

## LETTERS TO THE EDITOR

### Management of anaphylactic reactions to food

EDITOR,—The layout of the article by Patel *et al* on the management of anaphylactic reaction to food is misleading in that the first mention they make to the treatment of food related anaphylaxis is a description of cardiopulmonary resuscitation in the event of cardiac arrest.<sup>1</sup> We are sure it is not their intention to imply that this is the most common form of allergic reaction to food but they may have done so by ignoring the time honoured practice of describing the reactions as a progression from the mildest reaction to the most severe. We do not doubt the importance of the appropriate advice regarding life support but this advice should be put into context.

The role of modern non-sedating antihistamine applications in the treatment of mild and moderate reaction is given disappointingly little prominence, yet in conjunction with inhaled adrenaline they form the cornerstone of our treatment of reactions up to and including laryngeal oedema.

Inhaled adrenaline, in our experience, has been very effective in the treatment of mild to moderate allergic reactions. We also prescribe inhaled adrenaline for children with severe reactions, in addition to their prescription for an injectable form as described by Patel *et al*. We feel these children can 'buy time' by being more able to use rapidly an inhaler with which they and their companions are more likely to be familiar as a result of pre-existing asthma – itself a common association with severe anaphylaxis<sup>2</sup> – than they are with uncapping a needle and syringe in a stressful situation. Indeed, one of us has had over 20 years' experience of running allergy clinics, and there has been no patient who needed to resort to injectable adrenaline having used the inhaled form at the onset of reaction. The lower threshold for using the inhaled adrenaline at the first sign of symptoms such as tingling in the lips or throat may well have forestalled more severe responses.

The severity of a reaction is not necessarily predicted by the pattern of previous reactions. In the light of this unpredictable gradient of reaction we feel it is reasonable and responsible practice to recommend graded treatments that vary from antihistamines alone through inhaled adrenaline to immediate use of injected adrenaline.

We have no experience of parents using an injectable form of adrenaline as an excuse to exclude their child from school and it is certainly not our impression that parents use the increased level of medication as an excuse for decreased vigilance. The parents of our patients appear to be well informed and highly motivated people who are keen to learn more, not to do less. We have been involved with school nurses and community health services in the preparation of an information sheet and protocol for the treatment of anaphylaxis. This is provided to the parents to take to the school and to the general practitioner who is, in most cases, the first point of contact after an accidental exposure has occurred.

The issue of food related allergic reactions and anaphylaxis is a topic of widespread media and public interest at present. It may be that a forum needs to be developed for the development of nationwide guidelines for the medical management of the entire problem not just emphasis on the management of cardiopulmonary arrest. From our point of view the major concern for the near future will be the loss of Medihaler-Epi because of the ban on chlorofluorocarbon usage.

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- 1 Patel L, Radivan FS, David TJ. Management of anaphylactic reactions to food. *Arch Dis Child* 1994; 71: 370–5.
- 2 Yunginger JW, Sweeney KG, Sturmer WQ, *et al*. Fatal food induced anaphylaxis. *JAMA* 1988; 260: 1450–2.

#### Dr Patel and coauthors comment:

It is correct to point out that our article concentrated on severe reactions; this was our intention. It is the management of these potentially life threatening events which has been of special interest. Antihistamines are indeed believed to be useful for mild reactions, and their role in severe reactions is discussed. We agree that inhaled adrenaline may have a role, for the reasons given in our review. It is correct to say that the severity of a reaction is not necessarily predicted by the pattern of previous reactions, but as far as life threatening reactions are concerned, a previous severe reaction is the best predictor. Thus, in 13 children with life threatening reactions to food, there was a previous history of a serious anaphylactic reaction in all 13.<sup>1</sup>

Since our review was published, there have been three developments. The first is the publication of data giving further concern about the efficacy and safety (risk of cardiac arrhythmia) of injected adrenaline in anaphylaxis.<sup>2</sup> The second is the publication of the latest *British National Formulary*, which has an expanded section on anaphylaxis.<sup>3</sup> The third is a useful badge which can be sewn on to children's garments warning that the child has a food allergy\*. At present there are four such sew on patches, marked 'No Milk Please', 'No Nuts Please', 'No Nuts or Milk Please', and 'BEWARE – Food Allergies'. These labels seem to us a useful and practical way to help warn or remind carers of a potential problem.

- 1 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.
- 2 van der Linden PWG, Hack CE, Struyvenberg A, van der Zwan JK. Intentional diagnostic sting challenges: an important medical issue. *J Allergy Clin Immunol* 1994; 94: 563–4.
- 3 British Medical Association and the Royal Pharmaceutical Society of Great Britain. *British national formulary*. No 28. London: BMA and the Royal Pharmaceutical Society of Great Britain, 1994: 133–5.

\*Purchasable from Eyecatches, 31 Berrow Drive, Edgbaston, Birmingham B15 3UA; (tel: 021-456 5742).

### Hypoglycaemia complicating treatment regimens for glycogen storage disease

EDITOR,—In 1978 we reported the risks of hypoglycaemia in children with glycogen storage disease (GSD) treated with nocturnal

nasogastric feeding regimens.<sup>1</sup> The use of such treatment combined with regular drinks during the day in order to mimic the basal hepatic glucose production rate in infants and young children with GSD, has proved to be effective in reversing many of the metabolic abnormalities and improves growth. However, the stimulation of insulin secretion and suppression of alternative fuels for cerebral metabolism will result in profound symptomatic hypoglycaemia if the nasogastric tube feeds are suddenly interrupted. In 1978 we reported the devastating sequelae of tube disconnection in a boy with GSD type 1. Sixteen years later these unfortunate incidents still occur and we have cared for two additional children with GSD type 1 who suffered profound hypoglycaemia with irreversible brain damage and death. Both were treated with nocturnal nasogastric tube feeds and in both cases parents forgot to switch on the pump.

Pump feed systems were initially designed for nutritional support and the early ones were not very sophisticated as interruption of the feeds does not usually have serious consequences in that situation. However, in children with GSD, because of the potential risks of hypoglycaemia, great care needs to be taken to ensure that the pump is set up correctly, that alarm systems will indicate when there is electrical or mechanical pump failure, and the tubing used for the delivery system and the nasogastric tube is secure. Fortunately a new generation of pumps including these features are now available and should be used in preference to the earlier systems. It is essential that the enteral feeding pump complies with BS 5724:1 (medical electrical equipment: specification for general safety requirements). It should have an accuracy of delivery within  $\pm 10\%$  of the nominal volume in a given time, comprehensive alarm systems, safety interlocks to prevent tampering while running, and battery back-up in case of mains failure. The following alarm features are considered essential:

- Feeding container is empty
- Feeding tube occlusion alarm
- Drop sensors blocked
- Pump set improperly loaded
- Preset dose has been administered
- Unit is left on 'hold' for longer than three minutes
- Safeguard against the risk of overinfusion due to misloading of pump feed onto the pump

(Technical performance and safety assessments of these devices are published by the Medical Devices Directorate of the Department of Health: 0171-636 6811 ext 3141.)

In addition to extensive alarm features the nasogastric tube should have a 'Luer lock' fitting or similar securing device to prevent accidental disconnection.

However, even with these precautions, accidental removal of the nasogastric tube overnight may still occur. We recommend the use of a baby intercom alarm as this may alert parents to changes in their child's breathing or movements which might be indicative of developing hypoglycaemia. The morbidity and mortality of GSD type 1 has been greatly reduced by effective treatment, but it must be emphasised that these treatments are not without risks, and should only be used with meticulous attention to detail.

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### Guidelines and clinical standards

EDITOR,—Professor Lilleyman's article on clinical standards touches on the trend towards guidelines.<sup>1</sup> There is, understandably, no mention of variations in their quality. Although the risk of exposing to litigation those who do not follow guidelines may result in greater compliance, we also face the risk of being 'locked into' some inappropriate or unnecessary practices. One way of reducing this risk might be clearly to distinguish between areas in which there seems to be a compelling case for the suggested course of action and others where a personal or arbitrary stance is adopted.

One such example is advice from the British Committee for Standards in Haematology (BCSH) that the platelet count be raised to at least  $50 \times 10^9/l$  before lumbar puncture.<sup>2</sup> This recommendation is based solely on the routine practice of the authors' hospitals (M E Murphy, personal communication). It may well be unnecessary in one large group of thrombocytopenic children — those with acute leukaemia at presentation. In our centre we have no policy to administer platelets to these children before lumbar puncture, yet have never seen a haemorrhagic complication. Is there a risk of clinically important haemorrhage in these children? Perhaps there is, and we have simply been fortunate over the last 20 years. What is missing is information to support the recommendation. No doubt others can think of examples of similarly questionable but didactic recommendations.

Of course, guidelines can be modified with experience (and the BCSH will look at this particular issue again) but one wonders if the process of modification may be simpler and more timely if areas of ignorance or doubt are not denied by the desire to provide an all encompassing impression of security and direction.

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1 Lilleyman JS. Clinical standards in the reformed NHS. *Arch Dis Child* 1994; 71: 275-6.

2 British Committee for Standards in Haematology, Working Party of the Blood Transfusion Task Force: Murphy MF, Brozovic B, Murphy W, et al. Guidelines for platelet transfusions. *Transfusion Medicine* 1992; 2: 311-8.

### Professor Lilleyman comments:

Dr Reid makes a good point with which I totally agree. There is, though, another aspect to the type of *ex cathedra* based guidelines he worries about, and that is whether anyone takes any notice of them — or even reads them. I would guess that few clinicians have changed their practice of giving or not giving platelet cover for lumbar punctures based on the reference he cites, and that few plan to.

But the fact that the recommendation exists could come back to haunt a clinician

who faces litigation and who did not follow it. So I endorse the suggestion that guidelines should indicate areas of uncertainty. But I also believe that to be effective they should be backed by a system of external peer audit so that compliance with them can be assessed. Guidelines drifting quietly into the literature mostly get filed or forgotten.

### Pseudomonal rectovaginal abscesses in HIV infection

EDITOR,—Borgstein and Broadhead have interestingly described a series of nine cases of acquired rectovaginal fistula in children with presumed vertically acquired HIV infection.<sup>1</sup> They suggest this may be caused by localised perianal sepsis. We would like to report a child with AIDS and severe perianal abscesses due to *Pseudomonas aeruginosa* infection.

A 6 month old infant presented with a rash, hepatosplenomegaly, and a severe bronchiolitic illness associated with disseminated cytomegalovirus infection. HIV antibody, p24 antigen, and the polymerase chain reaction were all strongly positive. The cytomegalovirus infection was successfully treated with intravenous ganciclovir, followed by continued maintenance via a Hickman line. Further illnesses included respiratory infections with respiratory syncytial virus and influenza C. At the age of 13 months the child presented with pus draining from the vagina. A 3-4 cm indurated abscess was noted in the vulva. A further 2-3 cm perirectal abscess was noted on the same side. A swab of the discharge grew a heavy growth of *P aeruginosa*. This was treated with oral ciprofloxacin for 10 days, with complete resolution of both abscesses. There have been no further recurrences, and the child is alive now at 18 months of age.

Rectal and vulvovaginal abscesses are a major problem in immunosuppressed children without HIV infection, and are most commonly caused by anaerobic organisms.<sup>2</sup> Frequent bacterial infections occur in children with HIV infection, including skin and perirectal abscess formation,<sup>3</sup> with pseudomonal infections seen more commonly in children with AIDS. All nine cases reported by Borgstein and Broadhead fulfilled the clinical criteria for AIDS related complex/AIDS, and we suggest that the rectovaginal fistulas seen in these infants may have been secondary to pseudomonal infection.

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- 1 Borgstein ES, Broadhead RL. Acquired rectovaginal fistula. *Arch Dis Child* 1994; 71: 165-6.
- 2 Brook I. Anaerobes. In: Patrick CC, ed. *Infections in immunocompromised infants and children*. London: Churchill Livingstone, 1994: 522-37.
- 3 Krasinski K. Bacterial infections. In: Pizzo PA, Wilfert CM, eds. *Pediatric AIDS*. Baltimore: Williams and Wilkins, 1994: 241-53.

### Acquired rectovaginal fistula

EDITOR,—We have seen examples in Zimbabwe of rectovaginal fistulas in HIV positive infants<sup>1</sup> similar to those described by Borgstein and Broadhead (see table).<sup>2</sup> They were seen over a two year period; they and their mothers were HIV positive.

The fistulas occur just behind the fourchette in the vagina communicating with the rectum just above the levator muscles, the lowest point at which the vagina and rectum are most closely applied. In case 3 the histological findings described non-specific acute and chronic inflammatory tissue from the edge of the fistula. No 'owl's eye' intranuclear inclusions were seen, excluding cytomegalovirus as a cause.

An abnormally opening fistula in ano may be responsible, or ulceration breaking through the anterior wall of the ano-rectum into the vagina. Anal ulceration in adults may be caused by cytomegalovirus, cryptococcus, and herpes simplex. These ulcers are often indolent and run a protracted course. Chronic intersphincteric abscesses and fistulas also occur.<sup>3 4</sup>

All cases were managed by constructing a defunctioning sigmoid colostomy which has given symptomatic relief in all patients. Case 3 showed no evidence of the fistula healing. Death probably occurs fairly soon after the onset of the fistula as only one of the patients has come for regular follow up.

It may be possible to close the fistula surgically, although one would expect a high incidence of wound breakdown and of faecal fistula when the colostomy is closed.

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- 1 Oliver MJ. Spontaneously occurring rectovaginal fistulae in children and adults with HIV infection. *East and Central African Journal of Surgery* 1995 (in press).
- 2 Borgstein ES, Broadhead RL. Acquired rectovaginal fistula. *Arch Dis Child* 1994; 71: 165-6.
- 3 Cope R, Debow JM. Anorectal pathology and acquired immune deficiency syndrome. *Br J Surg* 1992; 79: S3.
- 4 Scholefield JH, Northover JMA, Carr ND. Male homosexuality, HIV infection and colorectal surgery. *Br J Surg* 1990; 77: 493-6.

### Clinical details of four cases of acquired rectovaginal fistula

Case No	Age	Weight (kg)	Duration of signs of fistula (weeks)	Associated features	Course
1	4 Months	5.7	2	Splenomegaly	Lost to follow up
2	8 Months	5.9	1	Failure to thrive, bilateral chronic otitis media, rash on face and trunk	Lost to follow up
3	2-3 Years	11.0	1	Cervical lymphadenopathy, chronic otitis media	Losing weight
4	4 Months	5.0	3	Chronic otitis media, pneumonia	Lost to follow up