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 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 the risks of paralysis, 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.1 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 to work in “emergency medicine”. Emergency medicine or accident and emergency department

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

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 study (in the absence of patient consent or approval) of referrals of patients to the Accident and Emergency 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 TPSS 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 Appar score).

This score has been validated by medical students (Davis et al) in the department and has shown to have good inter-rater reliability (Spearman’s rank correlation 0.82) and to have also significant constructive validity when compared with patients who presented with postoperative admissions. 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 TPSS 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 acceptable and potentially important route for 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.1

We recently used intranasal diamorphine as pain relief in a 57 year old woman. This woman suffered from chronic renal failure, and underwent frequent hemodialysis. 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

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

We 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

<table>
<thead>
<tr>
<th>Grade</th>
<th>Consultant</th>
<th>Non-consultant career grade</th>
<th>Training grade</th>
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<td></td>
<td>2</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.

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 “casuality”, belong firmly in the last millennium.

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We recently used intranasal diamorphine as pain relief in a 57 year old woman. This woman suffered from chronic renal failure, and underwent frequent hemodialysis. 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...
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 practice treatment in UK emergency departments regarding acute pulmonary oedema. Admissions and death attributable to heart failure, unlike those attributable to chronic heart disease, were predicted to increase. 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 is already 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 reappraised the evidence concerning the effectiveness of NIV in acute respiratory failure and found further evidence that both supports its use and highlights current controversies. Although Cross comments on individual studies he has not presented 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) treated with NIV had reduced intubation rates with a number needed to treat (NNT) of three and reduced mortality with an NNT of five. In the case of chronic obstructive pulmonary disease (COPD), CPAP improves oxygenation in patients with chronic obstructive pulmonary disease (COPD) 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 acidotic 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 who were all acidotic on arrival (pH < 7.35), 89% improved both clinically and in terms of arterial pH before leaving A&E (unpublished data). 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 based on severity of the disease. 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 pulmonary 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 acidotic on arrival and of these 25% had completely corrected their pH by the time they arrived on the ward. Similarly, in an audit of 104 patients presenting to A&E in Leeds with acute pulmonary oedema who were all acidotic on arrival (pH < 7.35), 89% improved both clinically and in terms of arterial pH before leaving A&E (unpublished data).

Takeda points out that NIV has been shown to “decrease the need for endotracheal intubation”4. However, these data are derived from studies he does not mention the meta-analysis This combined the data and found a significant reduction in intubation rates for those receiving CPAP (NNT=5) 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 LVF5 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 Pao, 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 28 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, and further study is needed to confirm its safety in these patients.

In our department we treat 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 intubation emergency department patients, and we welcome the attention drawn to this by the review article.
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. Some patients (particularly those with COAD) will have a prolonged requirement for NIV and it is, therefore, important to consider training in an NIV machine 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 training in an NIV machine 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.

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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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).