PostScript

LETTERS

Is morphine indicated in acute pulmonary oedema

Recent referrals to our intensive care unit have led us to question the indication for morphine in acute pulmonary oedema. Acute pulmonary oedema is a common, life-threatening emergency. Appropriate prompt therapy can provide rapid improvements in symptoms by reducing pre-load and after-load, or increasing myocardial contractility. Oxygen, loop diuretics, and nitrates are well-established therapeutic options. Most textbooks of acute medicine^{1,2} also recommend that intravenous morphine (or diamorphine in the UK) is given to "cause systemic vasodilatation and sedate the patient",² despite the absence of evidence supporting its efficacy. Treatment with morphine may be associated with respiratory depression in an already hypoxic patient, potentially exacerbating cardiac insufficiency. Respiratory failure secondary to opiates in pulmonary oedema has previously been reported elsewhere.³

In vivo experiments have confirmed that intravenous morphine results in significant peripheral vasodilatation and reduction in systemic vascular resistance.⁴ Further studies reveal that these effects are mediated via histamine receptors rather than opiate receptors however,⁵ and directly correlate with the rise in plasma histamine concentrations associated with morphine administration.⁶

In view of the potential iatrogenic morbidity and non-specific pharmacological action of morphine in acute pulmonary oedema, we question the recommendation of its use. There are more potent vasodilators available without the side-effects of respiratory depression. We suggest that it is only used in acute pulmonary oedema, with caution, when analgesia is required in association with acute myocardial infarction. The use of titrated intravenous diuretics and nitrates to promote vasodilatation is preferable.

Matthew Hall, Richard Griffiths, Bal Appadu
Peterborough District Hospitals, Cambs, UK

Correspondence to: Matthew Hall; drmatthall@hotmail.com

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References

- 1 Hope RA, Longmore JM, Hodgetts TJ, et al. Oxford handbook of clinical medicine. Third Edition. Oxford University Press, 1997.
- 2 Kumar P, Clark M. *Clinical Medicine*, Fourth Edition. W.B.Saunders, 1999.
- 3 Chambers JA, Baggeley CJ. Pulmonary oedema-prehospital treatment. Caution with morphine dosage. *Medical Journal of Australia* 1992;157:326.
- 4 Vismara LA, Leaman DM, Zelis R. The effects of morphine on venous tone in patients with acute pulmonary oedema. *Circulation* 1976;54:335.
- 5 Grossmann M, Abiose A, Tangphao O, et al. Morphine-induced venodilation in humans. *Clinical Pharmacology and Therapeutics* 1996;60:554.

6 Rosow CE, Moss J, Philbin DM, et al. Histamine release during morphine and fentanyl anesthesia. *Anesthesiology* 1982;56:93.

Emergency rooms differ in the detail

I read with interest the article by Schull.¹ I have recently moved to Trinidad and find that the problems in A&E are the same as the UK: overcrowding, waiting times, lack of facilities of trained staff. Each of these problems differ in detail.

Overcrowding and waiting times are less severe in Trinidad than the UK. In my department (a paediatric facility seeing 40,000 patients per year) our average time to see a doctor is less than half an hour. Is this a reflection of good practice? In most departments in Trinidad, staffing is at a junior level. Doctors in the Emergency Room provide limited care for patients before referral. This leads to shorter waiting times, but patients suffer through multiple referrals before receiving definitive care. This is more in the adult departments, where the average waiting time is less than that quoted, while the admission rate is higher (40% for adult departments compared to 10% for the children's hospital). Quicker care is not necessarily better care.

The availability and use of inpatient facilities has an impact on throughput. In most departments in Trinidad, overcrowding on the wards is a part of life. Space is 'made' on wards by accommodating patients two to a bed, or making room for trolleys. The only area in which this policy is not feasible is ICU. The availability of ICU beds is much less than in developing countries and threshold for admission much higher.

Finally, staffing is a problem. Juniors with no specific interest in Emergency Medicine staff most departments. An audit of our paediatric emergency room suggests that senior staff can reduce both the admission rate and waiting time, but patients stay longer while receiving more comprehensive care.

In summary, the problems of all Emergency Rooms are similar, but vary in detail. Achieving better waiting times in the Emergency Room may be at the expense of the quality of care in the entire system, if managed in isolation.

I Sammy

Department of Clinical Surgical Science, University of the West Indies, Trinidad and Tobago; psam@tsstt.net.tt

Reference

- 1 Schull MJ. Sex, SARS, and the Holy Grail. *Emerg Med J* 2003;20:400-401.

Glucagon use in β blocker overdose

The *Best Evidence Topic Reports* series is intended to provide evidence-based answers to clinical questions. The recent best evidence topic report by Boyd¹ concluded that there is not enough evidence to support the use of glucagon in β blocker overdose. However, clinical toxicology is an area in which the

evidence basis is often lacking and one therefore needs to rely on a combination of practical experience, case reports and assessment of biological plausibility. There is a sound theoretical basis for the use of glucagon in the cardiovascularly compromised patient who has taken a β blocker overdose. Glucagon activates adenylyl cyclase and exerts an inotropic and chronotropic effect² by a pathway that bypasses the β receptors.

Each of us has personal experience of the dramatic improvement in cardiovascular parameters that can occur following the administration of glucagon in this clinical situation.

Patients seldom take an overdose solely of a β blocker and the purist evidence base sought by Boyd is unlikely to be achievable. There is a wealth of clinical experience in support of administration of glucagon. Nobody would suggest that naloxone should not be used for opiate overdose yet the evidence base for its use is as flimsy as that of glucagon in β blocker overdose. We suggest that to attempt to undertake a randomised clinical trial of the use of glucagon in the compromised β blocker overdosed patient would be unethical.

By acting on the recommendation of this best evidence topic report, the unwary reader may deny patients a potentially life-saving treatment, which is universally recommended by toxicologists.²⁻⁴

Niall O'Connor

Our Lady of Lourdes Hospital, Drogheda, Ireland

Shaun Greene

Emergency Department, Guys and St Thomas' NHS Trust

Paul Dargan, Alison Jones

National Poisons Information Service, Guys and St Thomas' NHS Trust

Correspondence to: Niall O'Connor; niall.oconnor@maile.hse.ie

References

- 1 Boyd R, Ghosh A. Glucagon for the treatment of symptomatic β blocker overdose. *Emerg Med J* 2003;20:266-7.
- 2 Vale A. β Blockers. *Medicine* 1999;27(4):27.
- 3 Jones AL, Dargan PI. *Churchill's Pocketbook of Toxicology*. London: Harcourt Publishers, 2001:39.
- 4 White CM. A review of potential cardiovascular uses of intravenous glucagon administration. *J Clin Pharmacol* 1999;39:442-7.

Survey of blood gas interpretation

Hospital clinicians frequently request arterial blood gas (ABG) analysis to aid in the diagnosis and management of patients.

We carried out a one-day, survey to see how well ABG's were interpreted. We asked 66 participants to complete a written questionnaire during their normal working duties. No one declined to take part. Respondents were asked to give the normal ranges for ABG parameters. Five different ABG results were presented and respondents asked to describe (free text) the findings and to give any number

Table 1 Specialty and grade of respondents

Specialty	Cons/Mid Grade	SHO/HO
Anaesth	3	
Emergency Dept	5	8
Medicine	7	15
Orthopaedics	2	3
Psychiatry		1
Radiology	7	
Surgery	6	9

of differential diagnoses for each (free text). Responses were scored one point for each correct pH and for identifying correct metabolic/respiratory cause. A further point was awarded if acute, chronic, or compensated/mixed descriptions were correctly used. They received a further single mark for each appropriate differential diagnosis. Test examples had been chosen from real patient data. Correct results had previously been agreed by the surveyors and an independent ITU consultant.

54% of doctors correctly identified the normal range of values. 71% correctly described the abnormality shown in each example. 27% correctly produced two appropriate differential diagnoses. Surgeons and anaesthetists did better than most in the identification of normal ranges and the interpretation of results. ED staff obtained better results when listing potential diagnoses. Of ED staff, best overall results were returned by the three consultant respondents.

Results from this small snapshot survey imply the need for continued education of medical staff in the interpretation of ABG results. Given that only a quarter of our doctors offered correct differential diagnoses, we suggest that teaching should be directed towards the practical clinical use of gases as well as the theoretical background.

Í O' Sullivan, R Jeavons

Emergency Department, Bristol Royal Infirmary, Bristol, UK

Correspondence to: Dr Íomhar O' Sullivan, Emergency Department, Bristol Royal Infirmary, Bristol BS2 8HW; íomhar.O'Sullivan@ubht.swest.nhs.uk

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BOOK REVIEWS

Critical care transport field guide

M Czarniecki NREMT-P, CCEMT-P, Jones, Bartlett. Publishers: Sudbury, MA USA, 2001. ISBN 0-7637-1580-8

This small pocket book measures only 15 cm by 7.5 cm and is intended as a pocket reference book. It is designed to assist the reader in recalling knowledge acquired or confirmed from other sources.

I am afraid I found it quite confusing. The pages are printed in both landscape and portrait format which means having to constantly re-orientate the book. It is divided into 25 sections, covering everything from intra aortic balloon pumps, drug incompatibilities, and burns management.

It seemed at first sight to be a very useful collection of data but on closer examination it was most disappointing. The laboratory and other normal values are not quoted in SI units. The American values for things like blood glucose will be of little value to those working in the UK and much of the rest of the world.

Much of the detail is specific to the hospital concerned giving details of the colour of top for the blood sample required for each parameter. The section on blood transfusion has an administration check list, which has details that are specific to the procedures at the hospital concerned and are not generic.

There is a whole section on mnemonics and other aide memoirs. A few of these could be helpful, in the majority I would find easier to remember the lists rather than the mnemonic!

There are old favourites like C3,4,5, keeps the diaphragm alive and PEA ITT VOD being the differential cause of pulseless electrical activity, namely; Potassium, Embolus, Acidosis, Ischaemia, Temperature, Tamponade, Tension pneumothorax, Volume Oxygen, Drugs; which I personally find most unhelpful.

My overall impression was sadly, that there are other similar products on the market which are more user friendly, and which have more material relevant to the field without confusion with American normal values.

K Hines

Eastwood Medical Centre, London;
ken.hines@gp-f86641.nhs.uk

Forensic Medicine: clinical and pathological aspects

Edited by J Payne-James, A Busutil, W Smock. London: Greenwich Medical Media Ltd, 2003. ISBN 1-84110-026-9

I do acknowledge some personal bias, but I believe that it really would be quite hard to write a boring book on forensic medicine. The subject matter is simply so interesting. And what interests most people, fascinates many. This hefty tome comprises 51 chapters written by an assorted collection of international authors. Given the diversity of both the subjects covered and the contributing authors, the editors have done well to maintain a uniform style throughout. They should be particularly congratulated for managing to avoid an excess of photographs, which might be construed in some way as being voyeuristic.

The relationship between the specialties of A&E and Forensic Medicine has sometimes been somewhat awkward, particularly in the UK. This was typified by some heated correspondence which appeared in the *BMJ* a few years ago about a wound which had been described as a "neatly incised laceration"! Many of the chapters of this book are of direct relevance to A&E. The A&E specialist may wish to skip some subjects, such as the history of Forensic Medicine or the forensic investigation of war crimes. However, there is a wealth of material on injury, toxicology, and legal medicine. References, whilst not exhaustive, are reasonably representative.

Weighing in at 3.2 kilograms, this book will not easily find its way into the pocket of a busy clinician. With its attractive design and interesting content, it does deserve a place on the bookshelf. Take care though, if

you open it to quickly look something up, you might easily find yourself distracted and become engrossed within the pages.

J Wyatt

A&E consultant and Honorary Fellow in Forensic Medicine;
jonathan.wyatt@rcht.cornwall.nhs.uk

ECGs for the emergency physician

Edited by A Mattu, W Brady. London: BMJ Books, 2003. ISBN 0727916548

"Can you just check this ECG?" is one of the most frequently asked questions in the Emergency Department. *ECGs for the Emergency Physician* will help you become an expert at answering ECG queries: a core skill for ED physicians. Mattu and Brady have put together 200 ECGs that illustrate virtually all electrocardiographic diagnoses. This is achieved in a self-assessment format that is instructive and interesting. The first hundred ECGs are "easier" and are useful revision for SHOs preparing for the MFAEM exam, or SpRs looking to improve their ECG diagnostic skills. The second hundred are certainly more "challenging" as the authors suggest. I recommend these as continuing education for Emergency Medicine specialists: no matter how well honed your skills, there is something here that will make you stop, think and learn.

An A4-sized book, having two ECGs per page allows good reproduction of the data, and the answers are remote from the cases, so that a quick look is deliberately made more difficult. The answers are correspondingly concise, clear, and informative. You have the impression that the authors regularly examine ECGs in their EDs. Of comparable texts, this book is the most relevant to ED physicians. Basic knowledge is assumed though: this is not a text for medical students. I look forward to learning if BMJ books plan ABGs for the Emergency Physician or CXRs for the Emergency Physician, to complete the core data interpretation skills needed by ED physicians.

It is hard to find fault with this book, except to say that to read the lot in one go will have you dreaming of PR segment abnormalities and Brugada syndrome.

After you have read this book, I suspect the next person to ask you to "Just check this ECG" will be overwhelmed by your knowledge!

A Fletcher

Northern General Hospital, Sheffield;
alan-fletcher@supanet.com

CORRECTION

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The journal has been notified of an error in the paper entitled Simple monograms to calculate sample size in diagnostic studies (*Emerg Med J* 2005;22:180-1). The error occurs on the example line on the specificity nomogram (fig 1 part B). A correct version of this figure is available at <http://emjonline.com/supplemental>. It should be noted that the error only affects the example and not the underlying nomogram itself.