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Link between angiographic extent and severity of coronary artery disease and degree of sensorineural hearing loss

Atherosclerotic coronary artery disease (CAD) is the leading cause of morbidity and mortality around the world. Atherosclerosis is a systemic, chronic, progressive disease that mainly involves medium-sized arteries. Clinically, it can become apparent as ischemic heart disease, cerebrovascular disease, or peripheral arterial disease [1]. Presbycusis, or age-related hearing loss, is the cumulative effect of aging on hearing. Presbycusis is defined as progressive bilateral symmetrical age-related sensorineural hearing loss, with onset during the 3rd or 4th decade of life [2, 3, 4]. The etiology of presbycusis is not fully understood, yet it is thought to be multifactorial. Many authors have asserted that presbycusis has a genetic basis, and there is a general presumption based on clinical observation that presbycusis is an inherited disorder and that genetic factors may influence both the rate and severity of hearing loss [5, 6].

On the other hand, atherosclerosis may diminish the vascularity of the cochlea, thereby reducing its oxygen supply [7]. Hypoperfusion of the cochlea may also lead to sensorineural hearing loss. In a long-term follow-up study that examined elderly individuals, low-frequency hearing loss (low pure-tone average, 0.25–1 kHz) was associated with cardiovascular disease events including CAD and stroke in both genders but more prominently in women [16].

In this study, we aimed to investigate the relationship between the angiographic severity and extent of CAD, which is a surrogate of atherosclerotic burden, and the overall degree of sensorineural hearing impairment, including both hearing loss and speech discrimination.

Patients and methods

Patient selection

In this study, 381 consecutive patients who underwent coronary angiography for symptoms suggesting ischemic heart disease and who had ischemia detected by a noninvasive stress test were screened. Seventy-nine patients were excluded because they met one of the exclusion criteria defined below, and 37 patients were excluded because they did not meet the inclusion criteria. The remaining 265 patients [mean age, 61.5±13.0 years; median age (25th–75th percentile), 59 years (50.5–67)] including 146 male (55.1%) and 119 female subjects (44.9%) constituted the study population. This was a sample of adult patients who may represent the general population. The study protocol was approved by the local ethics committee.

The patients were included in the study if they were 18 years of age or older, if their coronary angiogram was clear enough to enable efficient calculation of the Gensini score, and if they gave consent to participate in the study.

The patients with acute coronary syndrome, cardiomyopathy, congenital heart disease, severe valvular heart disease, a history of previous revascularization (percutaneous transluminal coronary angioplasty or coronary artery by-pass graft surgery) were excluded from the study. From an audiological point of view, patients with a history of exposure to ototoxic drugs or excessive noise, a history of Meniere's disease, acoustic neuroma or otological surgery, and patients with an air-bone gap of more than 20 dB hearing level units (HL) or non-type-A tympanogram or nonstapedial reflex were ex-

Tab. 1 Classification of hearing loss and distribution according to age and sex

	LL	UL	n[a]	Male gender, n (%)	Mean age ± SD	Median age (25th–75th percentile)
Normal hearing group (NHG)	−10	25	89	37 (41.6)	55.4±11.7	56 (48–62.5)
Mild (group I)	26	40	103	55 (53.4)	58.8±11.3	58 (50–67)
Moderate (group II)	41	55	40	28 (70.0)	64.8±9.7	65 (60–70.7)
Moderately severe (group III)	56	70	26	20 (76.9)	58.2±8.9	60 (51.7–64.5)
Severe (group IV)	71	90	7	6 (85.7)	68.9±11.3	69 (57–81)
Profound[b]	91	–	0	–	–	–

LL and *UL* denote lower and upper limits of hearing loss, respectively, in dB(HL) defining each group, *SD* standard deviation. [a] Denotes the number of patients in each group, mean and median ages are expressed as years. [b] As none of the patients in our study had profound hearing loss, no such group was assigned

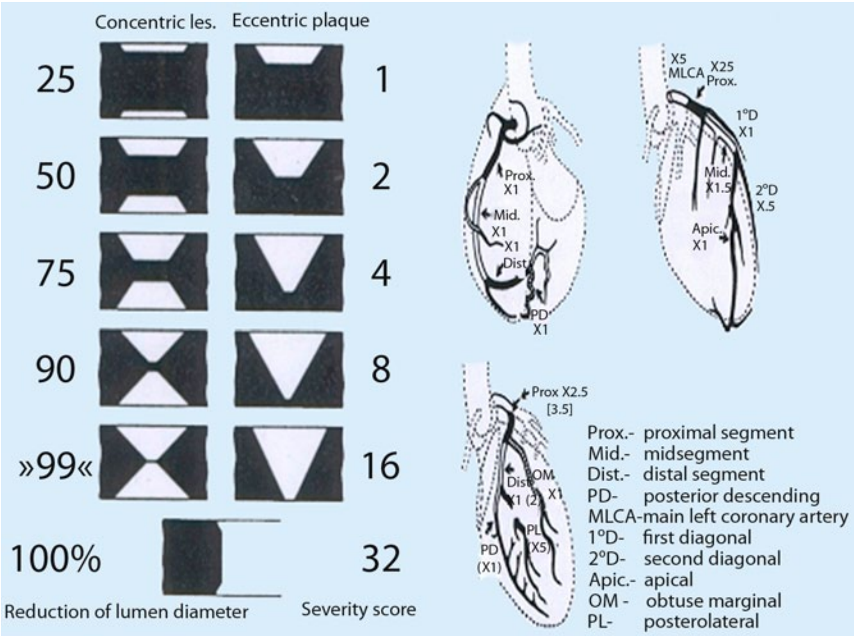

Fig. 1 ▲ Schematic explanation for the calculation of the Gensini score. (With permission from [8])

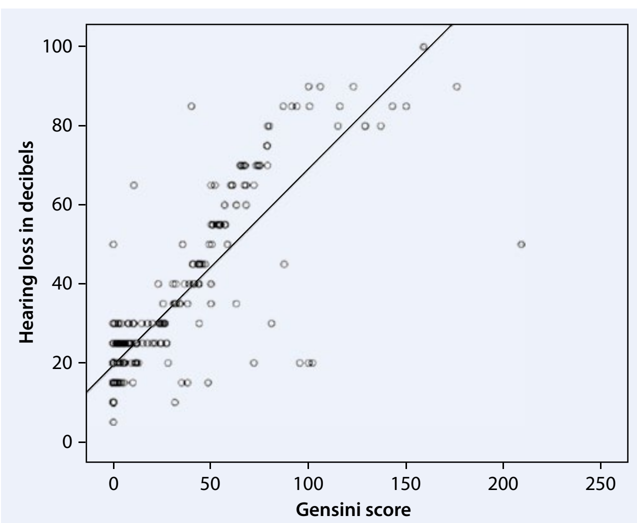

Fig. 2 ◀ Scatterplot depicting the positive correlation between the degree of hearing loss at 2,000 Hz and the Gensini score (for the right ear)

cluded. Patients with asymmetrical hearing loss (a difference in terms of hearing loss of more than 20 dB(HL) between the two ears) were also excluded.

Coronary angiography and assessment of CAD

Selective coronary angiography was performed using the femoral approach employing the Judkins technique and utilizing an Innova angiographic system (General Electric, USA). Multiple views were obtained for all patients, with visualization of the left anterior descending and left circumflex coronaryrtery in at least four projections, and the right coronary artery in at least two projections. Coronary angiograms were recorded in DICOM format. The extent and severity of the CAD were evaluated by using the Gensini score [8]. In this scoring system, a severity score is derived for each coronary stenosis based on the degree of luminal narrowing and its topographic importance. Reductions in luminal diameter of 1–25%, 26–50%, 51–75%, 76–90%, 91–99%, and total occlusion are scored as 1, 2, 4, 8, 16, and 32, respectively. Each principal vascular segment is assigned a multiplier that represents its functional importance in maintaining myocardial blood supply. The multipliers are: 5 for the left main coronary artery; 2.5 for the proximal segment of the left anterior descending coronary artery (LAD) and circumflex artery; 1.5 for the mid-segment of the LAD; 1 for the right coronary artery, the distal segment of the LAD, the posterolateral artery, and the obtuse marginal artery; and 0.5 for other segments (■ **Fig. 1**). Angiographic scoring was performed by two interventional cardiologists who were blinded to audiological measurements and clinical data. The Gensini score for each angiogram was the average of the scores assigned by these two observers. In case of discrepancy between the two observers, the angiogram was rescored. The study population was divided into three groups according to the Gensini score as follows: Gensini score 0, normal coronary arteries; Gensini score between 0 and 20, mild CAD; Gensini score >20, significant CAD [9].

Audiological assessment

All patients who underwent coronary angiography also underwent an audiometric evaluation before the procedure per the study protocol. Hearing impairment was not the presenting symptom in any of the patients. All audiological tests were performed in the Ear, Nose, and Throat Department, Division of Hearing and Speech, by the same audiologist to avoid variation in technique. The audiologist was also blinded to angiographic and clinical data. Detailed hearing assessment included ear, nose, and throat examination, tympanograms, stapedial acoustic reflexes, pure-tone hearing levels, and speech discrimination scores (SDS). An interacoustics AC-40 audiometry device, TDH-49 earphones, and B71 bone vibrator for bone conduction were used in the audiological testing. All subjects underwent air-conduction and bone-conduction pure-tone threshold measurement at 125–8,000-Hz frequencies, and degree of hearing loss was noted in dB(HL). The frequency of 8,000 Hz was excluded from the analysis because hearing loss at this relatively high frequency could be more likely to be age-related, and thus confounding. The degree of hearing loss was classified as previous-

Abstract · Zusammenfassung

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Link between angiographic extent and severity of coronary artery disease and degree of sensorineural hearing loss

Abstract

Aims. Atherosclerosis is a systemic disease that can affect the whole arterial tree. An important cause of neuronal degeneration is atherosclerosis, which may lead to sensorineural hearing loss. We aimed to investigate the relationship between the angiographic severity and extent of coronary artery disease, which is a surrogate of atherosclerotic burden, and the degree of sensorineural hearing loss.

Patients and methods. Out of 381 consecutive patients who underwent coronary angiography for symptoms suggesting ischemic heart disease and who had ischemia detected by a noninvasive stress test, 265 patients [mean age, 61.5±13.0 years; median age (25th–75th percentile), 59 years (50.5–67)], including 146 male (55.1%) subjects met the eligibility criteria and were enrolled. Audiological measurements (hearing levels and discrimination scores) were performed before the coronary angiography. The Gensini score was calculated for each angiogram.

Results. There was a statistically significant positive correlation between the degree of hearing loss at all frequencies analyzed (250, 500, 1,000, 2,000, 4,000 Hz) and the Gensini score ($p<0.05$ for all frequencies), which remained significant after adjustment according to age and other risk factors. A statistically significant negative correlation was observed between the Gensini score and the speech discrimination score ($p<0.05$).

Conclusion. The findings of this study suggest that the angiographic severity and extent of coronary artery disease are significantly and independently correlated with the degree of hearing loss. Sensorineural hearing loss was more prominent in patients with higher Gensini scores. We propose that the findings of this study warrant further research and should be verified in large-scale studies.

Keywords

Atherosclerosis · Coronary artery disease · Sensorineural hearing loss · Neuronal degeneration · Angiography

Beziehung zwischen Ausmaß und Schweregrad der koronaren Herzkrankheit in der Angiographie und Grad der sensorineuralen Schwerhörigkeit

Zusammenfassung

Ziel. Die Atherosklerose ist eine systemische Erkrankung, die alle Arterien erfassen kann. Eine bedeutende Ursache der neuronalen Degeneration ist die Atherosklerose, die zur sensorineuralen Schwerhörigkeit führen kann. Ziel der vorliegenden Studie war es, den Zusammenhang zwischen dem Schweregrad und dem Ausmaß der koronaren Herzkrankheit (KHK), die einen Surrogatparameter der atherosklerotischen Last darstellt, und dem Grad der sensorineuralen Schwerhörigkeit zu untersuchen.

Methoden. Von 381 konsekutiven Patienten, bei denen eine Koronarangiographie wegen Symptomen erfolgte, die den Verdacht auf eine ischämische Herzerkrankung nahelegten, oder wegen einer Ischämie, die mit einem nichtinvasiven Belastungstest festgestellt wurde, erfüllten 265 Patienten [Durchschnittsalter: 61,5±13,0 Jahre, Altersmedian (25.–75. Perzentile): 59 (50,5–67) Jahre], darunter 146 Männer (55,1%), die Auswahlkriterien und wurden in die Studie aufgenommen. Vor der Koronarangiographie wurden audiologische Messungen durchgeführt (Hörschwelle und Wert für Sprachverständlichkeit). Für jede Angiographie wurde der Gensini-Score berechnet.

Ergebnisse. Es bestand eine statistisch signifikante positive Korrelation zwischen dem Grad der Schwerhörigkeit bei allen ausgewerteten Frequenzen (250, 500, 1000, 2000, 4000 Hz) und dem Gensini-Score ($p<0,05$ für alle Frequenzen), die auch nach Berücksichtigung des Alters und anderer Risikofaktoren signifikant blieb. Andererseits wurde eine statistisch signifikante negative Korrelation zwischen dem Gensini-Score und dem Wert für Sprachverständlichkeit festgestellt ($p<0,05$).

Schlussfolgerung. Den Ergebnissen der vorliegenden Studie zufolge sind der Schweregrad und das Ausmaß der KHK signifikant und unabhängig mit dem Grad der Schwerhörigkeit korreliert. Die sensorineurale Schwerhörigkeit war bei Patienten mit einem höheren Gensini-Score deutlicher ausgeprägt. Nach Ansicht der Autoren rechtfertigen die Ergebnisse der Studie weitere Untersuchungen und sollten in großangelegten Studien verifiziert werden.

Schlüsselwörter

Atherosklerose · Koronare Herzkrankheit · Sensorineurale Schwerhörigkeit · Neuronale Degeneration · Angiographie

ly described by Clark et al. [10]. This classification and the distribution of patients according to the degree of hearing loss are summarized in **Tab. 1**.

Speech discrimination is described as a person's ability to not only hear words but to identify them. The procedure involved presentation of 50 selected monosyllabic words at an easily detectable intensity level, which was defined as 40 dB above the pure-tone average for each individual patient. The SDS was defined as the percentage of words correctly identified.

Statistical analysis

Statistical analysis was performed by using SPSS 15.0 for Windows (SPSS, Chicago, Ill., USA). The normal distribution of variables was verified with the Kolmogorov–Smirnov test. The Gensini score and the degree of hearing loss (in decibels) did not display a normal distribution. To demonstrate the relationship between the Gensini score and the audiological measurements, Spearman's rho correlation analysis was performed. Comparisons between the groups were made with Kruskal–Wallis test or the Mann–Whitney U test where appropriate. The χ^2 test was used to investigate whether distributions of categorical variables differ with-

Tab. 2 Biochemical parameters with regard to degree of hearing loss[a]

	Hearing loss	Mean	Median	SD	p
FBG	NHG	110.9	98.7	41.6	0.248
	Mild (group I)	118.4	103.7	50.6	
	Moderate (group II)	107.3	104.3	22.2	
	Moderately severe (group III)	116.3	106.4	35.3	
	Severe (group IV)	131.0	110.9	47.2	
LDL	NHG	132.1	132.0	36.4	0.946
	Mild (group I)	133.5	132.2	42.2	
	Moderate (group II)	127.8	129.9	30.5	
	Moderately severe (group III)	132.9	123.4	39.8	
	Severe (group IV)	128.8	124.7	19.9	
HDL	NHG	47.5	45.9	11.8	0.031
	Mild (group I)	45.4	43.6	11.0	
	Moderate (group II)	41.0	41.7	9.2	
	Moderately severe (group III)	42.8	41.6	10.9	
	Severe (group IV)	38.2	34.8	9.3	
TG	NHG	147.5	129.1	83.4	0.204
	Mild (group I)	161.9	139.1	112.6	
	Moderate (group II)	136.4	123.8	59.7	
	Moderately severe (group III)	181.5	153.2	79.4	
	Severe (group IV)	137.0	124.3	61.6	
TC	NHG	206.5	205.1	43.8	0.343
	Mild (group I)	205.0	205.7	44.4	
	Moderate (group II)	192.5	192.2	37.6	
	Moderately severe (group III)	202.6	184.6	43.5	
	Severe (group IV)	191.7	189.0	20.7	

[a] There is no significant difference between the groups except for HDL cholesterol levels. All biochemical parameters are expressed as mean ± SD, in mg/dl *NHG* normal hearing group, *FBG* fasting blood glucose, *LDL* low-density lipoprotein cholesterol, *HDL* high-density lipoprotein cholesterol, *TG* triglycerides, *TC* total cholesterol, *SD* standard deviation

in groups. Data are shown as mean ± SD and/or median (25th–75th percentile) for continuous variables and absolute numbers (%) for dichotomous variables. A p value less than 0.05 was considered statistically significant.

Results

Baseline characteristics and biochemical and audiological findings

The study population consisted of 265 patients, the mean age of whom was 61.5±13.0 years, and 146 (55.1%) of whom were male.

When the patients were classified according to the degree of hearing loss, it was seen that 89 subjects had normal hearing (normal hearing group, NHG), 103 subjects had mild hearing loss (group I), 40 subjects had moderate hearing loss (group II), 26 subjects had moderately severe hearing loss (group III), and 7 subjects had severe hearing loss (group IV). The audiological definitions of hearing loss and the age and sex distributions of these groups are summarized in **Tab. 1**. The differences between the groups in terms of age and sex were significant (p<0.001 and p=0.001, for age and sex, respectively, see **Tab. 1**).

There was no significant difference between the groups with regard to traditional risk factors for atherosclerotic CAD, namely, diabetes mellitus, hyperlipidemia, hypertension, tobacco use, and family history of premature CAD. As for the biochemical findings, the mean HDL cholesterol level of group IV was significantly lower than that of the NHG and group I (p=0.031). Other biochemical parameters did not significantly differ across the groups (**Tab. 2**).

Angiographic findings and degree of hearing loss

According to the Gensini score, 32.8% of the subjects had normal coronary arteries, 24.5% of the subjects had mild CAD, and 42.7% of them had significant CAD. Comparison of the groups with respect to the Gensini score revealed that CAD became progressively more significant as the hearing status worsened; the NHG had the lowest average Gensini score, and group IV had the highest (mean Gensini scores, 3.63±8.69, 16.75±18.86, 55.76±32.93, 82.79±32.68, and 121.57±44.26 for the NHG, group I, group II, group III, and group IV, respectively, p<0.001).

There was a statistically significant positive correlation between the degree of hearing loss (in decibels), which was calculated according to the pure-tone hearing thresholds for 125, 250, 500, 1,000, 2,000, and 4,000 Hz and the Gensini score (**Tab. 3**). This correlation maintained its significance after adjustment for age and other risk factors. As an example, **Fig. 2** depicts the positive correlation between the degree of hearing loss and the Gensini score at 2,000 Hz for the right ear.

A traditional three-frequency (500, 1,000, and 2,000 Hz) pure-tone average was also obtained in order to assess overall hearing levels. The pure-tone average for 500, 1,000, and 2,000 Hz was positively and significantly correlated with the Gensini score (r=0.713, p<0.001 and r=0.668, p<0.001 for the left and the right ear, respectively).

A low-frequency pure-tone average (125, 500, 1,000 Hz) was also calculated, and it was found that the average hearing loss at these low frequencies was also positively and significantly correlated with the Gensini score (r=0.594, p<0.001 and r=0.358, p<0.001 for the left and the right ear, respectively).

There was a statistically significant negative correlation between the SDS and the Gensini score (r= −0.841, p<0.01 and r= −0.813, p<0.01 for the left and the right ear, respectively). This correlation also maintained its significance after adjustment for age and other risk factors.

Tab. 3 The correlation between the Gensini score and the degree of hearing loss in dB(HL)

Frequency (Hz, for left)		Correlation with Gensini score	Frequency (Hz, for right)		Correlation with Gensini score
125	r	0.141	125	r	0.153
	p	**0.023**		p	**0.013**
250	r	0.159	250	r	0.102
	p	**0.010**		p	0.101
500	r	0.408	500	r	0.321
	p	**0.000**		p	**0.000**
1,000	r	0.750	1000	r	0.205
	p	**0.000**		p	**0.001**
2,000	r	0.828	2000	r	0.853
	p	**0.000**		p	**0.000**
4,000	r	0.806	4000	r	0.868
	p	**0.000**		p	**0.000**

Discussion

Atherosclerosis and sensorineural hearing loss

Atherosclerotic cardiovascular disease is a major health problem around the world. Atherosclerosis is an inflammatory process that may affect the entire arterial tree. 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). Thus, cochlear ischemia is followed almost immediately by hearing loss [11]. According to Fang [12], there is a negative correlation between the diameter of the arterial vessels of the internal auditory meatus and the degree of hearing loss. In addition, the lumen of the vessels reduced more in patients with atherosclerosis than those without this condition, and fibroid thickness was seen in the tunica adventitia [12]. Pertinent to these findings, the data obtained from our study demonstrated that the severity of CAD, which reflects the atherosclerotic burden, is significantly correlated with audiological parameters: Sensorineural hearing impairment was more prominent in patients with higher Gensini scores and vice versa. These findings suggest that there is a relationship between sensorineural hearing impairment and the severity of atherosclerotic CAD, a surrogate of systemic atherosclerosis. One explanation may be that the progress of the vessel changes in patients with presbycusis might be accelerated by atherosclerosis. Atherosclerosis is a systemic disease that may diminish blood supply of the cochlear-neural structures, and this might be a plausible explanation for this relationship. It can be hypothesized that audiological assessments may be predictive of the severity of CAD, or systemic atherosclerosis in general. This hypothesis must be tested further in large-scale studies.

In a recent, population-based, retrospective cohort study [13], diabetic patients were significantly more likely to experience sudden sensorineural hearing loss when compared with nondiabetic age- and sex-matched controls. Of note, comorbidities such as retinopathy and CAD increased the likelihood of sudden sensorineural hearing loss [13]. Ciccone et al. [14] reported an association between vascular endothelial dysfunction and sensorineural hearing loss, suggesting a vascular etiology for this condition.

It has previously been reported in the literature that the audiometric pattern may be an indicator of cardiovascular health status [15, 16, 17]. Of note, in a long-term follow-up study by Gates et al. [16], low-frequency hearing loss (low pure-tone average, 0.25–1 kHz) was associated with cardiovascular disease events including CAD and stroke. In addition to the findings of Gates et al., we found significant correlations of the severity of CAD not only with low-frequency hearing loss, but with overall and relatively high-frequency (i.e., 4,000 Hz) hearing loss, and with speech discrimination as well. We propose that our findings warrant further research and should be verified with large-scale studies.

Study limitations

This study has certain limitations. A major limitation was that the cochlear blood flow measurements using laser Doppler velocimetry were not performed because of cost restrictions. Genetic evaluation for hereditary hearing loss syndromes was also not performed.

Conclusion

In conclusion, the results of this study suggest that the degree of sensorineural hearing loss may be correlated with the severity and extent of atherosclerotic CAD. This may be explained by the fact that atherosclerosis is a diffuse and systemic process that affects the entire arterial tree. Increased atherosclerotic burden, as reflected by a higher Gensini score, may explain the diminished blood supply to the cochlea, eventually leading to sensorineural hearing loss. These findings need to be verified in further research, involving large-scale studies.

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Compliance with ethical guidelines

Conflict of interest. A.F. Erkan, G.K. Beriat, B. Ekici, C. Doğan, S. Kocatürk, and H.F. Töre state that are no conflicts of interest.

All studies on humans described in the present manuscript were carried out with the approval of the responsible ethics committee and in accordance with national law and the Helsinki Declaration of 1975 (in its current, revised form). Informed consent was obtained from all patients included in studies.

References

1. Lahoz C, Mostaza JM (2007) Atherosclerosis as a systemic disease. Rev Esp Cardiol 60:184–195
2. Jennings CR, Jones NS (2001) Presbycusis. J Laryngol Otol 115:171–178
3. Bhatt KA, Liberman MC, Nadol JB (2001) Morphometric analysis of age-related changes in the human basilar membrane. Ann Otol Rhinol Laryngol 110:1147–1153
4. Bovo R, Ciorba A, Martini A (2011) Environmental and genetic factors in age-related hearing impairment. Aging Clin Exp Res 23:3–10
5. Gates GA, Couropmitree NN, Myers RH (1999) Genetic associations in age related hearing thresholds. Arch Otolaryngol Head Neck Surg 125:654–659
6. Huang Q, Tang J (2010) Age-related hearing loss or presbycusis. Eur Arch Otorhinolaryngol 267(8):1179–1191
7. John U, Baumeister SE, Kessler C, Völzke H (2007) Associations of carotid intima-media thickness, tobacco smoking and overweight with hearing disorder in a general population sample. Atherosclerosis 195:144–149
8. Gensini GG (1983) A more meaningful scoring system for determining the severity of coronary heart disease. Am J Cardiol 51:606
9. Oishi Y, Wakatsuki T, Nishikado A et al (2000) Circulating adhesion molecules and severity of coronary atherosclerosis. Coron Artery Dis 11:77–81
10. Clark JG (1981) Uses and abuses of hearing loss classification. ASHA 23:493–500
11. Mom T, Chazal J, Gabrillargues J et al (2005) Cochlear blood supply: an update on anatomy and function. Fr ORL 88:81–88
12. Fang YY (1993) Image analysis of arterial vessels of the internal auditory meatus during presenile and aged with hypertension and atherosclerosis. Zhonghua Er Bi Yan Hou Ke Za Zhi 28(2):91–93, 124
13. Lin SW, Lin YS, Weng SF, Chou CW (2012) Risk of developing sudden sensorineural hearing loss in diabetic patients: a population-based cohort study. Otol Neurotol 33(9):1482–1488
14. Ciccone MM, Cortese F, Pinto M et al (2012) Endothelial function and cardiovascular risk in patients with idiopathic sudden sensorineural hearing loss. Atherosclerosis 225(2):511–516
15. Friedland DR, Cederberg C, Tarima S (2009) Audiometric pattern as a predictor of cardiovascular status: development of a model for assessment of risk. Laryngoscope 119:473–486
16. Gates GA, Cobb JL, D'Agostino RB, Wolf PA (1993) The relation of hearing in the elderly to the presence of cardiovascular disease and cardiovascular risk factors. Arch Otolaryngol Head Neck Surg 119:156–161
17. Liu D (1988) Influence of hypertension and coronary heart disease on the hearing in the aged. Zhonghua Er Bi Yan Hou Ke Za Zhi 23:342–345, 384–385