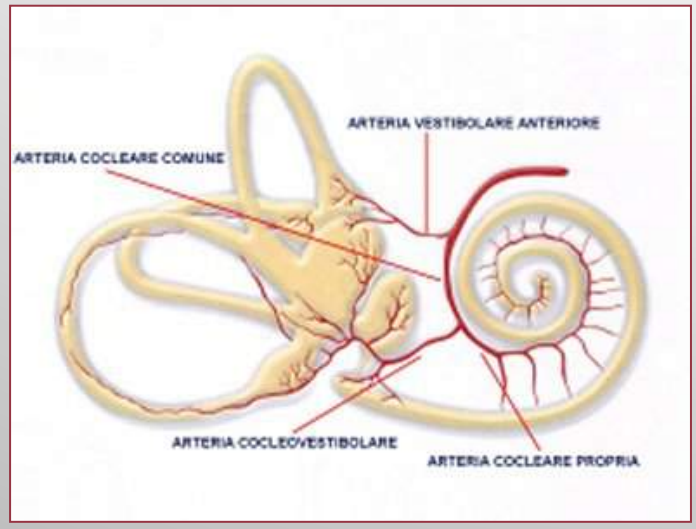
Correlazione tra imaging di gliosi cerebrale e patologie cocleo vestibolari (ipoacusia improvvisa e neurite vestibolare)

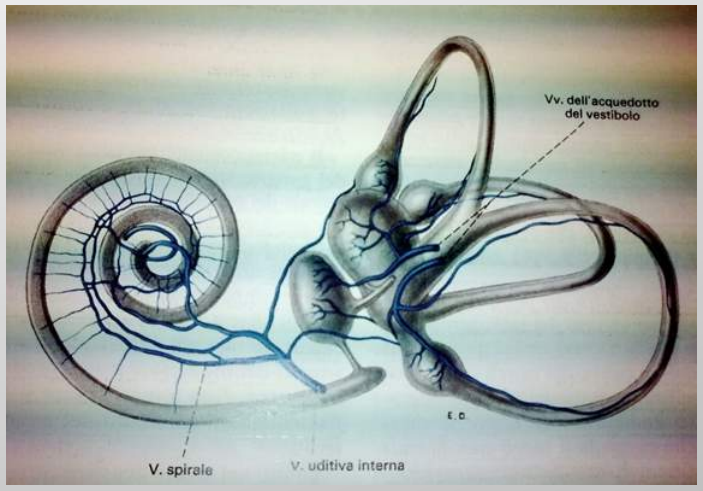
SAPIENZA
UNIVERSITÀ DI ROMA

Facoltà di Medicina e Odontoiatria

Prof. Massimo Fusconi

Circolazione arteriosa orecchio : terminale
Circolazione venosa orecchio : assenza di collaterali





tronco basilare
dell'arteria cerebellare antero-inferiore
dall'arteria uditiva interna

vena uditiva interna
seno petroso superiore o nel seno trasverso.

>150 µ

Acta Otolaryng 73: 259–266, 1972

DEGENERATIVE CHANGES OF THE ARTERIAL VESSELS OF THE INTERNAL AUDITORY MEATUS DURING THE PROCESS OF AGING

A Histological Study

U. Fisch, M. Dobozi and D. Greig

From the Department of Otolaryngology, University of Zürich, Zürich, Switzerland

Abstract. Degenerative changes are observed histologically in the wall of the arterial vessels of the internal auditory meatus following the first decade of life in man. These changes consist mainly in a progressive thickness of the tunica adventitia accompanied by an increase of collagenous tissue and loss of fibroblasts. The increasing compactness of the adventitial layer may lead to complete acellularity and loss of structure (hyalinization). A possible functional implication of the adventitial changes observed in the arteries of the internal auditory meatus of the aging men may be a difficulty in contractibility related to the increasing amount of collagenous or hyaline tissue present in their walls.

The histological changes occurring during the aging process in the arterial system of the internal auditory canal have not yet been specifically investigated. Bosatra (1956) described minimal quantitative and qualitative modifications (particularly subendothelial hyperplasia of the tunica intima and hypertrophy of the internal elastic membrane) in the wall of the "internal auditory artery" of the aging man. The vascular segment investigated by this author was, however, removed outside of the internal auditory meatus and according to its position, ramification and size, corresponds to a branch of the anterior inferior cerebellar artery only partly involved in the blood supply of the internal meatus itself.[1] The purpose of this investigation was to describe the histological structure of the arterial vessels of the internal auditory meatus during the different decades of life. Particular attention was paid to those vessels carrying the blood to the inner ear.

[1] In order to avoid confusion, the official anatomical nomenclature has discarded the name of "internal auditory artery" as early as 1955 (Fisch, 1969).

MATERIAL AND METHOD

78 temporal bones without apparent pathology, other than the changes imputable to the age of the patients, were selected from temporal bones collection of the E.N.T. Department of the University of Zurich. The age of the patients ranged from 2 weeks to 88 years. The distribution of the specimens according to the decades of life is shown in Table I. On each specimen representative sections of three categories of small arteries *lying* in the internal auditory meatus were taken (Fig. 1):

(*a*) Vessels with an average outer diameter of 150 microns, corresponding to the labyrinthine artery or arteries.

(*b*) Vessels with an average outer diameter of 100 microns, corresponding to the arteria cochleae propria or arteria vestibuli anterior.

(*c*) Vessels with an average outer diameter of 60 microns, corresponding to the Vasa nervorum, suppling the VIII nerve.

The diameter of the vessels as well as the thickness of their vascular layers were measured using a Zeiss screw micrometer ocular. The investigated sections were stained with

<150 µ

(a) Vasi con un diametro esterno medio di **150 µ** , corrispondente ad **a. uditiva.**

(b) Vasi con un diametro esterno medio di **100 µ ,** corrispondente **all'a. cocleae propria** o **a. vestibolare anteriore.**

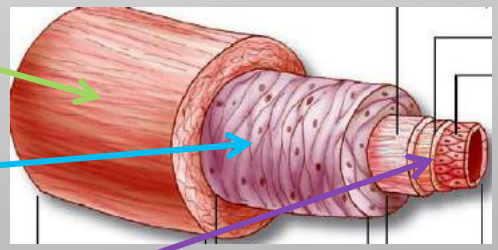
(c) Vasi con un diametro esterno medio di **60 µ** , corrispondente al vasi nervorum

150 µ fibre muscolari + connettivo - VS ai vasi extracranici
Inoltre membrana elastica esterna manca

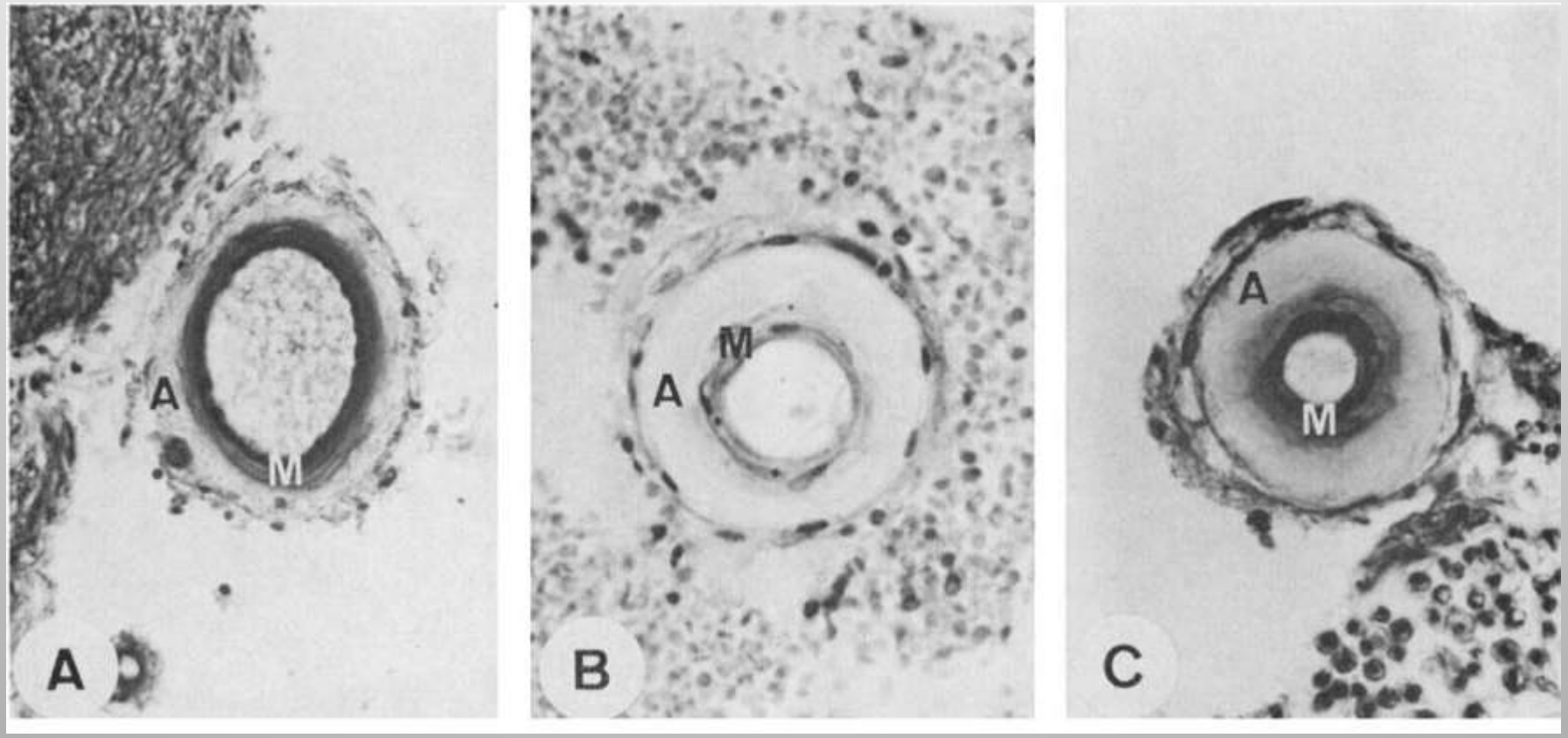
La tunica avventizia
tessuto connettivo di dimensione variabile, con pochi fibroblasti
strato sottile di tessuto aracnoidale

La tunica media
2-8 strati disposti in modo circolare di fibre muscolari occasionale tessuto collageno

La tunica intima
strato endoteliale circondato
membrana elastica

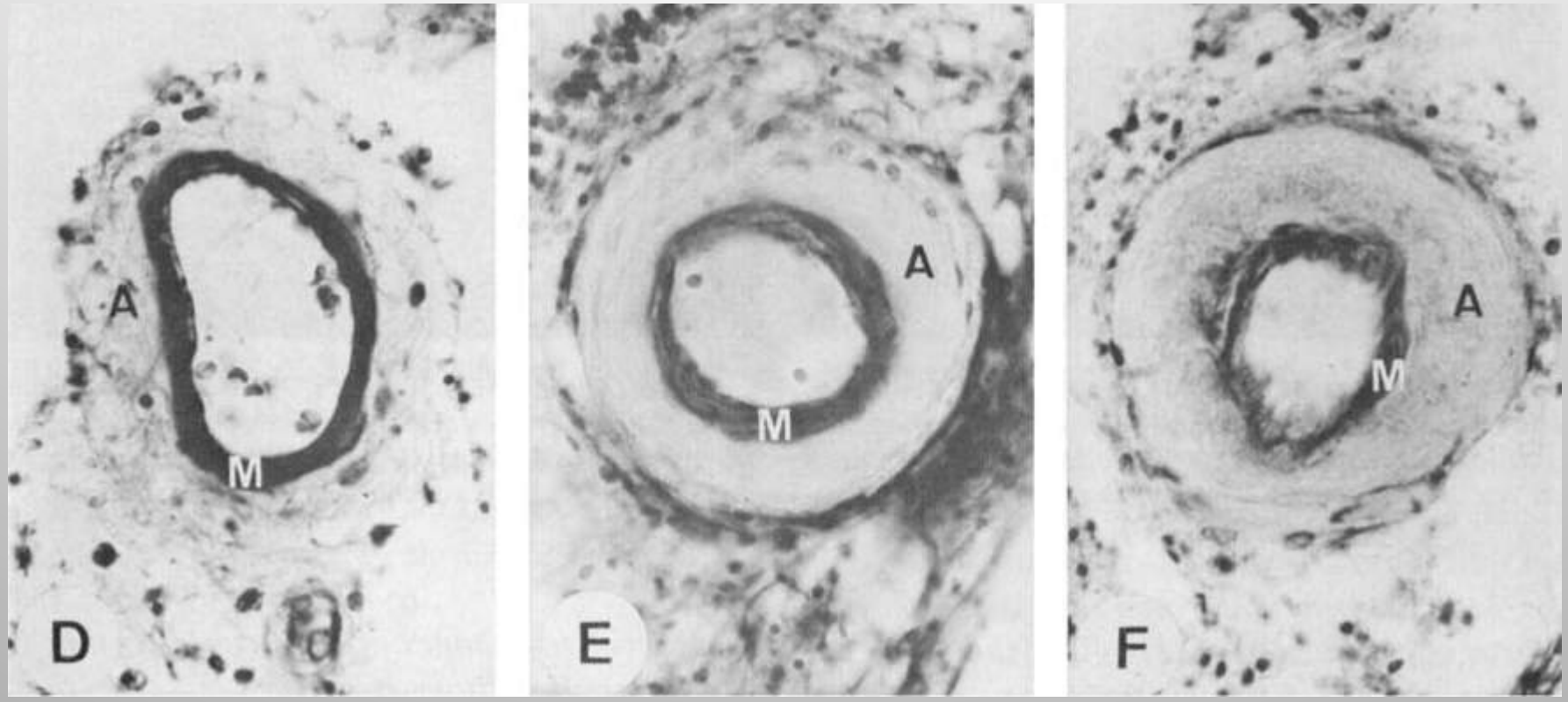


60 µ



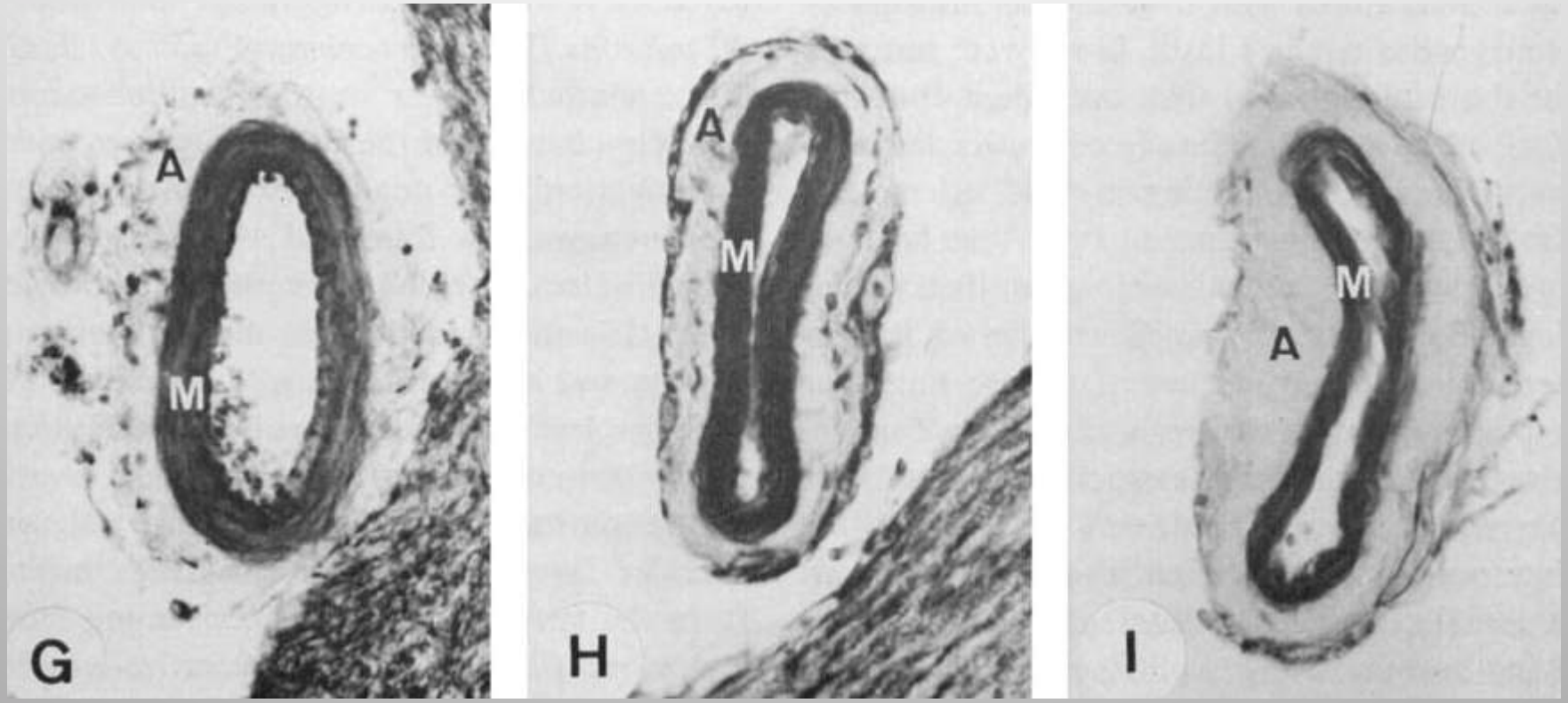
Il processo degenerativo il tessuto avventizio è accompagnato da un perdita progressiva di fibroblasti e può infine portare a una perdita totale di struttura (**ialinizzazione**).
Calibro può mostrare una forte riduzione con l'aumentare età ma un'occlusione totale di questi vasi è stato osservato raramente.

100 µ



I più sorprendenti cambiamenti sono nell'tunica avventizia. Lo spessore di questo strato inizia ad aumentare dopo il primo decennio di vita e dopo 60 anni può raggiungere il 60-70%
l lume dei vasi, in contrasto con quello del Vasa nervorum, non è influenzato da i cambiamenti strutturali relativi all'età dei pazienti.

150 µ



cambiamenti degenerativi nell'avventizia ma meno importanti dei vasi da 60 e 100 **µ**
Tonaca muscolare lievemente ridotta dall'età

cambiamento che si verifica con l'età nei vasi arteriosi nel canale uditivo interno.

1

Una progressiva degenerazione della tunica avventizia:

•Aumento spessore e compattezza dell'avventizia

•perdita progressiva di fibroblasti,

•perdita di struttura (ializzazione).

2

Simile modello degenerativo in vasi da 60 – 100 -150 µ

i vasi più piccoli coinvolto prima e in misura maggiore.

3

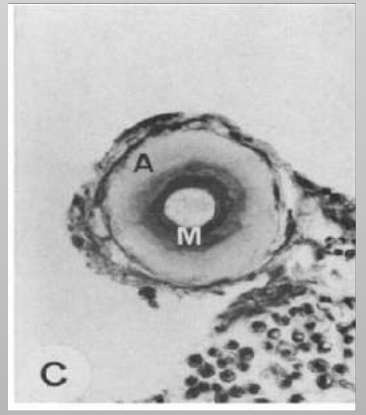
Di conseguenza, una **riduzione del lume** è stato osservato solo per età nei vasi da **60 µ**

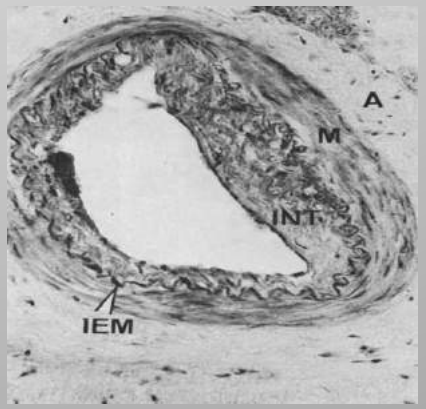
4

Una possibile spiegazione: il particolare stress meccanico a cui viene esposta la tunica avventizia durante contrazione dei vasi.

5

vasi arteriosi di dimensioni simili nella regione del **ganglio genicolato (rami del stylomastoide o arteria petrosa)** : una grave riduzione del lume a causa di un ispessimento della tunica intima, è stato riscontrato che si verifica con l'aumentare dell'età .



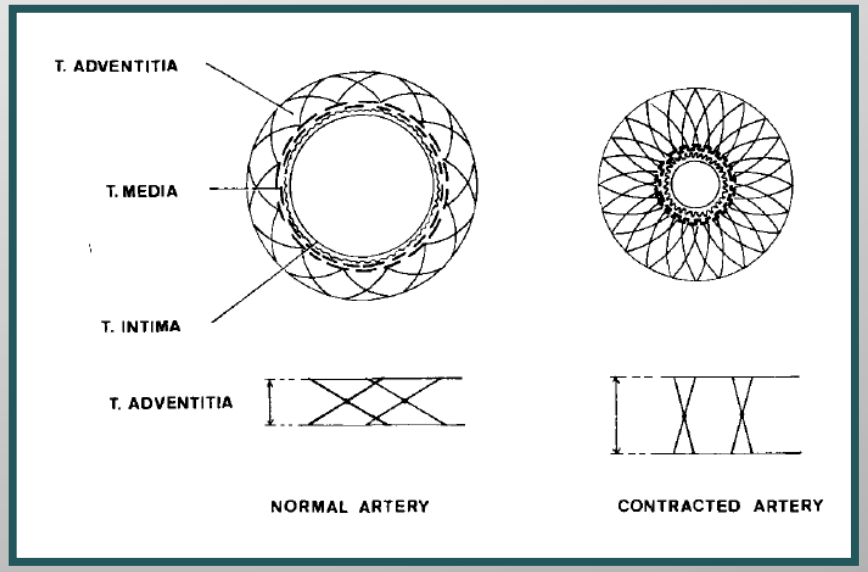


cambiamento che si verifica con l'età nei vasi arteriosi nel canale uditivo interno.

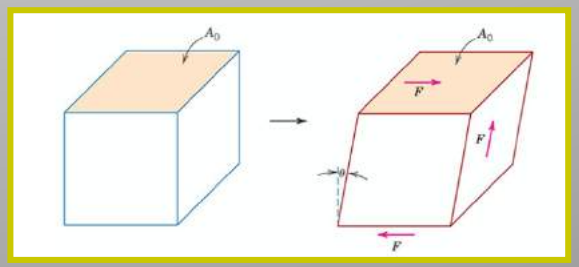
Parete delle arterie più piccole del **polmone**
Hayek (1952)

arterie **intracerebrali** più piccole
Wolkoff (1933) e Baker & Iannone (1959)

Arterie **condotto uditivo interno**
(Fisch 1972)



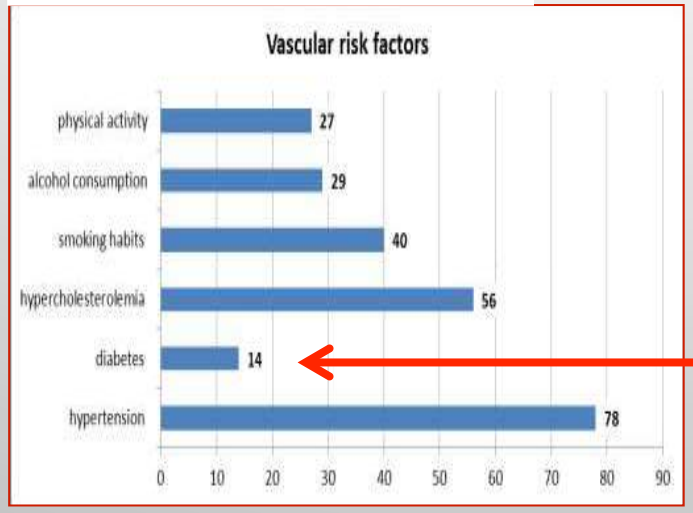
il microcircolo va incontro a degenerazione ialina
tipico risultato dello **shear stress**.



LEUCOARAIOSI o malattia dei piccoli vasi (SVD)

aumentati livelli plasmatici di fibrinogeno

FATTORI DI RISCHIO

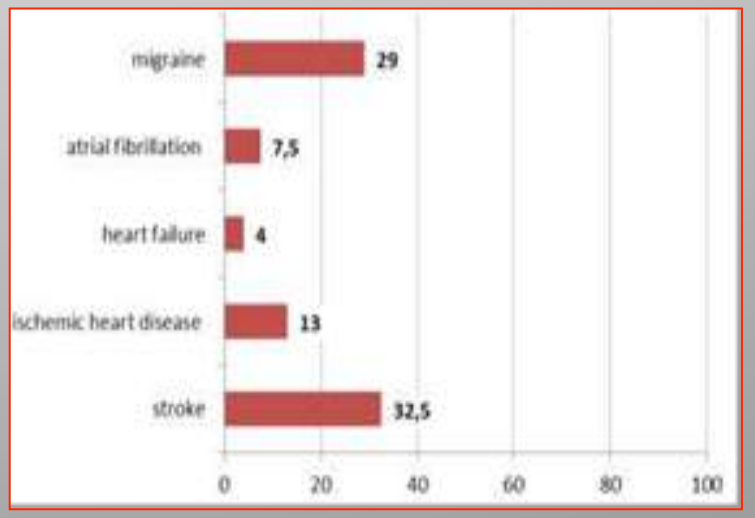


L' encefalo, (solo il 2% del peso corporeo),

riceve il 15% della gittata cardiaca (in condizioni di riposo, e a qualunque età)

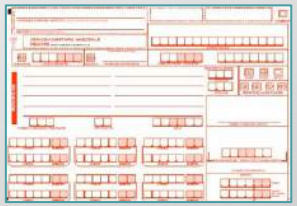
responsabile del 20% del consumo di ossigeno

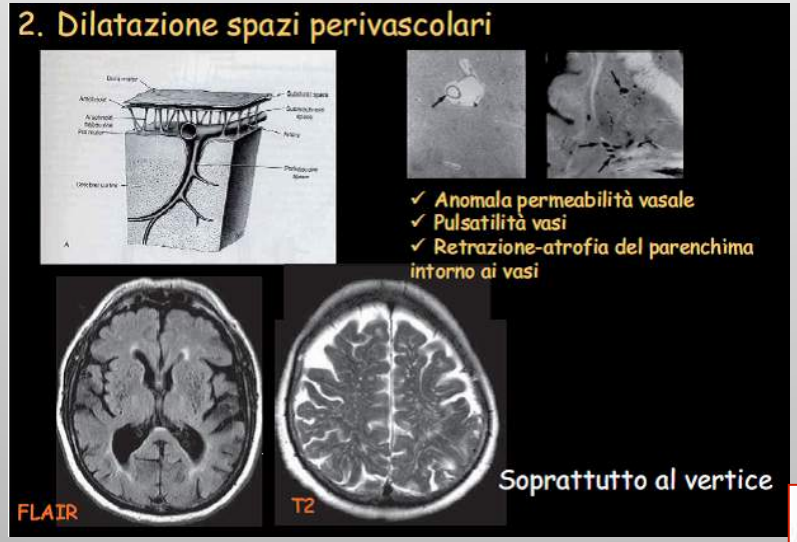
Il cervello riconosce solo la aerobiosi



LEUCOARAIOSI o malattia dei piccoli vasi (SVD)

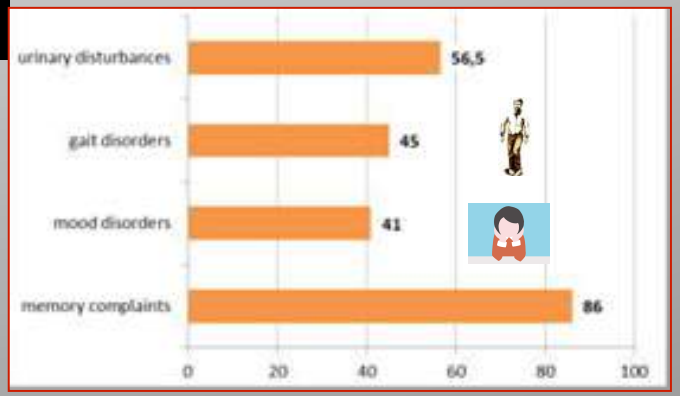
lesioni localizzate:
nel sottocorticale del cervello
o più profonde come la sostanza periventricolare;





ne conseguono infarti lacunari, lesioni della sostanza bianca, grandi emorragie o micro-emorragie

CARATTERISTICHE PERSONALI



LEUCOARAIOSI o malattia dei piccoli vasi (SVD)

Il Rotterdam Study (F-E de Leeuw 2001) ha stabilito:

soggetti 60-70 anni

13% **non presenta** lesioni della sostanza bianca sottocorticale

32% **non presenta** lesioni della sostanza bianca periventricolare.

soggetti 80-90 anni

0% **non presenta** lesioni della sostanza bianca sottocorticale

5%, **non presenta** lesioni della sostanza bianca periventricolare

le lesioni sottocorticali e periventricolari aumentano, dello 0,2% e 0,4% per anno, rispettivamente.

LEUCOARAIOSI o malattia dei piccoli vasi (SVD)

- Si consideri che per gli studi sulla SVD solitamente vengono arruolati pazienti di oltre 60 anni ritenendo una sua manifestazione prima di questa età poco frequente.

- Utilizzando la TC o la RM, tali lesioni sono state riscontrate solo nel 22% dei soggetti con età inferiore a 40 anni.

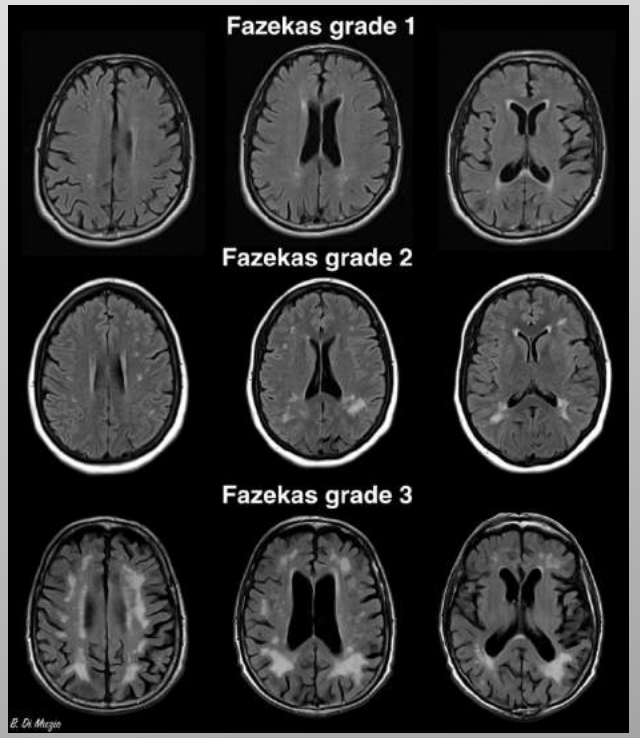
Scala di Fazekas

La scala di Fazekas è usata per misurare la gravità della malattia della sostanza bianca, visibile mediante RM con immagini pesate in T_2 FLAIR.

In particolare si distinguono due tipi di lesioni della sostanza bianca con 3 gradi relativi:

- Sostanza bianca periventricolare
  - 0= assente
  - 1= lesioni puntiformi, lesioni a punta di matita
  - 2 = lesione ad aloni lisci
  - 3= lesione ad alone irregolare periventricolare che si estende alla sostanza bianca

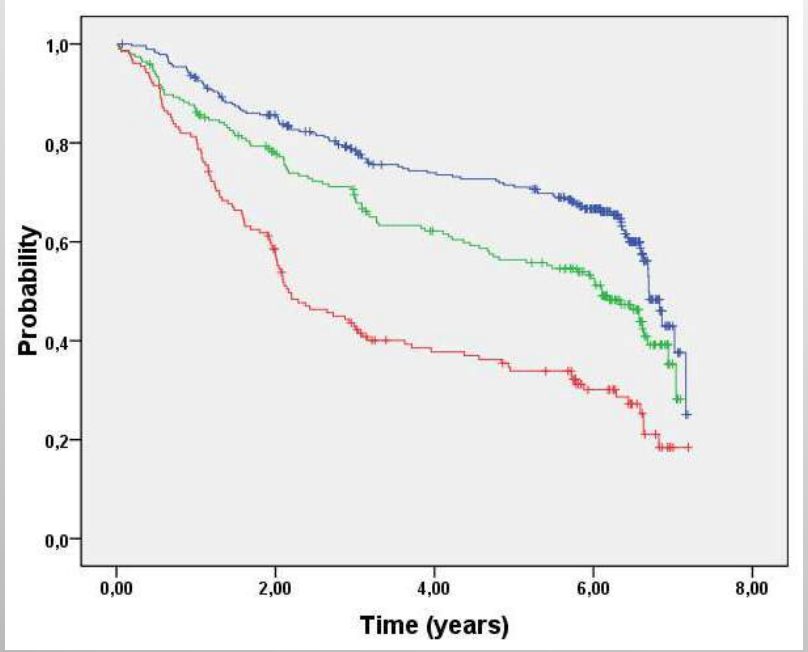
- Sostanza bianca profonda
  - 0 = assente
  - 1 = foci puntiformi
  - 2 = inizio confluenza
  - 3= grandi aree confluenti



Scala di Fazekas

- Fazekas I è considerato normale dopo i 60 anni.
- Fazekas II è considerato anormale nei pazienti <75 anni.
- Fazekas III è anormale in qualsiasi fascia di età e sta ad indicare scarso controllo dei fattori di rischio

Scala di Fazekas



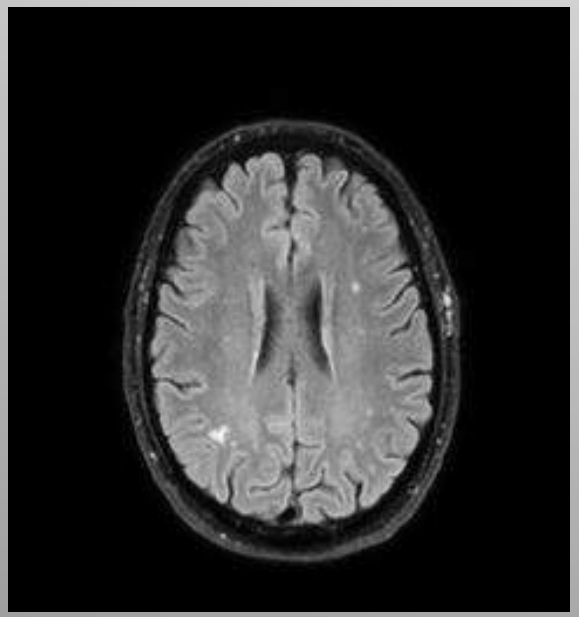
Fazekas scale
MEDIANS FOR SURVIVAL TIME (years)

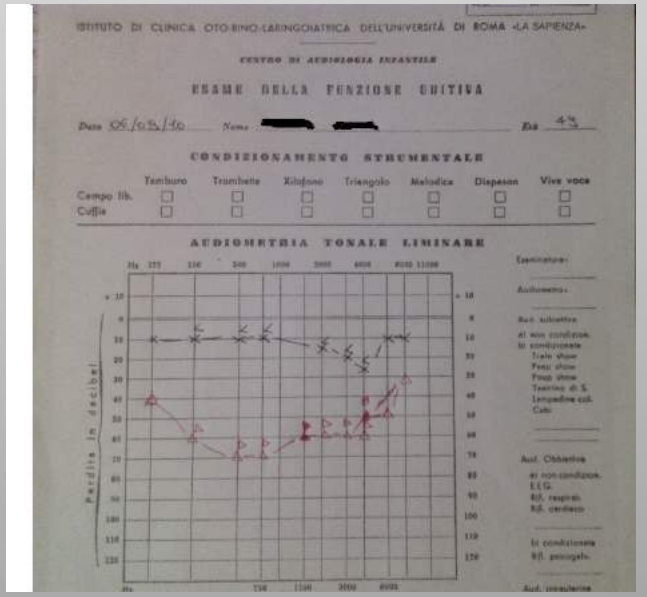
Mild 6.7 (95%CI 6.5 – 6.8)

Moderate 6.1 (95%CI 5.2 – 7.0)

Severe 2.2 (95%CI 1.6 – 2.7)

Attualmente non è noto il meccanismo d' azione trombotico dell' omocisteinemia. L' ipotesi più accreditata è che live




ISTITUTO DI CLINICA OTO-RINO-LARINGOIATRICA DELL'UNIVERSITÀ DI ROMA «LA SAPIENZA»

CENTRO DI AUDIOLOGIA INFANTILE

ESAME DELLA FUNZIONE UDITIVA

Data 06/05/40 Nome [redacted] Età 4 a

CONDIZIONAMENTO STRUMENTALE

	Tamburo	Trombetta	Xilofono	Triangolo	Melodica	Diapason	Viva voce
Campo lib.	☐	☐	☐	☐	☐	☐	☐
Cuffia	☐	☐	☐	☐	☐	☐	☐

AUDIOMETRIA TONALE LIMINARE

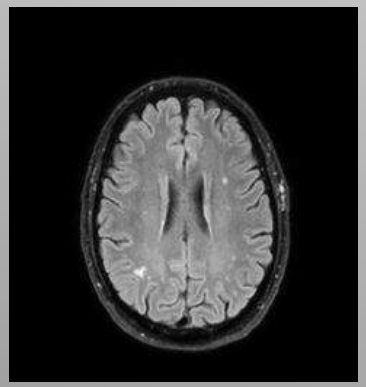
Perdita in decibel

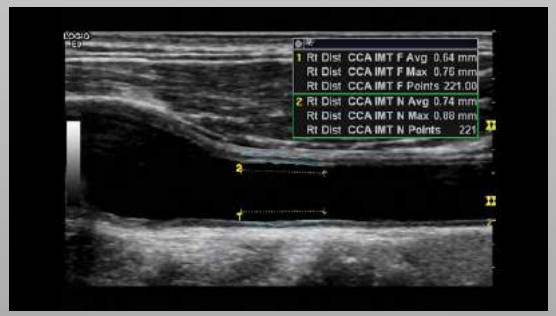
leucoaraiosi VS eco coolor doppler carotide

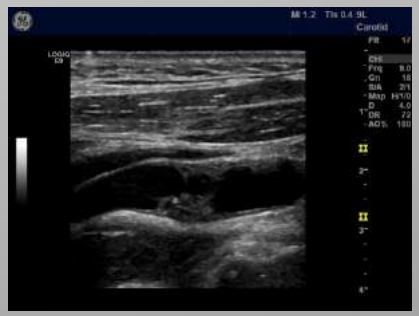
- aumentato spessore del complesso intima-media
- presenza di placche carotidee

associati a presenza di alterazioni della sostanza bianca

Rotterdam Study (Bretler et al., 1994)







leucoaraiosi VS eco coolor doppler carotide

Clinical Radiology xxx (2017) 1–6

Contents lists available at ScienceDirect

Clinical Radiology

journal homepage: www.clinicalradiologyonline.net

Association between leukoaraiosis and cerebral blood flow territory alteration in asymptomatic internal carotid artery stenosis

Y.-F. Chen [d], Y.-S. Kuo [c,f], W.-C. Wu [a,b,c,d,*], S.-C. Tang [e], S.-F. Jiang [d]

[a] Graduate Institute of Medical Device and Imaging, National Taiwan University, Taipei, Taiwan
[b] Graduate Institute of Clinical Medicine, National Taiwan University, Taipei, Taiwan
[c] Graduate Institute of Biomedical Electronics and Bioinformatics, National Taiwan University, Taipei, Taiwan
[d] Department of Medical Imaging, National Taiwan University Hospital, Taipei, Taiwan
[e] Department of Neurology, National Taiwan University Hospital, Taipei, Taiwan
[f] Department of Radiology, Cathay General Hospital, Taipei, Taiwan

ARTICLE INFORMATION

Article history:
Received 8 August 2017
Accepted 6 December 2017

AIM: To test the hypothesis that leukoaraiosis (also known as white matter lesion) is associated with cerebral blood flow territory change as revealed by territorial arterial spin-labeling (TASL) magnetic resonance imaging (MRI) in patients with asymptomatic internal carotid artery stenosis (aICAS).

MATERIALS AND METHODS: The institutional review board approved this study. Thirty-three patients with aICAS were included prospectively and divided into high-grade (ultrasonographic stenosis ≥70%, $n=17$) and low-grade ($n=16$) groups; 16 healthy subjects were also included. Cerebral flow territory was delineated for left ICA, right ICA, and vertebral arteries using TASL MRI and fuzzy clustering. Two licensed neuroradiologists independently and dichotomously rated the hemispherical asymmetry of flow territories. Flow territories were finalised by consensus, and when asymmetry was present, these were divided into normal and abnormal areas where the raters separately assessed leukoaraiosis based on fluid-attenuated inversion recovery images and the Fazekas scale.

RESULTS: The inter-rater agreement in the evaluation of flow territory asymmetry with TASL imaging in conjunction with time-of-flight angiogram is substantial (Cohen's kappa=0.82). Multinomial logistic regression (reference group=healthy subjects) indicates that global leukoaraiosis is not a predictor of aICAS after controlling for age, whereas in high-grade patients, the deep white matter lesion is more severe in the area receiving collateral circulation than in the area with normal flow territory (Wilcoxon signed-rank test, $p=0.03$).

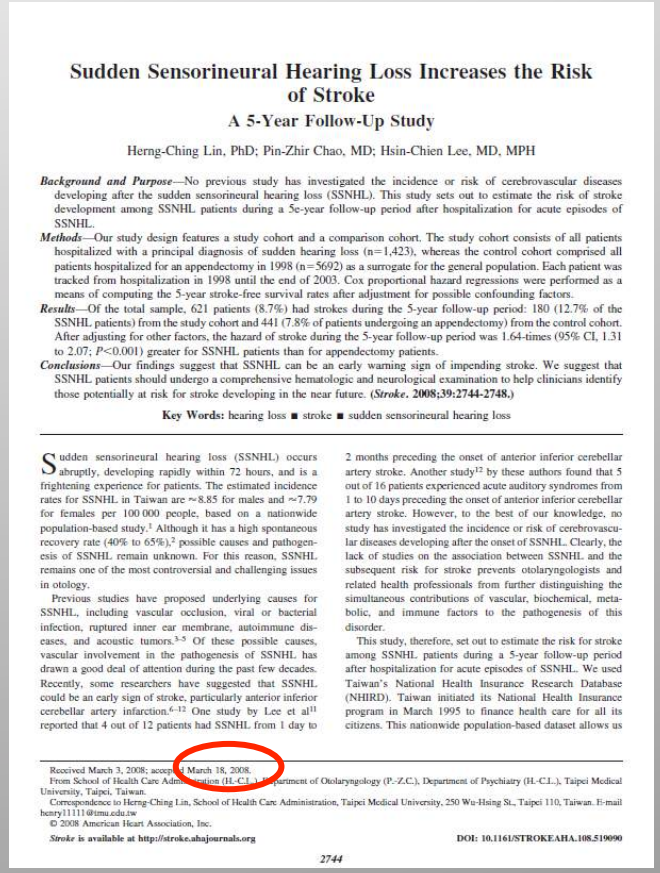
CONCLUSION: TASL MRI is clinically feasible in aICAS and shows that more severe deep white matter lesions are associated with collateral circulation in high-grade patients.

Background and Purpose: the incidence or risk of cerebrovascular diseases developing after (SSNHL) during a 5e-year follow-up period after hospitalization

Hypertension	0.001
Diabetes	0.001
Hyperlipidemia	0.007

Age:

45–64	0.001
65–74	0.001
Over 74	0.001



Sudden Sensorineural Hearing Loss Increases the Risk of Stroke

A 5-Year Follow-Up Study

Herng-Ching Lin, PhD; Pin-Zhir Chao, MD; Hsin-Chien Lee, MD, MPH

Background and Purpose—No previous study has investigated the incidence or risk of cerebrovascular diseases developing after the sudden sensorineural hearing loss (SSNHL). This study sets out to estimate the risk of stroke development among SSNHL patients during a 5e-year follow-up period after hospitalization for acute episodes of SSNHL.

Methods—Our study design features a study cohort and a comparison cohort. The study cohort consists of all patients hospitalized with a principal diagnosis of sudden hearing loss (n=1,423), whereas the control cohort comprised all patients hospitalized for an appendectomy in 1998 (n=5692) as a surrogate for the general population. Each patient was tracked from hospitalization in 1998 until the end of 2003. Cox proportional hazard regressions were performed as a means of computing the 5-year stroke-free survival rates after adjustment for possible confounding factors.

Results—Of the total sample, 621 patients (8.7%) had strokes during the 5-year follow-up period: 180 (12.7% of the SSNHL patients) from the study cohort and 441 (7.8% of patients undergoing an appendectomy) from the control cohort. After adjusting for other factors, the hazard of stroke during the 5-year follow-up period was 1.64-times (95% CI, 1.31 to 2.07; $P<0.001$) greater for SSNHL patients than for appendectomy patients.

Conclusions—Our findings suggest that SSNHL can be an early warning sign of impending stroke. We suggest that SSNHL patients should undergo a comprehensive hematologic and neurological examination to help clinicians identify those potentially at risk for stroke developing in the near future. (***Stroke*., 2008;39:2744-2748.)**

Key Words: hearing loss ■ stroke ■ sudden sensorineural hearing loss

Sudden sensorineural hearing loss (SSNHL) occurs abruptly, developing rapidly within 72 hours, and is a frightening experience for patients. The estimated incidence rates for SSNHL in Taiwan are ≈8.85 for males and ≈7.79 for females per 100 000 people, based on a nationwide population-based study.[1] Although it has a high spontaneous recovery rate (40% to 65%),[2] possible causes and pathogenesis of SSNHL remain unknown. For this reason, SSNHL remains one of the most controversial and challenging issues in otology.

Previous studies have proposed underlying causes for SSNHL, including vascular occlusion, viral or bacterial infection, ruptured inner ear membrane, autoimmune diseases, and acoustic tumors.[3–5] Of these possible causes, vascular involvement in the pathogenesis of SSNHL has drawn a good deal of attention during the past few decades. Recently, some researchers have suggested that SSNHL could be an early sign of stroke, particularly anterior inferior cerebellar artery infarction.[6–12] One study by Lee et al[11] reported that 4 out of 12 patients had SSNHL from 1 day to 2 months preceding the onset of anterior inferior cerebellar artery stroke. Another study[12] by these authors found that 5 out of 16 patients experienced acute auditory syndromes from 1 to 10 days preceding the onset of anterior inferior cerebellar artery stroke. However, to the best of our knowledge, no study has investigated the incidence or risk of cerebrovascular diseases developing after the onset of SSNHL. Clearly, the lack of studies on the association between SSNHL and the subsequent risk for stroke prevents otolaryngologists and related health professionals from further distinguishing the simultaneous contributions of vascular, biochemical, metabolic, and immune factors to the pathogenesis of this disorder.

This study, therefore, set out to estimate the risk for stroke among SSNHL patients during a 5-year follow-up period after hospitalization for acute episodes of SSNHL. We used Taiwan's National Health Insurance Research Database (NHIRD). Taiwan initiated its National Health Insurance program in March 1995 to finance health care for all its citizens. This nationwide population-based dataset allows us

Received March 3, 2008; accepted March 18, 2008.

From School of Health Care Administration (H.-C.L., H.-C.L.), Department of Otolaryngology (P.-Z.C.), Department of Psychiatry (H.-C.L.), Taipei Medical University, Taipei, Taiwan.

Correspondence to Herng-Ching Lin, School of Health Care Administration, Taipei Medical University, 250 Wu-Hsing St., Taipei 110, Taiwan. E-mail henry111116@tmu.edu.tw

Stroke is available at http://stroke.ahajournals.org DOI: 10.1161/STROKEAHA.108.519090

La compromissione della perfusione dell’’orecchio interno/labirinto è ampiamente riconosciuta come possibile meccanismo patogenico

- diabete,
- fumo,
- contraccettivi orali,
- terapia ormonale sostitutiva,
- menopausa

- l’aumento della viscosità ematica:

trigliceridi,

LDL,

colesterolo totale

Cadoni 2004,
Bil et al 1984,
Mattox et al 1999

Marcucci 2005
Herng-Ching Lin 2008

Review

Audiology & Neurotology

Audiol Neurootol 2014;19:256–260
DOI: 10.1159/000363213

Received: March 25, 2014
Accepted after revision: April 21, 2014
Published online: July 26, 2014

Cardiovascular Risk Factors as Causes for Hearing Impairment

Yahav Oron Katya Elgart Tal Marom Yehudah Roth

Department of Otolaryngology, Head and Neck Surgery, Edith Wolfson Medical Center, Sackler School of Medicine, Tel Aviv University, Holon, Israel

Key Words
Hearing loss · Aging · Stria vascularis · Diabetes · Hypertension · Hyperlipidemia · Smoking

Abstract
The purpose of this paper is to provide a contemporary review of the correlation between cardiovascular risk factors (CVRFs) and hearing impairment (HI). We conducted a comprehensive review of the literature in order to assess the effects of the different CVRFs on HI. We focused on the pathological findings in the inner ear and their correlation with cochlear function in population-based studies. We found that CVRFs adversely affect hearing acuity. HI diagnosis should be accompanied by detecting and treating CVRFs, according to the presented outline, which may augment hearing rehabilitation and improve the general health and the well-being of the patient.

Introduction

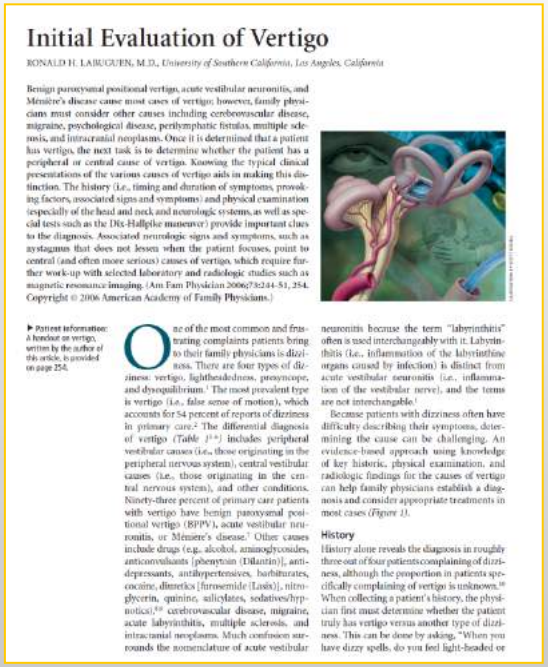
The major cardiovascular risk factors (CVRFs) include an older age, male gender, hypertension, hyperlipidemia, lack of physical activity, smoking, diabetes mellitus (DM) and positive family history of coronary heart disease [Oparil and Oberman, 1999]. These CVRFs were also investigated as risk factors for the development of hearing impairment (HI). The purpose of this paper is to review the current knowledge regarding the relationship between CVRFs and HI and to suggest possible prevention and treatment options. CVRFs are usually more common among older patients, as is presbycusis. Thus, it might be difficult to distinguish the effects of age and noise exposure on hearing acuity from the effects which CVRFs might have on hearing.

Blood Supply to the Inner Ear and Auditory Nerve

The internal auditory artery (IAA) is the source of blood supply to the cochlea and to the auditory nerve. The IAA is usually a branch of the anterior inferior cerebellar artery and it is an end artery, without anastomosis to compensate for any obstruction of the blood vessel [Baloh, 2001; Kim and Hyung, 2009] (fig. 1). The IAA feeds the stria vascularis (SV) at the lateral wall of the cochlea. At the cochlear apex this blood supply becomes sparse [Friedland et al., 2009]. The SV has a major role in the production of the endocochlear potential, which is essential for normal hearing [Wangemann, 2006]. More medi-

KARGER

Yahav Oron, M.D.
Department of Otolaryngology, Head and Neck Surgery



Initial Evaluation of Vertigo

RONALD H. LABUGUEN, M.D., *University of Southern California, Los Angeles, California*

Benign paroxysmal positional vertigo, acute vestibular neuronitis, and Ménière's disease cause most cases of vertigo; however, family physicians must consider other causes including cerebrovascular disease, migraine, psychological disease, perilymphatic fistulas, multiple sclerosis, and intracranial neoplasms. Once it is determined that a patient has vertigo, the next task is to determine whether the patient has a peripheral or central cause of vertigo. Knowing the typical clinical presentations of the various causes of vertigo aids in making this distinction. The history (i.e., timing and duration of symptoms, provoking factors, associated signs and symptoms) and physical examination (especially of the head and neck and neurologic systems, as well as special tests such as the Dix-Hallpike maneuver) provide important clues to the diagnosis. Associated neurologic signs and symptoms, such as nystagmus that does not lessen when the patient focuses, point to central (and often more serious) causes of vertigo, which require further work-up with selected laboratory and radiologic studies such as magnetic resonance imaging. (Am Fam Physician 2006;73:244-51, 254. Copyright © 2006 American Academy of Family Physicians.)

▶ **Patient information:** A handout on vertigo, written by the author of this article, is provided on page 254.

One of the most common and frustrating complaints patients bring to their family physicians is dizziness. There are four types of dizziness: vertigo, lightheadedness, presyncope, and disequilibrium.[1] The most prevalent type is vertigo (i.e., false sense of motion), which accounts for 54 percent of reports of dizziness in primary care.[2] The differential diagnosis of vertigo (*Table 1*[3-6]) includes peripheral vestibular causes (i.e., those originating in the peripheral nervous system), central vestibular causes (i.e., those originating in the central nervous system), and other conditions. Ninety-three percent of primary care patients with vertigo have benign paroxysmal positional vertigo (BPPV), acute vestibular neuronitis, or Ménière's disease.[7] Other causes include drugs (e.g., alcohol, aminoglycosides, anticonvulsants [phenytoin (Dilantin)], antidepressants, antihypertensives, barbiturates, cocaine, diuretics [furosemide (Lasix)], nitroglycerin, quinine, salicylates, sedatives/hypnotics),[8,9] cerebrovascular disease, migraine, acute labyrinthitis, multiple sclerosis, and intracranial neoplasms. Much confusion surrounds the nomenclature of acute vestibular neuronitis because the term "labyrinthitis" often is used interchangeably with it. Labyrinthitis (i.e., inflammation of the labyrinthine organs caused by infection) is distinct from acute vestibular neuronitis (i.e., inflammation of the vestibular nerve), and the terms are not interchangable.[1]

Because patients with dizziness often have difficulty describing their symptoms, determining the cause can be challenging. An evidence-based approach using knowledge of key historic, physical examination, and radiologic findings for the causes of vertigo can help family physicians establish a diagnosis and consider appropriate treatments in most cases (*Figure 1*).

History

History alone reveals the diagnosis in roughly three-out-of-four patients complaining of dizziness, although the proportion in patients specifically complaining of vertigo is unknown.[10] When collecting a patient's history, the physician first must determine whether the patient truly has vertigo versus another type of dizziness. This can be done by asking, "When you have dizzy spells, do you feel light-headed or

International Journal of Advances in Medicine
Rahul RK et al. Int J Adv Med. 2016 Feb;3(1):106-109
http://www.ijmedicine.com

pISSN 2349-3933 | eISSN 2349-3941

Research Article

DOI: http://dx.doi.org/10.18203/2349-3933.ijam20160109

Prevalence, risk factors and clinical presentations of patients with peripheral vertigo: a retrospective study from a tertiary care hospital

Ravendran Krishnan Rahul, Chakramakal Joseph Andrews*, Karuthedath Sridevi, Arayamparambil Rajagopalan Vinayakumar

Department of ENT, Amrita Institute of Medical Sciences, Amrita Nagar, Thiruvananthapuram 695 555, Kerala, India

Received: 16 November 2015
Accepted: 17 December 2015

***Correspondence:**
Dr. Chakramakal Joseph Andrews,
E-mail: noblejohn@gmail.com

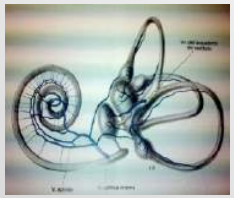
ABSTRACT

Background: A retrospective study was conducted to find out the clinical presentations and risk factors of peripheral vertigo.
Methods: Sixty patients with peripheral vertigo (age 20-70 years) who had presented with signs of vertigo were included in the study. A thorough history was taken from the subjects. The prevalence, clinical presentation and risk factors were subjected to statistical analysis.
Results: Total 60 patients of age 20-70 years (15 males and 45 females) were included in the study. Based on the distribution of risk factors, age group 40–59 was highest followed by the age group of 60-70. The prevalence of incidence was significantly found in females ($P < 0.01$). The clinical manifestations presented were spinning, about to faint, light-headedness, disequilibrium and both spinning and about to faint. Spinning plus about to faint was significantly ($p < 0.01$) found as the major clinical manifestation with a female dominance (19/25). This was highly significant ($p < 0.05$) in the age group of 40-59 years (8/19). Disequilibrium (10%) was the least clinical presentation. Among the risk factors, the patient with ear infection was significantly found (31.6%) with predominance for female (32/10).
Conclusions: The result concluded that prevalence of incidence was significantly found in females of age group 40-59 with clinical presentation of both spinning and about to faint. Risk factor of ear infection was significantly found. These observations will help a surgeon to recognize which all patients need inpatient management or emergency intervention. Furthermore, the need for awareness about the prompt treatment of ear infection is emphasized.

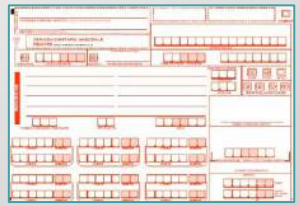
Keywords: Vertigo, Vertebrobasilar insufficiency, Light-headedness, Spinning, Disequilibrium, Labyrinthitis

INTRODUCTION

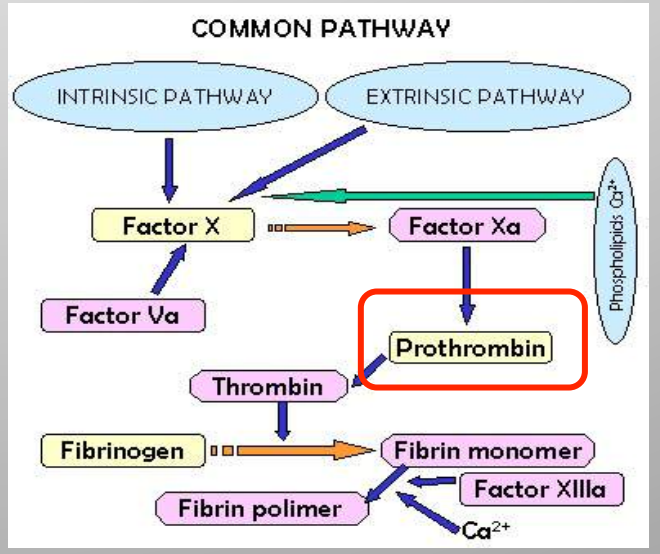
Vertigo is a very common complaint in clinical practice. The epidemiological knowledge on vertigo mainly on its prevalence or incidence character is limited. Peripheral causes of vertigo arise from abnormalities in the vestibular end organs (semi-circular canals and otoliths), the vestibular nerve, and the vestibular nuclei. Most of these causes are benign and readily treatable. Vertigo has anticonvulsive etiology and hence the patients may consult various specialties including psychiatry, otolaryngology, neurology and cardiology. Although all these disciplines play an important role in the evaluation of the patient, a good history and focal physical examination in the primary care setting can usually reveal the diagnosis. The management approaches employed by these clinicians will be varied considerably. Vertigo is often an unnamed symptom and is frequently associated with serious handicap and considerable psychological morbidity.[1] In addition to diagnosis of vertigo, the goal of the primary clinician should be to recognize which patients need inpatient management or emergency intervention. This

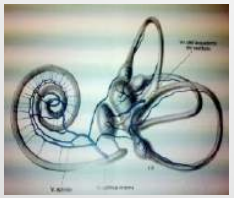


protrombina mutata: Fattore II della coagulazione



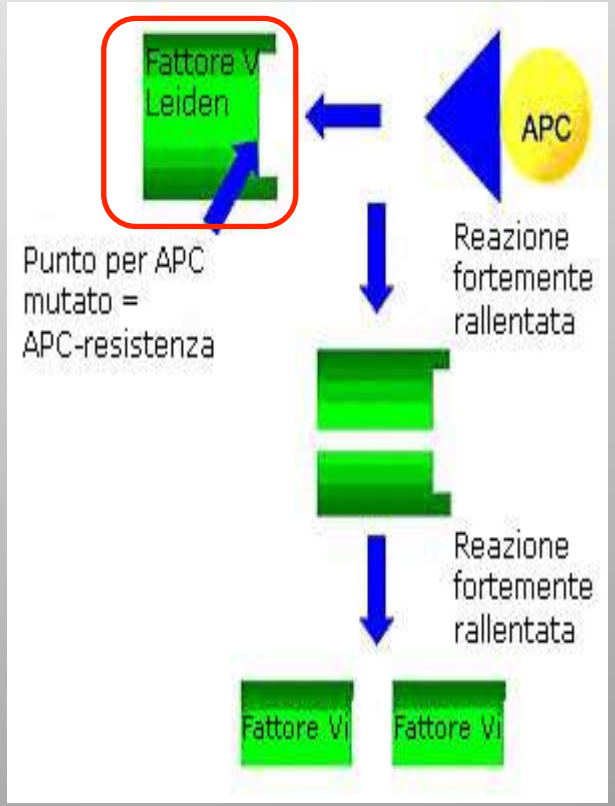
- Sul gene che codifica la protrombina mutazione (G20210A) eterozigote
- aumento del livello ematico di protrombina.

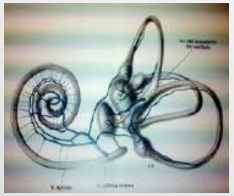




fattore V Leiden

- Sul gene che codifica il fattore V Leiden mutazione (G1691A) eterozigote
- impossibilità da parte della Proteina C di inattivare l' azione protrombotica del Fattore V





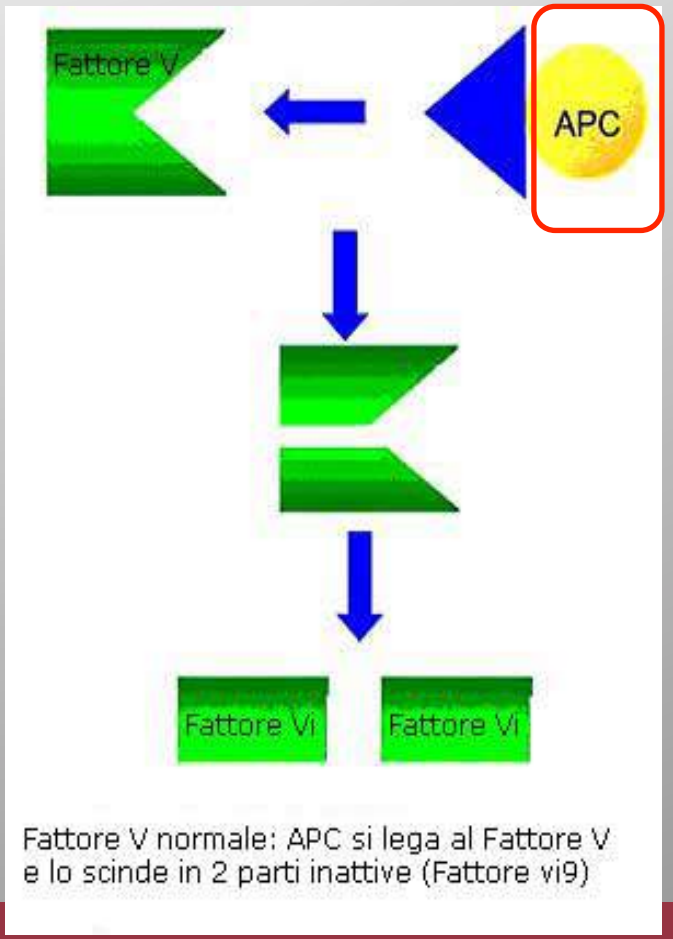
DEFICIT PROTEINA C

- Sul gene che codifica proteina C mutazione omozigote

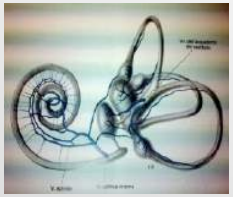
  (porpora fulminans o CID) è fatale senza terapia sostitutiva e anticoagulante.

- Riduzioni acquisite:

  malattie epatiche, gravi infezioni, chemioterapia e con la terapia con coumadin.



Fattore V normale: APC si lega al Fattore V e lo scinde in 2 parti inattive (Fattore vi9)

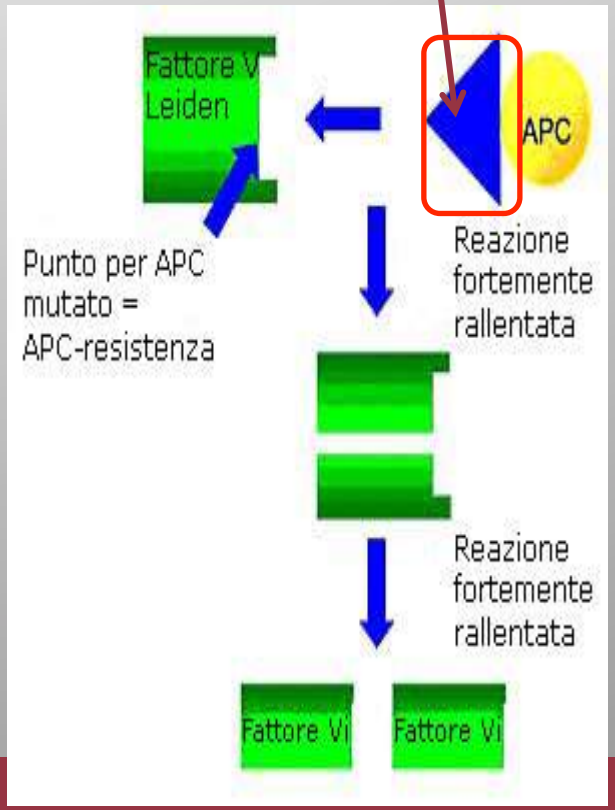


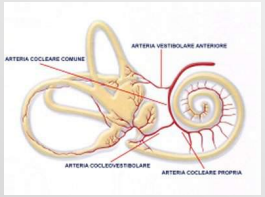
DEFICIT PROTEINA S

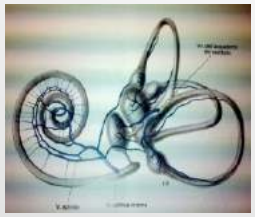
- Sul gene che codifica proteina S plasmatica mutazione omozigote

- Riduzioni acquisite:

  gravidanza, infezioni gravi, HIV, uso di contraccettivi orali, terapia con coumadin







i*peromocisteine*mia

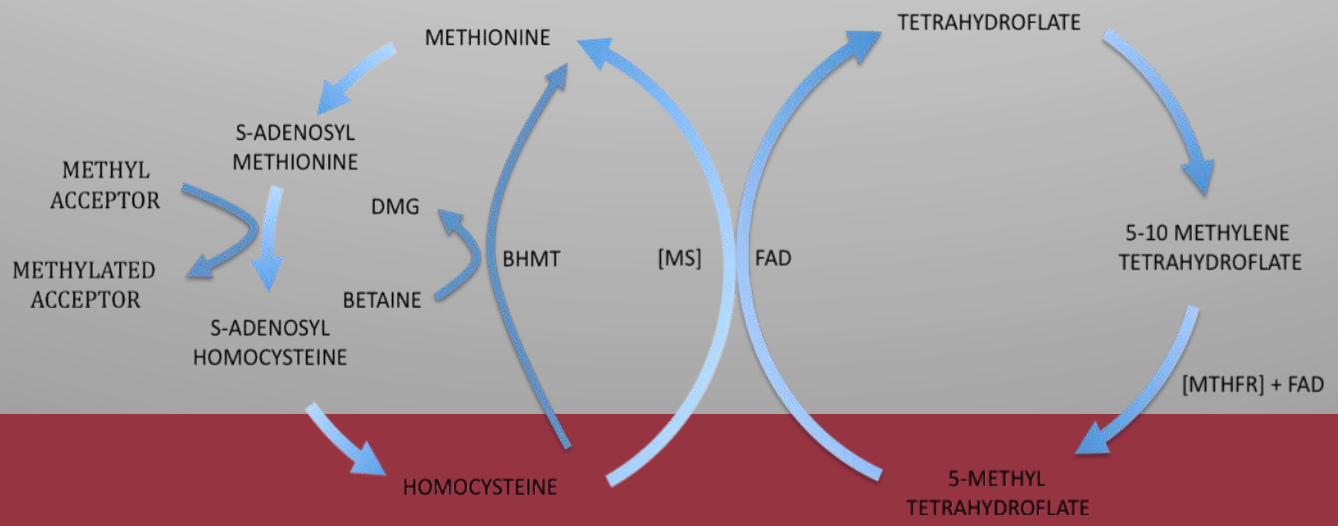
Sul gene che codifica l'MTHFR può esserci una mutazione

- 677CC (Wild Type)
- 677CT (Eterozigote)
- 677TT (Omozigote) Malattia

- sostituzione Citosina (C) >Timina (T) nel nucleotide 677 del gene per l'MTHFR porta alla formazione di un enzima termolabile

- L’enzima MTHFR è responsabile della riduzione del 5-10-metiltetraidrofolato in 5-metiltetraidrofolato, necessario per la trasformazione di omocisteina in metionina.

l'omocisteina si accumula nel sangue e può provocare danni alle pareti dei vasi (il colesterolo riesce quindi a depositarsi bene) .



Iperomocisteinemia acquisita

- **disordini alimentari,**
- **ipotiroidismo,**
- **malattie renali.**

(Frosst et al 1995)

L'acido folico è presente nelle frattaglie (rene, fegato), come folati nelle verdure a foglia verde (lattuga,spinaci,broccoli), nei legumi e nelle uova.

La sua presenza è scarsa nella frutta e nel latte. L'acido folico si può perdere durante la cottura.

malattia ischemica cardiaca

- **Wald et al.: meta analisi condotta su 72 studi**

  omozigosi per la mutazione MTHFR:

- Wald DS, Law M, Morris JK. Homocysteine and cardiovascular disease: evidence on causality from a meta-analysis. BMJ. 2002 Nov 23;325(7374): 1202

malattia ischemica cardiaca

- **Klerk et al.: omozigosi mutazione MTHFR:**
- **In posizione C677T significatività**
- **In quelle popolazioni (europea) in cui l'integrazione di folati nei prodotti alimentari non è obbligatoria**

Klerk M, Verhoef P, Clarke R, Blom HJ, Kok FJ, Schouten EG; MTHFR Studies Collaboration Group. MTHFR 677C-->T polymorphism and risk of coronary heart disease: a meta-analysis. JAMA. 2002 Oct 23-30;288(16):2023-31.

non è noto il meccanismo d'azione trombotico dell'omocistenemia

- Danneggi l'endotelio dei vasi,
- antagonizza la sintesi e la funzione dilatante dell'ossido nitrico endoteliale,
- favorisce la proliferazione delle cellule muscolari lisce della tonaca muscolare del vaso con accumulo di LDL , le due circostanze coniugare comportano un ristringimento del vaso con rallentamento del circolo fino alla trombosi

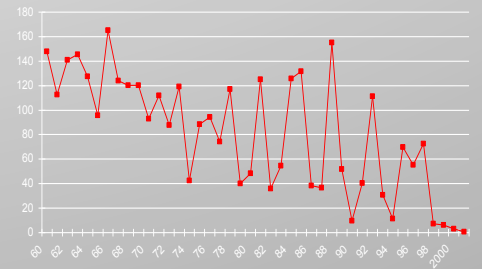
CONCLUSIONI

- Il microcircolo dell'orecchio interno è di tipo terminale e garantito dai piccoli vasi come quello cerebrale
- entrambi si riforniscono dalle stesse arterie maggiori,
- le stesse cause della SVD cerebrale possono agire sui vasi dell'orecchio interno estremamente piccoli,
- perciò un micro embolo, uno spasmo, una minore efficienza dell'autoregolazione o la degenerazione ialina del microcircolo possono determinare l'ipoacusia improvvisa.

Il virus si presume raggiunge l'orecchio interno

1. diffusione ematogena, liquido cerebrospinale dall'orecchio medio.
2. riattivazione di un virus latente infezione dell'orecchio interno.
3. un'infezione virale sistemica o distante che innesca una risposta anticorpale cross-reagisce con un antigene dell'orecchio interno.

1 2 l virus della parotite epidemica (meno del 10%) morbillo e la rosolia



famiglia di virus Herpes VS neurite labirintica/ SSHL

- herpes simplex types 1 and 2,
- varicella zoster virus (VZV),
- citomegalovirus (CMV),
- virus Epstein-Barr
- herpesvirus 6, 7 e 8 (HHV-6, HHV-7 e HHV-8).

La maggior parte degli adulti è sieropositiva per molti di questi virus.
riattivazione nell paziente del virus latente.