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) 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 nurse:patient ratio of 8:1), he was treated with 1 mg of phystostigmine. This was immediately and dramatically followed by a period of complete lucidity lasting 90 minutes. He did not become agitated again and his confusion settled. He did not require any antipsychotics (the ward to which he was admitted has a policy of not using antipsychotics, as it is felt that the risks of paralysus, 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.4 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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Pain in young children attending the accident and emergency department

Editor,—We read with interest the article by Macarthy et al.5 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 fashion than those who do not.

Our pain score is loosely based on both the TTPS’ 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 and Rostron) 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 conditions. 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 TPS score as the author suggests.

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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 accepted and potentially an acceptable and potentially important route for adults. It is rapidly absorbed from 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, and underwent frequent haemodialysis. 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. Radiographs


2 Royal Colleges of Physicians of the United Kingdom. Acute medicine: the physician’s role. A workplan by and for 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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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.5 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.1 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 nausea and vomiting which 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

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, 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: 2.4 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 is little evidence to support the conclusion that NIV is “extremely useful in an emergency department setting in the first line treatment of acute respiratory failure.” As Cross points out, most of the trials in this area have been performed in an intensive care or high dependency setting where the patients were selected by virtue that other treatments had failed. Many patients presenting to accident and emergency (A&E) with acute respiratory distress will get better after initiation of other treatments such as bronchodilators and controlled oxygen therapy (in the case of chronic obstructive airways disease (COAD)) or oxygen, nitrates and diuretics (in the case of acute pulmonary oedema). In a study of 954 COAD patients presenting to A&E in Leeds, only 25% were acidic on arrival and of these 25% had completely corrected their pH by the time they arrived on the ward.3 Similarly, in an audit of 104 patients presenting to A&E in Leeds with acute pulmonary oedema, only 6% were acidic on arrival (pH < 7.35). 89% improved both clinically and in terms of arterial pH before leaving A&E (unpublished data).

Cross points out that NIV has been shown to “decrease the need for endotracheal intubation”. However, these data are derived from

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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 the 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] can 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 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 patients 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 undoubtably 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

Evrtn—I would like to highlight an example of “speciality care in place” 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 anaesthetic gases administered, morbidity and mortality. Thus it covers not only the “flat unresponsive 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 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 of the data 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 bulging 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 well defined and end 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 and available data 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.

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Letters, Book reviews, Correction, Notices

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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
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).

Books received
Physostigmine as treatment for severe CNS anticholinergic toxicity

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