

LETTERS TO THE EDITOR

Resuscitation from hypothermia

EDITOR,—The three cases of accidental hypothermia successfully resuscitated without neurological deficit using cardiopulmonary bypass reported by Ireland *et al*¹ provide a timely reminder that circulatory collapse in severe hypothermia must be treated with active rewarming (by whatever technique). Such patients, however, also require appropriate supportive critical care. In two of the patients reported, ventilatory support appears to have been inappropriate or delayed.

In case 2, the patient was successfully resuscitated from a VF arrest but then left self-ventilating through a T piece in spite of an unstable cardiac rhythm, severe hypothermia, and presumably acidosis and hypoxaemia. This is likely to be unsatisfactory both in terms of missing the opportunity to centrally rewarm using warmed humidified gases via a ventilator and in providing increased respiratory work (caused by the resistance of the tube and loss of intrinsic PEEP). Low volume ventilation with warmed 100% oxygen would have ensured better oxygen delivery and reduced respiratory muscle oxygen consumption, and would have contributed to the rewarming process.

In case 3, a patient with severe hypothermia, a GCS of 6/15 and "poor respiratory effort" was treated with high flow oxygen until some time after admission, when he developed an unrecordable blood pressure, bradycardia, and anuria, upon which he was intubated and ventilated. Many critical care authorities now recommend that hypothermic patients with a decreased conscious level and impaired airway reflexes or inadequate gas exchange require early intubation and ventilation.² This patient would have benefited from ventilatory support with warmed humidified oxygen from the time of arrival in the A&E department.

Cardiopulmonary bypass may have a place as a means of active rewarming where available, when other methods have failed, and in the presence of severe circulatory disturbance. Such highly complex techniques should not, however, detract from a basic ABC approach to resuscitation, and adequate ventilatory support must not be neglected in the haste to restore an adequate circulation and rewarm the patient if late deaths from multiple organ failure are to be minimised.

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1 Ireland AJ, Pathi VL, Crawford R, Colquhoun IW. Back from the dead: extracorporeal rewarming of severe accidental hypothermia victims in accident and emergency. *J Accid Emerg Med* 1997;14:255-7.

2 Simcock AD. Near drowning and hypothermia. In: Goldhill DR, Withington PS, eds. *Textbook of intensive care*. London: Chapman and Hall, 1997.

The authors reply

Dr Parke's comments about the role of ventilation with warmed humidified oxygen in severe hypothermia are well made and deserve to be highlighted. We also fully agree that in-

tial resuscitation according to the guidelines recommended by the Resuscitation Council UK is vital in the absence of effective spontaneous respiration or circulation.

The point of our article was to show that, if available, extracorporeal rewarming is the method of choice for victims of profound hypothermia with absent or inadequate circulation and can be used in A&E.

In case 2 the presence of spontaneous respirations was recorded to outline the patient's physiological status at the time; however, the underlying unstable cardiac rhythm was ventricular fibrillation and although not clearly stated in the text, compressions and ventilation with a "thumper" were reinstated before starting bypass. Once on bypass, ventilation was not required as oxygenation was provided from the extracorporeal membrane oxygenator. Compressions were, however, maintained to prevent cardiac distension and pulmonary oedema.

In case 3 the early involvement of senior specialists in intensive care medicine did indeed lead to timely intubation and ventilation before rewarming and oxygenation via bypass. While supporting Dr Parke's recommendation for ventilatory support from the time of arrival, the precise timing of the induction of anaesthesia in this unstable, profoundly hypothermic patient was a matter of clinical judgement for the clinicians involved at the time.

We do not agree that extracorporeal rewarming is only indicated when other methods have failed. In profound hypothermia with cardiac arrest, it is our opinion that, if available, extracorporeal rewarming deserves immediate consideration as a highly effective first line treatment after commencement of standard resuscitation procedures.

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Treatment of focal status epilepticus with lignocaine

EDITOR,—I read with interest the letter by Kato *et al*.¹

When working at Murgwanza Hospital in Tanzania in 1989, a patient with a very similar history presented with persistent focal status epilepticus who failed to respond to intravenous diazepam and pheytoin but instantly responded to a bolus of 100 mg of lignocaine and remained well with a lignocaine infusion over the next 24 hours. The same patient presented again in the same way several months later. However, on the second occasion he failed to respond to intravenous lignocaine and his fits persisted for more than a week. Consequently, I am doubtful that lignocaine is definitely the drug of choice in the treatment of status epilepticus with focal seizures.

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1 Kato H, Kishikawa H, Emura S, Takashima T, Ohmori K. Treatment of focal status epilepticus. *J Accid Emerg Med* 1997;14:201.

The authors reply

We would like to reply to Dr Emerton's letter. The treatment for focal status epilepticus has not yet been established and currently

conforms to that used in generalised status epilepticus. Generalised tonic-clonic status epilepticus is usually treated with intravenous diazepam to abort the status, followed by infusion of phenytoin to prevent recurrence of seizures.¹ Patients who do not respond to this treatment are generally treated with phenobarbitone or general anaesthetics, although with both of these endotracheal intubation may be needed to maintain respiration.¹ Lignocaine has the benefit of being devoid of significant depressing effect on consciousness and respiration at therapeutic doses.² It may be indicated as a first line drug in patients in whom depression of respiration or consciousness is particularly undesirable or as a second line drug in those episodes of status epilepticus that do not respond to diazepam. However, the precise role of lignocaine in the management of focal status epilepticus is not yet clear, because very little research has been done on its use for this purpose. We hope our letter will provide a stimulus to further investigations into the anticonvulsant activity of lignocaine in focal status epilepticus.

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1 Aggarwal P, Wai JP. Lidocaine in refractory status epilepticus: a forgotten drug in the emergency department. *Am J Emerg Med* 1993;11:243-4.

2 Morris HH. Lidocaine: a neglected anticonvulsant? *South Med J* 1979;72:1564-6.

Alcohol intoxication in a toddler

EDITOR,—We have recently had an interesting case of alcohol intoxication. The patient was an 18 month old girl. She was playing in her bedroom and took a 200 ml bottle of paracetamol elixir from the top of a bookcase high in her room. This had been dispensed some months earlier and was not fitted with a childproof cap. Her mother found her playing with the open bottle. Most of the contents had gone from the bottle but there was a significant amount on the child's clothes. Her mother brought her immediately to hospital and on the way she started to become drowsy. On arrival in accident and emergency she was pale and flaccid with a Glasgow coma scale of 3/15. She had vomited but was maintaining her own airway. She had a blood pressure of 80/60, a pulse of 96/min, and a respiratory rate of 20/min. Her oxygen saturation was 99% on 100% oxygen by face mask. The initial serum potassium was 2.5 mmol/l and the blood glucose was 2.4 mmol/l. Her plasma urea and electrolytes were otherwise normal. Blood gases showed: pH 7.356, Pco₂ 2.15 kPa, Po₂ 24.80 kPa, HCO₃ 8.8 mmol/l, oxygen saturation 99.3%. The blood paracetamol concentration was 1.1 mmol/l at 4 h postingestion.

Traditionally, paediatric paracetamol elixir (British Pharmacopoeia, 1980) is formulated to provide 120 mg of paracetamol in 5 ml. This mixture also contains 10% (by volume) of 96% ethanol. Although many proprietary liquid paracetamol formulations contain no alcohol, the British Pharmacopoeia formulation is still sometimes dispensed from pharmacies.

Serum alcohol was not measured but serum osmolality was 350 mmol/kg. Using an algorithm,¹ the ethanol concentration can be