PostScript

LETTERS

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What to do about psychological distress in emergency department senior house officers?

The article by McPherson *et a*[1] generates some interesting questions concerning disproportionately high levels of psychological distress among emergency department senior house officers (SHOs). The combination of shift work, a challenging working environment, broad case mix, and newly acquired decision latitude may explain the findings.

We did have some reservations about the article. We are unfamiliar with the general health questionnaire (GHQ) and brief COPE questionnaire. A more detailed description and explanation of terms would have been valuable. We felt that SHOs on nights (if not those on holiday) should have been included to reduce sample bias. Confining the study to units based in district general hospitals raises questions regarding generalisation. It would have been interesting to know the degree of shop floor senior cover in the units studied, and to examine whether this influenced distress levels.

How can we apply this useful work to our own practice? If we acknowledge the core finding, and accept that there is a problem among our junior colleagues, we then need to ask whether intervention is required. SHOs are required to have regular contact with a consultant supervisor, but there is potential tension between the roles of supervision, and support. Formal mentoring schemes offer an alternative, but their value in the emergency department has been questioned.[2] It may be that the best way to support SHOs is to be aware of their potential vulnerability to psychological distress, and to encourage a team based and pastoral atmosphere within our departments. This will permit doctors recognising a need for support to seek it out for themselves, from people who they feel are appropriate for the problem in hand. This is the approach we have, in the past, taken within our own unit. However, as a response to this article we will incorporate a session on stress management into our SHO teaching, perhaps in conjunction with administration of the GHQ and brief COPE… once we find out more about them.

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The hidden dangers of dietary supplements

With increasing health awareness, many parents give children dietary supplements. Getting children to take tablets is difficult therefore manufacturers have produced supplements in ''novelty'' shapes, for example cartoon characters.

In our emergency department there have been two cases to date of children presenting with accidental overdoses of vitamin and iron supplements. The toddlers, aged 2 and 3, had taken 20 and 15 tablets respectively. The children had thought that the supplements were sweets and indulged themselves. Both were admitted and blood serum iron concentrations taken. Fortunately, neither child needed further treatment, both making an uneventful recovery.

Severity of iron ingestion depends on the total elemental iron taken. Preparations are available incorporating iron in a number of different compounds. Ferrous sulphate contains 20% elemental iron, ferrous fumarate 33%, and ferrous gluconate 12%. To calculate the total elemental iron ingested, the iron compound involved and the number of tablets taken must be known.[1]

Elemental iron ingestion above 20 mg/kg is likely to produce features of toxicity, above 60 mg/kg possible fatality. Preparations for children are available with elemental iron content of 6 and 12 mg. For a typical 3 year old therefore, ingestion of less than 25 tablets may result in toxicity requiring treatment; 70 tablets potential fatality.

From 12 March 1998 to 31 December 2001, there were 90 product accesses to combined iron and multivitamin tablets on Toxbase, 12 of which were from Northern Ireland (Scottish Poisons Information Bureau, The Royal Infirmary, Edinburgh, personal correspondence April, 2002; NHS National Poisons Information Service, Edinburgh Centre, Personal correspondence Feb, 2002).

Products of this type are licensed as food supplements, not as a medicine, as they do not purport any medicinal claim. The quality of the product is governed by the Food Standards Agency. Children are legally permitted to purchase the product.

Carefully worded packaging and increased consumer awareness is necessary to prevent a fatality.

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Reference

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Aetiology of cerebral oedema in diabetic ketoacidosis

The excellent evidence based review of the emergency management of diabetic ketoacidosis (DKA) in adults by Hardern and Quinn perpetuates the premise that ''unnecessarily large volumes of intravenous fluids should be avoided because of the high case fatality rate of cerebral oedema''.[1] This presupposes that the rate of fluid delivery is causally related to the development of cerebral oedema, which has not been proved. The large 15 year paediatric study in the USA that analysed 6977 hospitalisations for DKA found among the 61 cases of cerebral oedema (0.9%) that after multiple logistic-regression analysis with random and matched controls, the only variables statistically associated with cerebral oedema were higher initial serum urea nitrogen concentrations and lower partial pressures of carbon dioxide at presentation.[2] In addition, smaller increases in serum sodium concentration during treatment and the use of bicarbonate were also implicated. Importantly, the rate of fluid, sodium, and insulin administration were not associated with the development of cerebral oedema, nor was the initial serum glucose or its rate of change.

Clearly these findings relate to patients aged 18 years or less but most occurrences of cerebral oedema in DKA are in children and adolescents, with only rare cases in adults. However, the underlying aetiology should be no different. One unifying hypothesis is that the cerebral oedema is related to cerebral vasoconstriction, brain ischaemia, and hypoxia, as hypocapnoea causing cerebral vasoconstriction and extreme dehydration would both decrease cerebral perfusion. In addition, as children's brains have higher oxygen requirements than adults this may explain their unique susceptibility.

Perhaps clinicians should focus more on recognising the warning signs of cerebral oedema such as headache, lethargy, and deterioration in conscious level, prior to seizures, incontinence, pupillary changes, bradycardia, and respiratory arrest as brain stem herniation occurs.[3] Early hyperosmolar treatment and presumably supplemental oxygen with exemplary supportive care are then essential. Finally, accepting that cerebral oedema may well be idiosyncratic rather than iatrogenic could have important medicolegal connotations too.

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Glucagon use in symptomatic β blocker overdose

I was interested to read the best BET ''Glucagon for the treatment of symptomatic β blocker overdose'' by Boyd and Ghosh.[1] As the authors recognised, the six studies tabulated were of mixed overdose or had multiple therapeutic interventions and could not answer the question posed. However, had the search strategy included individual drug names (for example, propranolol, atenolol) more relevant papers would have been found, including two cases of pure β blocker overdose successfully treated with glucagon alone.[2 3] The evidence for glucagon in treating symptomatic β blocker overdose will probably never reach a higher level than case reports. This is true of most ''antidotes'' because of ethical constraints on toxicology studies. Glucagon, however, has been shown to be effective in treating symptomatic β blocker overdose in various controlled animal studies.

About 20 deaths per year in the UK are attributed to β blocker overdose. The authors state that glucagon is expensive. It is true that large doses may be required and that this may outstrip hospital supplies. However, at an initial dose of 5–10 mg (100 μg/kg) intravenously at £19.95/mg, the cost[4] compares favourably with thrombolysis as a potential lifesaving intervention. Atropine has been shown to be spectacularly ineffective in this setting and alternatives such as β agonists, phospho-diesterase inhibitors, insulin-euglycaemia, and pacing have significantly more associated complications than glucagon without improving outcome.

Glucagon treatment for symptomatic β blocker overdose should not yet be discarded on grounds of cost or lack of evidence.

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Emergency management of contact lens associated corneal abrasions

Corneal abrasions in contact lens wearers may have sight threatening consequences. Contact lenses can compromise the corneal epithelium and act as pathogenic vectors, facilitating the development of bacterial keratitis. Most corneal abrasions heal quickly when treated with topical antibiotics, which act as lubricants and antimicrobial agents. However, in contact lens wearers there may be rapid progression to corneal scarring or even perforation.

Two patients with contact lens related corneal abrasions, who were initially treated with topical fusidic acid or chloramphenicol, have presented with corneal stromal abscesses. The abscesses developed 12 hours and three days respectively after diagnosis of simple corneal abrasion. Visual acuity was perception of light and hand movements. Both required admission for intensive topical fortified guttae gentamicin and guttae cephalosporin.

Pseudomonas aeruginosa and proteus were grown, which were resistant to chloramphenicol and fusidic acid. Best corrected visual acuities were 2/60 and 6/36 after resolution of the infections; one patient has proceeded to corneal grafting.

A 15 year study of resistance in bacterial isolates from corneal scrapings found that 30.4% of isolates were resistant to chloramphenicol[1] (54% of Gram negative organisms), with a significant increase in resistance during this period. Once microbial keratitis is established, a combination of topical fortified aminoglycoside and cephalosporin or fluoroquinolone is indicated[2]; no trend for increasing resistance to these antibiotics was observed in the aforementioned study.[1]

Contact lenses are the most important risk factor for the development of bacterial keratitis.[3] In the emergency department, a history of contact lens wear should be sought, with urgent review of worsening abrasions. We advise that all contact lens related red eyes should be referred to the ophthalmology department, as clinical signs may initially be subtle and corneal scraping may be warranted. Timely commencement of guttae ofloxacin with the first sign of infection, may greatly reduce the chance of poor outcome.

Contributors

Shauna Quinn treated the second patient, reviewed the literature and wrote the paper. Jeffrey Kwartz treated both patients and contributed to the discussion of core ideas. He was the supervisor and is the guarantor.

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BOOK REVIEWS

Radiology for anaesthesia and intensive care

Richard Hopkins, Carol Peden, Sanjay Gandhi. London: Greenwich Medical Media, 2003, pp 332. ISBN 1-84110-119-2

This book is clearly aimed at anaesthetists preparing to sit the FRCA examination. Any doubt regarding this is quickly dispelled by the two chapters that follow the introduction: ''About the FRCA Examination'' and ''The Pre-Operative Assessment''. Hardly surprising, therefore, that it is of limited relevance to emergency medicine.

Each of the book's seven main chapters begins with a general introduction to set the scene and introduce underlying concepts, before moving on to a series of realistic ''case illustrations'', in question and answer format, accompanied by an explanation and additional background information. This is a clear and effective layout, but because the whole case is often presented on a single page it is sometimes a little too easy to read the answer before the question, or perhaps I'm just a natural cheat.

There are, however, some useful sections. I found the chapter on imaging the chest, particularly in relation to chest radiological interpretation, interesting and educational, but the abdominal section was too heavily weighted towards computed tomography and contrast studies to be of substantial use.

A sizeable section of the book is dedicated to trauma radiology, but while the chapter on the cervical spine is informative and up to date, that on chest and abdominal trauma covers management at a basic level, with little imaging of interest.

As emergency physicians take on a greater role in the management of head injury, and computed tomography becomes more widely available, I find myself interpreting more and more head scans. For this reason I thought that the chapter on computed tomography of the head was probably the best in the book. It makes a good introduction to those who are approaching this subject for the first time, and has some excellent scans, clearly described with useful clinical detail. There is also a short final chapter on ultrasound in intensive care units, which overlaps considerably with the recent development of ''FAST'' scanning in the emergency department. I am doubtful, however, that a textbook can teach more than the basic principles underlying such an essentially dynamic skill.

For those about to sit the MFAEM or FFAEM exams there is some useful information in this book, but probably not sufficient to justify the purchase price. The two major problems are the inevitable anaesthetic slant, and the limitations of the medium itself. This anaesthetic slant is constantly manifest in the presentation of cases that are particularly relevant to anaesthesia (lots of rheumatoid arthritis, for example), followed by questions such as ''are there any precautions necessary prior to anaesthesia?'' Some might argue that with the increasing performance of intubation by emergency physicians these questions are now becoming more relevant, but on the other hand detailed imaging is a rare luxury before rapid sequence induction in our departments.

The limitations of the medium are inherent to all books that profess to teach radiology: particularly in an A5 format large radiographs are reduced to small pictures in which the detail is lost. This may be one of the reasons why the section on head computed tomography is so effective: the pictures are about the same size as the original films. For me, however, there is no substitute for handling and examining the real thing. Until digital radiology finally arrives in the south west of England, that is.

J R Benger

Sudden death and the myth of CPR

Stefan Timmermans. Philadelphia: Temple University Press, 1999, $19.95, pp 256. ISBN 1-56639-716-2

Most emergency physicians will sometimes recognise a feeling of futility during cardiopulmonary resuscitation (CPR)—the algorithm is followed despite the fact that most of those present know the attempt is doomed to failure, or frankly inappropriate.

Stefan Timmermans is a Belgian healthcare sociologist who spent time in American emergency departments observing the rituals surrounding CPR. His book questions the notion of CPR for all, and the over-optimistic prognosis of survival from out of hospital cardiac arrest that is portrayed in the media, and by some medical authorities. The book describes the attitudes and feelings of doctors, nurses, and paramedics, their definitions of good and bad resuscitation attempts, and the way in which they feel constrained by guidelines and lawyers.

The chapters are wide ranging and include the evolution of resuscitations techniques, death awareness, and what constitutes a "good" death, as well as discussion on advance directives and the presence of relatives during resuscitation attempts. The author divides resuscitation attempts into four distinct categories, or *trajectories*, which will be familiar to all practising emergency physicians: the *legal death trajectory*, where resuscitation is performed mainly as a legal matter; the *elite death trajectory* where the victim is presumed to have high social viability and receives aggressive resuscitation irrespective of clinical viability (for example, the young); the *temporary stabilisation trajectory*, in which the patient is resuscitated despite the fact that the short term prognosis is poor; and the *stabilisation trajectory*, in which prompt resuscitation leads to a better outcome.

The book is written from a sociologist's perspective, and therefore does not aim to provide answers—just observations. Yet despite the North American setting, it raises questions that are highly applicable to UK practice, and this book should be required reading for all ALS providers.

J E France

Handbook of paediatric emegency medicine

P O Brennan, K Berry, C Powell, M V Pusic, editors. Oxford: BIOS Scientific Publishers, 2003, pp 458. ISBN 1-85995-242-4,

> Knowledge is a process of piling up facts; wisdom lies in their simplification. Martin Luther King, Jr (1929–1968)

Upwards of two million children will attend accident and emergency departments in the United Kingdom every year. Many thousands more will attend general practice for advice or treatment after acute illness or injury. Large numbers of practitioners in many different settings therefore need to be prepared to deal with children with a variety of urgent and emergency conditions. As an old Chinese proverb states "Small children do not pretend to be sick". The problem is that the vast majority of children have minor to moderate illness, much of which is self limiting. Indeed many of the injured children require little more than symptomatic relief and general supportive care.

The problem therefore is identifying the wheat from the chaff. In other words, how does one identify the critically ill child, or the child who is brewing something serious? Age and experience help. Certainly knowledge is useful. More often the wisdom of Solomon is required. There is no doubt that experience brings greater wisdom, and with it ability to deal with children effectively. I suppose that is really what I like about this book. The authors have brought their collective experience and wisdom, gathered over the years (I am not brave enough to state how many, but I know it is considerable!) to produce an extremely readable text that is well laid out and well presented. The salient features are highlighted in boxes and the use of diagrams is good. I personally would have liked to have seen more radiographs and clinical pictures, but then again this may not be the purpose of a handbook. This may best be left to a colour atlas, or better still actual clinical practice. Computed tomograms of the head are poorly produced and this is again disappointing.

This book covers virtually all the salient features of paediatric emergency medicine. There are no glaring omissions, although one always has pet subjects one would wish to see incorporated. It would be churlish to let these personal idiosyncrasies detract from the overall good feel I have for this text.

There is no doubt that this book will provide useful reading at all levels of experience. Reading it and being familiar with the contents will bring greater knowledge. Wisdom, I'm afraid will have to come with time. The only major problem with this book is that it is a bulky, heavy hardback. As such it won't fit into a pocket conveniently and may well end up on the shelf. By being left on the shelf it runs the risk of being ignored and this, I think, would be a tragedy.

Martin Luther King would be proud of this effort.

T Beattie

Core cases in critical care

Edited by S Ridley, G Smith, A Batchelor. London: Greenwich Medical Media, 2003, £29.50, pp 250. ISBN 1-84110-161-3

In 230 pages and a few monochrome illustrations this paperback covers the top 20 clinical cases that are the stock in trade of every intensive care unit. The authorship is a reassuring collection of UK intensivists, a *who's who?* of the Intensive Care Society. I liked the standardised format; case histories are followed by discussion of the main issues with reference to pathophysiology, treatment options, and outcome. A panel of key learning points rounds off each chapter, and the recommended further reading is appropriate and proportionate.

A number of the cases bear upon emergency care and many are set in the resuscitation room. The importance of securing the ABCs is emphasised before discussion of theoretical concepts, not always the case in books of this sort. This reflects the interests of the authors, many of whom are active in education at the interface between intensive care and emergency medicine. Relevant cases include burns, trauma, and overdose, but pyrexia is included uncomfortably in the chapter on status epilepticus. The chapters are up to date; the roles of inhaled nitric oxide and prostacyclin in ARDS are set out. And there is a review of the evidence on non-invasive ventilation in COPD. Activated protein C is (to this reviewer at least) a very new treatment in septic shock, and its brief mention is testimony to the book's contemporary quality. The Swan Ganz catheter is placed in its correct context, alongside alternatives including the pulse induced continuous cardiac output monitor. I was also pleased to see the role of corticosteroids set out in accordance with current thinking on the treatment of sepsis.

This reviewer has an aversion to diagrammatic representation of pulmonary physiology, lung capacities, closing volumes, and zones of perfusion. The authors avoid such esoteric concepts, and there is no assumption of knowledge of molecular biology in the chapter on sepsis and multiple organ failure. Cardiac care is the major omission from what is otherwise a reasonably broad based content.

Trainees in intensive care medicine from all parent specialties will find this a useful and accessible resource. It sets out to present a consensus approach to common clinical problems, and is not a comprehensive textbook. For any specialist registrar about to start a secondment in the ICU this little book would be a good investment.

P Nee