Derleme

Hearing Impairment in Vascular Disorders

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Abstract

Objective: Cochlea is provided with the arterial blood by the labyrinthine artery that is an end-branch of the vertebrobasilar system. Therefore, it is very susceptible to any compromise of the systemic and local circulation. During the last decades, a good proportion of the audio-vestibular disorders, that is previously labelled as "idiopathic" are proved to be of vascular origin through the advances in the study of microcirculation of the inner ear. This study aims to review the relevant literature on the relationship of inner ear pathologies, hearing loss in particular, and local and systemic vascular diseases in order to put together the current knowledge on the subject in a comprehensive manner.

Material and Methods: A literature search have been conducted through "Pubmed" and full texts of relevant literature have been obtained.

Results: A total of 82 landmark publications have been selected and used for the study.

Conclusion: In this literature review, characteristics of cochlear blood flow and its disturbances are summarized with their clinical correspondences as to include recent findings.

Key words: inner ear, cochlea, hearing, vascular

Overview of the Cochlear Blood Supply

Arterial blood supply to the cochlea is provided by a sole vessel of labyrinthine artery. In post-mortem studies, this artery has been found mainly arising from the anterior inferior cerebellar artery (AICA), a branch of the basilar artery in 83 % of the specimens, occasionally from the basilar artery itself (14%), or rarely its posterior inferior cerebellar artery (PICA) branch (1,2). Basilar artery is formed by two merging vertebral arteries (Fig. 1).

Cochlear artery, a branch of the labyrinthine artery, enters internal acoustic canal following the cochlear branch of the VIIIth nerve, and gives off two branches within the cochlea: spiral modiolar artery and vestibulo-cochlear artery. Cochlear veins also follow the same route with the same names as arteries, e.g. spiral modiolar vein and drains into the vein of the cochlear aqueduct.

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At the level of spiral lamina, arterioles divide into fine collaterals in radial fashion forming three distinctive group of capillary network, forming rich anastomoses with each other (Fig. 2) (3). In addition to this brief anatomical description, below given characteristics of the cochlear blood flow (CBF) are also found very important and highly relevant to central auditory or cochlear disturbances of vascular origin:

1. Cochlea is considered as an end-organ in terms of vascularization, as the blood is supplied mainly by a single artery, as it is the case for retina, heart and kidneys. This type of vascularisation makes the organ particularly vulnerable to any circulatory constriction (4). Insufficient cochlear blood supply may also increase susceptibility to noise induced hearing loss and accelerates natural going process of the cochlea (5).
2. There are well developed circulatory compensatory mechanisms for vital organs, which are activated when the oxygen supply is constrained. It has been shown that 30% decrease in hematocrite achieved 75% increase in cerebral blood flow, including of the brainstem, where auditory pathways and nuclei are located. Similar degree of compensation is estimated for the inner ear as well (6).
3. Cochlear blood flow is under the control of autonomic regulation that occurs along the basilar

artery, the anterior inferior cerebellar artery and at the points of cochlear artery where the spiral modiolar artery branches off (7). Autoregulation of CBF has been demonstrated in guinea pigs (8). In this experiment, it was found that when this autoregulatory system is pharmacologically blocked, CBF responds proportionally to the changes in the systemic blood pressure. It has been shown through animal experiments that cochlear microvessels are rich in myofibrils that respond to this autonomic influence. This feature also offers therapeutic possibilities as to improve CBF by extrinsic agents, on the other hand,

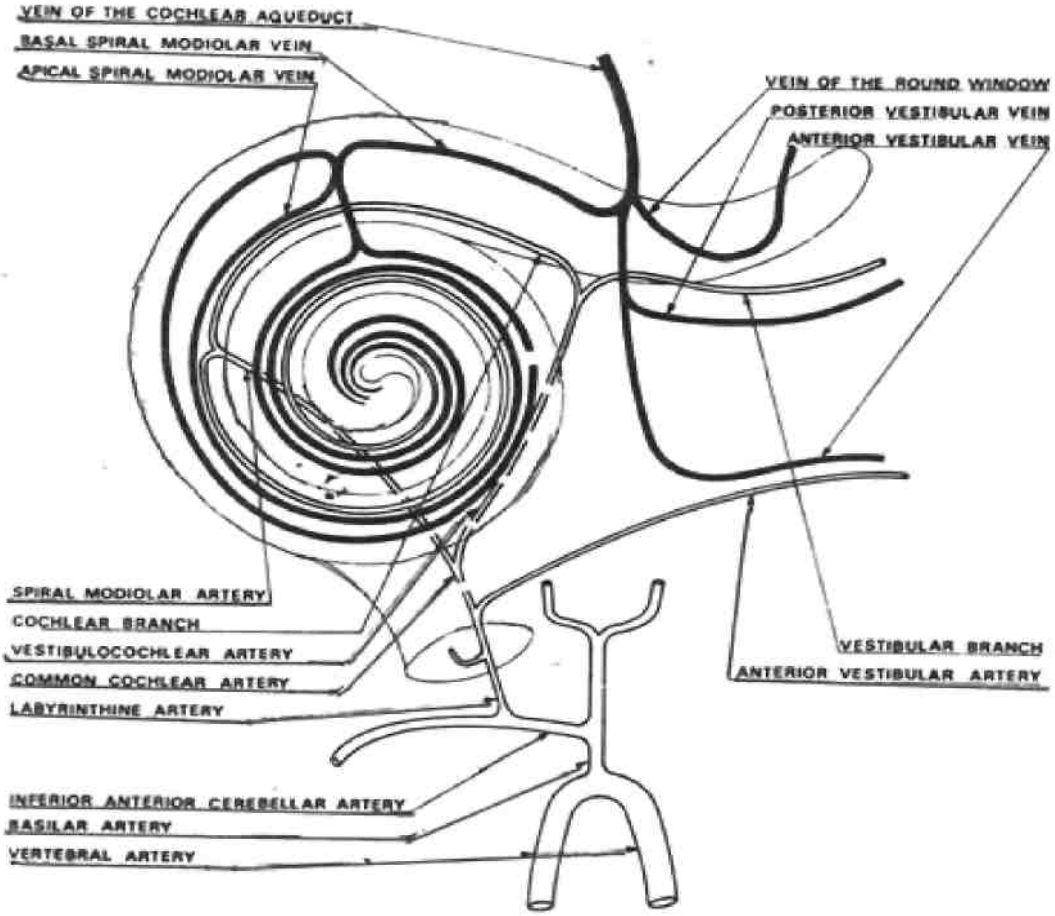

Fig 1. Blood supply to the organ of Corti. a. Spiral Prominence Vessels, b. Scala Vestibuli Vessels c. Stria Vascularis Vessels (From Donaldson J.A. and Duckert L.G. Anatomy of the ear. In Otolaryngology. Eds. Paparella MM, Shumrick DA, Gluckman JL, Meyerhoff WL; third ed., W.B. Saunders; Philadelphia, 1991).

making them susceptible to constrictions under intrinsic and extrinsic influences (9).

4. There is no direct blood supply to the neuro-epithelium of the organ of Corti. It receives oxygen and nutrients either from the spiral prominence vessels underlying basilar membrane or spiral limbus vessels, both branches of the spiral prominence vessels and adjacent to the organ (Fig. 2) (3).

5. Temporal bone histopathologic studies showed that small vessels are coursing freely through the perilymphatic space of the cochlea, especially in the apical turn (10).

6. Following parameters have been identified by the researchers studying (CBF) as the indicatives of significant circulatory disturbances (11,12,13):

a. External artery constriction in the lateral wall,
b. Irregularities within the vessel lumen,
c. Red blood cell (RBC) aggregation,
d. Decrease in RBC velocity,
e. Changes in RBC density.

Investigation Methods of CBF

Vertebro-basilar system is well visualized through conventional arteriography up to the labyrinthine artery. The pathologies in the vessels of vertebrobasilar system as well as resultant ischemia, however, better studied through digital subtraction angiography (DSA), single photon emission computerized tomography (SPECT), magnetic resonance angiography (MRA) and computerized tomographic angiography (CTA) (14, 15). Microcirculation of the labyrinth, on the other hand, is assessed on research bases using videomicroscopy (7) and laser Doppler flowmetry (16, 17). Changes in the CBF have also been found to well correlate with the changes in the outer hear cells (OHC) electromicroscopically (18) and increase in the latency of the distortion product oto-acoustic emissions (DPOAE), both of which indirectly reflect the status of the microcirculation (19).

Local Vascular Diseases Effecting Cochlear Blood Flow

Local primary vascular or other space occupying pathologies may cause compression or stenosis of the main supplying vessels of the labyrinth, such as internal carotid artery, vertebral artery, basilar artery or jugular vein, thereby indirectly effecting labyrinthine circulation without any intrinsic general or systemic vascular pathology of the inner ear. Sometimes, they cause ischemia and infarction of the end organs of cochlea and vestibule.

Experimental occlusion of the cochlear blood vessels themselves in animal model has been demonstrated to reduce the cochlear blood flow by 35%, resulting in drastically reduced cochlear oxygenation and auditory dysfunction that is well documented by means of electrocochleography and auditory brainstem response (ABR) measurements (20).

Reduced blood flow to the inner ear infrequently manifests with sudden hearing loss, either by directly affecting hair cells and/or stria vascularis or the VIII th nerve. In a series of 392 patients with sudden hearing loss, 5% of the cases are attributed to pure vascular causes (21). Conductive deafness due to pathologies of the neighboring vessels has also been reported.

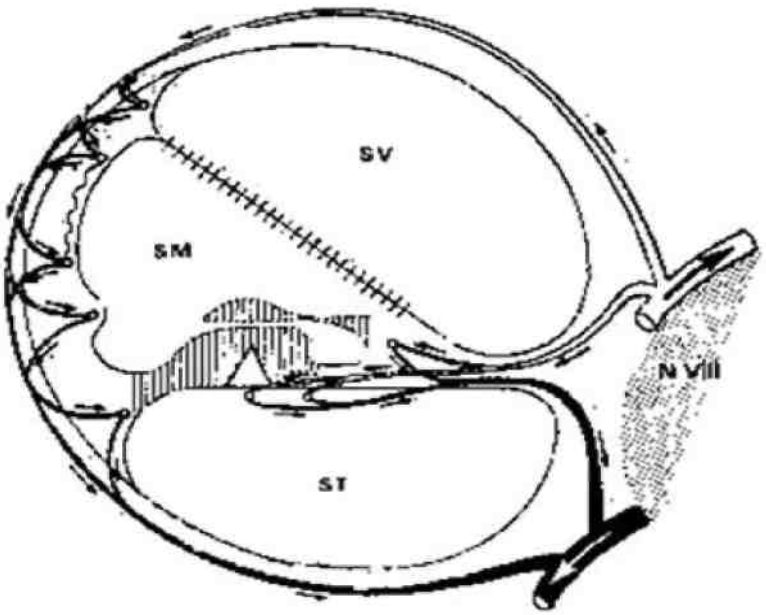

Fig 2. Cross-section of one turn of cochlea. (From Donaldson JA and Duckert LG. Anatomy of the ear in Otolaryngology. Eds. Paparella MM, Shumrick DA, Gluckman JL, Meyerhoff WL; third ed., W.B. Saunders; Philadelphia 1991).

A. Pathologies of the adjacent vessels:

1. Congenital or idiopathic vascular disorders:

a. Aberrant carotid artery within the middle ear as a result of dehiscent hypotympanum may cause variable conductive or mixed hearing loss, however it is a very rare condition (22, 23, 24).

b. Arterial loops of AICA compress the VIIIth nerve on occasions, causing typical cerebello-pontine angle (CPA) mass findings with the signs and symptoms of sensorineural hearing loss (SNHL) of retro-cochlear origin (25, 26). Pulsatile tinnitus is also reported to be one of the hallmarks of this anatomic variation (27).

c. Ectatic vertebral and basilar artery has been shown to mimic CPA tumors, Méniere's disease and other peripheral or central conditions with inner ear symptoms, by compressing brainstem and the VIIIth nerve (26, 28).

d. Aneurysm of AICA may also cause sudden hearing loss, although it is extremely rare, mostly originates from the loops around the internal acoustic meatus (IAM). Less than 50 cases have been reported so far. Signs and symptoms are indistinguishable from the CPA tumors (29, 30).

e. High jugular fossa is usually seen on the right side, encroaches upon labyrinth, effects cochlear aqueduct, vestibular aqueduct and erodes posterior semicircular canal. It typically causes low frequency SNHL as well as occasional conductive hearing loss (31, 32).

f. Greatly enlarged jugular fossa is also situated higher than normal level and accompanied by a giant sigmoid sinus and sometimes with a diverticulum. Symptomatology and aetio-pathogenesis is very similar to those of high jugular fossa. In addition, giant jugular fossa may affect cochlear circulation by causing turbulent flow and decreased venous return (31).

g. Cavernous malformation of the internal acoustic canal (33).

h. Dissection of the vertebral artery (34, 35, 36).

i. Anomalous carotid artery (37).

j. Arterial anomalies of the middle ear associated with stapes ankylosis (38).

k. Aneurysm arising from the petrous portion of the internal carotid artery (39).

I. Infarction of internal auditory artery: Isolated infraction of internal auditory artery has also been reported with the histopathologic correlates of degeneration in the vestibule-cochlear nerve (40).

m. Susac's syndrome is an idiopathic disorder characterized by the triad of encephalopathy, fluctuating hearing loss, and visual loss resulting from microangiopathy of the brain, cochlea, and retina (41).

2. Vascular tumors:

a. Hemangio-endothelioma of the temporal bone (42).

b. Cavernous hemangioma of the IAC (43).

c. Glomus jugulare tumors (paragangliomas) cause conductive hearing loss at early stages and may invade labyrinth, causing SNHL (44).

d. Non-paraganglioma jugular foramen lesions (45).

e. Angiosarcoma of the temporal bone (46).

g. Jugular foramen schwannoma (47).

B. General restrictions of vertebro-basilar system:

1. Thrombosis of AICA:

Thrombosis of AICA affects almost all the structures in the brainstem, including auditory pathways and nuclei at varying degrees as well as the VIIIth nerve and labyrinth itself. Isolated vestibulo-cochlear damage, due to involvement of the cochlear and vestibular nuclei as a result of brainstem infarction also has been reported (48). However, it is more likely that vestibule-cochlear involvement is accompanied by ponto-cerebellar findings such as dysarthria, dysmetria and decreased facial sensation (49). Some cases of AICA infarction may also present with recurrent symptoms that mimic Méniere's disease (50). There is a thrombus formation within the cochlear vessels as well, accompanied by widespread serous fluid collection and inflammatory cell infiltration both within the labyrinth and the myelin sheath of the VIIIth nerve. Sudden hearing loss that usually accompanies the pathology is due to cochlear involvement; whereas late auditory dysfunction tends to be caused by the residual brainstem lesion if the patient survives (51).

2. Vertebro-basilar ischemia is usually caused by atherosclerosis and may lead to sudden hearing loss by labyrinthine infarction with accompanying vertigo that could be bilateral (52, 53, 54). Although very rarely, infarction in the territory of PICA may also be associated with audio-vestibular symptoms (55).

Systemic Cardio-Vascular Diseases

There is a well established correlation between SNHL and systemic cardiovascular diseases. Patients with hearing loss of unknown etiology have been found 8 times more prone to have concomitant ischemic heart disease than their healthy peers. Susmano and Rosenbush

speculated that, this strong correlation may imply a possible genetic defect that is expressed in both coronary and labyrinthine arteries, which are both end-arteries (56). This theory may explain the co-existence of some congenital cardiac diseases with SNHL, such as Jarvell-Lange-Nielsen's Syndrome, Romano-Ward's Syndrome and valvular-pulmoner stenosis. Beyond this hypothetical co-existence, there is a plethora of studies reporting impaired cochlear blood flow and SNHL as a result of cardiovascular diseases. Bachor et al in their post-mortem studies of temporal bones found a statistically significant correlation between the congenital heart defects and abnormalities in the audio-vestibular vessels, which are more pronounced in the cochlea than the vestibule (10).

A. Intrinsic diseases of the cardio-vascular system:

Vertebral giant cell arteritis effects whole vertebro-basilar system including labyrinthine artery, presenting with Méniere-like symptoms (57).

Diabetes mellitus may cause low frequency hearing loss. However, it is argued that, the hearing loss seen in insulin-dependant diabetes mellitus patients is more related to peripheric neuropathy rather than microangiopathy as once had been thought (58). Recently, in diabetic rat inner ears, Liu et al have shown upregulation of nitric oxide (NO) that functions in the vascular tone, and increase in the expression of vascular endothelial growth factor (VEGF), that induces angiogenesis and plays a crucial role in diabetic microangiopathies (59).

Common systemic cardio-vascular diseases: Coronary heart disease, intermittent claudicatio and systemic hypertension are all associated with SNHL especially in elderly patients. They more likely accelerate the ongoing cochlear ageing process rather than causing sudden deafness, as well as causing retrocochlear hearing loss and central auditory dysfunction by way of predisposing cerebro-vascular accidents (58). Patients with hypertension showed deterioration of hearing thresholds at 8 kHz and, compared with normotensive subjects, a higher frequency of abnormal otoacoustic emissions (60). Likewise, stress, hyperlipidemia, glucose intolerance, renal apparatus and even gastroenteric diseases with a functional component can attribute to the development of hearing loss by constituting risk factors for systemic cardio-vascular diseases and/or hemodynamic imbalance (61, 62). Nevertheless, there is no obvious cause-effect relationship established between hearing loss and cardio-vascular insufficiencies. That is possibly due to varying degree of compensatory involvement of the above mentioned local auto-regulatory mechanisms.

4. Systemic vasculitis: Any systemic vascular disease that involves small arteries and arterioles can easily affect cochlear vessels. They include a broad spectrum of diseases which are characterized by disseminated blood vessel inflammation in different organs and systems, mostly by various immunopathological mechanisms. Systemic vasculitis usually causes sudden or progressive SNHL or mixed hearing loss by predisposing different forms of otitis media as well (63). Once they affect the inner ear, the damage is usually grave and irreversible. Long-standing systemic arteriolar insufficiencies tend to cause low frequency hearing loss, presumably due to resultant strial atrophy, whereas aforementioned cardio-vascular diseases typically cause or contribute to high frequency hearing loss by hair cell loss.

a. Systemic lupus erythematosis (SLE): Typically causes severe vasculitis in the apical turn of the cochlea. Outer hair cells are lost by ischemic necrosis and microinfarctions. Clinical manifestation is typical of rapidly progressing SNHL (63, 65).

b. Wegener's granulomatosis: Commonly causes otitis media with effusion (35-47%), sometimes with chronic granulomatous changes in the middle ear (64). It may also cause cochlear damage and VIII th nerve damage by directly affecting their arterioles through necrotizing vasculitis (66).

c. *Polyarteritis nodosa (PAN)* tend to cause and more severe inner ear changes, that sometimes leads to infarctions and fibrotic changes and less serious form of OME than Wegener's granulomatosis (65).

d. Cogan's syndrome: Active vasculitis and fibrosis of the medium and small arteries are typical of this immune-mediated idiopathic disease. Audio-vestibular system is frequently involved with associated vertigo, tinnitus and hearing loss (67, 68).

e. Tromboangiitis obliterans may cause sudden hearing loss (52).

f. Systemic sclerosis (Scleroderma): In one particular study, 77% of the cases with systemic sclerosis showed abnormally low hearing levels (69).

B. Changes in the blood pressure:

It has been shown that, orthostatic fall in the blood pressure of the 10 mmHg in elderly may cause a 60% decrease in cerebral blood flow, because local humoral mechanisms are not quick or efficient enough to compensate for the insufficient supply and orthostatic hypotension is known as to cause TTS (temporary threshold shift) by means of cochlear hypoxia (70). Likewise, rapid reduction of the high blood

pressure has also been reported to cause temporary audio-vestibular symptoms (71). However, the relationship between the cochlear blood flow and hypoxia and measured auditory function is very inconsistent and erratic (12,20, 72,73).

Hemorrhagic hypotension has been demonstrated effectively reducing the blood flow in the apical turn of the cochlea in guinea pigs by Tyagi et al (74).

C. Special circulatory conditions:

1. CBF rate: Yamasoba et al found an association between the sudden hearing loss and slow blood flow within the vertebro-basilar system (1). Hypoventilation has also been shown to be associated with the concurrent decrease in the CBF and endocohlear potential, suggesting a cause-effect relationship between these two entities (75). Cochlea is also found more susceptible to ischemia than the VIIIth nerve.

2. Increased blood viscosity also well correlate with hearing loss, adversely effecting RBC velocity and cochlear oxygenation. Incidence of sudden hearing loss in polycythemia and macroglobulinemia is found significantly higher in adults with SNHL than the controls of the same age group (6, 76, 77).

3.Decreased RBC deformability is characteristics of blood dyscrasia such as thalassemia and sickle cell anemia and associated with cochlear hearing loss. Its occurrence has also been shown in upper respiratory tract infections, respiratory diseases,diabetes, smoking and acidosis (6, 78).

4. Effect of noise on CBF has always been an area of controversy. However, there is enough experimental evidence to suggest that, noise at non-physiological or potentially damaging levels, either continuous or intermittent, can produce constrictive effects on cochlear vessels, thus reducing CBF (5,73,79,80). It has also been demonstrated that acoustic overstimulation is capable of indirectly effecting CBF by angiotensin mediated systemic blood pressure elevating effect (81), which is an initial temporary increase of CBF followed by arterial constriction if noise exposure become chronic and continuous. A product of noise exposure, 8- iso-prostaglandin F (2alpha), has also been demonstrated to reduce inner ear blood flow (82).

Conclusions

Vascular disorders, both local and systemic, can cause hearing loss by;

1. Creating a mass that interferes with the sound conduction,

2. Preventing the vessels of the vertebro-basilar system from providing cochlea and auditory pathways with a level of blood supply, that is quantitatively and qualitatively sufficient for their survival and normal functioning.

3. Making cochlear hair cells more susceptible to external effects, such as noise trauma.

Systemic cardio-vascular diseases tend to effect cochlear blood flow by one of several mechanisms, such as increased blood viscosity, thrombo-emboli and vasoconstriction and sometimes more than one of these factors are involved in the process.

Although there are local compensatory mechanisms, activated by ischemic conditions, their efficiency is very variable and reduced by age.

Damarsal Hastalıklarda İşitme Kaybı

Özet

Amaç: Koklea, vertebrobaziller sistemin bir uç dalı olan A. Labyrinthy tarafından tek başına kanlandırılır. Bu nedenle de yerel ve sistemik dolaşımdaki her türlü kısıtlanmaya duyarlıdır.

Son yıllarda iç kulağın mikro dolaşımının incelenmesinde yaşanan gelişmelerle daha önce "idyopatik" olarak sınıflandırılan odyovestibüler hastalıkların önemli bir bölümünün aslında damarsal kaynaklı olduğu anlaşılmıştır. Bu çalışmada, iç kulak patolojileri ve özellikle de işitme kaybı ile yerel ve sistemik damarsal hastalıkların ilişkisi üzerine mevcut literatürün araştırılması ve böylelikle konuyla ilgili güncel bilgilerin anlaşılabilir bir şekilde bir araya getirilmesi amaçlanmıştır.

Gereç ve Yöntem: "Pubmed" aracılığı ile bu konuyla ilgili literatür araştırılmıştır.

Sonuçlar: Mevcut araştırma için nirengi oluşturan 82 önemli çalışma seçilmiş ve kullanılmıştır.

Sonuç: Bu derleme çalışmasında, son bulguları da içerecek şekilde, koklear kan akımının özellikleri ve bozuklukları klinik yansımaları ile özetlenmiştir.

Anahtar kelimeler: iç kulak, koklea, işitme, damarsal

References

1. Yamasoba T, Kikuchi S, O`uchi T, Higo R, Tokumaru A. Sudden sensorineural hearing loss associated with slow blood flow of vertebrobasilar system. Ann Otol Rhinol Laryngol 1993; 102: 873-877.
2. Lownie SP, Parnes LS. Isolated vestibulocochlear dysfunction of central or peripheral origin. Laryngoscope 1991; 101:1339-1342.
3. Donaldson JA. Duckert LG. Anatomy of the ear

in “Otolaryngology”. Eds: Paparella MM, Shumrick DA, Gluckman JL, Meyerhoff WL, third ed. WB. Saunders; Philadelphia, PA 1991:23-58.
4. Sidman JD, Prazma J, Pulver S, Pillsbury HC. Cochlea and heart as end-organs in small vessel disease. Ann Otol Rhinol Laryngol 1988; 97:9-13.
5. Hawkins JE. The role of vasoconstriction in noise induced hearing loss. Ann Otol Rhinol Laryngol 1971; 80:903-913.
6. Hildesheimer M, Bloch F, Muchnik C, Rubinstein M. Blood viscosity and sensorineural hearing loss. Arch Otolaryngol Head Neck Surg 1990; 116: 820-823.
7. Wangemann P, Wonneberger K. Neurogenic regulation of cochlear blood flow occurs along the basilar artery, the anterior inferior cerebellar artery and at branch points of the spiral modiolar artery. Hear Res 2005; 209:91-96.
8. Brown JN, Nuttall AL. Autoregulation of cochlear blood flow in guinea pigs. Am J Physiol 1994 ; 266:458-467.
9. Franz P, Helmreich M, Stach M, Franz-Italon C, Böck P. Distribution of actin and myosin in the cochlear microvascular bed. Acta Otolaryngol 2004; 124:481-485.
10. Bachor E, Selig YK, Jahnke K, Rettinger G, Karmody CS. Vascular variations of the inner ear. Acta Otolaryngol 2001; 121:35-41.
11. Axelsson A and Dengerink H. The effects of noise on histological measures of the cochlear vasculature and red blood cells: A review. Hearing Research 1987; 31:183-192.
12. Prazma J, Vance SG, Bolster ED, Pillsbury HC, Postma SD. Cochlear blood flow, the effect of noise at 60 minutes’ exposure. Arch Otolaryngol Head Neck Surg 1987; 113:36-39.
13. Quirk WS, Seidman MD. Cochlear vascular changes in response to loud noise. Am J Otol 1995; 16:322-325.
14. Krishnan A, Mattox DE, Fountain AJ, Hudgins PA. CT arteriography and venography in pulsatile tinnitus: preliminary results. AJNR Am J Neuroradiol 2006; 27:1635-1638.
15. Koyuncu M, Elhami AR, Akan H, Sahin M, Basoglu T, Simsek M. Investigation of the vertebrobasilar arterial system in vertigo by vestibulocochlear test, SPECT and angiography. Auris Nasus Larynx 2001; 28:23-28.
16. Nakashima T, Naganawa S, Sone M, Tominaga M, Hayashi H, Yamamoto H, et al. Disorders of cochlear blood flow. Brain Res Brain Res Rev 2003; 43:17-28.
17. Iwasaki S, Nagura M, Miyashita H, Umemura K, Hoshino T. Focal damage to cochlear microcirculation measured using a non-contact laser blood flowmeter in guinea pigs. Acta Otolaryngol 1998; 118:666-672.
18. Olszewski J, Chudzik W, Miłoński J, Kuśmierczyk K. Qualitative and quantitative studies in electron microscopy on influence of experimental ischemia of the vertebral arteries on the outer hair cells function in guinea pigs. Acta Otorhinolaryngol Belg 2003; 57:151-154.
19. 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.
20. Scheibe F, Haupt H, Baumgartl H. Effects of experimental cochlear thrombosis on oxygenation and auditory function of the inner ear. Eur Arch Otorhinolaryngol 1997; 254:91-94.
21. Morrison AW. Acute deafness. Br J Hosp Med 1978; 19:237-249.
22. Campbell G, Renner G, Estrem SA. Bilateral aberrant internal carotid arteries. Otolaryngol Head and Neck Surg 1992; 107:124-128.
23. Ciorba A, Bianchini C, Ortore RP, Bovo R, Martini A. Aberrant internal carotid artery in the middle ear: two case reports. B-ENT 2007; 3: 191-194.
24. Windfuhr JP. Aberrant internal carotid artery in the middle ear. Ann Otol Rhinol Laryngol Suppl. 2004; 192:1-16.
25. Parnes LS, Shmotakahara SG, Pelz D, Lee D, Fox AJ. Vascular relationships of the vestibulocochlear nerve on magnetic resonance imaging. Am J Otol 1990; 11:278-281.
26. Odkvist LM, Thuomas KA, Niklasson M. Macrovascular causes underlying otoneurological disturbances. Acta otolaryngol (Stockh) 1994; 115:145-148.
27. Chadha NK, Weiner GM. Vascular loops causing otological symptoms: a systematic review and meta-analysis. Clin Otolaryngol 2008; 33: 5-11.
28. Liu CH, Lin SK, Chang YJ. Cochlear vertebral entrapment syndrome: a case report. Eur J Radiol 2001; 40:147-150.
29. Rinehart R, Harre RG, Roski AR, Dolan KD. Aneurysm of the anterior inferior cerebellar artery producing hearing loss. Ann Otol Laryngol 1992; 101:705-706.
30. Sarkar A, Link MJ. Distal anterior inferior cerebellar artery aneurysm masquerading as a cerebellopontine angle tumor: case report and review of literature. Skull Base 2004; 14:101-106.
31. Good CD, Phelps PD, Lim DP. Case report: greatly enlarged jugular fossa with progressive sensorioneural hearing loss. J Laryngol Otol 1995; 109:350-352.
32. Haupert MS, Madgy DN, Belenky WM, Becker JW. Unilateral conductive hearing loss secondary to high jugular bulb in a pediatric patient. Ear-Nose-Throat J 1997; 76:468-469.
33. Bricoli A, De Micheli E, Gambin R, Alessandrini F, Iuzzolini P. Cavernous malformation of the internal auditory canal. A case report. J Neurosurg Sci 1995; 39:153-158.
34. Strome SE, Hartshorn DO, Carroll WR, Sheard N, Disher MJ. Otologic manifestations of vertebral artery dissection. Otolaryngol Head Neck Surg 1997; 116:234-237.
35. Nagahata M, Hosoya T, Fuse T, Aoyagi M, Yamaguchi K. Arterial dissection of the

vertebrobasilar systems: A possible cause of acute sensorineural hearing loss. Am J Otol 1997; 18: 26-31.
36. Horii A, Okumura K, Kitahara T, Kubo T. Intracranial vertebral artery dissection mimicking acute peripheral vertigo. Acta Otolaryngol 2006; 126:170-173.
37. Synder SO. Unilateral sudden hearing loss as a result of anomalous carotid anatomy. J Vasc Surg 1990; 12:341-344.
38. Pirodda A, Sorrenti G, Marliani AF, Cappello I. Arterial anomalies of the middle ear associated with stapes ankylosis. J Laryngol Otol 1994; 108: 237-239.
39. Umezu H, Seki Y, Aiba T, Kumakawa K. Aneurysm arising from the petrous portion of the internal carotid artery: case report. Radiat Med Med Imaging Radiat Oncol 1993; 11:251-255.
40. Kim JS, Lopez I, DiPatre PL, Liu F, Ishiyama A, Baloh RW. Internal auditory artery infarction: clinicopathologic correlation. Neurology 1999; 52:40-44.
41. Gross M, Banin E, Eliashar R, Ben-Hur T. Susac syndrome. Otol Neurotol 2004; 25: 470-473.
42. Eliashar R, Saah D, Osin P, Sichel JY. Hemangioendothelioma of the temporal bone in a child. Int J Pediatr Otolaryngol 1997; 76:468-469.
43. Pappas DG, Schneiderman TS, Brackmann DE, Simpson LC, Chandra-Sekar B, Sofferman RA. Cavernous hemangiomas of the internal auditory canal. Otolaryngol Head Neck Surg 1989; 101: 27-32.
44. Roland PS, Glasscock III ME. Surgery of the cranial base in "Otolaryngology". Eds: Paparella MM, Shumrick DA, Gluckman JL, Meyerhoff WL. Third ed. Saunders; Philadephia, PA 1991; 1789-1807
45. Megerian CA, McKenna MJ, Nadol JB.Jr. Non-paraganglioma jugular foramen lesions masquerading as glomus jugulare tumors. Am J Otol 1995; 16:94-98.
46. Hindersin S, Schubert O, Cohnen M, Felsberg J, Schipper J, Hoffmann TK. Angiosarcoma of the temporal bone. Laryngorhinootologie 2008; 87: 345-348.
47. Yamakami I, Ono JA. Sigmoid sinus dural arteriovenous malformation resulting from jugular foramen schwannoma--case report. Neurol Med Chir (Tokyo) 1998; 38:43-46.
48. Kido T, Sekitani T, Okinaki Y, Tahara T, Hara H. A case of cerebellar infarction occurred with the 8th cranial nerve symptoms. Auris-Nasus-Larynx (Tokyo) 1994; 21:111-117.
49. Lee H, Kim HJ, Koo JW, Kim JS. Progression of acute cochleovestibulopathy into anterior inferior cerebellar artery infarction. J Neurol Sci 2009; 278: 119-122.
50. Park JH, Kim H, Han HJ. Recurrent audiovestibular disturbance initially mimicking Ménière's disease in a patient with anterior inferior cerebellar infarction. Neurol Sci 2008; 29:359-362.
51. Hinojosa R, Kohut RI: Clinical diagnosis of anterior inferior cerebellar artery trombosis. Ann Otol Rhinol Laryngol 1990; 99:261-271.
52. Baloh R.W. Vestibular disorders due to cerebrovascular disease in "Disorders of the Vestibular System". Eds: Baloh RW, Halmagyi GM. Oxford University Press; New York, N.Y. 1996; 418-429.
53. Lee H, Baloh RW. Sudden deafness in vertebrobasilar ischemia: clinical features, vascular topographical patterns and long-term outcome. J Neurol Sci 2005; 228:99-104.
54. Sauvaget E, Kici S, Petelle B, Kania R, Chabriat H, Herman P, Tran Ba Huy P. Vertebrobasilar occlusive disorders presenting as sudden sensorineural hearing loss. Laryngoscope 2004; 114:327-332.
55. Lee H. Sudden deafness related to posterior circulation infarction in the territory of the nonanterior inferior cerebellar artery: frequency, origin. Eur Neurol 2008; 59:302-306.
56. Susmano A, Rosenbush SW. Hearing loss and ischemic heart disease. Am J Otol 1988; 9:403-408.
57. McKennan KX, Nielsen SL, Watson C, Wiesner K. Meniere's Syndrome: An atypical presentation of giant cell arteritis (Temporal arteritis). Laryngoscope 1993; 103:1103-1107.
58. Gates GA, Cobb JL, D`agostino RB, Wolf PA. The relation of hearing in elderly to the presence of cardiovascular disease and cardiovascular risk factors. Arch Otolarngol Head Neck Surg 1993; 119:156-161.
59. Liu F, Xia M, Xu A. Expression of VEGF, iNOS, and eNOS is increased in cochlea of diabetic rat. Acta Oto-Laryngologica 2008; 128: 1178-1186.
60. Esparza CM, Jáuregui-Renaud K, Morelos CM, Muhl GE, Mendez MN, Carillo NS, et al. Systemic high blood pressure and inner ear dysfunction: a preliminary study. Clin Otolaryngol 2007; 32:173-178.
61. Pirodda A, Brandolini C, Ferri GG, Modugno GC, Esposti DD, Borghi C. Inner ear dysfunction of uncertain origin: a multidisciplinary approach could give something more. Med Hypotheses 2009; 72: 188-189.
62. Axelsson A, Borg E, Hornstrand C. Noise effect on the cochlear vasculature in normotensive and spontaneously hypertensive rats. Acta Otolaryngol 1983; 96:215-225.
63. Yoon TH, Paparella MM, Schachern PA. Systemic vasculitis: a temporal bone histopathologic study. Laryngoscope 1989; 99: 600-609.
64. Per-Lee JH, Parsons R. Vasculitis presenting as otitis media. South Med J 1969; 62:161-165.
65. Rowe-Jones JM, Macallan DC, Sorooshian M. Polyarteritis nodosa presenting as bilateral sudden onset cochleo-vestibular failure in a young woman. J Laryngol Otol 1990; 104:562-564.
66. Yildirim N, Arslanoglu A, Aygun N. Otologic

and leptomeningeal involvements as presenting features in seronegative Wegener granulomatosis. Am J Otolaryngol 2008; 29:147-149.
67. Cheson BD, Bluming AG, Alroy J. Cogan's syndrome: Systemic vasculitis. Am J Med 1977; 65:549-555.
68. Selivanova O, Haxel BR, Mann WJ. Cogan's syndrome: a diagnostic challenge. HNO 2006; 54: 619-623.
69. Amor-Dorado JC, Arias-Nuñez MC, Miranda-Filloy JA, Gonzalez-Juanatey C, Llorca J, Gonzalez-Gay MA. Audiovestibular manifestations in patients with limited systemic sclerosis and centromere protein-B (CENP-B) antibodies. Medicine (Baltimore) 2008; 87:131-141.
70. Hansen S. Postural hypotension--cochleo-vestibular hypoxia--deafness. Acta Otolaryngol (Stockh) 1988; 449:165-169.
71. Chao TK. Sudden sensorineural hearing loss after rapid reduction of blood pressure in malignant hypertension. Ann Otol Rhinol Laryngol 2004; 113:73-75.
72. Misrahy GH, Shinabarger EW, Arnold JE. Changes in cochlear endolymphatic oxygen availability, action potential, and microphonics during and following asphyxia, hypoxia and exposure to loud sounds. J Acoust Soc Am 1958; 30:701-704.
73. Nuttall AL, Hultcrantz E, Lawrence M. Does loud sound influence the intracochlear oxygen tension. Hear Res 1981; 5:286-293.
74. Tyagi I, Tsutomu N, Ito A, Yanagita N. Effect of hemorrhagic hypotension on blood flow to the basal and upper turns of the cochlea. Auris-Nasus-Larynx (Tokyo) 1995; 22:93-95.
75. Yamamoto H, Makimoto K. Sensitivity of the endocochlear potential level to cochlear blood flow during hypoventilation. Ann Otol Rhinol Laryngol 2000; 109:945-951.
76. Hall SJ, McGuigan JA, Rocks MJ. Red blood cell deformability in sudden sensorineural deafness: another aetiology? Clin Otolaryngol 1991; 16:3-7.
77. Browning GG, Gatehous S, Lowe GDO. Blood viscosity as a factor in sensorineural hearing impairment: Lancet 1986; 18:121-123.
78. Burch-Sims GP, Matlock VR. Hearing loss and auditory function in sickle cell disease. J Commun Disord 2005; 38:321-329.
79. Nakai Y, Masutani H. Noise-induced vasoconstriction in the cochlea. Acta Otolaryngol (Stockh) 1988; Suppl.447:23-27.
80. Attanasio G, Buongiorno G, Piccoli F, Mafera B, Cordier A, Barbara M, et al. Laser Doppler measurement of cochlear blood flow changes during conditioning noise exposure. Acta Otolaryngol 2001; 121:465-469.
81. Wright JW, Dengerink HA, Miller JM, Goodwin PC. Potential role of angiotensin II in noise-induced increases in inner ear blood flow. Hearing Research 1985; 17:41-46.
82. Miller JM, Brown JN, Schacht J. 8-iso-prostaglandin F (2alpha), a product of noise exposure, reduces inner ear blood flow. Audiol Neurootol 2003; 8:207-221.