Letters to the Editor

Nasal ventilation

Sir,

In a paper in the June issue of this journal,¹ the author mentioned briefly the “crucial recognition” that continuous positive airway pressure (CPAP) “is effective therapy for obstructive sleep apnoea”.

Although CPAP is a well-established management option for sleep apnoea, its limitations and lack of universal acceptance should be borne in mind. Other modalities of treatment should also be considered when appropriate. Definition of sleep apnoea, determining its severity, and management options remain unclear to many healthcare providers. Sleep apnoea is the periodic cessation (apnoea) or reduction (to 50% of tidal volume or less) of air flow at the nostrils and mouth during sleep. The number of apnoeic-hypopnoeic episodes per hour of sleep (called the apnoea-hypopnoea index, or AHI) is used to indicate the severity of the syndrome (mild: 5–20, moderate: 21–50, severe: >50). It is usually caused by nasopharyngeal narrowing or collapse (peripheral or obstructive sleep apnoea) and much less commonly due to disturbance of the neurological control of respiration (central sleep apnoea). It is thought that obstructive sleep apnoea (OSA), the more common type of sleep apnoea, affects 2–4% of males and 1–2% of females in middle age.

The syndrome presents clinically with observed apnoea, disturbed sleep and, in OSA, loud snoring. Daytime somnolence is a significant part of the syndrome² and is used with other parameters to indicate its severity. OSA is also associated with cognitive impairment and increased susceptibility to road traffic accidents.³ Claims that OSA is a cause of ischaemic heart disease, cerebrovascular accidents and pulmonary hypertension are based on very weak evidence.⁴ Risk factors include obesity, male gender, alcohol ingestion, nasal obstruction and retrognathia. The most common cause of paediatric OSA is adenotonsillar hypertrophy.

History and clinical examination are aimed at identifying the syndrome components, severity, causes and associated risk factors. The severity of the syndrome may be assessed from a parents' or a partner's account or a tape recording of breathing during sleep. The Epworth Sleepiness Scale is being used by some units to quantify the problem. This scale assesses daytime tendency to doze in eight different situations and, indirectly, measures the quality of night-time sleep. The chance of dozing is scored as slight (1), moderate (2), or high (3). The situations covered are: watching TV, sitting inactive, sitting and reading, sitting and talking to someone, sitting quietly after lunch without alcohol, lying down in the afternoon, as a passenger in a car for an hour without a break and in a car whilst stopped for a few minutes in traffic. Some hospital units have restricted management of sleep apnoea in adults to cases with Epworth Sleepiness Scale scores of 12 or more.

At this point, it may be obvious that a specific cause is responsible, eg, nasal obstruction, large tonsils, long redundant soft palate, small receding mandible or other craniofacial abnormalities. It would seem logical to address the obvious cause(s) directly by surgery, orthodontic devices or weight reduction as appropriate.

Polysomnography is the overnight sleep study used to assess different parameters related to OSA syndrome. Other versions of the investigation include pulse oximetry and home sleep study. Polysomnography is resorted to if the diagnosis needs to be confirmed, if central apnoea is suspected, if there are severe symptoms with respiratory or cardiovascular complications, and if there are contraindications to, refusal or failure of other options, such as surgery. In other words, when the management choices have been narrowed to the use of CPAP. Other uses for polysomnography include pre-operative assessment if post-operative failure to restore spontaneous breathing is anticipated. In such cases, arrangements for post-operative assisted ventilation in the intensive care unit should be made. Polysomnography is also used to monitor the efficacy of a certain modality of management.

Polysomnography consists of simultaneous measurement during sleep of eye movements (electro-oculogram), electro-encephalogram, electrocardiogram, chest and abdominal respiratory movements, nasal and oral air flow and oxygen saturation with finger or ear lobe oximeter. The following information is available from the study:

- the AHI
- the drop in oxygen saturation during the apnoeic/hypopnoeic episodes. This is also used to grade the syndrome, ie, >85%: mild, 80–85%: moderate, <80%: severe
- duration of periods of oxygen desaturation
- sleep efficiency, ie, the proportion of the study spent asleep
- associated cardiovascular complications, eg, arrhythmias.

CPAP ventilation during sleep is the treatment option in severe cases of obstructive sleep apnoea and in less severe cases if other modalities of treatment have failed, been rejected or are contraindicated. Central apnoea may be managed more effectively with intermittent rather than continuous positive airway pressure ventilation.⁵ Positive pressure ventilation acts as a pneumatic splint for the airways. Air is generated by a ‘blower’ and is delivered by means of a tight fitting mask placed on the nose and mouth. The appropriate pressure, between 7–15 cmH₂O, is determined during a night stay in a sleep laboratory.

CPAP is effective in improving the AHI and the oxygen saturation but has the following disadvantages: life-long treatment, cumbersome noisy machine, uncomfortable mask, nasal and pharyngeal irritation, sore eyes from leakage around the mask, abdominal bloating from leakage into the oesophagus, a feeling of claustrophobia experienced by many patients and overall compliance of 50–90%. New developments include customised masks, smaller quieter machines and nasal BiPAP (bi-level positive airway pressure) in which the pressure is reduced during expiration thus improving comfort and compliance.

Surgery may be used if a specific obstruction is detected in the nose or pharynx during clinical or endoscopic examination under simulated sleep induced by light general anaesthesia (sleep naso-endoscopy). Surgical management for OSA aims to relieve the obstruction by increasing the surface area, to bypass the pharyngeal airway, or to remove a specific pathological lesion. Papers on the

outcome of surgery are usually based on reporting particular procedures and/or related modifications rather than on randomised controlled trials.

The following are some of the surgical procedures and their indication or rationale:

- adeno-tonsillectomy for adenoid and tonsillar hypertrophy
- uvulo-palato-pharyngoplasty (UPPP) which consists of excision of the uvula, redundant part of the soft palate and the tonsils in case of obstruction caused by these structures. Modifications and improvements to this procedure include laser-assisted uvulo-palatoplasty (LAUP)
- nasal surgery to relieve nasal obstruction caused, for example, by nasal septal deviation, polyps or large inferior turbinates
- tracheostomy to bypass pharyngeal obstruction
- inferior sagittal mandibular osteotomy and genio-glossal advancement with hyoid myotomy and suspension (GAHM) to create an enlarged retro-lingual airway
- laser mid-line glossectomy and linguoplasty to create an enlarged retro-lingual airway
- maxillo-mandibular osteotomy and advancement to enlarge both retro-lingual and retro-palatal airways
- epiglottoplasty in selected cases of laryngomalacia.

Mandibular advancement prostheses (oral appliances), provided by an orthodontist, are worn at night to pull the mandible and tongue base forward if the oropharynx is narrowed by retrognathia or tongue base hypertrophy. They are less effective than CPAP in improving the AHI and oxygen saturation but are better tolerated and accepted by patients.⁶

Conservative measures include weight loss, medications to relieve nasal obstruction, modification of sleep position, and avoidance of evening alcohol and hypnotics.

In conclusion, effective management of sleep apnoea will depend on careful assessment of its severity, identification of its causes and risk factors and awareness of the available management options.

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- 1 Simonds AK. Nasal ventilation. *Postgrad Med J* 1998;74:343–6.
- 2 Douglas NJ, Polo O. Pathogenesis of obstructive sleep apnoea / hypopnoea syndrome. *Lancet* 1994;344:653–5.
- 3 Royal College of Physicians. *Sleep apnoea and related conditions. A report of working party.* London: Royal College of Physicians, 1993.
- 4 Wright J, White J. The effectiveness of continuous positive airways pressure for the treatment of obstructive sleep apnoea. (Cochrane Review). In: *The Cochrane Library*, Issue 3. Oxford: Update Software, 1998.
- 5 Ferguson KA, Ono T, Lowe AA, Keenan SP, Fleetham JA. A randomised crossover study of an oral appliance vs nasal continuous positive airway pressure in the treatment of mild–moderate obstructive sleep apnoea. *Chest* 1996;109:1269–75.
- 6 Thommi G, Nugent K, Bell GM, Liu J. Termination of central sleep apnoea episodes by upper airway stimulation using intermittent positive airway pressure. *Chest* 1991;99:1527–9.

Time delay to thrombolytic therapy

Sir,

In their informative article, Constantine and Thenabadu¹ identify reasons for delay in initiating thrombolysis, including failure to diagnose acute myocardial infarction at the time of admission. This resulted from a lack of specific electrocardiogram (ECG) changes or a failure to recognise them. This concurs with previously published observations regarding diagnostic difficulty when the admitting physician is faced with a patient with prolonged chest pain, a history of ischaemic heart disease and previous myocardial infarction with suspicious but equivocal ECG changes.² On other occasions, the history is atypical but an ECG suggests infarction. Uncertainties in deciding whether chest pains are due to unstable angina or infarction inevitably delays thrombolysis. Comparison with a previous ECG can be helpful in establishing a correct diagnosis.

A further proposal to reduce in-patient delay for those patients being admitted for the second or subsequent time would be to issue at-risk patients with their ECG and to encourage them to carry them at all times. Immediate comparison would facilitate the detection of fresh ECG changes, removing diagnostic doubt and thereby allowing appropriate treatment to be efficiently administered.

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Accepted 3 December 1998

- 1 Constantine GR, Thenabadu PN. Time delay to thrombolytic therapy - a Sri Lankan perspective. *Postgrad Med J* 1998;74:405-7.
- 2 Varma S, Large GA. Patients from coronary care units should be given their electrocardiograms and carry them at all times. *BMJ* 1997;314:1764-5.

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

Sir,

We appreciate the response to our paper on time delay to thrombolytic therapy. The proposal to reduce in-patient time delay by encouraging high-risk patients to carry their ECG at all times is important. However, our experience has been negative. Introducing a 'risk score' based on ECG, echocardiography and simple electrophysiological interpretation like duration of loss of sinus arrhythmia may be more practicable in our context.

Troponin I and troponin T are highly sensitive and specific markers of myocardial necrosis. Recently, Hamm *et al*¹ showed that troponin-negative patients were at low risk for myocardial events after an episode of acute chest pain. Troponin T and troponin I may become important markers in the management of patients with acute chest pain in the future.

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- 1 Hamm CW, Goldmann BU, Heeschen C, Kreyman G, Berger J, Meinerz T. Emergency room triage of patients with acute chest pain by means of rapid testing for cardiac troponin T or troponin I. *N Engl J Med* 1997;337:1648.

Can combined defects be diagnosed on spirometry alone?

Sir,

I read with interest the article on hospital doctors' assessment of baseline spirometry, by Stephenson and colleagues, in a recent issue of this journal.¹ It is indeed a matter of great concern that only 12% of the respondents could accurately interpret all the five vitalographs given to them. What is still more disturbing is the fact that the correct interpretation for the third vitalograph was considered as 'combined defect'. This vitalograph, in a 77-year-old man, shows a mild reduction in forced vital capacity (FVC) (63% of predicted) and a severe reduction in both the forced expiratory volume in the first second (FEV₁) and the FEV₁/FVC ratio (30% and 38%, respectively). The volume-time curve provided is also flattened, indicating diminished expiratory flow. All these features are consistent with an obstructive rather than a combined defect. As per standard guidelines on interpretation of spirometry,² an obstructive defect should be diagnosed when the decrease in FEV₁ is out of proportion to any decrease in vital capacity, as in this case. One must resist the temptation of diagnosing a combined defect based on spirometry alone. It is well known that FVC may be reduced in patients with airway obstruction, especially in those with severe airflow limitation. This is largely related to air-trapping and a consequent increase in residual volume (RV). Estimation of RV, total lung capacity (TLC) and RV/TLC ratio is necessary to diagnose such an abnormality and to look for any coexisting restriction.

I therefore believe that, based on the database provided in this vitalograph, one can diagnose only a severe obstructive defect. Additional information is needed before one can consider this defect to combine obstructive and restrictive defects. The results of this study indicate that 50 of 78 respondents incorrectly interpreted this vitalograph, the number of incorrect responses being far more than in any of the other four records. It would be interesting to know the number of respondents who diagnosed an obstructive defect in this record. Such spirometry reports often cause some confusion during interpretation; however, with adequate knowledge of currently recommended interpretative strategies,² one can usually reach a correct conclusion and ask for additional investigations in case of any doubt.

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Accepted 7 December 1998

- 1 Stephenson N, Morgan R, Abdel-Rahman, Abdel-Wahab E, Turnbull CJ. Hospital doctors' assessment of baseline spirometry. *Postgrad Med J* 1998;74:537-40.
- 2 American Thoracic Society. Lung function testing. Selection of reference values and interpretative strategies. *Am Rev Respir Dis* 1991;144:1202-18.

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

Sir,

Dr Aggarwal has some very valid comments to make, including the fact that a diagnosis of a combined defect should, strictly speaking, not be based on spirometry alone and that

further investigations, including measurements of TLC, should be requested.

A restrictive pattern means that lung volumes are small and the primary criteria for this diagnosis is a reduction in TLC. TLC cannot be measured by spirometry but a reduced VC may be used to suggest the presence of restriction. If there is any doubt about the cause of a reduced VC, TLC should be measured.

However, in the context of this study, looking at doctor's interpretation of baseline spirometry with no other information available, we believe it was justified to ask whether the vitalograph showed a combined defect in the differential interpretation. The vitalograph had kindly been provided by the Cardiorespiratory Department as examples of normal, restrictive, obstructive and combined defects and *validated independently* by two respiratory physicians. Figure 3 did show a reduced FEV₁, consistent with an obstructive defect, however, the vital capacity was reduced, raising the question of a combined defect. We appreciate that measurement of TLC would be necessary to confirm this (although not a single participant raised this issue).

We fully concur with the author that, with adequate knowledge, interpretation of spirometry should lead to a correct conclusion. The problem, however, as the study has illustrated, is that hospital doctors do not have adequate knowledge and 72% felt that they had not had adequate teaching. We should not be complacent that doctors can interpret spirometry and address the issue of adequate training and wider dissemination of the current guidelines on interpretation of spirometry.

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Interprofessional learning

Sir,

Parsell and Bligh, in their article on interprofessional learning, describe examples of current practice in the UK, focusing especially on examples involving medical students.¹ The examples quoted are based largely on developing communication skills, and on social and ethical issues. Whilst this style of interprofessional learning experience is well recognised, other methods of shared learning should not be forgotten.

A cardiac arrest draws together acutely all the components of working together as a multiprofessional team. Cardiac arrest team members share a common goal - the successful resuscitation of the patient. The team is multiprofessional, many contain a mixture of doctors, nurses, operating department practitioners, resuscitation officers and others. Team members have had to adjust to the change in the traditional professional boundaries of resuscitation practice. No longer is it the case that the doctor is the only provider of advanced life support skills. In fact, in some trusts, the doctor is no longer the leader of the resuscitation team.² Successful resuscitation is dependent on working together as a team and on sharing and respecting each others individual skills and contributions.

At the University of Birmingham, shared learning is introduced in the first year training

of medical, dental, nursing and physiotherapy students in the form of a resuscitation course. Students are taught in small multiprofessional groups. They are taught by senior students whom have undertaken a resuscitation training instructor course. Emphasis is placed on a problem-solving approach to common emergencies and practical resuscitation skills. Quality control is ensured by external assessment through the Royal Life Saving Society UK (an aquatic rescue and resuscitation training organisation).

The course has been exceedingly well received by the students. Feedback questionnaires reveal that students prefer being taught in multiprofessional groups: 96% enjoyed the course, 94% rated the teaching as excellent or good, and 93% preferred being taught by their senior colleagues rather than clinicians. A remarkable 52.7% requested to receive training to become instructors.

In summary, shared learning focusing on the management of a cardiac arrest has been successfully introduced at the University of Birmingham. The programme has been well received by students and staff. It is an alternative approach to interprofessional learning and should help to facilitate improved working relationships between the professional groups involved.

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Accepted 9 December 1998

- 1 Parsell G, Bligh J. Interprofessional learning. *Postgrad Med J* 1998;74:89–95.
- 2 Williams A. Team leaders at cardiac arrests. *Resuscitation* 1998;37:S29.

Metabolic acidosis

Sir,

The patient described by Darwich and Allaf¹ is unlikely to suffer from simple metabolic acidosis. It is more likely that their patient suffered from a mixed disorder, namely, metabolic acidosis and respiratory alkalosis. This is because, if we calculate the expected

pCO₂ for the degree of hypobicarbonataemia in their patient according to the formula²:

$$pCO_2 = ((1.5)(HCO_3^-) + 8) \pm 2$$

the value we obtain (21.5±2) is significantly higher than the measured pCO₂ of 9.5. A superimposed respiratory alkalosis with metabolic acidosis would explain the low pCO₂ found in their patient.

It would be interesting to know whether an underlying septic process was present that was treated with empirical antibiotic therapy.

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- 1 Darwich MS, Allaf W. Metabolic acidosis. *Postgrad Med J* 1998;74:427–8.
- 2 Narins RG, Kupin W, Faber MD, Godkin DA, Dunfee TP. In: Areiff AI, Defronzo eds. *Fluid electrolyte and acid-base disorders*, 2nd edn. New York: Churchill Livingstone, 1995; pp 105–98.

Prevalence of *Helicobacter pylori* infection in an Albanian population

Sir,

The decline in the incidence of *Helicobacter pylori* infection in developed countries¹ should lead to a reduction in the burden of associated disease, notably peptic ulceration and gastric cancer. Patterns of *H. pylori* incidence are difficult to demonstrate but can be deduced from prevalence studies.

High prevalence of infection with *H. pylori* has been reported in eastern European countries,² where there may be high rates of infection even in younger age groups. This pattern is similar to that in developing

countries, suggesting continued high incidence rates. We report below the results of a small study conducted by the University Hospital Centre, Tirana, Albania, in collaboration with Leicester General Hospital, UK.

Serum samples were obtained from a population sample of 120 subjects in six age groups ranging from under 20 years to 70 years. This sample was drawn from mixed rural and urban housing in Albania. Samples were frozen and subsequently tested for immunoglobulin G antibodies to *H. pylori*, using the Pyloriset Dry latex agglutination test (Orion Diagnostica). Results overall and by age are shown in the table.

In common with other eastern European countries, Albania was found to have a very high prevalence of *H. pylori* infection. Infection was common in all age groups, with no correlation between age and positive status, suggesting that incidence is not declining as in western nations. There may be no decrease in the burden of *H. pylori*-associated disease where incidence rates remain high.

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Accepted 6 January 1999

- 1 Parsonnet J. The incidence of *Helicobacter pylori* infection. *Aliment Pharmacol Ther* 1995;9(suppl 2):45–51.
- 2 Matysiak-Budnik T, Megraud F. *Helicobacter pylori* in eastern European countries: what is the current status? *Gut* 1994;35:1683–6.

Table Prevalence of *H. pylori* infection in an Albanian population

	Age group (years)						Total
	<20	21–30	31–40	41–50	51–60	61–70	
<i>n</i>	20	20	20	20	20	20	120
No +ve (%)	16 (80)	15 (75)	14 (70)	14 (70)	15 (75)	16 (80)	90 (75)

Book reviews

Neurology for the MRCP Part 2, David Smith. 176 pp. Arnold, 1998. £15.99, paperback

This small book is designed to help MRCP candidates who may have seen a relatively small number of clinical neurological cases and therefore "view the subject with trepidation". It covers four areas, namely 'grey' cases, data interpretation, slide interpretation, and viva topics.

The clinical material is clearly set out and the illustrations, in particular, are instructive and well chosen. Likewise the data interpretation section succeeds in its aims of being informative and challenging. The other two sections are less impressive. The 'grey' cases, for example, are too clear cut, but probably the whole point of a 'grey' case is that the system which is primarily involved may not be immediately obvious, and therefore placing a grey case into a neurological text is somewhat self-defeating.

There are only five viva cases, on the topics of Guillain Barré syndrome, narcoleptic syndrome, status epilepticus, multiple sclerosis and stroke. This section is hardly comprehensive and therefore could have been excluded in favour of more data interpretation studies.

Overall this is a useful book for MRCP candidates, even though some of the subjects covered, such as striatonigral degeneration and narcolepsy, are unlikely to be MRCP topics. I would commend this book to candidates, in particular for the clinical slides and their interpretation.

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Clinical guidelines and the law. Negligence, discretion and the law, Brian Hurwitz. 132 pp. Radcliffe Medical Press, Abingdon, 1998. £18.50, paperback. ISBN 1 85775 044 6

The dramatic growth of claims of clinical negligence, the personal trauma of litigation and the possibility of disciplinary action has affected every medical practitioner. The view that "there, but for the grace of God, I might stand" is one held by many of us. Within this environment the promise held out by clinical guidelines for the investigation of symptoms and management of disease has offered some comfort, especially when linked with the need to practice evidence-based medicine. The emergence of local and national guidelines and their availability to patients through the Internet has ensured that clinical decisions will often be challenged and desperate

patients and their relatives are ever more ready to seek information on the latest approaches to the management of various diseases.

Does this book provide clearcut answer to questions about the place of guidelines in the prosecution or defence of the clinician? It does not, and cannot do so because medicine cannot be practised as though it were similar to "painting by numbers". There is still a place for the role of experience and judgement. However, that role is being eroded and no clinician can practice without the safety net of recognised guidelines within his specialty. This also means that the role of the generalist will rapidly decline.

Brian Hurwitz's book is thought-provoking and disturbing. He draws on a wide range of clinical and legal cases. Its publication is timely and it should find a place on most doctors' bedside cabinets.

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PasTest final FRCA practice papers, Tim Issitt, Sarah Chieveley-Williams. 240 pp. PasTest, 1998. £17.50, paperback

The examination for the fellowship of the Royal College of Anaesthetists has recently reverted back from a three-part to a two-part examination, thus making most of the previous revision aids out of date. This book fills this gap in a very satisfactory manner.

Both the authors are of that soon-to-be-extinct-breed of senior registrar with a medical as well as anaesthetic postgraduate qualification. For the trainee, the book details the structure of the final examination and the marking system. It also gives the eligibility criteria for taking the examination, which unfortunately have already changed since the book was published! Senior House Officers may now take the examination, which previously only registrars could sit. (This change was implemented to help overseas visiting candidates who would have found increasing difficulty obtaining a registrar post.)

The introduction is particularly useful, explaining the format of the short answers, multiple-choice questions and the two viva sections of the examination. It also gives an indication of the scope of the syllabus, including basic sciences and clinical measurement which were previously emphasised in other parts of the older style of examination.

Three complete practice exams are then given with the correct solutions. Model short answers and viva responses are also supplied. These are well done, giving examples of good

short answers, not simply brief inadequate responses. There is a good breadth of multiple-choice questions which are usually well worded and the answers are explained. There are useful examples of X-rays, electrocardiograms, laboratory results, and clinical scenarios. No book of this type can be exhaustive, but any lacunae of knowledge should be identified.

Trainers will benefit from this book. The index is thorough and most useful for local revision teaching. The book may act as a stimulus to update certain topics. A few references are supplied and a personal list of helpful books and journals is given. Some authors and editors may be upset at finding themselves absent from this section but the writers of the book do make it clear that this view is an individual one.

This book should not, nor could be, used as a shortcut for detailed study, but it will appeal to many examination candidates and perhaps some postgraduate tutors.

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Clinical cases for the MRCS and AFRCS, G J Morris-Stiff, D J Bowrey, M C A Puntis, B I Rees, A A Shandall. 200 pp, illus. Arnold, 1998. £29.99, paperback

I thought this was an excellent book. The high quality of the illustrations – both X-rays and clinical photographs – is immediately apparent and the clarity of the format is excellent. The answers are clearly and concisely written; inevitably some of the answers consist of a very brief overview of a large topic, eg, head injury or abdominal trauma, but within the confines of space these brief summaries are apposite and do seem to focus on aspects of the topic which tend to feature in examinations. The authors set themselves a demanding task in trying to select 142 cases to demonstrate the entire spectrum of surgery (the whole of orthopaedics and trauma in 15 cases is fairly minimal), but generally they have produced a well-balanced selection. The acid test was definitely passed when my own surgical trainee not only praised the book but also intends to buy a copy! I congratulate the authors and the publishers on this high quality book and firmly believe that candidates will find it extremely useful in preparing for the MRCS/FRCS examinations.

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