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 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. Referral to an ITU was considered but it was felt that this patient had the advantage of therapeutic and diagnostic basis of therapeutics (edition expires 30th September 2000).

Physostigmine is a tertiary ammonium compound that reverses anticholinergic effects. Its rapid onset of effect and duration of action of one to two hours.

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

We have been working on developing an observational tool for the assessment of pain in children to the department. We know from experience of auditing such pain scores in the A&E department 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.

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.

We 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 way of providing analgesia for children in severe pain. We believe 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, 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 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
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 an injection 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.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 if the cause of nausea 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

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, patients with 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 addition to decreasing 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 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.3 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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EDITOR,—We read with interest the review by Cross of the use of non-invasive ventilation (NIV) in the emergency department. We have reappraised the evidence concerning the effectiveness of NIV in treating respiratory failure and found further evidence that both supports its use and highlights current controversies.

Although Cross comments on individual studies and 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.2 This combined the data and found a significant reduction in intubation rates for those receiving CPAP (NNT=24) 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) in patients with LVF3 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 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, nitrites 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.4 Similarly, in an audit of 104 patients presenting to A&E in Leeds with acute pulmonary oedema whom were all 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...
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] 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 being 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 associated with the use of 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 machine 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.

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

Bronch— I would like to highlight an example of “speciality 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 anatomy, morbidity and mortality. Thus it covers not only the “flat overdoes 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 other junior doctors in surgery in the role as 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 contributors.

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 and experience 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 examination in critical care, anaesthesia or accident and emergency medicine. When junior doctors wore white coats it would have been crammed into a reeling 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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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
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).