LETTERS TO THE EDITOR

Phyostigmine as treatment for severe CNS anticholinergic toxicity

EDITOR,—We report the successful use of phyostigmine to treat central anticholinergic toxicity: a use described before but rarely seen. We treated a patient admitted after an overdose of amrisulpride 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) but 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 phyostigmine. This was immediately 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. Referral to an ICU was considered but it was felt that the risks of paralysis, intubation and ventilation (and the possible need for interhospital transfer) outweighed those associated with phyostigmine treatment. This latter option had the advantage of therapeutic and diagnostic potential.

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Physostigmine is a tertiary ammonium compound that reverses anticholinergic effect via acetylcholineesterase 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.1 We do not propose that physostigmine 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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Physostigmine as treatment for severe CNS anticholinergic toxicity

EDITOR,—We report the successful use of phyostigmine to treat central anticholinergic toxicity: a use described before but rarely seen. We treated a patient admitted after an overdose of amrisulpride 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) but 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 phyostigmine. This was immediately 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. Referral to an ICU was considered but it was felt that the risks of paralysis, intubation and ventilation (and the possible need for interhospital transfer) outweighed those associated with phyostigmine 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 Phyostigmine is a tertiary ammonium compound that reverses anticholinergic effect via acetylcholineesterase 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.1 We do not propose that physostigmine 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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Pain in young children attending the accident and emergency department

EDITOR,—We read with interest the article by Macarthy et al.1 We too have experienced difficulties assessing and scoring children’s pain in the accident and emergency (A&E) setting. We feel that while subjective assessment has been shown to be the gold standard of pain assessment in some settings, for example, postoperative pain, the unexpected nature and anxiety associated with attendance at the A&E department makes this type of scoring invalid.

We have been working on developing an observational scale for the assessment of pain in children presenting to the A&E department.

We know from experience of auditing analgesic use in A&E that children who have a pain score allocated receive more analgesia in a more timely basis than those who do not.

Our pain score is loosely based on both the TPPS and CHEOPS score and relies on observations of various parameters in five categories (1) cry/vocal expression, (2) colour, (3) facial expression, (4) posture, (5) movement.

Each score receives a value of 0, 1 or 2 to give a maximum total of 10 (similar to the mechanism of an APgar score).

This score has been validated by medical students (Davis et al2) in the department and has shown to have good inter-rater reliability (Spearman’s rank correlation 0.82) and to have also significant construct validity when compared with patients who presented with other and examinations.

We feel that this score can be extended from the age of one year right through the paediatric population and not be just restricted to under fives, as we have experienced problems with subjective pain scoring in all age groups presenting to the department.

We endorse the suggestion that exploration of such pain scores in the A&E department should be actively pursued and intend to further validate our Alder Hey score against the modified TPPS score as the author suggests.

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Emergency medicine or accident and emergency?

EDITOR,—What is emergency medicine? Is it trauma, medical and paediatric emergency “emergency patients ...by specially trained doctors” (11 versus 6) are in fact to work in acute general medicine.6 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 pain relief in a 57 year old woman. This case is an 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.1

We recently used intranasal diamorphine as pain relief in a 57 year old woman. This woman suffered with chronic renal failure, and underwent frequent dialysis. She had fallen onto her right hand. Her right elbow was tender, swollen and deformed. She was supporting this elbow with her uninjured arm. 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. Radiographs

revealed a displaced four part supracondylar fracture of the right humerus with an intracapsular 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.6 The rectal route can be embarrassing and uncomfortable. Rapid analgesia may be necessary and the intranasal route also has delayed action.7 CPAP has been 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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The role of non-invasive ventilation in the emergency department

Editor,—Anthony Cross highlighted the effectiveness of non-invasive ventilation (NIV) in the emergency department in his review.1 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 also exists an article by Takeda et al from Tokyo.2 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 increasing 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 change our current treatment practice in UK emergency departments regarding acute pulmonary oedema. Admissions and death attributable to heart failure, unlike those attributable to chronic heart disease, are predicted to increase.7 CPAP has been 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.

Letters, Book reviews, Correction, Notices


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.

Cross also suggests that “early intervention [with NIV] to 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.


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

Evans—I would like to highlight an example of “speciality-specific 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 anatomy, 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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BOOK REVIEWS


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 junior doctors in the ICU.

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 the resuscitation unit? All was revealed however on reading the book. 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 it 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 hydration 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).