Letters to the Editor

Raised ABR threshold after suction aspiration of glue from the middle ear: three case studies

Dear Sir,

I have read with interest the paper by Mason *et al.* in the August issue of the Journal.

Auditory brainstem response (ABR) thresholds measured immediately after suction aspiration of middle ear fluid were compared with hearing assessment, in some cases with repeat brainstem response audiometry (BRA), at a later date. In six of 14 ears the latter threshold was improved by 15 dB or more. These results are interpreted as indicating a possible temporary threshold shift due to suction noise.

There is, however, another possible explanation. We have previously shown that the time-course of hearing threshold improvement following myringotomy, aspiration of middle ear secretion and insertion of a ventilation tube is frequency dependent (Mair *et al.*, 1989). Threshold improvement in the low frequency range of 0.25-1 kHz is immediate, is delayed at 4-8 kHz and further delayed, by two to eight weeks, in the extra-high frequency range of 9-20 kHz.

Since click-evoked ABR thresholds at moderate to high intensities originate from the 2–8 kHz region of the cochlea (Eggermont and Don, 1980; Burkard and Hecox, 1983), a persistent threshold elevation at 4 and 8 kHz immediateley after myringotomy, suction and grommet insertion would result in a high ABR theshold. The use of 1 kHz pure-tone pips as ABR stimulus, especially when high-pass masking is not employed, results in high thresholds even in normal-hearing adults, a correction factor of 30–40 dB having been indicated (Fjermedal and Laukli, 1989).

The threshold differences reported by Mason *et al.* (1995) may be due to the frequency dependent timecourse of threshold improvement following tubulation and, possibly, the inherent difficulties associated with response identification with low-frequency stimuli.

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Authors' reply

Dear Sir,

We would like to respond to the valuable comments made by Dr Mair in his letter regarding our August publication entitled:

'Raised ABR after suction aspiration of glue: three case studies', Mason, J. D. T., Mason, S. M. and Gibbin, K. P.

The causation of temporary hearing losses following myringotomy, aspiration of middle ear secretion and insertion of grommets is likely to be of complex origin. We suggested that sucker noise associated with aspiration of glue was a significant factor in the short term supported by (1) there was no threshold shift in children with dry ears and (2) repeat recordings (only 30 minutes later) of the clickevoked ABR in one of our case studies showed marked improvement in threshold. However, other factors cannot be excluded as indicated in our paper (e.g. pressure changes at the round window) and also as suggested by Dr Mair (e.g. temporary retention of secretion and/or mucosal oedema; Mair *et al.* (1989)).

It is interesting to note the frequency dependence time course of threshold improvement after middle ear intervention reported by Mair *et al.* (1989). Their results using subjective audiometry are consistent with our ABR findings. However, our experiences in the operating threatre suggest an even higher elevation of the click-evoked ABR threshold immediately after surgery (average of 30 dB) compared to subjective results reported by Mair *et al.* in the 24 hour post-operative period (around 10 dB).

We agree with Dr Mair regarding the difficulty of identification of the ABR with low-fequency tonepip stimuli such as 1 kHz, particularly with stimuli close-to-threshold. In our experience of routine ERA with children an offset of 10 to occasionally 20 dB for the 1 kHz-ABR is more typical with good recording conditions (e.g. with sedation and anaesthesia). We support Dr Mair's opinion that this difficulty with response identification is likely to be a contributing factor as to why the 1 kHz ABR threshold is raised above the expected hearing level, although other explanations are possible as indicated in our paper.

J. Mason, S. Mason, K. Gibbin, Department of Otolaryngology, Leicester Royal Infirmary, Leicester LE1 5WW.

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Mair, I. W. S., Fjermedal, O., Laukli, E. (1989) Air conduction thresholds and secretory otitis media: a conventional and extra-high frequency audiometric comparison. *Annals of Otology, Rhinology and Laryngology* 98: 767-771.

Middle ear pressures in patients with nasopharyngeal carcinoma and their clinical significance

Dear Sir,

I read with great interest the paper by Low in the May 1995 issue of the Journal. He draws attention to the case of patients with nasopharyngel carcinoma, developing post-irradiation middle ear effusions (MEE). He observed irradiation-induced MEE only in ears with pre-irradiation moderate to severe negative middle ear pressures. He therefore suggests that post-irradiation MEE occurs only in those cases where there has been a direct involvement of the Eustachian tube or its associated muscles by the tumour prior to radiotherapy. A combined effect of disease and treatment is thus suspected. However, irradiation-induced MEE does occur even in patients where the pathology involved has no influence on the patency of the Eustachian tube (Anteunis et al., 1994). In patients with unilateral parotid gland tumours, post-irradiation hearing loss has been documented in a prospective survey and sensorineural hearing loss was also noted. I do agree with Low that, in the presence of middle ear pathology, bone conduction thresholds not only reflect the cochlear (dys-) function but also the effect of altered middle ear transmission on the inner ear in addition to the usual test-retest variabilities. However, I disagree with him when he tries to explain all or most shifts in sensorineural thresholds with these mechanisms. Changes may occur in middle ear, inner ear, auditory nerve and brainstem after conventional radiotherapy and substantial conductive as well as sensorineural hearing losses may result from it.

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Reference

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Author's reply

Dear Sir,

I thank Dr Anteunis for the comments on my paper (Low, 1995). I agree that after radiotherapy, middle ear effusion (MEE) can sometimes develop in ears with normal Eustachian tube and associated muscles. However, I am still of the opinion that after radiotherapy, there is a tendency for MEE to develop in ears with tumour involvement of the Eustachian tube and associated muscles.

As explained in my paper (Low, 1995), I have reasons to believe that the pathogenesis of MEE in nasopharyngeal carcinoma (NPC) is more complicated than merely Eustachian tubal obstruction, whether mechanically or functionally. We recently performed magnetic resonance imaging studies along the lengths of the Eustachian tubes in patients with NPC prior to radiotherapy (Low *et al.*, 1995). We found a tendency for the Eustachian cartilage to be eroded by tumour in those patients with MEE. This led us to postulate that abnormal compliance of the Eustachian tube plays a major role in the pathogenesis of MEE in patients with NPC prior to radiotherapy.

I believe that at least in some cases, the MEE which results after radiotherapy may also be a result of altered Eustachian tubal compliance. It is not difficult to imagine that irradiation itself can change the compliance of the Eustachian tube, especially when the tumour has already involved the Eustachian tube and its associated muscles.

Afer radiotherapy for NPC, minor shifts in sensori-neural and conductive hearing were observed in many patients, resulting either in hearing loss or hearing gain (Low, 1995). I attributed these hearing changes to test-retest variabilities or changes in middle ear pressures which can explain both the post-irradiation hearing deterioration and improvement. I agree with Dr Anteunis that changes may occur in the ear, auditory nerve and brainstem after radiotherapy. These changes can account for the hearing loss but not the hearing gain observed after irradiation.

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