Clinical Topics

Changing patterns of communicable disease in England and Wales

Part III—Increasing infectious diseases

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Food poisoning, malaria, pertussis, sexually transmitted diseases, and typhoid fever have increased in England and Wales and cholera has reappeared after an absence of over 70 years.

Food poisoning

Food poisoning was made notifiable in 1938, but not until 1949 were data from this source collected centrally and brought together with reports from medical officers of health and laboratory data in a series of publications describing annual trends.¹ The overall trend in the past 20 years is illustrated by notifications which reached a peak of over 12 000 in 1955 and then declined to about 5000 in 1972 but later increased; in 1978 there were 9739 notifications (fig 1). Study of bacterial food poisoning reported by laboratories shows that this recent increase was mainly due to a rise in salmonella food poisoning caused by serotypes other than *Salmonella typhimurium*, particularly *S hadar* (fig 1). This followed changes in animal husbandry, in particular the intensive production of poultry, and a rise in the consumption of poultry meat.² Salmonella infections due to many new serotypes were introduced into the breeding stocks in the 1960s by the importation of infected day-old chicks and hatching eggs and into the poultry flocks by contaminated animal feeding stuffs; infection then spread to the human population. In outbreaks of salmonellosis in which the vehicle of infection was identified, 12% were attributed to poultry in the five years 1960-4 but this rose to 70% in 1976-8. Although reports of *S typhimurium* infections have not increased, the appearance of multiple antimicrobial resistant strains in outbreaks of illness in calves and man in Britain is worrying because in some countries the acquisition of multiple resistance by salmonellae has been accompanied by enhanced virulence and communicability.³

In contrast to salmonella food poisoning staphylococcal food poisoning decreased from about 100 incidents each year in the early 1960s to usually less than 20 each year in the 1970s, although there was a rise in 1975 and 1976, possibly attributable to the hot summers in those two years. This overall decline probably resulted from improvement in food hygiene and personal hygiene but it may also have been partly associated with changes in food habit. For example, salted preserved meats, which selectively favour the growth of staphylococci

FIG 1—Incidence of food poisoning and salmonellosis in England and Wales 1960-79: notifications and reports of salmonella infections and reports of *Staph aureus* and *Cl perfringens* infections.

Notifications of food poisoning and laboratory reports (incidents) of salmonella infections (upper diagram) and laboratory reports (incidents) of *Staph aureus* and *Cl perfringens* infections (lower diagram).

when contaminated, have decreased in popularity since frozen cooked meats have become readily available. *Clostridium perfringens* food poisoning also declined in 1978, probably for similar reasons, although the fall was much less striking (fig 1).

The first cases of botulism in England and Wales since 1955 were reported in Birmingham in 1978⁴; they were due to *Cl botulinum* type E. Four patients, two of whom died, developed the disease after eating canned salmon from a 7½-oz tin which was subsequently shown to be contaminated with the organism

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and to have a damaged rim with a small leak. The salmon was probably contaminated after processing while the can was cooling. Extensive investigations failed to discover any other affected cans.

Three newly recognised causes of food poisoning in England and Wales were described during the 1970s. Two bacterial causes, *Bacillus cereus* food poisoning,⁵ usually associated with rice, and *Vibrio parahaemolyticus* food poisoning,⁶ associated with sea food, usually imported, were together responsible for 20 to 30 incidents each year. The third newly recognised cause of food poisoning was viral: a large outbreak of gastroenteritis associated with eating cockles occurred in the autumn of 1976 and small round virus particles were shown in the faeces of affected patients.⁷

Malaria

The incidence of malaria in England and Wales during the present century was reviewed by Bruce-Chwatt and Abela-Hyzler.⁸ Indigenous malaria was reported after the first and second world wars but ceased in 1948. Imported malaria declined to its lowest recorded level of less than 100 cases a year in the early 1960s but rose sharply at the beginning of the 1970s and in 1979 reached 2053, the highest figure since 1920. Malaria surveillance in England and Wales was undertaken by the Malaria Reference Laboratory, London School of Hygiene and Tropical Medicine, where data derived from statutory notifications, laboratory reports to the Communicable Disease Surveillance Centre, and reference reports were collected. These showed that the dramatic rise in the 1970s was most evident in people making short visits abroad, particularly tourists, and in immigrants. The rise corresponded with the greatly increased air passenger traffic between India and Africa and the United Kingdom and with the resurgence of malaria in many parts of the world.

Of 2053 cases reported in England and Wales in 1979, 1001 (49%) were in people who had acquired the disease in the Indian subcontinent and in 978 (98%) of these the cause was *Plasmodium vivax*. Many of these cases were in settled immigrants returning to their countries of origin for holidays who did not take prophylaxis because they failed to appreciate that the incidence of the disease in their homeland had increased over fiftyfold since they left in the 1960s. Africa was the second most frequent continent of infection; in 500 (24%) cases the disease was acquired there in 1979, mainly (287 cases) in West Africa. In 311 (69%) of these cases the cause was *P. falciparum*. There were five deaths in 1979, all of which were due to *P. falciparum* acquired in Africa.

Malaria is now an important clinical problem in travellers arriving in England and Wales from endemic areas, and clinicians need particular vigilance to detect and treat falciparum malaria in patients from Africa, early enough to prevent death. Although chloroquine resistance is common in south-east Asia and South America, there have been only a few isolated reports of resistance in east Africa,⁹ where it still remains the drug of choice.

Pertussis

Whooping cough was made notifiable in England and Wales in 1940. The incidence and severity of the disease from 1940 to 1976, the effects of immunisation introduced nationally in 1957, and the complications of immunisation were reviewed by the Joint Committee on Vaccination and Immunisation¹⁰ of the Department of Health and Social Security. The notification rate increased from 1950 to 1953 but by 1957 had fallen to pre-1950 levels. The rate then more than halved from 1958 to 1961 after the introduction of immunisation on a national scale, and the downward trend continued until 1976. Neurological complications after immunisation were reported with varying

frequency, but, as the Joint Committee pointed out, no wholly reliable estimate of the incidence of these complications could be made retrospectively because the neurological conditions also occurred in children of the same age but unrelated to immunisation. Nevertheless, the evidence available indicated that the risks of serious complications were slight and were outweighed by the advantages of immunisation.

Whooping cough remains a serious disease, especially in infancy.¹¹ Despite the availability of an effective vaccine, the public controversy surrounding its complications led to a dramatic fall in national acceptance rates from 70% to 80% in 1965-74 to about 30% in 1978. A natural epidemiological experiment followed: in 1977-9 the largest outbreak of whooping cough since the mid-1950s took place (fig 2), confirming the efficacy of immunisation. A local study in Hertfordshire showed that it conferred 92% protection in children under 4 years.¹² Although the case fatality rate was lower than in any previous epidemic, a decline which has been evident since the 1940s,¹⁰ morbidity in the next few years will probably resemble that of the 1950s unless infant immunisation acceptance rates improve substantially.

FIG 2—Annual notifications of whooping cough in England and Wales 1940-79, and vaccination acceptance rates 1964-79.

Sexually transmitted diseases

Trends in the incidence of sexually transmitted diseases have been measured by data provided by special clinics since they were set up in 1916, and these have provided the main source of information for a series of reports in the *Annual Reports* of the Chief Medical Officer.¹³

In the 1950s the trends in sexually transmitted diseases reached the lowest levels ever recorded; total new cases of syphilis declined to about one-sixth of the number of cases recorded in the postwar peak of 1946 and gonorrhoea to about one-third. It appeared that control of these diseases was at last being achieved and that eventually eradication was possible. The last two decades, however, have seen these hopes evaporate as the trends in incidence reversed in England and Wales and in other Western countries.¹³ By 1969 the postwar peak in gonorrhoea had been exceeded and by 1972 the total number of new cases of all diseases seen at STD clinics was more than double that of 1960 (see table) and over three times that of 1955.

The number of cases of early syphilis, primary and secondary syphilis, was small compared with postwar experience but these nearly doubled between 1960 and 1978 (see table). The increase was almost entirely confined to men, especially homosexuals, although a rise also took place in young women in 1976 and 1977 which was not sustained in 1978. The trends in syphilis in England and Wales compare favourably with those in other Western countries, where much greater increases were reported.¹⁴ This measure of control may have been achieved by

Sexually transmitted disease 1960-78 for England and Wales: cases seen in STD clinics (DHSS clinic returns SBH 60)

Year	Total new cases	Primary and secondary syphilis		Gonorrhoea		Non-specific genital infection in men
		Men	Women	Men	Women	
1960	129 506	819	175	26 618	7 152	22 004
1961	141 361	965	234	29 519	7 588	24 472
1962	138 210	995	229	28 329	7 109	26 494
1963	146 657	951	176	27 895	8 154	25 001
1964	152 721	1172	209	29 050	8 615	27 521
1965	159 028	1469	274	27 876	8 805	29 141
1966	164 763	1132	242	27 921	9 562	30 462
1967	176 274	1075	246	30 645	11 184	32 318
1968	195 352	1085	235	32 595	12 367	35 721
1969	227 744	1043	222	36 379	14 881	41 189
1970	253 326	969	193	37 811	16 933	47 292
1971	236 737	963	224	37 929	18 059	60 708
1972	245 413	1030	192	36 095	18 879	64 271
1973	271 205	1329	220	38 901	21 357	70 440
1974	272 645	1425	260	38 466	21 288	71 400
1975	279 356	1445	216	38 452	22 134	71 401
1976	290 553	1535	243	38 115	22 011	74 944
1977	304 099	1578	295	37 793	22 289	77 998
1978	310 763	1543	222	36 241	21 265	79 875

the long-established clinic-based contact tracing system combined with early effective treatment and follow-up of cases. Another achievement was the very low incidence of congenital syphilis: fewer than 23 cases in the first year of life were reported in each year since 1960, and in 1978 there were only seven, a success attributed to the antenatal serological screening programme.

No such success attended efforts to control gonorrhoea. There was a continuous rise from the mid-1950s reaching over 60 000 cases in 1975, after which the increase levelled off with a slight decline to about 57 500 cases in 1978. The increase since 1960 has been proportionately greater in women, in whom the number of cases in 1978 was about three times that of 1960 whereas in men the increase was about 36%. The increase in women was greater in women aged 15-24 years. In 1976 penicillinase-producing strains of gonococci appeared in a localised outbreak in Liverpool,¹⁵ but this was successfully brought under control and has not recurred. Subsequently, isolated cases have been reported from many parts of the country but in most of these there was a history of infection in West Africa or the Far East, where β -lactamase-producing gonococci are endemic. Nevertheless, the emergence of penicillin-resistant strains of gonococci remains a serious threat to the control of the disease in the United Kingdom.

Non-gonococcal urethritis in men was first reported by clinics as a separate entity in 1951, when nearly 11 000 cases were recorded, a figure which doubled by 1960 (see table). In 1971 these cases were included under the more general heading of non-specific genital infection in clinic returns, but most of the subsequent increase to over three and a half times the 1960 figure in 1978 was due to an increase in non-specific urethritis. It is now twice as common as gonorrhoea in men and has proved equally difficult to control. The causative organisms are not known, but many of the cases are probably due to chlamydial infection.¹⁶

Typhoid and paratyphoid fevers

Enteric fever, including typhoid and paratyphoid fevers, was first made notifiable in 1911 and separated into these two diseases in 1938. In 1973 notifications and laboratory reports were integrated to provide information about typhoid, paratyphoid A, and paratyphoid B by country of infection. Some of this information was available for typhoid fever from 1967.

In 1938 there were nearly 1000 notifications of typhoid fever with 144 deaths; this declined to under 100 cases in 1960 with three deaths. A large outbreak in Scotland in 1964 of over 500 cases due to canned corned beef¹⁷ led to improved hygiene in the canning factories to prevent post-processing contamination

and eliminated this source of infection. In the succeeding 10 years to 1974 notifications remained almost constant at about 150 cases a year, but in an increasing proportion the infection was acquired abroad—80% by 1974. The numbers of cases increased from 1975 to 1979 to nearly twice the annual average of the 1960s, and in 1979 there were 235 cases, in 85% of which infection had been acquired abroad, principally in the Indian subcontinent (fig 3).

Paratyphoid A fever has never been endemic in the United Kingdom but it has increased in incidence recently due to infection acquired in the Indian subcontinent (fig 4). Like typhoid, paratyphoid B fever changed from an indigenous disease to an imported disease (fig 4), but, unlike typhoid fever, in most of the imported cases the infection was acquired in the

FIG 3—Notifications and laboratory reports of typhoid fever in England and Wales 1960-79. From 1967 the number and percentages of cases infected abroad are shown.

FIG 4—Notifications and laboratory reports of paratyphoid fever in England and Wales 1960-79. From 1973 the numbers and percentages of cases infected abroad of paratyphoid A and paratyphoid B are shown.

Mediterranean or Middle East. The overall decline in paratyphoid fever from 1960 to 1978 may be attributed to the identification of two major sources of infection: Chinese frozen whole egg¹⁸ and desiccated coconut¹⁹ and the subsequent pasteurisation of imported bulk egg products and heat treatment of desiccated coconut. Two notable outbreaks took place associated with infection in cattle, one a milk-borne outbreak of over 750

cases in 1965²⁰ and the other a waterborne outbreak of over 80 cases in 1970.²¹

In 1977-9 there were 129 cases of enteric fever infected in England and Wales comprising 87 typhoid fever, 8 paratyphoid A fever, and 34 paratyphoid B fever. Nearly half of the affected patients were aged under 15 years, and most of these were children in immigrant households. Among the adult cases of typhoid fever four were probably infected by duodenal intubation in a gall-stone clinic, four were in an outbreak associated with a chronic carrier in a restaurant, and three were in laboratory workers, one in the husband of a laboratory worker, and one in a nurse. Another laboratory worker was reported with paratyphoid A fever and another nurse with paratyphoid B fever.

Cholera

Cholera disappeared from the United Kingdom at the turn of the century. The last indigenous case was reported in 1893 and there were imported cases up to 1909. The global spread of the el tor biotype of *Vibrio cholerae* began in the Far East in 1961, reaching Europe in 1971,²² and following this pandemic the disease was introduced once again into England and Wales. Twenty imported cases were reported from 1970 to 1978 but in none was there any spread of infection. There were no deaths. Further importations may take place but spread is unlikely because case-to-case spread does not usually occur and sewage contamination of water supplies or food stuffs, the usual mode of spread, is improbable with modern hygiene and sanitation.

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What is the risk of cross-infection from a school drinking fountain; if there is such a risk what bacteria and viruses might be responsible?

In modern drinking fountains the nozzle is so placed that the user would not normally touch it: a jet of water is emitted at a convenient angle for catching it directly in the mouth. The older type of "drinking fountain" was a misnomer, usually consisting of a drinking mug often suspended from a chain and bowl or container. When there was no mug the user had to cup his hands under the outlet and, if the gadget was manually operated, it was necessary to place the mouth directly over, and possibly in contact with, the outlet. While this eventuality raises obvious aesthetic objections, especially where doting owners refresh their dogs in this manner, there is no evidence to suggest that there is any practical risk of cross-infection. Theoretically, dysenteric organisms, staphylococci, streptococci, and viruses that are excreted in the saliva as in, for instance, mumps, herpes, Epstein-Barr virus, and the enteroviruses, could be transmitted through the old type of apparatus, but the possibility is so negligible as to be discounted.

For what sort of patient with low back pain are injections of a sclerosant solution recommended, what is the solution used, and into what are the injections given?

Sclerosant injections are usually recommended for patients with minor (in the pathological sense) mechanical low back lesions that produce regularly recurring pain. They are not recommended for serious or sinister spinal problems or for patients with definite neurological features. The commonly used solution is phenol dextrose glycerine diluted with local anaesthetic, and it is injected into the tenoperiosteal junctions of the supraspinous, iliolumbar, posterior sacroiliac, or sacrotuberous ligaments. Small volumes are injected into each site, in a course of three injections. The injection is sometimes painful but

always safe so long as the point of the needle is touching bone, thereby avoiding any chance of injecting the spinal canal and major nerves. It is postulated that painless fibrosis is provoked which by its contraction stabilises the joints concerned, and relief is thus apparent some two weeks after the trial injection. The occasional dramatic and rapid relief of pain suggests that sometimes the phenol actually blocks a minor pain-transmitting nerve.

A 16-year-old girl with cerebral palsy since birth has recently found reference in an American encyclopaedia to brain pacemakers attached to the cerebellum. Might this procedure help her or can you suggest any other treatments?

Cerebral palsy is a descriptive term for various non-progressive neurological deficits present from birth. Spastic weakness is the most common, though is often associated with cerebellar incoordination and extrapyramidal deficits such as athetosis, mental retardation, or epilepsy. Electrical stimulation of the cerebellum by electrodes applied directly to the cerebellar cortex has been used to treat the spasticity of cerebral palsy,¹ and it is claimed to produce a decrease in muscle tone. The technique is not widely available and should be regarded as experimental and not recommended for routine use. Treatment with conventional spasmolytics such as dantrolene (Dantrium) or baclofen (Lioresal) may improve the spasticity of cerebral palsy, but the clinical response is often unsatisfactory because of associated athetosis, for which there is no effective treatment. Choline improves some forms of cerebellar ataxia² and may offer hope for patients with predominantly cerebellar difficulties. Occupational, speech, and physio therapies may be of benefit in selected cases.

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