Mr Graham's second point is practical and of great importance. There is nothing to suggest the patient which is the subject of this paper had ever had a spontaneous CSF leak or meningitis. If it was to occur on the other side we agree it would present an excellent opportunity to put in a multichannel device while sealing the leak.

As a result of reports of successful intracochlear multichannel implantation in common cavities, we considered this procedure for our patient but the limited benefit at the age of ten, the risk of macrocephaly being an indication of raised intracranial pressure, decide us in favour of a single channel extracochlear device.

The purpose of this paper was to recommend this approach to other surgeons, who for whatever reason might favour a conservative implant procedure in such cases.

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Bilateral sudden sensorineural hearing loss following non-otologic surgery

Dear Sir,

Post-operative otovestibular disturbances.

De la Cruz and Bance (*JLO* **112:** 769–771) reported a case of post-surgical hearing loss, only finding 25 similar case reports. They described seven mechanisms by which sudden sensorineural deafness might occur, concluding that all theories were speculative at best. There is, however, a well-documented and perfectly satisfactory explanation, endolymphatic hydrops, which curiously they ignored completely.

Abundant audiological evidence for cochlear hydrops appears in the three cited cases of Cox and Sargent: *Case 1*. Aural fullness, lightheadedness, flattish bilateral hearing loss, *Case 2*. Fullness, profound flat loss on right maximal at 500 and 1000 Hz, typical low tone and high tone loss on left with normal hearing at 2000 Hz, *Case 3*. Muffled hearing and tinnitus, fullness in right ear, severe flat loss on right maximal at 500 Hz, *Their case*. Nausea/vomiting/vertigo, severe fluctuant right deafness maximal at 250 Hz with characteristic 4000 Hz peak, profound left fluctuant loss maximal below 2000 Hz.

Even separately these features strongly indicate hydrops. *Cases 2 and 3* followed spinal surgery. Panning *et al.* (1983) noted typical fluctuant peaked losses after urological operations under spinal anaesthesia; which they could not explain; a similar case appeared in Cox and Sargent's review. Cochlear hydrops due to low CSF pressure was the only mechanism proposed (Gordon, 1983). This idea is not new.

De la Cruz and Bance located one previous report of sporadic sudden deafness in otosclerosis (DuVall *et al.*, 1981). In the discussion to this report, the only proposed mechanism in the non-stapedectomized ear was hydrops, for which Paparella stated there was ample clinical and pathological evidence. A previous review in this journal (Hochermann and Reimer, 1987), the first to claim a bilateral loss, labelled it " 'Meniere-like' low frequency hearing impairment". Audiosensitivity, an often ignored symptom of hydrops (Gordon, 1983; 1997), was also noted.

Walsted (1998) prospectively performed serial audiometry on 34 patients undergoing spinal anaesthesia, 60 having neuromas removed from their opposite ears and 32 having neurosurgery. Reversible low-tone or peaked losses typical of hydrops were common, especially after large losses of CSF. No other type of deafness was seen.

There is an extensive literature on post-operative nausea and vomiting (Watcha and White, 1992). It would be surprising if its cause was unrelated to the occasional case of deafness, especially if associated with vertigo, as above. Although there is no consensus as to its origin, there are many facts implicating dehydration or labyrinthine fluid pressure changes. Risk factors are previous motion sickness, opioids that sensitize the labyrinth, motion or change in position, otological surgery, middle ear pressure change (with nitrous oxide), spinal anaesthesia, hypotension and dizziness. Sickness is reduced by drugs effective in motion sickness and otovestibular disorders. Extra fluids dramatically reduced post-operative thirst, dizziness and nausea (Yogendran *et al.*, 1995).

De la Cruz and Bance's case developed musical hallucinations shortly after vertigo and tinnitus. In a recent case (Marneros *et al.*, 1997), also otosclerotic, hallucinations were clearly related to alcohol intake and withdrawal, implicating labyrinthine pressure changes, and were abolished by stapedectomy. A review of musical hallucinations (Gordon, 1997) shows that irrespective of any concurrent psychiatric, psychological, otological, pharmacological, religious or mystical states, there is always an associated hyperirritable inner ear or incipient hydrops. The pathological basis is perilymphatic hypotension from dehydration, weight loss, hypotension or loss of CSF. Thus a lumbar puncture led to a mystical state (Judson and Wiltshaw, 1983), including a powerful rhythm or vibration: 'It could be likened to a pulse or even a tune sounding through the universe'.

Despite many appeals in neurological journals (Gordon, 1998). I have not found neurological examples of musical hallucinations. If any otologist knows of a case of musical hallucinations where thorough examination excludes fluctuant low tone deafness and all symptoms of hydrops or ear disease, could they please report them, as otherwise psychiatrists, reliant on a neurological model of auditory hallucinations, are in deep trouble.

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Author's reply

I thank Mr Gordon for his illuminating and interesting commentary on our case. There are a few points in his letter I would like to address. Firstly, the ear has only a limited number of possible responses to insult of any kind, 'muffled hearing loss', 'tinnitus', 'fullness' are common accompaniments of any sudden hearing loss, and cannot be taken as definitive evidence of hydrops, although I do agree they are suggestive. Much of the literature Mr Gordon cites describe hearing changes after loss of CSF, which was not the situation in our reported case with no spinal anaesthesia and no opening of the dura. Thirdly, I have a very difficult time believing that musical hallucinations are an end organ phenomenon, a result of 'hyper irritable inner ear or incipient hydrops'. In my experience with these hallucinations, they are often complex musical arrangements, and many times patients will describe them as a tape recording. In fact, often they are songs from childhood, and at least two musician patients have been able to listen to them accurately enough to transcribe them in musical notation. It is difficult to comprehend how an irritable inner ear could produce music of this rhythmic arrangement. The most important point against a peripheral mechanism is that most of the cases I have encountered of musical hallucinations (four I can remember) have followed total ablation of the inner ear by a translabyrinthine removal of the inner ear, by definition excluding a peripheral mechanism, at least on the operated side.

Nevertheless, I do appreciate the insights Dr Gordon brings to this discussion and thank him for sharing his observations.

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Eye protection for ENT junior doctors

Dear Sir,

The importance of eye protection is well recognized in surgical fields (Bell and Clement; 1991, Berridge *et al*, 1993). Otolaryngology risks contamination in the operating theatre (Hinton *et al.*, 1991) and during ward procedures. Epistaxis is a common problem managed on the ward by junior doctors. Close proximity to a patient with epistaxis who may be sneezing or coughing inevitably results in a wide dispersion of blood. It would be reasonable to expect doctors packing noses to be provided with eye protection in expectation of this hazard. A recent audit of 20 SHO's working in different departments countrywide, including inner-city areas, was performed by telephone questionnaire.

Fourteen of those questioned said no protection was provided. Goggles or visors were provided for only six. However these were irregularly utilized and only one doctor regularly wore eye protection. Sixteen had been recently splashed in the face and eyes. Conjunctival transmission of hepatitis B can occur and a case of HIV contracted in this way has been documented (Gioannini *et al.*, 1988).

It is argued that no distinction should be made between high risk and ordinary patients and universal precautions should be adopted in operating theatres (Wastell, 1992). These practices should be maintained on the wards. Glasses are known to provide insufficient protection against blood splashes (Brearley and Buist, 1989) and a full face visor should be available for doctors managing epistaxis.

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