Epidemiology

Report from the PHLS Communicable Disease Surveillance Centre

Meningococcal meningitis received wide publicity during August, a case of human rabies was reported, and a further 25 cases of the acquired immune deficiency syndrome (AIDS) were reported.

In September a notable outbreak of salmonella food poisoning took place affecting nearly 200 delegates at a medical conference in Cardiff; a single case of salmonellosis was traced to contaminated enteral feed; overall, laboratory reports of salmonellosis showed an increase of nearly 2000 compared with the first three quarters of 1985. Lyme disease received publicity because of reports of cases from the New Forest, and a postal survey of dermatologists showed that there were at least 68 cases in Britain in the past five years. The incidence of the acquired immune deficiency syndrome continues to increase, and analysis of cases by date of presentation to medical care showed no evidence of any falling off in the rate of increase.

Meningococcal meningitis

There were 583 notifications of meningococcal meningitis in England and Wales up to the end of August 1986 with a projected total for the year of about 700 (1·40 per 100 000 per year) compared with 548 (1·09 per 100 000 per year) in 1985. The reason for this increase is unknown but it was associated with localised outbreaks due to *Neisseria meningitidis* group B type 15, particularly in the north west and the west country, as described earlier this year.⁴ The most notable of these outbreaks took place in Gloucestershire, where in 1986 there were 12 notifications up until August, an annual rate of $2 \cdot 36$ per 100 000 per year. The size of the present epidemic is small compared with previous epidemics, however; notifications were about half those of the peak of the previous rise in 1974 and about one sixteenth the size of the very large epidemic in 1940 (fig 1).

In Norway, where the B15 strain was prevalent earlier than in England and Wales, the number of notifications rose in 1974, particularly in northern Norway. Unexpectedly, the high level of notified cases continued at between six and eight per 100 000 per year in the 1980s, unlike previous epidemics which were of shorter

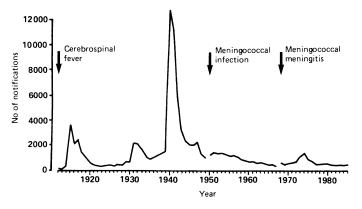


FIG1—Notifications of meningococcal infection and meningococcal meningitis in England and Wales 1912-85.

duration with a rapid fall in cases to pre-epidemic levels. A new notification system was introduced in Norway in 1975 which increased ascertainment of the disease from about half to three quarters of cases, but this could explain only a small part of the difference between this high level and that of two to three per 100 000 in the early 1970s.

The much higher notification rates seen in Norway than in England and Wales may be due partly to different methods of notification. In the new Norwegian system notifications are made both by clinicians and laboratories, and the notifiable condition is "meningococcal infection" including both meningitis and septicaemia, whereas in England and Wales notifications are made by clinicians and only "meningococcal meningitis" is notifiable. If, therefore, trends in England and Wales were to follow the Norwegian pattern the rise in notifications is likely to be less than in Norway and to persist at lower levels.

No vaccine is yet available against the group B meningococcus and primary cases cannot, therefore, be prevented. It is possible, however, to attempt to limit the spread to secondary cases and for this purpose chemoprophylaxis with rifampicin is usually recommended for the close household contacts of a case due to a sulphonamide resistant organism (though in Norway this has been replaced by a course of penicillin in therapeutic doses). Less close contacts, such as social contacts outside the household, should not usually be given chemoprophylaxis. The classic studies which suggested a relation between the incidence of meningococcal meningitis and overcrowding in barrack rooms has led to the suggestion that organised social gatherings in crowded premises should be avoided during epidemics. There is, however, no evidence that such restrictions limit the spread of infection. In schools, if two or more cases occur closure is sometimes suggested, but again there is no scientific evidence to justify such action, and most authorities recommend appropriate prophylaxis.

In localities with a high incidence of the infection clinicians need to be alert to ensure early diagnosis and prompt treatment. In these places it has been suggested that general practitioners should start intramuscular penicillin treatment in suspected cases immediately rather than allowing this to be delayed until the patient's arrival in hospital.

Rabies

A British born woman of 46 was admitted to hospital in Portsmouth on 15 August with rabies and died two weeks later. She was a resident of Lusaka, Zambia, where in mid-June a stray dog had entered the compound surrounding her house and attacked her guard dog. In attempting to separate the dogs she was bitten—but she believed the bite came from her own dog, who was vaccinated against rabies, and so she did not seek medical advice about postexposure rabies prophylaxis.

This was the 16th case of human rabies reported in the United Kingdom since 1940. One patient reported in 1946 was infected in Greece, one in 1964 in Indonesia, and the remaining 14 were infected in the Indian subcontinent or Africa. Rabies is endemic in the semidomesticated dog population in these regions, in contrast to Europe, where it is endemic in the fox population and where the dogs are fully domesticated and usually protected by immunisation and much less likely to be infected.

Recently in Europe a rabies related virus, similar but not identical to the Duvenhage virus first isolated from a man bitten by a bat in South Africa in 1970, has been reported in bats in countries bordering the Baltic Sea—Denmark, West Germany, Poland, and Finland. One associated human case of rabies was reported in a bat research worker in Finland in 1985. The World Health Organisation has indicated that this presents no hazard to the general population and that for people exposed to risk the currently available rabies prophylaxis also protects against this virus.

Rabies was present in the dog population throughout the nineteenth century in Britain, but it never became established in foxes or other wildlife. Thus when muzzling of dogs, destruction of strays, and quarantine of imported animals was vigorously applied at the turn of the century the disease was quickly eradicated. The disease was reintroduced in dogs in 1918 after the first world war but was again eradicated in 1921. Since then Britain has remained free of animal rabies (except in quarantined animals) by the strict control of importation of animals and by compulsory vaccination and quarantine of imported dogs and cats.

Rabies in man almost always follows the bite, scratch, or lick of a rabid animal. Human to human transmission by corneal transplantation from donors who died of undiagnosed rabies has been described, and airborne spread is theoretically possible. For this reason and also because of the possible contamination of broken skin or mucous membranes with saliva the close contacts of human cases, including health care staff, are usually offered vaccination, as was done in the Portsmouth incident.

Vaccination before possible exposure is recommended for special groups who are at risk, such as people concerned with the importation and quarantine of animals. For these groups, which are listed in the Rabies Memorandum published by the Department of Health and Social Security in 1977, vaccine is available free from National Health Service sources. Pre-exposure vaccination should also be considered, however, for people leaving Britain to travel or work in remote areas where rabies is enzootic and where postexposure vaccination is not available quickly if required; for these individuals vaccine should be obtained from commercial sources. In Britain human diploid cell vaccine is recommended, and a course usually consists of 1.0 ml given by deep subcutaneous injection in two doses four weeks apart with a reinforcing dose after 12 months and at one to three yearly intervals while the risk of exposure continues, though intradermal injection of 0.1 ml is sometimes given using the same schedule.

Travellers from Britain to any country where rabies is present may be at risk, especially if they ill advisedly allow themselves to come into close contact with animals. If they are bitten or scratched the wound should be flushed with clean water immediately and cleaned with soap or detergent, and medical advice on prophylaxis should be sought. For those not previously immunised recommendations are: human rabies immunoglobulin when indicated, 20 IU per kg body weight, up to one half locally around the wound and the other half intramuscularly, plus human diploid cell vaccine, six doses of 1 ml by deep subcutaneous injection on days 0, 3, 7, 14, 30, and 90. Advice on prophylaxis and supplies of immunoglobulin and vaccine are available from the Public Health Laboratory Service in England and Wales, from the Communicable Diseases (Scotland) Unit in Scotland, and from the Department of Health and Social Services in Northern Ireland.

Salmonellosis

Over 414 delegates attended the conference of the British Diabetic Association in Cardiff on 4-6 September, and on the last day many became ill with abdominal pain, severe watery diarrhoea, and vomiting. *Salmonella typhimurium* was isolated from stools of some of those affected and preliminary investigation suggested that the infection was due to the consumption of chicken served at a cold buffet lunch on 5 September. Indeed, *S typhimurium* was later isolated from the chicken by the Public Health Laboratory, Cardiff,

and was shown by the PHLS division of enteric pathogens to be phage type 9—the same phage type as that isolated from the patients.

Another incident, fortunately affecting only one person, took place at the end of August in Oxford. A child, who had been admitted to hospital after a road traffic accident, developed profuse bloody diarrhoea, and *S derby* of an unusual antibiotic sensitivity pattern was isolated from his stools. He had received enteral feed the previous day, and the same organism was isolated from the empty discarded bottle of the feed. The Public Health Laboratory, Oxford, then examined unopened bottles and isolated an identical salmonella from one of eight bottles of the same production batch as was fed to the child but not from any of 40 bottles of another production batch. The affected batch, which was distributed only to National Health Service hospitals, was immediately withdrawn and no further cases were reported. Investigation of the possible source of contamination of the feed is continuing.

These two incidents highlight the important risk of salmonellosis in mass catering and in the mass production of foodstuffs, which have become increasingly prominent features of our food supply in recent years. Although the main sources and modes of spread of salmonella infection have been known for many years, attempts to control the disease have largely failed, as is illustrated by the almost continual rise in notifications of food poisoning and in laboratory reports of salmonellosis since the 1940s (fig 2).

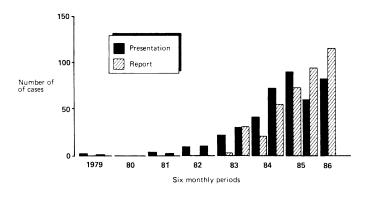


FIG 2—Food poisoning and salmonellosis in England and Wales 1941-84. *Only bacteriologically confirmed incidents included.

Salmonellosis is a zoonosis and therefore requires concerted veterinary and medical action to achieve its control, but this is unlikely to develop until there is greater medical pressure for its prevention. Many doctors still regard salmonellosis as an unimportant mild diarrhoeal illness, a view which the delegates to the Cardiff conference will dispel, especially those admitted to hospital, and which national statistics do not support. In the past decade 1976-85 there were over 100 000 reported salmonella infections, about 1500 (1.5%) of which were extraintestinal, including bacteraemia, meningitis, bone and joint infections, and abscesses; in these 10 years there were 447 deaths in which salmonellosis was certified as the main cause of death.

Lyme disease

Lyme disease has been reported in many European countries and appears to be common in southern Sweden and Austria, where about 400 cases were recorded in Vienna in 1984. Little is known about the incidence of the disease in Britain; the first reported case was from south east Scotland in 1977, 13 cases were reported in East Anglia in 1984, mainly from the Thetford area, and more recently further cases have been reported from the New Forest.² To obtain an indication of the incidence of erythema chronicum migrans in Britain members of the British Society for Dermatologists were sent a questionnaire in August 1986 asking how many cases they had seen in the past five years. Although only just over half the members had responded by the end of September, they recorded at least 68 cases. Of these, 13 patients acquired the disease abroad and the British cases were recorded mainly in East Anglia, Hampshire, Sussex, Oxfordshire, Yorkshire, south Wales, and Scotland. Dermatologists are invited to return the questionnaire to the Communicable Disease Surveillance Centre or the Communicable Diseases (Scotland) Unit if they have not already done so, so that a more complete picture may be obtained.

Serological tests for Lyme disease have been developed, although special studies have shown that these are not always positive in culture proved cases. The tests are available at the department of microbiology, Charing Cross Hospital, or through the Public Health Laboratory Service at the Leptospira Reference Unit, Public Health Laboratory, Hereford. It is proposed to begin soon the routine collection of reports of positive laboratory results through the existing national laboratory reporting system so that cases presenting with arthritic, neurological, or other manifestations, as well as skin disease, may be included in a continuing national surveillance System. In the mean time the Communicable Disease Surveillance Centre (telephone 01 200 6868) and the Communicable Disease (Scotland) Unit (telephone 041 946 7120) would be pleased to learn from clinicians of cases coming under their care.

The acquired immune deficiency syndrome (AIDS)

During September 1986 a further 22 cases of AIDS were reported to the Communicable Disease Surveillance Centre and Communicable Disease (Scotland) Unit, giving a cumulative total of 512 with 250 deaths. These cases are tabulated by patient characteristics (table), which have been revised since the last report in the $BM\mathcal{J}$ (13 September, p 680). The characteristics are now mutually exclusive and the cases associated with sub-Saharan Africa have been allocated to the most probable group, 10 of them to "heterosexual contact."

There were two cases of AIDS in children (table). One of these was infected by a blood transfusion in the United States and the other was a child of seropositive African parents; of the 19 cases in haemophiliacs one was under 15 years old. These data probably under-represent the true incidence of AIDS in children because many paediatricians and other clinicians outside the specialty of genitourinary medicine are probably unfamiliar with the national reporting system for AIDS. To ensure more complete reporting of paediatric AIDS and disease related to the human immunodeficiency virus in children paediatricians have been invited to report cases through the newly established British paediatric surveillance unit reporting system. This reporting scheme is a joint development of the Communicable Disease Surveillance Centre, the British Paediatric Association, and the Institute of Child Health, and the data collected on AIDS will be collated by the Communicable Disease Surveillance Centre with reports from all other sources and presented in the centre's monthly reports on AIDS.

The monthly reports of cases of AIDS over the past 12 months

Acquired immune deficiency syndrome in Britain to 30 September 1986

Patient characteristic	No of cases			No - of
	Male	Female	Total	deaths
Homosexual/bisexual man	457	0	457	209
Intravenous drug abuser	5	1	6	2
Homosexual and intravenous drug abuser	2	0	2	2
Haemophiliac	19	0	19	18
Recipient of blood	7	2	9	7
Heterosexual contact:				
Britain	2	2	4	3
Abroad*	5	7	12	7
Paediatric (intrauterine or perinatal infection)	0	2	2	1
Other	0	1	1	1
Total	497	15	512	250

*Includes 10 associated with sub-Saharan Africa.

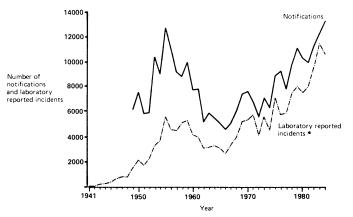


FIG 3—Cases of AIDS by date of presentation and date of reporting in UK 1979-86. No information was available in 46 cases.

ranged from 16 to 76 and do not provide a satisfactory indicator of the trend in the epidemic. A better measure is the date of presentation to medical care aggregated into six month periods. In fig 3 these data are shown together with date of presentation up to 30 June 1986. There is a continuing upward trend with no evidence of any decline in the rate of increase. Since over 40% of cases were reported more than six months after first presentation to medical care, more cases are likely to be reported later with dates of presentation in 1985 and 1986. There was a similar relation between date of reporting and presentation in the second half of 1984 and first half of 1985, and if this remains constant the number of reported cases in six month periods will become a reasonably accurate measure of the trend.

The reporting of cases and suspected cases of AIDS is essential to maintain national surveillance of the disease. Clinicians are therefore invited to telephone or write, in confidence, to the director or one of the other medical epidemiologists at the Communicable Disease Surveillance Centre (61 Colindale Avenue, London NW9 5EQ. Tel 01 200 6868) or Communicable Disease (Scotland) Unit (Ruchill Hospital, Glasgow G20 9NB. Tel 041 946 7120) if they become aware of a case so that the necessary details for surveillance may be recorded. Most physicians in genitourinary medicine and medical microbiologists are familiar with the national reporting system and would be able to provide information about the system to other clinicians if asked.

References

1 Communicable Disease Surveillance Centre. Report from the PHLS Communicable Disease Surveillance Centre. Br Med J 1986;292:1447-8.

2 Williams D, Rolles CJ, White JE. Lyme disease in a Hampshire child-medical curiosity or beginning of an epidemic? Br Med J 1986;292:1560-1.

Is arthritis a recognised manifestation of cat scratch disease?

Cat scratch disease is characterised by enlargement of lymph nodes, usually in the head, neck, or axillae. There may also be fever and splenomegaly. The diagnosis is supported when a papule develops at the site of a scratch with enlargement of regional lymph nodes occurring simultaneously or up to about a month later. The enlarged nodes typically yield sterile pus but the presence of Gram-negative bacilli in skin lesions and from the nodes has been reported.¹ None the less, there is no benefit from antibiotics, the condition subsiding spontaneously within two months. None of Margileth's series of 707 patients had arthritis.² Rarely, superficial cat scratches have been followed by septic arthritis, particularly in immunosuppressed patients.— C J ELLIS, consultant physician, Birmingham.

1 Wear DJ, Margileth AM, Hadfield TL, et al. Cat scratch disease: a bacterial infection. Science 1983;221:1403.

2 Margileth AM. Cat scratch disease. In: Cecil textbook of medicine. Philadelphia: Saunders, 1985.