified three different subdivisions: parahippocampal, retrosplenial, and subgenual subdivisions. The parahippocampal cingulum brings inputs from the posterior cingulate cortex and parietal areas to the medial temporal lobe. The retrosplenial part connects the prefrontal cortex, anterior cingulate cortex, and posterior cingulate cortex. The subgenual subdivision is likely to connect the anterior cingulate region to limbic areas such as the amygdala, the insula, and the uncus [21].

The anterior cingulum carries information that is important in attention and volitional control of cognitive and motor functions. It mirrors frontal lobe functions, is important in self-awareness, and subserves functions such as error recognition, conflict detection, and problem solving. The more ventral part is involved in emotion and visceral function, while the more dorsal portion is involved in cognition and higher-order motor function. It is the site of internally driven eye movements. The posterior cingulum carries information that plays an integrative role in visuospatial processing.

Damage to the anterior cingulum (including cingulotomy) causes several different emotional and/or behavioral deficits, including lack of affective response to pain, decreased anxiety, executive dysfunction, reduced spontaneous behavior, akinetic mutism, reduced intentional saccades, and depression. Right side damage may lead to paranoia, dysphoria, and a feeling of being frightening, whereas a left side damage leaves one with a feeling of chill without anxiety. Ventral lesions may cause intense fear and are related to phobia, post-traumatic stress disorder, and obsessive-compulsive disorder; dorsal lesions may cause a feeling of anticipation of movement. Anterior cingulotomy may be an effective surgical treatment for intractable pain and severe refractory obsessive-compulsive disorder [22, 23].

Damage to the posterior cingulum is associated with retrosplenial amnesia (anterograde and retrograde components of memory), topographical disorientation, and loss of verbal memory and metamorphopsia (blurring of the right sides of the objects).

6.1.8 **External (ExC) and Extreme (EC) Capsules** (Figs. 2.4, 2.8, and 2.9)

The external capsule (ExC) lies lateral to the putamen and medial to the claustrum. It is a thin sheet of association fibers connecting the cerebral cortex to the striatum, largely covered by the insular folds. Fibers in the medial aspect are derived from the IFOF and cross over the foot of the corona radiate [24]. The UF provides fibers to the anterior portion of the ExC and reaches the rostral end of the corpus striatum. The posterior fibers form the bulk of the ExC and are largely derived from the ILF. Some fibers from the fron-

tal lobe enter the ExC and reach the nuclei in the tegmentum of the mesencephalon and the substantia nigra. The ExC is a route for cholinergic fibers from the basal forebrain to the cerebral cortex

The extreme capsule (EC) lies between the claustrum medially and the insula laterally. It spans from the inferior frontal cortex (Broca's area) through the middle part of the superior temporal gyrus into the inferior parietal lobule (angular gyrus/Geschwind's territory) adjacent to the middle longitudinal fascicle [25]. The EC is important in language processing, specifically language expression.

Isolated lesions of the ExC and EC are rare. Strokes involving the external capsule and the extreme capsule can cause transient partial motor symptoms, transient speech arrest, and/or dysarthria [26]. Language impairment most frequent in left-sided lesions. Mild-to-moderate contralateral hemiparesis may develop. Sensory deficits usually do not with ExC or EC damage. Anterograde and retrograde axonal degeneration may involve the striatum due to ExC hemorrhage [27, 28].

6.2 Projection Tracts

Projection tracts are long tracts that connect cortical and subcortical centers, essentially connecting the cerebral hemispheres with the cerebellum, brainstem, and spinal cord. Almost all information reaching the cerebral hemispheres arrives via projection tracts.

6.2.1 Internal Capsule (IC) (Figs. 2.4, 2.8, and 2.9)

The IC is classically subdivided into five anatomical divisions: the anterior limb (ALIC), genu, posterior limb (PLIC), and retrolenticular and sublenticular divisions. Five major types of fiber projections pass through the various divisions of the IC thalamocortical, corticothalamic, corticopontine, corticobulbar, and corticospinal [29, 30].

6.2.1.1 Anterior Limb (ALIC)

The ALIC contain *anterior thalamic radiation* (ATR), superolateral division of the *medial forebrain bundle* (slMFB), *fronto-pontine motor tracts* (*FPT*), and *anterior thalamic fibers*. These fibers are mostly horizontal on an axial plane. The ATR connects the periaqueductal gray matter, dorsomedial, and the anterior thalamic nuclei with the prefrontal cortex and particularly with the DLPFC. The slMFB (a.k.a. Arnold's bundle) originates in the ventral segmental area (VTA) and lies lateral to the ATR reaching orbitofrontal cortex, DLPFC and parts of the limbic system (nucleus accumbens and the ventral striatum) to the anterior and dorsomedial thalamic nuclei [31]. Anterior thalamic fibers connect the anterior and the dorsomedial thalamic nuclei to the orbitofrontal and the limbic system. FPT fibers connect the premotor and the prefrontal areas to the pontine nuclei coursing through the medial cerebral peduncle.

The slMFB of the ALIC mediates reward seeking and euphoric feelings, whereas ATR mediates opposite states such as those of sadness and psychic pain. Damage to the ALIC has been associated with deficits in storage and retrieval of verbal memory and decreases in motor initiation. The slMFB is involved with reduced affect (anhedonia) and depression. In the past, capsulotomies were performed to treat obsessive-compulsive disorders, severe anxiety, and panic disorders; today, the slMFB is a common target for deep brain stimulation treatment of psychiatric disorders [32]. Damage to the FPT results in conjugate eye deviation toward the site of lesion.

6.2.1.2 Genu

The genu contains mostly corticobulbar motor fibers, with a few anterior and inferior thalamic fibers passing through this area. Damage to the genu causes contralateral motor deficits in the head/neck and the face muscles, generating dysarthria, dysphagia, and faciolingual weakness. Bilateral injury to thalamofrontal fibers can cause abulia, somnolence, and cognitive impairment.

6.2.1.3 Posterior Limb (PLIC)

The PLIC is the fiber bundle running between the thalamus medially and the lenticular nuclei laterally. The anterior third of the PLIC just posterior to the genu carries superior thalamic fibers that connect frontal and parietal cortices to the ventral and lateral thalamic nuclei (thalamocortical sensory fibers). The middle third of the PLIC is comprised of fibers forming the *corticospinal tract* (*CST*). The posterior third of the PLIC contains posterior thalamic fibers connecting the posterior parietal and occipital cortices to the pulvinar and the lateral geniculate body of the thalamus.

The CST is the most important fiber bundle running through the PLIC [33]. The CST originates in primary motor cortex, passes through the PLIC and cerebral peduncle, and terminates in the gray matter of the spinal cord. The PLIC carries information about fine motor voluntary functions of all major muscle groups. A precise somatotopic organization of major muscle groups is maintained throughout the CST from the cortex down to the spine. Motor fibers from face, arm, trunk and foot cortical areas are organized in an anteroposterior gradient in the lateral middle third of the PLIC. In the cerebral peduncle, somatotopy is maintained with fibers in the face area being more medial and the foot area most lateral. Some 20% of the fibers in the CST are somatosensory. CST lesions will result in different degree of severity of contralateral hemiparesis, hemiplegia, and hemianesthesia. Conjugate eye deviation can also occur due to damage to afferents to superior colliculi.

6.2.1.4 Optic Radiation (Retrolenticular Division)

The optic radiation is contained in the retrolenticular division of the PLIC and is also known as the geniculocalcarine tract. The optic radiation is comprised of fibers originating from the superior colliculi and lateral geniculate bodies of the thalamus, traversing posteriorly to reach occipital cortex. The radiation is comprised of two sets of fibers: the superior fibers course posteriorly toward the primary visual cortex, whereas the inferior fibers wrap around the temporal horn of the lateral ventricle to reach the primary visual cortex (Meyer's loop) [34].

The optic radiation maintains a precise visuotopic organization such that superior fibers contain information from the inferior half of the visual field, whereas the inferior fibers represent the superior half of the visual field. Damage to the optic radiations leads to homonymous hemianopsia in the contralateral visual field.

6.2.1.5 Acoustic Radiation (Sublenticular Division)

Fibers from the lateral lemniscus reach the inferior colliculi and then travel to the medial geniculate bodies via the sublenticular division of the posterior PLIC. This pathway is part of the lemniscal pathway of auditory processing. A nonlemniscal pathway from the medial geniculate body connects auditory stimuli to the nuclei of the limbic system for higher more complex auditory processing. The acoustic radiation facilitates speech and nonspeech recognition with left lateralization for speech sound recognition. Defects in the acoustic radiation lead to cortical deafness (bilateral lesions), auditory agnosia (lesions also to the IFOF), and sound agnosia.

6.2.2 Fornix (Figs. 2.9 and 2.12)

The fornix is a thin fiber tract connecting the hippocampal efferents (fimbria) to the anterior septal nuclei, the hypothalamus, and the mammillary bodies. The two "crura" originate in each hippocampus, arch up and below the corpus callosum, and join just under the isthmus of the CC. Anteriorly it then divides at the level of the anterior commissure (AC) into precommissural (anterior to the AC) fibers that reach the hypothalamus and the septal nuclei and postcommissural (posterior to the AC) fibers that reach the mammillary bodies (a.k.a. columns of the fornix) [35].

The function of the fornix is not yet completely understood. It is thought to carry memory information from the hippocampus to structures of the limbic system and is part of the Papez circuit. It is recognized widely that fornix is important in emotion regulation and memory [36].

Damage to the fornix can result in memory dysfunction—particularly recent memory—resulting

in anterograde amnesia. Right fornix lesions have been associated with nonverbal memory and visual retention disturbances including deficits in visuospatial organization and topographical memory. Common diseases characterized by unilateral or bilateral lesions are chronic long standing epilepsy, herpes encephalitis, Wernicke-Korsakoff syndrome, and Alzheimer's disease [35, 37].

6.3 Commissural Tracts

The commissural tracts consist of fibers that interconnect homologous or heterologous cortical regions between the two hemispheres. There are five major commissures that link the two hemispheres: the corpus callosum (CC), anterior commissure (AC), posterior commissure (PC), hippocampal commissure (HC), and tectal commissure (TC). The corpus callosum and anterior commissure can be easily seen on DTI images; the others are not easily visible due to their small sizes. The AC, the PC, and the HC are phylogenetically older, whereas the CC appears only in placental mammals.

6.3.1 Corpus Callosum (CC) (Figs. 2.2–2.4 and 2.7–2.12)

The CC is a large compact white matter bundle that connects mostly homologous/homotopic cortical areas of the mammalian brain. There is evidence that some callosal fibers project to heterotopic regions. It is subdivided largely into the rostrum, genu, body, isthmus, and splenium [38]. The major divisions also reflect changes in fiber density and degree of myelination, both reflecting the functional diversity of different parts of the CC [39].

The *rostrum* lies at the undersurface of the genu of the CC extending anteriorly from the AC and lying immediately posterior to the septal nuclei. The *genu* is the most anterior portion of the CC that abruptly curves up from the rostrum and around to continue into the body. It contains fibers (a.k.a. forceps minor) that connect the anterior frontal lobes and the anterior cingulate cortices. Posteriorly it lines the anterior surface of the septum pellucidum. Fibers in the *body* lie between the cingulum bundle above and the occipito-fronto fasciculi below. They interconnect the premotor and supplementary motor areas. The *isthmus* contains fibers from the pre- and postcentral gyri thereby connecting primary motor cortices and the primary somatosensory cortices. Motor fibers are topographically arranged in the order lip, hand, and foot along the anterior-posterior gradient. Primary auditory fibers also pass through the isthmus [40, 41]. The fibers in the *splenium* form the forceps major. Anteriorly to posteriorly, the fibers connect parietal, temporal, and occipital cortices [42]. Finally, the *tapetum* consists of decussating fibers in the splenium arch around the atria of the lateral ventricle along the lateral surface of the temporal horns connecting the temporal lobes. These fibers can be seen on coronal images.

The CC permits interhemispheric transfer of information through both excitatory and inhibitory mechanisms. It plays a role in the development of language lateralization and hemispheric asymmetry. Demanding cognitive tasks however require greater hemispheric recruitment which is achieved through a predominant excitatory function of the CC promoting a unified experience in which we perceive and perform actions [43].

Lesions in the anterior CC may cause a frontal variant of the alien hand syndrome due to disconnection of the SMA resulting in grasping, compulsory manipulation of tools, and groping (affected hand constantly reaching for nearby objects). Anterior callosal lesions can also cause intermanual conflict (hands competing each other for purposeful movement) [44]. A posterior variant of callosal alien hand syndrome, caused by lesions to the posterior third of the CC including lesions in the forceps minor (parietal lobe), is characterized by uncoordinated hand movements, involuntary levitation accompanied by hemianesthesia, visuospatial neglect, and optic ataxia [45]. Neurodegenerative conditions such as corticobasal degeneration and hemorrhagic strokes in the splenium may result in such symptoms. Disruption of temporal lobe bi-hemispheric connections can cause alexia without agraphia (cannot read but can spell and recognize when spelled aloud).

In cases of callosal agenesis or surgical callosotomy, interhemispheric transfer of information is compensated through the AC and PC. However, disconnection syndromes [46] may result in symptoms such as ideomotor apraxia (inability to perform verbal commands but able to imitate visual movements perfectly), tactile anomia, agraphic aphasia, verbal anosmia, and memory impairments, particularly recall and working memory.

6.3.2 Anterior Commissure (AC) (Figs. 2.5 and 2.12)

The AC phylogenetically is the oldest forebrain commissures. It is readily seen as a compact cylindric highly myelinated bundle of white matter below the septum pellucidum, just anterior to the third ventricle. It is anterior to the basal ganglia and passes through the amygdalae. It is just caudal to the anterior columns of the fornix and crosses through the lamina terminalis. Its anterior fibers run medial to the UF and interconnect the olfactory bulbs, orbitofrontal cortex, and amygdala; its posterior fibers connect middle, inferior temporal gyri, fusiform gyri, and parahippocampal gyri.

The AC carries information about olfaction and sexual behavior, memory, and emotion. The AC is thought to serve as a compensatory pathway for transferring interhemispheric information in cases of CC agenesis and acquired lesions of the CC and in patients with callosotomies (e.g., for treatment of epilepsy) [47, 48].

Damage to the AC is rare, and there is little information regarding lesions of the AC [49].

6.3.3 Posterior Commissure (PC)

The PC is a small bundle of myelinated fibers seen in the dorsal upper end of the cerebral aqueduct. It connects the pretectal nuclei, the interstitial nuclei of Cajal, and the nuclei of Darkschewitsch bilaterally. Most of these nuclei are loosely considered the periaqueductal gray matter. Fibers also project bilaterally to the Edinger-Westphal nuclei, fibers that convey parasympathetic information to the third cranial nerve and are responsible for direct and consensual pupillary light reflex [29].

The PC carry information that mediates bilateral pupillary light reflex. Some fibers connect the MLF allowing for upward gaze. They allow consensual papillary light reflex and facilitate conjugate eye movements. A unilateral lesion of the PC results in upward saccade paralysis with preservation of the vestibule-ocular reflex. Lesions to the nuclei of the PC can cause bilateral eyelid retraction [50].

The *tectal commissure* consists of fibers that are partially intermingled with the fibers in the caudal aspect of the PC. These fibers are interconnect the superior colliculi and are thought to transfer information related to interocular functions. Damage to tectal commissure fibers is thought to inhibit the development of saccades.

6.3.4 Hippocampal Commissure (HC)

The HC is also known as the commissure of the fornix or psalterium Davidi. It lies under the splenium of the CC and unites the crura of the fornices. It connects the subicular and parahippocampal cortices.

The HC is essential for normal hippocampal development and unites hippocampal and entorhinal cortices bilaterally. It may also play a role in memory. Seizure spread to opposite hemisphere via the HC is suspected in temporal lobe epilepsy.

6.3.5 Habenular Commissure

The habenular commissure is a tiny white matter tract situated just in front of the pineal gland that interconnects the habenular nuclei. The habenular nuclei (lateral and medial groups) receive input from the stria medullaris of the thalamus and send outputs to septal nuclei, basal ganglia, and midbrain structures such as the ventral tegmental area, the raphe nuclei, and the substantia nigra. The habenular nuclei are known to be involved in pain, sleep-wake cycles, mood, and learning and are activated by reward negative stimuli [51]. Anxiogenic addiction may result from overstimulation of dopaminergic receptors in habenular nuclei [52].

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Part II

Cerebellum and Brainstem

The focus of the next five chapters is to illustrate brain structures derived from the embryologic mesencephalon, metencephaolon (pons) and myelencephalon (medulla oblongata). From these structures, the brainstem and cerebellum are formed. The brainstem is a relatively small structure that contains many different structures packed into a small volume: cranial nerve nuclei, reticular and raphe nuclei, ascending and descending fibers, and transverse crossing fibers. In an attempt to label as many clinically important structures as possible, labels on axial images may look crowded. Sagittal and coronal anatomy was avoided for the same reason. Most structures are very hard to identify on these planes and we think knowledge of their positioning on the axial slices is sufficient to correlate lesions and the constellation of symptoms presented. The cerebellum is presented in three planes, using both common cerebellar nomenclatures.

As in Part I, the anatomical chapters show labeled clinical 3T (Chaps. 7 and 8) and high resolution 7T (Chap. 10) MRI images. Vascular anatomy is illustrated using digital subtraction angiography technique (Chap. 9). The functional significance of most of the clinically relevant structures has been detailed (Chap. 11), including major syndromes related to the brainstem and cerebellar pathology. However, the details of the cranial nerves are presented separately in Part III.

Structural Anatomy

7

Nivedita Agarwal and John D. Port

The images presented within this chapter are from the same single healthy 25-year-old female subject that was presented in Chap. 1. Voxel dimensions are 1 mm in all dimensions (isotropic), with reconstructions created in the true axial and true coronal planes. Selected images were chosen for labeling that best represented the local anatomy in a given region. Images were magnified to focus on the brainstem and cerebellum and were adjusted to accentuate difference between the gray and white matter so that labeling is more obvious.

Note that these images appear blurry; while 1 mm isotropic pixel dimensions are adequate for imaging the cerebral hemispheres, this resolution is insufficient to capture the detailed anatomy of the small brainstem and cerebellar structures. Such anatomy is better imaged with a 7 tesla MRI scanner (see Chap. 10). However, most MRI facilities have 1.5T and 3T scanners; thus we chose to label images as the radiologist would see them. Furthermore, we chose to reconstruct images in the planes most commonly viewed by radiologists; as such, our labels are more relevant to routine clinical MRI than the labels presented in traditional histological atlases, which are typically aligned perpendicular to the long axis of the brainstem [3–7].

Each page contains the labeled images on the left-hand side. In order to keep the labels small, label numbers are specific to each brain region (cerebellum, brainstem). A small image on the top right of the page documents the locations of the slices, and a key in the lower right-hand side of the page lists the individual structures.

N. Agarwal, M.D. (✉)
S. Maria del Carmine Hospital, Azienda Provinciale per i Servizi Sanitari, Rovereto (TN), Italy

Center for Mind/Brain Sciences (CIMeC), University of Trento, Rovereto (TN), Italy

Department of Radiology, Section of Neuroradiology, University of Utah, Salt Lake City (UT), USA
e-mail: Nivedita.agarwal@apss.tn.it

J.D. Port, M.D., Ph.D.
Department of Radiology, Mayo Clinic, Rochester, MN, USA

7.1 Cerebellum Overview

Embryologically, the cerebellum develops from the dorsal aspect of the rhombencephalon, specifically the dorsal metencephalon. Once formed, the cerebellum then develops into a midline portion known as the vermis and two more lateral cerebellar hemispheres. Three primary lobes develop: the primary fissure separates the more cranial anterior lobe from the caudal posterior lobe, and the pos-

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Table 7.1 General structure and function of the cerebellum

Cerebellar lobe	Vermis (archicerebellum, spinocerebellum)	Cerebellar hemispheres (neocerebellum)	Function
Anterior lobe	Lingula Central lobule Culmen	Wings of lingula Wings of central lobule Quadrangular lobule	Sensorimotor
Posterior lobe (cerebrocerebellum)	Declive Folium Tuber Pyramid Uvula	Simple lobule Superior semilunar lobule Interior semilunar lobule Biventral Tonsil	Cognition, language
Flocculonodular lobe (paleocerebellum, vestibulocerebellum)	Nodulus	Flocculus	Eye movements, balance

terolateral fissure separates the posterior lobe from the flocculonodular lobe. The posterior lobe has two fissures, a horizontal fissure that further divides the posterior lobe between the folium/superior semilunar and tuber/inferior semilunar lobules and a prepyramidal fissure that divides the posterior lobe between the tuber/inferior semilunar and pyramid/biventral lobules. The cerebellar tonsils consist of the uvula and flocculonodular lobe. These lobes contain a total of 18 smaller lobules, 9 in the vermis and 9 in the cerebellar hemispheres. The general structure and function of the cerebellum are detailed in Table 7.1.

The vermis receives input from the spinal cord. It is important in the control of the muscle tone and axial limb movement, maintaining posture of the antigravity muscles. The cerebellar hemispheres receive input from the brain through the pontine nuclei. These areas are responsible for the non-motor functions of the cerebellum such as cognition, language and emotion processing, and modulation. The flocculonodular lobe is heavily connected with the vestibular nuclei and brainstem nuclei for the important head and eye movement coordination.

Table 7.2 Comparison of Ito's and Larsell's nomenclature for the cerebellar vermis

Ito [1]	Larsell [2]
Lingula	I
Centralis	II, III
Culmen	IV, V
Declive	VI
Folium	Superior VII A
Tuber	Inferior VII B
Pyramis	VIIIA, VIIIB
Uvula	IX
Nodulus	X

The complex anatomy of the cerebellum has been categorized in many different ways. From the literature, two separate but similar naming systems have become more prominent than the others, specifically, those of Ito [1] and Larsell [2]. A comparison of these nomenclatures is presented in Tables 7.2, 7.3, and 7.4.

The nomenclature of Larsell [2] was used to label the cerebellar figures (Figs. 7.1–7.14) in this chapter. For the sake of brevity, only additional structures not found in Tables 7.2, 7.3, and 7.4 will be listed in the key for each cerebellar page.

Table 7.3 Comparison of Ito's and Larsell's nomenclature for the cerebellar hemispheres

Ito [1]	Larsell [2]
Vinculum	HI
Central lobule	HII, HIII
Quadrangular lobule, anterior portion	HIV, HV
Quadrangular lobule, posterior portion	HVI
Semilunar lobule, superior portion	HVIIA (Crus I)
Semilunar lobule, inferior portion	HVIIA (Crus II)
Gracile	HVIIB
Biventer	HVIII
Tonsil	HIX
Flocculus	HX

Table 7.4 Nomenclature of the cerebellar fissures

1′	Precentral fissure
2′	Preculminate fissure
3′	Primary fissure
4′	Superior posterior fissure
5′	Horizontal fissure
6′	Prepyramidal fissure
7′	Secondary fissure
8′	Posterolateral fissure
9′	Inferior posterior fissure

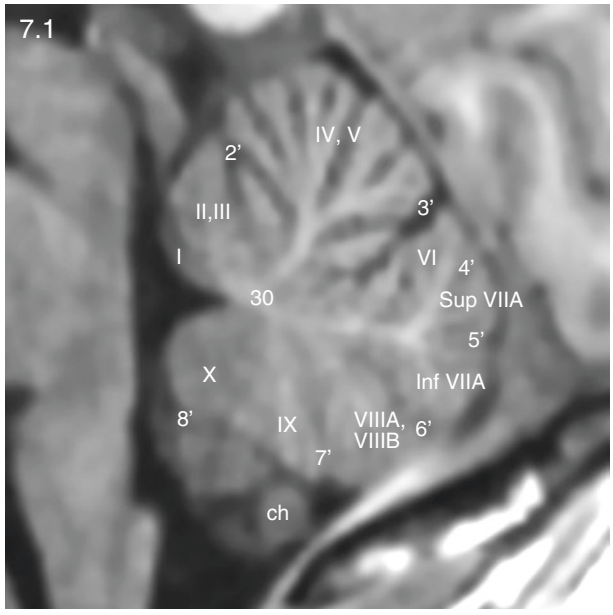
7.2 Brainstem Overview

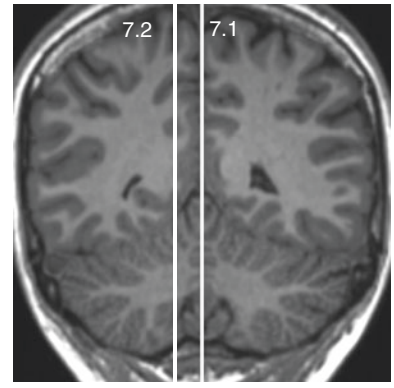
The brainstem develops from caudal two of the three embryological vesicles: the more rostral mesencephalon (a.k.a. midbrain) and the more caudal rhombencephalon. The rhombencephalon further subdivides into the metencephalon (a.k.a. pons) and the myelencephalon (a.k.a. medulla oblongata). All nuclei and tracts respect a columnar organization along the rostral-caudal axis of the brainstem. Thus, it is important to remember that some cranial nerve nuclei and most white matter tracts run through the entire length of the brainstem. It is a combination of symptoms that helps to localize pathology in one of the three parts of the brainstem.

In the developing central nervous system, a basal plate and an alar plate are formed delimited by the sulcus limitans. The alar plate is the dorsal part of the neural tube, whereas the basal plate is the ventral portion of it. The alar plate continues caudally into the sensory dorsal part of the spinal cord, and the basal plate continues to form the motor part of the spinal cord. In the brainstem the alar plates move out laterally and contain the general somatic, general and special visceral afferents of the cranial nerves. Also ascending sensory tracts are seen more laterally. The basal plate contains the motor axons and contain the general somatic and general and special visceral efferents. Also the motor descending fibers are mostly found in the medial part of the brainstem.

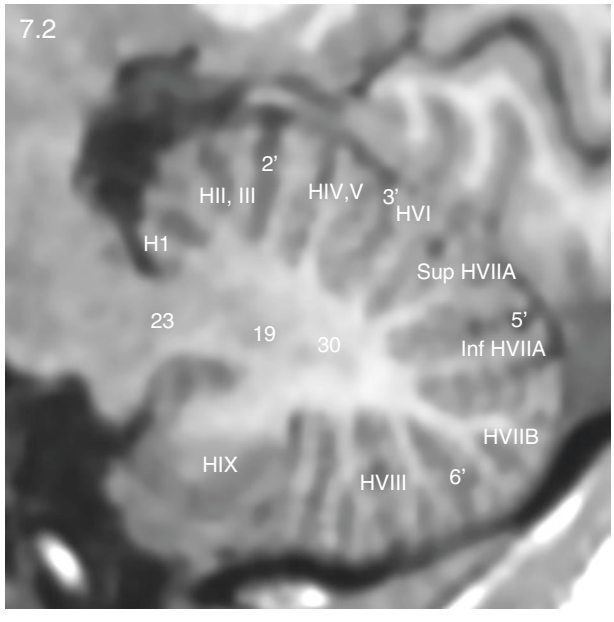
The brainstem contains most of the encephalic reticular centers. The brainstem reticular nuclei are divided into three longitudinal columns: median, central (or medial), and lateral. Median nuclei are the raphe nuclei and are cholinergic, locus coeruleus contain noradrenergic fibers, and the PAG and the reticular nuclei are mostly serotoninergic.

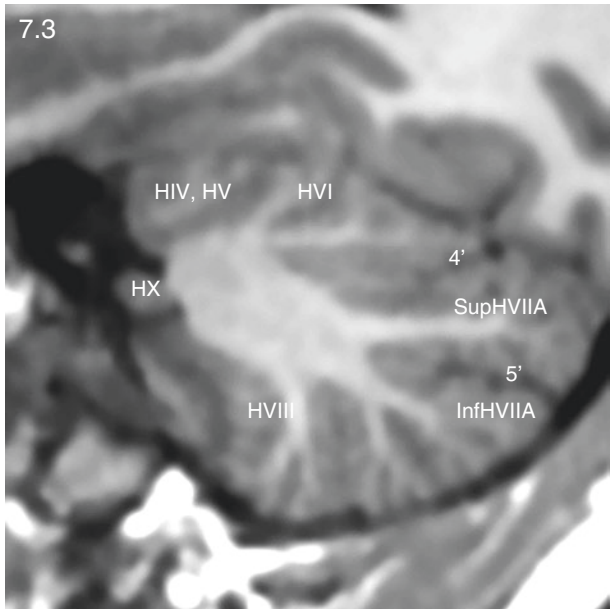
In this section, the gray matter contents of each brainstem region are reviewed. In Chap. 8, the white matter tracts in each brainstem region are discussed. The functions of the cranial nerves are separately detailed in Chaps. 12–23 and will not be referred to in this chapter. Finally, the functions of the gray matter structures and white matter tracts will be discussed in Chap. 11.

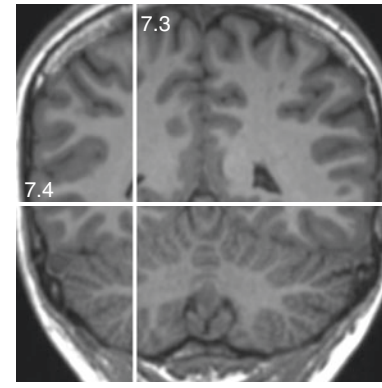


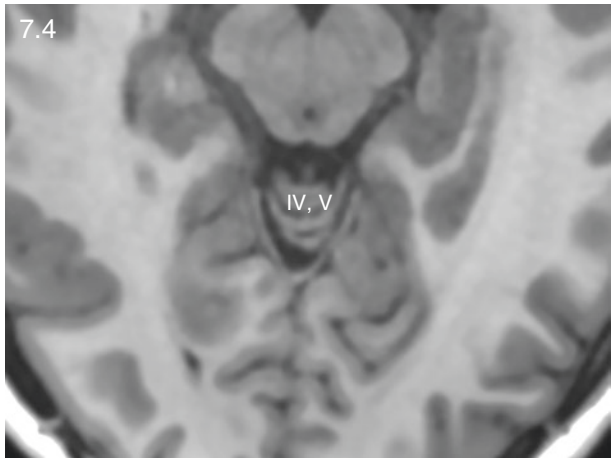


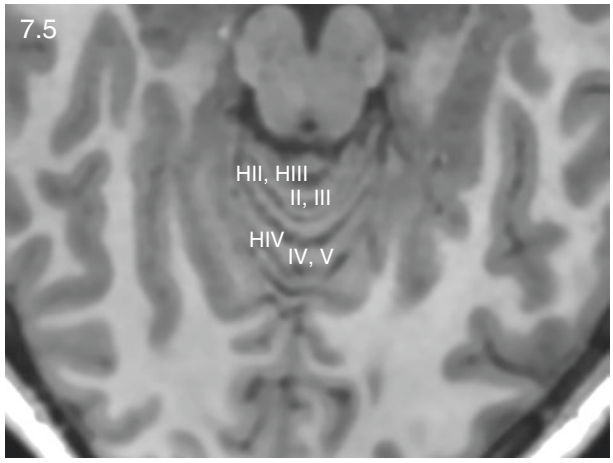
ch	Cerebellar hemisphere
19	Dentate nucleus
23	Middle cerebellar peduncle (brachium pontis)
30	Corpus medullare

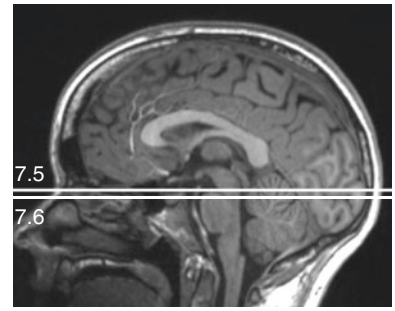


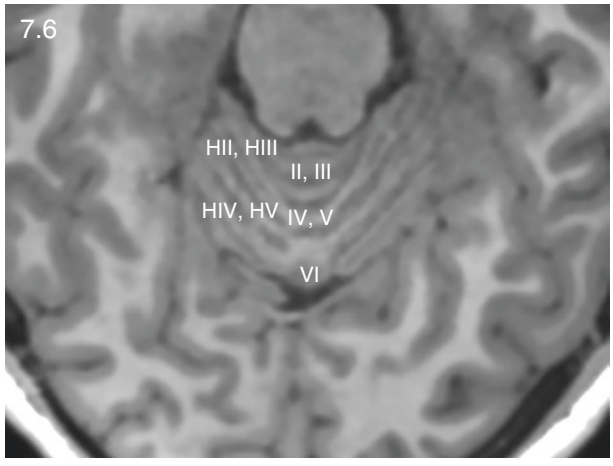

7.3
HIV, HV
HVI
4’
HX
SupHVIIA
5’
HVIII
InfHVIIA

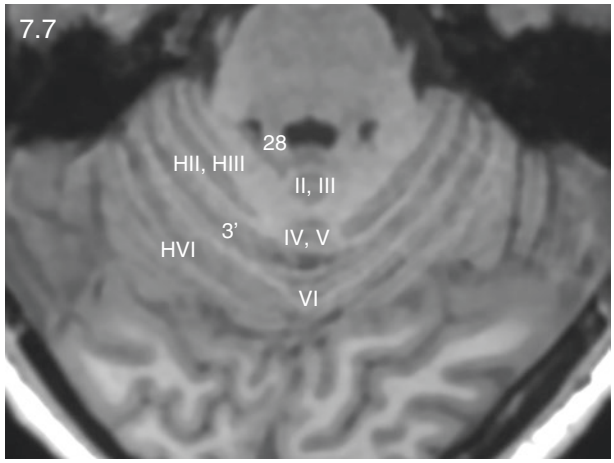

7.3
7.4

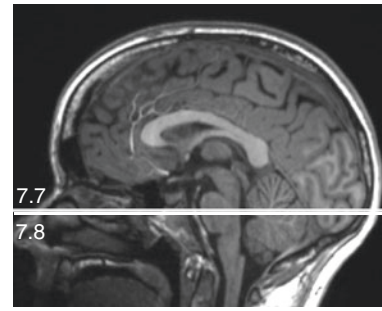

7.4
IV, V


7.5
HII, HIII
II, III
HIV
IV, V

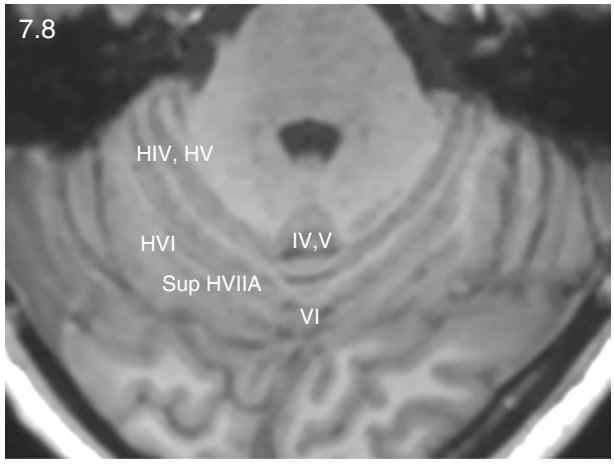

7.5
7.6

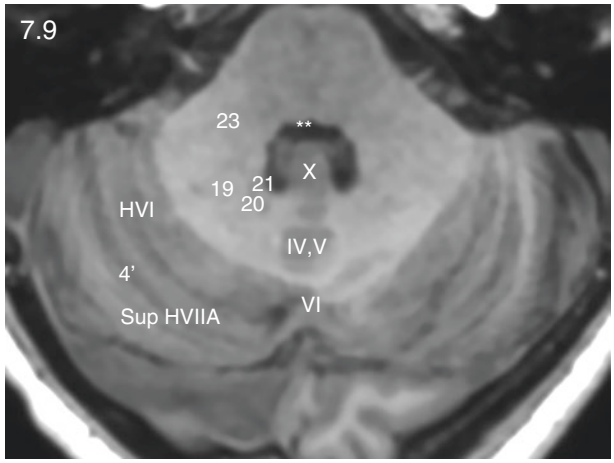

7.6
HII, HIII
II, III
HIV, HV
IV, V
VI

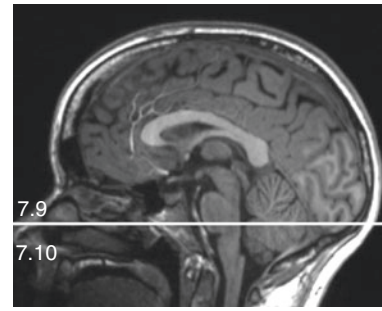




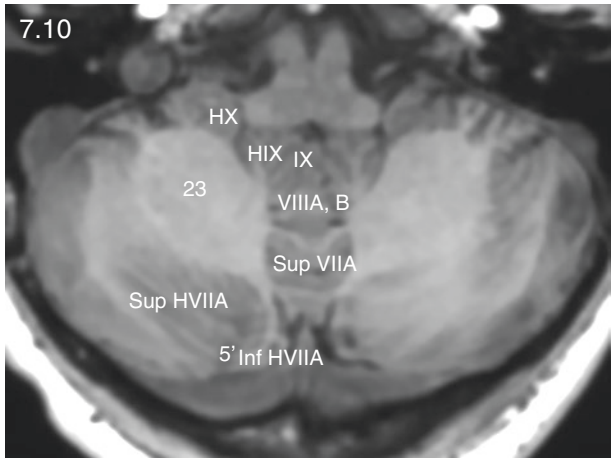
28	Superior cerebellar peduncle (brachium conjunctivum)

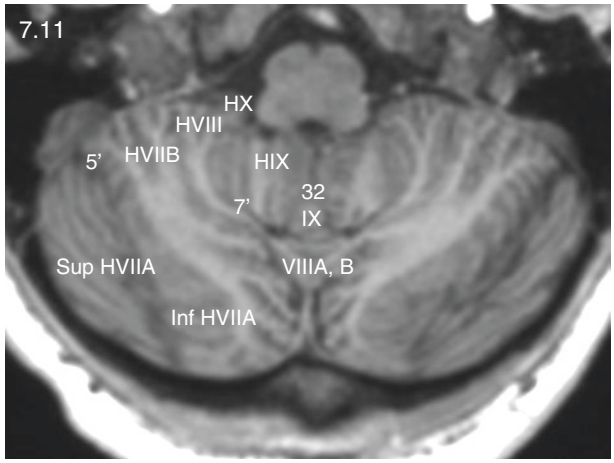


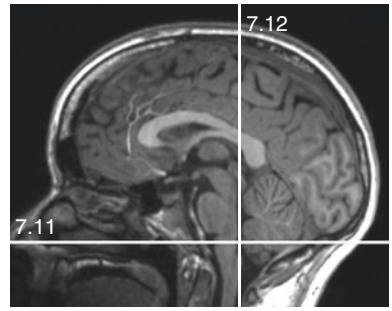




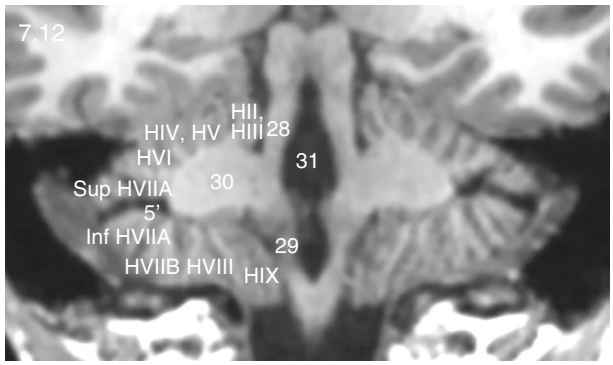
19	Dentate nucleus
20	Globose and emboliform nuclei
21	Fastigial nucleus
23	Middle cerebellar peduncle (brachium pontis)
**	Facial colliculus

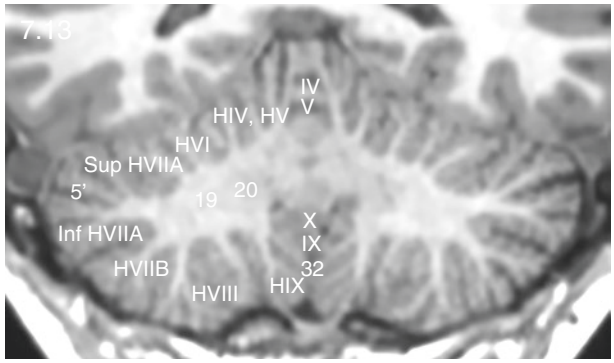


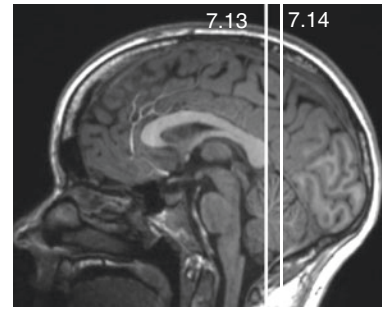




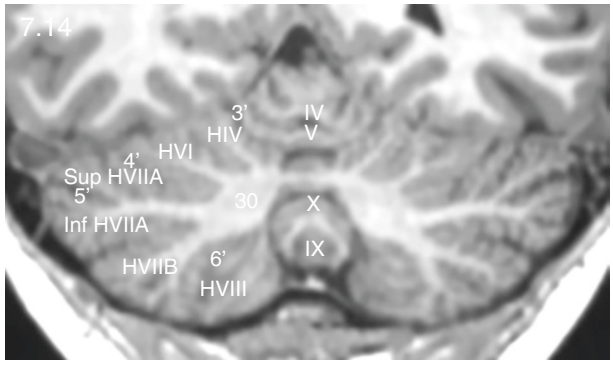
28	Superior cerebellar peduncle (brachium conjunctivum)
29	Inferior cerebellar peduncle
30	Corpus medullare
31	Fourth ventricle
32	Vallecula of the cerebellum

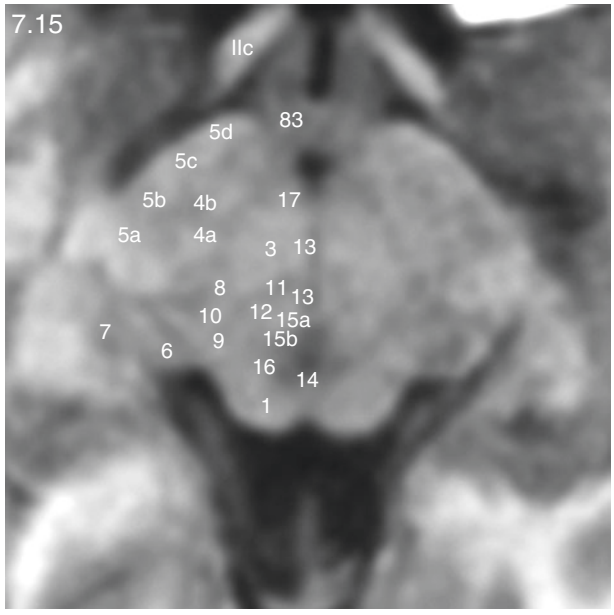


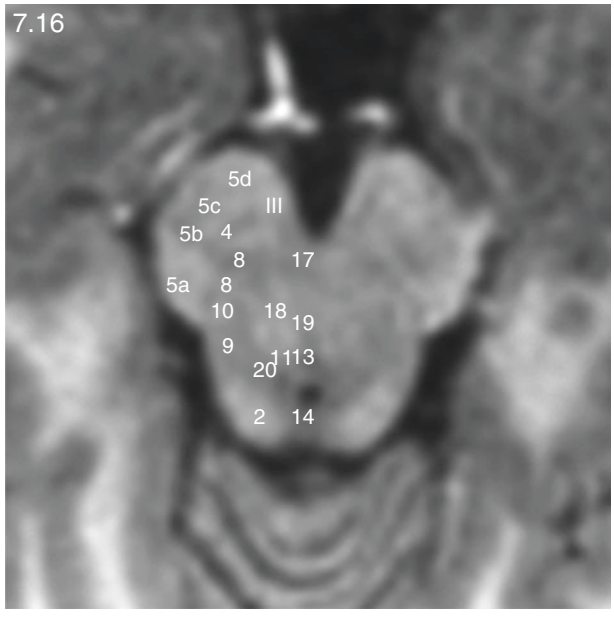


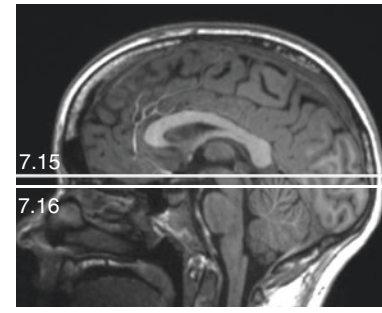


19	Dentate nucleus
20	Emboliform nucleus
30	Corpus medullare
32	Vallecula of the cerebellum

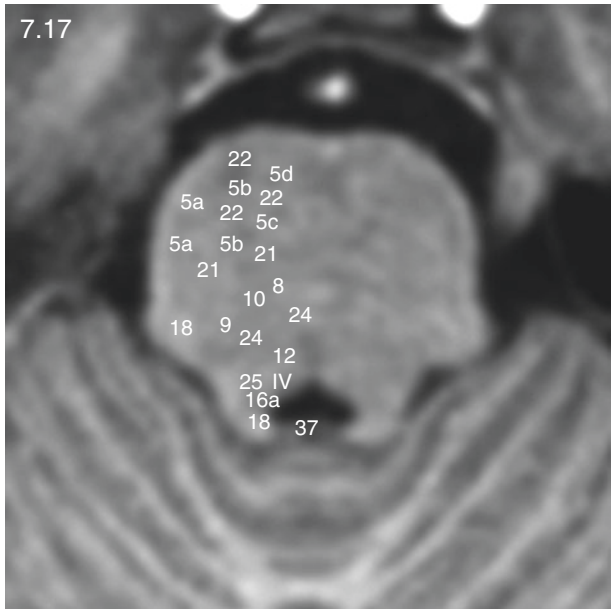


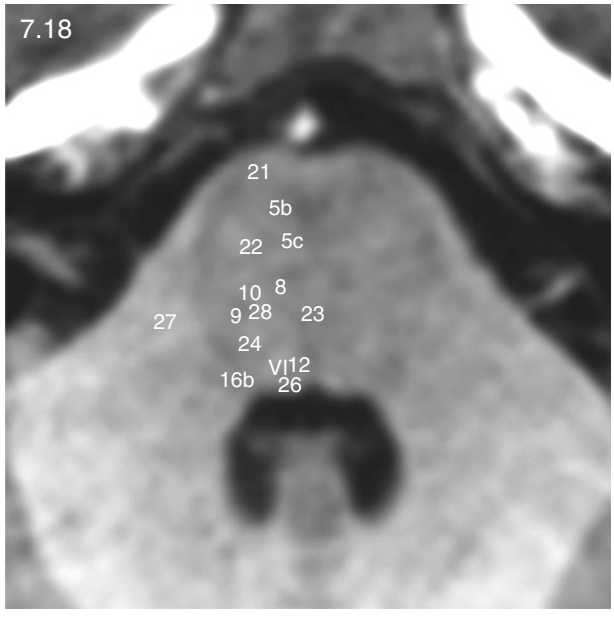


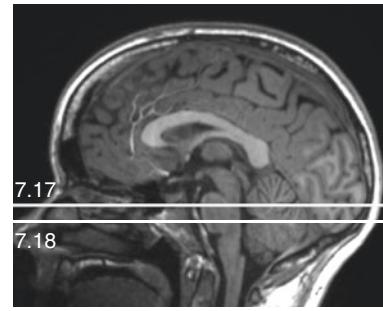




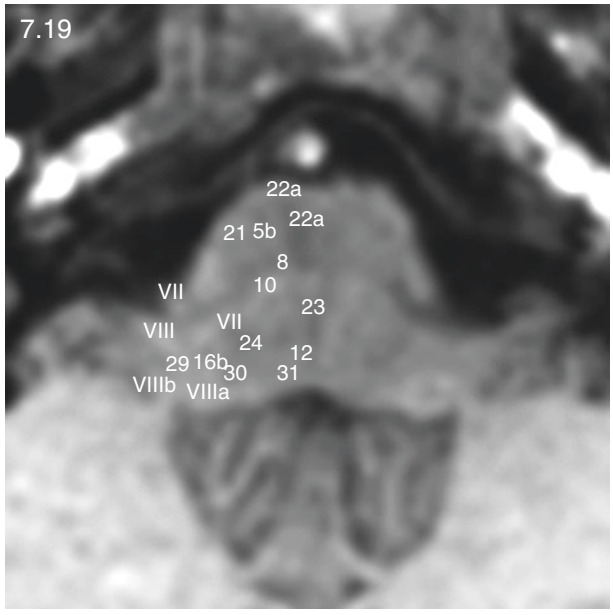
1	Superior colliculus
2	Inferior colliculus
3	Red nucleus
4	Substantia nigra
4a	Substantia nigra, pars compacta
4b	Substantia nigra, pars reticulata
5a	Cerebral peduncle, parietotemporopontine tract
5b	Cerebral peduncle, corticospinal tract
5c	Cerebral peduncle, corticonuclear tract
5d	Cerebral peduncle, frontopontine tract
6	Medial geniculate nucleus
7	Lateral geniculate nucleus
8	Medial lemniscus
9	Lateral lemniscus
10	Spinothalamic tract
11	Central tegmental tract
12	Medial longitudinal fasciculus
13	Dorsal raphae nucleus
14	Periaqueductal gray matter
15a	Oculomotor complex, principle motor nucleus
15b	Oculomotor complex, Edinger-Westphal nucleus
16	Trigeminal complex
17	Ventral tegmental area
18	Brachium conjunctivum
19	Decussation of the superior cerebellar peduncle
20	Reticular nuclei
83	Mammillary body
IIc	Optic tract
III	Oculomotor nerve

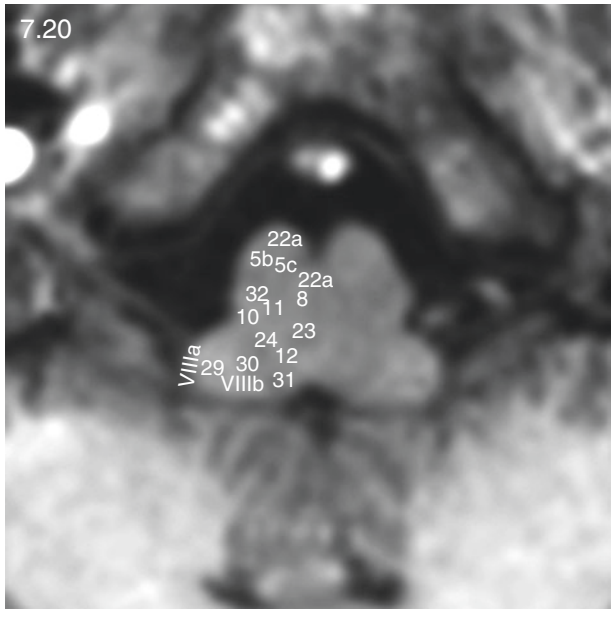


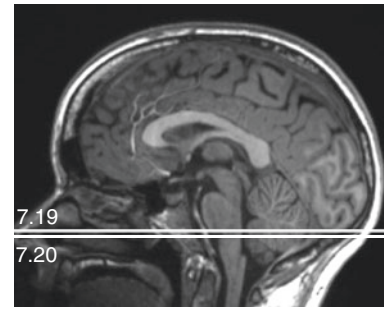




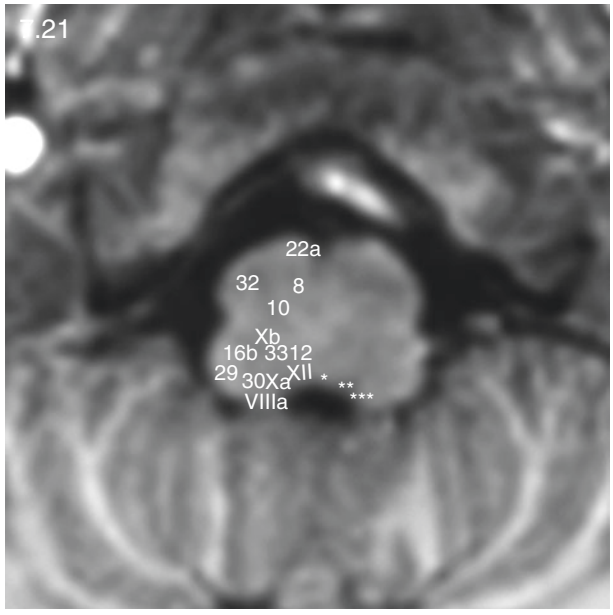
5a	Cerebral peduncle, parietotemporopontine tract
5b	Cerebral peduncle, corticospinal tract
5c	Cerebral peduncle, corticonuclear tract
5d	Cerebral peduncle, frontopontine tract
8	Medial lemniscus
9	Lateral lemniscus
10	Spinothalamic tract
12	Medial longitudinal fasciculus
16a	Trigeminal complex, mesencephalic nucleus
16b	Trigeminal complex, principle sensory nucleus
18	Superior cerebellar peduncle (brachium conjunctivum)
20	Reticular nuclei
21	Pontocerebellar fibers
22	Pontine nuclei
23	Pontine raphe nuclei
24	Pontine reticular nuclei
25	Locus coeruleus
26	Facial colliculus
27	Middle cerebellar peduncle (brachium pontis)
28	Superior olivary nucleus
37	Superior medullary velum
IV	Trochlear nucleus
VI	Abducens nucleus

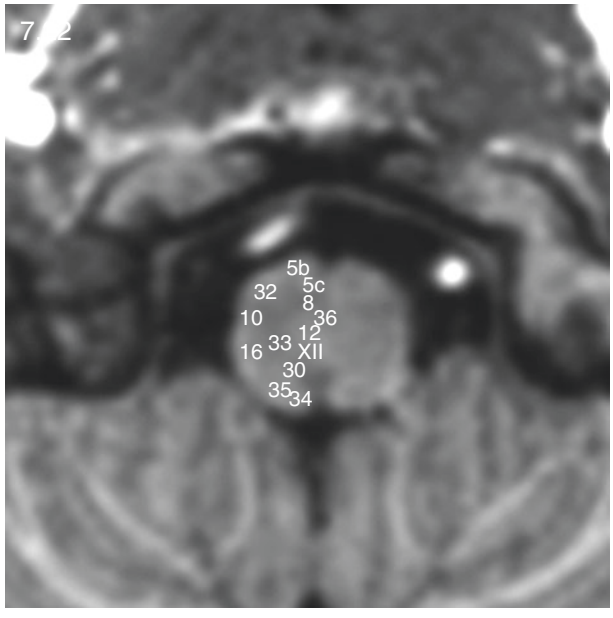


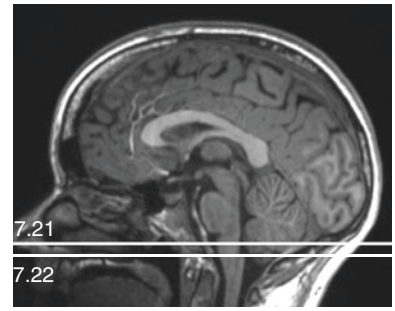




5b	Cerebral peduncle, corticospinal tract
5c	Cerebral peduncle, corticonuclear tract
8	Medial lemniscus
10	Spinothalamic tract
11	Central tegmental tract
12	Medial longitudinal fasciculus
16 b	Trigeminal complex, principle sensory nucleus
21	Pontocerebellar fibers
22a	Pontine nuclei, arcuate nuclei
23	Pontine raphe nuclei
24	Pontine reticular nuclei
29	Restiform body
30	Tract of nucleus solitarius
31	Nucleus prepositus hypoglossi
VII	Facial nerve
VIII	Vestibulocochlear nerve
VIIIa	Vestibulocochlear nerve, vestibular nuclei
VIIIb	Vestibulocochlear nuclei







5b	Cerebral peduncle, corticospinal tract
5c	Cerebral peduncle, corticonuclear tract
8	Medial lemniscus
10	Spinothalamic tract
12	Medial longitudinal fasciculus
16	Trigeminal complex
16 b	Trigeminal complex, principle sensory nucleus
21	Pontocerebellar fibers
29	Restiform body
30	Tract of nucleus solitarius
32	Inferior olivary nucleus
33	Bulbar reticular nuclei
34	Nucleus gracilis
35	Nucleus cuneatus
36	Decussation of Internal arcuate fibers
VIIIa	Vestibulocochlear nerve, vestibular nuclei
Xa	Vagus nerve, dorsal motor nucleus
Xb	Vagus nerve, nucleus ambiguus
XII	Hypoglossal nerve
*	Hypoglossal trigone
**	Vagal trigone
***	Vestibular trigone

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White Matter Anatomy

8

Andrea Poretti

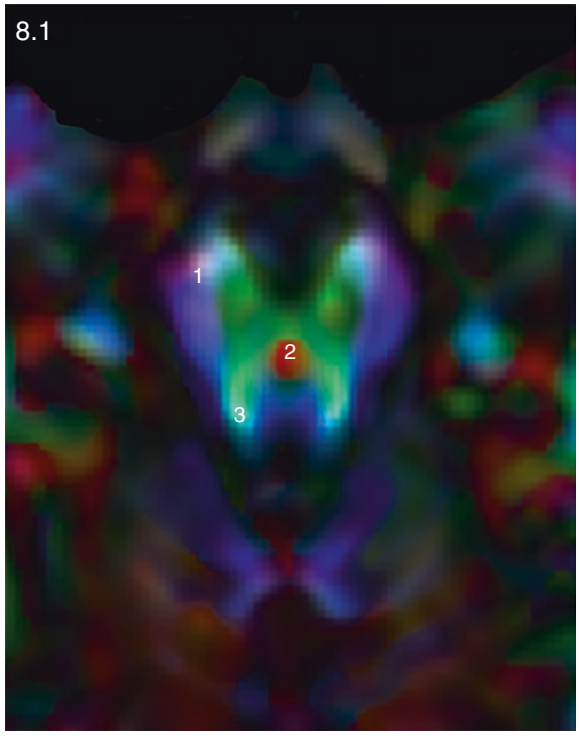
Details of the diffusion tensor imaging (DTI) technique and acquisition details were described in detail in Chap. 2. For this chapter, selected images were chosen for labeling that best represented the local anatomy in a given region. Note that we chose to reconstruct images in the planes most commonly viewed by radiologists; as such, our labels are more relevant to routine clinical MRI than the labels presented in traditional histological atlases [1–5].

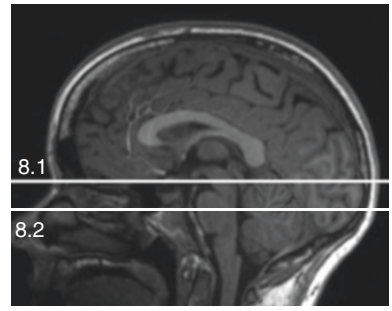
Each page contains the labeled images on the left-hand side. A small image on the top right of the page documents the locations of the slices, and a key in the lower right-hand side of the page lists the individual structures.

Andrea Poretti was deceased at the time of publication.

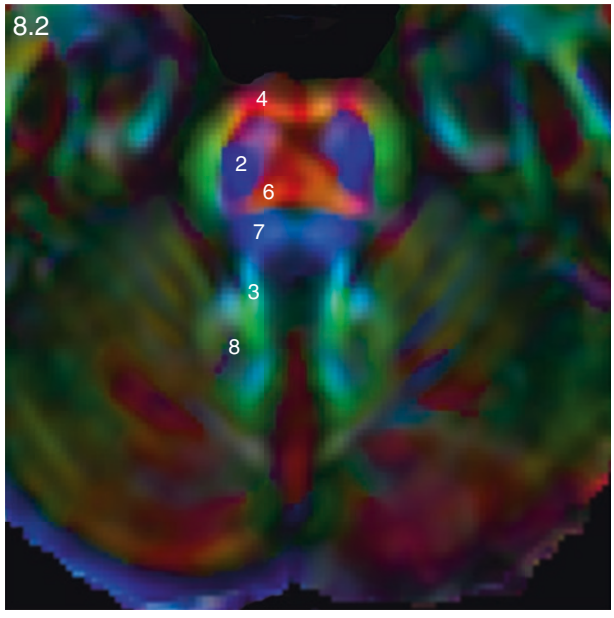
A. Poretti, M.D.
Section of Pediatric Neuroradiology, Division of Pediatric Radiology, Russell H. Morgan Department of Radiology and Radiological Science, Johns Hopkins University School of Medicine, Baltimore, MD 21287-0842, USA
e-mail: nivedita.agarwal@apss.tn.it

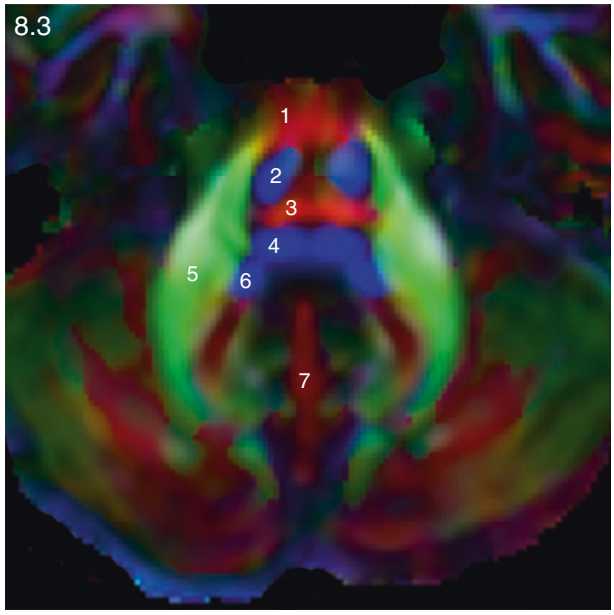
N. Agarwal, J.D. Port (eds.), *Neuroimaging: Anatomy Meets Function*,
DOI 10.1007/978-3-319-57427-1_8

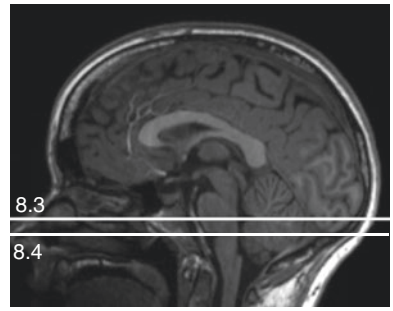




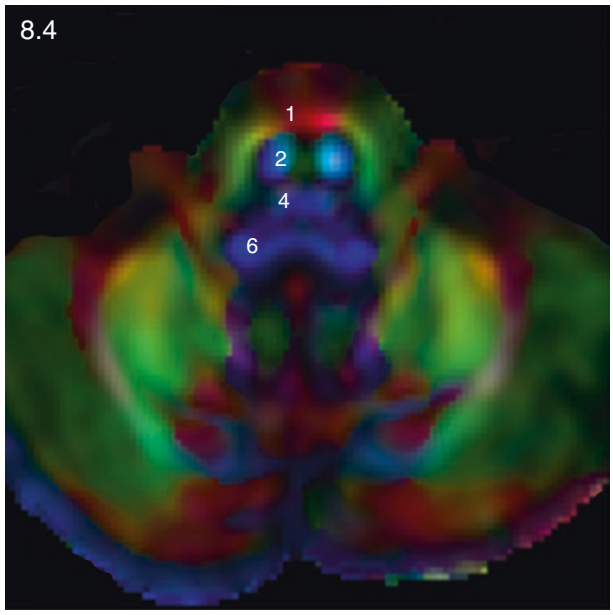
1	Cerebral peduncles
2	Decussation of superior cerebellar peduncles
3	Superior cerebellar peduncles
4	Anterior transverse pontine fibers
5	Corticospinal tracts
6	Posterior transverse pontine fibers
7	Medial lemniscus
8	Dentate nuclei

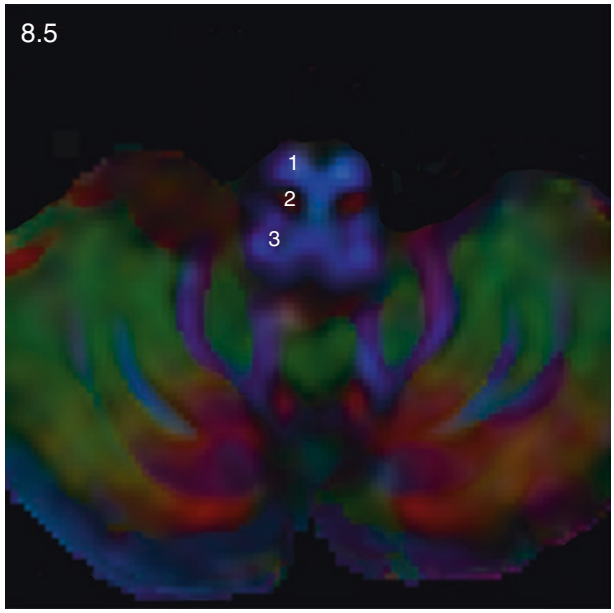


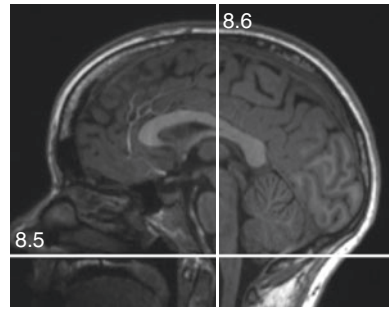




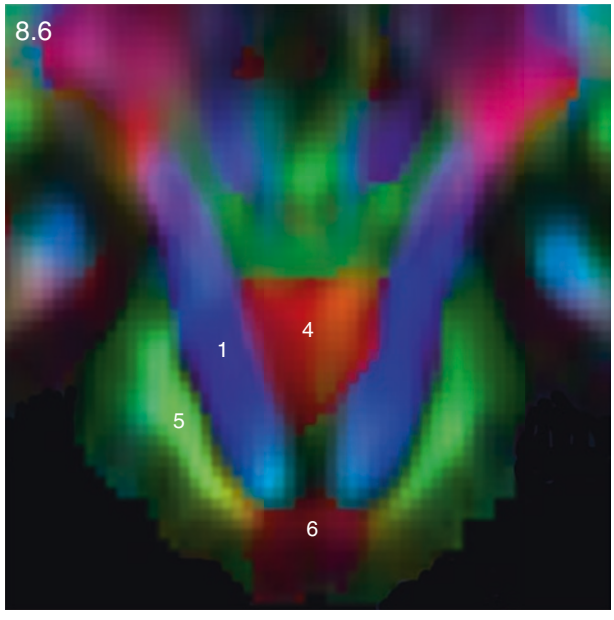
1	Anterior transverse pontine fibers
2	Corticospinal tracts
3	Posterior transverse pontine fibers
4	Medial lemniscus
5	Middle cerebellar peduncles
6	Inferior cerebellar peduncles
7	Cerebellar vermis

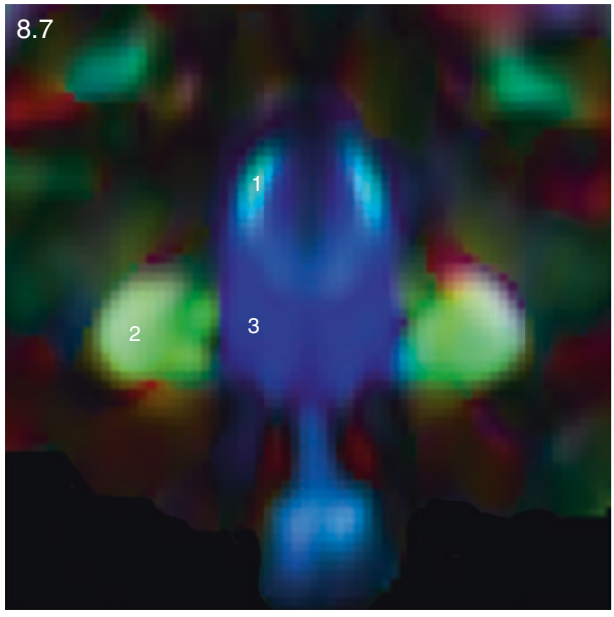


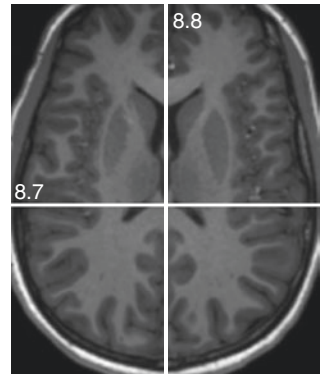




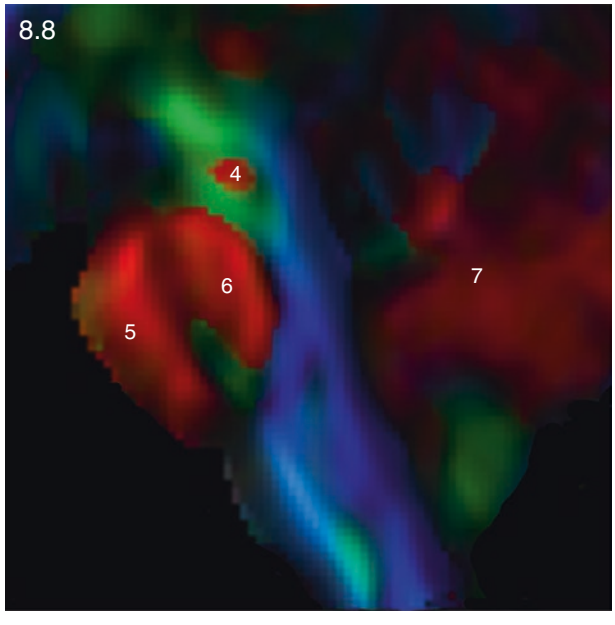
1	Corticospinal tracts
2	Inferior olivary nuclei
3	Inferior cerebellar peduncles
4	Posterior transverse pontine fibers
5	Middle cerebellar peduncles
6	Anterior transverse pontine fibers

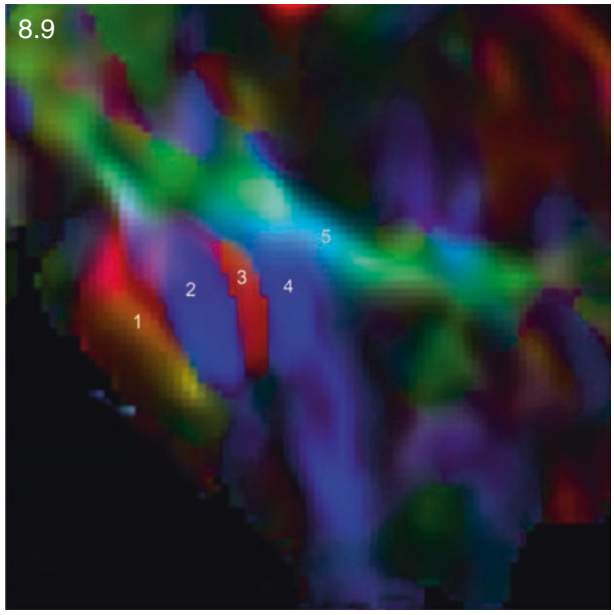


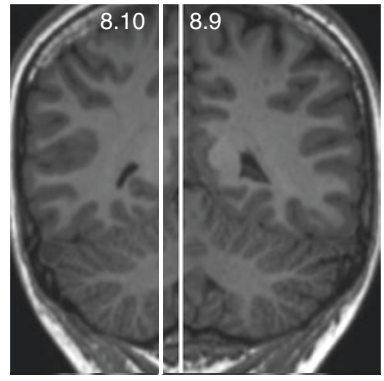




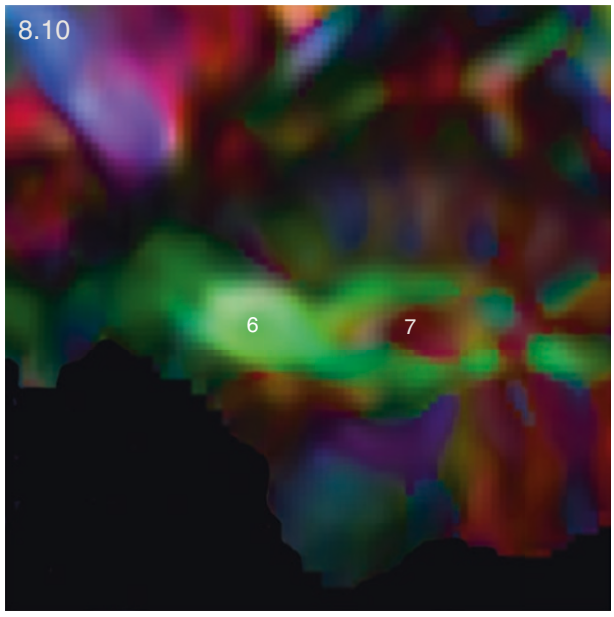
1	Superior cerebellar peduncles
2	Middle cerebellar peduncles
3	Medial lemniscus
4	Decussation of cerebellar peduncles
5	Anterior transverse pontine fibers
6	Posterior transverse pontine fibers
7	Cerebellar vermis







1	Anterior transverse pontine fibers
2	Corticospinal tracts
3	Posterior transverse pontine fibers
4	Medial lemniscus
5	Superior cerebellar peduncles
6	Middle cerebellar peduncles
7	Dentate nucleus



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Infratentorial Vascular Anatomy

9

Salvatore Mangiafico, Andrea Rosi, and Arturo Consoli

The posterior cerebral circulation is comprised of the distal vertebral arteries that join to form basilar artery at the level of the anterior aspect of the medulla oblongata. The basilar artery ends by bifurcating in two posterior cerebral arteries that functionally are a part of the supratentorial circulation (as described in Chap. 3).

The arteries of the posterior circulation are considerably more variable than the supratentorial arteries. The cerebellar arteries ideally form in a bilateral symmetric fashion, but often cerebellar arteries are very small or fail to form at all. Arteries are usually tortuous, and this makes it even more difficult to recognize them on angiography images. In this chapter, we will describe arteries of the cerebellum, brainstem, and basal ganglia [1–6].

S. Mangiafico, M.D. (✉)
Interventional Neurovascular Unit, Careggi University Hospital, Florence, Italy
e-mail: mangiax@libero.it

A. Rosi, M.D.
Residency Program in Radiology, University of Florence, Careggi University Hospital, Florence, Italy
e-mail: andrea.rosi87@gmail.com

A. Consoli, M.D.
Diagnostic and Interventional Neuroradiology Service, Hopital Foch, Suresnes, France

Interventional Neurovascular Unit, Careggi University Hospital, Florence, Italy
e-mail: onemed21@gmail.com

9.1 Major Infratentorial Arteries

9.1.1 Brainstem

The brainstem is vascularized mainly by the basilar artery. The basilar artery arises from the conjunction of the two vertebral arteries at the base of the medulla oblongata. The vertebrobasilar axis and the cerebellar arteries provide for arterial supply of the brainstem, cerebellar hemispheres, and the vermis. Its main branches include:

- Median and paramedian perforating arteries: these perforators penetrate in the brainstem close to and around the anterior sulcus of the brain stem.
- Short (anterolateral) and long (lateral) circumferential arteries: these arteries penetrate in the brainstem more laterally and are also known as collicular arteries in the mesencephalon, short and long pontine arteries, and anterolateral bulbar arteries.

9.1.2 Cerebellum

The cerebellum is vascularized by the distal portion of the cerebellar arteries that—after surrounding the brainstem—spread over to the cerebellar hemispheres and the vermis (Figs. 3.13, 9.1 and 9.2).

Superior cerebellar artery (SCA) arises from the basilar artery in front of the mesencephalon

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just before its bifurcation. It may be a single trunk or a double trunk arising symmetrically from the basilar artery. It passes laterally around the mesencephalon under the third and the fourth cranial nerves. Branches are:

- Marginal artery of the SCA: it is the first cortical branch of the SCA. It arises outside the cerebellar-mesencephalic fissure before the bifurcation of the common trunk. It usually is inconstant, and its importance depends mostly on the concomitant hypoplasia of AICA with whom is anastomosed to supply the anterior hemispheric surface (petrous surface) of the cerebellum. Direct perforators to the middle cerebellar peduncle arise from it.

The ramification of the main trunk of the SCA in its cranial and caudal branches and the emergence of precerebellar arteries take place inside the cerebellar-mesencephalic fissure.

- Precerebellar arteries: these arteries supply the deep territory of the SCA.
- Superior vermian arteries: these are typically two and arise from the cranial (or medial) branch of the SCA and anastomose posteriorly with the inferior vermian arteries of the PICA. They distribute to the superior vermis (and the cerebellar hemisphere close to the paravermian region).
- Cortical arteries: these arise from the hemispheric branch (or lateral) and reach the rest of the cerebellar hemispheric surface (tentorial surface). The most common pattern is the three hemispheric arteries: anterior, medial, and posterior that distribute evenly along the hemispheres.

Anterior inferior cerebellar artery (AICA) originates at the level of the pons, just below the sixth cranial nerve and alongside the seventh and the eighth cranial nerves, reaches the middle cerebellar peduncle, and ends at the level of the petrous surface of the cerebellum. The AICA remains very close to the pons, close to the pontine-bulbar sulcus, and reaches the lateral recess of the fourth ventricle at the level of foramen of Luschka. Branches are classified as follows:

- Perforating arteries of the brainstem.
- Choroidal arteries for the lateral segment of the choroidal plexus and arteries to the cranial nerves. After crossing the seventh and the eighth cranial nerves and after having crossed the foramen of Luschka, AICA surrounds the flocculus and reaches the cerebellopontine sulcus. It terminates at the level of the middle cerebellar peduncle into a rostral and a caudal branch and supplies the petrosal surface of the cerebellum.

The AICA originates in most cases as a single trunk; rarely however it may arise as two branches (duplication). The rostral trunk normally lies above the flocculus reaching the surface of the cerebellum-pontine fissure, whereas the caudal trunk lies inferiorly to the flocculus to supply the petrous surface of the cerebellum. If the PICA is absent, the caudal branch supplies almost the entire ipsilateral hemisphere and the inferior portion of the vermis.

Posteroinferior cerebellar artery (PICA) arises from the vertebral artery, the V4 intracranial segment. More rarely it may originate below the foramen magnum coursing in between the lower cranial nerves. It surrounds the cerebellar tonsils in the cerebello-medullary cistern. At this level, arteries that supply the choroid of the fourth ventricle and the dentate nucleus arise. From here it ascends toward the superior end of the tonsil, passes posteriorly to it to form a cranial loop. It divides into two branches; one reaches medially the inferior vermis, and the other courses laterally in the inferior aspect of the cerebellar lobes.

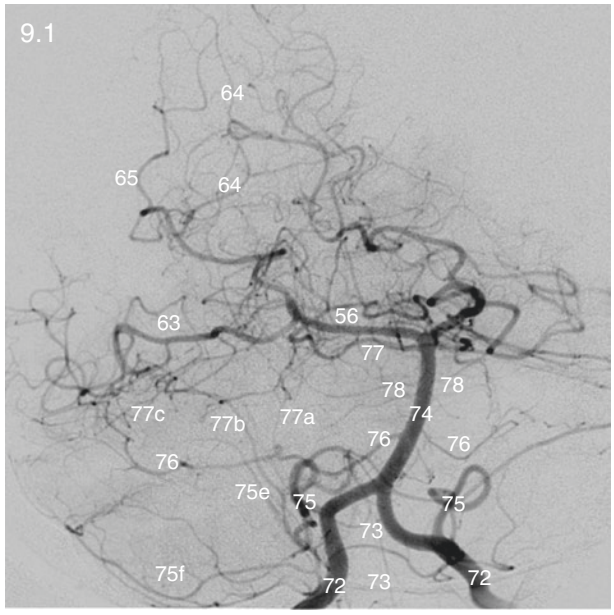

Fig. 9.1 Arteries of the posterior circulation and anterior spinal artery. Oblique projection

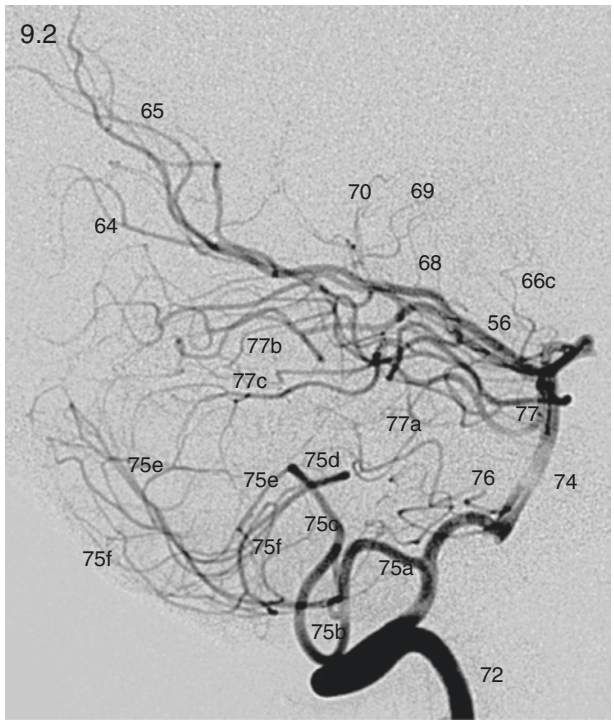

Fig. 9.2 Arteries of the posterior circulation and the posterior choroidal arteries. Lateral projection

56	Posterior cerebral artery (PCA)
63	Posterior inferior temporal artery
64	Parieto-occipital artery
65	Calcarine artery
66c	Perforating thalamic arteries, posterior thalamic or thalamoperforating arteries
68	Thalamogeniculate artery
69	Medial posterior choroidal artery
70	Lateral posterior choroidal artery
72	Vertebral artery (VA)
73	Anterior spinal artery (ASA)
74	Basilar artery (BA)
75	Posterior inferior cerebellar artery (PICA)
75a	PICA, bulbar segment
75b	PICA, tonsillar segment
75c	PICA, telo-velo-tonsillar segment
75d	PICA, cranial loop
75e	PICA, inferior vermian arteries
75f	PICA, hemispheric branches
76	Anterior inferior cerebellar artery (AICA)
77	Superior cerebellar artery (SCA)
77a	SCA, marginal artery
77b	SCA, superior vermian arteries
77c	SCA, hemispheric branches
78	Long pontine arteries

Of note, the degree of variability in the anatomy of the posterior circulation (point of origin of different arteries, the size of the PICA-AICA complex, degree of asymmetry, etc.) often leads to nonstandard vascularization of the brainstem and cerebellum.

9.1.3 Infratentorial Artery Pathology

Occlusion syndromes of the basilar artery:

- Locked-in syndrome (ventral pontine lesion): quadriplegia, aphonia without loss of conscious, and loss of all voluntary movements except for vertical eye movements and blinking
- Top of the basilar syndrome (infarct of the mesencephalon, thalamus, and bilateral parts of the occipital and temporal lobes): somnolence, hallucinations, memory loss, delirium, unilateral or bilateral loss of vertical gaze, nystagmus, oscillatory ocular movements, and visual deficits such as hemianopsia, cortical blindness, and Balint's syndrome (optic ataxia and simultanagnosia)

9.2 Major Infratentorial Veins

9.2.1 Superficial Veins of the Posterior Cranial Fossa

The superficial cortical veins are well recognized over the external surface of the cerebellum that can be further divided into three surfaces: tentorial, petrous, and the suboccipital (Figs. 9.3, 9.4, 9.5 and 9.6).

9.2.1.1 Cerebellar Tentorial Surface Territory

- Superior vermian veins
- Paramedian superior cerebellar veins (anterior group is at the level of the tentorial incisure that drain into the tentorial through the precentral vein, and the posterior group drains into the sinus torcular)
- Subtentorial veins of the lateral surface: cerebellar hemispheric veins drain into the transverse sinus or in the tentorial sinus.

9.2.1.2 Cerebellar Suboccipital Surface Territory

- Inferior hemispheric cerebellar veins and inferior vermian veins drain into the transverse sinus and into the torcular.
- Superficial anterior tonsillar veins drain in the deep venous system by connecting with the veins in the cerebello-medullary fissure and then to the superior petrous vein and then onward into the superior petrous sinus.

9.2.1.3 Cerebellar Petrous Surface Territory

- The superficial veins on the anterior surface of the cerebellum drain into superior petrosal vein, that is, a tributary of the superior petrous sinus and, through this, of the sigmoid sinus.
- The superior petrous vein is formed at the level of the cerebellopontine angle by the conjunction of the transverse pontine vein, cerebellopontine vein, and the vein of the middle cerebellar peduncle.

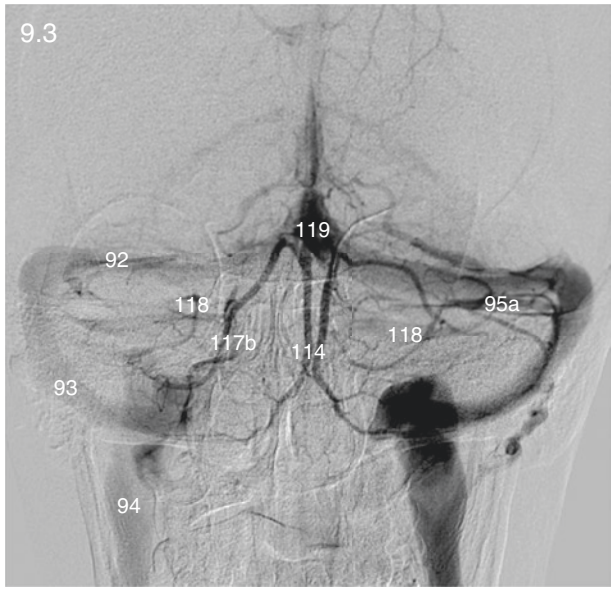

Fig. 9.3 Veins of the posterior cranial fossa. Anteroposterior projection

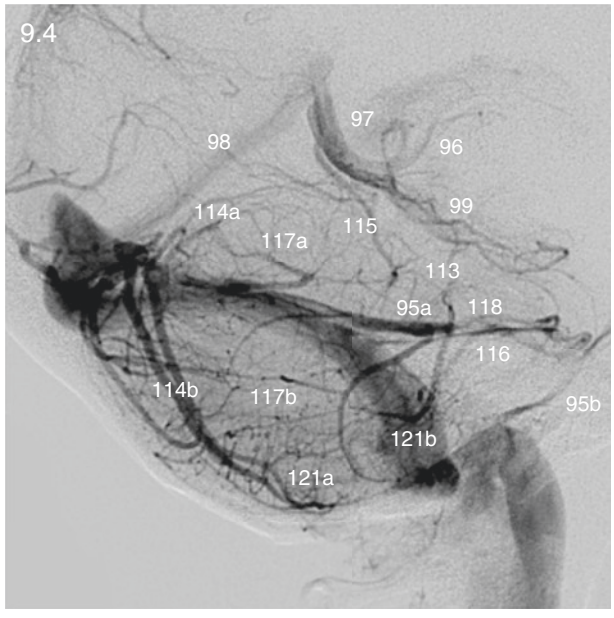

Fig. 9.4 Veins of the posterior cranial fossa, cerebellum. Lateral projection

92	Transverse sinus
93	Sigmoid sinus
94	Internal jugular vein
95a	Superior petrosal sinus
95b	Inferior petrosal sinus
96	Internal cerebral vein
97	Vein of Galen
98	Straight sinus
99	Basal vein of Rosenthal
113	Lateral mesencephalic vein
114	Vermian veins
114a	Vermian veins, superior
114b	Vermian veins, inferior
115	Precentral cerebellar vein
116	Transverse pontine vein
117a	Hemispheric cerebellar veins, superior
117b	Hemispheric cerebellar veins, inferior
118	Petrosal vein
119	Confluence of sinuses or torcular Herophili
121a	Superficial tonsillar veins, posterior
121b	Superficial tonsillar veins, anterior

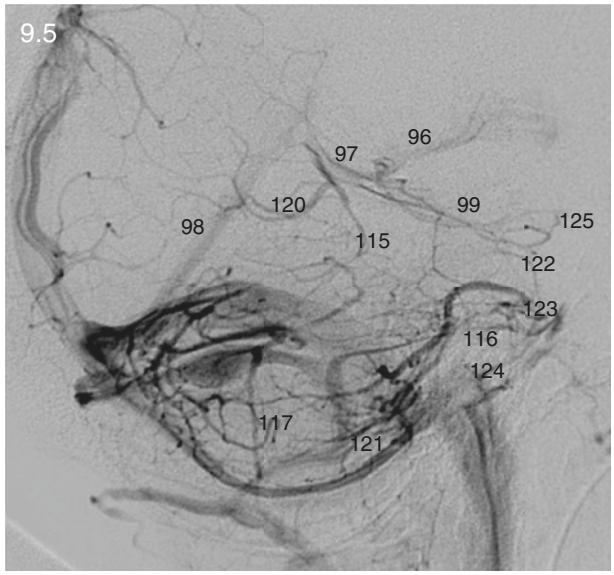

Fig. 9.5 Veins of the posterior cranial fossa, cerebellum, and the brainstem. Lateral projection

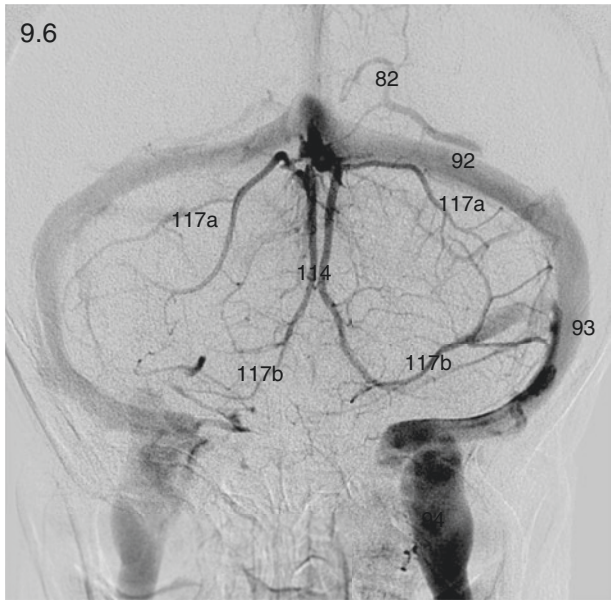

Fig. 9.6 Veins of the posterior cranial fossa. Occipitofrontal projection

82	Occipital veins
92	Transverse sinus
93	Sigmoid sinus
94	Internal jugular vein
96	Internal cerebral vein
97	Vein of Galen
98	Straight sinus
99	Basal vein of Rosenthal
114	Vermian veins
115	Precentral cerebellar vein
116	Transverse pontine vein
117	Hemispheric cerebellar veins
117a	Hemispheric cerebellar veins, superior
117b	Hemispheric cerebellar veins, inferior
120	Internal occipital vein
121	Superficial tonsillar veins
122	Anterior mesencephalic vein
123	Anterior pontine vein
124	Anterior bulbar vein
125	Interpeduncular veins

9.2.2 Deep Vein Territory of the Posterior Cerebral Fossa

The deep venous system is made up of the scissural veins interposed between the cerebellum and the brainstem in the deep cerebellar-mesencephalic, cerebellopontine and the cerebello-medullary fissures. They run parallel to the cerebellar peduncles and surround the fourth ventricle (Figs. 9.4 and 9.5).

9.2.2.1 Cerebellar Deep Drainage

- Cerebello-mesencephalic vein: anastomosis between the cerebello-mesencephalic vein and the lateral mesencephalic vein allows a direct posterior venous drainage to the vein of Galen.
- Cerebellopontine vein: It surrounds the superior cerebellar peduncle and connects the roots of the superior petrous vein, cerebello-medullar vein, and the deep inferior cerebellar hemispheric veins with the superior petrous sinus.
- Cerebello-medullary vein: It lies laterally to the fourth ventricle and connects to the middle cerebellar peduncle and then to the cerebellopontine vein to form the superior petrous vein. These interconnected veins ensure deep ascending venous circulation which together with the vein of the cerebellopontine fissure drains into the precentral cerebellar vein, and the basal vein (through the lateral mesencephalic vein, and the vein of Galen).

9.2.2.2 Brainstem Deep Drainage

- Anterior mesencephalic, pontine, bulbar veins
- Posterior mesencephalic veins: these are quadrigeminal veins that drain into the precentral cerebellar veins
- Lateral pontine territory: lateral transverse pontine vein drains directly into the superficial petrous vein.

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Detailed Anatomy at 7T

10

Isabella M. Björkman-Burtscher,
Karin Markenroth Bloch, and Pia C. Sundgren

The cerebellum and brainstem images presented in this chapter were acquired with an actively shielded Philips 7T Achieva (Best, The Netherlands) MR scanner with a dual-channel transmit and 32-channel receive head coil (Nova Medical, Wilmington, USA). For increased field homogeneity, dielectric pads were used during image acquisition. The axial T2-weighted images were obtained with a turbo spin echo sequence (TSE), repetition time (TR) 4636 ms, echo time (TE) 78 ms, and voxel dimensions of 0.3 × 0.3 × 1 mm.

The increased SNR from 7T MRI scanner was used to increase image resolution, thereby allowing for the better visualization of the internal structures of the brainstem and cerebellum compared to images obtained from clinical 1.5T and 3T MR scanners. The increased in plane resolution of 0.3^2 mm at 7T as compared to 1.0^2 mm in Chap. 7 removes the blurriness and allows for appreciation of the thin interleaved cortical, white matter, and cerebrospinal fluid layers in the cerebellum and vermis.

However, even with the high resolution of 7T MR scans, the images are not sufficient to distinguish the different brainstem nuclei from the fiber tracts that together form a complex network in these densely packed areas of the central nervous system. Furthermore, the appearance of the anatomical structures also depends on fiber orientation, as bundled fibers in plane may appear more clearly as stripes with lower signal intensity on the T2 images compared to nuclei and fibers running oblique or perpendicular to the main magnetic field. This is clearly seen in the decussation of the superior cerebellar peduncle (Fig. 10.1), in the transverse pontine fibers (Figs. 10.5 and 10.6), or the junction between the vermis and cerebellar hemispheres (Fig. 10.9). Finally, 7T MR scans also suffer from imaging artifacts such as flow-related artifacts in the cerebrospinal fluid or more subtle banding artifacts as illustrated in the right cerebellar hemisphere in Fig. 10.7.

I.M. Björkman-Burtscher, M.D., Ph.D. (✉)
Department of Medical Imaging and Physiology, Skåne University Hospital, Lund, Sweden

Department of Diagnostic Radiology, Lund University Bioimaging Center, Lund University, Lund, Sweden
e-mail: isabella.bjorkman-burtscher@med.lu.se

K.M. Bloch, Ph.D.
Lund University Bioimaging Center, Lund University, Lund, Sweden
e-mail: Karin.markenroth_bloch@med.lu.se

P.C. Sundgren, M.D., Ph.D.
Department of Diagnostic Radiology, Clinical Sciences Lund, Lund University, Lund, Sweden

Department of Medical Imaging and Physiology, Skåne University Hospital, SE.221 85 Lund, Sweden
e-mail: pia.sundgren@med.lu.se

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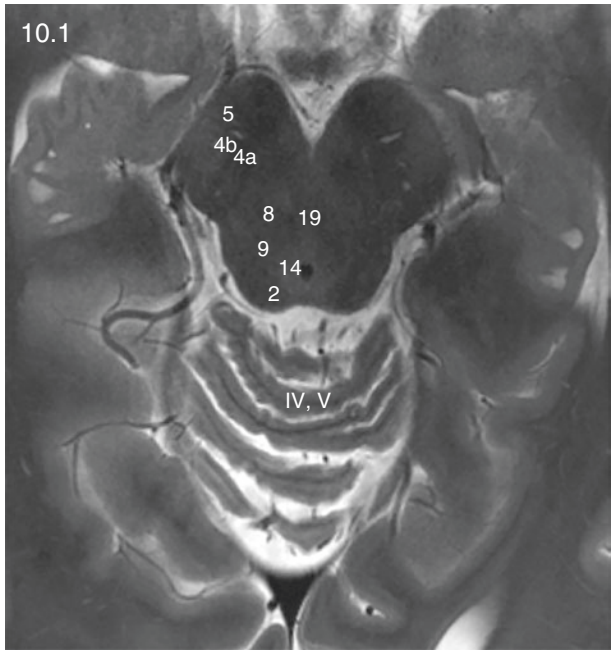
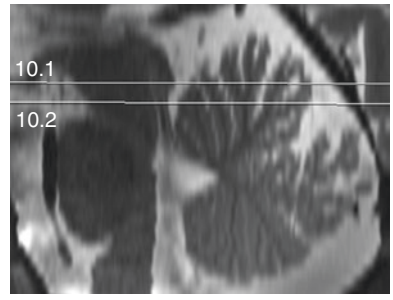
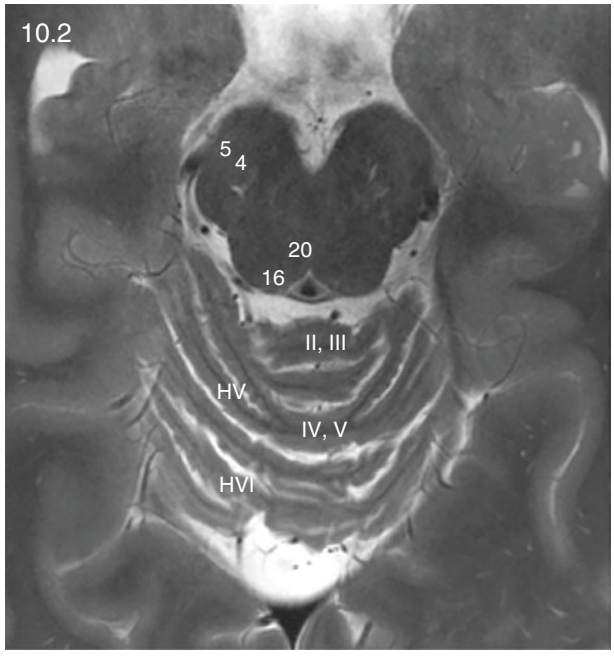
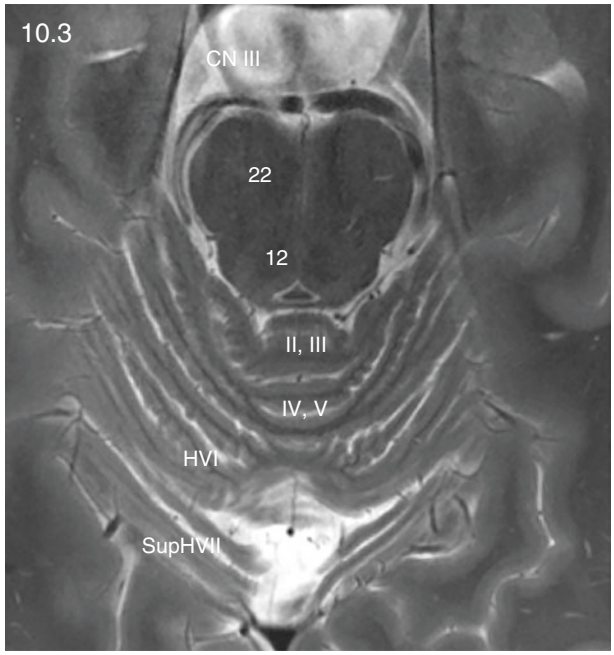
Label numbers are specific to each brain region and are identical to the cerebellum and brainstem labels found in Chap. 7. In this chapter, a more limited number of structures are outlined for anatomical orientation, and therefore not all individual structures from Chap. 7 appear in the figures and related keys. As before, the nomenclature of Larsell [1] was used to label the cerebellum in this chapter. For the sake of brevity, only additional structures not found in Tables 7.2 through 7.3 will be listed in the key for each page.

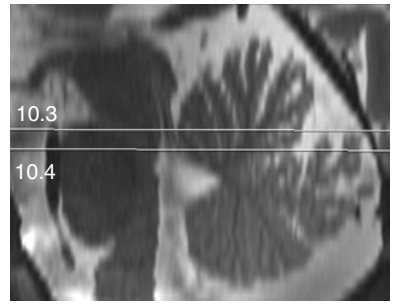




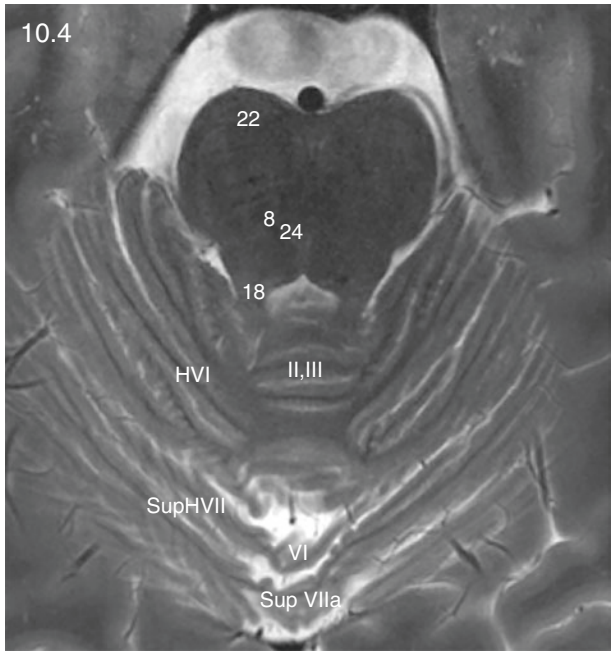
2	Inferior colliculus
4	Substantia nigra
4a	Substantia nigra, pars compacta
4b	Substantia nigra, pars reticulata
5	Cerebral peduncle
8	Medial lemniscus
9	Lateral lemniscus
14	Periaqueductal gray matter
16	Trigeminal complex
19	Decussation of the superior cerebellar peduncle
20	Reticular nuclei

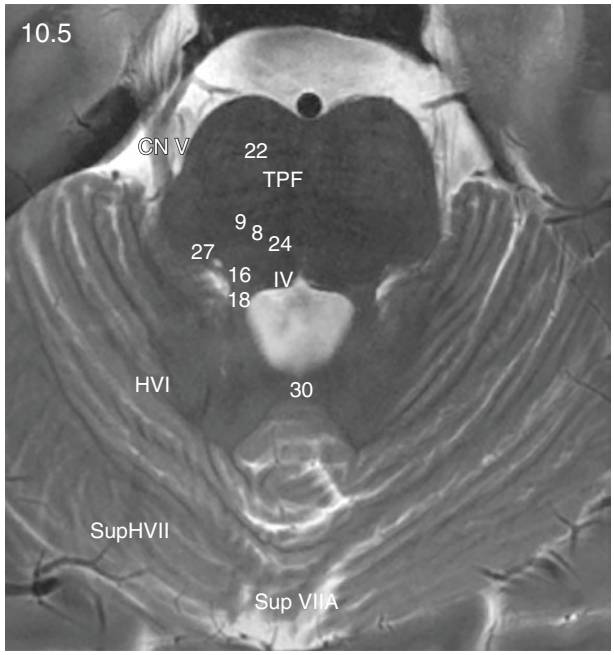


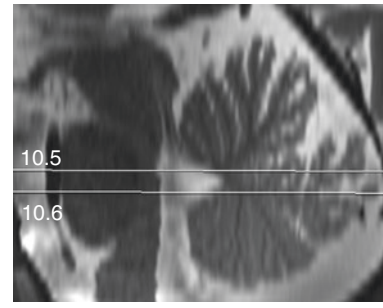




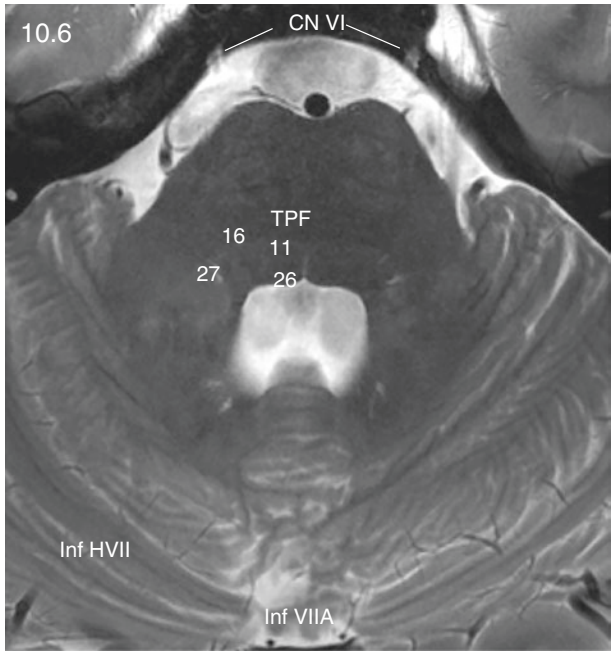
8	Medial lemniscus
12	Medial longitudinal Fasciculus
18	Brachium conjunctivum
22	Pontine nuclei
24	Pontine reticular nuclei

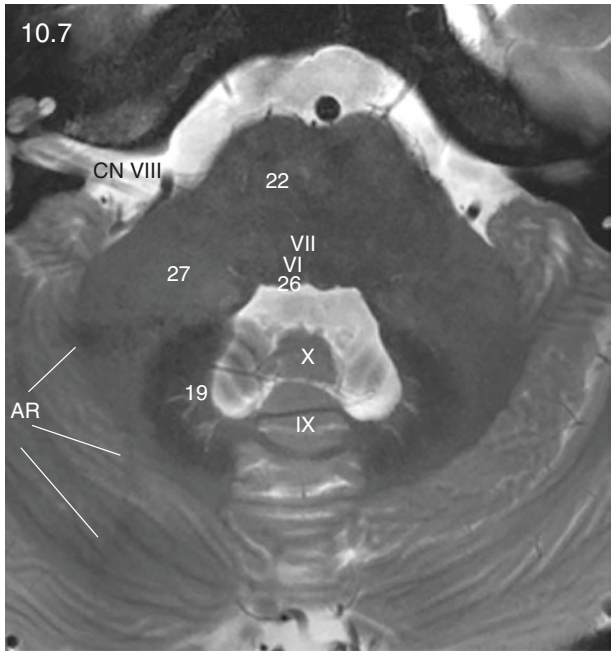


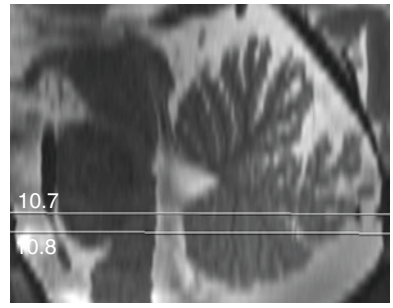




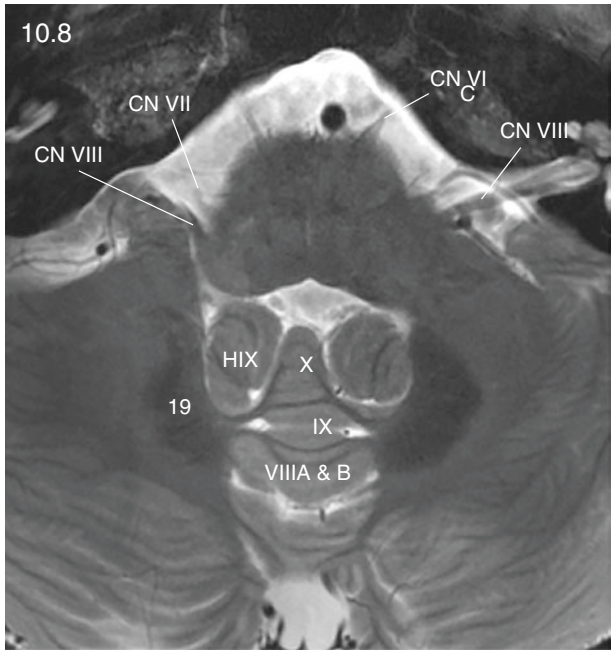
8	Medial lemniscus
9	Lateral lemniscus
11	Central tegmental tract
16	Trigeminal complex
18	Brachium conjunctivum
22	Pontine nuclei
24	Pontine reticular nuclei
26	Facial colliculus
27	Middle cerebellar peduncle (brachium pontis)
30	Corpus medullare
TPF	Transverse pontine fibers
IV	Trochlear nucleus

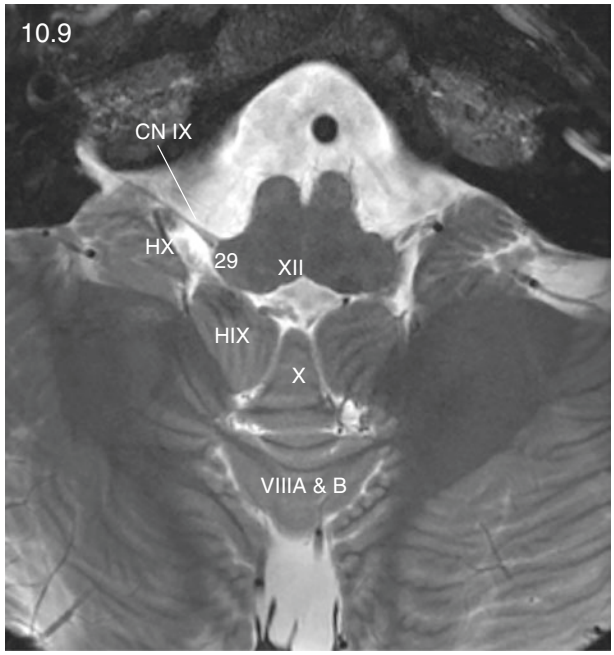


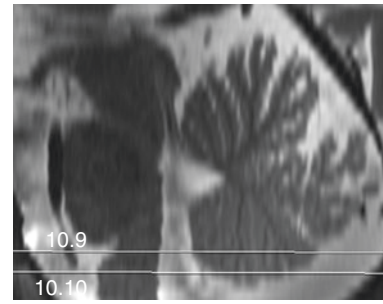




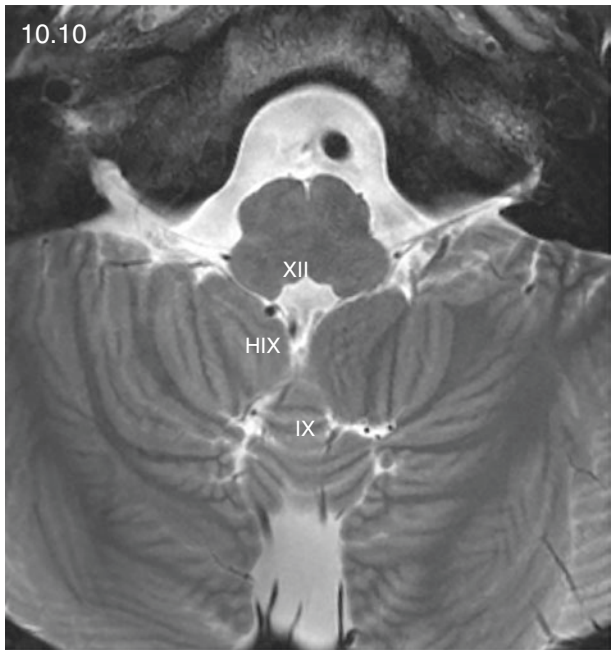
19	Dentate nucleus
22	Pontine nuclei
26	Facial colliculus
27	Middle cerebellar peduncle (brachium pontis)
VI	Abducens nucleus
VII	Facial nucleus
AR	Imaging artifacts







29	Restiform body
XII	Hypoglossal nucleus



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Functional Anatomy of the Cerebellum and Brainstem

11

Nivedita Agarwal

In this chapter, we will briefly review the functional anatomy of the cerebellum and brainstem, cross-referencing Chaps. 7 and 8 with references to relevant figures. We will describe the major functions of each structure and identify specific deficits that can localize disease to a specific brain region. As the longitudinally oriented white matter tracts span more than one part of the brainstem, they are discussed separately at the end of the chapter.

Where possible, important syndromes have been outlined. These descriptions are not intended to be comprehensive, but rather to present a sample of function and pathology. For more comprehensive descriptions, the reader is referred to standard texts of neurology.

N. Agarwal, M.D.
S. Maria del Carmine Hospital, Azienda Provinciale per i Servizi Sanitari, Rovereto (TN), Italy

Center for Mind/Brain Sciences (CIMeC), University of Trento, Rovereto (TN), Italy

Department of Radiology, Section of Neuroradiology, University of Utah, Salt Lake City (UT), USA
e-mail: Nivedita.agarwal@apss.tn.it

11.1 Cerebellum

On axial and coronal images (Figs. 7.1, 7.14), a midline portion or the vermis can be recognized from the more lateral cerebellar hemispheres. In addition, the primary fissure separates a more cranial anterior lobe from the caudal posterior lobe. The posterolateral fissure separates the posterior lobe from the flocculonodular lobe.

11.1.1 Motor Functions

As detailed in Chap. 7 (Table 7.1), the vermis (archicerebellum or spinocerebellum) receives input primarily from the spinal cord. It is important in the control of muscle tone and axial limb movements, maintaining posture of the antigravitational muscles. Asymmetric homotopic representation of the sensorimotor cortex is represented in the vermis and the paravermis. The flocculonodular lobe (paleocerebellum, vestibulocerebellum) is heavily interconnected with the vestibular nuclei and brainstem nuclei for the important head and eye movement coordination.

11.1.2 Non-motor Functions

The cerebellar hemispheres (neocerebellum) receive input from the brain through the pontine nuclei. These areas are responsible for the non-motor functions of the cerebellum such as cognition,

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language and emotion processing, and modulation. fMRI studies have identified specific cerebellar regions that process cognitive and emotional information. In addition to a precise homotopic mapping of the cognitive functions, a functional asymmetric representation of executive function is also maintained in the cerebellum. For instance, language is lateralized to the right posterior hemisphere. Visuospatial functions appear to be lateralized more to the left cerebellar hemisphere.

11.1.3 Functional subdivisions

Anatomical subdivision predominantly considers cerebellar nomenclature of Ito [1] and Larsell [2], as detailed in Chap. 7 and seen in Figs. 7.1–7.3.

Lobule I: specific function is unknown. Lesions in the lingula of primates disrupt vestibulocerebellar and spinocerebellar integration, resulting in axial muscle disequilibrium [3]. A malformed thick lingula may represent an endophenotype to increased risk of alcohol and substance use by creating enhanced tolerance to these drugs [4].

Lobules II–V (anterior lobe): the primary sensorimotor region of the cerebellum is located in this lobe and in the adjacent part of lobule VI [5]. The anterior lobe also receives input from the prefrontal cortex (area 46) for skilled motor functions. Sensorimotor homunculi have been precisely mapped to this lobe together with the paravermian area [6]. Right-handed finger tapping, flexion-extension, and pronosupination tasks will activate right lobules IV, V, and VIII.

Lobules VI and VII: receive contralateral input mostly from association areas such as prefrontal cortex, posterior parietal, superior and middle temporal areas, cingulate gyrus, and retrosplenial cortex [7, 8]. Thus, most cognitive tasks such as verb generation, mental rotation, and 2-back tasks for memory can activate both VI and VII lobules. Lobule VI also represents the language motor articulatory apparatus (Urban 2003). Lobules VI and VII are also considered the "oculomotor vermis" involved in controlling eye movements.

Lobule VIII: secondary sensorimotor region of the cerebellum [9].

Lobule IX: essential for visual guidance of movement. Recent functional connectivity magnetic resonance imaging (fcMRI) data indicate that it contributes to the default mode network [10].

Lobule X: gaze stability, smooth pursuit, and vestibulo-ocular reflex. These require active inhibition of unwanted saccades. Eyeblink conditioned response requires memory of eye movement partly stored in the vestibulocerebellar regions.

Higher level cognitive functions mainly are subserved mainly by the cerebellar hemispheres (Crus I and Crus II) and partly by some lobules in the vermis such as lobule VI, Lobule VIIA and VIIB). Language areas are predominantly represented in the right hemisphere regions of lobule VI, Crus I, and Crus II. Working memory tasks activate bilateral regions of lobule VI and Crus I and parts of VIII with cognitive areas situated laterally. Affective and emotional functions limbic cerebellum such as response to pain, panic, and air hunger activate most midline vermis, lobule VI, and Crus I [9].

Oculomotor regions of the cerebellum include lobules VI, VII, and IX together with the fastigial nucleus, Crus I and Crus II, flocculonodular lobe, and the uvula. These areas help maintain steady gaze. They are involved in the control of saccades, horizontal smooth pursuit, and the vestibulo-ocular reflex (eyes move conjugately in the opposite direction to horizontal head motion). While specific cerebellar regions have not yet been identified, brainstem-cerebellar circuits underlie conditioned and unconditioned eye blinking. Eye blinking has also been linked to cerebellar mechanisms of learning through reciprocate connections with the amygdala and other limbic areas [11].

Three pairs of pontine nuclei are present in the deep white matter of the cerebellum (Fig. 7.9). The *dentate nuclei* are involved in speech control, activate strongly with increasing complexity of sensorimotor tasks such as discrimination tasks, motor grip control, and are only weakly involved during simple motor tasks [12]. Multiple non-motor tasks also involve the dentate such as visuospatial tasks and verbal working memory. The *fastigial nuclei* are important in programming eye saccades. The *interpositus (emboliform*

and globose) nuclei are involved in eye blinking responses and critical to learning and memory of conditioned responses [13]. Limb movement coordination is also modulated by these nuclei, and lesions may lead to limb ataxia.

Three pairs of peduncles supply input and output to and from the cerebellum (Figs. 7.12, 8.2–8.4). The *inferior cerebellar peduncles (restiform bodies)* are composed of fibers that receive and transmit signals to and from the spinal cord and brainstem nuclei, specifically, sensory proprioceptive signals from the spinocerebellar tracts and motor vestibular-ocular signals from the brainstem nuclei. The *middle cerebellar peduncles (brachium pontis)* are composed of fibers that carry input from the contralateral cerebral hemisphere, mainly relayed through the pontine nuclei. The *superior cerebellar peduncles (brachium conjunctivum)* are composed of fibers that carry primarily output signals to the contralateral cerebral hemisphere relayed through the red nucleus.

11.1.4 Cerebellar Pathology

Motor deficits of the cerebellum include primarily sensorimotor and oculomotor deficits. *Sensorimotor deficits* involve disorders of timing, lack of control of corticomotor excitability, deficits in sensorimotor synchronization, and making motor predictions that impair coordination of movement (dysmetria, dysdiadochokinesia). These result in typical "cerebellar-like" disequilibrium with lack of control of axial muscles, gait ataxia, and impaired coordination (dysmetria) of the extremities [11]. Ataxic dysarthria, often seen in patients with cerebellar stroke that damage the muscles of the phonatory apparatus, is pathologic speech characterized by slow, monotonous, scanned, indistinct, jerky, explosive, and slurred production due to alterations in phonation and articulation of consonants and vowels. *Oculomotor deficits* involve loss of the vestibulo-ocular reflex in comatose patients with brainstem damage resulting in "doll's eyes" (eyes do not move to the opposite direction when the head is turned sideways). Lesions in the flocculus can cause gaze-evoked nystagmus. Lesions in the nodulus can result in periodic alternating nystagmus. Stroke and tumors in the cerebellum can cause ipsilateral reduction of the eyeblink-conditioning reflex. Similarly, cerebellar cortical degeneration and other diseases involving reduction of eyeblink conditioned response should point to a cerebellar involvement.

Non-motor deficits of the cerebellum, forming constellations of symptoms, are grouped into syndromes. Lesions in the dentato-thalamocortical pathway and emotional/affective loops lead to *"dysmetria" of thought*, with impairments of the cerebellar modulation of intellect and emotion [14]. Several levels of language deficits may occur in different neurodevelopmental, neurodegenerative and vascular diseases leading to ataxic dysarthria, disprosody and speech apraxia [15]. Neurodevelopmental abnormalities such as autism and schizophrenia are associated with cerebellum maldevelopment [16].

Cerebellar mutism syndrome (CMS) is characterized by the inability to produce and process speech without apraxia or aphasia typical of cerebral cortical lesions. Severe dysarthria and/or anarthria, unrelated to damage to muscles of the phonatory apparatus, leads to loss of verbal fluency and modulation of speech [17]. Lesions in the posterior midline and paramedian vermis, dentate nuclei, and superior cerebellar peduncle can result in CMS [18]. CMS is mostly reversible but may result in a more chronic complex cognitive and affective dysfunction.

Cognitive cerebellar affective syndrome (CCAS) is chronic changes in cognition and affect due to damage to midline structures of the posterior vermis and parts of the posterior lobe. CCAS is characterized by symptoms including: (a) impairment of executive functions such as deficient planning, verbal fluency, abstract reasoning, and working memory, (b) impaired visuospatial memory, (c) personality changes with blunting of affect and disinhibited and inappropriate behavior, and (d) la nguage deficits including agrammatism, mild anomia, and dysprosodia [19]. This is not accompanied by "cortical" symptoms such as aphasia, agnosia, and apraxia.

Rostral vermis syndrome (anterior lobe syndrome) is characterized by selective atrophy of the anterior vermis. The posterior or caudal vermis and most of the cerebellar hemispheres appear normal. The most common symptoms are wide-based stance and hesitating gait, gait ataxia and little or no involvement of the arms, speech, and ocular motility (arm incoordination, nystagmus, and dysarthria). This syndrome is best exemplified in patients with chronic alcoholic degeneration.

Caudal vermis syndrome occurs with damage to the flocculonodular lobe. It is characterized by axial disequilibrium and staggering gait, markedly impaired tandem gait, little or no limb ataxia, and spontaneous nystagmus and head rotation to the side. This syndrome is more commonly seen in pediatric populations with midline tumors such as medulloblastoma.

Cerebellar hemispheric syndrome is a severe constellation of symptoms including severely affected coordination of ipsilateral limbs as well as fine tremor arising only during voluntary movement. This syndrome is more commonly seen with cerebellar neoplasms, infarcts, and hemorrhage.

Pancerebellar syndrome presents with bilateral signs of cerebellar dysfunction characterized by various degree of signs and symptoms including incoordination of limbs, nystagmus, and dysarthria.

11.2 Brainstem

The brainstem develops from caudal two of the three embryological vesicles: the more rostral mesencephalon (that develops into the midbrain) and the rhombencephalon. The rhombencephalon further subdivides into the metencephalon (that develops into the pons and cerebellum) and the myelencephalon (that develops into the medulla oblongata). All nuclei and tracts respect a columnar organization along the rostral-caudal axis of the brainstem. Thus, it is important to remember that some cranial nerve nuclei and most white matter tracts run through the entire length of the brainstem. It is a combination of symptoms that helps to localize pathology in one of the three parts of the brainstem.

In the developing central nervous system, a basal plate and an alar plate are formed delimited by the sulcus limitans. The alar plate is the dorsal part of the neural tube, whereas the basal plate is the ventral portion of it. The alar plate continues caudally into the sensory dorsal part of the spinal cord, and the basal plate continues to form the motor part of the spinal cord. In the brainstem the alar plates move out laterally and contain the general somatic and general and special visceral afferents of the cranial nerves. Ascending sensory tracts are also seen more laterally. The basal plate contains the motor axons and contains the general somatic and general and special visceral efferents. The motor descending fibers are also mostly found in the medial part of the brainstem. The brainstem contains most of the encephalic reticular centers. The brainstem reticular nuclei are divided into three longitudinal columns: median, central (or medial), and lateral. Median nuclei are the raphe nuclei and are cholinergic, locus coeruleus contains noradrenergic fibers and the periaqueductal gray matter (PAG), and the reticular nuclei are mostly serotoninergic.

In this chapter, the contents of each brainstem section have been further subdivided into gray matter and white matter structures for easy reference. The functions of the cranial nerves have been detailed separately in Chaps. 12–23 and will not be further discussed in this chapter. The function of the white matter tracts will be discussed at the end of the chapter.

11.2.1 Midbrain Function

Superior colliculi (SC): The SC receive their primary input from retinal visual pathways and the frontal eye fields. Their most important function is controlling eye movement in "egocentric" (body specific) space allowing for voluntary saccadic movements. The SC also receive sensory input from other modalities such as auditory and somatosensory, helping to orienting head and

eyes toward objects that are voluntarily seen or heard [20].

Inferior colliculus (IC): Tonotopic synapses in the auditory system. The IC receives input bilaterally from the dorsal cochlear nuclei. There is a certain degree of asymmetry in the input, with ipsilateral fibers being more numerous. Auditory information is then transmitted to the ipsilateral medial geniculate body. The IC also receives and sends information through multiple parallel loop connections to the ipsilateral auditory cortex. The IC represents the main multisensorial hub and allows for the startle response. It is important in the spatial localization of sound [21]. Contralateral nonpulsatile tinnitus, diminished capability to localize sounds, and presbycusis all have been associated with IC pathology.

Pretectal nuclei: A group of nuclei just anterior to the superior colliculi in the midbrain and has extensive connections with the thalamus, Edinger-Westphal nucleus, pons, and inferior olive to name a few. They receive direct input from the retinal ganglion cells and are important in the classic pupillary light reflex. They allow eyes to follow a moving object (smooth pursuit) and allow for the accommodation reflex (through the CN III). They also participate in REM sleep [22].

Edinger-Westphal nuclei: Parasympathetic center for motor innervation of intraocular muscles, constriction of pupil, and accommodation reflex [23].

Red nucleus (RN): The RN is involved in motor coordination. Major inputs are from the contralateral cerebellar hemisphere and ipsilateral motor cortex. Major outputs are to the inferior olive (in the medulla), the cerebellum (via

Overview of Main Structures in the Midbrain

Major landmarks
- Upper boundary of the midbrain is the plane of the mammillary bodies and superior colliculi; lower boundary is the plane of the inferior colliculi and pons.
- Axial (Figs. 7.15, 7.16): the dorsal tectum (or the quadrigeminal plate), the central tegmentum, and the ventral or basal segment that is formed by the cerebral peduncles.

Major Nuclei
- Cranial Nerve
  - Motor
    Oculomotor nuclei (III) (Chap. 14)
    Trochlear nerve nuclei (IV) (Chap. 15)
    Pretectal nuclei
    Edinger-Westphal nuclei: parasympathetic center for motor innervation of intraocular muscles, constriction of pupil, and accommodation reflex (Chap. 14)
  - Sensory
    Mesencephalic trigeminal nuclei (V) (Chap. 16)
- Superior colliculi
- Inferior colliculi
- Red nuclei
- Substantia nigra
- Periaqueductal gray matter
- Ventral tegmental area
- Reticular activating system
  - Median centralis superior (median raphe nucleus)
  - Nucleus raphe dorsalis (dorsal raphe nucleus)

Major Tracts
- Motor tracts
  - Cerebral peduncles
  - Tectospinal tract
  - Rubrospinal tract
  - Medial longitudinal fasciculus
- Sensory tracts
  - Medial lemniscus
  - Spinothalamic tract
  - Lateral lemniscus
  - Central tegmental tract

the central tegmental tract, forming the "triangle of Guillian and Mollaret"), and the spinal cord (rubrospinal tract) [24]. The rubrospinal tract is more important in primates and may be vestigial in humans.

Substantia nigra (SN): The substantia is anatomically divided into a more medial *pars compacta* (SNpc) and a more lateral *pars reticulata* (SNpr). The SNpc sends dopaminergic output to striatum, initiating movement by disinhibiting thalamocortical pathways. The SNpc is also important in habit learning, goal-directed behavior, reward, pleasure, addictive behavior, and sleep-wake circadian cycle [25]. The SNpr sends GABAergic signals to the basal ganglia and other brain structures including the superior colliculus, modulating saccadic eye movement. Subthalamic nuclei may enhance spontaneous firing of SNpr neurons and are an important site for deep brain stimulation in patients suffering from tremor, rigidity, and bradykinesia [26].

Ventral tegmental area (VTA): The VTA is a group of gray matter nuclei in the ventral part of the mesencephalic tegmentum that give rise to dopaminergic mesolimbic (nucleus accumbens, SNpc, hippocampus, amygdala, olfactory cortex) and mesocortical (prefrontal cortex) reward mechanism circuitry.

Periaqueductal gray matter (PAG): The PAG contains opioid receptors. Spinothalamic tracts carrying pain and temperature information reach PAG. Together with the activation of the raphe nuclei, neurotransmitters and neuromodulators such as encephalin, substance P, and serotonin are released, activating neurons in the VPL of the thalamus reducing pain input. It is one of the sites of deep brain stimulation in chronic neuropathic pain. The PAG is also part of neuronal circuits underlying the expression of positive symptoms [27] in schizophrenia and manic depression. PAG activity may also be involved in maternal behavior through activation of oxytocin and vasopressin activity of the hypothalamus and the orbitofrontal cortex [28].

Reticular activating system (RAS): The RAS is an ill-defined group of different interconnected nuclei in the brainstem. The RAS includes raphe nuclei in the median part of the brainstem as well as the gigantocellular and parvocellular nuclei in the more lateral part of the brainstem. The RAS regulates cardiovascular and respiratory function, organizes eye movement, regulates sleep-wake cycle, and determines level of consciousness/arousal [29, 30]. It also helps maintain balance and posture.

The raphe nuclei are serotoninergic nuclei within the RAS that project to the medial forebrain and hypothalamic nuclei. They are involved in different types of reward systems and sleep-wake cycle. Dysfunction of the raphe nuclei (in particular dorsal raphe nucleus) is implicated in depression and obsessive-compulsive disorder and can cause narcolepsy and cataplexy [31].

11.2.2 Midbrain Pathology

Lesions in the midbrain cause several different symptoms including paralysis of vertical gaze, diplopia (double vision due to oculomotor and trochlear nerve weakness), rubral tremor, loss of conjugate movements in the horizontal gaze, altered consciousness, and akinetic mutism. While these symptoms can occur in solitary, more often they occur as constellations of symptoms that can be grouped into syndromes.

Benedikt syndrome: Involves damage to the RN + oculomotor nucleus. Presents with ipsilateral complete third cranial nerve palsy and contralateral involuntary motor movements such as hemiathetosis, hemichorea, or intention tremor.

Claude's syndrome: Involves damage to the RN + brachium conjunctivum + oculomotor nucleus. Presents with ipsilateral complete third cranial nerve palsy and contralateral cerebellar signs (asynergia, ataxia, dysmetria).

Nothnagel's syndrome: A variant of Claude's syndrome with sparing of the fascicular third cranial nerve palsy.

Weber's syndrome (superior alternating syndrome): Involves damage to the cerebral peduncles + SN + oculomotor nucleus. Presents with complete ipsilateral third cranial nerve palsy, contralateral hemiparesis, and Parkinson-like symptoms.

Top of the basilar syndrome: It is a complex syndrome involving the midbrain, thalamus, and parts of temporal and occipital cortex. Symptoms vary and include various disorders of eye movements, anisocoria, mild somnolence to deep coma, agitated delirium, hemianopsia, and cortical blindness.

Neuromyelitis optica (*Devic's syndrome*): Caused by aquaporin-4 autoantibodies that selectively damage astrocytes in the PAG, leading to PAG neuronal dysfunction. Symptoms originally described include acute monophasic loss of monocular vision (optic neuritis) and myelitis. However, patients may present with either of the two with or without brain involvement. Rarely brain lesions may be associated with the NMO spectrum, and they are usually in areas rich with aquaporin channels such as in the area postrema, hypothalamus, nucleus of tractus solitarius, and peri-ependymal region of the ventricular system mostly around the third and the fourth ventricles [32].

Parinaud syndrome (*Koerber-Salus-Elschnig syndrome, dorsal midbrain syndrome*): Due to a lesion of the superior colliculi. Causes vertical gaze paralysis. Increased intracranial pressure due to obstructive hydrocephalus or a failed ventriculoperitoneal shunt can also cause bilateral downward gaze, also known as the *sun-setting sign* [33]. Bilateral damage to pretectal nuclei can also cause Argyll Robertson pupils that are characterized by bilateral small pupils that can accommodate to near vision but do not react to bright light.

Rubral or *Holmes tremor*: Due to a lesion in the red nucleus. Presents with a fine low frequency tremor at around 4.5 Hz both at rest and intentional. This tremor may respond to levodopa treatment.

Progressive supranuclear palsy: Occurs with selective neurodegeneration of the brainstem tegmentum and tectum. Patients typically present with postural instability, pseudobulbar palsy (dysarthria and dysphagia), slow saccadic eye movements, supranuclear gaze palsy, postural rigidity, and dysexecutive frontal syndrome. Neurodegeneration is seen in the substantia nigra, dorsal midbrain tectum, subthalamic nuclei, and other diencephalic structures [34].

11.2.3 Pons Function

Nucleus (or locus) coeruleus (NC or LC): The NC nuclei are in the dorsal pons at the pontomesencephalic junction just lateral to the caudal end of the aqueduct of Sylvius. The LC lies just below the floor of the IV ventricle and contains neuromelanin. It is the largest noradrenergic nuclei in the brain and has extensive connections with all parts of the brain. Noradrenergic receptors are widely distributed in the neocortex, basal forebrain, limbic system (amygdala, hippocampus), hypothalamus, several parasympathetic cranial nerve nuclei, and reticular formation. The LC provides noradrenergic modulation to brain areas, modulation that promotes wakefulness, increases level of arousal, improves memory retrieval, and modulates autonomic/neuroendocrine functions [35].

Pedunculopontine nuclei (PPN): The PPN are a collection of neurons arranged longitudinally in the rostral pons and the lower mesencephalon tegmentum. The nuclei are medially bordered by

Overview of the Main Structures in the Pons

Major landmarks

- Upper boundary of the pons is the plane of the inferior colliculi; lower boundary is the plane of the pontomedullary junction.
- Axial (Figs. 7.17–7.19).

Major Nuclei

- Cranial Nerve
  - Motor
    Trigeminal nuclei (V) (Chap. 16)
    Superior salivatory nuclei: (Chap. 18)
    Inferior salivary nucleus: (Chap. 20)
    Abducens nuclei (VI) (Chap. 17)
    Facial nuclei (VII) (Chap. 18)

  - Sensory
    Mesencephalic trigeminal nucleus (V) (Chap. 16)
- Nucleus (locus) coeruleus
- Pedunculopontine nuclei
- Pontine nuclei
- Superior olivary nuclei
- Raphe nuclei
  - Median: nucleus raphe magnus, nucleus raphe pontis, and nucleus reticularis centralis superior
  - Central: nucleus reticularis pontis caudalis, nucleus reticularis pontis oralis, and nucleus reticularis tegmenti pontis (centralis inferior)
  - Lateral: nucleus reticularis parvocellularis (extension of the nucleus reticularis medullae oblongata and centralis into the pons), medial parabrachial nucleus, and lateral parabrachial nucleus
- Paramedian pontine reticular formation

Major Tracts
- Transverse pontine fibers (pontocerebellar fibers)
- Corticospinal tracts
- Spinothalamic tracts
- Middle cerebellar peduncle (brachium pontis)
- Superior cerebellar peduncle or the brachium conjunctivum
- Lateral lemniscus
- Medial lemniscus

the superior cerebellar peduncles and the central tegmental tract. The medial lemniscus borders the nuclei laterally [36]. The nuclei are considered important components of the reticular activating system. They are important for the initiation and the maintenance of gait. In patients with advanced Parkinson's disease including akinesia [37], the PPN serve as targets for deep brain stimulation.

Superior salivatory nuclei: Vasodilatory parasympathetic preganglionic fibers from these visceromotor nuclei innervate the nasal and palatine salivary glands as well as the lacrimal gland through vidian nerve and the sublingual and submandibular salivary glands through the chorda tympani.

Inferior salivatory nuclei: Lie just caudal to the superior salivatory nucleus. They contain vasodilatory and secretory-motor preganglionic parasympathetic fibers to the parotid glands, running through the tympanic nerve and tympanic plexus, eventually reaching the otic ganglion.

Pontine nuclei (PN): The pontine nuclei contain mostly GABAergic neurons that receive selective input from corticopontine fibers and send their output to specific cerebellar deep nuclei and folia (mossy fiber afferents) [38]. The majority of pontocerebellar projections are contralateral. These projections primarily control the motor activity in the contralateral cerebellar hemisphere. Visual and somatosensory cortical areas also project to the pontine nuclei.

Superior olivary nuclei (SON): The SON are highly specialized nuclei positioned in the lower pontine tegmental area. They play an important role in auditory processing, computing interaural time differences and interaural sound intensity level differences. The SON are the first major sites of binaural convergence of sounds. They are intimately connected to the trapezoid body, lateral lemniscus, and cochlear nuclei.

Raphe nuclei: These nuclei found in the pontine tegmentum are part of the reticular formation and share function similar to those in the mesencephalon (see midbrain above). Most raphe projections to the cerebellum are bilateral and involved in bilateral motor activity [39].

Paramedian pontine reticular formation (PPRF): Together with the raphe nuclei and the reticular formation in the mesencephalon, they widely project to other brain areas and contribute to the state of arousal. The PPRF lies just anterior to the MLF and receives input from the superior colliculus and the frontal eye fields. It is important in coordinating horizontal and vertical saccades.

11.2.4 Pons Pathology

Lesions in the pons cause several different symptoms including rubral tremor, hearing loss, facial nerve weakness/paralysis, gaze weakness, reduced arousal, and internuclear ophthalmoplegia. Like the midbrain, symptoms often occur in complexes depending on the size and region of the pons that is damaged.

More focal lesions result in more focal deficits. Damage to the NC results in sleep disorders, reduced levels of arousal (coma), and memory retrieval failure in neurodegenerative diseases such as Parkinson's, Alzheimer's, Lewy body, and Huntington's diseases, amyotrophic lateral sclerosis, and Rett syndrome [40]. Opiate withdrawal symptoms are caused in part by increased noradrenergic activity in the NC. Damage to the pedunculopontine nuclei may affect planning of movements, changes in arousal, attention, and reward-seeking behavior. Ipsilateral hearing loss, elevated threshold on audiograms, and impaired speech discrimination [41, 42] can result from injury to the superior olivary nuclei. Finally, damage to the medial longitudinal fasciculus results in internuclear ophthalmoplegia characterized by normal conjugate gaze toward the side of the lesion and loss of adduction of the ipsilateral eye when looking toward the opposite eye accompanied by nystagmus of the opposite eye.

Larger lesions involve more pontine structures and are grouped into syndromes.

Locked-in syndrome: Involves bilateral lesions in basis pontis + corticobulbar tract. Results in quadriplegia and aphonia, sparing vertical eye movements.

Ventral pontine syndrome (*Millard-Gubler syndrome*): Results in contralateral hemiplegia, ipsilateral diplopia in the horizontal gaze "toward" the lesion (lateral rectus paresis), and ipsilateral peripheral facial nerve paresis (may be spared in ventral median lesions, also called Raymond syndrome).

Dorsal pontine syndrome (*Raymond-Cestan syndrome*): Results in contralateral hemiplegia, conjugate horizontal gaze paralysis (gaze away from the lesion), cerebellar ataxia, coarse rubral tremor and contralateral hypoesthesia of the face and extremities, and internuclear ophthalmoplegia (due to lesion of the MLF).

Lateral pontine syndrome (*Marie-Foix syndrome*): Results in ipsilateral cerebellar ataxia (red nucleus and superior cerebellar peduncle), contralateral hemiparesis (corticospinal tract), and contralateral hemihypoesthesia for pain and temperature (spinothalamic tract).

Inferior medial pontine syndrome (*Foville syndrome*): Results in dysarthria (clumsy hand syndrome), ataxic hemiparesis, rare pseudobulbar symptoms, and "one-and-a-half syndrome" (lesion in the ipsilateral PPRF or MLF) [43].

Functional Anatomy of the Medulla

Major landmarks
- Upper boundary of the medulla is the plane of the pontomedullary junction; lower boundary is the plane at the caudal end of the medullary olives.
- Axial (Figs. 7.20–7.22).

Major Nuclei
- Cranial Nerve
  - Motor
    - Nucleus ambiguus (IX, X, and XI)
    - Dorsal motor nucleus of the vagus (parasympathetic component of the X)
    - Hypoglossal nucleus (XII)
  - Sensory
    - Nucleus of the solitary tract (nervus intermedius, IX, X)
    - Spinal trigeminal nucleus (V)
      - Pars oralis (nucleus in the pons)
      - Pars interpolaris (sends fibers to the cerebellum)
      - Pars caudalis (pain and temperature fibers from the face)
    - Cochlear nuclei (VIII)

Dorsal
Ventral
Vestibular nuclei (VIII)
Medial
Inferior
- Inferior olivary nuclei
- Area postrema
- Arcuate nuclei (belongs to the pontine nuclei)
- Nucleus prepositus hypoglossi
- Raphe nuclei
- Reticular formation
  - Central column: nucleus reticularis gigantocellularis
  - Lateral column: nucleus reticularis medullae oblongata centralis and nucleus reticularis lateralis (nucleus funiculi lateralis)

Major Tracts
- Restiform body (inferior cerebellar peduncle)
- Medial lemniscus
- Spinothalamic tract
- Medullary pyramids (corticospinal tract) before decussation
- Central tegmental tract

11.2.5 Medulla Function

Inferior olivary nuclei (ION): The ION serve as a major source of input to the cerebellum (climbing fibers). Olivocerebellar fibers decussate and reach the contralateral cerebellum through the inferior cerebellar peduncle. Major function includes fine control of motor timing and spatial learning and in time perception [44].

Area postrema (AP): The AP is one of the seven circumventricular organs devoid of the blood-brain barrier which puts it directly in contact with the main bloodstream. It is just anterior to the obex in the floor of the IV ventricle. It is connected to the nucleus of the solitary tract and the dorsal nucleus of the vagus. It is stimulated by the visceral afferents in the gastrointestinal tract and plays an important role in autonomic functions. Stimulation of the area postrema generates the sensation of nausea.

Arcuate nuclei: These nuclei are in the ventromedial region of the medulla. They are respiratory chemosensitive nuclei capable of directly sensing CO_2 levels in the CSF space.

Nucleus prepositus hypoglossi (NPH): Holds the position of the gaze once the eye has reached its final position. NPH together with the flocculus and the medial vestibular nucleus form the "neural integrator" of horizontal conjugate eye movement.

Nucleus reticularis gigantocellularis: Plays an important role in the pharmacologic and physiologic regulation of cardiovascular functions [45]. Exerts modulatory role on the baroreceptor reflexes. Strongly connected with the nuclei of the IX, X, XII, and nucleus of the solitary tract.

Raphe nuclei: The medullary raphae nuclei integrate and modulate multiple respiratory, heart rate, blood pressure, and temperature parameters. They are strongly connected with the nuclei of the IX, X, and XII cranial nerves and the nucleus of the solitary tract.

11.2.6 Medulla Pathology

Lesions in the medulla cause several different symptoms including intractable vomiting, sudden unilateral hearing loss and/or disequilibrium, nystagmus, palatal myoclonus, and altered consciousness with irregular cardiorespiratory rhythms. Like the midbrain and pons, symptoms often occur in complexes depending on the size and region of the pons that is damaged.

More focal lesions result in more focal deficits. Damage to the area postrema results in intractable vomiting. This region is also rich in aquaporin-4 receptors and is occasionally involved in neuromyelitis optica. Arcuate nucleus lesions and/or developmental anomalies of these nuclei have been implicated in sudden infant death syndrome (SIDS). Damage to the nucleus prepositus hypoglossi will cause gaze-evoked

nystagmus. Unilateral lesions result in partial loss of ipsilateral and contralateral gaze holding deficits, while bilateral lesions allow for conjugate eye movements, but eyes are unable to maintain new position. Lesions in the nucleus reticularis gigantocellularis may cause bradycardia and hypotension.

Larger lesions involve more medullary structures and are grouped into syndromes.

Lateral medullary syndrome (*Wallenberg syndrome, PICA occlusion*): Results in ipsilateral facial and contralateral extremity hypoalgesia and thermoanesthesia (damage to spinal trigeminal tract), vertigo, nausea and vomiting (damage to vestibular nuclei), ipsilateral cerebellar signs (damage to restiform body), paralysis of the pharynx, vocal cord and palate (damage to nucleus ambiguus), and an ipsilateral Horner's syndrome (damage to sympathetic tracts).

Medial medullary syndrome (*Dejerine syndrome, anterior spinal artery syndrome*): Results in ipsilateral tongue deviation (fascicular CN XII), contralateral hemiparesis (damage to pyramids), contralateral loss of position, and vibratory sensation (damage to medial lemniscus). Can rarely cause upward beat nystagmus (damage to medial longitudinal fasciculus).

11.3 White Matter Tracts Spanning the Brainstem

The choice of tracts reported below is tracts that have been identified using high-resolution DTI imaging on both a 3T and a 7T [46, 47].

Spinothalamic tract: Carries crude tactile and pain sensation from the ipsilateral spinal cord to the VPL nucleus of the thalamus.

Trigeminal spinal tract: Carries crude tactile and pain sensation from the ipsilateral face area to the VPM nucleus of the thalamus.

Medial lemniscus: Carries fine discriminative tactile and kinesthetic proprioceptive sensation from the spinal cord to the thalamus. Fibers ascend from the dorsal columns of the spinal cord and synapse in the nuclei gracilis and cuneatus. Axons from those nuclei cross to the contralateral side as internal arcuate fibers and then continue rostrally in the medial lemniscus to the VPL nucleus of the thalamus. Medial lemniscus is formed at the level of the medulla and can be seen in the entire brainstem.

Lateral lemniscus: Carries acoustic information from the cochlear nuclei to contralateral inferior colliculi.

Medial longitudinal fasciculus (MLF): The MLF interconnects the III, IV, and VI cranial nerve nuclei. It also integrates information from the frontal eye fields and the vestibular-cochlear nuclei to integrate head with eye movements. A lesion in MLF causes ipsilateral loss of adduction of the eye and horizontal nystagmus in the abducting eye looking away from the lesion resulting in internuclear (between nuclei III and VI) ophthalmoplegia (INO). A right INO refers to lesion on the right side. Adduction of the eye may (Cogan posterior INO) or may not (Cogan anterior INO) be spared during convergence of the eyes. Cogan anterior INO may be seen with midbrain lesions.

Central tegmental tract (CTT): Contains descending fibers from the red nucleus to the inferior olivary nucleus (rubro-olivary tract). Fibers from the inferior olivary nucleus reach the contralateral dentate nucleus via the inferior cerebellar peduncle. Fibers from the dentate nucleus reach the contralateral red nucleus via the superior cerebellar nucleus. This triangular connection is also called the Guillain-Mollaret triangle (dentate-rubro-olivary network). Lesions to these structures including damage to the CTT can cause a classic oculomotor tremor (or myoclonus), a delayed likely temporary hypertrophy of the olivary nuclei [48]. These structures are also involved in sudden infant death syndrome (SIDS) [49].

Cerebral peduncles (Fig. 8.1): Contain large motor tracts from the cerebral cortex to the infratentorial brain regions and the spinal cord. The corticospinal tract appears T2 hyperintense due to its large axonal size containing greater amount of water content. Lesions will cause selective motor damage that will result in contralateral bulbar palsy/hemiparesis or hemiplegia.

Superior cerebellar peduncles (brachium conjunctivum) (Figs. 8.1, 8.2): These peduncles con-

tain the dentate-thalamic tracts and cerebellorubral tracts (connecting the interpositus and the globose nuclei to the contralateral red nuclei). Lesions above the brachium conjunctivum cause contralateral cerebellar symptoms, and lesions below cause ipsilateral cerebellar symptoms.

Middle cerebellar peduncles (brachium pontis) (Fig. 8.3): These peduncles contain pontocerebellar projections relayed from the pontine nuclei to the contralateral cerebellar hemisphere. They carry information used for motor coordination.

Inferior cerebellar peduncles (restiform body) (Figs. 8.3, 8.4): These peduncles are composed of fibers that receive and transmit signals to and from the spinal cord and brainstem nuclei, specifically, sensory proprioceptive signals from the spinocerebellar tracts and motor vestibular-ocular signals from the brainstem nuclei.

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Part III

Cranial Nerves

It can be argued that there are 10 cranial nerves rather than 12 on the basis of their origin and constitution. Specifically, the olfactory and the optic nerves are different from the other ten cranial nerves in that they both consist of central nervous system cellular components and carry secondary axons rather than primary axons.

The numbering of cranial nerves has classically started from I to XII in a cranio-caudal order of nerve root emergence. However this has been recently challenged as new data shows that cranial nerve VI actually emerges at the same level or below the VII and VIII cranial nerves. It is good to know even though the authors do not propose a change in the nomenclature of the cranial nerves [1].

Cranial nerve anatomy and function is complex, deriving from both the somite and visceral origins of head and neck tissues that are innervated by them. In humans, most head and neck structures derive from five pharyngeal (or brachial) clefts, arches and pouches. Each arch carries its own cranial nerve that innervates all structures developing from the corresponding arch, cleft and pouch. Muscles that develop from the branchial arches (pharyngeal, laryngeal and facial expression muscles) are innervated by special visceral efferents (SVE). Muscles that derive from somites (skin, tongue, mucosa) are innervated by general somatic efferents (GSE). Somatic sensation from head, neck, meninges and sinus mucosa reach the brain by general somatic afferents (GSA). Parasympathetic innervation to visceral structures is considered general visceral efferents (GVE). The five special senses such as olfaction, vision, taste, hearing and balance are considered special afferents (SA). Visceral sensory afferents are sensory information derived from chemoceptors and baroceptors transported to the brain (VS). The following table shows the six different functions that cranial nerves subserve.

Cranial nerve	Nerve nomenclature	Functions
I	Olfactory	SA
II	Optic	SA
III	Oculomotor	GSE, GVE
IV	Trochlear	GSE

(continued)

(continued)

Cranial nerve	Nerve nomenclature	Functions
V	Trigeminal	GSA (face, oral cavity, sinuses, ant.2/3 of the tongue), SVE (muscles of mastication)
VI	Abducens	GSE
VII	Facial	GSA (skin, mucosa, external auditory meatus etc.), GVE (parasympathetic), SVE (facial muscles), SA (taste)
VIII	Vestibulocochlear	SA
IX	Glossopharyngeal	GSA, GVE, SVE, SA (taste), VA(chemo and baroceptors)
X	Vagus	GSA, GVE, SVE, SA (taste), VA (chemo and baroceptors)
XI	Spinal accessory	GSE
XII	Hypoglossal	GSE

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Cranial Nerve I: Olfactory

12

Nivedita Agarwal

12.1 Anatomy

The olfactory nerve (CN I) is the earliest sensory system to develop in the embryo. It develops from the olfactory (or nasal) placode in the rostro-lateral head region, the cells of which then differentiate into the olfactory epithelium [4].

Nuclei: The olfactory nerves do not have a nucleus. From the olfactory epithelium, a lineage of olfactory sensory nerves (OSNs) exits the epithelium and the nasal pit to travel posteriorly toward the telencephalon. The olfactory bulb (OB) forms from a predetermined part of the telencephalon and is an outpouching of this brain region. The unmyelinated free axon terminals of OSNs traverse the cribriform plate and synapse onto secondary olfactory cells such as the mitral and the tufted cells and form the OB. The OB is well seen just above the cribriform plate surrounded by the CSF space (Figs. 12.1 and 12.2).

Branches: There are no branches of the olfactory nerve. The secondary olfactory axons of OB form the olfactory tract found in the olfactory sulcus in between the gyri recti and the orbitofrontal gyrus. The olfactory tract splits into three at the level of the anterior perforated substance: lateral, medial, and intermediate:

- Lateral olfactory stria: sends axons to the contralateral and ipsilateral primary olfactory cortex (piriform cortex) and the amygdala. From there axons travel to the secondary olfactory cortex (entorhinal cortex) that connects to the hippocampus, insula, and frontal lobe through the uncinate fasciculus.
- Medial olfactory stria: projects to the septal area (subcallosal area, paraterminal gyrus) and mediates emotional and autonomic responses to odors.
- Intermediate olfactory stria: projects to the anterior perforated substance.

The diagonal band of Broca, a white matter bundle from the septal nuclei to the amygdala, contains fibers from all three stria.

N. Agarwal, M.D.
S. Maria del Carmine Hospital, Azienda Provinciale per i Servizi Sanitari, Rovereto (TN), Italy

Center for Mind/Brain Sciences (CIMeC), University of Trento, Rovereto (TN), Italy

Department of Radiology, Section of Neuroradiology, University of Utah, Salt Lake City (UT), USA
e-mail: Nivedita.agarwal@apss.tn.it

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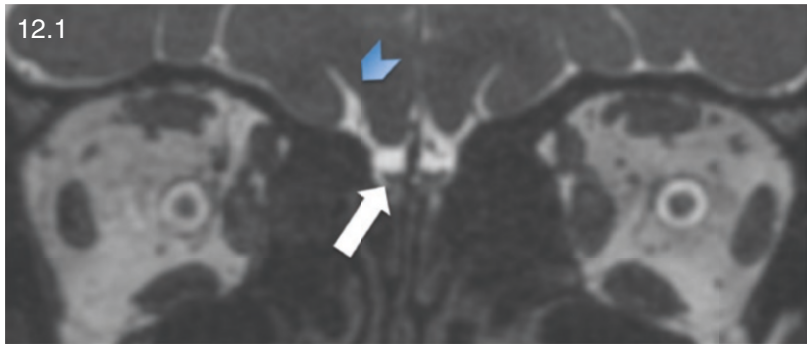

Fig. 12.1 Coronal CISS image highlights the presence of both olfactory bulbs lying just above the ethmoidal roof (*arrow*). Note the presence of normal olfactory sulci (*arrowhead*)

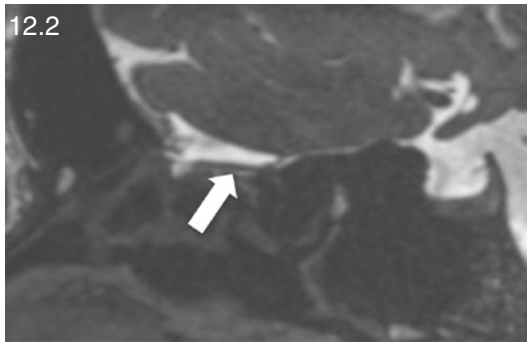

Fig. 12.2 Sagittal CISS image showing the olfactory tract (*arrow*) lying just above the ethmoid roof and below the gyrus rectus

12.2 Function

The olfactory nerve is one of the five special senses, consisting of only SA fibers. The olfactory nerve carries information involved in:

- Recognition of odors
- Emotional response to odors (including memories of pleasant and unpleasant sensations)
- Autonomic response to odors (increased peristalsis and gastric secretion)

12.3 Pathology

Individual symptoms: Damage to the olfactory nerve results in the following symptoms:

- Loss of smell at various levels from iposomia to anosmia. If ipsilateral, lesion is anterior to the piriform cortex.
- Cacosmia: perception of unpleasant smells.
- Parosmia (also troposmia): perversion of smell.
- Olfactory hallucinations (aka uncinate fits) (Fig. 12.4).

12.3.1 Syndromes

Kallmann syndrome: A rare genetic disease, of which one component is the absence of olfactory bulb and tract in the olfactory groove [3]. There is variable presence of olfactory sulcus (Fig. 12.3).

Foster-Kennedy syndrome: A constellation of symptoms associated with frontal lobe tumors. Results in ipsilateral anosmia, ipsilateral optic atrophy and contralateral papilledema [2].

CHARGE syndrome: A rare genetic disorder causing coloboma, heart defect, atresia choanae, retarded growth, genital abnormalities, and ear abnormalities. Patients also present with hyposmia or anosmia [1].

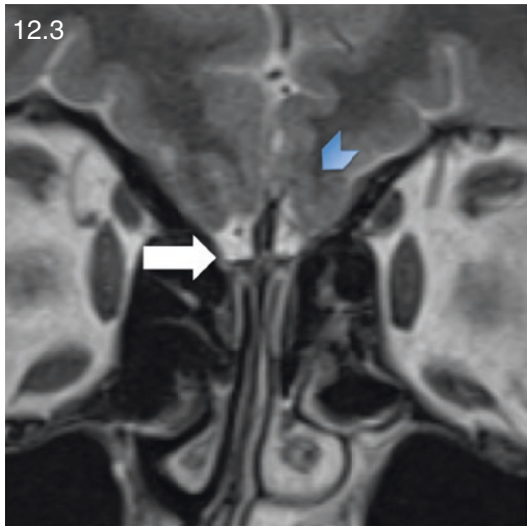

Fig. 12.3 Patient with a diagnosis of Kallmann syndrome, suffering from anosmia. Characterized by the absence of the olfactory bulbs (*arrow*) on T2W coronal images. Note the lack of the olfactory sulcus (*arrowhead*)

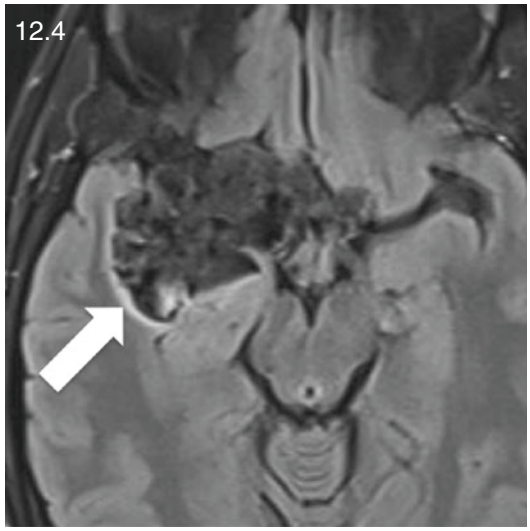

Fig. 12.4 A 31-year-old incidental finding: axial FLAIR image shows a hypointense lesion in the right Sylvian sulcus with gliosis of the adjacent temporal lobe structures, home to primary and secondary olfactory cortices. The patient was suffering from occasional olfactory hallucinations which he dismissed as benign

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Cranial Nerve II: Optic

13

Nivedita Agarwal

13.1 Anatomy

The optic nerve develops as an outpouching of the telencephalon. The axons from the retinal ganglion cells in the eighth layer of the retina form the nerve fiber layer that converges at the optic disc. These axons then continue posteriorly to form the optic nerve (Fig. 13.1). The nerve courses obliquely and medially through the orbit toward the bony optic canal. The optic nerves bilaterally come together to form the optic chiasm. Two optic tracts arise from the optic chiasm to reach the lateral geniculate bodies laterally (Fig. 13.2). Axons from the ganglion cells in the temporal side of the retina do not cross; rather, they run ipsilaterally in the optic tract and reach the occipital cortex. In contrast, axons from the ganglion cells in the nasal side of the retina cross over at the level of the chiasm to reach the contralateral hemisphere [1].

Nuclei: The optic nerves do not have a nucleus. The retinal ganglion cells pick up information from photoreceptors in the retina. Their axons form CN II chiasm, and tracts. The optic tracts form tertiary synapses at several nuclei in the brainstem including the lateral geniculate bodies (retinogeniculate fibers), pretectal area (retinopretectal fibers), superior colliculus (retinocollicular fibers), and suprachiasmatic nuclei of the hypothalamus (retinohypothalamic tract). The retinogeniculate fibers project posteriorly looping around and alongside the occipital horn to the calcarine cortex in the cuneus (area 17) of the occipital lobe.

Branches: There are no branches as in other CNs. There are however four pathways to consider:

- Retinogeniculate pathway
- Retinopretectal pathway
- Retinohypothalamic pathway
- Retinocollicular pathway

N. Agarwal, M.D.
S. Maria del Carmine Hospital, Azienda Provinciale per i Servizi Sanitari, Rovereto (TN), Italy

Center for Mind/Brain Sciences (CIMeC), University of Trento, Rovereto (TN), Italy

Department of Radiology, Section of Neuroradiology, University of Utah, Salt Lake City (UT), USA
e-mail: Nivedita.agarwal@apss.tn.it

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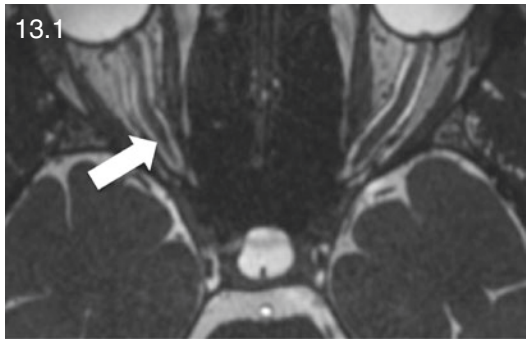

Fig. 13.1 Optic nerves depicted on an axial CISS image. The nerves are hypointense surrounded by subarachnoid space which may not always be clearly visible. In this particular patient suffering from idiopathic hypertension, the spaces are enlarged as a result of increased intracranial pressure

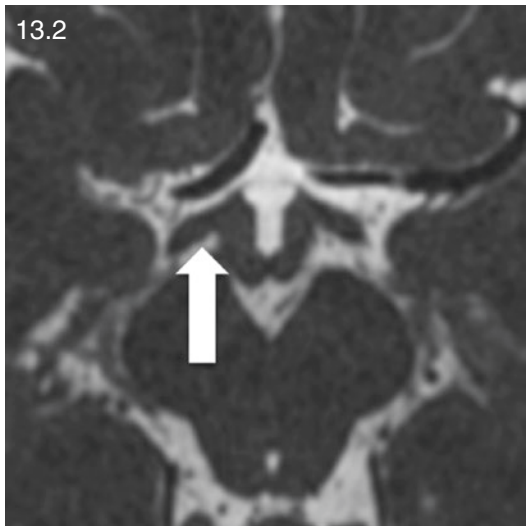

Fig. 13.2 Axial CISS image showing optic tracts. The arrow points to the right optic tract which is retrochiasmatic

13.2 Function

The optic nerve is one of the five special senses, consisting of only SA fibers. CN II is important in providing the sense of vision. The nerve preserves a retinotopic map of the outside visual field such that the nasal area of each visual field projects to the temporal (or lateral) half of each retina and the peripheral or visual field projects to the nasal half of the retina. While the nasal retinal fibers decussate at the level of the optic nerve traveling into the opposite optic tract, the temporal retinal fibers course along CN II and into the optic tract without decussating. Thus, each optic tract contains information from both eyes (e.g., the right optic tract contains peripheral half of the visual field from the right eye and the medial visual field from the left eye). This information remains constant and well maintained at all levels including the lateral geniculate bodies, optic radiations and, in the cortex of the primary visual cortex. The optic radiations can be further divided into the parietal lobe optic radiations that subserve the inferior visual fields and the temporal lobe optic radiations that contain information from the superior visual fields.

- Retinogeniculate pathway: the primary pathway for conscious vision.
- Retinopretectal pathway: responsible for pupillary light reflex and for conjugate pupillary response.
- Retinohypothalamic pathway: visual information reaches the suprachiasmatic nuclei of the hypothalamus promoting rhythmic circadian sleep-wake cycle and promoting neuroendocrine functions.
- Retinocollicular pathway: allows conjugate eye movements and eye movement reflexes.

13.3 Pathology

Individual symptoms: Damage to the optic nerve results in the following symptoms depending on location [2, 3]:

- **Prechiasmatic**
  - Partial optic nerve: scotoma
  - Complete right optic nerve: complete monocular blindness
- **Chiasmatic**
  - Bitemporal hemianopsia (loss of vision from the peripheral visual fields)

- **Retrochiasmatic**
  - Right optic tract: left homonymous hemianopsia (loss of left peripheral vision and right nasal vision)
  - Right geniculate body: left homonymous sectoranopia
  - Right optic radiation: left homonymous hemianopsia with macular sparing
  - Right parietal optic radiation: lower left homonymous quadrantanopsia (Fig. 13.3) illustrates a lesion in the left optic radiation
  - Right temporal optic radiation: upper left homonymous quadrantanopsia
  - Right calcarine gyrus upper bank: left inferior homonymous quadrantanopia
  - Right calcarine gyrus lower bank: right superior homonymous quadrantanopia
  - Right tip of the occipital lobe: left homonymous central scotomas
  - Fig. 13.4 illustrates vasogenic edema due to compression of the both optic tracts

Common etiologies of CN II lesions that can cause visual field defects and visual loss are:

- Inflammatory/demyelinating disease
- Intracranial hypertension with optic disk edema
- Tumors of the optic nerve sheath (meningioma and astrocytomas) Fig. 13.5
- Compressive lesions to any part of the visual pathway including granulomatous tissue (pseudotumor, sarcoidosis, or vascular and lymphatic malformations)

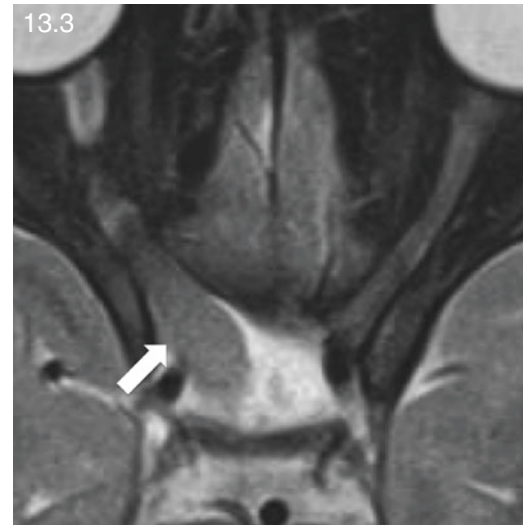

Fig. 13.3 A 4-year-old boy with neurofibromatosis type I: axial T2W image shows an elongated mass involving the right optic nerve. The lesion is an astrocytoma, a glial tumor

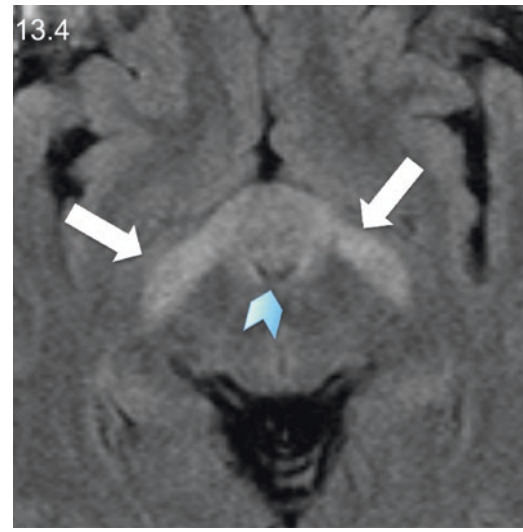

Fig. 13.4 Sudden blindness followed by loss of consciousness in a patient with solid pituitary mass (*blue arrow*). Optic tracts are bilaterally edematous (*white arrows*)

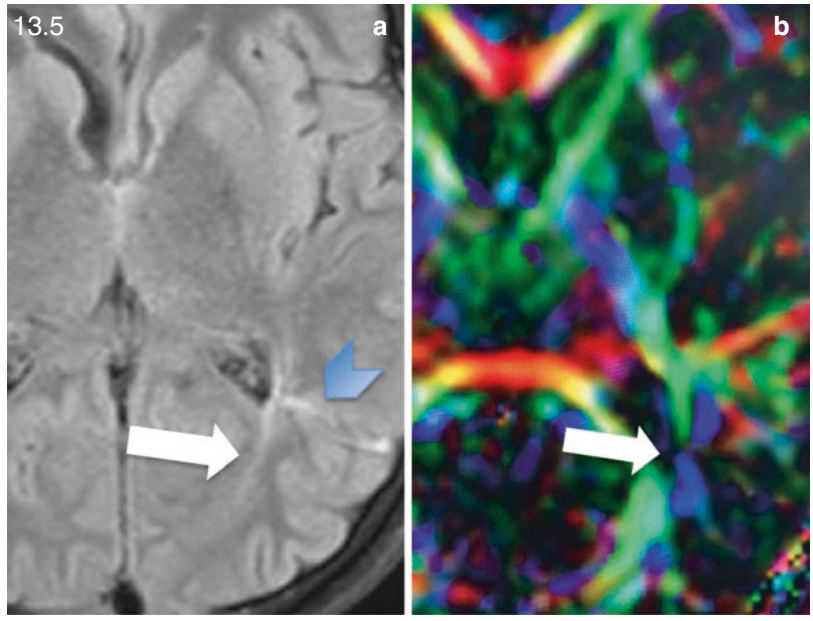

Fig. 13.5 Patient with recurrent right homonymous hemianopsia. (**a**) Axial FLAIR image shows a focal developmental venous anomaly surrounded by gliotic changes alongside the wall of the left occipital horn of the lateral ventricle. (**b**) DTI image shows a focal interruption of the optic radiations (in green), possible cause of the specific visual field defect

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Cranial Nerve III: Oculomotor

14

Karen S. Chen, Ari M. Blitz, and Nivedita Agarwal

14.1 Anatomy

Cranial nerve III develops from the basal plate of the embryonic midbrain. Motor fibers rise from the oculomotor nuclei. From the ventral midbrain within the interpeduncular cistern, the oculomotor nerve (CN III) courses anterolaterally between the posterior cerebellar artery and superior cerebellar artery through the suprasellar cistern [1] (Fig. 14.1). The cisternal segment continues medial to the uncus before entering the interdural space within the cavernous sinus, ensheathed in dura along its superolateral wall and lateral to the posterior clinoid process [2] (Fig. 14.2). The foraminal segment of CN III passes anteriorly through the superior orbital fissure before exiting into the extra-foraminal segment within the orbit. The superior medial aspect of the oculomotor nerve comprises parasympathetic fibers which terminate in the ciliary ganglion in the retrobulbar intraconal space [3]. The inner aspect comprises motor neurons which innervate the extraocular muscles [4]. The nerve subdivides into a superior and an inferior division.

14.1.1 Nuclei

- *Oculomotor nuclei*: located in the midbrain, generate signals that control the activity of the superior, medial, and inferior rectus muscles, the inferior oblique muscle, and the levator palpebrae muscle
- *Edinger-Westphal nuclei*: located in the midbrain, generate signals that control pupillary constriction and lens accommodation

K.S. Chen, M.D. (✉)
Department of Neuroradiology, Russell H. Morgan Department of Radiology and Radiological Sciences, Johns Hopkins University School of Medicine, Baltimore, MD, USA
e-mail: kchen21@bwh.harvard.edu

A.M. Blitz, M.D.
Department of Neuroradiology, Russell H. Morgan Department of Radiology and Radiological Sciences, Johns Hopkins University School of Medicine, Baltimore, MD, USA
e-mail: ablitz1@jhmi.edu

N. Agarwal, M.D.
S. Maria del Carmine Hospital, Azienda Provinciale per i Servizi Sanitari, Rovereto (TN), Italy

Center for Mind/Brain Sciences (CIMeC), University of Trento, Rovereto (TN), Italy

Department of Radiology, Section of Neuroradiology, University of Utah, Salt Lake City (UT), USA
e-mail: Nivedita.agarwal@apss.tn.it

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14.1.2 Branches

- *Superior division*: innervates the superior rectus, levator palpebrae.
- *Inferior division*: inferior and medial rectus, inferior oblique muscles. This division also carries the presynaptic parasympathetic fibers. Postsynaptic fibers are carried by six to ten nasociliary nerves that reach ciliary muscle and sphincter pupillae.

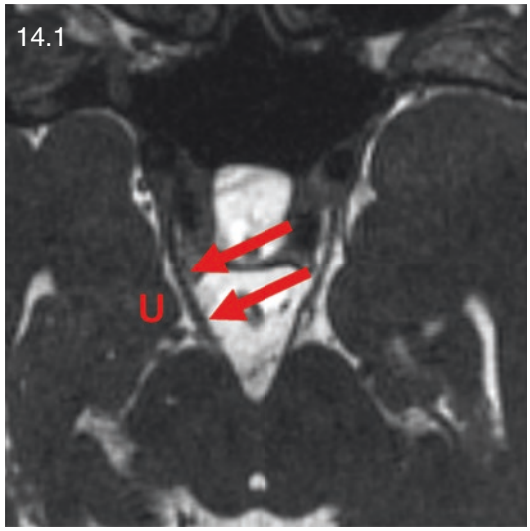

Fig. 14.1 Axial CISS demonstrates the cisternal segment of the right CN III (*arrows*) arising from the ventral midbrain and passing anteriorly medial to the uncus (U), a surface landmark on the medial aspect of the temporal lobe

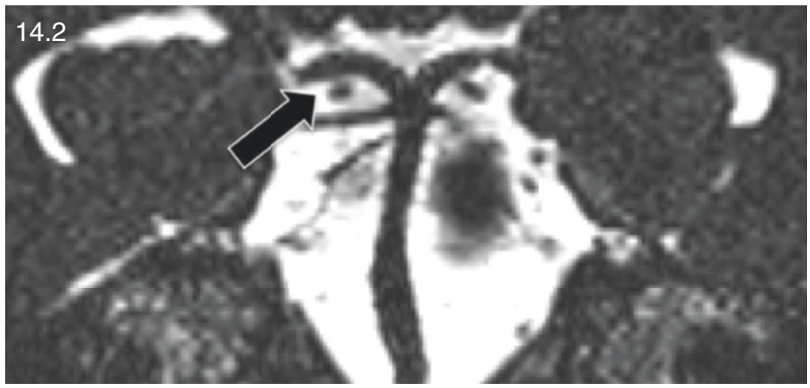

Fig. 14.2 Coronal CISS image: cisternal segment of CN III lies in the "oculomotor triangle" defined superiorly by the posterior cerebral artery and inferiorly by the superior cerebellar artery

14.2 Function

- Motor function (GSE): controls the superior rectus, medial rectus, inferior rectus, levator palpebrae, and inferior oblique muscles. The medial rectus muscle allows for adduction of the eye, the inferior rectus muscle moves the eyeball downward, the superior rectus moves the eyeball upward, the inferior oblique moves it upward and outward, and levator palpebrae moves the eyelid upward.
- Parasympathetic function (GVE): activation leads to pupillary constriction and lens accommodation.

14.3 Pathology

Individual symptoms: Damage to the oculomotor nerve results in the following symptoms depending on location:

- *Nuclear lesion*: Isolated nuclear lesions are extremely rare. Ipsilateral lesion will cause ipsilateral oculomotor palsy and bilateral paralysis of the superior recti and the levator palpebrae (both have a bilateral innervation), resulting in incomplete ptosis bilaterally.
- *Cisternal lesions*: Lesions to the cisternal segment will cause only ipsilateral symptoms (Figs. 14.3 and 14.4). Uncal herniation or compression by a PCOM or SCA aneurysm will inhibit pupillary constriction in the ipsilateral eye (mydriasis) without necessarily affecting ocular movement:
  - Anisocoria: asymmetry of pupils (fixed mydriatic pupil on the affected side).
  - Extraocular muscle palsy: unopposed muscle tension by the superior oblique and lateral rectus results in down- and outpositioning of the globe.
- *Cavernous sinus lesions*: Due to lateral extension of a pituitary mass into the cavernous sinus, skull base meningioma, epidermoid cyst, arachnoid cyst, schwannoma, or neurofibroma that compresses CN III in its cisternal or intradural segment. Other lesions include carotid-cavernous sinus fistula, cavernous sinus thrombosis, carotid artery aneurysm, and meningitis [5].
- *Superior orbital fissure lesions*: Most pathology results in similar deficits as lesions in the cavernous sinus. These include hypertrophic idiopathic inflammatory lesions (pseudotumor); bony lesions such as fibrous dysplasia, trauma, and osseous metastasis may also affect contents of the superior orbital fissure (CN III, IV, V1, and VI and superior ophthalmic vein).
- *Intraorbital lesion*: Isolated CN III nerve palsy does not occur from orbital lesions. Although rare, selective paresis of either division of the III nerve may occur. Malignant lesions such as intraorbital melanoma or metastatic disease may reach intracranially through perineural spread along CN III.

14.3.1 Syndromes

Parinaud syndrome: upgaze palsy from compression of the superior colliculi on the tectal plate by a pineal mass which inhibits the oculomotor nerve nuclei in the dorsal midbrain [6].

Benedikt syndrome: upper midbrain (red nucleus) lesion causing ipsilateral CN III palsy, contralateral flapping hand tremor, and ataxia [6].

Weber syndrome: infarct of the cerebral peduncle with contralateral hemiplegia and ipsilateral CN III palsy [6].

Nothnagel syndrome: CN III with ipsilateral ataxia [6].

Claude syndrome: CN III with contralateral ataxia [6].

Tolosa-Hunt syndrome: inflammation of cavernous sinus resulting in painful ophthalmoplegia [7].

Superior orbital fissure syndrome: total ophthalmoplegia, CN V1 pain, paresthesias, sensor loss, chemosis, and proptosis of the ipsilateral eye.

Cavernous sinus syndrome: symptoms relative to CN III, IV, VI, V1, and V2 and sympathetic innervation from the ICA. Pupils are in the midline, fixed due to loss of both sympathetic and parasympathetic component.

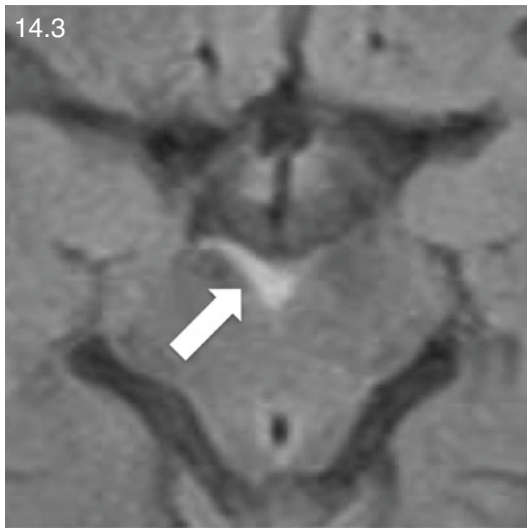

Fig. 14.3 Subarachnoid hemorrhage in the interpeduncular fossa in a patient with thunderclap headache, ptosis, and mydriasis in the right eye

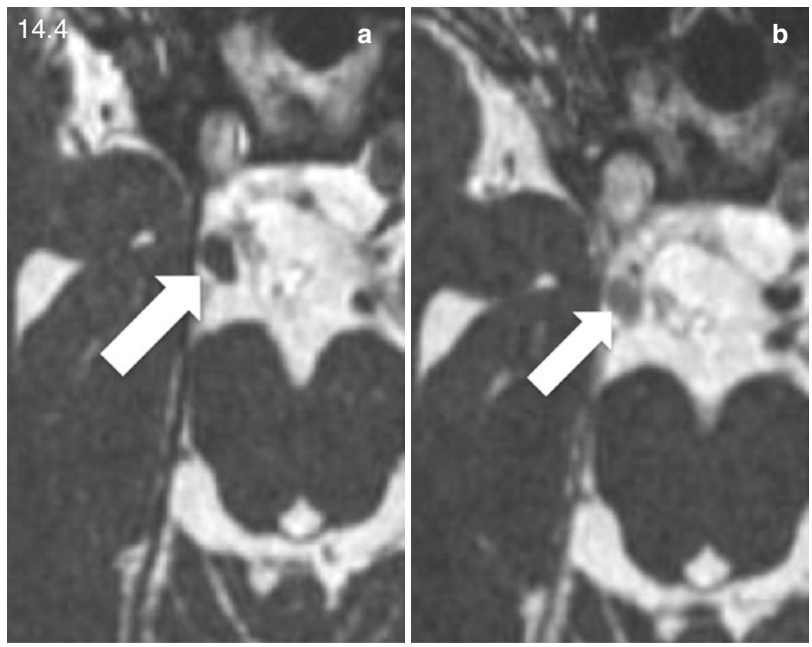

Fig. 14.4 46-year-old with recurrent and reversible ophthalmoplegia in the right eye. (**a**) Precontrast CISS image shows a focal mass on CN III. (**b**) This mass enhances with contrast on postcontrast CISS image, raising the possibility of a focal neurinoma of CN III in the absence of metastatic disease

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Cranial Nerve IV: Trochlear

15

Karen S. Chen, Ari M. Blitz, and Nivedita Agarwal

15.1 Anatomy

The fourth cranial nerve develops from the posterior part of the basal plate of the embryonic midbrain. Unique among the cranial nerves, the trochlear nerve (CN IV) exits from the dorsal brainstem to innervate the contralateral superior oblique muscle. This is because the trochlear nuclei are located anterior to the aqueduct of Sylvius and the parenchymal fascicular segment decussates before exiting just below the inferior colliculi [1]. CN IV is a very small nerve and is seldom visible on routine MR. High-resolution heavy-weighted T2 images may be required to see parts of CN IV, especially in the dorsal part of the mesencephalon. The cisternal segments of the nerves continue anteriorly through the quadrigeminal cistern and laterally within the ambient cistern where they pass under the free edge of the cerebellar tentorium before entering the lamina propria (Fig. 15.1). Like the oculomotor nerve above it, the trochlear nerve continues into the interdural segment with its dural covering along the lateral wall of the cavernous sinus. The foraminal segment then enters the superior orbital fissure, followed by the extra-foraminal segment that runs along the orbital roof to innervate the superior oblique muscle between its posterior third and anterior two-thirds [2].

Nuclei: The trochlear nuclei are located in the dorsal midbrain. They generate signals that control the activity of the superior oblique muscle.

Branches: There are no branches as in other CNs.

K.S. Chen, M.D. (✉)
Department of Neuroradiology, Russell H. Morgan Department of Radiology and Radiological Sciences, Johns Hopkins University School of Medicine, Baltimore, MD, USA
e-mail: kchen21@bwh.harvard.edu

A.M. Blitz, M.D.
Department of Neuroradiology, Russell H. Morgan Department of Radiology and Radiological Sciences, Johns Hopkins University School of Medicine, Baltimore, MD, USA
e-mail: ablitz1@jhmi.edu

N. Agarwal, M.D.
S. Maria del Carmine Hospital, Azienda Provinciale per i Servizi Sanitari, Rovereto (TN), Italy

Center for Mind/Brain Sciences (CIMeC), University of Trento, Rovereto (TN), Italy

Department of Radiology, Section of Neuroradiology, University of Utah, Salt Lake City (UT), USA
e-mail: Nivedita.agarwal@apss.tn.it

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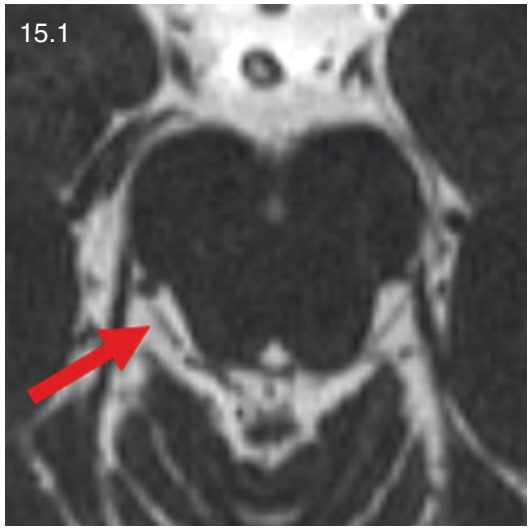

Fig. 15.1 Axial CISS demonstrates the cisternal segment of the right CN IV (*arrow*) arising from the dorsal aspect of the midbrain just caudal to the inferior colliculus

15.2 Function

The trochlear nerve has only GSE motor fibers. It innervates the superior oblique muscle, activation of which causes the eye to depress when adducted and to intort (inward movement of the eye) when abducted.

15.3 Pathology

Individual symptoms: Damage to the trochlear nerve results in the following symptoms depending on location:

- *Nuclear lesions*: Isolated lesions are very rare. Trauma, ischemia, tumor, and inflammatory diseases may cause nuclear lesions. Patients will present with vertical strabismus and vertical diplopia. Patients usually present with their head tilting in a direction opposite to the muscle paresis. Lesions will affect the contralateral superior oblique muscle if the lesion is in the nucleus and the fascicles before decussation. Congenital absence of the nerve is possible [3].
- *Cisternal lesions*: Neoplasm, aneurysmatic compression, ischemia, increased intracranial hypertension, or traumatic avulsion/contrecoup forces are main causes of trochlear nerve damage (Fig. 15.2). Inflammation/neuritis is also possible. Lesions will cause ipsilateral palsy of the superior oblique muscle. There may be associated hypotrophic superior oblique muscle due to chronic denervation (Fig. 15.3).
- *Cavernous sinus lesions*: Neoplasm, carotid-cavernous fistula, ICA aneurysms, inflammatory granulomatous disease, pituitary lesions, or apoplexy may cause damage to the trochlear nerve in its intradural segment [4]. Direct injury from trauma or surgery may also damage the nerve [5]. Isolated damage to CN IV is unlikely, and usually associated neurologic findings may be found.
- *Superior orbital fissure lesions*: Most pathology results in similar deficits as lesions in the cavernous sinus. These include hypertrophic idiopathic inflammatory lesions (pseudotumor); bony lesions such as fibrous dysplasia, trauma, and osseous metastasis may also affect contents of the superior orbital fissure (CN III, IV, V1, and VI and superior ophthalmic vein)
- *Intraorbital lesions*: Rarely intraorbital lesion will cause damage to the trochlear nerve alone.

15.3.1 Syndromes

Tolosa-Hunt syndrome: inflammation of cavernous sinus resulting in painful ophthalmoplegia [6].

Brown syndrome: tenosynovitis of the superior oblique tendon. It may mimic trochlear nerve palsy. It is often seen with rheumatologic disorders.

Superior oblique myokymia: an idiopathic condition characterized by intermittent vertical diplopia and oscillopsia. It may be associated with monocular blurred vision [7].

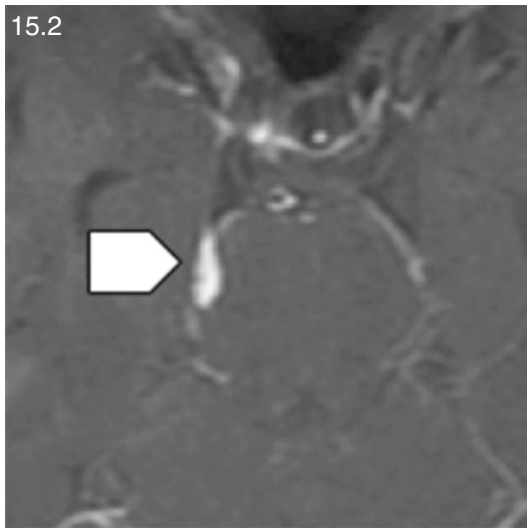

Fig. 15.2 Small enhancing trochlear nerve lesion in the medial aspect of the right cerebellar tentorium

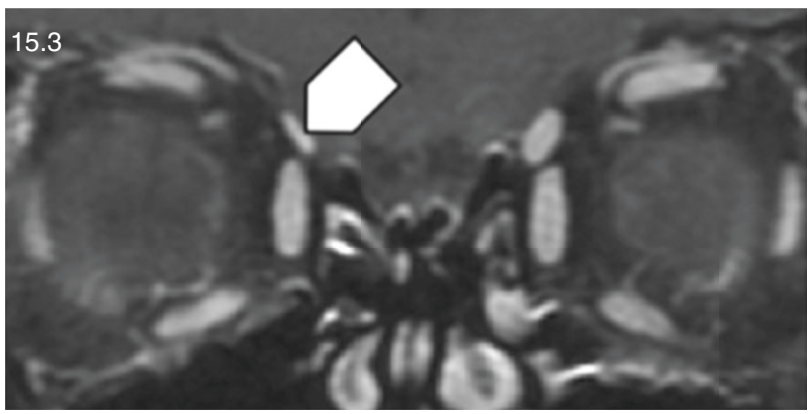

Fig. 15.3 Same patient as 15.2. Note the severe hypotrophy of the right superior oblique muscle due to damage to the ipsilateral trochlear nerve

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Cranial Nerve V: Trigeminal

16

Richard Wiggins

16.1 Anatomy

Cranial nerve V develops from the first pharyngeal arches. The trigeminal nerves exits the brainstem on both sides of the pons Fig. 16.1 and Fig. 16.2. They then course through the prepontine cistern and through the porous trigeminus to enter the trigeminal cistern (Meckel's cave) Fig. 16.3 and Fig. 16.4. The trigeminal (semilunar or Gasserian) ganglion is within the trigeminal cistern, and the postganglionic fibers then separate into the three divisions of the trigeminal nerve: V1 (ophthalmic), V2 (maxillary), and V3 (mandibular) Fig. 16.5. The V1 and V2 divisions continue anteriorly within the inferior and lateral dural sleeves of the cavernous sinus lateral walls (Fig. 16.6). The V1 branch leaves the cavernous sinus and extends superiorly and laterally and passes through the superior orbital fissure and then exits the supraorbital notch above the orbit. The V2 division leaves the cavernous sinus and travels through foramen rotundum to the pterygopalatine fossa and then along the infraorbital canal to exit at the infraorbital foramen, inferior to the orbit. The V3 division is the largest trigeminal branch and extends inferiorly from the trigeminal cistern, through foramen ovale into the infrazygomatic masticator space (infratemporal fossa).

Intracranial course: The primary neuron reaches the principal sensory nucleus. Second neuron fibers descend to form the spinotrigeminal tract to the C2 level. They then cross the midline and ascend as ventral spinotrigeminal tract to reach the ventroposteromedial nuclei of the thalamus (pain and temperature). Fibers carrying fine touch and pressure from the principal sensory nucleus cross the midline to reach the VPM of the thalamus via the trigeminal lemniscus. From here a third sensory neuron will reach the sensory cortex in the parietal lobe. CN V motor fibers descend along the corticobulbar tract (the lower third of the precentral gyrus) traversing the corona radiata, internal capsule, and cerebral peduncle decussating in the pons to reach the contralateral trigeminal motor nucleus.

R. Wiggins, M.D.
Departments of Radiology and Imaging Sciences, Otolaryngology, Head and Neck Surgery, and BioMedical Informatics, University of Utah Health Sciences Center, 30 North, 1900 East, #1A071, Salt Lake City, UT 84132-2140, USA
e-mail: Richard.Wiggins@hsc.utah.edu

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16.1.1 Nuclei

- Main (principal) sensory nucleus: large sensory nucleus within the posterior pons
- Mesencephalic nucleus: sensory nucleus extending superiorly into the mesencephalon
- Spinal trigeminal nucleus: sensory nucleus extending inferiorly into the medulla:
  - Pars caudalis: from C2 to the obex
  - Pars interpolaris: from the obex to the hypoglossal nucleus
  - Pars oralis: from the hypoglossal nucleus to the pons
- Trigeminal motor nucleus: small motor nucleus medial and anterior to the trigeminal main sensory nucleus

16.1.2 Branches

- V1 (ophthalmic) nerve (GSA):
  - Frontal nerve:
    Supraorbital nerve
    Supratrochlear nerve
  - Lacrimal nerve
  - Nasociliary nerve:
    Anterior ethmoid nerve
    Infratrochlear nerve
    Posterior ethmoid nerve
    Ciliary ganglion nerve
- V2 (maxillary) nerve (GSA):
  - Zygomatic nerve
  - Nasopalatine nerve
  - Greater palatine nerve
  - Lessor palatine nerve
  - Superior alveolar nerve
  - Pharyngeal nerve
  - Infraorbital nerve
- V3 (mandibular) nerve (SVE):
  - Efferent nerves from main branch to tensor tympani, medial pterygoid, and tensor veli palatini muscles
  - Anterior division:
    Buccal nerve (sensory)
    Lateral pterygoid muscle nerve
    Anterior temporal nerve
    Posterior temporal nerve
    Masseter muscle nerve
  - Posterior division:
    Inferior alveolar nerve:
    Anterior belly of the digastric nerve
    Mylohyoid nerve
    Lingual nerve
    Auriculotemporal nerve

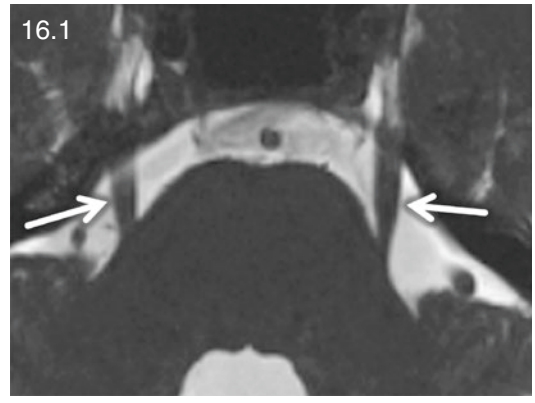

Fig. 16.1 Axial thin section T2W MRI image through the prepontine cistern demonstrates the large fibers of the trigeminal nerves (*arrows*) extending from the pons anteriorly toward the trigeminal cistern

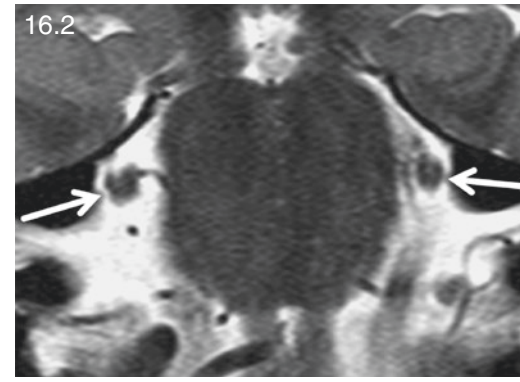

Fig. 16.2 Coronal T2W MRI image through the belly of the pons demonstrates the large fibers of the trigeminal nerves (*arrows*) on both sides of the pone, extending anteriorly

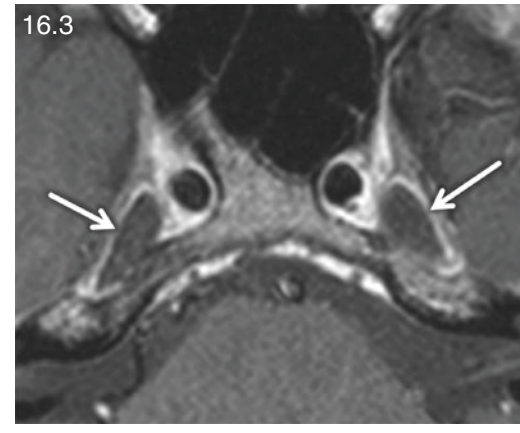

Fig. 16.3 Axial T1W MRI image through the trigeminal cisterns (*arrows*) shows the normal peripheral enhancement of the dural surfaces, without central enhancement

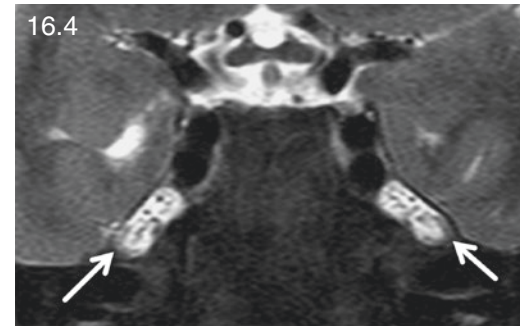

Fig. 16.4 Coronal T2W (CSF bright) MRI image through the anterior trigeminal cisterns demonstrates the normal semilunar-shaped trigeminal ganglions (*arrows*) inferiorly and laterally within the trigeminal cisterns

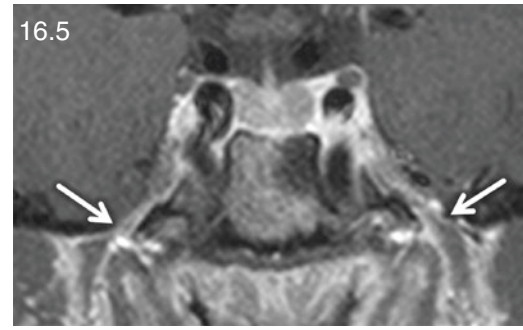

Fig. 16.5 Coronal T1W postcontrasted MRI image immediately anterior to the trigeminal cistern shows the normal V3 (mandibular) divisions extending inferiorly through foramen ovale (*arrows*). The nerves themselves do not normally enhance, although there is normally enhancement of the vasculature surrounding the nerves in the foramen; this is nicely demonstrated on the left side

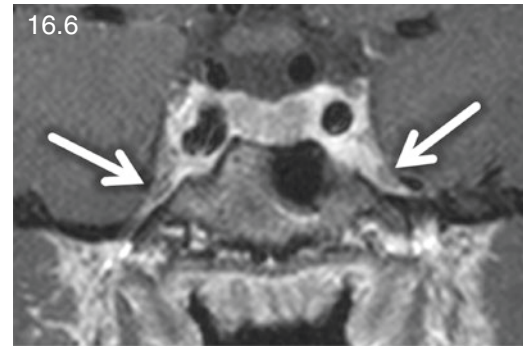

Fig. 16.6 Coronal T1W postcontrasted MRI image through the pituitary gland demonstrating the normal cavernous sinuses on both sides of the sella with normal internal carotid artery dark oval flow voids. The nonenhancing foci seen laterally and inferiorly are the normal V2 division nerves (*arrows*) of the trigeminal nerves within the lateral and inferior dural fascial sleeves of the cavernous sinus. The nonenhancing V1 divisions are seen immediately superior to V2 on both sides of the cavernous sinus

16.2 Function

- Motor function (SVE): Motor fibers extend with V3 division from the trigeminal motor nucleus and supply the muscles of mastication (anterior division: masseter, temporalis, medial pterygoid, lateral pterygoid) as well as the anterior belly of the digastric muscle and mylohyoid muscle (posterior division).
- Sensory function (GSA): carries general sensory information from the face:
  - V1 (ophthalmic) nerve: sensory information from the forehead and scalp skin with sympathetic fibers for pupil dilation.
  - V2 (maxillary) nerve: sensory information from the midface (sinuses, nasal cavities, maxillary teeth, palate, and mouth).
  - V3 (mandibular) nerve: anterior division supplies sensory innervation to the inner lining of the cheek. Posterior division supplies sensory innervation to the anterior 2/3 of the tongue, mandibular teeth, lower lip, and chin.

16.3 Pathology

Individual symptoms: Damage to the trigeminal nerve results in the following symptoms depending on location:

- Trigeminal neuropathy: Any pathology of the trigeminal nerve, including motor to the muscles of mastication and sensory to the face. Trigeminal neuralgia (tic douloureux) is a type of trigeminal neuropathy, most commonly a sudden onset of facial pain or less commonly a constant aching or burning facial pain that can be both mentally and physically debilitating [3], [4].
- Supranuclear lesions: Contralateral facial paresthesias and/or anesthesia. Muscles of mastication carry bilateral innervation but predominantly control contralateral muscles. Unilateral lesion of the motor fibers will cause deviation of the jaw toward the affected side.
- Pontomedullary lesions: Isolated lesions of the pontomedullary junction can result in tri-

geminal neuropathy, such as ischemia, neoplasm, and demyelinating pathologies. Symptoms usually are ipsilateral and include paresis/fasciculations of the affected muscles, ipsilateral hemianesthesia, and paresthesias.

- Cisternal lesions: Lesions within the prepontine cistern can present with trigeminal neuropathy, such as a trigeminal schwannoma or a meningioma [1]. Trigeminal neuralgia (tic douloureux) is most commonly caused by a vascular loop syndrome, with a vessel significantly displacing the main trunk of CN V at the root entry zone at the anterior and lateral pons [2], [5]. Preganglionic lesion will cause ipsilateral motor paresis and ipsilateral sensory deficits with weak corneal reflex.
- Extracranial lesions: Consider neoplasms and trauma as major causes of CN V dysfunction. Trigeminal neuropathy can be seen with perineural tumor spread (PNTS) especially with squamous cell carcinoma (SCCA), most commonly seen along V2 and V3 branches.

16.3.1 Syndromes

Gradenigo syndrome: petrous apicitis leads to V and VI nerve damage causing ipsilateral facial pain and numbness accompanied by ipsilateral lateral rectus palsy.

Herpes zoster ophthalmicus: latent varicella virus in the trigeminal ganglion spreads along any division of the fifth nerve to cause pain and vesicles in the affected territory.

Superior orbital fissure syndrome: total ophthalmoplegia, CN V1 pain, paresthesias, sensory loss, chemosis, and proptosis of the ipsilateral eye.

Cavernous sinus syndrome: symptoms relative to CN III, IV, VI, V1, and V2.

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Cranial Nerve VI: Abducens

17

Karen S. Chen, Ari M. Blitz, and Nivedita Agarwal

17.1 Anatomy

The sixth cranial nerve develops from the basal plate of the embryonic pons. From the ventral pontomedullary junction, the abducens nerve courses superiorly in the prepontine cistern where it may encounter veins and arteries of the posterior circulation such as the anterior inferior cerebellar artery (Fig. 17.1). From the radiologic perspective, the interdural segment is notably longer than the other ocular motor cranial nerves and enters the petroclival venous confluence, passes through Dorello's canal, and then enters the cavernous sinus, lateral to the cavernous segment of the internal carotid arteries. The foraminal segment of CN VI, along with CN III and IV, passes through the superior orbital fissure and passes into the extra-foraminal segment in the orbit where it innervates the lateral rectus between its posterior third and anterior two-thirds [1].

Nuclei: The abducens nuclei are located in the caudal pons. They generate signals that control the activity of the lateral rectus muscles.

Branches: There are no branches as in other CNs.

K.S. Chen, M.D. (✉)
Division of Neuroradiology, John Hopkins Hospitals, Baltimore, MD, USA
e-mail: kchen21@bwh.harvard.edu

A.M. Blitz, M.D.
Department of Neuroradiology, Russell H. Morgan Department of Radiology and Radiological Sciences, Johns Hopkins University School of Medicine, Baltimore, MD, USA
e-mail: ablitz1@jhmi.edu

N. Agarwal, M.D.
S. Maria del Carmine Hospital, Azienda Provinciale per i Servizi Sanitari, Rovereto (TN), Italy

Center for Mind/Brain Sciences (CIMeC), University of Trento, Rovereto (TN), Italy

Department of Radiology, Section of Neuroradiology, University of Utah, Salt Lake City (UT), USA
e-mail: Nivedita.agarwal@apss.tn.it

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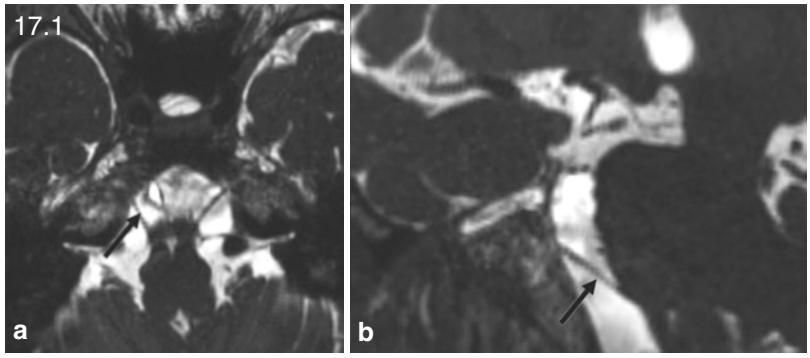

Fig. 17.1 Axial oblique CISS image (**a**) demonstrates the cisternal segment of the right CN IV (*arrow*) arising from ponto-medullary junction and coursing anterior and best seen in sagittal section (**b**) superiorly toward the dorsal aspect of the clivus

17.2 Function

The abducens nerve has only GSE motor fibers. It innervates the lateral rectus muscle, the activation of which causes abduction of the eye allowing for conjugate horizontal gaze.

17.3 Pathology

Individual symptoms: Damage to the abducens nerve results in the following symptoms depending on location:

- *Nuclear lesions:* Mass lesions that compress at the skull base such as meningioma, epidermoid cyst, arachnoid cyst, and schwannoma can cause compression at the level of the dorsal pons. A *nuclear* lesion always causes impairment of the ipsilateral gaze of both eyes due to interneuron connections with the contralateral CN III through the medial longitudinal fasciculus. Inflammatory and ischemic pontine (Millard-Gubler syndrome) lesions also cause fascicular parenchymal lesions causing ipsilateral gaze deficit.
- *Cisternal lesions:* Due to its unusually lengthy course of the nerve, CN VI is particularly prone to traumatic injury. Changes in intracranial pressure (in particular idiopathic intracranial hypertension), diabetic ischemia, aneurysmal compression from basilar artery or neurovascular conflict (e.g., presence of dolicho basilar artery), neoplasms (tentorium of clival meningioma), and idiopathic hypertrophic pachymeningitis are some of the most frequent etiologies. Miller-Fischer variant of Guillain-Barrè can cause CN VI palsy.
- *Cavernous segment:* Carotid-cavernous sinus fistula, cavernous sinus thrombosis, and carotid artery aneurysms result in mass effect-induced CN VI palsy [2].
- *Lesions of the petrous apex:* Petrous apicitis, fractures of the skull base, bone tumors, and metastasis can involve CN VI. Inflammatory lesions of the petrous apex such as cholesteatoma, cholesterol granuloma, and mucocele also result in CN VI damage.
- *Intraorbital lesions:* Isolated lesions are rare. Inflammatory granulomatous tissue in the orbit or in the retro-orbital tissue can cause CN VI palsy. Myasthenia gravis or thyroid orbitopathy involves muscle enlargement causing horizontal diplopia falsely attributed to CN VI palsy.

17.3.1 Syndromes

Tolosa-Hunt syndrome: inflammation of cavernous sinus resulting in painful ophthalmoplegia [3].

Gradenigo syndrome: petrous apicitis, lateral rectus palsy, and ipsilateral retro-orbital/facial pain [4].

Raymond's syndrome: CN VI palsy with contralateral hemiparesis [5].

Millard-Gubler syndrome: CN VI and VII palsy with contralateral hemiparesis [5].

Foville's syndrome: CN V–VIII palsy with ipsilateral Horner's syndrome [5].

Möbius syndrome: malformation of the brain stem with underdevelopment of the abducens and facial nuclei causing horizontal gaze palsy and ipsilateral facial paresis.

Duane retraction syndrome: congenital absence of the abducens nerve causes unilateral horizontal gaze palsy.

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Cranial Nerve VII: Facial

18

Laura B. Eisenmenger and Richard H. Wiggins III

18.1 Anatomy

Cranial nerve VII develop from the second pharyngeal arches. The facial nerves exit the pons inferior and ventrolaterally, immediately superior to the pontomedullary junction [1, 2], and course superiorly and laterally through the cerebellopontine angle (CPA) (Fig. 18.1) to enter the internal auditory canal (IAC), traveling superiorly and anteriorly (Figs. 18.2 and 18.3). The fibers then turn anteriorly (the labyrinthine segment) from the lateral IAC to the anterior genu, where the greater superficial petrosal nerve branches off, extending anteriorly and medially. The remaining fibers then course posteriorly and laterally (the tympanic segment) immediately inferior to the lateral semicircular canal [3, 4]. The nerve penetrates the mastoid bone, turns inferiorly at the posterior genu, and descends posterior to the external posterior auditory canal (EAC). Two branches originate from the descending mastoid segment, the stapes muscle nerve, and the chorda tympani. The main facial nerve remaining fibers then exit the skull base through the stylomastoid foramen (Fig. 18.4). The facial nerve then pierces the posterior fascia of the parotid gland and passes lateral to the retromandibular vein while ramifying into terminal branches extending out over the face to the voluntary and involuntary muscles of facial expression [5–7].

Intracranial course: Primary motor fibers descend mostly in the corticobulbar tract arising from the lower third of the precentral gyrus in the corona radiata, internal capsule, and cerebral peduncle. The upper part of the face receives bilateral innervation, whereas the lower third receives contralateral motor input.

18.1.1 Nuclei

- *Motor nucleus:* Ventrolateral pontine tegmentum, anterior and lateral to the sixth motor nucleus
- *Superior salivary nucleus:* Located posterior to the motor nucleus
- *Solitary nucleus:* Located posterior and lateral to the superior salivatory nucleus

L.B. Eisenmenger, M.D. (✉)
Department of Radiology and Imaging Sciences, University of Utah Health Sciences Center, 30 North, 1900 East, #1A071, Salt Lake City, UT 84132-2140, USA
e-mail: Laura.Eisenmenger@hsc.utah.edu

R.H. Wiggins III, M.D.
Departments of Radiology and Imaging Sciences, Otolaryngology, Head and Neck Surgery, and BioMedical Informatics, University of Utah Health Sciences Center, 30 North, 1900 East, #1A071, Salt Lake City, UT 84132-2140, USA
e-mail: Richard.Wiggins@hsc.utah.edu

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18.1.2 Facial Nerve Segments

- Cisternal
- Canalicular
- Labyrinthine
- Tympanic
- Mastoid
- Extracranial

18.1.3 Branches

- *Anterior genu:*
  - Greater superficial petrosal nerve (GVE)
- *Descending mastoid segment:*
  - Stapes muscle nerve (GVE)
  - Chorda tympani (GVE + SA)
- *Intraparotid branches (SVE):*
  - Temporal
  - Zygomatic
  - Buccal
  - Mandibular
  - Cervical
- *Extraparotid Branches:*
  - Posterior auricular nerve

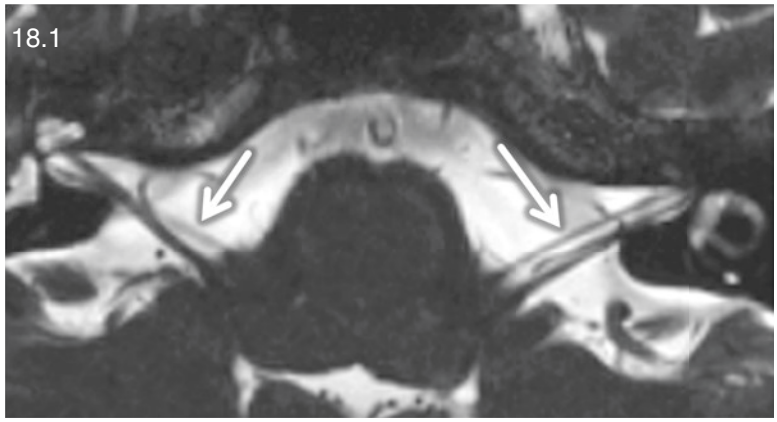

Fig. 18.1 Axial high-resolution CISS T2 MR image at the level of the cerebellopontine angle cistern and internal auditory canal. The facial nerve roots (*arrows*) exit anterior to the vestibulocochlear nerve from the inferior and anterior pons, above the pontomedullary junction bilaterally. The facial nerve remains anterior to the vestibulocochlear nerve as it crosses through the cerebellopontine angle cistern

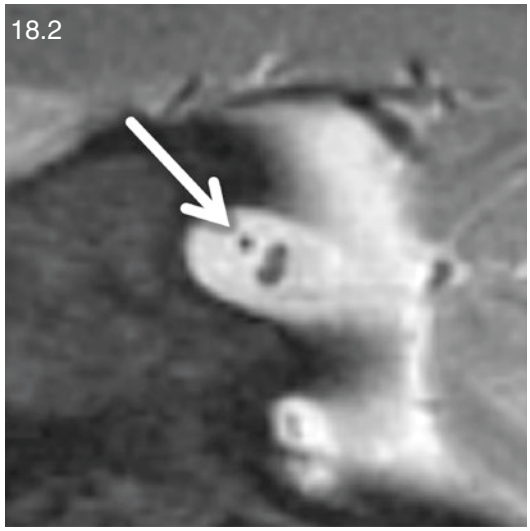

Fig. 18.2 Oblique sagittal T2 MR image through the porus acusticus reveals the characteristic "ball in catcher's mitt" appearance of the facial nerve (*arrow*) and the vestibulocochlear nerve. The facial nerve is the "ball," and the vestibulocochlear nerve is the "catcher's mitt", inferior and posterior to the facial nerve within the opening of the IAC

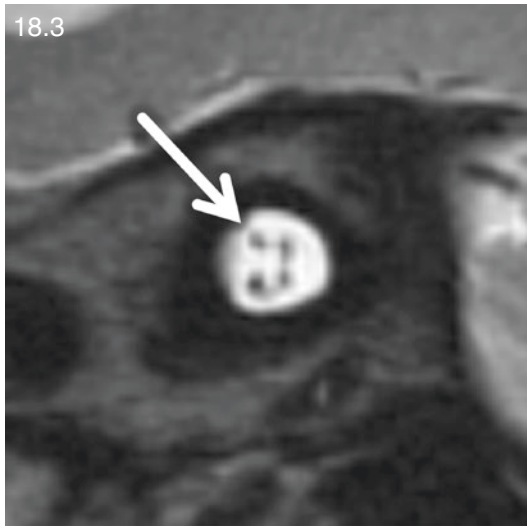

Fig. 18.3 Oblique sagittal T2 MR images through the mid-internal auditory canal (IAC). The four nerves which travel in the IAC are clearly identified. The common mnemonic used to remember the nerves in the IAC is 7-up (anterosuperior, facial nerve (*arrow*)) and coke-down (cochlear nerve anteroinferior, inferior to the facial nerve). The posterior nerves are the superior (posterior to the facial nerve) and inferior (posterior and inferior to the facial nerve) vestibular nerves

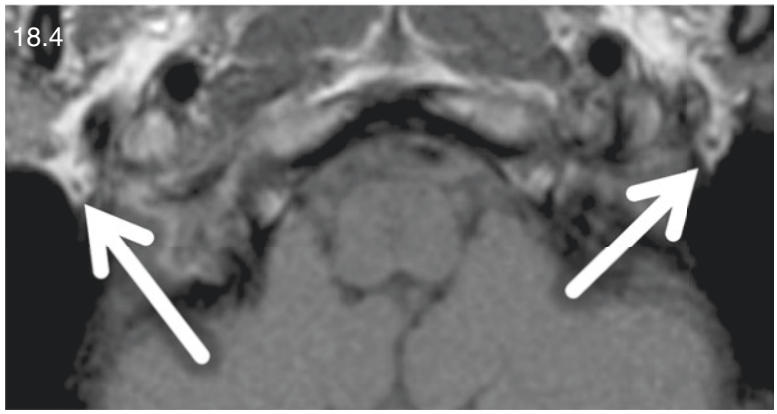

Fig. 18.4 Axial T1 noncontrasted MR image at the level of the stylomastoid foramen shows the exiting low-signal facial nerve (*arrows*) surrounded by high-signal fat in the "bell" of the stylomastoid foramen. The fat surrounding the facial nerve may be obscured if perineural tumor spread is present

18.2 Function

- Branchial motor function (SVE): Motor fibers extend through the temporal bone through the stylomastoid foramen to supply the voluntary and involuntary muscles of facial expression.
- Visceral motor function (GVE): Parasympathetic fibers travel along the greater superficial petrosal nerve to the pterygopalatine ganglion to supply innervation to the lacrimal gland and nasal mucosa. There are also parasympathetic fibers that travel with the chorda tympani to supply innervation to the submandibular and sublingual glands.
- Special sensory function (SA): Sensory fibers extend through the middle ear along the chorda tympani to join the posterior division of V3 to form the lingual nerve. The chorda tympani component of the facial nerve carries taste information from the anterior two thirds of the tongue.

18.3 Pathology

Individual symptoms: Damage to the facial nerve results in the following symptoms depending on location:

- *Supranuclear lesions:* Contralateral paresis/palsy of lower portion of the face (central paresis) with flattening of the nasolabial fold (unopposed contraction of the contralateral side of the orbicularis oris), possible drooling of saliva, and general sparing of the upper half of the face (corrugation of the forehead and voluntary eyelid closure possible).
- *Pontine lesions:* Isolated lesions of the pons can result in a facial nerve neuropathy. Etiologies such as ischemia, neoplasm, and vascular malformations such as a cavernous malformation or demyelinating pathologies are possible.
- *Cisternal lesions:* Masses within the cerebellopontine cistern can present with facial nerve neuropathy, such as a facial nerve schwannoma, meningioma, or metastatic disease. Neurovascular compression can result in a facial hemispasm. Lesions in the tympanic segment will cause a "peripheral" paralysis characterized by ipsilateral muscular weakness of the upper and lower face (flattening of the nasolabial fold and lagophthalmos).
- *Intratemporal lesions:* Lesions such as facial nerve schwannomas have varied appearances when found within the temporal bone based on the surrounding anatomic landscape of the involved segments [8]. Herpetic infection of the facial nerve (Bell's palsy) is demonstrated on imaging with normal CT osseous borders, but avid enhancement throughout the intratemporal bone and a tuft of enhancement within the lateral IAC. Traumatic fractures may injure the facial nerve within the temporal bone.
- *Extracranial lesions:* Consider neoplasms as major causes of extracranial CN VII dysfunction. Perineural tumor spread can be seen with minor salivary gland carcinomas, such as adenoid cystic carcinoma, or with squamous cell carcinoma.

18.3.1 Syndromes

Millard-Gubler syndrome: Lesion in ventral pons involves CN VI, VII, and corticospinal tract. The patient will present with contralateral hemiplegia, ipsilateral facial, and abducens palsy [9].

Foville's syndrome: Inferior medial pontine syndrome characterized by ipsilateral facial nerve paralysis, ipsilateral conjugate gaze paralysis, and contralateral hemiplegia [9].

Herpes Zoster Oticus (Ramsey-Hunt Syndrome): Characterized by ipsilateral facial nerve palsy and erythematous vesicular rash over the ipsilateral ear and the mouth. The varicella zoster virus (HSZ) lies in the geniculate ganglion. Patients may also complain of vertigo, tinnitus, and hearing loss since the geniculate ganglion is close to the inner ear structures [10].

Möbius syndrome: See Chap. 17.

Herpetic palsy (Bell's Palsy): Most frequent cause of sudden ipsilateral peripheral facial nerve paresis or paralysis of unknown etiology. It is generally self-limited and may be linked to the Herpes simplex virus (HSV) [11].

Geniculate neuralgia (aka tic douloureux of the nervus intermedius of Wrisberg): Patients present with paroxysmal otalgia. It may be caused by compression of the nervus intermedius and the geniculate ganglion, requiring microsurgical decompression or classic antiepileptic drugs [12].

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Cranial Nerve VIII: Vestibulocochlear

19

Miriam E. Peckham and Richard H. Wiggins III

19.1 Anatomy

The vestibule and cochlea develop from the embryonic otic placode. The vestibulocochlear nerve exits the pons anteriorly and laterally, superior to the pontomedullary junction at the restiform bodies, and extends superiorly and laterally through the cerebellopontine angle to enter the internal auditory canal (IAC) through the porus acusticus [1] (Fig. 19.1). Within the medial half of the IAC, the vestibulocochlear nerve separates into three distinct nervous structures: the cochlear nerve, the superior vestibular nerve, and the inferior vestibular nerve, which are most commonly identified separately at the midpoint of the IAC.The cochlear nerve is located anteriorly and inferiorly, below the facial nerve, the superior vestibular nerve is located posteriorly and superiorly, and the inferior vestibular nerve is located posteriorly and inferiorly (Figs. 19.2 and 19.3) [2]. Laterally within the IAC, there is a variably ossified horizontal septation (crista falciformis) separating the superior facial nerve and superior vestibular nerve from the inferior cochlear nerve and inferior vestibular nerve. There is also a variable superior vertical fascial septation (Bill's bar) at the lateral IAC, separating the facial and superior vestibular nerves.

The cochlear nerve extends anteriorly at the lateral IAC, through the cochlear nerve canal to enter the modiolus of the cochlea, which is populated by the spiral ganglia (Fig. 19.4). The superior vestibular nerve divides within the lateral IAC into three separate nerves: the utricular nerve to the utricle (a subunit of the vestibule with the saccule), the superior ampullary nerve to the ampulla of the superior semicircular canal, and the lateral ampullary nerve to the ampulla of the lateral semicircular canal. The inferior vestibular nerve divides into the saccular nerve to the saccule (a subunit of the vestibu-le with the utricle), and the posterior ampullary nerve to the ampulla of the posterior semicircular canal.

Intracranial course: The vestibular nuclei have several outputs and are a part of several tracts such as the vestibule-cerebellar tracts, medial and lateral vestibulospinal tracts, and the medial longitudinal fasciculus that help in providing sense of balance and maintaining pull against gravity [3]. Cochlear nuclei efferents largely cross over and form the lateral lemniscus to reach the inferior colliculi. The inferior colliculi projects to the medial geniculate bodies, and through the auditory radiations (geniculotemporal fibers), reach Heschl's gyri.

M.E. Peckham, M.D. (✉)
Department of Radiology and Imaging Sciences, University of Utah Health Sciences Center, 30 North, 1900 East, #1A071, Salt Lake City, UT 84132-2140, USA
e-mail: miriam.peckham@hsc.utah.edu

R.H. Wiggins III, M.D.
Departments of Radiology and Imaging Sciences, Otolaryngology, Head and Neck Surgery, and BioMedical Informatics, University of Utah Health Sciences Center, 30 North, 1900 East, #1A071, Salt Lake City, UT 84132-2140, USA
e-mail: Richard.Wiggins@hsc.utah.edu

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19.1.1 Nuclei

- Cochlear Nuclei:
  - *Ventral cochlear nuclei:* Anterior and lateral to the inferior peduncle
    Posteroventral cochlear nucleus
    Anteroventral cochlear nucleus
  - *Dorsal cochlear nuclei:* Posterior and lateral brainstem surface around cerebellar peduncle
- Vestibular Nuclei:
  - *Medial vestibular nucleus:* Superior medulla near floor of fourth ventricle within the medulla oblongata, within the inferior portion of the area acustica in the rhomboid fossa
  - *Lateral vestibular nucleus:* Superior medulla lateral to medial nucleus, within the lateral angle of the rhomboid fossa
  - *Inferior vestibular nucleus:* Inferior medulla, inferior to medial and lateral nuclei
  - *Superior vestibular nucleus:* Inferior pons, superior to inferior and medial nuclei

19.1.2 Branches

- Cochlear nerve (SA)
- Superior vestibular nerve (SA)
  - Utricular nerve
  - Superior ampullary nerve
  - Lateral ampullary nerve
- Inferior vestibular nerve (SA)
  - Saccular nerve
  - Posterior ampullary nerve

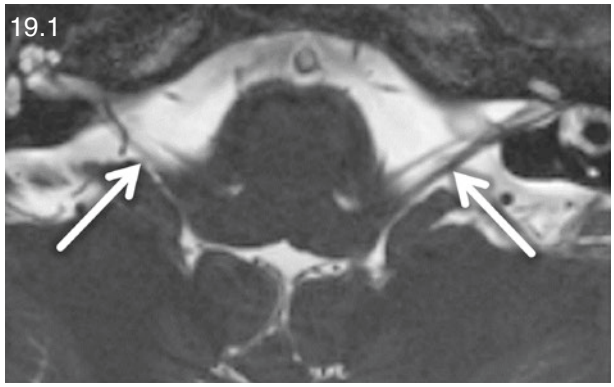

Fig. 19.1 Axial thin section CISS (CSF bright) MRI image through the level of the CPA demonstrates the origin of CN8 (*arrows*) at the restiform bodies

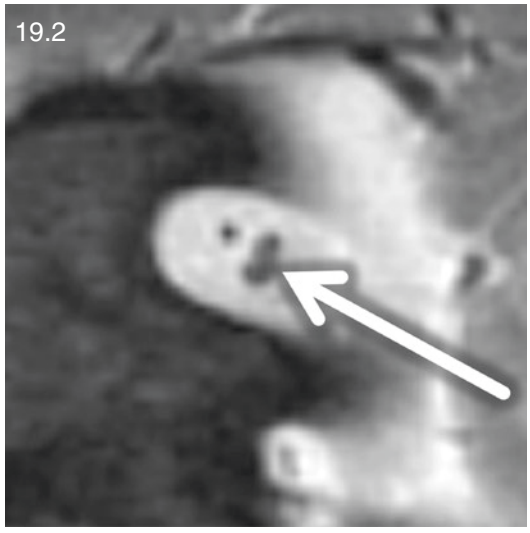

Fig. 19.2 Oblique sagittal T2W (CSF bright) MRI image through the porus acusticus (opening of the IAC) demonstrates the vestibulocochlear nerve (*arrow*) inferior and posterior to the facial nerve

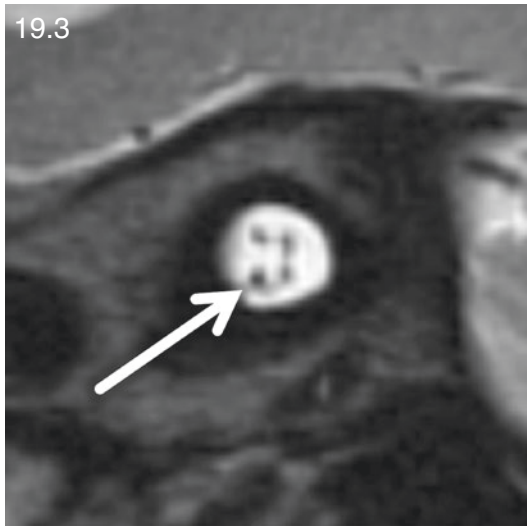

Fig. 19.3 Oblique sagittal T2W (CSF bright) MRI image through the midportion of the IAC demonstrates the cochlear nerve (*arrow*) inferiorly and anteriorly. The facial nerve is seen superiorly and anteriorly within the IAC, superior to the cochlear nerve, reminding us of the memory phrase, "7-up, coke-down" (facial nerve up and cochlear nerve down), describing the position of these two nerves within the lateral IAC. The posterior superior and inferior vestibular nerves are separating at this midpoint posteriorly within the IAC

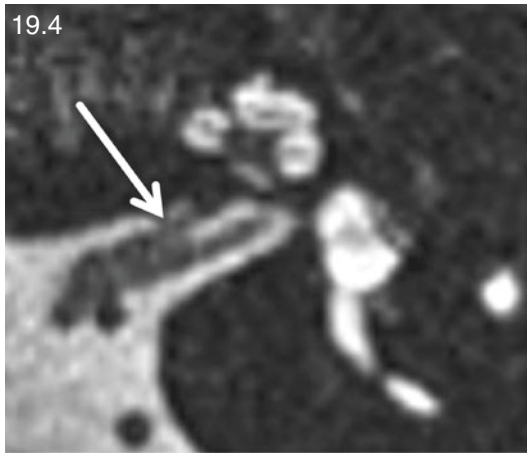

Fig. 19.4 Axial thin section magnified T2W (CSF bright) MRI image through the IAC demonstrates the cochlear nerve (*arrow*) anteriorly within the IAC, extending toward the bowtie-shaped modiolus within the center of the cochlea

19.2 Function

- Special sensory function (SA):
  - Cochlear nerve: Transmits auditory sensory information from the cochlea to the brain
  - Vestibular nerve: Transmits balance and spatial orientation information from the vestibule and semicircular canals to the brain. Increases tone in antigravitary extensors and holds eyes on target while the head moves.

19.3 Pathology

Individual symptoms: Sensorineural hearing loss, pulsatile or nonpulsatile tinnitus, and vertigo [4]. Damage to the vestibulocochlear nerve results in the following symptoms depending on location:

- *Supranuclear lesions:* Isolated lesions may cause subtle defect in the spatial localization of sound. Bilateral cortical lesions can cause pure word deafness (auditory verbal agnosia).

- *Pontomedullary lesions:* Isolated lesions of the pontomedullary junction can result in a vestibulocochlear nerve neuropathy, such as ischemia, neoplasm, vascular malformations, and/or demyelinating pathologies. Ipsilateral lateral lemniscus lesions will cause bilateral hearing loss more on the contralateral side.
- *CPA cisternal lesions:* Lesions within the cerebellopontine angle (CPA) cistern can present with vestibulocochlear nerve neuropathy, such as a vestibulocochlear nerve schwannoma, which represents approximately 90% of all pathologies of the CPA. There are several CPA pathologies which can mimic a vestibulocochlear nerve schwannoma, such as meningioma and metastatic disease, both pial and dural metastatic spread. Metastatic disease of the choroid plexus within the lateral cistern of the fourth ventricle and metastatic disease of the flocculus may also present on imaging as a mimic of vestibulocochlear schwannomas. One important mimic is the facial nerve schwannoma, which can not only present identically to a vestibulocochlear nerve schwannoma on imaging, but also on clinical presentation, as 50% of facial nerve schwannomas within the IAC may present with a component of hearing loss. This important distinction suggests that the cochlear nerve is more sensitive to external compression within the IAC than the facial nerve is sensitive to internal derangement, as these cases do not all present with facial paralysis [4, 5].
- *Intratemporal/intra-auricular lesions:* Lesions within the inner ear can also present with vestibulocochlear disorders. Rarely, schwannomas may occur within the inner ear structures, as an intracochlear or intravestibular mass. Labyrinthine ossificans can be thought of as an endpoint to damage to the inner ear. This can be found secondary to trauma to the inner ear or damage to the vascular or nervous supply to the inner ear. Early changes are demonstrated as loss of cerebrospinal (CSF) like bright signal intensity on CSF bright MRI sequences within the inner ear structures. Late labyrinthine ossificans changes are shown on CT as increased density within the inner ear structures. Trauma may also demonstrate blood products within the inner ear structures.

19.3.1 Syndromes

MISME (Multiple Inhereted Schwannomas, Meningiomas, and Ependymomas, or Neurofibromatosis, Type II) Presence of bilateral IAC, as these can also occur from the facial nerves cranial nerve schwannomas.

Neurovascular compression syndrome: Vertigo and pulsatile tinnitus are the most frequent symptoms. Compression of the nerve at the root entry zone in its rostro-ventral and caudal aspects by a vascular loop of the anteroinferior cerebellar artery is usually the cause. Hearing loss may not be present initially [6].

Some *syndromic inner ear malformations* are highly correlated with vestibule-cochlear nerve hypoplasia such as labyrinthine and cochlear aplasia, otocyst deformation, and complete aplasia of the semicircular canals.

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Cranial Nerve IX: Glossopharyngeal

20

Luisella Sibilla and Nivedita Agarwal

20.1 Anatomy

The glossopharyngeal nerve develops from the third pharyngeal arch. It exits the brainstem laterally from the post-olivary sulcus and leaves the skull through the pars nervosa of the jugular foramen anterior to the X and XI nerves (Fig. 20.1). It then courses between the internal jugular vein and the internal carotid artery, descends beneath the styloid process, and courses along the lower border of the stylopharyngeus muscle. From here it penetrates the pharyngeal constrictor muscle and reaches the posterior third of tongue terminating in several general somatic sensory branches such as lingual, tonsil, and pharyngeal branches.

The glossopharyngeal nerve has a communicating branch to the vagus nerve. The superior and inferior petrosal ganglia of the glossopharyngeal nerve lie mostly within the jugular foramen [1, 2].

20.1.1 Nuclei

- Nucleus ambiguus: The motor nucleus located in the upper medulla, just dorsal to inferior olivary nucleus. Contains efferent cell bodies to stylopharyngeus muscle and to superior pharyngeal constrictor
- Inferior salivatory nucleus: Parasympathetic nucleus stimulating the salivary glands
- Inferior solitary tract and nucleus: Sensory nucleus for the special visceral information
- Nucleus of the spinal tract of the trigeminal nerve: Sensory nucleus for the general somatic information

20.1.2 Branches

- Tympanic nerve (nerve of Jacobson) (GSA): It branches off the inferior petrosal ganglion and, through the inferior tympanic canaliculus, ascends the middle ear cavity forming the tympanic plexus on the cochlear promontory.
- Tympanic plexus (GVE): The tympanic nerve together with superior and inferior

L. Sibilla, M.D. (✉)
Section of Neuroradiology, Department of Radiology, Sahlgrenska University Hospital,
Gröna stråket 2 plan 2, 413 45 Gothenburg, Sweden
e-mail: luisella.sibilla@vgregion.se

N. Agarwal, M.D.
S. Maria del Carmine Hospital, Azienda Provinciale per i Servizi Sanitari, Rovereto (TN), Italy

Center for Mind/Brain Sciences (CIMeC), University of Trento, Rovereto (TN), Italy

Department of Radiology, Section of Neuroradiology, University of Utah, Salt Lake City (UT), USA
e-mail: Nivedita.agarwal@apss.tn.it

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caroticotympanic nerves forms the tympanic plexus providing via the lesser superficial petrosal nerve parasympathetic innervation to the parotid gland via otic ganglia. Other branches of the plexus include a branch to the greater superficial petrosal nerve and general somatic branches to the inner aspect of the tympanic membrane, eustachian tube, and mastoid cells.

- Carotid sinus nerve (Hering's nerve) (GVA): The nerve carries information from chemoreceptors that detect blood oxygen and carbon dioxide levels. Fibers travel via the inferior petrosal ganglion to the nucleus solitarius to control respiratory rate.
- Pharyngeal branches (GSA): Together with the branches of the vagus nerve, these form the pharyngeal plexus (see CN X).
- Stylopharyngeal branch (SVE): It innervates the stylopharyngeus muscle and parts of the superior constrictor pharyngeal muscle.

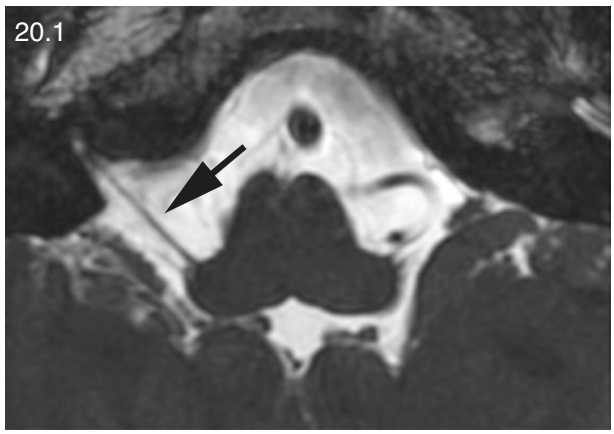

Fig. 20.1 Thin glossopharyngeal nerve exiting the lateral aspect of the medulla (*arrow*) coursing to the pars nervosa of the jugular foramen

20.2 Function

- Branchial motor function (SVE): The muscular branch from the nucleus ambiguus innervates the stylopharyngeal muscle to elevate the pharynx during phases of swallowing and speech. It also innervates the superior pharyngeal constrictor together with muscular branches of CN X.
- Visceral motor function (GVE): Fibers from the inferior salivatory nucleus innervate the parotid gland to provide parasympathetic innervation to the parotid gland via the lesser superficial petrosal nerve (promotes salivation).
- General somatic sensory function (GSA): It carries general sensory information from the posterior one-third of the tongue, the tonsil, the skin of the external ear, the internal surface of the tympanic membrane, and the pharynx via the superior and inferior petrosal ganglia to the spinal trigeminal nuclei.
- Special sensory function (SA): It provides taste sensation from the posterior one-third of the tongue, the circumvallate papillae, posterior pharynx, and the Eustachian tube. This information travels via the inferior petrosal nerve to the nucleus solitarius.
- General visceral sensory function (GVA): carries visceral changes in blood O_2 and CO_2 from the carotid body (chemoreceptors) and increased blood pressure information from the carotid sinus (baroreceptors) via the carotid sinus nerve to the inferior petrosal ganglia to reach the nucleus solitaries and the inferior salivatory nucleus, respectively. The former triggers control of respiratory depth and rate, while the latter will cause a vagal response with consensual activation of the dorsal nucleus of CN X.

20.3 Pathology

Individual symptoms: Damage to the glossopharyngeal nerve results in the following symptoms depending on location [3, 4]:

- Supranuclear lesions: Unilateral lesions usually are asymptomatic because corticobulbar innervation is bilateral. Bilateral lesions may give rise to pseudobulbar palsy characterized by dysphagia, spastic dysarthria, and pathologic bursts of crying with loss of emotional control. Dysfunctional or absence of gag reflex may indicate CN IX paralysis (tongue retraction and elevation of the pharynx).
- Medullary lesions: Isolated lesions of the dorsal nuclei can cause dysphagia and dysarthria, stroke, tumors, and inflammation.
- Demyelinating lesions are most frequent to consider as etiologies of the disease.
- Cisternal lesions: Tumors and neurovascular compression can cause glossopharyngeal neuralgia. It typically presents with an excruciating pain in the tonsils that radiates to the ear, triggered by various stimuli as swallowing and yawning. It can be associated with bradycardia and syncope.
- Extracranial lesions: Consider neoplasms, inflammation, infections, compressive injury, and trauma as major causes of CN IX dysfunction. Isolated lesions will rarely arise from diseases in the extracranial compartment due to dense anastomosis of branches with other cranial nerves such as X and XI.

20.3.1 Syndromes

Eagle syndrome: Deep throat pain and/or ear pain due to elongated or calcified styloid process causing entrapment of CN IX.

Vernet syndrome (aka jugular foramen syndrome): Damage to cranial nerves at the level of the jugular foramen (IX, X, and XI) results in ipsilateral paresis of the sternocleidomastoid and trapezius muscle, dysphonia, dysphagia, homolateral vocal cord paralysis, and loss of sensation and taste from the ipsilateral posterior one-third of the tongue, uvula, larynx, and pharynx.

Collet-Sicard syndrome: This is Vernet syndrome and lesion of the XII cranial nerve leading to symptoms similar to Vernet syndrome with additional ipsilateral paresis of the tongue.

Schmidt syndrome: Collet-Sicard syndrome and contralateral hemiparesis/plegia.

Villaret's syndrome (aka retroparotid space syndrome): Ipsilateral palsy of the nerves IX, X, XII, and XII and ipsilateral Horner' syndrome (damage to the sympathetic chain causes ipsilateral ptosis, miosis, and anhidrosis).

Wallenberg syndrome (aka lateral medullary syndrome): Ipsilateral vestibular and cerebellar signs and symptoms, ipsilateral laryngeal, pharyngeal paralysis, dysphagia, dysarthria, contralateral deficits of pain and temperature, and ipsilateral Horner's syndrome.

Opalski syndrome: Wallenberg syndrome and ipsilateral hemiplegia/paresis (due to extension of the lesion caudal to the pyramidal decussation).

Babinski-Nageotte syndrome: Wallenberg syndrome and contralateral hemiplegia/paresis.

Frey syndrome (aka auriculotemporal syndrome): Complications of parotid surgery may cause damage to auriculotemporal nerve (parasympathetic component to the parotid gland). Aberrant anastomosis with the parasympathetic component in the facial nerve can cause ipsilateral abnormal seating and flushing of the skin around the ear and the angle of the mandible while eating [5].

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Cranial Nerve X: Vagus

21

Luisella Sibilla and Nivedita Agarwal

21.1 Anatomy

The vagus nerve develops from the third and fourth pharyngeal arches. The vagus nerve exits the medulla from the post-olivary sulcus and leaves the skull through the pars vascularis of the jugular foramen (Fig. 21.1). The superior jugular ganglion lies within the jugular foramen giving off meningeal branches. Just below the jugular foramen are the inferior nodose ganglia involved in visceral afferent and special sensory information. Then it descends in the carotid sheath between the internal carotid artery and the internal jugular vein giving off branches to the viscera [1, 2].

L. Sibilla, M.D. (✉)
Section of Neuroradiology, Department of Radiology, Sahlgrenska University Hospital,
Gröna stråket 2 plan 2, 413 45 Gothenburg, Sweden
e-mail: luisella.sibilla@vgregion.se

N. Agarwal, M.D.
S. Maria del Carmine Hospital, Azienda Provinciale per i Servizi Sanitari, Rovereto (TN), Italy

Center for Mind/Brain Sciences (CIMeC), University of Trento, Rovereto (TN), Italy

Department of Radiology, Section of Neuroradiology, University of Utah, Salt Lake City (UT), USA
e-mail: Nivedita.agarwal@apss.tn.it

21.1.1 Nuclei

- Dorsal nuclei of the vagus nerve (motor nucleus of vagus): lies in dorsal medulla just lateral to the midline in the floor of the fourth ventricle (vagal trigone). It contains parasympathetic secretomotor fibers to mucosa in the thorax walls, abdomen (esophageal, gastric, cardiac, splenic, and hepatic plexuses), and pharyngeal and laryngeal mucosa.
- Nucleus ambiguus: a motor nucleus located in the upper medulla, dorsal to the inferior olivary nucleus. Contains efferent cell bodies to striated muscles of the larynx and pharynx (pharyngeal constrictors).
- Superior and inferior solitary nuclei: superior solitary nucleus receives gustatory information together with CN VII and CN IX. Inferior solitary nucleus receives visceral sensation via vagal branches to nodose ganglia.
- Nucleus of the spinal tract of the trigeminal nerve: receives general somatic information from the ipsilateral face.

21.1.2 Branches

- Auricular branch (aka Arnold's nerve) (GSA): sensory innervations to the skin of the ear canal, tragus, and auricle
- Pharyngeal nerve (SVE): supplies the muscles of the soft palate and pharynx

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- Superior laryngeal nerve (SVE): supplies the inferior pharyngeal constrictor and cricothyroid muscles of the larynx
- Superior cervical cardiac branch (GVE): supplies the cardiac plexus
- Inferior cervical cardiac branch (GVE): supplies the cardiac plexus
- Recurrent laryngeal nerve (SVE and GSA): innervates all intrinsic laryngeal muscles except the cricothyroid and supplies GSA to vocal cords and the subglottis
- Thoracic cardiac branches (GVE): supply ganglia and plexuses in the thoracic walls
- Branches to the pulmonary plexus (GVE): supply ganglia and plexuses in the pulmonary and bronchial walls
- Branches to the esophageal plexus (GVE): supply plexuses in the esophageal walls
- Anterior vagal trunk (GVE): supplies the hepatic, celiac, and gastric plexuses
- Pharyngeal plexus (SVE): pharyngeal nerve of CN X, pharyngeal branches of CN IX, and external laryngeal nerve of CN X form a pharyngeal plexus

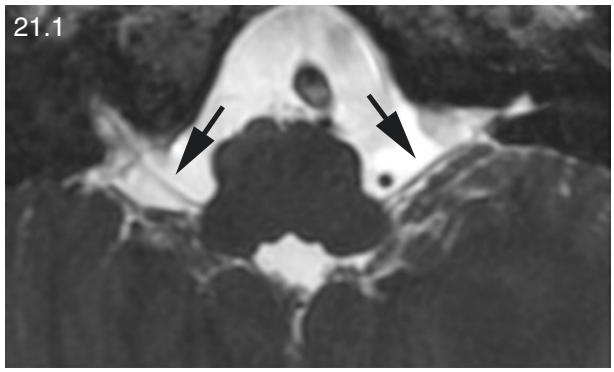

Fig. 21.1 The vagus nerves (arrows) exit the medulla and travel laterally to the pars vascularis of the jugular foramina

21.2 Function

- Special visceral motor function (SVE): from nucleus ambiguus to all intrinsic muscles of the larynx, all pharyngeal and soft palate muscles except for stylopharyngeal muscle (CN IX) and tensor veli palatini (CN V). The nucleus is supplied by upper motor neurons bilaterally in the corticobulbar tracts.
- Visceral motor function (GVE): parasympathetic component from dorsal motor nucleus to muscle and glands of the pharynx, larynx, and thoracic (heart, lungs, esophagus) and abdominal viscera (gastric and celiac plexus). The vagus nerve modulates several essential parasympathetic visceral functions, such as determining optimal respiratory and cardiac rates and aiding in digestion by controlling salivation and gut peristalsis.
- General somatic sensory function (GSA): sensations from posterior fossa dura mater, nasal concha, skin on the external pinna of the ear, external acoustic meatus, external surface of the tympanic membrane (Arnold's nerve), pharyngeal and laryngeal mucosa reach the superior jugular ganglia and then to the nucleus of the spinal trigeminal nucleus.
- Special sensory function (SA): taste sensation from the epiglottis and hard and soft palate reaches the nucleus solitarius via the inferior ganglion.
- General visceral sensory function (GVA): visceral afferent sensations from thoracic and abdominal viscera, from larynx, trachea, esophagus, carotid, and aortic arch baroreceptors reach the caudal nucleus solitarius via the inferior nodose ganglion.

21.3 Pathology

Individual symptoms: Damage to the vagus nerve results in the following symptoms depending on location [3, 4]:

- Supranuclear lesions: Unilateral lesions of the motor and sensory cortices are usually asymptomatic due to the bilateral innervation of the nucleus ambiguus. Bilateral lesions will cause moderate to severe dysarthria and dysphonia and may result in pseudobulbar palsy.
- Medullary lesions: Unilateral lesions in the dorsal lower medulla usually involve other cranial nerve dysfunction such as CN IX and XI. Most frequent etiologies involve stroke and inflammatory/demyelinating diseases. Symptoms usually cause dysarthria and dysphonia without contralateral hemiparesis. Damage to the inferior nucleus solitarius can give a host of vagal parasympathetic dysfunction symptoms such as bradycardia and hypotension, and sudden decrease in skin temperature can result with increased vagal stimulation leading to a vasovagal syncope. Syringobulbia may cause vagal nerve lesions.
- Cisternal lesions: These are rare and seldom occur in an isolated fashion.
- Extracranial lesions: These may affect any part of the vagal nerve in its course along the thoracic and abdominal viscera causing increased sympathetic activity. Unilateral lesions in the recurrent laryngeal nerve will cause hoarseness due to paralysis of all laryngeal muscles except the cricothyroid, whereas bilateral damage will cause inspiratory stridor and severe dysphonia to complete aphonia.

21.3.1 Syndromes

Vernet syndrome (aka jugular foramen syndrome): see Chap. 20.

Avellis syndrome (laryngeal hemiplegia): usually it is caused by a medial medullary stroke that involves the nucleus ambiguus with or without contralateral hemiparesis/plegia.

Collet-Sicard syndrome: see Chap. 20.

Schmidt syndrome: see Chap. 20.

Wallenberg syndrome: see Chap. 20.

Amyotrophic lateral sclerosis: degeneration of lower motor neurons in the brain stem nuclei is characterized by a bulbar syndrome (dysarthria, dysphagia).

Chiari I malformation: rarely, if complicated by syringobulbia, it can cause lower cranial nerve damage.

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Cranial Nerve XI: Spinal Accessory

22

Laura B. Eisenmenger and Richard H. Wiggins III

22.1 Anatomy

The spinal accessory nuclei are derived from the basal plate of the C1–C6 embryonic spinal segments. The spinal accessory nerve is the only cranial nerve that originates outside of the cranial vault and therefore is the only cranial nerve to both enter and exit the skull [1, 2]. The s*pinal root* is derived from neurons within the anterior horn of the cervical spinal cord which join together to form spinal rootlets, which exit the lateral spinal cord between the anterior and posterior upper cervical cord nerve roots. These rootlets combine to form roots which then join to form the spinal accessory nerve [3]. The spinal accessory nerve travels superiorly through the foramen magnum and then turns laterally to exit the skull base through the pars vascularis of the posterior and lateral jugular foramen (Fig. 22.1) [4, 5]. There is some debate about the presence of a *cranial root* of the spinal accessory nerve. It is thought to arise from the caudal part of the nucleus ambiguus and exit the post-olivary sulcus of the medulla, coursing along with the X cranial nerves (Fig. 22.2). It eventually blends with the spinal CN XI at the level of the superior jugular ganglion. The nerve then travels through the carotid sheath (carotid space) of the suprahyoid neck inferiorly, then most commonly extends laterally between the internal jugular vein and the posterior belly of the digastric muscle. The nerve continues inferiorly, deep to the sternocleidomastoid muscle, giving a sternocleidomastoid muscle branch. The remaining fibers then continue inferiorly and posteriorly to terminate in the trapezius muscle nerve

Nuclei: The spinal accessory nuclei are located in the upper cervical spinal cord, often referred together as the spinal nucleus. The cranial root nerves originate from the caudal part of the nucleus ambiguus.

22.1.1 Branches

- Sternocleidomastoid muscle nerve
- Trapezius muscle nerve

L.B. Eisenmenger, M.D. (✉)
Department of Radiology and Imaging Sciences, University of Utah Health Sciences Center, 30 North, 1900 East, #1A071, Salt Lake City, UT 84132-2140, USA
e-mail: Laura.Eisenmenger@hsc.utah.edu

R.H. Wiggins III, M.D.
Departments of Radiology and Imaging Sciences, Otolaryngology, Head and Neck Surgery, and BioMedical Informatics, University of Utah Health Sciences Center, 30 North, 1900 East, #1A071, Salt Lake City, UT 84132-2140, USA
e-mail: Richard.Wiggins@hsc.utah.edu

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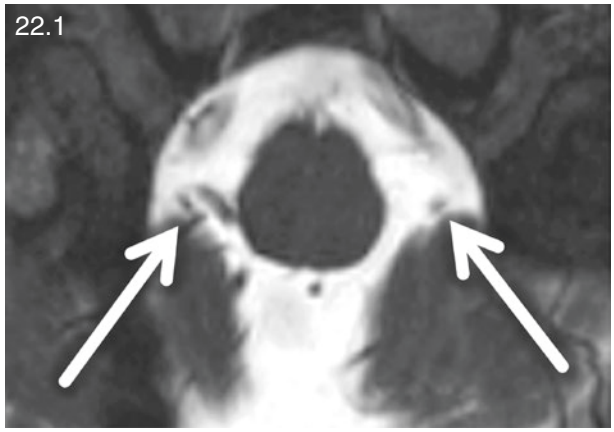

Fig. 22.1 Axial thin CISS MRI image at the level of the lower medulla shows the spinal root of CN11 (*arrows*) traveling cephalad through the foramen magnum to the join bulbar root of CN11 before joining in the lateral basal cistern and entering the pars vascularis of jugular foramen.

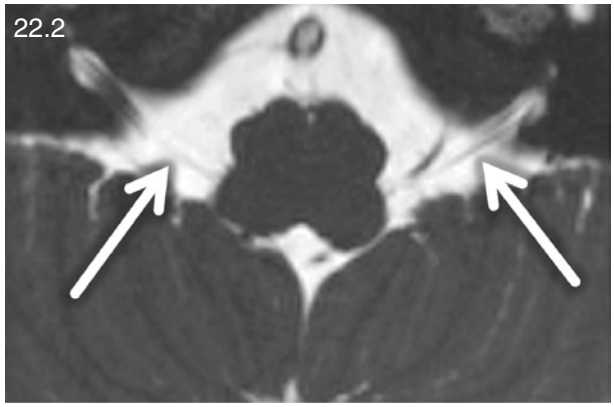

Fig. 22.2 Axial thin CISS MRI image at the level of the medulla shows the cranial root of CN11 (*arrows*) emerging from the post-olivary sulcus just inferior to CN10. The cranial root travels anterolaterally through the basal cistern with CN9 and CN10.

22.2 Function

Branchial motor function (GSE): Motor fibers innervate the sternocleidomastoid muscle to turn the head and the trapezius muscle to raise the scapula and shrug the shoulders.

22.3 Pathology

Individual symptoms: Damage to the spinal accessory nerve results in the following symptoms depending on the location:

- *Upper cervical spinal lesions*: Isolated lesions of the upper cervical spinal cord can result in spinal accessory neuropathy, such as ischemia, neoplasm, and demyelinating pathologies.
- *Cisternal lesions*: Lesions within the premedullary cistern may present with spinal accessory neuropathy, such as meningiomas and metastatic disease.
- *Jugular foramen lesions*: Lesions centered at the jugular foramen can present with spinal accessory dysfunction. At this location, a thin-bone algorithm (edge enhancement) CT and a thin-section contrasted MRI can distinguish pathologies. A jugular foramen paraganglioma causes permeative destructive (decreased density) changes to the jugular foramen walls, has variable phleboliths and/or slow flow and high-velocity flow voids leading to a salt and pepper (bright T1 and dark T1) appearance on MRI, and will have a superolateral vector of spread into the middle ear cavity. A jugular foramen

meningioma will cause permeative sclerotic (increased density) to the surrounding osseous walls, has dural tails on contrasted MRI, and a centrifugal spread in all directions from the jugular foramen. A jugular foramen schwannoma will cause smooth, scalloped changes to the jugular foramen, shows intratumoral cysts when larger, and has a vector of spread along the expected course of cranial nerves 9–11 superiorly and medially toward the midbrain and inferiorly and laterally into the carotid sheath (carotid space).

- *Cervical soft tissue lesions*: Traumatic changes and pathologies of the cervical soft tissues may also present with spinal accessory nerve dysfunction. Isolated CN11 dysfunction or injury is most commonly caused by neck dissection due to the close association of CN11 with the spinal accessory nodal chain [6]. The initial presentation of CN11 neuropathy is downward and lateral rotation of the scapula with the shoulder droop from loss of trapezius muscle tone [7]. With time, ipsilateral sternocleidomastoid and trapezius muscle atrophy progresses with compensatory hypertrophy of ipsilateral levator scapulae muscle. Hypertrophic levator scapulae muscle related to CN11 dysfunction should not be confused for a cervical mass.
- *Others*: Other pathologies within the cervical soft tissues that may lead to CN11 dysfunction include nerve sheath tumors, such as glomus vagale paragangliomas, schwannomas, and neurofibromas, as well as squamous cell carcinoma nodal disease or primary lateral extension, carotid artery dissection. Spinal accessory nerve schwannomas not associated with neurofibromatosis are very rare. When they do occur, they can involve either intrajugular or intracisternal portions of CN11 [8–10].

22.3.1 Syndromes

Collet-Sicard syndrome: see Chap. 20.
Vernet syndrome: see Chap. 20.
Schmidt syndrome: see Chap. 20.
Villaret's syndrome: see Chap. 20.

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Cranial Nerve XII: Hypoglossal

23

Craig M. Johnson and Richard H. Wiggins III

23.1 Anatomy

The hypoglossal nerve is derived from the basal plate of the embryonic medulla oblongata. It arises from the hypoglossal nucleus within the posterior and inferior medulla. The efferent nerve fibers then course anteriorly through the medulla, exiting at the pre-olivary (ventrolateral) sulcus to exit from the anterior medulla, and extend laterally to enter the hypoglossal canal (Fig. 23.1), located inferior to the jugular foramen and jugular tubercle within the inferior occipital bone [1, 2]. This segment is accompanied by a prominent venous plexus. The fibers exit the skull and then descend within the carotid space between the internal jugular vein and the internal carotid artery. The nerve then courses toward the hyoid bone, medial to the posterior belly of the digastric muscle, and then along the hyoglossus muscle, where muscular branches provide motor innervation to the majority of the tongue musculature.

Intracranial course: Upper motor neurons originate from lateral precentral gyrus and descend along the corticobulbar tract via the corona radiata and the internal capsule to reach the hypoglossal nuclei bilaterally except for the genioglossus muscle which is crossed and unilateral.

Nuclei: The hypoglossal nuclei are located in the dorsal medulla.

23.1.1 Branches

- Intrinsic tongue muscles (GSE)
  - Vertical tongue muscle nerve
  - Transverse tongue muscle nerve
  - Superior longitudinal muscle nerve
  - Inferior longitudinal muscle nerve
- Extrinsic tongue muscles (GSE)
  - Genioglossus muscle nerve
  - Hyoglossus muscle nerve
  - Styloglossus muscle nerve

C.M. Johnson, M.D. (✉)
Department of Radiology and Imaging Sciences, University of Utah Health Sciences Center, 30 North, 1900 East, #1A071, Salt Lake City, UT 84132-2140, USA
e-mail: Craig.Johnson@hsc.utah.edu

R.H. Wiggins III, M.D.
Departments of Radiology and Imaging Sciences, Otolaryngology, Head and Neck Surgery, and BioMedical Informatics, University of Utah Health Sciences Center, 30 North, 1900 East, #1A071, Salt Lake City, UT 84132-2140, USA
e-mail: Richard.Wiggins@hsc.utah.edu

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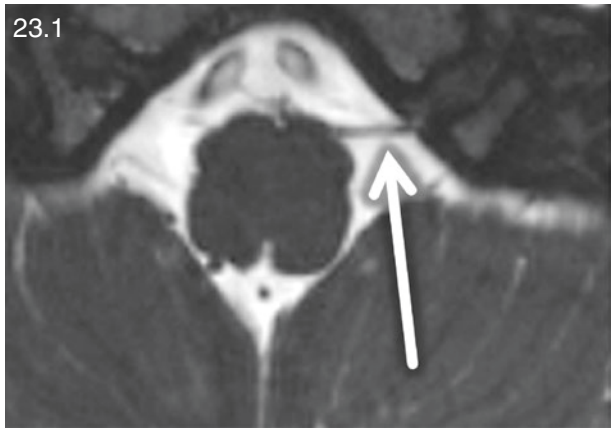

Fig. 23.1 Axial thin-section CSF bright sequence demonstrates the hypoglossal nerve (*arrow*) extending through the medullary cistern laterally toward the hypoglossal canal

23.2 Function

The hypoglossal nerve has only GSE motor fibers. It innervates the intrinsic and all of the extrinsic tongue muscles except the palatoglossus (which is innervated by CN X).

23.3 Pathology

Individual symptoms: Damage to the hypoglossal nerve results in the following symptoms depending on the location:

- *Supranuclear lesions*: Isolated lesions are rare and are typically associated with other neurologic findings. They are not associated with tongue fasciculations or atrophy. Spastic dysarthria and pseudobulbar palsy especially with bilateral cortical lesions are possible. When asked to protrude the tongue, the tongue will deviate away from the side of the lesion [3].
- *Lower medullary lesions*: Isolated lesions of the lower medulla such as ischemia, neoplasm, and/or demyelinating pathologies can result in hypoglossal nerve neuropathy. Pathology at this level will cause the tongue to deviate towards the side of the lesion.
- *Cisternal lesions*: Lesions within the medullary cistern may present with hypoglossal nerve neuropathy, such as meningiomas and/or metastatic disease.
- *Jugular foramen and extracranial lesions*: The most common lesion to involve the hypoglossal nerve is the hypoglossal schwannoma which accounts for only 5% of non-vestibular intracranial schwannomas. This lesion is a benign tumor of differentiated Schwann cells which may arise anywhere along the course of the hypoglossal nerve. A sharply marginated, fusiform mass which may exhibit a dumbbell shape is seen on CT or MRI [4, 5]. The most common MRI characteristics of this lesion include T1 isointensity to gray matter, T2 hyperintensity, and uniform enhancement. Intramural cysts may be identified if the lesion is large. Multiple lesions including skull base metastasis, glomus jugular paraganglioma, vascular variant persistent hypoglossal artery, or jugular foramen meningioma can mimic a hypoglossal schwannoma. Skull base traumatic fractures can also present with hypoglossal nerve dysfunction.
- *Others*: Hypoglossal nerve motor denervation can be secondary to hypoglossal schwannoma or any other skull base lesion causing mass effect on the hypoglossal nerve. Imaging demonstrates asymmetry of the tongue with linear demarcation corresponding to the hypoglossal nerve innervation. The appearance on CT and MRI of hypoglossal nerve motor denervation depends on the time course. In the initial stage, CT shows unilateral tongue swelling, while MRI demonstrates T2 hyper-

intensity and T1 hypointensity with variable enhancement consistent with edema. As the time course of denervation becomes chronic, there is resolution of the swelling and development of fatty atrophy of the affected half of the tongue. MRI demonstrates increasing T1 hyperintensity consistent with fatty atrophy of the tongue musculature and resolution of enhancement. Careful evaluation is recommended to not mistake this entity, with abnormal enlargement or enhancement of the tongue, for a tongue base tumor.

23.3.1 Syndromes

Dejerine syndrome (aka medial medullary syndrome): Occlusion of anterior spinal artery can cause ipsilateral loss of proprioception (damage to the medial lemniscus), ipsilateral paresis of the tongue (tongue deviates to the opposite side), and contralateral hemiplegia.

Pseudobulbar palsy: Bilateral supranuclear lesions can cause bilateral tongue paresis and dysarthria.

Collet-Sicard syndrome: See Chap. 20.

Schmidt syndrome: See Chap. 20.

Villaret's syndrome: See Chap. 20.

Jackson syndrome: Ipsilateral hypoglossal palsy, incomplete vagal paresis (dysarthria), and contralateral hemiparesis/plegia.

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Part IV

Surgical and Endoscopic Illustrative Anatomy

Neuroradiologists are trained to identify and localize disease on images created with X-rays and radio waves. This information is then used by neurosurgeons to treat their patients. However, the imaging planes used for diagnosis seldom reflect the actual neurosurgeon's view of the disease.

In an attempt to better educate neuroradiologists on how their images relate to neurosurgical anatomy, selected conditions of particular interest to neurosurgeons are presented in this section, as visualized through the eyes of the surgeon. It is useful to understand these relationships in order to better assist surgical planning.

These drawings were produced over a period of several years, the result of a passionate combination of the two disciplines of artistic drawing and decades of neurosurgical experience. All sketches were created on cardboard using the classic techniques, freehand drawing with pencil, colored pencil, and china pencil.

For each section, the following have been illustrated:

- Normal anatomy
- Major Clinical Symptoms & Related Clinical Syndromes
- Indications for surgery
- Goals of the surgery
- Surgical approach(es)
- Examples of normal and 'pathological' anatomy

Sellar Region

24

Vinicio M.F. Valente

24.1 Normal Anatomy

The hypophysis is an endocrine gland housed in the sella turcica. The sella turcica is a small concavity in the posterosuperior part of the sphenoid body. The optic chiasm is found immediately above, and the vascular structures of the anterior circulation lie around the sellar region. The boundaries of the hypophysis are (1) tuberculum sellae anteriorly that separates it from the ethmoid-sphenoidal planum, (2) cavernous sinus medially, (3) dorsum sellae posteriorly, and (4) the floor of the sella turcica caudally separating it from the sphenoid sinus (Figs. 24.1 and 24.2).

24.2 Major Clinical Symptoms

- Hormonal: hypersecretion of hormones (Cushing syndrome, acromegaly, hyperthyroidism).
- Focal neurologic symptoms: related to compression of the visual pathways lying just above the hypophysis (bitemporal homonymous hemianopsia, various degrees of visual field deficit).
- Non-localizing neurologic symptoms: intracranial hypertension with possible hydrocephalus, optic atrophy, and bilateral or unilateral papilledema.

24.3 Indications for Surgery

- Secreting microadenomas (≤10 mm in maximum diameter). Non-secreting microadenomas are "leave-alone" lesions and need to be periodically monitored using MRI.
- Macroadenomas (>10 mm in maximum diameter) with the exception of prolactin-secreting adenomas which are usually treated medically. Surgical treatment is required in cases of drug-resistant prolactinomas or in patients with exceptionally large symptomatic prolactinomas.
- Recurrent sellar tumors of any etiology.

24.4 Objectives of the Surgery

- To remove lesions causing hypersecretion of hormones while preserving normal hypophyseal function.
- To remove mass effect on adjacent structures.
- To preserve visual function and/or limit visual field defects.
- To provide histologic specimen where necessary to reach final diagnosis.

V.M.F. Valente, M.D.
Section of Neurosurgery, Hospital of Cosenza (Azienda Ospedaliera di Cosenza), Cosenza, Italy
e-mail: viniciovalente@alice.it

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24.5 Surgical Approach(es)

- Endoscopy is considered the gold standard, with the trans-rhinosphenoidal approach preferred (Figs. 24.3–24.10).
- Transcranial approach is reserved for lesions with extremely large dimensions with extrasellar component (suprasellar cistern, cavernous sinuses, and the middle cranial fossa). In case of hydrocephalus, ventriculoperitoneal shunting is also necessary [1–3].

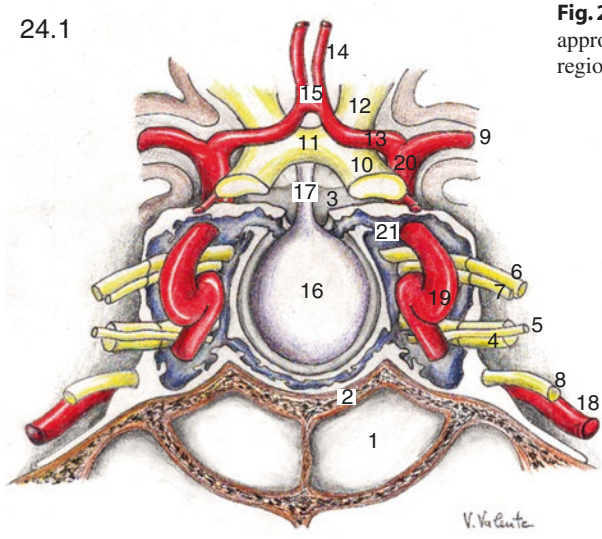

Fig. 24.1 Anatomical view from a trans-rhinosphenoidal approach showing the common structures in the pituitary region

1	Sphenoid sinus
2	Floor of the sella
3	Sellar tentorium
4	Ophthalmic branch of the trigeminal nerve (V_1)
5	Abducens nerve (VI)
6	Oculomotor nerve (III)
7	Trochlear nerve (IV)
8	Maxillary branch of the trigeminal nerve (V_2)
9	Middle cerebral artery (MCA)
10	Optic nerve
11	Optic chiasm
12	Optic tract
13	A1 seg of ACA
14	A2 seg of ACA
15	Anterior communicating artery (ACOM)
16	Hypophysis
17	Hyophyseal stalk
18	Petrous internal carotid artery (ICA)
19	Cavernous ICA
20	Supraclinoid ICA
21	Cavernous sinus

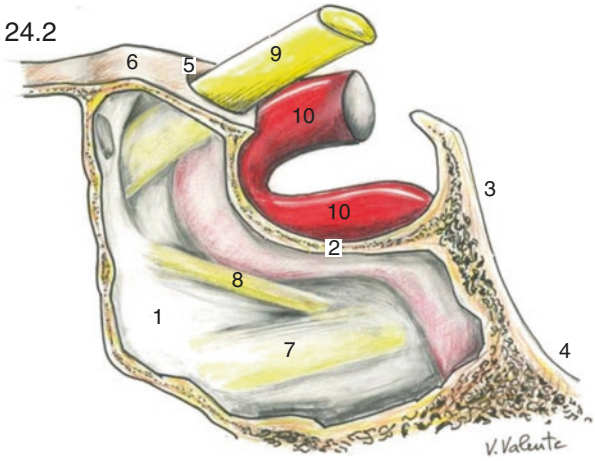

Fig. 24.2 Sagittal view showing the common structures in the pituitary region

1	Sphenoid sinus
2	Floor of the sella
3	Sella dorsae
4	Clivus
5	Optic foramen
6	Sellar tuberculum
7	Ophthalmic branch of the trigeminal nerve (V_1)
8	Abducens nerve (VI)
9	Optic nerve
10	Carotid siphon

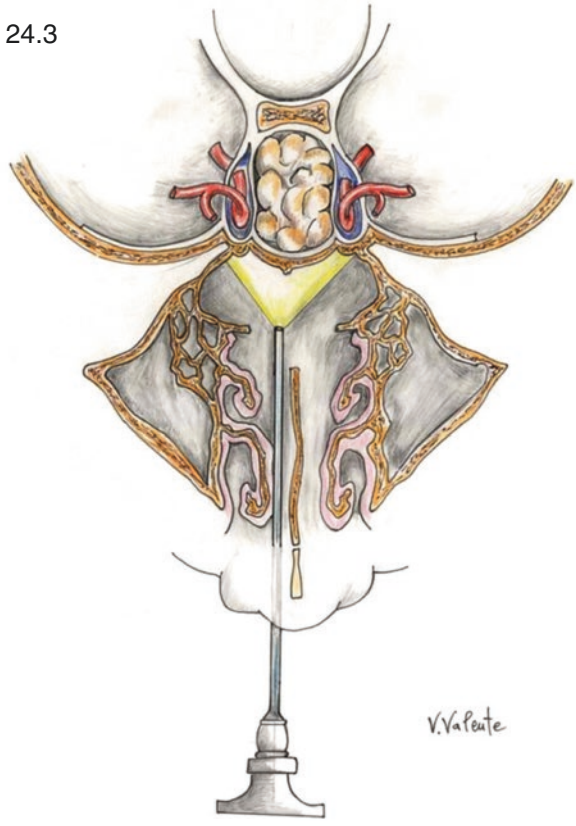

Fig. 24.3 Anterior view showing a trans-rhinosphenoidal endoscopic surgical approach

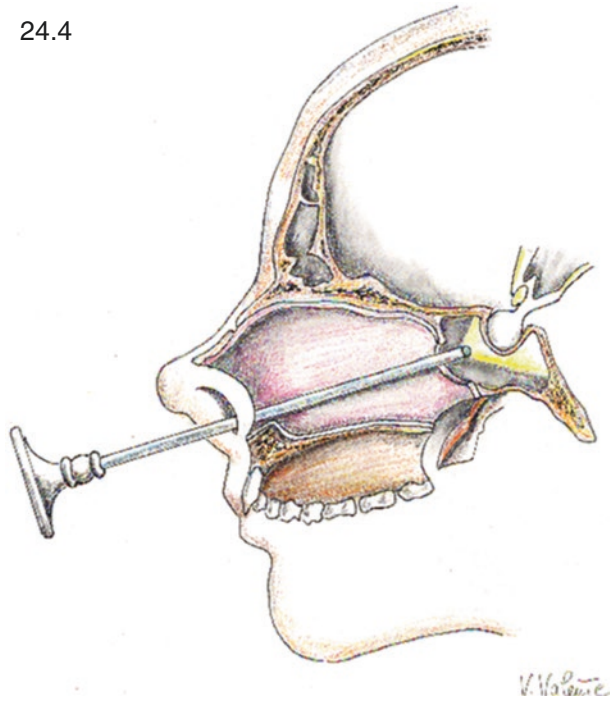

Fig. 24.4 Sagittal view showing a trans-rhinosphenoidal endoscopic surgical approach

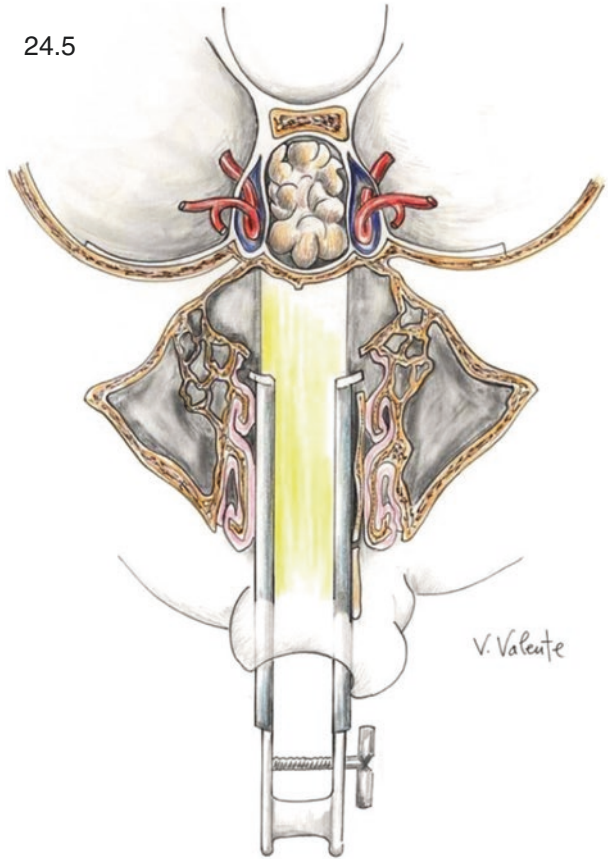

Fig. 24.5 Anterior view showing a microsurgical approach

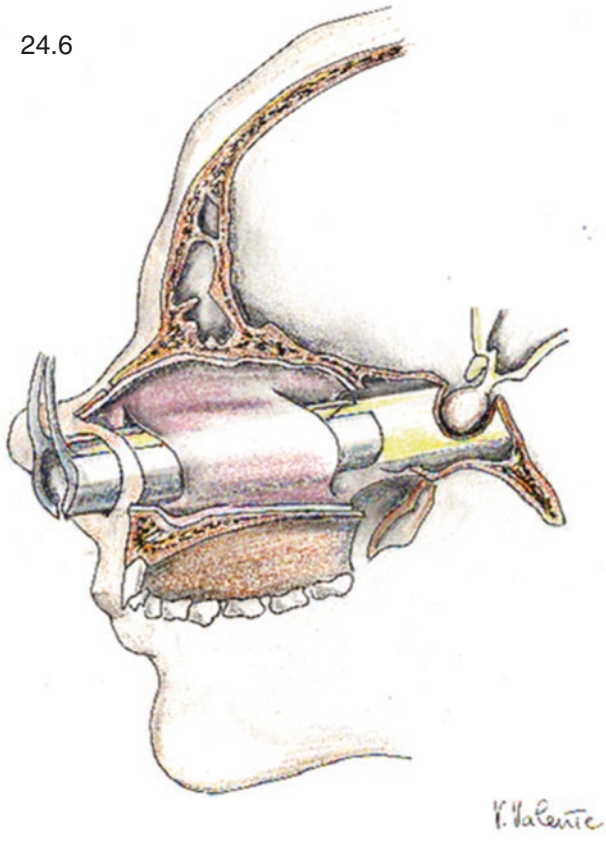

Fig. 24.6 Sagittal view showing a microsurgical approach

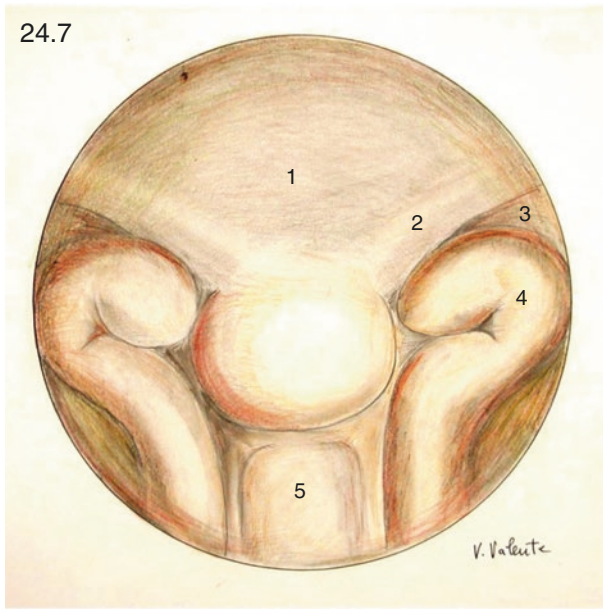

Fig. 24.7 Endoscopic surgical view of the sphenoid sinus

1	Ethmoidal-sphenoidal plane
2	Bony impression of the optic nerves
3	Clinoids as seen from the sinus
4	Bony impression of the siphon
5	Clivus

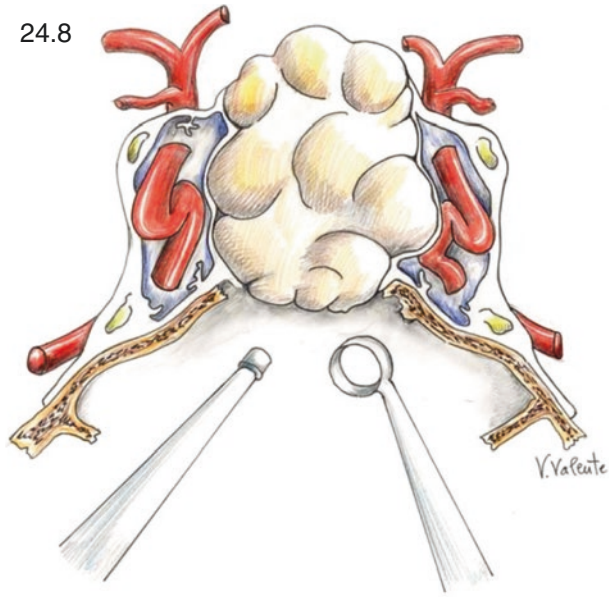

Fig. 24.8 Trans-rhinosphenoidal view of a pituitary adenoma

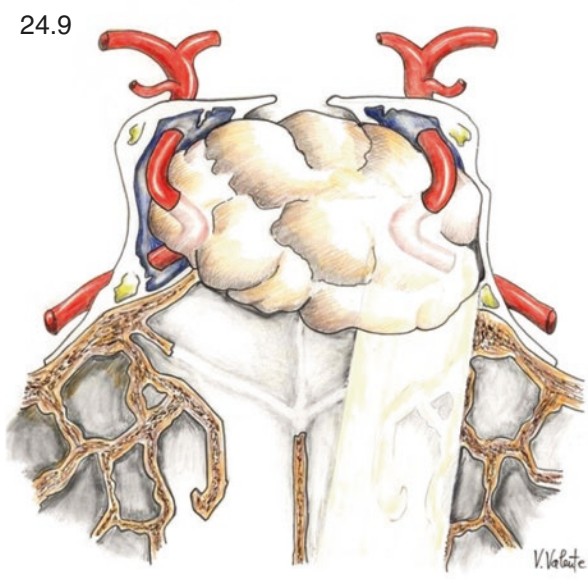

Fig. 24.9 Macroadenoma invading the cavernous sinus, partially involving the carotid siphons

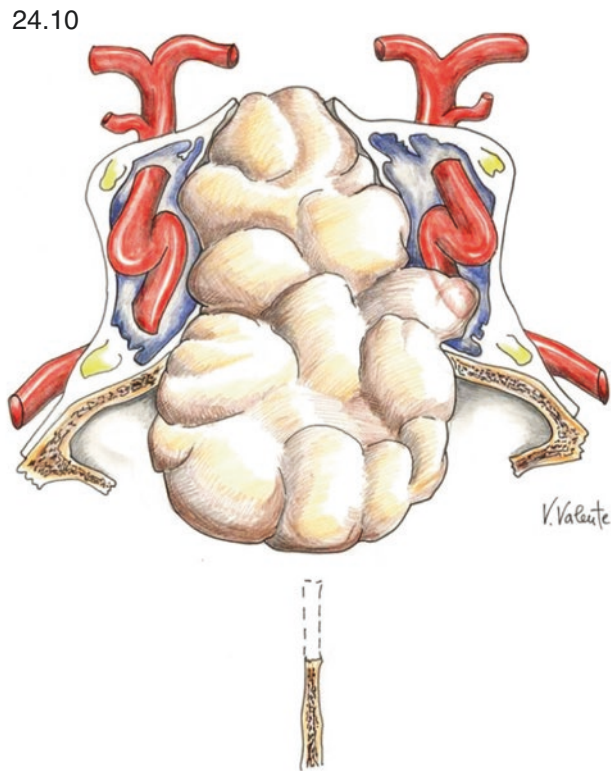

Fig. 24.10 Macroadenoma invading the sphenoid sinus

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Pineal Region

25

Vinicio M.F. Valente

25.1 Normal Anatomy

The pineal region is a complex region of deep anatomical structures that houses the pineal gland. This region is bordered anteriorly by the posterior commissure; posteriorly by the confluences of the internal cerebral veins, inferior sagittal sinus, vein of Galen, and straight sinus; inferiorly by the quadrigeminal plate (tectum); superiorly by the splenium of the corpus callosum; and laterally by the posteromedial aspects of both temporal lobes (Fig. 25.1).

25.2 Major Clinical Symptoms

- Pinealomas become symptomatic only when they compress or involve adjacent structures.
- Focal neurologic symptoms: relative to the compression of the quadrigeminal plate/tectum, visual problems with color recognition, restricted movement of the eyes specially in the vertical direction (Parinaud's syndrome), and deficits in conjugate accommodation and rotatory nystagmus.
- Non-localizing neurologic symptoms: relative to the compression of the tectum and cerebral aqueduct, obstructive hydrocephalus with intracranial hypertension may present as predominant symptom, bilateral or unilateral papilledema.

25.3 Indications for Surgery

- Removal of pineal tumors is technically challenging and relatively risky because of their deep location and the presence of important surrounding vascular structures. The development of microsurgical techniques has improved surgical outcome [1].
- Tumors with small dimensions are monitored over time with MRI.
- In case of development of hydrocephalus, symptomatic endoscopic treatment and third ventricle cisternotomy are proposed with possible tumor biopsy.
- Tumors with large dimensions or fast-growing lesions as seen on follow-up scans are treated surgically using the infratentorial supracerebellar approach.

V.M.F. Valente, M.D.
Section of Neurosurgery, Hospital of Cosenza
(Azienda Ospedaliera di Cosenza), Cosenza, Italy
e-mail: viniciovalente@alice.it

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25.4 Objectives of the Surgery

- To remove mass effect on adjacent structures
- To preserve visual function and/or reverse visual symptoms (or is it to reverse oculomotor function?)
- To provide histologic specimen where necessary to reach final diagnosis

25.5 Surgical Approach(es)

- The most commonly used and relatively safest approach is the infratentorial supracerebellar approach [2] (Figs. 25.2, 25.3 and 25.4).

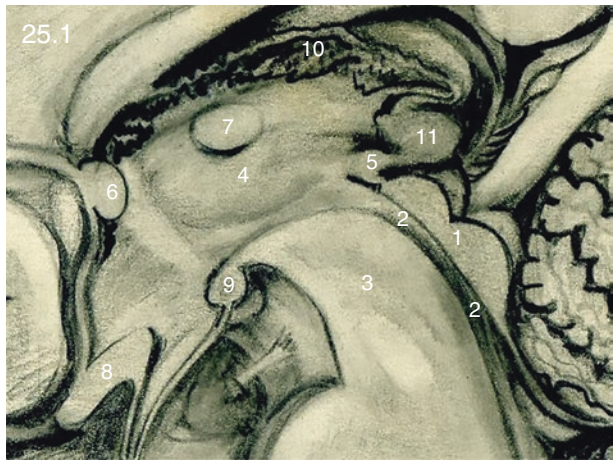

Fig. 25.1 Midline sagittal view of the structures located in and around the pineal gland

Structure labels for Fig. 25.1

1	Quadrigeminal plate
2	Cerebral aqueduct
3	Midbrain
4	Third ventricle
5	Habenula
6	Anterior commissure
7	Thalamic adhesion
8	Optic chiasm
9	Mammillary body
10	Choroid plexus
11	Pineal gland

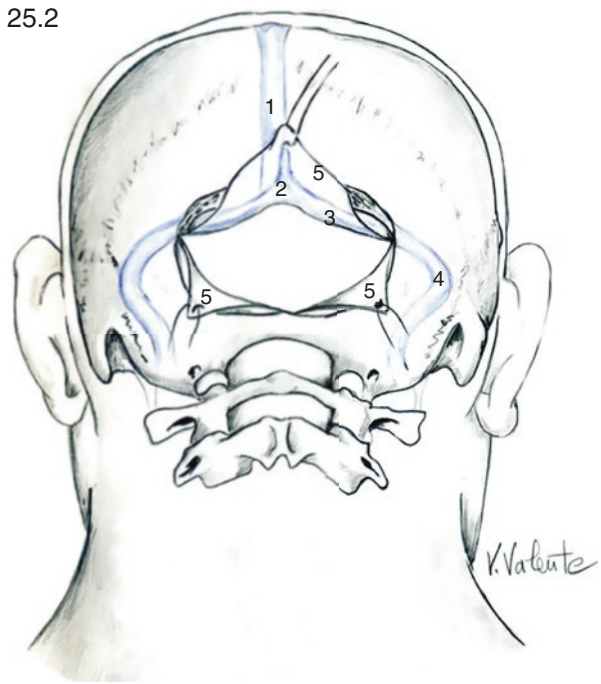

Fig. 25.2 Infratentorial supracerebellar approach, posterior surgical view

Structures for Figs. 25.2 and 25.3

1	Superior sagittal sinus
2	Confluence of sinuses (torcular herophili)
3	Transverse sinus
4	Sigmoid sinus
5	Dura mater
6	Inferior sagittal sinus
7	Straight sinus
8	Vein of Galen
9	Cerebellum
10	Pineal region mass
11	Third ventricle

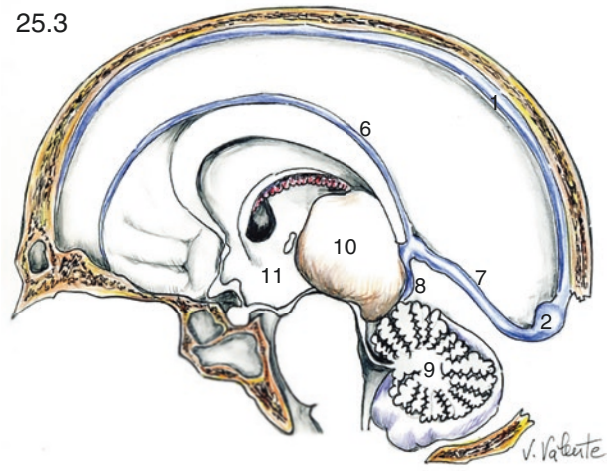

Fig. 25.3 Infratentorial supracerebellar approach, lateral anatomical view

25.4

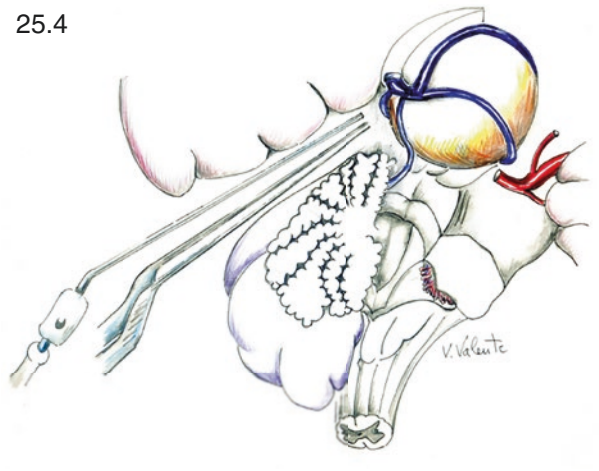

Fig. 25.4 Infratentorial supracerebellar approach, oblique surgical view

25.5

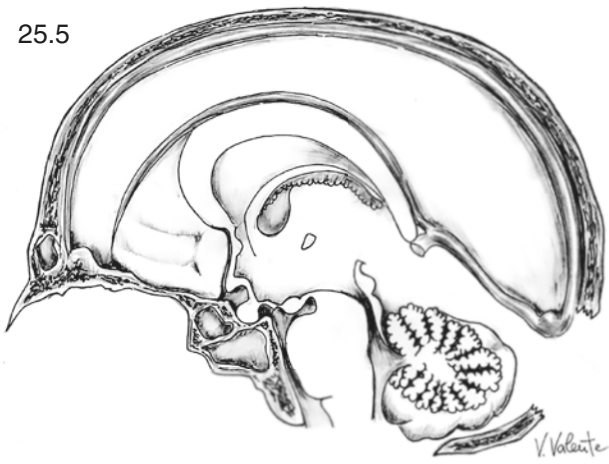

Fig. 25.5 Postoperative surgical corridor shown in the sagittal plane

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Cerebellopontine Angle

26

Vinicio M.F. Valente

26.1 Normal Anatomy

The cerebellopontine angles (CPAs) are regions in the lateral aspects of the posterior fossa. The lateral border of the CPA is the medial aspect of the petrous bone, centered on the internal auditory canal. The medial aspect of the CPA is formed by the middle cerebellar peduncle (brachium pontis). Anteriorly the CPA is bordered by the pons and posteriorly by the cerebellar hemisphere. The CPA contains a number of important structures including cranial nerves (V, VII, and VIII) and vessels (basilar artery, anterior inferior, and superior cerebellar arteries) (Fig. 26.1).

26.2 Major Clinical Symptoms

- Neurovascular compression syndromes: These result from abnormal contact of a cranial nerve by a vessel, usually an artery. Arterial pulsations irritate the perineurium causing inflammation of the nerve resulting in pain and/or focal deficit due to malfunction of the nerve. The two most important neurovascular compression syndromes of surgical interest are:
  - Trigeminal neuralgia (superior cerebellar artery and the V).
  - Hemifacial spasm (antero-inferior cerebellar artery and the VII).
- Acoustic schwannomas: The most frequent symptoms of an acoustic nerve schwannoma are tinnitus and hypoacusis. Even vestibular nerve schwannoma can present with tinnitus and hypoacusis. These symptoms are followed by vertigo, nystagmus, and inability to maintain a stable erect position (i.e., positive Romberg sign). With increasing tumor size, adjacent structures can be compressed. The first structure is the facial nerve and may give rise to homolateral peripheral facial paralysis. Next, compression of the trigeminal nerve can cause hypoanesthesia in the trigeminal nerve territory. If the tumor is very large, cerebellar symptoms may become obvious, with patients exhibiting dysmetria and homolateral adiadochokinesia. Voluminous lesions may also compress the brainstem, obstructing the fourth ventricle, causing hydrocephalus.

V.M.F. Valente, M.D.
Section of Neurosurgery, Hospital of Cosenza
(Azienda Ospedaliera di Cosenza), Cosenza, Italy
e-mail: viniciovalente@alice.it

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26.3 Indications for Surgery

- Neurovascular compression syndromes: trigeminal neuralgia
  - Medical therapy first: carbamazepine (antiepileptic drugs stabilize nerve membrane potentials).
  - Trigeminal ganglion ablation (thermocoagulation, alcohol ablation, balloon ablation by compressing the ganglion).
  - Surgery to separate the offending artery from the trigeminal nerve.
- Neurovascular compression syndromes: hemifacial spasm
  - Medical therapy first: botulinum toxin injection.
  - Surgery to separate the offending artery from the facial nerve.
- Acoustic schwannomas:
  - Wait-and-watch approach for lesions less than 1.5 cm.
  - Gamma knife surgery consists in treating patients with gamma radiation. With newer technology, radiation dose is significantly reduced, and in the hands of experts, excellent outcomes have been reported with few postradiation complications. It is considered a first-line treatment of choice in patients with tumors less than 3 cm and without signs of brainstem compression or risk of hydrocephalus. There is very low risk of facial nerve deficit (<2–3%) and high chances of conserving auditory sensation.
  - Surgery to remove the acoustic schwannoma.

26.4 Objectives of the Surgery

- Neurovascular compression syndromes: separate the affected cranial nerve from the compressing artery.
- Acoustic schwannoma:
  - To remove the mass from the affected nerve, preserving auditory and vestibular function.
  - To remove mass effect on adjacent structures (facial nerve, brainstem).
  - To provide histologic specimen where necessary to reach final diagnosis.

26.5 Surgical Approach(es)

- Neurovascular compression syndromes: Usually a retrosigmoidal approach is used to visualize the vascular compression. The vessel is lifted off of the cranial nerve, and a Teflon pledget is placed in between the vessel and cranial nerve to separate them [1] (Figs. 26.2–26.5).
- Acoustic schwannoma: There are different approaches (retrosigmoidal, translabyrinthic, extradural subtemporal) (Figs. 26.6–26.9). The choice depends on the dimensions of the tumor, on the exact location of the schwannoma (intra- or extracanalicular), and on the presence of neurological deficits before surgery [2, 3].

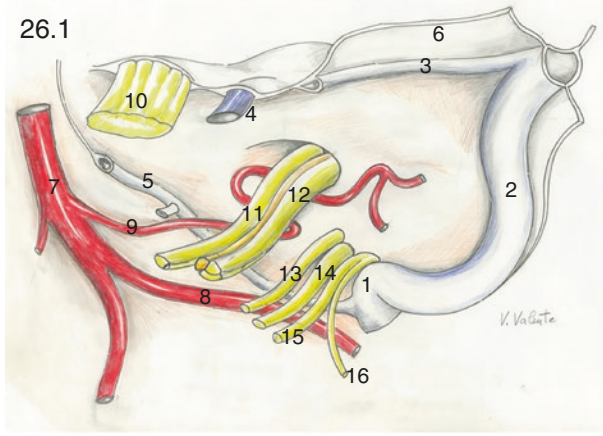

Fig. 26.1 Anatomical view showing normal structures

1	Jugular bulb
2	Sigmoid sinus
3	Superior petrosal sinus
4	Petrosal veins
5	Inferior petrosal sinus
6	Tentorium
7	Basilar artery
8	Right vertebral artery
9	Anterior inferior cerebellar artery
10	Trigeminal nerve (V)
11	Facial nerve (VII)
12	Vestibulocochlear nerve (VIII)
13	Glossopharyngeal nerve (IX)
14	Vagus nerve (X)
15	Accessory nerve of the vagus (XI)
16	Spinal root of the accessory nerve

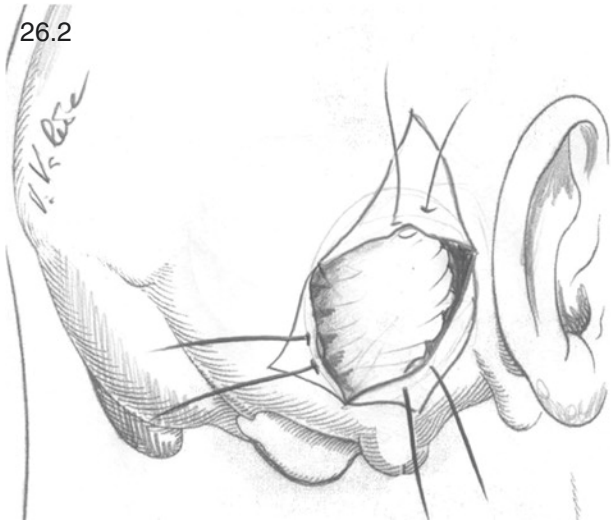

Fig. 26.2 Retrosigmoid surgical approach to neurovascular compression syndrome

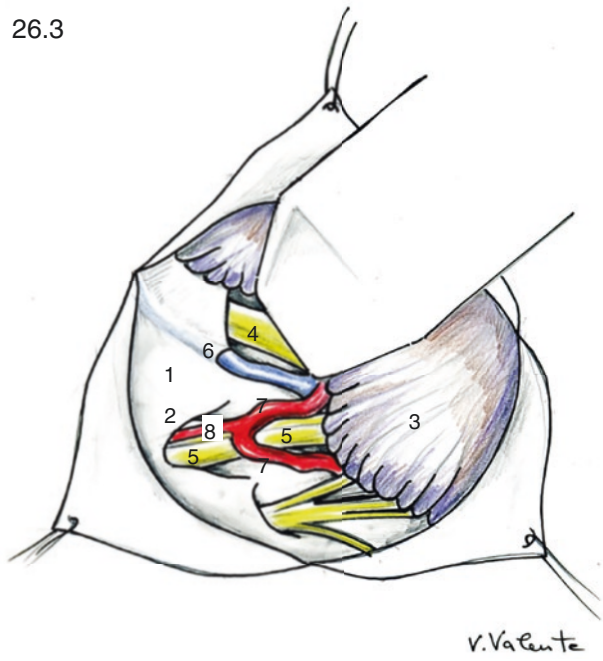

Fig. 26.3 Surgical view of normal CPA anatomy

Structure labels for Figs. 26.3, 26.4, and 26.5

1	Posterior aspect of the dura
2	Internal auditory canal
3	Cerebellar hemisphere
4	Trigeminal nerve (V)
5	Facial and vestibulocochlear nerves (VII & VIII)
6	Petrous vein
7	Anterior inferior cerebellar artery loop
8	Internal auditory artery
9	Superior cerebellar artery
10	Bipolar tongs
11	Dissector
12	Pledget inserted between the artery and nerve

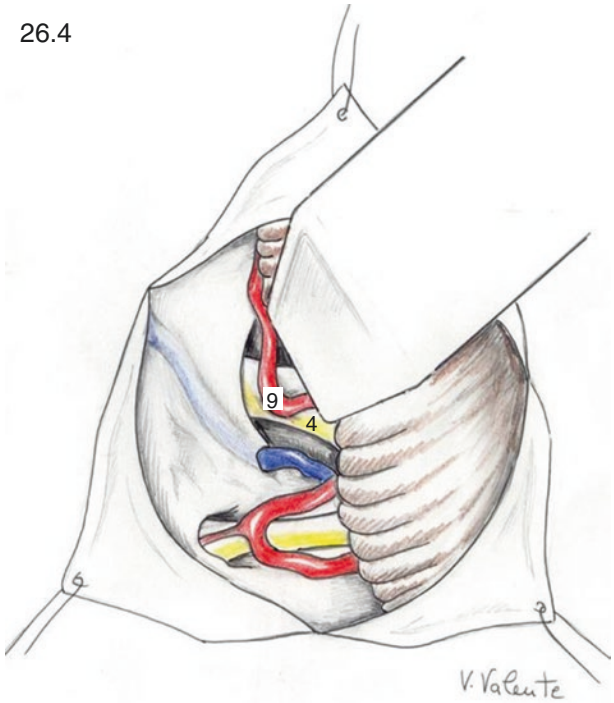

Fig. 26.4 Surgical view showing compression of the trigeminal nerve root entry zone by the superior cerebellar artery

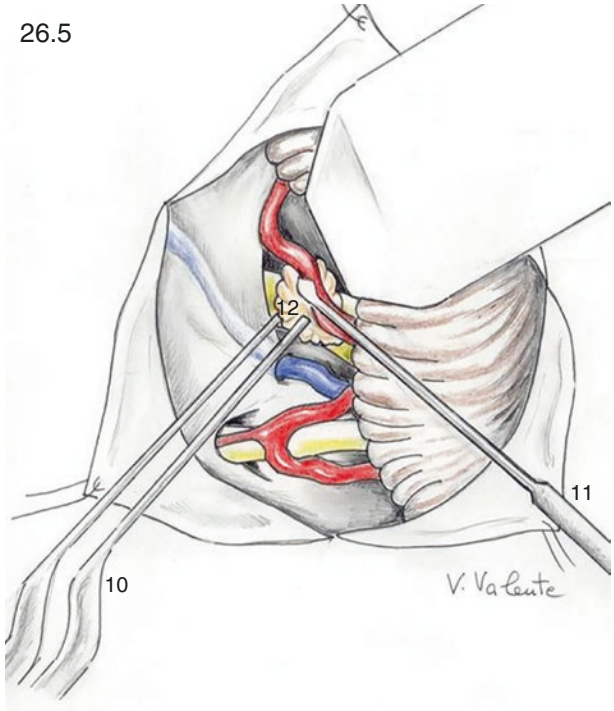

Fig. 26.5 Surgical view showing placement of a Teflon pledget between the superior cerebellar artery and the trigeminal nerve

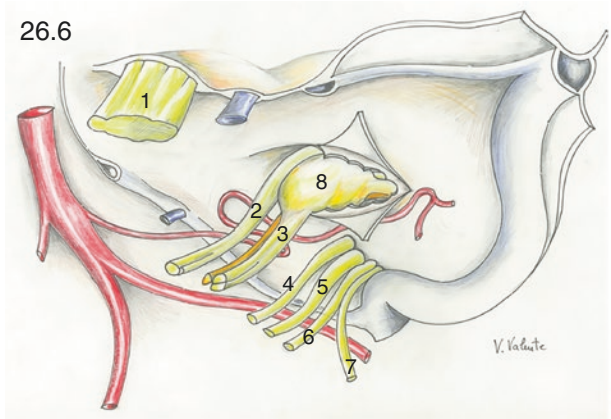

Fig. 26.6 Anatomical view showing Type 1 (intracanalicular) vestibulocochlear nerve schwannoma

Structure labels for Figs. 26.6, 26.7, 26.8, and 26.9

1	Trigemnal nerve (V)
2	Facial nerve (VII)
3	Vestibulocochlear nerve (VIII)
4	Glossopharyngeal nerve (IX)
5	Vagus nerve (X)
6	Accessory nerve of the vagus (XI)
7	Spinal root of the accessory nerve
8	Schwannoma

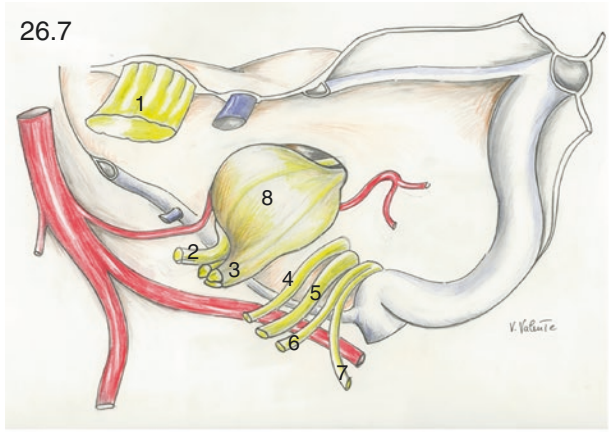

Fig. 26.7 Anatomical view showing Type 2B vestibulocochlear nerve schwannoma, facial nerve medial to schwannoma

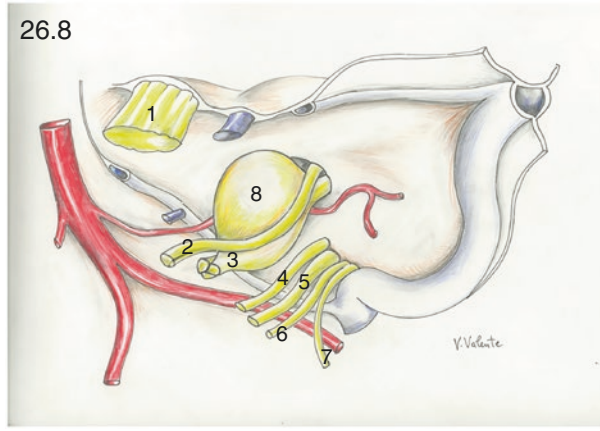

Fig. 26.8 Anatomical view showing Type 2B vestibulocochlear nerve schwannoma, facial nerve lateral to schwannoma

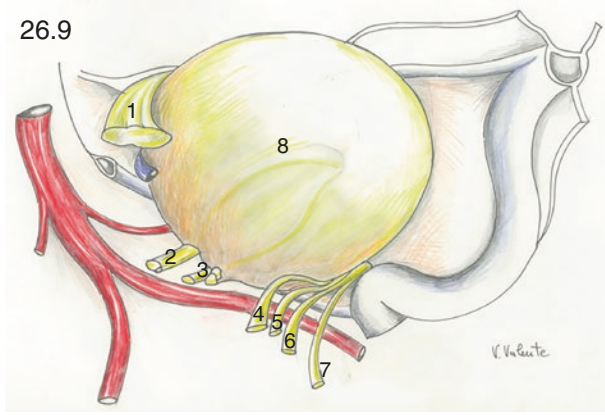

Fig. 26.9 Anatomical view showing Type 3 vestibulocochlear nerve schwannoma

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Hydrocephalus

27

Vinicio M.F. Valente

27.1 Normal Anatomy

The normal adult brain weighs on average 1500 g and is immersed in cerebrospinal fluid (CSF). There is approximately 125–150 ml of CSF within and surrounding the brain and spinal cord. CSF, in addition to its metabolic functions, plays an important role mechanically supporting the brain and buffering it from injury. CSF is produced in the ventricles by the choroid plexus at a rate averaging approximately 20 ml/h. CSF then flows from the ventricles through the foramina of Luschka and Magendie into the subarachnoid spaces surrounding the brain and spinal cord, initially flowing caudally but then rostrally where it is resorbed into the dural sinuses through the arachnoid (Pacchionian) granulations (Fig. 27.1).

Any pathology that (1) causes increased CSF production (e.g., choroid plexus papillomas), (2) interferes with CSF flow leaving the ventricles (e.g., tumor, aqueductal stenosis), or (3) interferes with CSF resorption through the arachnoid granulations (e.g., subarachnoid hemorrhage) can cause hydrocephalus. The most frequent causes are neoplastic, hemorrhagic (subarachnoid hemorrhage), and infectious (e.g., TB) [1, 2].

Another clinical entity, normal pressure hydrocephalus, remains of unknown etiology and can be treated surgically in patients with cognitive symptoms, urinary incontinence and ataxia [3].

27.2 Major Clinical Symptoms

The symptoms of hydrocephalus result primarily from the intracranial hypertension and primarily include:

- Headache: diffuse, continuous but more prominent in the morning due to less efficient CSF resorption in the recumbent position.
- Neck pain and dizziness: due to tonsillar herniation through foramen magnum.
- Nausea: due to stimulation of the vagal centers and independent of head movements.
- Vomiting: projectile, more prominent in the morning, independent of meals.
- Papilledema: seen on fundoscopy.

Secondary symptoms can include:

- Diplopia and/or convergent strabismus. Due to pressure on CN VI.
- Dizziness, imbalance, and gait problems.
- Cognitive deterioration that can progress quickly to coma if the condition is not diagnosed and treated.

27.3 Indications for Surgery

- If the symptoms above are rapidly progressing in a patient with a known cause for hydrocephalus, immediate shunting surgery may be

V.M.F. Valente, M.D.
Section of Neurosurgery, Hospital of Cosenza (Azienda Ospedaliera di Cosenza), Cosenza, Italy
e-mail: viniciovalente@alice.it

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indicated to relieve the intracranial pressure and save the patient's life.

27.4 Objectives of the Surgery

- To relieve the elevated intracranial pressure, returning it to a more normal pressure, either temporarily or on a more permanent basis as is determined by the cause of the hydrocephalus.

27.5 Surgical Approach(es)

- Extraventricular drain (shunt) placement is used to temporarily relieve the mounting CSF pressure causing neurological symptoms, and is reserved for emergency treatment (Fig. 27.2).
- If the underlying cause of the hydrocephalus cannot be cured (e.g., obstructive hydrocephalus due to a brain tumor), then the shunt can be internalized and made permanent. CSF is drained from the ventricles into the right atrium (ventriculoatrial shunt) and peritoneal cavity (ventriculoperitoneal shunt) or from the spine into the peritoneal cavity (spino-peritoneal shunt) (Figs. 27.3 and 27.4).
- Endoscopic third ventriculostomy (ETV) is a permanent option for patients with obstructive hydrocephalus distal to the third ventricle [2] (Fig. 27.5).

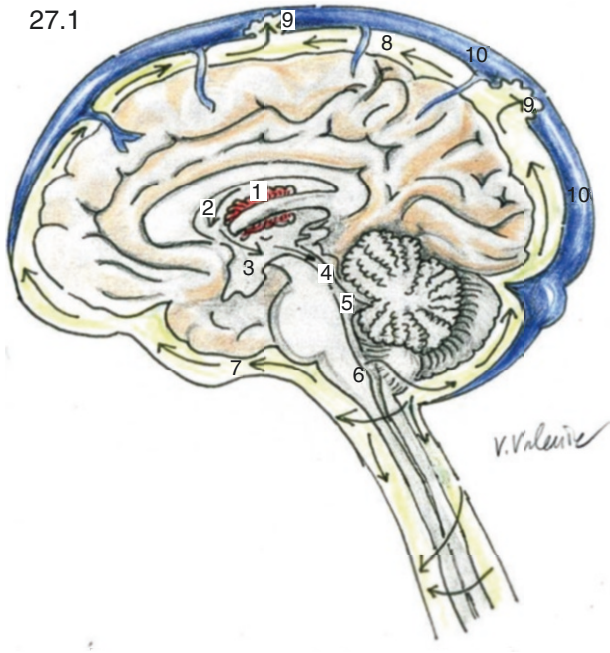

Fig. 27.1 Anatomical view showing normal CSF flow pattern

1	Choroidal plexus
2	Lateral ventricles
3	Third ventricle
4	Cerebral aqueduct of Silvius
5	Fourth ventricle
6	Foramina of Magendie and Luschka
7	Basal cisterns
8	Cortical CSF space
9	Arachnoidal granulations
10	Superficial venous sinus

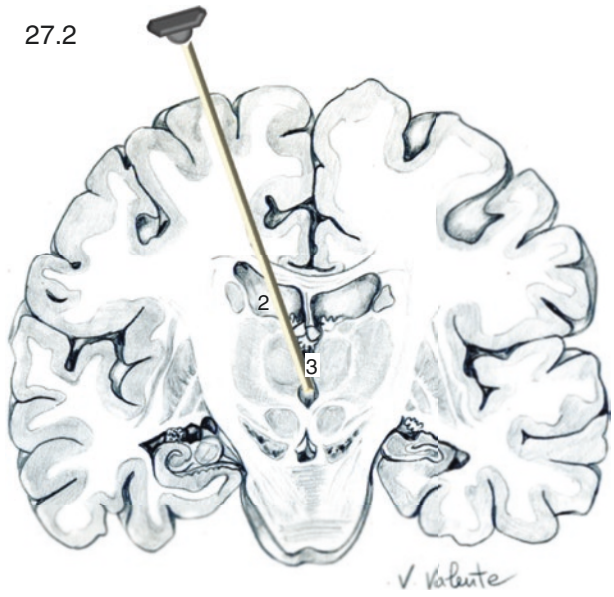

Fig. 27.2 Ideal placement of a trocar for a frontal shunt tube

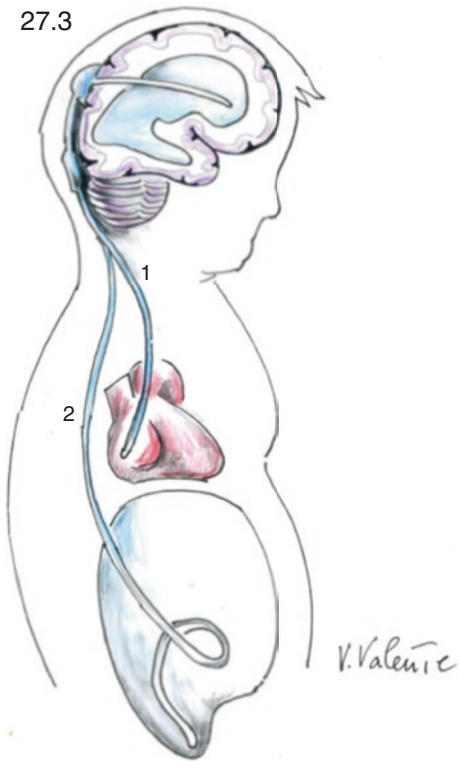

Fig. 27.3 (1) Ventriculoatrial shunt; (2) ventriculoperitoneal shunt

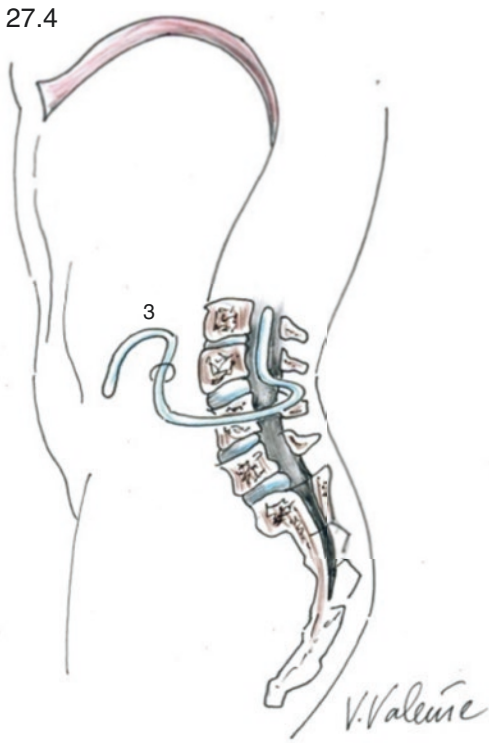

Fig. 27.4 (3) Spino-peritoneal shunt

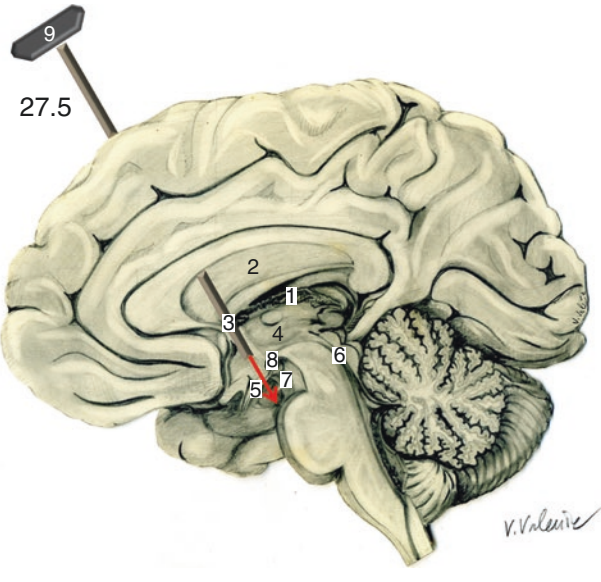

Fig. 27.5 Endoscopic third ventriculostomy

1	Choroid plexus
2	Lateral ventricle
3	Foramen of Monroe
4	Third ventricle
5	Third ventricle floor
6	Aqueduct of Silvius
7	Interpeduncular cistern
8	Mammillary body
9	Endoscope

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Orbit

28

Vinicio M.F. Valente

28.1 Normal Anatomy

The orbital cavity is formed by the maxillary bone inferomedially, zygomatic bone inferiorly and laterally, frontal bone superiorly, and a combination of the lacrimal bone, ethmoid bone, and sphenoid bones medially. The sphenoid bone also forms the walls of the orbital apex. Orbital contents include the globe; optic nerve (anatomically a continuation of the CNS); extraocular muscles and their associated cranial nerves III, IV, and VI; lacrimal gland; and various supporting small arteries and veins. The orbit is divided into a preseptal compartment anterior to and including the conjunctiva and a retrobulbar compartment posterior to the conjunctiva [1] (Fig. 28.1).

The neurosurgical approach to orbital masses is limited to the retrobulbar cavity. Surgery in this region is challenging due to the presence of adipose tissue in the orbital cavity that limits the dissection and surgical exposure of periorbital structures.

28.2 Major Clinical Symptoms

- Exophthalmos (axial or disaxial), unilateral with or without other visual symptoms. While occasionally the globe is pulsating with the patient's heartbeat (pulsating exophthalmos), most orbital masses cause a nonreducible and non-pulsating exophthalmos.
- Symptoms accompanying the exophthalmos are usually a combination of the following:
  - Visual field deficits
  - Global vision deficit
  - Defects in accommodation
  - Fundus alterations
  - Venostasis
  - Pain
  - Palpable mass

28.3 Indications for Surgery

- Surgical indications in the orbits highly depend on the compressive effect to structures and the type of temporal evolution of growth of neoplasias [2, 3].

28.4 Objectives of the Surgery

- To decrease or remove mass effect on the orbital contents, thereby preserving visual function.
- To provide a histologic specimen where necessary to reach a final diagnosis.

V.M.F. Valente, M.D.
Section of Neurosurgery, Hospital of Cosenza
(Azienda Ospedaliera di Cosenza), Cosenza, Italy
e-mail: viniciovalente@alice.it

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28.5 Surgical Approach(es)

- Smaller more anterior masses are approached by the ophthalmologist via a trans-conjunctival approach.
- The best access for larger or more posterior retrobulbar masses is the extradural transfrontal approach (Figs. 28.2, 28.3, 28.4 and 28.5).

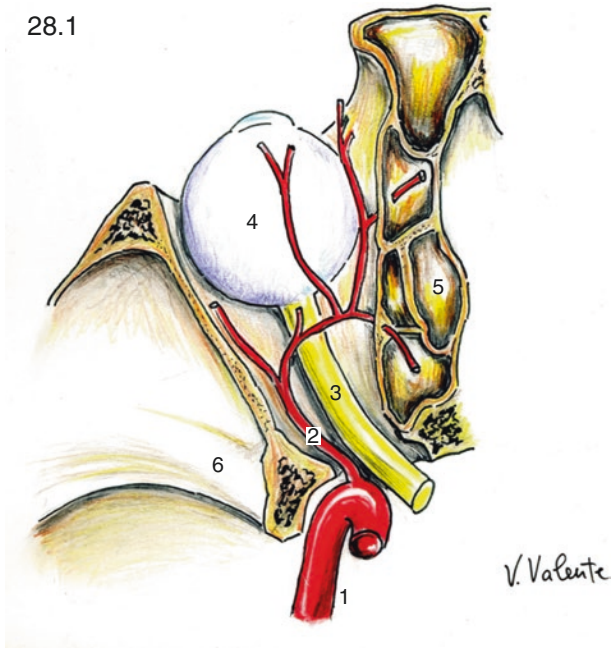

Fig. 28.1 Anatomical view of the orbit

Structure lables for Figs. 28.1, 28.2, 28.3, 28.4 and 28.5

1	Carotid siphon
2	Ophthalmic artery
3	Optic nerve
4	Eye globe
5	Ethmoid air cells
6	Lesser wing of the sphenoid bone
7	Frontal dura mater
8	Frontal sinus
9	Spatula
10	Orbital roof
11	Superior orbitotomy, open Tenon's capsule
12	Tenon's capsule
13	Superior rectus and oblique muscles
14	Orbital mass

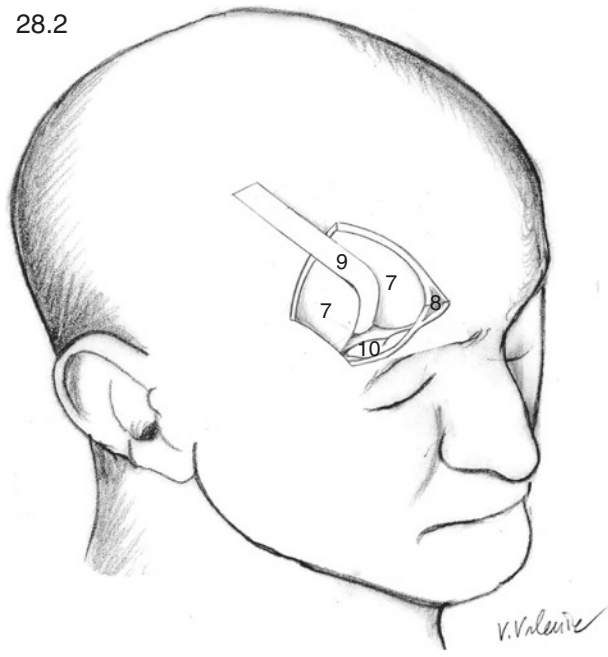

Fig. 28.2 Surgical view of transconjunctival approach to the superior orbit

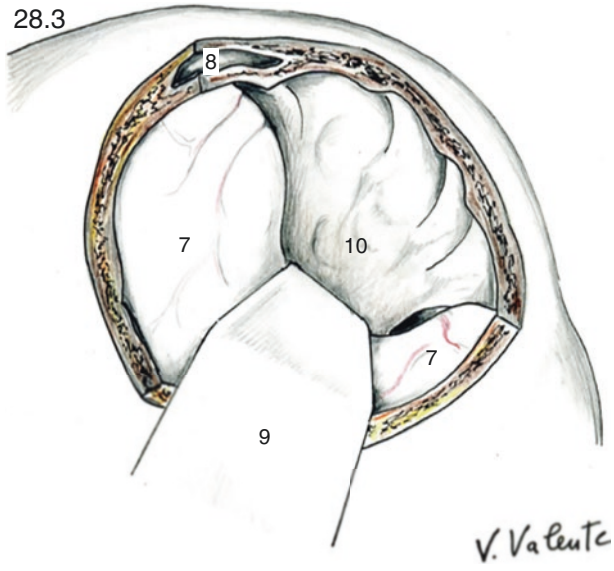

Fig. 28.3 Surgical view, exposure of the orbital roof

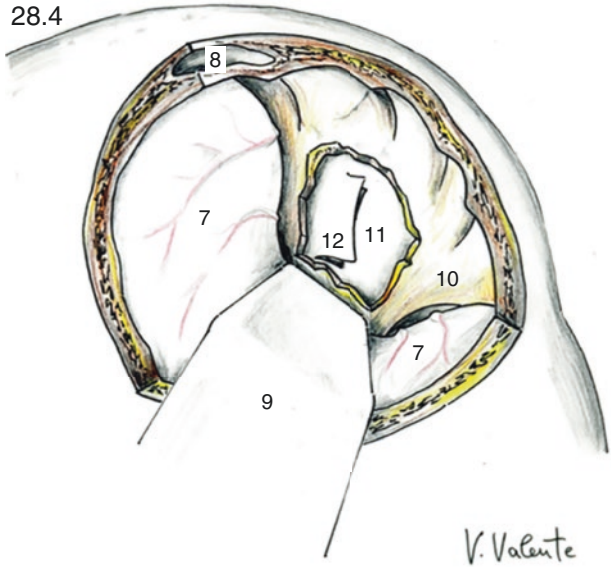

Fig. 28.4 Surgical view, removal of the orbital roof and exposure with incision of the Tenon's capsule

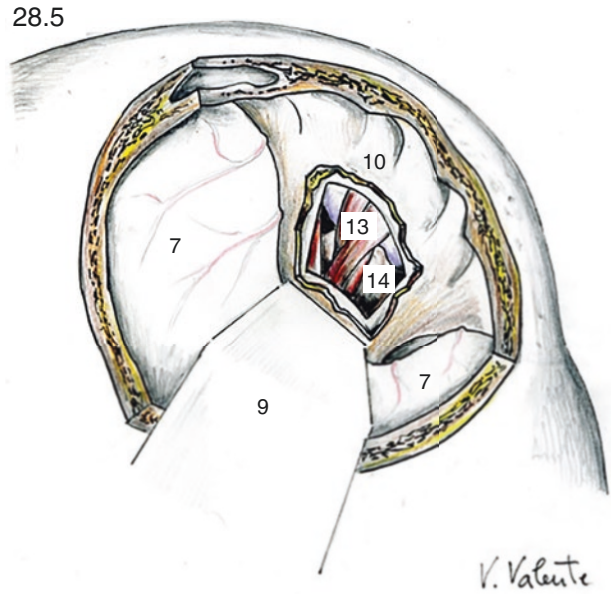

Fig. 28.5 Surgical view, exposure of the retrobulbar orbital tissue (note the superior oblique and rectus muscles) and an intraconal retrobulbar mass

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Vascular Surgery

29

Vinicio M.F. Valente

29.1 Normal Anatomy

The anatomy of the cerebral vasculature has been demonstrated in detail in Chaps. 3 (cerebral vascular anatomy) and 9 (vascular anatomy of the cerebellar and brainstem) (Figs. 29.1, 29.2, 29.3 and 29.4). Illustrations of vascular anatomy as seen from the viewpoint of the vascular surgeon are shown below (Figs. 29.6–29.15).

29.2 Major Clinical Symptoms

- Ruptured aneurysms: Patients present with acute sharp headache (worst headache of life) that may be accompanied by projective vomiting and loss of consciousness. The severity of the symptoms is best rated using the Hunt and Hess scale (Table 29.1). This scale also guides surgeon on the timing of surgery and the approach.
- Unruptured aneurysms: These are usually incidental findings in asymptomatic patients being scanned for other pathologies. If symptomatic, focal neurological deficits may be seen due mostly their mass effect. For example, aneurysms in the carotid siphon may cause a peripheral third cranial nerve deficit with anisocoria.

Table 29.1 Hunt and Hess scale

Grade	Clinical symptoms
I	Asymptomatic or mild headache with minimal nuchal rigidity
II	Moderate or severe degree of headache and nuchal rigidity and no neurologic deficit other than cranial nerve palsy
III	Sleepiness, confused, and mild focal neurologic deficit
IV	Obtunded, stupor, moderate hemiparesis or hemiplegia, and autonomic dysfunction
V	Deep coma, decerebration rigidity, and severe autonomic dysfunction

Hunt WE, Hess RM (1968) Surgical risk as related to time of intervention in the repair of intracranial aneurysms. J Neurosurg 28(1):14–20

29.3 Indications for Surgery

- Ruptured aneurysm(s): Treatment is indicated within at least 72 h after hemorrhage. Several factors help decide whether the treatment is

V.M.F. Valente, M.D.
Section of Neurosurgery, Hospital of Cosenza
(Azienda Ospedaliera di Cosenza), Cosenza, Italy
e-mail: viniciovalente@alice.it

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endovascular or surgical. Factors include the clinical condition of the patient, aneurysmal location, and type and size of the aneurysms.
- Unruptured aneurysm(s): Treatment can be deferred to when there are more favorable clinical conditions. Aneurysms above 7 mm are given highest priority [1, 2].

29.4 Objectives of the Surgery

- To isolate an aneurysm from the arterial circulation in order to minimize/eliminate the risk of future rupture with associated subarachnoid hemorrhage.
- To prevent further hemorrhage in ruptured aneurysms [2, 3].

29.5 Surgical Approach(es)

The decision whether endovascular treatment or whether it is surgical approach is largely based upon topographic criteria (where the aneurysm is located):

- Carotid siphon aneurysms: these are usually treated with an endovascular approach.
- Anterior cerebral artery: for A1, A1-A2 angle and proximal A2 a pterional approach is used whereas for distal A2, pericallosal and calloso-marginal arteries a frontal parasagittal is used (Figs. 29.18, 29.19 and 29.20).
- Middle cerebral artery, anterior communicating artery, and more distal anterior cerebral artery aneurysms: surgical treatment, especially when they are more distally located (Fig. 29.5).
- Posterior circulation aneurysms: endovascular approach, especially at the level of the PICA and basilar tip (Figs. 29.16 and 29.17).

It is important to note that such decisions are usually made on a case by case basis, and good collaboration between the neurosurgeon and the interventional neuroradiologist is mandatory in identifying the best treatment approach.

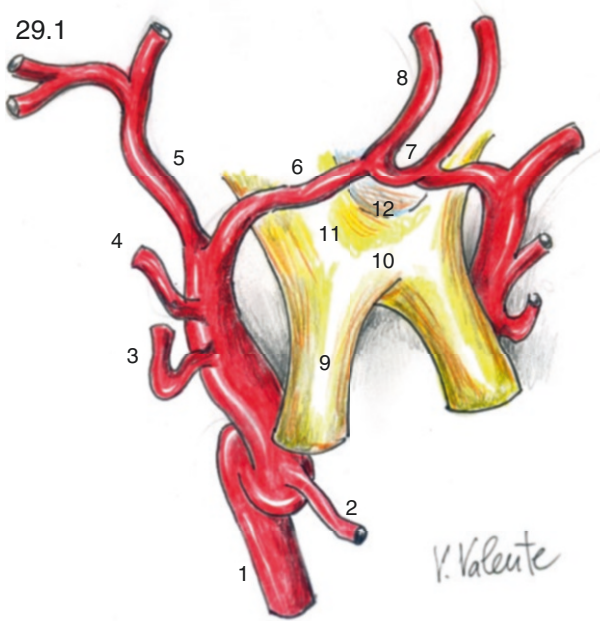

Fig. 29.1 Proximal anterior cerebral artery distribution

1	Internal carotid artery
2	Ophthalmic artery
3	Posterior comunicating artery
4	Anterior choroidal artery
5	Middle cerebral artery (M1)
6	Anterior cerebral artery (A1)
7	Anterior comunicating artery
8	Anterior cerebral artery (A2)
9	Optic nerve
10	Optic chiasm
11	Optic tract
12	Lamina terminalis
13	Pericallosal artery (A3)
14	Fronto-orbital artery
15	Fronto-polar artery
16	Callosomarginal artery
17	Corpus callosum

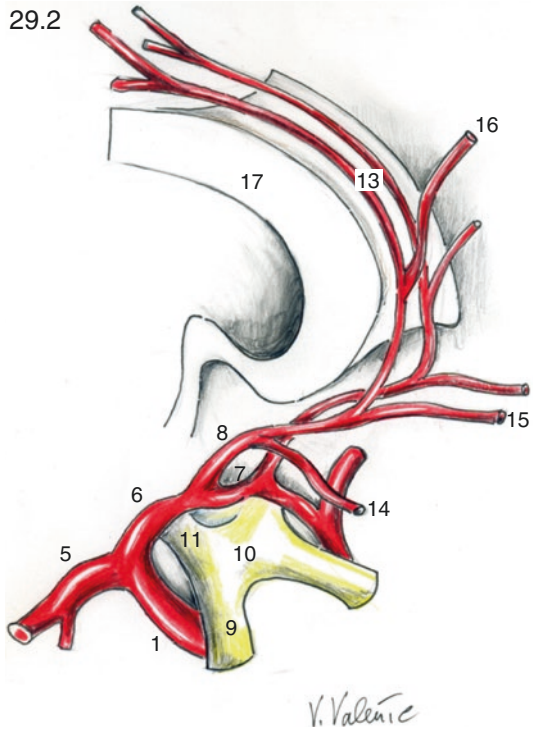

Fig. 29.2 Distal anterior cerebral artery distribution

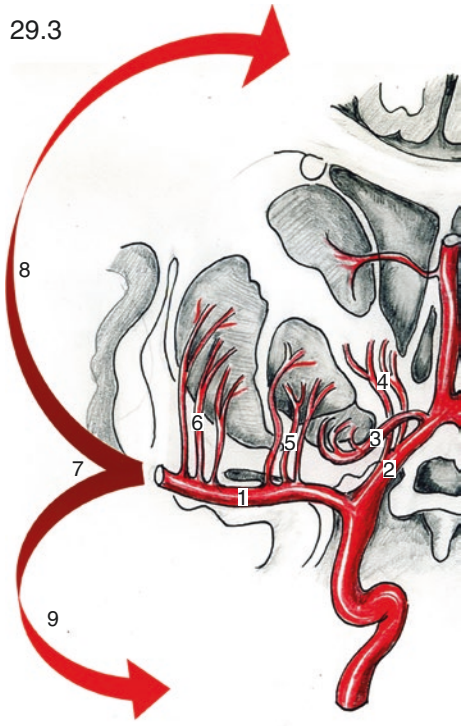

Fig. 29.3 Middle cerebral artery distribution

1	Middle cerebral artery (M1)
2	Anterior cerebral artery (A1)
3	Recurrent artery of Heubner
4	Internal striate arteries
5	Medial lenticulostriate arteries
6	Lateral lenticulostriate arteries
7	Middle cerebral artery bifurcation
8	MCA, frontal branch (M2)
9	MCA, temporal branch (M2)
10	Basilar artery
11	Posterior cerebral artery (P1)
12	Superior cerebellar artery
13	Anterior inferior cerebellar artery
14	Posterior inferior cerebellar artery
15	Vertebral artery
16	Brainstem perforating arteries

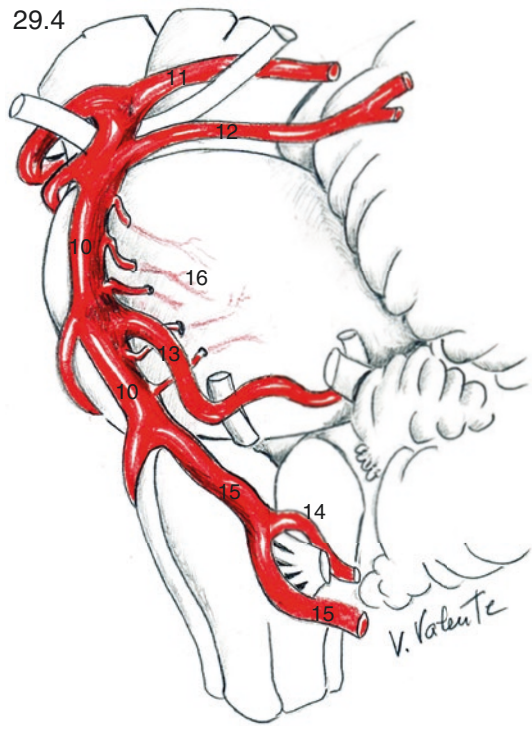

Fig. 29.4 Posterior cerebral artery distribution

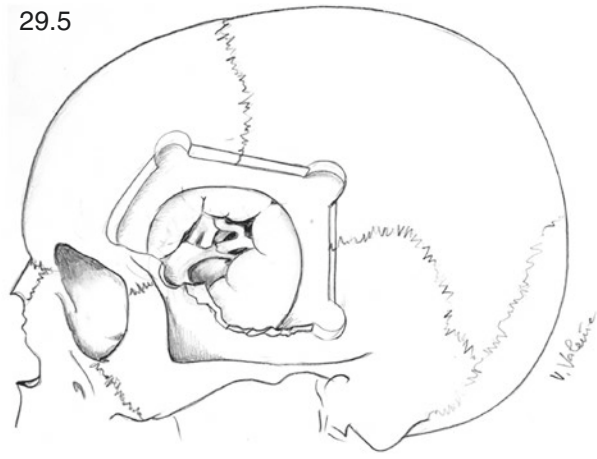

Fig. 29.5 Pterional craniotomy approach to the cerebral circulation

Structure labels for Figs. 29.6–29.15

1	Internal carotid artery
2	Ophthalmic artery
3	Posterior communicating artery.
4	Anterior choroidal artery
5	Middle cerebral artery (M1)
6	Anterior cerebral artery (A1)
7	Anterior comunicating artery
8	Anterior cerebral artery (A2)
9	Optic nerve
10	Optic chiasm
11	Optic tract
12	Lamina terminalis
13	Aneurysm
14	Oculomotor nerve (III)
15	Tentorium
16	Anterior clinoid process
17	Frontal lobe
18	Temporal lobe
19	Sylvian fissure
20	Recurrent artery of Heubner
21	MCA, frontal branch (M2)
22	MCA, temporal branch (M2)
23	Lateral lenticulostriate arteries

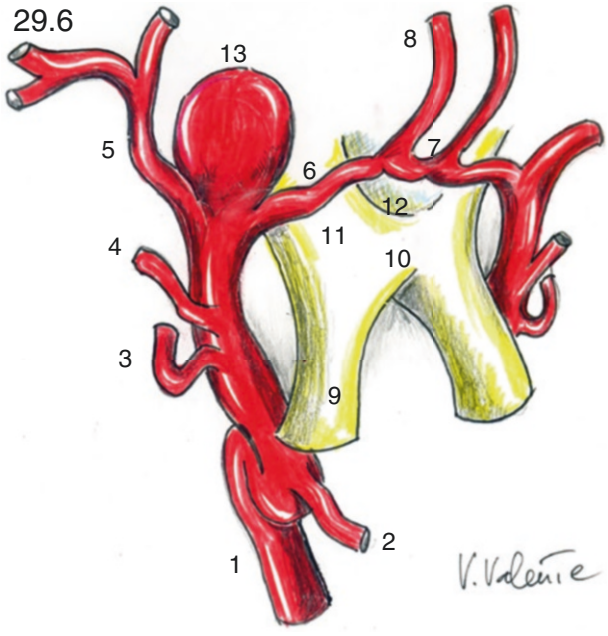

Fig. 29.6 Right ICA terminus aneurysm

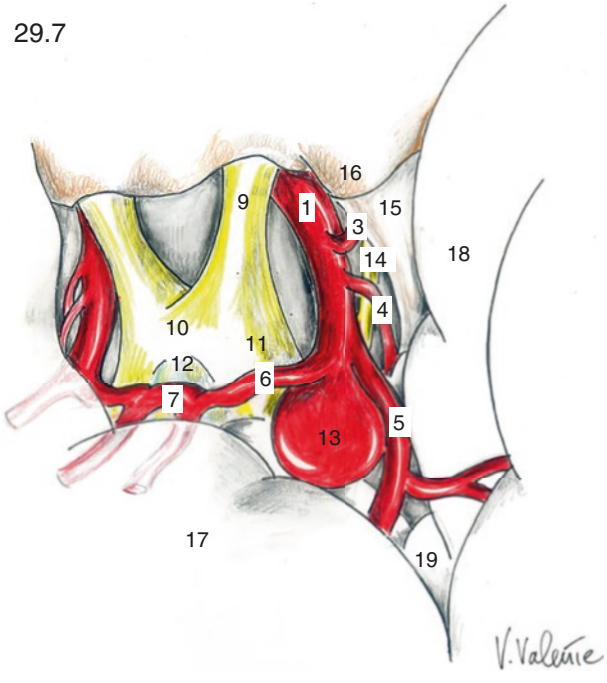

Fig. 29.7 Right ICA terminus aneurysm, surgical view

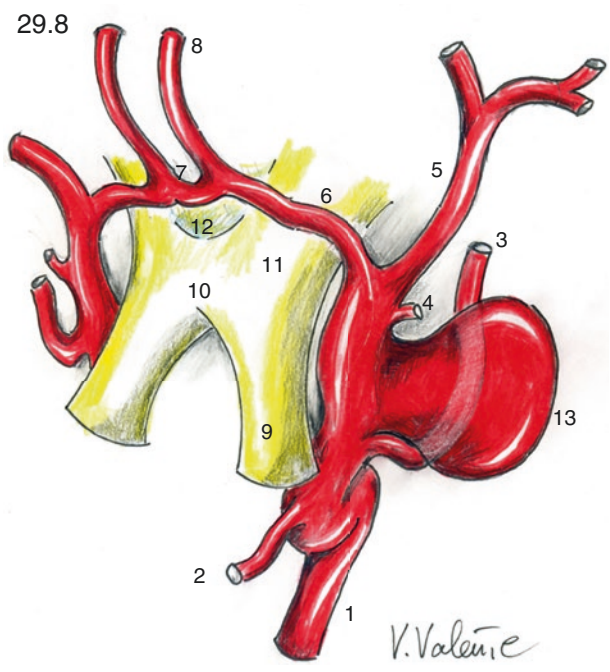

Fig. 29.8 Left PCOM aneurysm

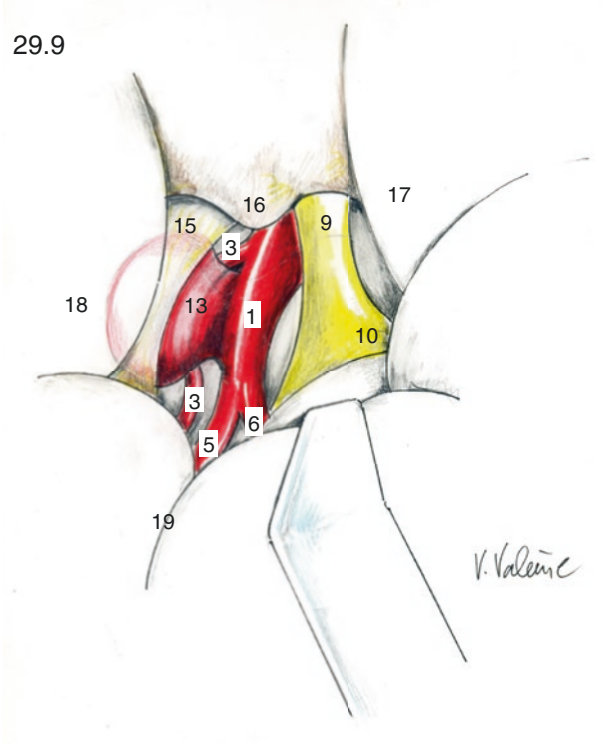

Fig. 29.9 Left PCOM aneurysm, surgical view

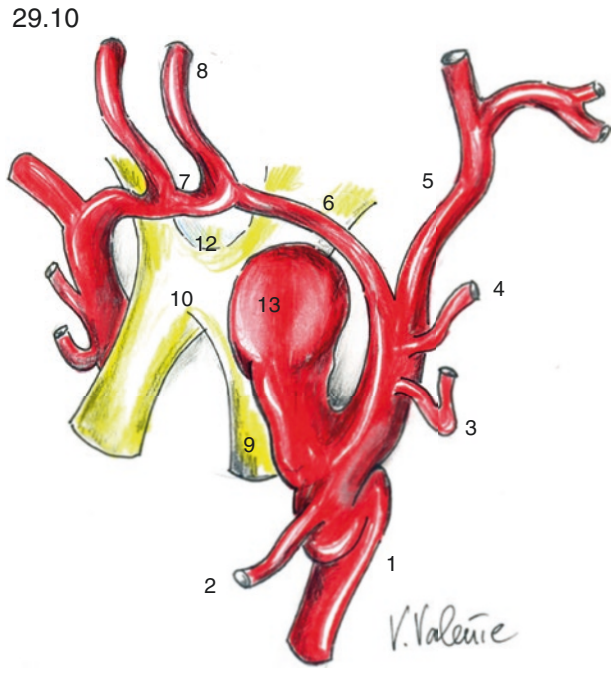

Fig. 29.10 Left ophthalmic artery aneurysm

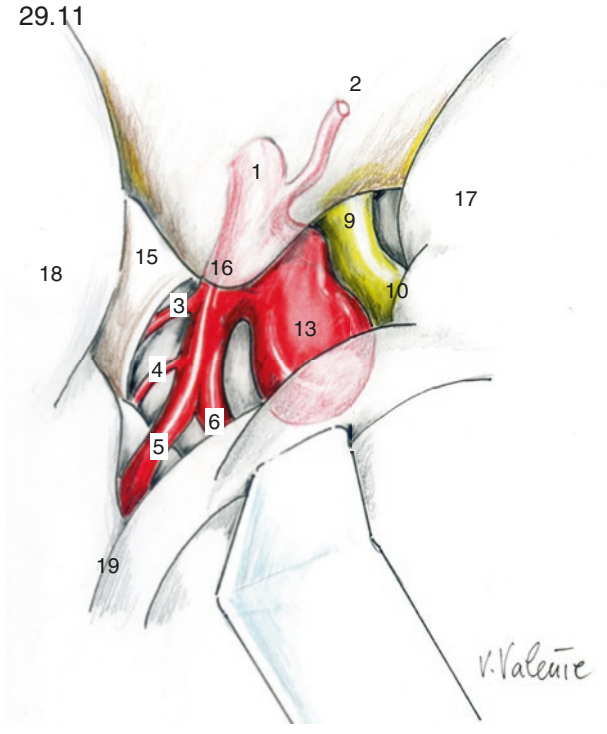

Fig. 29.11 Left ophthalmic artery aneurysm, surgical view

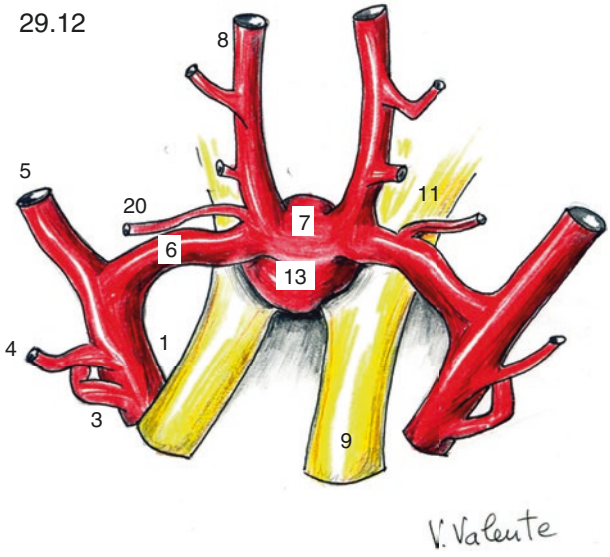

Fig. 29.12 ACOM aneurysm adherent to optic chiasm

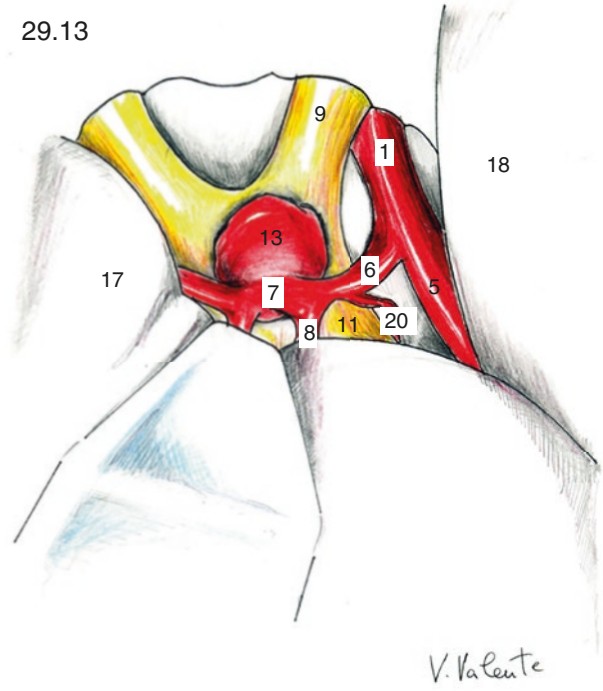

Fig. 29.13 ACOM aneurysm adherent to optic chiasm, surgical view

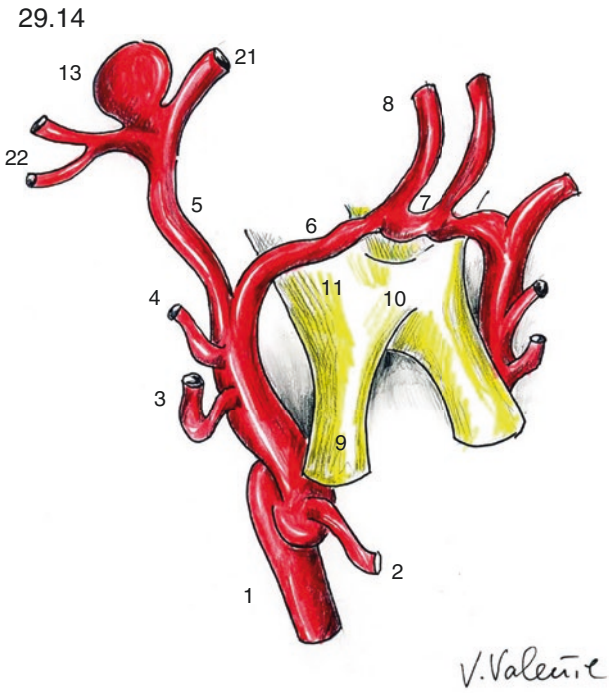

Fig. 29.14 Right MCA bifurcation aneurysm

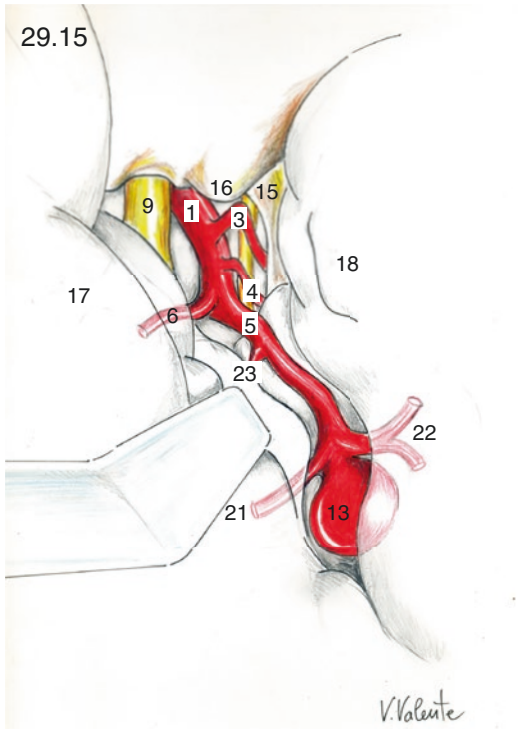

Fig. 29.15 Right MCA bifurcation aneurysm, surgical view

29.16

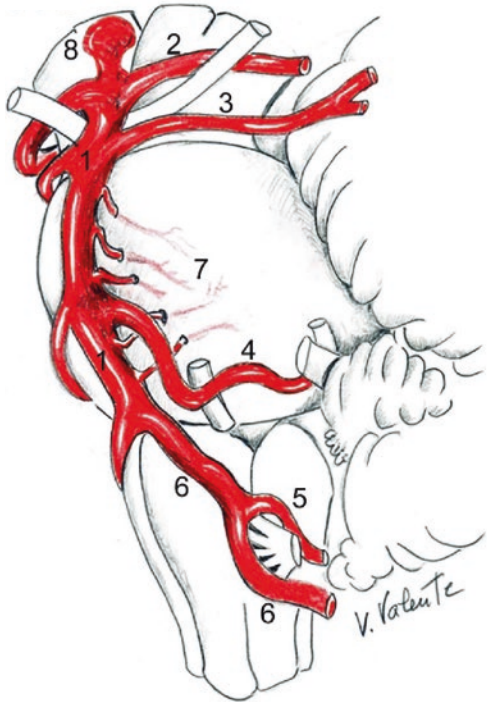

Fig. 29.16 Basilar tip aneurysm

1	Basilar artery
2	Posterior cerebral artery (P1)
3	Superior cerebellar artery
4	Anterior inferior cerebellar artery
5	Posterior inferior cerebellar artery
6	Vertebral artery
7	Brainstem perforating arteries
8	Aneurysm
9	Temporal lobe
10	Frontal lobe
11	Anterior clinoid process
12	Tentorium
13	Optic nerve
14	Internal carotid artery
15	Posterior communicating artery
16	Oculomotor nerve

29.17

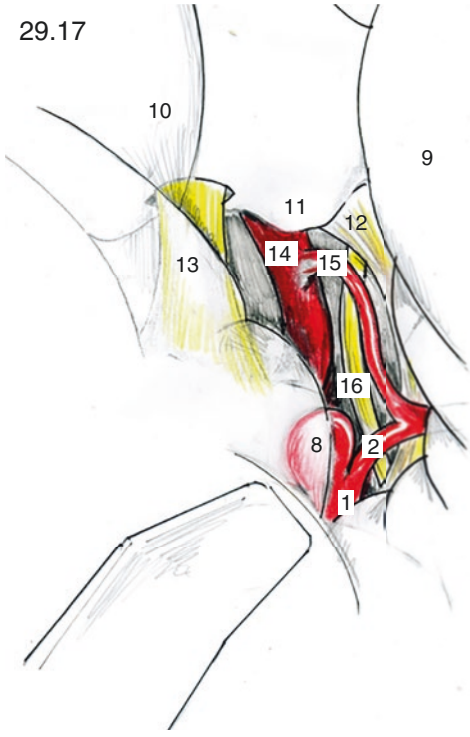

Fig. 29.17 Basilar tip aneurysm, surgical view

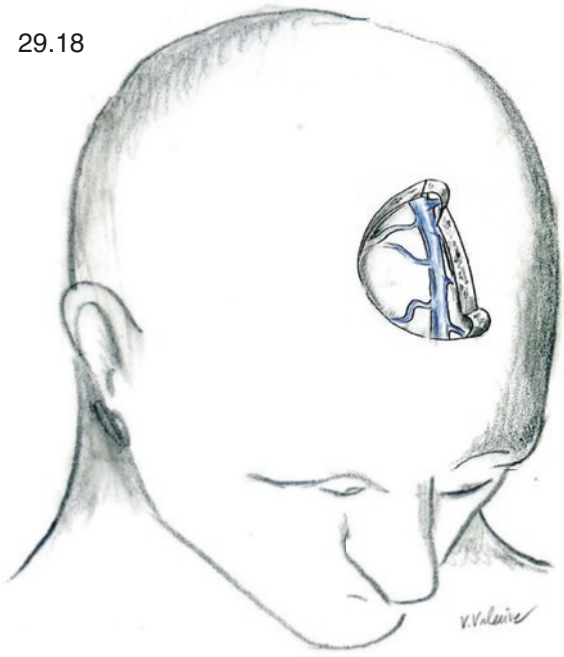

Fig. 29.18 Right frontal parasagittal approach

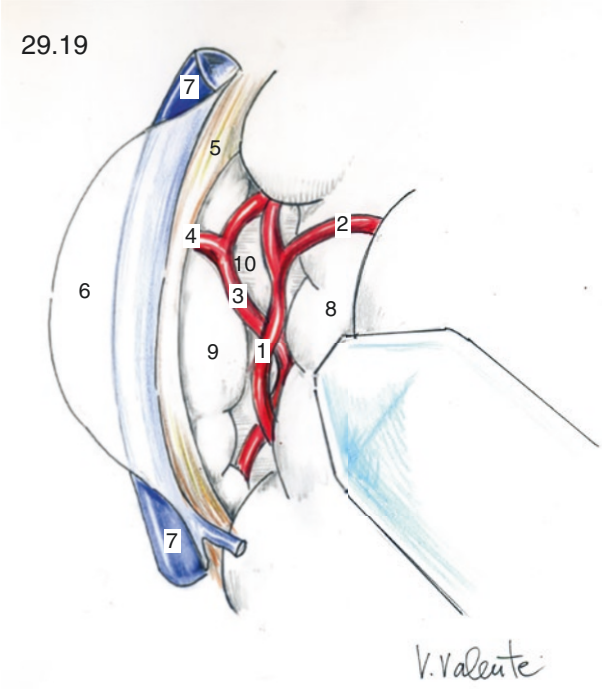

Fig. 29.19 Distal ACA anatomy, surgical view

Structure labels for Figs. 29.19, 29.20

1	Right pericallosal artery (A3)
2	Right callosomarginal artery
3	Left pericallosal artery (A3)
4	Left callosomarginal artery
5	Falx
6	Dura mater
7	Superior sagittal sinus
8	Right cingulate gyrus
9	Left cingulate gyrus
10	Corpus callosum
11	Aneurysm

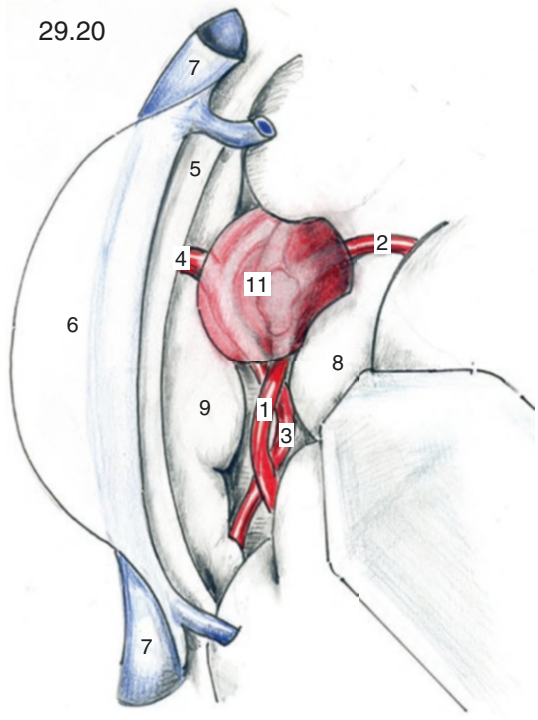

Fig. 29.20 Pericallosal artery aneurysm, surgical view

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