Matters arising


Swash replies:

Wilson suggests that damage to the peri-patellar nerve plexus could account for the defective movement sensation I have described after open lateral meniscectomy. This defect in sensation is not accompanied by any discernible change in static sense of position in the joint, although there is an associated alteration of motor control. These features are difficult to explain by dysfunction of any single class of sensory receptor. The concept that interruption of nerve pathways in the peri-patellar plexus could result in interference with the accession of afferents emerging from intra-articular receptors, and with secondary afferents from muscles acting across the knee, is attractive and may be susceptible to experiment by local anaesthetic blockade of this plexus. These matters are important not only for theoretical reasons, but in the management of sports injuries, and in the understanding of falls in elderly persons with osteo-arthritisic joints.

Reference


Alternating unilateral jaw spasm due to metoclopramide

Sir: Thompson et al. described eight cases of unilateral jaw spasms of which three were masticatory in nature. Three of the eight cases were painful. None of the eight cases was drug induced. The following case illustrates once again that drug induced movement disorders often mimic the naturally occurring ones, even those that are rare.

A 66 year old woman was seen because of painful jaw spasms which began 2 hours previously. She described having spasms on each side in a seemingly random pattern. In the emergency room, she was witnessed by other observers to have two episodes of involuntary right jaw spasm, each lasting about 5 minutes. When I examined her no neurological abnormality was found and she had full range of motion of the jaw without any pain. She could not induce a spasm. After approximately 10 minutes she developed left masseter spasm with the jaw deviated mildly to the left. In addition, there was a mild dystonic contraction of the orbiculari oris. During this time speech was dysarthric due to jaw clenching and the patient complained of severe pain. A repeat neurological exam was otherwise normal. There was no other evidence of dystonia and movements in other body parts did not exacerbate the jaw spasm. Intravenous diphenhydramine 50 mg was given and the episode abated over 1-2 minutes. Diphenhydramine 50 mg was given orally in addition at that time and thrice daily for the next 3 days. There were no recurrences.

The patient had taken metoclopramide 10 mg tid for one month, ending one month before this episode, along with librax (chloridiazepoxide hydrochloride 5 mg, and clidinium bromide 2.5 mg) two tablets per day. Two days before the spasms she had resumed metoclopramide 10 mg tid and the day before she had stopped the librax. Other medications included macrodantin, sul-famethoxizole, trimethapham for a urinary tract infection and amiloride 5 mg hydrochlorothiazide 50 mg (Moduretic) for hypertension. Her past medical history included an ileal resection for bowel infarction, spastic colitis, hypertension, total abdominal hysterectomy and bilateral salpingoophorectomy for endometrial carcinoma 16 years previously, cholecystectomy and appendectomy 48 years previously, kidney stones and recurrent urinary tract infections. She stated that she had suffered similar jaw spasms approximately 18 years before but she could not recall any details. For several years, she had had no neurological symptoms and between episodes of jaw spasms her neurological examination was normal. She had no jaw or dental problems. The onset of the syndrome in close association to starting metoclopramide, a dopamine blocking agent, stopping the anticholinergic agent clidinium hydrochloride and the striking relation to diphenhydramine make the diagnosis of an acute dystonic reaction as certain as possible. The normality of her post-ictal examination also supports this.

This case is unusual for several reasons. As discussed in the paper by Thompson et al., unilateral jaw spasms are themselves rare. Acute dystonic reactions, like primary dystonia is quite symmetric when it involves the jaw. Acute dystonic reactions are rarely painful. Oftentimes acute dystonic reactions can be overcome, at least temporarily, by volition, although once the conscious focus is lost the dystonia re-emerges. Acute dystonic reactions tend to last for hours if untreated. Finally, an intermittent alternating dystonia is (I think) unreported. An explanation for this extraordinary reaction is not apparent.

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Reference


Pseudotumour cerebri with amiodarone

Sir: The letter by Fikkers et al prompts us to report a similar case of pseudotumour cerebri in a patient taking amiodarone.

A 52 year old man with Wolff-Parkinson-White syndrome was treated by amiodarone because of recurrent exercise-induced paroxysmal tachycardia. In November 1985 the dose was raised to 400 mg/day, five days a week. In January 1986 he noticed gradual loss of vision in the left eye; he felt tired and irritable. There was no headache, nausea or vomiting. The neurological examination on 3 February 1986 showed bilateral papilloedema; there were no focal or lateralising signs. The general examination was unremarkable. The blood pressure was 140/85 mm Hg and the electrocardiogram showed a sinus rhythm. Ocular examination revealed corneal deposits typical of amiodarone keratopathy. Visual acuity and colour vision were normal. There was a partial field defect in the nasal inferior quadrant of the left eye, confirmed by computerised perimetry. Pattern-shift visual evoked responses were within normal limits. Fluoroangiography showed dilatation of the peripapillary capillaries and increased fluorescence beyond the edge of the papill on the late views, consistent with bilateral papilloedema.

A CT scan of the brain and orbitae was normal. A dural sinus occlusion was excluded by a normal intravenous digital subtraction angiography of the intracranial vessels. Blood and urine tests were normal. At lumbar puncture, the opening pressure
was 175 mm H2O; the analysis of the cerebrospinal fluid was entirely normal (protein content 31 mg/dl with normal agar-electrophoresis). There was no evidence of sinusitis or otitis.

A diagnosis of pseudotumour cerebri was made. A possible side effect of amiodarone was suspected; it was the only drug taken by the patient. A query to the manufacturer (Labaz-Brussels) yielded no mention of previous similar reports. Nevertheless, we decided to withdraw the drug. The patient was put on verapamil 120 mg/day. During the following weeks the papilloedema gradually resolved. A repeat fluoangiography on 11 April 1986 showed disappearance of the oedema of both optic discs. The visual field defect was also reduced.

Our observation supports the view that amiodarone can be responsible for the development of pseudotumour cerebri. As in the case of Fikkers et al, there was a close temporal relation between the introduction and withdrawal of amiodarone and the appearance and resolution of the papilloedema.

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Reference
1 Fikkers BG, Bogousslavsky J, Regli F, Glasson S. Pseudotumour cerebri with amiodarone.  

Book reviews

Cerebrovascular Surgery Volumes 1 and 2  
Edited by JM Fein, ES Fliam (Vol 1 Pp 300; $96.90 Vol 2 Pp 300; $96.90.) Berlin:  

This short monograph, at one DM per page, attempts to help the reader to increase his clinical yield from CT scanning in stroke. Prof Zulch has selected a large number of good quality CT brain scans to illustrate the abnormalities to be expected when infarcts occur in the territories of individual arteries. There is a very useful section, well illustrated by line drawings, on the pathogenesis of stroke explaining how the site and size of an infarct is determined. The roles of arterial stenoses or occlusions, the type and quality of an anastomoses and the influence of general haemodynamic state are described in a straightforward and practical manner.

In most cases, the CT scans are paired with coronal pathological slices of similar lesions. The value of these combinations would have increased considerably if the pathological slices had been in the same plane as the accompanying CT scan.

The text is at times rather too brief and on occasion too personalised. The rather insular English reader may find the large number of references to the German literature somewhat vexing. In general the book succeeds in its aim and after its perusal, the reader should have far more insight into the causes of pathological changes underlying the lesions seen on CT scanning. This is timely in view of the rapid decline in frequency of detailed conventional cerebral angiography in cerebrovascular disease.

D J Thomas

Diagnosis and Treatment of Global Aphasias.  
Clinical Updates on Speech and Pathology Series. By Michael Collins. (Pp 196; £19.00.)  

Part of the Clinical Updates in Speech-Language Pathology Series, this book is written primarily for speech therapists. The authors’ aim is to consolidate current knowledge about global aphasia by drawing on a variety of sources and they divide the book logically into chapters of the definition of aphasia, assessment procedures and treatment and concludes with case illustrations. There is a clear reference section at the end.

D L McEllan

Matters arising

The information sources are cited with little critical appraisal. The treatments outlined are based more on American than British models and are lacking in details required by speech therapists. Some speech therapists might find the title Global Aphasia misleading since the assessment of treatment procedures is at such a high level of complexity as to be more appropriate for moderate to severe aphasias. The text provides a useful overview for students and a good source of references but the student would have to search elsewhere for a more detailed account of treatments.

D Neary


This is a collection of papers, many rewritten and updated, given by invited speakers at a symposium on Feedback and Motor Control held at the University of Glasgow in July 1984. There are seven main sections, the first six of which present data from animal experiments ranging from molluscs to mammals. The section titles are motor system organisation, central control of sense organ excitability, sensory input during movement, the role of reflexes, the control of movement, the control of equilibrium, and feedback and motor control in man. Each section is given a reasonably substantial introduction that puts its contents in context and points up controversial issues. Indeed, many of the papers themselves incorporate reviews of previously published work in addition to the author’s own recent observations.

This book makes fascinating reading. It is a useful source of references; its reasonable cost has been achieved by the use of reduced typescript but most of the figures have clearly been chosen so that they will reproduce well. Those interested in motor control will want their own copy to browse through, and it is ideal for the general scientific reader wanting to catch up with the latest observations, thinking and controversies about sensorimotor integration and motor control.

The editors are to be congratulated on a symposium publication that is not simply a momento of a pleasant occasion but a valuable record of current scientific thinking on a topic of major importance.

D L McEllan
Pseudotumor cerebri with amiodarone.

M Van Zandijcke and A Dewachter

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