enzyme-linked immunosorbent assay using fimbrial haemagglutinin as antigen. *J Infect Dis* 1982;**146**:741-5.
8 Gillenius P, Jäätmaa E, Askelöf P, Granström M, Tiru M. The standardization of an assay for pertussis toxin and antitoxin in microplate culture of Chinese hamster ovary cells. *J Biol Stand* 1985;**13**:61-6.
9 Granström M, Granström G, Gillenius P, Askelöf P. Neutralizing antibodies to pertussis toxin in whooping cough. *J Infect Dis* 1985;**151**:646-9.
10 Orenstein WA, Bernier RH, Dondero TJ, *et al*. Field evaluation of vaccine efficacy. *Bull WHO* 1985;**63**:1055-68.
11 Medical Research Council. The prevention of whooping-cough by vaccination. *Br Med J* 1951;i:1463-71.
12 Laurell G, Mellbin T, Rabo E, Vahlquist B, Zetterquist P. Systematische Impfung mit kombiniertem Impfstoff (Diphtherie, Tetanus, Pertussis) im Säuglingsalter. Serologische und klinische Studien. *Klin Wochenschr* 1957;**35**:920-4.
13 Cherry JD. The epidemiology of pertussis and pertussis immunization in the United Kingdom and the United States: a comparative study. *Curr Probl Pediatr* 1984;**14**:1-78.
14 Preston NW, Mackay RI, Bamford FN, Crofts JE, Burland WL. Pertussis agglutinins in vaccinated children: better response with adjuvant. *Journal of Hygiene (Cambridge)* 1974;**73**:119-25.
15 Butler NR, Wilson BDR, Benson PF, Dudgeon JA, Ungar J, Beale AJ. Response of infants to pertussis vaccine at one week and to poliomyelitis, diphtheria, and tetanus vaccine at six months. *Lancet* 1962;ii:112-4.
16 Blennow M, Granström M, Olin P, *et al*. Preliminary data from a clinical trial (phase 2) of an acellular pertussis vaccine, J-NIH-6. *Dev Biol Stand* 1986;**65**:185-90.

(Accepted 26 January 1988)

SHORT REPORTS

Small bowel volvulus in two siblings

We report two cases of small bowel volvulus occurring in brothers, one with tragic consequences. Cases of intestinal atresia, probably as a result of intrauterine volvulus, have been reported in siblings,[1,2] but volvulus alone has not.

Case reports

Case 1—A 4½ year old boy presented with a three hour history of vomiting, abdominal pain, and drowsiness. He had previously been well, and his medical history was unremarkable. On examination he was not feverish but was tachycardic, tachypnoeic, and peripherally shut down. His abdomen was soft and not obviously tender or distended. Before much investigation had been carried out, however, his condition deteriorated further, and in spite of vigorous attempts at resuscitation he died. Postmortem examination showed that virtually all of the small intestine was ischaemic, with twisting of the root of the mesentery. The cause of death was recorded as volvulus of the small bowel.

Case 2—The younger brother of the patient in case 1 was referred to this hospital at the age of 4 with a history of two episodes of sudden abdominal pain associated with profuse vomiting. These episodes settled quickly without treatment, and no haematological or radiological abnormalities were found when he was examined, although his symptoms had subsided by this time. Because his parents were naturally worried that the symptoms were caused by small bowel volvulus, as in their first son, a laparotomy was performed. This was decided on because even if other investigations—for example, barium enema or barium meal and follow through—had yielded normal results the parents would not have been reassured until a laparotomy had been performed. Surprisingly, at laparotomy the bowel was found to be malrotated and the small bowel to be suspended on a long, narrow based mesentery, which showed signs of previous episodes of volvulus. The malrotation was treated, and he made a good recovery, experiencing no further problems.

Comment

Malrotation of the small intestine is caused by disordered movement of the intestine around the superior mesenteric artery during embryological development.[3] Two main abnormalities that produce clinical syndromes may occur. In the first narrowing of the base of the mesentery occurs, which may allow the midgut to twist and cause a volvulus. This may occur acutely, causing complete obstruction, or intermittently, producing bouts of obstruction that resolve spontaneously. In the second Ladd's bands are present, which rarely cause acute symptoms.

Malrotation can cause symptoms at any age, but most cases are seen in childhood. Most children who develop symptoms related to volvulus do so within the neonatal period, presenting with features of intestinal obstruction. Unless the obstruction is partial persistent vomiting occurs from the onset. The vomitus may or may not contain bile. Abdominal distension is often absent.[4] Blood in the stool or vomit is a serious sign of impending bowel infarction. Features in plain abdominal radiographs vary, but duodenal dilatation and malposition of bowel loops are usually present. Dehydration occurs rapidly, and resuscitation and early laparotomy are essential. Children with malrotation who present later in childhood may do so with features of intermittent obstruction, such as intermittent episodes of vomiting and abdominal pain,[3] as in case 2.

Laparotomy is indicated when symptoms occur, and ischaemic bowel may need resection. When ischaemia is extensive resection may not be feasible, and after the volvulus has been untwisted a second investigatory operation should be considered. Ladd's operation (or one of its various modifications) is usually the procedure of choice.[5]

Volvulus during intrauterine life may cause intestinal atresia, which has been reported in siblings.[1,2] In the "apple peel syndrome" there is duodenal or high jejunal atresia, and an autosomal recessive type of inheritance has been postulated. Volvulus occurring in infants without atresia has not previously been reported in siblings.

The outcome in the first case was most unfortunate, but the parents' wish to have a laparotomy performed on their second son may have saved him from the same fate.

1 Mishalany HG, Najjar FB. Familial jejunal atresia: three cases in one family. *J Pediatr* 1968;**73**:753-5.
2 Dickson JAS. Apple peel small bowel: an uncommon variant of duodenal and jejunal atresia. *J Pediatr Surg* 1970;**5**:595-600.
3 Walker-Smith JA, Wright V. Congenital abnormalities of the gastrointestinal tract. In: Weatherall DJ, Ledingham JGG, Warrell DA, eds. *Oxford textbook of medicine*. Vol 1. Oxford: Oxford University Press, 1983:12-144.
4 Atwell JD. Pitfalls in the diagnosis of intestinal obstruction in the newborn. *Proc R Soc Med* 1971;**64**:26-9.
5 Cathcart RS, Williamson B, Gregorie HB. Surgical treatment of midgut non-rotation in the adult patient. *Surg Gynecol Obstet* 1981;**152**:207-10.

(Accepted 16 February 1988)

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Effect of topical corticosteroids on seasonally induced increases in nasal mast cells

Symptoms of allergic rhinitis caused by grass pollen become more severe as the pollen season progresses despite falling pollen counts, a phenomenon known as "nasal priming."[1]

We have shown that there is a substantial and significant increase in the density of mast cells in the nasal mucosa of patients who have seasonal allergic rhinitis in summer compared with that in winter,[2] which would facilitate an interaction between cell bound immunoglobulin and allergen and offer an explanation for nasal priming. The topical administration of corticosteroids is an established and effective treatment for allergic rhinitis, but its mode of action remains unknown. We investigated the effect of a topical corticosteroid, beclomethasone dipropionate, on seasonally induced increases in the density of mast cells.

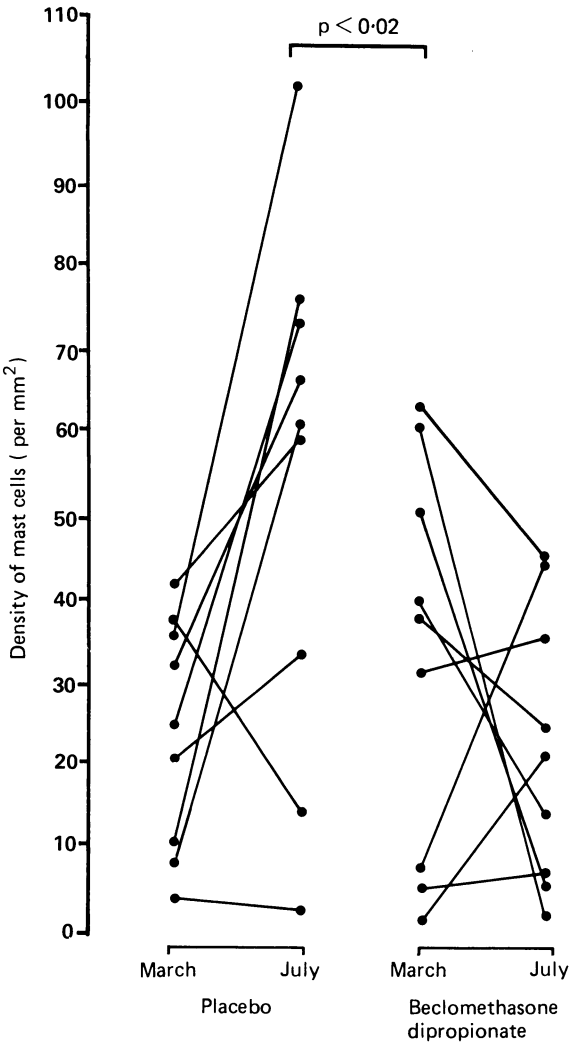
Patients, methods, and results

Nine men and nine women (mean age 23·7 years) were studied. All had a five year history of seasonal rhinitis and gave positive results to skin prick tests with mixed grass pollen (2·5% weight/volume) and negative results to skin prick tests with house dust (150% weight/volume) and house dust mite (1·2% weight/volume.) A biopsy sample was taken from the inferior turbinate of each patient during the first week of March 1986 before the grass pollen season started. Patients were randomised (with a random number table) in May 1986 to receive double blind treatment with beclomethasone dipropionate (Allen and Hanburys Ltd, Greenford, UK) or placebo 100 μg twice a day to each nostril from identical

spray devices. Treatment started immediately for 10 weeks. Nasal biopsies were repeated in the second week of July 1986.

All biopsy samples were coded and examined blind. After being fixed in Carnoy's solution the sections were stained with α-naphthol AS-D chloroacetate esterase staining reaction. In addition, adjacent sections were stained with toluidine blue at pH 0·5. The mast cells were counted by light microscopy. The cross sectional area of each biopsy section was determined with a planimetric method and the cell count per mm^2 calculated (Imagan Standard, Leitz Instruments, Luton, UK). The coefficient of variation of the mast cell counts in sections was 4·2%.

The mean paired differences in the density of mast cells in the two groups (March and July) were compared by Student's t test. The extent of agreement between the two staining techniques was investigated by plotting the difference between the two methods against the mean of the two methods.[3] Though no agreement between the staining techniques was seen, in July both methods showed similar significant differences in the density of mast cells in the group treated with beclomethasone dipropionate compared with the group given placebo (figure; $p<0·02$, Student's t test).



Density of mast cells (per mm^2) in biopsy samples from single patients treated with placebo and beclomethasone dipropionate before treatment at start of pollen season and after three months.

Comment

Our results suggest that beclomethasone dipropionate inhibits the increase in density of mast cells that occurs in the nasal mucosa during the pollen season. Atopic patients have been shown to have increased numbers of mast cell progenitors in the circulation,[4] and the migration of such progenitors into the nasal mucosa may be inhibited by corticosteroids. Alternatively, these drugs may act by inhibiting the growth and differentiation of mast cell progenitors in situ. The maturation and migration of such progenitor cells are thought to be dependent on T cell lymphokines.

Interleukin 3, the lymphokine believed to have the main role in regulating mast cells, has now been identified in humans.[5] Though the mechanism of action of corticosteroids is likely to be complex, our study suggests that corticosteroids may influence maturation or migration, or both, of mast cells, possibly by inhibiting the production of interleukin 3.

Whatever its mode of action, we believe that treatment with beclomethasone dipropionate should be started before the pollen season begins to prevent increases in mast cell density and nasal priming.

We are grateful for financial support to perform this study from the Joint Research Board of St Bartholomew's Hospital and the Asthma Research Council.

1 Connell JT. Quantitative intranasal pollen challenge. II. Effect of daily pollen challenge, environmental pollen exposure, and placebo challenge on the nasal membrane. *J Allergy* 1968;**41**:123-39.
2 Viegas M, Gomez E, Brooks J, Davies RJ. Effect of the pollen season on nasal mast cells. *Br Med J* 1987;**294**:414.
3 Bland MJ, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986;i:307-10.
4 Denburg JA, Telyzyn S, Belda A, Dolovich J, Bienenstock J. Increased numbers of circulating basophil progenitors in atopic patients. *J Allergy Clin Immunol* 1985;**76**:466-72.
5 Yang YC, Ciarletta AB, Temple PA, *et al*. Human IL3 (multi CSF): identification by expression cloning of a novel hematopoietic growth factor related to murine IL3. *Cell* 1986;**47**:3-10.

(Accepted 8 February 1988)

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Kinesiology and food allergy

The report on food intolerance and food aversion noted that a wide variety of symptoms have been incorrectly attributed to the effects of food and recommended that diagnostic tests for food allergy should be strictly evaluated.[1] In the proceedings of a recent symposium kinesiology was reviewed under the heading "Untested or invalid methods for diagnosis,"[2] though in the subsequent discussion it was claimed that the effect on which the test depends "exists and is repeatable."[3]

Patients, methods, and results

The kinesiology test is performed as follows. Allergens to be tested are prepared in stoppered neutral glass bottles. The patient holds the bottle in one hand, and a positive test is indicated by a decrease in muscle power in the contralateral arm. No physiological basis for this effect is known. To see if the effect "exists and is repeatable" a set of 12 prick test solutions (Bencard, Brentford, Middlesex) of milk, cheese, candida, maize, yeast, ethanol, beef, lamb, pork, cod, orange, and grasses was prepared and correctly labelled, and another set of 12 identical bottles was prepared and labelled A-L. These contained (in random order and in duplicate) prick test solutions of milk, cheese, candida, maize, and yeast, the remaining two bottles containing saline solution. The tester was asked, when the response was positive to one or more of the labelled bottles, to test all the "blind" bottles on the same patient and to score the results on a form provided.

The table shows the results obtained with 20 consecutive patients who entered the trial. On open testing six patients gave a positive reaction to milk (cases 1, 2, 10, 11, 13, and 14). These six patients then had 12 blind challenges with milk, of which only one (case 2, second blind challenge) gave a positive result. Among the 14 remaining patients who gave negative results to milk on open testing there were four positive reactions on blind testing. If the reactions to milk, cheese, candida, maize, and yeast were summed eight of 50 blind tests (16%) gave positive results among those patients who gave positive results on open testing with these antigens, and 24 of 150 (16%) gave positive results among patients who gave negative results on open testing. Saline solution caused positive reactions in seven of 40 blind tests (18%).

Because the blind testing was done on duplicate samples we were able to see if the concordant results within the pair (that is, both positive or both negative) exceeded the frequency that would be predicted by chance. This analysis showed that the number of concordant results within duplicates was similar to that that would be expected by chance, given an overall positive response rate of 39 of 120.

Comment

Our experience indicates that the kinesiology response is not reproducible under conditions of blind testing and therefore cannot be a reliable indicator of food allergy. This does not deny that patients who have undergone the test