- 59 Schiano TD, Parkman HP, Miller LS, *et al.* Use of botulinum toxin in the treatment of achalasia. *Dig Dis* 1998;16:14–22.
- 60 Fowler CJ. Disorders of the pelvic floor. In: Moore P, ed. *Botulinum toxin treatment*. Oxford: Blackwell, 1995:263–9.
- 61 Carruthers A, Carruthers JDA. Botulinum toxin in the treatment of glabellar frown lines and other facial wrinkles. In: Jankovic J, Hallett M, eds. *Therapy with botulinum toxin*. New York: Marcel Dekker, 1994:577–95.
- 62 Naumann M, Hofmann U, Bergmann I, *et al.* Focal hyperhidrosis. *Arch Dermatol* 1998;134:301–4.
- 63 Schneider P, Binder M, Kittler H, *et al.* Double blind trial of botulinum A toxin for the treatment of focal hyperhidrosis of the palms. *Br J Dermatol* 1998;138:553–4.
- 64 Odderson IR. Axillary hyperhidrosis: treatment with botulinum toxin A. *Arch Phys Med Rehabil* 1998;79:350–2.
- 65 Quardo MA, Borodic GE. Treatment of myofascial pain with botulinum A toxin. *Anesthesiology* 1994;80:705–6.
- 66 Hobson DE, Gladish DF. Botulinum toxin injection for cervicogenic headache. *Headache* 1997;37:253–5.
- 67 Barnes MP. Management of spasticity. *Age Ageing* 1998;27:239–45.
- 68 Das TK, Park DM. Effect of treatment with botulinum toxin on spasticity. *Postgrad Med J* 1989;65:208–10.
- 69 Pullman SL, Greene P, Fahn S, *et al.* Approach to the treatment of limb disorders with botulinum toxin A. Experience with 187 patients. *Arch Neurol* 1996;53:617–24.
- 70 Dengler R, Neyer U, Wohlfarth K, *et al.* Local botulinum toxin in the treatment of spastic foot drop. *J Neurol* 1992;239:375–8.
- 71 Memin B, Pollak P, Hommel M, *et al.* Treatment of spasticity with botulinum toxin. *Rev Neurol (Paris)* 1992;148:212–14.
- 72 Hesse S, Friedrich H, Domasch C, *et al.* Botulinum toxin therapy for upper limb spasticity: preliminary results. *Journal of Rehabilitation Science* 1992;5:98–101.
- 73 Konstanzer A, Ceballos-Baumann AO, Dressnandt J, *et al.* Botulinum toxin A treatment in spasticity of arm and leg. *Nervenarzt* 1993;64:517–52.
- 74 Dunne JW, Heye N, Dunne SL. Treatment of chronic limb spasticity with botulinum toxin A. *J Neurol Neurosurg Psychiatry* 1995;58:232–5.
- 75 Bhakta BB, Cozens JA, Bamford JM, *et al.* Use of botulinum toxin in stroke patients with severe upper limb spasticity. *J Neurol Neurosurg Psychiatry* 1996;61:30–5.
- 76 Sampaio C, Ferreira JJ, Pinto AA, *et al.* Botulinum toxin type A for the treatment of arm and hand spasticity in stroke patients. *Clinical Rehabilitation* 1997;11:3–7.
- 77 Grazko MA, Polo KB, Bhaman J. Botulinum toxin for spasticity, muscle spasms, and rigidity. *Neurology* 1995;45:712–17.
- 78 Simpson DM, Alexander DN, O'Brien CF, *et al.* Botulinum toxin type A in the treatment of upper extremity spasticity: a randomised, double blind, placebo controlled trial. *Neurology* 1996;46:1306–10.
- 79 Pierson SH, Katz DI, Tarsy D. Botulinum A toxin in the treatment of spasticity: Functional implications and patient selection. *Arch Phys Med Rehabil* 1996;77:717–21.
- 80 Burbaud P, Wiart L, Dubos JL, *et al.* A randomised, double blind, placebo controlled trial of botulinum toxin in the treatment of spastic foot in hemiparetic patients. *J Neurol Neurosurg Psychiatry* 1996;61:265–9.
- 81 Hesse S, Lucke D, Malezic M, *et al.* Botulinum toxin treatment for lower limb extensor spasticity in chronic hemiparetic patients. *J Neurol Neurosurg Psychiatry* 1994;57:1321–4.
- 82 Snow BJ, Tsui JKC, Bhatt MH, *et al.* Treatment of spasticity with botulinum toxin: a double blind study. *Ann Neurol* 1990;28:512–15.
- 83 Benecke R. Botulinum toxin for spasms and spasticity in the lower extremities. In: Jankovic J, Hallett M, eds. *Therapy with botulinum toxin*. New York: Marcel Dekker, 1994:557–65.
- 84 Yablson SA, Agana BT, Ivanhoe CB, *et al.* Botulinum toxin in severe upper extremity spasticity among patients with traumatic brain injury: an open labelled trial. *Neurology* 1996;47:939–44.
- 85 Pavesi G, Brianti R, Medici D, *et al.* Botulinum toxin type A in the treatment of upper limb spasticity among patients with traumatic brain injury [letter]. *J Neurol Neurosurg Psychiatry* 1998;64:419–20.
- 86 Palmer DT, Horn LJ, Harmon RL. Botulinum toxin treatment of lumbrical spasticity: a brief report. *Am J Phys Med Rehabil* 1998;77:348–50.
- 87 Cosgrove AP, Graham HK. Botulinum toxin A prevents the development of contractures in the hereditary spastic mouse. *Dev Med Child Neurol* 1994;36:379–85.
- 88 Cosgrove AP, Corry AP, Graham HK. Botulinum toxin in the management of the lower limb in cerebral palsy. *Dev Med Child Neurol* 1994;36:386–96.
- 89 Koman CA, Mooney JF, Smith B, *et al.* Management of cerebral palsy with botulinum A toxin: preliminary investigation. *J Pediatr Orthop* 1993;13:489–95.
- 90 Wall SA, Chait LA, Temlett JA, *et al.* Botulinum A chemodenervation: a new modality in cerebral palsied hands. *Br J Plast Surg* 1993;46:703–6.
- 91 Koman CA, Mooney JF, Smith B, *et al.* Management of spasticity in cerebral palsy with botulinum A toxin: report of a preliminary, randomised, double-blind trial. *J Pediatr Orthop* 1994;14:299–303.
- 92 Sutherland DH, Kaufman KR, Wyatt MP, *et al.* Injection of botulinum A toxin into the gastrocnemius muscle of patients with cerebral palsy: a 3-dimensional motion analysis study. *Gait Posture* 1996;4:269–79.
- 93 Gooch JL, Sandell TV. Botulinum toxin for spasticity and athetosis in children with cerebral palsy. *Arch Phys Med Rehabil* 1996;77:508–11.
- 94 Wong V. Use of botulinum toxin injection in 17 children with spastic cerebral palsy. *Pediatr Neurol* 1998;18:124–31.
- 95 Arens LJ, Goldschmidt RB, Leary PM. Botulinum toxin A in the hemiplegic upper limb: a double blind trial [letter]. *Dev Med Child Neurol* 1997;39:491–2.
- 96 Corry AP, Cosgrove AP, Walsh EG, *et al.* Botulinum toxin A in the hemiplegic upper limb: a double blind trial. *Dev Med Child Neurol* 1997;39:185–93.
- 97 Corry AP, Cosgrove AP, Duffy CM, *et al.* Botulinum toxin A compared with stretching casts in the treatment of spastic equinus: a randomised prospective trial. *J Pediatr Orthop* 1998;18:304–11.
- 98 Reiter F, Danni M, Lagalla G, *et al.* Low dose botulinum toxin with taping for the treatment of spastic equinovarus foot after stroke. *Arch Phys Med Rehabil* 1998;79:532–5.
- 99 Hesse S, Reiter F, Konrad M, *et al.* Botulinum toxin type A and short term electrical stimulation in the treatment of upper limb flexor spasticity after stroke: a randomised, double blind, placebo controlled trial. *Clin Rehabil* 1998;12:381–8.
- 100 Jankovic J, Schwartz K. Response and immunoresistance to botulinum toxin injections. *Neurology* 1995;45:1743–6.
- 101 Whitaker J, Butler AG, Semlyen JK, *et al.* Botulinum toxin for people with dystonia treated by an outreach nurse practitioner: a comparative study between a home and a clinic treatment service. *Arch Phys Med Rehabil* 2000 (in press).

EDITORIAL COMMENTARIES

Acoustic neuroma surgery as an interdisciplinary approach

For such a relatively rare tumour, acoustic neuroma continues to hold a remarkable fascination for both neurosurgeons and neuro-otologists. The Third International Conference on Acoustic Neuroma in Rome last June continued for 5 days!

The early controversies in the management of acoustic neuromas were largely about the surgical approaches which might be used and about the outcome of facial nerve function. In this period the improved outcomes which could be achieved by utilising the combined expertise of otologists and neurosurgeons, were clearly demonstrated. Likewise, the advantages to be gained by monitoring facial nerve function were also firmly established. More recently, controversy has shifted to the question of hearing preservation and, in particular, to the unresolved problem of what constitutes “useful” hearing. The place of intraoperative brain stem auditory evoked response (BAER) audiometry in improving hearing outcomes is still uncertain, largely because of the known difficulties in

obtaining reliable recordings. However, on empirical grounds it is likely that if hearing preservation rates are to improve, BAER audiometry will play a significant part in this improvement. The paper¹ by Tonn *et al* in this issue (pp 161–166) once more emphasises the benefits of interdisciplinary teamworking. It also indicates that BAER monitoring may indeed have a part to play in improved hearing outcomes. However, it also raises other perhaps more interesting questions. The series is large and the analysis of the patient material indicates that 78.5% of the series had “good” hearing preoperatively (grade 1–3 on the Gardner-Robertson scale). This is a remarkably high proportion of patients with “good” preoperative hearing. In the Cambridge series (now 725 patients) the number of patients who have “good” preoperative hearing are in a substantial minority. Most of our patients have had either “dead” ears or very poor hearing. This is a significant difference and I suspect that it reflects the unsatisfactory level of delay inherent in current United Kingdom

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.

D G HARDY

Department of Neurosurgery, Box 166, Level 2, Block A, Addenbrooke's Hospital, Hills Road, Cambridge CB2 2QQ, UK

1 Tonn J-C, Schlake H-P, Goldbrunner R, *et al*. Acoustic neuroma surgery as an interdisciplinary approach: a neurosurgical series of 508 patients. *J Neurol Neurosurg Psychiatry* 2000;69:161-6.

2 Moffat DA, DaCruz MJ, Baguley DM, *et al*. Hearing preservation in solitary vestibular schwannoma surgery using the retrosigmoid approach. *Otolaryngol Head Neck Surg* 1999;121:781-8.

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