Arch. Oto-Rhino-Laryng. 212, 301–307 (1976)

Archives of
Oto-Rhino-Laryngology

On the Mechanism of the Menière Attack

G. F. Dohlmann

49 Newton Street, Weston, Mass. 02193, USA

Summary. Three theories are discussed dealing with the origin of the Menière attack: 1) The hypothesis of pressure induced changes is disproved. 2. The stimulation of the crista epithelium by a mechanical cupula deviation has been investigated. The results showed that the changes in action potential frequency of the ampullary nerve induced by maintained bending of the cupula, always declined and returned to resting potential frequency within a very short period of time usually within 1 min. Preventing the "elastic return" of the cupula did not change the time constant of this responce decline. This proves that a Menière nystagmus lasting for hours, can not be elicited by a mechanical deformation of the cupula. 3) The paralyzing influence on vestibular nerve fibres by K^+ ions in endolymph, leaking into perilymph spaces has earlier been demonstrated by the author. When the distended membranous labyrinth ruptures at predestined weak spots, leaking endolymph may reach and paralyze different nerve branches. This can explain all the main symptoms of a Menière attack.

Key words: Menière's disease – Labyrinthine pressure – Cupular physiology – Perineural potassium.

Zusammenfassung. Da weder die Überdehnung des Labyrinths allein, noch die Druckerhöhung im häutigen Labyrinth oder eine Anoxie der Sinnesendstellen bei gestörter Durchblutung den Menièreschen Anfall erklären können, wird ein Mechanismus dargestellt, welcher auch experimentell belegbar ist. Durch die Ausweitung des Endolymphschlauches kommt es an bestimmten Stellen zur Ruptur der Wand. Die kaliumreiche Endolymphe dringt in den Perilymphraum ein, und zwar am Helicotrema und in der Ampullenumgebung. Hier lähmt sie die durchziehenden Nerven und verursacht so das Erscheinungsbild des Anfalls in allen seinen Abarten in Abhängigkeit vom Orte der Ruptur.

Three theories dealing with the explanation of the Menière attacks, the first and oldest hypothesis originates from the discovery by Hallpike und Cairns. They as-

sumed that the distented endolyphatic system presses the capillary blood vessels against the bony walls causing obstruction of the blood supply of the sensory areas. This presumes that the increase in endolymph pressure would be equivalent to the capillary blood pressure. Studies by Perlman and Lindsay showed that short obstructions could produce nystagmus with a recovery of hair cell function, but that obstructions producing nystagmus for hours or days, as in a Menière attack always would leave a dead labyrinth. After a Menière attack the labyrinthine and cochlear functions as a rule return to the level before the attack. Thus, vascular obstruction can be excluded as the cause.

However, an increase in endolymph pressure is undoubtedly the basis for the distention of the membranous inner ear. This fact has given rise to the assumption that some other mechanism than anoxia is active in pressure induced vestibular and cochlear irritation. Tonndorf has assumed a mechanical suppression of the movements of the basilar membrane in the cochlea producing a low tone hearing loss, but that cannot explain the vestibular symptoms. Henriksson and Gleissner have experimentally increased the endolymphatic pressure in frogs. They interpreted their results as an irritation of the hair cells by pressure. However, later Gleissner found that it is impossible to change pressure mechanically without producing endolymph movements, accordingly producing response from bending of the cupula. No super-

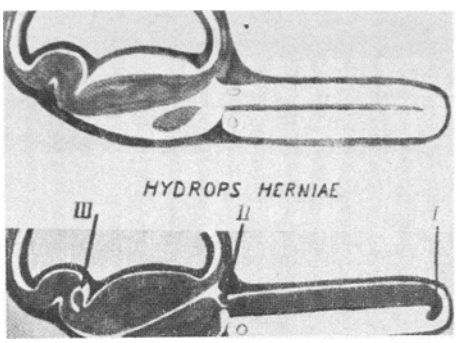

Fig. 1a. Schematic drawing of the distention of the Endolymphatic space in Menière's disease. The membranous walls are distended until they reach the support of the surrounding bony walls. Further increase in endolymph volume can occur only in three areas where the expanding membranes are lacking bony support: I at the helicotiema, II at the coecum, and II at the entrance into one of the entrances of a semicircular canal

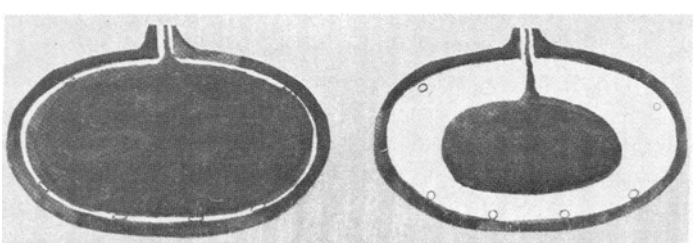

Fig. 1b. Schematic drawing of suggested mechanism of action on the capillaries from expanding membranous inner ear (After Hallpike and Cairns)

imposed responses to an increase in pressure could be recorded. Recent experiments have convinced us that even a considerable pressure when applied to the whole labyrinth was unable to change the resting frequency of the nerve. Thus, there is no evidence forthcoming that pressure per se can influence the function of the hair cells. Further, Beentjes has been able to show that the mammalial labyrinth can not withstand a higher pressure than 2 cm of water without rupturing (Fig. 1a, b).

The second theory is also based on an increased endolymphatic pressure causing the distention of the membranous inner ear. This distention is then assumed to produce a bending of the cupula through a distortion of the ampulla with ensueing nystagmus. An other possibility mentioned is that ruptures in the membranous walls might result in a sudden deflection of the cupula due to a pressure release in the endolymph with the onset of a resulting nystagmus, which was presumed to last as long as the deformation remained.

The crucial question that arises is then: Can a mechanical deformation produce functional changes resulting in the well known clinical symptoms: nystagmus and vertigo as well as a temporary hearing loss, lasting for hours, days or longer as in Menière's disease? It therefore seemed necessary to record peripherally from the first order neurons from the crista the results of a application of controlled mechanical bending of the cupula, maintained in a deviated position. For these experiments the action potentials in the nerve from the horizontal canal in frogs were used. When the canal wall was indentated, a sharp increase in the resting potential frequency followed. This change in frequency returned to normal resting level usually within 20–40 s. This decline in frequency has always earlier been explained as the result of an elastic return of the cupula to its upright normal position, pushing the endolymph back to its earlier position. Relieving the pressure on the canal is then assumed to move the endolymph and the cupula in the opposite direction producing a decrease

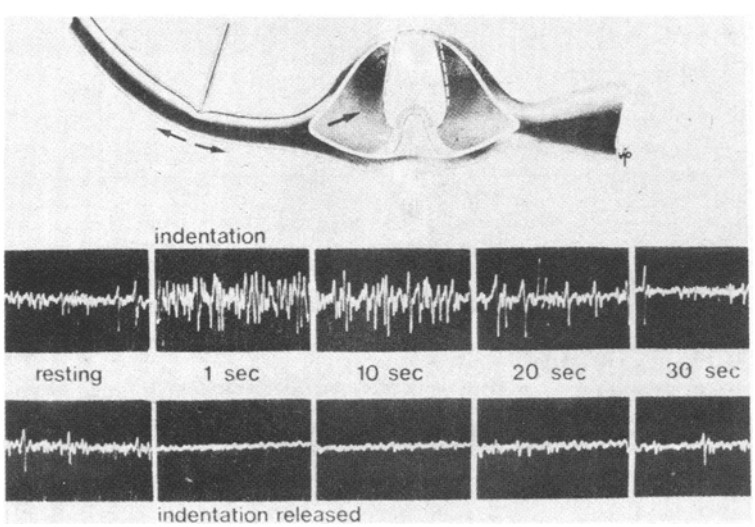

Fig. 2. Drawing of a canal with ampulla. The canal wall is indentated. Upper recording shows the action potential frequency during the first 30 s. Lower recording shows the decrease in frequency and returning to normal resting potential frequency after removing the pressure against the canal

of potential frequency, again returning to resting level within approximately the same time of about 20 s (Fig. 2).

In order to maintain the position of an imposed bending of the cupula, two procedures were used. In the first experiment the canal was clamped, to prevent any spinglike recoil of the cupula and endolymph. The second series of manipulations were based on a mechanical deviation produced by blocking the cupula in a bent position by deep indentations of the ampullar walls on either side of the cupula (Fig. 3, 4).

In both experiments the manipulations prevented all responses to rotational stimuli.

The experimental results were invariably the same:

1. The sudden increase in action potentials in indentation of the canal was the same if the canal was patent or if it was clamped or even if the cupula was blocked in any position.

2. The frequency decline back to resting level was invariably the same, within about 20—40 s. In all experiments: if the canal was patent and responding to rotational acceleration, if the canal had been clamped, or the cupula had been locked in

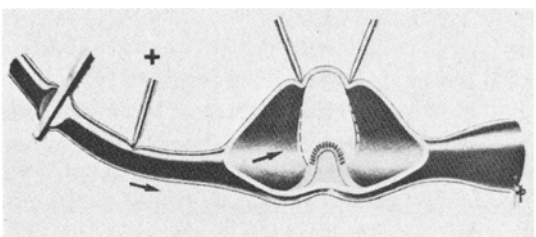

Fig. 3. Clamping of the canal. Indentation of canal wall produced the same increase in action potential frequency and the same frequency decline as in the patent canal

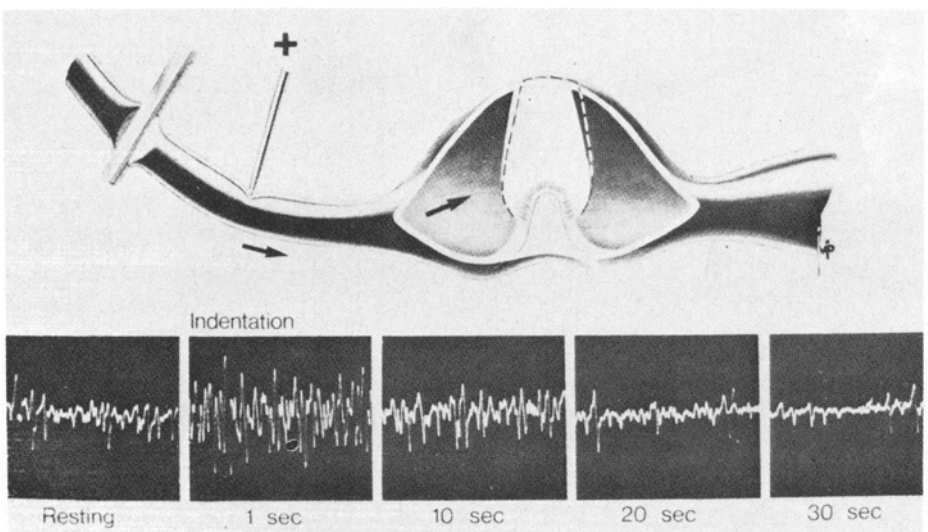

Fig. 4. The cupula is fixed in position by deep indentations of the ampullary wall

position, the action potential frequency had returned to normal resting level within less than a minute.

In conclusion the experiments showed that it is impossible to produce a peripheral change in action potential frequency from the vestibular sense organs by a maintained mechanical deformation of the ampulla or bending of the cupula. Accordingly a maintained bending of the cupula is incapable of producing a nystagmus which could last longer than the response decline of about 1 min. This excludes the possibility of producing a nystagmus by mechanical cupula displacement which lasts for hours or days as in a Menière attack.

Based on the histology of the de Buhlet, Hallpike, Lindsay, Schuknecht and others, the distention will first hit the weakest parts of the membranous wall. Reissners membrane ist the thinnest and will therefore be the first to bulge. When scala media has been distended to the extent that scala vestibuli has been obliterated, the membrane will rest against the bony walls and will resist any further distention. That is, except in two places. At the helicotrema at the top and at the coecum at the bottom of the cochlear duct. At these points the distended membrane lacks bony support and can therefore be further distended, like a hernia, to that point at which the wall might rupture.

In the vestibule with its openings into the semicircular canals the conditions are similar. The saccule and utricle are distended until their membranous walls have reached the surrounding bony walls. At this stage the thin utricular walls facing the openings to the ampullae are lacking support, they will therefore bulge as a herniation in the way it has been described by Lindsay and Schuknecht. At these points the distended membranes may rupture.

As the cause for an attack by pressure, experimental support is lacking and the hypothesis in itself is extremely unlikely from what is known.

The third hypothesis is based on the increase in endolymph volume causing the distention and the ruptures in certain predetermined places: at the helicotrema and the herniations towards the ampullae of the semicircular canals. The effect of a leakage of endolymph through the ruptures into the perilymphatic space around the membranous ampulla into the space where the nerves pass to the crista is the decisive factor in the explanation of the symptoms in Menière's disease.

It is well known that potassium ions can paralyse the propagation of action potentials in a nerve fibre, even in a concentration $1/5$ of the concentration of potassium chloride found in the endolymph. It was shown in experiments on frogs that endolymph from one labyrinth, applied to the vestibular nerves of the other side, immediately inhibited all activity. It has also been shown that endolymph-like potassium chloride solutions can produce a nystagmus in monkeys, pigeons and cats if dripped into the perilymph of the vestibule (Fig. 5).

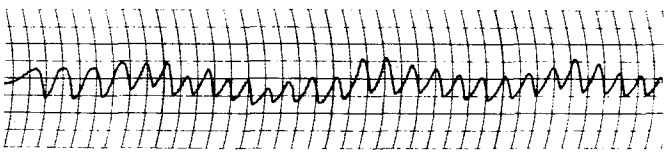

Fig. 5. Nystagmogramm from a squirrel monkey during nystagmus attack produced by isotonic KCE in the perilymph

Thus, correlating the site of a rupture with the paralysing effect of the endolymph potassium, the symptoms expected from each single location of the rupture is predictable. The thinnest membrane in this system is Reissner's membrane. Accordingly the first rupture and the first symptoms would be expected from elements in the vicinity of the helicotrema, representing the low tone region. Overwhelming experience from early cases of Menière whow a fluctuating hearing loss mostly presenting a falling or a flat audiogram. The second potential localization of a rupture is where a herniation develops towards the posterior vertical ampulla. The effect of a paralysis of that nerve branch would evidently be a rotatory nystagmus towards the diseased side. Then next, the nerve branches to the horizontal and anterior vertical ampullae, which are morphologically close might be affected, each separately or in combination. The anterior vertical canal would produce a rotatory nystagmus towards the same side. The sensory elements of the horizontal crista give the well known effect of a horizontal nystagmus beating to the opposite side a demonstrated in every cold caloric reaction.

Reviewing from the literature the descriptions of the different types of nystagmus characterizing the Menière attack, it is evident that the nystagmus varies. Thus, it turned out that there are principally two types of nystagmus: one rotatory to the same side and one horizontal to the opposite side. Further it has been shown that the nystagmus during an attack might change, starting as a pure horizontal to the opposite side, changing to a more or less pure rotatory nystagmus to the diseased side or vice verse, with a mixture of both these characteristics in between. The mechanism for such changes might be found in diffusion of potassium from the region around one nerve branch spreading to another. It has been evidenced that ruptures might heal, the potassium will then be absorbed by the perilymphatic tissue and the attack is over, whereas a non-healing rupture produces a permanent fistula, with deterioration of the brain cell function as result.

In conclusion: the distention, location of the rupture and the paralysing effect of the endolymph potassium, explains and determines the symptoms from the cochlea, from one of the vertical canals or from the horizontal. It therefore seems that the theory of nerve blocking by endolymph potassium is the most likely explanation for the mechanism of the Menière attacks. It might explain all the main symptoms of an attack by one single mechanism, which has the advantage of having been experimentally confirmed.

References

Beentjes, B.: The cochlear aqueduct and the pressure of cerebrospinal and endolabyrinthine fluids. Acta oto-laryng. **73**, 112 (1972)

Hallpike, C., Cairns, H.: Observations on the pathology of Menière's syndrome. J. Laryng. **53**, 625 (1938)

Henriksson, N., Gleisner, L.: Vestibular activity at experimental variation of labyrinthine pressure. Acta oto-laryng. **61**, 380 (1966)

Henriksson, N., Gleisner, L., Johansson, G.: Experimental pressure variation in the membranous labyrinth of the frog. Acta oto-laryng. **61**, 281 (1966)

Schuknecht, H. F.: Pathology of Menière's disease and correlation of pathology with symptoms of Menière's disease. In: Menière's disease (ed. J. Pulec). Philadelphia-Toronto-London: J. B. Saunders 1968

Diskussionsbemerkungen

E. A. Schnieder (Solingen): Kalium lähmt den Nerven reversibel. Wie kann eine Schädigung, die keine morphologischen Schäden setzt, einen so lang anhaltenden Schwindel erzeugen?

G. F. Dohlmann (Weston): Kalium schädigt den Nerven. Eine experimentelle Labyrinthausschaltung beim gesunden Ohr erzeugt einen lang anhaltenden Nystagmus. Nicht so ist es beim Menière-Kranken. Nach der Labyrinthausschaltung sistiert der Nystagmus sehr bald. Auch die Intoxikation der Perilymphe im Experiment löst Nystagmus aus, allerdings nur kurzzeitig. Die permanente Fistel aber kann die Erklärung für den lange dauernden Anfall sein.

K.-H. Vosteen (Frankfurt a. M.): Wenn die Ausspülung des Kaliums aus der Perilymphe den Nystagmus im Experiment zum Erlöschen bringt, liegt darin ein therapeutisches Prinzip. Wir können die Perilymphdurchsatzrate ja nur erhöhen, indem wir die Durchblutung verbessern.