LETTERS TO THE EDITOR

Resuscitation from hypothermia

EDITOR,—The three cases of accidental hypo- 
thermia successfully resuscitated without 
neurological deficit using cardiopulmonary 
bypass reported by Ireland et al provide a 
timely reminder that circulatory collapse in 
severe hypothermia must be treated with 
active rewarming (by whatever technique). 
Such patients, however, also require appropri- 
ate 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 resus- 
citated from a VF arrest but then left self ven-
tilating through a T-piece in spite of an unsta-
bile cardiac rhythm, severe hypothermia, and 
premature acidosis and hypoxaemia. This is 
likely to be unsatisfactory both in terms of 
missing oxygen delivery centrally and in warm 
using warmed humidified gases via a ventila-
tor and in providing increased respiratory 
work (caused by the resistance of the tube and 
loss of intrapleural pressure). 

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 hypother-
mia, 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.1 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 when available, where other methods have failed, and in the presence of severe circulatory distur-
bance. 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. 

TIMOTHY R J PARKE 
Accident and Emergency Department, Southern General Hospital, Glasgow


The authors reply

Dr Parke’s comments about the role of ventila-
tion with warmed humidified oxygen in severe hypothermia are well made and deserve to be highlighted. We also fully agree that ini-
tial resuscitation according to the guidelines 
recommended by the Resuscitation Council 
UK is vital in the absence of effective sponta-
neous 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 circu-
lation and can be used in A&E. 

In case 2 the presence of spontaneous respi- 
ration 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 
shown in the text, compressions and ventila-
tion with a “thumper” were reinstituted 
before starting bypass. Once on bypass, ventilation 
was not required as oxygenation was provided 
from the extracorporeal membrane oxygena-
tor. Compressions were, however, maintained 
to prevent cardiac distension and pulmonary 
collapse. 

In case 3 the early involvement of senior 
specialists in intensive care medicine did 
indeed lead to timely intubation and ventila-
tion before rewarming 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 uncontrollable, 
profoundly hypothermic patient was a matter of 
clinical judgement for the clinicians involved 
at the time. 

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

ALASTAIR J IRELAND 
VIVEK L PATHI 
RUDY CRAWFORD 
IAN W COLQUHOUN 
Departments of Accident and Emergency Medicine and Cardiothoracic Surgery, Royal Infirmary, Glasgow

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 1995 I treated a patient with 
very similar history presented with persistent focal status epilepticus who failed to respond to intravenous diazepam and phenytoin but 
instead 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. 

DAVID EMERTON 
Accident and Emergency Department, North Tees General Hospital, Stockton on Tees, Cleveland

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 

forms to that used in generalised status 
epilepticus. Generalised tonic-clonic status 
epilepticus is usually treated with intravenous 
diazepam to arrest the status followed by 
infusion of phenytoin to prevent recurrence of 
seizures.2 Patients who do not respond to this 
treatment are generally treated with pheno-
barbitone or lorazepam. However, though with both of these endotracheal intubation 
may be needed to maintain respiration.3 

Lignocaine has the benefit of being devoid of 
significant depressive effect on consciousness 
and respiratory function.1 It may 
be indicated as a first line drug in patients 
in whom depression of respiration or conscious-

ness is particularly undesirable or as a second 
line drug in those episodes of status epilepti-
cus that do not respond to diazepam. How-
ever, 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 antagonistic activity 
of lignocaine in focal status epilepticus. 

H KATO 
H FUJITA 
H KISHIKAWA 
S EMURA 
T TAKASHIMA 
K OHMORI 
Saga Medical School, Saga 849, Japan

Alcohol intoxication in a toddler

EDITOR,—We have recently had an interesting 
case of alcohol intoxication. The patient was 
a 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 
cap proof cap. Her mother found her playing 
with the open bottle. Most of the contents 
had gone from the bottle but there was a 
considerable amount on the child’s clothes. 
Her mother brought her immediately to hospital 
and on the way she started to become 
unresponsive. 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 
gas showed: pH 7.356, PCo2 2.15 kPa, Po2 
24.80 kPa, HCO3 8.85 mmol/l, oxygen satura-
99.3%. The blood paracetamol concentra-
tion was 1.1 mmol/l at 4 h post ingestion 

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 formula-
tion is still sometimes dispensed from phar-
macies. 

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