ORIGINAL ARTICLE

Paracetamol overdose: an evidence based flowchart to guide management

C I Wallace, P I Dargan, A L Jones

Emerg Med J 2002;19:202–205

A flowchart for the management of patients with paracetamol poisoning is presented to help clinicians in the emergency department.

Paracetamol is the commonest drug taken in overdose in the United Kingdom. While the management of early paracetamol poisoning is straightforward, the management of late presenting cases, cases presenting after a staggered overdose, and patients with risk factors for paracetamol poisoning can be much more complex. The authors have developed and present here an evidence based flowchart that will guide clinicians step by step through the investigation and treatment of all patients presenting to hospital after this common, but often difficult to manage overdose. As well as a management guideline this flowchart can be used as an educational tool.

BACKGROUND

Paracetamol is the commonest drug taken in overdose in the United Kingdom, accounting for 48% of all poisoning admissions to hospital and an estimated 100–200 deaths per year.^{1–6} However, junior doctors' knowledge about the management of paracetamol poisoning is poor.⁷

The management of patients who present early (less than 15 hours) after ingestion of a single paracetamol overdose is straightforward. If the patient has taken a potentially toxic dose of paracetamol, management is guided by the plasma paracetamol concentration; treatment with N-acetylcysteine in patients with a toxic plasma paracetamol concentration provides complete protection against paracetamol induced hepatotoxicity.⁸ However, when cases stray from this simple scenario (such as with staggered overdoses, patients with high risk factors for paracetamol poisoning, or late presentation), management decisions are more complex.^{9–11}

Current guidelines for paracetamol poisoning are based on the consensus recommendations of the UK National Poisons Information Service (NPIS), they have also been adopted by the Royal College of Paediatrics and Child Health as a Good Practice Consensus Statement. The guidelines have been circulated to all accident and emergency departments in the form of a poster in prose format and we support their use.¹²

Our aim is to provide an evidence based, easy to follow, and visually attractive management guideline for paracetamol poisoning, aimed at emergency and general physicians (particularly junior doctors) dealing with this common, but

often difficult to manage overdose. The flowchart that we present will guide the clinician through the management of a patient presenting with a paracetamol overdose in a stepwise fashion. It can also be used as an educational tool because it indicates the evidence (or lack of it) for each management step. It therefore augments the current NPIS paracetamol poster.¹² The flowchart is a guideline however, and not a protocol, and individual decisions will need to be made for every patient based on their particular circumstances.

METHODS

We conducted a literature search of Medline, Toxline, and Embase using the terms "paracetamol" and "acetaminophen" with "intoxication", "poisoning" and "overdose". No language was barred and no other limitations were placed. The retrieved abstracts were reviewed and the most pertinent articles were reviewed in more detail. In addition, we took into account the consensus recommendations from the UK Toxicology Group (National Poisons Information Service (NPIS), Paracetamol Information Centre, and British Association of Accident and Emergency Medicine) on which the current UK guidelines for the management of paracetamol poisoning are based. This evidence base was then used to construct an algorithmic flowchart to guide the clinician through the management of both simple and complex paracetamol poisoning in a stepwise fashion.

RESULTS

See figure 1 for the flowchart used to guide the management of patients with paracetamol poisoning, together with the supporting references from the literature.^{8–45}

The paracetamol flowchart is structured around a few crucial branchpoints in the following order. Is the patient presenting after a single or staggered overdose? What is the time after ingestion? What are the results to the relevant investigations? Based upon the results of these questions, the clinician is guided through the appropriate steps in investigation and treatment of the paracetamol overdose. So that the flowchart can be used as a stand alone tool to guide patient management we have included the standard UK plasma paracetamol treatment nomogram,^{8 12 37} together with information boxes on risk factors for paracetamol poisoning,^{13–19} doses of N-acetylcysteine^{8 40} and management of adverse reactions to N-acetylcysteine.^{41 42}

Correspondence to:
Dr A Jones, National
Poisons Information
Service, Guy's and St
Thomas' NHS Trust,
Avonley Road, London,
SE14 5ER, UK;
alison.jones@
gstt.sthames.nhs.uk

Accepted for publication
29 October 2001

Figure 1 Paracetamol overdose: a flowchart to guide management. (The numbers in superscripts relate to the supporting references).

DISCUSSION

Paracetamol remains the most common agent taken in overdose in the UK,^{4,5} but junior doctors' knowledge about the management of paracetamol poisoning remains poor,⁷ despite the availability of UK guidelines as a poster in prose format.¹² The management of paracetamol poisoning has been reviewed in detail elsewhere and these review articles complement our management flowchart for readers who wish to study the background literature in more detail.^{9,10,23,46}

A study by Hardern *et al.*⁴⁷ showed that the management of paracetamol poisoning is improved if staff have access to guidelines, but found no difference between the performance of prose and flowchart formats. However, two further studies, one in the US⁴⁸ and one in the UK⁴⁹ have shown that physicians prefer practice guidelines in the form of evidence based algorithms that are "user friendly". The management of paracetamol overdose entails multiple steps in both investigation and treatment. We feel that the presentation of these management decisions in the algorithmic, flowchart format that we present means that each step in the process can be focused upon separately (whereas the entire body of prose guidelines may need to be assimilated before understanding the individual steps). The management flowchart deals with both the well defined early cases and more complex cases such as patients with risk factors for paracetamol poisoning, staggered overdoses, and late presenters.

This flowchart will guide physicians through the management of the majority of patients presenting with a paracetamol overdose from the time of presentation to hospital to the time that they are medically fit for discharge. There are however situations where further advice tailored to the management of an individual patient from either a clinical toxicologist at the National Poisons Information Service, or a hepatologist at a liver transplant unit may be required and this has been indicated on the flowchart. This particularly applies to patients presenting either after a staggered paracetamol overdose or later than 24 hours after a single paracetamol overdose, where both the efficacy and mechanism of action of N-acetylcysteine are controversial.^{8,11,21} Patients with established hepatotoxicity, with markers of severe toxicity outlined in the flowchart, such as coagulopathy, should be discussed early with a hepatologist, as meticulous supportive care is critical to a good outcome in such cases.^{43,50}

CONCLUSION

Paracetamol is by far the commonest substance involved in self poisoning in the UK. While the management of early paracetamol poisoning is straightforward, the management of late presenting cases, cases presenting after a staggered overdose and patients with risk factors for paracetamol poisoning can be much more complex. We have developed an evidence based, easy to follow management guideline in the form of a flowchart that will guide clinicians step by step through the investigation and treatment of all patients presenting to hospital after a paracetamol overdose.

ACKNOWLEDGEMENT

The authors would like to thank Professor Laurie Prescott and Dr Alex Proudfoot for reviewing the flowchart and for their helpful comments.

Contributors

Craig Wallace and Paul Dargan were responsible for the literature review and designed the management flowchart. Alison Jones reviewed the literature review and the management flowchart. All three authors were involved in the writing of the paper and all three authors will act as guarantors.

Authors' affiliations

C I Wallace, P I Dargan, A L Jones, National Poisons Information Service, Guy's and St Thomas' NHS Trust, London, UK

Conflicts of interest: AJ has acted as an advisor to Glaxo Smith Kline, Cumberland Pharmaceuticals. Oxford Pharmaceuticals support the outreach educational activity of the London Centre of the National Poisons Information Service. AJ and PD have acted as advisors to Orphan Drugs (Europe) and have received funding to attend meetings from Glaxo Smith Kline.

REFERENCES

- 1 Fagan E, Wana G. Reducing paracetamol overdoses. *BMJ* 1996;**313**:1417–18.
- 2 Atcha Z. Paracetamol related deaths in England and Wales, 1993–1997 (Office for National Statistics). *Health Stat Q* 2000;**1**:5–9.
- 3 Gunnel D, Hawton K, Murray V *et al.* Use of paracetamol for suicide and parasuicide in the UK and France: are restrictions on availability justified? *J Epidemiol Community Health* 1997;**51**:175–9.
- 4 Thomas SHL, Horner JE, Chew K *et al.* Presentation of poisoned patients to accident and emergency departments in the North of England. *Hum Exp Toxicol* 1996;**15**:466–70.
- 5 Bialas MC, Reid PG, Beck P *et al.* Changing patterns of self-poisoning in a UK health district. *Q J Med* 1996;**89**:893–901.
- 6 Hawton K, Fagg J. Trends in deliberate self poisoning and self injury in Oxford, 1976–1990. *BMJ* 1992;**304**:1409–11.
- 7 Hulbert DC, Bray GP, Beckett MW. The management of paracetamol poisoning by junior doctors. *J Accid Emerg Med* 1995;**12**:66–7.
- 8 Prescott LF, Illingworth RN, Critchley JA *et al.* Intravenous N-acetylcysteine: the treatment of choice for paracetamol poisoning. *BMJ* 1979;**2**:1097–100.
- 9 Vale JA, Proudfoot AT. Paracetamol (acetaminophen) poisoning *Lancet* 1995;**346**:547–52.
- 10 Prescott LF. Paracetamol overdosage: pharmacological considerations and clinical management. *Drugs* 1983;**23**:290–314.
- 11 Jones AL. Mechanism of action and value of N-acetylcysteine in the treatment of early and late acetaminophen poisoning: a critical review. *J Toxicol Clin Tox* 1998;**36**:277–85.
- 12 NPIS. *Management of acute paracetamol poisoning*. Guidelines agreed by the UK National Poisons Information Service 1998. Supplied to Accident and Emergency Centres in the United Kingdom by the Paracetamol Information Centre in collaboration with the British Association for Accident and Emergency Medicine.
- 13 Prescott LF. Paracetamol, alcohol and the liver. *Br J Clin Pharmacol* 2000;**49**:291–301.
- 14 Bray GP, Mowat C, Muir DF *et al.* The effect of chronic alcohol intake on prognosis and outcome in paracetamol overdose. *Hum Exp Toxicol* 1991;**10**:435–8.
- 15 Wright N, Prescott LF. Potentiation by previous drug therapy of hepatotoxicity following paracetamol overdose. *Scott Med J* 1973;**18**:56–8.
- 16 Smith JA, Hine ID, Beck P *et al.* Paracetamol toxicity: is enzyme induction important? *Hum Exp Toxicol* 1986;**5**:383–5.
- 17 Minton N, Henry JA, Frankel RJ. Fatal paracetamol poisoning in an epileptic *Hum Exp Toxicol* 1988;**7**:33–4.
- 18 Buhl R, Jaffe HA, Halroyd KA, *et al.* Systemic glutathione deficiency in symptom-free HIV-seropositive individuals. *Lancet* 1989;**ii**:1294–8.
- 19 Whitcomb DC, Block GD. Association of acetaminophen hepatotoxicity with fasting and ethanol use. *JAMA* 1994;**272**:1845–50.
- 20 Wendel A, Jaeschke H, Gloger M. Drug induced lipid peroxidation in mice – protection against paracetamol induced liver necrosis by intravenous liposomally entrapped glutathione. *Biochem Pharmacol* 1982;**31**:3601–5.
- 21 Prescott LF. *Paracetamol (acetaminophen). A critical bibliographic review*. London: Taylor and Francis, 1996.
- 22 Linden CH, Rumack BH. Acetaminophen overdose. *Emerg Clin North Am* 1984;**2**:103.
- 23 Prescott LF, Critchley JA. The treatment of acetaminophen poisoning. *Ann Rev Pharmacol Toxicol* 1983;**23**:87–101.
- 24 Henretig FM, Selbst SM, Forrest C, *et al.* Repeated acetaminophen overdosing causing hepatotoxicity in children: clinical reports and literature review. *Clin Pediatr* 1989;**28**:525–8.
- 25 Agran PF, Zenk KE, Romanowsky SG. Acute liver failure and encephalopathy in a 15 month old infant. *Am J Dis Child* 1983;**137**:1107–14.
- 26 Swetnam SM, Florman AL. Probable acetaminophen toxicity in an 18-month old infant due to repeated overdosing. *Clin Pediatr* 1984;**23**:104–5.
- 27 Smith DW, Isakson G, Frankel R, *et al.* Hepatic failure following ingestion of multiple doses of acetaminophen in a young child. *J Pediatr Gastroenterol Nutr* 1986;**5**:822–5.
- 28 Douidour SM, Ali-Khali I, Hakersang RW. Severe hepatotoxicity, acute renal failure and pancytopenia in a young child after repeated acetaminophen overdosing. *Clin Pediatr* 1994;**33**:42–5.
- 29 American Academy of Clinical Toxicology; European Association of Poison Control Centres and Clinical Toxicologists. Position statement: single-dose activated charcoal. *J Toxicol Clin Toxicol* 1997;**35**:721–41.
- 30 Buckley NA, Whyte IM, O'Connell DL, *et al.* Activated charcoal reduces the need for N-acetylcysteine treatment after acetaminophen (paracetamol) overdose. *J Toxicol Clin Toxicol* 1999;**37**:753–7.

- 31 **Singer AJ**, Carracio TR, Mofenson HC. The temporal profile of increased transaminase levels in patients with acetaminophen induced liver dysfunction *Ann Emerg Med* 1995;**26**:49–53.
- 32 **Harrison PM**, O'Grady JG, Keays RT, *et al.* Serial prothrombin time as a prognostic indicator in paracetamol induced fulminant hepatic failure *BMJ* 1990;**301**:964–6.
- 33 **Record CO**, Iles RA, Cohen RD, *et al.* Acid-base and metabolic disturbances in fulminant hepatic failure *Gut* 1975;**16**:144–9.
- 34 **Blantz RC**. Acetaminophen, acute and chronic effects on renal function. *Am J Kidney Dis* 1996;**28**:S3–6.
- 35 **Eguia L**, Materson BJ. Acetaminophen-related acute renal failure without fulminant liver failure. *Pharmacotherapy* 1997;**17**:363–70.
- 36 **Prescott LF**, Proudfoot AT, Creegen RJ. Paracetamol induced acute renal failure in the absence of fulminant liver damage. *BMJ* 1982;**284**:421–2.
- 37 **Rumack BH**, Matthew H. Acetaminophen poisoning and toxicity. *Pediatrics* 1975;**55**:871–6.
- 38 **Keays R**, Harrison PM, Wendon JA, *et al.* Intravenous acetylcysteine in paracetamol-induced fulminant hepatic failure: a prospective controlled trial. *BMJ* 1991;**303**:1026–9.
- 39 **Harrison PM**, Keays R, Bray GP, *et al.* Improved outcome of paracetamol induced fulminant hepatic failure by late administration of acetylcysteine. *Lancet* 1990;**335**:1572–3.
- 40 The Medicines Committee of the Royal College of Paediatrics and child Health and Neonatal and Paediatric Pharmacists Group. *Medicines for Children* 1999. London: Royal College of Paediatrics and Child Health Publications, 1999.
- 41 **Dawson AH**, Henry DA, McEwen J. Adverse reactions to N-Acetylcysteine during treatment for paracetamol poisoning. *Med J Aust* 1989;**150**:329–31.
- 42 **Bailey B**, McGuigan MA. Management of anaphylactoid reactions to intravenous N-Acetylcysteine. *Ann Emerg Med* 1998;**31**:710–15.
- 43 **Makin A**, Williams R. The current management of paracetamol overdosage. *Br J Clin Pharmacol* 1994;**48**:144–8.
- 44 **Prescott LF**, Wright N, Roscoe P, *et al.* Plasma paracetamol half-life and hepatic necrosis in patients with paracetamol overdosage. *Lancet* 1971;i:519–22.
- 45 **Gazzard BG**, Hughes RD, Widdop B, *et al.* Early prediction of the outcome of a paracetamol overdose based on an analysis of 163 patients. *Postgrad Med J* 1977;**53**:243–47.
- 46 **Makin A**, Williams R. The current management of paracetamol overdosage. *Br J Clin Pharmacol* 1994;**48**:144–8.
- 47 **Hardern RD**, Hodgson LC, Hamer DW. Flow diagram or prose: does the format of practice guidelines matter? *Eur J Emerg Med* 1998;**5**:241–4.
- 48 **Stone TT**, Kivalahan CH, Cox KR. Evaluation of physician preferences for guideline implementation. *Am J Med Qual* 1999;**14**:170–7.
- 49 **Hardern RD**, Hampshaw S. What do accident and emergency medical staff think of practice guidelines. *Eur J Emerg Med* 1997;**4**:68–71.
- 50 **O'Grady JG**, Wendon J, Tan KC, *et al.* Liver transplantation after paracetamol overdose. *BMJ* 1991;**303**:221–3.

PostScript

LETTERS

Diagnostic errors in an accident and emergency department

I commend the author for comprehensively investigating a complex area but found some important pieces of information missing in the study.¹ Firstly, there is no information regarding the total number of patients seen in the accident and emergency (A&E) department during the study period. This information would put into better perspective the number of patients (934) who had recorded diagnostic errors and would allow for more scientifically valid comparison of the findings of this study by other A&E departments. Secondly, there is no record of the number of cases where there was dispute over the diagnosis between A&E clinician and radiologist. Furthermore, it seems the author *alone* made the final decision regarding the diagnosis in such cases. This is a very subjective method of diagnosis with little scientific validity. Moreover, there is no information as to the specific diagnosis made and subsequent management of this group of patients. The management of this subset of cases is a dilemma for A&E clinicians and more information from the author on their management will be informative. Finally, hospital policy for reporting A&E radiographs changed during the study period. Did this have any effect on the number of diagnostic errors recorded? Data comparing the number of diagnostic errors before and after the change of policy to immediate reporting of radiographs would provide useful scientific evidence for radiologists to decide whether to give priority to A&E radiographs.

A Wakai

Department of Accident and Emergency Medicine,
Beaumont Hospital, Dublin 9, Ireland

References

- 1 Guly HR. Diagnostic errors in an accident and emergency department. *Emerg Med J* 2001;18:263-9.

Author's reply

I thank Dr Wakai for his comments. The total number of new patients seen over the four year period in which this study took place was 244 442. I have no record of the number of cases where there was dispute over the diagnosis between A&E clinician and radiologist, but the number was very small, and usually related to a radiology trainee, rather than a consultant radiologist. The subsequent management of patients in whom diagnostic errors had been made was left to the individual consultant and I have no specific data on this but it obviously varied with the severity of the diagnostic error and the circumstances in which the error was discovered. Clearly, if the diagnostic error was discovered when the patient reattended the A&E department, or a follow up clinic, it was dealt with there and then, but if an error was discovered by a radiological report, probably

most patients were sent an appointment to reattend one of the A&E clinics, though some patients would have been telephoned and asked to return immediately. For very minor errors, for example, minor avulsion fractures, the GP would have been informed that the patient would not have been advised to return.

The change in radiological reporting that occurred part of the way through the study was, of course, only one change that occurred over the four year period. There were also changes in staffing and as the idea behind the original collection of data was for continual quality improvement, the results of the study each six months led to changes in teaching, etc. For what it is worth, the incidence of diagnostic errors appeared to fall for the 12 months after the introduction of hot reporting, but subsequently rose again. It is difficult to attribute this completely to the change in radiological reporting. In addition, as the study notes, it proved very difficult to obtain details of every diagnostic error and the data are certainly incomplete. I am not sure that conclusions on the effectiveness of changing the radiological reporting system based on incomplete data would be scientifically valid.

Dr Wakai rightly states that diagnosis based on the opinion of a single person is not valid. To this must be added the difficulties in defining diagnostic error and the incompleteness of the data.

With a relatively low incidence of diagnostic errors, a study to accurately determine the incidence of these and to draw scientifically valid conclusions about their types, causes, etc, would require 100% follow up of many thousands of patients with all potential diagnostic errors being submitted to a panel to determine the exact diagnosis. Such a study would be very expensive and has never been done.

My study was, I hope, more than just "one consultant's experience of diagnostic errors he has encountered", as I actively tried to seek out all diagnostic errors as part of a quality improvement exercise. It must be regarded as a best attempt at determining all diagnostic errors for audit purposes but with no additional resources allocated. As such, I hope that it will be useful when discussing quality of service in A&E departments, but it did not

accurately define every diagnostic error that occurred over the four year period.

H R Guly

Emergency Department, Derriford Hospital,
Derriford, Plymouth, PL6 8DH, UK

Prehospital rapid sequence intubation

We read with interest the recent paper by Mackay and colleagues regarding the safety of prehospital rapid sequence induction by emergency physicians¹ and would like to make two observations. Despite grading more of the patients as Cormack-Lehane 1 and 2 (95% compared with 81.5% in the emergency physician group) the anaesthetists were still using the gum elastic bougie more often (60.4% versus 51.0%). The use of the Cormack-Lehane scoring system is not necessarily predictive of intubation difficulty. Pre-hospital evaluation of intubation in France has showed that glottic exposure alone is an incomplete reflection of the difficulty encountered. In fact using a seven point scoring system, the influence of glottic visualisation was only moderate when assessing the subsequent degree of difficulty of intubation.² Given that this is the case then should the use of an aid to intubation, such as the gum elastic bougie be part of the standard operating procedure for prehospital intubation? This may further reduce the number of repeat attempts at intubation, which the authors themselves comment as probably being under-reported in the study.

The authors also state that the laryngeal mask airway is not routinely carried. This is surprising given that, as an airway adjunct, while not providing protection from gastric aspiration, it may be available to provide oxygenation in circumstances where the provision of a definitive airway may be difficult. Its potential role in the prehospital setting should not be overlooked.³

P J Shirley, D Pogson

Intensive Care Unit, Royal Adelaide Hospital, North
Terrace, Adelaide, SA 5000, Australia

References

- 1 Mackay CA, Terris J, Coats TJ. Prehospital rapid sequence induction by emergency physicians: is it safe? *Emerg Med J* 2001;18:20-4.
- 2 Adnet F, Borron SW, Racine SX, et al. The intubation difficulty scale (IDS) Proposal and evaluation of a new score characterising the complexity of endotracheal intubation. *Anesthesiology* 1997;87:1290-7.
- 3 Sasada MP, Gabbott DA. The role of the laryngeal mask airway in pre-hospital care. *Resuscitation* 1994;28:97-102.

Authors' reply

We thank the authors of this letter for their comments. While we accept that simply grading the view at laryngoscopy is not the only factor predicting difficulty of intubation, it is convenient and well understood and may reflect potential problems.¹ We agree that a gum elastic bougie should be used as a routine to aid prehospital intubation.

If you have a burning desire to respond to a paper published in *EMJ*, why not make use of our "rapid response" option?

Log on to our web site (www.emjonline.com), find the paper that interests you, and send your response via email by clicking on the "eLetters" option in the box at the top right hand corner. Providing it isn't libellous or obscene, it will be posted within seven days. You can retrieve it by clicking on "read eLetters" on our homepage. The editors will decide as before whether to also publish it in a future paper issue.

A laryngeal mask airway may certainly have a role as a backup device, but is not always easy to insert, particularly in the multiply injured patient requiring cervical stabilisation.² Comparative studies are required to determine the best approach to a failed prehospital intubation.

C Kelly (Mackay), T Coats

Helicopter Emergency Medical Service, Royal London Hospital, Whitechapel, London, UK; cathy.kelly@luht.scot.nhs.uk

References

- 1 Cook TM. A new practical classification of laryngeal view. *Anaesthesia* 2000;**55**:274–9.
- 2 Gabbot DA, Sasada MP. Laryngeal mask airway insertion using cricoid pressure and manual in line neck stabilisation. *Anaesthesia* 1995;**50**:674–6.

Intranasal naloxone for life threatening opioid toxicity

Heroin overdose is a major cause of death in Western countries. Many lives are saved by the administration of naloxone by emergency department and ambulance staff. In Australia, there have recently been calls by drug and alcohol dependence agencies and coroners for the extension of this treatment to other emergency service and community workers. Parenteral administration of naloxone however has some problems. It entails administration by way of an injection, mandating training of personnel and secure storage of equipment. There is also risk of transmission of blood-borne diseases such as hepatitis C to the treating person by way of needlestick injuries.

Currently available pharmacology data suggest that naloxone has high bioavailability through the nasal mucosa, with onset of action and plasma bioavailability curves that are very similar to the intravenous route.¹ Work in the field of drug addiction has shown that intranasal naloxone is effective in detection of opioid dependence² and is as effective as parenteral naloxone for the reversal of opioid effects.³ To date, the intranasal administration of naloxone for the emergency treatment of opioid overdose has not been reported in the literature.

Six cases of isolated acute heroin overdose were treated with intranasal naloxone, in addition to ventilatory support, in the Department of Emergency Medicine of Western Hospital, Melbourne, Australia. All patients had return of adequate spontaneous respiration within two minutes, with a median of 50 seconds (table 1). Doses used ranged from 0.8 to 2 mg and were at the treating doctor's discretion.

If intranasal administration of naloxone could be shown in larger series to be effective and practical, there is the potential to extend

this treatment to a wide variety of community workers without the risk of needlestick injury and with minimal training. This may well translate into an increase in lives saved.

A prospective clinical trial comparing the effectiveness and safety of the intranasal route for administration of naloxone to the intramuscular route in the prehospital setting is planned to begin in December 2001.

A-M Kelly

Joseph Epstein Centre for Emergency Medicine Research and Department of Emergency Medicine, Western Hospital, Australia and The University of Melbourne, Australia

Z Koutsogiannis

Joseph Epstein Centre for Emergency Medicine Research and Department of Emergency Medicine, Western Hospital, Australia

Correspondence to: Professor A-M Kelly, Joseph Epstein Centre for Emergency Medicine Research, Western Hospital, Private Bag, Footscray 3011, Australia; Anne-Maree.Kelly@wh.org.au

References

- 1 Hussain AA, Kimura R, Huang CH. Nasal absorption of naloxone and buprenorphine in rats. *Int J Pharmacol* 1984;**21**:233.
- 2 Loimer N, Hofmann P, Chaudhry HR. Nasal administration of naloxone for detection of opiate dependence. *J Psychiatr Res* 1992;**26**:39–43.
- 3 Loimer N, Hofmann P, Chaudhry HR. Nasal administration of naloxone is as effective as the intravenous route in opiate addicts. *Int J Addict* 1994;**29**:819–27.

Anti-D immunoprophylaxis within the accident and emergency department

The debate on anti-D prophylaxis rages on. Recently the subject was discussed in a green top guideline from the Royal College of Obstetricians and Gynaecologists.¹ There are still approximately 50 deaths per annum attributable to rhesus isoimmunisation in the UK. In reviewing the reasons why these deaths still occur, the Consensus Conference on Anti D in 1997 admitted that the 1991 Recommendations are not being adhered to by all units and that a substantial proportion of accident and emergency (A&E) departments did not administer anti-D when appropriate (Consensus Conference on Anti-D Prophylaxis, Edinburgh, UK 8–9 April, 1997).

The conference discussed but did not conclude on the need for anti-D prophylaxis where threatened miscarriage and resolution occurs in the first trimester, or when spontaneous miscarriage occurs at this time without instrumentation. The College guidelines go further in advocating non-use of anti-D when pregnancy bleeding occurs in the first trimester with a viable fetus and supports the use of anti-D when "bleeding is heavy or repeated, when abdominal pain is present or when gestation approaches 12 weeks".

There is a need here for more precision. Many SHOs in A&E have limited gynaecological experience and under the new guidelines will be expected to determine which patients require anti-D.

Furthermore, the present recommendation for non-use of anti D is based largely on two observational studies, (Grade C recommendation). In this era of evidence based medicine is this sufficient basis for a change in policy?

In the past anti-D immunoprophylaxis was routinely given to all rhesus negative women

with early pregnancy bleeding. This has not been shown so far to be significantly associated with adverse side effects and the cost implications are not prohibitive.

Perhaps the way forward is shown in a more recent RCOG guideline, on the management of early pregnancy loss.² The same dilemma is dealt with in a caveat "if there is clinical doubt then anti D should be given". Until more conclusive information is to hand, rather than obfuscating the issue, a return to a policy of administering anti-D to all rhesus negative women with early pregnancy bleeding seems a more plausible option.

M J Kavanagh, T Dada

Accident and Emergency and Obstetrics and Gynaecology Departments, St James's University Hospital, Beckett Street, Leeds LS 9 7TF, UK

References

- 1 Royal College of Obstetrics and Gynaecology Guidelines. Use of anti-D immunoglobulin for Rh prophylaxis. 2000 (www.rcog.org.uk/guidelines/antid.html)
- 2 Royal College of Obstetrics and Gynaecology Guidelines. Management of early pregnancy loss. 2000 (www.rcog.org.uk/guidelines25.html)

Teaching and learning

We read with interest the paper by Dr Lockey describing the different learning approaches that may be taken by students.¹ We are aware that the field of educational psychology is woolly and littered with many definitions and it may be difficult to give a brief overview of learning approaches. The author has made a valid point in suggesting that as doctors we are expected to teach but are rarely trained in the teaching process. The author goes on to describe how there are essentially two learning approaches adopted by students: "surface" and "deep". We are then told how deep learning is superior to surface and that as educators we should attempt to promote deep learning.

This is fine. However, Dr Lockey has made an important omission in his paper. The author has failed to describe a third and very important learning approach. That is the "strategic" approach as described by Miller and Partlett.²

The strategic learner is a success driven person who approaches the learning process as a game where a high mark is the end point. These people will focus only on what they perceive to be relevant to exam success and disregard additional information. They may attempt exam prediction or even attempt to obtain inside information from authority figures. This approach results in poor long term recall and patchy subject knowledge. McManus *et al* have shown that medical students with the most clinical experience do not perform best in final exams but deep and strategic approaches do correlate well with exam success.³ The worry here is that as medical students these people may flourish in exams but as clinicians lack the knowledge base or understanding to work safely or effectively.

R McLaughlin

Emergency Department, Royal Victoria Hospital, Grosvenor Road, Belfast BT12 6BA, UK

R Bell

Radiology Department, Royal Victoria Hospital

References

- 1 Lockey A. Teaching and learning. *Emerg Med J* 2001;**18**:451–2.

Table 1

Patient	Dose IN naloxone	Time to spontaneous respiration
1	0.8 mg	40 seconds
2	1.6 mg	2 minutes
3	1.6 mg	30 seconds
4	2 mg	1 minute
5	1.6 mg	90 seconds
6	0.8 mg	30 seconds

- 2 Entwistle N. *Styles of teaching and learning*. Manchester: Wiley, 1981.
- 3 McManus I, Richards P, Winder B, *et al*. Clinical experience, performance in final examinations, and learning style in medical students: prospective study. *BMJ* 1998;**316**:345–50.

Factitious hypoglycaemia in hypotension

Capillary blood glucose evaluation is routinely performed on patients presenting to the accident and emergency department. However, the limitations of this test are not widely known. We recently cared for a shocked patient who was hypoglycaemic (capillary glucose 1.3 mmol/l, venous laboratory glucose 2.3 mmol/l) on presentation. He was treated with repeated boluses of intravenous glucose and a single dose of intravenous glucagon (1 mg) as capillary blood samples remained hypoglycaemic. With continued resuscitation a further venous glucose sample revealed his formal blood sugar to be increased (30.8 mmol/l) while capillary levels were still in the hypoglycaemic range (1.8 mmol/l). We were unaware of the possibility of inaccuracy in this situation and discussion with colleagues revealed a similar lack of awareness.

Atkin *et al*¹ showed in a prospective study of hypotensive (systolic blood pressure <80) patients in the emergency department that

32% of patients were incorrectly diagnosed as hypoglycaemic by finger stick measurements. Indeed, on laboratory measurement of venous samples, two patients were hyperglycaemic. They recommended that venous blood samples measured with glucose reagent strips should be the preferred method of bedside blood glucose estimation in hypotensive patients as these results were comparable to laboratory values. The reason for the discrepancy between capillary blood glucose measurements and venous blood glucose measurements remains unclear. It has been proposed that, in the shocked patient, both peripheral vasoconstriction causing shunting of blood from the periphery and continued peripheral consumption lead to decreased capillary blood glucose concentrations.

While the risks of hypoglycaemia are widely appreciated, it is becoming increasingly recognised that hyperglycaemia is not desirable and may indeed worsen outcome.² The mechanism involved is uncertain but is probably related to increased cellular lactic acid production.³

Hypotension is frequently encountered in acutely ill patients and the limitations of a routinely used test need to be recognised and highlighted.

A MacDuff, I S Grant

Western General Hospital, Crewe Road South,
Edinburgh EH4 2XU, UK

References

- 1 Atkin SH, Dasmahapatra A, Jaker MA, *et al*. Fingerstick glucose determination in shock. *Ann Intern Med* 1991;**114**:1020–4.
- 2 Voll CL, Auer RN. The effect of postischaemic blood glucose levels on ischaemic brain damage in the rat. *Arch Neurol* 1988;**24**:658–46.
- 3 Myers RF, Yamaguchi M. Effects of serum glucose concentration on brain response to circulatory arrest. [Abstract]. *J Neurol Pathol Exp Neurol* 1976;**35**:301.

CORRECTION

An editorial error occurred in this article by Dr Wallace and others (2002;**19**:202–5). In the flowchart, along the staggered overdose pathway, all doses should be described on a dose/kg/day and not a dose/kg basis. Also, patients who present after a paracetamol overdose with an unknown quantity of paracetamol should definitely be treated as though they may have taken a potentially hepatotoxic dose. The correct version of the flowchart is available on the journal web site (www.emjonline.com).