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

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1 Laureno R, Karp BI. Pontine and extra pontine myelinolysis following rapid correction of hyponatraemia. *Lancet* 1988;**i**:1439–41.
2 Boon AP, Carey MP, Salmon MV. Central pontine myelinolysis not associated with rapid correction of hyponatraemia. *Lancet* 1988; **ii**:458.
3 McKee AC, Winkelman MD, Banter B. Central pontine myelinolysis in severely burned patients, relationship to hyperosmolality. *Neurology* 1988;**38**:1211–7.
4 Tormey WP. Central pontine myelinolysis 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 Holmes[1] 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 generalised 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-rolling" movement of the thumb against the first finger, was absent (According to Denny-Brown[2] these movements of our patient's first finger and thumb differentiate midbrain tremor from PD tremor). The tremor amplitude increased on stretching the arms out and performing 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 adjacent 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, that is, combined resting, postural and intentional tremor, may develop.

Direct evidence that postural maintenance rather than rest was involved 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 hypokinesia at the injected side, nor in PD patients elsewhere, Rondot *et al*[3] and Rondot (personal communication).

Sabra and Hallett[4] argued that in cases of severe tremor appearing on postural maintenance, Holmes' term "rubral tremor" should be avoided; "severe postural cerebellar 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.[5] Antagonist muscles in these patients showed Parkinson-like alternating activity. Both findings 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 postural tremor.

We cannot exclude the possibility that, 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 tremor[6 7] may belong to this group. Patients with midbrain tremor will be studied carefully in an attempt to resolve this issue.

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1 Holmes G. On certain tremors in organic cerebral lesions. *Brain* 1904;**27**:327–75.
2 Denny-Brown, D. *The basal ganglia and their relation to disorders of movement*. London: Oxford University Press, 1962:66–67.
3 Rondot P, Korn H, Scherrer. Suppression of an entire limb tremor by anesthetizing a selective muscular group. *Arch Neurol* 1968; **196**:421–9.
4 Sabra AF, Hallett M. Action tremor with alternating activity in antagonist muscles. *Neurol* 1984;**34**:151–6.
5 Fahn S, Koller WC, Hallett M. What is it? Case 4. *Movement Disorders* 1986;**4**:299–308.
6 Samie MR, Selhorst JB, Koller WC. Post-traumatic midbrain tremors. *Neurol* 1990; **40**:62–66.
7 Findlay LJ, Gresty MA. Suppression of "rubral" tremor with levodopa. *BMJ* 1980;**28**:1043.

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 Newsom-Davis in 1967,[1] with measurement 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.[2] Most recent studies have used the seventh or eighth intercostal spaces just anterior to the costal margin.[3 4]

In some papers, notably Newsom-Davis's original work,[2] there was slight concern over the possibility of stimulating nerves other than the phrenic, especially those supplied 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[4]), does not affect the CMAP seen. Other muscles which also may be stimulated, such as larissimus dorsi, lie too far away from the electrodes to affect the signal. Stimulation of the serratus anterior muscle was also suggested as a possible confounding contraction[2] but anterior placement 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 have successfully studied over 250 children before and after surgery. Chest electrodes are 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 recognising 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. Postoperatively his trace was that seen in figure 2. This shows an apparent latency of 2·6 ms with a normal appearance to the CMAP.

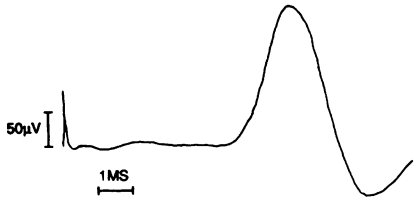

Figure 1 Preoperative study. The stimulus given was 8 mA for 100 μs duration. Recording electrodes were placed over the seventh intercostal space in the anterior axillary line and the eighth rib, just laterally.

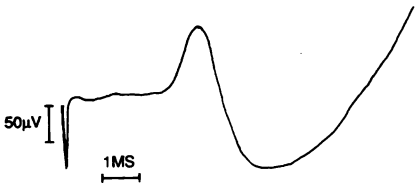

Figure 2 Postoperative study. The stimulus given was 10 mA at 100 μs duration. Electrode positioning was the same as figure 1.

Clinical observation at the same time, however, showed no diaphragmatic contraction in association with the stimulus, but a very vigorous contraction of the pectoral muscles was observed. Fluoroscopic examination of the diaphragm confirmed that it was paralysed. We suggest that the absence of any visible diaphragmatic movement, coupled with our knowledge of a "normal" preoperative latency, that is, 3–7 ms, implies that the electrical signal picked up was not from the diaphragm but may represent pectoral stimulation. The nerve supply to the pectoral muscles arises from C6, 7 and 8 for pectoralis minor and C7, 8 and T1 for the sternal portion of pectoralis major. In stimulating the phrenic nerve in small children, the probe often has to be held quite low in the neck and stimulation of these lower nerve roots may well occur. As the nerve supply to the serratus anterior is from C5, 6 and 7, this may also contract, but the electrode position in this CMAP recording makes it unlikely, as does the very short latency.

We suggest that in all studies of phrenic latency, especially in children, care is taken to ensure that there is clinical evidence of diaphragmatic contraction so that misinterpretation does not occur. Electrode positioning should be no higher than the seventh intercostal space to minimise the risk of pectoral artefact.

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1 Newsom-Davis J. Phrenic nerve conduction in man. *J Neurol Neurosurg Psychiat* 1967; 30:420–6.
2 Markand ON, Kincaid JC, Pourmand RA, *et al.* Electrophysiologic evaluation of diaphragm by transcutaneous phrenic nerve stimulation. *Neurology* (Cleveland) 1984;34:604–14.
3 Mier A, Brophy C, Moxham J, Green M. Phrenic nerve stimulation in normal subjects and in subjects with diaphragmatic weakness. *Thorax* 1987;42:885–8.
4 Ross Russell RI, Mulvey D, Laroche C, Shinebourne EA, Green M. Bedside assessment of phrenic nerve function in infants and children. *J Thorac Cardiovasc Surg* 1991;101: 143–7.

BOOK REVIEWS

All titles reviewed here are available from the BMJ Bookshop, PO Box 295, London WC1H 9TE. Prices include postage in the United Kingdom and for members of the British Forces Overseas, but overseas cutomers should add £2 per item for postage and packing. Payment can be made by cheque in sterling drawn on a United Kingdom bank, or by credit card (Mastercard, Visa or American Express) stating card number, expiratory date, and your full name.

Infections of the Central Nervous System. Edited by WM SCHELD, RJ WHITLEY AND DT DURACK (Pp 937; Price $199.00). 1991. New York, Raven Press. ISBN 0 88167 766 3

However one looks at it, this is a weighty tome (3·4 Kg; 52 authors: 51 American and one Swiss; and editors associating, in their three persons, professorships of Medicine, Internal Medicine, Neurosurgery, 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 "larding" here "of lean facts with the fat of others' work". 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 source where one would 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 neurology of infective endocarditis, the whole field of what should now perhaps be called "neuro-helminthology", 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 others. Going through the pages of this volume I succumbed often to this temptation. In the process I gained insight about how trypanosomes got into the CSF, about the upper motor neurone lesion in tetanus, and about salivation being defective in botulism. I saw, in reproduced hieroglyphics, the first account of trismus (in the Edwin Smith Surgical Papyrus). I discovered that there was a disease called "Rocky Mountain Spotless Fever" and even learned how *Lagochilascaris minor*, a nematode of ocelots and opossums, destroyed the brain of a 14 year old boy in Brazil, in 1986!

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 Guillain-Barré syndrome, or on the neurology of rheumatoid arthritis, Sjögren syndrome, polyarteritis 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 vu depuis longtemps*", especially in a section describing how "lysates from the amebocytes of the horseshoe crab (*Limulus polyphemus*)" currently assist in the identification, in the CSF, of endotoxins produced by Neisseria meningitidis, Haemophilus influenzae and other gram-negative bacteria.

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Surgery of the Sellar Region and Paranasal Sinuses. Edited by M SAMII. (Pp 583; Price DM 398.–). 1991. Heidelberg, Springer-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 disciplines to consider the pathology, diagnostic procedures, 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 on a variety of pathologies in this most 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 variety of surgical disciplines provide information on a number of approaches to the various pathologies around the sellar and paranasal sinuses; and, it is inevitable 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 patients' outcome and what are the likely future surgical developments in this region of the