phases in the brain stem in this case, at and beyond the margin of the area involved with CPN. No multinucleated cells were seen.

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4 Tormey WP. Central pontine myelolysis and changes in serum sodium. Lancet 1990; 335:1169.

Could midbrain “resting” tremor be caused by postural maintenance at rest?

James Parkinson described resting tremor suggesting that tremor in Parkinson’s disease (PD) persists even when the patient no longer has to maintain limb posture. So-called midbrain or rubral tremor characteristically includes resting, postural, and intention tremor. Gordon Holmes1 noticed that midbrain resting tremor ceased when the limb was completely at rest. Holmes’ observation suggests that midbrain resting tremor, contrary to resting tremor in PD, is caused by postural maintenance. We describe a patient with presumed midbrain tremor showing evidence that PD resting tremor and midbrain resting tremor may have a different neurophysiological background.

At the age of 63, a 67 year old man was suddenly struck by a left-sided third nerve palsy and a right-sided hemiparesis which disappeared after a few weeks. After this period resting, postural, and intentional tremor appeared in the right arm. He developed coarse, irregular myoclonic head shaking to the right and frequent generalized shuddering tremor lasting a few seconds. The patient was unsuccessfully treated with Sinemet. With orphenadrine 50 mg four times daily the tremor diminished, as did the shuddering attacks. Four years later the patient noticed that the entire limb tremor would disappear if he pushed firmly on the upper edge of the homolateral trapezius muscle.

On physical examination the right arm showed a complex resting tremor (Webster grade 2–3) with flapping flexion-extension at the wrist and elbow, and pronation-supination of the forearm. In our patient the first finger was beating against the thumb, though the classic “pill-pulling” movement of the thumb against the first finger, was absent (According to Denny-Brown2 these movements of our patient’s first finger and thumb differentiate midbrain tremor from PD tremor). The tremor amplitude increased on stretching the arm and performed the finger-nose test. With distraction, when the patient was at rest or lying on a bed the tremor sometimes disappeared. The right arm was hypokinetic (grade 1) and rigid (grade 2).

EMG showed regular 5 Hz bursts in the trapezius, supraspinatus and splenius capitis muscles, with the trapezius muscle constantly discharging 10 to 20 ms before the supraspinatus muscle. There were alternating 5 Hz bursts in the biceps and triceps muscles. CT head scan did not reveal any focal abnormalities.

The resting and action tremors were completely abolished by local intramuscular injection of 10–20 cc bupivacaineadrenaline solution into the supraspinatus and the adja-
cent part of the trapezius muscle. The effect on the tremor lasted for days to weeks, although it diminished after the first few days. The patient was treated 17 times with intervals of two weeks to three months. Unfortunately the 17th injection caused a troublesome pneumothorax, so that the patient refused further injections.

Although we do not have direct anatomical proof of a mesencephalic lesion in our patient, the clinical picture consisting of acute ipsilateral third nerve palsy and contralateral hemiparesis warrants a diagnosis of Benedikt’s syndrome as a result of mesencephalic stroke. In these patients a so-called midbrain tremor, which may be combined resting, postural and intentional tremor, may develop.

Direct evidence that postural maintenance rather than movement in our patient was provided by local intramuscular anaesthetic infiltration after which both action and resting tremor disappeared. Although we cannot explain why the beneficial effect was so long lasting, the effect itself is well known. According to Rondot,3 postural tremors may spread from one muscle to the other muscles of the limb. Intramuscular anaesthesia of the muscles in which the rhythmic activity originates stops the rhythmic phenomena in all muscles of the corresponding limb. This procedure was neither effective in suppressing the resting tremor in 3 of our PD patients with classic resting tremor, rigidity and hypokinetics at the injected side, nor in PD patients elsewhere, Rondot et al4 and Rondot (personal communication).

Sabra and Hallett5 argued that in cases of severe tremor appearing on postural mainten-
ance, Holmes' term “rubral tremor” should be avoided; “severe postural cere-
bellar tremor” is more appropriate because it is mainly the superior cerebellar peduncle which is involved. The most typical vascular form of this postural tremor is associated with a contralateral third nerve palsy.6 Antagonist muscles in these patients showed Parkinson-like alternating activity. Both find-
ings are also present in our patient. Although Sabra and Hallett mention only one of their 32 patients having a tremor at rest, our case suggests that postural tremor at rest may be part of such a condition.

We cannot exclude the possibility, depending on the site and extent of the lesion, other patients with so-called midbrain tremor show the characteristic resting tremor of PD. Dopa responsive midbrain tremor7 may belong to this group. Patients with midbrain tremor will be studied carefully in an attempt to resolve this question.

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6 Samie MR, Selhorst JB, Koller WC. Post-

Transcutaneous phrenic nerve stimulation

Transcutaneous phrenic nerve stimulation, with measurement of the terminal latency of the nerve, is a well recognised technique for assessing phrenic integrity. The technique used is essentially that described by News-
sworth-Davis in 1967,7 with modification of the diaphragmatic compound muscle action potential (CMAP) using surface electrodes placed over the chest wall. The exact position of the electrodes has been the subject of some discussion. Newsom-Davis originally recorded from the eighth intercostal space in the anterior axillary line. In other studies the fifth and sixth spaces, also in the anterior axillary line, the eighth space and the xiphisternum, and the seventh or eighth space near the costochondral junction have been used.8 Most recent studies have used the seventh or eighth intercostal spaces just anterior to the costal margin.9,10

In some papers, notably Newsom-Davis’ original work,11 there was slight concern over the possibility of stimulating nerves other than the phrenic, especially the accessory nerve by the brachial plexus, and producing a CMAP that did not reflect diaphragmatic contraction. This was not borne out clinically and brachial plexus stimulation, while common (especially in children), is unlikely to affect the CMAP seen. Other muscles which may also be stimulated, such as lariissimus dorsi, lie too far away from the electrodes to affect the signal. Stimulation of the serratus anterior muscle was also suggested as one of the confounding contraction but anterior place-
ment of the electrodes should avoid this as the origins of the muscle are from the lateral borders of the upper 8–10 ribs.

We are involved in a prospective study of phrenic nerve function in children having cardiac surgery, and over the period of a year we have successfully studied over 250 children before and after surgery. Chest electrodes were placed over the seventh intercostal space and over the eighth rib in the anterior axillary line. In a small number of children we are now examining an artefactual trace which was initially thought to be part of the diaphragmatic CMAP; we now recognise that it is clearly not. Figure 1 shows the preoperative trace of a normal five year old boy, with latency measured at 5 ms. Post-operatively his trace was that seen in figure 2. This shows an apparent latency of 2–6 ms with a normal appearance to the CMAP.

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However one looks at it, this is a weighty tome (3.4 Kg; 52 authors: 51 American and one Swiss; and editors associating, in three persons, to departments of Medicine, Internal Medicine, Neurology, Paediatrics, Microbiology and Immunology). The book is encyclopaedic in scope, beautifully produced and illustrated, extensively referenced, entertaining as well as learned and—no mean achievement these days—written in fluent, jargon-free English. If “language be the dress of thought” the authors emerge attractively clad. It is the sort of book neurologists should seek to own, not borrow. Lending books is, anyway, of all kindnesses the one that meets the least return.

No “fardering” here “of lean facts with the fat of others’ works”. Various disorders are first reviewed in depth, with special emphasis on pathogenesis, clinical differential diagnosis and treatment. Tuberculosis and syphilis of the nervous system are reviewed with the breadth of vision and pathological insight of many an older textbook devoted exclusively to these themes but spiced with fascinating information derived from newer immunological or imaging techniques. I know of no other book one could find, in a single volume, up-to-date reviews (and I mention but a few) dealing with the physiology of CSF production and reabsorption, the infection of CSF shunts, viral vaccines that protect the nervous system, space-occupying lesions due to fungi, slow viral infection, the neuropathology of endocarditis, the whole field of what should now perhaps be called “neuro-helmintholo-
gy”, HIV infections, immuno-prophylaxis against Neisseria meningitidis and against Bordetella pertussis, pitfalls in the practical management of neonatal meningitis, and the “imaging of intracranial infection”.

A good illustrated dictionary was once described as the sort of book where—when looking for one word—one was tempted, en passant, to check the meaning of many, many others. Going through the pages of this volume I succumbed often to this temptation. In the process I gained insight about how trypanosomes get into the CSF, about the upper motor neuron lesion in tetanus, and about salivation being defective in botu-
lism. I saw, in reproduced hieroglyphics, the first account of trisnus (in the Edwin Smith Surgical Papyrus). I discovered that there was a disease called “Rockefeller Fever” and even learned how Lapochi-
lascaris minor, a nematode of ocellots and opossums, destroyed the brain of a 14 year old boy in Brazil, in 1915.

I have but one criticism. It is that the net seems at times to be cast too wide. How else explain the presence of sections on the Guillian-Barré syndrome, or on the neuro-
dology of rheumatoid arthritis, Sjogren syndrome, polyrarthritis and sarcoidosis? Even “botulism” and “tetanus” seem interlopers in this perspective. These sections are so good, however, that I suppose all will have to be forgiven. What cannot be forgiven though is the reference to Taenia solium (admittedly not in the main chapter devoted to cysticercosis) as a “porcine” tapeworm (p 720).

Older readers will sense the time warp when encountering, in this ultra-modern text, the use of units pertaining to an earlier era. It was a surprise to see CSF protein concentrations given as mg% in some chapters (and as g/dl in others). CSF glucose concentrations (given as mg/dl—or as mg%) gave a sense of “pas vue depuis longtemps”, especially in a section describing how “lyses from the amebocytes of the horseshoe crab (Limulus polyphemus) currently assist in the identification, in the CSF, of endotoxins produced by Neisseria meningitidis, Haemo-
philus influenzae and other gram-negative bacteria.

C PALLIS

Surgery of the Sellar Region and Para-
nasal Sinuses. Edited by MJ SAMLI. (Pp 583; Price DM 398–). 1991. Heidelberg, Sprin-
ger-Verlag. ISBN 3 540 53697 3.

The book contains papers from the Fourth International Congress of the Skull Base Study Group held in Hanover. The aim was to bring together experts from many disci-
plines of the medical-surgical, diagnostic, procedural, surgery and other therapies used in this area of the skull base. This book was written to give an overview of modern practices and procedures being carried out and the variety of pathology and fascinating interesting and intricate area. There are excellent sections on the anatomy, a wealth of pathological entities are well described and there is a number of papers indicating the sophisticated modern radiological approaches. A area of surgical disciplines provide information on a number of approaches to the various pathologies around the sellar and paranasal regions. At an area that there is considerable overlap and repetition. It is unfortunate that in this book there are some excellent papers written as chapters while others are just the authors’ talks without the detail this type of specialist publication requires. The papers are variable, some being excellent and others being of questionable value with no clear message.

The book to some extent achieves its editor’s aims in supplying an up-to-date report on the state of the art. However, while the book carries much information it is difficult for a reader to get a clear picture of what is being done, how it affects patient outcome and what are the likely future surgical developments in this region of the

Letters to the Editor

BOOK REVIEWS

Figure 2 Postoperative study. The stimulus given was 10 mA at 100 µs duration. Electrode positioning was the same as figure 1.

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Transcutaneous phrenic nerve stimulation.

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