Vascular Temporal Bone Lesions

Hervé Tanghe

Contents

Abstract

This chapter discusses the possible causes of pulsatile tinnitus (PT). First the possible causes are classified into groups. The imaging strategy is explained, with the otoscopcally findings as starting point. Thereafter, only the vascular etiologies are highlighted in detail. Attention is also given to the neurointerventional treatment of some of these diseases.

1 Definition and Classification of Pulsatile Tinnitus

Tinnitus is a "sound in one ear or both ears, such as buzzing, ringing or whistling, occurring without an external stimulus" (American Heritage Dictionary 2000). Tinnitus can be classified as pulsatile (coinciding with the heartbeat) or continuous, and subjective (perceived only by the patient) or objective (perceptible to another person). Pulsatile tinnitus is less common than continuous tinnitus.

Pulsatile tinnitus (PT) can be caused by (1) vascular anatomical variants; (2) vascular tumors; (3) vascular malformations; (4) acquired vascular lesions; (5) vascularized chronic inflammatory tissue of the middle ear; (6) other diseases like otosclerosis, Paget disease, benign intracranial hypertension. Some other causes may be transient, related to drugs, hypertension, anaemia, and pregnancy (Table 1) (Weissman and Hirsch 2000; Madani and Connors 2009; Vattoh et al. 2010).

Vascular anatomical variants frequently are not associated with symptoms.

H. Tanghe (✉)
Department of Radiology,
Section of Neuroradiology & ENT Radiology,
Erasmus Medical Centre, Erasmus University Rotterdam,
's-Gravendijkwal 230, 3015 CE,
Rotterdam, The Netherlands
e-mail: h.l.j.tanghe@erasmusmc.nl

M. Lemmerling and B. De Foer (eds.), *Temporal Bone Imaging*, Medical Radiology. Diagnostic Imaging,
DOI: 10.1007/174_2013_935, © Springer-Verlag Berlin Heidelberg 2014
Published Online: 12 January 2013

Table 1 Overview of vascular temporal bone lesions

Pseudo lesions
Vascular anatomical variants
Tumors with rich vascularity
Acquired vascular lesions
Vascularized chronic inflammation tissue
Vascular malformations

Table 2 Imaging strategy in pulsatile tinnitus

Otoscopy: intratympanic mass	Small lesion	Only CT
	Vascular variants	Only CT
	Large lesion	CT and MRI
Otoscopy: no intratympanic mass		CTA and CTV

2 Imaging Strategy in PT

The pre-imaging evaluation of a patient consists of a detailed history, looking for hearing loss, vertigo, headaches, and a medical examination including a neuro-otologic and audiologic evaluation.

There is a wide variation in the reported incidence of structural abnormalities in patients with PT ranging from 44 to 91 % (Madani and Connors 2009). Diagnostic imaging, even performed optimally, fails to find a reason for PT in many patients (Verbist et al. 2011). If an intratympanic mass is seen at otoscopy, then the imaging analysis can start with a CT, thin sections with bone algorithm in the axial and coronal plane, of the temporal bone and adjacent structures of the skull base. The anatomy of the carotid canal and the jugular foramen is carefully studied. For the anatomical vascular variants and small soft tissue masses, like a small glomus tympanicum no further study is needed. For larger masses, an MRI examination is complementary and the CT study can be done without intravenous contrast. Various imaging strategies have been proposed for the investigation of PT in the otoscopically normal patient. Combined CT angiography (CTA) and CT venography (CTV) with a multidetector CT is a good approach for demonstrating arterial, venous, skull base and middle ear disease. MR and MR angiography cannot be trusted to confidently exclude all causes (Krishnan et al. 2006; Mattox and Hudgins 2008; Narvid et al. 2011; Verbist et al. 2011) (Table 2). Our protocol for the imaging analysis op PT consists of an CTA & CTV. The lower scan border is the level of vertebra cervical 7 and the upper border is the end of the skull. The lower border is chosen to include the carotid bifurcation in the protocol. The upper border is chosen for the detection of a possible high flow intracranial AVM, whereby the venous drainage is causing PT. Post-processing includes maximum intensity projections in the axial, coronal and sagittal planes. There are also separate multiplanar bony algorithm reconstructions of the right and left temporal bone with thin slices of 0.75 mm in the axial and coronal plane to detect an anatomical vascular variant or another intratemporal cause of the PT. For the detection of a dural arteriovenous fistula (DAVF) you have to look to: asymmetric arterial feeding vessels, "shaggy" appearance of a dural venous sinus, transcalvarial venous channels, asymmetric venous collaterals and abnormal size and number of cortical veins. The presence of asymmetrically visible and enlarged arterial feeders has a high sensitivity and specificity for the diagnosis of a DAVF (Fig. 1). A conventional digital subtraction angiography (DSA) is necessary in cases of a negative CTA, but a strong clinical suspicion and in the presence of vascular malformations to demonstrate the angioarchitecture and in case of endovascular treatment (Narvid et al. 2011).

3 Vascular Anatomical Variants

Vascular anatomical variants of the temporal bone are a frequent cause of PT or a vascular retrotympanic mass that often clinically cannot be differentiated from a paraganglioma. This differentiation is important prior to biopsy or therapy. Often these variants are an incidental asymptomatic finding on a CT done for other reasons (Table 3). The radiological diagnosis is possible with MRI, MR angiography, and with CT. For some variants, like the dehiscent jugular bulb, the bone detail, available with CT is important. On MR spin echo sequences, the signal void of the abnormally located artery or vein may be identical to the signal void of the cortical bone and the air in the ear and the anomaly can be missed. A gradient echo sequence gives a high signal from the vessel with better differentiation from the cortical bone.

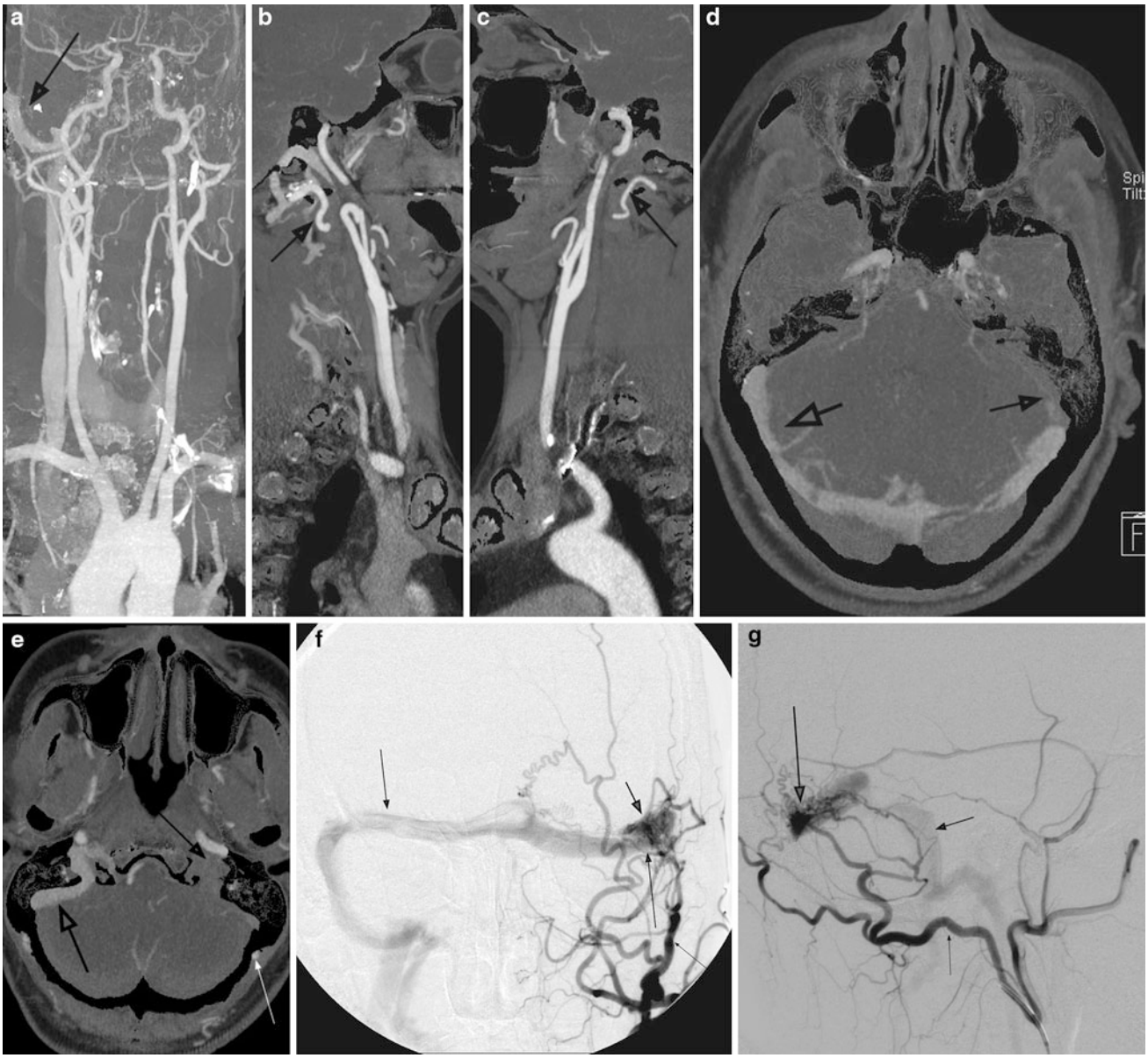

Fig. 1 **Use of CTA: dural A-V fistula *left* with contralateral *right* venous drainage.** CT Siemens Flash Definition double source/double energy with use of CT-angiography subtraction. **a** CTA- subtraction. Early venous drainage in *right* transverse, sigmoid sinus (*arrow*). **b** *Left* common carotid artery shows hypertrophy of the occipital artery (*arrow*). **c** *Right* common carotid artery with a normal occipital artery (*arrow*). **d–e** Axial reconstruction shows in the arterial phase early venous drainage *left* (*large arrow*), no contrast in *left* sigmoid sinus (*middle-sized arrow*) en hypertrophy of *left* occipital artery (*small arrow*). **f–g** DSA confirms the dural A-V fistula with nidus in the *left* transverse sinus (*large arrow*), the occlusion of the *left* sigmoid sinus (*middle-sized arrow*), the contralateral venous drainage (*middle-sized arrow*) and the feeding vessels, most important the hypertrophied occipital artery (*small arrow*)

Table 3 Vascular anatomical variants

Arterial	Venous
The aberrant ICA	High jugular bulb
Hyostapedial artery variants	Dehiscent jugular bulb
Laterally displaced ICA	Jugular bulb diverticulum
Isolated agenesis of the ICA	
Pharyngo-tympano-stapedial artery	
Splitting of the petrous ICA	

Table 4 CT appearance of the "aberrant ICA"

1	Absence of the vertical part of the carotid canal
2	Enlarged inferior tympanic canaliculus
3	The vertical segment of the ICA enters the temporal bone through (2), running more posterior and laterally than normal
4	Absence of the normal bony posterior margin of the horizontal part of the carotid canal
5	Soft tissue density in the middle ear at the level of the promontory, joining the horizontal carotid canal through the bony defect mentioned in (4) and simulating a paraganglioma
6	"Stenosis" at the entry point in the horizontal carotid canal

3.1 Arterial Variants

3.1.1 The Aberrant Internal Carotid Artery

The aberrant internal carotid artery (ICA) also called the aberrant flow of the internal carotid artery in the tympanic cavity (Lasjaunias et al. 2001) enters the skull base through an enlarged inferior tympanic canaliculus with a characteristic narrowing of the vessel. The fundamental cause is a segmental agenesis, absence of the cervical part of the internal carotid artery with the subsequent absence of the vertical part of the bony carotid canal. The ascending pharyngeal artery with its tympanic branch (going through its own enlarged inferior tympanic canaliculus) serves as a collateral pathway to the normal horizontal part of the ICA. At the end of the inferior tympanic canaliculus, the artery enters the middle ear cavity and is located behind the tympanic membrane at the promontorium as a "vascular mass," not covered by bone, before entering the horizontal part of the carotid canal through a bony dehiscence. An aberrant ICA can be associated with a persistent stapedial artery (vide infra; Tanghe 1994; Weisman and Hirsch 2000).

This anomaly can present with objective or subjective pulsatile tinnitus and conductive hearing loss. On otoscopy the ENT surgeon finds a vascular appearing retrotympanic mass that mimics a paraganglioma. A misdiagnosis as a glomus tympanicum must be avoided: the operation can result in a debacle (Tanghe 1994).

The CT features are characteristic (Table 4): (1) absence of a normal-appearing vertical segment of the carotid canal; (2) absence of the bony wall in the posterolateral portion of the horizontal segment of the carotid canal; (3) a round soft tissue mass in the middle ear at the promontorium. Especially in a coronal section this mass can look similar to a glomus tympanicum; (4) on axial CT this mass is in continuity with the horizontal segment of the carotid canal; (5) the aberrant ICA enters the tympanic cavity through an enlarged inferior tympanic canaliculus, located more lateral and posterior compared to a normal vertical ICA segment (Tanghe 1994) (Fig. 2). On conventional MRI, the diagnosis of an aberrant ICA is difficult because of the lack of contrast between the low signal of bone, the signal void of the ICA, and the air in the middle ear and mastoid portion. Flow-sensitive MR images or MR angiography (MRA) allow the diagnosis. Conventional angiography is not necessary for the diagnosis (Weisman and Hirsch 2000).

3.1.2 Partial Persistence of the Stapedial Artery

The so-called "persistent stapedial artery" is in fact a partial persistence of the stapedial artery, different from the full persistent variant which is vary rare (Lasjaunias et al. 2001). The partial persistent variant is an intratympanic origin of the middle meningeal artery. The artery originates from the ICA between the vertical and the horizontal portion of the carotid canal. It enters the middle ear cavity anteroinferiorly, runs along the promontory, passes between the crura of the stapes and enters the tympanic segment of the facial canal causing an enlargement of this canal. At the level of the first genu, it leaves the facial canal through its own foramen and enters the extradural space of the middle cranial fossa, becoming the middle meningeal artery (MMA) (Tanghe 1994). It can be associated with an aberrant ICA (Fig. 3) and with an aneurysmal enlarged ICA (Fig. 4).

This vascular variant is most often an incidental finding at operation or on a CT examination (easily overlooked) and rarely symptomatic with pulsatile tinnitus.

A persistent stapedial artery cannot be diagnosed with MR or even MRA. The CT features are pathognomonic (Tanghe 1994) (Table 5): (1) absence of the foramen spinosum, because there is no MMA to go through; (2) erosion of the promontory; (3) widening of the proximal tympanic segment of the facial canal to accommodate for the artery; and (4) absence of the MMA from the internal maxillary artery on angiography.

3.1.3 Splitting of the Petrous ICA

In this case, two channels carry the flow into the petrous portion. Both channels belong to the ascending pharyngeal artery of the ICA system and the internal carotid artery can still be considered segmentally agenetic (Lasjaunias et al. 2001) (Fig. 5).

3.1.4 The Intrameatal Vascular Loop

The presence of an intrameatal vascular loop of the anterior inferior cerebellar artery (AICA) in the internal auditory canal is a frequent finding on MRI or in cadaver sections (Fig. 6). Some authors consider this as a possible cause of

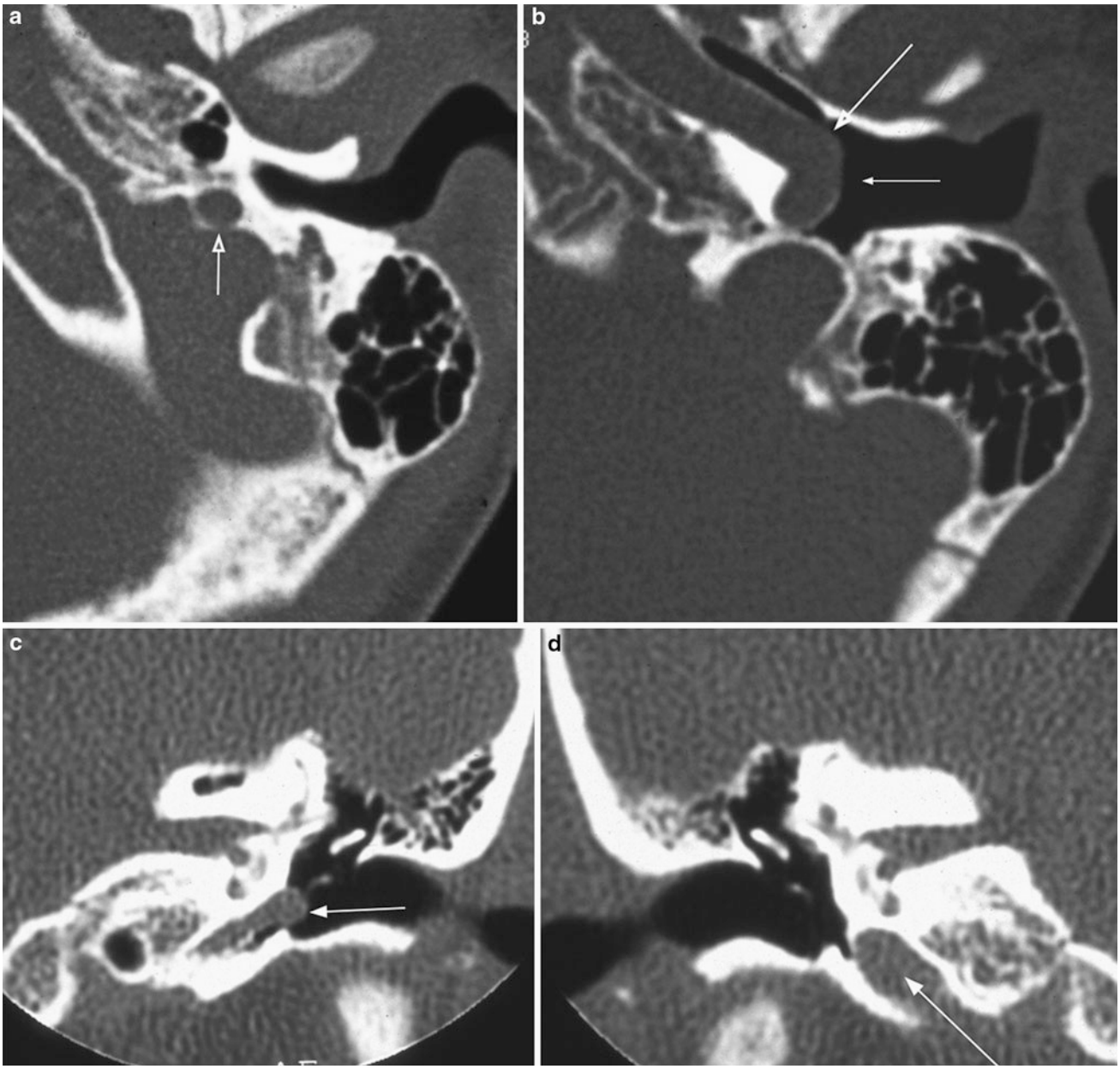

Fig. 2 **Aberrant internal carotid artery**. **a** Axial CT at the level of the enlarged inferior tympanic canaliculus. This canal is located more lateral and is smaller than the normal vertical part of the carotid canal (*arrow*). **b** Axial CT at the level of the horizontal part of the carotid canal. Part of the lateral bony wall is absent (*large arrow*) and the carotid artery comes close to the tympanic membrane (*small arrow*). **c** Coronal CT at the level of the genu in a different patient. The carotid artery reaches the tympanic membrane (*arrow*). The inferior tympanic canaliculus is smaller than the normal vertical part of the carotid canal at the opposite site in **d** (*arrow*)

PT and advocate a major surgical treatment (De Ridder et al. 2005). However, this intravascular loop is encountered in 40 % of the people. There are many persons with deep vascular loops into the internal auditory canal that do not suffer from pulsatile tinnitus. Conversely, not all patients with pulsatile tinnitus have loops into the IAC. More research and understanding of the underlying mechanisms of pulsatile tinnitus is needed before proposing a craniotomy as a treatment for this symptom.

3.2 Venous Variants

3.2.1 The High or High Riding Jugular Bulb

The jugular bulb is considered high when it extends above the level of the floor of the internal auditory canal. On a T1 W MR sequence after gadolinium this variant can simulate an enhancing tumor in the medial temporal bone. But the bony margins of the jugular foramen are intact and well corticated. In case of doubt a CT with bone algorithm can

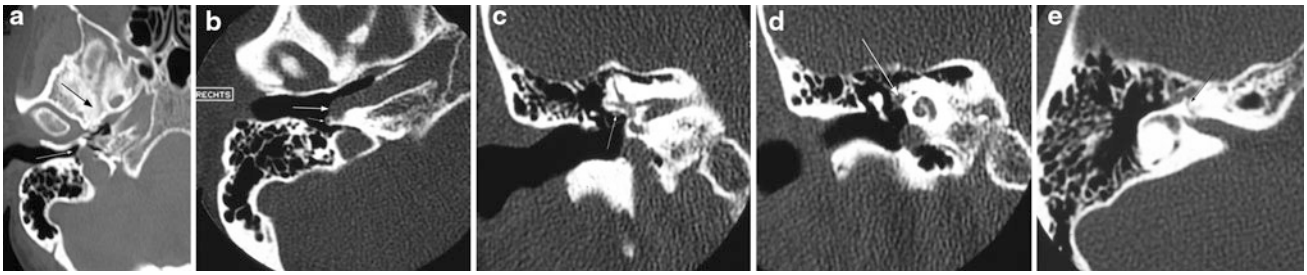

Fig. 3 **Persistent stapedial artery, associated with an aberrant internal carotid artery**. **a** Axial CT. The foramen spinosum is absent (*large arrow*), compared with the normal left side. The carotid artery is in contact with the tympanic membrane through a bony defect at the end of inferior tympanic canaliculus (*small arrow*). There is no bony margin between the inferior tympanic canaliculus and the jugular foramen. **b** Axial CT. The carotid artery lies in the middle ear with a large bony defect in the horizontal part of the carotid canal (*large arrow*). Notice the origin of the persistent stapedial artery (*small arrow*). **c** Coronal CT at the level of the oval window; **d**, **e** coronal and axial CT at the level of the proximal tympanic segment of the facial canal. The facial canal at the level of the oval window is still normal in size (*small arrow*), but it is enlarged at his proximal tympanic segment, to accommodate for the persistent stapedial artery (*large arrow*). The labyrinthine segment of the facial canal is normal (*small arrow* in **e**)

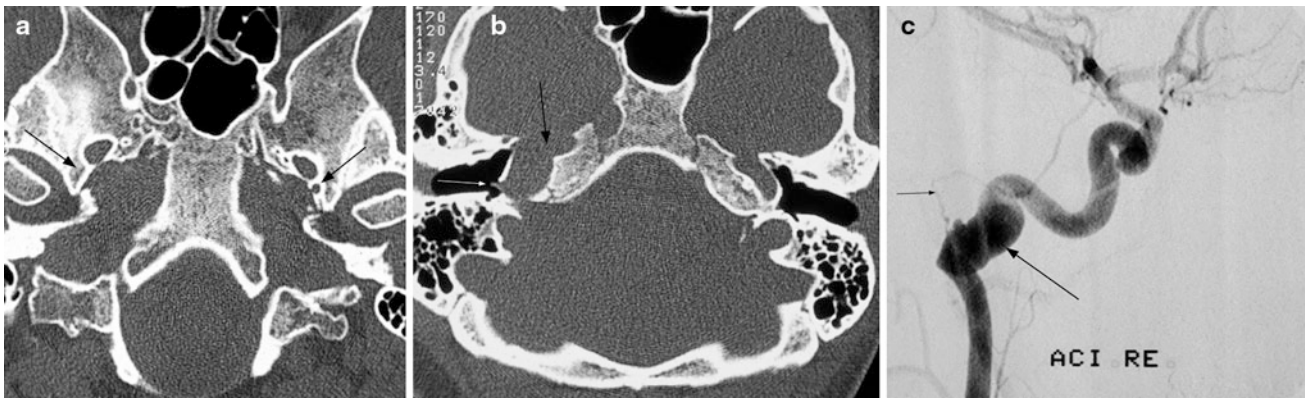

Fig. 4 **Persistent stapedial artery, associated with aneurysmal enlargement of the carotid artery**. **a** Axial CT of the skull base. The foramen spinosum is absent on the right side (*arrow*) and normal on the *left side* (*arrow*). **b** Axial CT at the level of the horizontal part of the carotid canal. The canal is enlarged (*large arrow*). Also notice the origin of the persistent stapedial artery (*small arrow*). **c** The angiogram in the same patient shows the fusiform aneurysmal enlargement of the carotid artery (*large arrow*) and the persistent stapedial artery (*small arrow*)

Table 5 CT appearance of the partial persistent stapedial artery

1	Absence of the foramen spinosum
2	Erosion of the promontory
3	Widening of the proximal tympanic segment of the facial canal

be done (Tanghe 1994; Weissmann and Hirsch 2000; Vattoh et al. 2010).

3.2.2 The Dehiscent Jugular Bulb

A dehiscent jugular bulb lacks a complete cortical covering and lies in part in the hypotympanum of the middle ear. It usually presents as a vascular retrotympanic mass behind the posteroinferior quadrant of the tympanic membrane (Tanghe 1994; Weissmann and Hirsch 2000; Vattoh et al. 2010).

On MR the diagnosis is difficult because MR cannot demonstrate the presence or absence of the cortical covering of the jugular foramen. The dehiscence can be inferred from a more lateral position of the bulb on coronal images and by the presence of a lateral lobulation.

On CT the dehiscent jugular bulb is visible as a mass low in the medial part of the middle ear. This mass is in continuity with the jugular bulb in the foramen trough a bony defect (Fig. 7).

3.2.3 The Jugular Bulb Diverticulum

A jugular diverticulum is a protrusion of the jugular bulb superior and medial to the jugular foramen which extends above the inferior border of the round window. It does not extend into the middle ear, but can be associated with PT, because of turbulence of the venous flow. The tympanic

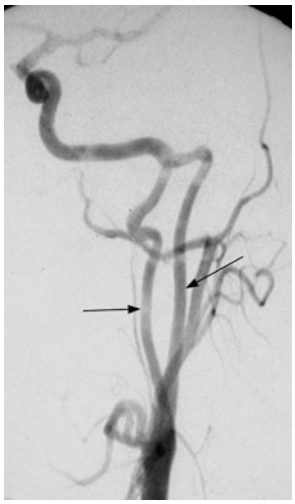

Fig. 5 Splitting of the internal carotid artery. Angiogram. The carotid artery is split into two parts (*arrow*) from its origin at the carotid bifurcation, until the horizontal part of the carotid canal

membrane is normal and the anomaly cannot be seen at otoscopy (Weissmann and Hirsch 2000; Madani and Connor 2009; Vattoth et al. 2010) (Fig. 8).

4 Acquired Vascular Lesions

Numerous acquired vascular lesions can cause PT. A detailed description is beyond the scope of this chapter (Lo and Solti-Bohman 1996; Weissmann and Hirsch 2000; Sismanis and Girevendoulis 2008; Madani and Connor 2009) (Table 7).

Aneurysm of the petrous portion of the carotid artery can cause PT but is rare. Intracranial aneurysms are far more frequent (Table 6).

Atherosclerotic stenotic disease at the carotid bifurcation in the neck may produce turbulence of flow but gives only occasionally PT. Despite its high prevalence as a cause of asymptomatic carotid bruit, atherosclerosis is not a common cause of symptomatic pulsatile tinnitus (Sandok et al. 1982).

Fibromuscular dysplasia of the internal carotid artery most frequently gives intracranial ischemia, but PT is the second most frequent manifestation (Sismanis et al. 1994).

Other acquired vascular lesions that can give tinnitus are: spontaneous dissection of the internal carotid artery, idiopathic intracranial hypertension, a vascular compression of the eight nerve in the cerebellopontine angle.

5 Vascular Tumors

Paraganglioma is the most common tumor causing PT or a vascular tympanic membrane (Lasjaunias et al. 2001). Other less common vascular tumors are: meningioma, haemangioma of the facial nerve, endolymphatic sac tumor (see the chapter on imaging of the jugular foramen),

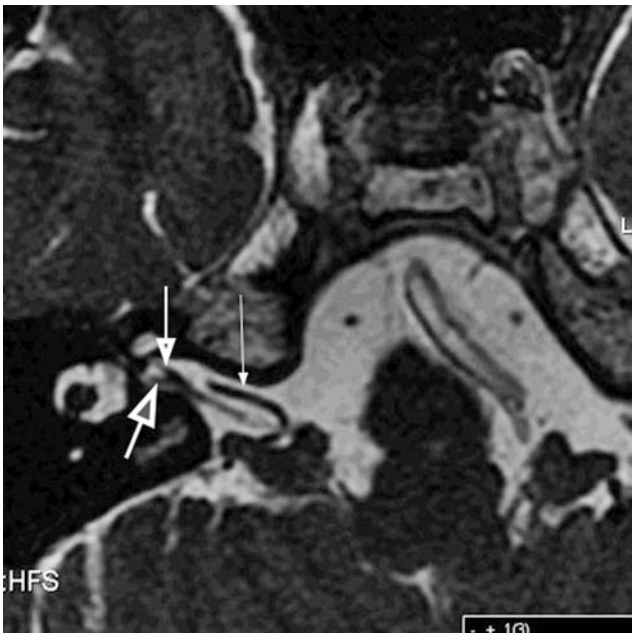

Fig. 6 Intrameatal vascular loop MRI 3D FIESTA. The presence of an intrameatal vascular loop of the anterior inferior cerebellar artery (AICA) (*small arrow*) in the internal auditory canal is a frequent finding on MRI or in cadaver sections. *Middle-sized arrow* cochlear nerve, *large arrow* inferior vestibular nerve

cavernous haemangioma of the middle ear and vascular metastasis like Grawitz tumor (Madani and Connor 2009) (Table 7). A meningioma located in the temporal bone can clinically mimic a paraganglioma. When it protrudes in the middle ear, the ENT surgeon sees on otoscopy a vascular retrotympanic mass, indistinguishable from a paraganglioma. Meningioma is characterized by the presence of sclerotic changes on CT and the absence of flow voids on MR, in contrast to a glomus tumor (Vattoth et al. 2010). Meningioma and metastasis can be as vascular as paraganglioma. For a detailed discussion of the noninvasive imaging features of these tumors, see the chapter on the imaging of the jugular foramen.

An angiography is rarely needed for diagnostic purposes and is usually done to perform a preoperative embolization in cases where an operation is chosen as the treatment.

Paragangliomas have a characteristic angiographic appearance with enlarged feeding arteries, an early and intense tumor blush and early draining veins (Fig. 9a). The intratumoral arterioles in the periphery of the tumor are smaller than those in the centre. There are multiple intratumoral direct communications between arterioles and venules with arterio-venous shunting. Large paragangliomas (type C2 or more, according to the classification of U. Fisch) (Fisch and Mattox 1988) have a lot of feeding vessels and large intratumoral A-V shunts, making them almost impossible to resect without a pre-operative embolization (Connors and Wojak 1999) (Fig. 9b and c).

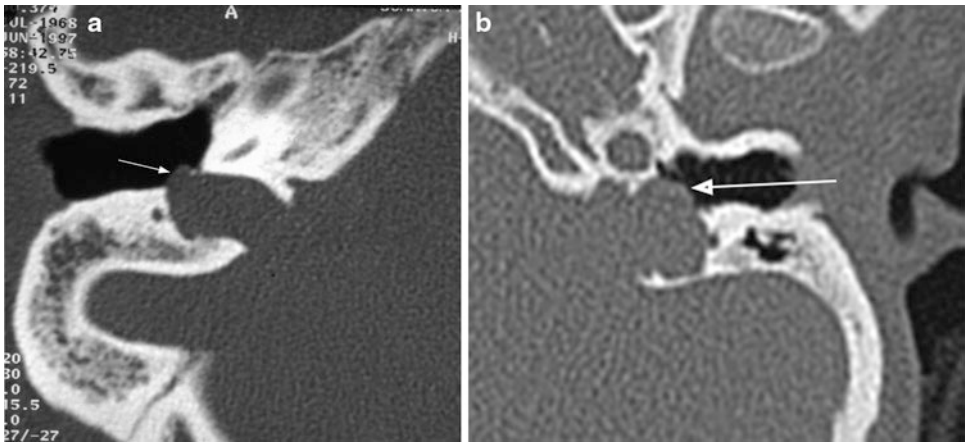

Fig. 7 **Dehiscent jugular bulb.** **a** Axial CT. The jugular bulb protrudes in the middle ear through a bony defect in the jugular foramen (*arrow*). **b** Axial CT in a different patient: a woman of 19 yr with disease of Crouzon. Same abnormality (*arrow*)

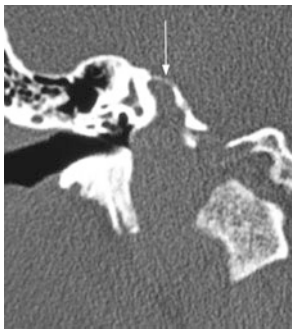

Fig. 8 **Jugular bulb diverticulum.** Coronal CT. There is a protrusion of the jugular bulb superior and medial to the jugular foramen. Such a protrusion is called a diverticulum (*arrow*). It does not extend into the *middle* ear

Table 6 Acquired vascular lesions

Petrous carotid aneurysm
Atherosclerotic carotid artery disease
Fibromuscular dysplasia
Spontaneous carotid dissection
Thrombosis of dural sinus with intracranial hypertension

Table 7 Tumors of the temporal bone that may present with pulsatile tinnitus or a vascular retrotympanic mass

Paraganglioma
Haemangioma
Meningioma
Endolymphatic sac tumor
Vascular metastasis

In 75–85 % of the cases the angioarchitecture of a paraganglioma is multicompartmental (Table 8).

The pre-embolization angiographic protocol consists of a study of the ICA, the vertebral, internal maxillary, occipital and ascending pharyngeal artery. It is further important to look at the relationship of the tumor to the internal jugular vein, the inferior petrosal sinus and the other dural sinus. Especially the detection of extraluminal compression or intraluminal tumor extension is important for the surgeon. This information can be obtained from the venous phase of the vertebral and carotid angiography and from the CT and MRI.

The embolization is carried out with a microcatheter with superselective catheterisation of the different feeding vessels. As embolisation material can be used particles polyvinyl alcohol (PVA), with a size of 150–250 μ or Onyx. In case of a good intratumoral catheter position smaller particles, less than 90 μ, can be used to reach the centre of the tumor trough the small-sized peripheral intratumoral arterioles. To prevent complications, the dangerous anastomoses among the vertebral, internal carotid and external carotid arteries must be known (Moret et al. 1982; Connors and Wojak 1999; Lasjaunias et al. 2001) (Fig. 10). Furthermore, it is important to remember that several cranial nerves receive their blood supply by the vessels we intend to embolize: the IX, X, XI and XII cranial nerve by the ascending pharyngeal artery, the facial nerve by the middle meningeal and accessory meningeal artery, the III, IV, V, VI cranial nerve by the accessory meningeal artery. Use of too small particles or of glue in conjunction with an improper catheter position can result in definitive cranial nerve palsy.

Until early in this millennium microsurgical resection, endovascular embolization, conventional radiation therapy, or any combination of these, were the only treatment modalities for glomus jugulare tumors. However, with the establishment of effectiveness of stereotactic radiotherapy

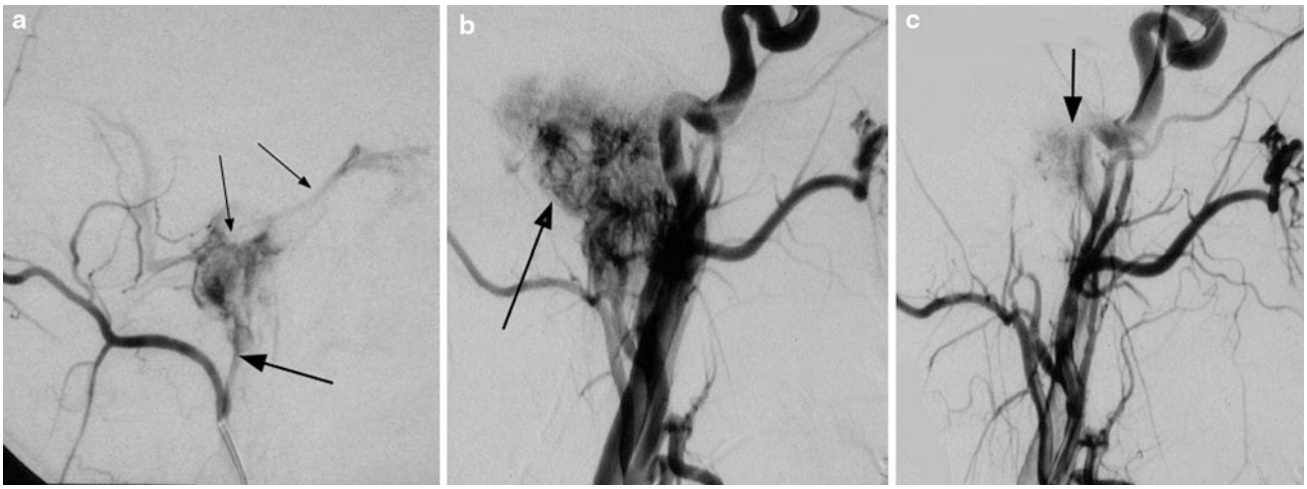

Fig. 9 **Glomus jugulotympanicum. a** Occipital artery. The posterior part of the tumor is fed by the stylomastoid artery (*large arrow*). The angiographic appearance is characteristic, with an enlarged feeding artery, an early and intense tumor blush and early draining veins (*small arrow*). **b**, **c** Angiogram of the common carotid artery before and after embolization. The tumor is very vascular with an intens tumor blush (*arrow* in **b**). The small tumor blush *left*, after embolization, comes from feeding from the internal carotid artery via the caroticotympanic ramus (*arrow* in **c**). This vessel usually cannot be catheterized selectively

Table 8 Angioarchitecture of paraganglioma: compartment and feeding vessels (Krishnan et al. 2006). For each compartment the feeding vessels are arranged starting from the principal vessel to additional feeding vessels with increasing volume of that tumor compartment

Compartment	Feeding vessels
Infero-medial	Inferior tympanic branch (ascending pharyngeal artery)
	Neuromeningeal trunk (ascending pharyngeal artery)
	Lateral clival branch of the ICA
	Meningeal branches of the vertebral artery
Postero-lateral	Stylomastoid artery (occipital artery or posterior auricular artery)
	Meningeal branches of the occipital artery
	Meningeal branches of the vertebral artery
Anterior	Anterior tympanic artery (internal maxillary artery)
	Caroticotympanic artery (ICA)
	Cavernous branches of the ICA
Superior	Middle meningeal artery
	Accessory meningeal artery

(SRT) in the treatment of cerebral arteriovenous malformations and in other highly vascular brain tumors, the vascular character of paragangliomas made them appropriate targets for SRT. Compared with conventional radiotherapy, SRT has fewer complications, and shorter course of treatment and hospital stay (Genc et al. 2010; Ivan et al. 2011). Other authors demonstrated that gross total resection of large paraganglioma (type C and D) is possible with low mortality and may be curative (Makiese et al. 2012).

6 Vascular Malformations

Vascular malformations may cause PT, usually objective and sometimes subjective. Often the patient has in addition an audible bruit or a pulsatile thrill. When these signs are present, the angiography can be the initial imaging study. It is important to know that not only intracranial or skull base vascular malformations, but also cervical vascular lesions like vertebral arteriovenous fistula can cause PT. Every high

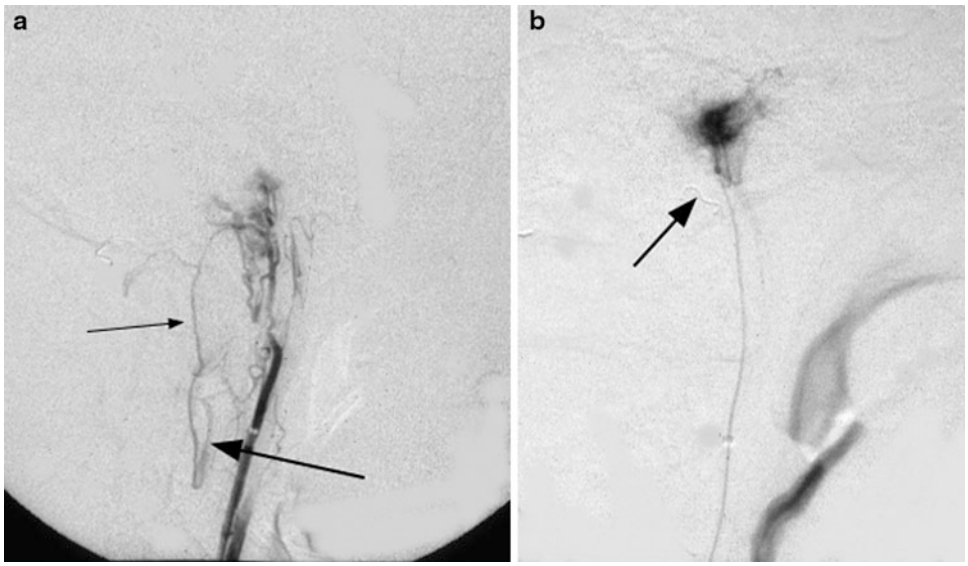

Fig. 10 **Dangerous anastomosis. a** Selective injection of the inferior tympanic artery with the microcatheter, shows the tumor blush in the *middle* ear. There is also a visualisation of the vertebral artery (*large arrow*) at the C2–3 level via the dens arcade (*small arrow*). **b** After closure of the dens arcade with a microcoil (*arrow*), the feeding artery can safely be embolized

Table 9 Vascular malformations that can cause pulsatile tinnitus

Dural arteriovenous fistula
Carotid-cavernous fistula: direct or dural
Vertebral arteriovenous fistula
External carotid fistula
Brain AVM

Table 10 Classification of dural arteriovenous fistula (DAVF) according to the type of venous drainage (Cognard et al. 1995)

Type I	Antegrade into the venous sinus
Type IIa	Into the venous sinus with reflux in the sinus or retrograde into the sinus
Type IIb	Antegrade into the venous sinus with cortical venous drainage
Type IIa + b	Retrograde into the venous sinus with cortical venous drainage
Type III	Direct cortical venous drainage
Type IV	Type III with venous ectasia
Type V	Spinal perimedullary venous drainage

flow vascular lesion of which the venous drainage comes through or near the jugular bulb can cause PT (Table 9). Therefore, the angiographic protocol should not only include the internal and the external carotid artery but also the vertebral artery. In some cases, the vascular lesion can be located contralateral to the side of the PT. For example, a left-sided dural carotid-cavernous fistula can drain through the right cavernous sinus into the right inferior petrosal sinus to the jugular bulb. Both cavernous sinus plexuses exist anteriorly and posteriorly (Lasjaunias et al. 2001).

Dural arteriovenous fistula's (DAVF) are the most common cause of objective PT in the patient with a normal otoscopic examination (Weissman and Hirsch 2000). DAVF are abnormal shunts located in the dura. They can occur at any site in the dura, but most frequently they are located near a venous sinus. They are classified according to their location, or to the type of venous drainage. The criterion standard to diagnose and classify cranial DAVFs is DSA. This is an invasive, relatively expensive, and time-consuming procedure. A noninvasive alternative is CTA although it may not

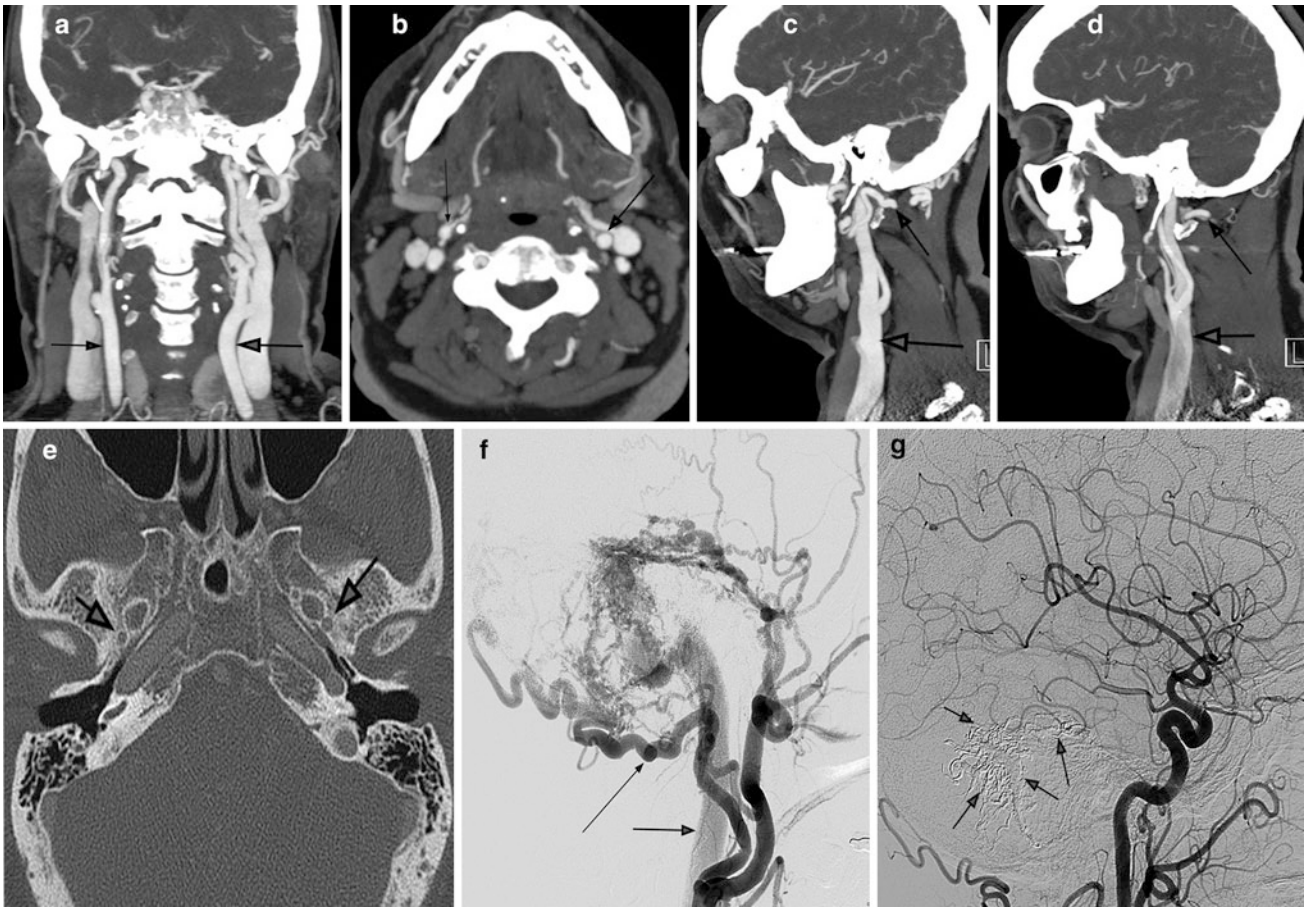

Fig. 11 **Dural arteriovenous fistula of the transverse sinus type I.** **a–d** CT-angiography, **e–f** DSA with embolisation with Onix. **a** The *left* common carotid artery (*large arrow*) is larger than the *right* (*small arrow*). **b** The *left* external carotid artery (*large arrow*) is larger than the *right* (*small arrow*). **c** *Left* sagittal view. The *left* occipital artery is hypertrophied (*small arrow*), and also the *left* jugular vein (*large arrow*). **d** *Right* sagittal view. Normal *right* occipital artery (*small arrow*) and jugular vein (*large arrow*). **e** Because of the hypertrophy of the feeding *left* meningeal artery, the *left* foramen spinosum is enlarged compared to the *right* (*arrow*). **f** DSA before embolization: feeding occipital artery (among others) (*small arrow*). Draining transverse sinus (*large arrow*). **g** DSA after embolization. The radio-opaque Onix. is visible (*arrow*). The fistula is closed

rule out a small slow-flow DAVF (Willems et al. 2011). The classification is important for determining the natural history, the prognosis and the indication for treatment (Cognard et al. 1995; Ghandhi et al. 2012) (Table 10). Type I lesions have a benign natural history and these lesions are only treated when the pulsatile tinnitus cannot be tolerated by the patient. Type IIa lesions are less benign with intracranial hypertension occurring in 20 % of the cases. In type IIb the risk for an intracranial bleeding is 10 %, in type III the risk is 40 % and in type IV 65 %. Patients with a DAVF type V developed progressive myelopathy in 50 % of the cases. Figure 11 gives an example of the possibilities of CT-A in the diagnosis of DAVF, with a lot of abnormalities you can find in the CT-A.

The treatment of choice is endovascular, via the arterial or via the venous route. It is beyond the scope of this chapter to go further in detail into the endovascular treatment. Surgery is an option in case of failure of the endovascular treatment. When the nidus of the DAVF is small, than stereotactic radiosurgery is an option.

6.1 Vertebral Arteriovenous Fistula

Vertebral arteriovenous fistula (VAF) are less common lesions. The most frequent cause is trauma, but they may also occur spontaneously, sometimes associated with fibromuscular dysplasia and neurofibromatosis type I. The clinical manifestations can vary: spinal cord or vertebrobasilar ischemia, spinal cord compression, nerve root compression, and pulsatile tinnitus. A bruit is present in nearly 100 % of the cases. The treatment of choice is endovascular (Lasjaunias et al. 2001) (Fig. 12).

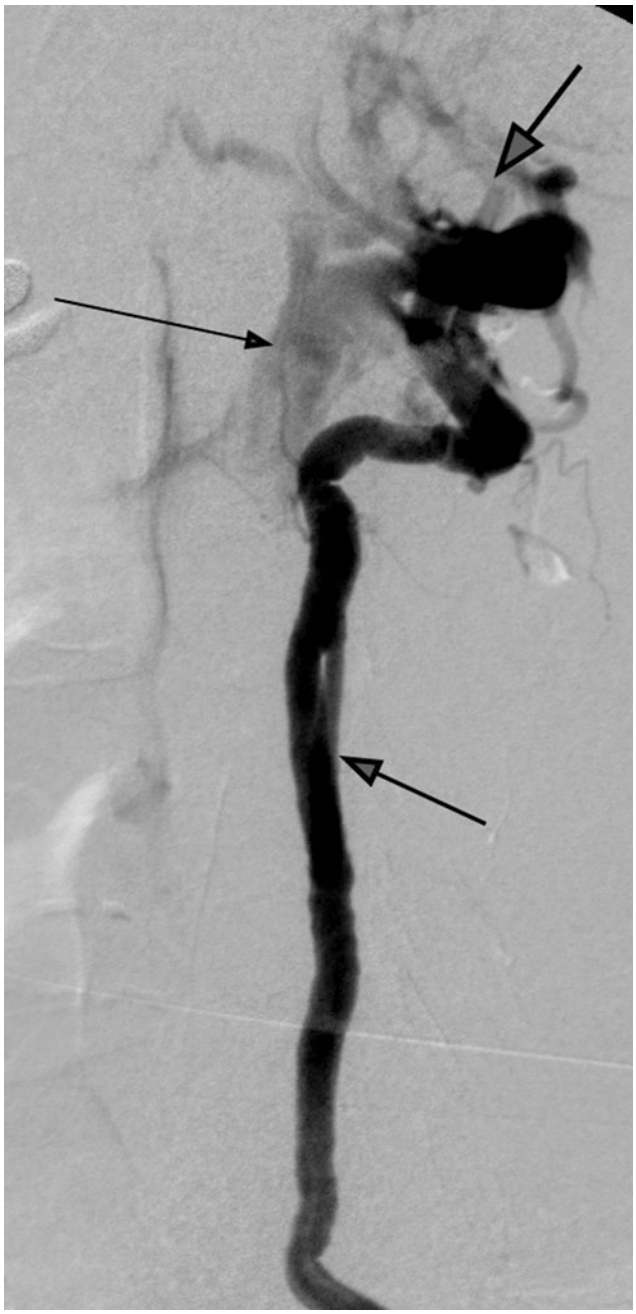

Fig. 12 **Vertebro-jugular fistula.** Patient with PT caused by a lesion just below the skull base. DSA *left* vertebral artery frontal view. High flow vertebra-jugular fistula. Hypertrophied feeding vertebral artery (*middle-sized arrow*). The fistel place is high cervical, just beneath the jugular foramen (*large arrow*). Early filling of draining veins, also epidural veins (*small arrow*)

References

American Heritage Dictionaries (2000) The American heritage dictionary of the english language, 4th edn. American Heritage Dictionaries, Boston

Cognard C, Gobin YP, Pierot L, Bailly AL, Houdart E, Casasco A, Chiras J, Merland JJ (1995) Cerebral dural arteriovenous fistulas: clinical and angiographic correlation with a revised classification of venous drainage. Radiology 194:671–680

Connors JJ III, Wojak JC (1999) Paragangliomas. In: Connors JJ III, Wojak JC (eds) Interventional Neuroradiology. Strategies and Practical Techniques, Saunders, Philadelphia

De Ridder M, De Ridder L, Nowé V, Thierens H, Van de Heyning P, Moller A (2005) Pulsatile tinnitus and the intrameatal vascular loop: why do we not hear our carotids? Neurosurgery 57:1213–1217

Fisch U, Mattox D (1988) Microsurgery of the skull base. Thieme, New York

Genc A, Bicer A, Abacioglu U, Peker S, Pamir M, Kilic T (2010) Gamma knife radiosurgery for the treatment of glomus jugulare tumors. J Neurooncol 97:101–108

Ghandi D, Chen J, Pearl M, Huang J, Gemmete JJ, Kathuria S (2012) Intracranial dural arteriovenous fistula: classification, imaging findings and treatment. Am J Neuroradiol 33:1007–1013

Ivan ME, Sughrue ME, Clark AJ, Kane AJ, Aranda D, Barani IJ, Parsa AT (2011) A meta-analysis of tumor control rates and treatment-related morbidity for patients with glomus jugulare tumors. J Neurosurg 114:1299–1305

Krishnan A, Mattox DE, Fountain AJ, Hudgins PA (2006) CT arteriography and Venography in pulsatile tinnitus: preliminary results. Am J Neuroradiol 27:1635–1638

Lasjaunias P, Berenstein A, Ter Brugge KG (2001) Surgical neuro-angiography, vol 1, 2nd edn. Springer, Berlin

Lo WWM, Solti-Bohman LG (1996) Vascular tinnitus. In: Som PM, Curtin HD (eds) Head and Neck Imaging, 3rd edn. Mosby—Year Book Inc, St. Louis

Madani G, Connor SEJ (2009) Imaging in pulsatile tinnitus. Clin Radiol 64:319–328

Makiese O, Chibbaro S, Marsella M, Tran Ba Huy P, George B (2012) Jugular foramen paraganglioma: management, outcome and avoidance of complications in a series of 75 cases. Neurosurg Rev 35:185–194

Mattox DE, Hudgins P (2008) Algorithm for evaluation of pulsatile tinnitus. Acta Oto-Laryngolocica 128:427–431

Moret J, Delvert JC, Bretonneau CH, Lasjaunias P, de Bicetre CH (1982) Vascularization of the ear: normal-variations-glomus tumors. J Neuroradiol 3:209–260

Narvid J, Do HM, Blevins NH, Fischbein (2011) CT angiography as a screening tool for dural arteriovenous fistula in patients with pulsatile tinnitus: feasibility and test characteristics. Am J Neuroradiol 32:446–453

Sandok BA, Whisnant JP, Furlan AJ (1982) Carotid artery bruits: prevalence survey and differential diagnosis. Mayo Clin Proc 57:227–230

Sismanis A, Girevendoulis A (2008) Pulsatile tinnitus associated with internal carotid artery morphologic abnormalities. Otol Neurotol 29:1032–1036

Sismanis A, Stamm MA, Sobel M (1994) Objective tinnitus in patients with atherosclerotic carotid artery disease. Am J Otol 15:404–407

Tanghe H (1994) Congenital malformations and anatomic variations of the ear. Rivista di Neuroradiologica 7:417–422

Vattoth S, Shah R, Curé JK (2010) A compartment-based approach for the imaging evaluation of pulsatile tinnitus. Am J Neuroradiol 31:211–218

Verbist BM, Mancuso AA, Antonelli PJ (2011) Pulse synchronous (pulsatile) tinnitus. In: Macuso AA, Hanafee WN, Verbist BM, Hermans R (eds) Head and neck radiology. Wolters Kluwer, Philadelphia

Weissman JL, Hirsch BE (2000) Imaging of tinnitus: a review. Radiology 216:342–349

Willems PWA, Brouwer PA, Barfett JJ, terBrugge KG, Krings T (2011) Detection and classification of cranial dural arteriovenous fistulas using 4D-CT angiography: initial experience. Am J Neuroradiol 32:49–53