PostScript

CORRESPONDENCE

Experiential phenomenon on cortical stimulation

I read with interest the short report "Human 'memories' can be evoked by stimulation of the lateral temporal cortex after ipsilateral medial temporal lobe resection", by Moriarty *et al.*¹

The cortical localisation of experiential memory phenomenon was summarised by Penfield and Perot.²

Subsequent to that, Halgren *et al.*³ used implanted depth electrodes to delineate medial temporal structures as a source of experiential phenomenon in patients with temporal lobe seizures. However, their work did not include simultaneous recordings from temporal neocortex and mesial structures. This subsequently was performed by Gloor *et al.*⁴

Based on this and a review of the images, there seems to be a fair amount of amygdala still present in this patient. Therefore, it was not surprising that simple seizures, as well as complex seizures, recurred within four months. Specific to the stimulation of the lateral temporal cortex after mesial temporal lobe resection, the anatomical work by Klingner and Gloor,⁵ along with the axial images, suggests that there was adequate anterior, inferior temporal, and insular cortex connected to residual amygdala to produce the experiential phenomenon on cortical stimulation.

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- 2 Penfield W, Perot P. The brain's record of auditory and visual experiences. A final summary & discussion. *Brain* 1963;86:595-696.
- 3 Halgren E, Walter RD, Cherlow DG, *et al.* Mental phenomena evoked by electrical stimulation of the human hippocampal formation and amygdala. *Brain* 1978;101:83-117.
- 4 Gloor P, Olivier A, Quesney LF, *et al.* The role of limbic system in experiential phenomena of temporal lobe epilepsy. *Ann Neurol* 1982;12:129-44.
- 5 Klingner J, Gloor P. The connections of the amygdala and of the anterior temporal cortex in the human brain. *J Comp Neurol* 1960;115:333-69.

Author's reply

Professor Lehman raises the interesting question of whether participation of the amygdala can account for the auditory hallucinations observed in our patient. He cites two previous studies in which electrical stimulation of the amygdala and hippocampus evoked experiential phenomena such as visceral sensations, déjà vu, and emotions.^{1,2} The experiential phenomena reported with stimulation of the amygdala were usually associated with after-discharges and did not include formed auditory hallucinations. In contrast, we reported formed auditory hallucinations evoked by neocortical stimulation, often without after-discharges, in a patient who had an extensive medial temporal resection.³ Our study suggests that medial temporal lobe involvement cannot account entirely for evoked auditory hallucinations. While our case does not preclude a contribution from the medial temporal lobe, we think it is more likely that neocortical structures are primarily involved. Further investigation will be required to determine the potential interaction between limbic and neocortical structures in experiential phenomena.⁴

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- 3 Moriarty JL, Boatman D, Krauss GL, *et al.* Human "memories" can be evoked by stimulation of the lateral temporal cortex after ipsilateral medial temporal lobe resection. *J Neurol Neurosurg Psychiatry* 2001;71:549-51.

- 4 Fish DR, Gloor P, Quesney FL, *et al.* Clinical responses to electrical brain stimulation of the temporal and frontal lobes in patients with epilepsy. *Brain* 1993;116:397-414.

BOOK REVIEW

Neuroanatomical basis of clinical neurology

By Orhan Arslan (Pp 432, £33.99). Published by Parthenon Publishing, Lancaster, 2001. ISBN 1 85070 578

Medical students find neuroanatomy difficult and often boring. A ruse that many teachers use is to refer to clinical implications. The author of this book has tried to condense that method into print. A classically organised, heavily illustrated text of neuroanatomy has been sprinkled with clinical vignettes, printed in blue. Sadly enough the result is disappointing. Firstly, the neurological vignettes are rather haphazardly chosen and often too long. In the section on the temporal lobe a single blue column throws together uncus herniation, Cheyne-Stokes respiration (sic!), uncinate fits, and Pick's disease. Secondly, the neurophysiological perspective, found in several other neuroscience textbooks for undergraduates, is dearly missing here. The problem is most sorely felt in the chapter on the reticular formation, where utter confusion reigns on disturbances of consciousness and its anatomical substrate. Thirdly, vignettes consist almost entirely of bare text; the few drawings of clinical signs are at best poor and often erroneous (there is no "ape hand" in carpal tunnel syndrome and chorea is chorea, whether Huntington's or Sydenham's). Lastly, more slip-ups occur in the text. Torticollis is said to be caused by muscle contracture, subarachnoid haemorrhage would usually be accompanied by aortic coarctation and polycystic kidney disease, and gliomas would become manifest mostly through headache and vomiting—to name a few.

What about the core of neuroanatomy? The magnetic resonance images are a nice feature but as always the book contains far more facts—such as the good old rubrospinal tract—than one has ever needed in decades of clinical practice. Even worse, some useful anatomical facts are missing: not only the caudal loop of central neurones to the facial nucleus but even perforating arteries and their relation to lacunar infarcts. In brief, neuroanatomy books should be written by clinicians, not the other way around.

Jan Van Gijn