LA STORIA DELLA VESTIBOLOGIA

Eugenio Mira

Clinica Otorinolaringoiatrica, Università di Pavia
Fondazione IRCCS Policlinico S. Matteo, Pavia

INTRODUZIONE

La vestibologia, come le neuroscienze e in generale tutte le scienze mediche, evolve grazie al contributo delle ricerche di base, delle osservazioni cliniche e delle innovazioni tecnologiche

Le origini

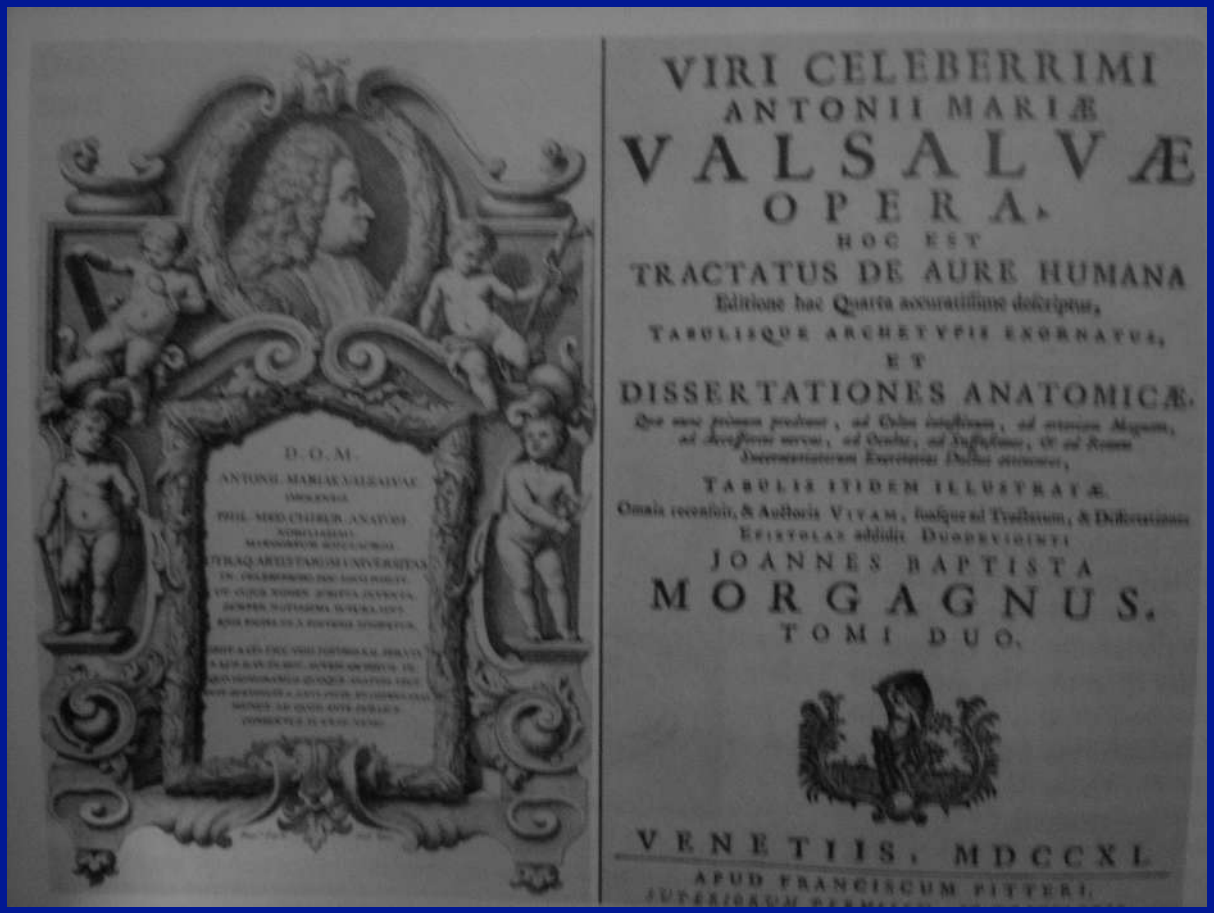
Antonio Maria Valsalva (1666 – 1723)

Allievo di Marcello Malpighi.

Docente di anatomia all' Università di Bologna (1705).

Autore del "Ttractatus de aure humana" (1704) in cui introduce la suddivisione dell' orecchio in esterno, medio e interno. Nell' orecchio interno dà una accurata descrizione della chiocciola e dei canali semicircolari, in cui descrive la presenza di una linfa ut aqua limpida.

A M Valsalva (1666 – 1723)

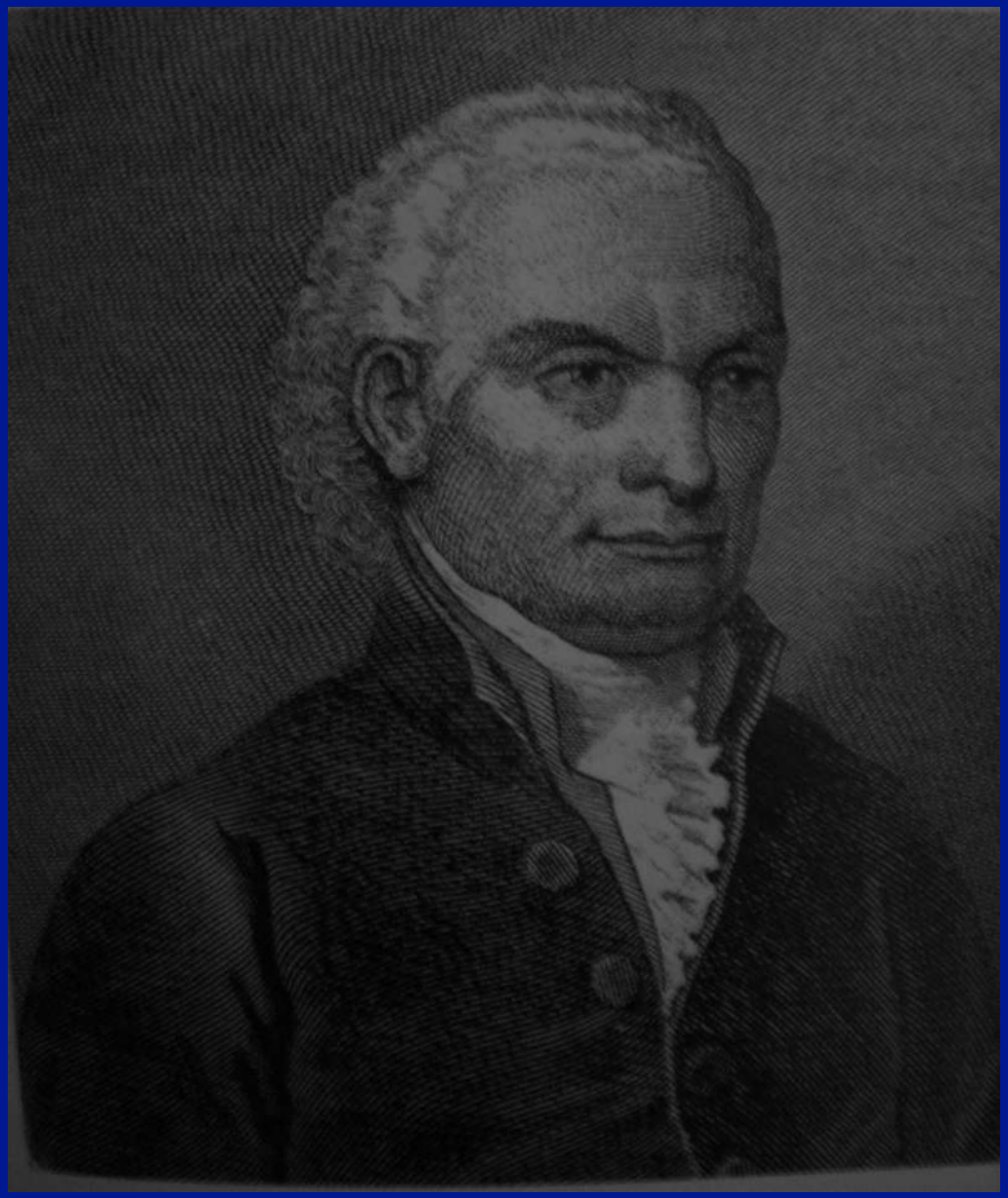


A M Valsalva (1666 – 1723)

Domenico Cotugno (1736 – 1822)

Docente di anatomia all' Università di Napoli (1766).

Autore del trattato "De aqueductibus auris humanae" (1775) in cui descrive gli acquedotti del vestibolo e della chiocciola e individua la presenza dei due liquidi labirintici.



Domenico Cotugno (1736-1822)

TAB. X.

FIG. I.

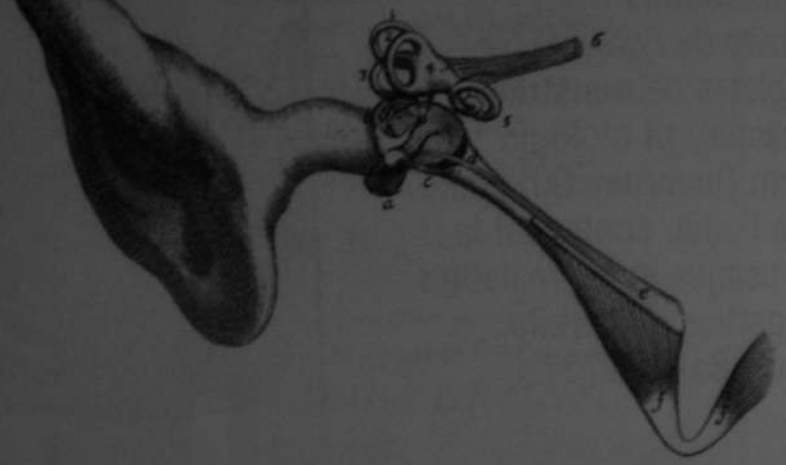
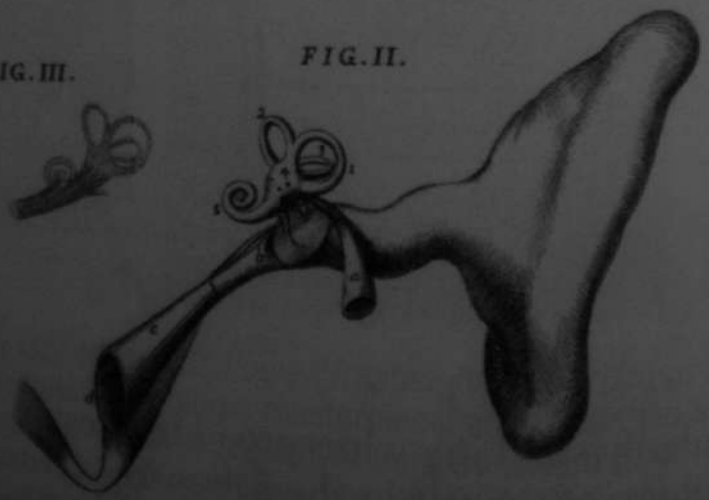


FIG. III.

FIG. II.



DOMINICI COTUNNII

PHIL. ET MED. DOCT.

DE

AQUÆDUCTIBUS

AURIS HUMANÆ

INTERNÆ

ANATOMICA DISSERTATIO

NEAPOLI, ET BONONIÆ

Ex Typographia Sancti Thomæ Aquinatis

MDCCLXXV.

Superiorum auctoritate.

Antonio Scarpa (1752 – 1832)

Allievo di G B Morgagni. Docente di anatomia e chirurgia alle università di Modena (1722) e di Pavia (1783). Autore di "De structura fenestrae rotundae auris, et de tympano secundario anatomicae observationes" (1722) e di "Anatomicae disquisitiones de auditu et olfactu" (1789) con descrizione accurata delle strutture dell' orecchio interno e del nervo e ganglio vestibolare.

Antonio Scarpa (1752-1832)

ANATOMICAE DISQUISITIONES

DE

AUDITU ET OLFACTU

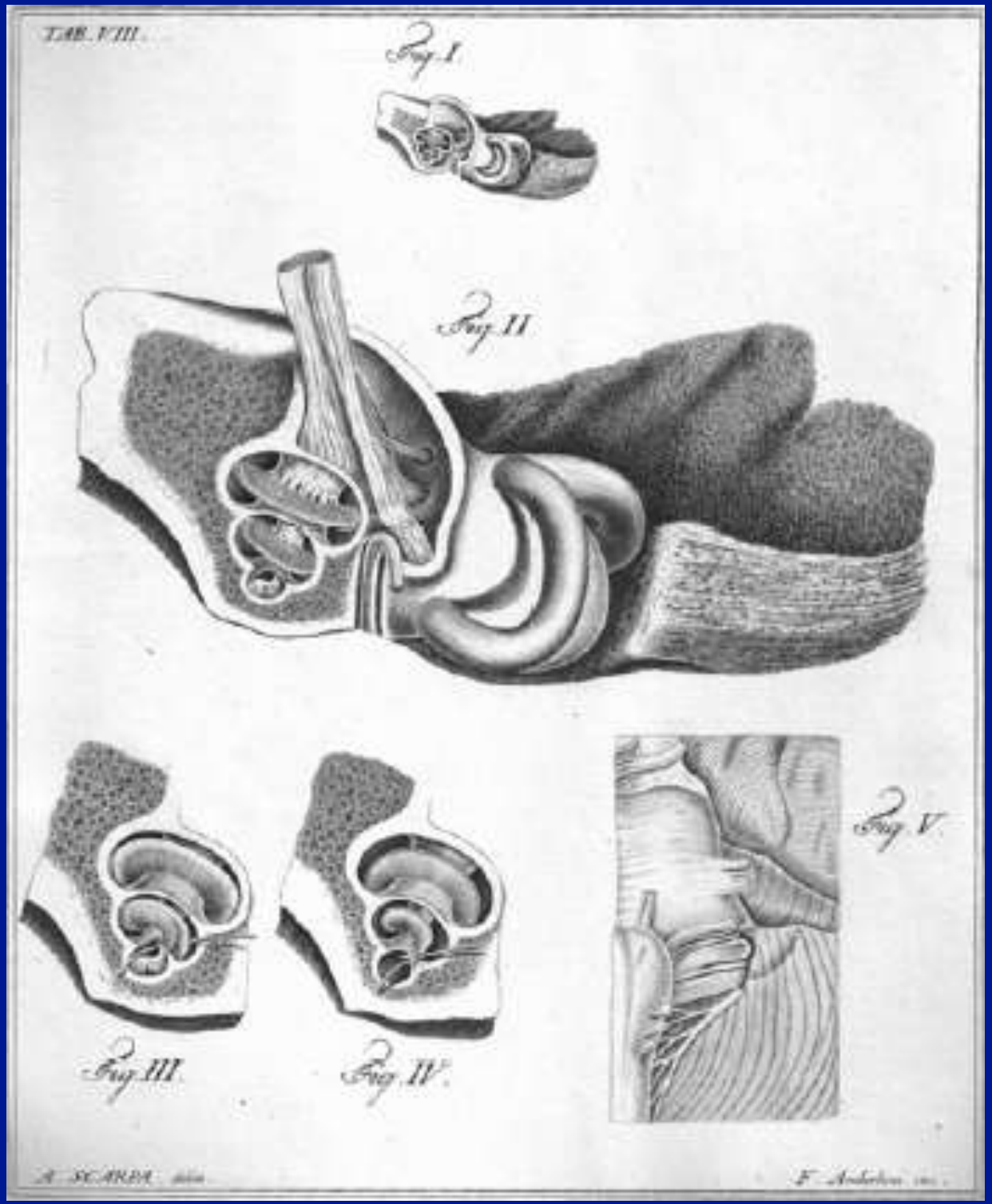
AUCTORE

ANTONIO SCARPA

In Taurinensi Archigymnasio Anatomes, et Chirurgiae Clinices Professore, Facultatis Chirurgicae nec non Societatum Anatomicae Parisin. Imp. Acad. Medico-Chir. Vindobon., Caesareo-Leopold. Nat. Curios., R. Scient. Acad. Berolin., R. Practicae Medic. Londin., R. Scient. Acad. Taurin. Ac. Ac. Sodali.

TICINI.

IN TYPOGRAPHEO PETRI GALEATTI PRAESIDIS REG. LITTER. SERENITT.
ANNO MDCCLXXXIII.


TAB. VIII.
Fig. I.
Fig. II
Fig. III.
Fig. IV.
Fig. V.
A. SCARPA delin.
F. Anderloni inc.

Marie-Jean Pierre Flourens (1794 – 1867)

Docente di fisiologia a Parigi. Uno dei padri della neurologia, dove descrisse le localizzazioni cerebrali, e della anestesia con cloroformio.

Con le sue osservazioni sui piccioni individuò la funzione dei canali semicircolari come organo correlato al movimento anziché all' udito.

Marie-Jean Pierre Flourens (1794-1867)

JOURNAL GÉNÉRAL
DE MÉDECINE,
DE CHIRURGIE ET DE PHARMACIE
FRANÇAISES ET ÉTRANGÈRES,

OU

RECUEIL PÉRIODIQUE

DES TRAVAUX

DE LA SOCIÉTÉ DE MÉDECINE DE PARIS;

RÉDIGÉ

PAR A. N. GENDRIN, L'UN DE SES MEMBRES.

TOME CIV. — VII^e DE LA III^e SÉRIE.

A PARIS,
CHEZ BAILLIÈRE, LIBRAIRE,
RUE DE L'ÉCOLE DE MÉDECINE, N° 13 *bis*.

Juillet M. DCCC. XXVIII.

1828

et les conduira enfin à ordonner l'exécution de la totalité de la loi sur l'exercice de la pharmacie.

INSTITUT. — ACADÉMIE DES SCIENCES.

Travaux relatifs à la Médecine et aux Sciences qui s'y rattachent.

1828

Séance du 11 août 1828.

Expériences sur les Canaux semi-circulaires de l'oreille des Oiseaux.

M. Flourens fait connaître des expériences qu'il a faites dans l'intention de déterminer quelles sont les fonctions du nerf acoustique dans les canaux semi-circulaires dans les oiseaux.

Les canaux semi-circulaires sont, dans les oiseaux comme dans l'homme, au nombre de trois, deux verticaux et un horizontal. Chez les pigeons, qui sont particulièrement les animaux qui ont servi aux expériences de M. Flourens, le plus grand de ces canaux est vertical et un peu oblique en avant.

M. Flourens a coupé le canal vertical antérieur d'un côté : l'animal a été immédiatement agité de ce côté d'un mouvement brusque continuel et involontaire en avant; cette agitation s'est calmée au bout de quelques instans, mais elle s'est reproduite aussitôt que l'animal a voulu faire un mouvement.

Après la section du canal semi-circulaire vertical postérieur d'un côté, l'animal est agité d'un mouvement vertical violent et continuel en arrière, et du côté où le canal a été coupé; enfin, la section du canal semi-circulaire horizontal détermine un mouvement horizontal également continuel.

OSSERVAZIONI DI FLOURENS (1794 – 1867)

Le nerf des canaux semicirculaires est un nerf spécial et propre.

……

Il est doué de la faculté singulière d’ agir sur la direction des mouvements.

OSSERVAZIONI DI FLOURENS (1824 – 1828)

La section de chacune de ces parties détermine un ordre ou une direction de mouvements toujours parfaitement conformes à la direction de la partie coupée. Ainsi la section des canaux horizontaux détermine un mouvement horizontal; celle des canaux verticaux un mouvement vertical.

Prosper Menière (1799 – 1862)

Medico ginecologo, internista, infettivologo infine direttore dell' Istituto Nazionale dei Sordomuti.

Nella seduta dell' 8 gennaio 1861 presentava alla Accademia Imperiale di Medicina una memoria in cui correlava due sintomi uditivi, ipoacusia e acufeni, e un sintomo sino ad allora ritenuto di origine neurologica, la vertigine, ad una patologia dell' orecchio interno.

Prosper Menière (1799-1862)

ANNEE 1861.

GAZETTE MÉDICALE

DE PARIS.

TRENTE-UNIÈME ANNÉE. — TROISIÈME SÉRIE. — TOME SEIZIÈME.

N° 38. — 21 SEPTEMBRE.

1861

TABLE DES MATIÈRES.

BUREAUX DE LA GAZETTE MÉDICALE, RUE CHANOINESSE, 12, PRÈS NOTRE-DAME

La Gazette Médicale de Paris (Gazette de santé et Clinique des hôpitaux réunies) paraît tous les samedis; un numéro, avec le supplément, comprend vingt-quatre pages in-4°, quarante-huit colonnes, ce qui équivaut à neuf feuilles in-8°. — Le prix de l'abonnement est, pour Paris et les départements, de 36 francs par an, 18 francs pour six mois, et 9 francs pour trois mois; pour l'étranger, 40 francs. Les abonnements ne peuvent dater que du commencement d'un trimestre, 1er janvier, 1er avril, 1er juillet, 1er octobre. — On s'abonne, à Paris, au bureau du Journal, et dans les départements, chez tous les directeurs de postes et de messageries. — On ne reçoit que les lettres affranchies.

sur la morve sont passées en peu de temps de leur première de d'évolution à la seconde; nous espérons bien qu'ils ne tarde- ons, eux et leurs collègues, à les faire passer à la troisième : ce a meilleure preuve de sagacité qu'ils auront montrée dans cette ssion, et, pour nous, la seule compensation que nous ayons à rer de nos efforts!

JULES GUÉRIN.

PATHOLOGIE AURICULAIRE.

MÉMOIRE SUR DES LÉSIONS DE L'OREILLE INTERNE DONNANT LIEU A DES SYMPTÔMES DE CONGESTION CÉRÉBRALE APOPLECTIFORME; par le docteur P. MENIÈRE, agrégé de la Faculté, médecin de l'institution impériale des Sourds-Muets. (Lu à l'Académie impériale de médecine, dans la séance du 8 janvier 1861) (1).

Il s'est présenté à mon observation, il y a déjà bien longtemps, un certain nombre de malades offrant un groupe de symptômes toujours les mêmes, symptômes d'apparence grave, donnant l'idée d'une lésion organique de la plus fâcheuse espèce, se renouvelant de temps en temps pendant des semaines, des mois, des années, disparaissant tout à coup et offrant pour résultat commun l'abolition d'un sens. Qu'on me permette la description d'un de ces états pathologiques que tout le monde a rencontrés, et l'on comprendra bientôt l'importance que j'ai dû y attacher en raison des suites qu'il avait, de l'infirmité qui en était la conséquence.

Un homme jeune et robuste éprouve subitement, sans cause appréciable, des vertiges, des nausées, des vomissements; un état d'angoisse inexprimable anéantissait les forces; le visage pâle et baigné de sueur annonçait une syncope prochaine. Souvent même le malade, après s'être senti chancelant, étourdi, était tombé à terre sans pouvoir se relever; couché sur le dos, il ne pouvait ouvrir les yeux sans voir les objets environnants tourbillonner dans l'espace; le plus léger mouvement imprimé à la tête augmentait les vertiges et les nausées; les vomissements se renouvelaient dès que le malade essayait de changer de position. Ces accidents, hâtons-nous de le dire, n'avaient aucun rapport avec l'état de plénitude ou de vacuité de l'estomac; ils survenaient au milieu d'une santé irréprochable; ils duraient peu, mais leur caractère était tel que les médecins appelés croyaient à une congestion cérébrale et prescrivaient un traitement en rapport avec cette vue étiologique.

Des accidents de même nature s'étant reproduits à plusieurs reprises, causèrent de graves inquiétudes, d'autant plus qu'entre chaque crise il restait une disposition aux vertiges, aux étourdissements. Le patient ne pouvait lever brusquement la tête, se tourner à droite ou à gauche sans perdre le sentiment de l'aplomb; sa marche devenait incertaine, il inclinait sans le vouloir vers un côté, souvent encore il était contraint de s'appuyer contre un mur, le sol lui paraissait inégal, il se heurtait au moindre obstacle, les deux jambes n'étaient plus également habiles à franchir les degrés d'un escalier; en un mot, les muscles de la station et de la marche ne fonctionnaient plus avec leur régularité accoutumée.

Tout mouvement un peu brusque déterminait des troubles fonctionnels du même ordre. Si le malade, au moment du coucher, se laissait aller brusquement à la position horizontale, aussitôt le lit et tous les objets environnants entraient dans un mouvement giratoire énorme, il se croyait sur le pont d'un navire balancé par un roulis de grande dimension, et les nausées se manifestaient aussitôt, absolument comme au début du mal de mer. Par contre, en se levant, s'il reprenait tout à coup la position verticale, les mêmes phénomènes se déclaraient, et si le malade voulait se mettre en marche, il tournait sur lui-même et ne tardait pas à tomber. On observait alors la pâleur du visage, un état syncopal, le corps se couvrait d'une sueur froide, et tout indiquait une angoisse profonde.

Jusque-là, rien de spécial n'avait attiré l'attention du malade et des médecins. On ne voyait en tout ceci qu'une congestion cérébrale devant céder à un régime sévère, à des évacuations sanguines, à des purgatifs; mais l'expérience prouvait bientôt que cette médication, acceptée avec empressement et suivie avec une extrême rigueur, demeurait impuissante, et l'on portait toujours les plus fâcheux pronostics.

Mais le patient attentif ne tardait pas à signaler l'apparition de certains phénomènes, par exemple des bruits dans les oreilles souvent très-forts, très persistants, et puis l'ouïe s'affaiblissait d'une manière notable d'un côté, quelquefois même des deux côtés, et c'est à cette occasion que l'on avait recours à un médecin s'occupant plus spécialement de maladies d'oreilles. J'explorais ces organes, je n'y découvrais le plus souvent aucune trace d'une lésion appréciable, mais aussi je constatais la coïncidence entre la surdité et les troubles cérébraux dont on m'avait fait part. J'eus de fréquentes occasions de voir des faits semblables, je m'attachai curieusement à rechercher leur valeur réelle, il se rencontra des circonstances si favorables cette enquête que je fus conduit à considérer cet ensemble de lésions cérébrales et auditives comme une seule maladie. Poursuivons cette démonstration.

Chez quelques malades plus attentifs à ce qui se passe en eux, il me fut possible, à l'aide de questions très-précises, d'établir que les vertiges, l'état syncopal, la chute subite du corps, les vomissements, avaient été précédés de bruits dans les oreilles, que ces bruits ne reconnaissaient aucune cause appréciable, qu'ils persistaient dans l'intervalle des accès, mais qu'ils coïncidaient souvent avec l'augmentation des étourdissements, et que jamais ces bruits ne prenaient la forme saccadée, artérielle, en un mot qu'ils n'étaient pas carotidiens. C'était déjà un indice de leur caractère nerveux, ils dépendaient d'un état particulier des nerfs acoustiques et non d'une cause sanguine; le système circulatoire n'y était pour rien. Je me croyais suffisamment autorisé à ne voir en ces phénomènes si graves, si inquiétants, que l'expression symptomatique de la lésion d'un appareil spécial, compatible avec la conservation de la santé générale, et en effet,

(1) Voir d'autres communications sur la même question, GAZ. MÉD., année 1861, p. 29, 88 et 239.

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le joindre à ses collections de la Martinique, mais le propriétaire des poulets, qui avait pu le capturer, ne voulut jamais s'en dessaisir, disant, pour excuser son refus, qu'il voulait faire expier lui-même à *l'horrible bête*, ainsi qu'il appelait le *gallicide*, ses méfaits de la nuit, et il exécuta de suite sa résolution en clouant sa capture sur une palissade.

Mais si le mille-pieds, en général, fait une guerre acharnée aux volailles, la guerre que celles-ci lui font à leur tour ne l'est pas moins. Dans toutes les contrées où sont des mille-pieds, les poules, avec leurs poussins, sont sans cesse à leur recherche, et, dès qu'elles en aperçoivent un quelque part, dans un coin, sous du bois ou quelque pierre, elles poussent leur cri d'alarme si connu. A ce cri, sorte de tocsin ou d'appel au secours, toutes les autres poules se rendent; puis, les unes et les autres réunies, elles se massent, se serrent, et marchent ainsi à l'ennemi, — à cet ennemi si cruel et si lâche, — si lâche, puisqu'il ne les attaque que la nuit, durant leur sommeil. La poule, le plus souvent, terrasse alors le mille-pieds; victime dans l'ombre, elle triomphe au grand jour. Une poule est belle à voir alors qu'étreignant, dans ses pattes, son nocturne égorgeur, elle l'accable sur la tête de coups de bec redoublés, — et le tableau sera complet si l'on voit en même temps des poussins imiter en tous points leur mère, dans cette juste représaille, avec les jeunes mille-pieds qu'ils auront eu, de leur côté, la bonne fortune de rencontrer. Or, cette rencontre ne doit pas être rare, car les jeunes mille-pieds, comme les poussins, marchent aussi à la suite de leur mère, et les familles de l'une et de l'autre espèce fréquentent les mêmes lieux. Que si l'insecte, dans les poursuites du volatile, ne trouve quelque refuge dans les accidents du sol, il cherchera à se glisser dans les plumes de son adversaire, et, s'il y parvient, les chances du danger auront changé; elles se seront retournées du côté du volatile, car l'insecte ne tardera pas à le saisir à la gorge, et l'on sait comme il procède alors pour achever sa victime.

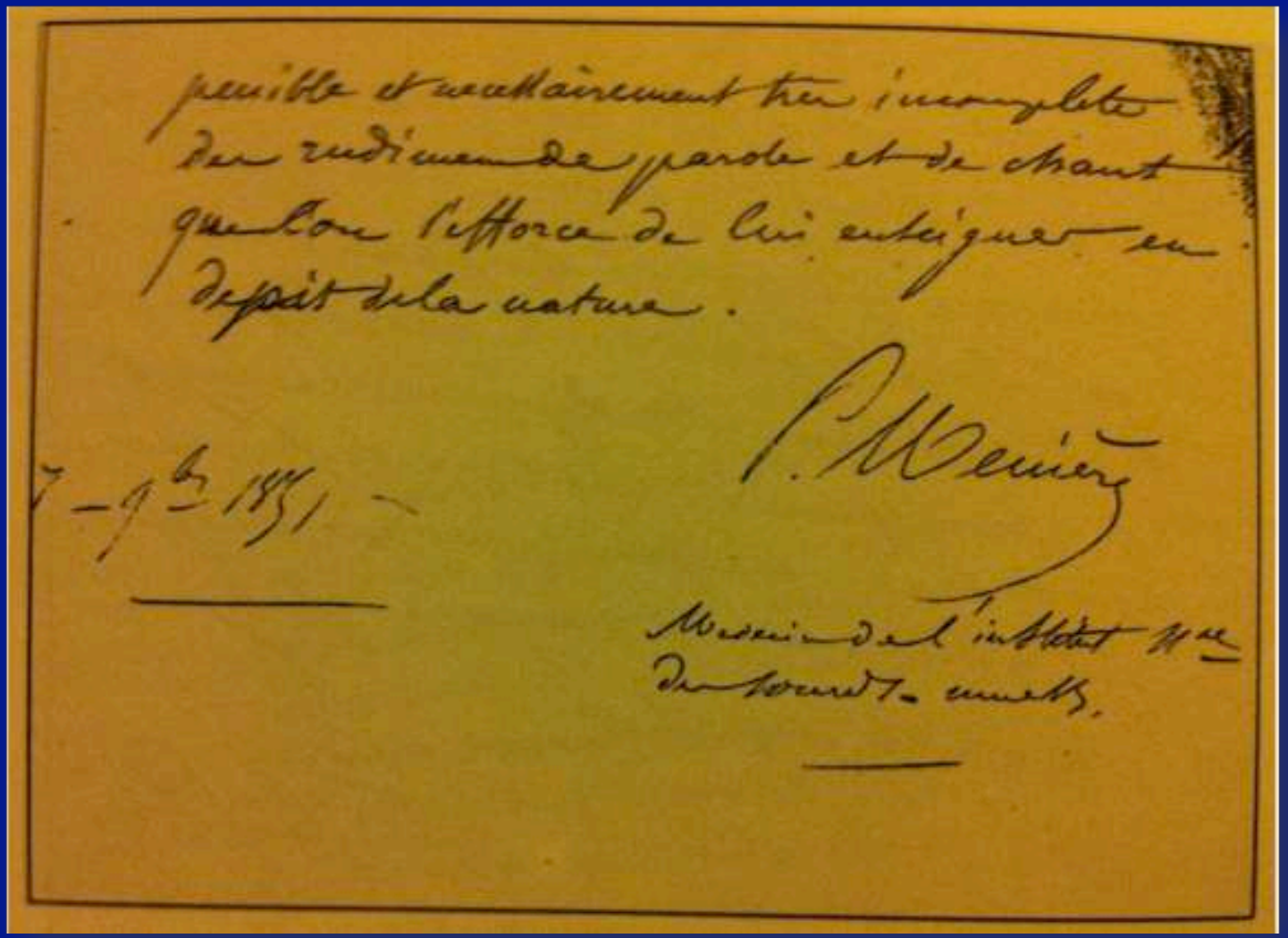
Disons ici ce que peut-être nous eussions dû dire plus tôt, à savoir que le mille-pieds suce, avec une avidité sans pareille, le sang de ses victimes, au fur et à mesure qu'il en divise les chairs.

Dans leurs combats entre eux, les mille-pieds ne sont pas moins terribles qu'envers les autres animaux. La victime est ordinairement le plus petit, lequel, par cela même, doit être le plus faible. Le vainqueur, dans tous les cas, est celui qui, le premier, est parvenu à s'implanter sur le dos de son adversaire, car, une fois là, il a bientôt atteint la gorge qu'il perfore comme chez les autres animaux. Un jour (26 décembre 1822), deux mille-pieds sont mis en présence dans un bocal en verre : l'un et l'autre, après avoir cherché comme à s'éviter, finissent par s'attaquer. Ils étaient, en apparence, d'égale force; le combat se prolongeait, et je ne pus en attendre la fin. Le lendemain, l'un des deux était mort; toute la partie antérieure du cou était rongée, dans l'étendue de près d'un pouce de longueur, et les organes correspondant à cette partie avaient entièrement disparu.

Morsure (1).

Cette morsure se présente sous la forme de deux piqûres plus ou moins

(1) Il existe sur les effets de la morsure des scolopendres ou mille-pieds



pénible et malheureusement très incomplète
des rudiments de parole et de chant
que l'on s'efforce de lui enseigner en
dépit de la nature.

P. Bonnier

7 – 9bre 1891 –

Membre de l'Institut 11 rue
des Saints-Pères.

Yamakawa, Kyoschiro: Über pathologische Veränderungen bei einem Menière-Kranken. (*Jahresvers. d. Japan. Oto-Rhino-Laryngol. Ges. [10. japan. med. Kongr.], Kyoto, Sitzg. v. 3.—4. IV. 1938.*) Z. Ot. usw. (Tokyo) **44**, dtsch. Zusammenfassung 181 bis 182 (1938) [Japanisch].

Bei einem Menière-Kranken mit vielen Anfällen von verschieden langer Dauer mit Zu- und Abnahme von Schwindel, Ohrensausen und Schwerhörigkeit konnte Verf. die Innenohren mikroskopisch untersuchen. Er fand Vorwölbung der Reissnerschen Membran nach der Scala vestibuli in allen Windungen, Konkrementbildung in der Stria vascularis, im Aquaeductus cochleae sowie im inneren Gehörgang. Das Lumen des Ductus und Saccus endolymphaticus erwies sich im Vergleich zur anderen Seite als eng und mit dicker Kolloidmasse gefüllt. Dagegen waren das Cortische Organ, die Nervenzellen des Ganglion spirale und vestibulare intakt, nur im Ductus cochlearis war Nubercula spärlich vorhanden. Verf. erklärt aus diesen Befunden den Anfall derart, daß die Sekretion der Stria vascularis durch den Konkrementreiz anfallsweise gestört wird, wodurch sich der Druck im Endolymphraum erhöht und die Funktion der Sinneszellen gestört wird. Durch Diffusion und Resorption der Endolymphe und Ausdehnung der Reissnerschen Haut wird der gesteigerte Druck ausgeglichen und die Sinneszellen wieder funktionsfähig. Wie die Nubecula im Duct. cochl. und die Kolloidmasse im Duct. und Saccus endolymphaticus zeigen, wird die vermehrte Lymphe dicker. Die Ursache der Konkrementbildung ist unklar, vielleicht auf die Blutbeschaffenheit zurückzuführen.

H. Beyer (Berlin).

Yamakawa K, aprile 1938

Section of Otology

President—F. J. CLEMINSON, M.Chir.

[*May* 6, 1938]

Observations on the Pathology of Ménière's Syndrome

By C. S. HALLPIKE, F.R.C.S.,[1] and H. CAIRNS, F.R.C.S.

(Ferens Institute of Otology, Middlesex Hospital, and Department of Neurosurgery, London Hospital)

IT is the purpose of this paper to describe—as is believed, for the first time—the pathological changes in the temporal bones in two cases of Ménière's syndrome. In both death occurred shortly after operation for section of the 8th nerve. For the purposes of the present paper, the surgical factors concerned in the operative failure of these cases will be dealt with only in so far as they are related to the interpretation of the histological changes which are now to be described. Their detailed consideration will be reserved for a further publication.

Hallpike CS & Cairns H. Proc Roy Soc Med 1938; 31: 1317-36

La prima neurootologia: i riflessi vestibolospinali e i relativi tests

Moritz Heinrich Romberg

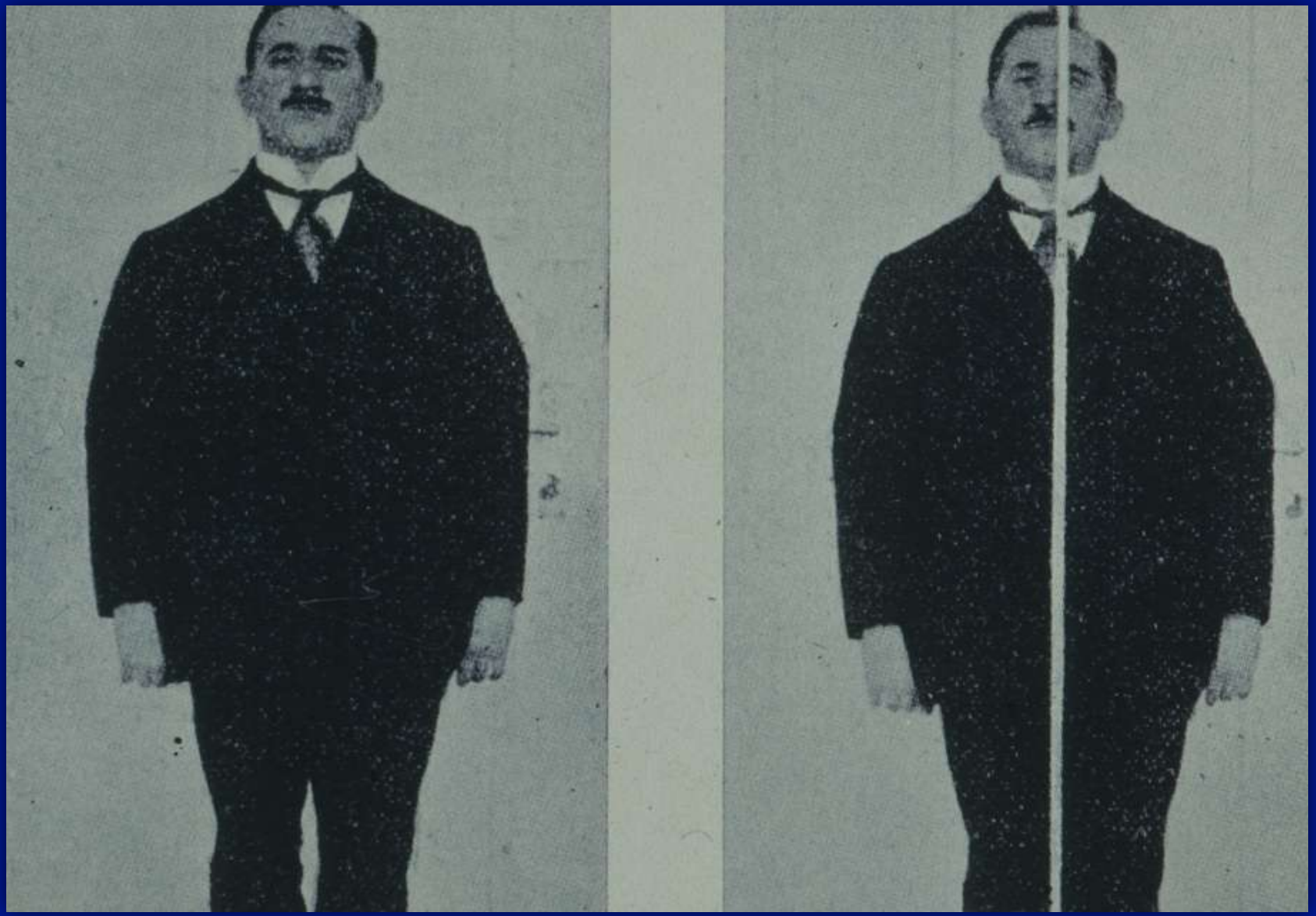
Valutazione dell'equilibrio (riflesso vestibolo spinale)

1840 Prova di Romberg

1910 Prova delle braccia tese (Barany)

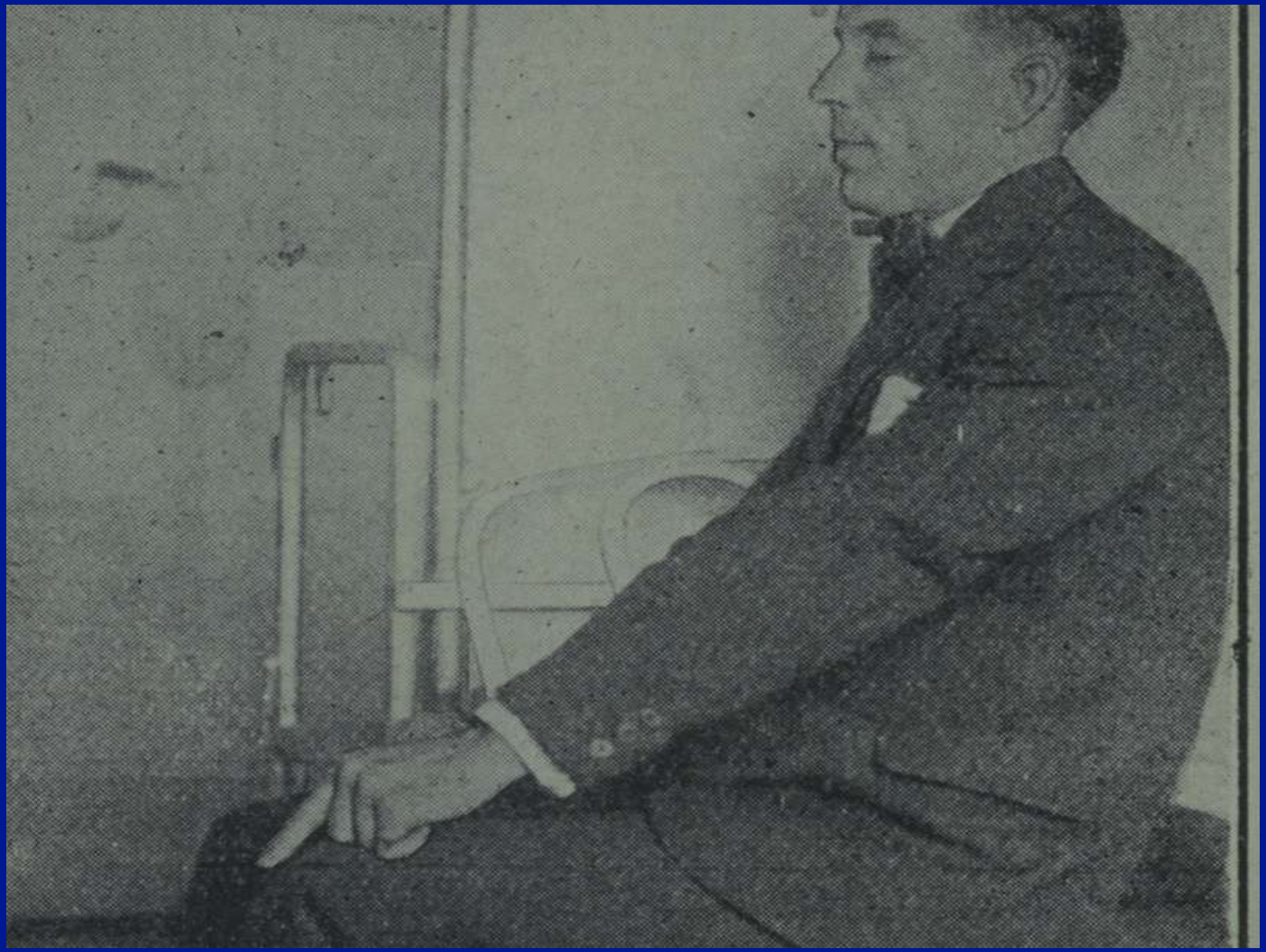
Marcia a stella di Babinski - Weil

Stepping test di Fukuda



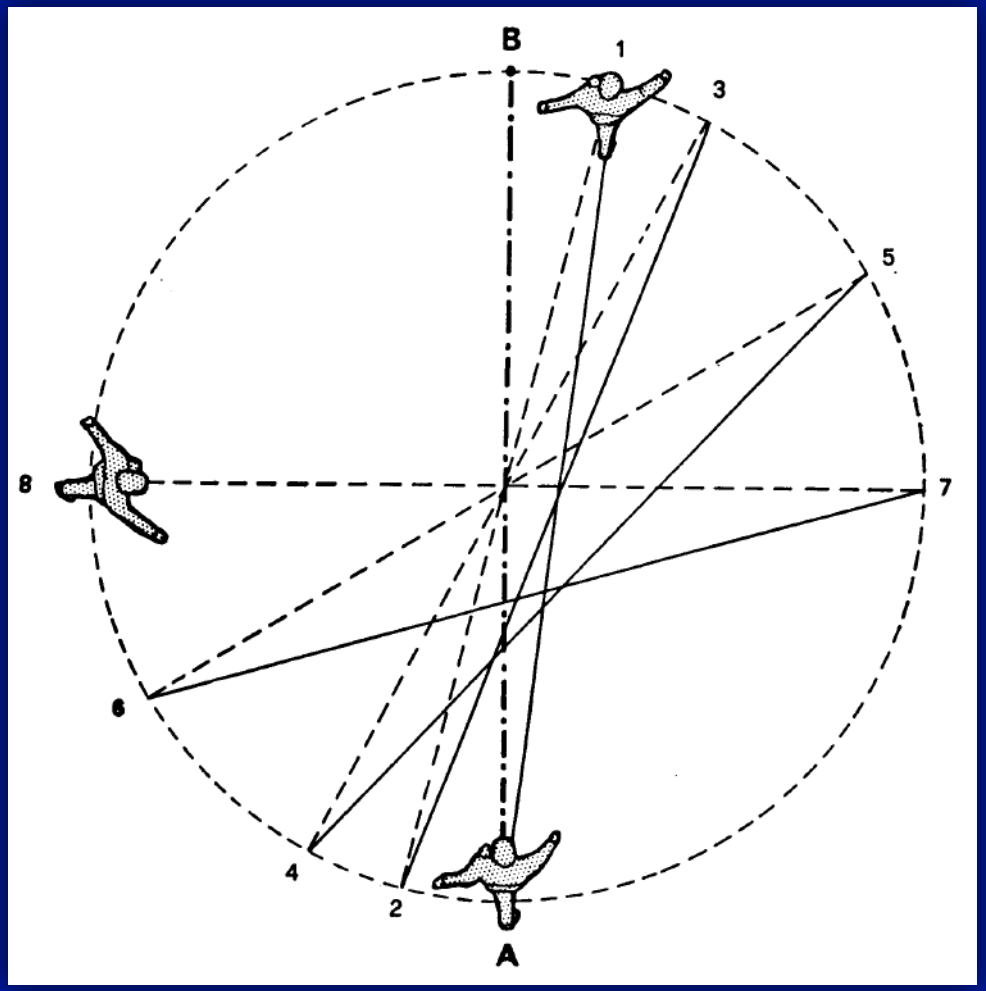
Prova delle braccia tese o degli indici di Barany

Si pone il paziente seduto, con le braccia e gli indici estesi in direzione anteriore, in asse con quelli dell'esaminatore che funge da controllo

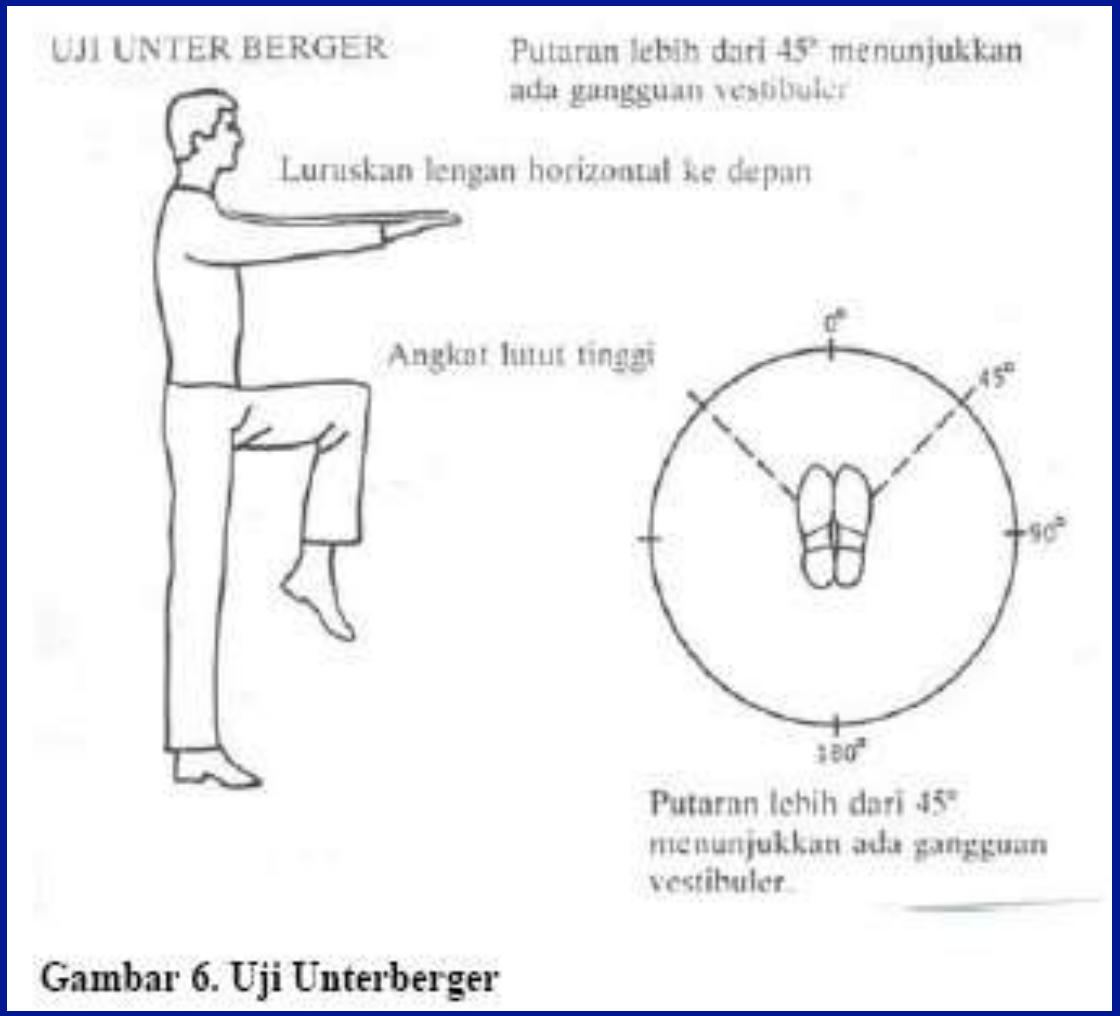


Prova della marcia a stella o di Babinski-Weil

Il paziente viene invitato a camminare avanti e indietro senza voltarsi, ad occhi chiusi per almeno 5 metri, eliminando ogni stimolo acustico o visivo che possa permettere eventuali correzioni.



J Babinski, GA Weil
1913



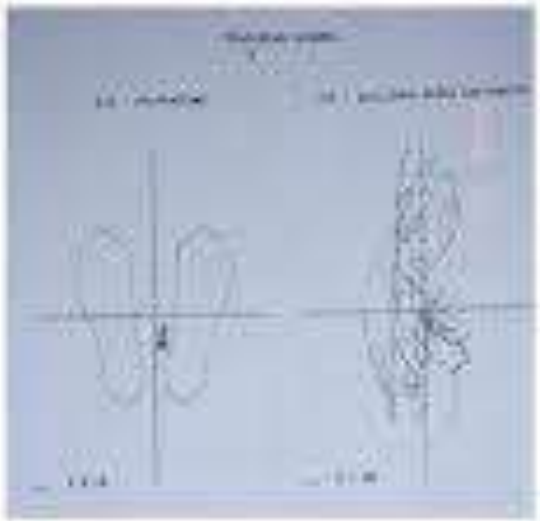
Gambar 6. Uji Unterberger

S Unterberger
1939

T Fukuda
1959

STABILOMETRIA STATICA

Il sistema stabilometrico statico è deputato alla valutazione della capacità di controllo posturale di un soggetto immobile in stazione eretta

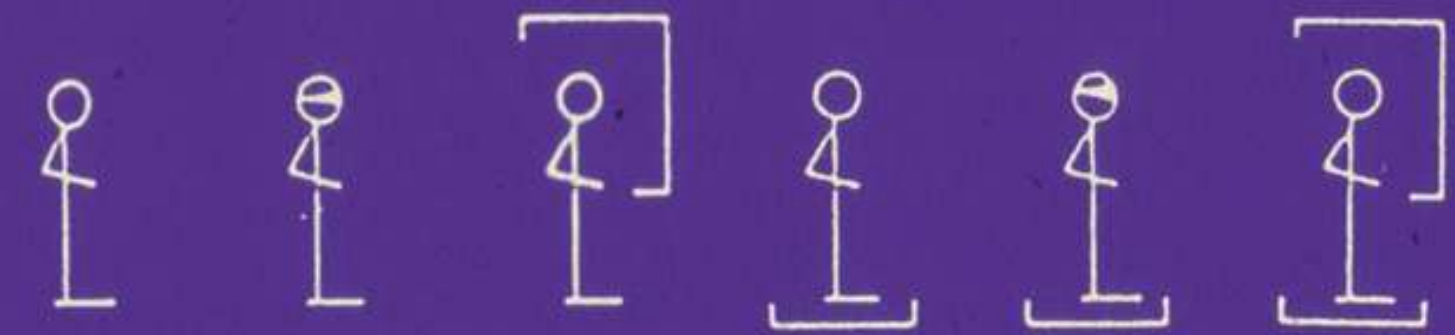


Anni ‘60

La posturografia dinamica e l’ Equitest

Nashner LM et al. Adaptation to altered support and visual conditions during stance: patients with vestibular deficit. J Neurosci 1982; 2: 536-44

Nashner LM. Analysis of movement control in man using the movable pkatform. Adv Neurol 1983; 39: 607-19



condizioni sensoriali	1	2	3	4	5	6
conflitto sensoriale	NO	NO	SI	SI	SI	SI
informazioni sensoriali corrette	VEST VISIONE SOMATO	VEST SOMATO	VEST SOMATO	VEST VISIONE	VEST	VEST
non corrette	NESSUNA	NESSUNA	VISIONE	SOMATO	SOMATO	VISIONE SOMATO

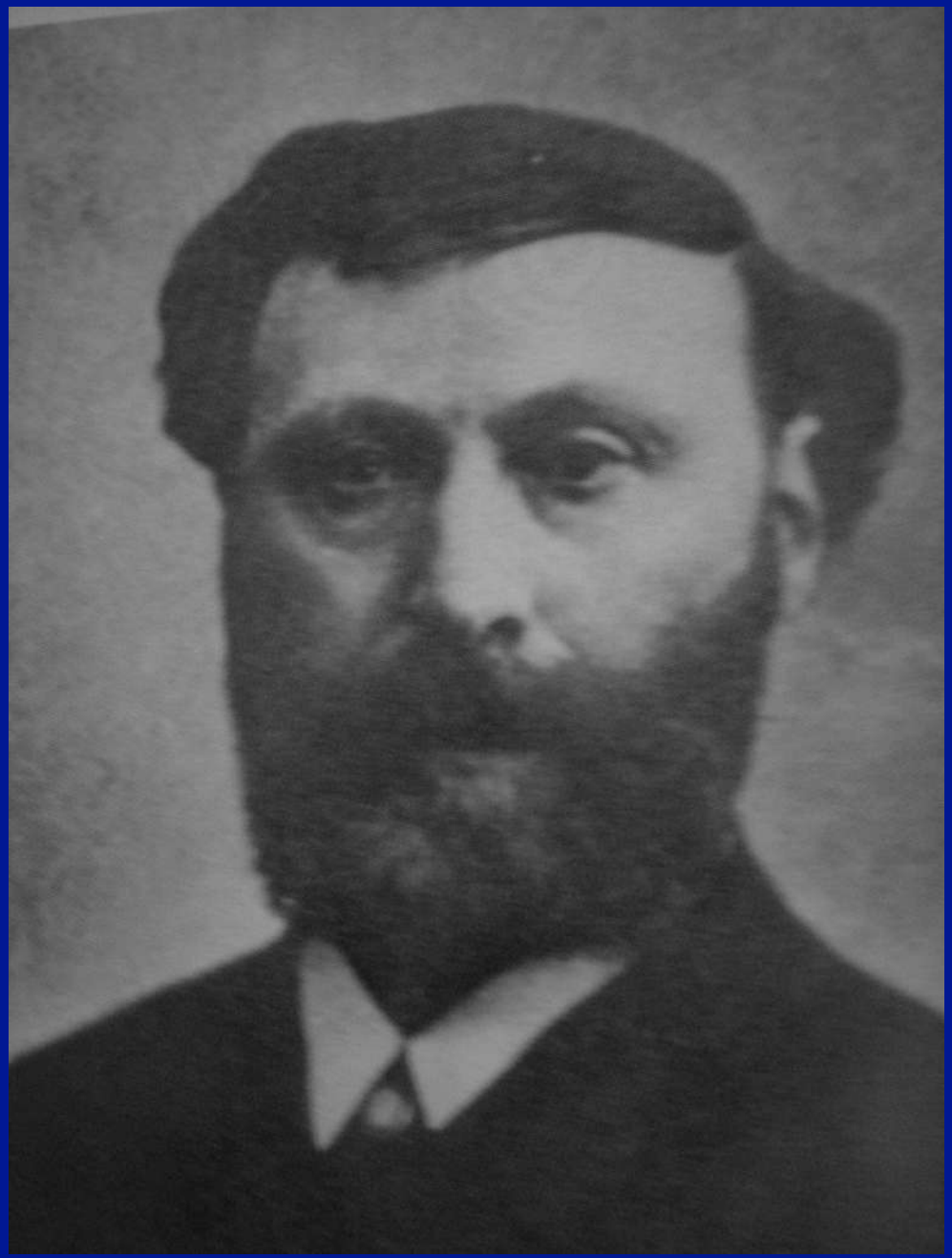
Anni ‘80

La Scuola di Vienna: il VOR, il nistagmo e le stimolazioni labirintiche

Adam Politzer (1835 – 1920)

Uno dei padri della moderna otologia e otochirurgia. La sua Scuola di Vienna si occupò di tutte le problematiche di anatomia, fisiologia e patologia dell' orecchio medio e interno.

Tra gli allievi Barany, Alexander, Ewing, Frenzel e altri nomi illustri della storia della vestibologia.



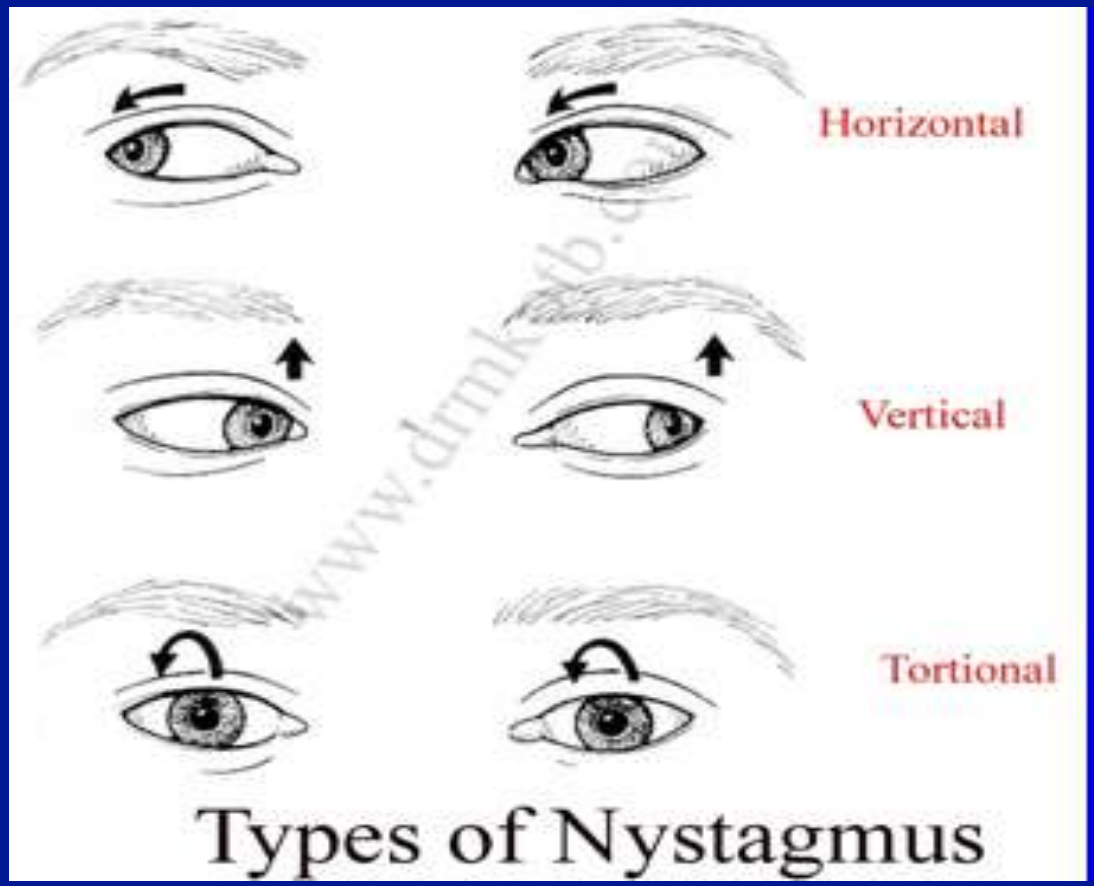
Adam Politzer (1835-1920)

Osservazione strumentale del nistagmo con abolizione o riduzione della fissazione

1906. Occhiali con vetri opachi (Barany)

1910. Occhiali con lenti di 20 diottrie (Bartels)

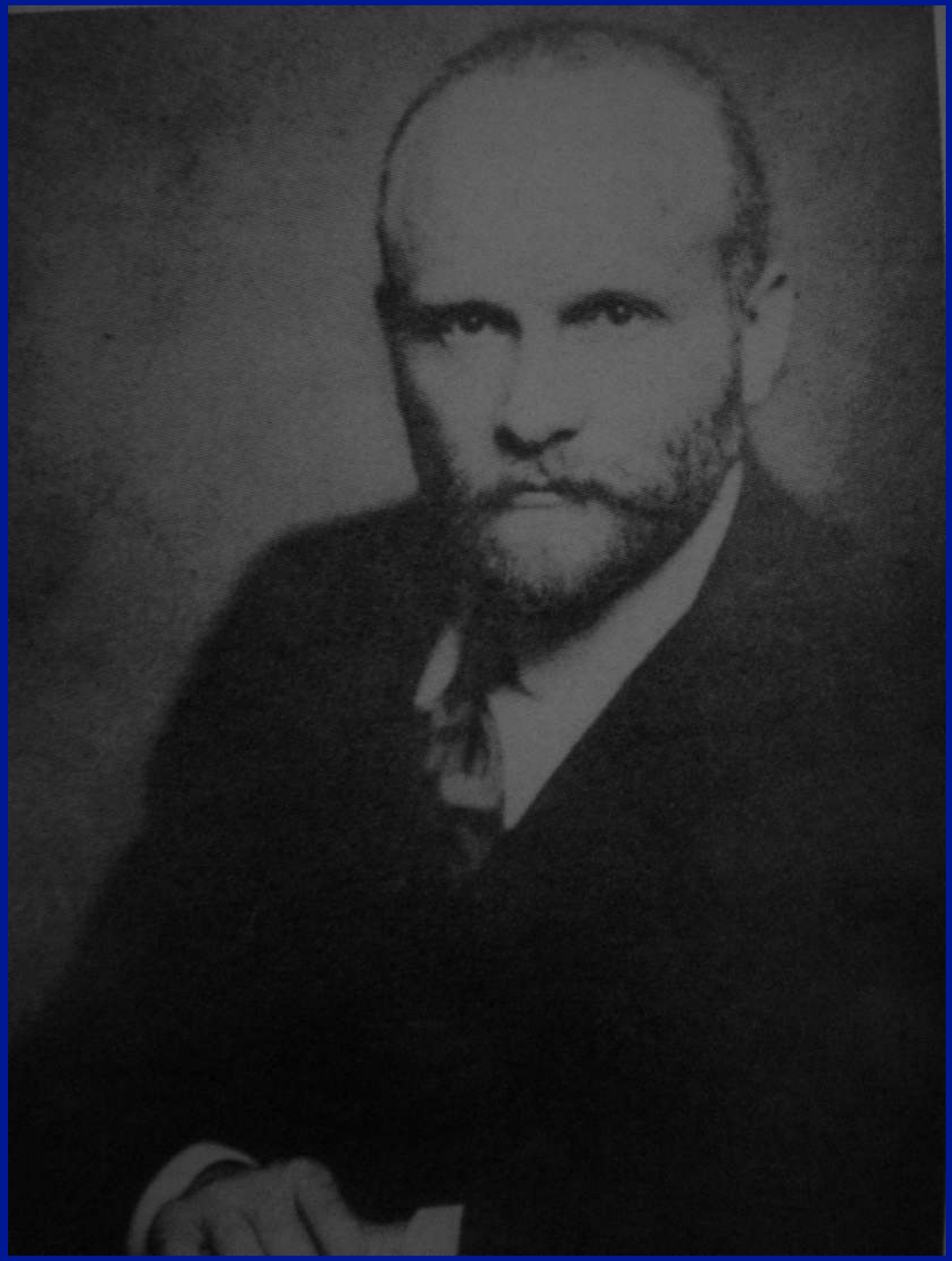
1935. Occhiali di Frenzel



Hermann Frenzel (1895-1967) e i suoi occhiali(1935)

Robert Bàràny (1876 – 1936)

Allievo della scuola di Politzer a Vienna, si occupò di tutte le problematiche di fisiologia, patologia e clinica del sistema vestibolare, periferico e centrale. Padre della stimolazione termica e rotatoria del labirinto. Durante la prigionia in Russia nella prima guerra mondiale, ricevette il premio Nobel. Liberato grazie alla CRI, passò in Svezia dove ottenne la cattedra di otorinolaringoiatria ad Uppsala.



Robert Barany (1876 – 1936)

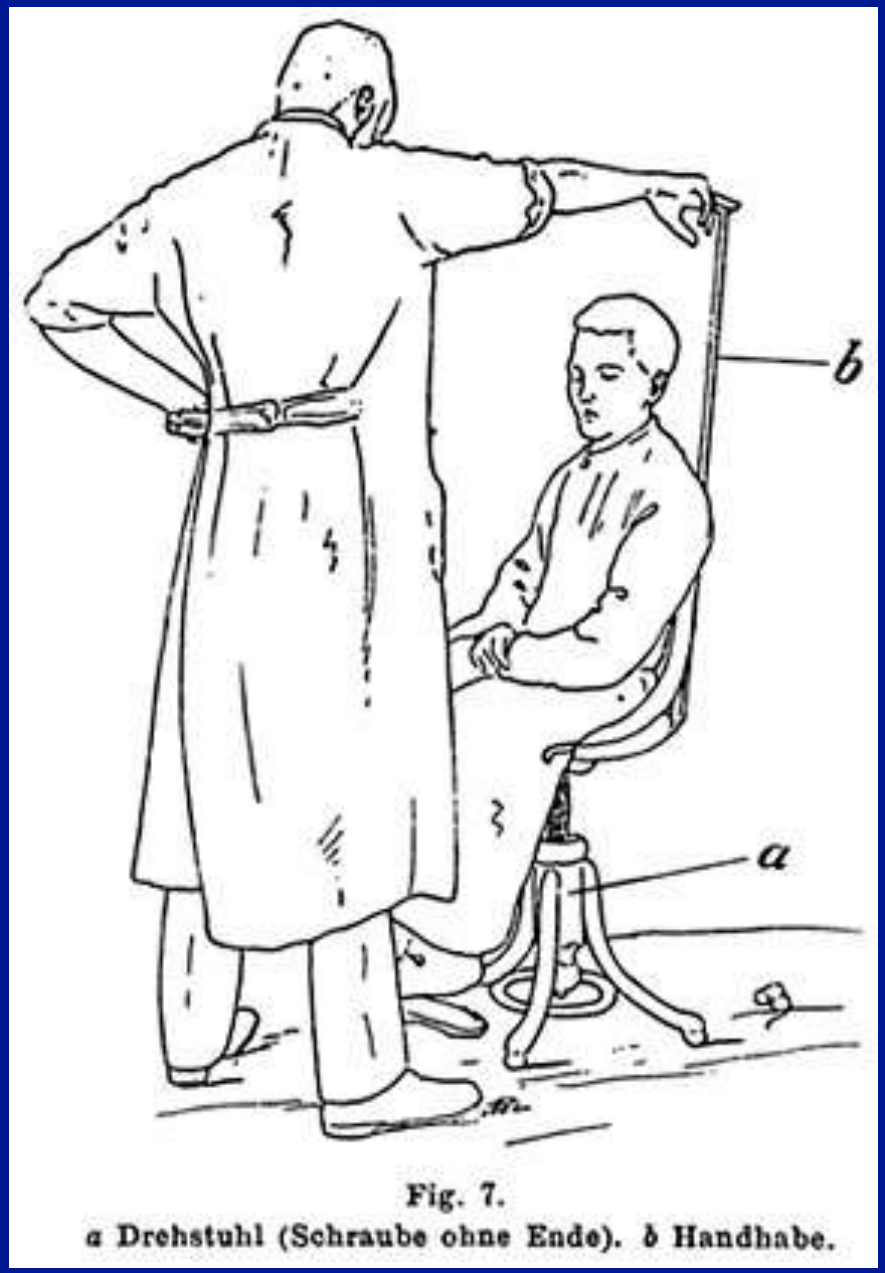
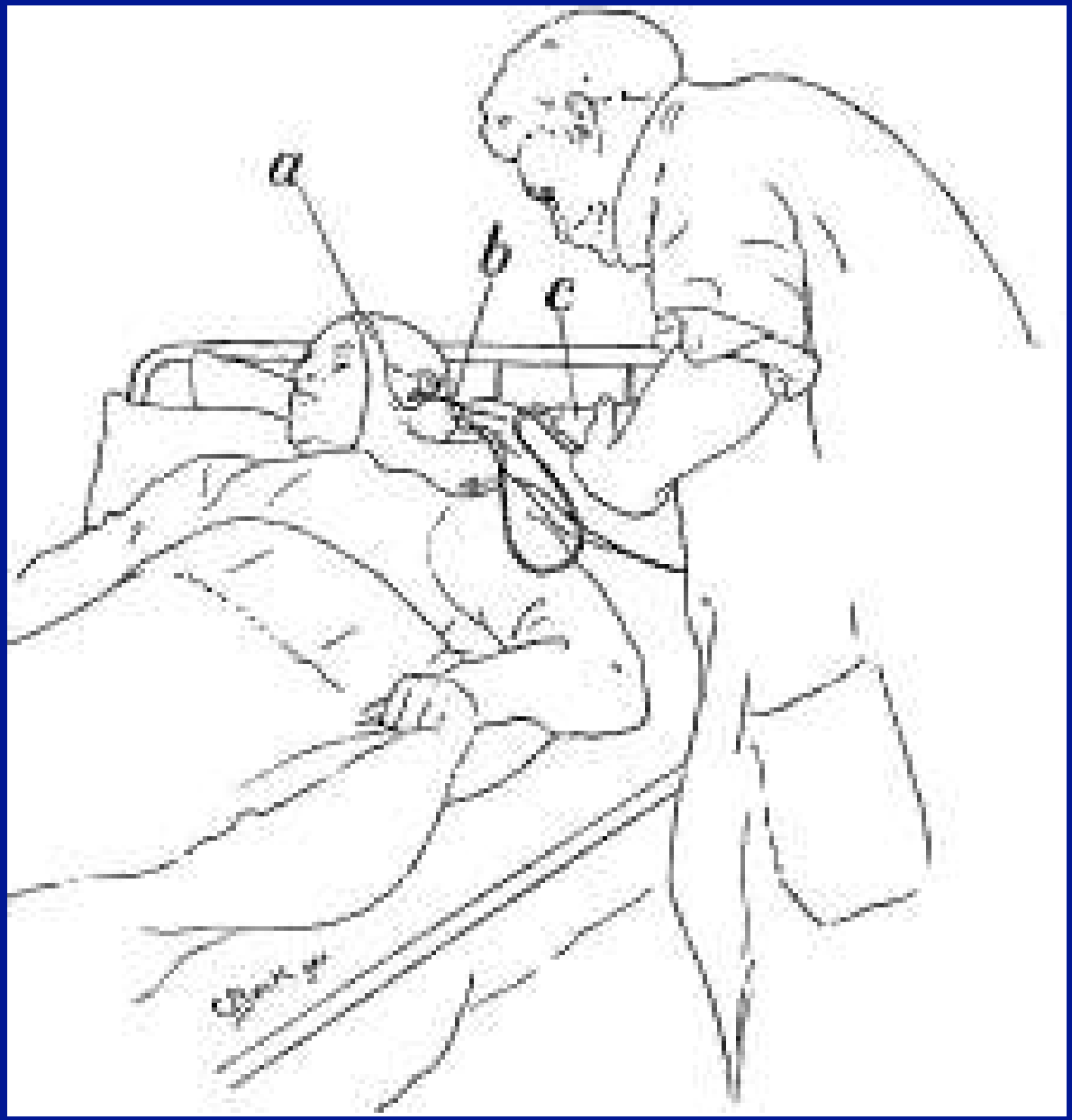


Fig. 7.
a Drehstuhl (Schraube ohne Ende). b Handhabe.


a
b
c

La registrazione e lo studio del nistagmo

La registrazione del nistagmo Prima fase: metodi meccanici ed ottici

Metodi meccanici a leva (Tuyl 1901, Ohm 1914)

Metodi meccanici pneumatici (Buys 1909, Wotzilka 1924, Grunberg 1924)

Metodi ottici (Dodge e Kline 1901,Wojatschek 1908, Dohlman 1925)

usati dagli altri AA., fissò una delle estremità della leva non più sull'occhio, ma su una *capsula di gomma* a diretto contatto col bulbo oculare.

Nel 1891 BERLIN si servì, invece, di una coppa di avorio che aderiva alla superficie dell'occhio, precedentemente cocainizzata, e portava fissata una setola di circa 6 cm. di lunghezza che serviva a riprodurre i movimenti oculari sulla faccia concava di un vetro d'orologio. BERLIN stesso riferiva che tale metodo non era idoneo per una buona registrazione a causa della eccessiva flessibilità della setola.

NIEDEN, per studiare il nistagmo dei minatori, usò pure il metodo meccanico, ma non ottenne tracciati migliori di quelli di BERLIN.

EWALD, per le sue ben note ricerche sul nistagmo vestibolare nei piccioni, preferì infilare un ago direttamente nel centro della cornea. Applicando all'altra estremità libera un filuzzo di paglia, egli riuscì a registrare su un cilindro i movimenti oculari.

Il sistema usato in precedenza da BERLIN, cioè quello di una *coppa appoggiata sull'occhio* venne perfezionato in seguito da DELABARRE nel 1898. Per evitare gli errori di registrazione dovuti ai facili spostamenti della coppa sulla superficie del bulbo, l'A. pensò di costruire delle coppe di gesso perfettamente adattate alla forma del bulbo oculare. Sulla superficie convessa veniva infilata un'asticciola, che riportava su un tamburo ruotante i movimenti oculari. Anche tale metodo presentò dei gravi inconvenienti: si osservarono fenomeni di irritazione e di infiammazione della congiuntiva per

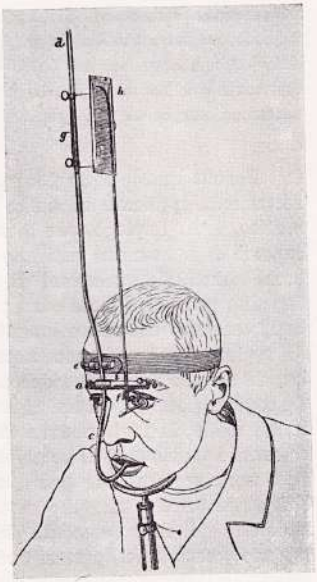


FIG. 6
Schema dell'apparecchio di TUYL.

messo di raggiungere dei risultati migliori, costruì un nuovo nistagmografo basato sul principio della registrazione pneumatica. Egli si servì di una specie di relais costituito da tre capsule di MAREY, ottenendo in tal modo un notevole ingrandimento del movimento oculare.

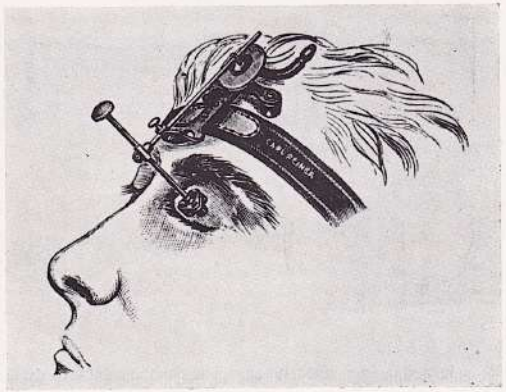


FIG. 11

Metodo di registrazione del nistagmo sec. WOTZILKA

Anche l'apparecchio nistagmografico di GRÜNBERG e di WOTZILKA, è basato sugli stessi principi di quello di BUYS. Questi AA. sfruttarono, per la registrazione dei movimenti oculari, il principio che in meccanica viene chiamato « Knaggensteurung ». La leggera pressione esercitata dalla cornea durante i movimenti oculari, crea delle variazioni di volume dell'aria contenuta nella capsula pneumatica che vengono trasmesse ad uno sfigmomanometro per la registrazione (fig. n. 11).

GRÜNBERG abbandonò pertanto l'impiego dell'apparecchio di registrazione a leva (illustrato nella figura n. 12) adottato nelle prime ricerche di nistagmografia. Egli, infatti, comprese che la registrazione dei movimenti oculari ottenuta con l'apparecchio a leva, presentava numerosi svantaggi nei confronti di quella ottenuta, col metodo pneumatico.

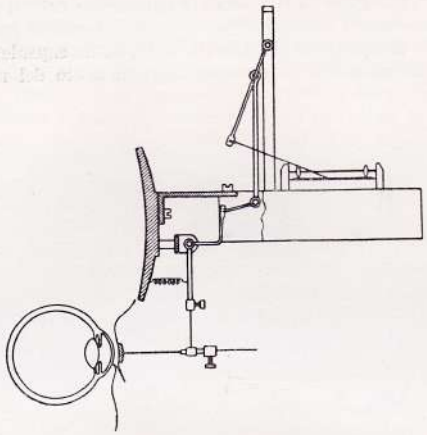


FIG. 12

Apparecchio a leva per la registrazione del nistagmo adottato da GRÜNBERG.

In sintesi possiamo ritenere che sia il metodo meccanico a leva che il metodo pneumatico non hanno permesso, malgrado i numerosi miglioramenti apportati dai singoli AA., di raggiungere quel grado di esattezza nella registrazione che gli stessi AA. desideravano. Le fonti d'errore consisterano proprio nel principio tecnico del metodo e per questo non poterono venire completamente eliminati.

II. - METODO OTTICO

Questo metodo di registrazione venne impiegato da numerosi AA. allo scopo di eliminare, per quanto fosse possibile, o di ridurre, gli inconvenienti che, nelle tecniche precedenti, avevano impedito una esatta registrazione dei movimenti oculari.

Il metodo di registrazione per via ottica avrebbe innanzitutto permesso di eliminare :

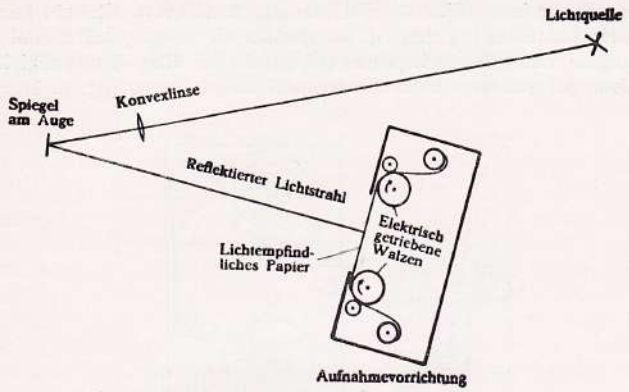


FIG. 14
Schema dell'apparecchio cinematografico di DOHLMAN.

chio del malato. Egli impiegò per le sue ricerche un apparecchio basato sul principio della registrazione *ottica* (fig. n. 14).

Sulla superficie oculare, DOHLMAN fissò una capsula da suzione di gomma sulla cui estremità era applicato uno specchietto convenientemente illuminato da una sorgente luminosa. La superfice dello specchio veniva opportunamente cambiata di posizione per registrare i movimenti oculari nei vari piani di rotazione (fig. n. 15). Nel 1934, KUILMAN costruì un apparecchio i cui principi tecnici erano simili a quelli dell'apparecchio usato da STRUYCHEN. Egli però perfezionò il sistema di registrazione, apportandovi alcune modifiche tali da permettere : *a)* la registrazione contemporanea della *componente orizzontale, rotatoria, verticale* di una scossa nistagmi-

FIG. 15
Capsula di gomma usata per la registrazione del nistagmo col metodo ottico di DOHLMAN.

L’ elettronistagmografia (1930-1970)

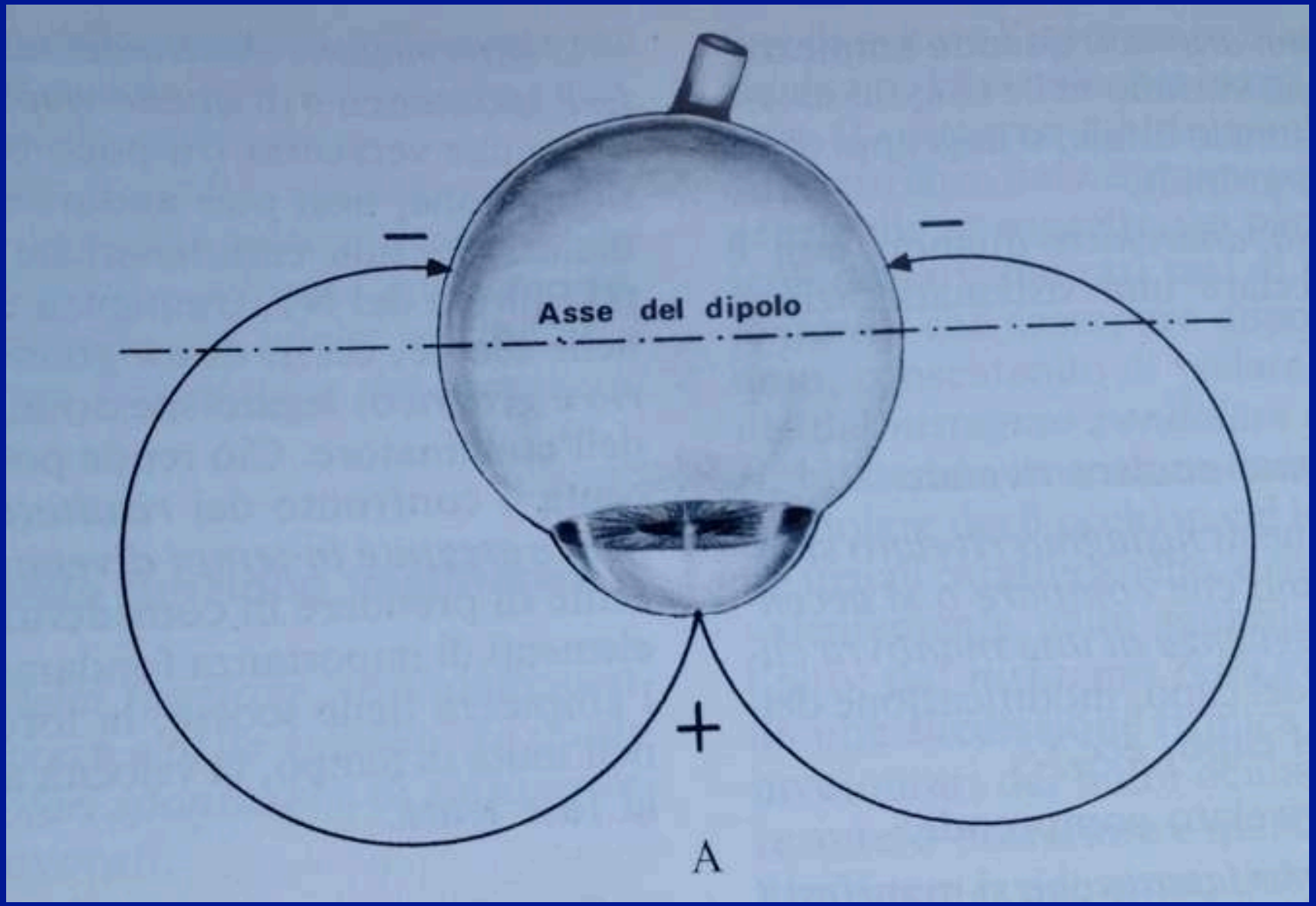
Schott E. Dtsch Arch Klin Med 1922; 140: 79-90

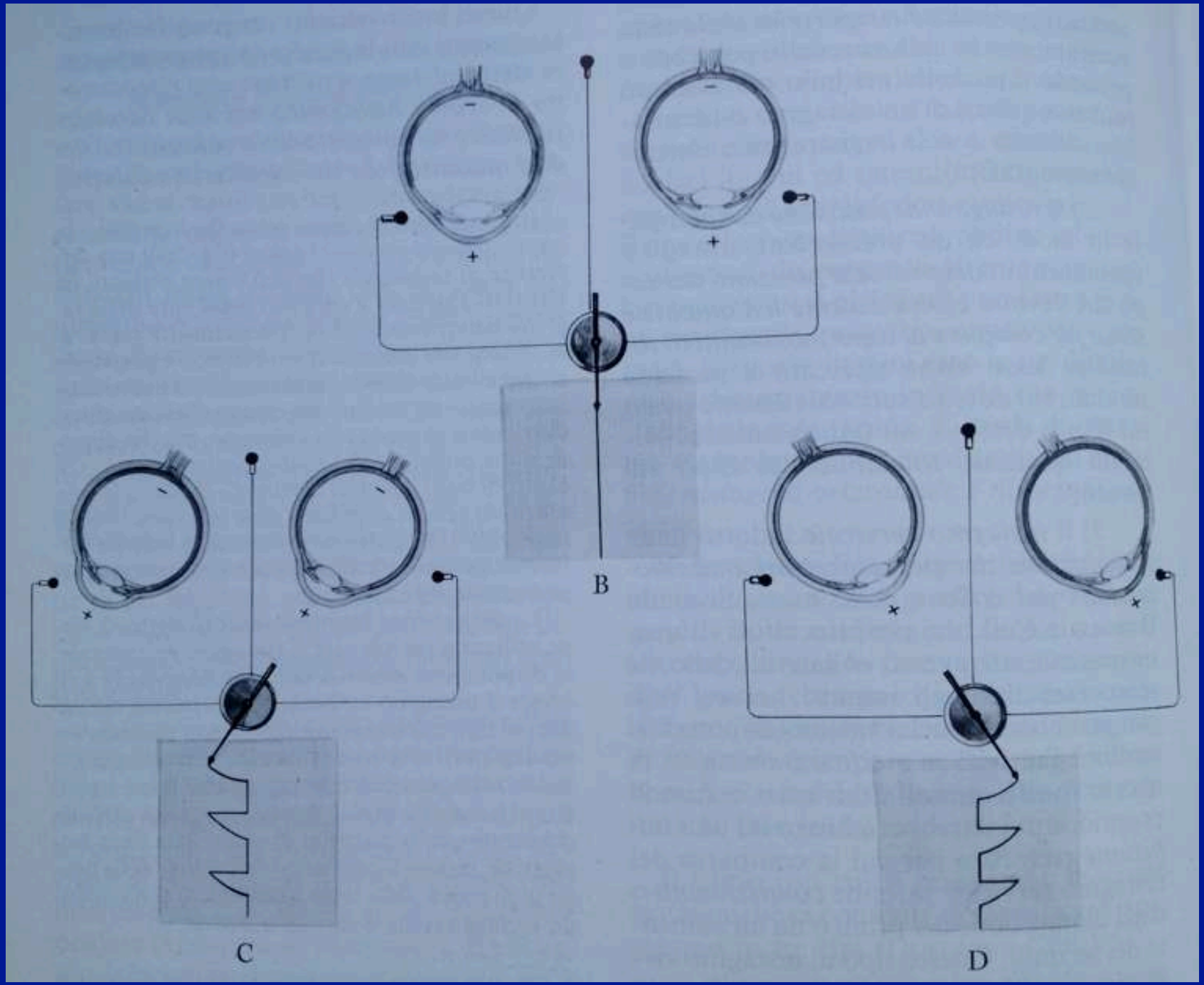
Meyers IL. Arch Neurol 1929; 21: 901-8

Mowrer OH et al. Am J Physiol 1936; 114: 423

Jung R. Arch Ohr usw Heilkh 1939; 18: 21-4

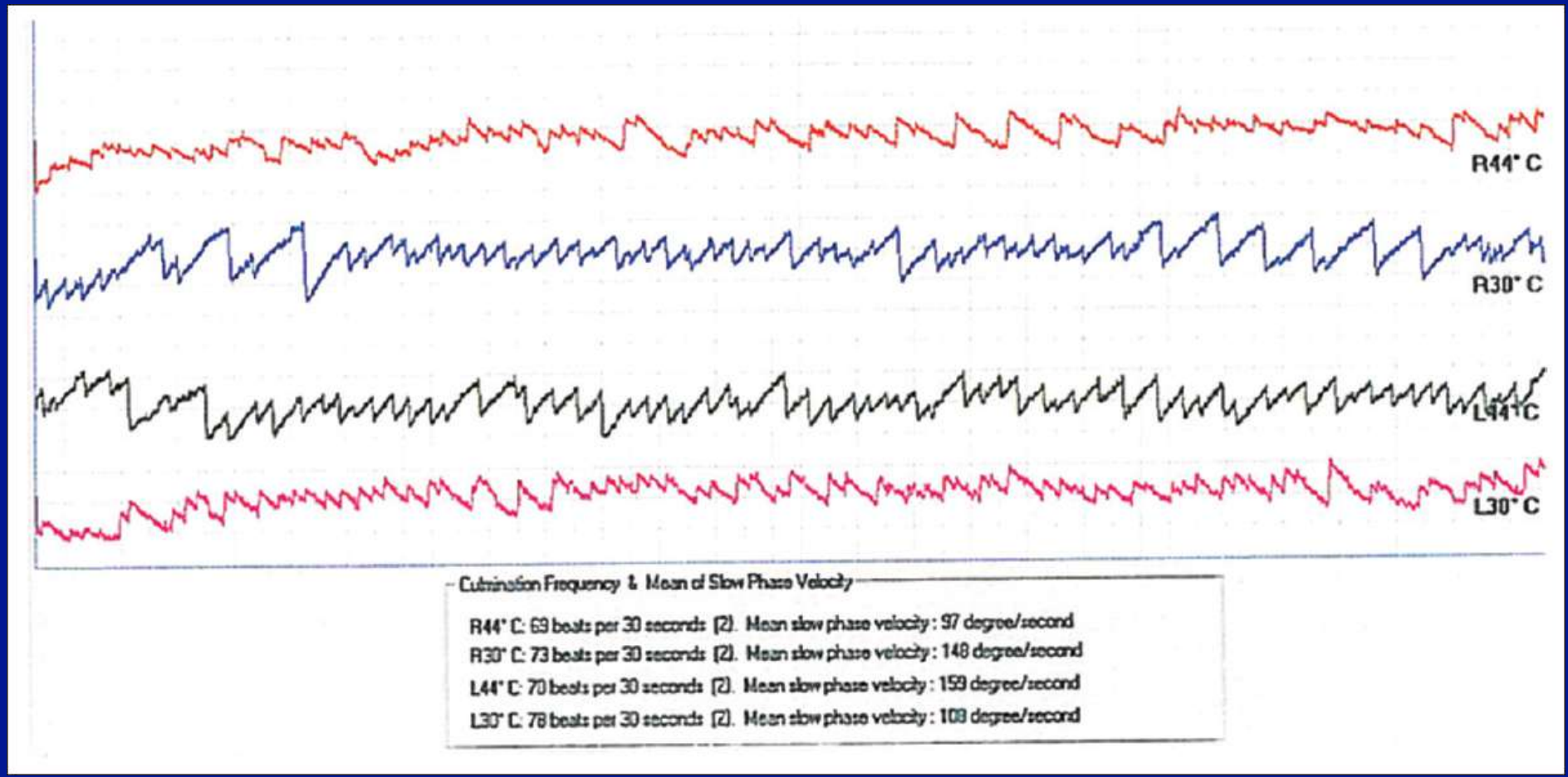
L’ occhio come dipolo corneo-retinico




B
C
D

Otological Research Unit
National Hospital
Queen Square, Londra
Anni ‘60

Il tracciato nistagmografico



I problemi tecnici dell'elettronistagmografia

Elettrodi:

quanti ? dove posizionati ? quale pasta conduttrice ?

Amplificazione del segnale:

corrente continua (DC) o corrente alternata (AC) ? quale costante di tempo ?

Registrazione:

carta termosensibile o penna ad inchiostro ? quale velocita' di scorrimento ?

L’ analisi morfologica del nistagmo

Scosse decapitate

Onde quadre

Intercisioni saccadiche

Pause

Petite ecriture

Nistagmo a ressort

I parametri quantitativi della risposta nistagmica

La durata, il numero delle sosse

La Gesamtamplitude: Jung R, Toennies JF. Klin Wschr 1948; 21: 513-21

La Slow Phase Velocity: Henriksson NG. Acta Otolaryngol (Stockh) 1955; 45: 25-41

La Culmination Frequency: Torok N. Acta Otolaryngol (Stockh) 1957; 48: 530-5

La Slow Cumulative Eye Position: Anzaldi E, Mira E. Acta Otolaryngol (Stockh) 1975; 80: 120-7

globale percorso dai bulbi oculari durante l'intera reazione nistagmica. Essa è un elemento di estrema importanza nella valutazione della risposta vestibolare, ancora più del numero delle scosse e della durata della reazione stessa.

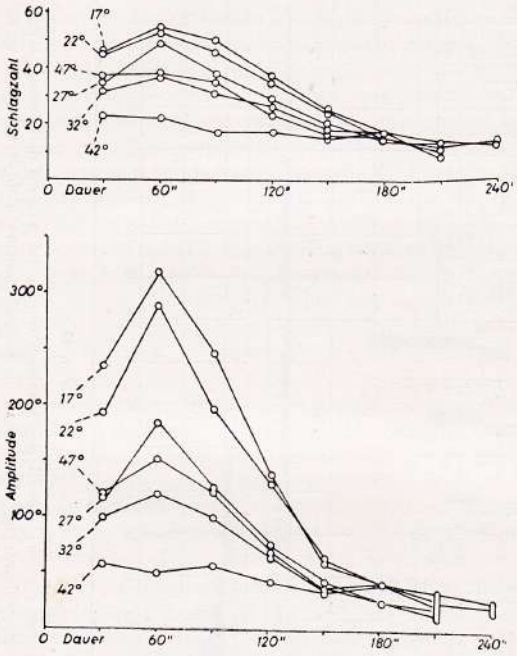


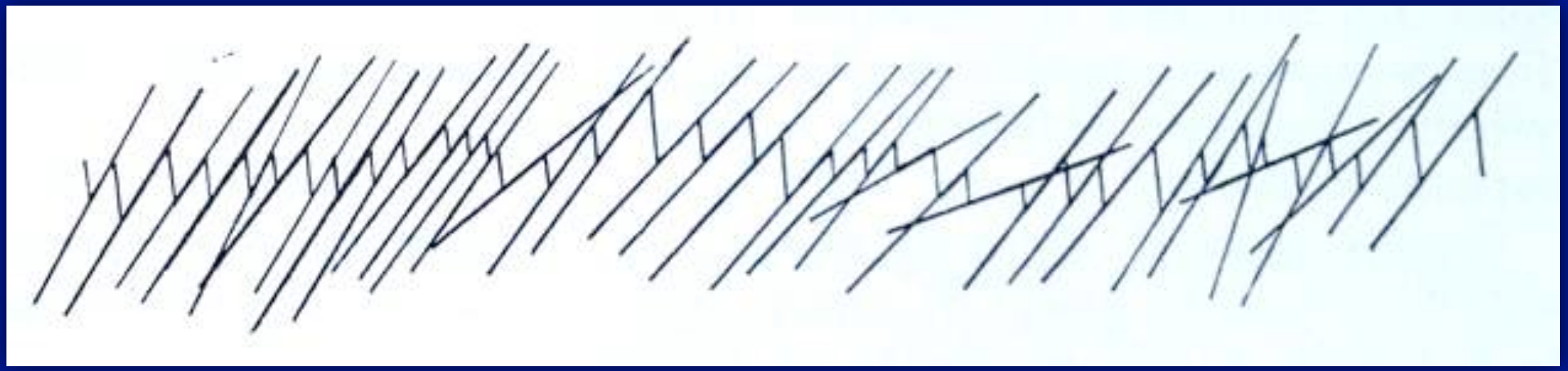
FIG. 45

Nel primo diagramma sono riportati i valori medi del numero delle scosse (Schlagzahl), e nel secondo diagramma quelli dell'ampiezza totale (Amplitude) calcolati in periodi di 30". Variando il valore fisico dello stimolo termico si ottengono corrispondenti modificazioni nei valori del numero e soprattutto dell'ampiezza delle scosse. Gli indici maggiori vengono raggiunti nei primi 90" della reazione nistagmica con punte massime al 60mo sec. (da MITTERMAIER).

1948

La derivata di Heriksson (1955)

La velocita’ angolare della fase lenta come parametro piu’ signficativo del nistagmo



Tra gli AA. che hanno adottato il metodo elettronico per il prelievo del potenziale del dipolo corneo-retinico, merita un particolare cenno HENRIKSSON: questo A. ha proposto e realizzato la registrazione della derivata nella curva nistagmica invece del comune nistagmogramma.

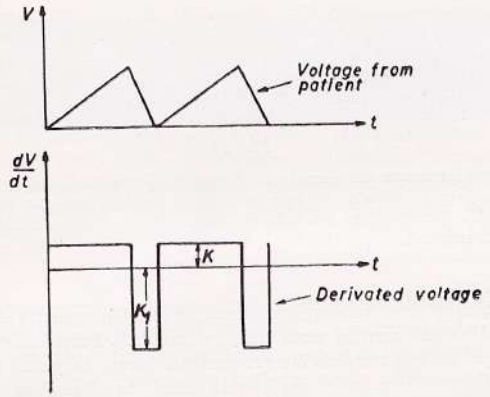


FIG. 23

Nel tracciato superiore sono disegnate le scosse del nistagmo registrato con il metodo comune. Nel tracciato inferiore sono riprodotte le stesse scosse come appaiono nel tracciato « derivato » (da HENRIKSSON).

Per realizzare questo tipo di tracciato egli si è valso di due accorgimenti tecnici. In primo luogo ha adottato una costante di tempo tale da consentire una rapidissima deviazione della penna scrivente negli istanti in cui il bulbo oculare inverte il suo moto di rotazione. In secondo luogo ha ridotto la velocità di scorrimento della carta a 0,25 cm/sec., velocità che consentiva il massimo accostamento possibile delle ordinate del tracciato.

Nel tracciato superiore della fig. n. 23 sono riprodotte delle scosse nistagmiche come vengono comunemente registrate, mentre in quello inferiore sono riprodotte col metodo della « derivata » secondo HENRIKSSON. Si nota che i segmenti a pendenza costante

1955

corrispondenti alla fase lenta e rapida, evidenti nel primo tracciato, vengono trasformati nel tracciato « derivato » in segmenti paralleli all'asse dell'isoelettrica .

Gli apici delle scosse riprodotti sul tracciato comune vengono resi in quello « derivato » con segmenti perpendicolari all'asse della

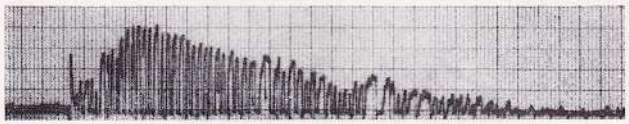


FIG. 24

Nistagmo provocato da stimolo acceleratorio (1ª fase). Come appaiono sul tracciato « derivato » le fasi lente delle scosse. L'ampiezza del grafico indica il comportamento della vel. ang. della fase lenta del nistagmo (da HENRIKSSON).

isoelettrica, perchè in corrispondenza di essi la velocità di rotazione del bulbo è zero (il moto infatti si inverte). Poichè la costante dei tempi dell'apparecchio è estremamente bassa, la penna ritorna in modo immediato sulla isoelettrica, anzi, la oltrepassa, perchè la pendenza della fase rapida è di segno opposto a quella della fase lenta. Nel tracciato così ottenuto i segmenti orizzontali corrispondenti alle due fasi vengono notevolmente accorciati con l'impiego di una ridotta velocità di scorrimento della carta per cui i valori della velocità angolare della fase lenta sono espressi dalle ordinate rispetto all'asse della isoelettrica.

Ne deriva che se la velocità della carta è molto ridotta, le due fasi sul tracciato « derivato » vengono a corrispondere praticamente a tanti punti che saranno al di sopra (fasi lente) e al di sotto dell'isoelettrica (fasi rapide).

La distanza degli apici della curva « derivata » dalla linea isoelettrica esprime il valore della velocità angolare che viene pertanto letta sull'asse delle ordinate del tracciato.

HENRIKSSON, HAMERSMA, FRENCKNER e PREBER hanno adottato anche un sistema onde poter eliminare la derivata della fase rapida, per cui sul tracciato viene soltanto registrata quella della fase lenta delle scosse (fig. n. 24).

1955

L’ evoluzione dell’ analisi del nistagmo: conversione analogico/digitale e uso del computer

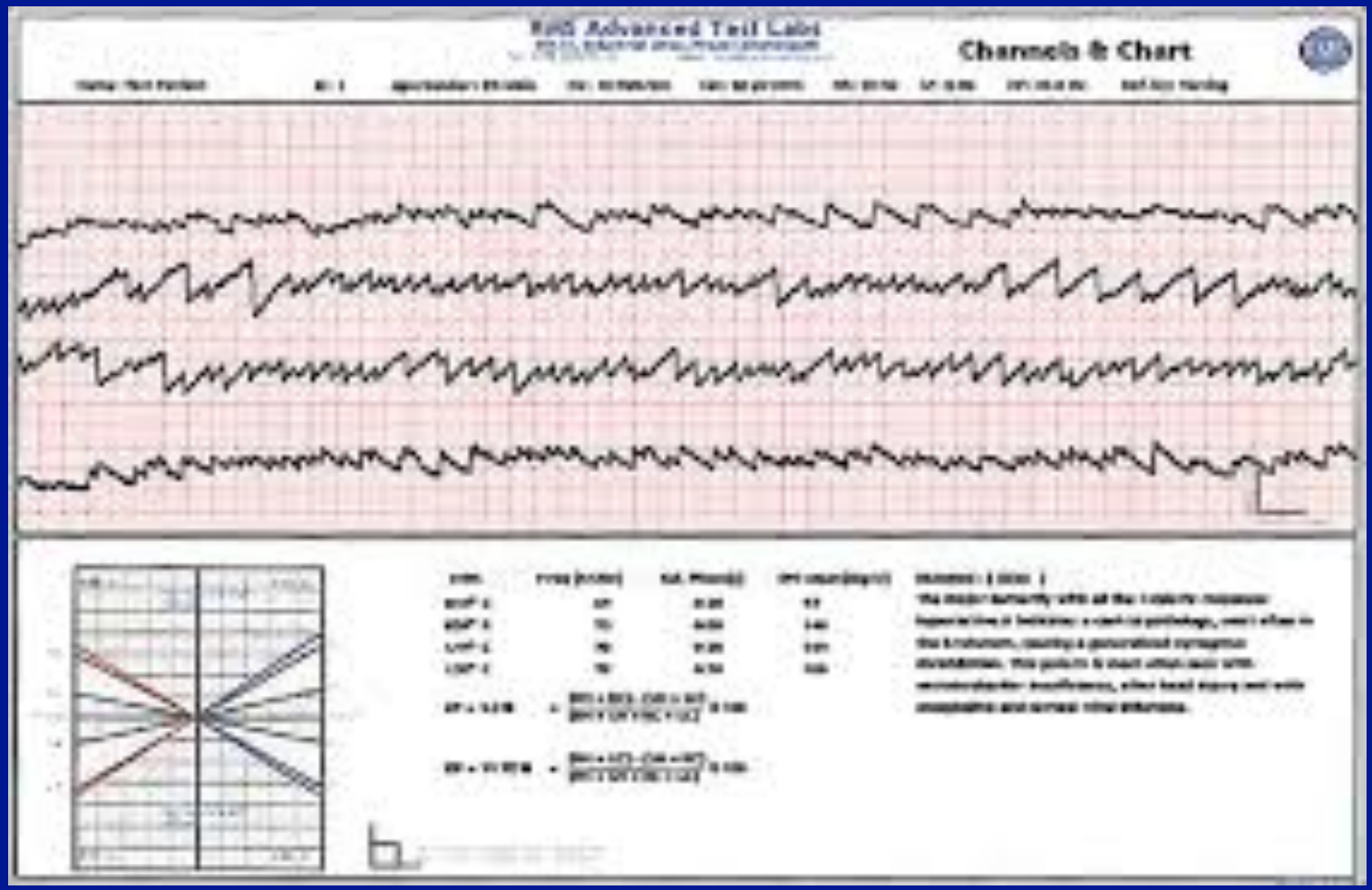
L’ analisi automatica del nistagmo

Herberts G et al. Computer analysis of electronystagmographic data. Acta Otolaryngol (Stokch) 1968; 65: 200-8

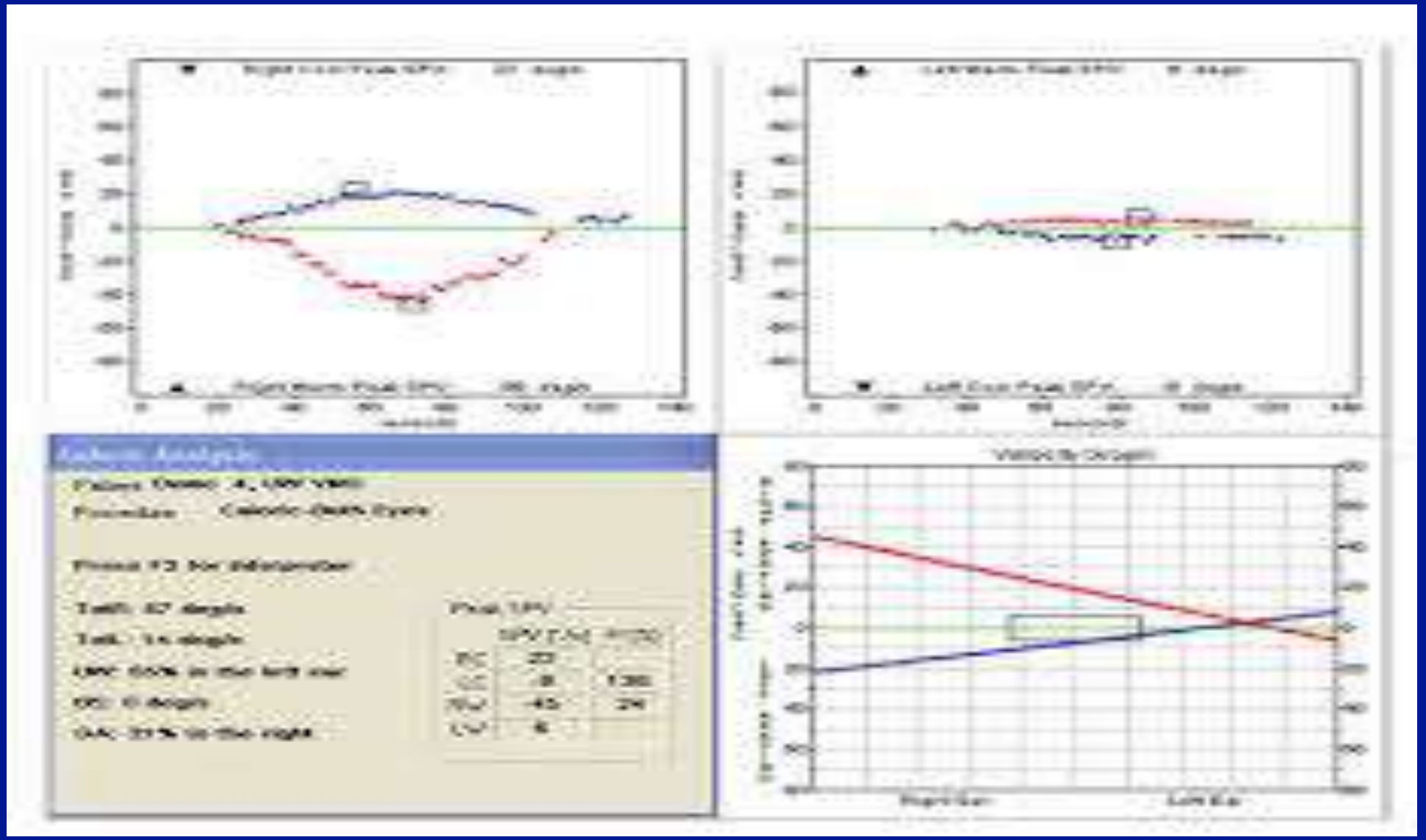
Tole JR, Young LR. MITNYS: a hibrid program for on-line analysis of nystagmus. Aerospace Med 1971; 42: 508-11

Anzaldi E, Mira E. An interactive program for the analysis of ENG tracings. Acta Otolaryngol (Stockh) 1975; 80: 120-7

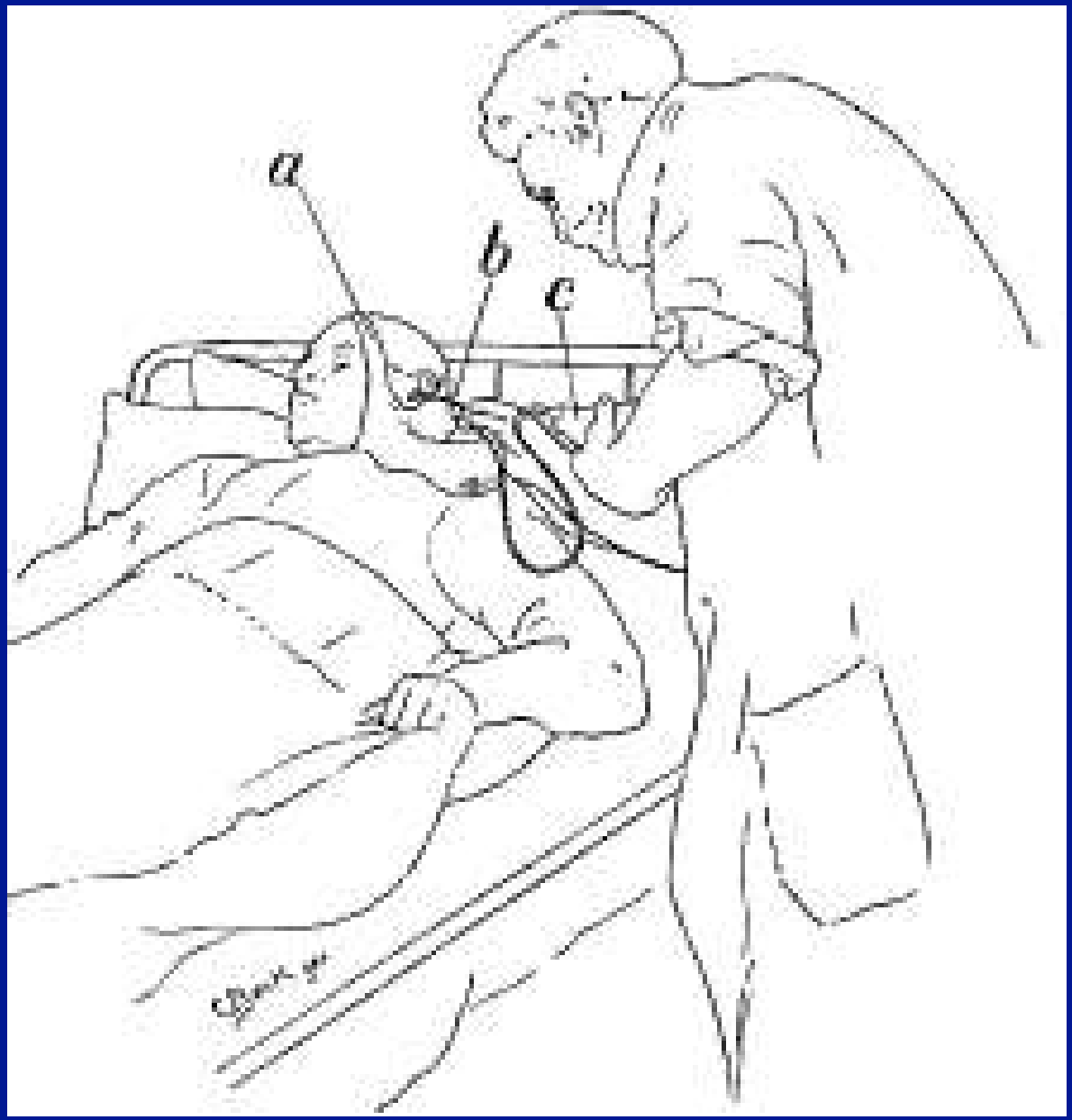
Analisi automatica di nistagmo da stimolazione termica



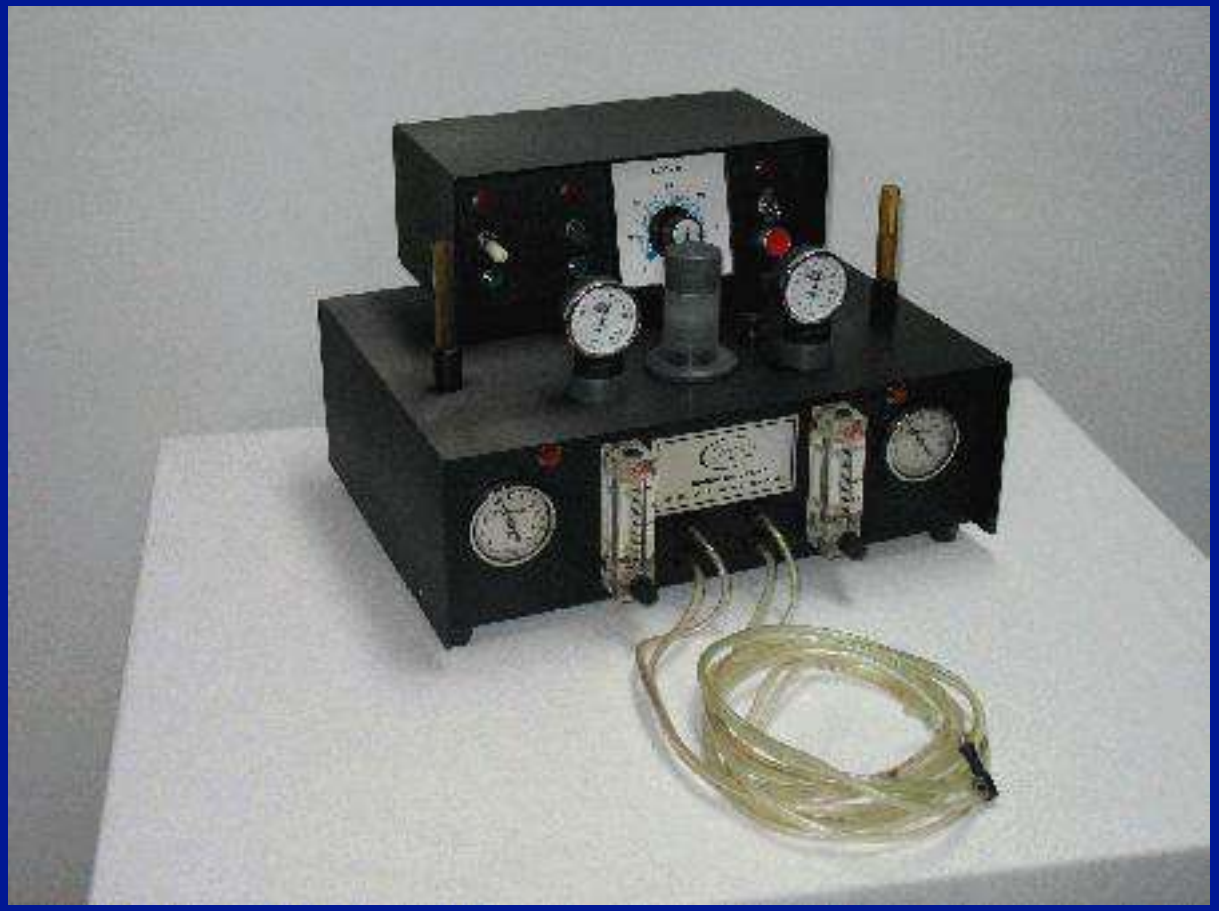
Analisi automatica di nistagmo da stimolazione termica



L’ evoluzione delle stimolazioni labirintiche termiche e rotatorie: otocalorimetro e sedie rotatorie elettromeccaniche


a
b
c

Otocalorimetro



Le stimolazioni termiche

Acqua o aria ?

Quanta acqua ? in quanto tempo ? calda o fredda ? a quale temperatura ?

Quale posiione del paziente ? quale posizione della testa ?

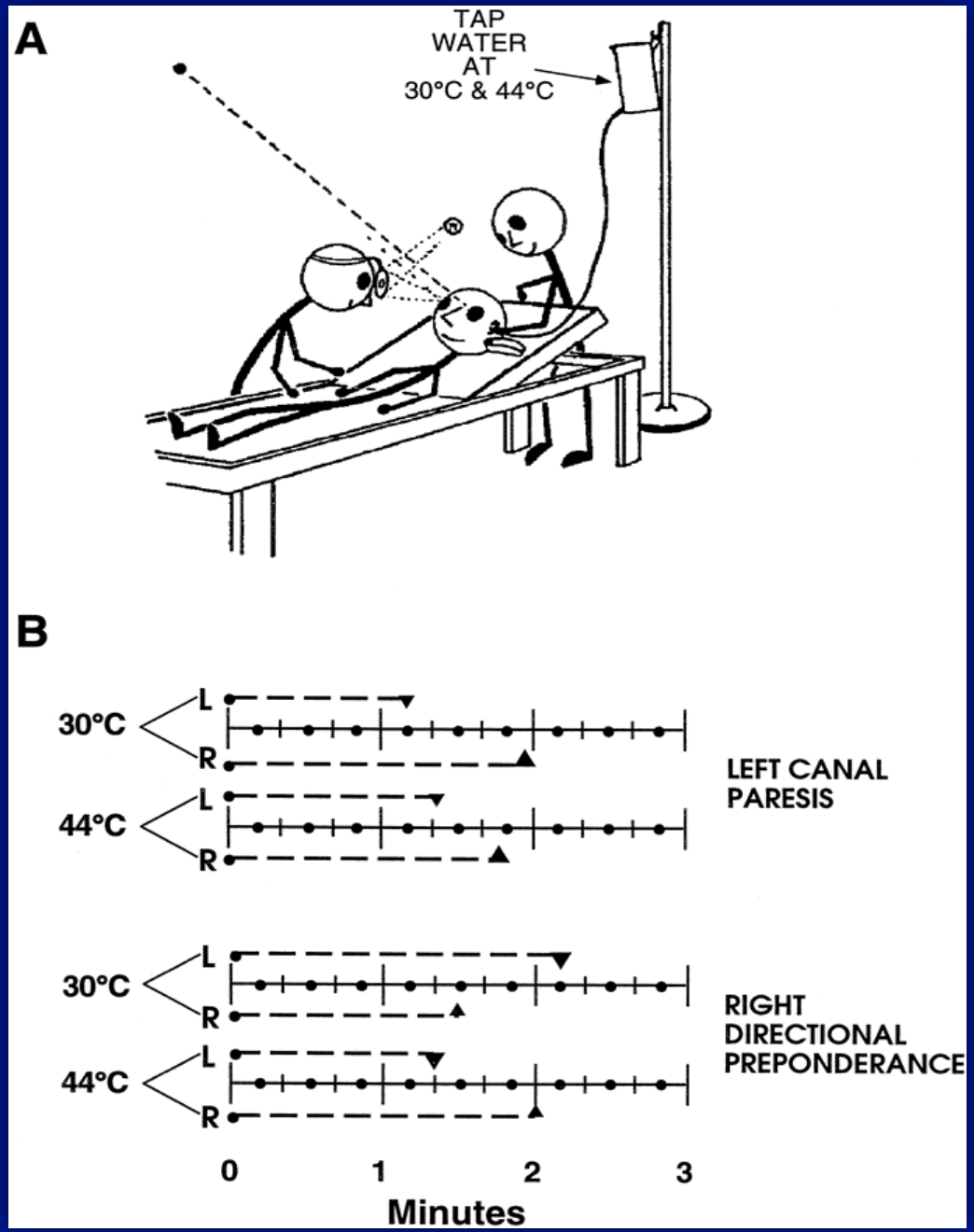
Prova di Veits

Prova minimale di Kobrak

Prova simultanea di Mulch

Stimolazione bitermica secondo Fitzgerald & Hallpike

Fitzgerald G & Hallpike CS Brain 1942; 115: 65-72



1942

La formula di Jongkees (1962)

Unilateral Weakness:

$$\frac{(RW+RC)-(LW+LC)}{RW+RC+LW+LC} \times 100 = UW$$

Directional Preponderance:

$$\frac{(RW+LC)-(LW+RC)}{RW+RC+LW+LC} \times 100 = DP$$

RW=right warm, RC=right cool, LW=left warm, LC= left cool

Registrazione elettronistagmografica

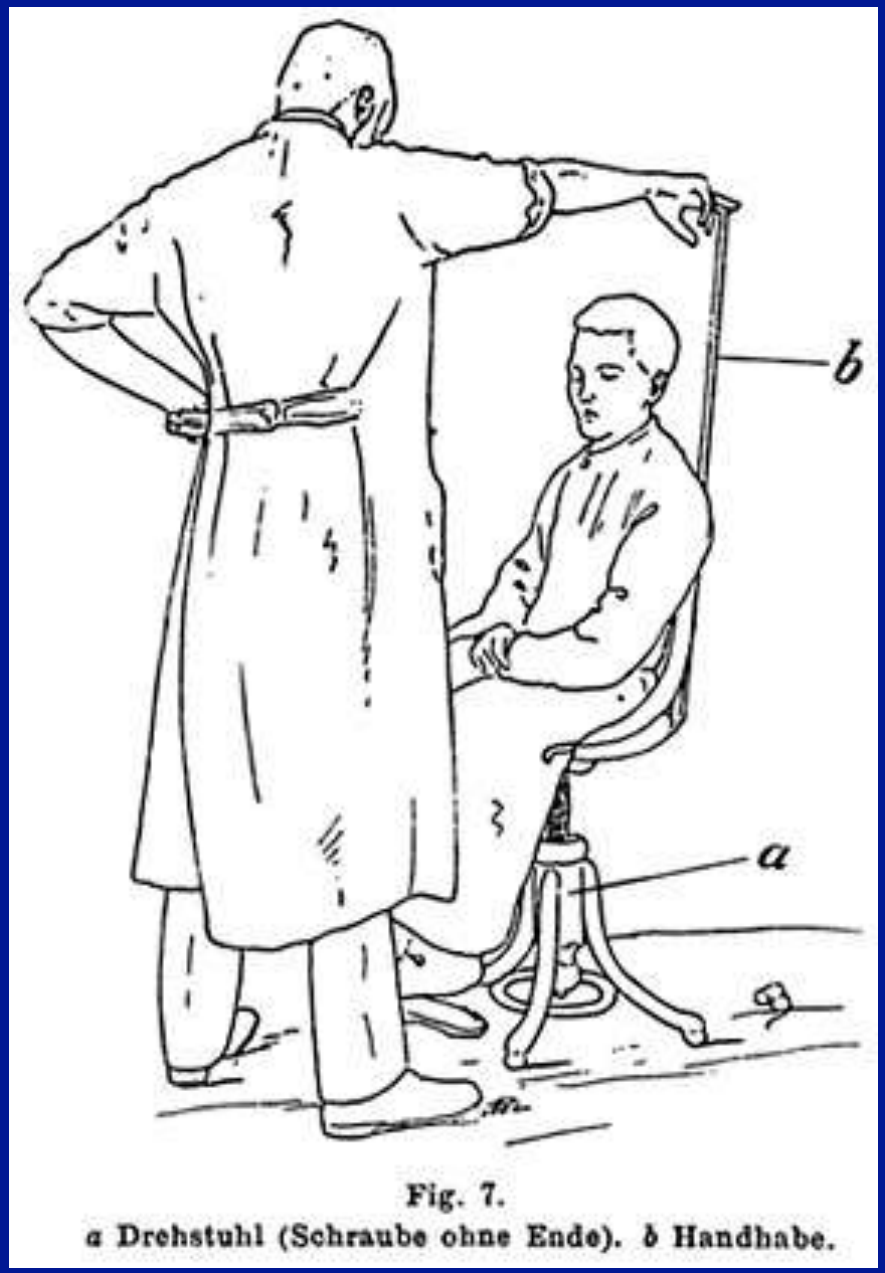


Fig. 7.
a Drehstuhl (Schraube ohne Ende). b Handhabe.

Sedia rotatoria

Le stimolazioni rotatorie

Ad accelerazioni minimali sec Montandon

Cupolometria sec van Egmont et al

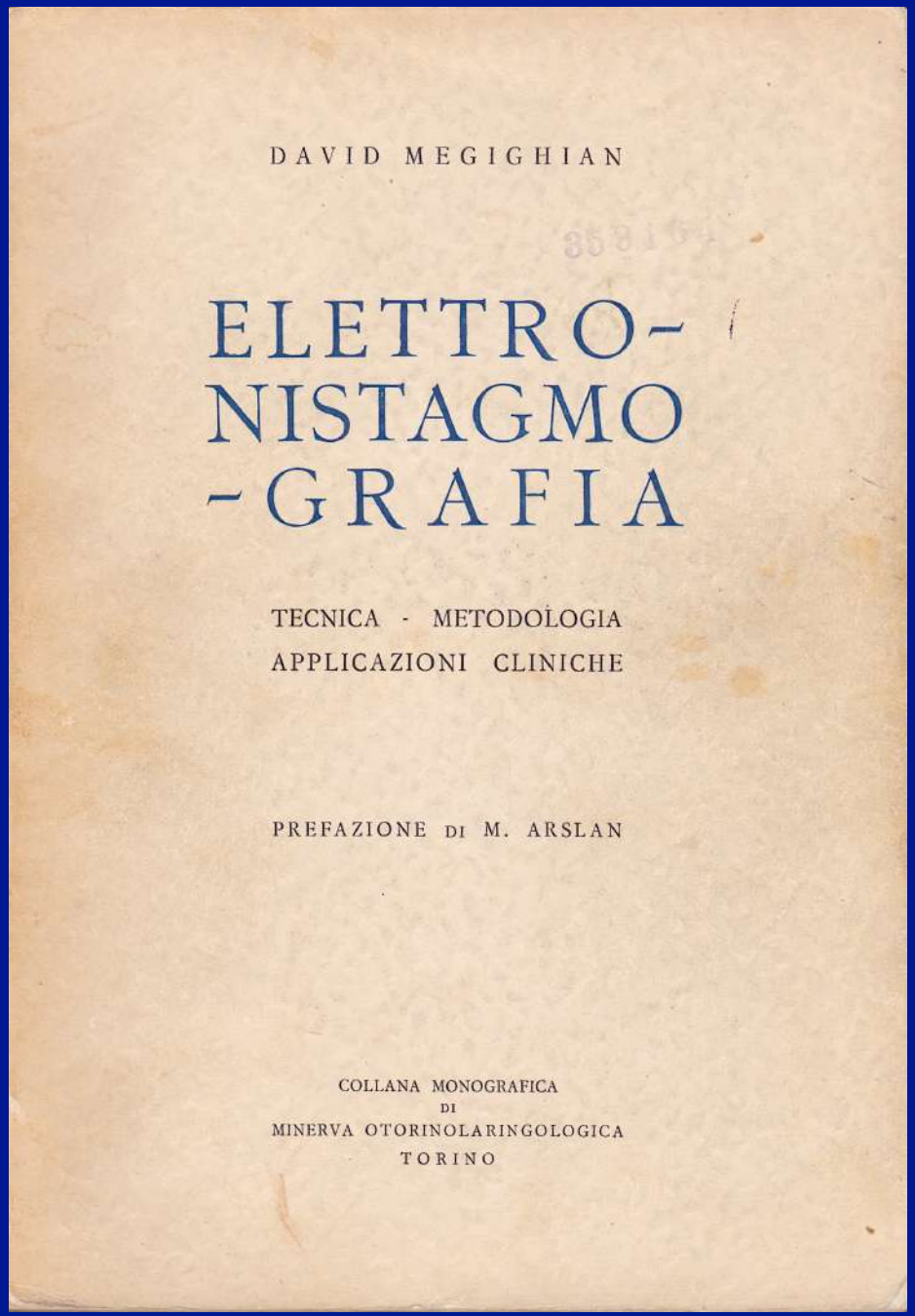
Post-rotatoria sec Buys-Fischer-Arslan

Trapezoidale sec Pirodda

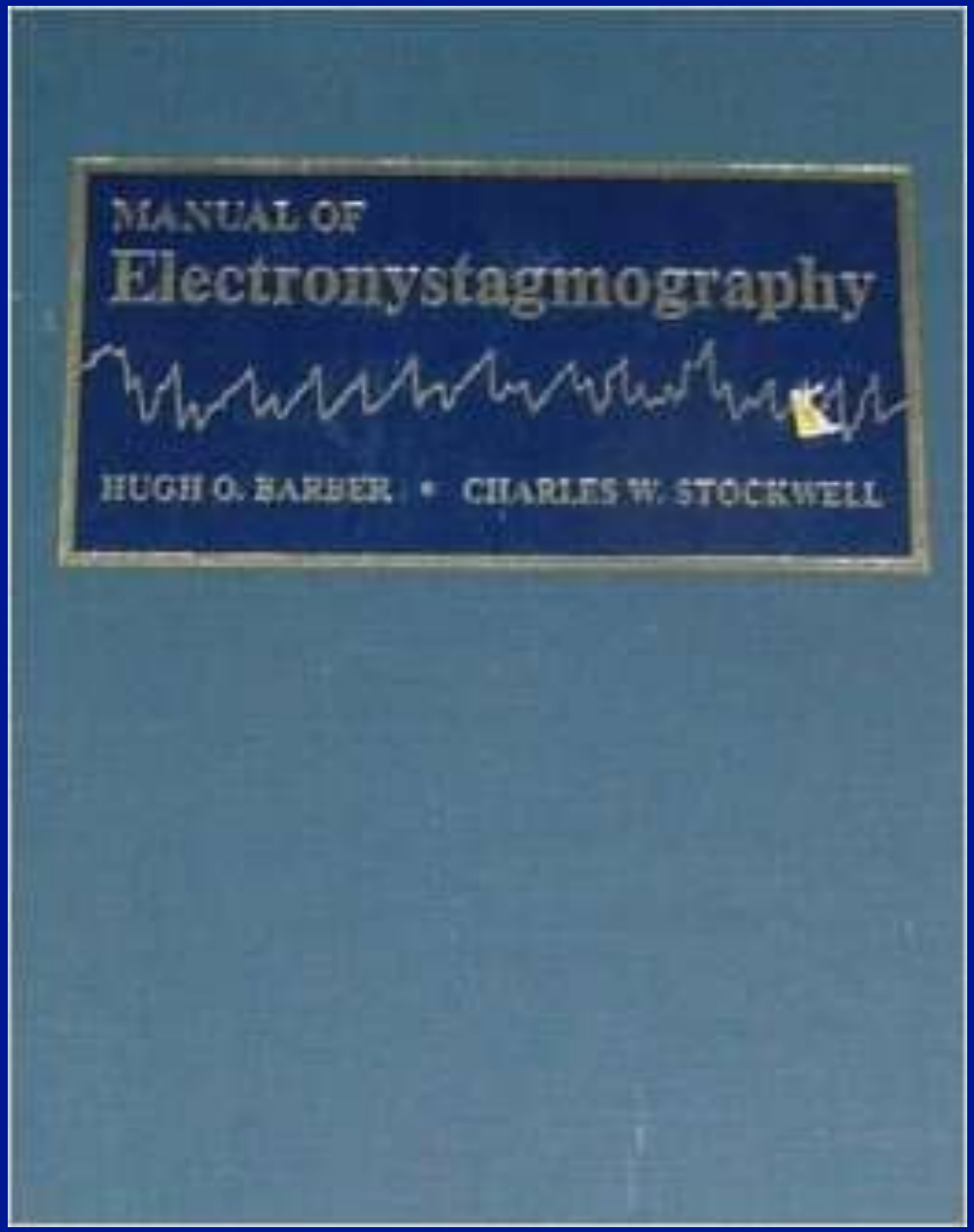
Sinusoidale smorzata o pendolare sec Greiner-Collard-Conraux

Sinusoidale armonica multifrequenziale

I sacri testi dell’ elettronistagmografia


DAVID MEGIGHIAN
ELETTRO-
NISTAGMO
-GRAFIA
TECNICA - METODOLOGIA
APPLICAZIONI CLINICHE
PREFAZIONE DI M. ARSLAN
COLLANA MONOGRAFICA
DI
MINERVA OTORINOLARINGOLOGICA
TORINO

1959


MANUAL OF
Electronystagmography
HUGH O. BARBER • CHARLES W. STOCKWELL

1976


JOSEPH U. TOGLIA, M.D.
JOHN EGYED, M.D.
ELECTRONYSTAGMOGRAPHY
Technical Aspects and Atlas
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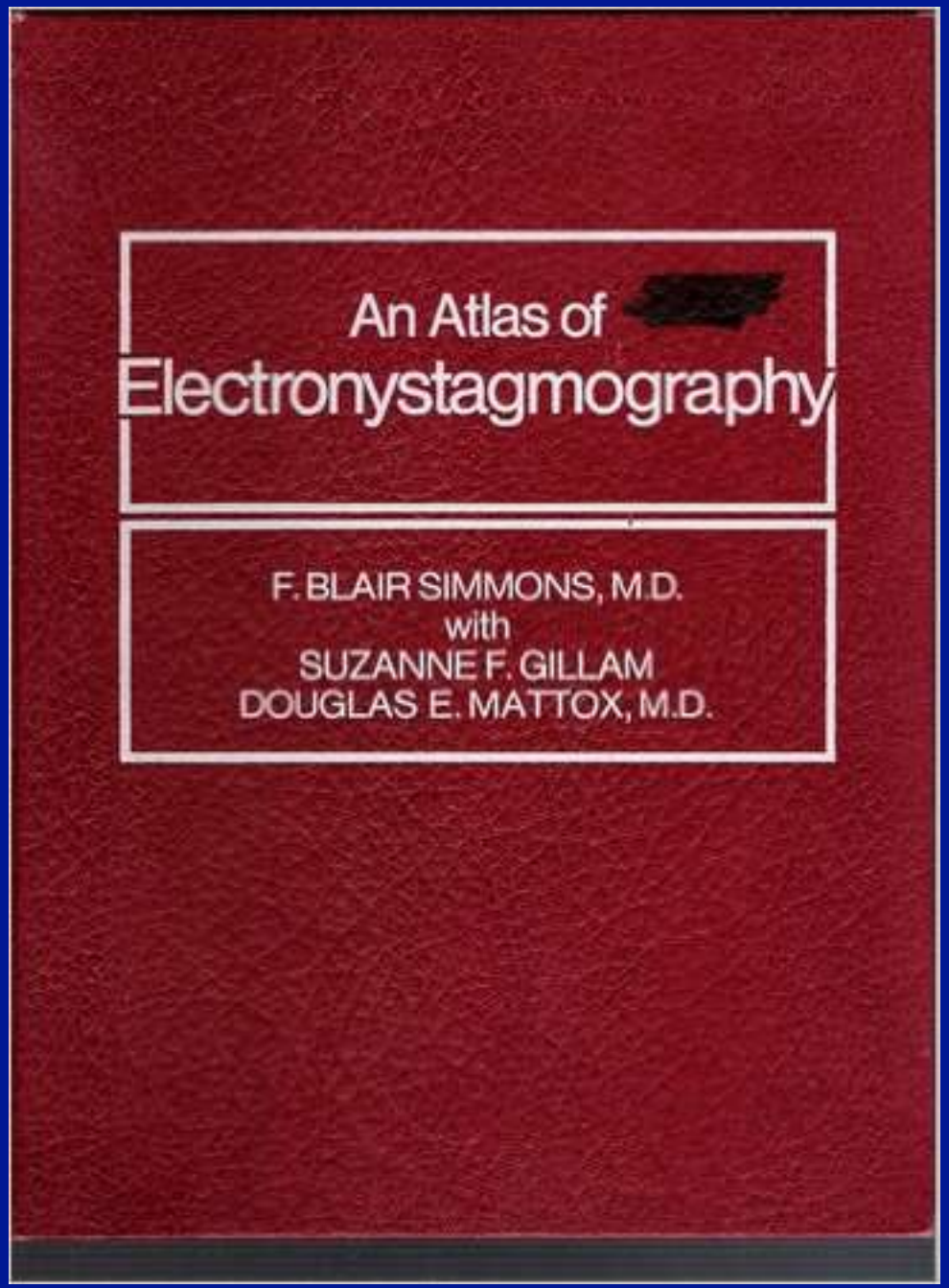
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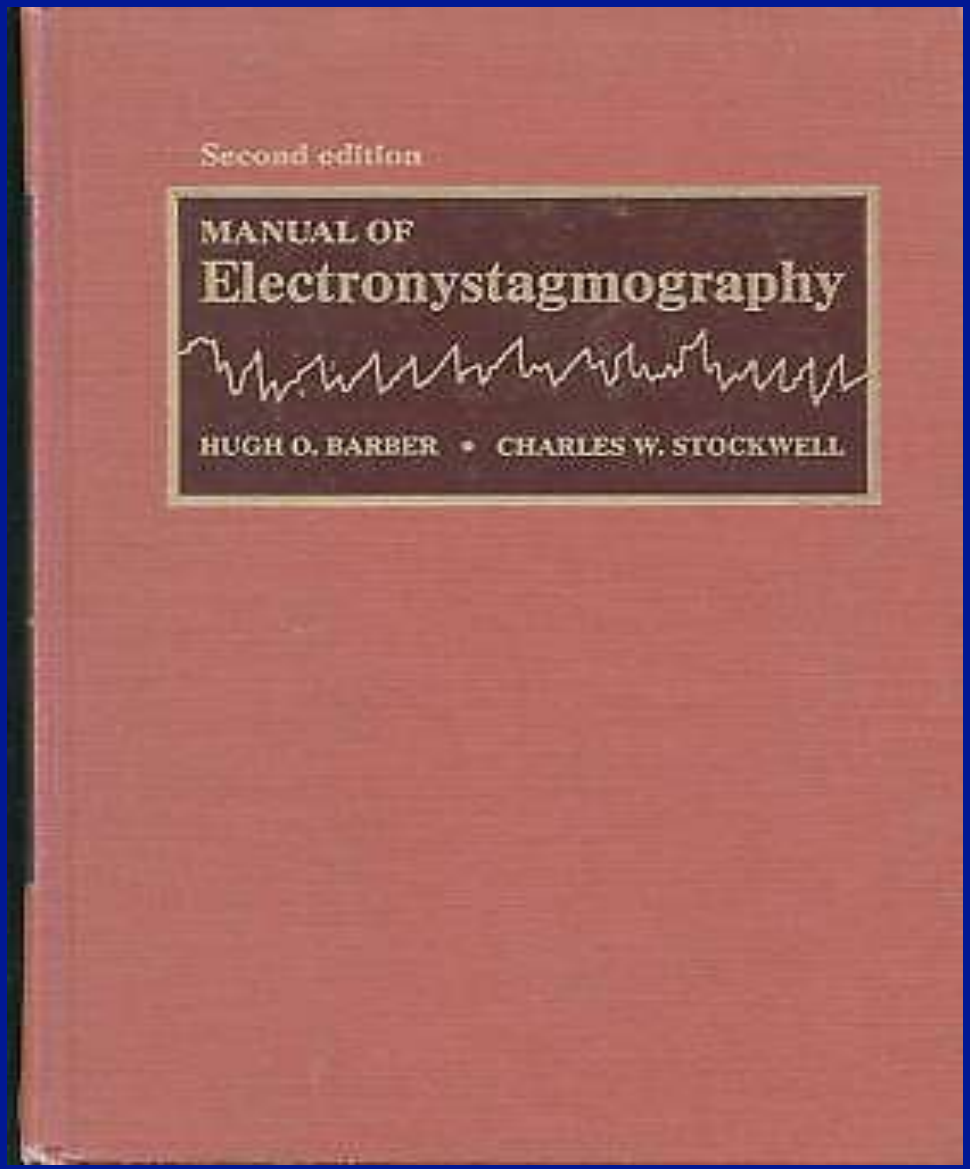
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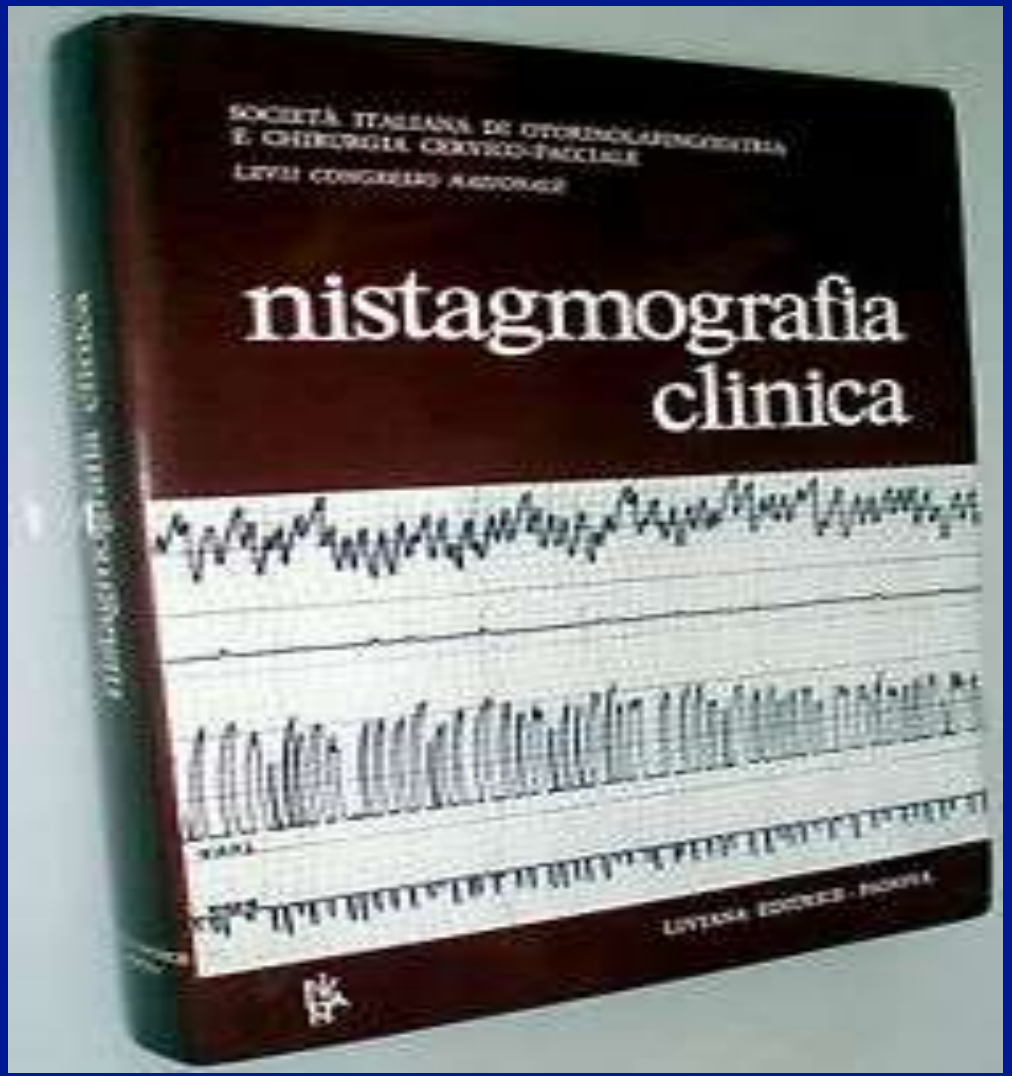

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La rivoluzione culturale e la nascita della *bedside vestibular examination*

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La *bed-side vestibular examination*

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Seconda meta' degli anni '70
La rivoluzione dell' esame vestibolare
La *bed-side vestibular examination*

I precursori

Anatomia: Wersall, Flock, Spoendlin, Iurato

Fisiologia: Lorente de No, Goldberg, Fernandez, Kornhuber, Pompeiano, Precht

Modelii matematici: Robinson

Seconda meta' degli anni '70
La rivoluzione dell' esame vestibolare
La *bed-side vestibular examination*

I clinici

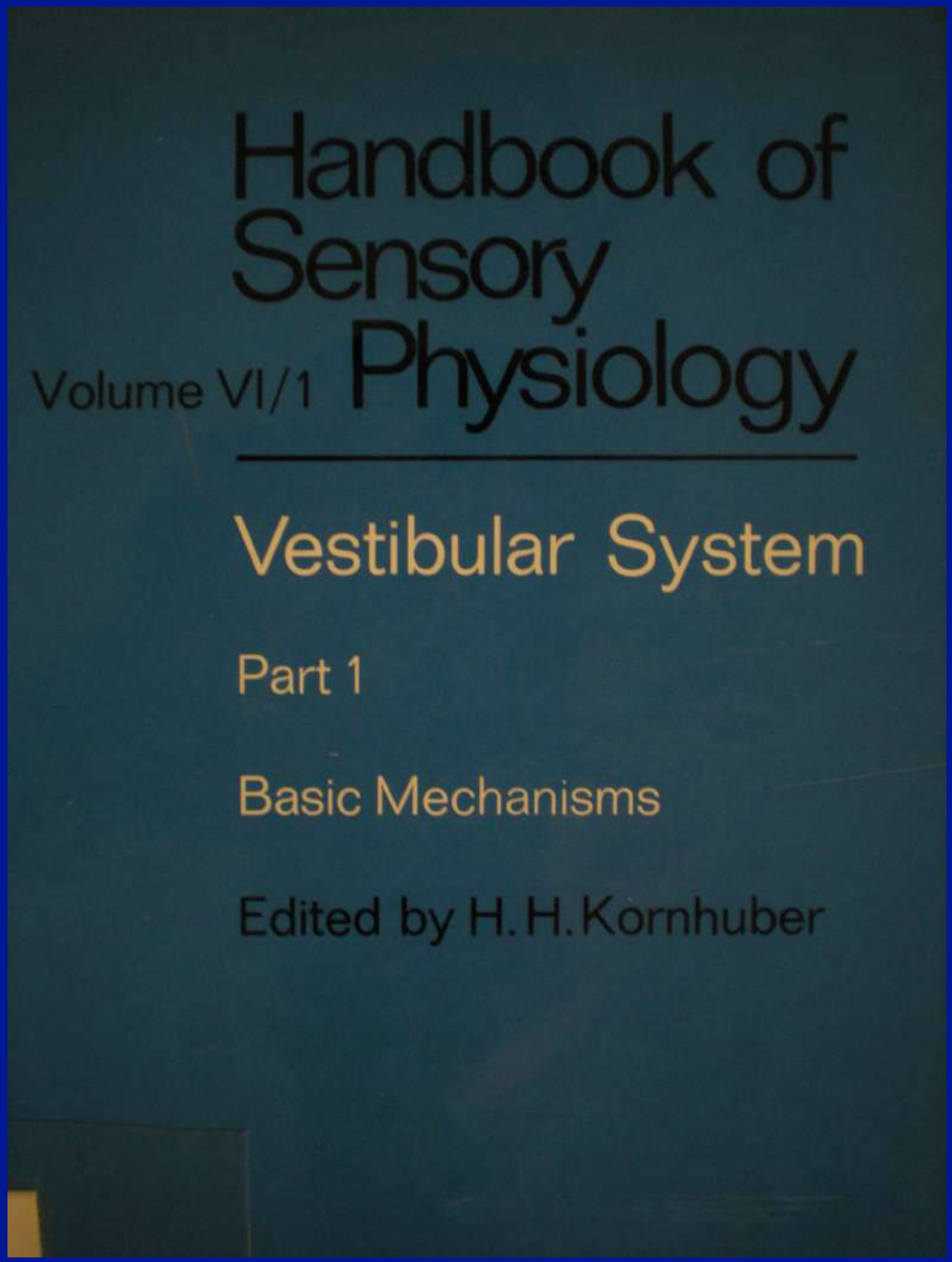
CS Hallpike, J Hood, A Bronstein, M Gresty,
D Robinson, D Zee, T Hain, L Minor
V Honrubia, RW Baloh
Th Brandt, M Dieterich, Th Lempert
M Halmagyi, I Curthoys,
A Bohmer, D Straumann

Seconda meta' degli anni '70
La rivoluzione dell' esame vestibolare
La *bed-side vestibular examination*

I sacri testi


M. AUBRY ET P. PIALOUX
MALADIES DE
L'OREILLE INTERNE
ET
OTO-NEUROLOGIE

1957


Handbook of Sensory Physiology
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Vestibular System
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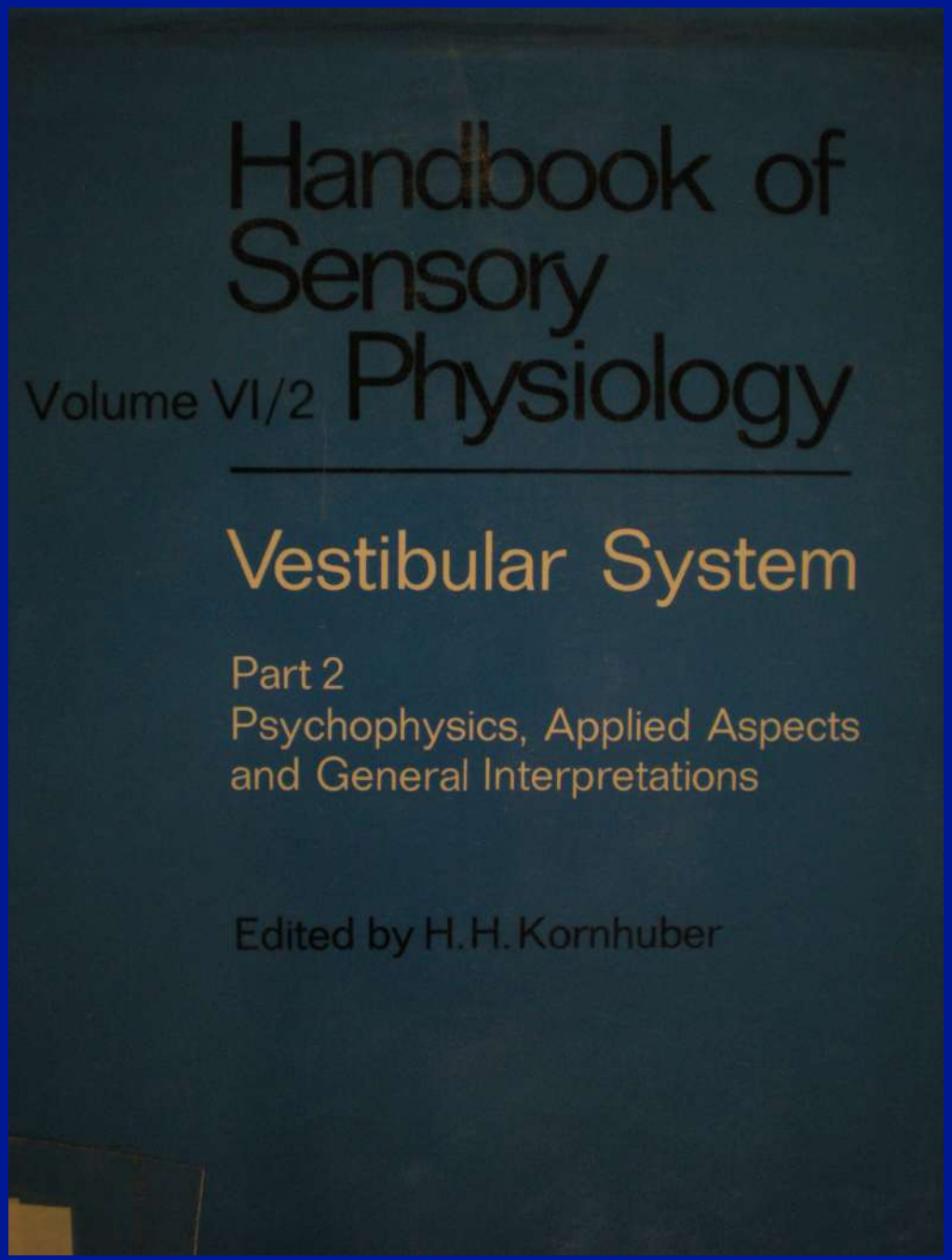
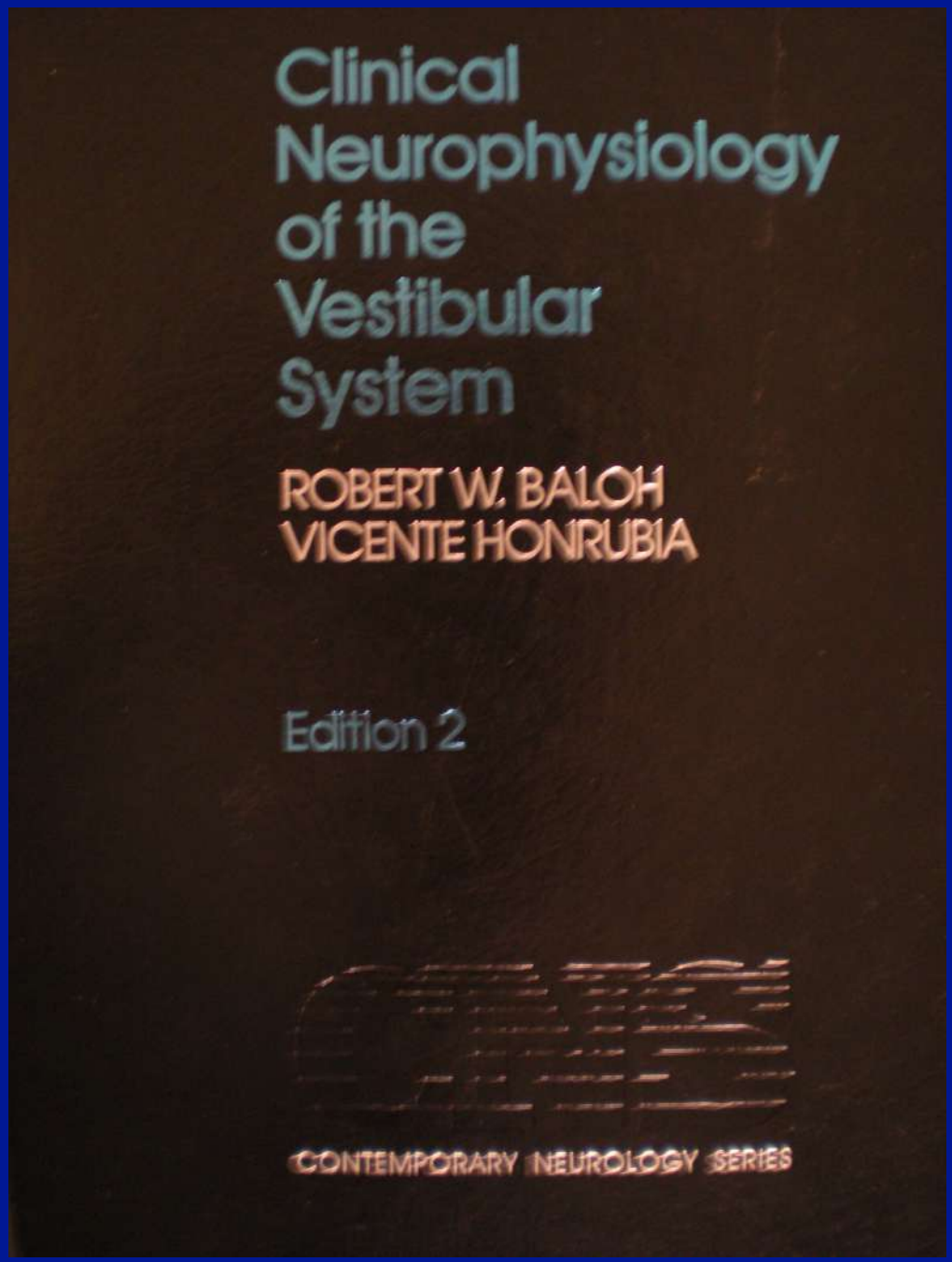
Basic Mechanisms
Edited by H. H. Kornhuber

1974


Handbook of Sensory Physiology
Volume VI/2
Vestibular System
Part 2
Psychophysics, Applied Aspects and General Interpretations
Edited by H.H. Kornhuber

1974


Clinical
Neurophysiology
of the
Vestibular
System
ROBERT W. BALOH
VICENTE HONRUBIA
Edition 2
CONTEMPORARY NEUROLOGY SERIES

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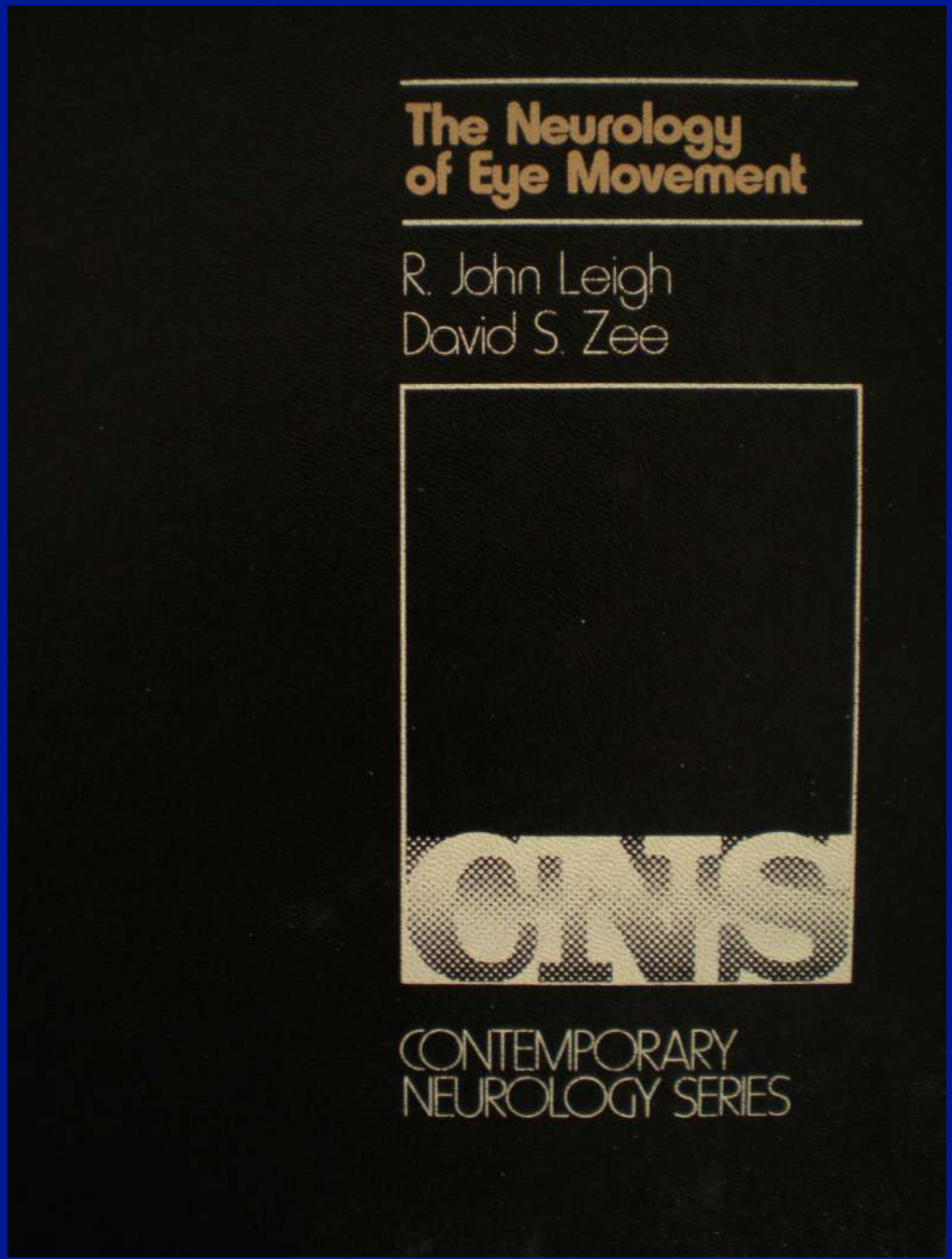
The Neurology of Eye Movement
R. John Leigh
David S. Zee
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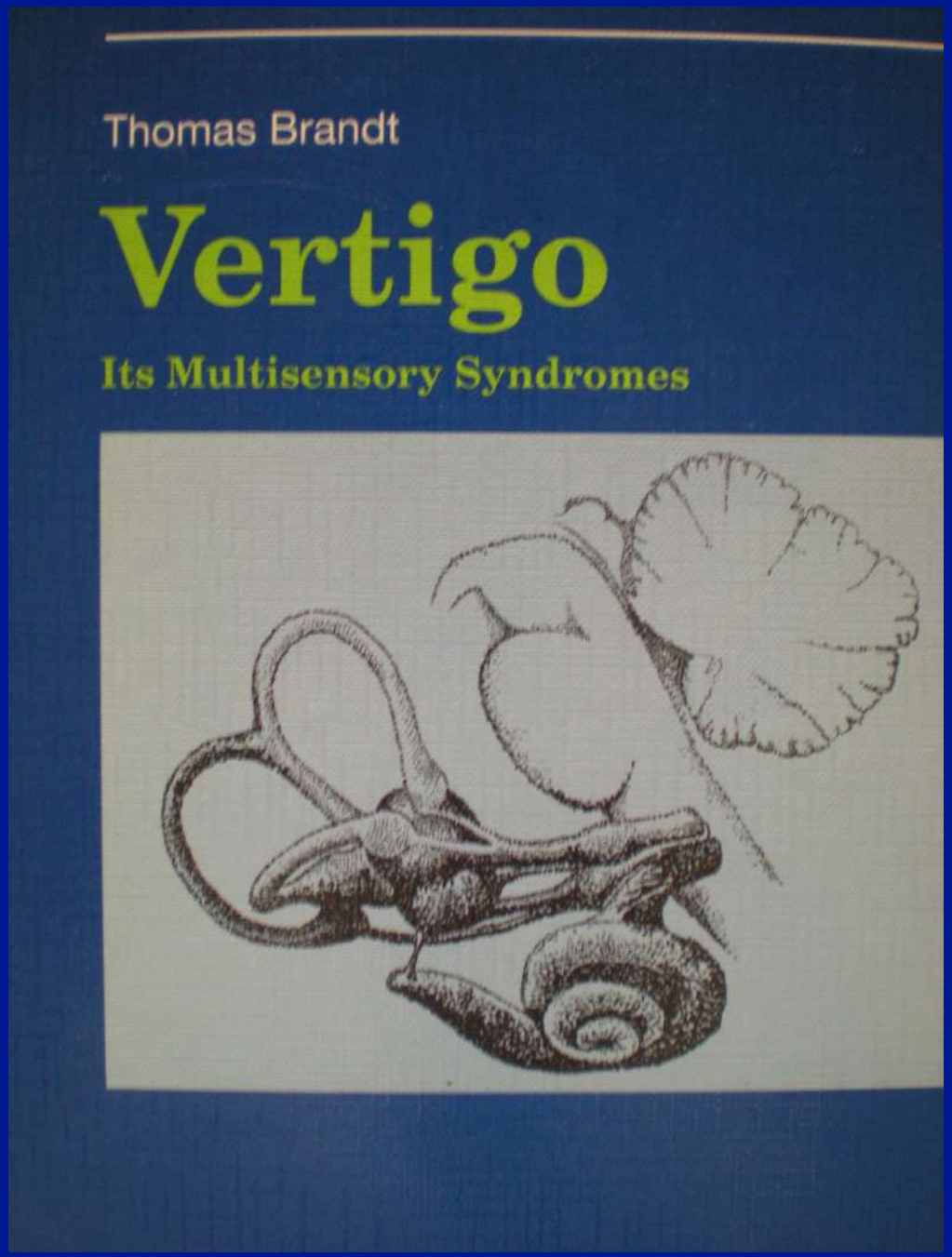
1983



1991

Nuove patologie:
neurite vestibolare,
BPPV, vertigine
emicranica, deiscenza
del CSS

LXXVIII

THE PATHOLOGY, SYMPTOMATOLOGY AND DIAGNOSIS OF CERTAIN COMMON DISORDERS OF THE VESTIBULAR SYSTEM

M. R. Dix, F.R.C.S.

AND

C. S. Hallpike, F.R.C.P., F.R.C.S.

London, England

It is generally agreed that within the last fifteen years some extension has been achieved of our understanding of the problems of human vertigo. Nevertheless, difficulties and confusion still persist and in the course of the present communication an attempt will be made to advance the process of its clarification, both by critical review and by a short account of some of our recent investigations. The foundations of the subject are to be found in the writings of Prosper Ménière, and these we take as our starting point.

Ménière's papers on vertigo are chiefly remarkable for the powers which their writer displays of describing and analysing the symptoms and signs of disease. It is easy now to realise that it was this mastery of symptomatology which, more than anything else, enabled him to identify, with an accuracy which has never been seriously questioned, the disease of the labyrinth which has since come to bear his name. Beyond, however, asserting with confidence that the disease was due to a lesion of the internal ear, limited to that organ and indestructive to life, he made no direct observations upon its pathological anatomy, and further information upon this point was not forthcoming until 1938. Since then the morbid anatomical basis of the disease has been established by means of histological examinations of the temporal bones in a number of clinically characteristic cases. It is fair to say that this opinion is not perhaps a universal one. On the contrary it is still sometimes said that Ménière did describe the pathological anatomy of Ménière's disease, and reference is made to the case which

From the Aural Department and the Otological Research Unit of the Medical Research Council, National Hospital, Queen Square, London.

Read before the Section of Otology, Royal Society of Medicine, March, 1952.

1952

Dix MR, Hallpike CS Ann Otol Rhinol Laryngol 1952; 61: 987

Vestibular Neuritis

Michael Strupp, M.D.,[1] and Thomas Brandt, M.D., F.R.C.P.[2]

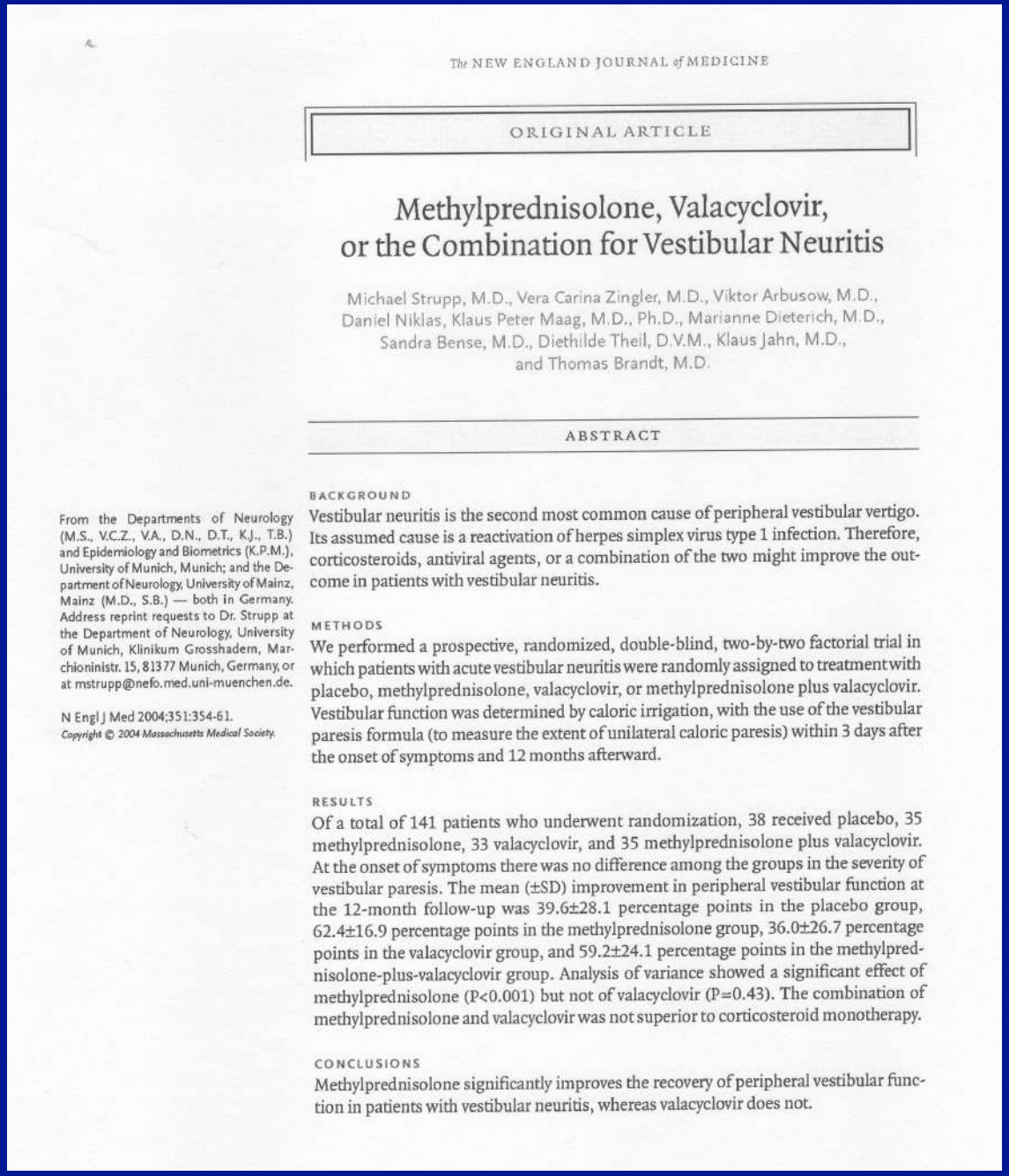
ABSTRACT

The key signs and symptoms of vestibular neuritis are rotatory vertigo with an acute onset lasting several days, horizontal spontaneous nystagmus (with a rotational component) toward the unaffected ear, a pathologic head-impulse test toward the affected ear, a deviation of the subjective visual vertical toward the affected ear, postural imbalance with falls toward the affected ear, and nausea. The head-impulse test and caloric irrigation show an ipsilateral deficit of the vestibuloocular reflex. Vestibular neuritis is the third most common cause of peripheral vestibular vertigo. It has an annual incidence of 3.5 per 100,000 population and accounts for 7% of the patients at outpatient clinics specializing in the treatment of vertigo. The reactivation of a latent herpes simplex virus type 1 (HSV-1) infection is the most likely cause, as HSV-1 DNA and RNA have been detected in human vestibular ganglia. Vestibular neuritis is a diagnosis of exclusion. Relevant differential diagnoses are vestibular pseudoneuritis due to acute pontomedullary brainstem lesions or cerebellar nodular infarctions, vestibular migraine, and monosymptomatically beginning Ménière's disease. Recovery from vestibular neuritis is due to a combination of (a) peripheral restoration of labyrinthine function, usually incomplete but can be improved by early treatment with corticosteroids, which cause a recovery rate of 62% within 12 months; (b) mainly somatosensory and visual substitution; and (c) central compensation, which can be improved by vestibular exercise.

KEYWORDS: Peripheral vestibular vertigo, dizziness, nystagmus, herpes simplex virus type 1, glucocorticoids, vestibular rehabilitation

Strupp M, Brandt Th "T.Vestibular neuritis."

Semin Neurol. 2009; 29: 509-19



The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Methylprednisolone, Valacyclovir, or the Combination for Vestibular Neuritis

Michael Strupp, M.D., Vera Carina Zingler, M.D., Viktor Arbusow, M.D., Daniel Niklas, Klaus Peter Maag, M.D., Ph.D., Marianne Dieterich, M.D., Sandra Bense, M.D., Diethilde Theil, D.V.M., Klaus Jahn, M.D., and Thomas Brandt, M.D.

From the Departments of Neurology (M.S., V.C.Z., V.A., D.N., D.T., K.J., T.B.) and Epidemiology and Biometrics (K.P.M.), University of Munich, Munich; and the Department of Neurology, University of Mainz, Mainz (M.D., S.B.) — both in Germany. Address reprint requests to Dr. Strupp at the Department of Neurology, University of Munich, Klinikum Grosshadern, Marchioninistr. 15, 81377 Munich, Germany, or at mstrupp@nefo.med.uni-muenchen.de.

N Engl J Med 2004;351:354-61.

ABSTRACT

BACKGROUND

Vestibular neuritis is the second most common cause of peripheral vestibular vertigo. Its assumed cause is a reactivation of herpes simplex virus type 1 infection. Therefore, corticosteroids, antiviral agents, or a combination of the two might improve the outcome in patients with vestibular neuritis.

METHODS

We performed a prospective, randomized, double-blind, two-by-two factorial trial in which patients with acute vestibular neuritis were randomly assigned to treatment with placebo, methylprednisolone, valacyclovir, or methylprednisolone plus valacyclovir. Vestibular function was determined by caloric irrigation, with the use of the vestibular paresis formula (to measure the extent of unilateral caloric paresis) within 3 days after the onset of symptoms and 12 months afterward.

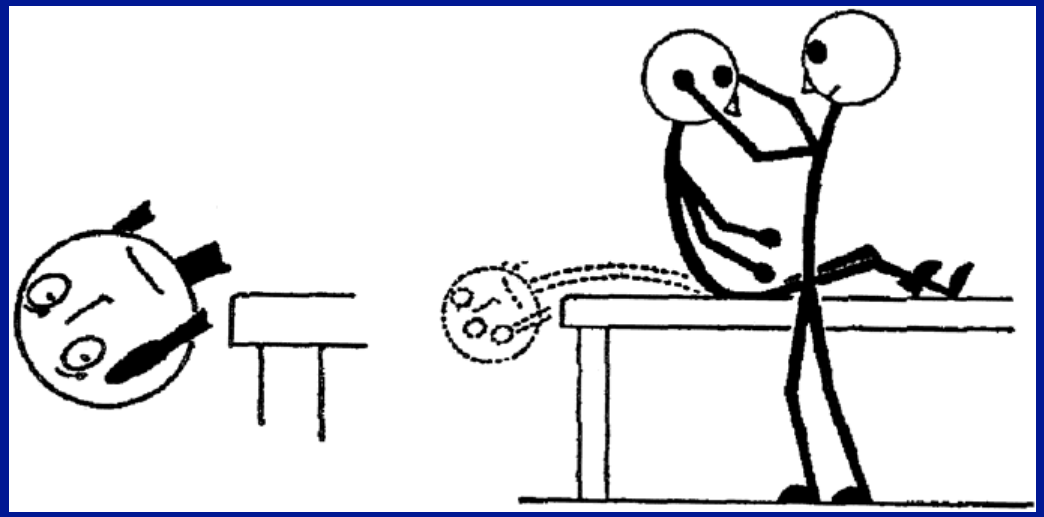
RESULTS

Of a total of 141 patients who underwent randomization, 38 received placebo, 35 methylprednisolone, 33 valacyclovir, and 35 methylprednisolone plus valacyclovir. At the onset of symptoms there was no difference among the groups in the severity of vestibular paresis. The mean (±SD) improvement in peripheral vestibular function at the 12-month follow-up was 39.6±28.1 percentage points in the placebo group, 62.4±16.9 percentage points in the methylprednisolone group, 36.0±26.7 percentage points in the valacyclovir group, and 59.2±24.1 percentage points in the methylprednisolone-plus-valacyclovir group. Analysis of variance showed a significant effect of methylprednisolone ($P<0.001$) but not of valacyclovir ($P=0.43$). The combination of methylprednisolone and valacyclovir was not superior to corticosteroid monotherapy.

CONCLUSIONS

Methylprednisolone significantly improves the recovery of peripheral vestibular function in patients with vestibular neuritis, whereas valacyclovir does not.

Strupp M et al "Methylprednisolone, valacyclovir, or the combination for vestibular neuritis" N Engl J Med 2004; 351: 34-61



BPPV: Manovra diagnostica di Dix-Hallpike, 1952

Cupulolithiasis

Harold F. Schuknecht, MD, Boston

This paper is dedicated to Julius Lempert whose ingenuity, skill, and enthusiasm set otology in motion in this century. It was a privilege and honor for me to have come momentarily under the tutelage of this superb otologic surgeon and teacher.

THE TERM cupulolithiasis is presented for the first time to designate a vestibular disorder which previously has been identified by several names including postural vertigo, positional vertigo, and positional vertigo of the benign paroxysmal type. Recent pathological studies support the concept that the disorder is caused by a deposit, presumably composed of mineral, on the cupula of the posterior semicircular canal which renders this organ sensitive to gravitational force and, therefore, subject to stimulation with changes in head position. The clinical features of cupulolithiasis are distinctive and serve to differentiate it from positional vertigo caused by lesions of the central nervous system. The diagnosis can be made by inducing the characteristic vestibular manifestations by provocative positional testing.

Bárány[1] first described the disorder as he observed it in a 27-year-old woman and he wrote as follows:

The attacks only appeared when she lay on her right side. When she did this, there appeared a strong rotatory nystagmus to the right. The attack lasted about thirty seconds and was accompanied by violent vertigo and nausea. If, immediately after the cessation of the symptoms, the head was again turned to the right, no attack occurred and in order to evoke a new attack in this way, the patient had to lie for some time on her back or on her left side.

Bárány and others originally attributed this disorder to lesions in the semicircular canals but, because the dizziness was precipitated not by head movement but by head position, they came to believe that the condition was due to a disorder of the otoliths.

Dix and Hallpike[2] in 1952 made a notable contribution to our understanding of positional vertigo. In a study of 100 patients with this symptom they found a high incidence of ear disorders (eg, otitis media, acoustic trauma). They noted that the vertiginous attacks occurred when the abnormal ear was placed undermost in the testing procedure. Their observations supported Bárány's opinion that this type of vertigo is of inner ear origin.

In 1956 Lindsay and Hemenway[3] described for the first time a symptom complex which they observed in seven patients. It was characterized by a sudden, severe, and prolonged vertiginous episode which subsided after several weeks and was followed by positional vertigo. All were in the fifth, sixth or seventh decade of life and the positional vertigo persisted for weeks to years. All had impaired response to caloric testing and some had hearing loss. These functional alterations existed in the ear located under-

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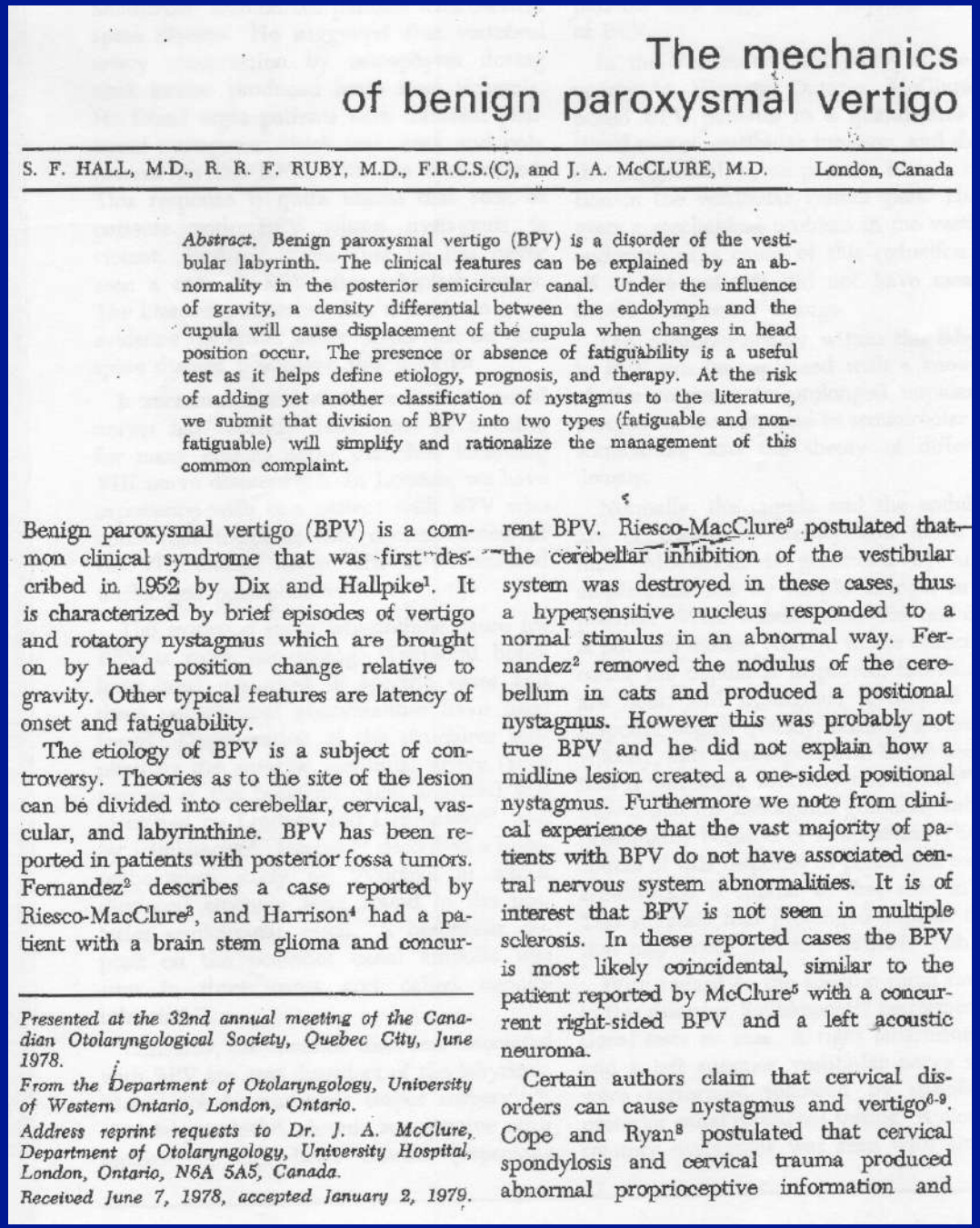
From the Harvard Medical School and the Massachusetts Eye and Ear Infirmary, Boston.

Reprint requests to Massachusetts Eye and Ear Infirmary, 243 Charles St, Boston 02114.

1969

Scuknecht HF Cupololithiasis Arch Otolaryngol 1969; 90: 112-20



The mechanics of benign paroxysmal vertigo

S. F. HALL, M.D., R. R. F. RUBY, M.D., F.R.C.S.(C), and J. A. McCLURE, M.D. London, Canada

Abstract. Benign paroxysmal vertigo (BPV) is a disorder of the vestibular labyrinth. The clinical features can be explained by an abnormality in the posterior semicircular canal. Under the influence of gravity, a density differential between the endolymph and the cupula will cause displacement of the cupula when changes in head position occur. The presence or absence of fatiguability is a useful test as it helps define etiology, prognosis, and therapy. At the risk of adding yet another classification of nystagmus to the literature, we submit that division of BPV into two types (fatiguable and non-fatiguable) will simplify and rationalize the management of this common complaint.

Benign paroxysmal vertigo (BPV) is a common clinical syndrome that was first described in 1952 by Dix and Hallpike[1]. It is characterized by brief episodes of vertigo and rotatory nystagmus which are brought on by head position change relative to gravity. Other typical features are latency of onset and fatiguability.

The etiology of BPV is a subject of controversy. Theories as to the site of the lesion can be divided into cerebellar, cervical, vascular, and labyrinthine. BPV has been reported in patients with posterior fossa tumors. Fernandez[2] describes a case reported by Riesco-MacClure[3], and Harrison[4] had a patient with a brain stem glioma and concurrent BPV. Riesco-MacClure[3] postulated that the cerebellar inhibition of the vestibular system was destroyed in these cases, thus a hypersensitive nucleus responded to a normal stimulus in an abnormal way. Fernandez[2] removed the nodulus of the cerebellum in cats and produced a positional nystagmus. However this was probably not true BPV and he did not explain how a midline lesion created a one-sided positional nystagmus. Furthermore we note from clinical experience that the vast majority of patients with BPV do not have associated central nervous system abnormalities. It is of interest that BPV is not seen in multiple sclerosis. In these reported cases the BPV is most likely coincidental, similar to the patient reported by McClure[5] with a concurrent right-sided BPV and a left acoustic neuroma.

Certain authors claim that cervical disorders can cause nystagmus and vertigo[6-9]. Cope and Ryan[8] postulated that cervical spondylosis and cervical trauma produced abnormal proprioceptive information and

Presented at the 32nd annual meeting of the Canadian Otolaryngological Society, Quebec City, June 1978.

From the Department of Otolaryngology, University of Western Ontario, London, Ontario.

Address reprint requests to Dr. J. A. McClure, Department of Otolaryngology, University Hospital, London, Ontario, N6A 5A5, Canada.

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1979

Hall SF, Ruby RR, McClure JA “The mechanics of benign paroxysmal vertigo” J Otolaryngol 1979; 8: 151-8

Horizontal canal BPV

JOSEPH A. McCLURE, M.D.* London, Canada

Abstract. Benign paroxysmal vertigo (BPV) is generally attributed to a differential density condition in the posterior semicircular canal. Although the posterior canal is implicated because of its dependent position, the possibility exists that either the horizontal or superior canal could become involved. This paper reports on seven patients with a clinical picture consistent with horizontal canal BPV. The characteristic features are brief vertigo and horizontal nystagmus precipitated by head movement into or out of one of the lateral positions. Position change toward the left lateral position induces left beating nystagmus and *vice versa* for position change toward the right. The direction of the nystagmus indicates a utriculopetal "endolymph flow" when the affected horizontal canal is undermost. This could be explained by particle movement or a "viscous plug" in the posterior aspect of the canal.

Benign paroxysmal vertigo (BPV) is a syndrome characterized by brief (usually less than 30 sec) episodes of vertigo and nystagmus brought on by certain changes of head position relative to gravity. Schuknecht[1] put forward a theory of differential density in the cupulo-endolymph system of the posterior semicircular canal to explain the pathophysiology. This was supported by a limited amount of pathological evidence whereby he was able to demonstrate a deposit of material on the cupula of one posterior canal in patients with BPV.

Several features of BPV suggest involvement of the posterior semicircular canal. For example, the vertigo and nystagmus are most intense with a change of head position in the plane of one posterior canal, the associated nystagmus is primarily rotatory, the cupular deposits in Schuknecht's pathological cases involved only posterior canals, and cutting the nerve to the involved posterior canal (singular nerve section) relieves the symptoms. In accordance with Schuknecht's differential density theory, it is not surprising that BPV originates in the posterior canal. Loose material in the endolymphatic system will eventually find its way to the most dependent part of the system which happens to be the ampulla of the posterior canal. This material, either loose in the endolymph or attached to the cupula, will move under the influence of gravity when the head is rotated in the plane of that canal.

The aforementioned pathophysiology seems to explain most aspects of BPV. However, one might expect on occasion to encounter a similar but modified syndrome if a differential density condition were to occur in either the superior or horizontal semicircular canals. In the case of horizontal canal involvement, the syndrome should be recognizable because one would expect a horizontal rather than a rotatory nystagmus. The paper will report on seven patients who demonstrate a clinical picture consistent with horizontal canal BPV.

*Associate Professor, Department of Otolaryngology, University of Western Ontario, London, Canada.

Presented at the 38th annual meeting of the Canadian Society of Otolaryngology – Head and Neck Surgery, Halifax, N.S., June 1984.

Address reprint requests to J. A. McClure, M.D., Department of Otolaryngology, University Hospital, London, Ontario, Canada, N6A 5A5.

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CASE REPORTS

Case 1 (E.J.) – This 58 year old lady was first seen in our clinic in February 1981 with a 10-15 year history of brief intermittent episodes of vertigo associated with position change. The most bothersome position changes included lying down on her left side and tilting her head forward with the left side down. Dix-Hallpike position testing induced a violent left beating horizontal nystagmus when the patient was moved into the left head hanging position and a violent right beating horizontal nystagmus when moved into the right head hanging position. Vestibular function tests showed a low intensity right beating spontaneous nystagmus with eyes closed and reduced caloric responses on the left. The positional nystagmus was recorded and is shown in Figure 1. The time course of the nystagmus was consistent with a horizontal canal BPV.

Case 2 (E.B.) – This patient, a 73 year old lady, presented in March 1981 with a 15 year history of severe brief vertigo associated with turning onto her right side. Two weeks prior to her assessment, the problem became more severe – to the point that any tilting of her head to the right precipitated the vertigo. Dix-Hallpike position testing revealed a violent right beating horizontal nystagmus when she was moved from the sitting to the right head hanging position, as shown in Figure 2. The time course of the nystagmus was consistent with a horizontal canal BPV. Vestibular function tests showed no significant spontaneous nystagmus with eyes closed, and borderline normal but symmetrical caloric responses. The patient was re-assessed six weeks after her initial visit, and by that time the positional vertigo had completely subsided.

1985

McClure JA "Horizontal canal BPV" J Otolaryngol 1985; 14: 30-5

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(Neurinoma dell'VIII escluso)

a cura di A. Dufour

Comitato Simposi Scientifici della BOOTS-FORMENTI S.p.A.

1985

"CUPULOLITIASI"

L. CIPPARONE, G. CORRIDI, P. PAGNINI

Cattedra di Audiologia dell'Università di Firenze

DEFINIZIONE

Con il termine di cupulolitiasi si intende una labirintopatia maculare con distacco di otoliti, a varia eziologia ed evoluzione favorevole, caratterizzata da brusche vertigini transitorie recidivanti di posizionamento legate ad un intenso ny a caratteri tipici.

La sindrome veniva in passato definita come "vertigine parossistica benigna di posizionamento". Con l'identificazione del meccanismo patogenetico è entrato in uso il termine di "cupulolitiasi" che meglio definisce i caratteri nosologici (1).

EZIOLOGIA

La cupulolitiasi ha origine da un distacco degli otoliti dalla macula dell'utricolo. Tale distacco può avvenire fondamentalmente con due diverse modalità:

a) un meccanismo diretto di natura traumatica, b) un meccanismo indiretto per degenerazione del neuroepitelio sottostante.

MECCANISMO DIRETTO

Nei traumi cranici diretti con testa in movimento, al momento dell'urto, la testa (e conseguentemente il neuroepitelio maculare) subisce una brusca decelerazione mentre gli otoliti tendono a mantenere la velocità per inerzia; si determina pertanto una accelerazione relativa degli otoliti rispetto alle macule. Se tale accelerazione supera la forza di adesione alle ciglia si verifica il distacco degli otoliti, che è reso possibile dalla loro massa elevata ed è in rapporto con la velocità iniziale del sistema. La cupulolitiasi è invece rara nei traumi da colpo di frusta cervicale, nei quali il movimento di otoliti e cellule è più consensuale.

---

(1) Il termine di "vertigine parossistica benigna" fu usato nel 1952 da Dix e Hallpike, che riportarono 100 casi di tale sindrome. Schuknecht (1969) formulò l'ipotesi attualmente più accreditata coniando il termine di "cupulolitiasi".

ELSEVIER

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Original Contribution

BENIGN PAROXYSMAL POSITIONAL VERTIGO OF THE HORIZONTAL CANAL: A FORM OF CANALOLITHIASIS WITH VARIABLE CLINICAL FEATURES

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*Department of Otolaryngology, University of Siena, Italy;
†Department of Audiology, University of Firenze, Italy
Reprint address: Dr. D. Nuti, Istituto di Discipline Otorinolaringologiche, Università di Siena, Policlinico Universitario, Viale Bracci, 53100 Siena, Italy; Tel: ++39-577-58470; Fax: ++39-577-47940

□ Abstract—Benign paroxysmal positional vertigo of the horizontal semicircular canal (HC-BPPV) is a well-defined syndrome characterized by direction-changing horizontal positional nystagmus. We report the clinical features of 5 patients who illustrate the possible variables of the syndrome. In most cases, nystagmus is geotropic and more intense when the pathological ear is lowermost; less often the syndrome presents with apogeotropic nystagmus that is more intense when the affected ear is uppermost. The nystagmus pattern may vary in time in the same patient, changing from apogeotropic to geotropic even in observations at short intervals. In some patients, the features indicate involvement of more than one canal, either simultaneously or in succession. It is sustained that the clinical findings can be explained by movement of endolymph caused by displacement of otoconia in the semicircular canals and that variants are due to different positions of the otoconia within the canals.

□ Keywords—horizontal semicircular canal; benign positional vertigo; paroxysmal nystagmus; canalolithiasis.

Introduction

Benign paroxysmal positional vertigo (BPPV) is the most frequent vestibular syndrome. In 1969, it was commonly known as "cupulolithiasis." Schuknecht (1) postulated that the disorder was caused by a deposit on the cupula of the posterior semicircular canal (PC) that made the cupula sensitive to gravitational forces. In 1979, Hall, Ruby, and McClure (2) suggested that the pathogenetic mechanism of BPPV was due to "something" moving inside the canal rather than adhering to the cupula of the PC. This theory, known as "canalolithiasis," was sustained by the observations of Parnes and McClure (3), who found many free-floating particles in the PC of patients undergoing surgical occlusion of the canal because of incapacitating BPPV. Theoretical considerations of the features of nystagmus during therapeutic maneuvers in most cases support the canalolithiasis theory (4–7).

In 1985, it was reported that BPPV might originate from the horizontal semicircular canal (HC) (8). The features of HC-BPPV are such that the pathogenesis can be explained on the basis of particles or a viscous plug moving in the posterior part of the HC (9). In 1989, we reported a series of 15 patients with HC-BPPV and described the clinical features of paroxysmal geotropic nystagmus, observing the possibility of a variant characterized by apogeotropic nystagmus (10). Three patients with persistent direction-changing nystagmus consistent with debris leaving the PC and becoming attached to the cupula of the HC were

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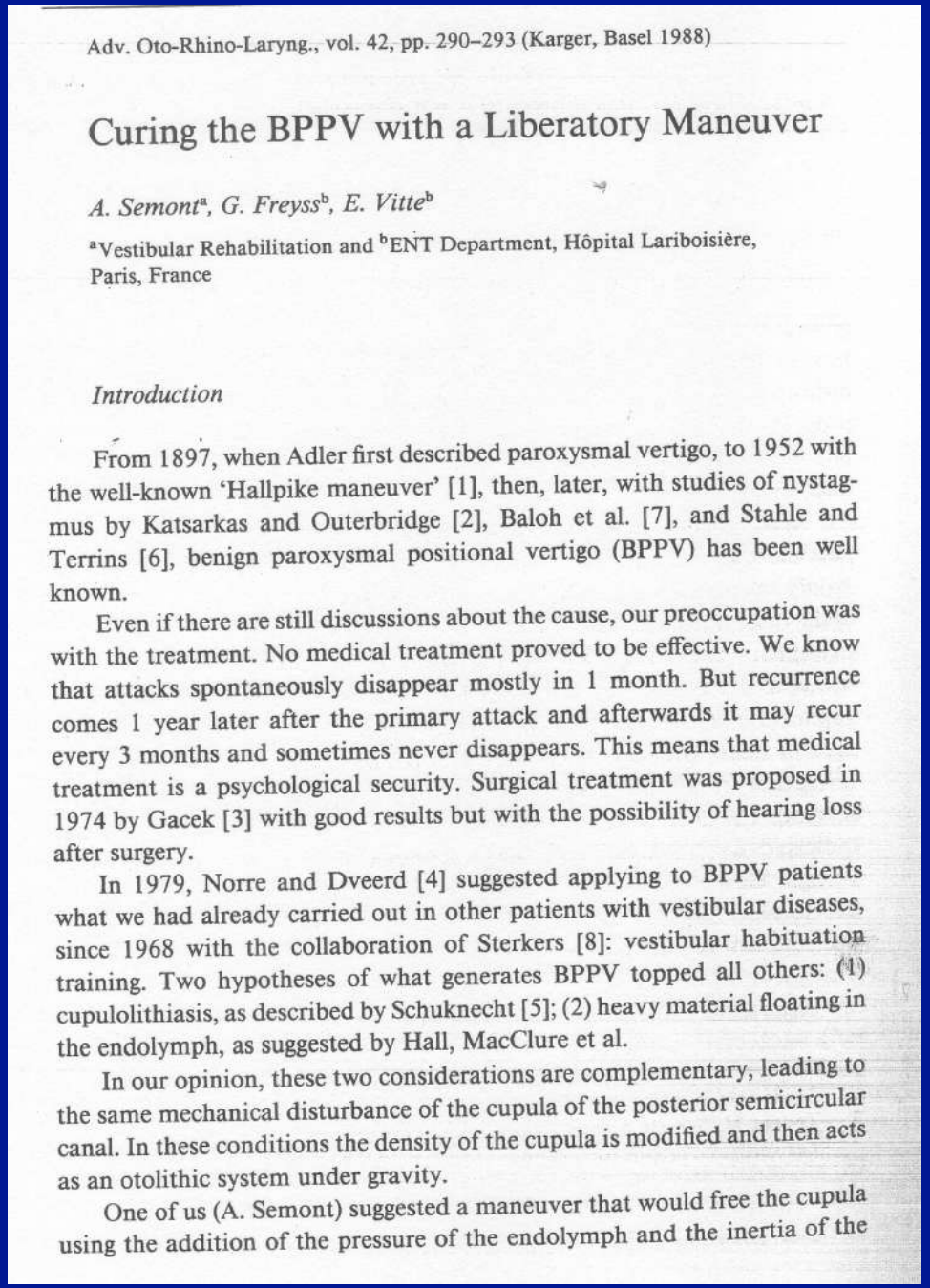
1996

Nuti D, Vannucchi P, Pagnini P "Benign paroxysmal positional vertigo of the horizontal canal: a form of canalolithiasis with variable clinical features" J Vestib Res 1996; 6 173-84



Adv. Oto-Rhino-Laryng., vol. 42, pp. 290–293 (Karger, Basel 1988)

Curing the BPPV with a Liberatory Maneuver

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Introduction

From 1897, when Adler first described paroxysmal vertigo, to 1952 with the well-known 'Hallpike maneuver' [1], then, later, with studies of nystagmus by Katsarkas and Outerbridge [2], Baloh et al. [7], and Stahle and Terrins [6], benign paroxysmal positional vertigo (BPPV) has been well known.

Even if there are still discussions about the cause, our preoccupation was with the treatment. No medical treatment proved to be effective. We know that attacks spontaneously disappear mostly in 1 month. But recurrence comes 1 year later after the primary attack and afterwards it may recur every 3 months and sometimes never disappears. This means that medical treatment is a psychological security. Surgical treatment was proposed in 1974 by Gacek [3] with good results but with the possibility of hearing loss after surgery.

In 1979, Norre and Dveerd [4] suggested applying to BPPV patients what we had already carried out in other patients with vestibular diseases, since 1968 with the collaboration of Sterkers [8]: vestibular habituation training. Two hypotheses of what generates BPPV topped all others: (1) cupulolithiasis, as described by Schuknecht [5]; (2) heavy material floating in the endolymph, as suggested by Hall, MacClure et al.

In our opinion, these two considerations are complementary, leading to the same mechanical disturbance of the cupula of the posterior semicircular canal. In these conditions the density of the cupula is modified and then acts as an otolithic system under gravity.

One of us (A. Semont) suggested a maneuver that would free the cupula using the addition of the pressure of the endolymph and the inertia of the

1988

Semont A, Freyss G, Vitte E "Curing the BPPV with a liberatory maneuver" Adv Oto-Rhino-Laryngol 1988; 42: 290-3

The canalith repositioning procedure: For treatment of benign paroxysmal positional vertigo

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The Canalith Repositioning Procedure (CRP) is designed to treat benign paroxysmal positional vertigo (BPPV) through induced out-migration of free-moving pathological densities in the endolymph of a semicircular canal, using timed head maneuvers and applied vibration. This article describes the procedure and its rationale, and reports the results in 30 patients who exhibited the classic nystagmus of BPPV with Hallpike maneuvers. CRP obtained timely resolution of the nystagmus and positional vertigo in 100%. Of these, 10% continued to have atypical symptoms, suggesting concomitant pathology; 30% experienced one or more recurrences, but responded well to retreatment with CRP. These results also support an alternative theory that the densities that impart gravity-sensitivity to a semicircular canal in BPPV are free in the canal, rather than attached to the cupula. CRP offers significant advantages over invasive and other noninvasive treatment modalities in current use. (OTOLARYNGOL HEAD NECK SURG 1992;107:399.)

First described by Barany[1] in 1921, the entity of benign paroxysmal positional vertigo (BPPV) was more fully defined in 1952 by Dix and Hallpike,[2] who originated the provocative test now generally referred to as the "Hallpike maneuver." This elicits a pathognomonic "classic" nystagmus characterized by: predominantly rotatory motion with the fast phase directed toward the undermost side, latency, limited duration, reversal on return to upright, and response decline on repetition of the provocative maneuver.

Dix and Hallpike[2] also localized the source of BPPV to the undermost ear in the Hallpike maneuver, leading the way to destructive surgical procedures.[3,4] These usually provided relief of symptoms, but involved significant risk and postoperative morbidity. Thus, there has been a search for an effective noninvasive treatment.

McClure and Willett[5] found antivertiginous medications ineffective in controlling BPPV. Cawthorne[6] advocated "vestibular habituation therapy" (VHT), whereby patients assumed head positions that provoked the symptoms of vertigo, an approach primarily aimed at increasing their tolerance to the vertigo rather than eliminating its cause. Brandt and Daroff[7] described a more specific exercise, which purportedly dispersed heavy debris within labyrinth. Toupet and Semont[8] developed a more aggressive approach termed the "liberatory maneuver," based on the theory that it freed the cupula of heavy debris. Norré and Beckers[9] reported only 52% of 23 BPPV patients treated with the liberatory maneuver were free of vertigo after 1 week, but Semont et al.[10] claimed "positive results" in 92.68%.

All of these methods, however, have significant drawbacks. VHT and other exercise treatments often extend for weeks and temporarily augment symptoms, causing patients to abandon treatment. An alternative of hospitalization with treatment under sedation is impractical. The violent character of the liberatory maneuver is of concern in a litigious society.

Pathophysiologic mechanism of BPPV. In 1962, Schuknecht[3] presented what has become known as the "cupulolithiasis" or "heavy cupula" theory, which held that the posterior semicircular canal (PSC) is rendered sensitive to gravity by dense particles attached to or impinging on its cupula.[11] Whereas the PSC localization has been verified,[4] the "heavy cupula" concept has been brought into question. Paramount are studies showing that when a heavy cupula is created by alcohol or deuterium ingestion,[12,13] the resulting nystagmus, in a particular head position, is sustained for long periods of time, rather than transient as in the classic nystagmus.

An alternative explanation holds that at least some BPPV cases result from free-moving densities in the endolymph of the PSC.[14,15] This is strongly supported by the direct surgical observation by Parnes and McClure[16] of free densities, resembling accumulations

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23/1/37658

1992

Epley JM "The canalith repositioning procedure: for treatment of benign paroxysmal positional vertigo" Otolaryngol HNS 1992; 107: 399-404

BENIGN PAROXYSMAL VERTIGO OF CHILDHOOD

(A Variety of Vestibular Neuronitis)

By

L. S. BASSER[1]

Introduction

In the past fifteen years and particularly in the last decade there has been a considerable and remarkable clarification of the varieties of vertigo occurring in adult life. This has been due in part to the establishment of consistently reliable techniques of estimating vestibular function and also to the increased attention given to these conditions.

The position in children is very different. Vertigo in childhood has received very little attention and in fact often goes unrecognized, as will be discussed. The general view is exemplified by Ford (1960), who refers to the purulent and rare serous forms of labyrinthitis complicating gross infections of the middle ear and mastoid process, to vertigo as a symptom of a number of conditions particularly brain-stem lesions and posterior fossa neoplasms, and to Ménière's disease which he states is "so rare in childhood that it does not require more than a very brief discussion." Confirming this view, the case quoted in illustration by him, as well as being atypical of Ménière's disease, also had a posterior fossa exploration during the course of her investigations. No other form of vertigo is mentioned. Harrison (1962*a*) has discussed other varieties of vertigo occurring in children.

The purpose of this paper is to draw attention to the problem and to describe a particular variety of paroxysmal vertigo occurring in childhood, not previously reported, not uncommon, with characteristic and readily recognizable features, and differing from other forms of paroxysmal vertigo found in adult life. It has both practical and theoretical importance.

[1]193 Macquarie Street, Sydney, N.S.W., Australia.

1964

Basser LS "Benign paroxysmal vertigo of childhood" Brain 1964; 87: 141-52

Brain (1984), **107**, 1123–1142

NEURO-OTOLOGICAL MANIFESTATIONS OF MIGRAINE

by ALTAN KAYAN[1] *and* J. DERRICK HOOD

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SUMMARY

Vestibulocochlear derangements have been studied in three groups of patients: 200 unselected patients with migraine (Series I), 80 migrainous patients referred because of their symptoms for full neuro-otological examination (Series II), and 116 patients with tension headache who served as controls (Series III). Significant differences were established between tension headache and migraine in respect of incidence and severity of symptoms and their time of onset in relation to the headache. In migraine, vestibulocochlear disturbances can occur as an aura, accompanying the headache or during headache-free periods, the highest incidence occurring during the headache. In Series I, 59 per cent reported vestibular and/or cochlear symptoms and these were of disabling severity in 5 per cent. Significantly, 50 per cent had a history of motion sickness and 81 per cent experienced phonophobia during the headache, the probable mechanism of which is discussed.

Persisting vestibulocochlear derangements were found in 77.5 per cent of Series II, largely vestibular and of both central and peripheral origin. Involvement of the vertebrobasilar vascular system appears to be the most likely explanation. Possible links between Ménière's disease, benign paroxysmal vertigo and migraine are discussed.

INTRODUCTION

The association of disorders of hearing and balance with migraine has long been recognized. As early as AD 131 Aretaeus of Cappadocia (cited in Sachs, 1970) gave a particularly colourful description of the occurrence of both during what was undoubtedly an attack of migraine. In more modern times, it was Liveing in 1873 who was the first to draw attention to the clear association of vertigo with migraine. Since then, Gowers (1907), Symonds (1926) and Graham (1968), amongst others, have all taken the view that vertigo and/or disturbance of hearing can truly be described as migrainous prodromata. In particular, Bickerstaff (1961) introduced the concept of 'basilar artery migraine', that is, attacks of migraine commencing with visual disturbances, vertigo, ataxia, slurred speech, tinnitus and sensory disturbances of the distal parts of the limbs and around the lips followed by throbbing, occipital headache.

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Correspondence to: Dr J. D. Hood, MRC Neuro-Otology Unit, National Hospital, Queen Square, London, WC1N 3BG.

1984

Kayan A, Hood D "Neurotological manifestations of migraine" Brain 1984; 107: 1123-42

The interrelations of migraine, vertigo, and migrainous vertigo

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Article abstract—*Objective*: To assess the prevalence of migrainous vertigo in patients with migraine and in patients with vertigo according to explicit diagnostic criteria that are presented for discussion. *Methods:* The authors prospectively evaluated 200 consecutive patients from a dizziness clinic and 200 patients from a migraine clinic for migrainous vertigo based on the following criteria: 1) recurrent vestibular symptoms (rotatory/positional vertigo, other illusory self or object motion, head motion intolerance); 2) migraine according to the criteria of the International Headache Society (IHS); 3) at least one of the following migrainous symptoms during at least two vertiginous attacks: migrainous headache, photophobia, phonophobia, visual or other auras; and 4) other causes ruled out by appropriate investigations. In addition, the authors compared the prevalence of migraine according to the IHS criteria in the dizziness clinic group with a sex- and age-matched control group of 200 orthopedic patients. *Results:* The prevalence of migraine according to the IHS criteria was higher in the dizziness clinic group (38%) compared with the age- and sex-matched control group (24%, $p < 0.01$). The prevalence of migrainous vertigo was 7% in the dizziness clinic group, and 9% in the migraine clinic group. In 15 of 33 patients with migrainous vertigo, vertigo was regularly associated with migrainous headache. In 16 patients, vertigo occurred both with and without headache, and in two patients headache and vertigo never occurred together. The duration of attacks varied from minutes to days. *Conclusion:* These results substantiate the epidemiologic association between migraine and vertigo and indicate that migrainous vertigo affects a significant proportion of patients both in dizziness and headache clinics.

NEUROLOGY 2001;56:436–441

Patients with both episodic vertigo and migraine have been reported from the early days of clinical neurology.[1,2] The International Headache Society (IHS) classification of migraine,[3] however, refers to vertigo as a migrainous symptom in adults only within the framework of basilar migraine, which may include vertigo as an aura symptom. A more comprehensive concept of migrainous vertigo appears to take shape from several published case series[4-16] under various names such as benign recurrent vertigo,[5,13,16] migraine-associated dizziness,[6,8] migraine-related vestibulopathy,[7] or vestibular migraine,[9] but is not yet clearly defined. So far, all studies on migrainous vertigo have been retrospective and, with a few exceptions,[7,9] have not been based on explicit inclusion and exclusion criteria. Only one study[6] has strictly adhered to the IHS criteria for the diagnosis of migraine. The understanding of migrainous vertigo is further compromised by the lack of prevalence estimates.

Among clinicians, not surprisingly, the acceptance of migrainous vertigo as a diagnostic entity is still rather poor. In addition to the methodologic shortcomings of previous studies, this may be due to a simple statistical consideration: with a prevalence of migraine of 15 to 17% in women and 5 to 8% in men[17] and a prevalence of dizziness in the general population of more than 20%,[18] one may question whether there is more than a coincidental association of migraine and vertigo.

What data are available to support a true association between migraine and vertigo? Vertigo has been found to be three times more common in patients with migraine than in controls.[19,20] Conversely, a high prevalence of migraine of 30 to 50% was found in patients presenting with vertigo in several case series.[4,15] This finding, however, has not yet been confirmed by a controlled study.

Therefore, we have conducted this study 1) to assess the prevalence of IHS migraine in patients with vertigo as compared with controls; 2) to identify patients with migrainous vertigo according to explicit diagnostic criteria in populations with vertigo and in populations with migraine as the chief complaint; and 3) to delineate the clinical features of migrainous vertigo.

2001

Neuhauser H et al "The interrelations of migraine, vertigo and migrainous vertigo" Neurology 2001; 56: 436-41

VERTIGINE EMICRANICA
Classificazione di Pagnini, 1999

VERTIGINE EMICRANICA ASSOCIATA (VMA)

- **1. COME AURA**
- **2. CONCOMITANTE**
  - **EMICRANIA senz' AURA**
  - **EMICRANIA con AURA**
  - **EMICRANIA BASILARE**

VERTIGINE EMIGRANICA EQUIVALENTE (VME)

- **3. EQUIVALENTE PRECOCE**
  - **VERTIGINE PAROSSISTICA dell' INFANZIA**
  - **TORCICOLLO PAROSISTICO dell' INFANZIA**
- **4. EQUIVALENTE INTERCRITICO** → **VERTIGINE RICORRENTE BENGNA dell' ADULTO**
- **5. EQUIVALENTE TARDIVO** → **VERTIGINE EQUIVALENTE EPIGONA**

Sound- and/or Pressure-Induced Vertigo Due to Bone Dehiscence of the Superior Semicircular Canal

Lloyd B. Minor, MD; David Solomon, MD, PhD; James S. Zinreich, MD; David S. Zee, MD

Objectives: To present symptoms, patterns of nystagmus, and computed tomographic scan identification of patients with sound- and/or pressure-induced vertigo due to dehiscence of bone overlying the superior semicircular canal. To describe anatomical findings and outcome in 2 patients undergoing plugging of the superior semicircular canal for treatment of these symptoms.

Design and Setting: Prospective study of a case series in a tertiary care referral center.

Patients and Results: Eight patients with vertigo, oscillopsia, and/or disequilibrium related to sound, changes in middle ear pressure, and/or changes in intracranial pressure were identified in a 2-year period. Seven of these patients also had vertical-torsional eye movements induced by these sound and/or pressure stimuli. The direction of the evoked eye movements could be explained by excitation or inhibition of the superior semicircular canal in the affected ear. Computed tomographic scans of the temporal bones identified dehiscence of bone overlying the affected superior semicircular canal in each case. Disabling disequilibrium in 2 patients prompted plugging of the dehiscent superior canal through a middle cranial fossa approach. Symptoms were improved in each case. One patient developed recurrent symptoms requiring an additional plugging procedure and developed sensorineural hearing loss several days after this second procedure.

Conclusions: We have identified patients with a syndrome of vestibular symptoms induced by sound in an ear or by changes in middle ear or intracranial pressure. These patients can also experience chronic disequilibrium. Eye movements in the plane parallel to that of the superior semicircular canal were evoked by stimuli that have the potential to cause ampullofugal or ampullopetal deflection of this canal's cupula in the presence of a dehiscence of bone overlying the canal. The existence of such deshiscences was confirmed with computed tomographic scans of the temporal bones. Surgical plugging of the affected canal may be beneficial in patients with disabling symptoms.

Arch Otolaryngol Head Neck Surg. 1998;124:249-258

From the Departments of Otolaryngology–Head and Neck Surgery (Dr Minor), Radiology (Dr Zinreich), and Neurology (Dr Zee), The Johns Hopkins University School of Medicine, Baltimore, Md; and the Department of Neurology, University of Pennsylvania School of Medicine, Philadelphia (Dr Solomon).

VESTIBULAR responses to sound and/or pressure transmitted to an inner ear were initially documented in studies during the first quarter of this century. The Tullio phenomenon is vertigo or other abnormal vestibular sensations accompanied by eye and/or head movements in response to sound. In the initial experimental studies by Tullio,[1] later elaborated by Huizinga[2] and Eunen et al,[3] fenestration of individual semicircular canals in pigeons led to sound-evoked eye and head movements in the plane of these canals. These responses were abolished by application of cocaine to the ampulla of the fenestrated canal. It was further noted that sound-evoked eye movements could be produced without surgical interventions on the labyrinth when greater intensity stimuli were used. Young et al[4] subsequently showed that vestibular nerve afferents in the squirrel monkey respond to sound with phase-locking thresholds typically higher than 100-dB sound pressure level and rate-change thresholds 10 to 30 dB higher than intensities required for phase locking.

Clinical studies initially identified the Tullio phenomenon in patients with congenital syphilis. The histopathological features of the temporal bone in these cases was shown to be gummatous osteomyelitis and labyrinthine fistulae.[5] Vestibular symptoms and/or eye movements evoked by sound have also been demonstrated in congenital deafness,[6] Ménière disease,[7] perilymph fistula,[8] head trauma,[7,9] Lyme disease,[10] and cholesteatoma with semicircular canal erosion and fenestration.[11]

1998

Minor LB, Solomon D, Zinreich JS, Ze DS "Sound- and/or pressure-induced vertigo due to bone dehiscence of the superior semicircular canal" Arch Otolaryngol 1998; 124: 249-58

Nuovi nistagmi

REBOUND NYSTAGMUS

BY

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SPONTANEOUS nystagmus characterized by a fast component in one direction followed sequentially by a slow component in the other is pathognomonic of disorders of the vestibular system and may result from a lesion of the peripheral organ or of its central connexions in the brain-stem, the cerebellum or the cerebrum.

In recent years electro-nystagmography has made possible its systematic recording and analysis and as a result certain useful criteria have been established which have proved helpful in the localization of the lesion giving rise to the nystagmus. By way of example, the tracings shown in fig. 1 typify spontaneous nystagmus encountered in (A) a peripheral lesion, (B) an eighth nerve tumour with involvement of the vestibular nuclei within the brain-stem, and (C) a high brain-stem lesion. The recordings are carried out with DC amplification and hence provide a complete description of the nystagmus indicating its magnitude and occurrence in the primary position of the eyes together with lateral gaze deviation to right and left. Although the wave forms of the nystagmus in all three are indistinguishable, certain striking and diagnostically significant changes take place when fixation is removed by recording in total darkness with the eyes open. Thus in (A) the nystagmus is markedly enhanced both in amplitude and frequency; in (B) it is inhibited particularly in respect of the slow component velocity; and in (C) it is completely abolished.

Despite these changes which have an important bearing upon the underlying nervous mechanism in each case (Hood, 1967), it is noteworthy that two important characteristics, clearly evident upon direct examination of the eyes, are common to all: first the strict adherence to Alexander's Law, inasmuch as the nystagmus is enhanced with gaze deviation in the direction of the fast component of the nystagmus, and secondly, the regularity of the nystagmus which persists unfatigued with specific deviations of gaze. This gaze deviation dependence and unfatigability is with few exceptions characteristic of any spontaneous nystagmus resulting from a lesion of the peripheral vestibular system or its central connexions at any level of the brain-stem. Recently, however, a spontaneous nystagmus has been observed which although clearly vestibular in appearance differs conspicuously in a number of respects from any spontaneous nystagmus encountered so far in patients with brain-stem

1973

Hood JD, Kayan A, Leech J "Rebound nystagmus" Brain 1973; 96: 507-26

Downbeat nystagmus: A type of central vestibular nystagmus

1981

Robert W. Baloh, M.D., and Joseph W. Spooner, M.D.

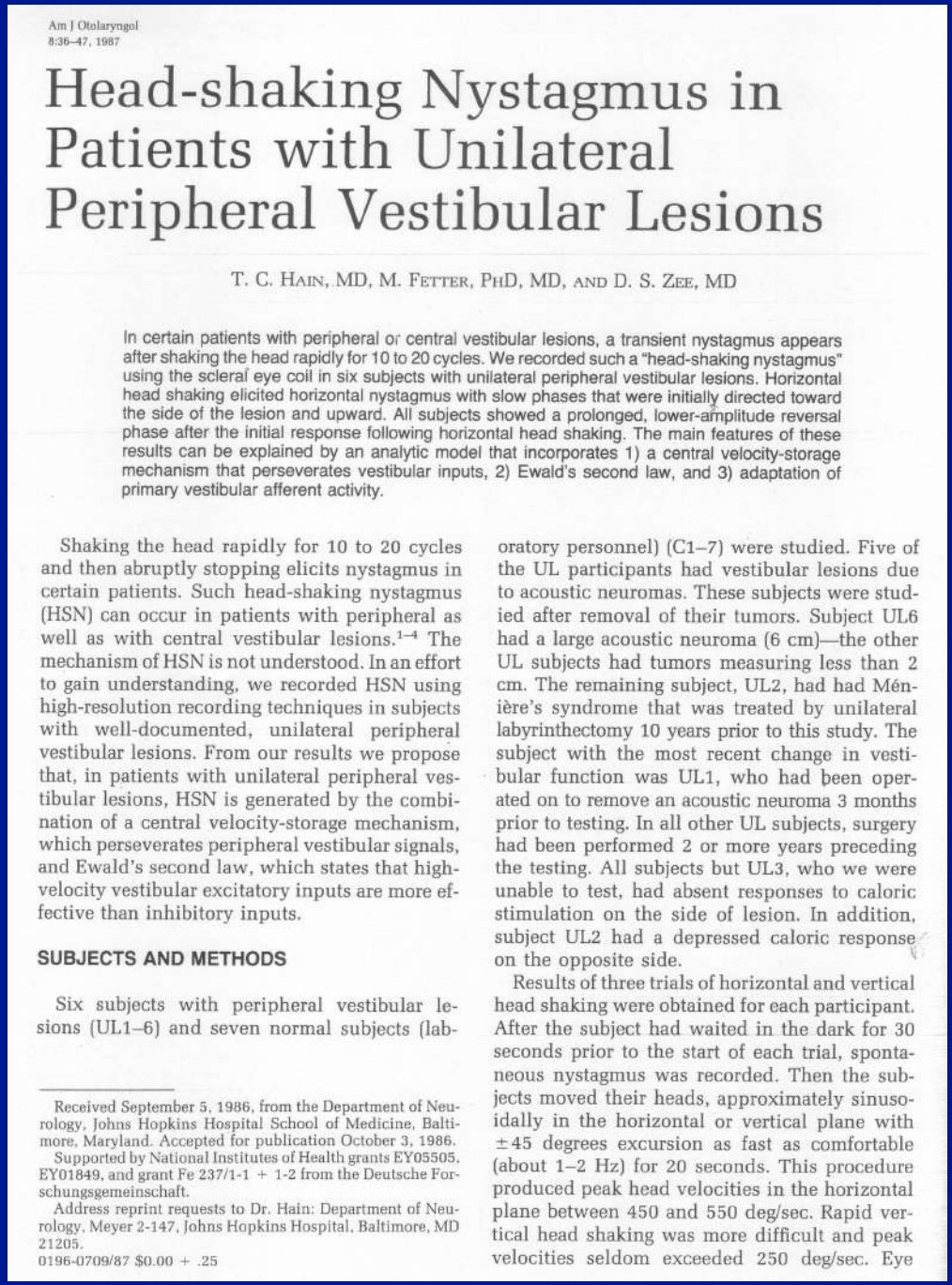
Downbeat nystagmus is a specific neurologic sign.[1,2] About one-third of the patients with this sign have a Chiari malformation, and most of the others have an identifiable lesion near the medullary-cervical junction. We define downbeat nystagmus as spontaneous nystagmus present with fixation in the primary position or on lateral gaze. When downbeat nystagmus is present on downgaze but not in the primary position or on lateral gaze, the implications are less specific, and it is usually associated with gaze paretic nystagmus in all directions of gaze; it is most commonly caused by tranquilizers or anticonvulsant drugs.

No pathophysiologic mechanism has been proposed that adequately explains how a lesion near the medullary-cervical junction causes downbeat nystagmus. Zee et al[3] suggested that downbeat nystagmus is a type of pursuit nystagmus. They observed selective impairment of downward pursuit in three patients with downbeat nystagmus and simulated downbeat nystagmus in an analog computer model of eye movement control by assuming that downgaze velocity information could not be transmitted to the neural integrator that generates downward smooth eye movements. Others suggested that upbeat and horizontal primary position nystagmus could also be varieties of pursuit nystagmus.[4-7] However, while some patients with asymmetric smooth pursuit have primary position nystagmus,[7] others do not.[8-10] Furthermore, patients with spontaneous or induced peripheral vestibular nystagmus and patients with periodic alternating nystagmus have impaired smooth pursuit in the direction of the nystagmus.[10,11] In the former instance, pursuit pathways are clearly normal, and in the latter an alternating pursuit deficit is unlikely. Thus, pursuit asymmetry seen with spontaneous nystagmus may be the result of the nystagmus rather than its cause.[10]

We studied horizontal and vertical ocular control systems in 17 patients with downbeat nystagmus to better understand the mechanism of its production.

Methods. Eye movements were recorded with direct current electrooculography (EOG). Horizontal movements were recorded by electrodes at the inner and outer canthi and vertical movements were recorded with electrodes placed above the eyebrow and below the lower eyelid. The bandwidth of the recording system was 0 to 35 Hz (-3 db). The velocity of slow components of jerk nystagmus, the velocity of smooth pursuit move-

Baloh RW, Spooner JW "Downbeat nystagmus: A type of central vestibular nystagmus" Neurology 1981; 31: 304-10



Am J Otolaryngol
8:36–47, 1987

Head-shaking Nystagmus in Patients with Unilateral Peripheral Vestibular Lesions

T. C. Hain, MD, M. Fetter, PhD, MD, and D. S. Zee, MD

In certain patients with peripheral or central vestibular lesions, a transient nystagmus appears after shaking the head rapidly for 10 to 20 cycles. We recorded such a "head-shaking nystagmus" using the scleral eye coil in six subjects with unilateral peripheral vestibular lesions. Horizontal head shaking elicited horizontal nystagmus with slow phases that were initially directed toward the side of the lesion and upward. All subjects showed a prolonged, lower-amplitude reversal phase after the initial response following horizontal head shaking. The main features of these results can be explained by an analytic model that incorporates 1) a central velocity-storage mechanism that perseverates vestibular inputs, 2) Ewald's second law, and 3) adaptation of primary vestibular afferent activity.

Shaking the head rapidly for 10 to 20 cycles and then abruptly stopping elicits nystagmus in certain patients. Such head-shaking nystagmus (HSN) can occur in patients with peripheral as well as with central vestibular lesions.[1–4] The mechanism of HSN is not understood. In an effort to gain understanding, we recorded HSN using high-resolution recording techniques in subjects with well-documented, unilateral peripheral vestibular lesions. From our results we propose that, in patients with unilateral peripheral vestibular lesions, HSN is generated by the combination of a central velocity-storage mechanism, which perseverates peripheral vestibular signals, and Ewald's second law, which states that high-velocity vestibular excitatory inputs are more effective than inhibitory inputs.

SUBJECTS AND METHODS

Six subjects with peripheral vestibular lesions (UL1–6) and seven normal subjects (laboratory personnel) (C1–7) were studied. Five of the UL participants had vestibular lesions due to acoustic neuromas. These subjects were studied after removal of their tumors. Subject UL6 had a large acoustic neuroma (6 cm)—the other UL subjects had tumors measuring less than 2 cm. The remaining subject, UL2, had had Ménière's syndrome that was treated by unilateral labyrinthectomy 10 years prior to this study. The subject with the most recent change in vestibular function was UL1, who had been operated on to remove an acoustic neuroma 3 months prior to testing. In all other UL subjects, surgery had been performed 2 or more years preceding the testing. All subjects but UL3, who we were unable to test, had absent responses to caloric stimulation on the side of lesion. In addition, subject UL2 had a depressed caloric response on the opposite side.

Results of three trials of horizontal and vertical head shaking were obtained for each participant. After the subject had waited in the dark for 30 seconds prior to the start of each trial, spontaneous nystagmus was recorded. Then the subjects moved their heads, approximately sinusoidally in the horizontal or vertical plane with ±45 degrees excursion as fast as comfortable (about 1–2 Hz) for 20 seconds. This procedure produced peak head velocities in the horizontal plane between 450 and 550 deg/sec. Rapid vertical head shaking was more difficult and peak velocities seldom exceeded 250 deg/sec. Eye

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0196-0709/87 $0.00 + .25

1987

Hain TC, Fetter M, Zee DS "Head-shaking nystagmus in patients with unilateral peripheral vestibular lesions" Am J Otolaryngol 1987; 8: 36-47

Neurology 1999;53:2158

Hyperventilation-induced nystagmus in patients with vestibular schwannoma

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OBJECTIVE: To analyze the nystagmus evoked by hyperventilation in patients with unilateral vestibular schwannoma and to use this information to predict the effects of hyperventilation on individual ampullary nerves.

METHODS: Three-dimensional scleral search coil eye movement recording techniques were used to record the magnitude and time course of eye movements in six patients with unilateral vestibular schwannoma and hyperventilation-induced nystagmus. The presenting complaints in five of these patients were vertigo or dysequilibrium.

RESULTS: The eye movement response to hyperventilation was a "recovery" nystagmus with slow-phase components corresponding to excitation of the affected vestibular nerve. Projection of the eye velocity vector into the plane of the semicircular canals revealed that fibers arising from the ampulla of the horizontal canal were most affected by hyperventilation with lesser activation of fibers to the superior canal and smaller, more variable responses from posterior canal fibers.

CONCLUSIONS: The three-dimensional characteristics of the nystagmus evoked by hyperventilation in patients with vestibular schwannoma provide insight into the vestibular end organs affected by the tumor and the mechanism responsible for the nystagmus. This finding indicates that hyperventilation resulted in a transient increase in activity from these partially demyelinated axons.

LB Minor, et al “Hyperventilation-induced nystagmus in patients with vestibular schwannoma “
Neurology 1999; 53: 2158

Vibration-Induced Nystagmus – A Sign of Unilateral Vestibular Deficit

Karl-Friedrich Hamann Elke-Maria Schuster

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1999

Key Words
Vibration · Peripheral vestibular excitability · Nystagmus

Abstract
Vibrations of 60 Hz and 100 Hz were applied on the mastoid of healthy subjects, patients with unilateral peripheral vestibular lesions, with central lesions of different localizations, and patients with benign paroxysmal positioning vertigo (BPPV). In patients with unilateral peripheral deficit a horizontal nystagmus with a small torsional component beating generally to the not affected side could be observed. This nystagmus did not show adaptation during 40 s. The occurrence was more frequent using 60-Hz stimulations. This vibration-induced nystagmus did never occur in healthy subjects, seldom in patients with central vertigo, and only exceptionally in patients with BPPV. It is concluded that the finding of a vibration-induced nystagmus reflects a side difference of peripheral vestibular excitability.

Introduction

Strictly speaking, vibrations are periodic movements within an unlimited frequency frame. Because of their omnipresence, a human being is permanently exposed to mechanical vibrations. In order to perceive vibration stimuli, human beings as well as many animals have mechanosensors in the skin, muscles (muscle spindles), tendons, the capsula of articulations, and in the inner ear. In clinical examination its capacity is tested by placing a tuning fork on certain defined areas or by applying special vibration coils. The sensation of vibration might be impaired by several diseases, e.g. polyneuritis.

The perception of vibration plays a role in the orientation of an individual in space as could be shown by Goodwin et al. [1]. By means of vibrations over muscles illusions of motion were evoked. This observation also proves that there must be a cortical representation of muscle spindles. These findings were confirmed and elaborated in detail by several authors [1–4]. The receptors for vibration stimuli were identified as muscle spindles [1, 2] whose stimulation leads to an activation of Ia fibers [4]. A direct effect on the vestibular system was 'definitively excluded' by Biguer et al. [3].

By vibration it is possible to induce, besides the motion illusion, postural changes that show a direct relation between the stimulated muscle and the direction of body destabilization [4]. In patients suffering from vestibular neuritis a change of horizontal eye position could be induced by 100 Hz vibration of neck muscles [5]. However, a nystagmus-like pattern of eye movements has never been observed. In contrast to these findings Kobayashi et al. [6] detected, by observation with Frenzel's glasses, a

Hamann KF, Schuster EM
"Vibration-induced-nystagmus: a sign of unilateral vestibular deficit"
ORL 1999; 61: 74-9

Non solo nistagmi: AVD, OTR, HIT

The dynamic illegible E (DIE) test: a simple technique for assessing the ability of the vestibulo-ocular reflex to overcome vestibular pathology

N. S. LONGRIDGE, M.B., F.R.C.S.(C)* and A. I. MALLINSON, M.Sc.† Vancouver, Canada

Abstract. Vestibulo-ocular reflex (VOR) compensation, shown by the Dynamic Illegible E test (DIE test), for vestibular damage (as shown by caloric reduction) did not coincide consistently with time from initial onset of vestibular disease. We theorize that the reason for this is the variable efficiency of different compensatory mechanisms used by individuals in overcoming vestibular pathology. Methods of compensation are discussed in detail. The DIE test measures the efficiency of the VOR by comparing visual acuity with head still and head moving. While many patients compensated satisfactorily for vestibular injury to the point where they were able to function adequately, other patients appeared not to compensate. There was no obvious prior indication of which patients would compensate well. There was a suggestion that younger patients did better, but neither duration of symptoms nor severity of pathology appeared to be factors in the ability to compensate. Although younger patients may fare better as a group, there are no predictive criteria for the amount or rate of recovery which an individual patient with vestibular pathology will make.

Vestibular compensation is a multifaceted process of change throughout a broad area of the central nervous system (CNS) including the vestibular nuclei, spinal cord, the visual system, the cerebellum, the inferior olive, and the commissural system. It is a process which takes place in several stages resulting in the reorganization of neural circuits manifesting as a gradual recovery of equilibrium[1].

Compensation does not necessarily result in a return to previous levels of ability. Even a patient who has compensated well for a vestibular injury will frequently admit to imbalance, insecurity or vertigo upon being subjected to an extreme vestibular stress, such as a very rapid head movement.

The Dynamic Illegible E (DIE) test measures the difference between visual acuity with head still and head passively moved *(Figure 1)*. It assesses the efficiency of the vestibulo-ocular reflex (VOR). We consider it to be more sensitive than standard caloric testing. In our initial study[2] we found that the DIE test was performed better by a group of normal volunteers than it was performed by an age- and sex-matched group of dizzy patients who had normal caloric responses. During numerous patient assessments we formed the clinical impression that patients with a long history of dizziness and caloric reduction had better DIE test scores than those with dizziness due to a recent vestibular insult. We considered this to indicate that the DIE test measured recovery of accurate VOR function.

In general, vestibular compensation refers to the ability of a patient to regain such vestibular function as is needed to carry out normal daily activities in a satisfactory manner. For the purposes of this paper, we use the DIE test as a means of estimating the degree of compensation of a patient by quantifying the degree of recovery of the VOR. We aimed to define how well the test indicated the degree of compensation after initial vestibular damage in relation to

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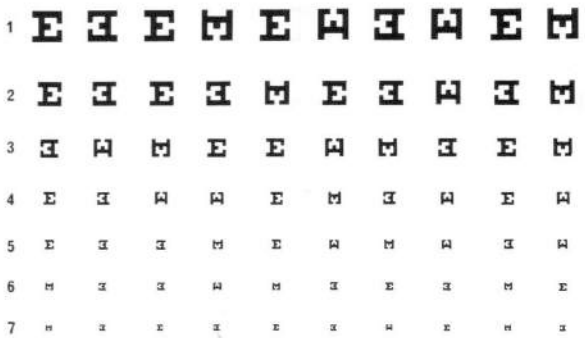

Fig. 1. The Dynamic Illegible E (DIE) test. Actual size 40 × 50 cm (16″ × 20″). Visual acuity ranges from 20/100 (row 1) to 20/80, 20/60, 20/40, 20/30, 20/25 and 20/20 (row 7).

1986

Visual acuity during head shaking
(normal subjects lose one or no lines)

Ocular Tilt Reaction with Peripheral Vestibular Lesion

G. Michael Halmagyi, FRACP,
Michael A. Gresty, PhD,
and William P. R. Gibson, FRCS

Following inadvertent destruction of the left vestibular labyrinth during stapedectomy, a patient developed a transient abnormality of posture consisting of leftward ocular counterrolling, leftward head tilting, and a right-over-left skew deviation. This postural pattern, known as the "ocular tilt reaction," is the normal compensatory response of the dependent utricle to tilting. In this patient, the unopposed action of the intact right utricle was presumably responsible for the appearance of a normal leftward ocular tilt reaction.

Halmagyi GM, Gresty MA, Gibson WPR: Ocular tilt reaction with peripheral vestibular lesion. Ann Neurol 6:80–83, 1979

Unilateral destruction or denervation of one functioning labyrinth invariably causes imbalance and a horizontal nystagmus beating toward the intact side. Postural changes are obvious in animals but are not usually so in humans. This report concerns a patient who did develop obvious abnormalities of head and eye posture following unilateral labyrinthine destruction during aural surgery.

A 37-year-old woman had experienced episodic vertigo and hearing loss in the left ear for three and a half years. The vertigo was rotational and was associated with low-pitched tinnitus and a further temporary impairment of hearing in the left ear. Examination of the ears and cranial nerves showed no abnormality apart from deafness. A pure-tone audiogram indicated that the patient had a marked conductive hearing loss in the left ear with an additional sensorineural impairment at higher frequencies. There was also a high-frequency sensorineural loss in the right ear. Roentgenograms of the mastoid, serological tests for syphilis, and a CT scan were normal. Caloric testing was limited to water at 44°C for 30 seconds because of intense nausea. Irrigation of the right ear resulted in nystagmus of 100 seconds' duration, and irrigation of the left ear in nystagmus for 105 seconds. The clinical diagnosis was endolymphatic hydrops secondary to otosclerosis [6], and after she had had several months of freedom from vertigo, a stapedectomy was undertaken on the left ear (W. P. R. G.). At operation a type II footplate was encountered. No attempt was made to remove it in toto, but a thin, 4.5 mm Teflon piston was placed snugly through a fenestration in its posterior part. The oval window was sealed using part of the chorda tympani, which had been severed during removal of the posterior bony meatal wall. The operation lasted 15 minutes.

Postoperatively the patient complained of vertical diplopia and vertigo, and was referred for a neuroophthalmological opinion (G. M. H.). On examination, visual acuity was 20/16 in each eye; color vision, pupils, lids, corneal reflexes, and orbicularis oculi muscles were normal. There was a head tilt to the left (Fig 1) and a right-over-left hypertropia that was most marked on rightward and upward gaze (Fig 2). The Bielschowsky head-tilt test did not cause any consistent change in the vertical deviation. First-degree horizontal nystagmus beating to the right was noted. With the head erect, the fundi were rotated 25 degrees in a clockwise direction (Fig 3) and there was a corresponding displacement of the blind spots in the visual fields. Other abnormal signs were past-pointing to the left with each arm, falling to the left in the Romberg test, and deviation to the left in the Fukuda vertical writing test.

Eye movements were examined in light and in total darkness by combined electrooculography and infrared video recording. This investigation confirmed the first-degree horizontal nystagmus beating to the right and revealed it to be of third degree in darkness. Optokinetic and pursuit eye movements were mildly impaired to the right, to a degree consistent with the spontaneous nystagmus to the right.

It was concluded that the patient had suffered operative injury to the left vestibular system. She was treated with bed rest for one week and then with graded mobilization. The diplopia resolved in two weeks, and two months later she was able to walk with a minimum of unsteadiness. There was still first-degree nystagmus to the right in the dark, but the head tilt, skew deviation, and tonic ocular counterrolling (OCR) had resolved. She had binocular single vision in all fields, a 40-second arc of stereopsis, and 12 prism diopters of horizontal and 2 diopters of vertical fusional vergence. Hearing in the left ear had improved in the low tones but had deteriorated in the high tones. Caloric testing showed total left canal paresis and a directional preponderance to the right.

Discussion

Selective loss of vestibular function in the operated ear is a recognized complication of stapedectomy [1]. In such cases, unopposed activity of the intact opposite horizontal semicircular canal results in a nystagmus beating toward the intact side. We assume that in this patient, unopposed activity of the intact right labyrinth was also responsible for the observed postural abnormalities. These abnormalities were: 10 degrees of leftward head tilting; 7 degrees of right-

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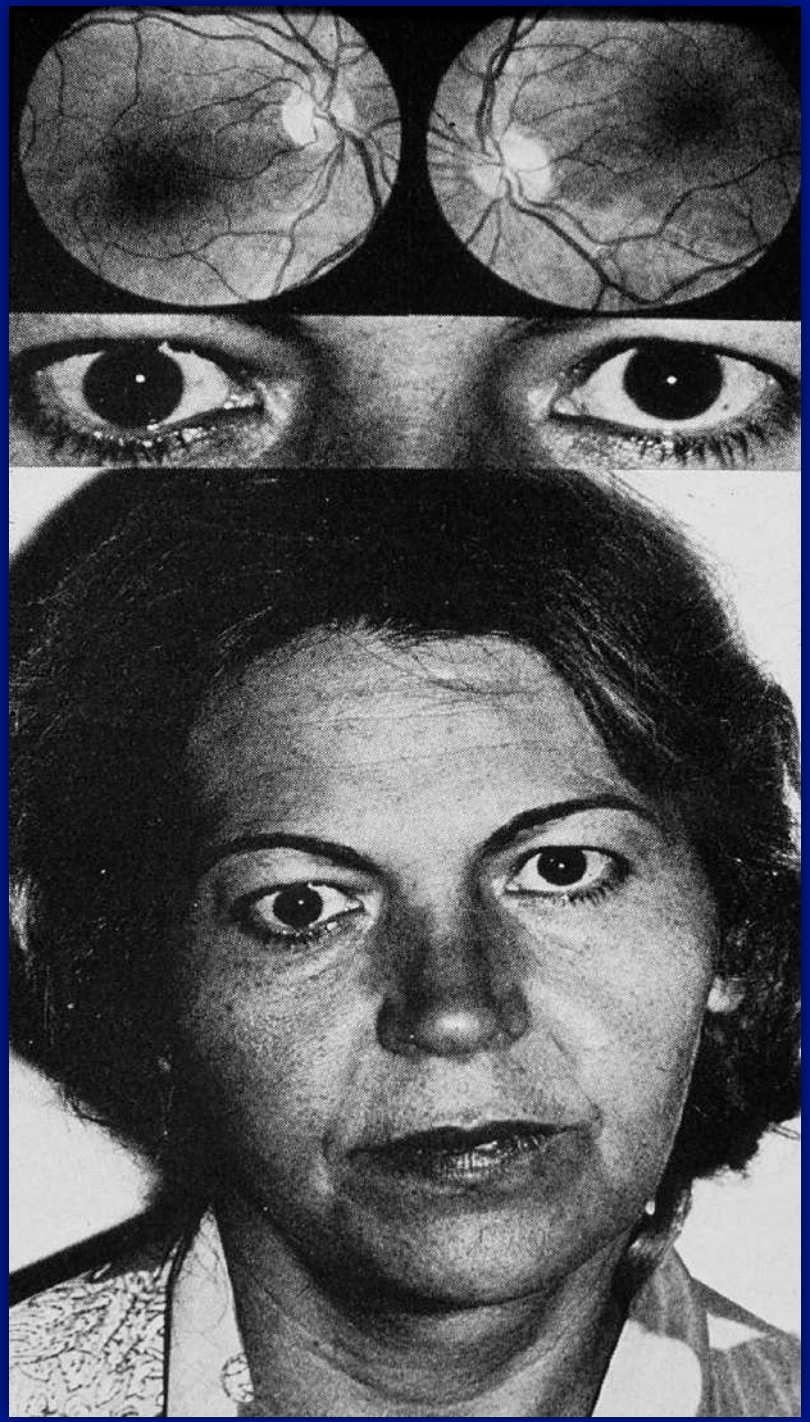
 0364-5134/79/070080-04$01.25

1979

Halmagyi GM, Gresty MA, Gibson WPR "Ocular tilt reaction with peripheral vestibular lesion" Ann Neurol 1979; 6: 80-3

Ocular Tilt Reaction OTR



COUNTERROLL

SKEW

HEAD TILT

A Clinical Sign of Canal Paresis

G. Michael Halmagyi, MB, BS, Ian S. Curthoys, PhD

• **Unilateral loss of horizontal semicircular canal function, termed *canal paresis*, is an important finding in dizzy patients. To our knowledge, apart from head-shaking nystagmus, no clinical sign of canal paresis has yet been described and the term derives from the characteristic finding on caloric tests: little or no nystagmus evoked by either hot or cold irrigation of the affected ear. We describe a simple and reliable clinical sign of total unilateral loss of horizontal semicircular canal function: one large or several small oppositely directed, compensatory, refixation saccades elicited by rapid horizontal head rotation toward the lesioned side. Using magnetic search coils to measure head and eye movement, we have validated this sign in 12 patients who had undergone unilateral vestibular neurectomy.**

(*Arch Neurol* 1988;45:737-739)

While impairment of vestibular function of one ear compared with the other is the pathophysiologic basis of most vertigo, clinical examination of patients complaining of vertigo is generally unrewarding. We describe a clinical sign that we have found in patients who have lost all vestibular function in one ear: compensatory refixation saccades elicited only by rapid head movements toward the affected side and not by head movements toward the intact side. We have verified this sign by objective head and eye movement recordings and suggest that it may be useful in the clinical evaluation of dizzy patients.

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PATIENTS AND METHODS

Patients

Twelve patients were examined before and from one week to one year after unilateral vestibular neurectomy for acoustic neuroma or intractable vertigo. All 12 had some caloric responses from the affected ear before surgery but no caloric responses, even with 0°C irrigation, after surgery. Each patient and 12 control subjects gave informed consent for these tests and the protocols had been approved by the hospital's Human Ethics Committee.

Clinical Examination

Patients would sit upright and fix their gaze on a target about 3 m away. The examiner would sit facing the patient and give the following instructions: "Please keep looking carefully at the target while I turn your head from one side to the other" (Fig 1, A). When the patients had accustomed themselves to the test and could relax their neck muscles, the examiner would try to turn the patient's head as quickly as possible to one side. It was easiest to do this if the head was already positioned 20° or so away from the side to which it was about to be turned. The examiner could then observe that during such rapid head rotations, a normal subject would not make any saccades, indicating that the subject's gaze had remained fixed on target. Patients with a total unilateral canal paresis, on the other hand, could only keep their gazes fixed on target when their heads were turned *away* from the lesioned side, ie, toward the intact side (Fig 1); when their heads were turned *toward* the lesioned side, they would invariably make one or more, clinically evident, compensatory, refixation saccades in the opposite direction to the head motion (Fig 3). The examiner can readily observe compensatory saccades if he keeps reminding the patient to (1) try to fix on target; (2) avoid making *anti*compensatory saccades (ie, saccades in the direction of head movement); and (3) avoid blinking. With a little perseverance, all of our patients were able to cooperate with these instructions.

Head and Eye Movement Examination

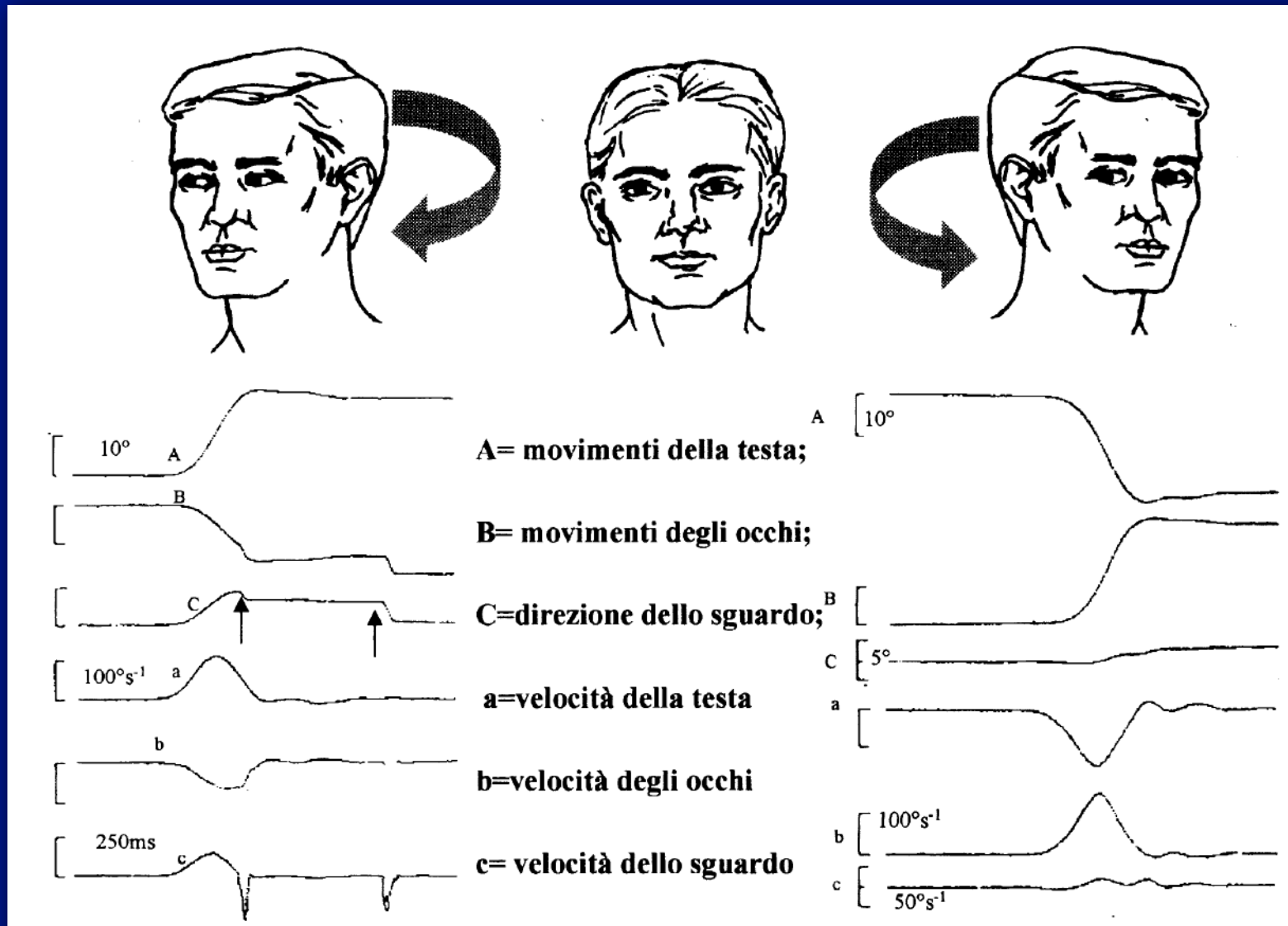
Horizontal displacement of the head and of the left eye were recorded, using magnetic search coils.[1] The test paradigm was identical to the clinical paradigm, except that the fixation point was 1 m, not 3 m, away and the examiner turned the patient's head from behind and not from in front.

Caloric Tests

Each ear was irrigated in turn with water at 30°C and 44°C from a closed-loop balloon system. For irrigation at 0°C following vestibular neurectomy, the ear was syringed with 50 mL of ice water. Vestibular nystagmus was recorded, using an infrared scleral reflection system and monitored with infrared, closed circuit television.

1988

Halmagyi GM, Curthoys IS "A clinical sign of canal paresis" Arch Neurol 1988; 54: 737-9


10°
A
B
C
100°s-1
a
b
250ms
c
A= movimenti della testa;
B= movimenti degli occhi;
C=direzione dello sguardo;
a=velocità della testa
b=velocità degli occhi
c= velocità dello sguardo
A
10°
B
C
5°
a
b
100°s-1
c
50°s-1
Lato patologico
Lato sano

Non solo movimenti oculari: VVS e DHI

Pergamon

Journal of Vestibular Research, Vol. 5, No. 1, pp. 35–45, 1995

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Original Contribution

THE SUBJECTIVE VISUAL VERTICAL AS A CLINICAL PARAMETER OF VESTIBULAR FUNCTION IN PERIPHERAL VESTIBULAR DISEASES

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Abstract – The subjective visual vertical, SV, was measured in the upright and side positions in 25 normal subjects and in 73 patients with various peripheral vestibular disorders. Significant deviations of SV (toward the affected ear) were found in 100% of the patients with vestibular nerve section and with Ramsay Hunt syndrome, in 89% of the patients with vestibular neuritis, and in 0% of the patients with benign paroxysmal positional vertigo. The deviation of SV gradually disappeared within a few weeks of the onset of the disease in all patients except in those with total VIIth nerve resection. SV is a parameter of tonic afferent differences between the two labyrinths similar to vestibular spontaneous nystagmus but is mediated by other parts of the inner ear (probably the otolith organs) and thus provides additional information on the labyrinthine function. SV measured in 90° side positions, however, did not reveal asymmetric vestibular sensitivity, which is in contrast to SV tested during eccentric rotation in patients after vestibular neurectomy.

Keywords – subjective visual vertical; side position; vestibular diseases; vestibular neurectomy.

Introduction

It has been known for more than 100 years that perception of vertical (and horizontal) does not depend solely on visual information but is affected by head position relative to gravity and by linear acceleration forces acting on the gravity vector. In a 90° side position, a vertical line in an otherwise dark surround is perceived as being tilted with the upper end shifted toward the feet (*A* phenomenon, (1)), and Mach (2) described the apparent tilting of houses and trees while travelling around a curve in a railroad. He clearly attributed this phenomenon to the vestibular organs, and, consequently, perception of vertical has been widely investigated for theoretical purposes (3–8). Even in the absence of gravitational forces, astronauts were able to judge the position of an illuminated line in the dark with precision, indicating that the proprioceptive system can contribute to this performance (9). Significant tilts of the subjective visual vertical have been described in patients with peripheral vestibular disorders including labyrinthectomy as well as in patients with brainstem, cerebellar, and thalamic lesions (10–13). Contradictory findings were reported in regard to the correlation between the tilt angles of the subjective visual vertical and static ocular torsion, which occurs as an oculomotor consequence of central and peripheral vestibular lesions (12,14). In addition to these measurements of the subjective visual vertical in a static upright body position, tilting the gravitational vector relative to body longitudinal axis (by excentrical centrifugation of the subject) has made it possible to detect differences in the roll-tilt perception sensitivity between the two labyrinths in vestibular neurectomized subjects (15). Nevertheless, the subjective visual vertical has found little attention in routine clinical testing to date.

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1995

Bohmer A, Rickenmann J
“The subjective visual vertical as a clinical parameter of vestibular function in peripheral vestibular diseases”
J Vestib Res 1995; 5: 35-45

Bedside Examination of the Otoliths static disturbances

Perception of upright

- Tests of the Subjective Visual Vertical
- Patient adjusts a vertical line in an otherwise dark room

The Development of the Dizziness Handicap Inventory

Gary P. Jacobson, PhD, Craig W. Newman, PhD

• Conventional vestibulometric techniques are inadequate for quantifying the impact of dizziness on everyday life. The 25-item Dizziness Handicap Inventory (DHI) was developed to evaluate the self-perceived handicapping effects imposed by vestibular system disease. The development of the preliminary (37 items) and final versions (25 items) of the DHI are described. The items were subgrouped into three content domains representing functional, emotional, and physical aspects of dizziness and unsteadiness. Cronbach's α coefficient was employed to measure reliability based on consistency of the preliminary version. The final version of the DHI was administered to 106 consecutive patients and demonstrated good internal consistency reliability. With the exception of the physical subscale, the mean values for DHI scale scores increased significantly with increases in the frequency of dizziness episodes. Test-retest reliability was high.

(*Arch Otolaryngol Head Neck Surg.* 1990;116:424-427)

Quantifying the effects of medical, surgical, and rehabilitative management of the dizzy patient is problematic. Caloric testing is ineffective in evaluating treatment, since a unilateral weakness observed during the initial examination usually persists on reexamination. Although pretreatment and posttreatment rotary chair and platform posturographic testing may serve to document the process of central vestibular system compensation, these procedures are unable to quantify the effects of unsteadiness on everyday function. It appears, therefore, that conventional diagnostic tests are inadequate for evaluating the handicapping effects imposed by vestibular system disease.

Many clinicians either have developed their own or employ published dizziness questionnaires[1] in an attempt to qualitatively assess a patient's dizziness complaint. Although these instruments provide a systematic approach for obtaining case-history information, they are not useful in measuring the effects of therapeutic intervention. In response to the need for developing a method for quantifying the effects of treatment for Meniere's disease, the Committee on Hearing and Equilibrium of the American Academy of Ophthalmology and Otolaryngology[2] proposed a formula that determines the frequency of dizziness episodes before and after treatment. The formula consists of a ratio of the average number of dizziness spells a patient experiences per month 24 months after therapy, divided by the average number of spells per month 6 months prior to therapy. This quantity is multiplied by 100. Although this formula takes into account the frequency of dizziness attacks, it does not attempt to evaluate the impact of dizziness on the way a patient functions in everyday life.

We feel the method described above is shortsighted and limited in evaluating the treatment of vertigo. It is our contention that quantifying the handicapping consequences of dizziness may be a more useful means of validating treatment procedures. For example, patients who have experienced only a few attacks of violent vertigo may be sufficiently alarmed to the extent of restricting their activities (eg, quitting their occupations or avoiding social activities) so as not to be vulnerable should another attack of vertigo occur. It is reasonable to conclude that effective management of vestibular system disease should be reflected by an individual's ability to resume his or her premorbid vocational and avocational activities.

The present study was a natural evolution from work conducted by one of the investigators (C.W.N.) who recently employed the Hearing Handicap Inventory for the Elderly[3] before and after a hearing aid–fitting protocol to quantify benefits derived from amplification.[4] Similarly, we are suggesting that perceived dizziness handicap may be used as an index of treatment success over a selected time interval. That is, a well-designed scale could be employed to quantify changes in behavior that result from therapeutic intervention. The present scale was modeled after the Hearing Handicap Inventory for the Elderly and has been called the Dizziness Handicap Inventory (DHI).

The purposes of the present investigation were fourfold. First, we sought to develop a self-assessment inventory designed to evaluate the precipitating physical factors associated with dizziness as well as the functional and emotional consequences of vestibular system disease. Additionally, we undertook the present study to determine the internal consistency reliability of the DHI and to examine self-perceived dizziness handicap as a function of the frequency of dizziness or unsteadiness episodes. Finally, it was our intent to

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1990

Jacobson GP, Newman CW "The development of the Dizziness Handicap Inventory" Arch Otolaryngol HNS 1990; 116: 424-9

Gli ultimi progressi della tecnologia:

VNG e computer analysis

VEMPS

video HIT

La registrazione del nistagmo: oltre l' ENG

La fotoelettronistagmografia: Torok et al. Ann Otol Rhinol Laryngol 1951; 60: 917-26

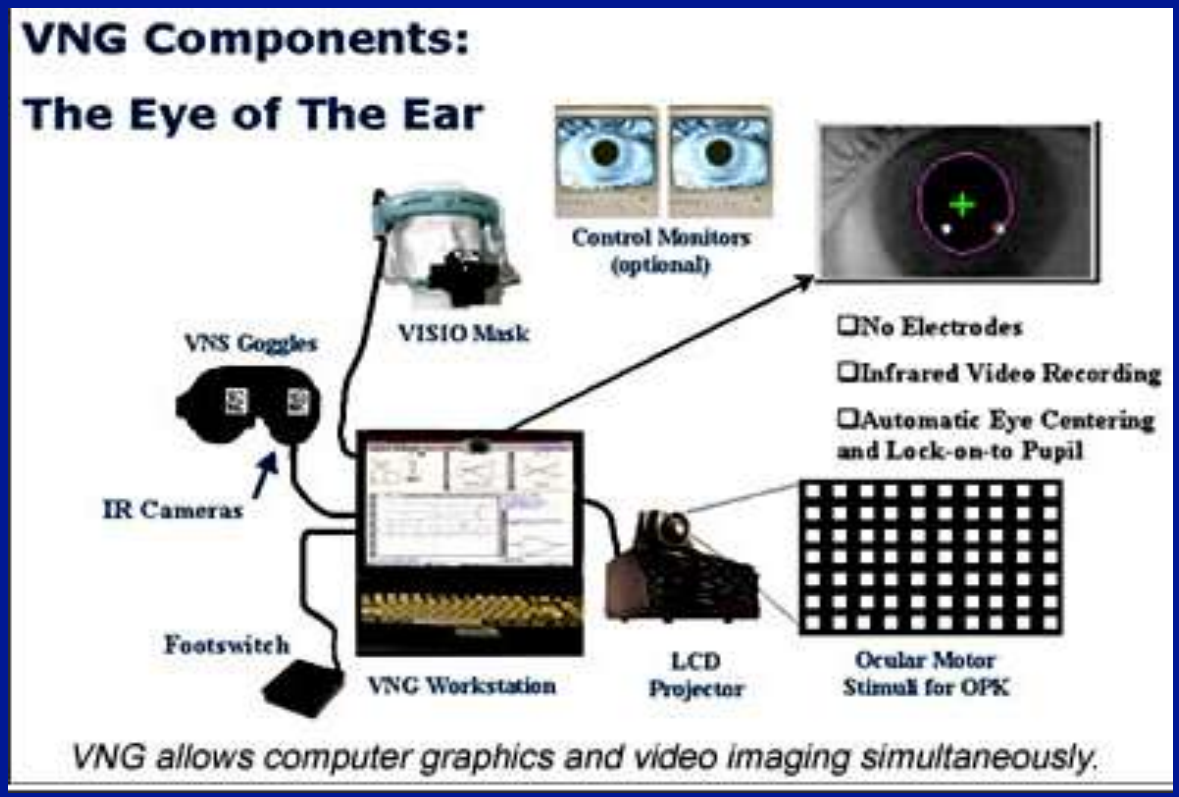
I *search coils*: Robinson DA. IEEE Trans Biomed Eng 1963; 10: 137-45

La videooculografia: Young LR et al. Ann NY Acad Sci 1981; 374: 80-92

Videonistagmografia

La nistagmografia nell’ era del computer

La digitalizzazione e l’ analisi computerizzata del tracciato nistagmografico



Myogenic potentials generated by a click-evoked vestibulocollic reflex

J G Colebatch, G M Halmagyi, N F Skuse

Abstract

Electromyograms (EMGs) were recorded from surface electrodes over the sternomastoid muscles and averaged in response to brief (0·1 ms) clicks played through headphones. In normal subjects, clicks 85 to 100 dB above our reference (45 dB SPL: close to perceptual threshold for normal subjects for such clicks) evoked reproducible changes in the averaged EMG beginning at a mean latency of 8·2 ms. The earliest potential change, a biphasic positive-negativity (p13-n23), occurred in all subjects and the response recorded from over the muscle on each side was predominantly generated by afferents originating from the ipsilateral ear. Later potentials (n34, p44), present in most but not all subjects, were generated bilaterally after unilateral ear stimulation. The amplitude of the averaged responses increased in direct proportion to the mean level of tonic muscle activation during the recording period. The p13-n23 response was abolished in patients who had undergone selective section of the vestibular nerve but was preserved in subjects with severe sensorineural hearing loss. It is proposed that the p13-n23 response is generated by activation of vestibular afferents, possibly those arising from the saccule, and transmitted via a rapidly conducting oligosynaptic pathway to anterior neck muscles. Conversely, the n34 and p44 potentials do not depend on the integrity of the vestibular nerve and probably originate from cochlear afferents.

(*J Neurol Neurosurg Psychiatry* 1994;57:190–197)

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The vestibular nuclei have powerful projections to the ocular motor nuclei, the cerebellum, the reticular formation, and the spinal cord.[1] In humans, the most accessible and best studied vestibular pathway is that between the semicircular canals and the ocular motor nuclei: the standard test of vestibular function, caloric induced nystagmus, measures the effect of horizontal canal activation on eye movements.[2] The reflex effects resulting from activation of the otoliths and the function of the direct vestibular projections to the spinal cord in humans are difficult to study and poorly understood.[3] The initial muscle excitation after an unexpected fall depends on otolith activation, possibly via connections to the reticular formation and thereby to the spinal cord.[4–6] Detailed studies on a single patient who had an otolithic Tullio phenomenon (sound-evoked activation of the vestibular apparatus) showed short-latency activation of leg muscles, probably via vestibulospinal pathways.[7] The limitations of our knowledge of vestibular influences on the muscles of the trunk and limbs led us to reinvestigate earlier reports of activation of the vestibular apparatus in normal subjects by loud clicks.

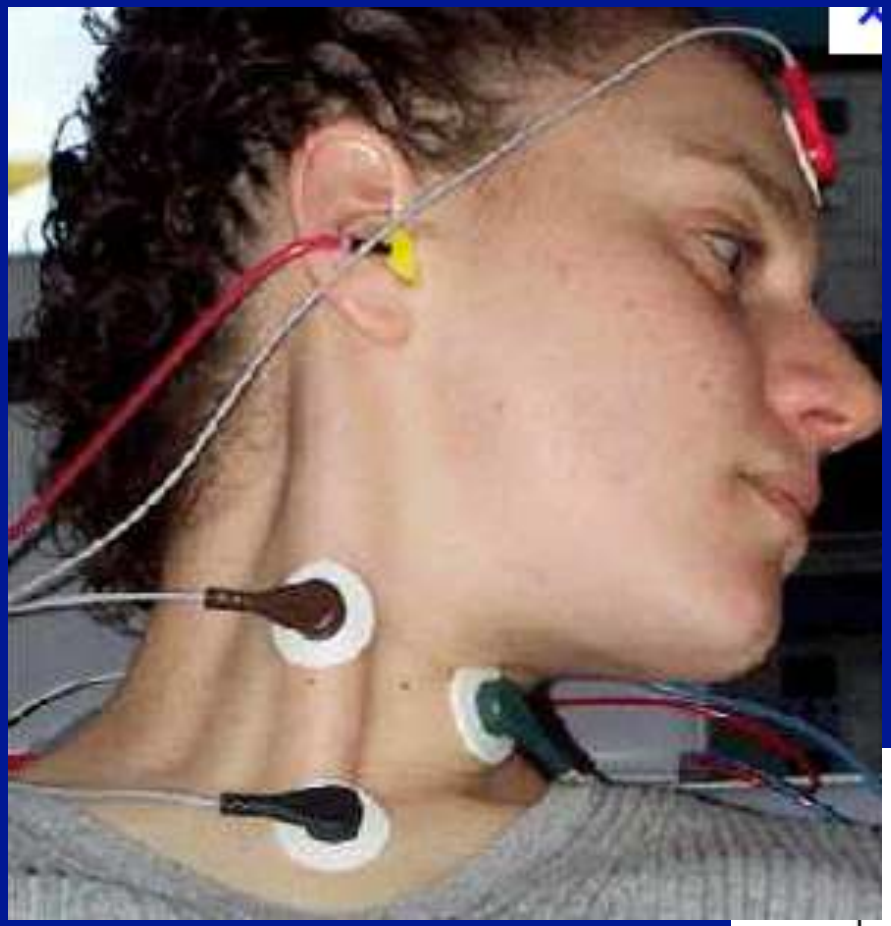
Bickford *et al*[8] described the characteristics of averaged responses to clicks with recordings with an active electrode just below the inion (the "inion response"). They concluded that the short latency potentials that they recorded were not, as they had first supposed, indicative of an auditory projection to the cerebellar vermis, but rather were generated by reflex changes in the level of EMG of posterior neck muscles, and thus were "myogenic" in origin. They also reported that the evoked response was present in patients with sensorineural deafness, leading them to propose that it arose from activation of the vestibular apparatus and not from activation of the cochlea. Subsequent publications by these workers described the inion response in patients with different acoustic and vestibular lesions and provided further evidence suggesting that it depended on activation of the otoliths, specifically, the saccule.[9,10] The role of the vestibular apparatus has been disputed by others. Meier-Ewert *et al*[11] argued that the inion response was a part of a generalised sonomotor response that originated from cochlear afferents. Additionally, averaged responses at the inion were not unique to click stimuli: Cody and Bickford[12] were able to record a similar-looking response to both light flashes and electric shocks, albeit at longer latency than the response to sound. These uncertainties have led to the view that the inion response is non-specific and it has not been accepted as a useful measure of vestibular reflex function.[13]

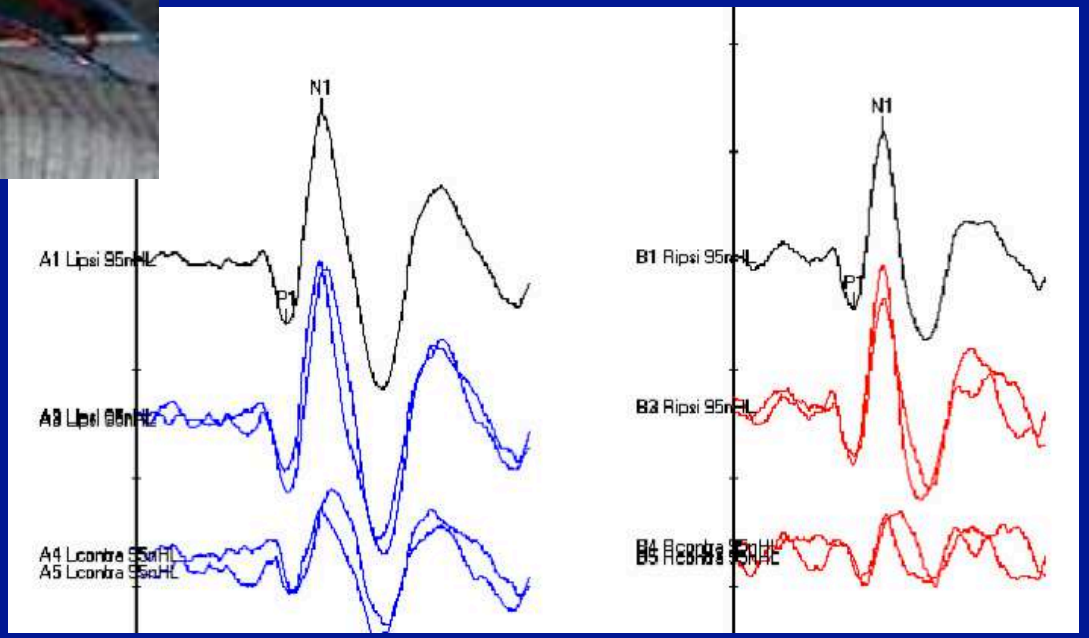
We have used different recording sites from those of Bickford and co-workers, to demonstrate the presence of short latency EMG changes in anterior neck muscles after clicks. Also, the response can be separated into two distinct components: only the earlier of these depends specifically on the integrity of vestibular afferents. The findings from one patient have been the subject of a brief report.[14]

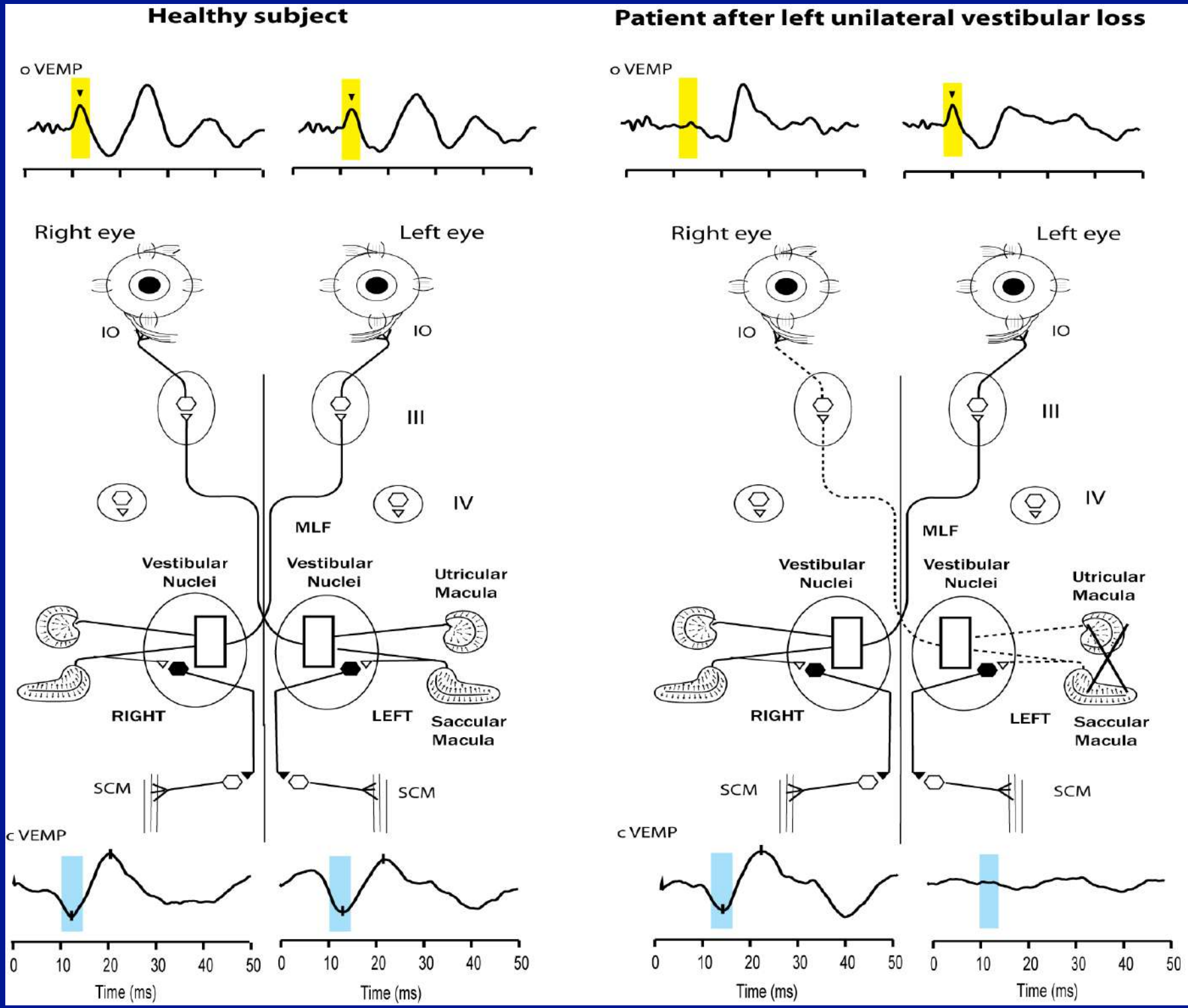
1994

Colebatch JG, Halmagyi GM, Skuse NF "Myogenic potentials generated by a click-evoked vestibulocollic reflex" J Neurol Neurosurg Psych 1994; 57: 190-7

cVEMP






Healthy subject
Patient after left unilateral vestibular loss
o VEMP
o VEMP
Right eye
Left eye
Right eye
Left eye
IO
IO
IO
IO
III
III
IV
IV
MLF
MLF
Vestibular Nuclei
Vestibular Nuclei
Vestibular Nuclei
Vestibular Nuclei
Utricular Macula
Utricular Macula
RIGHT
LEFT
RIGHT
LEFT
Saccular Macula
Saccular Macula
SCM
SCM
SCM
SCM
c VEMP
c VEMP
0 10 20 30 40 50
0 10 20 30 40 50
0 10 20 30 40 50
0 10 20 30 40 50
Time (ms)
Time (ms)
Time (ms)
Time (ms)

The video head impulse test

Diagnostic accuracy in peripheral vestibulopathy

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K.P. Weber, MD*
L.A. McGarvie, MBiomedE
G.M. Halmagyi, MD
I.S. Curthoys, PhD

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ABSTRACT

Background: The head impulse test (HIT) is a useful bedside test to identify peripheral vestibular deficits. However, such a deficit of the vestibulo-ocular reflex (VOR) may not be diagnosed because corrective saccades cannot always be detected by simple observation. The scleral search coil technique is the gold standard for HIT measurements, but it is not practical for routine testing or for acute patients, because they are required to wear an uncomfortable contact lens.

Objective: To develop an easy-to-use video HIT system (vHIT) as a clinical tool for identifying peripheral vestibular deficits. To validate the diagnostic accuracy of vHIT by simultaneous measures with video and search coil recordings across healthy subjects and patients with a wide range of previously identified peripheral vestibular deficits.

Methods: Horizontal HIT was recorded simultaneously with vHIT (250 Hz) and search coils (1,000 Hz) in 8 normal subjects, 6 patients with vestibular neuritis, 1 patient after unilateral intratympanic gentamicin, and 1 patient with bilateral gentamicin vestibulotoxicity.

Results: Simultaneous video and search coil recordings of eye movements were closely comparable (average concordance correlation coefficient $r_c = 0.930$). Mean VOR gains measured with search coils and video were not significantly different in normal ($p = 0.107$) and patients ($p = 0.073$). With these groups, the sensitivity and specificity of both the reference and index test were 1.0 (95% confidence interval 0.69–1.0). vHIT measures detected both overt and covert saccades as accurately as coils.

Conclusions: The video head impulse test is equivalent to search coils in identifying peripheral vestibular deficits but easier to use in clinics, even in patients with acute vestibular neuritis.
Neurology® 2009;73:1134–1141

GLOSSARY

BVL = bilateral vestibular loss; **HIT** = head impulse test; **IMU** = inertial measurement unit; **ITG** = intratympanic gentamicin; **vHIT** = video head impulse test; **VN** = vestibular neuritis; **VOR** = vestibulo-ocular reflex.

The head impulse test (HIT) is a useful bedside examination to identify a peripheral vestibular deficit for example in patients with vestibular neuritis (VN).[1-4] The clinician briskly rotates the patient's head to detect "overt" catch-up saccades after head rotation as a sign of semicircular canal paresis. "Covert" saccades are saccades that occur during the head rotation that may be imperceptible to the naked eye and hence confound the diagnosis.[5,6] In patients with acute VN, spontaneous nystagmus also interferes with assessment of bedside HIT.

Up to now, the scleral search coil technique has been the gold standard for HIT measurements.[7-9] It quantifies the VOR deficit and shows the associated pattern of overt and covert catch-up saccades in vestibular deficient patients.[6,10] However, search coil measurements require the subject to wear an uncomfortable contact lens, are time intensive, are expensive, and are not practical for acute patients.

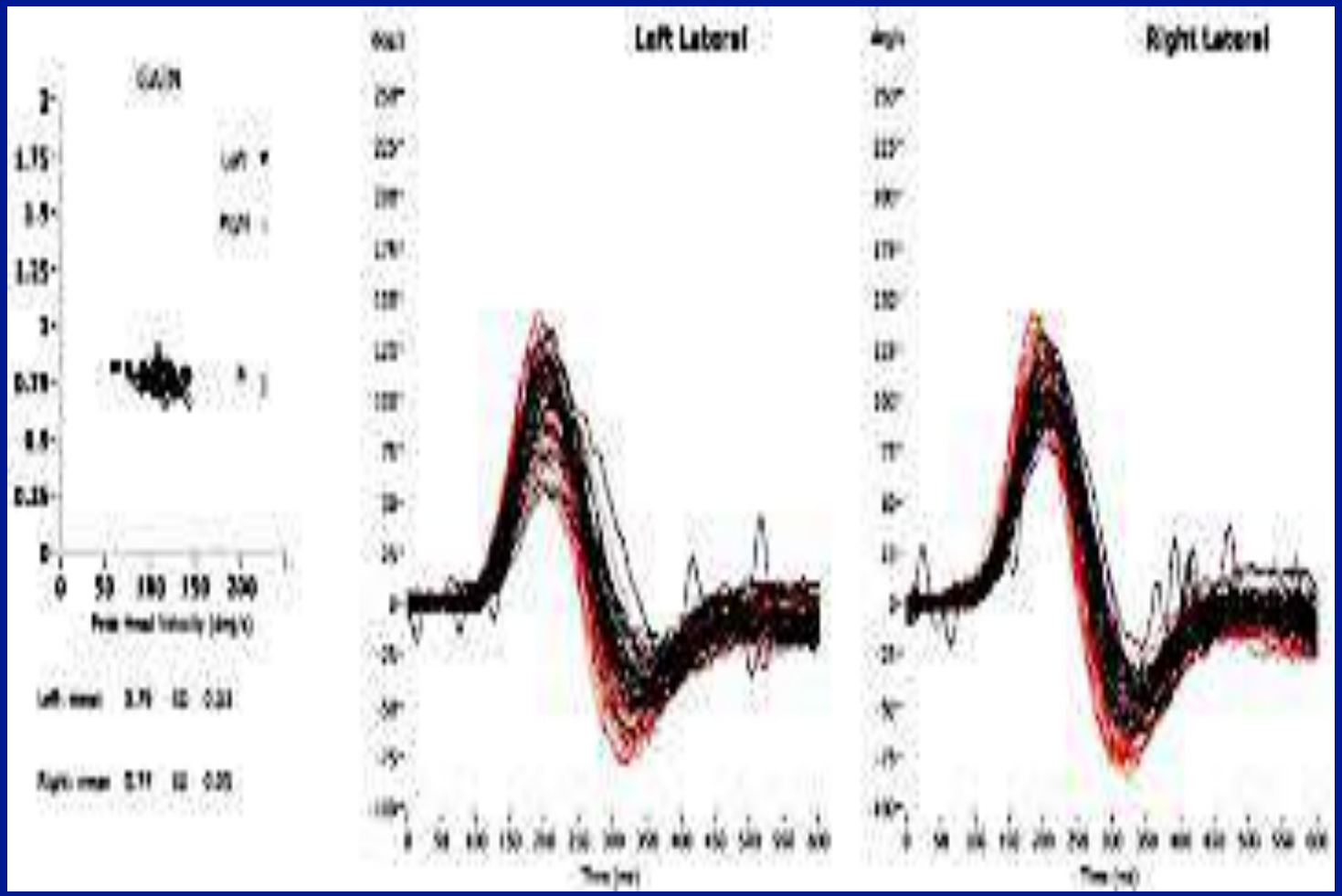
The goal of the study was to develop an easy-to-use high-speed video HIT system[11] (see video on the *Neurology*® Web site at www.neurology.org) as a clinical tool to identify a periph-

Supplemental data at www.neurology.org

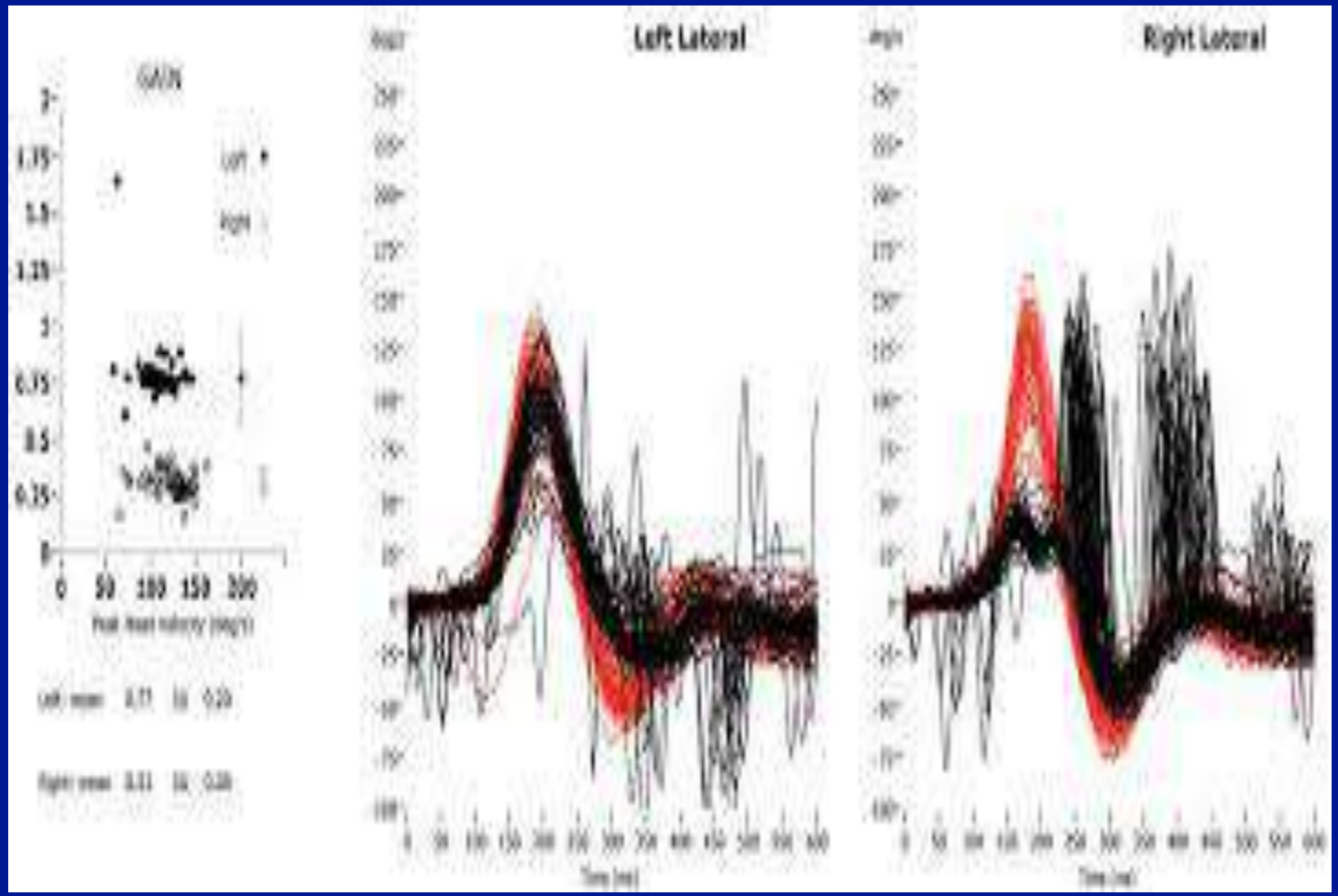
*These co-first authors contributed equally.
From the Vestibular Research Laboratory (H.G.M., I.S.C.), School of Psychology, University of Sydney; and Department of Neurology (K.P.W., L.A.M., G.M.H.), Royal Prince Alfred Hospital, Sydney, Australia.
Disclosure: Author disclosures are provided at the end of the article.

2009

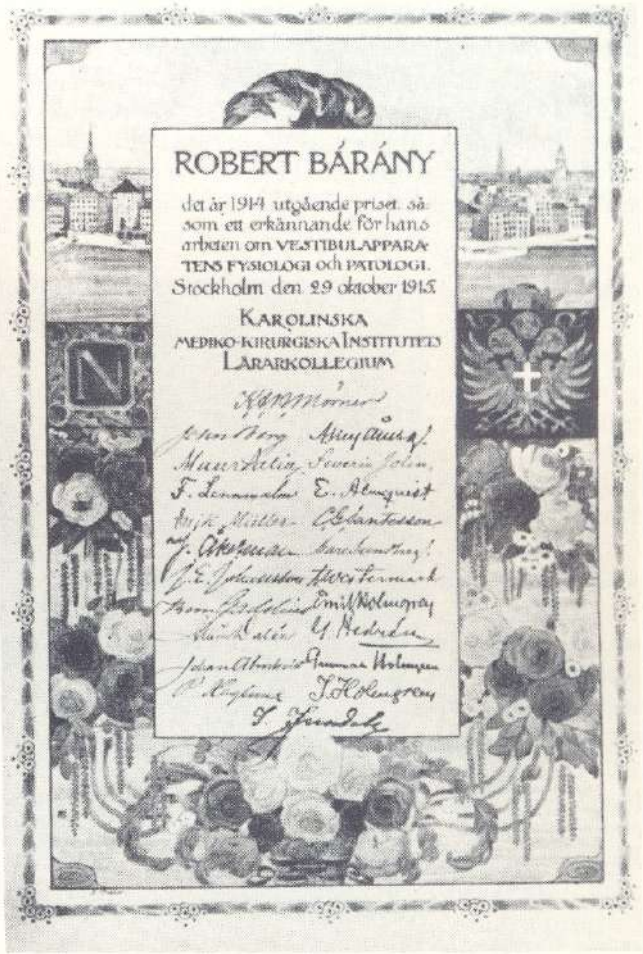
vHIT normale



vHIT patologico



1916



Nobel Prize awarded to Robert Barany in 1915 for his contribution to the understanding of the physiology of the vestibular system.

Unfortunately, his success was too spectacular

ROBERT BARANY

BÀRÀNY SOCIETY MEETINGS

1960 Padova
1963 Uppsala
1968 Uppsala
1970 Amsterdam
1971 Toronto
1972 Strasburgo
1974 Los Angeles
1975 Kyoto
1977 Londra
1978 Uppsala
1980 New York
1982 Basel
1983 Uppsala
1985 Ann Arbor
1987 Bologna
1988 Uppsala
1990 Tokyo
1992 Praga
1994 Uppsala
1996 Sydney
1998 Wurzburg
2000 Uppsala
2002 Seattle
2004 Parigi
2006 Uppsala
2008 Kyoto
2010 Reykiavik
2012 Uppsala
2014 Buenos Aires
2016 Seoul
2018 Uppsala

Italia

I nostri maestri e la nostra storia

Pietro Tullio (1881 - 1941)

Laureato in Medicina all' Universita' di Bologna (1905) e quindi libero docente e assistente di Fisiologia all' Università di Bologna (1917). Direttore degli Istituti di Fisiologia di Sassari, Cagliari, Messina, Bari, Parma, Genova. Si occupa di fisiologia dell' orecchio e pubblica "Studio sopra il comportamento dei riflessi sonori labirintici nel cane" in Arch Ital Otol 1938;50:274-9.

Pioniere dell' uso del microscopio binoculare nella chirurgia dell' orecchio.

VOL. L **Giugno 1938** FASC. VI

ARCHIVIO ITALIANO
DI
OTOLOGIA, RINOLOGIA E LARINGOLOGIA

LABORATORIO DI FISIOLOGIA DELLA R. UNIVERSITÀ DI PARMA
diretto dal Prof. P. TULLIO

XVIII

Studio sopra il comportamento dei riflessi sonori labirintici nel cane

P. TULLIO e G. ZANZUCCHI

Ad un illustre medico e fisiologo lombardo, *Filippo Lussana* (1), spetta il merito di aver collegato i disturbi vertiginosi, che si possono osservare in alcuni ammalati, con le lesioni dei canali semicircolari. La sua *monografia sulle vertigini*, edita in Milano, risale infatti al 1858, mentre in epoca successiva comparvero le osservazioni del *Ménière*, pubblicate nella *Gazette Médicale* nel 1861 (2). Inoltre il *Lussana* ritenne che i disturbi motori, che si osservano nei suddetti malati, fossero dovuti a vertigine auditiva, venendo nella concezione che i canali semicircolari possano essere stimolati dal suono e con ciò darne percezione della direzione.

Tullio (3), praticando un foro in vicinanza dell'ampolla dei singoli canali del piccione ed avvicinando il fischio di Edelmann, ottenne un movimento del capo nel piano del canale aperto, movimento che si può graficare (4) e si ripete sempre eguale per uno svariatissimo numero di volte, di intensità variabile in rapporto alla forza del suono e che scompare cocainizzando l'orecchio. Il movimento della testa è accompagnato sempre, per ogni singolo canale, da differenti moti delle ali, della coda e delle zampe (5).

I medesimi risultati si sono riscontrati anche nel coniglio (6), in

Fenomeno di Tullio
1938

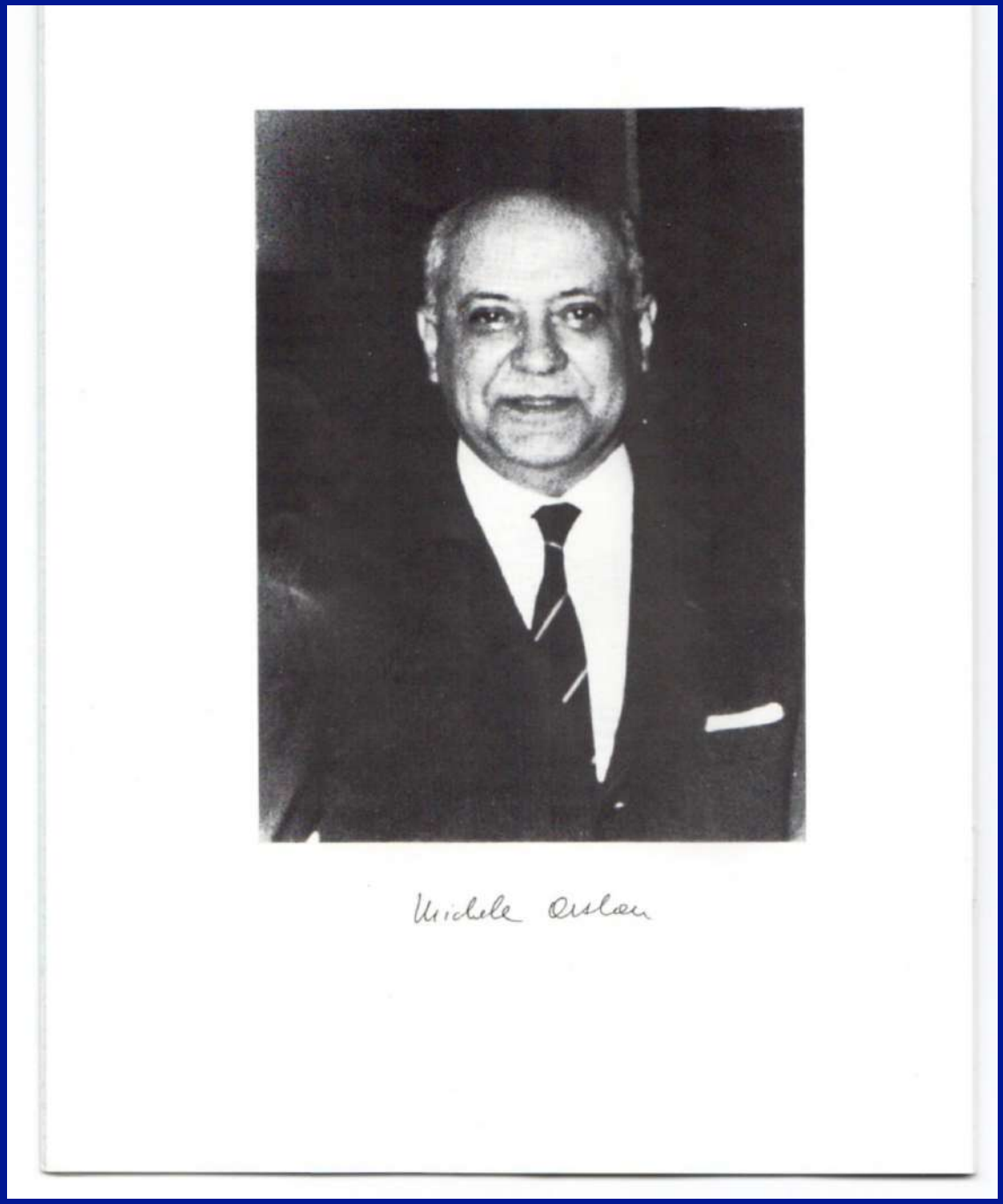
STORIA DELLA VESTIBOLOGIA – ITALIA 1900-2000

Michele Arslan 1904-1989

figlio di Yerwant Arslanian

padre di Edoardo Arslan e Antonia Arslan

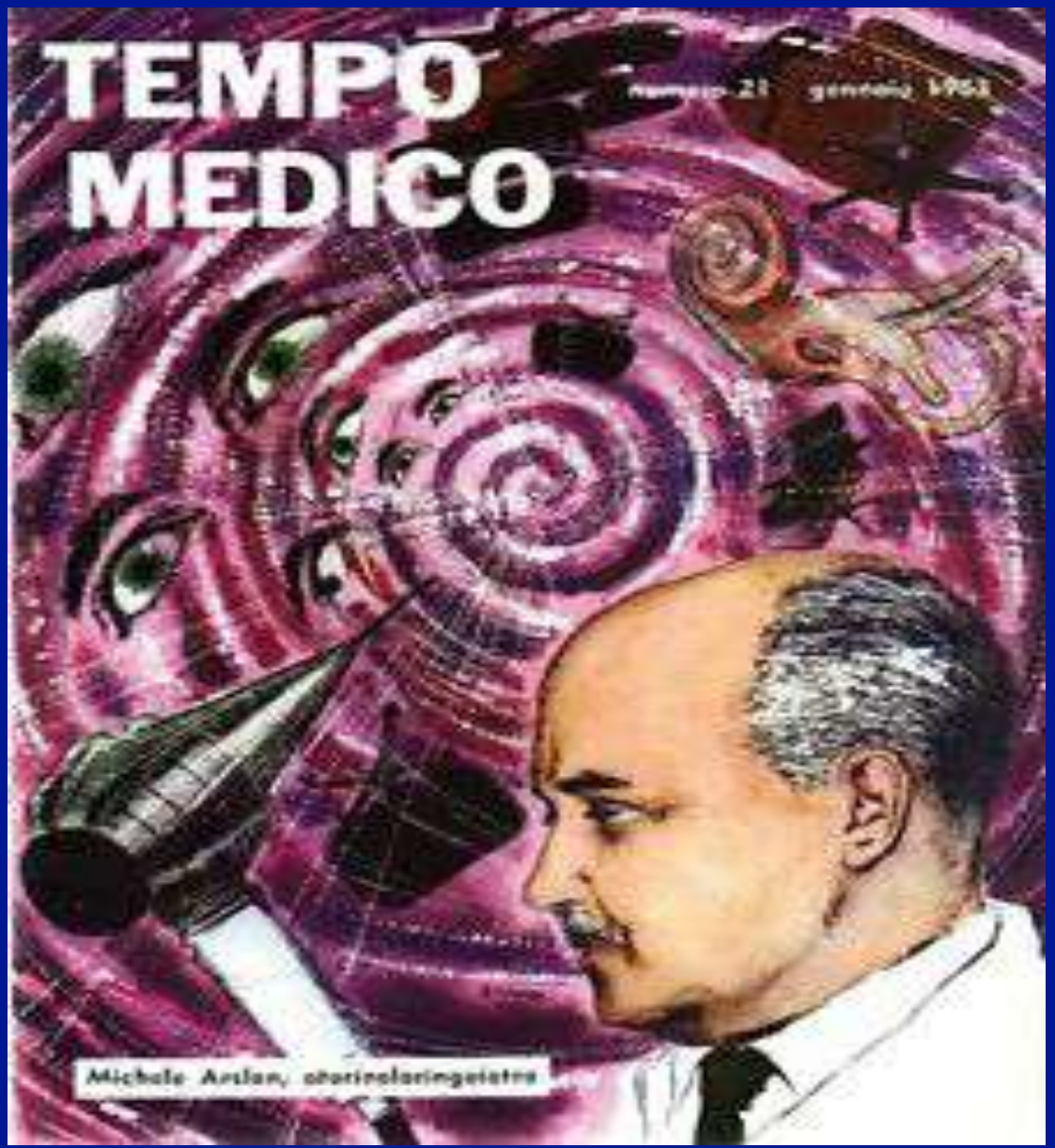
allievo: David Megighian (ENG)

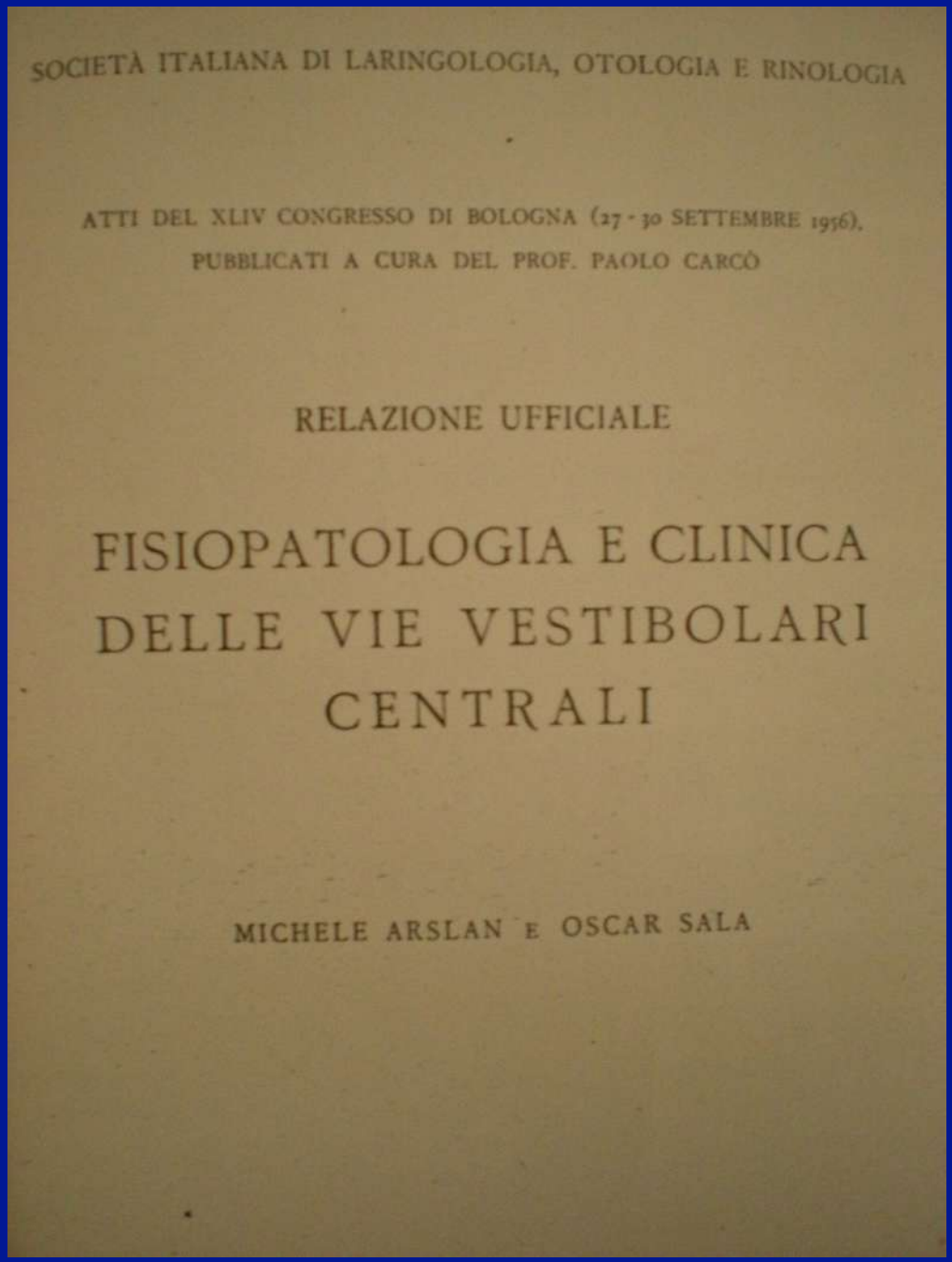


Michele Arslan

Michele Arslan
1904 - 1988

Michele Arslan




SOCIETÀ ITALIANA DI LARINGOLOGIA, OTOLOGIA E RINOLOGIA
ATTI DEL XLIV CONGRESSO DI BOLOGNA (27 - 30 SETTEMBRE 1956).
PUBBLICATI A CURA DEL PROF. PAOLO CARCÒ
RELAZIONE UFFICIALE
FISIOPATOLOGIA E CLINICA DELLE VIE VESTIBOLARI CENTRALI
MICHELE ARSLAN E OSCAR SALA

1956

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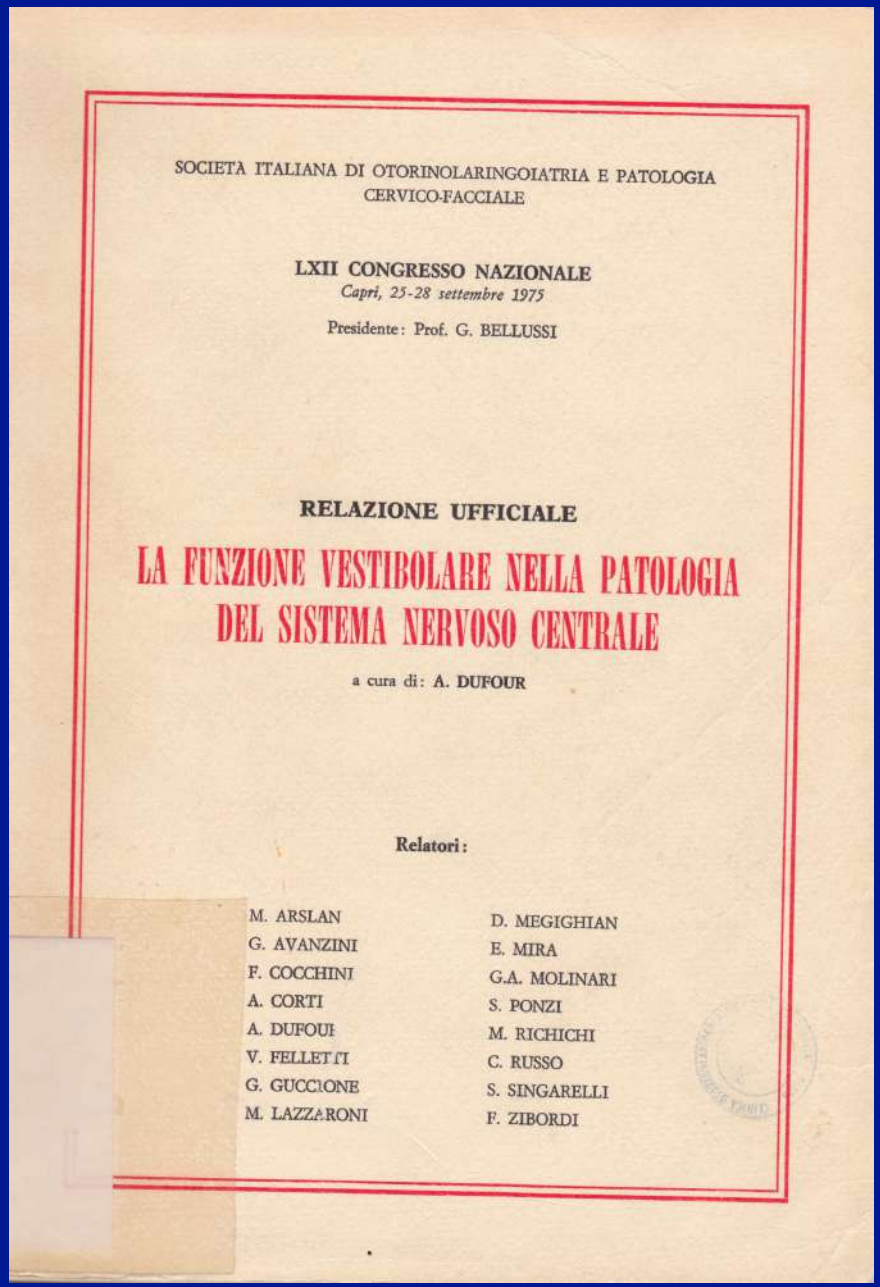
Aldo Dufour 1931-2000 (Milano, FBF e Besta)

allievi: Cocchini, Richichi

Oreste Pignataro (Milano)

Eugenio Mira (Pavia)

Aldo Dufour
1931 - 2000



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Capri, 25-28 settembre 1975

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1975

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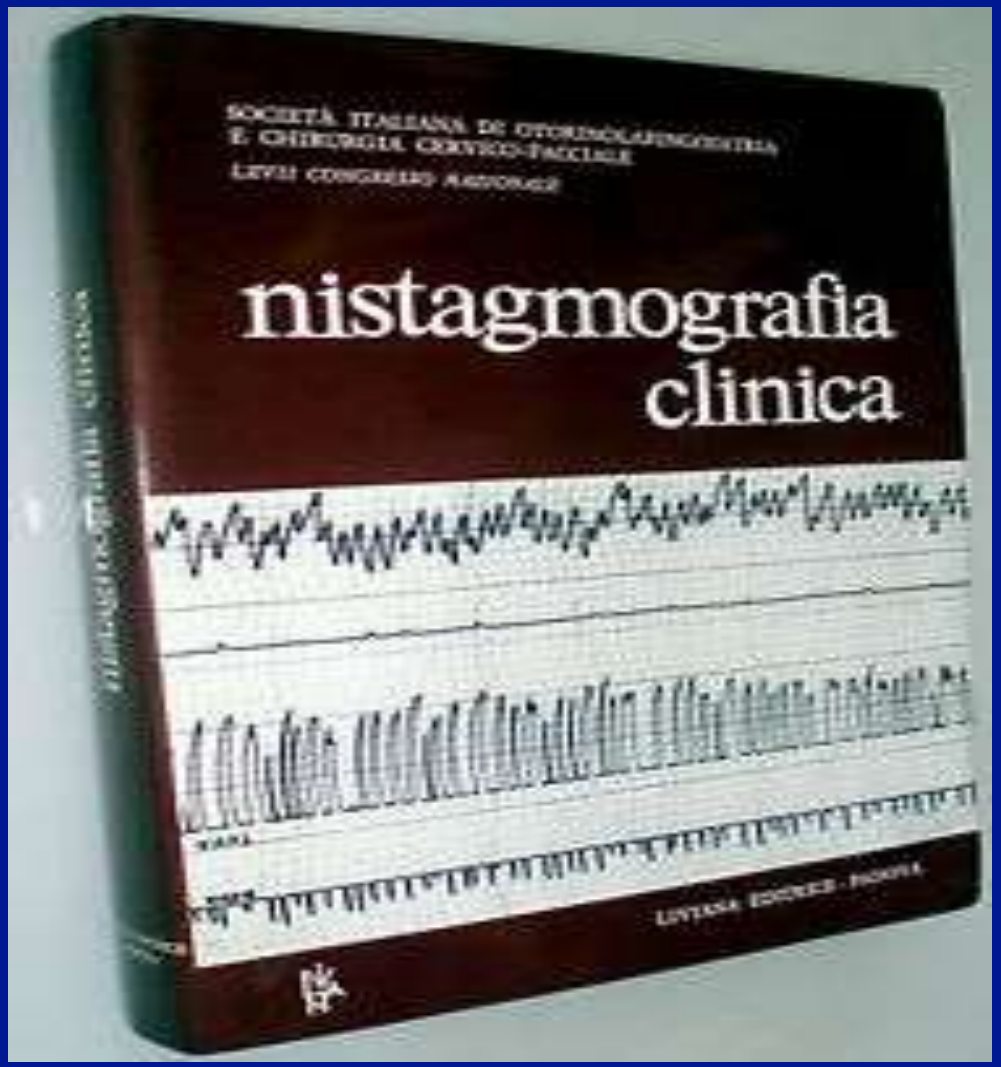
LIVIANA EDITRICE
PADOVA

1980



1980

LA VESTIBOLOGIA IN ITALIA, OGGI

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Paolo Pagnini Daniele Nuti

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Versino

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Ralli, Teggi, ….

VIS

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VESTIBOLOGIA
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A CURA DEL CRS AMPLIFON
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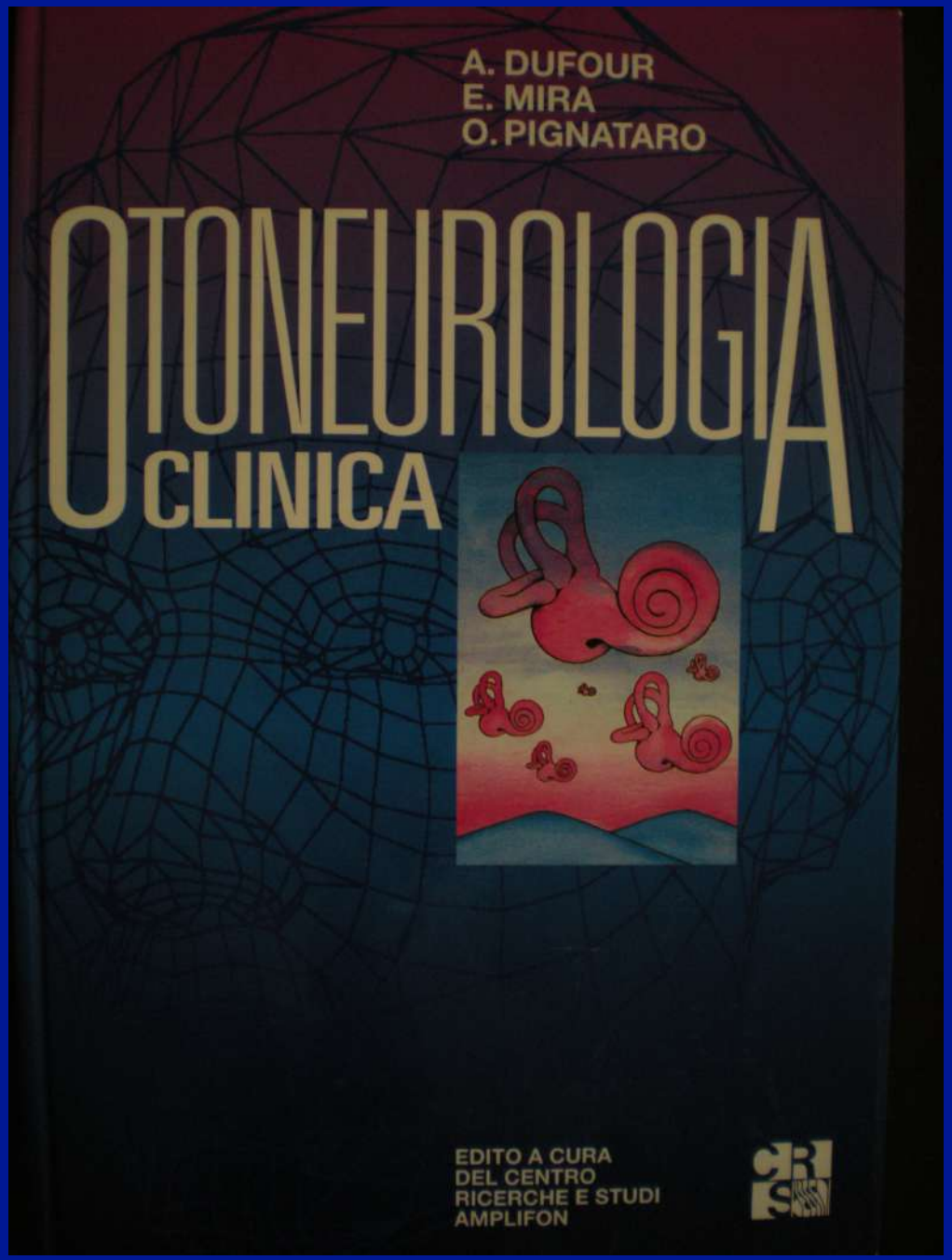
1980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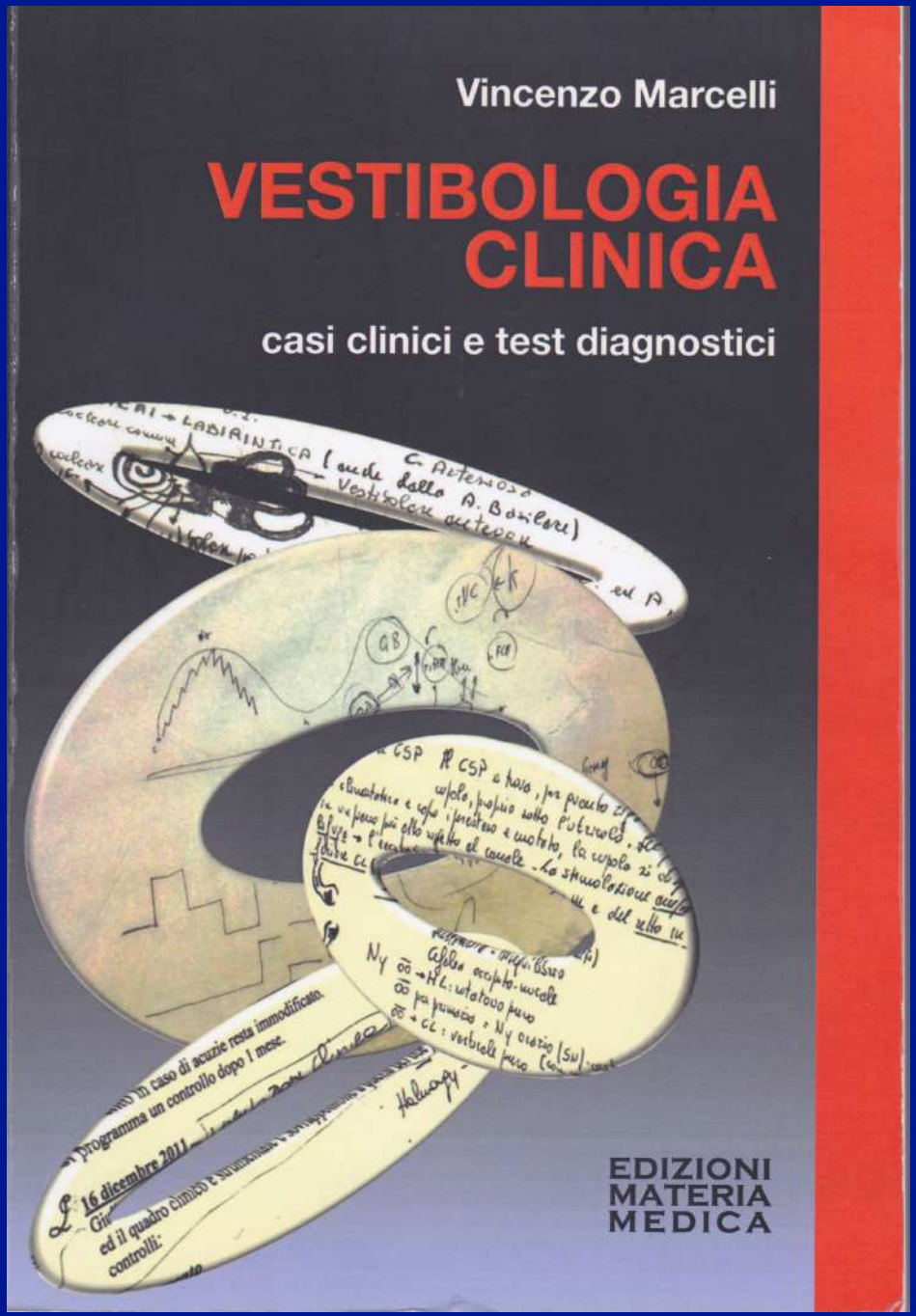
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amplifon

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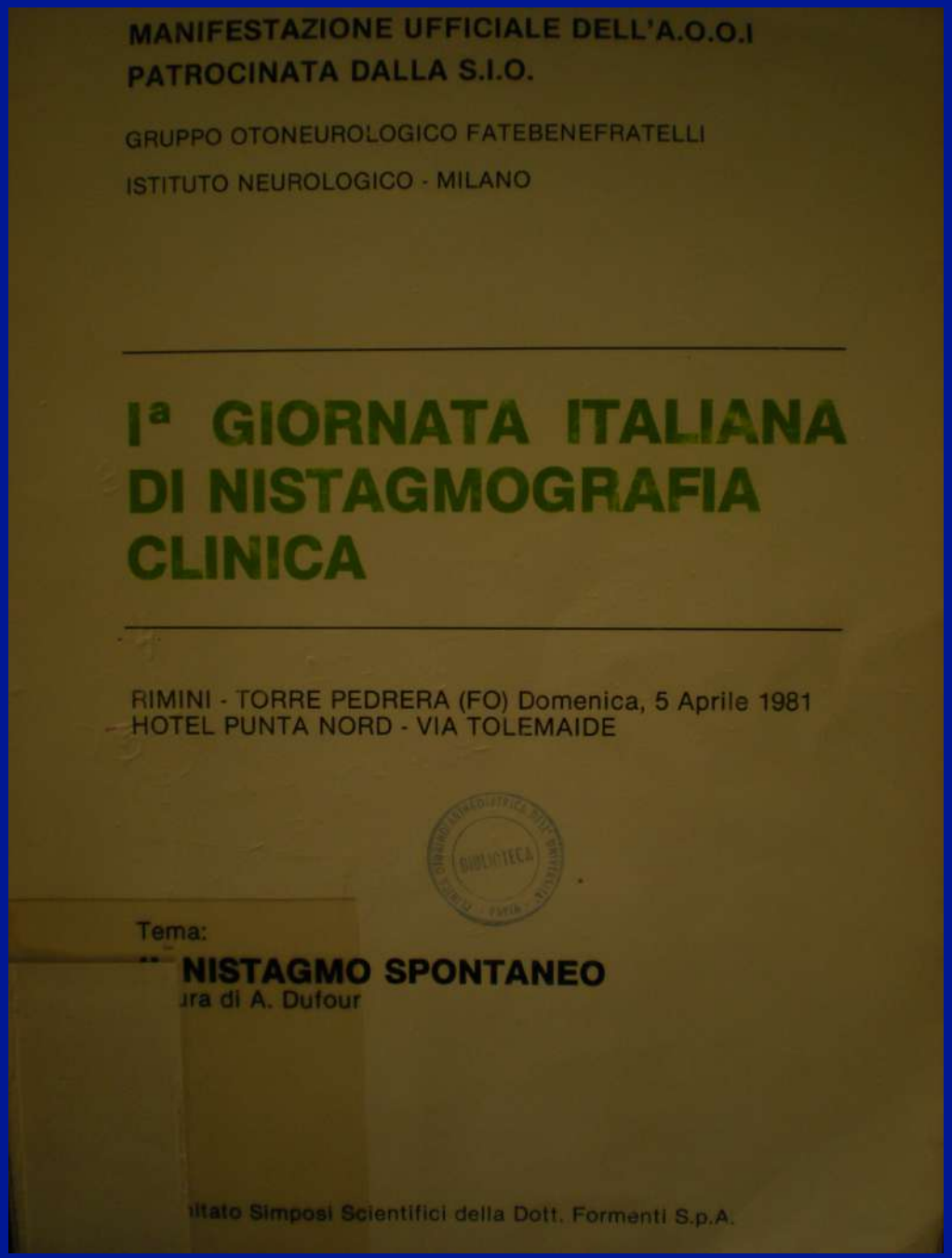

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PATROCINATA DALLA S.I.O.

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ISTITUTO NEUROLOGICO - MILANO

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Tema:

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iltato Simposi Scientifici della Dott. Formenti S.p.A.

1981

COME RAGGIUNGERCI

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Dall'autostrada Adriatica A14 (da nord: in direzione Lecce; da sud: in direzione Ancona), uscire a Pescara Ovest/Chieti e immettersi sull'Asse Attrezzato in direzione di Chieti.

Da Roma
Prendere l'autostrada A 25 Roma - Pescara, uscire a Chieti/Pescara e immettersi sull'Asse Attrezzato in direzione di Chieti.

Treno

Stazione ferroviaria di Pescara Centrale (è la stazione principale sita in Via Ferrari): Linea diretta Milano - Lecce. La stazione di Pescara Centrale è una delle più importanti della dorsale adriatica.
Per consultare gli orari dei treni visitare il sito web delle Ferrovie dello Stato www.ferroviedellostato.it/.

Bus

Sono numerosissimi gli autobus che ogni giorno partono e arrivano a Pescara.
Le aziende che si occupano dei principali collegamenti sono:

- **Autolinee Arpa** (Autolinee Regionali Pubbliche Abruzzesi):
  Principali collegamenti con: Roma, Napoli, Salerno, Chieti, L'Aquila, Teramo, tutta la provincia di Pescara.
  Per informazioni su tutte le corse per raggiungere Pescara consultare il sito web www.arpaonline.it/ ; Biglietteria Pescara Tel. +39 085 4215099
- **Autolinee Satam** (Gruppo La Panoramica):
  Principali collegamenti con: Napoli, Salerno, Bologna, Genova, Sanremo, Sulmona, Chieti. Sito web www.gruppolapanoramica.it/
- **Autolinee Baltour** Per maggiori informazioni visitare il sito web www.baltour.it/
- Le **autolinee Di Fonzo** coprono moltissime località Abruzzesi, creando una fitta rete tra loro e Roma: www.difonzobus.com/

Aereo

- Aeroporto di Pescara
  Si effettuano sia voli nazionali che internazionali: Roma, Milano, Torino, Londra, Francoforte, Bruxelles, Bergamo, Catania, Parigi,
- Aeroporto Ancona (156 km circa)

Gli hotel si trovano tra Pescara e Chieti. Le possibilità di pernottamento sono diverse e si suggerisce di valutarle in relazione al mezzo di trasporto prescelto per raggiungere la sede congressuale.

Gli hotel di Pescara (Hotel Plaza, Hotel Ambra, Hotel Alba, Hotel Duca d'Aosta) sono facilmente raggiungibili dalla Stazione Ferroviaria e dalla Stazione dei pullman (5minuti a piedi). Si trovano nel centro di Pescara a pochi minuti dal mare, dai ristoranti e dalla zona shopping.

Ulteriori sistemazioni alberghiere si trovano a Chieti Scalo presso Hotel Parco Paglia (a circa 900 metri dalla Sede Congressuale).L'Hotel è situato nella zona industriale per cui bisogna spostarsi in taxi o in auto propria per raggiungere i ristoranti o la zona shopping di Chieti e Pescara.

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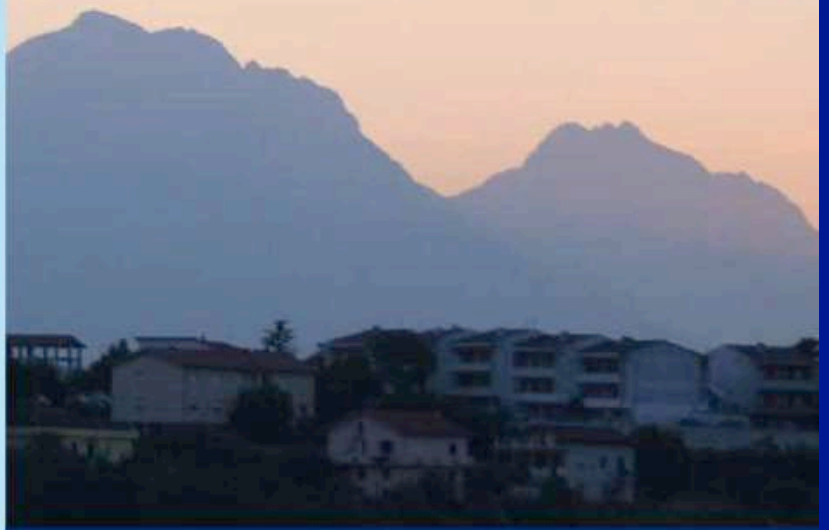

CHIETI 23 - 24 e 25 GIUGNO 2016

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Responsabile Scientifico: **Prof. Giampiero NERI**
Dipartimento di Neuroscienze e Imaging
Università degli Studi "G. d'Annunzio" - Chieti-Pescara

2016


"la storia"
siamo NOI
attenzione
nessuno
si senta
escluso
"La storia"
(F. De Gregori)