retention of its contents. Treatment is directed to the lower oesophageal sphincter and not at the body of the oesophagus. The sphincter may be disrupted by forceful dilatation or incised by a Heller's cardiomyotomy; most authorities favour the surgical approach.[5]

Diffuse spasm of the oesophageal muscle is thought to be even less common, with an incidence of about one-fifth that of achalasia.[6] Epidemiological data are, however, scanty, and the true incidence may be very much greater.[7] Asymptomatic persons may nevertheless show typical radiographic and manometric changes, with intermittent localised non-progressive contractions. Manometry shows non-sequential, frequently repeated pressure waves of high amplitude and long duration. In contrast with achalasia, these findings are usually restricted to the distal oesophagus, and the lower oesophageal sphincter is often normal. The sphincter may, however, be affected in one-third of patients, and a few may present a similar clinical picture even though the disorder is confined to a hypertensive or hyperacting lower oesophageal sphincter.[8] Apart from muscular hypertrophy no characteristic pathological changes are found in the oesophagus in diffuse spasm.

The cardinal symptoms of diffuse spasm are chest pain and dysphagia. They are not only intermittent but they vary in frequency and severity and do not necessarily worsen with time. Symptoms tend to occur in tense, nervous people and are often precipitated by emotional stress. The pain is not necessarily related to eating or drinking and may even occur at night. Manometric studies have shown that pain correlates with the length of a contraction rather than its height. Food rarely becomes impacted; if this happens the diagnosis should be reconsidered and an organic stricture looked for. Again, in contrast to achalasia the lumen is empty, and neither spillover into the lungs nor oesophageal carcinoma occurs as a complication. Medical treatment is usually ineffective, and patients can be advised only to eat slowly, chew food into small boluses, and avoid cold or fizzy drinks. Forceful dilatation is less successful than in achalasia and is probably best avoided. Surgical treatment should be reserved for severe cases; the short-term results of long oesophagomyotomy are favourable,[9] but we lack information from long-term follow-up, and surgery is probably of less benefit than in achalasia.

In spite of these differences between diffuse spasm and achalasia, there are certain similarities. There is no sex difference in either disease and both occur in adults of any age, though diffuse spasm is more common in older patients. Some patients with achalasia complain of chest pain and show manometric features of both disorders. In the United States of America the syndrome is called "vigorous achalasia," and some British authorities regard it as the early stage of the disease,[11] though it is probably no more than one end of the clinical range of achalasia.[3] Nevertheless, there may be a transition from diffuse spasm to achalasia.[12] Further evidence that these two disorders may share a common aetiology is provided by a recent report of two siblings, one of whom had diffuse spasm and the other achalasia.[13] Clearly we have much to learn about oesophageal dysmotility.

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Blows from the winter wind

A so-called "comfort index"[1] relates not to the activities of the festive season but to the weather affecting towns throughout the United States. Mortality from ischaemic heart disease was found to be associated with the comfort index but not with socioeconomic factors. In British towns West and Lowe[2] have shown that mortality from ischaemic heart disease is associated with socioeconomic conditions as well as temperature and rainfall; the effects of the three factors are independent of each other, and temperature is the most highly correlated with mortality. The gradients from south-east to north-west in Britain are from low to high mortality from ischaemic heart disease, from warm to cool and dry to wet weather, and from high to low socioeconomic standards.

Yet seasonal variations in temperature throughout Britain far exceed variations among towns. Last winter the freeze in the first week of the New Year affected the whole of the country. There were about 10% more deaths registered in the next week than the average for the five weeks after Christmas, and the whole cold spell produced about 10% more deaths than in the corresponding period of the previous winter. Mortality in the north-west was higher than in the south-east, but the freeze was associated with no greater relative increase in the north than in the south.

The effect of cold is greatest in the elderly; even without severe freezing weather there seems always to be an increase in mortality from ischaemic heart disease in those aged over 65, and even more in those over 75, shortly after the winter drop in temperature.[3] This susceptibility to cold is enhanced by polluted air or respiratory infections. Air pollution is now less severe than in the past. The Clean Air Act was drawn up after the 1952 London smog, which was blamed for 4000 excess deaths. The lesser smog in December 1962 produced about 500 excess deaths in London. But despite the "deadly cold,"[4] which continued through January 1963—cases of frostbite being admitted to London hospitals[5]—there were fewer deaths in that month than in the corresponding period the previous year,[6] probably because influenza was less prevalent.

If winter comes, can spring be far behind? While we all look forward to the spring and then to the summer, the heat waves of June 1968, August 1975, and June-July 1976 caused, at their peak, as many deaths in one day as the peak of the corresponding winters; this lethal effect was apparent for only about one day in 1966, but for several weeks in 1975 and 1976.[7]

We cannot alter the climate, and doctors have little influence on the socioeconomic environment of less privileged areas

such as the north-west of Britain. Nevertheless, since those at risk are mainly the elderly it should be possible, with the attention of doctors as well as families, to improve their "microenvironment" by giving them warm clothing and adequate domestic heating.[2] In a caring society few should be entirely lacking in comfort and joy at this time of the year.

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How necessary are elimination diets in childhood?

When most babies were breast-fed ailments in the newborn were commonly attributed to the thinness, inadequacy, or disagreeable qualities of the milk and the infant was changed to cows' milk preparations. Now that most babies are bottle-fed, cows' milk has become the culprit and is blamed for eczema, asthma, behaviour disorders, and constipation. In fact, the only disorders that have been proved to be due to cows' milk are bleeding from the gut[1] and chronic diarrhoea. Yet at times the outcry—even by well-established paediatricians—has become so emotional that before long, no doubt, demands will be heard that dried milks should be labelled "this preparation may harm your health."

As the questions and warnings about the real and imagined dangers of cows' milk have spread from doctors to the public, many mothers have looked for alternatives. About one-third of the infants receiving bottle feeds in the United States have a milk substitute based on soya bean protein. In Britain these preparations cost about five times more than cows' milk preparations. The results of short-term studies have suggested that growth of infants fed on soya protein is similar to that of babies reared on cows' or breast milk. The results of long-term studies of growth and intellect are not yet available, nor are there yet reliable estimates of the number of infants who react adversely to the new preparations.

The second effect of the widespread belief that cows' milk may explain almost any symptom in an infant has been some uncontrolled enthusiasm among paediatricians for the use of "elimination diets." For example, Atherton and colleagues have recently reported a dramatic success for a milk-free, egg-free diet in some children with infantile eczema.[2] Fortunately publicity is being given to the risks as well as the merits of dietary modification: the potential hazards of fashionable macrobiotic diets were reviewed in a *BMJ* article last year.[3] Another study showed how elimination diets can result in inadequate caloric intake and failure to thrive.[4] Gluten-free diets had been prescribed for large numbers of children without a specific diagnosis being made; detailed investigation showed that most did not have a serious underlying disorder and did not require a diet at all.

The value of elimination diets has been documented best in coeliac disease and intolerance to cows' milk protein. Infants less than a year of age with chronic diarrhoea need systematic investigation. Examination of the stool for a pathogenic *Escherichia coli* and giardia can be performed as a preliminary test in the outpatient clinic. Next a full blood count, a sucrose and lactose tolerance test, and a sweat test should be arranged, and finally a jejunal biopsy. There are no agreed diagnostic criteria for cows' milk protein intolerance, but the diagnosis is reasonably certain if symptoms recur on three challenges,[5] or if changes can be shown in the villi on microscopy of biopsy specimens before and after a challenge,[6] or changes in IgM plasma cells in the mucosa after a challenge.[7] If an elimination diet is prescribed it should be supervised by both a paediatrician and a dietitian, since how long the diet is needed cannot be assessed when it is first prescribed—regular challenges are necessary to determine how long dietary restriction must be continued. A rigid diet may be unpalatable and lead to deficient intake of calories.

The confusion that may be produced by uncontrolled prescription of elimination diets has been highlighted by the results of uncritical prescription of gluten-free diets (without a prior small intestinal biopsy) for children suspected of having coeliac disease. This approach is rare in Britain but apparently is still common in the United States,[4] despite the diagnostic confusion which may arise with the need for later reinvestigation to determine whether permanent gluten intolerance exists.[8] Perhaps a larger problem, in both Britain and the United States, is the unnecessary prescription of elimination diets for non-specific chronic diarrhoea, variously known as the peas/carrots syndrome, toddler's diarrhoea, and the irritable colon syndrome of infancy, in children over 1 year who are growing normally. Apart from examining the stool for pathogens and parasites no investigations are required and no treatment is needed provided the infant is growing normally—as shown by progress on growth charts. These children grow out of their symptoms by the age of 3 or 4 years. Most often, probably, the condition has some benign basis such as failure to chew properly—foods such as beans are characteristically seen in the stools. We have no evidence that removing some foods from the diet is of any real value in this self-limiting syndrome.

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