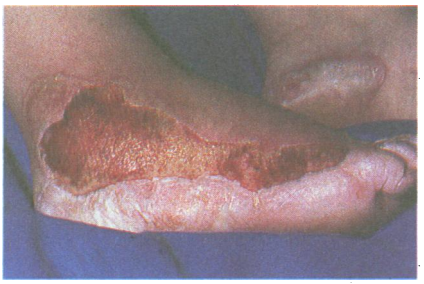

Trauma—Patients with diabetic or other types of neuropathy are at risk of developing trophic ulcers. Rarely they may be self induced—"dermatitis artefacta."

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In treating venous leg ulcers
(1) Take measures to eliminate oedema and reduce weight—make sure the patient understands these.
(2) Never apply steroid preparations to the ulcer itself or it will not heal. Make sure that both nurses and patients are aware of this.
(3) Beware of allergy developing to topical agents—especially to antibiotics.
(4) There is no need to submit the patient to a variety of antibiotics according to the differing bacteria isolated from leg ulcers, unless there is definite evidence of infection clinically.
(5) A vascular "flare" round the ankle and heel with varicose veins, sclerosis, or oedema indicates a high risk of ulceration developing.
(6) Make sure arterial pulses are present.

Epidemiology

Report from the PHLS Communicable Disease Surveillance Centre

A case of botulism was reported in August and another of infant botulism two months earlier; an outbreak of haemorrhagic colitis took place in the West Midlands in July and August; and two zoonoses received publicity during September, particularly in the veterinary press—rabies in bats and liver fluke disease

Botulism

A case of botulism, widely reported in the press, occurred in a man aged 49 who developed vomiting about 10 hours after eating a prepacked shelfstable kosher meal on a flight from Nice to Heathrow on 30 August. Later he developed diplopia, dysphagia, dysarthria, and increasing generalised paralysis and required artificial ventilation. The clinical diagnosis was made promptly and treatment with antitoxin given. A high titre of type A *Clostridium botulinum* toxin was detected in his serum and in gastric aspirate, which also contained the organism.

The implicated meal included a rice and vegetable dish which was said to have smelt offensive; the victim consumed less than a teaspoonful before discarding it. Other members of his family immediately rejected the dish after opening the sealed containers. All the meal was discarded and none was available for examination. These meals were withdrawn by all airlines and no further cases were reported. Unfortunately it was not possible to identify any rice and vegetable dishes from the same production batch for examination, but three of these dishes which were obtained were not sterile, being contaminated with *Bacillus* spp. Neither *C botulinum* nor botulinum toxin were detected.

Botulism is rare in the United Kingdom. Since the famous Loch Maree outbreak in Scotland in 1922, when eight people died after eating sandwiches containing duck paste, only 25 cases with 14 deaths have been reported in eight incidents. The most recent outbreak took place in Birmingham in 1978. Four people were affected, two of whom died, after eating canned salmon from a tin which was later shown to be defective. Most of these incidents were due to *C botulinum* toxin type A, but some were also due to types B and E.

Botulism has been reported more often in North America, where it is often associated with defective home preserved vegetables, in continental Europe, where it is usually associated with cured meats or vegetables, and in Japan, where it is usually associated with raw fermented and smoked fish.

Botulism should be suspected when neurological symptoms, characteristically visual disturbances, difficulty in swallowing and speaking, and flaccid paralysis occur in a mentally alert patient in association with gastrointestinal symptoms or with epidemiological evidence suggesting a contaminated food. The incubation period is usually less than 36 hours, though it depends on the dose of toxin consumed and it may be as long as eight days. Laboratory confirmation is by detecting toxin in the patient's serum (20-50 ml blood should be obtained before the administration of antitoxin), faeces, or vomitus and by the subsequent isolation of the organism from the suspected food or its container.

Advice and reference laboratory facilities are available at the PHLS food hygiene laboratory, Colindale (01 200 4400), and the public health laboratory, Luton (0582 571898). Supplies of trivalent equine botulinum antitoxins (types A, B, and E) for therapeutic purposes are available through designated centres listed in the *Health Service Purchasing Guide* (section D), the *Chemist and Druggist Annual Directory*, the *PHLS Directory*, and the *Pharmaceutical Supplies Bulletin* 1985;8:1-10. (See Communicable Disease Report of 14 February 1986 (86/07).) Epidemiological advice is available from the PHLS Communicable Disease Surveillance Centre (CDSC) (01 200 6868), from local public health laboratories

in England and Wales, and from the Communicable Diseases (Scotland) Unit (041 946 7120).

Infant botulism

Botulism in adults is almost always caused by the ingestion of preformed botulinum toxin produced by the organism growing in a foodstuff under anaerobic conditions. In 1976 in the United States an infection due to *C botulinum* was described in infants, in whom symptoms were caused by toxin produced by the organism during multiplication in the intestine. In this disease the affected infant, usually 2 to 6 months of age, becomes constipated and "floppy," its cry diminishes, it feeds poorly, there is generalised weakness with ptosis, and symmetrical lower motor neurone paresis follows. There is a wide variation in severity, from minor weakness and lethargy to the sudden infant death syndrome.

The first case in the United Kingdom was reported in London in 1978. Another case was recently reported in Bristol and was initially thought to be a case of vaccine associated poliomyelitis. The 5 month old child developed symmetrical lower motor neurone weakness of all four limbs three weeks after the first dose of oral poliomyelitis vaccine and later developed difficulty in breathing requiring mechanical ventilation. Infant botulism was suspected and later confirmed by the detection of *C botulinum* type F and toxin in faeces. Honey has been implicated as a source of infection in some of the cases in the United States, but neither of the British cases was associated with this food and their source of infection was not identified.

Advice and reference facilities are available from the sources quoted above. About 5 to 10 mg of faeces and 2-3 ml of serum are required for laboratory investigation, and the laboratory should be telephoned before the specimens are sent. Because only small quantities of toxin are likely to be present and the organism can be difficult to isolate results will not normally be available for several days.

Haemorrhagic colitis

Haemorrhagic colitis is another newly described disease also first reported in the United States. It is characterised by the sudden onset of severe abdominal cramps, bloody diarrhoea, and either a low grade fever or no fever. In 1982 two outbreaks in Michigan and Oregon were associated with beefburgers from the same source, and verotoxin producing strains of *Escherichia coli* serotype O 157 H7 were isolated from the affected people and from a sample of the implicated beefburgers. Since then outbreaks have been reported in the United States, Canada, and Japan, some of them associated with meat products and one with milk. There was a single outbreak in the United Kingdom, in East Anglia in July 1985, in which there was epidemiological evidence suggesting that handling locally produced vegetables, particularly potatoes, was associated with infection. These might have been contaminated by slurry. Sporadic cases have also been reported; a study of 83 apparently sporadic cases in 1985-6 in England and Wales showed that 32 of those affected had infections with verotoxin producing *E coli*, and 30 of these were serotype O 157.

Similar verotoxin producing strains of *E coli* have been isolated from patients with the haemolytic uraemic syndrome, which comprises acute renal failure, thrombocytopenia, and haemolytic anaemia. Most of these cases were sporadic but outbreaks have also been described. In the United Kingdom surveillance of the haemolytic uraemic syndrome by the joint scheme of the British Association of Paediatric Nephrologists the CDSC, and the PHLS division of enteric pathogens, augmented in mid-1986 by reports through the British Paediatric Surveillance Unit, has detected between 31 and 113 cases each year from 1983 to 1986. One outbreak of at least 15 cases was reported during the summer of 1983 in the West Midlands, but, despite intensive investigation, the vehicle of infection and the source were not identified.

In July and August 1987 another outbreak of infection with verotoxin producing *E coli* O 157 H7 occurred in the West Midlands after a christening party; 22 out of 93 people developed gastrointestinal symptoms, many with bloody diarrhoea, and one with the haemolytic uraemic syndrome. Although epidemiological evidence implicated cooked meats served at the party, no samples were available for microbiological study and the source of the organism was not found. Active case searching after this episode identified 12 sporadic cases of the haemolytic uraemic syndrome and of haemorrhagic colitis in the West Midlands, six of them with onset of symptoms in the last week of July; five of the 12 cases were associated with verotoxin producing *E coli* O 157 H7 infection. Detailed inquiry did not show a link between any of them and the christening party, nor was a common factor found. Many of those affected had eaten cooked meats of various types, however, and the distribution and sources of these products are being studied.

Clinicians should bear in mind the association of verotoxin producing *E coli* O 157 H7 with haemorrhagic colitis and the haemolytic uraemic syndrome and ensure full faecal microbiological examination of patients in whom they suspect these diagnoses so that the vehicles of infection and sources of this organism can be identified and controlled.

Bat rabies

In September the Ministry of Agriculture, Fisheries, and Food drew attention to the isolation of rabies virus from bats in continental Europe. Rabies transmission from vampire bats to cattle and man has been known since early this century and is confined to the American continent. European bats are insectivorous and likely to bite only if handled. Rabies virus was isolated on rare occasions from these bats in Germany, Yugoslavia, and Turkey in the 1950s and later in Poland, Denmark, Finland, and again in Germany. The recent isolates were similar to the Duvenhage virus, which was first isolated from a man bitten by a bat in South Africa in 1970. Only one human case of bat rabies has been reported in Europe, in a bat research worker in Finland in 1985.

Rabies has not been found in bats in the United Kingdom and does not present a public health problem. Nevertheless, it is wise not to handle sick bats, and anyone inadvertently bitten should seek advice about rabies prophylaxis. The available diploid cell rabies vaccine probably provides protection and should be used for postexposure prophylaxis, together with human rabies immunoglobulin if indicated, when rabies is suspected in the biting animal. Licensed bat handlers should be offered a pre-exposure course of rabies vaccine, which is obtainable from the PHLS Virus Reference Laboratory (01 200 4400).

Liver fluke disease (fascioliasis)

Another announcement from the Ministry of Agriculture reported that the incidence of liver fluke disease in cattle and sheep is likely to be the highest this autumn since the mid-1960s because the wet cool summer had led to an increase in the population of the snails, *Limnaea truncatula*, which are the intermediate host of the liver fluke, *Fasciola hepatica*.

In 1968 and 1969 two outbreaks of human fascioliasis comprising over 50 cases took place in Shropshire and Gloucestershire and were associated with eating watercress that had become contaminated by infected cattle. After these outbreaks a PHLS working party published guidelines for the hygienic production of watercress and recommended excluding animals from ground adjacent to watercress beds. Since then there have been no more than five human cases reported yearly; most occurred in people infected abroad, only two being indigenous cases in people who had eaten wild watercress. The human infection is manifested by fever, malaise, weight loss, and pain in the right upper abdomen with enlargement of the liver and eosinophilia. Cultivated watercress is now safe, and wild watercress is not sold for human consumption. Nevertheless, patients who prefer to "pick their own" may contract the disease, especially during the next few months.