Carbon monoxide poisoning

Sir, The recent review of carbon monoxide poisoning by Balzan et al provided helpful advice about this sometimes difficult diagnosis. We would, however, hesitate to follow the recommendations made about the treatment of CO poisoning. Their assessment of the benefit of hyperbaric oxygen (HBO) does not appear to be based on the presented evidence and the potential complications of transporting critically ill patients to HBO facilities do not receive the attention they merit.

The authors cite one randomised trial assessing HBO in patients who were not pregnant. Two of the indications proposed for HBO include two (impaired consciousness and carbon dioxide instability) that were the exclusion criteria in this trial. Case series are also cited. We do not believe these provide sufficient information to allow clinically useful comparison between different treatments. Therefore, we disagree rather than to test hypotheses. We conclude with the observation of Thom et al (our italics): "Questions that should be addressed include whether treating patients [with hyperbaric oxygen] more than 6 hours after poisoning is effective and whether the benefits outweigh the costs of transportation and treatment."

RD HARDERN 
AJ GRAY 
Emergency Department, Royal Manchester Children's Hospital, Manchester, UK


This letter was shown to the authors, who responded as follows:

Sir, In our article we attempted to reproduce widely accepted recommendations in the current literature for the use of HBO in CO poisoning. These are based mainly on clinical experience and retrospective case series comparing our cases in patients treated with HBO with historical controls receiving normobaric oxygen (NBO). These have shown clear-cut, often dramatic improvements with HBO in severely and partly poisoned.

I agree with Hardern et al that, in principle, any therapeutic measure and its indications should be tested in a large prospective randomised controlled trial. However, in view of the evidence in retrospective case series for the greater effectiveness of HBO, it is unlikely that any ethical committee would permit such a trial in severe CO intoxication. It is also unlikely that a properly informed patient or his delegate would give his consent to participate in such a trial. Probably for these reasons, two recent prospective randomised controlled trials comparing HBO with NBO have excluded patients with severe poisoning (those with loss of consciousness or myocardial instabilility) and concentrated on moderate intoxication. Both these studies have confirmed the utility of HBO, compared to NBO, in accelerating recovery and preventing delayed neurologique sequelae. But, does it make sense in clinical practice to treat moderately intoxicated cases with HBO and not severe cases for the simple reason that a trial cannot be performed as treatment is considered potentially life-saving? Would such a position be tenable in a court of law? Is it logical to treat moderately intoxicated women, except when they carry a foetus very vulnerable to hypoxia, when it has been shown that HBO is safe in pregnancy?

As regards the treatment of critically ill patients, it is accepted that supportive care must never be compromised in transport and that the logistics of every case must be considered individually and carefully evaluated in a local context. However I feel strongly that, in line with guidelines in the literature, comatose patients, or patients with prolonged loss of consciousness should, whenever possible, be offered HBO.

HBO treatment in patients presenting late is still debatable. Whether the cost of an HBO unit or the transport expenses are justified or not by the benefit of therapy depends on the logistics and the relative priorities of different healthcare setups.

RD HARDERN

Heart failure in the elderly

Sir, Although I enjoyed reading the well-researched review of the management of heart failure in the elderly, I disagree with the emphatic restatement of the conventional view that adjunctive treatment with spironolactone is absolutely contraindicated in patients already co-prescribed angiotensin-converting enzyme (ACE) inhibitors and loop diuretics. The reality is that amongst patients already co-prescribed ACE-inhibitors and loop diuretics, there will always be a few with a refractory, and potentially life-threatening individual sensitivity due, in some instances, to hyperaldosteronism, for which corrective treatment could either be resection of an adenocortical adenoma or co-prescription of spironolactone and ACE-inhibitors.

In my own experience, dating back to 1984, comprising 349 patients co-prescribed ACE-inhibitors and loop diuretics for heart failure, three women, now aged 81, 81 and 79, respectively were both receiving hypertension and hypokalaemia, the latter refractory to ACE-inhibitors. In the first patient, with a nadir serum potassium of 2.7 mmol/l, following co-prescription of spironolactone 25 mg/day, frusemide 40-80 mg/day and enalapril 10 mg/day followed by lisinopril 20 mg/day, the serum potassium was maintained in the range 3.9-4.4 mmol/l during the last six months of a 30-month period of triple therapy. Prior to commencement of triple therapy, her 24-hour urinary aldosterone was 7 nmol (in 1620 ml urine), and her 09.00 h serum cortisol was 516 nmol/l.

In the second patient, the co-prescription of spironolactone was precipitated by a fall in serum potassium to 2.2 mg/dl whilst taking enalapril 20 mg/day and frusemide 80 mg/day. Her 24-hour urinary aldosterone output was 25 nmol (in 1620 ml urine), but neither 24-hour urinary cortisols nor serum cortisols were requested. She had type 2 diabetes, an aldosterone level of 2.7 nmol/l, 11 years previously, rendered the diagnosis of Cushing's syndrome unlikely in the absence of the development of clinical stigmata over this period. Ultra soundography had also not identified any adrenal abnormality. This patient was subsequently prescribed spironolactone 25 mg/day in addition to frusemide 80 mg/day, with serum potassium of 1.6 mmol/l, urea of 16.5 mmol/l, creatinine of 139 mmol/l and body weight of 47 kg. Unfortunately, her 24-hour urinary aldosterone and cortisol levels were quantified when she was already taking spironolactone 25 mg/day, frusemide 120 mg/day, ramipril 10 mg/day, and lacedipine 4 mg/day, yielding values of 2.0 mmol and 71 nmol, respectively, in 800 ml urine. Computed tomography showed that she had right-sided hydrothorax, but no adrenalectomy. During the subsequent three months, spironolactone was progressively increased to 200 mg/day, frusemide reduced to 80 mg/day, whilst ramipril was maintained at 10 mg/day and lacedipine increased to 6 mg/day. Consequently, her blood pressure fell to 200/90 mmHg, but her plasma potassium remained at 2.9 mmol/l, with urea 17.1 mmol/l and creatinine 130 mmol/l. Due to the subsequent development of a pruritic maculopapular rash, losartan 50 mg bid was substituted for ramipril, and she is now undergoing titration of the spironolactone dose, due to a 75% reduction in frusemide requirements (based on transient development of reversible prerenal uraemia).

OMP IOLOBI
Tameside General Hospital, Ashton-under-Lyne, Lancashire OL6 9RW, UK

Sir, I make no apology for stating that the use of spironolactone (an otherwise highly useful diuretic), in conjunction with ACE-inhibitors should be discouraged in the elderly. This combination in older people
who often have impaired renal function due to the reduction in glomerular filtration with ageing will often result in life-threatening hyperkalaemia. Spironolactone is a synthetic steroidal potassium-sparing diuretic and aldosterone synthesis inhibitor, thus acting as a competitive antagonist. It is most effective in conditions producing high aldosterone levels, for example, hepatic cirrhosis, nephrotic syndrome and Conn’s syndrome. Heart failure does produce secondary hyperaldosteronism but there is no evidence of this in the patients Dr Jolobe describes, as the 24-hour urine aldosterone level is within the normal range and indeed if they were receiving ACE-inhibitors the aldosterone level, thereby limiting the effects of spironolactone. Spironolactone will, however, inhibit the exchange of sodium for potassium ions in the distal part of the distal tube which is under the control of aldosterone and therefore cause potassium retention. The amount of potassium retained depends upon renal function. It may be better to give potassium in a controlled way in the form of oral supplements if patients who are on loop diuretics and ACE-inhibitors remain severely hypokalaemic (<3.0 mmol/l). These patients require close monitoring of their electrolytes (at least three times per week) and usually require in-patient treatment until their condition is stabilised. However, such patients constitute a small minority and this is confirmed by Dr Jolobe’s three cases in 12 years. In the vast majority of patients (and particularly the elderly) with heart failure in taking loop diuretics and ACE-inhibitors, therefore, the addition of spironolactone would probably result in hyperkalaemia. I believe this ‘conventional view’ is worthy of ‘emphatic re-statement’.

DEBRA KING
Department of Geriatric Medicine,
Wirral Hospital, Arrowe Park, Upton, Wirral, Merseyside L49 5PE, UK

Psychiatry in trauma care

Sir,
The burden of morbidity and mortality following trauma cannot be overemphasized. Adeideji and Driscoll’s recommendations towards setting up a trauma care service with a multidisciplinary input are notable, as prompt, appropriate and effective intervention has been shown to reduce this morbidity and mortality to a minimum. The suggested multispecialty trauma team from the surgical, accident and emergency, orthopaedic and anaesthetic departments is a vital step towards achieving optimum trauma care. I wish to add, however, that survivors of trauma often also suffer psychological (or even psychiatric) sequelae.

Mental health problems have been found after all levels of trauma severity and different kinds of problems may present at various stages of recovery. While some disorders appear to be the direct result of of direct damage such as head injury, which can lead to long-term psychiatric problems like seizures, intellectual impairment, personality change, psychotic illness, mood and anxiety disorders, others clearly are not. In the non-head-injured, psychiatric symptoms and disorders are also recognised and may be frequent. Awareness of the personal and social consequences of the injury such as threat to life, loss of mobility, loss of income, disfigurement, prospects of a lengthy hospital stay and even bereavement may precipitate anxiety and depression following trauma. Acute stress reactions can cause severe distress to the patient, pre-hospital, during admission and post-discharge. Unresolved, this may progress to the development of a post-traumatic stress disorder (PTSD) with flashbacks, horrific and intrusive memories, nightmares, avoidance of reminders of the incident and increased arousal. Diagnostic criteria for PTSD are listed in the box.

While PTSD occurred in 10% of road traffic accident victims in an Oxford study, other researchers in Australia reported unrecognised PTSD in 33% of the traffic accident victims in a trauma unit following psychiatric evaluation. Furthermore, within a week of trauma, 60% of accident victims at an accident and emergency department reported significant psychological distress. Distress point-injury has been documented as being highly predictive of psychiatric morbidity and development of PTSD, however, no correlation between trauma severity (measured by the injury severity score) and onset of symptoms was detected. Incorporating the psychiatrist in holistic trauma management would greatly reduce severely disabling psychological and psychiatric sequelae of trauma and enhance patient care. Although the liaison with the psychiatrist and the trauma team do not need to be formally present, they can probably be called upon to play the true sense of multidisciplinary trauma care.

OYEDEJI A AYORINDEN
Department of Psychiatry,
Northwick Park Hospital,
Harrow, Middlesex HA1 3UJ, UK

Coital trauma

Sir,
In his review of coital emergencies, Banerjee omitted an account of the injuries to the female genital tract. Coital trauma in females is well documented. Such injuries were thought to be more likely in young virgin girls during first sexual intercourse, and also in post-menopausal women. Findings of recent studies, however, have shown a preponderance of such cases in women of reproductive age. Milder injuries result in superficial tears and lacerations of the vulva, vagina and vault. However, more extensive injuries can lead to involvement of pelvic soft tissue, urethra, bladder, ureter and rectum. Cases usually present with vaginal bleeding. An occasional case presenting as acute lower abdominal pain due to pelvic haematoma has been reported. Torrential haemorrhage from a torn blood vessel leading to shock and needing urgent resuscitation and blood transfusion has also been described.

Superficial laceration or a closed haematoma respond successfully to conservative treatment with vaginal packing. However, the majority of cases need an examination under anaesthesia for assessment of the extent of injury and its surgical repair.

RANI JARVIS
Bolton Centre for Sexual Health
Royal Bolton Hospital, Farnworth,
Bolton BL4 0TR, UK

Diagnostic criteria for post-traumatic stress disorder

A. The person experienced, witnessed, or was confronted with an event that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others. Response involved intense fear, helplessness, or horror

B. The traumatic experience is persistently re-experienced in one or more of the following ways:
   - recurrent and intrusive distressing recollections
   - recurrent and distressing dreams
   - acting or feeling as if the event were recurring (flashbacks)
   - intense psychological distress at exposure
   - physiological reactivity on exposure

C. Persistent avoidance of stimuli associated with the trauma and numbing of responsiveness:
   - efforts to avoid thoughts, feelings or conversations
   - efforts to avoid activities, places or people
   - inability to recall an important aspect of the trauma
   - feelings of detachment or estrangement
   - restricted range of affect
   - sense of foreshortened future

D. Persistent symptoms of increased arousal:
   - difficulty falling or staying asleep
   - irritability or outbursts of anger
   - difficulty concentrating
   - hypervigilance
   - exaggerated startle response

E. Duration of disturbance (symptoms in B, C, and D) more than one month.

F. The disturbance causes clinically significant distress or impairment in social, occupational or other important areas of functioning.