Unfractionated heparin and low molecular weight heparin in acute coronary syndrome without ST elevation: a meta-analysis

Overview—Acute coronary syndrome without ST elevation is attributed to unstable angina and non-Q-wave myocardial infarction. The role of unfractionated and low molecular weight heparin (LMWH) in the management of aspirin treated patients with acute coronary syndrome without ST elevation has not been determined satisfactorily by any single large study. This meta-analysis investigates this issue.

Design—Meta-analysis of 12 randomised trials, involving a total of 17 157 patients.

Main outcome measures—The primary efficacy outcome measures were death and non-fatal myocardial infarction. The primary safety outcome measure was the occurrence of a major bleed. Secondary outcome measures of interest were recurrent angina and the need for re-vascularisation.

Main results—In aspirin treated patients, short-term (up to seven days) treatment with unfractionated heparin or LMWH was associated with significant reductions in death and non-fatal myocardial infarction (summary odds ratio 0.53; 95% CI 0.38, 0.73, p=0.0001), equivalent to 29 events prevented per 1000 patients. No significant difference was found between unfractionated heparin and LMWH during short-term treatment. Long term LMWH conferred no reduction in deaths or non-fatal infarcts, but was associated with a significantly increased risk of major bleeding (odds ratio 2.26, (95% CI 1.63, 3.14), p<0.0001), equivalent to 12 major bleeds per 1000 patients treated.

Conclusion—The authors conclude that there are benefits of using unfractionated heparin or LMWH in aspirin treated patients presenting with acute coronary syndrome without ST elevation in the short-term, but cannot find evidence to support its use beyond the first seven days.

Critique—Acute coronary syndrome without ST elevation (unstable angina and non-Q wave myocardial infarction) is a common presentation to accident and emergency. Patients who present in this way are at significant risk of developing recurrent ischaemic events. Previous randomised trials, while sizeable, have been of insufficient power to determine with certainty the role for the use of heparin products in addition to aspirin in these patients. This meta-analysis circumvents many of the shortfalls encountered in individual randomised trials. The combination of using data from more than 17 000 patients with clear, objective endpoints enabled the authors to reach conclusions that should help to shape future patient management. No differences were observed between unfractionated heparin and LMWH in either efficacy (death or myocardial infarction) or safety (major bleeds), based on a meta-analysis of five trials involving a total of more than 12 000 patients. However, as the authors correctly point out, LMWH could be preferred for its theoretical pharmacokinetic advantages.

Early use of non-invasive ventilation for acute exacerbations of chronic obstructive pulmonary disease on general respiratory wards: a multicentre randomised controlled trial

Overview—Non-invasive ventilation (NIV) has been shown to be effective in reducing the need for intubation and the mortality associated with acute exacerbations of chronic obstructive pulmonary disease (COPD) in an intensive care setting. The authors attempted to determine whether the early introduction of NIV for acute exacerbations of COPD in the general respiratory ward would reduce the need for intubations and improve mortality.

Design—This was a multicentre randomised controlled trial, involving a total of 236 patients with mild to moderate acidosis prospectively recruited from 14 hospitals in the UK. Half of the patients were randomised to receive NIV, the other half to receive standard treatment.

Main outcome measures—The primary outcome measures were death and requirement for tracheal intubation (defined by strict failure criteria). Secondary outcome measures were improvement in breathlessness, respiratory rate, pH and pCO₂ at four hours after admission.

Main results—The use of NIV significantly reduced the need for tracheal intubation as defined by the failure criteria. Thirty two of 118 (27%) of the standard group failed, compared with 18 of 118 (15%) of the NIV group (p=0.02). Inhospital mortality was also reduced by NIV, 24 of 118 (20%) died in the standard group compared with 12 of 118 (10%) in the NIV group (p=0.05). Also, NIV led to a more rapid improvement in pH in the first hour (p=0.02), a greater fall in the respiratory rate at
four hours (p=0.035), and a reduction in the duration of breathlessness (p=0.025).

Conclusions—The authors concluded that patients with COPD can be managed effectively in the general respiratory ward with the introduction of NIV. They pointed to a marked reduction in the requirement for tracheal intubation and inhospital mortality for these patients.

Critique—The study has the considerable advantage over previous studies of having used very clear inclusion criteria for those admitted with acute exacerbation of COPD. Patients were admitted into the trial if the respiratory rate was over 23/min, PaCO₂ greater than 6 kPa, with a pH between 7.25 and 7.35. The exclusion criteria were also clear (low GCS, pneumothorax or where active treatment was deemed inappropriate). Randomisation was sound and there seemed to be independent statistical analysis. Although the primary outcome measures were clear, the secondary outcome measures were more subjective (including mask discomfort and breathlessness).

Availability of intensive care beds for patients with acute exacerbations of COPD within the UK health care system seems to be currently somewhat limited. NIV seems to have the potential to reduce demands on intensive care facilities while providing effective management for some patients with COPD. Widespread implementation of NIV would require extra training of the nursing staff. This paper has tackled a very important issue, and has provided a realistic solution for managing patients with COPD in our health care system.

Prospective audit of incidence of prognostically important myocardial damage in patients discharged from emergency department


This was a prospective observational study of patients presenting with chest pain of uncertain origin to a district general hospital accident and emergency (A&E) department. Some 110 of a possible 160 patients meeting the study’s criteria were reviewed by a single clinician within 48 hours of their A&E attendance, having had cardiac causes of chest pain “ruled out” clinically and electrocardiographically. At review, the patients were re-examined, had a repeat ECG and blood taken for cardiac troponin T. All repeat ECGs were said to be “normal”, but eight patients (7%) had detectable troponin T. In seven patients, troponin T levels exceeded 0.1 µg/l, indicating myocardial damage. All seven patients were referred for urgent cardiology follow up.

Comment—The findings of this study are consistent with those of previous studies that have shown that myocardial infarction is relatively frequently “missed” in A&E. This study nicely focuses upon troponins at a time when their measurement is playing an increasing role in the investigation of patients who present as an emergency with chest pain. It is worth noting that troponin T is not completely specific for myocardial ischaemic injury. Given its long half life, it might have been interesting if the authors had also measured troponin T levels in blood taken at the time of the initial presentation, as this might have uncovered problems that actually predated attendance at the emergency department. One major criticism of this paper is that the patient selection criteria excluded patients who were discharged with disorders believed to be of gastrointestinal origin. Troponin levels on these patients may have yielded further interesting results.

Evaluation of the utility of computed tomography in the initial assessment of the critical care patient with chest trauma


This was a prospective observational study of patients presenting with chest injuries to a level III Spanish trauma centre (equivalent to a Level I US trauma centre). The study was designed to investigate whether or not early thoracic computed tomography (CT) in chest trauma patients improves eventual outcome. The results confirmed that thoracic CT did detect more injuries in chest trauma patients than did conventional chest radiographs. However, there was no demonstrable effect on the final outcome of these patients in terms of mortality, time spent on mechanical ventilation or in intensive care.

Blunt splenic injuries: dedicated trauma surgeons can achieve a high rate of non-operative success in patients of all ages


This study focuses upon the non-operative management (NOM) of blunt splenic trauma. The conclusions drawn from the management of 251 consecutive patients with blunt splenic trauma to a Level I US trauma centre challenge two pieces of oft quoted dogma in relation to the NOM of blunt splenic trauma. It has previously been suggested that blunt splenic trauma in the paediatric population should be only managed by paediatric surgeons and that NOM is not feasible for those above 50 years of age. However, the findings of this study suggest that advanced age is not a contraindication when considering NOM of blunt splenic injury and furthermore that paediatric injuries can be managed safely by appropriately trained non-paediatric trauma surgeons.

Open or closed diagnostic peritoneal lavage for abdominal trauma? A meta-analysis


This meta-analysis attempts to determine whether it is preferable to use an open or closed technique when performing diagnostic peritoneal lavage (DPL) for abdominal trauma. Seven randomised control trials comparing open and closed DPL (involving a total of 1126 patients) were identified from a search of the English language literature between 1977 and 1999. Meta-analysis revealed that
closed DPL is comparable to open DPL in terms of accuracy and major complications. However, the advantage of a reduced time taken to perform closed DPL seemed to be offset by increased rates of technical difficulties and failure, requiring conversion to open DPL. The authors therefore conclude that an open method should be preferred to a closed one, but acknowledge that the role of DPL seems to be diminishing because of increasing use of other investigations (such as ultrasound).

**Bilateral simultaneous atraumatic quadriceps tendon ruptures associated with “pseudogout”**
R J Tedd, M R Norton, W G Thomas
*Injury* 2000;31:467–9

This case report serves as a reminder that “atraumatic” quadriceps tendon rupture can be difficult to diagnose and that such failure of diagnosis can result in even more difficulty in trying to treat it. A 58 year old diabetic gentleman is described whose initial presentation was to his general practitioner with a history of both knees suddenly giving way while walking. An initial diagnosis of peripheral neuropathy was made and the patient was started on an intensive rehabilitation programme. There was no improvement. He presented to accident and emergency with a painful swollen left knee three months after his initial complaint. A provisional diagnosis of septic arthritis was made. A septic screen was arranged, which proved negative. An aspirate of fluid from the knee joint showed a large number of calcium pyrophosphate crystals. Screening of the other knee confirmed a complete rupture of quadriceps tendon with presence of calcium pyrophosphate in the joint. Spontaneous rupture of the quadriceps tendon is generally believed to be attributable to weakening as a result of degenerative changes or repeated minor trauma, although there are previous associations in the literature with gout and hyperparathyroidism. Bilateral simultaneous quadriceps tendon rupture is a rare injury and in this patient was attributed to pseudogout.

**Sudden cardiac death during sport and recreational activities in Israel**
O Yanai, E D Phillips, J Hiss
*Journal of Clinical Forensic Medicine* 2000;7:88–91

This retrospective review of sudden cardiac death during strenuous physical activity in Israel serves as a reminder of the possibility of atherosclerotic cardiovascular disease in the relatively young. Unsurprisingly, atherosclerotic cardiovascular disease was found to be responsible for the majority of sudden cardiac deaths during sport in people aged 35 years and over. It was, however, also the second most frequent cause of death (after cardiomyopathy) in those aged less than 35 years.

**Purtscher’s retinopathy**
S Gibson, A Cox, D Ross
*Injury* 2000;31:472–4

Purtscher’s retinopathy was described in 1912, as multiple superficial white retinal patches and retinal haemorrhages surrounding a normal optic disc, attributed to a sudden increase in intracranial pressure transmitted to the optic nerve. This report describes the case of a 38 year old woman who presented to accident and emergency after a high speed road traffic collision. The patient sustained a seatbelt injury to her chest wall, but no other obvious direct limb or facial injuries. On detailed examination and screening, she was found to have a skull fracture, but computed tomography of the head was normal. Chest radiological findings were consistent with lung contusion and there was a wedge fracture of the thoracic spine. Subsequently she developed a scotoma of the left eye on the contralateral side to her skull fracture. Visual acuity testing revealed a marked deficit (reduced to finger counting), with an associated central visual field loss. There was no afferent papillary defect. Fundoscopy of the eye confirmed multiple retinal haemorrhages, cotton wool spots and “Purtscher flecken” close to optic disc. These areas of optic disc whitening are attributable to occlusion of deeper retinal capillary beds. Her visual acuity improved to 6/18 after some time. It was postulated that this woman developed retinal changes as a result of chest wall compression causing either a momentary increase in central venous (and hence retinal venous) pressure or a sudden increase in intracranial pressure transmitted to the optic nerve (Purtscher’s retinopathy), or both.
LETTERS TO THE EDITOR

Phystostigmine as treatment for severe CNS anticholinergic toxicity

EDITOR,—We report the successful use of phystostigmine to treat central anticholinergic toxicity: a use described before but rarely seen.1 We treated a patient admitted after an overdose of amisulpride and procyclidine (not his own medication). He became extremely agitated and was treated with intravenous benzodiazepines (total quantity in 26 hours equivalent to 125 mg diazepam) and remained agitated. Because of the risks of serious injury if his agitation was untreated and of further benzodiazepine use outside a critical care area (the ward to which he was admitted has a patient:nurse ratio of 8:1), he was treated with 1 mg of physostigmine. This was immediately and dramatically followed by a period of complete lucidity lasting 90 minutes. He did not become agitated again and his confusion resolved fully after a further 16 hours. Reference to an ITU was considered but it was felt that the risks of parallel, intubation and ventilation (and the possible need for interhospital transfer) outweighed those associated with phystostigmine treatment. This latter option had the advantage of therapeutic and diagnostic potential.

Procyclidine is an antimuscarinic drug with a half life of 8 to 16 hours. When taken in overdose the features of anticholinergic toxicity may be delayed.2 His agitation was unlikely to be attributable to amisulpride as this is a D2/D3 receptor antagonist but the patient had no signs of extrapyramidal side effects.3 Phystostigmine is a tertiary ammonium compound that reverses anticholinergic effect via acetylcholinesterase inhibition. Uniquely, for this class, it crosses the blood-brain barrier. It has a rapid onset of effect and duration of action of one to two hours.3 We do not propose that phystostigmine be routinely used to treat changed mental status after poisoning. We do, believe, however, that it has a very specific role in the treatment of patients with persisting central anticholinergic toxicity despite sedation with benzodiazepines.

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Emergency medicine or accident and emergency?

EDITOR,—What is emergency medicine? Is it the specialty that provides “The care of emergency patients . . . by specially trained doctors who deliver a wide range of services for all patients presenting to a separate emergency department” including “The initial assessment and early treatment in resuscitation, trauma, medical and paediatric emergency” and “acute care of ‘medical’ in its widest context including medicine, surgery, anaesthesia, paediatrics (and) psychiatry”? Or is it a subspeciality of general medicine responsible for the assessment and admission of acute adult medical conditions?

The Royal College of Physicians’ has named the latter “acute medicine”, yet confusion about the distinction remains.

I have reviewed 10 successive editions of BMJ Classified (14 October 2000 to 16 December 2000) in order to identify all job advertisements for “emergency physicians” or doctors to work in “emergency medicine”. Further study of the advertisements allowed me to establish which of these posts were to work in (accident and) emergency departments and which to work in medical assessment/admission units and/or general medicine (table 1).

Table 1 Number of advertisements for “emergency physician” or “emergency medicine” by grade and specialty

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<th>Grade</th>
<th>Consultant</th>
<th>Non-consultant career grade</th>
<th>Training grade</th>
<th>Total</th>
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<td></td>
<td>2</td>
<td>1</td>
<td>1</td>
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The results of this study show that the majority of positions in “emergency medicine” (11 versus 6) are in fact to work in acute general medicine.

I believe that it is now time for our speciality to adopt the name emergency medicine, to bring us in line with our colleagues in the USA, Canada, Australia, New Zealand and the Far East. If we do not, we are in danger of losing the title altogether to a subspeciality of general medicine. If this were to happen, we would be stuck with “A&E” which, along with its predecessor “casualty”, belong firmly in the last millennium.

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Intranasal diamorphine in adults

EDITOR,—We would like to describe a patient who benefited from intranasal diamorphine administration. This route has become an acceptable and potentially important route for providing analgesia for children who benefited from intranasal diamorphine administration. This route has become an acceptable way of providing analgesia for children. We feel it is also an acceptable and potentially important route for adults. It is rapidly absorbed from the venous plexi of the nasal mucosa and provides less variable pain relief than rectal or oral routes. Its aqueous solubility allows the use of small volumes.

We recently used intranasal diamorphine as pain relief in a 57 year old woman. This woman suffered from chronic renal failure, smoking and frequent headaches. She had fallen onto her right hand. Her right elbow was tender, swollen and deformed. She was supporting this elbow with her uninjured forearm. This combined with an arteriovenous shunt in the left arm made venous access difficult. She was crying out in pain and severely distressed. To allow immobilisation and investigation she was given intranasal diamorphine, at a dose of 0.1 mg/kg. This gave immediate pain relief.

revealed a displaced four part supracondylar fracture of the right humerus with an intra-articular component. Although the oral or intravenous routes remain the most favoured for analgesia, it is our experience that they are not always available. The oral route may be inaccessible, for example, in a hard collar or may take longer to work because of delayed gastric emptying. Intravenous and intramuscular routes are alternatives but a patient may refuse such analgesia because of a dislike of injections. The intramuscular route also has delayed action. The rectal route can be embarrassing and uncomfortable. Rapid analgesia may be necessary and the intranasal route provides this when intravenous access cannot be secured or is not strictly necessary.

We have used intranasal diamorphine on several occasions. During its use we monitor vital signs. Pain scores or direct questioning measures its efficacy. We believe that a prospective study of its use would permit identification of potential side effects or complications. It is our experience that these do not occur. Our experience is insufficient to identify any cause and vomiting would be a significant problem.

We feel it is an important adjunct in certain clinical situations and a valuable addition to pain management.

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The role of non-invasive ventilation in the emergency department

EDITORS.—Anthony Cross highlighted the effectiveness of non-invasive ventilation (NIV) in the emergency department in his review. As he concluded, studies certainly indicate that this treatment is beneficial in the treatment of acute exacerbation of chronic obstructive airways disease—with regard to the need for intubation, length of hospital stay and mortality.

The evidence for the use of NIV in the treatment of acute pulmonary oedema also exists, and is stronger than Dr Cross indicated. In addition to the three randomised controlled trials comparing chronic positive airway pressure (CPAP) with standard treatment of acute pulmonary oedema identified in the review, there is also an article by Takeda et al from Tokyo.1 If the results of this study are pooled with the three reviewed by Cross, the overall risk reduction for mortality becomes −13.8% (95% CI −24.2 to −3.4%). The interesting thing about this finding is that the confidence interval does not cross zero. This is the first time that pooled results have shown that CPAP treatment for heart failure, in addition to reducing intubation rates, also decreases mortality—at least in the short-term (only two of the studies included long term follow up). There is also evidence that CPAP treatment benefits are greater in those with increasing severity of pulmonary oedema.

There remains a need for a large prospective randomised controlled trial into the effectiveness of NIV compared with standard treatment of acute pulmonary oedema. If the results confirm the trends suggested in the available smaller studies, it should be sufficient to encourage our current treatment practice in UK emergency departments regarding acute pulmonary oedema. Admissions and death attributable to heart failure, unlike those attributable to respiratory heart disease, are predicted to increase. CPAP has shown to be effective in reducing the number of these patients intubated, admitted to intensive care units or dying. Personally, I believe that there already exists evidence for the acquisition and use of CPAP/BiPAP machines to treat acute pulmonary oedema in all emergency departments in the UK.

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EDITOR.—We read with interest the review by Cross of the use of non-invasive ventilation (NIV) in the emergency department.1 We have appraised the evidence concerning the effectiveness of NIV for respiratory failure and found further evidence that both supports its use and highlights current controversies. Although Cross comments on individual studies he does not report a meta-analysis of trials using NIV in patients with acute respiratory failure.2 By combining the data in these trials the results achieve significance. Patients with chronic obstructive pulmonary disease (COPD) treated with NIV had reduced intubation rates with a number needed to treat (NNT) of three and reduced mortality with an NNT of five.

Further to this, the three trials of continuous positive airway pressure (CPAP) for the treatment of left ventricular failure (LVF), quoted by Cross, have been the subject of a systematic review.3 This combined the data and found a significant reduction in intubation rates for those receiving CPAP (NNT=4) and a non-significant trend towards decreased mortality.

There are however issues that have not yet been resolved. The one trial comparing CPAP with bi-level positive airway pressure (BiPAP) for the treatment of acute respiratory failure is the subject of non-invasive positive pressure ventilation (NIV) in patients with acute respiratory failure.4 CPAP in patients with LVF was terminated early because of a high rate of myocardial infarction in the BiPAP group. The BiPAP group contained more patients with chest pain and known coronary artery disease. They also had higher mean creatinine kinase and lower mean pH and PaO2 at enrolment. As Cross reports, these differences between the two groups did not achieve statistical significance, but there was a trend towards worse baseline variables in the BiPAP group. With only 27 patients in this study the possibility of a type 2 error also exists. Further trials are needed to determine the role of BiPAP in the treatment of LVF.

Two of the three studies of CPAP in LVF excluded hypotensive patients. CPAP may increase cardiac output in patients with cardiac failure,5 and further study is needed to confirm its safety in these patients.

In our department we have treated patients with respiratory distress using CPAP for LVF and BiPAP for exacerbations of COPD. Patients who are unable to cooperate or require immediate intubation are excluded. With the use of fully portable equipment NIV is a simple technique, which has the potential to benefit many emergency department patients, and we welcome the attention drawn to this by the review article.

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trials in which the intubation rate for the controls is usually very high (up to 74% in studies of COAD patients and up to 60% in those with patients presenting with acute pulmonary oedema). It is almost inevitable that NIV will reduce the intubation rate when the rate is already so high in the controls. In our audit in A&E departments in Leeds, only 11% of severe acute pulmonary oedema patients (respiratory rate >23/min and pH <7.35) were intubated after the usual therapy for this condition. It is much less likely that NIV, in the A&E department, would reduce this low intubation rate significantly.

Cros also suggests that “early intervention [with NIV] may avoid the risks and complications of endotracheal intubation.” There is no doubt that the complication rate has been shown to be reduced by NIV in published studies, but in others a non-significant trend towards increased mortality in those treated with NIV has been shown and attributed to delays in intubation. It is important, therefore, to point out that NIV is not a substitute for intubation but may delay or prevent it becoming necessary in a carefully selected group of patients.

Two other points not discussed in the review are also important. Firstly, like everything else in A&E practice, there are training issues, particularly when new or unfamiliar techniques, such as NIV, are used. Both doctors and nurses need to know when and how to use particular equipment and, perhaps more importantly, when not to. In particular they need to be fully trained in all the possible complications of NIV. A&E staff may not use the technique regularly and so skills will decay without proper training schemes in place.

Secondly, many of the NIV machines currently on the market do not come with a battery pack, and this may present difficulties when transfer to the ward or intensive care is required by a patient who has been started on NIV in the A&E department. Some patients (particularly those with COAD) will have a prolonged requirement for NIV and it is, therefore, important to consider investing in an NIV machine that can run from a battery.

In summary, most patients presenting to A&E with respiratory distress do not need ventilatory support. For those that do, endotracheal intubation and mechanical ventilation remains the gold standard. For a small group of patients with chronic lung disease needing urgent ventilatory support, NIV may be first line treatment. Chronic positive airway pressure undoubtedly has a role in patients with acute pulmonary oedema.

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You can’t anaesthetise patients—you are not employed as an anaesthetist

Editor,—I would like to highlight an example of “specialty interface problem” and how the problem may be tackled. Although I was an anaesthetist for over three years and possess the anaesthetic fellowship, I have come into criticism from anaesthetists for intubating patients using anaesthetic drugs in my role as Specialist Registrar in Emergency Medicine (year 5). The first time it happened I ignored the criticism, but it has occurred since in different English hospitals. There are several issues arising out of this.

(1) There is a need to inform anaesthetic colleagues that not only is anaesthesia a core secondment but specialist registrars are keen to put the skills into practice.
(2) There is a need for individual clinicians to audit their practice of intubating patients in the emergency department. My personal logbook (kept on Microsoft Access) covers patient name, date, indication for intubation, drugs and anaesthetics, morbidity and mortality. Thus it covers not only the “flat overdose patient” or “coma ?subarachnoid” but also patients who arrive in cardiac arrest unintubated.
(3) There is a risk management need, as a specialty, to nationally audit morbidity and mortality in relation to patients being intubated by emergency department staff. In addition to the criteria above, timeliness and appropriateness of intubation should be considered.

So what if we do not audit this particular area of activity? When the inevitable disaster happens the clinical governance committee of the hospital may judge rapid sequence induction and intubation to be a procedure for the elite few doctors who satisfy the following criteria: confident, competent, qualified and employed as anaesthetists?

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Seventy-five years ago an American advertising executive wrote that “one picture is worth ten thousand words”. The aphorism holds particularly true for busy junior doctors preparing for examinations. This little book offers 150 pictures; clinical images, radiographs, tables of monitored data and diagrams to explain basic principles. The book is aimed at medical students, critical care trainees and surgeons in training. However, the topics covered are not exclusive to intensive care medicine and many conditions relevant to the emergency physician are included. Trauma, toxicology and resuscitation topics are among the 272 cases contributed by an international group of 50 intensivists.

The authors set out to assist the reader in understanding what they call “the science and Gestalt of critical care medicine”. I take this to mean that the clinical formulation should be determined by interpretation in the appropriate context, taking account of any technical and other confounding variables. These aims are equally appropriate to emergency medicine but I’m not sure that they are achievable in a book of this size and type.

The format is a familiar one; questions are posed on one side of the page with answers and explanations overleaf. The quality of the images is above average and the subject matter is not overly esoteric. Answers are commendably brief and informative, allowing a wide range of material to be covered fairly quickly, though not in great depth.

Accessible is the best way to describe this book, light reading in quiet moments before postgraduate examinations in critical care, anaesthesia or accident and emergency medicine. When junior doctors wore white coats it would have been crammed into a pocket. Nowadays it will reside instead in numerous clinical areas or languish on a bedside table in the on call room. I recommend that if you come across it you should pick it up and have a read.

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When I picked up Trauma care I was expecting another run of the mill text on “how to resuscitate the multiply injured”. This expectation was shaken when the book fell open on the chapter “nutrition”. Now I know waiting times in A&E are long, but surely no one was advocating taking the breakfast trolley into “resus”? All was revealed however as I read on. The book takes a much wider view of trauma care than is normally experienced by those of us who work in A&E. It is not designed for the nurse wanting to know how to resuscitate the multiply injured, but instead is a text outlining the delivery of optimum trauma care from the moment of injury to the re-integration of the patient into the community. The approach is fresh and is enhanced by the use of case studies to illustrate the points being made. The most compelling thing is that a large amount of the book seems to be written from the perspective of the patient—the patient’s experience, the patient’s needs.

The chapters that focus on the psychological effects of trauma are perhaps the most sobering, but other elements of care that are important to the patient are also covered—elimination, tissue viability, hygiene and mouth care to name but a few. But don’t be fooled into thinking that this is purely a nursing textbook—the contributors come from all disciplines involved in the care and rehabilitation of the trauma patient, with many team members writing their own chapters in the book. The book is made complete with considerations in the final section of the actual service delivery and the staff that work within the service.
This book has succeeded in making me rethink my often narrow approach to trauma care, and I have set myself the task of recommending it to all the areas within the hospital that have contact with “trauma patients”. I hope that if I ever have the misfortune to be seriously injured I am looked after by people who have a similar patient orientated approach.

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Books received


CORRECTION
Mr R A Simpson should have been acknowledged as a scan coordinator for the Journal scan that appeared in the January 2001 issue of the journal (2001;18:71–3).

NOTICES
15th Annual Trauma Anesthesia and Critical Care Symposium
23–25 May 2002, Stavanger, Norway
Further details: e-mail: congress-secretary@traumacare2002.com, web site: www.traumacare2002.com, tel: +47 51 74 91 02, fax: +47 51 74 70 02.

18th Annual Scientific Meeting of the Australasian College for Emergency Medicine, incorporating the 22nd Annual Scientific Meeting of the Australasian Society for Emergency Medicine
30 September to 4 October 2001, Hobart, Tasmania
Further details: the programme of the meeting and details of how to register are at the web site www.cdesign.com.au/acem2001

World Congress on Drowning
26–28 June 2002, Amsterdam, the Netherlands
Further details: Congress Secretariat World Congress on Drowning 2002, Consumer Safety Institute, PO Box 75 169, 1070 Amsterdam, the Netherlands (tel: +31 20 511 45 14, fax: +31 20 511 45 10, e-mail: Secretariat@drowning.nl, web site: www.drowning.nl).