

thesia). 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.

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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 nasopharyngeal 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.

After 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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