of the features of our patients, such as exacerbation by movement (case 1) or percussion (case 2).

The tyro surgeon is warned to beware of right basal pneumonia mimicking an acute abdomen⁵ but pulmonary thromboembolism is not listed as a diagnosis of which to be wary, although it is recorded that cholecystectomy has erroneously been performed in patients with the condition.¹

It is not uncommon at necropsy to find pulmonary thromboembolism that was unsuspected during life. One reason for this is the varied presentation of the condition.^{6,7} We suggest that abdominal pain as a presenting feature deserves to be more widely known.

References

- 1 Israel H, Goldstein R. The varied manifestations of pulmonary thromboembolism. *Ann Intern Med* 1957; 47:200-26.
- 2 Weatherall DJ, Ledingham JGG, Warrell DA, eds. *Oxford textbook of medicine*. Oxford: Oxford University Press, 1983.
- 3 Crofton J, Douglas A, eds. *Respiratory diseases*. 3rd ed. Bath: Pitman, 1981.
- 4 Gorham LW. A study of pulmonary embolism. III. The mechanism of pain; based on a clinicopathological investigation of 100 cases of minor and 100 cases of massive embolism of the pulmonary artery. *Arch Intern Med* 1961;108:418-26.
- 5 Raines AJH, Ritchie HD, eds. *Bailey and Love's short practice of surgery*. 18th ed. London: H K Lewis, 1981:1062.
- 6 Stein PO, Willis PW, Demets DL. History and physical examination in acute pulmonary embolism in patients without preexisting cardiac or pulmonary disease. *Am J Cardiol* 1981;47:218-22.
- 7 Bell WR, Simon TL, Demets DL. The clinical features of submassive and massive pulmonary emboli. *Am J Med* 1977;62:355-60.

(Accepted 31 July 1984)

Clinical Algorithms

Urticaria and angio-oedema

A D ORMEROD

The urticarias are characterised by transient, localised, itchy swellings of the skin. The lesions begin suddenly as erythematous patches which become raised with central paling as oedema develops in the dermis; they last for a few hours but rarely for longer than 48 hours. Angio-oedema is the name given to the same process when it occurs in the subcutaneous tissues. The resulting deep swellings, which often affect the eyelids, lips, and tongue, cause a burning sensation or pain. Angio-oedema and urticaria often occur together, although each may occur on its own.

These conditions affect 15% to 20% of people at some time in their lives.¹ In most cases urticaria is short lived and requires only antihistamines without the need for detailed investigation. Acute urticaria is that which lasts less than two months. It often has an allergic cause and is often associated with atopy. Possible allergens include drugs, injections, food, drinks, and inhaled particles, but in 75% of cases no causal agent is found.¹ Treatment of patients with chronic urticaria is unsatisfactory, and as 20% are still affected after 10 years this can mean years of frustration for the patient and doctor.¹

Urticaria is only rarely due to general medical causes and very rarely due to malignancy. For this reason a thorough history and examination are required, but only patients with relevant symptoms or signs warrant further investigation. Routine investigation of all patients is rarely of value² but is sometimes carried out to reassure the patient and doctor that no causes have been missed.

Physical urticarias

A different approach is required for the physical urticarias,³ in which physical factors, such as friction or cold, trigger the rash, and for contact urticaria,⁴ in which contact with an allergen produces localised weals. A physical cause is more likely if individual lesions disappear within 30 minutes. The patient may not have noticed the physical cause, and it is worth using the

physical provocation tests (described in the flow chart) in all cases of uncertain cause. The commonest physical urticarias are symptomatic dermographism and cholinergic and cold induced urticaria. The morphology of the lesions provides few clues, but 2-3 mm papules suggest cholinergic urticaria while linear lesions occur in symptomatic dermographism. Persistent lesions which resolve with scaling or pigmentation suggest urticarial vasculitis.⁵ Lesions in areas exposed to light are a feature of solar urticaria while disease of the hand and mouth favours contact urticaria.

Preventing urticaria

Avoidance of the factors which commonly exacerbate urticaria will often lead to improvement. The most important is aspirin, which causes an exacerbation in 30% to 50% of patients. All patients should be warned not to take aspirin. Other substances which exacerbate urticaria in fewer patients are the food colouring tartrazine and the food preservative sodium benzoate. A challenge test battery has been used to identify patients in whom these factors are implicated.⁶ Using this, Warin and Smith achieved considerable improvement in 75% of patients with positive reactions by appropriate dietary manipulation; patients were able to relax the diet after five to six months. Provocation tests may be difficult to interpret, they are time consuming, and particularly when aspirin is used severe reactions are possible, making it desirable for the test to be done in an inpatient. An alternative is to administer a low salicylate, low benzoate, and tartrazine free diet for a trial period and continue it only in those showing benefit. Diagnosis of specific food allergy is helped by the patient keeping a diary recording diet and attacks. This is most useful in patients with well defined intermittent attacks. An exclusion diet with reintroduction of foods on successive days need be used only for severely affected patients who have not benefited from other measures.

Particular attention should be paid to any infection, including dental sepsis and chronic sinusitis. Eosinophilia should alert the clinician to the possibility of parasitic infection. Treatment of these infections sometimes leads to improvement in the urticaria. More commonly, the patient's drug treatment is responsible. Penicillin and other antibiotics cause urticaria by an allergic mechanism

Urticaria and angio-oedema

whereas codeine, tubocurarine, morphine, and dextran provoke urticaria by non-immunological potentiation of histamine release.⁷

Intradermal prick tests with routine allergy testing solutions have not proved to be of value,¹ but it is worth performing a scratch test or intradermal injection with candida antigen which is read after 15 minutes and again after four to six hours. Eighty per cent of the patients showing a positive reaction improve when treated with systemic nystatin, amphotericin lozenges, and a yeast free diet.⁸

Treatment

Early in the course of the disease treatment with antihistamines should help all patients to some degree. There is little to choose between the large number of antihistamines available. The duration of action should be considered and the more sedative drugs given at night. The peripheral effect of most antihistamines is delayed by one to three hours so treatment should be continuous rather than intermittent. Doses should be timed according to when the urticaria is more troublesome. In the physical urticarias hydroxyzine has shown some advantages over other antihistamines.⁹ The sedative action of antihistamines can be advantageous in the anxious patient and those whose sleep is disturbed, but when sedation is not required terfenadine, a recently introduced antihistamine, is effective without affecting driving ability; astemizole may prove to have similar advantages. Patients with chronic urticaria who do not respond to antihistamines may benefit from terbutaline and ketotifen,¹⁰ but side effects of tremor

and palpitations are common. There is conflicting evidence for the role of cimetidine, which can exacerbate urticaria when used alone, perhaps because of inhibition of n-methyl transferase.⁹ Two studies showed that the combination of hydroxyzine and cimetidine was more effective than hydroxyzine alone.^{11 12}

In hereditary angio-oedema, a condition associated with deficiency of C₁ esterase inhibitor, treatment with danazol can prevent attacks, which are potentially fatal if they affect the larynx. It is therefore important to investigate this possibility when persistent subcutaneous swellings occur in childhood, when there is a family history, or when there is recurrent severe colicky abdominal pain which arises due to gastrointestinal disease.

- 1 Warin RP, Champion RH. *Urticaria*. London: Saunders, 1974.
- 2 Jacobson KW, Branch LB, Nelson HS. Laboratory tests in chronic urticaria. *JAMA* 1980;243:1644-6.
- 3 Jorizzo JL, Smith EB. The physical urticarias: an update and review. *Arch Dermatol* 1982;118:194-201.
- 4 Krogh GV, Maibach HI. The contact urticaria syndrome—an updated review. *J Am Acad Dermatol* 1981;5:328-42.
- 5 Warin RP. Urticarial vasculitis. *Br Med J* 1983;286:1919-20.
- 6 Warin RP, Smith RJ. Challenge test battery in chronic urticaria. *Br J Dermatol* 1976;94:401-6.
- 7 Munroe EW. Urticaria. *Int J Dermatol* 1981;20:32-41.
- 8 James J, Warin RP. An assessment of the role of *Candida albicans* and food yeasts in chronic urticaria. *Br J Dermatol* 1971;84:228-37.
- 9 Black AK. New approaches in treatment of physical urticarias. *Clin Exp Dermatol* 1982;7:301-10.
- 10 Saihan EM, Littlewood SN. Ketotifen and terbutaline in chronic urticaria. *Br J Dermatol* 1983;109(suppl 24):31.
- 11 Munroe EW, Cohen SH, Kalbfleisch J, Shultz CL. Combined H₁ and H₂ antihistamine therapy in chronic urticaria. *Arch Dermatol* 1981;117:404-7.
- 12 Harvey RP, Wegs J, Schocket AL. A controlled trial of therapy in chronic urticaria. *J Allergy Clin Immunol* 1981;68:262-6.

Clinical curio: psychotherapy initiated after prenatal determination of fetal sex

Detection of fetal sex is now generally possible from late in the second trimester by high resolution real time ultrasonography.¹ This can be helpful in some sex linked conditions and obstructive uropathies; we also found it to be useful in another case. A 34 year old Chinese woman presented in her third pregnancy. She had had a history of neurotic depression since the birth of her second daughter and feared that she would have another girl. As in many communities, a boy was considered to be essential in the family. Her husband had forbidden her to have an abortion. Ultrasonography at 28 weeks showed the fetus to be female. The mother was not informed of this, but psychotherapy was initiated and was continued post partum, when she had thoughts of killing herself and the baby. She was discharged about two weeks after delivery when her psychiatric condition had become stable. At subsequent follow up visits she did not show any signs of distress or depression.

Ultrasonography, in addition to being non-invasive, had the advantage of allowing psychotherapy to be initiated before delivery, thus possibly preventing a severe psychotic reaction.—K C MAK, medical officer, J S K WOO, v c w WONG, University of Hong Kong.

1 Scholly TA, Sutphen JH, Hitchcock DA, Mackey SC, Langstaff LM. Sonographic determination of fetal gender. *AJR* 1980;135:1161-4.

A 57 year old man underwent a transurethral prostate resection in 1981. Despite preoperative assurance by the urologist that his potency would in no way be affected, it has worsened steadily. No emotional or organic cause can be found. What treatment is recommended?

Probably the patient was having problems with potency before the operation in order to need assurance from the surgeon that surgery to the prostate would not adversely affect him in this respect. It is always wise to warn a patient before transurethral surgery to the prostate and bladder neck that he is very likely to have retrograde ejaculation after the operation. In explaining this to the patient it is reasonable to add that if there are no existing problems with penile erection then there is no organic reason why the surgery should adversely affect him in this respect. No firm guarantee, however, can be given as there is a small but not insignificant percentage of men in this age group who do

experience impotence after both upper and lower abdominal surgery including prostatectomy, whether the latter is performed transurethral or by the open route. If this patient is having good spontaneous erections then the problem of impotence is likely to be psychogenic. Referral of both the patient and his wife to an expert for psychosexual counselling is indicated. If this is not the case, or if psychotherapy fails, then there are now available a variety of implantable silicone rods which may be inserted into the corpora to effect a surgical cure of impotence. With careful patient selection the results are excellent.¹⁻³—J C GINGELL, consultant urologist and lecturer in urology, Bristol.

- 1 Jonas U, Jacobi GH. Silicone-silver penile prosthesis: description, operative approach and results. *J Urol* 1980;123:865-7.
- 2 Small MP. Small-Carrion penile prosthesis. A new implant for management of impotence. *Mayo Clin Proc* 1976;51:336-8.
- 3 Scott FB, Boyd GJ, Karacan I, Olsson P, Beutler LE, Attia SL. Erectile impotence treated with an implantable inflatable prosthesis, 5 years of clinic experience. *JAMA* 1979;241:2609-12.

What treatment is advised for blisters on the feet of an 11 year old girl with epidermolysis bullosa?

Epidermolysis bullosa is a rare disease with at least 20 different subgroups. As the most severe forms are fatal this 11 year old patient presumably has one of the less severe forms. Her symptoms probably result from pressure. Attention to good footwear is, therefore, essential, and advice from the medical appliance office could be most helpful in minimising "excessive" trauma of the feet from walking and possibly inappropriate footwear. The blisters occasionally may be tense and create discomfort. The child's parents must therefore be instructed to clean the surface of the blister with an antiseptic lotion, such as chlorhexidine (Hibiscrub) or povidone-iodine (Betadine), and then to burst the blister with a sterile needle. It may be necessary to rupture the blister in two or three places, so that all the fluid is adequately drained, and 24 hours later the blister roof may be removed. Topical antiseptics should be applied to any raw area; possibilities include chlorhexidine (Naseptin) cream and silver sulphadiazine (Flammazine). Such treatments will prevent the risk of secondary infection. There is no evidence that topical steroid creams are of benefit. The mainstay of treatment is therefore general advice to minimise excessive trauma and to prevent secondary infection. There is no cure.—W J CUNLIFFE, consultant dermatologist, Leeds.