practice for the management of acoustic neuromas. In this country, when patients first become aware that something may be wrong, they go in the first instance to their general practitioners, then, often after a substantial wait, are referred to and eventually seen by an ear, nose, and throat surgeon. By the time the necessary investigations have been performed, the various results assembled, and an appointment made further time has elapsed. Referral onwards to the "acoustic team" may involve significant further delay. When a decision to operate has eventually been made, a further period of delay on a waiting list may then occur. By the time the patient eventually comes to surgery the hearing has not infrequently by then deteriorated to the level at which any efforts directed to its preservation are usually pointless. This is clearly very unsatisfactory. In Germany, as in most developed countries elsewhere, they do things differently and such delays are not inherent in their systems. The system we have adopted in the United Kingdom is often euphemistically described as "gatekeeping" but in fact acts as a covert form of "rationing" and is clearly to the detriment of our patients. If in the United Kingdom we are to improve our rate of hearing preservation, a first step must be to treat the continuing risk to hearing as a matter of some urgency. It must be better for our patients if we are able to eliminate the various delays between initial presentation and surgery. Wherever possible we should be able to intervene while acceptable levels of hearing are still present.

The controversy about how to measure "acceptable" hearing continues. The use of the term "good" or "useful" hearing is no longer acceptable and, if used in scientific papers, a clear definition of what is meant by the term should also be set out. In our paper,² the difficulties of comparative assessment of hearing results are set out. It is now, I think, generally accepted that concepts, such as "serviceable hearing" or "good functional hearing" are insufficiently accurate to serve as useful comparators for results. It is generally agreed among British otologists and neurosurgeons that a less than 30 dB loss on pure tone audiogram and a speech discrimination score of 70% or

more represents the best definition of "good", "serviceable", "good functional" or "socially useful" hearing and I would make a plea for this definition to be accepted as the gold standard for these terms. If such criteria are used then the number of cases with "good" or "socially acceptable" preoperative hearing is likely to be much fewer than reported in most recent series and the postoperative outcomes will be correspondingly somewhat worse. Unless preoperative and postoperative hearing results are reported in this objective manner comparison of hearing outcomes becomes very difficult. All papers which do not report their results in this manner should now perhaps carry a "government health warning".

In our series, under these strict criteria, our postoperative hearing preservation rate is poor (less than 5%). The only preoperative factor that predicted a favourable hearing preservation outcome was the presence of normal preoperative BAER morphology. The outcomes in the paper by Tonn *et al* seem substantially better but direct comparisons are not possible as audiological and speech discrimination results are not explicitly given. However, it may nevertheless be correct that our hearing preservation results are indeed poorer than those of our more progressive neighbours. In surgery great technical breakthroughs are rare: the usual method of progress is by incremental improvements applied to a multiplicity of separate factors but it is much easier to identify those factors which might count if we have clear and comparable reporting criteria beforehand. It is also true that we should not first shoot ourselves in the foot by accepting such long delays before embarking on our surgery so that the exercise has already become pointless.

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Cerebral dysfunction and psychotic symptoms in Alzheimer's disease

Psychotic phenomena are far from uncommon in Alzheimer's disease; delusions are present in up to three quarters of patients, hallucinations in up to a half. They appear as the disease progresses into its middle phase and are associated with cognitive decline. A better understanding of the psychopathogenetic mechanisms through which psychotic experience is mediated would have implications that would extend well beyond Alzheimer's disease; in this respect it could provide a neurological model for the so called functional psychoses. Some studies have used functional neuroimaging to identify those parts of the brain implicated in the psychotic process. The findings have been inconsistent. The paper by Mega *et al* in this issue (pp 167-171)¹ describes a methodology that represents a distinct advance. The neuropsychiatric inventory², a caregiver based instrument, was employed to control for concomitant behaviours other than those that are psychotically driven. Ten behavioural variables were rated, only

two of which, delusions and hallucinations, are psychotic. A close match was achieved for non-psychotic behaviours as well as for sex, cognitive performance, and education, but only at a cost: the initial pool of 210 patients was reduced to psychotic and non-psychotic groups of 10 patients each.

The groups so formed underwent single photon emission computed tomography (SPECT) neuroimaging. In the psychotic group perfusion was significantly lower in 11 regions, most of which were clustered in the prefrontal cortex bilaterally, the left striatum, and the left parietal cortex. Reductions in regional perfusion in the temporoparietal cortex are characteristic of Alzheimer's disease³: reductions in prefrontal cortical perfusion would not necessarily have been predicted, so inevitably these findings provoke speculation concerning the role of executive function (or rather dysfunction) in the appearance of psychotic phenomena. These are not necessarily generated in the

prefrontal striatal areas (for example, there is some evidence to implicate the middle temporal cortex in the production of hallucinations⁴) but Mega *et al* are right to emphasise the critical significance of executive failure: apsychoic hallucination is not simply an isolated perceptual aberration; it owes its distinctive psychotic quality to the failure of insight, influential judgement, and self monitoring capacity that are characteristic of executive dysfunction.

Psychotic symptoms are not usually present during the early stages of Alzheimer's disease. Does their emergence signify the advance of Alzheimer's pathology into the prefrontal regions? Future studies that seek to replicate these

findings should include measures of executive function in their study design.

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