POLIOMYELITIS IN ENGLAND AND WALES
A CHADWICK PUBLIC LECTURE GIVEN ON FEBRUARY 20, 1951
BY
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MR. CHAIRMAN, LADIES, AND GENTLEMEN,

I would preface my talk to you this afternoon by expressing my keen appreciation of the privilege I feel it to be to lecture under the auspices of the Chadwick Trust. My lecture, is, in such circumstances, given under the aura of Sir Edwin Chadwick’s great and undying achievements in the field of public health and my only wish is that I were worthier of the honour it is to lecture in association with his name.

Poliomyelitis is an increasingly important disease owing to its growing prevalence throughout the world, to its serious consequences for so many of those stricken by it, and to the cloud of uncertainty which envelops so much of its epidemiology.

Some consider that the origin of the disease dates back to ancient times and that, though perhaps unrecognized in the earlier days, it has been with us in various guises for many centuries. Others have regarded it as a comparatively recent development among the ills that affect mankind.

The mounting significance of poliomyelitis in the last half-century and especially in the past decade has attracted considerable attention and much research into it has been carried out in this and other countries.

Unfortunately, though, adequate research is hampered because no simpler method of propagating the virus exists than inoculation into monkeys; at the same time means of isolating the virus in the laboratory have not yet been developed.

My task this afternoon lies not in the laboratory, clinical and other more scientific aspects of poliomyelitis, of which there are many others far more qualified to speak than I am; but in tracing briefly for you the history of the disease in England and Wales.

It is, however, justifiable and likely to be of interest if I give you shortly a few of the highlights in the history of this disease prior to its first tentative recognition in this country.

The possibility has emerged, from archaeological and other researches, of the occurrence of poliomyelitis in ancient times, but it must remain a possibility, as evidence allowing of fully reliable diagnosis is lacking.

There is the instance of the skeleton of 3,700 B.C., found in a village S.E.
of Cairo, by Flinders Petrie in which the shortening of one leg and other
findings afford some support to a diagnosis of poliomyelitis [1].

There are, too, the delineations on an Egyptian stele of the eighteenth
dynasty (1580–1350 B.C.). These were investigated by Ove Hamburger [2], a
student of Egyptian art and at the same time a physician. They include three
figures, a priest—Ruma, of the temple of Astarte in Memphis—a woman and a
child. The foot of the man is in a position of equino-varus and the slight flexion
of the hip and knee is not enough to raise the heel so high. There is shorten­
ing of the femur, tibia and fibula. The staff normally carried in front and
parallel with the body is borne crosswise from the shoulder through the bend
of the elbow and is alongside the withered leg as a support. The condition
seen may be due to other diseases such as coxitis, certain nervous diseases,
hip-joint trouble and the like, but the distinct probability of poliomyelitis
being the cause must be admitted.

Hippocrates describes a winter epidemic of paralysis on the island of Thasos
which also is not without interest.

Archæological investigations in 1921 in a mediaeval Norse colony—
Herjolfnes—by a Danish expedition, resulted in the discovery in a cemetery of
25 skeletons of the beginning of the fifteenth century. Some 6 of these gave
evidence of disease productive of physical deformities and there is consider­
able ground for the belief that poliomyelitis was responsible [3].

During the eighteenth century were many allusions to conditions sug­
gestive of poliomyelitis. Here again though they might be due to other diseases.

Some importance attaches to Boerhaave's reference in 1761 to reports which
had reached him of paralysis, of a peculiar description, in certain parts of Asia,
believed to be due to cold [4].

In 1823 Shaw commented on the occurrence of sudden attacks of paralysis
in Army children in India and at the same time called attention to the case
of a young man, in whom an attack of paralysis in India as a child had caused
physical deformity [5]. Goodeve later (1879) made reference to frequent paralytic
illnesses among English children in India, and indeed there was a suggestion
that the disease might have been introduced into England in the latter part of
the eighteenth century by returning Anglo-Indians, and have spread thence
to the Continent.

It will be recollected that Sir Walter Scott was in 1773 attacked by the
disease and suffered, as the result permanent lameness of the right leg.

It is interesting that there is some evidence of a prevalence of the disease in
America in 1795 and MacNalty tells us of a letter written by Madamé le Marquis
de la Tour du Pin who was, with her husband, a fugitive in Albany from the
French revolution. She says: "My little Seraphine was taken from us by a
sudden illness very common in this part of the country, a kind of infant para­
laxis. She died in a few hours without losing consciousness. The physician
from Albany gave us no hope that she would live and declared that this malady
was then very common in the country and that no remedy was known. The
young Schnuyler who, only the day before, had been playing with my daughter
A. E. Richmond

during the afternoon succumbed to the same trouble a few hours later and rejoined her in heaven."

In this and other countries, in the later seventeen hundreds, other physicians such as Underwood, Shaw, Abercrombie and Badham in England, Jorg in Germany, Monteggia in Italy, have described a number of cases which were not unlike poliomyelitis.

It is Michael Underwood to whom is generally awarded the credit of first describing the disease in treatises he wrote between 1784 and 1799 [6]. He paints a comparatively accurate picture of the disease and shows the confused state of affairs at this period, in regard to the various paralytic conditions between which differentiation today is comparatively easy.

In 1823 Shaw, in his "Nature and Treatment of the Distortions to which Bones of the Spine and Chest are Subject," contributed a chapter on the causes of partial paralysis and wasting of one of the limbs in infancy, which frequently produce spinal distortion. Herein groups of cases are mentioned, which appear to have been of a poliomyelitic type.

We now arrive at an outstanding event in the history of poliomyelitis in this country and that is a report by Badham [7] on 4 cases which occurred in 1835 at Worksop in Notts. The main features emphasized by Badham were firstly, the youth of the patients, all 2 to 2½ years, secondly, the occurrence of apparent cerebral symptoms in the very early stages, thirdly the lack of any degree of impairment of health in any of the cases, and fourthly, some indications of congested, oppressed or irritated brain.

Just about this time a few cases were being reported on the Continent and, in 1840, Jacob Heine of Cannstatt, one of a family of skilled orthopedists and mechanicians, published the first monograph on poliomyelitis to see the light of day [8]. The details of Badham's cases had focused Heine's attention on certain paralytic cases in his own practice. This led to the study of the disease which he subsequently carried out. Heine comes down to posterity as a pioneer in modern knowledge of this disease.

He recorded its features and described it as "an affection of the central nervous system, namely the cord, of an irritative and congestive sort." Though, at the time, some were opposed to Heine's concepts, these were nevertheless later confirmed.

There followed, from various sources, clinical contributions of varying degrees of importance as a result of which, among other things, the cerebral type of the disease came into prominence.

In 1887 an outbreak of 44 cases was observed by Medin in Stockholm, where previously, as in Sweden as a whole, the disease had been infrequent [9]. Medin's observations contributed much additional information about this disease. It became known as the "Heine-Medin disease" in view of the great extension in our knowledge due to these two men.

Caverley in America in 1894, brought to notice the non-paralytic type and other points, while Wickman, a German extended and clarified Medin's con-
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cepts and recognized many cases of the abortive (including the non-paralytic) types.

As far as the pathological aspect is concerned, Cornil in France in 1863 [10] carried out the first positive microscopical examination of a case of poliomyelitis and others did valuable work of this kind including Von Reinecke and Von Recklinghausen who described lesions in the anterior horns and lateral columns of the spinal cord. From then onwards various others made effective contributions to our knowledge of this aspect of the subject, of whom perhaps Wickman should be specially mentioned for his systematic study of poliomyelitis in Sweden in the earlier years of the twentieth century.

As regards virology, from the time of Landsteiner and Popper who in 1908 and 1909 first transmitted poliomyelitis to monkeys much has been accomplished by workers in a number of countries. On the other hand, essential progress has been much handicapped for reasons I have already mentioned. As regards advances made, among others the establishment of three main immunological groups of the virus with various strains of these is of special moment.

Nothing in the history of poliomyelitis is so baffling as its change in the earlier years of the twentieth century from a sporadic to often an epidemic disease. Various theories have been put forward to account for this but proof of either is wanting.

The older literature contains no references to epidemics, and after Heine's monograph there was no mention of such until 1880, although there were reports of groups of cases here and there, occurring within a brief period in a small area.

Some cite a small outbreak in 1868 in the Modum district of Norway described by Bull as the first epidemic [11]. It was thought at first to be cerebrospinal meningitis and there were 14 cases with 5 deaths. Others quote a Swedish outbreak at Umea in 1881 described by Bergenholtz.

In the years following, outbreaks of varying sizes were increasingly reported, but it was not until the startling outbreaks of 1903 to 1906 in Norway and Sweden that there was a realization of the dramatic proportions which the disease might on occasions assume.

From then onwards serious epidemics occurred, apart from Norway and Sweden, in England, America, Holland, Germany, Austria, Canada, Australia and elsewhere.

Turning our attention more particularly to developments in England and Wales, the first important mention of poliomyelitis in the annual reports of the medical officer to the Local Government Board is to be found in that for 1910–11. Here reference is made to the occurrence of sporadic cases; and also to the appearance of small groups of cases here and there such as those in Herts (1897), London (1904), Upminster (1908), reported on by Pasteur, Batten and Treves respectively.

Evidence had accumulated as to the infectious character of the disease and as to adults as well as children being attacked. It was appreciated too that
paralysis did not always occur and that mild cases were often difficult to recognize.

By the end of 1911, poliomyelitis had occurred in some twenty-one counties with a definite outbreak at Bristol in 1909 of 37 cases investigated by Parker, and in the same year a number of cases in London described by Parsons. In 1910 there were outbreaks in Cumberland (Carlisle) 34 cases and 7 deaths, Northumberland, Nottinghamshire, Leicestershire (80 cases), Dorset, Gloucester and York. In 1911 there were epidemics in Devon and Cornwall, Huntingdon, Hampshire, Bedfordshire, Suffolk, Yorkshire, Westmorland, Derbyshire and Wales.

It was therefore clear that there was a scattered prevalence of poliomyelitis throughout much of the country, and equally clear that it should be made a notifiable disease. This was first effected in London for a limited period. Other sanitary authorities followed this lead.

Finally, in March 1912 the disease became legally notifiable and it became possible to assess its incidence throughout the country with a reasonable degree of accuracy. It must, however, be borne in mind that, in comparing statistics of the earlier periods with those of later years, the existence of non-paralytic poliomyelitis was not appreciated as fully in the earlier years as later, and that as time went on notifications of this type of the disease increased with its fuller recognition.

In the annual report for 1911-12, special attention was paid to Farrar's investigations in Leicestershire and Dorset in 1910 and to those of Reece in 1911 in Devon and Cornwall. The latter traced 154 cases in the two counties and of these 34 were fatal. Pathological investigations by Dr. Mervyn Gordon showed typical lesions and Levaditi in Paris infected monkeys with material from two cases. Apart from the very interesting reports by Farrar and Reece, Gordon contributed a critical review of the work done on the virus, and Dr. MacEwan a review of the clinical characters and epidemiology of the disease. MacEwan too carried out an interesting investigation in 1913 into the prevalence of poliomyelitis in parts of Lancashire and Westmorland the results of which pointed to the spread of infection by personal contact. The large bulk of the cases were in children and there were no multiple cases in families.

At this time, Sir Arthur Newsholme—medical officer to the Local Government Board—voiced a warning as to the possibility of cases of poliomyelitis being at times misdiagnosed as cerebrospinal meningitis and in March 1912 the first of a series of memoranda on poliomyelitis saw the light of day together with a similar publication on cerebrospinal fever.

Up to 1915 and for some years after, it was true to say that England and Wales did not suffer from large epidemics of poliomyelitis such as those which had affected Sweden, Norway, and the U.S.A. and, in fact, isolated, or small groups, of cases only occurred.

There was nevertheless no lack of appreciation of the increasing importance of the disease and of the dire possibilities should its epidemicity increase to
large proportions. The part probably played by healthy carriers and abortive cases was also realized.

There was much discussion as to why poliomyelitis was becoming seriously prevalent in certain countries with previously low incidences and why its epidemic character was being so much enhanced. Some thought it to be due to an increased virulence of the causal organism; others that it was owing to a more general distribution of the virus in many countries. Flexner had incidentally demonstrated the feasibility of increasing the virulence of the organism by passage through monkeys.

In the 1915–16 annual report of the medical officer to the Local Government Board, Bruce Low surveys the international scene. He quotes Flexner's description of the disease—"one of the saddest of diseases"—as also Buzzard's emphasis on "its peculiar cruelty to its victims." He stresses how the influx of children into the orthopedic department of a hospital, with paralyses, deformities, curvatures and so on which had developed in the course of a brief illness, may often have been the first indication of an outbreak of poliomyelitis in the locality.

Bruce Low also emphasized the extent to which knowledge of the disease was increasing, and mentioned an American suggestion that all articles on the subject written prior to 1907 should be destroyed and forgotten.

He reminds us of the first production of poliomyelitis in monkeys by Landsteiner and Popper in Europe 1909; of its similar production independently a little later by Flexner, Laws, and Strauss in New York; also of its transference from monkey to monkey by Leisner and Wismer in Vienna in the same year.

In 1919 the separate notification of polio-encephalitis came into force largely owing to previous confusion as to whether it should or should not be included in poliomyelitis for notification purposes, and to help distinction from encephalitis lethargica.

This system remained operative until 1950 when a further change was made and polio-encephalitis is now included in poliomyelitis, distinction being made between the paralytic and non-paralytic types. In this lecture the term poliomyelitis includes polio-encephalitis unless otherwise stated.

Notification as already mentioned was not in full operation until 1913. In this year, there were the largest number of annual notifications in the decade 1911–20, viz. 744. As will be seen from Chart I the lowest were 228 in 1918. There is little else to be said of this period except to mention, specially, small localized outbreaks in Esher, the Dittons, Epsom (MacNalty), Cambridge, all in 1917, and in Matlock (Sturdee) in 1920. Perhaps too should be recorded the results of a small investigation, in line with those of more recent work, and pointing to a greater prevalence of the disease in those living in the more sanitary environments.

In 1921 with 539 notifications including 51 of polio-encephalitis there was a relatively high rate in parts of Cornwall, and in the country district around Wantage (Parsons). In the following year notifications fell to 386, but rose in 1923 and 1924 to 644 and 860 respectively. In 1923 there was an interesting
outbreak at Hastings. A fall to 422 notifications in 1925 heralded the record year of 1926 with 1,297 cases (including 138 of polio-encephalitis). This was equivalent to about 3 per 100,000 of the population compared with 1 and 2 per 100,000 in previous years. The trouble started in mid-July, large towns were not specially affected and it was chiefly small urban communities in rural districts which were struck.

The distribution of the disease was fairly general but Leicester, Broadstairs and Essex—81, 74 and 58 cases respectively—provided outbreaks of special note. In the decade following 1926, notifications varied between 1 and 2 per 100,000 annually. In this period various small outbreaks were studied, among others Grimsby and Spilsby in Lincolnshire in 1930, Glossop Borough, Oxfordshire and contiguous parts of Berks in 1932, Berks and Somerset in 1934, Gloucester in 1935, and Southall in 1936.

It still remained that little advance was being made in the study of the natural history of the disease, owing to the lack of an easily applied test, to
uncertainty as regards recognition in the earlier stages, and to the problem of abortive cases.

In 1931 the chief medical officer to the Ministry of Health drew attention to recent suggestions by Kling as to the possibility of infection by food and water, these being based on work by Levaditi, Schmitz and Willemin in the Bas Rhin department, and by others in Sweden, Saxony, and Roumania, and by the experimental infection of monkeys by injection and the recovery of the virus from the faeces.

The outstanding feature of the 1937–46 decade was the occurrence in 1938 of the largest annual number of cases yet recorded—1,661 with 178 deaths, an incidence per 100,000 of about 4. Prevalence was below the average up to the end of July when there was a sharp rise in Essex. From then onwards there was a rapid increase throughout the whole country—with 83 per cent of the cases occurring in the last two quarters of the year.

The incidence was highest in the Eastern and South Eastern Regions—6·3 and 5·0 per 100,000. The North suffered least—1·7 per 100,000. Oxfordshire 21·2 per 100,000, the Isle of Wight 17·5, and Lincolnshire 11·8 were the counties most severely attacked. There was no fall to the mean of the preceding seven years until late in February.

In 1947 came the holocaust of 7,766 cases with a total incidence of 18 per 100,000—4¼ times that of 1938 and the disease more widespread than it had ever been before.

The epidemic began in late May, six weeks earlier than the normal experience to date, the spread appearing to be radial from certain main early groupings—London—Birmingham—West Riding—East Lancs—and Tyneside. The peak was reached in the week ending September 6 with 708 notifications. Thenceforward there was a gradual decrease, but winter notifications remained higher than normal, including those in the first four to five months of 1948. There was on the whole a tendency towards higher rates, 40 and over per 100,000, in sparsely populated areas. The highest incidence in a county of over 200,000 was in Bedfordshire (32·1) and the lowest in Devonshire (9·7).

There was no close correspondence between the areas principally affected in 1938 and 1947, but Lincs, Oxford, Sussex E., Isle of Wight and the East Riding of Yorks, coupled with Carmarthen and Radnor had relatively high rates in both years.

Comparing the years of high prevalence of poliomyelitis, 1947 showed a tendency to a wide distribution side by side with a decline in the local intensity of epidemics.

Hopes were aroused in 1948 of a prolonged respite from the more severe ravages of the disease but in 1949—5,967 notifications were equivalent to a rate of 14 per 100,000. With the general rise in incidence in June there were several sharp outbreaks in widely scattered and relatively small communities—for instance, Newhaven, week ending June 18; Cambourne—Redruth, week ending June 25 and following week; Okehampton, week ending July 9; New Forest, week ending July 16. Weekly notifications never reached those recorded in the 36th week of
1947 (708) but the epidemic lasted much longer. The seasonal distribution was no exception to the rule.

In late May of 1950 with but little breathing space since the experiences of 1949, uncorrected weekly notifications reached 50 and doubled themselves the following week. A month later they were 212. The peak was attained in the week ending August 26 (589) and from then onwards there was a gradual fall to 53 in the last week of the year. The total corrected notifications for 1950 were 7,753 only a few short of those of the record year 1947.

The distribution of the disease was very general but it is noteworthy that in three counties, Lancashire, Warwickshire, and the West Riding of Yorkshire, notifications in the first quarter of the year were comparatively high and that these counties alone produced 22 per cent of the total notifications for the year.

Outbreaks of special importance which occurred were those in Birmingham, Bristol, the Isle of Wight and East Kesteven (Lincs).

An examination of my second chart is of interest as indicating the regional distribution of poliomyelitis in England and Wales in the past four years. From this it will be seen that the highest incidence attained in this period was in 1950.
in the Midland region comprising Herefordshire, Shropshire, Staffordshire, Warwickshire and Worcestershire, and that it had in this region been preceded by relatively much lower rates in the other three years. The same is to a material extent true of the South-Western Region—Cornwall—Devonshire—Gloucester—Somersetshire—Wiltshire—which in 1950 showed the next highest incidence in the four-year period. Excluding the non-epidemic year 1948, the Midland, Northern and North-Western regions and Wales—in 1949—gave the lowest figures for the period referred to.

I have nothing to add to this brief story of the advance of poliomyelitis in England and Wales as a whole save to mention that as regards seasonal incidence there has been little change over the years, and in the past four years approximately 1·4 per cent of cases have occurred in the first quarter, 2·5 per cent in the second, 68·7 per cent in the third and 26·5 per cent in the fourth, with the largest proportion of cases in August 32·2 per cent and September 27·2 per cent.

To regard the picture from a more particular, as distinct from general, aspect, we have recently commenced a study of the incidence of poliomyelitis in all the various boroughs and urban and rural districts—1,472 in all—in the twenty-eight-year period 1922–49 and the summated incidences for this period in the various areas may be of interest.

Before going any further though, I would remind you that we are dealing with notified cases only and that there may possibly have been cases of abortive and unrecognised types which have gone unrecorded.

I have to confess also that the summated incidences are estimated on the mean of the populations of the boroughs and districts concerned in 1922 and 1949 and not on the true mean. It can, however, be assumed that the figures given you represent reasonably accurately the incidences of the disease in the twenty-eight-year period referred to.

Considering first the various county areas in England and Wales, the situation is pictorially represented in the map which has been prepared for you. Anglesey takes pride of place with only 16 per 100,000 and indeed nine of the Welsh counties fall within the two groups of lowest rates. Radnor, however, had a very big incidence of 152.

Of the English counties, Westmorland headed the list with the extremely high rate of 196. In the first year of the series there was evidence of a core of infection in this county and with a steady fall of cases throughout the period and much increased numbers in 1940, 1947 and 1949 coupled with a small population, it achieved its unenviable position in the list. Rutland, a large sparsely populated rural area, came next with 155 cases per 100,000, but in actual numbers 29 only, confined to ten out of the twenty-eight years. Lincoln (Lindsey) 151 per 100,000, Oxford 144, Berkshire 138, and the Isle of Wight 128 followed in that order.

The Isle of Ely and the Soke of Peterborough emerged from the contest with almost invisible stains on their escutcheons and these large thinly populated rural areas even in 1947 and 1949 never produced more than 4 notifications in a year, and in seventeen and eighteen years respectively out of the twenty-eight
had no cases at all. Durham followed 55, and then Worcester, Stafford, Lancashire 58, 60, 61 per 100,000 respectively.

As regards county boroughs Oxford produced the highest rate 197, Grimsby following with 178, and then Canterbury 151, Leicester 138 and Hull 133. Blackburn, West Bromwich and Oldham, 33 in each case, shared first prize for the lowest incidence, closely followed by Wallasey 37, Doncaster 39, and Dudley 40 per 100,000. On a basis of notifications per square mile Leicester had the largest rate 25·3 and Barrow-in-Furness the lowest 1·1. The London County rate was 83 per 100,000, Holborn having the largest incidence 263 and Finsbury the lowest 43 per 100,000. Notifications per square mile were highest in Holborn 141 and lowest in Greenwich 10.
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In the Municipal boroughs and urban districts Broadstairs must be awarded the wooden spoon with 642 per 100,000, largely on account of the historical school outbreaks there in the autumn of 1926 Ripponden (Yorks) and Fowey (Cornwall) followed with 498 and 438 respectively, instances of the effect of a comparatively small number of cases in small populations on rates per 100,000.

Hexham (Northumberland), Knaresborough (Yorks) and Shildon (Durham) came next. As regards incidence per square mile Willesden showed the highest rate 26.7 of the municipal boroughs and urban districts.

In the rural districts Saffron Walden (Essex) headed the black list with 370 per 100,000 largely on account of 39 notifications in 1947–49. Billesdon (Leicester) followed—311—a small population with a large number of cases in 1926. Then came Cuckfield (Sussex) 286 per 100,000 closely followed by Uppingham (Rutland), North Westmorland and Ringwood and Fordingbridge (Hants) in that order.

I would now ask you to turn your attention for the moment to certain municipal boroughs and urban and rural districts which distinguish themselves by remaining free of notifications of poliomyelitis for the whole of the twenty-eight-year period we are considering.

They were as follows:

**Municipal Boroughs and Urban Districts**
- Bollington (Cheshire)
- Padstow (Cornwall)
- Torpoint (Cornwall)
- Axminster (Devon)
- Lynton (Devon)
- Brightlingsea (Essex)
- Godmanchester (Hants)
- Tenterden (Kent)
- New Hunstanton (Norfolk)
- Higham Ferrers (Northants)
- Brackley (Northants)
- Ellesmere (Salop)
- Wem (Salop)
- Halesworth (Suffolk)
- Saxmundham (Suffolk)
- Appleby (Westmorland)
- Scalby (Yorks N.R.)
- Tickhill (Yorks W.R.)
- Washington (Durham)
- Barrowford (Lancs)
- Rainford (Lancs)

**Rural Districts**
- Ely (Isle of Ely)
- Thornley (Isle of Ely)
- Wath (Yorks N.R.)

Of the twenty-one boroughs and urban districts named, nineteen are of small size with, in practically every case, populations of under 5,000 and densities under 1,000 per square mile. They are, in general, bordered very largely by rural areas with, in some instances, the sea in addition. Any abutment on other urban districts, where it does occur is slight, and, in practically all cases, the bordering districts concerned showed low incidences of the disease in the twenty-eight-year period we are considering. There are, however, one or two exceptions to this, notably the very high figures in North and South Westmorland rural districts which bound the Appleby urban district in large degree.
Washington, Barrowford, Rainford and Higham Ferrers come into a different category and are characterized by proximity to urban rather than rural districts. Washington in Co. Durham—the largest—is bordered by a number of districts in some of which there were materially high rates of the disease. The remaining three all have populations of 5,000 or less, and as regards densities, Barrowford and Rainford with low figures of 234 and 475 per square mile and Higham Ferrers 1,000. There were a number of instances of districts with significant incidences adjacent to them. Rainford was particularly fortunate in retaining its virginity with Upholland urban district over the boundary with a rate of 324 per 100,000.

As regards the three rural districts, Ely, Thorney and Wath, the first named has a population of some 33,000 spread over about one hundred square miles, roughly 360 persons per square mile. The incidence of poliomyelitis in the surrounding areas was low. Thorney some 34 square miles, with a density of 7, and Wath, with a density of much the same size—both had rather larger poliomyelitis rates in proximity to them but managed to retain their freedom from the disease.

Finally and before passing on from our twenty-eight-year survey, there are one or two other points worthy of attention which emerge. There has in the past been some discussion as to whether there is a greater prevalence of poliomyelitis in the more thickly populated areas or the reverse. Time has not yet allowed of a complete examination of our records over the whole country from this point of view. We took, however, a 10 per cent random sample with the following results:

<table>
<thead>
<tr>
<th></th>
<th>Average density per square mile</th>
<th>Incidence of cases per 100,000</th>
<th>Cases per square mile</th>
</tr>
</thead>
<tbody>
<tr>
<td>County boroughs</td>
<td>12,518</td>
<td>70.7</td>
<td>8.8</td>
</tr>
<tr>
<td>Municipal boroughs</td>
<td>2,655</td>
<td>91.0</td>
<td>2.5</td>
</tr>
<tr>
<td>Rural districts</td>
<td>166</td>
<td>101.0</td>
<td>0.02</td>
</tr>
</tbody>
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I think you will agree that these figures are statistically significant and indicate a much lower prevalence of the disease in the thickly populated county boroughs compared with the rural areas, and indeed a decidedly lower rate in such boroughs in comparison with the municipal boroughs and urban districts.

The gap between the latter and the rural districts is not so marked. There is, too, something of interest in the age incidence of poliomyelitis which is graphically indicated in Chart 4. You will note the material shift from the younger to the older age-groups, and the evidence in support of doing away with the old title of infantile paralysis. In the great Swedish epidemics of the first decade of the century and in the early American and Australian epidemics the attack rate was very high in the age-group 0–5 and very low in adolescents. In recent epidemics the rate has decreased in infants and increased in those of school age and over. It seems, however, that in communities with less advanced sanitation, the earlier type of age distribution is still found, e.g. Malta (Seddon), Japan (Paul) and Mauritius (McFarlane). In other civilized countries
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AGE INCIDENCE
PERCENTAGE OF CASES IN THREE AGE GROUPS

The change in age incidence has reached much the same stage as in England and Wales.

Turning to case fatality rates, you will see from the chart that, in respect of poliomyelitis, these rates have tended to be lower in years of high incidence of the disease, and that the lowest rates in the period 1938 to 1949 were in the epidemic years 1947 and 1949. It must, however, be accepted that fatality rates are influenced by the extent to which non-paralytic cases are notified, and that in epidemic years there is probably a tendency towards more thorough search for these and to their better notification. As would be expected the fatality rates of polio-encephalitis are very high; such cases, though, form a very small proportion indeed of the total poliomyelitis notifications.

Lastly the occurrence of singleton cases in districts is worthy of comment. Here, just as so often happens in families and communities, there were in the twenty-eight years a material number of districts producing in any one year a
There is much of value in the results of an enquiry into hospital cases in the year 1949 which were published in the monthly Bulletin of the Ministry of Health and Public Health Laboratory Service of October and November 1950 by Bradley and Gale. This investigation covered the large bulk of all such cases in the year referred to.

Time does not permit of any detailed study of the information given in this valuable report, and I will merely summarise it as follows.

The fatality rate was highest, 26·9 per cent in those over 45 years of age and lowest 4·2 per cent in those of 1-4 years. 79·4 per cent of all cases were paralytic, and of those paralysed who did not die, 40·5 per cent were slightly, 38·5 per cent moderately and 21 per cent severely paralysed.

Broadly speaking, of those admitted with suspected poliomyelitis 5 or 6 per cent were likely to die, 9 or 10 per cent to be severely paralysed, 17 or 18 per...
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30 cent to be paralysed but with proper treatment able to work, and 65-70 per cent practically all right.

Respirator treatment was necessitated in 10 per cent of cases—in actual numbers 559. Of these 213 required such treatment temporarily only, 29 permanently, and 317 died.

With regard to the therapeutic aspects of this disease, progress in treatment over the years has been disappointing and no specific treatment of any proved value has developed. The new antibiotics appear in no case to have any special activity against the virus.

Much reliance was at one time placed on convalescent serum, and, largely as the result of encouraging reports from Australia, Canada and elsewhere, it was in extensive use for some years. By 1935 doubts of its value had much increased and Walshe in the British Medical Journal of 19.10.35 stated that after many years of trial, the weight of opinion was that it was of no proven value.

Just about this time the treatment recommended by Sister Kenny, an Australian, attracted much attention. This lady was by the courtesy of the L.C.C. allowed to demonstrate her treatment on a series of cases at the Queen Mary's Hospital for Children at Carshalton and the trial started on 13.7.37. The results were watched by an honorary advisory committee of orthopaedic surgeons and neurologists.

Stating it very briefly, they reported that they could not admit Miss Kenny's claim that a complete cure could be promised in any group of cases, and felt that very early attempts to initiate voluntary movements and also early and frequent passive movements while harmless were not of proven value. They agreed with Miss Kenny on the value of hydrotherapy in the form of hot and cold douches, also that under certain conditions splints could be dispensed with. Their ideas, however, did not conform to hers as regards the entire abolition of splints and surgical appliances in treatment. Miss Kenny's treatment makes a heavy demand on staff and time, but a modified technique is now in use in many orthopaedic departments in this country.

In regard to treatment, mention must not be omitted of the development of the respirator for dealing with those suffering from respiratory paralysis.

Drinker of Harvard was the first in the field and his iron lung was demonstrated to University College Hospital Physiological Society in May 1931. There is a reference to its use at the Wingfield Orthopaedic Hospital, Oxford, in the Lancet of 12.11.32, and in 1934 it was used in the Great Ormond Street Hospital. It gradually came into more general employment.

Later, other types were developed such as the Bragg-Paul pulsator which operates by positive as distinct from negative pressure.

A modification of the Drinker by an Australian—Both—is also in extended use. From the historical point of view the generous action of Lord Nuffield in 1938 in providing iron lungs for hospitals genuinely in need of them must not go unrecorded.
In general I suppose the experts will agree that treatment has not changed much over many years and that strict rest and symptomatic measures in the early stages followed by steps to prevent deformity and to encourage the action of muscles or parts, which will include active and passive exercises, massage, hydrotherapy, and finally orthopedic surgery, are the main features of present-day therapy. Electrical methods of treatment have fallen into disrepute and disuse.

I would not like to conclude this review of history of poliomyelitis in this country without making some mention of those unhappy victims of permanent paralysis as the result of this disease. In the past four years alone with a total of some 25,000 cases of the disease, at least 2,500 must have been left with severe residual paralysis and unfitted to return to normal life to say nothing of
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those less severely affected. Much is done to help these cases and to rehabilitate them as far as possible and there is no lack of activity to this end. Time does not allow of any detailed reference to this, but I would like especially to draw your attention to the Infantile Paralysis Fellowship with its headquarters at 37, Tavistock Place, London, W.C.1.

This was founded some eight or nine years ago owing to the efforts of 2 sufferers from poliomyelitic paralysis, Mr. F. Morena and Miss Patricia Carey. Members and associates of this fellowship have in the course of time grown to some 7,000 in number and there are I believe 26 branches in England, a branch in Northern Ireland and in Wales, and a group in Scotland. It is an association of sufferers from poliomyelitis which aims at the encouragement and development of their interests and abilities, assistance in their training and re-education, alleviation of the loneliness of the friendless among them, and putting those requiring it in the way of advice and assistance. It endeavours also to arouse the social conscience on their problems and difficulties, to help on, when necessary, legislation on behalf of sufferers and to urge the energetic pursuit of research into the causes, prevention and cure of the disease.

Mr. Chairman, ladies and gentlemen, I have detained you over long and must bring my lecture to a close. There is much that I have omitted in what has been mainly a factual account of poliomyelitis in England and Wales.

For this account, I have been dependent on the work and investigations of others, and there is nothing of originality in what I have said. If I were to list my benefactors in full, you would be kept here much longer than you desire.

I would, however, mention specially my indebtedness to the Annual Reports of the Chief Medical Officer to the Local Government Board and later to the Ministry of Health and to the Registrar-General's Annual Statistical returns, which afforded the basis of the figures I have given you. I have too had considerable recourse to the Poliomyelitis Survey by an International Committee organized by Jeremiah Millbank and published in the U.S.A. in 1923; also to investigations by Dr. F. G. Crookshank who was, incidentally, a Chadwick lecturer on poliomyelitis in 1918 and to publications by Sir Arthur MacNalty who also later gave a Chadwick Lecture on the same subject.

I would like finally to thank Mr. F. H. Bradley of the Ministry of Health who went to much trouble over many weeks to help me in the statistical work entailed in the preparation of this lecture.

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