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Paralytic poliomyelitis in England & Wales, 1970–84

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SUMMARY

In 1962 the Public Health Laboratory Service (PHLS) became responsible for the Poliomyelitis Surveillance Scheme for England and Wales, which since 1970 has included the World Health Organisation (WHO) enquiry into Acute Persisting Spinal Paralysis. All the records have been kept, including those of patients who were later considered not to have had poliomyelitis. This paper reviews the cases between 1970–84 of patients normally resident in England and Wales, where the clinical features of the illness were considered by the clinician in charge to be those of poliomyelitis and in which either poliovirus was isolated or there was serological evidence of recent infection. Seventy cases met these criteria. Two patients died. A wild strain of poliovirus was isolated in 19 cases; a vaccine-like strain in 27; an intermediate strain in 5; and in 19 cases the strain was not known or there was no isolate. Eleven patients had a history of overseas travel; 17 had been vaccinated recently; and 12 had been in contact with a recent vaccinee. In the remaining 30 cases, the source of the infection was not found. Other details, including the age distribution, vaccination history and the laboratory findings are discussed.

INTRODUCTION

Poliomyelitis was made a notifiable disease in 1912. When inactivated vaccine was introduced in 1956 the then Ministry of Health set up a scheme of surveillance to detect possible cases of vaccine-induced poliomyelitis. There was concern that vaccine strains might become virulent, however the results indicated that the vaccine in routine use was safe and effective (Geffen & Spicer, 1960). When live attenuated vaccine was introduced in 1962, the surveillance scheme was revised and the Epidemiological Research Laboratory (ERL) of the PHLS was made responsible for it. In 1970 when the WHO began their 10 year enquiry into acute

persisting spinal paralysis (WHO Consultative Group, 1982), ERL undertook responsibility for both schemes. In 1984 this responsibility was transferred to the Communicable Disease Surveillance Centre (CDSC) of the PHLS.

Reports on the surveillance of poliomyelitis in England and Wales have been published for the years 1962–4 (Miller & Galbraith, 1965), 1965–8 (Miller, Reid & Diamond, 1970), 1969–75 (Smith & Wherry, 1978), and 1976–7 (Collingham, Pollock & Roebuck, 1978). Individual case histories have also been published, and annual reports produced by ERL have been reproduced in the Communicable Disease Report of the PHLS (unpublished).

METHODS

The poliomyelitis surveillance scheme for England and Wales

Information was derived from a number of sources to ensure that no cases were missed. Usually the clinician in charge either notified the Medical Officer for Environmental Health (MOEH) (Medical Officer of Health before 1974) officially or told him informally of a suspected case. In turn, the MOEH informed CDSC. Not all the suspected cases were acutely ill, and occasionally the disease was recognized only when a child was investigated for wasting of a paralysed limb. Directors of Public Health and other laboratories sent weekly returns of all virus isolates to CDSC (or ERL before 1977). More than 300 reports of poliovirus isolates were obtained each year, practically all from recently vaccinated children with unassociated illnesses. All were scanned and any suspicious findings were followed up. Directors of Public Health and other laboratories also sent samples of any poliovirus isolates associated with clinical features suggesting paralytic poliomyelitis or other neurological illness to the Virus Reference Laboratory (VRL). Weekly notifications published by the Office of Populations, Censuses and Surveys, Registrar General, UK (OPCS) were checked as well as copies of death certificates. The principal investigator worked closely with the Department of Health and Social Security.

When a case (or suspected case) was identified, a form requesting the details of medical history and clinical condition of the patient was sent to the Director of the reporting laboratory who forwarded it to the clinician in charge of the case for completion. Two other forms requesting the epidemiological details and the vaccination histories of the patient and contacts were sent to the MOEH, and the Director of the laboratory was asked to complete a fourth form giving details of all laboratory investigations. A follow-up form was sent to the clinician 6–8 weeks after the initial illness to confirm the original diagnosis and to assess the degree of residual disability. Finally, results of marker and other laboratory tests by the VRL were added to the data.

Vaccine-associated cases were classified by WHO as 'recipient' when the illness began 7–30 days after vaccination or as 'contact' if the illness began after the patient had been in contact with someone who had been vaccinated 7–60 days before the onset of symptoms. This classification depended on the history and not on the strain of virus isolated.

Until 1974, an annual report was sent to the Joint Committee on Vaccination and Immunisation of DHSS. Since 1970, after deletion of the patient's name, the completed forms have been returned annually to WHO.

Laboratory investigations

The earliest available passage of virus was sent to the VRL, to confirm its identity, and to determine if the strain was vaccine-associated. Mixtures of more than one poliovirus serotype were not infrequently found in faecal samples from patients who had been vaccinated.

Strains were differentiated by marker tests. Until 1980 the Relative Ceiling Temperature (RCT) 40 marker test was used. This test was based on the observation that non-vaccine strains of poliovirus are capable of growing and replicating at temperatures up to and above 40 °C, whereas vaccine strains are inhibited at this temperature. Additionally, another marker test (dextran inhibition) was used for the differentiation of type 1 strains in which the presence of dextran sulphate in the medium inhibits the growth of vaccine strains but has no effect on wild strains. After 1980, these marker tests were replaced by the Van Wezel intratypic serum differentiation test (Van Wezel & Hazendonk, 1978). Some strains did not react clearly in the RCT 40 test. These ambiguous strains were classed as intermediate.

In some cases, where the virus isolation was unsuccessful or had not been attempted, the diagnosis of poliovirus infection was confirmed retrospectively by serology. Serum samples from the patient were examined for neutralizing antibodies to types 1, 2 and 3 poliovirus.

The present review

All the records have been kept since 1962, including those of patients who were later considered not to have had poliomyelitis. When the vaccine was first introduced wild poliovirus was endemic in the population, and a distinction could not be made between illness caused by the wild and the vaccine strains. The WHO 10-year enquiry (WHO Consultative Group, 1982) into acute persisting spinal paralysis imposed a degree of consistency in the data collection. Thus 1970 was selected as the first year of this review.

The following case definition was used.

1. The clinical features of the illness were considered to be those of paralytic poliomyelitis by the clinician in charge.

2. Either poliovirus was isolated or there was serological evidence of recent poliovirus infection.

3. The patient was normally resident in England and Wales regardless of where the infection was acquired.

RESULTS

Between 1970 and 1984, 88 cases were reported to the PHLS. Three were excluded at the time as they were not considered to be poliomyelitis. During our review we excluded 15 further cases which did not meet the above case definition. Six were non-residents who had been infected abroad and had either become ill while visiting this country or had come to England and Wales solely for treatment. One was excluded because the diagnosis was subsequently altered by the clinician and in the remaining eight cases there was insufficient laboratory evidence to support the diagnosis of a recent poliovirus infection. Seventy cases of paralytic poliomyelitis thus met the criteria.

Table 1. *Paralytic poliomyelitis England and Wales 1970–84. Number of cases reported to PHLS, in published reviews and in this review*

Year of paralysis	Cases reported to PHLS	Cases in previously published reviews	Cases in this review
1970	7	4	6
1971	4	5	3
1972	4	4	4
1973	6	6	3
1974	6	2	4
1975	3	3	2
1976	16	13	14
1977	14	13	13
1978	3	...	1
1979	6		4
1980	3	—	3
1981	6	...	4
1982	4	—	4
1983	2		2
1984	4	—	3
Totals	88	50	70

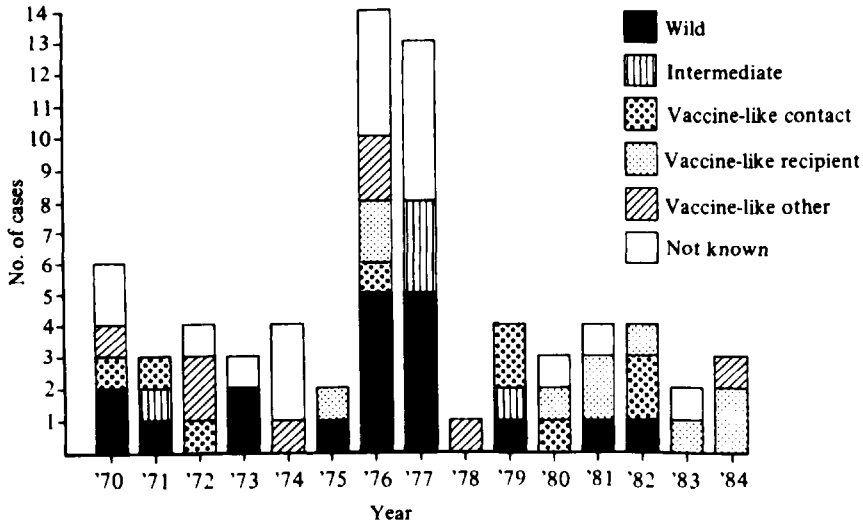

Fig. 1. Number of cases of paralytic poliomyelitis and virus strain isolated 1970–84.

Two patients, both unvaccinated, died from infection with naturally-occurring wild virus. One was a man aged 44 years who died in 1976 following a visit abroad. The second was a girl aged 7 who died in 1971 and whose source of infection was not traced.

Table 1 shows the number of cases reported each year to the PHLS, the number in the published reports and the number in this review according to the year in which they became paralysed. In the two published reports covering the years 1970–7 six residents of England and Wales who acquired the infection abroad were

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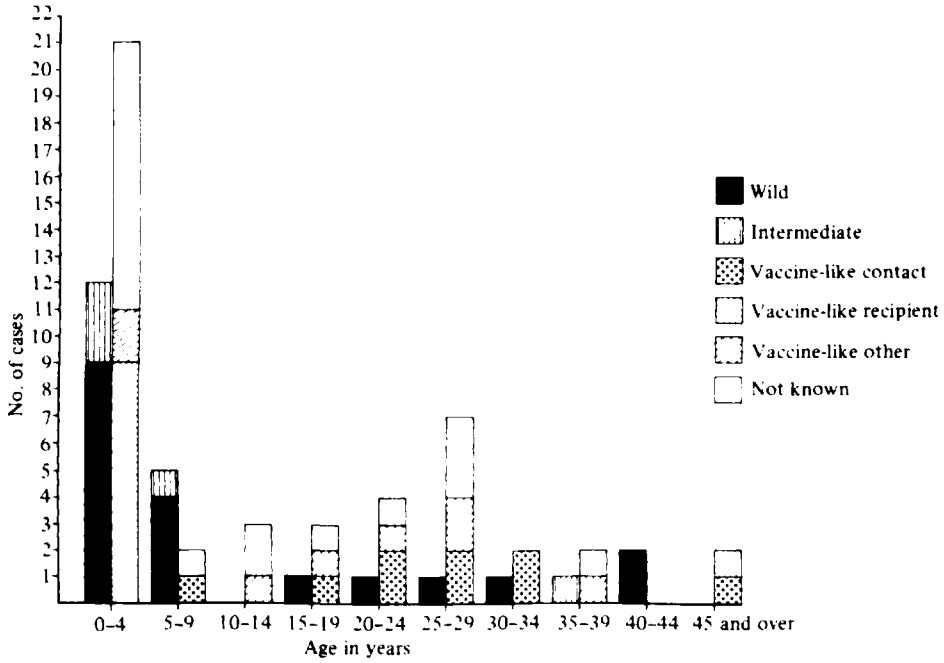

Fig. 2. Number of cases of paralytic poliomyelitis according to age and the virus strain isolated.

excluded by Smith & Wherry (1978) and one case was omitted by Collingham *et al.* (1978) because it was reported too late. Other discrepancies are the result of late notification or reporting of cases.

Fig. 1 shows the annual distribution according to whether the virus isolated was a naturally-occurring (wild), vaccine-like or intermediate strain. The annual number was fairly constant, except for the years 1976 7 when a small outbreak occurred with an increase in both naturally-occurring and vaccine-like strains (Collingham *et al.* 1978). This was concentrated during the last quarter of 1976 and the first quarter of 1977, followed by a period of 13 months during which no cases were reported. Outside this period no seasonal variation was found among either the wild or vaccine associated cases.

There was little geographical clustering apart from two small clusters during the 1976 7 outbreak. There was no direct contact between the patients in this outbreak.

Fig. 2 shows the age distribution of the cases. Children under the age of 5 years were the most vulnerable to naturally-occurring poliomyelitis. Of the 17 patients who had recently been vaccinated, 14 were under a year old, whereas none of those with a history of vaccine contact were under 5 years of age.

Table 2 shows that the source of infection was not always clear. Eleven patients had been abroad within the incubation period of their illness. In 5 a wild strain was isolated, in 3 it was intermediate or vaccine-like, and in 3 it was not known. Of the 17 patients who had recently been vaccinated, a vaccine-like strain was isolated in 10. Of the 12 who had a history of contact with a recent vaccinee, a vaccine-like strain was isolated in 9. Thus, among 30 of the 70 cases no source could be found,

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Table 2. *Source of infection according to virus strain isolated*

Source of infection	Wild	Inter-mediate	Vaccine-like	Not known	No isolate	Total
History of overseas travel	5	1	2	3	—	11
History of receiving vaccine	—	1	10	3	3	17
History of contact with recent vaccinee	—	—	9	2	1	12
Source unknown	14	3	6	2	5	30
Total	19	5	27	10	9	70

including 14 where a wild, 3 where an intermediate and 6 where a vaccine-like strain was isolated. Seven of the 30 patients were itinerants living in caravans but had not travelled abroad; a wild virus was isolated in 4, a vaccine-like strain in 1, and intermediate in 1 and in the last the character of the serotype was not known.

The dates of exposure were not known in the naturally-occurring infections so that the incubation periods could not be calculated. Paralysis usually occurs 2 or 3 days after the first vague symptoms. In 12 of the 17 recipient cases, it began 14–24 days after vaccination and in 9 of the 12 household contact cases 19–30 days after vaccination of the contact.

Vaccination status

Thirteen of the 70 cases had a history of partial or complete vaccination in the past. Eight were said to have received inactivated vaccine many years earlier but this was confirmed in only one patient who had had three doses 14 years before the illness. In 2 of these 8 cases the infection was caused by a wild strain, in 5 it was vaccine-like (3 were vaccine-associated cóntact cases) and in 1 the strain was not known.

Five patients had received oral polio vaccine and in all but one the dates of vaccination could be confirmed. Two were infants who had not completed the course and were infected by a wild strain of the virus. One was a child of 7 years who became paralysed about 10 days after receiving a fourth dose of vaccine but a virus was not isolated. The remaining two were a boy of 11 years who had an incomplete course 8 years previously and a boy aged 14 years who was said to have had a complete course, but this could not be confirmed. In both cases a vaccine-like strain of virus was isolated but the source of infection was not known.

Laboratory results

Serology was performed in 43 cases and 38 polioviruses were isolated from 34 of them. Table 3 shows the results of the poliovirus antibody tests and their relation to the strain of poliovirus isolated. Any patient may have antibodies to more than one type of poliovirus and more than one type of poliovirus may be isolated from any patient. The results shown refer to each type either on its own or in combination with other types. Where marker tests were able to differentiate wild from vaccine-like strains they correlated well with a history of likely exposure to wild

Table 3. *Relationship between poliovirus isolates and serology where there was a fourfold rise or antibody ⩾ 1 in 32 was detected*

Poliovirus serotype isolated	Polio antibodies: Same serotype	Polio antibodies: Same serotype plus one or both other poliovirus serotypes	Polio antibodies: Different serotype
Type 1			
Wild	7	3	—
Vaccine-like	2	1	—
Intermediate	3	—	—
Not known	1	—	—
Type 2			
Wild	—	—	—
Vaccine-like	4	1	—
Intermediate	—	—	—
Not known	3	—	—
Type 3			
Wild	1	—	—
Vaccine-like	2	3	2
Intermediate	—	3	—
Not known	1	1	—

Table 4. *Interval in days from onset of illness to first sample of blood where taken for antibody estimation*

Interval (days)...	Same day	1	2	3	4	5	6	7	8–13	14	Not known
Result of antibody testing											
Fourfold rise	—	1	1	—	2	3	1	2	2	1	2
One or more raised titre (⩾ 32)	1	—	—	—	1	1	—	2	5	17	1

virus or with vaccine association. Table 4 shows that a fourfold rise in antibody titre was demonstrated relatively infrequently, reflecting delays in taking the first blood sample.

DISCUSSION

The conquest of poliomyelitis by vaccination has been one of the greatest public health achievements of this century and during the 15 years of this review the annual number of cases of paralytic poliomyelitis has remained at about 3 or 4 a year, apart from the small outbreak in 1976–7 when the number reached double figures. Nevertheless it is still endemic in many parts of the world and once paralysis is established there is no cure. Poliomyelitis caused by wild virus is a reminder of the need for a comprehensive vaccination programme and careful monitoring of all cases.

The laboratory diagnosis of poliovirus infection is not easy, particularly in sporadic cases. One of the problems arises from the early appearance in the

patient's serum of antibodies to the invading virus. Ideally, laboratory confirmation of infection should be provided by isolation of the agent coupled with a rising titre of the specific antibody in the serum, but unless the first blood sample is taken at the earliest opportunity it may be too late to demonstrate a significant rise in antibody titre indicating recent infection.

The differentiation between wild and vaccine strains of poliovirus has not been easy in the past. The RCT 40+ marker test results were often unsatisfactory because the results with many of the strains were equivocal. This was particularly true of the 1976–77 type 1 isolates. These strains did not resemble vaccine virus; nor did they greatly resemble wild virus. Their most obvious characteristic was their lack of homogeneity. These strains were classified as 'intermediate', an unsatisfactory term which indicates, however, that their origin remains doubtful, especially since it is known that wild strains exist which have an RCT less than 40 °C (RCT 40−) and that the character of Sabin vaccine strains can change from RCT 40− to RCT 40+ after one passage through the human gut. These paradoxical results indicate that the RCT 40 test was not entirely satisfactory whereas the test developed by Van Wezel & Hasendonk (1978) gave readily reproducible, clear and unequivocal results.

In naturally-occurring illness, the correlation between isolation and serology usually presents few problems as the patients are apparently devoid of antibody at the time of infection and the response is to a single serotype of invading wild virus. Where virus isolation has failed, the development of a significant rise in antibody to a single serotype can be regarded as diagnostic. The presence of detectable antibodies to one serotype in the absence of antibodies to the other two in a single serum, although unsatisfactory as an indicator of recent infection, demonstrates an encounter with the agent at some time in the past. Serology is of no help in the diagnosis of an illness regarded clinically as poliomyelitis, except in those cases where the antibody is known to have been present before the onset making it unlikely that poliovirus is the cause since the serum antibodies would have rendered the patient immune.

The oral vaccine used in the UK contains all three poliovirus serotypes and the development of antibodies is associated with the colonization of the gut by all of them. Recipients of oral vaccine can be expected to excrete all three serotypes in the faeces for up to 3 weeks or longer. The presence of virus in the faeces of vaccinees in most cases is therefore of no clinical significance and merely indicates a temporary carrier state. Recovery of poliovirus from the faeces of patients suffering from a paralytic illness following vaccination consequently gives no confirmation that the serotype(s) isolated are related specifically to the illness, in contrast to naturally-occurring infection where only one serotype is involved. In addition, administration of even one dose of vaccine often initiates a multiple antibody response, so that serological evidence may be of no value in indicating the serotype of poliovirus responsible for the illness. As a result of these difficulties, a diagnosis of vaccine-associated poliomyelitis in a recipient of vaccine is usually reached by a process of elimination. The WHO criteria have been adopted as a practical guide to decide whether a case can be regarded as vaccine-associated. When exhaustive laboratory investigations have failed to implicate other potential

causes of paralytic illness the likelihood that the case is due to vaccine becomes much stronger.

In 61 of the 70 cases one or more poliovirus serotypes were isolated from the faeces. In 10 of these the marker test was not done but 3 had received vaccine within 30 days of the illness and 2 had been in close contact with someone who had been vaccinated within 60 days. In 19 cases a wild virus was isolated. Contrary to our expectations, only 5 had a history of recent travel abroad so that the remaining 14 must have acquired their infection in this country. This number includes 4 from itinerant families living in caravans. In poliomyelitis epidemics only a small proportion of those infected become paralysed, the majority are symptomless or suffer only a vague flu-like illness. Although wild strains have largely been replaced by vaccine-like strains, they can be found occasionally in the environment. It is therefore possible that cases of symptomless wild polio may still occur. This could account for the sporadic, apparently unconnected cases which arise from time to time. Vaccine-like strains were isolated from 10 patients who had been vaccinated and from 9 who had been in contact with others recently immunized. In 8 cases a source could not be identified.

Thus, although careful enquiries had been made at the time of the illness, no source of infection could be identified in 30 of the 70 cases. More than 95% of the adult population have detectable circulating antibodies to one or more types of poliovirus (Roebuck & Chamberlain, 1982) but immunization has rarely exceeded an annual rate of 85%. The difference must be due to infection through casual contact or from other environmental sources.

Eight of the 13 patients who had a past history of complete or incomplete vaccination were said to have had inactivated vaccine. The number of persons who had acquired their immunity only through this route is not known, so it is not possible to say from our data whether the immunity gained from the inactivated vaccine given before 1962 might have waned.

No reasons could be found why, on average, 1 recipient and 1 contact case of paralytic poliomyelitis occurred following some 2 million doses of oral vaccine given every year. Undoubtedly the number of contact cases could be reduced if all non-immunized adults were vaccinated at the same time as the children in their care. Similarly the number of cases of wild poliomyelitis could be reduced if all those visiting endemic areas were adequately protected before travelling. The fact that wild cases contracted in this country still occur demonstrates the need to maintain the present level of immunity.

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