LETTERS TO THE EDITOR

Intramuscular or intravenous adrenaline in acute, severe anaphylaxis?

EDITOR,—The consensus guidelines on the emergency medical treatment of anaphylactic reactions by the Project Team of the Resuscitation Council (UK) are an excellent guide for first medical responders, whether general practitioners or emergency department staff. They are pragmatic, safe, and emphasise the importance of first line treatment with oxygen, adrenaline (epinephrine) and fluids, and as Hughes and Fitzharris in their BMJ editorial suggest, rightly deserve to “... adorn the walls of emergency departments, general practitioners' surgeries, and outpatient clinics.”

The guidelines usefully remind us that a panic attack or a vasovagal syncopal episode may be confused with anaphylaxis with the danger of inappropriate treatment. Additional differentiating features not mentioned in the text that suggest a faint rather than anaphylactic collapse are the rapidity of onset, maintenance of a central pulse, and prompt response to the recumbent position.

It is refreshing to see the debate over the delivery of adrenaline move forward a stage, with the subcutaneous route no longer recommended as the absorption is delayed and variable, at least in well children with a history of systemic anaphylaxis, when compared with the intramuscular route. The guidelines thus quite correctly favour the early administration of intramuscular adrenaline at a dose of 0.5 ml of 1:1000 for adults, to all patients with clinical signs of shock, airway swelling, or definite breathing difficulty. Intramuscular adrenaline given early, or when venous access is difficult and if the patient is unmonitored, is safe and effective even in less experienced hands.

Noting that we also reappraise some of the perennial dogma that limits the use of intravenous adrenaline in acute, severe anaphylaxis. The introduction’s criticism of the inappropriate use of intravenous adrenaline, singling out accident and emergency department and intensive care units and hospitalised patients is unjustified. It is presumably based on rare, outdated reports that include a questionnaire circulated to junior resident staff showing that in the 10% who responded there was misunderstanding about the dosage, route, and dilution of adrenaline, and a purely anecdotal letter castigating the use of intravenous adrenaline as it is “...a hazardous procedure rarely warranted in anaphylactic reactions”! As has been pointed out before, published reports on the apparent dangers of intravenous adrenaline consistently fail to emphasise that other causes such as hypoxia, hypotension, or the direct actions of the inflammatory mediators themselves released during anaphylaxis may be responsible for the cardiovascular complications. In addition, adverse outcomes to adrenaline occur when it has been given too rapidly, inadequately diluted to prevent excessive dosage.

Also, the statement that intravenous adrenaline should only be reserved for profound shock or special indications, for example during anaesthesia, is perplexing. Seventy-five per cent of anaphylactic deaths are due to hypoxia from upper airway oedema and hypotension, bronchospasm, many within the first hour of onset of symptoms and only 25% from circulatory collapse. In addition, there is nothing unique about anaphylaxis during anaesthesia other than the standard mode of delivery of all drugs is intravenously, and thus unexpected anaphylactic reactions may be rapid and catastrophic.

However, many of the same drugs are also used regularly in emergency medicine departments and intensive care units to facilitate interventional airway care. Continuous electrocardiographic, blood pressure, and pulse oximetry monitoring are now routine in all these areas and allow for the safe use of intravenous adrenaline. As Hughes and Fitzharris point out, the guidelines are somewhat vague in empirically recommending repeated intramuscular adrenaline every five minutes, or intravenous adrenaline as slowly as seems reasonable in the absence of clinical improvement or if deterioration occurs, particularly with profound shock.

Emergency medicine has come a long way in the last decade to include a much higher level of senior supervision, routine access to the minimum standards of monitoring suggested above, and widespread collective expertise in managing anaphylaxis. It is now time to consider whether any monitored area, whether the emergency department, intensive care unit, or high dependency unit, in experienced hands low dose, high dilution intravenous adrenaline is the optimal care for a patient with severe or rapidly progressive, life threatening anaphylaxis whether from shock or acute respiratory distress. Administration of 0.75–1.5 µg/kg of 1:100 000 adrenaline intravenously at 1 ml/kg per minute reverses all the life threatening effects of anaphylaxis and improves further most cell mediator release by raising intracellular cyclic AMP. The 1:100 000 dilution, as the guidelines suggest, not only allows precise titration to response, but avoids inadvertent rapid or excessive dosage. In addition, the therapeutic response to the adrenaline is immediate and assured.

The Project Team of the Resuscitation Council (UK) must be congratulated on an anaphylaxis guideline that other causes such as hypoxia, hypotension, or the direct actions of the inflammatory mediators themselves released during anaphylaxis may be responsible for the cardiovascular complications. In addition, adverse outcomes to adrenaline occur when it has been given too rapidly, inadequately diluted to prevent excessive dosage.

We agree with Dr Brown for his kind general remarks about our consensus guidelines, and also for giving us the opportunity to clarify one sentence in our introduction. We said “There has been a vague for inappropriate use of intravenous epinephrine (adrenaline), both by paramedics and in accident and emergency departments, when epinephrine...
made the habit of always using adrenaline IV access without delay”.

hazardous and is recommended only for an 
reserved for medically qualified personnel 
given in paragraph 4.4 should usually be 
and by experienced clinicians.

that it should be used in the most serious cases 
very dangerous”. We must reiterate that we 
venous adrenaline (epinephrine) is potentially 
strongly that the advice against using intra-

colleagues were similar, but there seems also 
recommendations for expert management 
bronchoconstriction and nebulised inhaled bronchodila-

airway oedema and hypoxia from severe bron-

and Augustine and Gavalas and his 
patients. This is far from advising against its 
emergency departments or elsewhere. In retrospect 
we see that we should have been more explicit 
points. We agree totally with Dr 
emergency departments or elsewhere. In retrospect 
was indeed our intention to reference it 
the other specialist recommenda-

We do not accept that the guidelines need 
urgent revision. Neither does the Project Team with its wide representation accept that 
recommendations as they stand are insulting 
the specialty of accident and emergency. 
We are conscious that we all have the same 
aim: both and safer treatment of an important medical emergency.

Future inpatient management of patients with minor head injuries

We read, with interest, the letter from Pau and Buxton, regarding the need for 
neurosurgeological referral of patients with an 
admitting diagnosis of minor head injury. We agree that this is a low risk group.

We performed an audit of patients admitted to 
our observation ward with the primary 
admitting diagnosis of minor head injury. From 
October 1997 to July 1999, 668 such patients were 
admited under our care. Of these patients, 
only two were subsequently transferred to the 
regional neurosurgical unit after the finding of 
intracranial haematoma on computed tomographicogram. This finding, and our general experience, leads us to conclude that patients with an 
admitting diagnosis of minor head injury do not require neurosurgical referral, in the 

In the UK there are on-site neurosurgical facilities, this practice is not widespread anyway.

Pau and Buxton’s conclusion is in keeping with the recommendations of the Report of 
the Management on the Management of Patients with Head Injuries. One of the logistical 

Our guidelines stated in paragraph 4.4 that: “Intravenous adrenaline (epinephrine) in a 
dilution of at least 1:10 000... is hazardous and should be used in the most serious cases and 
by experienced clinicians. Our guidelines stated in paragraph 4.4 that: “Intravenous adrenaline (epinephrine) in a 
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dilution of at least 1:10 000... is hazardous and should be used in the most serious cases and 
by experienced clinicians.

The concerns of Mr Gavalas and his 
were intended specifically for those first medical responders who are 
inexperienced in the management of this 
emergency. They are unlikely to have monitoring 
facilities available. For these, cautious rec-

The recommendations of the report have 
provoked widespread debate and polarity of 
views within the specialty. It probably repre-

There has been successful government legislation concerning road traffic accident 
prevention and injury protection—for example, the breathalyser laws, compulsory crash 
helmets for motorcyclists, and the seat belt laws. These are still supported by publicity. 
The legislation could be expanded. 

We as an emergency specialty, however, have a responsibility in accident prevention 
and injury protection. We all need to be active, 
as I have been, in the local press recently high-lighting, for example, the need for cyclists to 
wear helmets. We recognise this problem only too well.

Our department has a nurse who travels 
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Playing in the back seat

EDITOR,—We read with interest the letter of fatal intra-abdominal injury associated with incorrect use of a seat belt and would like to present another aspect of seat belt misuse.1 A pair of 7 year old identical twins presented to the accident and emergency (A&E) department after being involved in a head-on road traffic accident. Both had been restrained in four point child seats in the back of the car.

In the department, one was complaining of abdominal pain, the other none. On examination of the twins, both were haemodynamically stable. Twin 1 had bruising to her abdomen, the other none. This bruising was linear in nature across her abdomen, “the seat belt sign”. She had no focal tenderness.

In view of the seat belt sign, twin 1 was admitted and an abdominal ultrasound was performed at this stage. The ultrasound showed a small amount of fluid in the pouch of Douglas, but no other free fluid or lacerations of visceral organs.

Overnight she remained painful, but haemodynamically normal. A further ultrasound was performed the next day which showed free fluid within her abdomen.

Although there was performed that confirmed turbid fluid and this was converted to a laparotomy. Three lesions were found in her jejunum, one full thickness and two partial. A 15 cm resection of her jejunum was performed with continuity. She made an uneventful recovery.

On detailed questioning while in the A&E department, she stated that she had been playing a game with her twin sister and had wriggled out of her shoulder straps. This had converted her four point child seat into a lap belt. Twin 2 had remained properly restrained in her child seat.

Lap belt use has been recognised as a mechanism of blunt injury to the small bowel. The seat belt sign is associated with an increased likelihood of intestinal injury.2 This lap belt injury in children usually occurs when children are too large for their safety seats and too small for adult seat belts.3 This case highlights the importance of the seat belt sign, but more importantly that children within their own safety seats need to be restrained properly within them. Incorrect usage results in injury similar to adult lap belts.

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Oesophageal rupture

EDITOR,—Drs Onyeka and Booth present an interesting case of tension pneumothorax associated with Boerhaave’s syndrome.1 I have previously described a similar case in a 47 year old man who survived his ordeal in 1996.2 As the authors note, awareness of the condition is the mainstay of diagnosis.

Aside from these two cases, tension pneumothorax has been described in association with rupture of a Barrett’s oesophagus3 and after rupture of an oesophageal diverticulum.4 It is worth noting that by far the commonest cause of oesophageal rupture is after endoscopy. Regardless of the cause of rupture I agree with the authors that a gastrografin oesophagram is the diagnostic procedure of choice.

Conservative management of oesophageal rupture is now a well established option in iatrogenic rupture and has been described in Boerhaave’s syndrome,5 although as the authors note, immediate surgery comprising drainage and repair is the mainstay of curative treatment.

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6 Troum S, Lane CE, Dalton ML. Surviving Boerhaave’s? This trial also included severely poisoned patients and incorporated sham treatments. This study found in 199 patients that three days of HBO (2.8 atmospheres for 60 minutes) offered no advantages compared with three days of NBO (100 minutes 100%). Another study is continuing in the United States and the interim results have found no difference in the incidence of persistent neurological sequelae between those treated with HBO compared with NBO, although there is an increased incidence of delayed sequelae in one of the blinded treatment arms.

The authors also recommend careful neurological and cognitive re-examination. It is worth highlighting that cognitive testing in carbon monoxide poisoning is far from standardised. Many studies utilise different screening tests, different time intervals to re-screening, and different HBO regimens. This lack of standardisation makes it difficult to compare studies and no doubt contributes to our inability to provide definitive recommendations in the management of carbon monoxide poisoning.

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Evidence based and guideline based medicine

EDITOR,—Evidence based and guideline based medicine are assessed in our Faculty of Accident and Emergency Medicine exit examination. Particular type of study. Lacking a photographic memory, I put forward simple acronyms that have helped me to facilitate timely and efficient appraisal for everyday use when selectively scanning relevant journals. Preceding these specific acronyms is a “stand-
ards' acronym that follows Crombie's suggestion that standard questions should be used as a filter for all papers.¹

I hope they are of use to fellow practitioners of evidence-based medicine, and further suggestions will be gratefully received.

### Standards
- Stated aims?
- Tests and measures appropriate?
- Arithmetic (do the numbers add up?)
- Design appropriate?
- Relevance to your practice?
- Different results from previous reports?
- Sample size/power adequate?

### Diagnosis
- Diagnostic test needed?
- Independent blind comparison?
- Gold standard used regardless of test result?
- Numerogram (2x2 table) constructable?
- Sensitivity and specificity important?
- Inferences possible?
- Safe, cheap, and helpful?

### Prognosis
- Prospective study?
- Objective and blinded outcome criteria?
- Groups adjusted for prognostic factors?
- Numbers recruited and followed up adequate?
- Outcomes likely?

### Therapy
- Trial ethically approved?
- High recruitment and follow up?
- Equal treatment and assessment in each group?
- Randomised and how?
- Appropriate population?
- Potential benefits for patients?
- End points applicable?
- Absolute risk reduction/number needed to treat?

### Systematic review
- Systematic search strategy?
- Randomised and relevant trials?
- Each trial assessed?
- Valid results in all trials?
- Inconsistent populations or results?
- Evidence of benefit via odds ratios/number needed to treat?
- Was hypothesis satisfied by the review results?

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