presence of neoplasms or hyperplasia involving endocrine glands. The organs most often affected are the parathyroids, pancreatic islets and pituitary gland. Occasionally neoplasms of the thymus gland have been reported in this syndrome.\(^\text{24}\) Myasthenia gravis is frequently associated with thymic hyperplasia and approximately 10–15% of the patients have a thymic tumour.\(^\text{1}\) Our patient presented with a thymic gravis had a thymoma and characteristic features of MEN-1 syndrome.

A previously healthy 28 year old male was admitted to hospital with a six weeks history of increasing dyspnoea, problems with mastication and nasal regurgitation, followed by diplopia. There was a progression of symptoms during the day and after exertion and reduced after rest. The patient’s mother had died in the past because of hyperparathyroidism.

Physical examination showed a slightly overweight man (height 1.72 m, weight 85 kg) with little facial hair. Internal investigations of the pituitary organs were normal. Neurological investigation revealed slight dysarthria, bilateral ptosis, facial weakness and paresis of abduction and elevation of both eyes. These symptoms fluctuated, depending on the degree of exertion. Leucocytosis was not detectable. The patient was intubated because dyspnoea was considered to be improved by pyridostigmine 60 mg four times daily, and bromocriptine 2.5 mg twice daily.

This case represents a combination of myasthenia gravis, a benign thymoma, hyperparathyroidism and a pituitary tumour, probably a prolactinoma. The coexistence of hyperparathyroidism and a pituitary tumour is characteristic of MEN-1 syndrome. The evolution of this syndrome may takes years and it is inherited as an autosomal dominant trait with a very high degree of penetrance.\(^\text{2}\) The mother of our patient also had hyperparathyroidism. Less common findings reported in patients with MEN-1 syndrome included lipoma, carcinoid tumours, thymic tumours and pinealoma.\(^\text{3}\) The neoplasms of thymic origin mainly composed of carcinoid tumours with a relatively poor prognosis.\(^\text{4}\) None of these cases had myasthenia gravis.

Only one patient has been described with the combination of myasthenia gravis, thymoma and hyperparathyroidism; sporadic forms of the MEN-1 syndrome was suggested by the author.\(^\text{1}\) Moreover, our patient had a pituitary tumour which underlines the diagnosis of MEN-1 syndrome. The insulin level and insulin/glucose ratio were slightly elevated, these values, however, did not prove to be a pancreatic islet tumour. Perhaps the thymoma was responsible for this disturbance as the tumour cells reacted positively with antibodies to insulin and the value returned to normal postoperatively.

The importance of this remarkable finding is not clear, but cell surface receptors for some polypeptide hormones have been mentioned in thymoma cells.\(^\text{5}\) This reaction was negative in five other thymomas that we have investigated in recent years. The association of tumours of the thymic and pancreatic gland is not surprising as these structures have a common embryonic origin from the third pharyngeal pouch.

Matters arising

Korsakoff’s psychosis in the presence of multiple sclerosis: an unusual cognitive state

After the publication of our letter in your journal, some inaccuracies were brought to our attention by Professor E K Warrington in whose department the patient was tested. The difference between the WAIS IQ and the estimated premorbid IQ (NART) test indicates a decline in the performance on non-verbal tests rather than general intellectual ability as stated in the article. A score of 19/30 in the McKenna and Warrington Graded Naming Test is within the average range and not indicative of nominal dysphasia. The statement: “in both Korsakoff’s syndrome and multiple sclerosis verbal IQs decline more than performance IQs...” is erroneous and it would be correct to say that the performance IQ is more likely to deteriorate.

Finally, although not an inaccuracy, the Camden Memory Tests quoted in the letter are still in the process of development and low scores should be interpreted as corroborating poor performance on the standard memory tests. These corrections in no way detract from the conclusions of our letter and we feel that it is worth highlighting the need to search for other pathologies in patients with alcohol related brain damage if their clinical presentation is in anyway unusual.

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Magnetic resonance imaging in patients with progressive myelopathy following spinal surgery

The explanation by Mr Adams’ of the postoperative myelopathy with a biomechanical mechanism producing postoperative traction of the dura and the spinal cord during cervical movements is an adjunct to our paper; in

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which merely an observation was made, rather than an explanation. We agree that the biomechanical mechanism can explain the progressive myelopathy, beginning late in the post-operative course. However, it is hard to believe that myelopathy beginning five years or more following surgery can be caused by arch-noiditis. On the other hand, the possibility of spinal cord injury during operation is unlikely. The biomechanical mechanism of postoperative myelopathy, so well described by Mr Adams, can explain that the myelopathy develops more slowly after surgery of spinal stenosis and disc disease, rather than after intramedullary surgery.

We also agree with the great importance given by Mr Adams to the careful preoperative evaluation of the candidates for cervical spine surgery by conventional radiography. In addition, we advise pre- and postoperative magnetic resonance imaging (MRI) examinations of these patients. MRI has the advantage of demonstrating the spinal cord and its relation to the bony spinal canal, and is easier to recognise the problems of the postoperative spine and spinal cord. Further experience in this field is necessary to solve these problems.

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BOOK REVIEWS


Dr Gunderson is chief of the Neurology Service at Walter Reed Army Medical Centre in Washington DC. In the preface to his new publication the author explains how it was that "brief handouts" to help his students became expanded over a period of 20 years to become, in 1982, his first book entitled Quick Reference to Clinical Neurology (J B Lippincott Co). The Essentials of Clinical Neurology represents a further expansion and revision of that work.

Dr Gunderson's stated aim is to avoid a book that espoused "to become encyclopaedic" but he hoped, rather, to "restrict the material" to that which he "would like every medical student to have been exposed to during a four-week clinical neurology rotation". (I presumed this means by this "heard about" rather than "seen" because the subject index includes reference amongst others to such things as acid, malaise in the 19th century, and Hogolhenst plaques.) The book is designed for the use of undergraduate, junior house officer or "practising physician" alike. A tall order? I rather think so—the many lists and tables do indeed look like hard hammering on the graduates and I would doubt whether this approach is either comprehensive enough or sufficiently attractive in its literary style for the post-graduate. Indeed, it is rather military—the section on "Physical Examination" in the index—1. The patient should disrobe (!).

Other chapters cover specific complaints (eg, The Dizzy and Dae Patient), management of specific syndromes (Neuropathies and Stroke), for example, and selected diseases (eg Infections and Tumour). Whilst there is an immense amount of valuable information, indeed encyclopaedic in undergraduate terms, unfortunately there are important omissions and, I think, errors. For example, routine examination of the upper limb omits the testing of muscle tone (page 10) and there is no mention of the characteristic pyramidal distribution of upper motor neuron weakness, only that "hemiparesis, quadriparesis or paraparesis" form the usual pattern. Patients who trip over often have spasticity, not "foot weakness" (page 67). Does muscle biopsy (page 48) only "occasionally differentiate" muscle disease from neuropathy? It is stated (on page 255) that "about 25 per cent of patients will have status epilepticus as their first symptom of epilepsy". Surely this is a central spinal centre. The chapter on epilepsy, in fact, raises another problem with the book and that is that old and new nomenclature is mixed side by side.

Chapter 26 (Disorders of Myelin) closes with a few paragraphs on "Brachial Neuritis" (page 474). Nowadays called Neurol Amyotrophy or Shoulder-girdle Neuritis, this disorder was never, as stated, "attributed to the design of the British (aircraft)". It was indeed initially described during the Second World War but the majority of patients were serving in the United Kingdom at the time. Many sufferers were soldiers, but the authors of the original papers all noted the association of the condition with incidental operations, convalescence from infective illness or prophylactic inoculations. The close similarity to "serum neuritis" was also noted. The possibility that this disorder was an immune based demyelinating disease was recognised very early on—not recently, as implied. I don't think the condition ever spreads, as stated, to a leg, although there is a complementary form of radiculopathy of "ido-pathic" variety affecting the lumbo-sacral outflow.

I doubt whether the style of this book will attract many students or junior doctors in the UK. There is too much for the undergraduate, and the book is not sufficiently comprehensive or accurate for the discerning postgraduate. A reader would be irritated by the numbering and lettering of every paragraph. I think they are more likely to be encouraged by the good selection of more attractive, compact text books of this kind already available. Further references have to be made to the larger comprehensive text.

J A SPILLANE


Patients with spinal injuries have only been kept alive since the development of spinal centres in 1944 at the end of the Second World War. Initially only paraplegic patients survived with the most serious centres initially and bidity and mortality decreased and paraplegic patients began to experience a nearly normal life expectancy. Initially few tetraplegic patients were admitted to spinal centres and those that were, died soon after injury. However, following the same principles tetraplegic patients were kept alive and since 1980 even those on ventilators have been kept alive and discharged home. In the rehabilitation of high quadrigriplesia the United States is ahead of the United Kingdom and this book reports the combined experience of centres that are leading the work in this field. The Craig Hospital, San Diego Valley Medical Center and the Institute of Rehabilitation and Research (Houston, Texas) have information on 216 individuals who have been discharged home, many of whom support the others, an extraordinary record of achievement.

The priority in patient care in the United States differs from that in the United Kingdom in that patients are admitted to neurological and neurosurgical centres initially and only some weeks later come to rehabilitation centres; thus the authors' experience of the initial treatment of spinal injury is limited—in the United Kingdom patients are admitted immediately after injury. The book is split into four sections, Acute Care, Rehabilitation, Discharge Planning and The Real World. Unfortunately the chapters on acute care do not deal with the associated complications found in some 75% of cases, the management of head injuries, the problem of hyperalcaemia and temperature regulation. In mentioning the various forms of amputations, the authors state that the patient should be anticoagulated at 1-5 times the control level, a view that would not gain universal acceptance.

Also, the description of a teardrop fracture being due to a small anterior fracture is intriguing. "The Radiograph may show a posterior displacement of the superior vertebral body on the inferior one, but more often one sees a separation of the disk space at one motion segment or an avulsion fracture of the anterior superior part of the inferior vertebral. This is called a teardrop fracture. A teardrop fracture is a crush fracture with extrusion of fragment. On the other hand the chapters by Carter on Available Respiratory Options, Anderson on Psychological Issues, the chapters on the medicolegal aspects and the whole approach to discharge home, are excellent. The chapter on the long term outlook, though short, is an extraordinary tribute to care whereby after nine years 63% of their initial group of respiratory dependent patients are still alive. This is an exceptional piece of work.

The whole book is fragmented with respiration management dabbled about in different places. It does not follow conventional presentation and there is no discussion of the fundamental problems of chest wall movement, postural problems associated