

Book reviews

Case histories for the MRCP, Paul Goldsmith, pp 296. Butterworth Heinemann, 1998. £15.99, paperback. ISBN 0 7506 3868 0. ****

The MRCP exam is the gateway to a consultant job in medicine. It is one of the most coveted achievements in the life of a junior doctor. The pass rate is low and it is usually attained after much hard work and sweat. The College has now removed one of the obstacles which is the limit of only six attempts at Part 2. However, candidates are still going to the exam as well prepared as possible because early success prevents unnecessary expense and enhances self-esteem.

There are already a large number of revision aids available in the bookshops which has enabled the standard of preparation for the exam to rise. However, as the College inevitably will try to beat 'the market' by introducing different styles and questions, then so the market must try to keep one step ahead of the college. This new book of case histories for the MRCP does indeed provide a much-needed source of high quality, challenging, case histories. The case histories are renowned to be the trickiest part of the written paper and usually include some obscure conditions not routinely seen in clinical practice. This book covers many of these including von Hippel Landau, Buruli ulcer and Lemierre's disease to name just a few – I was able to educate myself on the latter condition.

The book's great asset is the long explanations given with the suggested answers that

remove the need to look up unknown conditions in major reference books. Just enough information is given. Each set of 10 cases provides an ideal mock exam with a good range of everyday and lesser known medical problems. There are one or two minor spelling errors which is inevitable in any text.

The book has been shown to local juniors who found it difficult but useful. The scoring system could be more 'upbeat' in order to boost morale and the presentation is somewhat 'boring'. Perhaps just a few illustrations/X-rays/ECGs might have relieved it. It is really of personal preference whether all the answers should be at the end of the text or whether they should be given at the conclusion of each set of histories.

This is, as it is meant to be, a practical and functional presentation which I am sure will be of great help to the next generation of those aspiring to acquire MRCP.

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Microbiology in clinical practice, 3rd edn, D C Shanson, pp 500. Butterworth Heinemann, 1999. £29.99, paperback. ISBN 0 7506 3110 4. **

The book is targeted at medical students and junior medical staff and comprises three broad areas: laboratory microbiology and antimicrobial therapy, clinical conditions, and infection control. Now in an enlarged

format, the contents will be familiar, as much of the text, tables and illustrations are similar to the last edition. As before, there is a compromise between systematic microbiology (genus by genus) and system-based clinical microbiology (organ by organ) which inevitably leads to some duplication. The rather short section on *Clostridium difficile* disease, for example, appears in both the chapter on anaerobic infections and that devoted to gastrointestinal disease. Someone looking for a review of the microbiology, clinical features and treatment of a specific disease will therefore need to dip into several different sections of the book.

There are several new and expanded sections, notably an excellent new chapter on molecular biology and additional sections on herpes viruses, hepatitis and HIV infection. Several newer topics such as variant Creutzfeldt-Jakob disease have been added. However, the overall impression is that much of the original text needed more thorough revision. Microbiologists madden their clinical colleagues by constantly renaming microorganisms but the taxonomy here is both substantially out of date and inconsistent. Sections on 'more recently recognised infections' and some of the antimicrobial advice also look very dated. There are now several competing books around this price which have a clinical focus more in line with current teaching, but this edition will still find a place on the library shelf.

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Letter to the Editor

New weakness in a critically ill patient

Sir,

In a paper published in the May issue of your Journal, the authors describe a patient who was ventilated for acute severe asthma and who subsequently developed a profound weakness thought to be due to a myopathy.¹ The authors attribute this to 'critical illness myopathy', and although in the discussion they mention that corticosteroids might have played a role in previous cases, they do not discuss whether this may have been a contributory factor in their own patient.

The patient was originally treated with intravenous hydrocortisone 800 mg/day, increased to 2.4 g/day when the patient was transferred to the Intensive Care Unit, although the authors do not state for how long the patient received this dose. I have previously described four similar patients who developed a severe 'hydrocortisone myopathy', and reviewed the risk factors for development of steroid myopathy.² I found that these patients had usually had higher doses of hydrocortisone than non-affected patients, the former having received a total dose of more than 5.0 g of hydrocortisone, and the latter less than 4.0 g. I also speculated that muscle paralysis played a role in the development of this syndrome. Since writing that paper, I have no longer paralysed these patients when ventilated (relying on sedation instead), and have rigorously restricted the total dose of hydrocortisone. I have not subsequently seen a case of severe myopathy in a ventilated asthmatic.

Surprisingly, there is no clear-cut evidence for corticosteroids being of benefit once acute severe asthma has supervened, but their use is generally advised, and I do so routinely. I would question the rationale for using up to 2.4 g/day of hydrocortisone, as this is roughly

equivalent to 600 mg of prednisolone, and I know of no dose-response studies suggesting that such large doses have additional benefits over 200 mg/day of hydrocortisone (roughly 50 mg prednisolone).

It would be interesting to know the total dose of hydrocortisone received by this patient during the course of the acute illness. I suspect that a lot of this patient's weakness, rather than being due to 'critical illness', was actually due to enormous amounts of corticosteroids against a background of prolonged neuromuscular blockade.

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- 1 Craig JJ, Mirakhor M, Sawhney BB, Patterson VH. New weakness in a critically ill patient. *Postgrad Med J* 1999;75:301-3.
- 2 Shee CD. Risk factors for hydrocortisone myopathy in acute severe asthma. *Respir Med* 1990;84:229-33.

This letter was shown to the authors who responded as follows:

Sir,

The asthmatic woman we described who developed severe myopathic weakness during a period of mechanical ventilation received 5.4 g of hydrocortisone in total. As Shee postulates, our patient's weakness was most likely the result of high-dose glucocorticoids on a background of prolonged exposure to muscle paralyzing agents. We feel, however, that this condition should not be called 'hydrocortisone myopathy', as Shee refers to it, because a similar condition has been described with low-dose glucocorticoid exposure.¹ We have also found this to be the case, and in fact out of the seven cases of acute myopathy in criti-

cally ill patients that we have seen over the last two years, two patients received only 160 and 200 mg of hydrocortisone in total, and one patient had no exposure to glucocorticoids at all. These three patients all had similar findings on electrophysiological studies and muscle biopsy as described for critical illness myopathy. We therefore concur with Ruff that this condition is the result of a heterogeneous range of clinical insults, although usually two of the following three conditions are present:

- the patient is treated with a non-depolarizing blocking agent
- glucocorticoids are used
- the patient has a severe febrile illness or sepsis.²

Based on this information, Shee's practice of restricting the dose of paralyzing agents and glucocorticoids administered in these patients is therefore probably best practice at present.

In conclusion, we would reiterate that the myopathy occurring in this patient group is only one of many other possible causes of weakness, some of which are amenable to therapeutic intervention, and all of which can be difficult to separate purely on clinical grounds. Appropriate investigations must therefore be performed to be able to advise accurately on diagnosis and prognosis for these patients.

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- 1 Gutmann L, Blumenthal D, Gutmann L, Schochet SS. Acute type II myofiber atrophy in critical illness. *Neurology* 1996;46:819-21.
- 2 Ruff RL. Acute illness myopathy. *Neurology* 1996;46:600-1.

International Postgraduate Diary

Queen Charlotte's & Chelsea Hospital, London

13 December 1999: Infant nutrition perspectives (study day)

Details: Course Registration Service, PO Box 3219, Barnes, London SW13 9XR, UK. Tel + 44 181 741 1311; fax +44 181 741 0611; e-mail: CourseRegs@aol.com

University of Warwick Short Courses

13–16 December 1999: Techniques and applications of molecular biology

Details: Dr Charlotte West, Department of Biological Sciences, University of Warwick, Coventry CV4 7AL, UK. Tel + 44 1203 523540; fax + 44 1203 523701; e-mail: ca@dna.bio.warwick.ac.uk

Columbia University College of Physicians and Surgeons, New York

3/4 December 1999: Update in gastroenterology, hepatology & nutrition

26 April 2000: 15th Annual schizophrenia conference

5/6 May 2000: 12th Annual orthopaedic trauma course. Current techniques in upper and lower extremity trauma

22–25 May 2000: 4th Annual conference. Botanical medicine in modern clinical practice

Details: Center for Continuing Education, Columbia University College of Physicians and Surgeons, 630 West 168th Street, Unit 39, New York, NY10032, USA. Tel + 1 212 781 5990; fax + 1 212 781 6047; e-mail: cme@columbia.edu

Barrow Neurological Institute, Phoenix, AZ, USA

2–4 March 2000: 26th Annual symposium: recent advances in neurosurgery

5–7 March 2000: 26th Annual symposium: What's important? What's new? Neurology/neuroimaging

Details: Neuroscience Conference Coordinator, Barrow Neurological Institute, 350 West Thomas Road, Phoenix, AZ 85013, USA. Tel +1 602 406 3067; fax +1 602 406 4104; email deskildson@the BNI.com

Falk Symposia

27/28 January 2000: Chronic hepatitis: new concepts of pathogenesis, diagnosis and treatment (Cologne, Germany)

18–24 February 2000: VIII Gastroenterology week (Titisee, Germany)

4–6 May 2000: Hepatology 2000 (Munich, Germany)

Details: Falk Foundation eV—Congress Division, Leinenweberstr 5, PO Box 6529, D-79041 Freiburg, Germany. Tel +49 761 130340; fax +49 761 1303459; email: symposia@falkfoundation.de