

counsellor who will spend much time comforting patients after traumatic events. I suggest that general practitioners used to have the leisure to do this but are no longer able to do so, as their time is used up in dealing with surgery consultations, social security assessments, health promotion activities, public health measures, devolved secondary care, community care, and NHS administration.

The deputy was probably seeing a variety of "emergencies"—mostly cases of self limiting illness in fit people. Deputies are experts at this type of general practice. They are not experts in resuscitation or counsellors, they cannot stay and comfort bereaved people for long; they have to be off to the next sore throat.

Had Rawlinson's neighbour had access to an out of hours emergency centre she would have travelled quickly and received prompt care from fresh staff trained in emergency care; her husband could have contacted a counsellor or, perhaps more appropriately, a minister of religion, friend, or neighbour in his bereavement. A social worker might have organised his social care needs. Surely this is preferable to the current system, whereby society prefers the false belief that everything could be delivered by one's own general practitioner if only he or she attended.

I work 80 hours a week. My reserves of care and compassion are regularly exhausted. I send deputies to people who I believe are going to use up more of myself than I and the rest of my patients can afford. A bereaved relative told me today that I was a wonderful doctor, but I doubt if I will be doing the same job after the age of 45. The personal service that Rawlinson and Rawlinson's neighbour yearn for is delivered only at terrible cost to general practitioners and their families. The time has come to acknowledge this and change to a better system.

FAY WILSON
General practitioner

Birmingham B10 9QX

1 Rawlinson JN. Deputising general practitioners' role in emergencies. *BMJ* 1995;311:394. (5 August.)

Treatment of acute anaphylaxis

Surviving the journey is a good prognostic indicator

EDITOR,—Malcolm Fisher's article on acute anaphylaxis was prompted by an anecdote concerning a doctor's panic when his young daughter developed an acute allergic reaction.¹ The father ended up "ranting and raving" in the emergency department because his daughter had not received adrenaline immediately. Fisher uses this scenario to underline the need for the rapid administration of adrenaline in anaphylaxis. Nevertheless, he freely admits that, with the protean and sometimes life threatening nature of anaphylaxis and anaphylactoid reactions, randomised controlled trials of treatment are not feasible.

Given the emotive and anecdotal origin of Fisher's article, it is perhaps important to be aware of other anecdotal issues in the treatment of anaphylaxis. Each year we admit roughly 100 patients with acute allergic reactions of the sort described in Fisher's article to our short stay observation ward; a minority of these patients receive adrenaline, but the overwhelming majority are fit for discharge within 24 hours. The patients typically receive nebulised salbutamol, parenteral or oral steroid, and parenteral antihistamine. The problems that we encounter are usually those of rebound or biphasic systemic anaphylaxis (in about 7%), which merits treatment with oral antihistamine and steroid for about two days.²

Just like the doctor described in Fisher's article, I too received the fright of my life once, only my fright was induced by adrenaline during dentistry.

Adrenaline is as toxic as any drug: it can cause dangerous arrhythmias and profound distress in lucid patients. My thesis is that most unheralded deaths from uniphasic anaphylaxis occur within a few minutes of exposure and that those patients who survive to reach the emergency department are effectively in a separate category: observation over a few minutes or an experienced doctor can identify that minority requiring aggressive treatment with adrenaline. This may be a dangerous and heretical theory based on anecdote, but I believe that the universal prescription of adrenaline suggested in all contemporary texts is equally anecdotal and warrants closer examination. I accept that adrenaline is the drug of first choice in life threatening anaphylaxis, but I do not believe that all urticarial, angio-oedematous, or bronchospastic reactions are potentially lethal. The story in Fisher's article is important, but the girl survived: nearly all patients do, with precisely the treatment she received.

LC LUKE

Consultant in accident and emergency medicine

Royal Liverpool and
Broadgreen University Hospitals NHS Trust,
Liverpool L7 8XP

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Remove the patient from contact with the allergen

EDITOR,—In his article on the treatment of acute anaphylaxis Malcolm Fisher omits one important point: that further contact with the allergen should be prevented immediately. In the case of stings or intramuscular injections a tourniquet should be applied proximal to the site; bee stings should be removed, and any drugs being given intravenously at the time that anaphylaxis occurred should be stopped. Food allergens should be removed from the mouth, and if the patient is still conscious the mouth may be rinsed. Vomiting of recently ingested food can also be induced by putting two fingers in the throat of patients who are still alert and realise that they have swallowed something to which they are very allergic.

As with other allergic diseases, such as asthma and rhinitis, the efficacy of avoiding the relevant allergen should not be underestimated.

GLENIS K SCADDING
Consultant physician in clinical immunology,
allergy, and rhinology

Royal National Throat, Nose and Ear Hospital,
London WC1X 8DA

1 Fisher M. Treatment of acute anaphylaxis. *BMJ* 1995;311:731-3. (16 September.)

Benign allergic reactions should not be treated with adrenaline

EDITOR,—The case report of an anaphylactic reaction, presumably to nuts, and Malcolm Fisher's review of anaphylaxis and its treatment are timely.¹ The management of children's anaphylactic reactions to foods has recently been reviewed,^{2,3} and our experience in childhood allergy—in particular, peanut allergy—prompts us to emphasise some additional points and to urge clarification of terminology.

Fatal and near fatal anaphylaxis related to foods most commonly occurs in patients who have had previous severe reactions, which makes the history crucial rather than "of little value," as Fisher seems to suggest.¹ A high risk of anaphylaxis related to food is associated with poorly controlled asthma and the requirement of oral corticosteroids and with delay in the administration of adrenaline.⁴

Doctors who may encounter an anaphylactic

emergency must be aware that β blocking drugs may potentiate anaphylaxis⁵ and that fatal and near fatal reactions to foods sometimes proceed in the absence of signs of more minor reactions—for example, collapse and cyanosis without urticaria or pruritus.⁴ This again emphasises the importance of the history and awareness of allergy on the part of the subject, care givers, teachers, and doctors.

Patients often come to us with a diagnosis of anaphylaxis only for us to find that the reaction was confined to urticaria of short duration after exposure to a large dose of allergen. To label this benign reaction as anaphylaxis is misleading and alarmist: it may render the subject vulnerable to being overtreated (with adrenaline) when reassurance and observation or an antihistamine would suffice. We accept that the converse also applies with regard to the misdiagnosis of compromise of the airway or hypotension as a mild allergic reaction to be treated with antihistamines. Nevertheless, we urge that the term anaphylaxis be restricted to catastrophic, life threatening allergic reactions and accurate terms for urticaria, mild angio-oedema, and laryngeal oedema be used. Laryngeal oedema is, of course, life threatening and requires treatment with inhaled or injected adrenaline.

JONATHAN O'B HOURIHANE

Clinical research fellow

JOHN O WARNER

Professor of child health

School of Medicine,
Child Health,
Southampton General Hospital,
Southampton SO16 6YD

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3 Hourihane JO'B, Warner JO. Management of anaphylactic reactions to foods. *Arch Dis Child* 1995;72:274.

4 Sampson HA, Mendelson L, Rosen JP. Fatal and near fatal anaphylactic reactions to food in children and adolescents. *N Engl J Med* 1992;327:380-4.

5 Jacobs RL, Rake GW, Fournier DC, Chilton RJ, Culver WG, Beckmann CH. Potentiated anaphylaxis in patients with drug-induced beta-adrenergic blockade. *J Allergy Clin Immunol* 1981;68:125-7.

Avoid subcutaneous or intramuscular adrenaline

EDITOR,—Malcolm Fisher states that intravenous adrenaline should be used only in severe cases of anaphylaxis as it may cause arrhythmias¹ and cites a publication by Waldhausen *et al.*² In this paper the doses given intravenously were up to 20 times the initial dose recommended by the Association of Anaesthetists of Great Britain and Ireland.³ Previous authors have also expressed misgivings about using intravenous adrenaline on the basis of anecdotal reports in which the speed of administration was not stated and other causes of arrhythmias were not excluded.⁴ Like any other drug, adrenaline may be dangerous if given too fast or in an excessive dose, but it is illogical to restrict its use because of concerns over complications caused by inappropriate administration.

Ideally, intravenous adrenaline should be the first line treatment for all patients with anaphylaxis treated by medically trained staff. It is safe and effective if given in a controlled titrated manner at an initial dose of 0.5-1 ml of 1/10 000 solution.³ This ensures rapid delivery to its site of action and avoids the problem of variable absorption after its administration subcutaneously or intramuscularly in patients in whom tissue perfusion may be compromised. In addition to treating the pathological vasodilatation that contributes to hypotension in anaphylaxis, adrenaline stabilises mast cells and therefore treats both the effect and the cause of the condition when distributed systemically.

In a child with anaphylaxis, difficulty in securing venous access should not be regarded as an impediment to the use of adrenaline by infusion. As recommended by advanced paediatric life support