

# Cochlear blood supply: an update on anatomy and function

Thierry Mom <sup>1</sup>, Jean Chazal <sup>2</sup>, Jean Gabrillargues <sup>3</sup>, Laurent Gilain <sup>1</sup>, Paul Avan <sup>4</sup>

<sup>1</sup> ORL and Head&Neck Surgery Department - Gabriel Montpied Teaching Hospital - Clermont-Ferrand - France

<sup>2</sup> Neurosurgery A Department - Gabriel Montpied Teaching Hospital - Clermont-Ferrand - France

<sup>3</sup> Radiology Department (Neuroradiology Unit) - Gabriel Montpied Teaching Hospital - Clermont-Ferrand - France

<sup>4</sup> Director of the sensory Biophysics Laboratory - EA 2667 - School of Medicine - Auvergne Clermont I University - Clermont-Ferrand - France

## ABSTRACT

Thorough familiarity with the cochlear blood supply is extremely useful to improve the rate of hearing preservation after pontocerebellar tumor removal and to understand the pathophysiology of sensorineural hearing loss caused by vascular disease. Current knowledge on the cochlear blood supply is reviewed herein. The vascular anatomy of the cochlea shows that preservation of the internal auditory artery is crucial to hearing preservation after pontocerebellar tumor removal. Vascular cochlear partition is relevant to the symptoms of hearing loss due to vascular causes. The close dependency of the endolymphatic potential on the stria vascularis explains the vulnerability of otoacoustic emissions to ischemia. In conclusion, detailed knowledge of cochlear vascular anatomy is essential in order to preserve hearing during pontocerebellar tumor surgery. Evaluations of cochlear blood flow measurements using laser Doppler velocimetry may provide insights into the pathophysiology of sensorineural hearing loss caused by vascular disease.

*(Fr ORL - 2005 ; 88 : 81 - 88)*

**Keywords:** Blood flow, Cochlea, Auditory-facial pedicle, Otoacoustic emissions, Sensorineural hearing loss.

---

Submitted for publication: august 2004

Accepted for publication: july 2005

**Corresponding author: Thierry Mom**

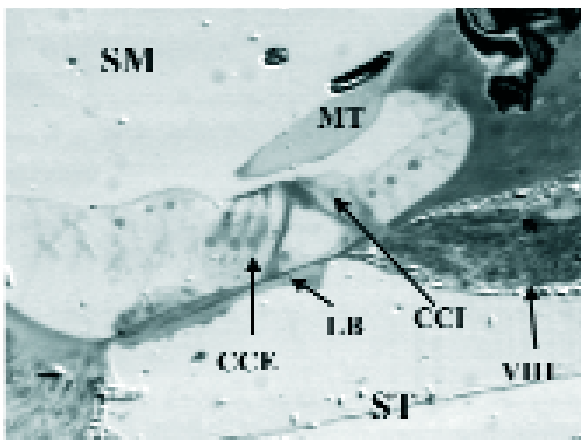
Service d'ORL et de Chirurgie Cervico-Faciale,  
Hôpital Gabriel Montpied - Centre Hospitalier  
Universitaire  
30 place Henri Dunant - 63 000 Clermont-Ferrand  
France  
e-mail: tmom@chu-clermontferrand.fr

### INTRODUCTION

Normal blood supply to the cochlea is crucial to auditory transduction, the mechanism by which sounds are converted to nerve impulses that travel along the auditory pathways to the gyri temporales transversi (Heschl's gyri). A unique feature of cochlear function is that intrinsic mechanical factors modulate the initial acoustic vibration before stimulating the auditory nerve. This initial phase of auditory transduction, which is dependent on cochlear micromechanics, explains the precision with which auditory stimuli are processed. It is dependent on contraction of the outer hair cells (OHCs) of the organ of Corti. OHC contractions are effective only when amplified. Amplification is ensured by the stria vascularis, which generates the endolymphatic potential (Figure 1). Thus, cochlear ischemia is followed almost immediately by hearing loss.

**Figure 1: Diagram of the organ of Corti seen under the light microscope.**

*The stria vascularis is not shown.*



*OHC: outer hair cells; IHC: inner hair cells; BM: basilar membrane; TM: tectorial membrane; SM: scala media. ST: scala tympani. VIII: auditory nerve fibers*

Among early experimental studies, those conducted by Perlman et al. [1] documented ischemia-induced alterations in cochlear function and histology. Subsequent animal studies shed light on the pathophysiology of cochlear ischemia, from which information on cochlear physiology was inferred [2,-7]. In humans, documentation of cochlear ischemia is diffi-

cult to obtain. Recognizing cochlear damage caused by ischemia alone among causes of sensorineural hearing loss requires invasive studies, which carry a risk of increased functional loss. The histological sections obtained by Schucknecht [8] show atrophy of the stria vascularis in some patients with presbycusis, but proof of a causative role for ischemia is lacking. Abrupt sensorineural hearing loss was associated with slow blood flow in the vertebrobasilar system in humans [9]. Typical ischemia-related hearing loss occurs after pontocerebellar tumor surgery with preservation of the auditory nerve and cochlea.

Advances in the etiological diagnosis of sensorineural hearing loss have been elusive, because preserving the entire cochlear vasculature in anatomic preparations is a daunting task. The cochlea receives its blood supply from a tight network of extremely slender vessels embedded in the highly compact bone of the otic capsule. As discussed below, clinicopathological correlations in ischemia-induced hearing loss remain unproven. A prerequisite to further progress is the development of a reliable tool for measuring cochlear blood flow without inducing cochlear damage.

### Cochlear micromechanics: outer hair cell feedback loop and otoacoustic emissions.

Knowledge of the function of the organ of Corti is important to a good understanding of the key role played by the cochlear blood supply in auditory function. Studies by von Békésy established that the lamina basilaris, on which the organ of Corti sits, vibrates from the base to the apex of the cochlea in response to sound. This vibration propagates along the lamina basilaris, stopping roughly at the site that matches the frequency of the initial sound stimulus. At this cut-off point, resonance occurs between the sound stimulus and the lamina basilaris, which substantially amplifies the vibration of the lamina basilaris.

This simple physical match between the sound input and the lamina basilaris is not sufficient to explain the accuracy with which the cochlea deciphers the frequency, rhythm, and intensity of sound stimuli [10]. Cochlear sound processing is not proportional to sound intensity: the cochlea can produce massive amplification of a very soft sound (characterized by a vibration no greater than that associated with thermal acceleration, for instance) or minimal amplification of a loud sound. As early as 1948, Gold hypothesized that this lack of proportionality indicated the existen-

## Cochlear blood supply

---

ce of a cochlear feedback loop [11]. Thus, Gold introduced the concept of a cochlear amplifier. Since then, studies have shown that, in addition to resonance at the cut-off point on the lamina basilaris, considerable amplification of vibrations occurs as a result of changes in the OHCs. When OHCs sense the vibration of the lamina basilaris, they depolarize and contract at their specific frequency [12]. OHCs are arranged from the base to the apex according to their contraction frequency. Force produced by the OHCs adds to the input force, substantially amplifying the vibration of the lamina basilaris at the cut-off point (Figure 1). At sites closer to the base, in contrast, the vibration is blunted. Although the cochlear feedback loop is dependent on the OHCs, the work produced by the OHCs must be amplified by the endolymphatic potential to yield a meaningful effect. Knowledge of the steps involved in OHC function is needed to understand the key role for the cochlear blood supply. Vibration of the lamina basilaris causes a shearing movement of the OHC stereocilia, thereby opening potassium channels. The resulting potassium inflow induces cell membrane depolarization, which in turn causes cell contraction that affects the lamina basilaris. This mechanism is possible because the stria vascularis secretes an enormous amount of potassium into the endolymph. The high potassium concentration generates the endolymphatic potential (about 90 mV) that allows the depolarizing potassium current to occur. If the highly specialized capillary network in the stria vascularis is not supplied with blood, the endolymphatic potential drops and the potassium inflow becomes minimal. Thus, cochlear ischemia is followed within a few seconds by dramatic impairment of cochlear function [1,3,5]. In the longer term, an inadequate supply of oxygenated blood impairs cochlear function, most notably by affecting local homeostasis. Alterations in the turnover of microcilia components would probably occur [13].

Otoacoustic emissions are simply energy generated by the OHCs when function of the organ of Corti is preserved. Ischemia [3-5] or furosemide [14-15] causes stria vascularis dysfunction, thereby inducing a drop in otoacoustic emissions. Otoacoustic emission recording may be the best available tool for the noninvasive investigation of cochlear blood supply. Distortion-product otoacoustic emissions have proved extremely reliable for detecting variations in cochlear blood flow in animals [3-5] and seem very promising in humans requiring pontocerebellar surgery [16].

*In sum*, adequate cochlear blood supply is essential to ensure optimal function of the OHC feedback loop, not only by permitting aerobic function and maintaining the homeostasis of the organ of Corti, but also by feeding the stria vascularis.

### Arterial blood supply

The arterial blood supply to the cochlea was first described in Europe, most notably in Germany by Siebenmann in 1894, and in Japan by Nabeya [17]. Other studies have provided detailed information on the gross [18-19] and microscopic [20-21] characteristics of cochlear arteries.

The cochlea and the vestibule are supplied by arteries from the same source, namely, the internal auditory artery (labyrinthine artery or arteria labyrinthi). The internal auditory artery usually arises from the middle cerebral artery (arteria cerebelli inferior anterior), a branch of the basilar artery (arteria basilaris); in some individuals, it arises directly from the basilar artery. There may be two internal auditory arteries; indeed, this variant was found in nearly half the individuals included in an autopsy study [19]. The internal auditory artery arises from the meatal loop of the middle cerebral artery, which is consistently present and penetrates more or less deeply within the internal acoustic meatus (Figure 2). The meatal loop usually sits on the cochlear nerve (Figure 3) and is often sandwiched between this nerve and the facial nerve.

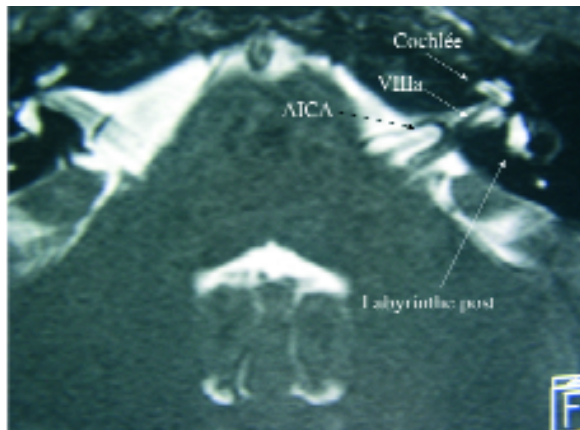
It also gives off the subarcuate artery (arteria subarcuata), which runs in the petromastoid canal, passing through the arch of the superior semicircular canal. The subarcuate artery does not give any branches to the labyrinth; in some individuals, however, the meatal loop gives off a trunk that divides into the subarcuate artery and internal auditory artery. Furthermore, the subarcuate artery may supply branches to the cerebellum. Therefore, every effort should be made to preserve the proximal subarcuate artery when removing pontocerebellar tumors, in particular to increase the changes of hearing preservation. In contrast, the subarcuate artery can be safely clipped or coagulated posterior to the porus acusticus internus, at the point of entry into the petromastoid canal.

The internal auditory artery usually arises from the apex of the meatal loop. It runs along the upper aspect of the cochlear nerve toward the fundus of the internal acoustic meatus. In about 10% of individuals, it sits on the floor of the internal acoustic meatus [19]. The portion of the internal auditory artery located within

## Cochlear blood supply

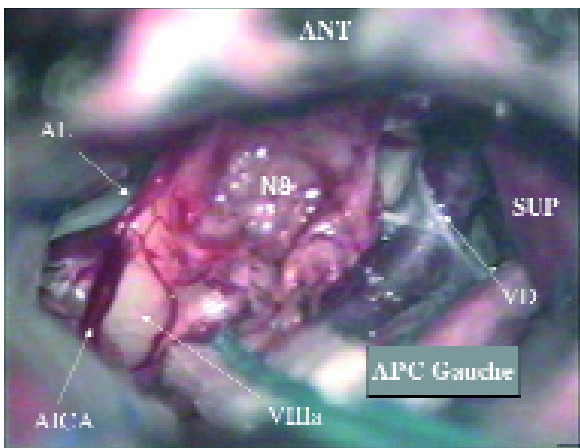
**Figure 2: Magnetic resonance imaging, section through the pontocerebellar angles, T2-weighted CISS sequence.**

The auditory nerve (VIIIa) is readily identified in the internal acoustic meatus. Here, the meatal loop of the middle cerebral artery penetrates along the proximal third of the internal acoustic meatus.



**Figure 3: Intraoperative view of the left pontocerebellar angle at the end of surgery to remove an auditory nerve neurinoma (ANN) via the retrosigmoid approach.**

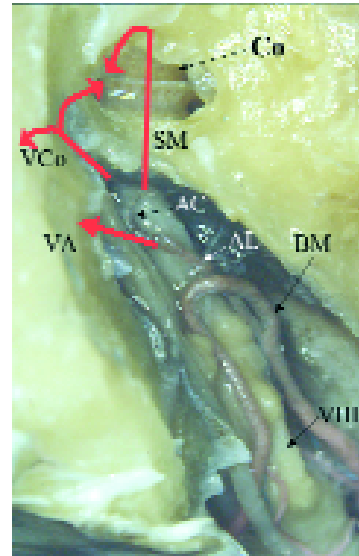
The meatal loop of the middle cerebral artery is easily identified on the auditory nerve (VIIIa). The internal auditory artery (IAA) arises from the apex of the loop. SCV: superior cerebellar vein.



the meatus gives off several branches, which have been described in detail [18-19, 22-23].

The first branch is the anterior vestibular artery (arteria vestibularis anterior), which supplies the posterior and lateral semicircular canals, the utricle, and the posterior part of the saccule. The cochlea is supplied

**Figure 4: Branches from the internal auditory artery.**



CA: cochlear artery; IAA: internal auditory artery; ML: meatal loop; Co: cochlea; SMA: spiral modiolar artery; AVA: anterior vestibular artery; VCoA: vestibulocochlear artery; VIII: auditory nerve. The facial nerve has been resected.

by the spiral modiolar artery (arteria spiralis modioli) and vestibulocochlear artery (arteria vestibulocochlearis), which arise from the common cochlear artery (arteria cochlearis). The common cochlear artery stems from the internal auditory artery near the site where the cochlear nerve penetrates into the modiolus; it runs through the modiolus and supplies the apex of the cochlea, the second turn, and part of the basal turn. The vestibulocochlear artery arises after the spiral modiolar artery and travels to the vestibule, where it gives off a vestibular branch and a cochlear branch. The vestibular branch supplies the posterior semicircular canal and the saccule, whereas the cochlear branch feeds the proximal part of the base of the cochlea. Figure 4 shows the distribution of the main branches of the internal auditory artery.

As pointed out by Tange [24], this distribution suggests that the clinical features may vary according to the site of arterial obstruction. Thus, obstruction of the spiral modiolar artery would be expected to cause hearing loss predominating in the low frequencies and obstruction of the vestibulocochlear artery hearing loss predominating in the high frequencies and accompanied with vertigo. In practice, the balance between these two arteries probably varies across individuals, and anastomoses between the two arteries

## Cochlear blood supply

---

may exist. When removing a neurinoma from the auditory nerve, the various arterial branches supplying the cochlea cannot be identified at the fundus. The internal auditory artery, in contrast, is usually identified during microsurgical dissection. This artery has a muscular media [21, 23] and can therefore develop spasm responsible for cochlear dysfunction [25]. Therefore, during microsurgical dissection of neurinoma of the auditory nerve, in addition to preserving the cochlear nerve and vessels, surgeons should take care to prevent arterial spasm.

Within the cochlea, both spiral and radial vessels are found. The arteries are terminal, forming no anastomoses. They have been described in detail in several animal species and in humans [20-21, 26]. Large arteries penetrate into the cochlea via the modiolus. The spiral modiolar artery gives off radial branches to the lateral cochlear wall, including the stria vascularis. As the arteries decrease in size, they lose their muscular layer, so that spasm necessarily causes extensive cochlear ischemia. The capillary network in the stria vascularis is extremely rich at the base of the cochlea, compared to the apex. The physiological and pathological impact of this difference in capillary abundance is unclear. The key role played by the stria vascularis in ensuring proper function of the OHC feedback loop suggests that this loop may be essential to the perception of high-frequency sounds but may be less important for low-pitched sounds.

The stria vascularis consists roughly of three cell layers: the basal layer facing the perilymphatic space, the intermediate layer, and the marginal layer facing the endolymphatic space. The basal cells are held together by tight junctions that make the stria vascularis impermeable to perilymph. Similarly, the intrastrial space is sealed away from the endolymph by tight junctions linking the marginal cells [27]. The stria vascularis is the only structure in the body where blood vessels are isolated by completely leak-proof cell layers. However, cross-layer communication occurs via gap junctions, which allow nutrients and metabolites to travel from the perilymph [28]. The endolymph is probably secreted from the perilymph rather than from blood [29]. Secretion of potassium into the endolymph is ensured primarily by energy-dependent ion pumps coupled to ATPases [30-31]. Cochlear ischemia stops ion pump function nearly instantaneously, inducing a drop in the endolymphatic potential and thereby causing hearing loss. This effect on endolymphatic potential, which is reliably demonstrated by the probe microphone method,

occurs within a few seconds of cochlear blood flow arrest [4]. This exquisite sensitivity of the cochlea to ischemia was reported as early as 1961, by Konishi et al. [32]. The ion pumps in the stria vascularis are also extremely sensitive to loop diuretics. Furosemide overdose impairs the OHC feedback loop as rapidly as does ischemia and therefore causes cochlear dysfunction [14-15].

### Cochlear veins

Venous drainage of the cochlea occurs via the modiolus. Most mammals have a spiral modiolar vein (vena spiralis modioli). In contrast, no main vein is visible among the nerves in the internal acoustic meatus. The venous blood empties either directly into the inferior petrosal sinus (sinus petrosus inferior) or internal jugular vein (vena jugularis interna) or travels through other venous sinuses via the vein of the vestibular or cochlear aqueduct (vena canales endolymphaticus/perilymphaticus) [17,22]. The multiplicity of venous drainage channels probably explains why resection of the internal jugular vein or sigmoid sinus (e.g., during surgery for jugular paraganglioma) does not cause hearing impairment.

### Cochlear lymphatics

Little is known about the cochlear lymphatics, which were long confused with the endo- and perilymphatic system [33]. Lymphoma can arise in the internal acoustic meatus, indicating that lymphoid tissue is present at this site.

### Cochlear blood flow measurement

A tool capable of detecting ischemia-induced hearing loss would be extremely useful in clinical practice. Such a tool would allow physicians to use, and to monitor the effectiveness of, treatments specifically targeted at organ ischemia. In patients with sudden hearing loss, for instance, the distinction between ischemic and nonischemic causes is usually impossible at present. The only exception is neurinoma of the auditory nerve: if the tumor is known, the presence of cochlear ischemia can be inferred from the pattern of otoacoustic emissions. Laser Doppler velocimetry is the least invasive method for measuring

## Cochlear blood supply

---

cochlear blood flow. This method consists in using the Doppler effect to study the speed of a reflected red laser beam directed at the tissue under study [34]. In a given region, higher blood flow is associated with faster blood movement, so that the beam reflected by the erythrocytes shifts toward the blue part of the color spectrum. After calibration, the differences in cochlear blood flow compared to the normal pattern can be determined. Laser Doppler velocimetry allows ready measurement of cochlear blood flow in animal species whose otic capsule opposite the stria vascularis is thin and therefore causes little attenuation of the incident and reflected laser beams. In the Mongolian gerbil, for instance, the cochlear bone is so thin that the stria vascularis can be glimpsed through the otic capsule under the microscope [5]. As a result, cochlear blood flow measurement is highly reliable in this species [3-5]. When the bone is thick, considerable laser beam attenuation occurs. To overcome this problem, the laser Doppler probe can be modified according to bone thickness [35]. A far simpler solution, however, consists in placing the probe opposite the round window so that the laser beams do not need to travel through the bone. This method has proved remarkably effective in rabbits [36] but remains to be studied in humans.

### CONCLUSION

Knowledge of cochlear blood supply helps to understand the pathophysiology of some patterns of sensorineural loss. In practice, the only currently available tool for suspecting ischemia is otoacoustic emission recording, which exists as two variants, standard otoacoustic emission and distortion-product otoacoustic emission recording. Noninvasive methods for measuring cochlear blood flow are urgently needed as a means of improving the effectiveness of treatment for sensorineural hearing loss. At present, neurinoma of the auditory nerve is the only condition in which current data on the anatomy and physiology of the cochlear blood supply translate into clinical applications.

*The authors thank Professor Michel MONDAIN, CHRU of Montpellier, to have handed the picture in Figure 1.*

### REFERENCES

1. Perlman HB, Kimura R, Fernandez C. Experiments on temporary obstruction of the internal auditory artery. *Laryngoscope* 1959; 69: 591-613.
2. Thalmann R, Miyoshi T, Thalmann I. The influence of ischemia upon the energy reserves of inner ear tissues. *Laryngoscope* 1972; 82: 2249-2272.
3. Ren T, Brown NJ, Zhang M, Nuttall AL, Miller JM. A reversible ischemia model in gerbil cochlea. *Hear Res.* 1995; 92: 30-37.
4. Mom T, Avan P, Romand R, Gilain L. Monitoring of functional changes after transient ischemia in gerbil cochlea. *Br Res.* 1997; 751: 20-30.
5. Mom T, Avan P, Bonfils P, Gilain L. A model of cochlear function assessment during reversible ischemia in the Mongolian gerbil. *Brain Res Protocols* 1999; 4: 249-257.
6. Mom T, Bonfils P, Gilain L, Avan P. Origin of cubic difference tones generated by high-intensity stimuli: effect of ischemia and auditory fatigue on the gerbil cochlea. *J Acoust Soc Am.* 2001; 110(3 Pt 1): 1477-1488.
7. Mom T., Telischi FF, Martin GK, Stagner BB, Lonsbury-Martin BL. Vasospasm of the Internal Auditory Artery: Significance in Cerebellopontine-Angle Surgery: *Am J Otol.* 2000; 21: 735-742.
8. Schuknecht HF. *Pathology of the ear.* Harvard Univ Press, Cambridge, MA, USA, 1974. pp 303-317.
9. Yamasoba T, Kikuchi S, Higo R, O'Uchi T, Tokumaru A. Sudden sensorineural hearing loss associated with slow blood flow of the vertebral basilar system. *Ann Otol Rhinol Laryngol.* 1993; 102: 873-877.
10. Patuzzi RB, Robertson D. Tuning in the mammalian cochlea. *Phys Rev.* 1988; 68: 1009-1082.

## Cochlear blood supply

---

11. Gold T. Hearing II. The physical basis of the action of the cochlea. *Proc R Soc Lond. (Biol)* 1948; 135: 492-498.
12. Brownell WE, Bader CR, Bertrand D, de Ribaupierre Y. Evoked mechanical responses of isolated outer hair cells. *Science* 1985; 227: 194-196.
13. Schneider ME, Belyantseva IA, Azevedo RB, Kachar B. Rapid renewal of auditory hair bundles. *Nature* 2002; 418: 837-838.
14. Mills DM, Rubel EW. Variation of distortion product otoacoustic emissions with furosemide injection. *Hear Res.* 1994; 77: 183-199.
15. Martin GK, Jassir D, Stagner BB, Lonsbury-Martin BL. Effects of loop diuretics on the suppression tuning of distortion-product otoacoustic emissions in rabbits. *J Acoust Soc Am.* 1998; 104: 972-983.
16. Mom T, Chazal J, Gilain L, Gabrillargues J, Avan P. Monitoring auditif utilisant les produits de distorsion acoustique au cours d'une chirurgie de l'angle ponto-cérébelleux: à propos d'un cas pilote. *Rev SFORL* 2004; 85: 102-104.
17. Nabeya D. A study in comparative anatomy of the blood-vascular system of the internal ear in Mammalia and in Homo (Japanese) Thèse. Kyoto, 1923.
18. Mercier P. Anatomie chirurgicale de l'angle ponto-cérébelleux. Thèse. Lyon, 1980.
19. Mazzoni A, Hansen CC. Surgical anatomy of the arteries of the internal auditory canal. *Arch Otolaryngol.* 1970; 91: 128-135.
20. Tange RA, Hodde KC. Microvasculature of the stria vascularis in the round window area in the rat. A scanning electron microscopy study. *ORL J Otorhinolaryngol Relat Spec.* 1985; 47: 225-8.
21. Jahn AF, Santos-Sacchi J. Physiology of the ear, Raven Press, New York, 1988. Circulation of the inner ear: I. Comparative study of the vascular anatomy in the mammalian cochlea. Axelsson A, Ryan AF. pp 295-315.
22. Guerrier Y. Anatomie chirurgicale de l'os temporal, de l'oreille et de la base du crâne. La Simarre, Joué-lès-Tours, 1988. pp 200-203.
23. Mom T, Gabrillargues J., Gilain L, Chazal J, Kemeny JL, Vanneuville G. Anatomie du pédicule vasculo-nerveux facio-cochléo-vestibulaire. Intérêt dans la prise en charge thérapeutique des schwannomes vestibulaires. *Neurochirurgie* 2002; 48: 385-397.
24. Tange, R.A.: Vascular inner ear partition: a concept for some forms of sensorineural hearing loss and vertigo. *ORL J Otorhinolaryngol Relat Spec.* 1998; 60: 78-84.
25. Mom T, Telischi FF, Martin GK, Stagner BB, Lonsbury-Martin BL. Vasospasm of the internal auditory artery: significance in cerebellopontine-angle surgery. *Am J Otol.* 2000; 21: 735-742.
26. de Lorenzo AJD. Vascular disorders and hearing defects. University Park Press, Baltimore, 1972. Vascular pattern of the membranous labyrinth: Smith CA. pp 1-18.
27. Jahnke K. The fine structure of freeze-fracture intercellular junctions in the guinea pig inner ear. *Acta Otolaryngol Suppl.* 1975; 336: 1-40.
28. Forge A. Gap junction in the stria vascularis and effects of ethacrinic acid. *Hear Res.* 1984; 13: 189-200.
29. Drescher DG. Auditory Biochemistry. Springfield, USA, 1985. Origin and electrochemical composition of endolymph in the cochlea: Sterkers O. pp 473-487.
30. Van den Abbeele T, Tran Ba Huy P, Teulon J. Modulation by purines of calcium-activated non-selective cation channels in the outer hair cells of the guinea-pig cochlea. *J Physiol. (Lond).* 1996; 494(Pt1): 77-89.
31. Kerr TP, Ross MD, Ernst SA Cellular localization of Na<sup>+</sup>, K<sup>+</sup>-ATPase in the mammalian cochlear duct: significance for cochlear fluid balance. *Am J Otolaryngol.* 1982; 3: 332-338.

## Cochlear blood supply

---

32. Konishi T, Buttler RA, Fernandez C. Effect of anoxia on cochlear potentials. *J Acoust Soc Am.* 1961; 33: 349-356.
33. Rouvière H, Delmas A. Anatomie humaine descriptive, topographique et fonctionnelle. Tome 1-Tête et cou. 12ème édition. Masson, Paris, 1985. pp 429-430.
34. Shepherd AP, Oberg PA. Laser-Doppler blood flowmetry, Kluwer Academic Publishers, Boston, 1990. TSI's LDV blood flowmeter: *Borgos J.* pp 73-92.
35. Asami K, Nakashima T., Morisaki H, Akanabe K, Kuno K, Yanagita N. Effects of hypercapnia on cochlear and cerebral blood flow in rabbits. *ORL J Otorhinolaryngol Relat Spec.* 1995; 57: 239-244.
36. Mom T, Telischi FF, Lonsbury-Martin BL, Martin GK. Measuring cochlear blood flow and distortion product-otoacoustic emissions during reversible cochlear ischemia: a rabbit model: *Hear Res.* 1999; 133: 40-52.