Balancing Risks: Childhood Inoculations and America's Response to the Provocation of Paralytic Polio

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Summary. Polio provocation has concerned health professionals for nearly a century. Before an effective polio vaccine was licensed in 1955, evidence that certain paediatric injections could precipitate a polio infection and severe forms of paralysis informed medical debates, experiments and shifts in public health policy. This article explores how the theory was received and approached in the United States and the consequences of its protracted resolution. It contends that although medical professionals sought to maximise health benefits for American citizens, varying conceptions of what constituted an appropriate balance of risk inspired diverse health policy outcomes.

Keywords: United States; poliomyelitis; provocation; precipitation; immunisation; public health; tonsillectomies; injections; gamma globulin

'We certainly will not ask any doctor to hold up a diphtheria shot when it is needed', explained a New York City Health Department spokesperson in the summer of 1951, 'but we are asking everyone to be particularly cautious about injecting any substance during a possible polio epidemic'. This heedful assertion hints at a debate that simmered for generations, affecting American health professionals and civilians. Just as public health programmes were burnishing their credibility at mid-twentieth century, an adverse health link was discovered between paediatric injections and poliomyelitis. According to observers, some inoculated children faced a higher risk of developing paralytic polio in the limb receiving an injection. Termed polio provocation, this clinical manifestation was troubling for Americans, since it undermined the benefits of public health programmes and threatened the sanctity of the doctor–patient relationship. Medical professionals were forced into an uncomfortable position of considering whether injections intended to prevent some illnesses might also be causing another.

This article engages with historical scholarship at the intersection of polio and public health. Polio historians have examined the nature of epidemics and the consequences for stricken individuals and their families before a national response to the disease was undertaken.³ They have also focused on the discovery of the Salk and Sabin vaccines and how they

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¹'State Acts to Curb Polio Spread Peril', *New York Times*, 13 June 1951, 30.

² James Colgrove, *State of Immunity: The Politics of Vaccination in Twentieth-Century America* (Berkeley and Los Angeles: University of California Press, 2006), ch. 3.

³Naomi Rogers, *Dirt and Disease: Polio Before FDR* (New Brunswick: Rutgers University Press, 1996).

reduced the incidence of polio in America. ⁴ Complementing these works, public health historians have studied the character of polio immunization campaigns and the range of outcomes. ⁵ Despite these important contributions, historians have not examined polio provocation, which arose in the 1920s, waned in the late 1950s, and resurfaced in the 1980s.

By drawing on archival records, historical newspapers, and medical journals, this article traces the evolution of polio provocation and assesses how health professionals imagined, debated and negotiated its effect. It reveals the challenges of balancing health risks at a time of clinical uncertainty and the inadvertent consequences of medical programmes designed to reduce suffering and save lives. The risk posed by declining herd immunity from halting paediatric immunisations versus the risk of inciting paralytic polio from adhering to public health orthodoxy framed the debate. The article argues that although health professionals attempted to minimise illness and disease exposure, divergent ideas of what constituted an acceptable balance of risk inspired varying outcomes. ⁶ Even though polio provocation was freighted on a considerable body of empirical evidence, uncertainty about its epidemiological mechanism and doubts as to the veracity of scientific reporting allowed room for subjective interpretations. Despite disagreement, most American health professionals achieved a compromise in which inoculation practices were reformed to reduce the risk of inciting polio. However, failure to unlock the epidemiological mechanism behind polio provocation until the 1990s rationalised sustained uncertainty, which affected international public health programmes for decades.

The Origins of Polio Provocation

For the first half of the twentieth century, many Americans considered polio a frightening disease. Although most polio infections passed unnoticed or with mild symptoms, in some cases the virus entered the blood stream and targeted the motor neurons of the spinal cord. Depending on the location and severity of the attack, complications could result in paralysis or death. Polio's notable seasonal occurrence, affinity for children, and legacy of disability tested the fortitude of survivors, families, and communities alike. At the height of the epidemic', a Minnesota physician remembered, 'the people of Minneapolis were so frightened that there was nobody in the restaurants. There was practically no traffic, the stores were empty. It just was considered a feat of bravado almost to go out and mingle in the public. To curtail epidemics, parents were advised to keep children rested,

⁴David M. Oshinsky, *Polio: An American Story: The Crusade that Mobilized the Nation Against the 20th Century's Most Feared Disease* (Oxford: Oxford University Press, 2005); Jane S. Smith, *Patenting the Sun: Polio and the Salk Vaccine* (New York: William Morrow & Company, Inc., 1990).

⁵Colgrove, *State of Immunity*, ch. 4; Sydney Ann Halpern, *Lesser Harms: The Morality of Risk in Medical Research* (Chicago: University of Chicago Press, 2006), ch. 2.

⁶Mary Douglas and Aaron Wildavsky, *Risk and Culture:* An Essay on the Selection of Technological and Environmental Dangers (Berkeley: University of California Press,

^{1983),} chs 1 and 3; Martin S. Pernick, A Calculus of Suffering: Pain, Professionalism, and Anesthesia in Nineteenth Century America (New York: Columbia University Press, 1985); Lee Clarke, Acceptable Risk? Making Decisions in a Toxic Environment (Berkeley: University of California Press, 1989), ch. 8; Bob Heyman (eds), Risk, Health, and Health Care: A Qualitative Approach (London: Arnold, 1998), ch. 2.

⁷Oshinsky, *Polio: An American Story*, 8.

⁸Ibid., 27.

⁹Richard Aldrich in *A Paralyzing Fear: The Triumph over Polio in America* (New York: TV Books, 1998), 113.

clean, and away from sources of contamination, such as swimming pools or movie theatres. The continual threat of polio ultimately increased anxiety and shaped cultural norms.

As part of a national effort to fight polio, President Franklin D. Roosevelt and his law partner Basil O'Connor, formed the National Foundation for Infantile Paralysis (NFIP) in 1938. The mandate of this charity was to sponsor public education programmes, acute and convalescent polio care, and medical research. To support its ambitious mission, the NFIP collected donations from Americans through the annual March of Dimes fundraising drive, which exploited prevailing fears about polio to generate revenue. Through flashy publicity campaigns and powerful cinematic productions, polio was recast as a spectre that haunted playgrounds and infected innocent children. Americans' growing concern about the disease bolstered the NFIP mandate and inspired research into causes and prevention.

With the financial backing of the NFIP, medical professionals and researchers examined whether social or environmental factors exacerbated the risk of polio infection. Older causation theories implicating immigrants or poor hygiene were slowly supplanted by new concepts, such as the physiological consequences of bruising, fatigue, sore throats, common colds, pregnancy, or diet. ¹³ By the 1940s, polio was no longer conceived by most Americans as a disease rooted in insalubrious tenements, but an affliction targeting prosperous, active, and aspiring people. March of Dimes campaigns, directed at predominantly middle-class donors, helped to fuel the association. ¹⁴ In fact, many medical experts attributed President Roosevelt's paralysis of 1921 to his arduous travel and strenuous cold water swim off the coast of Campobello Island. ¹⁵ The shifting conception of polio combined with new causation theories normalized the disease as arising from a complex interrelationship between personal characteristics and precipitating factors.

Since polio was endemic in America during the first half of the twentieth century and could be present on objects, surfaces, or skin, it posed an unremitting danger to those who did not enjoy protection from appropriate disease-fighting antibodies. ¹⁶ However, for some observers, the risk of polio infection appeared to be compounded in the aftermath of specific forms of surgery or immunisation. Although doctors did not have the technology to assess the epidemiological mechanism, strong evidence between polio and certain medical interventions began to be recognised.

Among the first procedures to be implicated was tonsil surgery.¹⁷ In 1910, doctors observed that children who underwent throat surgery during a polio epidemic also faced an elevated risk of contracting bulbar polio seven to fourteen days after the operation. British and American doctors later corroborated this finding in 1937.¹⁸ Although supporters

¹⁰Ibid., 53–69.

¹¹Smith, Patenting the Sun, 82.

¹²Rogers, *Dirt and Disease*, 30.

¹³Benjamin P. Sandler, *Diet Prevents Polio* (Lee Foundation for Nutritional Research, 1951); R. K. P., 'Factors That Predispose to Polio', *New York Times*, 19 August 1951, 135.

¹⁴Oshinsky, *Polio: An American Story*, 90.

¹⁵Smith, *Patenting the Sun*, 45–7.

¹⁶Oshinsky, *Polio: An American Story*, 8.

¹⁷C. K. Mills, 'The Tonsillectomy-Poliomyelitis Problem', Laryngoscope, 56, 1951, 1188–94.

¹⁸Fischer and Stillerman, 'Acute Anterior Poliomyelitis in New York in 1935, A Review of Six Hundred and Eighty-Six Cases,' American Journal of Diseases of Children, 54, 1937, 984–1004; Gerald N. Grob, 'The Rise and Decline of Tonsillectomy in Twentieth-Century America', Journal of the History of Medicine and Allied Sciences, 62, 4, 2007, 383–421; D. M. Anderson and J. H. Dixon, 'Acute Bulbar Paralysis: Report of Two Cases', British Medical Journal, 1938, 1077–9.

of this hypothesis were not wholly opposed to tonsil surgery, they warned fellow clinicians that 'operations in the nose and throat area should not be performed during epidemics'. ¹⁹ As case histories strengthened the possible adverse health link, medical researchers and epidemiologists attempted to measure the correlation. The director of the Harvard Infantile Paralysis Commission conducted a detailed study of over 2,000 case histories, concluding that tonsil surgery led to a significant danger of bulbar polio. ²⁰ In one instance, a family from Akron, Ohio, submitted five of their six children to tonsillectomies. Within two weeks, all five children developed bulbar polio and only two survived. ²¹ Through such heartwrenching case histories, doctors became aware of the need to balance the risk of provoking polio with the purported benefits of removing tonsils.

American doctors were divided over the clinical evidence implicating tonsil surgery. Proponents, such as Major-General E. A. Noyes, stated categorically 'that the policy of the United States Army has been to stop tonsil and adenoid operations during epidemics'. However, some practising otolaryngologists reasoned that there were too many 'disadvantages in postponing the tonsil and adenoid operation, including prolongation of general malaise and the danger of complications'. ²² Such assertions were justified by a small body of research, claiming no apparent correlation between throat surgery and polio provocation. ²³ Some health professionals attacked what they perceived to be the work of medical fearmongers and assured clinicians that 'the danger of contracting poliomyelitis following recent tonsillectomy [was] minimal'. ²⁴ The lack of consensus forced many American physicians and surgeons to measure the risks on a case-by-case basis.

Anxiety surrounding the hypothesis peaked in 1950 with an increase in the number of tonsillectomy operations and a corresponding spike in the incidence of bulbar polio. Fresh clinical evidence, including an article published by Dr Gaylord W. Anderson, a Harvard graduate and head of the University of Minnesota's School of Public Health, showed that tonsillectomies appeared to treble the risk of children developing polio paralysis. This new data established a theory of polio provocation, which in turn inspired some doctors to bring the issue to public attention. *Evening Capital* readers of Annapolis, Maryland, were warned by a local doctor that 'the only thing I can say is that I would not want to have my children's tonsil's removed when there was a considerable amount of polio in the area in which we lived'. Although not all doctors agreed with the theory, many heeded cautionary advice and postponed tonsil surgery until the summer polio season had subsided. The possibility of

¹⁹Alfred E. Fischer, 'Poliomyelitis After Tonsillectomy', British Medical Journal, 1939, 533.

²⁰W. L. Aycock, 'Tonsillectomy and Poliomyelitis: Epidemiologic Considerations', *Medicine*, 21, 1942, 65–94.

²¹T. Francis, C. E. Krill, J. A. Toomey, and W. N. Mack, 'Poliomyelitis Following Tonsillectomy in Five Members of a Family: An Epidemiologic Study', *Journal of the American Medical Association*, 119, 17, 1942, 1392–6; 'Poliomyelitis After Tonsillectomy', *The Lancet*. 1942, 552.

²²V. E. Negus, 'Poliomyelitis and Tonsillectomy', *The Lancet*, 1948, 72.

²³J. W. Traynor, 'Tonsillectomy and Poliomyelitis in Southern Iowa, 1948–1949', *Journal of the Iowa Medical Society*, 40, 1950, 400–402.

²⁴Claude D. Winborn and John R. Stansbury, 'Poliomyelitis and tonsillectomy,' *The Laryngoscope*, 57, 8, 1947. 575–79.

²⁵J. Alison Glover, 'Poliomyelitis and Tonsillectomy', Public Health 1950, 154–5; J. Alison Glover, 'The Incidence of Tonsillectomy in School Children', Proceedings of the Royal Society of Medicine, 31, 10,1938, 1219–36.

²⁶Gaylord W. Anderson, 'The Risk of Poliomyelitis After Tonsillectomy', *The Annals of Otology, Rhinology,* and Laryngology, 59, 3,1950, 602.

²⁷Edwin P. Jordan, MD, 'The Doctor Answers', *The Evening Capital*, 23 June 1950, 4.

polio infection was not only a health risk in its own right, but it had become a larger clinical consideration and a public relations problem for medical practitioners. The power of doctors to practise along orthodox lines was being eroded by an unseen viral enemy.

Coinciding with the tonsillectomy debate and exacerbating public anxiety about the consequences of medical interventions was evidence that certain paediatric injections could also predispose individuals to severe forms of polio. The first reports of this hypothesis were published in 1914 by German physicians, who noted that children receiving Neosalvarsan injections for the treatment of congenital syphilis later became paralysed in the recipient limb. ²⁸ Similar findings were reported among children receiving cholera vaccine in France and small-pox vaccine in Italy. ²⁹ Although these results were not reproducible in the laboratory due to technological limitations, the association between certain immunisations and polio provocation gained traction among many European health professionals.

Despite the international dissemination of medical knowledge, the proposed link between injections and polio provocation went unacknowledged in the United States. Penetration was partially hindered by language barriers, since some of the first European studies were not published in English or translated in literature reviews. Evidence also suggests that most American clinicians were not searching for such adverse health reactions. As one physician from Guy's Hospital, London, speculated: 'The most probable explanation for the absence of these cases from the American literature is that they have not been looked for particularly.'³⁰ Funding for medical research was also paltry during the 1920s and 1930s and this restricted certain lines of inquiry. ³¹ Linguistic barriers, narrow clinical pursuits and funding restrictions conspired to limit propagation of the hypothesis.

Despite impediments to dissemination, the provocation of polio by injections was briefly considered by American researchers in the mid-1930s in an effort to understand a polio vaccine disaster. In 1934, Philadelphia researcher Dr John Kolmer conducted a series of human medical experiments with his prototype live-virus vaccine. After testing the concoction on himself and his children, Kolmer proceeded to administer it to over 10,000 children in New York City. When it became apparent that some children receiving the test vaccine subsequently suffered adverse health reactions and polio paralysis, Kolmer terminated his study. In trying to understand what went wrong, some scientists reasoned that local tissue irritation caused by the injection had contributed to the unusually severe paralysis.

²⁸H. Kern, 'Ueber eine anstaltsendemie von Heine Medinscher krankheit', *Muenchen Medizinische Wochenschrift*, 61 (1914)' 1053–55; Alterthum, 'Lues Congenita und Poliomyelitis', *Deutsche Medizinische Wochenschrift*., 13, 30 March 1928, 522.

²⁹M. DeTeyssien, 'Acute Poliomyelitis Following an Injection of Anti-Cholera Vaccine', *Journal de médecine De Bordeaux*, 92, 1921, 77–8; M. Jarmulowicz, 'Two Cases of Poliomyelitis Following Anti-Smallpox Vaccination', *Review of Neurology*, 37, January 1930, 92–3; E. Molinari, 'Poliomyelitis: Common Prodromal Symptoms and Therapy of Paralysis Following Anti-Smallpox Vaccination: Colibacillary Etiology', *Gazzetta degli ospedali e delle cliniche*, 55 February 1934, 226–32.

³⁰W. H. Bradley, 'Discussion on Poliomyelitis following Inoculations' (Section of Epidemiology and State Medicine), *Proceedings of the Royal Society of Medicine*, 43, 1950, 775–82.

³¹Harry Marks, The Progress of Experiment: Science and Therapeutic Reform in the United States, 1900–1990 (Cambridge: Cambridge University Press, 1997), ch. 2.

³²H. V. Wyatt, 'Provocation Poliomyelitis: Neglected Clinical Observations from 1914 to 1950', Bulletin of the History of Medicine, 55, 4, 1981, 550.

³³Rogers, *Dirt and Disease*, 171–2.

³⁴Halpern, Lesser Harms, 48–57; John R. Paul, A History of Poliomyelitis (New Haven: Yale University Press, 1971), 393.

Although this conclusion bore remarkable similarity to earlier European findings, it did not stimulate American researchers to expand their inquiry.

After the Second World War, however, the attention given to polio provocation increased in tandem with the expansion of public health programmes for diphtheria, pertussis and tetanus. 35 The convergence of epidemiological surveillance and the application of statistical methods permitted quantifiable assessments of immunisation practices and potential adverse reactions. ³⁶ One of the first detailed studies emerged from Guy's and Evelina Hospitals, London. The study's author identified 17 cases of polio paralysis that had developed in the limb injected with pertussis, tetanus and typhus inoculations. The association was reportedly 'of sufficient importance to make one consider prophylactic measures'. 37 As American researchers pondered European reports, a supporting Australian study was released by Dr Bertram P. McCloskey. After reviewing case histories from South Australia and Victoria, McCloskey testified that inoculations appeared to be a leading causative factor in polio paralysis.³⁸ Based on 340 case histories, McCloskey described a link between the site of inoculation and paralysis, as well as the increased risk within three months of receiving an injection. Upon consulting with health officials at the Commonwealth Serum Laboratories and the superintendent of the Infectious Diseases Hospital, McCloskey concluded that they all 'agreed that there was certainly evidence of some association between prophylactic injections and development of poliomyelitis in the epidemic'. 39 Clinical observations derived from over four nations across two continents had established a theory that American researchers and doctors could no longer ignore.

The European and Australian studies were met by most American public health officials with scepticism. The New York City Assistant Health Commissioner claimed that there was 'no evidence at this time to substantiate the results'. Similarly, other health professionals reasoned that polio provocation was perhaps the product of geographical or population idiosyncrasies pertinent to other continents. 'It would be extremely unwise to accept conclusions arrived at on the basis of English and Australian experience as applicable to the conditions existing in New York City', explained the Commissioner of Health. 'We must make our own observations and reach our own conclusions.' For these health officials, it was rash to believe that a seemingly foreign theory was relevant to the United States until it was proven by examining local cases using local methods.⁴⁰

American epidemiologists and researchers scrambled to apply American realities to the problem. Among the first to investigate was the New York City Department of Health, which reviewed epidemiological data for over 2,000 local polio cases. ⁴¹ Meanwhile, Anderson, already a staunch supporter of the tonsillectomy theory of polio provocation, turned his attention to the role of injections by examining 2,709 case histories from a 1946 outbreak. Anderson's study was compiled from family interviews conducted retrospectively by nurses

³⁵DeTeyssien, 'Acute Poliomyelitis', 77–8.

³⁶Elizabeth W. Etheridge, Sentinel for Health: A History of the Centers for Disease Control (Berkeley: University of California Press, 1992); Mark E. Rushefsky and Deborah R. McFarlane, The Politics of Public Health in the United States (Armonk: M.E. Sharpe, 2005).

³⁷J. K. Martin, 'Local Paralysis in Children after Injections', Archives of Disease in Childhood, 25, 121, March 1950, 1–14.

³⁸Bertram P. McCloskey, 'The Relation of Prophylactic Inoculations to the Onset of Poliomyelitis', *The Lancet*, April 1950, 659–63.

³⁹McCloskey, 'The Relation of Prophylactic Inoculations', 662.

Arthur Gelb, 'Polio Link Sought to Immunity "Shots", New York Times, 29 September 1950, 23.

⁴¹Ibid

and medical students. He reasoned that this method 'increased the accuracy', since sufficient time had passed to achieve full disclosure unclouded by 'emotional stress'. Corroborating the Australian and English results, Anderson found that 'in poliomyelitis patients who have received some antigen during the month prior to onset there is a high degree correlation between site of paralysis and site of injection'. Even though his findings were not conclusive, they offered a strong indication rooted in American data that common public health injections appeared to exacerbate paralytic polio.

In addition to Anderson's findings, some American laboratory researchers bolstered the theory. Studies undertaken by the New York State Department of Health laboratory showed that mice injected with tetanus or pertussis toxoids suffered a higher risk of paralysis following exposure to encephalomyelitis virus. 'The frequency of paralysis in the inoculated extremity was 7 or more times greater than among the untreated controls', researchers concluded. 'Although these animal studies used a different virus to assess the adverse health link, they offered a reproducible model of a complex underlying epidemiological mechanism. It was becoming evident to some American health professionals that children receiving injections during the polio season were being burdened with a new health risk.

The epidemiological mechanism behind polio provocation was vigorously debated and as the Editor of *Pediatrics* conceded: 'just how this is brought about remains a mystery'. ⁴⁴ According to one hypothesis, inoculums injured tissue in a manner comparable to severe fatigue, which weakened the body and predisposed it to viral infection. Others posited that inoculations were perhaps contaminated with viral matter and were sources of infection. ⁴⁵ Another implicated syringes reused without proper sterilization as the root of the problem. A more refined proposal, advanced by Dr Harold K. Faber of the Stanford University School of Medicine in San Francisco, suggested that poliovirus present on the skin of children was being driven into the body during the injection, thereby seeding it into the tissue. ⁴⁶ The lack of certainty over what caused polio provocation increased anxiety and brought the issue to mainstream attention.

Although toxoid-based immunisations, such as those for pertussis, diphtheria and tetanus, appeared to play a leading role in polio provocation, other types of injections were implicated. Studies undertaken at the New York State Department of Health by Bureau of Epidemiology and Communicable Disease Control director Dr Robert F. Korns found a similar link with injections of sedatives, penicillin, hormones, vitamins and Novocain used in dental work.⁴⁷ As an established epidemiologist with a doctoral degree from Johns Hopkins University, Korns's findings carried considerable influence in medical and public

⁴²Gaylord W. Anderson and Audrey E. Skaar, 'Poliomyelitis Occurring After Antigen Injections', *Pediatrics*, 7, 6, June 1951, 741–59.

⁴³Donald J. Dean, Sophia M. Cohen, and Gilbert Dall-dorf, 'The Effect of Unrelated Vaccines on the Localization of Paralysis in Mouse Encephalomyelitis', Proceedings of the Society for Experimental Biology & Medicine, 77, 1951, 834–36.

⁴⁴Editorial, *The Journal of Pediatrics*, 38, 6, June 1951, 781–2

⁴⁵Martin, 'Local Paralysis in Children', 3.

⁴⁶Harold K. Faber, 'Postinoculation Poliomyelitis', *Pediatrics*, 7, 2, 1951, 300–4.

⁴⁷Harry Weaver, Editorial, June 1951, Series 3: Gamma Globulin Field Trial, Box 3, Surveys and Studies Records, March of Dimes Archives, White Plains, New York (henceforth denoted as MDA); Robert F. Korns, Robert M. Albrecht, Frances B. Locke, 'The Association of Parenteral Injections With Poliomyelitis', American Journal of Public Health, 42, 2, February 1952, 153–69; Robert M. Albrecht and Frances B. Locke, 'Effect of Physical Activity on Prognosis of Poliomyelitis', Journal of the American Medical Association, 146, 9, 1951, 769–71.

health communities. ⁴⁸ Like Anderson, he derived his evidence from case histories gathered from family and patient interviews. According to Korns, each informant 'was interrogated on the type of material injected, the site, the date, and the name of the attending physician' to assess how injections may have predisposed the individual to polio. ⁴⁹ Unlike earlier surveys, Korns attempted to increase the statistical rigour by including a control group comprised of neighbours and household contacts. His results appeared startling: common injections doubled the rate of sustaining polio paralysis for up to two months following the inoculation. ⁵⁰ For many health professionals it was apparent that the theory affected both private practice medicine and public health programmes.

As American studies confirmed European and Australian findings, many health professionals lobbied for a widespread suspension of immunisation programmes. Anderson reasoned that 'postponement of immunization during the periods when poliomyelitis is prevalent will avoid any possibility of its conditioning an unfavourable response to the infection'. Most state and national medical societies, including the American Medical Association concurred, thereby sending a strong message to their members that the risk of inciting polio was not to be ignored. During the early 1950s, the theory of polio provocation developed into a significant medical issue, spurring professional debate and surprising health policy changes.

Health Risks and Policy Debates

Informed by medical sources, resourceful journalists brought news that certain injections could provoke polio to the American public. The Dixon, Illinois, *Evening Telegraph*, advised parents to 'Postpone Vaccinations for Children During Warm Polio Months'. Another publication warned readers that 'doctors found that more children under 5 years old developed polio within a month after vaccinations for whooping cough [pertussis] and diphtheria than non-vaccinated youngsters'. ⁵³ National newspapers, such as the *New York Times*, warned readers that polio paralysis due to immunisations 'usually involved the limb in which the injection had been given'. ⁵⁴ As dramatic headlines bombarded Americans, parents were asked to weigh on behalf of their children the potential risks of immunisation with its purported benefits. ⁵⁵

Some families of those stricken with polio in the aftermath of injections voiced criticism of public health programmes and pharmaceutical manufacturers. Australian parents reportedly 'were naturally inclined to blame the inoculations' and demanded that their physicians consider the likelihood of the association. ⁵⁶ In the United States, some citizens pursued litigation against pharmaceutical companies, whom they believed were responsible for selling contaminated vaccines. In 1947, the American public learned that Helen Covington and her

⁴⁸David M. Herszenhorn, 'Robert F. Korns, 82, Researcher Who Helped Test Polio Vaccine', New York Times, 16 October 1995, B7.

⁴⁹ Proceedings of the Committee on Immunization of the National Foundation for Infantile Paralysis', 6 July 1951, Box 244, Folder 4, Dr Jonas E. Salk Papers, Mandeville Library, California, 1.

⁵⁰Ibid., 17.

⁵¹Anderson and Skaar, 'Poliomyelitis Occurring After Antigen Injections', 758.

⁵²Alton L. Blakeslee, 'Doctors Advise: Postpone Vaccinations for Children During Warm Polio Months', *Dixon Evening Telegraph*, 29 May 1951, 9.

⁵³ Ibid

⁵⁴R. K. P., 'Factors That Predispose to Polio', 135.

⁵⁵Colgrove, State of Immunity, ch. 3.

⁵⁶Betram P. McCloskey, 'The Relation of Prophylactic Inoculations to the Onset of Poliomyelitis', *The Lancet*, April 1950, 659.

husband Daniel of New Kensington, Pennsylvania, had brought a combined \$200,000 suit against Wyeth Inc. in the United States District Court. Covington contended that Wyeth's rabies vaccine contained 'organisms, germs and other ingredients which rendered it unfit, unsafe and unsuitable for the purpose for which, it was intended' and thus 'produced partial paralysis' in her leg. Although Covington did not attribute her paralysis to a polio infection, the implication that vaccinations could inspire a neurological condition only amplified public concerns surrounding injections.⁵⁷

As the foremost polio charity, the NFIP waded into the debate. ⁵⁸ Since the NFIP was recognised by most Americans as a leader in polio care and medical research, pressure was placed on the organisation to offer an official position. In June 1951, NFIP research director Dr Harry M. Weaver issued a tactful press release. ⁵⁹ He assured parents and health professionals 'that recent warnings linking immunization injections and the development of a paralytic form of poliomyelitis were based on tentative evidence'. He advised that until further studies were undertaken to substantiate the correlations observed by Korns and Anderson, it was best to avoid 'indiscriminate injections' during polio epidemics. ⁶⁰ Although Weaver did not define what he meant by 'indiscriminate', his press release acknowledged the merit of the prevailing medical consensus and the need for doctors to assess the possible dangers on a case-by-case basis.

The polio provocation theory led to extraordinary shifts in public health policy. A growing cultural sensitivity to risk and risk aversion in post-war America inspired these cautionary policies. ⁶¹ The Academy of Pediatrics Committee on Immunization and Therapeutic Procedures advised its members to stop conducting immunisations during polio epidemics. 62 The US Surgeon General offered similar words of caution by declaring that medical studies showed 'some risk' with administering injections during 'polio season'. 63 Due to the unpredictability of epidemics, New York State Health Commissioner Dr Herman E. Hilleboe issued a directive to all county and city public health officers that 'all elective immunization procedures on persons over six months of age be discontinued during the poliomyelitis season'. 64 Hilleboe reasoned that the risk of provoking polio was more serious than the risk of other childhood diseases. 65 Heeding this mandate, New York City health officials halted pertussis and diphtheria inoculations at child health stations, and advised private practice physicians to 'suspend standard immunization inoculations for children' 66 State laws mandating paediatric vaccinations before school admission were also relaxed. ⁶⁷ Acknowledging the unusual change in policy, the City Health Commissioner explained that 'since investigation has shown a probable relation between immunization during the polio season and the

⁵⁷ 'Woman Files Suit Against Drug Co.', Indiana Evening Gazette, 19 July 1947, 10; 'Couple Sues Firm for Antitoxin', Chester Times, 18 July 1947, 11.

⁵⁸Dr. Weaver, Editorial, June 1951, Series 3: Gamma Globulin Field Trial, Box 3, Surveys and Studies Records, MDA.

⁵⁹ 'Polio Fund's Views on Injection Ban', New York Times, 21 June 1951, 28.

⁶⁰Ibid.

⁶¹ Jens I. Zinn, 'Risk as Discourse: Interdisciplinary Perspectives', Critical Approaches to Discourse Analysis across Disciplines, 4, 2, 2010, 106–24.

⁶² 'Proceedings of the Committee on Immunization', 17 May 1951, 51.

^{63&#}x27;U.S. Decries "Shots" In "Polio Season", New York Times, 22 June 1951, 27.

⁶⁴Hilleboe to State Public Health Physicians, 11 June 1951, Series 3: Gamma Globulin Field Trials, Box 3, Surveys and Studies Records, MDA.

^{65&#}x27;State Acts to Curb Polio Spread Peril', New York Times, 13 June 1951, 30.

⁶⁶ 'Polio Fund's Views on Injection Ban', 28.

⁶⁷ 'School Vaccination Law Relaxed for Polio Season', New York Times, 31 August 1951, 16.

localization of paralysis, we are postponing immunization until the danger is over'. ⁶⁸ Due to public awareness and mounting evidence corroborating the theory, health officials and clinicians adjusted practices to reduce perceived risks.

Not all health officers agreed with New York's pre-emptive 'ban on polio season inoculations' and some openly challenged the directive. Westchester County Health Commissioner Dr William A. Holla attacked Hillaboe's policy, believing that evidence did not 'justify such a drastic measure' and that adherence to such policies 'would put medicine back into the dark ages'. Encouraged by county physicians, Holla pressed his case. He argued that polio provocation was 'based on untested theories ... associated with the use of the hypodermic needle'. Upon this rationale, he explained that one could also 'outlaw Novocain, blood tests, and treatment of hay fever and other allergies'. Instead, Holla advised Westchester County doctors to continue to 'give primary immunization against diphtheria, whooping cough and tetanus ... but omit restimulating injections (booster shots) until further notice'. 69 By issuing his own counter-directive, Holla freed county physicians from disciplinary action, while ultimately assuming personal liability for the consequences. Although appearing to dispute the theory, Holla's directive was a compromise, recognising that certain injections should be omitted during the epidemic season. Even among dissenters there was a grudging acceptance of a possible link between childhood inoculations and ensuing paralysis.

Like Westchester County public health officials, some doctors reacted to the debate over polio provocation with anger and frustration. One physician at the Jefferson Medical College in Philadelphia expressed disappointment at the lack of precision within the debate and branded it the product of 'hysteria'. He explained that Anderson's findings only implicated injections given within one month before the onset of illness, not those given prior to that time. He further reasoned that while inoculations might play a role in exacerbating polio under certain conditions, they played 'no part in the etiology' of polio. 'The public and many public health authorities,' he continued, 'are reacting to the serious detriment of the overall disease prevention picture.' For such clinicians, ambiguity and scare tactics were inspiring regressive health policies.⁷⁰

Sharing a sentiment of suspicion, a few state and county health departments rejected the polio provocation theory and pleaded for calm. In July 1951, the State Health Commissioner of Massachusetts issued a statement that condemned seemingly alarmist reports. 'These claims,' he explained, 'have been featured in the public press and some parents and your board of health have been disturbed in regard to immunizations which are now needed.'⁷¹ The Commissioner asked that parents 'weigh the danger' of postponing or omitting immunisations for diphtheria and pertussis with the possibility that such injections might cause polio. Promising citizens that the state would most likely witness fewer polio cases in the coming year, he urged them that 'there would be no reason to postpone any immunizations'. In this instance, parents, not doctors, were asked to balance the health

⁶⁸Arthur Gelb, 'City Clinics Halt Diphtheria "Shots" For Summer as Polio Precaution', New York Times, 12 June 1951, 1.

⁶⁹ Dr. Holla Disregards State Ban on Polio Season Inoculations', *The Herald Statesman*, 19 June 1951, Series 3:

Gamma Globulin Field Trial, Box 3, Surveys and Studies Records. MDA.

⁷⁰Edward L. Bauer, 'Correspondence', *Pediatrics*, 8, 3, 1951, 452–3.

^{71&#}x27;Health Director Advises Not to Delay Injections', Lowell Sunday Sun, 1 July 1951, 8.

risks. In turn, the Director of the Massachusetts Communicable Diseases Commission attempted to discredit the theory. He assured parents that 'the original observer in Australia [McCloskey] was able to collect only 30 cases in which there appeared to be a relationship between immunizations and increased paralysis'. He implied a similar lack of credibility to the results at Guy's and Evelina Hospitals in London. Although Anderson and Korns brought forward American data to substantiate the theory, such studies were not discussed. The some detractors of the theory, the uncertain perception of foreign research served as an expedient means to strengthen their own assertions.

Although a few health professionals rejected the polio provocation theory, evidence suggests that most accommodated its possibility. Like New York City, the District of Columbia advised doctors against administering booster shots during the summer and recommended they consider 'the relative risk of withholding an injection or giving one'. ⁷³ Yet despite divisions, each group of health professionals believed that their respective approach represented the best interests of the public and their patients. In fact, advocates of the polio provocation theory reasoned that the risk of paralysis due to an injection was too high to condone orthodox practices; for dissenters, the hazard of declining herd immunity was by far the greater risk. As the theory resurrected public discomfort about the potential dangers associated with certain immunisations, medical professionals were united in trying to maintain faith in public health programmes and the sanctity of the doctor–patient relationship. ⁷⁴

Polio Provocation and Medical Experimentation

The prospect of a human medical experiment roused further uncertainty and debate about polio provocation. In the spring of 1951, Dr William McD. Hammon of the University of Pittsburgh and his collaborator Dr Joseph Stokes, Jr., of the Children's Hospital Philadelphia asked the NFIP to support a study assessing the human blood fraction gamma globulin (GG) as a means to prevent paralytic polio. Hammon's clinical trial protocol required a proving ground in which residents were experiencing a high incidence, early-stage polio epidemic; half of the paediatric cohort would be injected with 4–11cc. of GG, while the other half would receive an equivalent volume of inert gelatin. By tracking emergent polio cases at the test site and later comparing paralysis rates in the two groups from a decoded inoculation schedule, Hammon believed that the safety and efficacy of GG could be ascertained.⁷⁵

However, since many American health professionals reasoned that paediatric injections should be avoided during polio outbreaks, Hammon's plan to administer large dose inoculations to thousands of healthy children challenged medical opinion. Although no studies specifically implicated GG or gelatin as provoking agents, the fact that similar practices and serums appeared to cause paralysis threatened the presumed safety of the study. Hammon reasoned that since GG was a licensed substance and was already used by a

⁷² Ihid

^{73 &#}x27;Counsel on Polio Shots Given by District Health Director', The Washington Post, 19 July 1951, B1.

⁷⁴Kenton Kroker, Jennifer Keelan, and Pauline M. H. Mazumdar (eds), Crafting Immunity: Working Histories of Clinical Immunology (Aldershot: Ashgate

Publishing, Ltd., 2008); Colgrove, *State of Immunity*, chs 3 and 4.

⁷⁵William McD. Hammon, Lewis L. Coriell, and Joseph Stokes, Jr., 'Evaluation of Red Cross Gamma Globulin as a Prophylactic Agent for Poliomyelitis', *Journal of the American Medical Association*, 150, 8, 25 October 1952, 742.

small number of doctors for polio prevention without evidence of its efficacy, the balance of risk was in favour of generating scientific knowledge. He hoped that learning whether or not GG was an effective agent against polio would reap wider benefits.

Opposition to Hammon's trial was manifest among many public health leaders and polio researchers. Although debate was initially sporadic and limited to private correspondence, it was soon consolidated through the NFIP Committee on Immunization. Hammon's experimental protocol was initially presented at the inaugural gathering of the Committee in May 1951. In attendance were prominent researchers and health professionals, including Dr Jonas E. Salk and Dr Albert B. Sabin, as well as representatives from the Division of Biologics Control, the American National Red Cross, and the American Medical Association. Out of a growing concern, NFIP officials purposefully invited some of the most prominent medical minds to discuss the potential benefits and risks of the GG experiment.⁷⁶

After reviewing the protocol, most Committee members were opposed to the plan. In particular, they were distressed by the risks associated with polio provocation and offered testimony as to their belief in the adverse health link. 'Some of us heard the papers of both Anderson and Korns in Baltimore', one attendee explained; 'I think most of us ... were pretty well convinced that Korns's data was as good as Anderson's data.' Based on these findings, one polio researcher warned that if Hammon's trial went ahead, one 'might almost expect an increase in the amount of paralysis' in the injected control group. ⁷⁷ Until the theory was better understood, Committee members deemed that an experiment with GG was too dangerous. ⁷⁸ Hammon was shaken by the lack of confidence, but committed to appeasing his critics. ⁷⁹ Even though the polio provocation theory stood to scupper the proposed experiment, Hammon and his allies were far from defeated.

Hammon and NFIP officials attempted to assuage concerns and build consensus for the protocol. To achieve these ends, NFIP officials invited Korns to present and defend his data at a subsequent gathering of the Committee. ⁸⁰ In July 1951, Committee members listened as Korns expounded on his earlier studies, methodology and the range of inoculations that appeared to instigate polio paralysis. Korns was confident that a link existed. 'As to how large a role this phenomenon plays,' he concluded, 'the best we can say is that it seems to be double the hazard of getting polio and double the hazard of getting paralytic polio.'⁸¹

Korns's presentation legitimised Committee delegates' prior trepidation and opposition to the GG experiment, but Hammon and his allies attacked the data and the conclusions. They chided Korns for failure to delineate his analysis by the localisation of paralysis and identified a potential weakness in the sampling method. They also inferred that Korns's overall correlations with non-immunising agents might be erroneous, since the results could not be corroborated with those published by Anderson. ⁸² Although Korns's study was shown to

⁷⁶ L. Pearce Williams, 'Chapter VIII: Passive Immunity to Poliomyelitis', Preliminary Draft, 1956, Series 1: Passive Immunity, Box 1, History of the NFIP Records, MDA, 14–15.

⁷⁷ 'Proceedings of the Committee on Immunization', 17 May 1951, 8, 12.

⁷⁸Paul, A History of Poliomyelitis, 393.

⁷⁹'Proceedings of the Committee on Immunization', 17 May 1951, 62.

⁸⁰ Proceedings of the Committee on Immunization',6 July 1951.

⁸¹Ibid., 17.

⁸² Ibid., 18-19, 21.

harbour deficiencies, the debate remained unresolved. As one Committee member keenly observed, 'the risk remains undefined, in my mind, as it was during the last meeting, and I think we should be guided as much by the consideration of whether or not it is worth taking that undefined risk.'⁸³

Hammon was emboldened after discrediting Korns's findings and accordingly reasoned that there was no reason why he could not proceed with his clinical trial. ⁸⁴ As a compromise to this request, Committee members agreed to authorise a small pilot study among 5,000 healthy children, which would serve as a litmus test for public reception and an appraisal of any adverse health reactions. 'There will not be enough people injected to find out whether it is going to do much good', one Committee member acknowledged. 'We are going to try to find out whether it would cause a lot of harm.' ⁸⁵ Although most attendees were unenthusiastic about enrolling children in a safety test of GG during a polio epidemic, a pilot study appeared as a lesser evil than a large clinical trial. With the assistance of allies, Hammon received peer approval to launch a pilot study.

When news emerged that the NFIP Committee on Immunization was 'unanimous' in approving Hammon's pilot study, some leading public health officials expressed dismay. Raderson emerged as one of the most vocal opponents to the plan. During an epidemiological training session at the Communicable Disease Center in Atlanta, Georgia, Anderson confronted Hammon's collaborator Stokes, inquiring why support for the GG experiment remained when the polio provocation theory appeared so robust. After this encounter Stokes reported to Hammon that Anderson 'still strongly disapproves of our study'. Although Stokes failed to counter Anderson's concerns, he sought to dissuade antagonism by drawing attention to the inevitability of GG's clinical use in polio prevention as the primary rationale for the study.

Despite outward displays of confidence, Hammon was frustrated by Anderson's objections and considered them the result of professional obstinacy. 'I am sorry that Gaylord Anderson still disproves of our study,' he replied to Stokes, 'but knowing him as I do I imagine he will continue to stubbornly remain of the same opinion despite all arguments.' While marginalizing professional criticism, Hammon privately admitted to Stokes that Anderson's fears about GG injections provoking polio might be well founded: 'I hope he is not right', he conceded.⁸⁸ Like other members on the Committee, Hammon could only hope that polio paralysis would not be exacerbated by his experiment.

New York State Health Commissioner Hilleboe was also distressed by the decision to undertake the GG experiment. Committed to an overall injection ban, Hilleboe could not fathom how NFIP officials, on the vanguard of polio prevention, could disregard prevailing medical opinion. He penned a protest letter to NFIP director Weaver, who in turn, sought to curtail dissent. In classic diplomatic style, Weaver thanked Hilleboe for his letter and

⁸³Ibid., 25.

⁸⁴Ibid., 21.

⁸⁵ Ibid., 24.

⁸⁶Paul, A History of Poliomyelitis, 393.

⁸⁷Stokes to Hammon, 13 August 1951, St65p, Hammon, W. McD. #5, Joseph Stokes, Jr., Papers,

American Philosophical Society, Philadelphia, Pennsylvania (henceforth denoted as APS).

⁸⁸Hammon to Stokes, 16 August 1951, St65p, Hammon, W. McD. #5, Joseph Stokes, Jr., Papers, APS.

mitigated his concern by explaining that members of Committee had already given polio provocation 'very serious consideration'. Moreover, he noted that Korns had reported to the Committee and that he might be available to clarify 'a number of details that I cannot go into in a letter'. Weaver's reference to Korns served not only to challenge Hilleboe, but to divest justification for contentious decisions onto others. Although acknowledging that injected placebo controls were best avoided whenever possible, Weaver concluded that they were necessary to assess 'the usefulness of gamma globulin'. ⁸⁹ He ultimately placated Hilleboe by linking the momentum to proceed with a claimed majority scientific opinion. By corresponding with concerned public health thought leaders, NFIP officials helped Hammon deflect criticism and quash debate.

In an effort to mitigate the potential health risks, Hammon revised his experimental protocol. In particular, he decided to exclude children under two years of age from the pilot study, since they were believed to be the most susceptible to polio provocation. He also defined a method to reduce viral contamination on the injection tables by using changeable paper covers. ⁹⁰ Moreover, skin around the injection site would be disinfected with iodine and wiped clean with alcohol. Hammon reasoned that these clinical procedures would reduce tissue irritation and help to prevent the poliovirus from being driven into the body. ⁹¹ To measure the potential harm of his experiment, Hammon required injections to be made in 'the right buttock so that it could be determined later whether there was any association between the site of inoculation and the distribution of paralysis'. ⁹² Although his revised protocol could not eliminate the risk of polio provocation, it represented an expedient response and acknowledgement that the theory had bearing on his trial.

In September 1951, Hammon and his allies launched the GG pilot study in Utah County, Utah. With the endorsement of the local and state medical societies, they enrolled over 5,000 civilian children to serve as human subjects. Although Utah parents were asked to volunteer their children and to sign a legal waiver, Hammon did not acknowledge the possible health risks or his measurement of polio provocation. Akin to other American researchers who conducted human experiments at mid-century, Hammon was anxious about how his proposed studies might be perceived and whether an honest admission of risks might limit volunteer enrolment. Medical ethics suffered under these circumstances, as the interests of children and their parents became subordinated to those of the researchers.

Despite Hammon's intention to assess polio provocation, the pilot study failed to provide an answer. After comparing the paralytic incidence rates in the GG group, the gelatin group

(Baltimore: Johns Hopkins University Press, 1997); Allen M. Hornblum, *Acres of Skin: Human Experiments at Holmesburg Prison* (New York: Routledge, 1999), ch. 4; Susan M. Reverby, "Normal Exposure" and Inoculation Syphilis: A PHS "Tuskegee" Doctor in Guatemala, 1946–48', *Journal of Policy History* (January 2010); Susan L. Smith, 'Mustard Gas and American Race-Based Human Experimentation in World War II', *Journal of Law, Medicine & Ethics*, 36, 3, Fall 2008, 517–21.

⁸⁹Weaver to Hilleboe, 21 August 1951, Series 3: Gamma Globulin Field Trial, Box 3, Surveys and Studies Records, MDA.

⁹⁰ 'Worker's Badly Needed', Houston Informer, 5 July 1952, 1.

⁹¹ Hammon *et al.*, 'Evaluation of Red Cross Gamma Globulin', Part 1, 743.

⁹²Ibid.

⁹³Ibid., 744.

⁹⁴Susan E. Lederer, Subjected to Science: Human Experimentation in America Before the Second World War



Fig. 1 Gamma Globulin Pilot Study, Farrer School, Utah, 1951. Courtesy of the March of Dimes Archives, White Plains, New York (Photograph #51–928).

Copyright: March of Dimes

and the uninjected group, Hammon reasoned that the data showed no alarming adverse reactions. ⁹⁵ However, he acknowledged that 'the actual number of cases occurring in the various groups [were] too small to permit the drawing of statistically significant conclusions'. He admitted that his imprecise evaluation was unfortunate, but conceded that his protocol was primarily designed to measure the efficacy of GG for polio. ⁹⁶ Although the pilot study left the polio provocation theory unresolved, the character of the experiment acknowledged the risk. Indeed, Hammon's decision to restrict the enrolment age and modify his protocol to reduce viral contamination showed a grudging recognition of polio provocation. However, for Hammon, the balance of risk favoured the generation of knowledge over the immediate safety of child subjects.

The Refinement and Renaissance of Polio Provocation

While the epidemiological mechanism of polio provocation remained beyond the reach of American researchers during the 1950s, most health professionals acknowledged the theory's merit and its possible health consequences. By 1952, it was refined to implicate

⁹⁵ Report of Gamma Globulin Passive Immunization Field Project, Utah County, Utah, 1951', Series 14: Poliomyelitis, Gamma Globulin Program, Box 12, Medical Program Records, MDA, 9.

⁹⁶ Proceedings of the Committee on Immunization', 4 December 1951, 10–11.

toxoid-based immunisations as the most likely provoking substances. Medical and health organisations, such as the NFIP, the American Academy of Pediatrics, the American Medical Association and the American Public Health Association reported their consensus. In a joint statement they agreed that

injections against the common infectious diseases such as diphtheria, whooping cough and tetanus be postponed during periods of high poliomyelitis incidence unless the risk of the disease to be prevented by the injections is greater than the presumed risk of converting an asymptomatic poliomyelitis infection into a paralytic one, or of localizing the paralysis to the injected extremity by means of the injection.⁹⁷

As a result, many American doctors and public health officials postponed common paediatric immunisations during polio epidemics. 98 Cognisance of local realities and cycles of disease slowly became part of clinical practice when administering toxoid-based immunisations.

As the polio provocation theory was further refined, other injectable substances, such as sedatives, vitamins and antibiotics were exonerated from suspicion. Public health thought leaders, recognising the growing uncertainty over what constituted a risk, attempted to educate parents about the distinctions. One 1952 article in the *Washington Post* explained the official position of the United States Public Health Service, asserting that 'no parent should object and no physician should hesitate to administer a needed antibiotic, drug, or other injection for treatment of a disease at any time'. ⁹⁹ Similarly, GG escaped crushing indictments in the aftermath of subsequent clinical studies undertaken in 1952 with over 50,000 children in Texas, lowa, and Nebraska. Hammon's buoyant characterization of these later studies rationalised a national GG immunization programme against polio in 1953 and 1954. ¹⁰⁰ Although refinement of the polio provocation theory did not discount the risks posed by other injections, it merely situated them as less significant than they were before.

Concerns over polio provocation slowly waned after the first effective polio vaccine developed by Salk was licensed in April 1955. Most Americans expressed great relief at the imminent victory over epidemic polio. One observer remembered 'A man was crying and shouting as he tossed down copies of a special edition of the newspaper. When everyone finally understood his words there was great joy—a giddiness that can only be experienced at

Prophylactic Agent for Poliomyelitis: 3. Preliminary Report of Results Based on Clinical Diagnoses', *Journal of the American Medical Association*, 150, 1952, 757–60; W. M. Hammon, L. L. Coriell, P. F. Wehrle, J. Stokes Jr. 'Evaluation of Red Cross Gamma Globulin as a Prophylactic Agent for Poliomyelitis: 4. Final Report of Results Based on Clinical Diagnoses', *Journal of the American Medical Association*, 151, 1953, 1272–85; 'National Advisory Committee for the Evaluation of Gamma Globulin in the Prophylaxis of Polio, An Evaluation of the Gamma Globulin in the Prophylaxis of Paralytic Poliomyelitis in the United States During 1953', Spring 1954, Series 3: Gamma Globulin Field Trial, Box 4, Surveys and Studies Records, MDA.

⁹⁷'Use of Polio Vaccine During Epidemic Season', *American Journal of Public Health*, 45, July 1955, 956.

⁹⁸ Health Department Warns Against Any "Shots" as Precaution Against Polio', The Norwich Sun (New York), 14 June, 1951, 1.

⁹⁹ Some "Shots" Held Perilous in Polio Areas', *The Washington Post*, 10 May 1952, B5.

¹⁰⁰W. M. Hammon, L. L. Coriell, and J. Stokes Jr. 'Evaluation of Red Cross Gamma Globulin as a Prophylactic Agent for Poliomyelitis: 2. Conduct and Early Follow-up of 1952 Texas and lowa-Nebraska Studies', Journal of the American Medical Association, 150, 1952, 750–6; W. M. Hammon, L.L. Coriell, P. F. Wehrle, C. R. Klimt, J. Stokes Jr., 'Evaluation of Red Cross Gamma Globulin as a

the end of a time of terror.' ¹⁰¹ As millions of children became vaccinated before epidemic season, the incidence of polio dropped considerably. ¹⁰² For some polio survivors, the victory was bitter-sweet: 'My first reaction was anger,' recalled one patient, 'but it was selfish anger. It was anger that it didn't happen sooner. ... But I was so grateful that no one else would have to go through this. It was really something to celebrate.' ¹⁰³ The later Sabin oral polio vaccine led to a further reduction in the number of reported cases. ¹⁰⁴ In a nation no longer threatened by polio outbreaks or the related risk of polio provocation, Americans turned their attention to more pressing public health threats. Although the epidemiological mechanism behind polio provocation remained uncharted, research into such questions appeared indulgent and unnecessary. The dawn of the polio vaccine temporarily relegated polio provocation to the archives of medical history.

The theory of polio provocation lay dormant for decades, but re-emerged in the 1980s when large aid agencies, such as the World Health Organization and Rotary International, expanded immunisation programmes in developing nations. In Africa, public health workers began to notice cases of polio paralysis following certain paediatric inoculations against common diseases. Intrigued by the evidence, Dr H. Vivian Wyatt of the Department of Clinical Medicine at Leeds University turned to timeworn medical journals in search of a possible answer. After reflecting on the polio provocation debates of the 1950s, Wyatt became convinced that health workers in Africa were observing the manifestation of a forgotten theory. 'It appears,' he explained, 'that "injection paralysis" has scientific grounds for its existence and that indeed the localization in the affected limb is a direct effect of the injection, as Congo mothers have so long asserted. '105 Wyatt was persuaded that polio provocation had clinical relevance and he hoped that his publications would inspire fresh research into the correlation and reforms to public health practice.

Wyatt's work brought a measure of attention to the theory, but it was not until polio epidemics swept through India in the 1990s that the concept was systematically re-examined. A clinical study among children in rural India found that 'injections given for treatment of fevers ... may play a role in precipitating paralytic poliomyelitis'. A subsequent assessment corroborated this data, concluding that 'the only significant risk factor for paralytic illness was having received any injection in the 30 days before onset'. Based on results echoing those from decades before, some health professionals advised that 'injections in polio-endemic countries should only be indicated when other therapeutic options have failed or are not available'. However, not all health officials were convinced by the data. One group of researchers at the National Institute of Communicable Diseases in

¹⁰¹Leigh Flower Bonner, Houston, Texas, e-mail message to author, 5 August 2010.

¹⁰² U.S. Polio Cases 1952–1962', U.S. Public Health Service Health Information Series, 8, 7, 1963; Oshinsky, Polio: An American Story, chs 12 and 13.

¹⁰³Charlene Pugleasa in *A Paralyzing Fear*, 133.

¹⁰⁴Oshinsky, *Polio: An American Story*, ch. 15.

¹⁰⁵H. V. Wyatt, 'Provocation Poliomyelitis: Neglected Clinical Observations from 1914 to 1950', *Bulletin of the History of Medicine*, 55, 1981, 543–37; H. V. Wyatt, 'Provocation of Poliomyelitis by Multiple

Injections', *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 79, 3, 1985, 355–8.

¹⁰⁶M. Varghese, I. Qadeer, and D. Mohan, 'Paralytic Poliomyelitis in a Rural Area of North India', *The National Medical Journal of India*, 10, 1, Jan–Feb 1997, 8–10.

¹⁰⁷K. A. Kohler, W. G. Hlady, K. Banerjee, and R. W. Sutter, 'Outbreak of Poliomyelitis Due to Type 3 Poliovirus, Northern India, 1999–2000: Injections a Major Contributing Factor', *International Journal* of *Epidemiology*, 32, 2, April 2003, 272–7.

India challenged the suggestion that the DPT (diphtheria, pertussis and tetanus) vaccine exacerbated the 1992 polio epidemic at a Delphi orphanage. ¹⁰⁹ We 'do not attribute the occurrence of febrile illnesses in 21% of children in the orphanage to the inflammation from the DPT', concluded Institute staff. ¹¹⁰ Akin to 1950s America, health professionals in India were divided over the interpretation of clinical data and corresponding health risks.

Evidence that certain injections could provoke polio in India inspired a range of criticisms levelled at international aid organizations and local government agencies. Wyatt maintained his commitment to promoting awareness of polio provocation and he worked with Indian health officials to assess the consequences. ¹¹¹ In an article published in the *Indian Journal of Medical Ethics* he asserted: 'Since 1980 unnecessary injections have probably produced paralysis in more than 600,000 Indian children who might, otherwise, have got away with non-paralytic polio and worsened paralysis in another million children.' ¹¹² In agreement with Wyatt, some Indian paediatricians speculated that perhaps thousands of children had become paralysed because of misinformed immunisation policies. One physician at the Indian Academy of Pediatrics openly condemned her government for pursing 'a polio immunization policy advocated by the World Health Organization which was unsuitable for Indian conditions'. ¹¹³ For some critics of Indian immunisation policies, the trust of parents and the well-being of children were being undermined by some health professionals' ignorance and complacency. Until polio provocation was better understood, there remained ample room for a subjective interpretation of clinical findings.

It was not until the late 1990s that the polio provocation debate shifted in response to fresh scientific discoveries. In 1998, Drs Matthias Gromeier and Eckard Wimmer at the State University of New York uncovered what appeared to be the epidemiological mechanism behind polio provocation: muscle injury sustained by an injection facilitated viral invasion and its transmission to the spinal cord. Through this and subsequent corroborative laboratory findings, polio provocation migrated from a clinical theory to a clinical model. However, the growing acceptance of polio provocation posed challenges for health professionals and aid agencies. In western and developed countries, which largely controlled epidemic polio through immunisation programmes that preserved herd immunity, substantiation of the theory did not necessitate health policy changes. By contrast, in nations where polio was endemic, immunisation priorities and sequence mattered. Changing characterisations of risk, ascribed to the power of evidence grounded in laboratory research and the expectation of increased longevity, placed pressure on policy makers to review the balance of risks. In particular, evidence suggested that polio vaccination needed to be undertaken a priori of other paediatric immunisations in order to reduce the

¹⁰⁹J. Singh, S. Khare, R.S. Sharma, and T. Verghese, 'An Explosive Outbreak of Poliomyelitis in an Orphanage in Delhi: Risk Factors for the Unusually High Attack Rates', *Indian Pediatrics*, 34, 1997, 135–9.

¹¹⁰J. Singh, S. Khare, R. S. Sharma, and T. Verghese, 'Reply', *Indian Pediatrics* 1997, 847–8.

¹¹¹H. V. Wyatt, S. Mahadevan, and S. Srinivasan, 'Unnecessary Injections and Paralytic Poliomyelitis in India', *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 86, 1992, 546–9.

¹¹²H. V. Wyatt, 'A View From The West: Health Warning: Injections Can Endanger Health', Issues in Medical Ethics, 4, 1, 1996, 14–15.

¹¹³ Ganapati Mudur, 'Flawed Immunisation Policies in India Led to Polio Paralysis', *British Medical Journal*, April 1998, 316.

¹¹⁴Matthias Gromeier and Eckard Wimmer, 'Mechanism of Injury-Provoked Poliomyelitis', *Journal of Virology*, 72, 6, June 1998, 5056–60.

chance of provoking polio. ¹¹⁵ Public health officials, researchers and international charities remain engaged with this issue as they negotiate its implications and reassess immunization practices. ¹¹⁶

Conclusions

The contentious history of polio provocation shows the challenges that health professionals faced when reacting to evidence of possible adverse reactions. Knowledge of polio provocation failed to penetrate American society in the 1920s, but by the 1950s it had become a minor cause célèbre. Although most doctors and public health officials believed certain paediatric injections could provoke polio, a failure to unravel the epidemiological mechanism allowed causation theories to spiral and divisions to congeal. For some health professionals, polio provocation was a fact, justifying injection bans and the consideration of seasonal epidemic factors; for others, it was a misguided hypothesis that risked regressive public health practices and the loss of herd immunity. Both sides believed that their respective positions reflected maximum public health benefit. For a few medical researchers, such as Hammon, knowledge derived from human experimentation promised the greatest advantage. He reasoned that the risk of provoking polio during his pilot study was acceptable on the grounds that generating knowledge about the value of GG would benefit the most people.

Despite fear and a lack of clarity surrounding polio provocation, most American health professionals at mid-century reached a compromise in which immunisation practices were reformed to account for the risk. For such thought leaders, the temporary reduction in herd immunity appeared less dangerous than the possibility of inciting polio. With the introduction of the Salk and Sabin vaccines, traditional public health practices resumed and polio provocation slipped into clinical dormancy. However, the 1980s renaissance showed the human costs when disagreements over clinical evidence and uncertainty about causation prevail. Although discovery of the epidemiological mechanism behind polio provocation in the 1990s established a clinical model, such findings did not resolve the issue. Instead, the politics of international aid and transnational public health posed new challenges. Balancing risks for polio provocation became more than an effort to save lives and reduce suffering, but a negotiation between how health professionals interpreted knowledge with how they chose to act on it.

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Sailakshmi Ganesan, U. V. Shenoy, and E. Narayanan, 'Provocative Poliomyelitis Causing Postpolio Residual Paralysis Among Select Communities of Two Remote Villages of North Karnataka in India: A Community Survey', *Indian Journal of Public Health*, 55, 4, 2011, 309–12.

¹¹⁵A. R. Bosley, G. Speirs, and N. I. Markham, 'Provocation Poliomyelitis: Vaccine Associated Paralytic Poliomyelitis Related to a Rectal Abscess in an Infant', Journal of Infection, 47, 1, 2003, 82–4.

¹¹⁶Neal A. Halsey, 'Commentary: Poliomyelitis and Unnecessary Injections', *International Journal of Epi*demiology, 32, 2, 2003, 278–9; Amitesh Narayan,

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