

Other TMD Related Research -- An Underlying Neurological Condition for Ménière's Disease?

Konrádsson, Nielsen and Carlborg are researching Ménière's Disease using the TMD technique. They find that posture induced changes in perilymphatic pressure are significantly greater in the diseased ear compared with the healthy ear (Konrádsson, 1999a; Konrádsson et al, 2000). They interpret this finding as a more efficient intracranial-cochlear pressure transfer in the diseased Ménière's ear, compared with the good ear.

“ The results also indicate that for the patients tested, the routes of communication are more effective in the diseased ear than in the healthy ear – a condition that may relate to the pathogenesis of Ménière's disease.”

The suggestion being made is that there is a greater degree of patency of the intracranial-cochlear routes in the ear with Ménière's Disease, and therefore, the cochlear is more susceptible to the continuous fluctuations in intracranial pressure. However, the opposite situation and the possibility of narrowing of these routes should also be considered. That is, the larger than expected change in the perilymphatic pressure both reflects the expected change in pressure with posture (as seen in the good ear) plus a component due to intracranial pressure waves causing a pressure 'pumping' action as a consequence of cochlear aqueduct narrowing. If this exists, I speculate that this pressure 'pumping' action could create intracranial-perilymph-endolymph pressure differentials and may be responsible for endolymphatic hydrops in some cases. It is not clear which of the two alternative explanations is valid and perhaps both are significant.

It is interesting to consider the work of the Konrádsson research group in the context of findings published by Boucarra et al (1998).

Konrádsson et al (2000) exposed 16 patients with Ménière's Disease to hypobaric conditions within a pressure chamber¹. A statistically significant relationship was found between those patients demonstrating the greatest change in perilymphatic pressure with posture as indicated by the TMD technique, and those showing the greatest improvement in hearing threshold at the low frequencies (250 Hz and 500 Hz) with treatment provided by hypobaric exposure. Konrádsson et al conclude that the TMD may have a predictive value regarding the outcome of hypobaric exposure for patients with Ménière's Disease. Furthermore, they conclude that there appears to be a relationship between the efficiency of the route of pressure transfer and the observed effect of hypobaric exposure. Again there appears to be an association between the intracranial-cochlear routes of communication (cochlear aqueduct) and the Ménière's Diseases process.

¹ The chamber pressure was set at 20 daPa/s to levels of -180 to -480 daPa (mean -285 daPa) relative to the prevailing atmospheric pressure, and the maximum hypobaric pressure was individually chosen so as to avoid opening of the Eustachian Tube and equilibration of the middle ear pressure. By this method the cochlear is exposed to a relative overpressure compared to that of the intracranial pressure, for duration which will depend on the degree of patency of the cochlear aqueduct (or any other intracranial-cochlear connecting pathways). The number of episodes of hypobaric exposure were 4 to 13 (mean 7.4) and the total duration was 24.4 ± 9.8 minutes. Just before normalisation of chamber pressure the recorded relative middle ear over pressure was 185 ± 105 daPa.

Boucarra et al (1998) compared a control group with normally hearing (n=7), with those with non-progressive sensorineural hearing loss (n=9), a group with fluctuating hearing loss which had been hospitalised during a phase of deterioration (n= 8), and similarly a group with Ménière's Disease² in an active phase (n=25). The control group and those with sensorineural hearing loss demonstrated normal perilymphatic pressure as assessed by TMD for the group as a whole. However, all the patients with a fluctuating hearing loss in an acute phase demonstrated raised perilymphatic pressure (inwards TMD). About 50% of the Ménière's Group similarly demonstrated raised perilymphatic by TMD assessment. Boucarra et al postulate the existence of two subgroups amongst the Ménière's Disease patients, and that these subgroups suggest the presence of normal and high pressures. Furthermore, they propose that two separate pathogenesis for Ménière's Disease may exist, or alternatively that a single pathogenesis exists with a fluctuating evolution of the pressure (i.e. about 50% of the patients were tested during a normal pressure phase). Their proposition is also that the high pressure group may have a pathogenesis common with the fluctuating hearing loss group that similarly show raised cochlear pressure.

Unfortunately Boucarra et al do not report the state of the intracranial-cochlear pressure exchange as assessed by using TMD and postural manoeuvres. However, if we assume that most of these Ménière's and 'fluctuating hearing loss' patients do have communicating intracranial-cochlear fluids, then raised perilymphatic pressure implies raised intracranial pressure and/or the presence of abnormal intracranial pressure waves. That is, the findings of Boucarra et al imply that in at least 50% of cases, the underlying pathology of Ménière's Disease is of a neurological origin.

Could Benign Intracranial Hypertension (BIH) be the neurological disorder underlying Ménière's Disease? This is unlikely since, although BIH is known to clinically present with Ménière's Disease like symptoms, adult BIH is predominately a female condition and Ménière's of a classical form is approximately equally prevalent in males and females. The Ménière's Group tested by Boucarra was 11 men and 14 females, although they note a small female predominance, this was not statistically significant and also does not support the BIH conjecture. So BIH would not appear to be the most likely neurological candidate. There are several other aspects of the clinical presentation of the BIH condition that makes it dissimilar to Ménière's Disease. Other striking differences are that the tinnitus with BIH is usually low frequency in character and it is likely to be bilateral. However, BIH cannot be considered to have a single pathogenesis as in 80% of cases the cause is of an ideopathic nature. It leaves the possibility that a subgroup of BIH population group will be the same as a Ménière's neurological sub-group. In addition other conditions need to be considered as candidates for Meniere's subgroups, such as Chiari I Malformation as proposed by Milhorat et al (1999), aqueductal stenosis as proposed by Barlas et al, 1983, or cerebral vascular disorders.

Finally, a clear distinction should be made between the existence of distinct neurological pathologies that underlay subgroups of Ménière's Disease and merely a neurological association. In the later case, the possibility that some ears are simply just more susceptible to intracranial pressure waves should not be overlooked.

Robert Marchbanks, 2001

² Ménière's Disease as defined by the American Academy of Otolaryngology, Head and Neck Surgery criteria.

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