
Poliomyelitis and the Neurologists: The View from England, 1896–1966

ANNE HARDY

The American vs. the European Experience

In 1907, following one of the earliest poliomyelitis outbreaks in New York City, the New York Neurological Society set up a poliomyelitis committee with a view to studying and illuminating the behavior of this newly epidemic disease.¹ The neurologists' interest in polio was of long standing, dating from the orthopedic surgeon Jacob von Heine's conclusion, in 1860, that the symptoms of infant paralysis pointed to "an affection of the central nervous system, namely the spinal cord."² Ten years later, Heine's deduction had been confirmed by the researches of Jean-Martin Charcot (1825–93), who demonstrated the destruction of the grey matter of the anterior horns of the spinal cord (within which the motor impulses arise and are transmitted) in cases of infant paralysis.³ This demonstration of the pathology of the disease placed it firmly within the concerns of neurology, where it remained until polio arrived as a major epidemic problem.

In America, according to John R. Paul, the New York Neurological Society's committee marked "almost the last time that neurologists as a professional body were to assume a position of dominance with respect

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1. John R. Paul, *A History of Poliomyelitis* (New Haven: Yale University Press, 1971), p. 107.

2. *Ibid.*, p. 53.

3. *Ibid.*, pp. 31–33, 54–56.

to poliomyelitis.”⁴ In 1909 Karl Landsteiner and his colleagues announced their discovery of the virus of poliomyelitis, and in the following year, Simon Flexner, director of the Rockefeller Institute for Medical Research, threw the resources of his well-funded and scientifically distinguished institution into the problem of polio. Thereafter the disease was taken over by “virologists, internists, pediatricians, orthopedists, specialists in physical medicine, and public health officials.”⁵ Although it took more than forty years for the efforts of America’s multiple medical specialists to resolve the problem of poliomyelitis, their involvement with the disease demonstrated very clearly its exceptional nature as an illness that required a high level of medical teamwork, both in its therapeutic and in its public health aspects.

The American experience of poliomyelitis overshadowed that of other Western countries in the decades to 1950. It was America that suffered the most spectacular epidemics—notably that in New York in 1916, in which some 2,400 people died. It was in the United States that public concern over the disease reached its highest peak, and that decades of laborious experimental research culminated in the development of Jonas Salk’s vaccine against the disease in the early 1950s. America had its own dramatic national figurehead for polio in President Franklin Roosevelt, himself crippled by the disease.⁶ The known history of poliomyelitis, both social and scientific, belongs largely to America. By contrast, the countries of Western Europe, with the single exception of Denmark, escaped the devastation wrought by major epidemics of the disease until after World War II.⁷

Yet Europe did not remain untouched by the disease. The English experience of poliomyelitis, for example, has been described in a recent study as negligible: “Everything to do with polio in Britain—not least the disease itself—was on a minor scale.”⁸ Despite “incipient panic” with the first big epidemic in 1947, the country’s response to polio is described as “phlegmatic” compared with that of New York in 1916, probably because of the more advanced state of knowledge available through American research.⁹ While the assessment of England’s polio problem as minor is

4. *Ibid.*, p. 107.

5. *Ibid.*

6. See *ibid.*; Naomi Rogers, *Dirt and Disease: Polio before FDR* (New Brunswick, N.J.: Rutgers University Press, 1992); Tony Gould, *A Summer Plague: Polio and Its Survivors* (New Haven: Yale University Press, 1995).

7. “World-wide Prevalence of Acute Poliomyelitis” (leading article), *Brit. Med. J.*, 1937, 2: 533.

8. Gould, *Summer Plague* (n. 6), p. 161.

9. *Ibid.*, pp. 161–62.

perfectly fair—there was an average of only 680 cases a year in the 1920s—this picture overestimates the extent to which response to the 1947 epidemic was tempered by more advanced knowledge. Although English medical observers kept up with American poliomyelitis research, and avidly reported developments in the medical journals, such knowledge had not by 1947 led to any significant advances in prevention, treatment, or cure. As the Ministry of Health's senior medical officer, W. H. Bradley, observed in 1948, the problem of polio the world over was one of "ignorance, impotence and insecurity."¹⁰

In fact, the British response to polio in 1947 was tempered far more by some fifty years of persistent, relatively low-key experience of polio and polio-alarms than by all the labors in all the laboratories of America. Beginning in the 1890s, a small number of British clinicians had maintained a close interest in polio, and in subsequent decades it was they, by and large, who responded to the periodic outbreaks of polio and public alarm, who kept abreast of developments in research, and who sought to maintain a degree of public awareness of the disease. One of the persistent questions that exercised these men was why Britain should have continued to escape major outbreaks of polio, and they were in constant expectation that the disease would establish itself in epidemic form, as it had in America, Scandinavia, and Australasia.¹¹ When it finally did manifest itself as a major epidemic in 1947, expectation of its arrival was well established. At the core of this group of medical men who maintained an interest in polio before 1947 were the neurologists centered on the National Hospital, Queen Square.¹² Whereas in America poliomyelitis had become a multiple-specialty interest by 1910, in England the dominance of the neurologists lasted for another forty years, and then continued to some extent in parallel with the upsurging interest of the virologists, the epidemiologists, and the many other medical specialists with legitimate concerns in epidemic polio.¹³

10. W. H. Bradley, "Reports of Official Delegates—England and Wales," in *Poliomyelitis: Papers and Discussions Presented at the First International Poliomyelitis Conference* (Philadelphia: J. B. Lippincott, 1949), pp. 335–36, quoted in Gould, *Summer Plague*, p. 161.

11. See, for example, "Rest for Acute Poliomyelitis" (leading article), *Brit. Med. J.*, 1926, 2: 947–48.

12. Until the 1920s, it was traditional for Queen Square staff to be associated with the practice of medicine or surgery in other institutions. With the establishment of neurological departments in most teaching hospitals after ca. 1920, and with the increasing amount of care required by neurological patients, this tradition gradually broke down: Gordon Holmes, *The National Hospital, Queen Square, 1860–1948* (Edinburgh: E. & S. Livingstone, 1954), p. 7.

13. The history of specialization, both of individual specialties and of broader patterns, has been neglected for twentieth-century England, and is beyond the scope of this article.

This was not a development resented by the neurologists. Indeed, in 1945 the distinguished neurologist F. M. R. Walshe (1885–1973) had, in a paper delivered to the Royal Society of Arts, deplored the fact that “in this country there is growing up the illusion that from beginning to end poliomyelitis is a matter for the orthopaedic surgeon.”¹⁴ Walshe stressed that a wide range of medical expertise was called for in the study and handling of the problems caused by poliomyelitis—the experimentalist, the physician, the public health expert, and the orthopedic surgeon each had his part to play. The expertise of the physician was required in diagnosis, for the initial recognition of polio and its differentiation from other febrile illnesses; the medical researcher contributed to understanding the disease process, and the public health man to the control of infection; and the orthopedic surgeon was needed to “salve” the paralyzed patient and to manage his convalescence. For Walshe, polio was a disease that above all required medical teamwork, in respect to both the individual patient and the wider community—this was, he noted, the model on which the Americans worked.¹⁵

The conception of polio as a disease requiring teamwork was not new in 1945, as the American model indicates, but it was new in the twentieth century, and derived from the recognition that polio was an infectious disease.¹⁶ This recognition, of which evidence was first provided by the Swedish pediatrician Ivar Wickman in his classic monograph on the Swedish epidemic of 1905, had been forced upon the Americans not just

For the history of orthopedic surgery, see Roger Cooter, *Surgery and Society in Peace and War: Orthopaedics and the Organization of Modern Medicine, 1880–1948* (Basingstoke: Macmillan, 1993). Older histories remain the principal resource for neurology and pediatrics; see Holmes, *National Hospital* (n. 12), and Herbert C. Cameron, *The British Paediatric Association 1928–1952* (London: British Paediatric Association, 1952). Epidemiology and virology did not achieve professional recognition in hospitals and universities in Britain until the 1920s; the history of these disciplines in Britain in the twentieth century remains unwritten.

14. F. M. R. Walshe, “Infantile Paralysis,” *J. Roy. Soc. Arts*, 1945, 93: 533.

15. *Ibid.*, pp. 532–33. These views were shared by a number of orthopedic surgeons. The surgeons, however, had a somewhat more limited interpretation of teamwork. Ernest Hey Groves, for example, professor of surgery at the University of Bristol, declared that the subject of poliomyelitis “should bring pathologist, neurologist, and surgeon into close touch with the general practitioner” (Ernest W. Hey Groves, “The Treatment of Infantile Paralysis with a Plea for the Reorganization of our Hospital System,” *Brit. Med. J.*, 1925, 1: 492–496, quotation on p. 492. See also Sir Robert Jones, “An Address on the Treatment of Paralysis in Children,” *ibid.*, 1922, 1: 705–708, especially p. 705; Harry Platt, “The Early Mechanical Treatment of Acute Anterior Poliomyelitis,” *ibid.*, 1924, 1: 266–67. I am indebted to Roger Cooter for the Platt reference.

16. Edward Farquhar Buzzard, “Opening Paper” (British Medical Association Annual Meeting; section of orthopaedics and the diseases of children; discussion on the early diagnosis and treatment of acute poliomyelitis), *Brit. Med. J.*, 1921, 2: 225–27.

by scientific research, but by the urgent necessity of placing blame for the terrible epidemics they suffered. As Naomi Rogers has pointed out in her study of New York's 1916 epidemic, America placed polio within a traditional sanitarian framework, constructing it as a disease of filth and poverty; in particular, public health officials came to emphasize the part played by flies in transmitting infection.¹⁷

The Nature of the Disease

Poliomyelitis is an acute viral disease, which occurs endemically and epidemically throughout the world, and which is characterized by a tendency to produce variable degrees of paralysis.¹⁸ Today, it is widely accepted as being transmitted by the fecal-oral route, and it is known that unrecognized infections exist to a far greater extent than does the clinical disease. Where the disease is endemic, among populations living with poor sanitation and hygiene, serologic surveys have demonstrated that nearly all children have acquired specific antibodies to the three poliovirus types by the age of five years; very young children are far less likely than older children and adults to suffer severe paralytic disease or death, and indeed, their infections are often subclinical or appear as no more than a cold, or mild influenza. With improvements in hygiene standards, however, and the associated social and economic changes, children are increasingly protected from immunizing infections in their early years, with the result that communities become vulnerable to epidemic outbreaks and more severe manifestations of disease.¹⁹

This transition from endemic to epidemic status, which occurred at different times in different Western countries in the years between 1880 and 1950, provoked widespread reassessment and reinterpretation of social and medical perceptions of polio, but also complicated these processes of explanation. Poliomyelitis, with its ability to maim and to kill, required a very different explanatory framework from its rarely encountered predecessor, infant paralysis.²⁰ Moreover, the new disci-

17. Rogers, *Dirt and Disease* (n. 6), chaps. 1, 5.

18. Robert L. Summitt, ed., *Comprehensive Pediatrics*, 3d ed. (St Louis: C. V. Mosby, 1990), p. 858.

19. Ralph D. Feigin and James D. Cherry, eds., *Textbook of Pediatric Infectious Diseases*, 3d ed., 2 vols. (Philadelphia: W. B. Saunders, 1992), 2: 1708; M. T. Parker and L. H. Collier, eds., *Topley and Wilson's Principles of Bacteriology, Virology, and Immunity*, 8th ed., 5 vols. (London: Edward Arnold, 1990), 4: 137.

20. The development of explanatory frameworks for disease is notably explored in the recent collection edited by Charles E. Rosenberg and Janet Lynne Golden, *Framing Disease: Studies in Cultural History* (New Brunswick, N.J.: Rutgers University Press, 1992).

plines of bacteriology and virology proved unable to offer solutions to the problems of polio's specificity and mode of transmission until at least the 1940s. Although Wickman had argued on the basis of epidemiological observation that the disease was transmitted primarily through subclinical infections, no tests for the presence of poliovirus could be developed; and although Simon Flexner had pronounced the source of infection to lie in the nasopharynx, precautions taken on this assumption had demonstrably little effect in restraining the spread of the disease.

In England, where there were no substantial epidemics and where the random scattering of cases among all social classes was well noted, there was no urgency to achieve a popular frame for the disease in the early decades of the twentieth century. Although there was initial alarm and interest in England at the time of the first American polio epidemics, as witnessed by the English adoption of notification rules at about the same time that the American health authorities required official reporting of the disease, popular anxiety was not sustained. Indeed, the English medical profession as a whole was slow to grasp the idea of polio as an infectious disease. As the Medical Officer of Health for the London borough of Islington noted in 1913, it was perhaps not surprising that people could not be brought to look on polio as infectious when medical men continued to state that it was nothing more than infant paralysis, "about which no fuss was ever made."²¹ Between 1918 and 1947 polio was largely neglected by the medical professionals most concerned with infectious disease, the medical officers of health and the epidemiologists. General practitioners, too, rarely came across the disease except as sporadic cases, and they frequently failed to diagnose them. It was left to the neurologists, in whose province the disease had traditionally lain as a mysterious affection of the nervous system, to coordinate, expound, and publicize, as best they might, the newer knowledge of polio. Between 1890 and 1945, the neurologists sought to frame poliomyelitis for the public and the profession, and to prepare both for the day when the disease would become epidemic. Working essentially from the Swedish model, and adapting the new concept of the healthy carrier, they consistently promoted the image of polio as a directly infectious disease.²²

21. Report of the Islington Medical Officer of Health, originally published in fall 1914; republished as "Notes of the Week: Disinfection after Poliomyelitis," *Med. Officer*, 1915, 13: 51.

22. For the origins of the concept of the carrier, and its early application in American public health practice, see Judith Walzer Leavitt, "'Typhoid Mary' Strikes Back: Bacteriological Theory and Practice in Early Twentieth-Century Public Health," *Isis*, 1992, 83: 608–29. See also John C. G. Ledingham and Joseph A. Arkwright, *The Carrier Problem in Infectious Diseases* (London: Edward Arnold, 1912).

Poliomyelitis in England

The history of polio in England between 1896 and the early 1960s can be divided into three phases, in each of which the neurologists played a pivotal role.²³ Between 1896 and 1918, polio was first discovered by the neurologists as an infectious disease, and subsequently as a minor epidemic by the public health authorities, the medical profession, and the general public. Between 1918 and 1946, the profile of the disease stabilized as a sporadic, small-scale epidemic of limited public health significance, whose enormous potential was recognized by a few. After 1946, this epidemic potential was realized—if not in terms of full-scale public panic, then in the sense of profound public concern and the detailed attention of the wider medical profession.

The First Phase: 1896–1918

The discovery of polio as an epidemic disease by English neurologists in the 1890s can in many ways be attributed to Thomas Buzzard (1831–1919), colleague and friend of Hughlings Jackson at the National Hospital for Nervous Diseases, Queen Square. Buzzard, whom David Ferrier described as a man of great caution and sound judgment, was one of the first British neurologists to develop an interest in the etiology—as distinct from the pathology—of the nervous diseases.²⁴ He was present at the International Medical Congress of 1890 where the Swedish pediatrician Oskar Medin (1847–1927) delivered his classic description of the Stockholm polio outbreak of 1887, which first alerted medicine to the epidemic possibilities of polio.²⁵ Buzzard had for some years been brooding on the relationship between his specialty and the dynamic new specialty of microbiology, and he assimilated Medin's study into the argument he was formulating.

In 1896, in his presidential address to the Clinical Society, Buzzard explored the linkages between microbiology and improvements in the treatment of nervous disorders, concluding with a discussion of the probable infectious origins of a number of these disorders. He noted the effects of leprosy, tetanus, and diphtheria, all them more or less recognized as being caused by microorganisms, and then discussed the

23. In the discussion that follows, the term *England* is used in the sense of “England and Wales” to the exclusion of Scotland and Ireland. This usage has been determined by the statistical material, which is derived from the records of the Registrar-General for England and Wales.

24. Ferrier's opinion is cited in an anonymous obituary: “Thomas Buzzard,” *Brit. Med. J.*, 1919, *I*: 59.

25. See Paul, *Poliomyelitis* (n. 1), pp. 74–76.

Stockholm epidemic and a number of his own cases of spinal paralysis: "With our present knowledge of the associations of epidemic disease," he observed, "it is impossible to doubt that the occurrence of such epidemics as these points to an origin by infection."²⁶ In eleven of Medin's cases, symptoms of paralysis of the cranial nerves, of polyneuritis, and of polyencephalitis were present alongside the typical spinal paralysis. In England, polyneuritis was not uncommonly associated with "anterior poliomyelitis"; thus, Buzzard continued (in an admittedly somewhat obscure passage), this occasional association "lends of itself some support to the view of an infective origin, for besides the very frequent acute polyneuritis connected with diphtheria, and the more chronic form usually seen in beri-beri, cases of acute multiple neuritis are not unfrequent in which the circumstances point forcibly to such a mode of production."²⁷ Significantly, he added, "Notably cases of acute 'ascending' . . . paralysis strongly suggest a septic origin."²⁸ Ascending paralysis is characteristic of the respiratory form of polio.

Buzzard's discussion of the variety of symptoms presented in cases of infant paralysis made a deep impact on a least one member of his audience. The physician William Pasteur, of the Queen's Hospital for Children, Hackney, had that very summer studied a series of cases of illness in a family of seven children, aged between eighteen months and eleven years, which he had classified as infant paralysis after some hesitation, because of the variety of types of paralysis presented by the different cases. Buzzard's paper convinced Pasteur that all his cases were poliomyelitis; their occurrence in succession over a period of ten days convinced him that infant paralysis was indeed an acute infectious disease.²⁹

Pasteur's cases apart, however, polio continued to keep a low profile in England at the turn of the century. Nonetheless, staff at the National Hospital were honing their expertise. The disease was, in fact, constantly present in London during the summer months, and could be observed without difficulty in hospital practice. In particular, a young physician named Frederick Batten (1865–1918) began to pay attention to the disease. His first appointment had been to the Children's Hospital at Great Ormond Street (1894), but he quickly specialized in the nervous diseases of children, and in 1899 was appointed to the staff at Queen Square. In 1902, Batten noted an undue prevalence of polio in London,

26. Thomas Buzzard, "Presidential Address," *Clin. Soc. Trans.*, 1896, 29: lviii.

27. *Ibid.*

28. *Ibid.*

29. William Pasteur, "An Epidemic of Infant Paralysis Occurring in Children of the Same Family," *Clin. Soc. Trans.*, 1897, 30: 143–50.

and in 1904 he collected some 45 cases at Great Ormond Street—all apparently isolated cases, and drawn from all over London. In 1908, a small group of 8 cases was reported from the then-tiny village of Upminster; and in June 1909 the largest outbreak so far in England occurred, with 32 cases being reported from Bristol between June and October.³⁰

The Bristol epidemic took place just before the publication of Landsteiner's discovery of the poliovirus in November 1909, but it was this discovery that galvanized the medical awareness of polio in England as elsewhere. A whole spate of minor epidemic outbreaks were recorded in the summer of 1910—at Maryport, Workington, Barrow-in-Furness, Carlisle, Newcastle, Manchester, Nottingham, Melton Mowbray, Cerne Abbas, and Weymouth. Batten was kept busy, circularizing local medical officers of health for information about outbreaks and recording the particulars—although, as he noted, this survey was inevitably defective, since polio did not come within the purview of the MOHs, and their information could not be other than incomplete.³¹ In 1911, this pattern was repeated: in the *British Medical Journal* alone, general practitioners, medical officers of health, and hospital physicians recorded outbreaks at Plymouth, Stonehouse, and Devonport; at Penryn in Cornwall, Swadlincote in Derbyshire, and Whittington in Staffordshire; in Huntingdonshire; at Downham in Norfolk, at Stowmarket, and in Birmingham. In July the Royal College of Physicians, at Batten's instigation, asked the London County Council to make poliomyelitis a notifiable disease, and the LCC complied.³² In October the city of Birmingham also added polio to its list of notifiable diseases; and in December the Local Government Board instructed such local authorities as had not already done so to make polio and cerebrospinal fever (with which it was often confused) notifiable "at an early date."³³ By 1912, therefore, polio had been placed within the province of the public health authorities.

Batten's involvement with polio extended beyond amateur monitoring and agitating for proper notice to be taken of the disease. He

30. See Frederick E. Batten, "The Epidemiology of Poliomyelitis," *Proc. Roy. Soc. Med.*, 1910–11, 4: 198–221; idem, "Acute Epidemic Poliomyelitis," *Pub. Health*, 1911–12, 25: 51–64, especially p. 56. See also idem, "Has Acute Anterior Poliomyelitis Been Unusually Prevalent during This Summer?" *Lancet*, 1902, 2: 707, and idem, "A Lecture on Acute Poliomyelitis and Encephalitis," *Lancet*, 1902, 2: 1677–79.

31. Batten, "Epidemiology of Poliomyelitis" (n. 30), p. 216.

32. See the remark by Shirley Murphy, the London County Council Medical Officer of Health, in the discussion that followed Batten's paper to the Royal Society of Medicine: *ibid.*, p. 223.

33. Editorial, "Acute Poliomyelitis and Cerebro-spinal Fever," *Brit. Med. J.*, 1911, 2: 1614; editorial, "Compulsory Notification of Poliomyelitis," *ibid.*, p. 245; untitled note, *ibid.*, p. 1028.

embarked on what amounted to a publicity campaign for the disease, lecturing to the Epidemiological Section of the Royal Society of Medicine, the British Medical Association, the Society of Medical Officers of Health, and the Medical Officers of Schools Association; and he alerted the neurological community through the journal *Brain*. To each of these audiences he addressed roughly the same speech, subsequently published in the various journals, in which he defined the disease, described its clinical features, detailed the nature of the virus, and discussed its epidemiology and the history of past outbreaks. Although his surveys ranged the available literature on poliomyelitis, his view of the disease was clearly taken from Wickman: for Batten, polio was an infectious viral disease, tending to affect children, and probably communicable from person to person, or carried by nonsymptomatic cases.³⁴ On each occasion, he ended by urging the greater involvement of the medical community that he was addressing. He told the epidemiologists at the Royal Society of Medicine, for example, that the clinicians had gone as far as they could in understanding the clinical aspects and pathology of the disease, and it was now for the epidemiologists to work out how it was communicated from person to person.³⁵ Similarly, he told the Society of Medical Officers of Health that “there is but one effective method of dealing with poliomyelitis, and that is by preventive medicine. Physicians and surgeons can do but little to relieve the disease, and it is for the practitioners of preventive medicine, of which this Society is representative, to take the matter in hand.”³⁶

Although the enthusiasm of English medical men for reporting local epidemics fell off considerably after 1911, Batten maintained a high level of involvement with the disease. He gave yet another version of his paper at the Seventeenth International Medical Congress in 1913; and he delivered an extended version as the Lumleian Lectures in 1916, which were published both in *Brain* and as a slim monograph, *Acute Poliomyelitis*.³⁷ Nor was Batten’s contribution to polio confined to raising awareness in the medical community and developing the tools for epidemiological study and practical control of the disease: in his own hospital practice he specialized in treating cases of child paralysis, and he displayed consider-

34. See, for example, Batten, “Epidemiology of Poliomyelitis” (n. 30), p. 218.

35. *Ibid.*, p. 226.

36. Batten, “Acute Epidemic Poliomyelitis” (n. 30), p. 62.

37. Frederick E. Batten, “Polioencephalitis and Poliomyelitis,” in *Seventeenth International Congress of Medicine* (London: Frowde, 1913), 14 vols., 17 (10): 145–61; *idem*, “Acute Poliomyelitis,” *Brain*, 1916, 39: 115–211; *idem*, *Acute Poliomyelitis: Its Nature and Treatment* (London: John Bale, Sons, and Danielsson, 1916).

able talent in developing light, adjustable, inexpensive celluloid splints for every form of infantile paralysis.³⁸

Before Batten's premature death at the age of fifty-three, in July 1918, his interests had begun to expand to take in "epidemic stupor" (encephalitis lethargica), a subject on which he worked with his friend and colleague Graham Still—so foreshadowing what was to become one of the brighter threads of English interest in polio in the 1920s, as one of a triumvirate of infectious diseases of the nervous system that had become prevalent between 1911 and 1918.³⁹ Indeed, Batten's work prepared the ground for the three principal interwar areas of interest in polio: as an epidemic infectious disease of the central nervous system; as a problem in schools; and as an orthopedic problem. The latter two were of particular interest to the medical community, but it was on the school issue that the neurologists made their appeal to the widest medical and public audiences.

The Second Phase: 1918–1946

In his analysis of the 1905 epidemic at Tingsryd in Sweden, Ivar Wickman had detailed the role of the local school as a disseminating point for poliomyelitis.⁴⁰ However, although (as already noted) Batten worked quickly to alert school medical officers to this potential problem, it was not until the mid-1920s that the problem arose in practice. Indeed, the pattern of polio's epidemic activity between the wars remained much as it had been in the years to 1914: a series of randomly scattered, relatively minor outbreaks against a background of randomly scattered sporadic cases. For the most part notifications averaged a few hundred per annum; exceptionally, in 1926, 1938, and 1940 they numbered more than a thousand.⁴¹ Later epidemiological studies revealed, however, that some areas suffered more persistently than others. Between 1919 and 1946, of

38. See Batten, *Acute Poliomyelitis* (n. 37), pp. 88–95; "F. E. Batten," *Munk's Roll* (London: Royal College of Physicians, 1955), 4: 426; William Osler, "Royal Society of Medicine: Exhibition of Cases," *Brit. Med. J.*, 1912, 2: 1389.

39. See, especially, Arthur S. MacNalty, *Epidemic Diseases of the Central Nervous System* (London: Faber & Gwyer, 1927).

40. See Paul, *Poliomyelitis* (n. 1), pp. 92–93.

41. It is worth remembering, however, that notifications reflected only a portion of the cases, many of which went unrecognized. It was commonly observed in orthopedic departments that the numbers of polio-crippled children presenting for treatment considerably exceeded the numbers that would have been predicted on the basis of notification. The number of children in special schools provided further evidence. See Groves, "Treatment of Infantile Paralysis" (n. 15), p. 493; John A. H. Brincker, "The Notifiable Diseases of the Nervous System as They Affect Children of School Age," *J. Roy. Sanit. Inst.*, 1927, 48: 92–96.

the 62 counties of England and Wales, 7 experienced three or more polio outbreaks;⁴² 14 experienced two; and 19 had one—leaving 22 counties without any epidemic at all. Similarly, certain towns and cities also suffered disproportionately: of 83 county boroughs, 20 had a sizable outbreak, but only 12 had more than one. There were 9 large towns where polio was epidemic three or four times in those twenty-eight years: Bristol, Oxford, Sheffield, Plymouth, Kingston-upon-Hull, Cardiff, Portsmouth, Manchester, and Birmingham; as Austin Bradford Hill (who compiled this information) noted, it might be significant that 6 of these were ports, while of the 20 worst-affected towns, half were ports.⁴³

This case-distribution was not so obvious to contemporaries. What concerned them most was polio as associated with that great British middle-class institution—the boarding school. Between 1926, when the first school epidemics were recorded, and 1947, boarding schools provided the focus for the medical debates about the nature of polio and the means of controlling it in England. For the educated British public, which habitually sent its sons (and often its daughters also) away to school in these years, the question was one of grave concern. The alarm began in the autumn of 1926, when polio appeared in school communities based around the Kent coastal town of Broadstairs. Broadstairs, which lies at the extreme eastern edge of the Kent seaboard, had then a reputation as a healthy seaside resort—all bracing sea-air and cross-Channel winds—and for this reason there were some twenty-six private day and boarding schools in the district, educating more than a thousand children. It was in certain of these that the outbreak concentrated; the poorer members of the local community almost entirely escaped.⁴⁴

The outbreak began suddenly, on 14 October, and within six days 31 cases of polio had been notified from twelve schools, mainly in the northern part of the district and near the sea. By the time the epidemic subsided (equally suddenly) on 29 October, 61 notifications had been confirmed, and a thirteenth school had become involved. Naturally, these outbreaks caused grave concern, and the schools' medical officers met together to decide on policy. A Ministry of Health medical officer and the eminent Harley Street pediatrician Reginald Miller (1879–1948) attended as consultants, and together they decided that they must isolate the schools as

42. The seven were Westmoreland, Lincolnshire, Essex, Kent, Hampshire, Somerset, and Cornwall.

43. Austin Bradford Hill, "Poliomyelitis in England and Wales between the Wars," *Proc. Roy. Soc. Med., Sect. Epidemiol. Prevent. Med.*, 1954, 47: 795–806, especially p. 798.

44. Alex M. Watts, "Infantile Paralysis in Broadstairs," *Pub. Health*, 1926–27, 40: 114–16. For Broadstairs and its schools see Barrie Wootton, *Early Broadstairs and St Peter's in Old Photographs* (Stroud, Glos.: Alan Sutton, 1992), pp. 71–79.

far as possible, advising parents to allow their children to stay at school.⁴⁵ The pattern of the outbreaks, which were of explosive but very limited duration in each affected school, was the factor deciding this policy.⁴⁶

While the letters to parents explaining this position were in the post, however, news of the outbreaks broke in the national press. On the morning of Reginald Miller's return home, he found three sets of Harley Street parents with children at school in Broadstairs on his doorstep demanding advice. Once Miller had explained the policy to them, they agreed to their children's remaining at school, and their decision effectively influenced other parents.⁴⁷ But the Broadstairs decision became topical within a fortnight, when a further outbreak was reported at Uppingham School in Rutland. Here the first case died on Monday, 8 November, and the Headmaster notified parents immediately, hoping it was an isolated case. On the following Friday, however, another boy developed mild symptoms, and the Headmaster, notifying parents, asked if the children could return home: by Saturday evening all five hundred boys had dispersed to their homes, and the school closed for a fortnight.⁴⁸

These developments were noted in the *Times* of 15 November, and the following day the paper published a letter from Lord Dawson of Penn, medical watchdog extraordinaire, and Dr. James Collier (1870–1935), physician at Queen Square.⁴⁹ The two eminent physicians castigated the closure of Uppingham as an error of judgment, of no benefit to the boys and a disservice to the community. Warmly advocating the isolation policy pursued at Broadstairs, they declared:

there is no sufficient reason for dealing with this illness differently from a visitation of scarlet fever, measles, or influenzal pneumonia, and for none of these diseases are we in the habit of shutting schools. . . . To send a whole school home for two weeks does not mean the problem will be over when the school reopens; the same precautions will have to be taken as were taken at Broadstairs from the outset of the outbreak.⁵⁰

45. Watts, "Infantile Paralysis" (n. 44), p. 116.

46. Reginald Miller (letter), "Epidemic Polio-encephalomyelitis in Schools," *Brit. Med. J.*, 1926, 2: 1018–19.

47. *Ibid.*

48. *Times* (London), 15 November 1926, p. 11; 18 November, p. 10.

49. Dawson of Penn is best known for his involvement in planning for the provision of health care by the state, but he had a wider career as a "man of affairs." He chose to regard his elevation to the peerage in 1920 as "an invitation from the State to the medical profession to take a larger share in national life," and thereafter concerned himself widely in medically related issues: "Viscount Dawson of Penn," *Munk's Roll* (London: Royal College of Physicians, 1955), 4: 446–49.

50. *Times*, 16 November 1926, p. 15.

It was impossible, they added, to watch boys as closely at home as at school, and besides there was the risk of spreading infection across the country.

This letter provoked prompt dissension and debate, both among parents and within the medical profession.⁵¹ The crux of the issue lay in the way polio was perceived by these communities, and in the differing interpretations of the infectiousness of polio. Reginald Miller, for one, was quick to list his objections, contrasting the limited duration of the Broadstairs outbreaks with the apparent chain-development at Uppingham. Was it worse, he inquired, if a child carrying polio infected another at school, or if it returned home and infected another there? Furthermore, Dawson and Collier had not allowed for the psychology of parents:

To ask them to regard this disease as in any way at all comparable with the infectious diseases they know is useless. They recognize no resemblance and, however many cases of permanent paralysis from it they see, and however many cases of children being found dead in bed they hear of, they will not be impressed with such resemblance. They do not look upon their school as a quarantine station, nor do they deem themselves fit to be responsible for the public health; they are simply anxious that their children should escape as quickly as possible and with the least risk.⁵²

And, he added, instancing his Harley Street parents, it was those who knew about the disease who were the most apprehensive.

Collier, however, now launched himself on a crusade that bore a distinct resemblance to Batten's earlier efforts, delivering papers on polio to a range of different audiences. If the school epidemics had initially sparked his interest, he was now developing a novel theory of polio transmission based on some of the latest epidemiological research. English observers of polio had hitherto tended to accept Wickman's well-established theory that polio was an infectious disease spread largely by apparently healthy carriers and by case-to-case infection. However, in the mid-1920s the epidemiologists had begun to explore concepts of herd immunity, both through experimental studies and through the more traditional medium of observation. In the Milroy Lectures for 1926, W. W. C. Topley expounded his work with Major Greenwood on experimen-

51. The debate continued in the pages of the *Times* for some weeks: see the letters from A. H. Henderson, 18 November 1926, p. 10; Sir Brian Donkin, 19 November, p. 10; Rev. R. H. Owen, 20 November, p. 12; J. A. Fort, 23 November, p. 10; Judge Lailey and Dr. S. G. Moore, 26 November, p. 15; Rev. R. H. Owen, 27 November, p. 13; Canon G. L. Richardson, 3 December, p. 10; and also the leading article on 1 December 1926, p. 15.

52. Miller, "Epidemic Polio-encephalomyelitis" (n. 46), p. 1019. See also Reginald Miller (letter), *Times*, 17 November 1926, p. 15.

tal epidemics in mouse populations; and at nearly the same time, the Medical Research Council published S. F. Dudley's work on invasion, carriage, and salting, which paralleled the mouse studies by observing small human communities. Topley and Greenwood had concluded that if a herd of infected mice were strictly isolated, infection would disappear, perhaps in a short time; that if a few unsalted immigrants were allowed to dribble into the infected herd from time to time, the infection could never be got rid of; and that each new addition of susceptible mice raised the virulence of infection.⁵³ Dudley, observing epidemic outbreaks in schools and military and naval depots, reached very similar conclusions—but with the additional reflection that bacterial variation in response to herd immunity resulted in the gradual vaccination of communities by carriers, with infection subsequently dying out despite increasing numbers of carriers.⁵⁴ All this evidence, in Collier's view, reinforced the necessity of isolating polio-infected communities so that all susceptibles could be cleared out and universal immunity established.⁵⁵

There was a difficulty, though, which hinged on the duration of carrier status. In February 1927 Collier was postulating a chain of carriers who perpetuated polio, carrying it over from season to season, and causing the sporadic cases that occurred every month of every year in London. The virulence of the carrier depended on contact with highly susceptible individuals, and contacts were, he argued, important in salting the community.⁵⁶ By July 1927, Collier had developed this theory further. He now argued that a carrier epidemic preceded each outbreak of polio; as the number of healthy carriers increased, he suggested, so susceptible contacts were infected and the outbreak developed. The practically simultaneous infection of all available susceptibles resulted in a characteristic epidemic pattern: "rapid blaze, high peak, and rapid fall."⁵⁷

Collier's theory, while it justified his argument for isolating polio-affected schools, also carried important public health implications, and it quickly attracted adverse reaction.⁵⁸ In fact, it was Collier's fellow-

53. William W. C. Topley, First, Second, and Third Milroy Lectures on Experimental Epidemiology, *Lancet*, 1926, 1: 477–84, 531–37, 645–51.

54. Sheldon F. Dudley, "The Spread of Droplet Infection in Semi-isolated Communities," Medical Research Council, *Special Report Series*, no. 111, 1927, pp. 1–61.

55. See Collier's summary of Topley and Dudley's research: James Collier, "Personal Observations upon the Clinical Features of Poliomyelitis," *Lancet*, 1927, 1: 321–26.

56. *Ibid.*, pp. 321, 325–26.

57. James Collier, "An Address on the Epidemiology and Pathology of Poliomyelitis," *Brit. Med. J.*, 1927, 1: 751–53. See also the discussion in F. M. R. Walshe, "Some Points in the Epidemiology of Poliomyelitis," *J. Roy. Sanit. Inst.*, 1927, 48: 81–86.

58. For the complexities of public health policy in respect to carriers, see Leavitt, "Typhoid Mary" (n. 22).

physician at the National Hospital, F. M. R. Walshe, who delivered the coup de grâce. Although Collier was a gifted clinician, his excursion into epidemiology was less successful, and he did not succeed in convincing school physicians and governors of the necessity of isolating schools. Walshe was a formidable opponent, and he believed that school closure must be determined on the individual circumstances of each outbreak.⁵⁹ Walshe, like Collier, had begun to take a public stand on polio in the wake of the 1926 outbreaks—but unlike Collier, he did not make the mistake of generalizing a pattern for epidemic polio from the Broadstairs outbreak. In a paper on the epidemiology of the disease he went back to earlier accounts, including Wickman's, to demonstrate the “convincing proof of case-to-case spread.”⁶⁰ In his conclusion he noted:

Even if we allow a preponderant role to healthy carriers, the lines of spread and the day to day incidence of cases in small semi-isolated communities show that the disease continues for an indefinite period to spread radially from a central focus so that every susceptible subject remains in danger of infection until the epidemic finally burns itself out.⁶¹

And as a postscript he added a reference to the latest American research, which reopened the case for the nonhuman spread of polio—for example, through the agency of milk. Such an agency, Walshe suggested, might have been responsible for the Broadstairs outbreak.⁶²

Both Collier and Walshe, like Batten before them, were happy to move from the immediate concerns of neuropathology to the clinical treatment of polio—and beyond, to issues of epidemiology and public health. Collier, indeed, made a remarkable statement in a speech to the Medical Officers of Schools Association in February 1927, in which he appeared to be staking neurology's claim for a defining voice on polio:

The epidemiological and public health aspects of poliomyelitis bristle with difficulties and dangers, ignorances and paradoxes, and so far from being

59. F. M. R. Walshe, “Poliomyelitis,” in “Report on a Discussion at the Medical Society of London,” *Brit. Med. J.*, 1927, 1: 282–83.

60. Walshe, “Some Points” (n. 57), p. 84.

61. *Ibid.*, p. 85.

62. *Ibid.*, pp. 83–86. The American research to which Walshe referred was that of A. C. Knapp, Edward S. Godfrey, and W. Lloyd Aycock, “An Outbreak of Poliomyelitis Apparently Milk-Borne,” *JAMA*, 1926, 87: 635–39. At about the time that Walshe made this point, Aycock took it further in his own investigation of the Broadstairs outbreak: William L. Aycock, “A Milk-Borne Epidemic of Poliomyelitis,” *Amer. J. Hygiene*, 1927, 7: 791–803. The Broadstairs outbreak seems to have been one of the few poliomyelitis outbreaks that was proved to be milk-borne.

hotly disputed in the daily newspapers, should be approached with extreme caution and in a spirit of constructive reverence exclusively by those whose experience gives them the right to undertake such a grave responsibility.⁶³

In this case, however, Collier's experience had to defer to Walshe's. It was Walshe who continued as neurological adviser on polio to the wider medical community. Indeed, by the early 1930s he too had developed a novel theory of polio—as an exclusively nervous disease rather than an acute specific fever—and he came under attack from yet another of his Queen Square colleagues, the highly respected James Purdon Martin (1893–1984).⁶⁴ Nonetheless Walshe's reputation as a polio expert remained untainted by this episode. In 1935, he contributed a paper on polio to a *British Medical Journal* series on the management of lesions of the nervous system in general practice, in which he warned against the ethical problems associated with the use of the iron lung; in 1946, he chaired an expert committee on the management of polio in schools.⁶⁵

The neurologists also maintained a powerful voice on the nature of treatment provided to polio patients in both the acute and convalescent phases of the disease, well into the 1950s. When Sister Elizabeth Kenny's theory and treatment of polio became topical in the later 1930s, the British medical profession reacted with reservations.⁶⁶ Despite the testing of Kenny's methods at Queen Mary's Hospital Carshalton, under the auspices of the LCC, and their cautious welcome by a committee of experts, severer criticism of Kenny surfaced in the early 1940s, notably in the pages of the *British Medical Journal*. Significantly, one *BMJ* leader observed that while there was indeed room for improvement in the treatment of polio, and for further study of its underlying pathology, these could not be entrusted to any enthusiasts: "We believe," the writer declared, "that understanding will be enhanced by critical enquiry upon

63. The published version of this paper omitted the second half of the sentence, but it was cited in full by John Brincker later in the year: see Collier, "Personal Observations" (n. 55), p. 321; Brincker, "Notifiable Diseases" (n. 41), p. 94. For an example of these "hot disputes" in the daily papers, see the *Times* correspondence cited in n. 51 above.

64. See F. M. R. Walshe, "A Paper on Poliomyelitis," *Brit. Med. J.*, 1933, 2: 1197–1200; James Purdon-Martin, "The Bearing of Recent Work on Certain Aspects of Poliomyelitis," *ibid.*, pp. 1200–1202. Purdon Martin's early experience of encephalitis lethargica as a houseman in Liverpool in 1920–21 aroused his interest in the basal ganglia, which remained his principal interest for the rest of his life: see "James Purdon Martin," *Munk's Roll* (London: Royal College of Physicians, 1984), 7: 323.

65. F. M. R. Walshe, "The Treatment of Acute Poliomyelitis," *Brit. Med. J.*, 1935, 2: 733–35; "Poliomyelitis in Boarding Schools," *Lancet*, 1946, 1: 972–73.

66. For Sister Kenny see Paul, *Poliomyelitis* (n. 1), chap. 32; Gould, *Summer Plague* (n. 6), pp. 85–89 et seq.

strictly neurological lines. So far this does not seem to have been done in the U.S.A.”⁶⁷

In fact, medical interest in polio was sustained through the war years by discussions of the Kenny method; by the work of F. M. Burnet; by acceptance of the findings of John R. Paul and his colleagues that the portal of entry for the poliovirus was the gastrointestinal tract, not the nasopharynx as previously supposed; and by Herbert J. Seddon's investigation of epidemic polio in Malta.⁶⁸ In England itself, meanwhile, polio continued quiescent, with notifications running no higher than usual. In 1944 the medical superintendent of Plymouth Isolation Hospital, discussing misdiagnoses in the common infectious fevers, noted that among polio cases arriving at his hospital, misdiagnoses ran at over 50 percent.⁶⁹ Besides the “mechanical faults which could be seen in the out-patients' department of any general hospital, and the usual make-weights which had apparently no connection whatever with the disease,” he found a “list of nervous diseases which would do credit to Queen Square,” including cerebellar tumor, cerebral tumor, disseminated sclerosis, traumatic neuritis (drunkard's palsy), a transverse lesion of the spinal cord, a hemichorea, and a postdiphtheritic palsy.⁷⁰ No doubt the superintendent voiced the feelings of many general practitioners in his despairing conclusion: “I feel quite incompetent to discuss the differential diagnosis in nervous diseases.”⁷¹

67. Editorial, “The Kenny Treatment of Poliomyelitis,” *Brit. Med. J.*, 1942, 2: 639. See also the editorials “Fact and Fancy in Poliomyelitis,” *ibid.*, 1943, 2: 141–42; “The Kenny Method Criticized,” *ibid.*, 1944, 1: 627.

68. For interest in Kenny see the citations in the preceding note. For notice of Burnet's work, see “New Light on Poliomyelitis from Australia” (leading article), *Brit. Med. J.*, 1940, 2: 123. For discussion of Paul's findings, see the editorial, “Poliomyelitis Virus in Stools and Sewage,” *ibid.*, p. 711; “The Route of Infection in Poliomyelitis” (leading article), *ibid.*, 1941, 2: 811–12. For Seddon's work on Malta, see “Epidemiology of Poliomyelitis” (report of a meeting at the Royal Society of Medicine), *ibid.*, 1943, 2: 686–87; Thomas Agius, Albert E. Bartolo, C. Coleiro, and Herbert J. Seddon, “Clinical Features of the Poliomyelitis Epidemic in Malta, 1942–43,” *ibid.*, 1945, 1: 759–63; Hyman G. G. Bernstein, John M. P. Clark, and Ronald E. Tunbridge, “Acute Anterior Poliomyelitis among Service Personnel in Malta: An Account of an Epidemic,” *ibid.*, pp. 763–67; “Poliomyelitis in Malta” (leading article), *ibid.*, pp. 773–74.

69. Donald F. Johnstone, “Some Mistaken Diagnoses in the Common Infectious Fevers,” *Brit. Med. J.*, 1944, 2: 555–59, see especially p. 558. Poliomyelitis was commonly diagnosed only after paralysis had set in; quite apart from the wide variety of types of paralysis consequent on polio, the relative rarity of the condition meant that many general practitioners, especially in country districts, were unfamiliar with its specific clinical features.

70. *Ibid.*

71. *Ibid.*

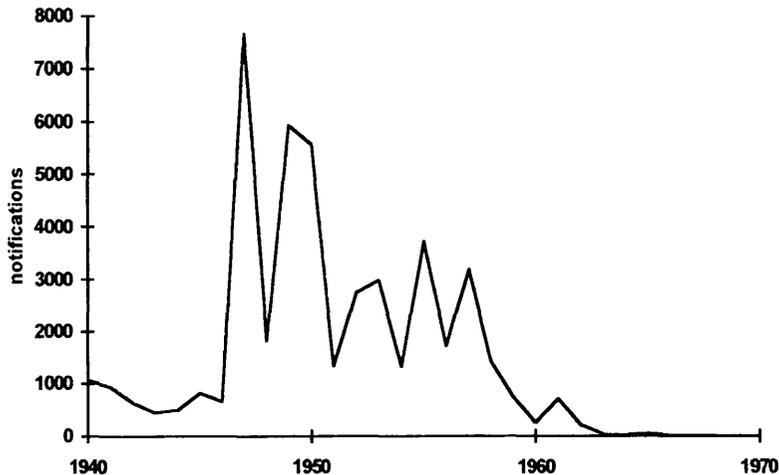


Fig. 1. Annual notifications of poliomyelitis (paralytic) in England and Wales, 1940–70. The disease was notifiable as “poliomyelitis” until 1949, and thereafter as two categories: “acute poliomyelitis (paralytic)” and “acute poliomyelitis (non-paralytic)”; this graph uses only the figures for “acute poliomyelitis (paralytic)” for the years 1950–70. *Source:* Compiled by Irvine Loudon from notification statistics given in *Registrar-General’s Statistical Review of England and Wales*.

The Third Phase: 1947–1966

The problems of differential diagnosis of polio for general practitioners and isolation hospital staff were about to become acute. In 1947, the established epidemic pattern of polio in England and Wales suddenly changed (see fig. 1). The level of polio notifications normally began to rise in late July: in 1947 the rise began in mid-June. In the week to 5 July, there were 79 notifications—a level rarely exceeded since the introduction of notification in 1912. In the following week there were 110 notifications; there were to be 7,766 before the year was out (corrected figures).⁷² The 1947 epidemic was unusual in two other respects: it claimed an unusually high proportion of adult victims, and it was largely concentrated in London. Both before and after 1947, outbreaks were scattered across the country. In fact, the changed behavior of polio after 1947 lay in an increase in the number of annual outbreaks, rather than in an increased attack rate. As W. H. Bradley pointed out, most of the statistically significant outbreaks before 1947 had occurred in just one place—only in 1926 and 1928 had two or three larger centers manifested themselves.

72. “Poliomyelitis” (leading article), *Brit. Med. J.*, 1947, 2: 135–36. See also *Registrar-General’s Statistical Review of England and Wales*, 1946 and 1947, 1951, 1 (*Text*): 124–25.

In 1947, however, 18 centers were involved besides London; in 1949, 11; and in 1950, 25. A change had occurred in the dynamics of the disease's spread, rather than in the virulence of the virus. Surveying the whole pattern of polio's behavior since 1922, Bradley echoed the feelings expressed by A. H. Gale and E. P. Hargreaves in their local study of polio in Devon and Cornwall between 1912 and 1952: that outbreaks tended to occur in the same rather ill defined areas, sometimes at intervals of many years, but that there was no obvious social characteristic common to these areas.⁷³

With the arrival of polio as a major epidemic in 1947 the English medical establishment took quick defensive action. Because few physicians had the opportunity of observing the preparalytic stage of polio, the instruction of general practitioners in this regard became a high priority. The *BMJ* published careful details of the symptoms and management of cases.⁷⁴ The Ministry of Health rushed into producing educational films, the first of which—*Polio Diagnosis and Management*—was aimed at general practitioners and emphasized the clinical features of the disease;⁷⁵ it included “an outstanding close-up . . . of a larger-than-life palate with just enough weakness on the left for it to be pulled a fraction to the right.”⁷⁶ The second film, *Johnny Green in His Fighting Chance*, was aimed at the general public.⁷⁷ The London teaching hospitals set up special units for the treatment of acutely ill polio patients, and the Emergency Bed Service agreed to coordinate the distribution of mechanical respirators both within and beyond London.⁷⁸

As in America thirty years earlier, the major epidemic outbreak precipitated an upsurge of interest in polio among a wide variety of medical specialists: the epidemiologists, who had been notably inactive before 1947, now sprang with relish into the fray, while virologists, medical officers of health, fever hospital medical officers, and the Ministry of Health all discovered a renewed enthusiasm for studying polio. Within this up-

73. William H. Bradley, “Poliomyelitis,” *J. Roy. Sanit. Inst.*, 1954, 74: 526; Arthur H. Gale and Edward P. Hargreaves, “History of Poliomyelitis in Cornwall and Devon,” *Brit. J. Prevent. Soc. Med.*, 1953, 7: 180–93.

74. “Poliomyelitis” (n. 72), p. 135; W. Howlett Kelleher, “Some Clinical Observations on the Present Outbreak of Acute Poliomyelitis,” *Brit. Med. J.*, 1947, 2: 291–95.

75. The Ministry of Health film, *Polio Diagnosis and Management* (1947), London: National Film and Television Archives, British Film Institute. See also Editorial, “New Poliomyelitis Measures,” *Brit. Med. J.*, 1947, 2: 260.

76. Editorial, “Poliomyelitis Film,” *Brit. Med. J.*, 1947, 2: 303.

77. The Ministry of Health film, *Johnny Green in His Fighting Chance* (1949), London: National Film and Television Archives, British Film Institute. See also “Epidemiological Notes: Film for the General Public,” *Brit. Med. J.*, 1949, 2: 342.

78. “New Poliomyelitis Measures” (n. 75), p. 260.

surge, however, the neurologists retained a place, both as coordinators and as contributors to the new research effort. In particular, a clinical study by William Ritchie Russell (1903–80), a consultant neurologist in Oxford, had a lasting impact on the advice offered to parents and practitioners on the management of suspected cases of polio. It had long been suspected by clinicians that polio patients' pains were greatest in the area of the body that would be the most severely paralyzed, and that great physical activity in the preparalytic stages was associated with severe paralysis; Russell's study provided statistical proof in confirmation of these suspicions.⁷⁹

It was Russell, too, who introduced into England the intermittent positive pressure respirator, developed by the Danes during the Copenhagen epidemic of 1952.⁸⁰ In the 1950s, Russell to a large extent assumed the mantle that Walshe had worn during the interwar period, as the most prominent neurologist advising on the management and treatment of poliomyelitis. His distinction in this field was recognized in 1966, when he was appointed to the professorship of clinical neurology at Oxford, newly endowed by the National Fund for Poliomyelitis Research (the charity that later became Action Research for the Crippled Child).⁸¹

By 1966, however, poliomyelitis was well along on the return journey to being a rare sporadic disease in England, as it was in America, Australia, and Western Europe. The development of Jonas Salk's inactivated-virus vaccine, and later of Albert Sabin's oral vaccine, followed by prolonged immunization campaigns, had largely replenished the vanishing natural immunities of Western populations—although the English, with their long history of suspicion of and reservation toward vaccines, lagged behind in the use of these measures.⁸² Denmark, for example, implemented immunization programs as soon as the field trials of the Salk vaccine had

79. Annotation, "Paralytic Poliomyelitis," *Brit. Med. J.*, 1949, 1: 488–89; William Ritchie Russell, "Paralytic Poliomyelitis: The Early Symptoms and the Effect of Physical Activity on the Course of the Disease," *ibid.*, pp. 465–71.

80. "William Ritchie Russell," *Munk's Roll* (London: Royal College of Physicians, 1984), 7: 514–15.

81. *Ibid.* See also "William Ritchie Russell," *Lancet*, 1980, 2: 1385. The structure of funding for polio research in Britain remains to be explored. Available funds were much smaller than in the United States: see Walshe, "Infantile Paralysis" (n. 14), p. 527.

82. See Roy M. MacLeod, "Law, Medicine and Public Opinion: The Resistance to Compulsory Health Legislation, 1870–1907," *Pub. Law*, Summer 1967, pp. 106–28, 188–211; Dorothy Porter and Roy Porter, "The Politics of Prevention: Anti-vaccinationism and Public Health in Nineteenth-Century England," *Med. Hist.*, 1988, 32: 231–52; Jane Lewis, "The Prevention of Diphtheria in Canada and Britain 1914–1945," *J. Soc. Hist.*, Summer 1987, 20: 163–76.

been completed in 1954, while the English immunization campaign did not get under way until the early summer of 1956.⁸³ In an effort to conserve the initially limited vaccine supplies for the most vulnerable population, the campaign was first aimed at the youngest children. Even when supplies became less of a problem, older children and young adults were slow to take up immunization; as Tony Gould has noted, it took the death of the footballer Jeff Hall from polio in 1959 to convince these age groups that immunization was desirable.⁸⁴ By 1963, however, the number of deaths recorded as being due to acute polio in England and Wales was down to single figures.⁸⁵ In the 1960s, therefore, polio joined the other infectious diseases of childhood in acquiring a nonthreatening status, and it dropped out of sight as a public health problem in England—although its presence as an orthopedic problem continued rather longer.⁸⁶

Conclusion

The differing experiences of America and England with poliomyelitis present a variety of historical contrasts—for example, of epidemic scale, of medical involvement, and of social concern. Although England's experience of epidemic polio was minor compared to that of America, it is not without its own suggestive history.

The medical definition of epidemics, and the ways in which societies differentiate between diseases, are highlighted by the English terminology of polio. Most English polio outbreaks before 1947 were very minor, and consisted in a random scattering of isolated cases over a period of time; the cases were numbered in tens and not in hundreds—yet these outbreaks were invariably described as epidemics. Clearly it was unfamil-

83. Gould, *Summer Plague* (n. 6), pp. 148, 164; "Medical Notes in Parliament: Poliomyelitis Vaccine," *Brit. Med. J.*, 1956, 1: 299, 579–80, 1551. Earlier British vaccination plans had been discouraged by "the Cutter incident," which occurred in April 1955, when 204 children in California and Idaho contracted polio after being treated with contaminated Salk-type vaccine issued by the Cutter Laboratories of Berkeley, California. The "incident" notwithstanding, Canada, France, Germany, and South Africa, as well as Denmark, had all begun to manufacture and administer polio vaccine by mid-1955. See Paul, *Poliomyelitis* (n. 1), p. 438. See also "Medical Notes in Parliament: The Salk Vaccine in Britain," *Brit. Med. J.*, 1955, 1: 1099; "Poliomyelitis Vaccine Trials Deferred," *Br. Med. J.*, 1955, 1: 1539–40, especially p. 1539.

84. Gould, *Summer Plague* (n. 6), pp. 173–74.

85. *Registrar-General's Statistical Review of England and Wales, 1963, 1965*, Pt. I, Table 22, p. 291.

86. The effects of poliomyelitis are with us still. The recent discovery of the late effects of polio poses problems for patients, clinicians, and neurologists; see Gould, *Summer Plague* (n. 6), pp. 205–15.

ilarity, but perhaps even more the fear of postpolio paralysis, that led to their definition as epidemics. In a city the size of Bristol in 1909, for example, 32 reported cases of measles spread over five months would not have been described as an epidemic; 32 reported cases of polio were. (Measles—endemic, familiar, universal, and generally disregarded—achieved epidemic status when its death toll began to rise; in Birmingham in 1909 the severity of an epidemic was judged by the 367 deaths registered in the first quarter of the year, compared with an average of 57 for the previous ten years.)⁸⁷

Further, polio's behavior in England raises questions about levels of personal and public hygiene. The rise of polio in the West is generally attributed to rising standards of personal and domestic hygiene,⁸⁸ but the differential distribution of the disease inevitably raises questions about the timing, nature, and location of these improving standards. Polio's failure to achieve a major epidemic in England before 1947 suggests that British hygiene standards may have risen only slowly to match those of America, Australia, and Scandinavia.

The sharpest contrast between England and America in regard to poliomyelitis lies in the responses of their respective medical professionals to the challenge it presented. The overwhelming response of Americans to the disease was partly a function of epidemic scale—the New York outbreak of 1907 was estimated at 750–1,200 cases—but it was also determined initially by the existence of laboratory research facilities and research funds, and later by the Roosevelt connection (more especially, by the activities of the National Foundation for Infantile Paralysis and its dynamic director, Basil O'Connor).⁸⁹ While the differences in response between England and America could be read as a consequence of differing scientific cultures, such a reading is probably simplistic. The factors of epidemic scale, and of Simon Flexner's scientific opportunism, in the first instance, and the accident of Roosevelt's involvement, in the second, were important elements in the disease's high American profile. The example of England reinforces the importance of epidemic scale as a factor determining levels of medical/scientific interest in poliomyelitis:

87. See *Med. Officer*, 1909, 1: 943.

88. See, for example, Feigin and Cherry, *Pediatric Infectious Diseases* (n. 19), p. 1708; Topley and Wilson, *Principles* (n. 19), 4: 174.

89. See Gould, *Summer Plague* (n. 6), for the history of the National Foundation's activities. F. M. R. Walshe drew attention to the disparate funding levels available to researchers in England and America, although he admitted that this was not the sole cause of the limited English contribution to poliomyelitis research: Walshe, "Infantile Paralysis" (n. 14), p. 527.

the floodgates of interest did not open until the disease achieved substantial epidemic status in 1947.

Whereas in America the nineteenth-century dominance of the neurologists over poliomyelitis was submerged by the tide of other interested specialties, that dominance continued in England in the face of the general indifference of other medical professionals as long as the disease remained essentially an endemic problem. This was partly a function of the traditional allocation of the disease to neurology, but it also resulted from the nature of that specialty, from the structure of the English medical profession, and from the largely endemic rather than epidemic behavior of the disease. The concern of the neurologist—as scientist, and hence as clinician—was with the disease process itself, from beginning to resolution. This overview gave him a general concern with the disease entity and its behavior that overrode the limited perspectives of the general physician coping with the acute fever, and of the orthopedic surgeon salvaging the crippled child. Unlike America, where the practice of medicine was already fragmenting among competing multiple specialties at the turn of the century, medical problems in England continued to be dealt with by general practitioners in the first instance.⁹⁰ Specialization as such began to advance in England only during the interwar years, with the establishment of professorial units in the universities, and with the support of the Medical Research Council.⁹¹ Against this background, polio's failure to materialize as a large-scale epidemic greatly reduced its interest value to general practitioners and research workers. The development of a team approach to poliomyelitis seems to have depended in part on the epidemic challenge presented by the disease, and in part on the existence of a sufficient reservoir of specialists whose professional interests stood to benefit from involvement with it.

90. Rosemary Stevens, *Medical Practice in Modern England: The Impact of Specialization and State Medicine* (New Haven: Yale University Press, 1966); idem, *American Medicine and the Public Interest* (New Haven: Yale University Press, 1971).

91. As indicated above (n. 13), patterns of specialization in twentieth-century England require more detailed exploration and elucidation.