A History of Poliomyelitis in New Zealand

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Jean C Ross

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PREFACE

I have endeavoured in this work to trace the course of polio in New Zealand, covering the social impact of the epidemics as well as the medical responses. Other than the annual reports of the Health Department and a few articles in the *NZ Medical Journal*, very little material exists in published form. I have made extensive use of the Health Department records held by National Archives. Unfortunately those in Wellington are poorly indexed, some files are missing and in many cases material is either duplicated or has not been kept.

I am indebted to many people. First of all to the members of the Immunisation Awareness Society whose comments made me realise how quickly we forget; to Geoffrey Rice, my supervisor at Canterbury University for his patience and guidance, and to Philippa Mein Smith, also of Canterbury University for her most helpful comments; to Denis Hogan of the Post-polio Society for his encouragement and enthusiasm; to the staff at National Archives in Wellington, Auckland and Christchurch, and at CCS headquarters in Wellington for their help in searching the archives; to the staff at the library at Christchurch School of Medicine and at Radio New Zealand Archives in Timaru. My especial thanks to my husband Keith, without whose help I would still be struggling with the word processor. I would also like to thank all those who wrote me letters or allowed themselves to be interviewed, particularly the members of the Post-polio Support Society in Christchurch. While little of these interviews appears directly in the text, they were invaluable for giving me understanding and insight, particularly as I searched the official records.

Finally I would like to dedicate this work to the memory of my father, Jim Ford, who was a polio statistic in 1921.

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ABBREVIATIONS

AHB	Auckland Hospital Board
AJHR	*Appendices to the Journals of the House of Representatives*
BMA	British Medical Association
CCS	Crippled Children's Society
CSL	Commonwealth Serum Laboratories
D-GH	Director-General of Health
MOH	Medical Officer/s of Health
n.d.	no date
NFIP	National Foundation for Infantile Paralysis
NZMJ	*New Zealand Medical Journal*
PPA	Protestant Political Association
PPS	Post-polio survey
up.	unpublished
WHO	World Health Organisation

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INTRODUCTION

Poliomyelitis[1] as an epidemic disease passed through the Western world like some great comet. Recognised only sporadically before 1900, epidemics of polio appeared in Western communities with increasing frequency and intensity in the first half of the twentieth century. Thousands died, many more were paralysed for life. Yet by 1960 the disease was no longer feared and indeed, within a decade, was all but forgotten except by those whose lives had been directly affected. So completely had the effects of this devastating illness passed from the collective memory, that by 1980 parents had to be urged and cajoled into having their infants immunised.

Little known or recognized before the twentieth century, polio has had a brief but spectacular history. It was the subject of a crusade which became "one of the greatest technical and humanistic triumphs of the age." [2] The story of polio is full of paradoxes. It was believed in the nineteenth century to be a recent manifestation, yet there is evidence of its appearance in antiquity. Known for many years as 'infantile paralysis' it was not confined to infants and was rarely paralytic. When it was paralytic, it caused the greatest morbidity and mortality amongst adults. Unlike the great scourges like typhoid, cholera or tuberculosis, epidemics of polio increased with improved hygiene and nutrition. Polio was for long considered a disease of the nervous system, but the causative agent in fact proved to be the first discovered of a huge group of entero-viruses - viruses affecting the gastro-intestinal system. Initially thought to be a rare affliction, it finally became apparent that almost all the population had suffered the disease at some time. The resultant effects of paralysis and contraction were the subject of heroic orthopaedic treatment, yet the most successful treatment was that devised by an untrained, unqualified 'bush nurse' from Australia. For many years an epidemic, or a threatened epidemic, could disrupt the day to day functioning of an entire country, yet it was later proved that the public health measures taken were quite ineffective. The fear it generated was partly because it was so capricious. It seemed to be the healthy and the strong who were struck down. As a noted authority wrote in 1940, "An attack of polio may be as inconsequential as measles or more agonising than death." [3]

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1 In the remainder of the text I shall use the shorter, and now more commonly used term 'polio' unless I am using a direct quotation.

2 J. R. Paul. *A History of Poliomyelitis.* Yale University Press, New Haven, 1971,p. 1. I have drawn extensively on Paul's work. It is a comprehensive and highly readable history by a man who played a major role in research on the poliovirus.

3 Philip Lewin ed. *Infantile Paralysis: Anterior Poliomyelitis.* W. B. Saunders and Co., Philadelphia, 1941, p. 3

New Zealand was as much affected as Australia, the United States or Scandinavia. An official report recalled that "epidemic poliomyelitis was the most terrifying epidemic condition in the country and the professional and public fear was justified as no specific measure of control was known." [4]

This study proposes to trace the history of polio in New Zealand - the course of the epidemics, its treatment, and the community's response.

[4] *Report to the Minister of Health of the Special Committee to Investigate the Safety of Poliomyelitis Vaccines.* Government Printer, Wellington, 1983, p. 1

CHAPTER ONE

POLIO - THE VIRUS AND THE DISEASE

Le microbe n'est rien, le terrain est tout.

Louis Pasteur.

> ***Poliomyelitis*** - A common acute viral disease characterised by fever, sore throat, headache and vomiting, often with stiffness of the neck and back. In the minor illness these may represent the only symptoms the patient has. The major illness, which may or may not be preceded by the minor illness, is characterised by involvement of the central nervous system, stiff neck, pleocytosis in the spinal fluid, and perhaps paralysis. There may be subsequent atrophy of groups of muscles ending in contraction and permanent deformity.[1]

So why did polio suddenly become the scourge of countries like Sweden, the United States of America and Australasia, at the very time when the great epidemic diseases were being brought under control? The answer lies in Pasteur's aphorism. The poliovirus is commonly found in almost all the inhabited parts of the world. The disease polio probably existed from prehistoric times. However improved sanitary and hygiene methods changed the epidemiology of both bacterial and viral enteric infections. Bacterial infections, like typhoid and gastro-enteritis, were markedly reduced but the opposite was the case with the viral enteric infections, which now began appearing as epidemics. Studies in developing countries have illustrated how this has come about. Polio results from exposure to the poliovirus. In unimmunised countries this usually happens in infancy. Where there is a very high infant mortality rate, the poliovirus, although present as an endemic disease, is 'hidden' by other infectious diseases. Polio is more or less in equilibrium with the population. The virus is continually present, some infants die or are paralysed but most acquire immunity through a subclinical infection. A high infant mortality rate is usually a reflection of the prevalence of endemic diarrhoeal diseases. It is not until the infant mortality rate drops below 75 per 1,000, that polio begins to manifest itself within a few years[2]. In New Zealand the infant mortality

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1 *Dorland's Illustrated Medical Dictionary.* W.B. Saunders Co, Philadelphia, 1957.

2 A. M. Payne. 'Poliomyelitis as a World Problem' in *Poliomyelitis: Papers and Discussions presented at the Third International Poliomyelitis Conference.* J.B. Lippincott and Co, Philadelphia, 1955, pp. 391 ff. See Appendix 2.

rate dropped below 75 per 1,000 around 1906, and epidemics of polio began to appear shortly after in 1914.

There are three major phases in the epidemiology of polio: endemic, epidemic, and post-vaccination. Where polio is endemic, virtually all children over four years of age are immune. The disease in this age group is usually mild, and rarely paralytic. As infant mortality drops with improving living standards, more children survive infancy without having been exposed to the virus and so becoming immune. Studies in Egypt and the Philippines have shown an incidence of polio comparable to the United States in the first half of the twentieth century - with an annual incidence of paralytic polio of about 28 per 10,000.[3] As more and more people encounter poliovirus for the first time in their late childhood or adulthood, epidemics increase in frequency and the incidence of paralysis becomes more common. The widespread use of vaccination, beginning in the mid 1950's, dramatically stopped the course of epidemics. However the prevention of epidemics depends on there being a large pool of immune people. As memories of the great epidemics fade, people become more casual about immunisation. If the level of immunity drops too far then the ever present poliovirus will reappear as polio.

The virus causing polio was first isolated in 1908. However the difficulties in growing viruses - viruses depend on living and multiplying cells to grow - meant it was a long and difficult process to replicate the disease. In 1911 the poliovirus was found during post-mortems in non-nervous tissue. The finding of the virus in the tonsils and the membranous linings of the throat, as well as in nasal secretions and salivary glands, gave weight to the nasopharyngeal portal of entry theory, that is, that the virus entered and left the body through the nerve fibres at the top of the nasal cavity. Findings of poliovirus in faecal samples initially prompted theories that polio was a waterborne disease like typhoid. But the typhoid bacillus multiplies if given the proper temperature and the availability of protein and other organic material. Such conditions of course do not affect the multiplication of viruses.

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[3] A.S. Evans, ed. *Viral Infections of Humans,* Plenum Medical Books, New York, 1982, p. 212

Around 1920 a number of severe viral diseases appeared, some old, some new. Besides the upsurge in polio epidemics of increasing frequency and severity, the 'Spanish flu' had spread its devastation throughout the world, and in Japan, outbreaks of Japanese Encephalitis occurred in 1924. Australian X disease appeared in that country in 1917, 1922 and 1926, with 50% of those affected being less than five years old. Many cases of 'lethargic encephalitis' were diagnosed in the decade between 1917 and 1927. There was a feeling among some medical researchers that perhaps new super viruses were developing, or that the human race was becoming more susceptible to central nervous system infections. More likely these were a sequelae of the 1918 Spanish Flu.[4]

In 1931, Frank Macfarlane Burnet and Jean McNamara made a discovery that, while slow to gain acceptance, eventually changed the thinking about virological and immunological disease. They found that the poliovirus was in fact three serotypes, each with its own special autogenic and biological characteristics. This discovery opened the way to the development of an effective vaccine. The discovery of the three serotypes also explained the occurrence (although rare) of attacks of polio on people who had previously had the disease. These second attacks are usually due to Type I or Type 3. It seems that Type I gives resistance to Type 2 and vice versa.[5] Exposure to a previously unencountered serotype is a possible explanation for an outbreak of polio amongst New Zealand soldiers in the Middle East in 1941 - 42.[6]

Poliovirus was the first of the entero-viruses to be discovered. It is a single stranded RNA virus which enters the body through the mouth. The primary site of multiplication is the lymphoid tissue of the alimentary tract, including the pharynx. Once lodged here the virus then diffuses outwards into the bloodstream and from there into other tissues and organs. The virus also spreads from the lumen of the gut and is excreted via the faeces. The most important aspect of enteroviral infection is the capacity of the virus to

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4 C.H. Andrewes. *The Natural History of Viruses.* Weidenfeld and Nicholson, London, 1967, pp. 90 ff. See also R.T. Ravenholt and W.H. Foege. '1919 Influenza, Encephalitis Lethargica, Parkinsonism', *The Lancet*, 16 October 1982, pp.860 - 4.

5 A.B. Christie. *Infectious Diseases: Epidemiology and Clinical Practice*, 3rd Edition, Churchill, Livingstone, Edinburgh, 1980, p. 572

6 J.E. Caughey and W.M. Porteous. 'An Epidemic of Poliomyelitis Occurring Among Troops in the Middle East.' in *Medical Journal of Australia*, 1 January 1946, pp. 5 - 10

spread to the central nervous system, and the subsequent potential for causing neurological disease.[7]

The poliovirus, like the other enteroviruses, is resistant to all known antibiotics or chemotherapy agents. Not only are laboratory disinfectants like alcohol, lysol and quaternary ammonium compounds ineffective, but formaldehyde and chlorination are not able to deactivate the virus under normal conditions. The virus is viable at room temperature for days, and for many years at freezing point. Ironically, in view of the widespread belief that somehow one caught polio through sunlight, the virus is killed by ultraviolet light, and temperatures of 50°C. Poliovirus is most commonly spread by personal contact with an infected person, usually by the oral route. The virus becomes established in the alimentary tract in the lymphatic tissue. In the mouth and throat the virus infects the tonsils, cervical nodes and the throat. These sites are the body's primary lines of defence and in fact most infections stop at this point with no apparent damage or symptoms. It has been estimated that only one per cent of poliovirus infections show clinical signs.[8] If the virus penetrates the gastro-intestinal tract, into the bloodstream, then the more serious disease results. Peripheral trauma can interfere with the resistance of the central nervous system, especially of that part with the closest connection with the site of injury. This was shown by the number of cases of bulbar (brainstem) paralysis which occurred in children on whom tonsillectomy had been recently performed. The still unhealed tissue proved to be an ideal site for the virus to penetrate the body's defences. Similarly inoculations during an epidemic could lead to paralysis of the limb injected. The connection of paralysis with vigorous exercise is less obvious, but the anecdotal evidence was so strong that caution about vigorous physical exercise following any minor 'flu-like' illness was advised.[9]

Following infection, initial symptoms like a cold or upset stomach, sometimes accompanied by fever, malaise, headache, nausea, vomiting, sore throat, or combinations of these symptoms, appear. Unless the virus is isolated in the laboratory or antibodies

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7 M.C. Timbury. *Notes on Medical Virology,* Churchill Livingstone, Edinburgh, 1983, pp. 49 ff

8 Evans, *op.cit.* p. 220.

9 W. Ritchie Russell, 'The Management of Acute Poliomyelitis' in *Poliomyelitis*, Robert Debre et al., WHO, Geneva, 1955, p. 138

measured, there is nothing to suspect polio rather than numerous other minor complaints, unless polio is suspected or known to be in the area.[10] In the pre-paralytic stage there is no single or specific diagnostic sign. Diagnosis of the minor illness is very difficult except in retrospect. This fact accounts for the apparently minor incidence of polio in non-epidemic years. Rarely these symptoms are followed by stiffness and pain in the back and neck, fretfulness and irritability lasting from two to ten days. Usually recovery is rapid and complete. These symptoms can be caused by other enteroviruses, however paralysis is rare in other enteroviral diseases. Sometimes paralysis follows the symptoms described above but quite often paralysis appears suddenly. Frequently this is biphasic or 'dromedary' - a minor 'cold' or 'flu' is followed by a few symptomless days, then the patient is afflicted with sudden severe paralysis. The main sign is flaccid paralysis caused by lower motor neuron damage. Incoordination, because of infection of the brainstem, and spasms of non-paralysed muscles can occur. The amount of damage to the central nervous system and muscles varies greatly. The muscles are usually affected to their maximum extent within a few days of the onset of paralysis with maximum recovery of the muscle usually in the first six months.

The muscle becomes paralysed when the nerve supply is cut off. The affected muscles are usually flaccid with the loss of the deep tendon reflexes. However there is no sensory loss. If the muscle is no longer used, it begins to atrophy. There may also be temporary paralysis caused by pain and muscle spasm. The leg muscles are the most frequently affected, as are the abdominals but any group of muscles can be involved. The paralysis can vary from slight weakness to complete loss of power, with nearly every skeletal muscle involved.[11] If the respiratory muscles are involved, breathing becomes difficult. The outcome of this paralysis was frequently fatal before the advent of respirators; and the bulbar form in which the soft palate and pharynx were paralysed, preventing the patient from clearing secretions from the throat, was almost always fatal especially when treated in a respirator. Bulbar and respiratory paralysis were frequently seen together

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[10] *ibid.*

[11] Christie, *op. cit.*, p. 579

and the mortality rate did not improve until better respirator and tracheotomy techniques were developed in the 1950's.[12]

A pathological examination shows the diffuse and usually extensive involvement of the brain and spinal cord. An initial inflammatory reaction is found throughout the brain and both the anterior and posterior horns of the spinal cord, giving rise to the meningitis-like symptoms.[13] However the virus seems to attack particularly the motor nerve cells of the anterior horn of the spinal cord, especially in the lumbar and cervical regions. Motor neurons in the thoracic area and medulla are also frequently affected. Destruction of the cells is not even, with healthy nerve cells being found in the same region as dead cells. Once motor nerve cells are destroyed they can not regenerate, giving rise to permanent paralysis. However any nerve cells in the region which remain vital, can, under appropriate conditions, continue to activate the muscle.

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12 A 740 537 62/3, 14 October 1955. See also W.S. Robertson, 'A Review of the Cases of Polioencephalitis in the Wellington Hospital' in *NZMJ*, Vol XXIV, No.123, 1925, pp. 237 ff

13 Lewin, *op. cit.* p. 59

CHAPTER TWO
WORLD HISTORY

Polio probably existed in prehistoric times, but one of the earliest records we have is an Egyptian wall painting c.1580 - 1530 B.C., which shows a priest with the characteristic atrophied leg held in the equinus position.[1] Hippocrates describes a paralysis of the legs which affected many in the late summer and early autumn on the Island of Helos. People were suddenly attacked by a kind or paraplegia and some died suddenly.[2] From then on no firm evidence of polio has been uncovered before the eighteenth century. Given the high rate of infant mortality and what we now know about the epidemiology of the disease, this is to be expected. In the eighteenth century, Dr Underwood of Philadelphia wrote of a cluster of cases of paralysis occurring in children after a brief illness, but the first clear account of an attack comes from Sir Walter Scott. He describes how, in 1772, as a healthy young child, he suffered an attack of fever which left him lame. This he blamed on "the paralysis which sometimes follows a fever and occasionally accompanies the cutting of teeth."[3] Teething was held to be the cause of polio, (as it has been for a myriad of other complaints), for at least another century. One reason for this is that up until at least the latter part of the nineteenth century the medical profession gave little importance to accurate diagnosis. Unless fevers were accompanied by a definitive rash or pustules like scarlet fever and smallpox, they were simply described as 'short', 'intermittent' or 'continued'. Similarly physicians defined paralysis by the part affected, rather than by the cause of the paralysis. One exception was a pathologist, Dr Abercrombie, who in 1828 noted cases of paralysis where there was no loss of sensory perception, but only muscle involvement: that is only the anterior part of the spinal cord was affected.[4]

In 1835 Dr John Badham described what was almost certainly polio occurring in a group of children in a confined neighbourhood near Sheffield. Jacob von Heine, an orthopaedic surgeon investigating paralysis among children in a German village in 1840,

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1 Lewin. *op. cit.*, frontispiece. See Appendix 1, page 107.

2 Lewin, *ibid*

3 Paul, *op. cit.*, pp. 17 ff

4 Paul, *op. cit.*, p. 8

found that the paralysis was frequently preceded by a febrile illness.[5] Interestingly his prescribed treatment excluded the standard purges, blisters and emetics and instead advocated rest with the affected limbs being wrapped in warm cloth.[6] It was the French, who, in describing a 'debility in the lower limbs' in children, coined the term 'infantile paralysis'. Undoubtedly paralytic polio was affecting children in many countries but was either unrecognised at all, or was simply described as 'acquired club-foot' or something similar. For instance there was an epidemic of 'strokes' in children in Greymouth, New Zealand, in 1880. Eleven children were affected in the space of a few weeks. The epidemic was not noted at the time and it was not until 50 years later that an observant general practitioner diagnosed 'the strokes' accurately.[7]

The second third of the nineteenth century saw the rise of a new type of medicine, stimulated by improvements in microscopes and advances in physiology and pathology. The development of the 'germ theory' by Pasteur and Koch, and the development of etiology, saw a shift in emphasis from treatment to causation. 'Germ theory' also led to great attention being paid to finding individual contacts or vectors of the disease, especially since the discovery of the role of insects in the transmission of malaria and yellow fever. The search for a 'single cause' for a disease became almost an obsession, and it was a long time before it was realised that susceptibility varied greatly with the individual. The place of immunity in the spread of disease was not appreciated until much later. In 1884, Medin gave the first description of a large epidemic of polio, in Sweden. He described the acute stage of the disease, clearly demonstrating the systemic phase which preceded paralysis. Prior to this, the disease was regarded primarily as a spontaneous degeneration and atrophy of anterior horn cells of the spinal cord. It was Medin's systematic observations which led to recognition of groups of cases which suggested that polio was in fact an infectious disease.[8]

The first substantial epidemic in the United States, with 132 cases, occurred in Vermont in 1894, while 1,031 children were affected in another major outbreak in Sweden in 1905.

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5 George Draper, *Acute Poliomyelitis,* P.B. Blakiston and Sons, Philadelphia, 1917, p. 1

6 Paul, *op. cit.* p. 8

7 F. Bennett, *A Canterbury Tale*, Oxford University Press, Wellington, 1980, p. 153

8 Lewin, *op. cit.,* p. 4

It was notable that in both epidemics most of the cases occurred in rural areas. It was recognised for the first time in 1905 that non-paralytic or 'abortive' cases were equal in number to, or greater than, the paralytic cases. Wickham, the researcher involved, realised the implications of this, in the spread of the disease. He asserted that polio was not entirely or even chiefly, a disease of the central nervous system. However he met with considerable opposition from his fellow doctors for bringing down a diagnosis of 'infantile paralysis' in the absence of paralysis.[9]

No etiological agent had yet been proved but the view was now widely held that polio was in fact an infectious disease. A description of the disease given in 1908 sums up the state of knowledge at that time:

> Polio is a peculiar gastro-intestinal infection, possibly the result of a specific infection, perhaps ... due to non-specific, miscellaneous, bacterial interrelation with poor nutritional conditions, under abnormal climatic surroundings resulting in the formation and absorbtion of poisonous substances which attack the central nervous system diffusely, the exact clinical results depending upon the concentration and effects of the poison at various points.[10]

1908 was in fact a landmark in the history of the disease. In this year the presence of the polio virus was demonstrated by the immunologist Karl Landsteiner in Vienna, although not the fact that there were three viral types, not one.[11] Unfortunately the discovery of the causative agent led researchers away from clinical epidemiology, itself an infant science. Instead of following up epidemics as they occurred, researchers emphasized the neurotropic aspects of the virus and the lesions it produced in the central nervous system. Investigators concentrated on experimental pathology in monkeys, the only mammal in which the disease could be induced. Both the expense of such research, and the distortions produced by it, severely handicapped the search for control of the disease.[12] For example, following a large epidemic in Sweden in 1911,

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9 Paul, *op. cit.*, p. 91

10 quoted in Paul, *op. cit.*, p. 139

11 R.E. McGrew, *Encyclopaedia of Medical History*, Macmillan, London, 1985, p. 274. The modern science of virology was still in its infancy. E.g. the influenza virus was not isolated until 1933.

12 Paul, *op. cit.*, p. 97

Swedish doctors presented a paper in Washington, in which the observations, had they been followed up sooner, may have hastened the discovery of control methods. The Swedes put forward the theory that immunity was acquired during subclinical infections during epidemics, and that this was the basis for the age incidence of clinical infections. Older children and adults had acquired resistance by being infected in previous epidemics, usually in a form which could not be diagnosed as polio. They had isolated poliovirus from patients for up to seven months after the original infection. This of course had considerable implications for the use of quarantine as a means of control. Further, poliovirus had been isolated from faecal and intestinal specimens, but because popular support for the theory that the nose was the point of entry of the virus (directly leading to the central nervous system) was so overwhelming, this evidence was ignored. Also, unfortunately, laboratory experiments which claimed that the stable fly was responsible for transmission obscured the Swedish study.[13]

The nasal mucosa had been the first site suggested for the penetration of the poliovirus into the body, and by 1917 this idea was firmly entrenched, as it was to remain for the next twenty-five years.[14] In this decade the correct concept of the disease was starting to take shape: the causative agent was a virus and it was an infectious or contagious disease with the potential to cause epidemics. But clinicians were no closer to being able to treat or alleviate the disease in its major form.

One worker who did pursue an epidemiological path was W.H. Frost. Investigating epidemics in the United States in 1910, 1911 and 1912, he used clinical observations, statistical analysis and laboratory experiments with sera. He used maps to plot the incidence of paralytic, abortive and suspicious cases as well as linking these with the known date of onset. Seasonal and meteorological charts were collected and extensive investigations were made into the sanitary conditions of affected persons premises and the sources of their milk and food supplies. His data confirmed that the disease spread through personal contact.

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[13] Paul, *op. cit.*, pp. 132 ff

[14] F.M. Burnet, *Virus as Organism*, Harvard University Press, Cambridge, 1946, p. 59

Unfortunately observations of a coincidental paralysis in domestic animals led Frost to conclude that the route of infection was through contact, through insects or through inhaled dust.[15] This lead to experiments which purported to show that polio was an insect borne disease, transmitted by the common stable fly. The emphasis on the relationship between filth (and by implication poverty) and disease was persisted with in the United States despite the evidence that polio did not conform to this pattern.[16] Insect vectors provided an ideal link between environmental sources of infection and the seemingly random appearance of polio in apparently sanitary surroundings.[17] Frost's work was known in New Zealand as the excellent report on the 1916 epidemic in the Wellington health district by Sydney Smith shows, but Smith was by no means convinced that Frost was right with his insect vector theory [see below, Chapter 3].

1916 also saw a major epidemic occur in the United States. The dramatic quarantine imposed had echoes in New Zealand for over thirty years. The premises of victims were placarded and the windows screened. The families of patients were quarantined within the house while family pets were kept out. There was ban on travel out of the epidemic area, especially by children under sixteen, for two and a half months.[18] Most authorities believed at this stage that polio was a respiratory disease with entry to the central nervous system via the nasopharynx. The unresolved question was why polio should be a disease of the late summer and autumn, when all other respiratory diseases occurred in winter.[19] Draper's textbook, published at this time and available in New Zealand, was the first to review the various clinical features of the disease.[20] For the first time simple laboratory tests for the acute disease were described in a way that general practitioners could replicate. The nineteenth century had seen polio as a wholly paralytic disease but by the time Draper wrote it had become generally accepted that polio was a common, contagious, viral infection, occurring in epidemics. It was appreciated that it was a disease not confined to infants but also occurring in children and occasionally in young

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15 Paul, *op. cit.*, p. 132

16 Naomi Rogers, 'Dirt, Flies and Immigrants: Explaining the Epidemiology of Poliomyelitis, 1910 - 1916 *Journal of the History of medicine and Allied Sciences*, Volume 44, p. 488

17 *ibid*, p. 493

18 H.F. Dowling, *Fighting Infection: Conquests of the 20th Century*, Harvard University Press, Cambridge, Mass., 1977, p. 205

19 Draper. *op. cit.*, p. 6

20 Draper. *op. cit.*, pp. 40 ff

adults. But just how it occurred and how it could be controlled was not resolved for four decades. Meanwhile, along with the rest of the Western world, New Zealand suffered a series of epidemics of increasing frequency and severity.

CHAPTER THREE
1916: THE FIRST EPIDEMIC IN NEW ZEALAND

In New Zealand the first reported case of infantile paralysis was that of a six month old child in 1887.[1] A report of a meeting held in November 1890, where Dr Pairman of Lyttelton exhibited a case of Infantile Paralysis and gave a short history of its progress and treatment, appears in the *New Zealand Medical Journal* for 1890,[2] and another case-history is given in 1893.[3] A letter from Dr Robert Fulton appeared in 1896 asking that "any member of the profession who has seen a case of this very unusual and extraordinary disease [infantile paralysis]," to send him the case notes, adding that he had notes of cases from Christchurch, Dunedin, Waimate, Milton, Lawrence, Tapanui, Outram and Invercargill.[4] The Director-General of Health, Dr T.H. Valintine, writing in 1923 said that "most of us can clearly recall schoolboys of our acquaintance who were affected by the ravages of Infantile Paralysis." [5] Valintine's schooldays were in the 1870's. Polio was not made a notifiable disease in New Zealand until 1914. Before 1908 deaths from polio were included in the total of deaths from diseases of the spinal cord and the central nervous system. However a small outbreak was recorded in 1895 when eight cases were admitted to Wellington Hospital, five of whom were children aged between 1 and 5 years of age. Between 1897 and 1900, seven children were admitted to Christchurch Hospital while in 1901, 'Landry's Paralysis' affected four children in Taranaki.[6]

New Zealand's first major polio outbreak occurred in 1914, when it became a notifiable disease, grouped under the heading 'Poliomyelitis, Cerebro-spinal Meningitis etc.'. Information about the disease at this time is extremely meagre. Even the outbreak in 1914, in which 25 deaths were recorded, receives only passing mention in the Health Department report for that year. But all that was to soon change. As one doctor was

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1 J.E. Caughey, 'Perils and Prospects of Polio?' in *New Zealand Parent and Child,* May 1955, p. 11

2 'Canterbury Letter' in *NZMJ,* Volume IV, 1890-91, p. 121

3 'Intelligence', *NZMJ,* Volume VI, 1893, p. 311

4 Letter, *NZMJ,* Volume IX, 1896, p. 183

5 H1, 131/9 3710, 1918-1925

6 F.S. Maclean, *Challenge for Health: A History of Public Health in New Zealand,* Government Printer, Wellington, 1964, pp. 317 ff

quoted in the *Press*: "Infantile Paralysis is really a far more serious disease than many which are compulsorily notifiable." [7]

The 1914 epidemic had shown that New Zealand was not immune from the rapid increase in polio epidemics world-wide. In 1905 in Sweden 1,500 people were affected, in 1907 in New York city 2,500. Epidemics were occurring with greater frequency and intensity in Australia. The New Zealand Minster of Public Health wrote in 1916 of the 'familiarity' of the term Infantile Paralysis and its 'widely manifest ravages'.[8] 1916 was not a good year for public health in New Zealand. Major epidemics of measles and influenza had occurred at Trentham Military Camp, where large numbers of troops had been gathered together in crowded conditions, to be followed almost immediately by an outbreak of cerebro-spinal meningitis. The outbreak of polio was followed by epidemics of enteric fever in Auckland and Northland, scarlet fever in Auckland and Christchurch, and diphtheria in Napier and Hastings.[9]

The polio epidemic began with a few cases in Auckland in December 1915. These increased in January and by the end of the month the first cases had occurred further down the island in Wellington, New Plymouth and Dannevirke. Gisborne was worst affected in February with 24 cases, Hawera with 20 cases in February and March and Wellington 41 cases in March and April. In all 960 cases were reported between January and June; 884 in the North Island and 76 in the South Island. Of the 1,018 reported cases in 1916, the majority suffered paralysis to some degree and 123 patients died.[10]

In line with overseas practice, Dr Sydney Smith, in a very comprehensive report on the epidemic in Wellington, divided the types of disease into eight different categories, plus the abortive or non-paralytic case. The majority of cases (about 70%) were spinal

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7 *Press* 6 February 1914

8 S.A. Smith, *Infantile Paralysis in the Wellington Health District, New Zealand,* Government Printer, Wellington, 1916, Foreword

9 *AJHR,* 1916, H-31, p. 1

10 *ibid,* pp. 2 ff. All the statistics used in this study come from the tables printed in the annual reports of the Director-General of Health, *AJHR,* 1915 - 1961, H-31. These vary slightly from those given by Maclean, *op. cit.* Because the diagnosis of polio was so difficult in the absence of paralysis, notifications were often revised months later. Statistics for Maori cases were sometimes listed separately but because this was not consistent, and was by 1947 dropped completely, I have amalgamated these figures. The statistics are more valuable for showing trends rather than absolute figures. See Appendices 2, 3, and 4, pp. 108ff.

paralysis. Following the onset of the characteristic fever, paralysis developed within a few days of the first symptoms. The general symptoms of fever and pain subsided followed usually by the gradual recovery of all or most of the paralysed muscles. In 60% of all cases of paralysis the affected limbs were the legs, often with the involvement of the abdomen and back muscles. Respiratory paralysis was very rare in these cases.

The second category noted by Dr Smith was the progressive type which conformed to the well-known descriptions of Landry's Acute Ascending or Descending Paralysis. In these cases the onset of paralysis was not limited. Paralysis progressed, usually from the legs but occasionally from the arms, until finally the whole body was affected. The patient usually died in these cases as the respiratory or bulbar centres became involved. Smith also noted that these cases frequently seemed to have two stages - a characteristic shortly to be described by Draper as 'dromedary'.[11] A typical case was that of a 17 year old farmhand, who after working in the hot sun complained of headache, fever and weakness. He spent a couple of days in bed, then feeling better, resumed work. That evening the symptoms recurred and he was sent to bed. Within two days both legs were completely paralysed, followed by paralysis of the trunk and arm muscles. Nine days after the initial onset of illness and four days after his relapse, the boy died of respiratory paralysis.[12] Two features of this and similar cases had a strong influence on medical thinking. It became widely believed (and continued to be believed to be so even amongst the medical profession right up until the 1950's), that somehow polio was connected with the sun. Repeated mention is made in case histories of the onset of polio symptoms following exposure to strong sunlight. The second feature noted was the relapse after apparent recovery. Smith was convinced that too early resumption of work was responsible for this and felt that the high proportion of deaths amongst adults was due to their premature resumption of duties. He strongly recommended that during an epidemic that any adult with gastro-intestinal symptoms "be rigidly forced to keep to their bed for at least a week." [13]

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[11] Draper's zoological knowledge was not as good as his clinical skills. The dromedary camel has a single hump. It is the Bactrian camel which has two humps.

[12] Smith, *op. cit.*, p. 63

[13] Smith, *op. cit.* pp 42 - 43

Smith went on to categorize a fulminant type - in which the patient was suddenly stricken with complete paralysis of limbs and respiratory centre - death being inevitable within a few hours; bulbar paralysis (paralysis of the medulla affecting swallowing) which usually occurred in conjunction with paralysis of other parts of the body; cerebral and ataxic paralysis(lack of muscular co-ordination), both described in overseas literature but with only one or two victims in the 1916 epidemic. A few patients had a neuritic type (inflammation of the nerves) characterized by intense neuritic pain. Because there was no loss of sensation it was concluded that the source of pain was the central nervous system. The closeness of the early symptoms of some polio cases to that of cerebro-spinal meningitis gave rise to yet another category: the meningitic type, showing intense headache, vomiting, retraction of the head, pain in the back and rigidity of the spine. Only the later development of paralysis or paresis (partial paralysis) confirmed a diagnosis of polio.

Of great interest to the health authorities were the abortive cases. Only 24 cases of non-paralytic polio were notified but Smith reckoned that this was grossly understated. He observed that a disease affecting children causing malaise, fever, headache, pain in the back, neck or limbs and often gastro-intestinal symptoms, had been widespread throughout the districts where polio had occurred. Attributed to 'influenza', Smith noted that a definite case of polio often occurred in families suffering from this flu. He came to the conclusion (quite accurately) that polio could manifest itself in all degrees from no paresis at all to complete paralysis.[14]

To the health authorities the epidemic of 1916 raised a number of problems. The cause of polio was known - the virus had been isolated eight years previously - but the mystery was how the virus spread.[15] Medical officers reported on all homes where a case of polio occurred. The houses were checked for general cleanliness, food sources, and sanitary arrangements. Food supply, especially milk, was a prime suspect as a means of transmission, but no common factor could be found. There was no consistency amongst the sanitary conditions found. Cases occurred where water supply and drainage were

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[14] Smith, *op. cit.*, p. 51

[15] See above, page 13

excellent as did cases where the sanitary arrangements were 'less than satisfactory'. Smith realised that the higher proportion of cases in rural areas might not be because of their poorer drainage and water supply but because the higher density of population in towns led to a greater chance of townspeople becoming immune as a result of exposure to a mild form of the disease. But he also felt that the fatigue from heavy work in hot sunshine while harvesting could be predisposing rural people to infection.[16] The number of children who apparently became infected after visits to beaches and bathing gave rise to concern but the only conclusion (other than the effect of the sun already mentioned) that could be drawn was that children were more susceptible to overheating and chilling than adults.[17]

Of far greater interest was the possible effect of the weather. A correlation between the weather and the incidence of polio had been noted. Outbreaks seemed to peak about 14 days after unusually high temperatures were recorded in a district. This echoed the incidence of epidemics in the United States where the disease was at its peak at times of highest temperatures. Smith suggested four possible ways in which the weather could be a cause: an increase in mould growth, the presence of extra dust in hot dry weather, and the possible 'lowering of vitality' among children in hot weather. The possibility of a direct effect of excessive exposure to direct sunlight, on the tissues of the central nervous system was also suggested. Not only had a number of children succumbed to polio after bathing in the sea, and sunbathing, but quite specifically it had been noted that on several occasions it was children who did not wear sun-hats who were affected. The fourth possible influence of the weather was on the number of insects. Experimental work more or less eliminated the possibility of transmission by stable flies. Fleas were very prevalent and were a possible vector, however the most likely vector was the house fly. Smith observed that a large number of victims had gastro-intestinal symptoms and that flies were quite possibly implicated in the transmission of infected material.[18]

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16 *ibid*, p. 58

17 *ibid* pp. 45 ff

18 *ibid*, pp. 48 ff

After discussing and dismissing cases of paralysis in lower animals, Smith turned to the possibility of human carriers. To him this was the only reasonable explanation for the spread of the disease, and he noted the appearance of cases along the main railway lines and steamer routes - these of course were the principal means of transport at that time.

But despite the best efforts of Smith and others the epidemiology remained obscure. The Inspector-General of Health, T.H. Valintine whose own ten year old daughter had been left slightly lame by polio, summed up his department's observations. He wrote that Infantile Paralysis occurred in the hot weather, more in the country districts and affected "the better classes, i.e. those who have gardens - not in slums or crowded parts of cities." [19] Inspectors made special enquiries about the patient's exposure to dust and hay. The prevalence of flies was to be noted, patients isolated and premises disinfected. However he noted in his annual report of 1915 that "in the absence of much precise knowledge of the causes of the spread of this disease, one cannot derive much satisfaction from using these sorts of weapons to check its spread." [20] Some of the community had no doubt as to the source of the disease - in keeping with current anti-German sentiment German doctors or doctors with German names were blamed.[21] Another theory suggested that "mistaken methods of feeding and clothing young children" were to blame. Prevailing fashions meant that "except for shoes and short socks hosts of little girls ... are naked to well above the knees in summertime ... the sudden changes in our climate ... impose a severe tax upon the vitality of even completely clothed adults. The strain upon the constitutions of half-naked growing children may be fairly presumed to be very much greater." [22]

The 1914 epidemic had been confined to the South Island and the immunity thus conferred meant that the 1916 epidemic had its greatest incidence in the North Island. It was also the first time that comprehensive statistics were produced. In the Wellington district 339 cases were reported; 58% of these were male. At this time males outnumbered females in the population but a slight predominance of male over female

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19 H1, 131/9 B89/90 3709, 3 March 1916

20 *AJHR* 1915 H-31, pp. 12 ff

21 e.g. letter to T.H. Valintine, H1, 131/9 3709-11, 8 June 1916

22 *Daily News,* Carterton, 15 February 1916

cases was to be a feature of all subsequent epidemics. Another trend which was to be repeated subsequently was the ratio of urban to rural cases. In rural areas the incidence was nine cases per 1,000 of population. In urban areas this was only 1 : 1,000. The term Infantile Paralysis was earned by the large proportion of under fives who were affected. 59.5% of all cases were five years old or under with 21% of all cases were in the one to two year old age group, and 92% of all cases were under 20. However the term Infantile Paralysis was misleading. There were 46 deaths from polio in the Wellington health district. Ten people, or 40% of those affected, over 20 years died while 23 children or 8% of those affected under 10 died.[23] But it was not death which was most feared. The prevailing attitudes of society towards the physically less than perfect may be inferred from a comment by a doctor in 1914, who said that he would almost go as far to say that he would prefer a child stricken with Infantile Paralysis to die because even if it recovered it would be permanently disabled or crippled.[24]

The public health authorities were not quite sure about the disease they were dealing with. An apparently 'new' disease, the medical profession was only aware of sporadic cases up until 1914. Despite the experience of Sweden and New York, it was not until after 1914 that the authorities even made the disease notifiable. By 1916 district medical officers had to report on households where cases occurred and public health measures, closely based on those used in New York, were instituted. These involved isolation of suspected cases and six weeks strict isolation for confirmed cases, preferably in hospital. Thorough disinfection of all body discharges and soiled linen was to be carried out with Jeyes Fluid,[25] while crockery and cutlery used by the infected person required boiling for one hour. The house of the victim was also to be thoroughly disinfected. Child contacts of the victim were excluded from school and public gatherings for two weeks following disinfection while adults were prohibited from occupations which involved handling food or contact with children, like teaching in day school or Sunday school. Mothers however were permitted to carry out normal household duties. General measures were instituted to promote cleanliness and sanitation. Dust removal was given

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[23] Smith, *ibid*, p. 19

[24] *Press*, 6 February 1914

[25] The ubiquitous Jeyes fluid featured in all epidemics right up until the 1950's, when copious amounts were used for rinsing hands in school lavatories in 1952 - 53.

high priority as was the elimination of flies. Common drinking cups and toothbrushes were to be discouraged. These measures were applied to outbreaks of infectious disease in general rather than being specifically applicable to polio. Gargling and spraying of the nose and throat with solutions of potassium permanganate,menthol, chloramine T,[26] hydrogen peroxide, or zinc sulphate were advocated as a preventative measure although these procedures never received the enthusiastic advocacy of the public health authorities which they did in the United States. Such treatments were of course totally ineffective and sadly, there were cases of temporary and permanent damage done to the nasal passages and olfactory organs caused by over enthusiastic treatment.[27] Warnings against fondling dogs and cats were made. The general public were told to avoid associating with people from infected areas. Newspapers carried warnings that as the disease was carried by direct contact with infected patients or carried by dust, biting insects, house-flies, bugs or fleas, scrupulous cleanliness was essential. Great care was urged to avoid contamination of food or drink, particularly milk.[28] It was a rather hit or miss approach and, as has been mentioned above, the authorities were not wholly convinced that all or any of these precautions was warranted. But unlike other epidemic diseases, the incidence of polio was puzzling. The Medical Officer of Health in Auckland noted that dust and flies were a problem in few cases. The disease had occurred in good houses with good surroundings - the slum area of the city had escaped.[29] It was accepted that polio did not behave like other infectious diseases, and that there was little point in trying to source the infection to slum areas or bad sanitation.[30]

Little information on the treatment of paralysis caused by fever appears in the textbooks of the period. The initial treatment at the onset of the first symptoms was to place the patient in complete rest for at least a week. The Department of Health recommended that calomel and Urotropin be given. (Urotropin had been observed to partially inhibit the disease in apes.) Systematic massage with olive oil (salad or sweet) for 15 to 20 minutes night and morning, possibly for months, and the wrapping of cold limbs in warm

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[26] Sodiumparatoluenesulphochloramide, an commonly used disinfectant with the bactericidal actions and uses of chlorine.

[27] H1, 131/9 B89-90 3709, 16 November 1917

[28] *Auckland Star*, 23 February 1916

[29] Report of MOH, Auckland to D-GH, Wellington, 29 January 1916, H1, 131/9 B89-90 3709

[30] Cf the attitude in the United States at this time; see Rogers, *op.cit.*

clothing and absorbent cotton was the general recommendation. Many children were treated at home in this manner with parents often spending hours a day for months or even years, patiently massaging the affected limb.[31] The general use of splints and braces to help with walking and standing, and also to avoid muscle contraction, was rapidly gaining favour in the first decades of the twentieth century. In the convalescent stage electrical treatments were in vogue; electric current was passed through the affected muscle to stimulate its recovery. Galvanic current was applied to the muscle by placing an anode on the spine or upper part of the muscle while the cathode was passed over the length of the muscle. Contact was made and broken frequently 40 to 60 times a minute by a gentle stroking process. *The Dictionary of Treatment* claimed "excellent results" although the method had lost favour by the late 1920's and Paul considered the benefits to the patient were psychological.[32]

However massage was the most widely used therapy. Nurse Bevilaqua, with an assistant, Miss Gulley, was brought over from Australia by the Department of Health. She had trained under Dr McKenzie of Melbourne who in turn had modified the treatment of Dr Keith Jones of Liverpool. Jones, who was regarded as the foremost authority in this field, had developed a splinting and re-education regime for paralysed muscles. Nurse Bevilaqua was reported to have obtained "extraordinarily good results [and] many a child who might otherwise have been a cripple has been able to regain full use of its limbs." [33] Her main duty was to train 'especially chosen masseurs, masseuses and qualified nurses'. In Auckland, the hospital authorities offered patients massage according to Nurse Bevilaqua's methods at a cost of 2/6d per visit.[34] The 1916 epidemic in fact underlined the need for experienced masseurs and hastened the registration of masseurs and the Dunedin School of Massage, later to become the School of Physiotherapy. Later several nurses, already in England with the army nursing service, were sent at public expense on special courses to help victims of polio. One went to a course on electrical treatment

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31 e.g. P.E. Muskett, *Illustrated Medical Guide,* New Zealand Edition, William Brooks and Co Ltd, Wellington, 1903; Paul, *op. cit.,* p. 325

32 Paul, *op. cit.*, p. 336

33 H1, 131/9 B89-90 3709, 3 November 1917

34 Memo (undated), A740 537 62/3

while two others who already had massage certificates went to the School of Swedish Exercise in Hornchurch to learn remedial exercises.[35]

Not surprisingly given the 'newness' of the disease, a number of cases were not diagnosed as polio at the time of the acute illness, although the patients later spent time in Rotorua or Trentham hospitals for treatment of their deformities. The war also affected the number of patients hospitalised. Priority was given to wounded soldiers. A 14 month old baby, paralysed in both legs and in the right arm, was pushed in a pram by her mother from Herne Bay to Auckland Hospital, only to be turned away. She was treated at home by immobilisation, hot oil massages and soaks in seawater.[36]

It was this decade which saw the emergence of orthopaedic surgery. Following the experience gained in dealing with the horrific injuries of World War I soldiers, surgeons had gained the confidence to rectify problems in the civilian population. Colonel Wylie came to New Zealand at the end of World War I at the head of an orthopaedic unit which was transferred to this country. A second orthopaedic unit soon followed and members were dispersed around the country. Dr J. Leslie Will came to Christchurch where he headed up an orthopaedic unit and also the new physiotherapy department.[37] As the military needs lessened, many children left crippled in the 1916 epidemic returned to hospital for surgical treatment. In particular as soldiers were discharged from the military hospitals at Rotorua and Trentham, children took their places. For instance in 1921, King George V Hospital in Rotorua had 61 long stay children in its care, most victims of the 1916 epidemic. It was not always easy to get treatment there however. In 1920 it was reported that there were only 11 infantile paralysis patients in King George Hospital when 72 beds were available. Parents reported difficulties in getting admission for their children. No information was to be found in Auckland and application for consideration had to be made to Wellington.[38] One girl, aged three when affected in 1916, had her first physiotherapy to her affected legs five years later when she spent nine

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35 H1, 167/5 20987, 22 October 1919

36 Post-polio Survey #94

37 F. Bennett, *Hospital on the Avon,* North Canterbury Hospital Board, Christchurch, 1962, pp. 167-173

38 *Auckland Star,* 13 October 20

months at Trentham Hospital. There she also had surgery on her foot, ankle and knee.[39] Another baby girl whose parents sought help everywhere, was at first hospitalised then treated at home with hot packs and massage. After seven years in a waist-to-ankle caliper she had her ankle reset during a stay at Rotorua, and had an achilles tendon transfer 31 years later.[40] A Hawera girl, affected in her back and legs, had her first operation in 1918 and later had her good leg operated on at Rotorua, to stop it growing.[41] Typically the child first spent months in plaster moulds and splintage to correct the deformity. 'Psycho-therapeutic treatment' including baths, massage and electric treatment was given. Finally, if all else failed, reconstructive surgery was tried.[42] This was "often successful in restoring some movement." [43] For instance a paralysed deltoid muscle could be replaced by loosening the thoracic attachments of the unparalysed pectoralis major, turning the muscle through 90 degrees and attaching it to the acromion and adjoining end of the clavicle so that it functioned as a deltoid. Transplanting or stretching tendons were probably the most widely used surgical techniques.

By July 1916 the epidemic had petered out. Nobody was quite sure when to expect another epidemic, although it seemed certain that there would be another before long. Over 100 children had died. Many more were permanently disabled. It was this legacy more than anything which caused people to dread its reappearance.

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39 PPS #203

40 PPS #429

41 PPS #187

42 *Evening Post* 16 February 1921

43 *Dictionary of Treatment*, p. 672

CHAPTER FOUR
1924: AN ORGANISED RESPONSE

For the next 45 years there were always some cases of polio in New Zealand and at least one death from the disease, each year. Given the difficulty of diagnosis of mild cases, it is highly likely that the disease was greatly under-reported in non-epidemic years. Even the paralytic form caused difficulty especially in the early decades of this century. One writer noted that patients were admitted with suspected "sapraemia, brain abscess, lumbago, pyomeningitis, tubercular meningitis, rheumatism, osteomyelitis and appendicitis," [1] and were later found to have polio. It is reasonable to assume that the reverse was also true.

In 1921 there was a small epidemic, with a total of 267 cases, beginning in Auckland in January, and gradually spreading throughout both islands, reaching Christchurch in July and Southland in December. Like the 1916 epidemic, over half the patients were under five, and only three cases were over 20.[2] Late in November in 1924, there was a sudden outbreak of cases in Petone. By December there were 59 cases in Wellington. The numbers climbed rapidly to over 340 in February and over 360 in March. The next three months saw an equally rapid drop, to ten cases in July. The epidemic had spread throughout New Zealand, but the highest number of cases were in Taranaki, Wellington and Canterbury.[3]

By this time a lot more was known about the disease than in 1916. It was recognised that the epidemics involved many more people than those with serious symptoms. However it was not fully understood how this related to the meningitic or paralytic forms. It was believed that somehow the mild disease ran concurrently with the serious form, with symptoms usually the same as those found initially in the prodromal ('dromedary' - see page 17 above) cases of polio - that is "headache, vomiting, epigastric pain, drowsiness, stiffness of back of neck, constipation and foul breath." [4] These symptoms

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1 W.J. Robertson, 'Polioencephalitis in Wellington Hospital' in *NZMJ*. Vol. XXIV #123, October, 1925, p. 238

2 *AJHR*, 1921-22, H-31, pp. 9-10

3 Maclean, *op.cit.* p. 321

4 Report from D-GH to Dr George Draper, 19 December 1925 H1, 131/9/10a n.p.

lasted for between 24 and 72 hours. If the infection was present in the paralytic form, then it usually occurred three to four days after the onset of the initial symptoms, beginning with muscular weakness developing into paresis and complete paralysis over 12 to 48 hours. The degree of paralysis in the acute stage was almost always more than the eventual residual paralysis, which was evident after three or four weeks. It was observed that the triceps, quadriceps, deltoid, ankle and dorsiflexors were the most commonly affected muscle groups, but no muscle appeared immune.[5]

The Health Department wrote an extensive report on the 1924 epidemic for Dr George Draper, whose book, *Acute Poliomyelitis*, was the standard text of the time. In this book, Draper had made many observations on the physical characteristics of his patients: the theory that certain types of individuals were susceptible to certain diseases; that susceptibility to disease was physiologically and anatomically determined, was currently fashionable. Draper noted that "it was common experience that polio patients are large for their age, well-nourished, plump, and with faces of rounded form." [6] He noticed abnormal dentition in 50 - 60% of his New York patients, and linked this to the widely held earlier belief that infantile paralysis was somehow connected to teething.[7]

Similar observations were made in Wellington. Robertson noted that:

> A great similarity of type was shown in the early cases - the fair, flaxen-haired, blue-eyed children [were] predominant. The weedy child, the guttersnipe and the ragamuffin were conspicuous by their absence; all cases with few exceptions, being plump, well-fed, perfect specimens of child-hood.[8]

Robertson also noted the similar history of onset in many cases. Children had been to the beach, or played in the sun all day; adults had been working in the sun and had 'sun-stroke'. He added hyperthesia (abnormal sensitivity), photophobia (extreme sensitivity to light) and irritability to the symptoms mentioned above. He was convinced that "the

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5 Robertson, *op.cit.* p. 238

6 Draper, *op.cit.* pp. 6 ff. It should be noted that 'plump' was a term of approbation at this time !

7 *ibid*

8 Robertson, *op.cit.* p. 235

sun's rays have at least a predisposing effect on some patients." [9] In spite of requests to do so, the Director-General, Dr Valintine, was reluctant to make these symptoms widely known, although he was happy to publicise the vulnerable age-groups. He felt that to talk about symptoms would cause anyone with a slight fever to think that they had the disease, and that this would do more harm than good.[10] His advice was to keep children as isolated as possible, in the open air, and to avoid crowds and assemblies. Children should wear suitable headwear, pink or red for preference, extending well down the neck.[11]

It was believed that the disease was spread mainly by children, therefore the Health Department's policy was to prevent children congregating when infection was prevalent. Because it was also thought that there was an age immunity as well as an acquired immunity, adults, even contacts, were not isolated, unless they had had symptoms or were in contact with numbers of children. Very few adults appeared to contract the disease and the economic effect of isolation was considerable.[12] The belief that if a child could be protected from infection until 16 years of age, he would "in all probability entirely escape attack",[13] formed the basis for the first major attempt in New Zealand to control the movements of a large section of the population.

The action was taken under Section 76 of the Health Act, 1920. This gave powers to each district Medical Officer of Health to take special steps for the purpose of preventing the outbreak or spread of any infectious disease. It was normal practice in the control of infectious diseases to close specific schools and to impose quarantine on individuals. A patient with an infectious disease would be kept away from school for at least six weeks from the onset of the disease. Contacts of the patient were quarantined for 14 days after disinfection of the house, following the removal of the infected person.[14] But

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9 *ibid,* p. 236

10 Valintine, memo to MOH, H1, 131/9/11a 16 January 1925

11 *ibid*

12 Report to Draper.

13 *ibid.* This theory proved to be completely wrong. Any adult who did not attain immunity as a child was highly likely to be severely affected if they became infected as adults. This became very evident in the 1950's. However the hypothesis of adult immunity was strongly believed by many authorities well into the 1940's. eg see F.M. Burnet, *op cit.*, 1946, p. 67

14 Memorandum to School Medical Officers, A49 H-A 61 25/29 17 April 1925

the 1924 - 25 polio epidemic was the first time that the Health Department had imposed a nationwide quarantine. When the first polio cases occurred in Petone and Wellington in November and December 1924, strict quarantine was applied to 30 families in an attempt to stop the disease spreading, but to no avail.[15] In a proclamation made on 13 January 1925, children under 16 were prohibited from all theatres, music halls and schools (including Sunday schools). They were prohibited from attending the races, sports and recreation grounds, and any places of public assembly, including public picnics and cadet parades. Initially the prohibition applied to Wellington, Patea and Hawkes Bay districts[16] but three days later it was extended to all the North Island and children were prohibited from crossing Cook Strait.[17] By 27 January, the Order included Christchurch.[18] Valintine felt that "the one great precaution to be taken is that children are not to be allowed to congregate ... I feel it is absolutely essential that the schools should remain closed." [19]

Support was forthcoming. The Medical Officer in Whangarei wrote to the Director-General that "the public is so accustomed to the triumphs of preventative medicine as shown in the general sphere of infectious disease, that they find it hard to accept a situation where science has to admit defeat, and they demand action for no other reason than it is action and without counting the cost." [20] F. Truby King lent his considerable mana to the scheme: "What the public expects in all such matters is a clear-cut, definite, consistent and authoritative lead." [21] All schools were to remain closed for at least the first two weeks in February. At the end of January it was announced that this would apply to all South Island schools also. While there had not yet been any cases of polio in the South Island, it was felt that this would be a wise precaution.[22] Dental clinics were allowed to stay open as it was considered important that dental treatment should

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15 H1, 131/9/10, B 91, 24 April 1925

16 *Dominion* 13 January 1925

17 *N.Z.Herald.* 16 January 1925

18 H1, 131/9/12a, 27 January 1925

19 H1, 131/9/13a, 8 January 1935

20 MOH, Whangarei to D-GH. H1, 131/9/12a 23370 1925, 24 January 1925

21 Truby King to D-GH. H1, 131/9/10, 17 January 1925

22 H1, 131/9/13a B 91. 21 January 1925

not cease entirely; however children had to wait outside, and if it rained, they were sent home.[23]

The public did not accept the restrictions without some protest. While there seemed to be general acceptance for the principle of quarantine, there was considerable correspondence in the newspapers, and to the Health Department on its application. Initially anyway, there was little concern expressed about the restrictions on schooling, but many asked for exemptions for their children to attend race meetings, picnics and picture theatres. Bands were upset that their junior bandsmen could not attend contests, or cadets under 16 parade, although those over 16 could. Many asked that, even if secular schooling was suspended, religious schooling be allowed to continue. There were other anomalies too. Youths over 16 were permitted to attend 'artisan', part-time and night classes at technical institutes, but youths over 16 were not allowed into secondary schools.[24]

There were some exemptions made. Special permits could be obtained to allow children already away from home to rejoin their families if they could produce a certificate to say that they were free from disease. Picture theatres were allowed to employ one boy each to sell sweets and programmes, in recognition of the importance of the boys' wages in many poor families.[25] Later, choir boys and altar boys were allowed to take part in church services.[26]

There were others of course who took advantage of the situation. A prominent member of the Protestant Political Association,[27] Mr. J. Burrell, complained to the local newspaper that over 130 Roman Catholic children were attending Mass in Palmerston North each Sunday. He asked why, if state schools and protestant Sunday schools were closed, Roman Catholics were allowed to flout the law. It was Roman Catholic parents,

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23 Memo from D-GH to Dental Clinics. H1, 131/9/13a, 23 January 1925

24 *Star-Sun* 26 February 1925

25 Memo D-GH to MOH. H1,131/9/13a, 25 February 1925

26 H1, 131/9/11a, 16 April 1925.

27 A very active sectarian group, dedicated to fighting 'rum, romanism and rebellion.' - *Oxford History of New Zealand,* 1st Edition, W.H. Oliver ed., p. 215

he insinuated, who were responsible for the spreading of the scourge. The police took no further interest in this case when a senior sergeant, who had attended the Mass in question, said he was not aware of any children present.[28] But inspectors were forced to take action after Howard Elliott, secretary of the P.P.A. complained to the Minister of Health that officials were deliberately favouring the Church of Rome in the face of evidence of breaches of Section 76.[29] Priests in Wellington were fined £3.3s and costs of 13/- for allowing children to congregate while attending school to pick up homework. There were some other successful prosecutions over children attending church : a Rev. J.W. Smyth was convicted and discharged for allowing his 12 year old son to attend church, on condition that he did not permit it to happen again.[30] But the proposed prosecution of a priest in Riverton was dropped on the advice of the Crown Solicitor, who felt that under Section 76, churches were not places of public assembly in the same way that schools, Sunday schools, racecourses and recreation grounds were.[31]

The question was also raised as to whether a tangi held on private grounds constituted a public assembly. On this occasion the opinion was in the affirmative. The Crown Solicitor likened the situation to a public picnic which, while it might be held on private land, in fact constituted a public gathering.[32] The parents of children found at a tangi at Te Horo were fined 5/-, while those of children at a Maori wedding were fined £3.[33] Concern was expressed by a number of members of the public about conditions at Ratana Pa. The Health Department was urged to stop Maori gathering at Ratana or anywhere else until the epidemic had passed: "now is the time to for clearing up the encampment- the opportunity is an extremely good one." [34] A letter was sent to Mr. Ratana in February asking for his co-operation.[35] There were problems with concert parties - and Maori groups were told that no distinction could be made between children as performers and children as audience. The organisers of the annual regatta at

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28 *Manawatu Times*, 17 March 1925, 18 March 1925

29 Elliott to Minister of Health, H1, 131/9/11a, 20 April 1925

30 Report of Solicitor-General to D-GH, H1, 131/9/11a, 20 March 1925

31 ibid

32 D-GH to MOH, Auckland, A49 H-A 61 25/29

33 The average wage for a male tradesman in 1925 was about £4/17/-, *New Zealand Official 1990 Yearbook*, p. 378

34 MOH, Wanganui to D-GH, H1, 131/9/11a

35 H1, 131/9/12a, 12 February 1925

Ngaruawhia were asked by the Minister of Health, Maui Pomare, not to ask for exemptions for their children as the Department did not want to be put in the position of appearing "to favour one race ahead of another." [36]

Other cases were more straightforward. In New Plymouth, parents of children found at racecourses were fined £2, costs 7/-; at Opononi, £3.10s and costs 22s. At Rawene, the fine for allowing children in a picture theatre was £5, costs 11s. Following motor races at Muriwai beach 15 people were prosecuted for allowing their children to attend. Most parents were admonished and discharged - the Bench did not feel that their attendance at a wide open beach was a serious breach of the regulations.[37]

By the end of March, the epidemic was obviously on the wane, and pressure mounted on the Department to lift the restrictions. Churches and individuals were anxious that children could attend church at Easter, (Easter Sunday was on 12 April that year), and make their Easter Communion. Private schools pointed out the seriousness of their position. So far they had received no fees for the first term, but if they could re-open immediately after the Easter break, then fees could be collected.[38] Others were anxious to have restrictions lifted before Anzac Day. The authorities did not wish to lift the general quarantine until after Easter, in order to limit movement over the Easter holiday break.[39]

By the beginning of April, a limited reopening of schools was allowed but attendance was not compulsory.[40] This partial revocation caused problems too. All schools in central Auckland were open but in Devonport (which was in the North Auckland Health district), only secondary schools re-opened, yet pupils were travelling from Devonport to central city schools. Similar problems arose on the other side of the city, as all south Auckland schools remained closed. The Waipawa, Wairarapa Hospital district allowed all schools in its district to open, but other Wairarapa and East Coast schools were

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[36] H1, 131/9/11a, 9 March 1925

[37] Report to D-GH, H1, 131/9/1a, 20 March 1925

[38] H1, 131/9/13a, 9 April 1925

[39] Memo D-GH to MOH, H1, 131/9/12a, 8 April 1925

[40] Memo D-GH to MOH, H1, 131/9/13a B 91, 4 April 1925

closed. Restrictions remained in Canterbury, Westland, Otago and Southland.[41] This was despite the fact that some areas had had no cases of polio at all. With schools closed, "children have been roaming about scarcely knowing what to do with themselves," children were congregating in the streets, square and at the beach, "and this state of things is leading to the moral and mental deterioration of the children." [42] Finally, quarantine restrictions were lifted on almost all schools on 25 April, to allow children to attend Anzac Day celebrations.

Teachers were given comprehensive instructions to follow. All floors were to be swept with sawdust dampened with disinfectant. Slates and pencils were to be cleaned with a solution of Jeyes Fluid. Common towels and drinking mugs were to be discouraged. If mugs were shared they were to be kept immersed in a solution of Condy's crystals. Other property, like pencils, pens, plasticine and so on, were not to be used in common. Teachers were to make sure that children carefully observed rules of personal hygiene - regular cleaning of teeth, washing hands and gargling night and morning with a solution of Condy's crystals and salt. Any child who appeared 'out of sorts' should be sent home at once and no child from a house where there was illness should be permitted to attend school.[43]

But not all children wanted to come back to school anyway. Attendance had become compulsory from June 1st but teachers reported that a number of children had found jobs during the closure of schools and they, or their parents, were reluctant for them to give up their jobs and go back to school.[44]

For others quarantine meant real hardship. Quarantining of adults, especially those whose work brought them in close contact with many children, or the manufacture or sale of foodstuffs such as bread or milk, which was consumed in the state in which it was bought, was done "more to allay parental alarm than with the belief that such adults are

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41 H1, 131/9/13a #2, 4 April 1925

42 H1, 131/9/11a, 8 April 1925.

43 Health Department instructions to teachers. H1, 131/9/13a 1925

44 H1, 131/9/13a B 91 #1, 13 June 1925

a menace to others." [45] With such widespread quarantine many families were badly affected economically. Hospital boards, which were charged with administering charitable aid, would not accept responsibility for hardship caused by the action of the Minister of Health. Therefore the Minister decided, as 'an act of grace', to "afford relief to persons in poor financial circumstances who have been isolated by the Medical Officer of Health." Between £2.10s and £4 per week was to paid for no more than two weeks, according to the size of the family and other relevant circumstances. But, cautioned the Department, "claimants, unless they are poor, are to be discouraged; otherwise a very dangerous precedent will be established." [46] There were still many cases of hardship. A labourer with 13 children was placed in isolation. If the breadwinner was forbidden to work, the Health Department could provide some help. However as he was given permission to work, he was not a charge on the Department. But the Department's belief that it was extremely rare for adults to carry the disease was not widely shared, and the man in question could not find anyone willing to employ him, or to work with him.[47] In another case, a family with three children was due to move from Opotiki to Tauranga where the husband was to take up a new job. One of his children was admitted to hospital where he could expect to stay for at least six weeks. The family faced medical costs of nearly £50 and two weeks in isolation. If the child was nursed at home, the whole family would be in quarantine for six weeks. While the family was forced to stay in Opotiki they had no income, and once they did move, they would receive no income for the first month.[48]

A mild, non-paralytic case of polio could expect to spend three weeks in hospital. If they had no further fever or other symptoms for the second or third week, they could go home, to spend a further week in isolation there. If they showed any sign of paralysis then the minimum stay in hospital was six weeks. Fever was reduced with salicylates, such as antipyrine or salicylate of soda. Purges, usually with calomel, were routinely administered. One text in common use felt that heroic measures such as "drycupping, blistering the spine, applying leeches, thermocautery, spinal ice-bags etc." should be

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45 H1, 131/9/10 B 91, 22 April 1925

46 Memorandum from Minster of Health, A49 H-A 60 25/29, 2 April 1925

47 Letter from Wairau Hospital Board to D-GH, H1, 131/9/10, 17 February 1925

48 Letter to Minster of Health, A49 H-A 61 25/29, 8 April 1925

abandoned, but that absolute rest until all tenderness had disappeared, was essential.[49] Blood serum was widely advocated. Serum was prepared from the blood of convalescent patients and was administered intrathecally. This was generally felt to give good results but to be really successful needed to be given before paralysis appeared. The most important thrust of treatment was to immobilise the affected, or possibly affected, muscles, to prevent elongation of the affected muscle and shortening of the opposing muscle, to prevent deformity. To this end splinting was done regardless of the presence of weakness or paralysis. Plaster spinal beds were used for all cases under three or four years of age, or any cases of abdominal weakness. Older patients were nursed on firm mattresses with their legs encased in plaster bed boots to above the knees, with the feet at right angles. Sandbags were also used to 'steady the limbs' and cradles used to relieve pressure. Arms were supported with 90o shoulder abduction (away from the normal position) semi-prone, on a Thomas frame. The immediate splinting of patients in such a manner was felt to "limit the extent of on-coming paresis, hasten recovery of paretic muscle and prevent deformity ... the whole body supported in a truly physiological position." [50]

Dr. Robertson recommended a whole plaster bed "on a pipe framework supported on two boxes with a bedpan underneath [so that] disturbance to the patient [was] minimal." [51] For severe cases, adrenaline was given intrathecally, along with frequent lumbar punctures to relieve the meningeal-type headaches and restlessness. The efficacy of all these measures of course was very difficult to assess. It was not realised at the time that the percentage of cases to develop paralysis was very low, so that any treatment, especially if applied at the pre-paralytic stage, would appear to have a very high success rate.

Once the acute stage was over 'tentative re-educative measures' could be introduced and gradually developed as the muscles recovered. Massage and electricity were used on selected cases only, and then only after three months. The patient's bed was gradually raised until the patient was in a sitting position before the patient was allowed to get up.

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[49] *Dictionary of Treatment*, p. 669

[50] Robertson, *op. cit.* pp. 239 ff

[51] *ibid*

When the patient was ready for walking they were fitted with long or short calipers, or abduction frames.[52]

A fifteen year-old boy felt stiff after playing football. By morning he could not walk unless he was holding on to something. He had weakness of the triceps in one arm, and the deltoid of the other. On hospitalisation, he was completely splinted for three and a half months. When the splints were removed, his arms were recovering but there was a large amount of wastage of this legs.[53] He was fitted with calipers and discharged after six months. As he was 15 at the time, his 160 days in hospital at 9/- per day, cost his parents £72. Had he been younger the charge would have been 6/6d per day.[54] The dislocation was not just physical. Another child was three when she was admitted to Wellington Hospital. She stayed there for five years. When she was discharged she cried and cried because it was strange at home, and she wanted to go back to hospital. She was able to walk with boots and irons on both legs. Eight years later, when she was 16, she went back to hospital, where her left leg was operated on by Alexander Gillies. After a further six months in hospital she was able to walk without boots or irons. Later she had further operations on one leg.[55] Others did not stay in hospital for so long - a six year-old girl in Hawkes Bay was hospitalised for one week. Her mother took her home, as she was getting no treatment, and her mother could not afford the hospital fees. Instead she was kept immobilised at home for years.[56] A two year-old girl who had been ill for some days, and who had been treated by her doctor for gastric trouble, was admitted with suspected tubercular meningitis. After a day it was noted that the child was very irritable and resented handling. Paralysis of her right arm and leg developed, then progressed further. Four days after admission, she died.[57] Baths in saltwater, brought especially from the sea, were the only treatment another baby received at home, because the doctor 'didn't know what else to do',[58] while another was not diagnosed for

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52 *ibid*

53 A perfectly healthy person immobilised for three months would experience considerable muscle wastage, and it would take about another three months to regain normal function: interview, A.R. Bean, 1 October 1992.

54 CH374 C20 158h 1926-27 #184

55 PPS #369

56 PPS #115

57 CH324 C20 158G #5210

58 PPS #223

eight and a half years. He was then admitted to hospital with a weak left leg and marked scoliosis (curvature of the spine). He was put in a plaster cast around his trunk up to his arms and discharged. He remained in this plaster for 27 months. After another three months in hospital the plaster was removed to show the scoliosis still marked. He was put into a Taylor's brace for 18 months and had massage and exercise. The nurses commented that he was 'a difficult child to keep under control.' His final discharge came in March 1936, eleven years after he contracted polio.[59]

The policy of admitting all cases of polio regardless of severity caused problems of overcrowding in many hospitals. Christchurch Hospital had up to sixty patients at a time. Because the hospital had 30 to 40 long-stay child patients a teacher had to be appointed, as well as an increase made in the massage staff.

Chiropractors, who were attempting to gain official recognition in the face of the entrenched opposition of the BMA[60], were making large claims as to their ability to successfully treat and cure polio. Public meetings were held throughout the country, including a large one on 2 March 1925 in the Auckland Town Hall, seeking an independent enquiry into the treatment and effects of treatment of polio, and to include chiropractic in such an enquiry.[61] The Chiropractic Association also wrote, successfully, to hospital boards asking them to support the setting up of a commission to enquire into chiropractic methods of treating polio.[62] However the acting Director-General of Health soon squashed any such attempts to legitimise the claims of chiropractic. He stated firmly that as there was conclusive proof that polio was an infectious disease caused by a specific virus, there was no point in setting up a commission to investigate the claims of chiropractors. Furthermore the Department was preparing to prosecute chiropractors for making false and misleading claims in the newspapers.[63]

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59 CH324 110 C.21 161a #5289

60 See L. Snookes, 'The Relationship Between Chiropractic and the Medical Profession in New Zealand', BA(Hons) essay, University of Canterbury, 1992.

61 H1,131/9/10 B 91, 3 March 1925

62 North Canterbury Hospital Board Minutes, 24 March 1925

63 A740 537 6213, 7 May 1925

Like all diseases of little known origin, there were many and varied explanations as to the cause of polio. Many linked the paralysis of polio with that caused by distemper in dogs, or with that caused by lead and arsenical poisoning - lead arsenate being widely used as a horticultural spray. Others who believed that 'man creates health or disease at the table', blamed the early feeding of fattening cereals, damp inferior flour, pasteurised milk, sugar, starchy foods, and preservatives.[64] What none of these theories accounted for was the cyclical nature of the epidemics: that is they recurred at intervals of several years, thus ruling out diet or poisoning as possible causes.

The inability of the medical profession to control the disease led to a proliferation of unorthodox remedies. Only about 5% of those who contract polio are paralysed in some way, and of these, about 40% can expect to make a complete recovery.[65] Any serious illness which has a 40% chance of complete recovery in the absence of a specific cure, is a fruitful field for alternative medicine. The 1908 Quackery and Other Frauds Prevention Act, which aimed to control fallacious quack advertisements, went some way to prevent the more extravagant claims for polio cures being published. Rubs and liniments of various kinds were the most generally favoured treatments and probably did as much as the treatments offered by the medical profession. Doctors, however were very conscious of anyone treading on their territory, and even alternative treatments offered through orthodox channels were treated brusquely, presaging the reception Elizabeth Kenny was to receive years later.

The Auckland Hospital Board was inclined to favour letting Dr E.C. Dukes, who claimed to treat polio successfully, access to those hospital patients whose parents agreed. However hospital doctors insisted that he work under their supervision, and Dr Dukes refused this condition. A masseur, Mr Crowhurst, claimed to be successful with his massage treatments and offered his services to the Board. However when the Board asked the honorary medical staff for favourable consideration to his working at the hospital, the staff refused to co-operate.[66]

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[64] All these suggestions and others far more bizarre are in a Health Department file H1,131/9/16a B 92, 'Suggested Causes and Cures'. Another file, H1,131/9/16 B 92 is missing.

[65] *AJHR* 1950 H-31 p. 104

[66] A740 532 62/3/1 Auckland Hospital Board Minutes, 6 March 1925

The medical profession was pushing for money to be spent on fundamental research. Doctors urged on the government the need for New Zealand to carry out its share of research work. The nature of the poliovirus, and its consequent control, took a long time to establish because of the difficulties in infecting animals other than humans. For many years monkeys were the only non-human species available to researchers, and the cost of obtaining and maintaining suitable primates put research into the virus beyond the means of most scientists, including those in New Zealand. In this country virtually all polio research was into the epidemiological nature of polio, and was carried out by public health officials as part of their responsibility for the control of notifiable diseases. In 1925, in the middle of the 1924 - 1925 epidemic, the British Medical Association campaigned for the Government to provide adequate funding for bacteriological and experimental research.[67] They claimed that New Zealand should be making a contribution towards world-wide research into polio and public meetings were held and resolutions passed urging the Government to establish a department under the control of a director and with researchers in the four main centres. As one editorial writer said, it was "incumbent on the State to leave no stone unturned to find a means of preventing ... this dread disease [which] leaves children condemned for life to drag tiny wasted limbs after them." [68] The campaign was supported by Dr Valintine, the Director-General of Health. He was against increasing funding for cancer research, preferring to investigate the causes of polio. Cancer, after all, he said, affected old people whereas polio "affects our young people before they have had a chance to do anything in the world." [69]

The BMA sought £10,000 from the government to fund research in the four main centres. The Association knew that 'able men', the equal of researchers anywhere in the world, were available in this country; the problem was simply an economic one. The Cabinet made a grant of £2,000 for a unit to be set up in Dunedin under the supervision of Professor Hercus at the Medical School, with Dr Charles Hector as research assistant. After a survey of the available literature from around the world, Professor Hercus was confident that New Zealand was up-to-date with the latest work being done. He saw the

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[67] Thirty years later, in the 1950's, the medical profession was far more realistic about the cost of research and development, and were content to await overseas developments in the production of the Salk and Sabin vaccines.

[68] *Poverty Bay Herald,* 20 February 1925

[69] Valintine to Hercus. H1, 148/6 12985

need to concentrate on developing a simple test for detecting those susceptible to the disease and to modify the virus, as Pasteur had done for rabies, to allow the development of a vaccine. He was confident that the problem would yield to steady, persistent research but there was no short cut method.[70]

A monkey house was built on the roof of one of the University buildings, an initial supply of monkeys was imported from Sydney (at a cost of £12 each), and the monkeys infected with poliovirus. Immediately there were problems. Some of the monkeys died from other complaints, others proved difficult to infect. Once the animals **were** infected they of course became immune and of no further use to the bacteriologist other than for post-mortem work. Although a further 46 monkeys were imported from Calcutta (for £3 each) the inability to get a constant and reasonable supply of monkeys doomed the project.[71] Professor Hercus's frustration at this limitation burst out in a letter to Dr Valintine: "Why will not our moral code allow us to experiment on some of the hopeless imbeciles in our mental hospitals." [72]

After three years research, (and the expenditure of £4,474 18s 8d), which proved no more than what was already well-known - that monkeys could be infected with washings from the naso-pharyngeal passages of infected humans - the research was abandoned. Dr Hector went on to study hayfever.[73] What became of the six remaining monkeys was not recorded. No further attempts at fundamental research were made. For the next thirty years New Zealand medical researchers returned to epidemiology, which, given the country's relatively small, stable and homogeneous population, had probably always been the area in which they could make the most effective contribution.

The 1924 - 25 epidemic was the most severe polio epidemic to affect New Zealand in both the attack rate, and the death rate in young children. There were 1,185 cases notified, or 9.7 per 10,000, of whom over 50% were under five years of age and nearly

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70 Hercus to Valintine, H1, 148/6 12985, 19 February 1925

71 C. Hercus and G. Bell, *Otago Medical School Under the First Three Deans,* E.S. Livingstone, Edinburgh, 1964, p. 187

72 H1, 148/6 12985, 19 February 1925

73 H1, 148/6 12985, 5 December 1928.

80% under ten. Nearly 200 people died, three quarters of these children under ten.[74] Quarantine measures had been instituted nation-wide, and the social life of the country, especially that of children, had been disturbed to an unparalleled degree. The Minister of Health announced publicly that he had "no doubt as to the value of closing schools during an epidemic of Infantile Paralysis." [75] The one positive result of the epidemic was to give impetus to the newish discipline of orthopaedics, ensuring its continuing existence as a part of the hospital establishment.

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[74] Maclean, *op. cit.* p. 321, *AJHR* 1925 H-31 p.13. Maori were virtually unaffected by this epidemic.

[75] *Press* 14 April 1925

CHAPTER FIVE
POLIO IN THE THIRTIES

If the consolidation of orthopaedics in the New Zealand medical scene was one legacy of the 1924 epidemic, then there was another negative, and sadder one: the number of children left paralysed. In the first decades of the twentieth century, New Zealand did not appear to have a problem with disabled children. The country did not have a history of industrially crippled children, and those who were born handicapped or who became so through disease, were the sole responsibility of their parents. As late as 1932 a newspaper could write that "until recently the problem of crippledom did not exist ... the community is justified in assuming that the care of crippled children is the responsibility of their parents and guardians." [1] Handicapped children were a source of shame and were kept hidden from sight. In the absence of a comprehensive state social security system families with no significant resources of their own could receive charity from church and other organisations to meet their immediate needs. Medical and surgical treatment was sometimes available through the local hospital but this was strictly means-tested and many families did not qualify. Even if children did qualify for treatment, difficulties of transport and accommodation for patient and parent could prevent the opportunity being taken. In the community there were no facilities for disabled people, no rehabilitation or vocational training. The disabled child would be cared for at home and eventually go to an institution when the family was no longer able to provide care.

The attitude of society was ambivalent - ranging from the sentimentality of the Tiny Tim variety: 'children ... dragging their tiny wasted limbs after them',[2] to outright hostility from those who did not want to see the disabled in public. This became very apparent during the setting up of the Crippled Children's Society. People walked out of public meetings and the organisers received abusive phone calls. Alexander Gillies counteracted this by sending his wife along to speak; people were reluctant to walk out on a lady.[3]

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1 quoted H.E. Carey, *History of the Crippled Childrens' Society's First 25 Years, 1935-1960*, n.p. Wellington, p. 3

2 *Poverty Bay Herald* 20 February 1925.

3 Radio New Zealand Archives, 'Crippled Children's Society Celebrates Its Golden Jubilee'. Recorded 1985

Gillies was determined to do something about the problem. An orthopaedic surgeon with considerable experience in treating war injuries,[4] he began to campaign on behalf of crippled children and disabled soldiers, immediately after his appointment as orthopaedic surgeon at Wellington Hospital in 1929. His aim was to dispel the attitude that a crippled person was destined to remain forever dependent on others, doomed to exist rather than to have a full and purposeful life.[5] He urged that surgical treatment be provided as needed, at little or no expense to the family, and that training be given to each crippled child for their future employment, so that they could be as independent as possible. In 1930, he joined the Wellington Rotary Club. Organised help for crippled children had been started by the Rotary Club of Syracuse, New York, in 1913. Later, Rotary International had urged that all Rotary clubs consider helping crippled children as a major community service effort. On 13 May 1930, Gillies addressed Wellington Rotary on 'The Cripple, and vocational training for the physically defective: the need in New Zealand for an organisation to help.' [6]

The Depression hampered efforts to get a society established but eventually Alexander Gillies with the help of Sir John Ilott, drafted proposals which were accepted by the Rotary Clubs of New Zealand at a National Convention, in Timaru in 1934. Rotary underwrote the cost of forming the Society and each Chapter undertook to set up a branch of the Crippled Children's Society in their area. Within six weeks of the Society's formation on 26 March 1935, fifteen branches had been set up- their boundaries the same as the relevant hospital boards. The local branches were autonomous, with the Dominion Executive of the parent body the link with the various hospital boards, government departments and other institutions. The branches were responsible for enroling members, raising funds and doing the actual work with crippled children. A survey, the first ever, was undertaken. The criteria set out by the Society were quite strict:

> a crippled child is a person under 21 years of age who being not mentally deficient or not educable has a defect, which causes or tends to deformity

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4 *ibid.* 60% of World War One surgical casualties had injuries to the limbs or spine.

5 *ibid*

6 Carey, *op. cit.* p. 3

> or interference with normal functions of the bones, muscles or joints. The defective condition may be congenital or acquired but does not include defects of the vital organs.[7]

Even so, 5,000 eligible children - far more than had been envisaged - were found.[8]

There were two factors which were especially important to the successful launch of the Society. Firstly, from its initial acceptance as a Rotary project, the idea had had the acceptance of established figures. Vice-presidents of the Society were the heads of the main churches, the heads of the BMA, the New Zealand Red Cross, Plunket Society, Women's Division of the Farmers' Union and Women's Institute. Secondly, polio was a disease which knew no social boundaries. High socio-economic status was no protection, as President Roosevelt himself showed.[9] Also polio struck down the previously fit. To hide away a formerly healthy and lively child was quite a different matter from concealing or sending away a child with a congenital deformity.

A number of benefactors gave generous donations - the most notable of which came from Lord Nuffield, the British motor magnate, and business associate of Charles Norwood. Lord Nuffield gave £50,000 - an enormous sum - as the nucleus of an endowment fund. He later made further donations totalling £17,500, and donated an iron lung (the first in the country), to Wellington Hospital.[10] Others who gave considerable monetary support were businessmen John Ilott, Roy McKenzie and Robert Anderson, pastoralist T.H. Lowry and Mr and Mrs W.R. Wilson. The latter donated their home at Takapuna, plus 13 acres of land including the beach frontage. The public were asked to donate funds for the maintenance of the home and this was soon done - £25,843 being raised in 1936. The Home, which was administered by the Auckland Hospital Board for the Crippled Children's Society, initially provided for 25 children who

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7 *ibid*, p. 20

8 *ibid* p. 14. The largest single group was disabled as a result of polio. This peaked in 1943 when 813 out of 3441 crippled children, or nearly 25% were polio victims. By 1955 this had dropped to 9% and in 1974 to less than 2%, Crippled Children's Society *Annual Report*, 1974, p. 3.

9 Roosevelt was crippled by polio in 1921. Although this was well-known, and Roosevelt himself publicly gave his support to organisations like NFIP, the stigma of 'cripple' was such that he was careful never to be photographed in a wheelchair, or using crutches or calipers.

10 The average annual wage for a tradesman in 1935 was about £225 - *1990 Year Book*

met the Society's criteria, that is physically not mentally handicapped. As space became available, crippled children from outside Auckland, and non-crippled convalescent children were admitted. By 1960 the number of beds had increased to sixty, and the Home had acquired a large bath house, physiotherapy department, solarium, gymnasium and chapel.[11] The Crippled Children's Society actually had a policy of not establishing institutions and taking on stipendiary staff, so the Wilson Home remained the only home under its auspices.

There were two main thrusts to the Society's work. One was to make sure that no child was deprived of appropriate treatment. To this end travelling clinics moved around each hospital district. An orthopaedic surgeon and orthopaedic technician from the hospital workshops assessed the needs of each child in their locality, thus saving the necessity for long journeys (and overnight stays) to Wellington or Auckland by parent and child. There were also problems over transfer of patients between Boards which happened when patients sought facilities not available at smaller hospitals. The larger hospitals required guarantees that their fees would be paid. In one case a child was admitted to Palmerston North Hospital, where she was referred to Wellington Hospital for specialist orthopaedic treatment. However she was refused admission until payment was guaranteed. This was done by the Manawatu branch of the Society. But the case did not finish there. Wellington Hospital provided the caliper ordered by the orthopaedic surgeon but disputed whether they should also provide the boots which the caliper fitted, or whether this was the responsibility of the Palmerston North Hospital.[12] The second thrust was to assist in developing crippled children into "self-supporting independent units in the Dominion's economic system." The Government was asked to relax the medical tests for admission into the Public Service, for candidates recommended by the Society. Approaches were made to the Department of Labour for special apprenticeship conditions in training and wages for disabled adolescents.[13] The local societies sought out work opportunities for disabled children in suitable occupations like artificial limb making, ticket writing, clerical work and so on. Employment options were mainly of the

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[11] History of the Wilson Home. A740 91 13/1/7 1960

[12] Carey, *op. cit.* p. 24

[13] *Crippled Children's Society 1st Annual Report*, p. 3

light manual variety but some branches helped children with their education. The New Plymouth branch for instance helped keep three boys, (victims of the 1924 epidemic), at New Plymouth Boys' High School as boarders[14] Offers of help came from other organisations like the Girl Guides, who set up 'home guides' and the Swimming Association which offered swimming instructors 'practically anywhere in New Zealand.' [15] In 1941 the policy of the Society was modified to include the aim of bringing treatment 'within the reach of every cripple or potential cripple', and gave equal emphasis to educational and vocational training.[16]

Less than two years after its formation, the Crippled Children's Society faced a major increase in the number of those needing help. There had been a few cases of paralytic polio each year since 1925, with two small peaks in 1927 when 17 people died, and 1932 when notified cases reached three figures and 19 people died.[17] In 1936, only two cases had occurred in the whole country up until December, when suddenly Dunedin was hit with 85 cases. The disease remained confined to the Otago Health District, with only isolated cases in the rest of the country. In April 1937, the epidemic struck Christchurch and almost simultaneously the North Island and continued until July. Between December 1936 and November 1937, nearly 900 cases were notified of whom 656 had some degree of paralysis and 46 people died. The attack rate of 5.7 per 10,000 was less than that of 1916 or 1924.[18] This could be attributed to the fact that the birth rate had been in decline since 1925 so that the number of children in the most vulnerable age-group of five to nine years of age, had decreased. There were two other interesting features of this particular epidemic. Polio was still regarded as a warm weather disease, with highest incidence in late summer. Yet this epidemic began in early summer and continued until mid-winter. The second interesting feature was that the attack rate amongst Maori was approximately the same as the European. This was unusual because the Maori population was still largely rural, with (in many cases) unsatisfactory sanitation. Their infant mortality rate was still well above the critical point for polio to switch from

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14 Carey, *op. cit.*, p. 38

15 *Crippled Children's Society Annual Report*, 1937 - 38, pp. 6-7

16 *Crippled Children's Society Policy*, 1941, p. 1

17 Maclean, p. 320

18 *AJHR* 1937 - 38 H-31 p. 13

being endemic to epidemic, yet the pattern of infection, the age of incidence and number of paralysed cases closely followed the European pattern. This epidemiological abberation has been studied but no wholly satisfactory explanation has emerged.[19]

The Crippled Children's Society was concerned that few Maori children were coming forward for assistance. In 1937 the Society asked the Maori Affairs Department to ensure that the Department's school nurses and school doctors made sure that eligible Maori children came to the Society for help. In 1938, following a meeting of representatives of the Taranaki, Manawatu, and Wanganui branches, the medical officers for these regions and representatives of the Maori Affairs Department, £250 was allocated by the Society for 'experiments' in how Maori crippled children could best be helped.[20] This committee, while noting the reluctance of Maori parents to seek advice, felt that compulsion was not desirable. However education in health and welfare was essential and 'would in time diminish the prejudices that now exist.' [21] The Society stated that "the Executive will not hesitate to devote any funds necessary to obtain treatment for Maori crippled children. It remains a human and personal factor not the economic factor which must be surmounted." [22]

In 1932, Dr A.B. Pearson, a hospital pathologist, had stated that "in the unlikely event of another epidemic occurring the Health Department and hospitals would be able to deal with it ... by concentration of most of the cases in hospitals and by the use of serum, then mortality and paralysis rates could both be kept down." [23] Unfortunately Dr Pearson was over-optimistic. With the outbreak of polio in Dunedin, admission of all non-polio cases was restricted to urgent cases only. All suspected polio cases were admitted to single isolation rooms until positively diagnosed. They were then moved to two wards, small children in one and bigger boys and girls in another. Non-paralysed patients were sent to a convalescent home.[24] Isolation for positive cases was still six

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19 *NZMJ*, Volume LVII #319, June 1958, pp. 242 ff

20 Carey, *op. cit.* pp. 28 ff

21 *Crippled Children's Society Annual Report*, 1938 - 39, p. 6

22 ibid

23 *Christchurch Sun* 6 January 1932

24 H1, 131/9 3703 B 89-90, 13/1/37

weeks, although by January this was reduced to four weeks from onset and the quarantine for contacts was reduced from 14 days to ten when Dunedin Hospital began to suffer from over-crowding. Mild cases were recovering rapidly and were free from clinical signs in a few days.[25] Doctors throughout the country were contacted by the Department, and given an updated memorandum first issued in the 1924 epidemic. They were reminded of the need for complete immobilisation of all acute cases regardless of the presence of weakness or paralysis.[26] This was in line with the accepted practice overseas where total immobilisation for six to eight weeks was the standard, and the majority of patients were kept off their feet for ten to twelve months. Passive exercise was introduced once all soreness had gone.[27] An initial recommendation to commence serum treatment was withdrawn, although serum was still being bought and held by hospitals in 1937. Serum treatment had been greatly in vogue in the early 1930's. Half a pint of blood was taken from people known to have had polio. The blood was allowed to separate and the resulting serum drawn off. Fifty millilitres were injected, ten millilitres intrathecally and forty millilitres intravenously.[28] Voluntary donors were sought without great success, and in 1933 the Government as paying 2/2d a pint for suitable blood.[29] A budget of £40 was allowed and the resulting serum was sold to hospitals at the rate of 6d per millilitre. Serum treatment was therapeutically correct in principle but it failed because of the small doses used, (by the time the serum was administered the body was manufacturing its own antibodies), and it was usually given too late in the course of the infection.[30] Results of serum treatment obtained overseas were patchy and by 1937 the consensus of medical opinion was that it had little value. Nasal sprays, particularly one of picric acid, were being advocated in the United States but the Health Department did not believe that they were of any use.[31]

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25 H1, 131/9 3702 B 89-90, 1/11/37

26 Memorandum for Medical Practitioners, H1, 131/9 12961 1937-43 .

27 e.g. Lewin. *op. cit.* pp. 136, 158

28 Pamphlet, H1, 131/9 B 89-90 3711, 21 March 1932

29 H1, 131/9 3700, 6 December 1932

30 Paul, *op. cit.* p. 199

31 Memorandum to Medical Practitioners, H1, 131/9 12961, 22 December 1936

Hydrotherapy baths however were in vogue in 1937. A Hubbard bath was installed in Wellington Hospital in 1937. This was six feet long and had a holding capacity of 150 gallons of water maintained at 90°C.[32] Christchurch and Dunedin each had baths, and baths were installed in the Wilson Home, Cook Hospital, Gisborne and Wanganui where £300 was set aside for building the bath, bath-house and heating.[33] But overall facilities were limited. In 1938 the physiotherapy department in Christchurch was explaining the need for a gymnasium for rehabilitation, and Miss Lambie, the Director-General of Nursing, returned from overseas appalled at the poor standard of equipment in the physiotherapy departments compared with Great Britain, Canada, United States and Europe.[34]

In contrast to the 1924 epidemic, the Health Department in 1936 circulated a description of symptoms to all the newspapers, and these were published along with the list of precautions previously issued, warning against getting over-fatigued, playing in the sun, going about without the head and neck covered, sharing common property and so on. By January 1937, the Department was confident that the epidemic had been confined to the southern part of the South Island. Dunedin Hospital had admitted nearly 150 cases, suspected or confirmed. The majority had been mild cases, with 16 of minor paralysis, 14 of severe paralysis and seven deaths from respiratory paralysis.[35] Schools throughout the country had been closed early in December and gatherings of children were discouraged but the massive quarantine effort of 1924 - 25 was not repeated. The only restriction on travel was on that by children out of Dunedin, although all excursion trains from Christchurch, Timaru and Waimate areas were cancelled, including the regular Christchurch - Timaru Sunday train.[36] Centenary celebrations at Riverton were cancelled after two years planning, and health camps were closed except for children from orphanages. Children under 13 were excluded from picture theatres, and parents were advised to keep children away from swimming baths, holding birthday parties and

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32 L. Barber & R. Towers, *Wellington Hospital 1847 - 1976*, Wellington Hospital Board, Wellington, 1987, p. 84

33 H1, 131/9a 9276, 14 July 1937

34 North Canterbury Hospital Board: Annual Report of the Physiotherapy Department, Series 1/6, Book 36, 31 March 1938

35 H1, 131/9 B 89-90 3702, 11 January 1937

36 *ibid*, 7 January 1937

camps, and so on. Unlike 1924, these restrictions were voluntary.[37] Even so the restraints were criticised by a group of Dunedin doctors, including three professors from the Medical School (one of whom was Professor Hercus), five hospital consultants, Dr McMillan, a local Member of Parliament, and the District Medical Officer of Health. They passed a resolution that the compulsory restrictions placed on the public should be lifted, especially those concerning travel. They declared that such restrictions were not in accord with current medical knowledge about the epidemiology of polio, and that they were not in the public interest. Further, the Department of Health should be educating the public about how epidemics occurred, and in particular the futility of trying to restrict the disease to one area of the country, by measures which interfered with the individual's right to freedom of movement.[38] Privately the Director-General of Health agreed that quarantine was of little practical value in preventing the spread of the epidemic, but that the decision to restrict children's travel was a political one.[39] But he was furious at the doctors' very public criticism. Not only was the local Medical Officer of Health opposing his Director-General by claiming to know more about polio, when it was the Director-General himself who had examined him in the only public health qualification that he held, but also Professor Hercus was being deliberately two-faced. While very publicly advocating the value of acquiring natural resistance by exposing young children to the virus, Hercus had privately acquired serum and inoculated his own children.[40]

A large number of cases in Canterbury and the North Island resulted in schools again being closed, although this time it was generally on a school by school basis. For instance, Waitaki Girls' High School was closed for two weeks after two pupils were diagnosed as mild cases. When the three year-old sister of a Martinborough school child was positively diagnosed, the Martinborough school was closed. Schools were closed as a group in three areas: the Wellington Education Board district (north of Cook Strait), Hawkes bay (north of Nuhaka and including East Cape), and the Waikato, until after the May school holidays. The local Medical Officers were allowed to exercise their discretion over admittance of children to picture theatres and Sunday schools over the holidays, but

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37 *ibid*, 5 January 1937

38 H1,131/9 B 89-90 3702, 31 December 1936

39 Dr Watt to MOH, Samoa, H1, 131/9 B 89-90 3702 8 January 1937

40 H1, 131/9 B 89-90 3703 20 January 1937

all gatherings of children to commemorate Anzac Day and to celebrate George VI's coronation were cancelled.[41]

Frequently school committees closed schools when parents kept their children away from school following rumours of possible cases. A request by the Auckland Hospital Board that the Department close all schools and picture theatres was declined by the Minister of Health, Peter Fraser, on the grounds that it "was not warranted at that stage." [42] After a possible outbreak at Kaikohe, the local kaumatua wanted to stop Maori children going to picture theatres. However, they opposed the closing of schools. It was felt that control would be lost over the children. While they were at school they could be watched, and healthy children kept from mixing with children from infected households.[43]

High school students had assignments posted to them by their form teachers, and pupils were expected to do a minimum of four hours study a day. For primary school children, the newspapers published lessons daily. A radio session on 2YA was held for an hour from 9.30am each day, but because many families did not have radios it was not a medium which could be used widely. The closure of schools was not felt in all quarters to be a bad thing. The Editor of the *Star-Sun* took the opportunity to make a general attack on the education system when he wrote that:

> "interruption of school lessons is a less serious matter than the critics imagine ... a great deal of the education given in the schools is concerned with inessentials so that the loss of the school time is by no means a measure of the actual gap in training." [44]

Most schools finally reopened following the May holidays, those in Wellington and Gisborne two weeks later. As had happened after the wholesale closures of 1925, the number of pupils returning dropped. At Wellington Technical College, attendance dropped 11% on the expected rolls. Parents, tired of having children around the home,

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41 A373 H.HN 5/21 1938/1A Letter from D-GH to MOH, 30 April 1937

42 A740 537 62/3, 20 April 1937

43 H1, 131/9 13706, 22 April 1937; MOH Auckland to DGH, H1, 131/9 3711 20731

44 *Star-Sun*, 7 April 1937

had urged them to take up jobs if they were available. In some cases girls had gone to attend commercial colleges.[45]

Ratana Pa was once again a focus for public suspicion. The local Medical Officer allowed small gatherings of Ratana adults as long as they were not contacts and children under 16 were excluded. Other Maori celebrations were cancelled: a visit by a Maori party from Tauranga to the opening of a new pa at Matamata was stopped, and Te Puea's concert party was refused permission to travel to Auckland.[46]

Anyone diagnosed with polio was isolated for at least four weeks, and most were admitted to hospital. Many of these patients were in hospital for months, and often, years. Initially parents had to pay for hospital treatment unless they qualified for free medical care, such as the children of war pensioners. In 1938, hospital treatment became free for everyone. Christchurch Hospital had polio patients in three wards: babies in one, older children in another (with boys at one end and girls at the other), and children over about 14 and adults in yet another. A ten-year old boy was admitted to the main children's ward after he collapsed getting out of bed. With one leg completely paralysed he was placed in isolation, in a felt-covered cast, unable to move anything other than his eyes. This lasted for a week. Some months later he commenced treatment with daily physiotherapy and hydrotherapy baths. School lessons were given daily by the hospital teacher. The hospital carpenters made boards to go across the children's knees as they sat on the floor. The main interruption to lessons was the arrival of the porters each day to take individuals to therapy. Community organisations 'adopted' the children. Scout and guide troops were formed, with the children doing what tests and badges they could. The Mayor raised funds to purchase a radio for the ward, and the Salvation Army came every Sunday to take a service. Parents were allowed to visit for two hours once a week, but sibling visits were completely forbidden. Later, after considerable pressure, the medical superintendent conceded a second weekly parental visit of one hour.[47]

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45 *Dominion*, 1 June 1937

46 H1, 131/9 B 89-90 3706, 13/5/37, 19/5/37

47 Denis Hogan, interview, 3 April 1992. The policy of hospitals to exclude or at least minimise parental visiting was in place throughout the entire period of this study. The policy was usually justified on the grounds that children became 'too upset' when their parents visited. The effect of prolonged hospitalisation on the children, and the lack of normal family ties with their parents and siblings is difficult to ascertain, although it is tempting assume that re-integration with their families must (continued...)

This routine of school and daily treatment seemed set to continue indefinitely. After about 18 months this particular boy was taken home by his mother and brought to the hospital every day for treatment - which often involved being pushed in a wheel-chair several miles to the hospital and back. The Christchurch Businessmen's Association, Rotary and the Crippled Children's Society all at times provided transport. However the outbreak of World War II saw the rapid reassessment and discharge of the remaining polio patients, and they too came back for daily treatments. Another five year old boy who collapsed getting out of bed, was kept at home for a week before the doctor decided he was suffering from polio, and sent the child to hospital. His legs were splinted and he was kept in isolation for four weeks as were his family at home. He was then given massage, electric impulse treatment and exercises. After five months he was able to stand by himself. After six months he was discharged and his parents were told to take him the physiotherapy at their expense. He showed no further improvement so after some months he was sent back to hospital where he was fitted with specially made boots and calipers. These he wore for eight years.[48]

Polio patients were under the general supervision of orthopaedic surgeons. Dr W.S. Robertson and J. Leslie Will were still at Wellington and Christchurch respectively, while Dr W. McCaw was in charge in Auckland and J. Renfrew White in Dunedin. Orthopaedic surgery seems to have been undertaken with more caution than earlier, with the most polio related operations being done in Dunedin. The lack of specific purpose in treatment is shown in the case of 'B'. Admitted to hospital at 14 months of age, after being unwell for 10 days she was discharged two years and 11 months later. On admission she was able to stand, but not walk, and her left arm was paralysed. During her time in hospital her arm was kept in a sling, and she was allowed out of bed for two hours each day. Not surprisingly her nurses reported that she was often 'naughty' and would not keep her arm in its sling. During her stay in hospital she contracted mumps and chickenpox. Other than daily doses of Syrup of Figs and Milk of Magnesia, her only

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[47](...continued)
have been difficult. However few of those interviewed remembered any problems in settling into home life after prolonged stays in hospital. Many of course were preoccupied with coping with their mobility problems - getting to out-patients appointments on public transport, adjusting to school buildings that made no provision for the disabled and so on. This also applies to their parents, and maybe this has caused them to forget problems of social adjustment after long-term hospitalisation.

[48] PPS #320

treatment was regular massage. After two years her arm was reassessed, as being still limp and showing little sign of regeneration. Eleven months later she was again reassessed. The surgeon decided that her arm had flaccid paralysis and had failed to grow. The treatment had been to no avail, so the child was discharged.[49]

By July 1937, the epidemic was over but there was a post-script. In February 1938, the ship *Maunganui* arrived at Invercargill from Australia, with the body of a child who had died of polio on the voyage. *Truth* charged the authorities with carelessness in releasing the ship, and running the risk of spreading polio in New Zealand from Melbourne where an epidemic was raging. However it was proved that normal quarantine procedures had been carried out, and short of prohibiting all travel between New Zealand and Australia, there was little more that the authorities could do. Ironically it was probably someone from New Zealand who had taken the disease to Melbourne.[50]

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[49] 1945-46 CH334 100 110 c21 162f #4445

[50] *Truth* 16 February 1938; H1, 131/9 92795, 25 February 1938. However Barry Smith states that the 1937 - 38 Victorian epidemic was caused by Type I poliovirus - see J. Caldwell et al., *What We Know About Health Transition: the Cultural, Social and Behavioural Determinants of Health,* Health Transition Series, No. 2, Volume II, Australian National University, Canberra, 1990, p. 866. But New Zealand research indicates that the 1937 epidemic here was probably caused by Type III poliovirus - see Caughey et al., 'A Study of Immunity to Poliomyelitis in New Zealand', *NZMJ*, Volume LV, #308, August 1956

CHAPTER SIX

THE 1940's AND SISTER KENNY

The two names which became inextricably linked with polio in the minds of the New Zealand public were those of Jonas Salk and Elizabeth Kenny. That Salk's name became a household one is not surprising: he had given his name to the vaccine that eliminated one of this century's most feared diseases. The familiarity of Sister Kenny's name, however is rather unexpected. A nurse with no formal training, from the back country of Queensland, she became renowned, reviled, respected and rejected in Australia, New Zealand and the United States for her theories on the treatment of polio, and her manner of promoting them.

Sister Kenny's treatment was not new. The use of hot packs to treat muscle spasm had been a standard remedy for many years, and as far back as 1884, Bernard Roth had advocated the early, vigorous treatment of polio patients with warm baths, massage, passive stretching and exercise.[1] There is much anecdotal evidence of home treatment with hot packs.[2] In 1925, a 21 year-old man described how in 1906 his mother had treated his polio affected legs with hot packs made by soaking blankets in hot water, and massage for two hours night and morning.[3] Another letter detailing a similar case in 1924, elicited the response from the Director of the Division of Public Hygiene that "the use of frequently repeated hot packs was well-known to the medical profession as one of the many forms of symptomatic treatment, but the important thing is to deal effectively and directly with the virus floating in the blood".[4] This was at the height of the fashion for serum treatment. In reality, orthodox medicine from about 1915 on, came to regard muscles affected by polio as extremely frail. The emphasis was on the weakness caused by the lower motor neuron type paralysis, and the deformities that resulted from the different strengths of antagonist muscles. This view resulted in the patient being completely immobilised by being encased in plaster, or by being stretched

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1 Papers and Discussions Presented at the Third International Poliomyelitis Conference, p. 336

2 This probably accounts for the numerous people who claim they were one of the first persons in New Zealand (or Australia) to receive Kenny treatment.

3 H1, 131/9/10 B 91, 19 February 1925

4 H1, 131/9 B 89-90 3711, 14 January 1932

out on a frame. Kenny reversed what was cause, and what was effect. She argued that deformities were the result of persistent muscle shortening following muscle spasm. If this spasm could be relaxed in the very early stages then deformity would not develop, or would be minimised. She also argued that some paralysis resulted from a psychological mechanism triggered by painful shortening or spasm of opposing muscles - a process she termed 'mental alienation'. Function could be improved by the relief of muscle spasm and muscle re-education.[5]

The system of immobilisation for numerous conditions besides polio, such as stroke, fracture, multiple sclerosis, peripheral nerve disorders and so on, was so entrenched that it was going to be very difficult to persuade orthodox medicine that the treatment not only did not have the desired effect, but could even be harmful. It would have been difficult for a fully trained medical doctor to overturn the received wisdom of the orthopaedic hierarchy; for a woman, a nurse, an Australian with a less than complete knowledge of neurology, it would have seemed almost impossible.[6] Yet she played a major role in ending 'a century of the abuse of rest', not just for polio, and caused a complete re-evaluation of what had become routine methods, often carelessly applied.[7] Even while she was being most scorned, her methods for treating paralysed limbs were becoming absorbed into mainstream care.[8]

The resistance she encountered from the medical profession was partly due to her own personality. A woman of statuesque proportions, she was equally solid in her opinions. She was aggressive and dogmatic and demanded that not only her treatment but also her theories be accepted.[9] She also did not understand the innate conservatism of conventional medicine, and resented all criticisms as either petty or stupid. In fact very few medical ideas have gained such rapid acceptance, or caused such a complete change

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5 M. Knapp, 'Contribution of Elizabeth Kenny to the Treatment of Polio' in *Archives of Physical Medicine*, Chicago, 1955, p. 257

6 Paul, *op. cit.* pp. 344, 340

7 V. Cohn, *Sister Kenny: the Woman Who Challenged the Doctors*, University of Minneapolis Press, Minneapolis, 1975, p. 257

8 E. Willis, 'Sister Elizabeth Kenny and the Evolution of the Occupational Division of Health Care', in *Australian and New Zealand Journal of Sociology* Volume 15 #3, November 1979, p. 34. Willis uses Kenny's experiences to illustrate the tight grip the medical profession keep on all areas of treatment. There are parallels in Kenny's experiences with the modern medicalisation of childbirth.

9 *Report of the American Medical Association*, n.p., 18 April 1944

in treatment of a disease as hers.[10] Following a negative report from a commission of enquiry into her methods, held in Australia, she went to the United States at the beginning of World War Two. The National Foundation for Infantile Paralysis was prepared to adopt Sister Kenny's methods as part of its programme but she alienated that organisation when she tried to dominate it. This, coupled with the efforts of several prominent doctors to discredit her, led to the setting up of a rival Sister Kenny Foundation in Minneapolis. Elizabeth Kenny revelled in her martyrdom, the press loved her aggressive approach, and the general public, seeing for the first time someone who could offer hope where before there had been none, flocked to her support. Her battles with the medical establishment became David and Goliath struggles. Articles and books, some bordering on the hagiographic, appeared.[11] Not all medical opinion was negative. One of her early American converts was Philip Lewin, who only a year or two previously had published a standard text which pushed immobilisation techniques to their extremes.[12] Another convert was Dr M. Knapp, of the University of Minnesota, who wrote in 1943 that "Sister Kenny's observations of symptomology are absolutely correct and regardless of future developments this much I do know: she has knocked us so completely out of our complacent groove of thought about infantile paralysis that some worthwhile advance is bound to result from both the revolutionary ideas and the frantic attempts of her opponents to refute them." [13]

Through determination and will-power, Sister Kenny managed to effect a revolution in the treatment of polio. By persuading hospitals to abandon prolonged immobilisation, she also encouraged patients to involve themselves actively in their own rehabilitation. By taking a positive approach, and by stressing those things which could be treated, like muscle shortening, reversible paralysis and incoordination, and ignoring those things for which no treatment was successful such as anterior cell degeneration with motor denervation, patients and their families were given hope. The mother of a six year old girl described what Kenny treatment meant to her. Her husband had had polio when he

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10 Knapp, *op. cit.*, p. 510

11 e.g. Elizabeth Kenny and Maria Ostenso, *And They Shall Walk*, Robert Hale, London, 1951. The best biography is by Victor Cohn.

12 Paul, *op. cit.*, p. 342; Lewin, *op. cit.*

13 quoted Paul, *op. cit.* p. 342

was 17. He had spent months in hospital immobilised with sandbags; treatment which he was sure had contributed to the residual weakness he had in his left arm and leg. When her daughter, paralysed from the waist down, was diagnosed as having polio, she "felt this was a cruel blow of fate ... I was grieved beyond belief." The only ray of hope was that instead of being allowed to waste away like her father the child was given Kenny treatment. The girl was admitted to Waikato Hospital where Mr Bell had just started as a Kenny technician (see below, p. 62). After four months she was discharged with weakness in one leg but without the dreaded irons and calipers, an outcome the mother always ascribed to the prompt application of Sister Kenny's methods.[14]

Sister Kenny's treatment was based on observation and treatment of symptoms, and had no basis in pathology. This was the basic objection of her sternest critics who relied wholly on pathological observation of damage, complete or incomplete, to the anterior horn cells. However more recent studies, and more sophisticated equipment have in fact shown that many other areas of the nervous system were affected, findings which were to have implications for the development of post-polio syndrome (see Chapter 9, p. 96 below).[15] Sister Kenny had first applied her methods on a systematic basis in Queensland in the 1930's. By 1937 there was some public pressure for Kenny treatment to be used in this country, but the Department of Health was not encouraging. Enquiries had been made in Australia, but the replies from the New South Wales, Queensland and Victorian health services were all negative. It was felt that her methods were not new and the success she appeared to be having were more the result of the confidence she inspired in her patients.[16] A report from London confirmed this and said that "after a very few trials" it had been felt that one or two of her ideas were good, and one or two definitely harmful.[17] Kew Hospital in Invercargill had however used Kenny treatment during the 1937 epidemic. They used hot packs, encouraged early movement and avoided splinting. The Health Department admitted that Kew's results were good but by 1942 they still felt that this was due to "the care and thoroughness" with which

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[14] Dorothy Alice Ford, *Journey From Strangers Rest*, Daphne Brassell, Wellington, 1989, p. 156

[15] M. Knapp, *op. cit.* p. 511. See Chapter 9 on Post-polio Syndrome.

[16] H1, 131/9 3703 B 89-90, 29 January 1937

[17] H1, 131/9/3 31291, 11 April 1940

ordinary methods were carried out.[18] Christchurch and Dunedin hospitals used a 'modified form' in the 1942 epidemic, and by 1943 the Health Department felt that perhaps there was something in it.[19] There had been a larger number of cases than usual in 1943, with nearly 200 reported and 24 deaths. Labour Member of Parliament, Mabel Howard took up the cause and urged the Minister of Health to arrange for nurses to receive Kenny training so that the method would be available to any parent desiring it for their child.[20] Her request for clinics to be set up was met with the response that this was not necessary as Sister Kenny's book outlining her principles and treatment was available, and in fact modifications of her treatment were in use in most New Zealand hospitals.[21] It was also felt that her method was "expensive, laborious, time-consuming and required long periods in hospital." [22] This last remark is rather ironic considering the length of hospital stays recorded during the 1937 epidemic. It is also doubtful whether Kenny's methods were being carried out as she prescribed. Radiant heat rather than packs, massage rather than passive movements, and a continuation of splinting, albeit in a much modified form, continued to be used by hospitals claiming to use Kenny methods.[23]

There was one centre in New Zealand where Kenny treatment was applied as exactly as Sister Kenny set out. This was at the Duncan Hospital for Neuromuscular Disease in Upper Hutt. Mr and Mrs Duncan of Hunterville financed Sister Dryden to study under Elizabeth Kenny in the United States. On her return they opened the Duncan Hospital in Silverstream, in May 1945.[24] This hospital could cater initially for eight patients, later increasing to 25. Offering free treatment to New Zealanders, the full responsibility for admission treatment and discharge lay with Sister Dryden, with occasional visits from Dr W. Robertson, orthopaedic surgeon at Wellington Hospital. Robertson was sympathetic to Kenny methods but generally the Duncan Hospital did not have a good relationship

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18 H1, 131/9 12961, 16 September 1942

19 Letter from D-GH to Medical Superintendent, Auckland Hospital, A740 532 62/3/2, 26 May 1943

20 *Parliamentary Debates*, Volume 262, 16 June 1943, p. 785

21 *ibid*

22 *Press*, 3 October 1942

23 *ibid*

24 *N.Z. Woman's Weekly*, 12 September 1946, pp. 6-7

with doctors. Their attitude was described as "not one of hostility but non-co-operative scepticism." [25] The hospital was the subject of enquiry by Dr J. Caughey, who, while supporting the general thrust of Sister Dryden's work, was very critical of the old building, which was a fire risk, the lack of equipment for dealing with acute respiratory paralysis, and the lack of regular medical supervision.[26] Following his suggestions, the hospital was moved to Durie Hill, Wanganui. An adjacent farm helped finance the operation, and the hospital continued under the direct supervision of Bill Bell (see below). The basis of treatment remained the Kenny method: hot packs, with an individual exercise regime for each patient. Treatment was carried on as long as necessary, with no-one considered incurable. Although close co-operation was maintained with Wanganui Hospital, it was believed that better results could be achieved from continuous in-patient treatment, commenced as soon as possible after the onset of the disease, than by out-patient visits to the Physiotherapy Department of a general hospital.[27] The Duncan Trust also paid for a number of doctors and nurses to train in the United States, in treatment of acute polio, to be prepared for future epidemics.[28]

The Health Department was waiting for the next 'big one'. They were in the unenviable situation of having to prepare for a disease for which there was no clear cause of infection, no universally adopted method of treatment, no adequate means of control, and which caused dread and panic in the general population. The health authorities were also the target of every medical crank, every believer in alternative remedies, and now populist politicians. No wonder Dr Watt had described polio as "the bane of all public health officials." [29]

Regular advertising campaigns were carried out in newspapers and on radio to encourage the population avoid 'summer sickness', a general term covering symptoms like diarrhoea and vomiting, fever and general malaise usually resulting from the inadequate storage of food, and poor handling techniques. 'Summer sickness' resembled the early and abortive

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[25] *Parliamentary Debates*, Volume 275, 24 September 1946, pp. 158 - 160

[26] H1, 131/9 23399, Report of Dr J. Caughey, 17 January 1950

[27] Conversation with S.W. Bell 6 July 1991; A740 537 62/3 1955

[28] H1, 131/9 25556, 29 October 1952

[29] H1, 131/9 3707, 12 March 1937

stages of polio, and it was felt that an attack on one might help the spread of the other, after it had been noted that the previous epidemics had all begun in the summer, peaking in the late summer and autumn months. Parents were warned to be cautious of people with colds, upset stomachs, headaches and fever, and to watch for children getting chilled, to be careful sun-bathing, and to keep their hats on.[30] However in November 1947 there was a rapid increase in polio cases in Auckland - to be followed quickly by cases in Waikato and Taranaki. The epidemic became wide-spread throughout the North Island that summer, although it did not peak in Wellington until the beginning of winter. Late 1948 saw the disease wide-spread in the South Island until finally the epidemic died away by July 1949. Southland, hard hit in 1937, remained virtually free of any cases, although Dunedin, equally hard hit in 1937, recorded a large number of cases ten years later. With 80% of the cases in Wellington occurring over the winter months, polio could no longer be described as a 'summer disease'. No longer either could it be regarded as a disease of young childhood. Up until 1937 the largest number of children paralysed were under five. Now the five to nine age-group had the highest rates of paralysis, and more and more cases were appearing in the young adult category.[31] This is reflected in the change at this time in both the popular and medical press from the use of the term 'infantile paralysis', to the more accurate 'poliomyelitis'.[32]

Sister Kenny had sent a training film to Sister Dryden in September 1947, and this was followed by a visit from Sister Kenny herself in December 1947. Her lectures on her techniques to general practitioners and hospital staffs were greeted with the usual mixture of scepticism and hostility. One of her audience in Auckland described her as "a large blowsy woman, absolutely convinced she was the Messiah for polio victims." [33] Someone who was convinced that Sister Kenny had a lot to offer was Mabel Howard, now Minister

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30 e.g. Broadcast, Dr Turbott. 5 December 1947

31 166 people were paralysed in the 15 - 24 age-group in 1947 compared with 97 in 1937; 125 people over 24 years were paralysed in 1947 compared with 41 in 1937. Maclean, *op. cit.* p. 328

32 The Health Department used both the terms'Infantile Paralysis' and 'Poliomyelitis'. For example up until 1920 the Annual Reports of the D-GH used 'poliomyelitis' in the tables and 'infantile paralysis in the text. In communications with docotors and with the public the terms were used interchangeably up until about this time. By 1950 all medical communications used 'poliomyelitis'. The public (and media) were slower to change but the introduction of the Salk vaccine - always referred to as a polio vaccine - saw the universal acceptance of 'poliomyelitis'. It was interesting that almost invariably all those I interviewed who had had polio carefully explained that 'it used to be called infantile paralysis'. The same comment appears again and again on the Post-polio Survey forms.

33 Letter, Dr R. Wright-St.Clair, 17 April 1991

of Health. Sister Kenny offered to send two fully trained technicians to assist in Auckland, with the Kenny Foundation in the United States paying their salaries and fares to and from the United States. These two technicians duly arrived and stayed for six months. At the end of their term they were asked to stay on, but could not because of other commitments at the Kenny Institute in Minneapolis.[34] They brought with them a centrifugal wringer used for making hot packs. This wringer was later purchased by the Auckland Health Board.

Also recommended by Sister Kenny was S.W. (Bill) Bell. Bell, a nephew of Kenny, worked with her in Australia from 1935. In 1941 he went to Minneapolis to demonstrate and teach at the Kenny Institute. He worked as a physiotherapist with the United States Army during the War, then returned to Australia. He initially worked with the Duncan Trust, then moved to Auckland to demonstrate and teach Kenny treatment there until the arrival of the two American technicians. In March 1948 he went to Waikato Hospital at the request of their Board. His stay there was highly successful, although his initial appointment was against the advice of the medical staff.[35] In asking for his contract to be renewed the Board chairman wrote that Bell had "an outstanding personality ... his methods of dealing with patients, physically and mentally [was] remarkable and he gained the complete confidence and sincere affection of all those patients, men, women, and children he came in contact with." [36] The medical superintendent was also won over: "the results are very encouraging ... medical officers in control of the polio cases have a very high opinion of Mr Bell's capabilities." [37] The Health Department offered Mr Bell three years employment, and he returned to Auckland where he helped train others in the Kenny method. Later he moved to the Duncan Hospital. In December 1947 the Health Department also sent Dr Walter Robertson to the United States to study Kenny treatment and other orthopaedic matters to see what could be usefully applied in New Zealand.[38] His subsequent report was very favourable. First, he noted that as in New Zealand, many hospitals in the United States were carrying out 'modern methods' -a

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34 H1, 131/9/2 20485, 8 January 1948

35 R.E. Wright-St Clair, *From Cottage to Regional Base Hospital: Waikato Hospital, 1887 - 1987*, Hamilton, 1987, p. 72

36 H1, 131/9/3 2057 312911, 1 August 1948

37 H1, 131/9/3b 2040 31293, 12 November 1948

38 H1, 131/9/3, 22 December 1947

combination of the old splinting regime 'with a dash of Kenny' - that is, hot packs in the acute stage then splinting of the affected muscles. Much of the criticism of Kenny treatment was actually based on the results of this incorrect technique. He also noted that Elizabeth Kenny had never claimed to be able to limit paralysis where that was the first manifestation of illness.[39] Robertson accepted her classification of cases into paralytic or non-paralytic.[40] In paralytic cases the return of function was entirely dependent on the degree of nerve cell damage. In non-paralytic cases the paretic (weak) muscles were in a state of inhibition due to pain and spasm in opposing groups of muscles. Robertson agreed with Kenny that no non-paralytic case should develop gross deformity if treated with her methods, nor should there be gross muscle atrophy.[41] After comprehensively reviewing the superior rehabilitation system in the United States. Robertson came to a number of conclusions: Kenny treatment on its own could not restore complete normal function to paralysed muscles but it could diminish the resultant deformity; deformities were minimal both in number and severity. Her methods eliminated the evils of bed splinting, and the pain and spasm experienced in the acute stage. Many patients were able to walk, even with paralysed legs, with a stick crutch only and no calipers, and with a much better pattern of walking than splinted cases. While the possible need for surgery or other orthopaedic measures was not eliminated, Robertson knew of no better form of treatment. It was a good and rational method in both acute and convalescent stages, with much to be gained and little to lose. He recommended that training be given to nurses and masseuses, to be held in readiness for epidemics.[42]

The Department however declined to act. Decisions on treatment were the prerogative of individual hospital superintendents, not the Department. The ratio of technicians to patients was generally held to be about one to six or one to eight, and therein lay the inherent problem with instituting Kenny treatment. It was highly labour intensive. Preparing the hot packs, applying them, and doing all the muscle re-education was tiring

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39 H1, 131/9/3a 2040 31292, Report of Dr W Robertson

40 Previously accepted classifications were non-paralytic, paralytic, neuritic or meningitic.

41 H1, 131/9/3a 2040 31292, Report of Dr Robertson.

42 *ibid*

and arduous. In some hospitals volunteers came to wring the blankets to make the packs, but there was a great shortage of people to implement the exercise part of her programme, and the cost of having enough staff was enormous.[43] The other problem was to have enough trained staff on hand to deal with a situation which might occur perhaps once in ten years. Auckland Hospital had only 19 full-time physiotherapists out of a full establishment of forty. The Masseurs Association reply to a plea from the Auckland Hospital Board for more help gave some reasons.[44] Low remuneration, poor general conditions and an out-moded department were not attractive to staff. They were also not happy with a doctor being in charge of the physiotherapy department and wanted direct consultation between the Board and the physiotherapists themselves. The situation in Auckland was critical but the shortage was nationwide. In September 1948, the Minister of Health appealed for all registered physiotherapists to help treat polio victims if only for a few hours each day.[45] Dr Robertson offered a course in Kenny treatment in Wellington of two to four weeks duration but few could accept because of the difficulty of getting locums.[46]

Certain that another big epidemic could be starting, the Department of Health was anxious about prevention as well as treatment. Within days of the first cases in November 1947, in Auckland, schools in the North Island were closed, followed on 8 December by schools in the South Island. On 11 December, children were prohibited from staying in motor camps and attending Sunday schools, and two days later inter-island travel by school children was forbidden. The usual quarantine applied: all home contacts under 16 were quarantined for 14 days from date of removal of the patient to hospital, and home contacts over 16 were quarantined for the same time if their work brought them into contact with children, or if they were food handlers.[47] In Auckland no persons, including parents, were allowed to visit any patients in wards set aside for polio cases. To avoid the overcrowding of 1937, a panel of physicians expert in the diagnosis of polio was set up to examine all suspected cases before their admission to

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43 Medical Officer in charge, Physiotherapy Department to Medical Superintendent, A.H.B., A740 537 6213, 9 April 1948

44 Chairman, A.H.B, to Auckland Branch, Masseurs Association and reply, A740 537 62/3, 18 February 1948

45 H1, 131/9/11, 24 September 1948

46 H1, 131/9/11 2040 31301, 1948-49

47 H1, 131/9 177730, 8 December 1947

hospital. Only in cases of respiratory paralysis could general practitioners get direct admission for their patients.[48] Following a Health Department warning about bathing in the Manukau and Waitemata harbours, the beaches were almost deserted as were the swimming baths, which were closed to children, but if chlorinated could be used by adults.[49] In Wellington swimming baths were closed to everyone.[50]

University Entrance and Scholarship examinations went ahead but other wise 5th and 6th form pupils visited their school once only, by appointment, to discuss courses and obtain textbooks.[51] Prize-giving ceremonies were private or postponed to the next year. Children were sternly warned that "all congregations [were] to be avoided, both at schools and in tramcars and other public conveyances." [52] Dental clinics remained open, but appointments were introduced to avoid waiting. There were no extractions or root canal operations performed. Children were forbidden to 'loiter' after treatment. Dental nurses were told to avoid late nights, picture theatres and other entertainments, and to take their recreation in the open air.[53] A Christmas parade in Hamilton caused some concern. Father Christmas would parade through the city on an advertised route, distributing modelling wax on behalf of a local store. It was decided that the parade could go ahead provided the children stood on the pavement at least six feet apart.[54] The ban on children in camping grounds also caused difficulty. The post-war period was a time of acute housing shortages and many camping grounds had permanent residents. Delivery boys under 16 could not take groceries into residents, but had to leave them in the sun at the entrance gates. Later in January, families prohibited from camping within the grounds were simply camping outside, where conditions were far less hygienic than if they had been allowed inside. The Christchurch *Star-Sun* urged that restrictions be introduced in the South Island before cases appeared,[55] and the Blackball District Coal Mines Union wanted visitors barred from visiting Blackball until the epidemic was over.

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48 Notice from Medical Superintendent, Auckland Hospital, A740 537 62/3, 4 December 1947

49 *N.Z. Herald*, 11 December 1947

50 H1, 131/9 17730, 19 December 1947

51 ACC 210 B14 94f 1947/83

52 E 29/54/8 1948/14

53 H1, 131/9 20485

54 ibid

55 *Star-Sun*, 3 December 1947

Not only had they had no rain for seven weeks, their primitive sanitary arrangements restricted the facilities available to visitors and also many Blackball residents had cancelled their holidays and refrained from going to other areas, and they felt that others should refrain from coming there.[56]

At the end of January, the Minister of Education announced that all schools would be closed until Easter - "the danger of a deadly epidemic must be met." Lessons would be by correspondence and supplemented by broadcasts: "every home will become a miniature school." [57] Assignments from the Correspondence School in the form of booklets for each class from Primer Three to Form II were distributed to pupils, who sent the completed work to teachers for marking. The Education Department arranged for the posting of assignments to be free of charge.[58] The broadcast lessons began with a session between 9.30 and 10 am for Primers to Standard Two: "The Adventures of David and June at the Seaside and The Adventures of Brian and Lois in the Country - programmes which will provide activity in the form of written work which the children will do as a follow-up." [59] Standard Three to Form II had talks on music appreciation, current news, nature study, social studies, literature and "two special quiz sessions to test their listening", between 11 and 11.30am.[60] Secondary schools broadcasts covering English, social studies, general science and mathematics were broadcast in the afternoon. The sessions were built around Correspondence School lessons but individual schools had to meet the cost of duplicating correspondence school material.[61] The Editor of the *Listener* did not think much of the idea of broadcasts to school children:

> It is greatly to be regretted that it has been found necessary to attempt teaching by radio and earnestly to be hoped it will not be necessary to carry the experiment very far. If the whole scheme disappears before we have learnt much from it, parents, teachers and the Broadcasting Service

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56 H1, 131/9/2 20485, 20 December 1947

57 Radio New Zealand Archives, 'Minister of Education Talks About the Polio Outbreak', 19 April 1948

58 ACC 210 B.14 94f E 29/54/8 1947-89

59 *Listener*, 30 January 1948

60 *ibid*

61 ACC 210 B14 94f E 29/54/8 1947-88

> will all be happy ... full co-operation is not to be expected, human nature being what it is.[62]

Most schools in New Zealand re-opened on 1 March 1948, although those in Auckland, and in some other individual places, did not re-open until 19 April. This caused great confusion. Children living in restricted areas, but attending schools elsewhere had to obtain a certificate stating that they had had no recent contact with polio, and then reside somewhere outside the restricted area for 14 days before attending school.[63] The list of restricted areas was confusing, and some pupils were already on their way to school. The principals at New Plymouth Boys' and Girls' High Schools described the situation as "a glorious muddle." [64] Post-primary schools were getting anxious about the work that pupils sitting outside examinations were missing. There were also anomalies. Children under 16 could go to work, commercial colleges were not restricted and evening classes carried on. The Auckland branch of the Secondary Schools Association wanted schools open to 6th formers, who, as they pointed out, were allowed to congregate anywhere except school.[65]

Criticism of the Health Department's actions during the epidemic, and the closure of schools in particular, was growing. The *New Zealand Observer* attacked the Minister of Health's comment that the polio situation was "disturbing", and that her Department was keeping a close watch on the incidence. It was her only comment in six months, it said, and time the Department was able to answer questions about patterns of distribution, age-groups and the effect of special precautions.[66] The *Taranaki Daily News* wrote that the Department had done nothing except close schools and cinemas - "it was the silence of ignorance or defeatism." The paper also said that the quite remarkable co-operation of the public was wearing thin.[67] A 'very tired worn-out mother' expressed the feelings of many parents when she wrote to the Minster of Health, pleading that schools be re-

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62 Editorial, *Listener*, 6 February 1948

63 H1, 131/9 19885, 27 February 1948

64 *Taranaki Daily News*, 2 March 1948

65 H1, 131/9/2 20485, 14 March 1948

66 *N.Z. Observer*, Volume LXVIII #37, 2 June 1948

67 *Taranaki Daily News*, 10 June 1948

opened for those that wanted to send their children,[68] and a 16 year-old girl wrote that she was anxious that assignments were not satisfactory preparation for University Entrance, especially when she had to work at home with three primary aged boys and a three year-old.[69]

Medical comment was critical too. The Medical Officer of Health for Whangarei said that the public was bewildered by the closure of schools. Initially all schools were closed when the nearest case was 200 miles away, now a school was not closed if a case was only ten miles away. Picture theatres were closed but not eating houses. Infected people could gather in doctor's surgeries, which also raised the question of whether it was helpful to seek early medical advice for a disease which could not be treated anyway. He was also concerned at the very real fear of a positive diagnosis in parents. He had had the new experience of "seeing terror in the face of patients" even at the mention of the possibility of polio. Newspapers were printing details which made it impossible to disguise the whereabouts of a case, and as a result the affected household were treated like lepers. All this, he felt, could well be a result of the publicity and restrictions imposed.[70] His counterpart in Christchurch pointed out the futility of the restrictions on inter-island travel - the polio virus had been detected in Christchurch in October, three months before the first definite case in the city.[71] Finally Dr J. Caughey, one of the country's foremost experts on the disease, wrote to the Minister of Health urging the reopening of schools. Isolation, he pointed out, did not stop the spread but merely slowed it down. "Polio must be accepted as a risk of childhood just as any other acute infectious disease." Because paralysis was worse in adults, it was actually better for children to mix and gain immunity.[72] The Polio Consultant Panel met and agreed to the reopening of primary and secondary schools, but kindergartens and swimming baths were to stay closed.

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[68] H1, 131/9/2 20485, 22 March 1948

[69] *ibid*, 22 March 1948

[70] MOH Whangarei to D-GH, H1, 131/9/12a 23370, 6 April 1949

[71] H1, 131/9/10 31300, 18 March 1948

[72] H1, 131/9 23399, 31 March 1948

As a result of the new policy, patients were nursed in isolation for three weeks. Their family, playmates and other close contacts were still in quarantine for 14 days, but no longer confined to the house. Essential shopping was permitted but no visitors were allowed nor were the contacts to go visiting. Necessary communication with others could be made in the open air; child contacts could play with other contacts only, and again this was restricted to the open air. The breadwinner was encouraged to take time off work, on holiday if possible, otherwise they were to work on their own, and take their own sandwiches. The emergency unemployment benefit was now payable in cases of hardship. Picture theatres, swimming pools, motor camps and so on were not closed except to quarantined persons, but if polio was in the area, the public were advised not to congregate.[73] Teachers made daily health inspections of their classes and pupils were encouraged to have their own handtowels, and were discouraged from sharing food. Competitive school sports were disallowed as over-fatigue was recognised as a factor in lowering resistance.[74] At the end of the epidemic, when the closing of schools was evaluated, it was found that in only three cases did a second case occur in a school within the incubation period of seven to 14 days, so that it was doubtful whether schools played any part in the spread of the disease.[75]

The 1947-48 epidemic was actually spread over three years. In its length it differed from the previous pattern. Formerly epidemics had started in early summer and ended by winter, and usually were confined to one part of the country. This time the disease spread slowly over the whole country. Several special studies were done which also threw some light on the nature of the disease. A survey amongst the still largely rural Maori of the East Cape recorded only one case in a child over 12, and children under five accounted for 42% of cases. This incidence was similar to patterns in Japan, North Africa, Palestine and the southern states of the United States.[76] Hospital stays had shortened (in Wellington for instance, the average stay was 38 days), although it was not uncommon for people with a single paralysed limb to be hospitalised for nine to 12

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[73] Memo from District Office, Wellington, H1, 131/9 23399, 7 September 1948

[74] *ibid*, 16 September 1948

[75] *AJHR* 1950, H-31, p. 104

[76] H1, 131/9/12 23370, 4 February 1949

months.[77] Increasing age of patients saw an increasing use of iron lungs. The success of these was not high, and 12 iron lung deaths were recorded, seven from asphyxia (lack of oxygen). Patients requiring iron lung treatment usually had widespread paralysis, and the few who did survive were almost always severely physically handicapped.[78] For the first time a comprehensive follow-up was made 12 months later of patients who had been paralysed. Of these, 26% had recovered completely and 36.6% had a minor disability: that is they were able to earn their living and take a normal part in life. Nearly 11% were seriously disabled: unable to take normal employment but not completely incapacitated. Complete disability was recorded for 1%, and 9.5% of all those paralysed, died of their illness. The remaining 15% were untraced and can be presumed to have recovered completely or with a minor disability.[79]

The official view of the epidemiology of polio in 1947 was similar to that held in 1937. But the prolonged nature of this epidemic gave the Health Department a chance to re-evaluate its public health measures. The general public too had become less tolerant of draconian regulations, and was much more assertive in its demands different and better treatments. The climate of opinion everywhere was changing.

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[77] H1, 131/9/12, 20 June 1950

[78] Survey, Dr D. Taylor, 1948, A740 537 62/3

[79] *AJHR* 1950, H-31, p. 104

CHAPTER SEVEN

THE 1950's

The pattern of epidemics in the first 50 years of the twentieth century indicated that New Zealand's fifth major epidemic could be expected around 1958, so it was with more than a little surprise, and much trepidation, that public health officials recorded an ominous rise in reported cases in June 1952. Instead of spreading gradually over the country as had happened before, cases were reported more or less simultaneously from all over the North Island, followed by a similar pattern in the South Island three months later. The incidence of polio was not as high as in 1916 or 1925, but the cases of paralysis were now spread across all age-groups. Almost as many were affected in the 15 to 24, and the 25 and over age-groups as were affected in each of the under 15 age-groups.[1]

In hospitals throughout New Zealand Kenny treatment was now firmly established although not always acknowledged. Almost all hospitalised patients in the 1950's were treated with a combination of hot packs, hot baths and passive movements. The completeness of this about-face in treatment is shown by the report of the Third International Congress on Polio:

> the idea that paralysed muscle should be fixed in a position of relaxation is now quite untenable, for it is now fully established that frequent passive movements improve the condition of all paralysed muscles. The best way to avoid deforming postures in the acute stages is not by fixation but by frequent passive movement and positioning. Passive movements with gentle stretching of tight muscles may be done for a few minutes every four hours from the first day of paralysis.[2]

The reign of the orthopaedic surgeon was over.[3] Physiotherapists controlled their own departments and the care of acute polio was now largely the province of physiotherapists,

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1 See Appendix 4, p. 114.

2 *Report of the Third International Conference on Poliomyelitis*, 1955, p. 85. The Report went on to give slightly less enthusiastic endorsement of hot packing suggesting that their use depended 'more on sentiment than science', but it did accept that they were effective in reducing the pain and discomfort of having a tight muscle stretched.

3 There is no clear evidence what orthopaedic surgeons thought of this change. For instance I could not find any of the monthly reports to their respective hospital boards by surgeons in charge of departments at Christchurch, Waikato or Wellington. Certainly Dr Robertson of Wellington Hospital was enthusiastic about the change in emphasis in treatment - see p. 62.

paediatricians, neurologists and specialists in physical medicine. The orthopaedic specialists acted as consultants in the acute stage. Not until several months later were they called in to advise on the need for braces and corrective surgery.

In a comprehensive report for the Health Department, Dr J Caughey summarized the current state of knowledge of the disease and its effects. Like Sydney Smith nearly forty years earlier, he divided the disease into several stages, but he reduced Smith's eight categories to four:

1. Silent or inapparent infection in which there were no clinical signs or symptoms but the virus could be recovered from the stools.
2. Minor illness with fever, headache, sore throat, listlessness, anorexia,vomiting, diarrhoea, constipation, pains in the back and limbs. The clinical picture was not typical, and serological and viral studies were the only conclusive diagnosis. He estimated that in 1948 the ratio of minor illness to paralysis was 300:1.
3. Pre-paralytic - this stage marked the beginning of central nervous system involvement. In addition to the symptoms of the minor illness, he noted more severe muscle pain and stiffness, tightness and spasm. Cerebro-spinal fluid might be normal or abnormal with changes in cells and protein. These cases might settle down in few days, or progress to paralysis, possibly leaving residual muscle shortening and tightening. The pre-paralytic stage could last four to seven days, but the symptoms could be so mild as to pass unnoticed.
4. Paralysis - the central nervous system became involved at the spinal cord mainstem or bulbar regions. Reflexes were either exaggerated or absent.Paralysis reached its maximum in about 48 hours. Spinal paralysis had asymmetrical effects on legs, arms, back and thorax. Paralysis affecting the bulbar region involved the cardiac and respiratory centres, causing difficulties in reglutition and nasal regurgitation.

Caughey hazarded some reasons for the development of a minor intestinal disease into one causing major central nervous system involvement. Age was the most obvious factor

- the older the patient, the more serious the paralysis.[4] Genetic influences played a part as did endocrine factors. Pregnant women with their higher cortisone levels were particularly vulnerable. Tonsillectomy and other ear, nose and throat operations increased patients vulnerability as did recent inoculations against other diseases. Unusual exertion also seemed to predispose invasion of the central nervous system. A final factor was nutrition - but not malnutrition. Research had shown that rodents deficient in thiamine actually had increased resistance to polio, which tied in with the long observed clinical impression that the well-developed, well-nourished person was more vulnerable.[5] The report also summarized the state of knowledge of muscle involvement, which validated much of Elizabeth Kenny's empirical observation.[6]

The 1952 epidemic in the United States had revealed a very high incidence of bulbar paralysis, prompting the Health Department to check on the number of respirators in New Zealand. The first respirator in use was the Drinker respirator designed in 1929. It was a rigid cylinder into which the patient was placed and negative and positive pressure applied within the cylinder at short regular intervals. It was soon called the 'iron lung'.[7] The iron lung was designed for paralysis of the diaphragm and the intercostal muscles. Unfortunately, patients with bulbar paralysis, or a combination of bulbar and respiratory paralysis were treated in iron lungs. The secretions which bulbar patients were unable to clear were drawn into the patient's lungs, and as a result many died from drowning.

Wellington had the first iron lung in the country, in 1937, and soon the other major hospitals had machines donated by Lord Nuffield. Auckland hospital built one of their own from textbooks, adapting a milking machine, for a cost much less than imported models. In 1953 £2,500 was allowed in the Health Department budget to establish a reserve of respirators.[8]

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4 In 1952 the fatality rate amongst those over 25 years old who were affected was 10%; amongst children up to the age of four, 3.4%. See Maclean, *op. cit.* p. 329.

5 Report by Dr J. Caughey, H1, 144/17/1, October, 1954

6 *ibid*

7 Paul, *op. cit.* p. 325

8 *Parliamentary Debates,* Volume 301, p. 2262, 13 November 1953

The results of the 1947 epidemic were not encouraging. At Wellington Hospital 11 patients were treated in respirators, and seven died. In Christchurch, only one of the four iron lung patients survived. Results at Waikato Hospital were no better. Of ten cases for whom the iron lung was used, seven died while in the machine and one died three weeks later.[9] It was concluded that the lungs had no therapeutic value but made the patient more comfortable.

All this changed following a major epidemic in Denmark. A shortage of iron lungs there led to some makeshift measures, which unexpectedly resulted in a greatly reduced mortality rate. New guidelines were developed, and were probably responsible for the much lower mortality rate in the 1950's despite the greater severity of paralysis.

When there was bulbar involvement, or combined paralysis of respiration and swallowing, tracheotomies were performed in conjunction with intermittent positive pressure respiration. Close watch was kept for moisture in the pharynx and trachea, or respiratory obstruction. Posture and suction were used as first aid but a tracheotomy was usually needed. As the airway was unlikely to remain adequate indefinitely with artificial respiration, constant medical care was necessary. Complications developed with great rapidity, and in the first few days of respiratory paralysis patients needed to be specialed (treated on a one-to one basis) by a house surgeon as well as a nurse. Frequent power cuts added to the difficulties of nursing patients in respirators, and staff always had to be ready to leave their other duties and rush to operate the machines manually.[10] Suction, anaesthetic apparatus, intratracheal tubes and tracheotomy equipment all needed to be close at hand. Respirators were used as soon as a patient's breathing became distressed. Positive pressure respirators, such as the Oxford hand bellows, were used while the tank respirator was open to allow for nursing procedures or physiotherapy. Sometimes a Cuirasse respirator (chest only) was suitable.[11] Patients came to rely psychologically as well as physically on their machines and considerable attention had to

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9 H1, 131/9/12 23370, 6 March 1950

10 Letter, Elizabeth Gell, 3 May 1991

11 H1, 131/9 26838, 28 September 1955

be given to 'weaning' patients off their machines. For those with permanent respiratory difficulty, glossopharyngeal or 'frog-breathing' was taught.

Quarantine measures continued to be applied to all diagnosed cases, however mild. Suspect patients were isolated and school contacts excluded until the diagnosis was confirmed. Peer group contacts, and family contacts who were teachers were also quarantined for two weeks from the last exposure to infection.[12] Inoculations, especially BCG and diphtheria were postponed. There had been increasing evidence that the risk of paralysis increased when there had been recent inoculation, and the Department of Health did not want to risk the success of their immunisation campaigns.[13] Tonsillectomies were also postponed. Otherwise there were no general restrictions. The press were kept fully informed about the epidemic and were asked not to sensationalise any stories. Strict personal hygiene, especially among food handlers, was stressed.[14] Up until 1949 the general restrictions were based on the droplet theory of transmission, with the nasal passages considered the most likely portal of entry. However by the 1950's it was recognised that the virus usually left the body by way of the faeces, and that numerous seemingly healthy carriers could transmit the virus for up to six weeks. But the general public was not reassured. The *Timaru Herald* referred to "public unease" at the complete about face by the Health Department,[15] while the *Otago Daily Times* questioned the "apparent disinclination of the health authorities to insist on what was previously insisted on to prevent the spread of the disease." It felt that even if restrictions were of dubious effectiveness, they were psychologically beneficial.[16] Public bewilderment at the Health Department's change of policy was articulated by the redoubtable Dr Smith of Rawene. When the local medical officer of health had refused to close all schools, and stop school buses, Dr Smith telegraphed the Minister of Health: "keeping any school open in Hokianga is ethically a reprehensible action and politically unsound in view of overwhelming public opinion." [17] But unlike 1933 when Smith had set up road blocks

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12 Circular letter, Department of Health, A740 537 62/3, 17 September 1952

13 H1, 131/9 23399, 3 August 1950. See also footnote 8, Chapter 8, p. 81.

14 *AJHR* 1953, H-31, p. 10.

15 *Timaru Herald*, 3 February 1953

16 *Otago Daily Times*, 3 January 1956

17 G. Kemble Welch, *Dr Smith, Hokianga's King of the North*, Blackwood and Janet Paul, 1965, p. 225

in the Hokianga to prevent a tour by Ratana and his party during an epidemic of 'sleeping-sickness', public opinion was not quite so overwhelming, and Hokianga's schools remained open.

The Health Department reacted to criticism that it was taking a fatalistic attitude by introducing a handwashing campaign. This was thought to have a two-fold benefit. First, it would help stop the spread of a number of diseases passed on by poor hygiene, especially polio, and second, it would involve the parents of all school age children which was psychologically important.[18] Teachers were asked to emphasize the importance of handwashing before eating and drinking. Parents were asked to provide each of their children with a clean handtowel each week. Paper towels would have been preferable but they proved to be too expensive for school committees to provide. A more basic problem immediately became apparent: handwashing facilities were woefully inadequate and not enough money was available from schools' maintenance grants to enable upgrading. Christchurch South Intermediate for instance, had 16 hand basins for 585 pupils. Another school in Christchurch had eight hand basins for 800 pupils. Nor was there anywhere to hang the hand towels. Some schools resorted to the unsatisfactory practice of attaching cuphooks to the children's desks.[19] This hand-washing campaign certainly made more sense than the earlier attempts at quarantine, although it probably made little difference to the spread of polio. Instances of more than one or two cases at any school had always been extremely rare.

Members of the general public were not so convinced as the Department that personal hygiene had a large bearing on the spread of the virus. The most popular theories on virus spread put forward to the Health Department at this time involved the sun. The cycle of sunspots was linked with the cycle of epidemics. This theory came apart somewhat when it was pointed out that each epidemic occurred before each sunspot cycle, and the 1952 - 53 epidemic occurred when there was little sunspot activity.[20] Another was sure that polio was caused by lying around in wet swimming togs and getting

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18 H1, 131/9 255336, 23 December 1952

19 *Press*, 3 February 1953

20 H1, 131/9 25556, 31 May 1951

chilled. The remedy suggested was that swimming togs be banned and sunbathing be permitted, at segregated sessions at swimming baths.[21] Someone else noted the prevalence of polio amongst Scandinavian and Anglo-saxon peoples for whom sunbathing was a popular past-time but non-existent amongst Negroes, Aborigines and the Spanish who customarily took a siesta.[22]

The epidemic which had started in June, peaked in late winter and early spring, and continued over the summer finally declining in early autumn. Only eighty deaths occurred as a result of this epidemic, much lower than the death toll in 1916 or 1924, due almost entirely to the improvements in nursing bulbar paralysis. Equally dramatic was the shift in incidence from the youngest age-group to young adults, and with a resulting increase in severity of paralysis. In 1937, in the under four years age-group, the paralysis rate per 10,000 population was 15; the comparable figure for 1952 was 7.3. For the 15 to 24 years old age-group, the 1937 figure was 3.4, the 1952 figure, 5.7; and for those over 24, 0.4 in 1937 and 1.8 in 1952.[23] Another trend which had become increasingly evident in 1947 was the higher attack rate in some rural areas. This trend continued in 1952: although having only 30% of the total population, rural areas recorded over a third of the total cases, nearly 40% of paralysed cases and half the deaths.[24]

Epidemiologically, the next major outbreak could have been expected in about 1962, but 1955 saw another epidemic occurring only two years after the last. In August and September, cases began appearing in Hamilton and New Plymouth. The disease spread rapidly throughout the North Island to peak in November. The epidemic was slower to take hold in the South Island, but did so with greater severity. By the time it had tapered off in July 1956, the number of cases notified (1,485) was the highest ever in New Zealand; 925 people had been paralysed and 73 people had died.

There were two features of the 1955-56 epidemic which gave rise to great concern. First it seemed that New Zealand was developing a pattern of ever more frequent epidemics.

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21 H1, 131/9 26838, 17 January 1956

22 H1, 131/9 25556, 3 September 1953

23 Maclean, *op. cit.* pp. 327ff

24 *ibid* p. 324

The prospect was that of an annual polio "season", as was already the case in the United States. Secondly, the age of those attacked was increasing. In 1955 - 56 nearly half of all those cases notified were aged 15 and over; in 1925 this had been 11%; in 1937, 20%; and in 1947 - 49, 31%. Increasing age meant increasing severity of attacks and the requirement for more intensive nursing. By 1955 there were 54 iron lungs in the country. Ten more tank respirators were ordered from Australia, and six positive pressure respirators were ordered to supplement the two available in Wellington.[25] The techniques for using respirators had improved greatly. Tank or Box respirators were being used for purely respiratory paralysis, while tracheotomies and intermittent positive pressure respiration was used for combined paralysis of respiration and medullary function. Both machines used were more comfortable for the patient than the early Nuffield Iron Lung, but they were still draughty and cold. In order to keep the stomach empty and prevent aspiration of the stomach contents, feeding was through an intravenous drip. Oxford Hand Bellows had to be used while the box was open for nursing procedures or physiotherapy. Complications developed with great rapidity and the first two to three days were critical. Twenty-four hour nursing care was essential[26]. Nursing care for the less dramatically paralysed was also intensive and the strain on hospital resources was considerable. In Auckland the panel of specialists who were to be called in to check all suspect cases (to prevent hospital isolation blocks being overloaded) was revived for the first time since September 1952. Other than in cases of respiratory paralysis all suspected polio cases were to be vetted by one of the panel before admission to hospital. The Department of Health paid the consultant's fee.[27] In Christchurch where the hospital was trying to house 573 patients instead of the normal maximum of 567, the Medical Superintendent was reported as saying that "The only way to take in more patients at the moment is by putting stretchers in the wards." A recent severe outbreak of staphylococcal infection had made the hospital even more wary of overcrowding.[28] The situation was grim, but reports coming in from the United States gave hope that the end to this ever-increasing problem was at hand.

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[25] H1, 144/17/1 25623, 26 July 1955 and *ibid* 9 November 1955

[26] Letter from Medical Superintendent-in-Chief, A.H.B., A740 537 62/3 29 September 1955

[27] A740 537 62/3 17 October 1955

[28] *Dominion*, 15 February 1956

CHAPTER EIGHT

VACCINATION: THE END OF THE LAST OF THE CHILDHOOD PLAGUES

The elimination of polio from the list of epidemic diseases affecting the Western world has been described as "one of those rare achievements ... of what could be done when science and technology were directed to the good of mankind." [1] Following the discovery of the virus causing polio, medical researchers soon realized that prevention through some kind of artificial immunisation rather than treatment of the disease, was the logical approach. There were two reasons for this. Bacteria, which are metabolically different from mammalian cells and which exist outside of the cells of their host, can be targeted specifically with a wide variety of drugs. But it is much more difficult to separate the functioning of viral cells from normal host cells because the virus bonds to those cells. The main approach to controlling viruses therefore was not to attack the virus directly but to stimulate the body's immune system in advance. The second reason for the preventative approach was the difficulty in actually treating an infected patient. Polio, especially in non-epidemic years, was notoriously difficult to diagnose because the symptoms were similar to 'gastric flu' or 'summer sickness'. It was not until paralysis occurred that a definitive diagnosis could be made, and by then of course, it was too late for any treatment to reverse damage already done to the nerve cells.

It was a long time between the discovery of the virus in 1908 and the release of a safe vaccine in 1955. For many years the difficulty was replicating the disease in laboratory animals. No animals were susceptible to the human disease. Eventually it was discovered that one breed of monkeys could be infected with the human virus, not by the oral route as were humans but by intra-nasal inoculation. Unfortunately this meant that not only did researchers not replicate exactly the human infection but also that successive inoculations in monkeys led to a change in the pathogenesis (disease causing ability) of the virus. The virus produced at the end say of 18 successive inoculations was quite different from that originally extracted from human victims. It was not until 1939 when a way of infecting chimpanzees was found, that researchers were able to more closely

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[1] Paul, *op. cit.,* Preface. It also showed the unsavoury side of the commercialization of medical research which has been well documented elsewhere: e.g. Paul; Jane S.Smith, *Patenting the Sun.*

replicate the human disease.[2] The discovery of two different serotypes by MacFarlane Burnet and Jean McNamara, in 1930, was an important breakthrough, but progress in developing a safe vaccine was set back years by an unfortunate incident in 1935. Scientific rivalry coupled with public pressure to prevent a disabling and life-threatening disease led to premature human trials of two vaccines developed separately in New York and Philadelphia. The methods of production had been based on inadequate experimental evidence. The result was a disaster: eleven people died and the scientists involved disgraced.[3]

About the same time the National Foundation for Infantile Paralysis came into being in the United States, with the backing of the world's most famous polio victim, President Roosevelt. Using high powered methods of fund-raising (of which the annual March of the Dimes was the most famous) the Foundation poured money into research projects from 1938. The biggest breakthrough came in 1949 when J. Enders of Harvard University grew the polio virus on a non-nervous tissue culture. Until then it had been thought that the poliovirus could only be grown on nerve tissue cells. Monkeys had to be infected, killed and their spinal cords ground up; and there were simply not enough monkeys in the world to provide sufficient material to make a vaccine viable on a commercial basis. But Enders' discovery meant that production of a commercial vaccine was a feasible object. Enders received the Nobel Prize for his work. The necessity previously to inoculate live monkeys had seriously hampered studies into the effects of poliovirus. Many more scientists were now able to pursue the quest for a vaccine. Researchers were able to type hundreds of individual strains into three immunological types. The discovery in 1952 of viraemia (the presence of virus in the bloodstream) in the incubation stage of the disease was another breakthrough. As well as adding to the understanding of the pathogenesis of the infection, it meant that theoretically circulating antibodies could overcome small amounts of virus early in the incubation period, and during the minor illness stage. If the viraemia could be blocked, the body would be protected against the subsequent invasion of the central nervous system by the virus.[4]

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2 Paul, *op. cit.*, p. 250

3 Paul, *op. cit.*, p. 260

4 Paul, *op. cit.*, p. 389.

In 1952 Jonas Salk used formaldehyde to destroy the disease-producing ability of the virus while retaining its ability to stimulate resistance to the active virus. Backed by the NFIP, Salk now needed to test his vaccines in humans - and in the post-war period emotions still ran high over using humans, especially children, for any kind of medical experiment. However in 1953 Salk immunised a group of children with his vaccine and subsequent tests of blood specimens showed a satisfactory response. A field trial involving 1,800,000 American school children, (450,000 actually received the vaccine), began in 1954. Results showing a successful immunological response were released amidst great fanfare at Ann Arbor in April 1955.[5]

Pressures to release the vaccine to the general public were intense. The dilemma facing the authorities can be understood when it is realised that it could take twenty years to develop a perfect vaccine. In that time, in the United States alone, a million people would be affected by polio, and twenty per cent of those with paralysis. But the Salk vaccine involved a calculated risk. Salk's work was based almost entirely on laboratory experiments with animals. There was no assurance, official or unofficial, that the vaccine was effective against polio in a natural epidemic.[6] The NFIP and the six pharmaceutical manufacturers involved urged caution, but the American public clamoured for its widespread use. It took the tragedy of the paralysis of 44 immunised children fifteen days after the announcement at Ann Arbor to bring the public back to cruel reality. The faulty vaccine was traced back to one laboratory, Cutter, which had not followed strictly the procedures for manufacturing. The immunisation programme was temporarily suspended although Canada and Denmark, (two countries which suffered badly from epidemics), pressed ahead.[7]

These events in the United States were followed closely in New Zealand by both the Health Department and the general public.[8] The trend for epidemics to become more frequent and for older age-groups to become affected, increased the pressure on the

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5 J.S. Smith, *Patenting the Sun: Polio and the Salk Vaccine*, William Morrow & Co., New York, 1990, p. 371

6 Memo from Ambassador, Washington, H1, 131/9 25556, 2 April 1955

7 Paul, *op. cit.*, p. 391

8 Just how closely was demonstrated in Auckland following the problems with the Salk vaccine. Parents were put off giving permission for their children to receive BCG tests at school. At one school 30% of parents refused tests and vaccinations for their children - *Auckland Star*, 17 June 1955.

health authorities to introduce the vaccine as soon as possible. The Health Department was cautious: after all, the last epidemic had caused 80 deaths over two years. In the same period there had been 772 deaths from tuberculosis and 614 road deaths. Over the last previous ten years, two serious epidemics had caused 164 deaths while 6,547 people had died from tuberculosis and 2,408 people had been killed on the roads.[9] But the emotional impact of the polio epidemics was considerable. This can be gauged from the composition of the special Cabinet sub-committee - the Prime Minister, the Minister of Finance and the Minister of Health - which authorised the Health Department to purchase the vaccine following official reports from the United States proving its effectiveness[10]. If the United States Report proved favourable to Salk then the Committee recommended that all children up to 15 years be vaccinated within three months[11]. It was proposed that first of all two hundred medical students should be vaccinated and their immunological status then checked. Cost was inhibiting factor - it was estimated that the cost of buying vaccine for this trial would be about £2 per injection[12]. The Health Department was in regular correspondence with the equivalent authorities in Australia. One opinion came from MacFarlane Burnet who cautioned against too much haste. It was still unclear how long immunity would last, although the Salk vaccine was expected to be the best around for at least five years. It seemed that to maintain effective immunity with Salk, shots would need to be repeated every few years. Not only would this be expensive but there was also the possibility that repeated injections of monkey kidney protein used to grow the virus, could cause allergic states in the subjects[13]. Burnet mentioned that an important body of scientific opinion held that a truly effective and long lasting immunization could only be obtained by giving living virus vaccines by mouth[14].

A further cause for hesitation was the variety of Type I virus used in the vaccine. Salk was convinced that his method of vaccine production was 100% safe. Therefore, to get

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9 Memo from D-GH, H1, 131/9 26838, 31 October 1955

10 Memo to D-GH, H1, 144/17 25622, 19 April 1955

11 H1, 131/9 25556, 31 March 1955

12 H1, 144/17 25622, 2 April 1955

13 H1, 144/17/1 2629 35354, 18 October 1955

14 Letter from F.M. Burnet, H1, 144/17 25622, 30 April 1955

the best immunity possible, he used the most virulent Type I virus - the Mahoney strain - to prepare his vaccine. The British had suspended trials with Salk vaccine following the Cutter laboratory tragedy. They were now developing a killed vaccine using a less dangerous and more predictable Type I virus, Brunhilde. The New Zealand Government set up a special Polio Vaccination Committee to study the multitude of tests, trials and reports being produced overseas. This committee was headed by Sir Charles Hercus, Dean of Medicine and Professor of Preventative Medicine, at Otago University, who had had an interest in the disease since his early research attempts in the 1920's. Also from Otago University, were Professor J. Caughey, a neurologist and Professor J. Miles, a microbiologist. They were joined by Dr J. Mercer, a pathologist and Dr S. Ludbrook, a paediatrician. Hercus was very confident in the future of Salk and was keen to keep in close touch with the Commonwealth Serum Laboratories. He reported that the Canadians, whose Connaught Laboratories at Toronto University had supplied Salk with his viral material, were very confident. The Medical Research Council of Britain, he felt was behind the times in modern viral research[15]. By December, the Vaccine Committee was able to report that Salk vaccination programmes were being run in Denmark, South Africa and Germany. The United Kingdom, Sweden and France were carrying out trials. The Canadian results were particularly encouraging. Amongst vaccinated children the incidence of paralytic polio was 1.07 per 100,000, compared with an incidence of 5.39 per 100,000 in unvaccinated groups. Further in only one case of polio in 860,000 vaccinated children, could the vaccine possibly have been responsible. The committee was impressed with the WHO study group report on the effectiveness and apparent safety of the Salk vaccine.

The Committee also investigated the possibility of local manufacture but it would take a long time to set up suitable facilities and by then ample supplies would be available from overseas at a reasonable cost. Australia, where manufacture had started at the Commonwealth Serum Laboratories in August, looked to be the most likely source[16]. In January 1956 the Minister of Health, Mr Hanan, announced that the vaccine should

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[15] H1, 144/17/1 2629 35354, 21 July 1955

[16] H1, 144/17/1, 16 December 1955

be available "next year." The Government wished to investigate all aspects of the various vaccines before deciding on any one for New Zealand[17].

The WHO report recommended that countries with a high incidence of polio should introduce vaccination at an early date despite doubts over how long the immunity would last.[18] In April the Minister of Health, Mr Hanan, assured the country that vaccination would be made available just as soon as supply could be guaranteed[19]. Further evidence of the vulnerability of New Zealand to another major epidemic was provided by surveys carried out by two teams of doctors - including Doctor Caughey of the vaccination committee. Both these surveys - one done in Auckland, one in Dunedin - showed that 80% of New Zealanders under 30 had immunity to one of the three types of virus, but only 20% had immunity to all three. In Auckland 50% of those surveyed had immunity to Type I, 52.5% immunity to Type II, and only 24% immunity to Type III. The figures for Dunedin respondents were similar for Types I and II, but 47% for Type III. The 1952 - 53 epidemic was almost certainly caused by Type II virus, 1947-49 by Type I, and the 1937 probably by Type III. All three epidemics had swept through the country yet a very high proportion of the vulnerable population had missed becoming immune and would be susceptible in another epidemic[20]. Although the samples used in both surveys were small, the results were in line with similar overseas studies.

Having accepted the need for vaccination, and the safety of the Salk vaccine the remaining major problem was one of supply. The United States prohibited the export of any vaccine, the Australians had only just begun manufacture and had none to spare, which left Britain as the source of supply. A logistical problem was the need to keep the vaccine at 0o - 4oC. Once removed from refrigeration the vaccine remained viable for less than 24 hours. Refrigerators, either commercial or domestic, were still not universally available, nor was refrigerated transport. Special containers had to be made to transport the vaccine and arrangements were made with cool stores around the country to store them when the mass program started. By mid 1956 the decision to begin

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17 *Press*, 21 January 1956

18 *Press*, 28 February 1956

19 *Press*, 10 April 1956

20 J. Caughey et al., 'A Study of Immunity to Poliomyelitis in New Zealand', *NZMJ*, Volume LV #308, August 1956, pp. 281ff

vaccination of 5 - 9 year-olds had been taken. Although the rate of incidence in this age group (8: 10,000) was little different to that in the 0 - 5 group (6: 10,000) there were several reasons for making them the initial target. First, because these children were at school they were easy to organise. Second, this age group had an "indifference to personal hygiene" which made them good spreaders of infection, and thus indirectly could lower the incidence among the younger age group. Finally, the amount of vaccine available would be enough to cover this number of children[21]. The initial plans had to be modified when the first batch of vaccine intended for New Zealand failed tests in Great Britain. The start of the program had to be delayed, and limited initially to nine year-olds.

Consent cards were sent out to the parents of all primary school children in July.[22] The acceptance rate by parents was over 90% which was an incredibly high rate for any public health measure anywhere, and could be compared with a 29% acceptance rate in England, 42% in Scotland, and 74% in New South Wales.[23] The initial plans were for two injections to be given at a three or four week interval with a booster dose at least seven months later[24]. Parents were reassured as to the safety of the vaccine: "We have obtained our material from the Old Country where it is being tested for safety far more meticulously than in the USA." [25] Medical Officers of Health received numerous requests from parents groups to talk about the vaccine. Local press releases were made and radio talks given.

The first vaccine arrived in New Zealand on 20 September, 1956. Because the vaccine was scarce, expensive, and with a short shelf life, speed was essential. The organisation of vaccination teams in the Auckland Health district was typical of the way the Health Department organised the vaccination campaign throughout the country. Two teams, each consisting of a doctor, a nurse inspector, a nurse and a clerical inspector covered

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21 H1, 144/17 25843, 16 August 1956

22 There was never any question of making vaccination compulsory. It probably would have been illegal, and anyway in the case of polio it was hardly necessary: it is doubtful if compulsory vaccination would have achieved a better rate. The problem was to satisfy the demand from parents of children in the other age-groups.

23 Maclean, *op. cit.*, p. 325

24 *ibid*

25 Letter from Minister of Health, A373 H-HN 18/18/2

the metropolitan area of Auckland. Their visit to each school was preceded by another nurse who supervised the children and made preliminary arrangements. Children were sent straight back to the class room after receiving their shots. Parents were assured their attendance was not necessary. The second week of the campaign saw three teams going to schools on the North Shore, Thames, Pukekohe, Warkworth and Wellsford. The third week, a team went to Coromandel, one to Te Puke and one to Great Barrier Island. The fourth week was spent going back to any school missed earlier. In this way 15,584 children were vaccinated. The exercise was then repeated to give the second dose to each child.[26] Teachers were used to do the clerical work: distribute consent cards, separate out refusals from consents, parade the class in order on the day and keep the completed cards with school records.[27]

The first vaccination was given in Wainuiomata on 21 September 1956. There was widespread publicity throughout the country. Nearly every newspaper carried photos of children receiving their vaccination. The *Auckland Star* for instance, on 27 September, had a full page of photos of children baring their arms, nurses holding up hypodermics, and so on, as well as brave comments from the children about how little the needle hurt.

This initial campaign cost about £1 per vaccination. A cheaper supply of vaccine became available from CSL in Melbourne for about 7/- per dose. It was decided that while supplies of the English vaccine would be used to continue the vaccination of school children, the Australian vaccine would be used to vaccinate pregnant women, nursing staff, laboratory staff and house surgeons.[28] The decision to give priority to these designated 'at risk' groups was an interesting one. While it could be shown that non-immune pregnant women were highly vulnerable to paralytic polio, the same could not be said of the occupational groups. A survey done in 1950 investigating the 1947 - 49 epidemic showed that in fact very few nursing and no medical staff caught the disease. There had been no cases in hospital staff in Southland, Wellington, South Canterbury, Northland or Nelson. A total of 16 nurses were reported to have had polio during the

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[26] *N.Z. Herald*, 22 September 1956

[27] H1, 144/17/7 2629 35357, 30 June 1959

[28] H1, 144/17/1

epidemic. Of these, three were of doubtful diagnosis, and another three had had no contact with polio patients, leaving a total of ten nurses affected in the whole country and no medical or laboratory staff at all.[29]. The attitude of the hospital boards was interesting too. The Superintendent-in-Chief of the Auckland Hospital Board wrote to his Board that, while he was happy for his staff to be vaccinated, the hospitals could not be responsible for vaccinating pregnant women because of the extra work and associated record keeping which would place 'undue strain' on his staff.[30] By May 1957 all hospital staff in Auckland had been vaccinated but the number of pregnant women taking advantage of vaccination was small. This was partly due to the lack of publicity directed at a widespread group, and to the difficulty women had of getting to Health Department clinics on designated days.[31] In some health districts, a pregnant woman had to produce a special medical certificate to prove she qualified.[32]

Despite the lack of evidence as to their need for vaccination ahead of other members of the community, the British Medical Association made representations to the Health Department to get priority for medical practitioners, their families and medical students.[33] This was in fact done wherever there was 'surplus' vaccine. In July 1957, the decision was made to extend vaccination to all 9-16 year-olds, and then widen the campaign to vaccinate the 2 - 5 age-group as well. It was also decided to give the third booster dose. The cost of the vaccine had dropped to 4/6d per dose but the 1957 campaign still cost £170,000 at a time when overseas funds were low. An all-out effort was to be made in the next two years to cover the rest of the community.[34] The cost for 1958 was budgeted to be £400,000 of which £327,000 was in overseas funds. In April 1958, the Minister of Health in the new Labour Government asked if the campaign, and therefore the costs, could be spread out. The answer from the Director-General was masterly. The Health Department would be willing to slow down the programme but the previous government had promised to give the vaccination campaign urgency and priority.

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29 H1, 131/9 12c 31303, 15 May 1950

30 H740 532 62/3/4, 19 October 1956

31 H1 144/17 26133, 1 May 1957

32 H1, 144/17/7 2629 35357, 14 July 1959

33 H1, 144/17/1, 6 August 1957

34 H1, 144/17/1, 3 July 1957

If the number of polio cases increased, the Opposition might make political capital out of the Labour Government's failure to pursue the campaign with all vigour. The Government would in fact be gambling against an outbreak occurring. Finally, he reminded the Minister, polio was a very emotional issue.[35] The campaign went ahead as planned.

By Spring 1959, all children between two and 16 had been covered. In July, using the Plunket Society, kindergartens and playcentres, the parents of all children between six months and two years were contacted. Polio vaccine was added to the recommended vaccine for diphtheria and whooping cough at three months. Consent cards were done away with, as it was assumed that a mother at a clinic with her pre-schooler was giving tacit consent.[36] Clinics were held at Plunket rooms, primary schools, kindergartens, mass x-ray units and departmental health clinics. Some areas had open clinics for pre-schoolers. This caused chaos in Invercargill. "The weather was beautiful, everyone arrived at once." The result was pandemonium and letters to the press. A change was made to alphabetical grouping but there were still delays of up to 20 minutes. Finally a change was made to an appointment system. When it was found that the output per vaccinator was increased by 300%, an appointment system was urged on all medical officers.[37]

A group which proved much harder to contact were the 17 to 21 year-old age group. Response to advertising on radio and in newspapers was poor. A large number were due for booster doses while others were yet to have their first. Large advertisements were placed in the papers. Quarter page advertisements were placed in the *Auckland Star* and *New Zealand Herald* for two days in a row. A four day campaign was undertaken on the radio. In Auckland advertising spots were taken on the Morning Session, the Shopping Reporter's Session, the Midday Session, Marina's Women's Hour and the Evening Session. Written appeals were made to Businessmen's Associations, Rotary Clubs, the Public Service Commission, Railways, Post and Telegraph and other employers and

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35 H1, 144/17 26754, 9 April 1958

36 H1, 144/17 2629 35357, 30 June 1959

37 Memo from D-GH to all MOH, H1, 144/17 2629 35357, 3 June 1959

bodies employing people in this age-group.[38] Vaccination teams, usually consisting of a doctor, two nurses and a clerk were sent out to universities and teachers' colleges. A clinic was set up at the Canterbury Winter Show. An old T.B. hut was erected in front of the Post Office in Cathedral Square in Christchurch to draw attention to the campaign.

The summer and autumn of 1961 saw a rise in the cases of polio in the Auckland area, and it was apparent that another major epidemic was underway. As the disease spread to Northland, Bay of Plenty, Waikato and Poverty Bay, the demand for vaccination grew. The monthly clinic in Hamilton was giving 500 - 600 injections per month but when the first cases in the Waikato were notified in April, the number of injections given rose to 1,100 in May and 1,500 in July.[39]

The vaccine had not been generally available to adults outside the 'at risk' groups, and public health officials had claimed that the difficulties in extending the campaign were 'insurmountable'. However public pressure following the latest outbreak was so great that within three weeks of this claim, the Minister of Health announced that adults could receive a course of three shots for 15s a dose plus the normal consultation fee.[40] But despite the almost universal coverage of the country with the Salk vaccine, the epidemic was barely being contained and earlier plans to concentrate all respiratory cases in the northern region in Auckland Hospital were instituted. A report on the 1961 epidemic in Auckland showed 61% of those diagnosed as having polio, were over 15. Admission to Auckland Hospital included 85 adults, none of whom had been immunised. There was some degree of paralysis in 65 of these, and over a third of this number had some respiratory involvement - 16 in fact required assistance with breathing and one died. This was nearly as many paralytic cases as in the major 1952 - 53 epidemic. The average severity of paralysis was greater but advances in respirator treatment had greatly improved the survival rate.[41] In all there were 214 notified cases in New Zealand in

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38 H1, 144/17/7 2629 35357, 1 October 1959

39 H1, 144/17/7 2629 35357, 16 June 1961

40 *N.Z. Herald,* 7 June 1961

41 W.R. Lang et al., 'Poliomyelitis in Auckland: a Report of the 1961 Epidemic', in *NZMJ,* Volume #350, October 1961, pp. 450ff

1961. Seven people died - none of whom had been immunised. Of those who had been paralysed, only four had been fully immunised, and nine partially. None was seriously paralysed. The age-group who had received the greatest protection were the 11 - 15 age-group.[42] Clearly, although the Salk vaccine was keeping the most vulnerable age-groups protected, it was containing, rather than preventing, the disease.

Once the Salk vaccine had been shown to safe and effective, the NFIP was lukewarm in its support for perfecting an attenuated vaccine (using a live, but weakened virus). However the WHO realised the advantages of such a vaccine and took up the cause. By 1958 - 59, twenty field trials of attenuated vaccine had been held in 15 countries, and by 1962 all three attenuated strains had been released for manufacture.[43]

The difficulties with Salk vaccine had been apparent from the start, because each child required three or four shots administered by a health professional. Salk was suitable for economically advanced countries but not for developing countries. Further, the duration and efficacy of the immunity induced depended on the strength of the antigens used in the vaccine's manufacture, the number of doses given and the exposure of the child to natural poliovirus infection. The vaccine stimulated antibody production, but this took some time, particularly in the very young. Salk protected against the occasional individual getting a secondary invasion of the spinal cord. A 'live' vaccine on the other hand not only would stimulate antibody production, but because it mimicked natural infection, it would promote local resistance in the gastro-intestinal tract by stimulating local secretory antibodies. Immunity would occur in a matter of days rather than months. Furthermore, administration by mouth would obviously be a lot simpler and cheaper than injections. The live virus guaranteed a trivial intestinal infection which the majority of people would get anyway. The problem was to ensure that this infection was not virulent enough to give secondary spinal complications. The Department of Health had been aware all along that an oral vaccine was ultimately going to be used in New Zealand, but

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[42] *AJHR* 1963, H-31, p. 9

[43] Paul, *op. cit.*, p. 45

until a safe vaccine was assured, it was felt that the country should go ahead with Salk, and in fact the importation of live oral vaccine was prohibited.[44]

Following successful trials overseas with the Sabin oral vaccine, the Epidemiology Committee (which included Dr Neil Begg of the Plunket Society, Professor J. Miles, a world authority on viruses, and Professor C.Dixon, who had played a major part in the world campaign to eradicate smallpox) considered whether to continue with Salk or to switch to Sabin vaccine, which was the attenuated vaccine which had received WHO approval.[45] While Salk was safe and effective in anyone who had completed the course, it was not known how long this immunity would last, nor were those not injected protected. The Sabin vaccine on the other hand, gave a better antibody response for longer, but there would always be a small risk using a live vaccine that the virus used would revert to a more virulent form.[46]

The likelihood of an impending epidemic decided the Committee on undertaking a mass re-immunisation of all children over a four day period. Everyone would be reimmunised to make sure that there would be no reservoirs of infection[47]

The changeover began with the administration of Sabin vaccine to all infants up to 12 months old, from April 1961. In December 1961, the Minister of Health announced that all school-age children would be re-vaccinated with Sabin as soon as possible, but that in the event of an outbreak, oral vaccine would be extended to appropriate age-groups in an endeavour to smother the disease.

Emphasising the term 'oral vaccine' rather than 'live vaccine', the new campaign was launched in April 1962. The Health Department undertook to vaccinate all school-age children while the Plunket Society undertook to contact the more difficult to reach infant and preschool group. Society members tried to visit the homes of all these children. Clinics were organised, usually in Plunket rooms or dental clinics. Shuttle services of cars

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44 H1, 144/17/6 25356 2629, 9 April 1960

45 Besides Sabin, two other attenuated vaccines had been trialled widely : Koprowski and Lederle-Cox; see Paul, *op cit* p. 459

46 *AJHR* 1962, H-31, p. 9

47 N.C. Begg, *The Intervening Years*, John McIndoe, Dunedin, pp. 108-9

were arranged to transport mothers and children to clinics. A backup list of those who missed their clinic in April was compiled, and further clinics were arranged for July. The result was that over 90% of the one to five year old age group was vaccinated - a result unparalleled anywhere else in the world at that stage.[48] Similarly over 95% of school-age children were given the vaccine between 10 April and 19 April. The publicity was considerable. Screen slides were shown in cinemas and newspaper advertisements were run. On radio, four talks of six minutes each were given. The Governor-General spoke as a parent, Dr Turbott as the Radio Doctor, June Opie as a victim of the disease and Neil Begg as head of the Plunket Society. The papers even ran stories on the production of the 'distilled' water used to mix with the vaccine in the paper cups. In September and November the oral vaccine was made available to the entire population.[49]

By January 1963 over two million New Zealanders had received vaccination with Sabin. A study undertaken in 1962 showed that of children who had received both Salk and Sabin, 99.6% had antibodies to all three polioviruses; of those who had received Salk only, 89% were immune; of those who had received Sabin alone, 92%. However only 69% of those who had had no vaccination at all had complete immunity. Sabin had estimated that 70 to 80% of children, particularly preschoolers, needed to be resistant to break the pattern of infection.[50] If this was correct, then the health authorities were right in expecting another epidemic very soon, without a vaccination campaign.

The results of the campaigns were quite exceptional. In 1955-56 there had been 1,485 cases; in 1961 there were 214. Between January and March 1962 there were five notified cases. Once Sabin was widely used, there were no further cases, and there has been no reported case of polio in New Zealand since then. A similar pattern was noted in the United States (from 38,000 cases annually to 570 after Salk to 15 after Sabin), United Kingdom, Australia and other developed countries.

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48 *ibid*

49 H 740 532 H 62/3/4, 22 August 1962

50 Report of Dr W.Hamilton, National Health Institute, Wellington, H 144/17/6 25356 1962

It was one of the most remarkable, and swiftest, health achievements of the twentieth century. Yet by 1964, health authorities were worried that young children were not getting their second dose of vaccine at 12 months. Only one in four children under three had full immunity. The *N.Z. Herald* felt that forgetfulness rather than apathy was the reason for children not returning for their booster dose, and it was quite clear who was forgetting: "The Health Department, the Plunket Society, the medical profession - all will have to be more positive in reminding mothers of their obligations not only to their children but also to the community." [51]

The Plunket Society, which under Neil Begg had emphasised the Society's preventative role, was particularly disturbed by the number of children not completing their vaccinations. It was pointed out that children were getting their triple vaccine (diphtheria, tetanus, whooping cough) but not the Sabin. Triple vaccine was available from general practitioners but polio vaccine was given at separate clinics run by the Health Department.[52] The Health Department had originally controlled the import and distribution of polio vaccine for two reasons. The vaccine was expensive with a short shelf life and required great care during transport and use. Further, the use of Health Department staff greatly reduced the cost of vaccination. Had general practitioners been used, a general medical services benefit would have been payable as well. By 1963 orders restricting the import of Salk vaccine (which was still recommended for pregnant women), were lifted and Glaxo Laboratories were given monopoly of such importation.[53] However by 1967, virtually all the population had been covered and the only candidates for vaccination were young babies. The improved Sabin vaccines were more stable, so it was decided to make oral vaccine available from general practitioners, to be given along with triple vaccine at three months, from 1st April 1967. The Health Department continued to be responsible for importation and the hospital boards undertook to supply general practices.[54]

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51 Editorial, *N.Z. Herald,* 20 November 1964

52 *N.Z. Herald,* 21 September 1965

53 H1, 144/17/6 35356 2629, 22 April 1963

54 *N.Z. Herald,* 31 March 1965

The public acceptance of the vaccine was almost total. In the thirty years since immunisation began, rates of immunisation have dropped, in some cases to the 70% level that Sabin saw as critical. This would seem to be from ignorance or apathy, although a small but vocal group, the Immunisation Awareness Society, has vigorously opposed polio vaccinations. Their grounds for doing this range from the ignorant to the bizarre. With the use and misuse of statistics, and by making extraordinary links between polio vaccine and myxomatosis, AIDS and a multitude of other diseases, the Society argues that immunisation is dangerous, ineffective and that all diseases are preventable by good nutrition and good living practices. The fact that the epidemiology of polio makes nonsense of this is completely ignored.

A more serious attack on the immunisation campaigns came in *New Zealand Truth* in 1983.[55] *Truth* alleged that the first Sabin vaccine given in New Zealand in 1961 and 1962 was 'contaminated' with S.V.40 (a monkey virus which could possibly be responsible for tumours, genetic abnormalities and chronic illnesses) and that this had been the subject of a coverup by officials. A committee was set up to investigate the claims, and found that the S.V.40 virus was a known constituent of the early Sabin vaccines as a result of the method of manufacture. However S.V.40 had not been shown at the time, or subsequently to be pathogenic in humans. Nor had there been any attempt at concealment by officials, with all the relevant files being open for inspection. The Committee found that risks of the adverse effects of the vaccine - estimated at one in three to five million doses - had been weighed against the need to stop the developing epidemic, and had been found to be wanting. In conclusion the Committee found that not only had there been no negative effects but that the positive effects of mass immunisation included the disappearance of fear and concern about polio, a greater clarity of diagnosis of some other similar conditions, and a wider public acceptance of prevention as an option to the solution of some health problems.[56]

In the 1930's there were three main causes of crippling in children : tuberculosis, rickets and polio. In the 1960's the main causes of crippling in children were congenital

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[55] *Truth*, 2 February 1983, 9 February 1983, 17 February 1983

[56] *Report of the Committee* ..... p. 5

deformities and accidents.[57] Polio was the last of the childhood plagues to be defeated.[58] The children who received the first polio vaccinations were a privileged group: for the first time in history healthy babies could expect to grow up to be healthy adults. The childhood epidemics had all been conquered with better hygiene, nutrition, antibiotics and vaccination, and the world did not yet know of the threat of AIDS, pollution, ozone depletion or other biological and environmental threats to come.

The feelings of many in the 1960's - parents and the medical professions alike - were summed up by Dr Neil Begg: "it is doubtful if future physicians will feel greater elation and relief than we did when we found that the killing, deforming, the terrifying disease of polio had been banished by effective immunisation." [59]

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[57] CCS Annual Report 1974, p. 3.

[58] While smallpox is the only disease to have been eradicated by vaccination, polio has been effectively controlled by vaccination.

[59] Begg, *op.cit.* p. 111

CHAPTER NINE
POSTSCRIPT: POST-POLIO

The history of polio in New Zealand should have finished with the successful vaccination campaigns of the late 1950's and early 1960's. The elimination of polio from this country is one of the public health achievements of this century. No longer would a casual gastro-intestinal complaint, headache or stiff neck give rise to the spectre of a life-threatening illness. Certainly those who had been affected by earlier epidemics were still around, but they had been absorbed into the general health system. The damage caused by polio was considered complete once the acute stage was over, and stabilised once optimum function had been achieved, usually several months after the acute stage. Typically those left with physical disabilities exercised strenuously, overcame handicaps, ignored pain, and showed great determination and considerable success in overcoming their disability. The most common long term effects are the result of muscle paralysis and paresis, leading to skeletal deformities, joint contractures and disability of movement. Circulatory problems lead to growth retardation of the affected limb, intolerance to cold and venous stasis caused by the pooling of blood in paralysed legs. Trouble with breathing still affects those who had respiratory paralysis. As well the constant use of calipers, or a wheelchair could lead to compression neuropathy and pain from wear and tear resulting from compensating movements.[1]

But polio victims coped with these residual problems. Many achieved considerable success in a wide range of activities despite their disabilities, while others managed to disguise their physical problems altogether. The overwhelming feeling amongst most was that they had struggled long ago to overcome considerable difficulties, and had won the battle.[2]

But in the 1970's and early 1980's an increasing number of former polio patients began to report further loss of muscle function, in both the affected and apparently non-affected muscles, and unaccustomed fatigue and pain. Changes in life-style, often

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1 C. Le Boeuf, *The Late Effects of Polio*, Commonwealth Department of Community Services and Health, Adelaide, 1990, p. 13

2 Post-polio Survey

involving a change of job or early retirement, and the acceptance for the first time of mechanical supports, or the addition of others, were becoming more and more necessary amongst those who had recovered from polio. This was accompanied by a severe psychological blow with the realisation that the problems the person believed they had beaten many years ago, had in fact returned. Initially they were told by doctors that the symptoms were imaginary, or the inevitable and normal effects of aging. But as the reports accumulated, particularly from the United States, certain patterns began to emerge, and the medical profession was forced to take notice. The symptoms fell into two main categories: those involving muscles affected by the original disease, but which had recovered or at least stabilised; and those involving muscles not affected originally but now 'giving out'.

By the early 1980's a number of theories had been put forward to explain what was happening. One of these was that the poliovirus had re-activated. This resulted in some sensational headlines but did not stand up to the known characteristics of the virus. The poliovirus in fact disappears from the nervous system about three weeks after the onset of the acute stage.[3] A second theory, and the most commonly postulated, was that the syndrome was simply the result of the normal ageing processes. This however did not stand up to investigation either. Development of the syndrome did not relate to chronological age; rather it appeared approximately 30 to 35 years after the person had had their acute attack.[4] Other theories suggested metabolic fatigue or malfunction of nerve cells from burn-out or overwork, scarring of the remaining nerve cells, or changes to the supporting tissues of the spinal cord.

Gradually epidemiological studies began to give some idea of how many people were affected, and a re-examination of research studies long since forgotten, suggested some reasons for the syndrome. Delayed weakness in former polio patients had been noted in medical literature since 1875, but little notice had been taken. However as the survivors of the big epidemics of the 1940's and the 1950's in particular aged, more and more began reporting new pain, new weakness, and excessive fatigue. The best data

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3 G. Moir, *Report of the IVth International Polio and Independent Living Conference, St Louis, USA, 1987*, n.p. p. 5

4 N. Cashman, I. Siegel and J. Antel, 'Post Polio: An Overview' in *Clinical Prosthetics and Orthotics*, Volume 11 #2, 1987, p. 74

available from the United States (and for many reasons the methodology is not particularly reliable), suggests that 25% of those who were paralysed in the acute stage, were showing signs of post-polio syndrome.[5] A survey promoted by the New Zealand Post-polio Society attracted some 700 respondents, almost all of whom reported a deterioration in their condition, independent of their age. This would represent about 10% of the approximately 9000 notified cases of polio in this country this century. Most of those affected in 1916 and many from 1924 would now be dead, and by its nature the survey was not a comprehensive follow-up of all those who had been affected years ago.[6] Further, many of the cases notified in each epidemic were not paralysed at any stage. All this would suggest that the New Zealand figure is probably similar to that of the United States. A Mayo Clinic study examined 276 former polio patients. Of these nearly a quarter had significant symptoms: new weakness and excessive fatigue were noted by most, and nearly half had muscle cramps and muscle pain.[7] The Post-polio Survey in New Zealand showed similar results.

By the late 1980's, a more complete description of the syndrome was generally accepted. To be considered a candidate for the syndrome the patient had to have had a history of polio with complete or partial recovery, and a minimum of ten years in a stable condition. They had to show progressive muscle weakness without any demonstrable neurological disease. The symptoms and signs of the syndrome are largely neuromuscular, respiratory or vascular, the most common being severe fatigue, out of all proportion to the exertion involved, and new muscle pain and weakness.[8] Those who had had respiratory paralysis, though not necessarily needing mechanical assistance in the acute stage, can now find that they need respirators,[9] or suffer from various lung complaints, as the ageing process or infection pushes what has been borderline respiratory compensation over the limit.[10]

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5 B. Cheever and B. Garee ed. *Post Polio, an Accent Guide,* Cheever Publishing Inc., Illinois, 1987, p. 27. Post polio syndrome is also known as post-polio sequelae, late pot-polio and late effects of polio (LEOP).

6 Post-polio Survey.

7 P. Baker, 'Neuro-muscular Symptoms in Patients with Previous Poliomyelitis: a New Zealand Study' in *NZMJ,* Volume 102 #864, March 1989, pp 132-134

8 *N.Z. Disabled,* May 1989, p. 41

9 R. Cheever and B. Garee, *op. cit.,* p. 10

10 Cashman, *op. cit.,* p. 76

Current theory suggests that the most likely reason for the symptoms found in former polio patients is overuse degeneration of the neuromuscular complexes. Groups of muscle fibres are connected by an axon to a motor neurone. If that motor neurone dies, then the muscle fibre activated by that neurone, dies also, which is what happens when the poliovirus attacks the central nervous system. However rarely were all the motor neurones in a particular area killed. Motor neurones which survived a polio infection enlarged or sprouted new pathways, supporting up to eight times the normal number of muscle fibres.[11] The muscle fibres reinervated (restimulated) in such a way compensate by overuse, resulting in complete or partial recovery of muscle bulk and strength. This overload of 'good' muscles results later in the premature breakdown of those muscles. Until the development of electromyography, it was not possible to measure the full extent of muscle denervation. Muscle weakness is only clinically apparent when more than half the corresponding number of motor neurones have been destroyed.[12] Muscles graded as 'good', in fact might have 25 to 40% less strength than normal. It is still possible at these reduced strengths for the muscle to carry out normal activity, but it requires the muscle to work twice as hard.[13] This also meant that many muscles not thought to have been affected at the acute stage, were in fact damaged, and the surviving fibres have acted to compensate. The continual use of weak muscles also led to the damage of those muscles and secondary tissues.

It had also been the generally held belief that the only important damage to the nervous system caused by the poliovirus was the decrease in motor neurones. The interest in post-polio syndrome led to a revival of interest in the research of David Bodian in the 1940's. He found during post-mortem studies that there were viral lesions not just on the anterior horn motor neurons, but throughout the central nervous system, including the brain, even when there had been no paralysis. These lesions in the cortex, however, were confined to the motor and pre-motor areas. Because the cognitive processes were unaffected by the virus, polio survivors were able to achieve the same social, academic and professional attainments as their unaffected peers. But the widespread nature of

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[11] *ibid*, p. 74

[12] *ibid*

[13] *ibid*

these lesions is believed to account for some of the symptoms experienced in post-polio syndrome, most notably the exaggerated stress response and increases sensitivity to pain.[14]

A major problem for people with post-polio syndrome to overcome is recognition that their symptoms are real. Most doctors and physiotherapists who worked during the big epidemics have retired. Teaching about polio is virtually non-existent. To most health professionals polio is a disease of historical interest only. Because polio is a medical backwater with a diminishing number of survivors, it has been difficult to get funding for post-polio clinics attached to hospital neurological centres. These clinics, found in the United States, Canada and Australia, are staffed by teams of neurologists, physiotherapists and rehabilitation experts. It has also been difficult to persuade health professionals unaware of the syndrome that the normal rehabilitation programmes of increasing muscle work-load is quite inappropriate for already overloaded muscles. Exercise programmes need to be applied extremely cautiously, with enough exercise to prevent muscle atrophy but not enough to cause strain.

Besides pressing for recognition of the syndrome, the Post-polio Society aims to increase lay and medical awareness of the condition and to provide practical, emotional and psychological support for its members, many of whom have struggled for years on their own, with increasing disability.

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[14] R.I. Bruno and N.M. Frick, 'Post Polio Sequelae: Physiological and Psychological Overview' in *Rehabilitation Literature*, 1986 #47, pp 106 - 111

CONCLUSION

Over about five decades, a period less then a person's lifetime, polio had become evident, developed into a major epidemic disease, and was conquered. It is a story full of paradoxes. Nearly everyone who had had polio never knew, and of those who did, most recovered completely. Only about 9,000 New Zealanders were noticeably affected over that time yet polio was more feared than tuberculosis or scarlet fever, both major causes of mortality in children and young adults over the same period. A disease for which there was no cure, its cost to the country in terms of quarantine, hospitalisation and ultimately, prevention, was enormous.

When polio first became evident in New Zealand it was regarded with great interest by the medical profession as a new addition to the many infectious diseases which afflicted children at that time. Careful investigation showed that it did not fit the normal pattern of infectious disease but appeared regardless of public health measures such as sanitation and clean water. Unlike the United States, the health authorities here accepted from the beginning that the disease knew no social boundaries, and that rural rather than urban communities seemed more susceptible. It was children *per se* who were believed to be responsible for spreading the disease not any class, or for that matter, race. To the general public polio was yet another of the hazards of infancy which could blight their child's life forever.

By the time of the next epidemic in 1924, the country had experienced the tragedy of the 1918 influenza epidemic, and had put in place public health measures to cope with any future epidemics. These measures were applied in 1924 and 1925 as polio spread throughout the country. The population acquiesced to regulations that were extraordinarily wide in their scope, and which affected everyone and caused hardship to many. There were some complaints when quarantine affected a person's particular interest but few challenged the principle of the policy. The quarantine imposed proved quite useless in stopping the spread of polio yet it was still not questioned by either the public or the medical profession, or the health authorities. Indeed when the third major epidemic broke out in 1936 the same measures were taken. Again few questioned the efficacy of a nationwide quarantine. Schools were closed, people were prevented from travelling,

men were ostracised in their jobs if they had had polio in their family. But ten years later in 1947 people were not quite so docile. Perhaps greater affluence, and greater mobility, following the restrictions of World War Two caused people to be less tolerant of curbs on their movements and on their holidays. At the same time the increasing age of the victims called into question the long-standing belief that susceptibility to polio was age-related. The wisdom of mass quarantine was now being challenged by members of the medical profession as more and more evidence accumulated overseas about the disease's epidemiology. By 1952 the Department of Health's strategy had changed. Quarantine was imposed on individuals and their direct contacts only. Instead a national hand-washing campaign was launched. While this had little effect on an epidemic in progress, it did make more sense than the previous policy, if only to curb other gastro-intestinal complaints. The health authorities were faced with the difficult task of persuading the general public that what had been absolutely essential five years before, was now not necessary. There was general, if somewhat bewildered acceptance of the new policy.

This acceptance was partly the result of changes in treatment. In the 1920's and 1930's anyone paralysed by polio was given little to hope for. The prospect was of years in hospital, immobilised by sandbags or plaster casts, followed by a lifetime in calipers or on crutches. Orthopaedic surgery was about the only hope of some return to normal function, but it was costly and not always successful. Parents of hospitalised children faced large bills for their children's treatment, and the cost of this, or of massage treatment was beyond the reach of many. But by the 1940's there was some hope. At long last something could be done, and done immediately. Sister Kenny offered more than treatment: she offered hope, she offered results and she broke the log jam of medical control. The B.M.A. had successfully routed alternative practitioners like chiropractors, but Kenny treatment caught the public imagination and the entrenched views of doctors were forced to change. Because of her, physiotherapists came to play a major part in the treatment of polio patients, and the whole concept of the management of all kinds of paralysis was turned upside down. Free hospital care and out-patient treatment also removed much of the anxiety about illness from people's minds. Coupled with improvements in the 1950's in the use of respirators, these changes allayed some of the fear that polio aroused.

Changes in attitudes to disabled people owe much to the survival of paralysed polio patients. The Crippled Children's Society was first established largely to help children paralysed by polio. By seeking to establish these children as independent citizens, and acting as their advocates, the Society did much to advance the cause of all disabled people, and perhaps more importantly, change public attitudes.

The vaccination campaigns of the late 1950's were notable in a number of ways. First, was the almost total acceptance by the country of the need for vaccination, which was exceptional by world standards. It is doubtful whether compulsory vaccination would have achieved a higher rate. Equally exceptional was the speed and competence with which the campaigns were carried out, involving as they did the commitment by the Government of considerable expenditure, and the co-operation of health officials, teachers, employers, the media and parents. Third, the very success of the campaigns has jeopardised that success. The complete control of polio by 1962 resulted within two years of a fall-off in response to calls for vaccination. Since then parents have had to be reminded and persuaded to continue with vaccinations. Polio vaccination levels have fallen close to the 70 per cent level at which we can expect outbreaks of the disease. In view of the dramatic consequences that polio epidemics had on everyone, not just the victims and their families, such rapid collective amnesia is remarkable. Vaccination is the only defence against polio. If there were an outbreak of polio in New Zealand tomorrow, treatment would be no better than that given forty or even fifty years ago.

The fear engendered by polio was out of all proportion to its actual impact. But the level of fear does not relate to the actual level of danger. Polio was a bizarre disease - made manifest as paralysis of the nervous system, it was in fact an intestinal disease. When it was thought to be rare or unknown, it was common; when it seemed common it was rare. Those whom it paralysed did not fade away from a debilitating, wasting disease, but were as healthy as anyone else. At a time when infectious diseases were being overcome with disinfectants, sewers, good food and clean water, polio could not be controlled. It made a mockery of every public health measure and advance in standard of living. It appeared in the cleanest places and the healthiest children.

Epidemic diseases that affect the whole community are more frightening than chronic diseases like tuberculosis. An epidemic disease that kills its victims, leaves a reduced population to mourn its dead and then gradually to forget. But the community could not forget its polio victims. Most did not die; they lived, their wasted limbs, their calipers and their crutches a constant reminder, of the fate which could befall anyone. In a society where the rights of the handicapped were non-existent, where the disabled were seen as a source of guilt and shame, death was even seen as a preferred outcome to being condemned to a lifetime of 'crippledom'.

In the 1980's and 1990's it is inevitable that comparisons should be made between the fear of AIDS and the fear of polio. Any similarities are only superficial. From its first appearance in the west, the cause of AIDS has been known. The virus was identified early and the means of transmission discovered and made widely known. By being careful in one's lifestyle, AIDS is avoidable. Polio on the other hand was a mystery disease. Although its viral nature was long known the public was not generally aware of this. Its means of transmission was not realised for many years. Onset was devastatingly sudden. To most people, both medical and lay, its appearance was quite capricious, its advent unavoidable.

Polio struck randomly, and it struck children, the innocent. AIDS, on the other hand, affects adults, and as a sexually transmitted disease, carries with it all society's prejudices about sex. This is compounded by its prevalence amongst homosexuals. Acquiring AIDS through intravenous drug use is also obviously avoidable, and associated with habits repugnant to most people. Even the transmission of AIDS by way of blood transfusions to those whose lifestyles are not alien to the majority, carries with it our culture's taboos. Blood has always had connotations of life force, a mystical perception not attached to other body fluids like saliva.

In following the course of polio epidemics in this country, we can trace changes in our attitudes to infection and quarantine, to our attitudes to the physically disabled, in our treatment of paralysis and provision of health care. Polio in New Zealand has been conquered but not eradicated. If we allow our vigilance to falter, by forgetting the

history of this 'last of the childhood plagues', we could see its appearance once again in our community.

APPENDICES

APPENDIX 1

POLIOMYELITIS IN THE ANCIENT WORLD

An Egyptian Wall drawing dating from c1500 - 1350 BCP, showing a man with a withered leg, and his foot in the *equinus* position - deformities characteristic of an attack of paralytic polio.

Original in the Carlsberg Glyptothek, Copenhagen.

APPENDIX 2

TABLES SHOWING INCIDENCE OF POLIOMYELITIS

Table 1
Poliomyelitis, 1915 - 61: New Cases Notified, by Months[1]

Month Year	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Totals
1915	..	1	..	1	..	1	2	..	..	..	1	1	10
1916	119	319	320	167	44	19	9	5	4	4	4	4	1,018
1917	10	2	3	4	5	..	..	3	2	18	5	2	54
1918	1	1	1	1	..	1	1	..	..	..	..	..	6
1919	1	2	..	1	1	3	..	..	2	..	..	1	11
1920	2	1	..	17	14	7	5	2	5	10	4	9	76
1921	46	84	60	26	12	3	3	6	3	5	9	10	267
1922	21	20	21	14	4	5	3	2	2	3	2	1	98
1923	5	1	4	..	3	1	..	..	1	2	..	..	17
1924	2	2	..	..	..	1	1	1	..	2	5	59	73
1925	224	340	366	120	54	22	10	9	6	5	3	..	1,159
1926	..	4	4	1	..	..	1	1	2	..	1	8	22
1927	3	5	5	4	4	2	2	..	..	..	2	2	29
1928	5	5	11	11	4	1	..	1	3	2	2	2	47
1929	3	10	14	9	6	6	1	3	2	..	1	..	55
1930	1	3	..	..	1	1	1	1	1	1	..	2	12
1931	2	1	2	1	..	1	4	2	3	4	4	1	25
1932	12	31	39	23	8	6	1	8	5	2	7	8	150
1933	10	4	15	9	2	..	1	..	1	..	2	1	45
1934	..	3	1	2	2	..	1	1	..	2	1	1	14
1935	1	1	1	1	..	..	..	1	..	2	..	1	8
1936	..	..	..	..	..	1	..	..	..	..	1	85	87
1937	70	53	107	244	163	95	30	14	14	10	11	5	816
1938	9	1	3	8	1	..	..	..	..	..	..	..	22
1939	3	2	2	11	7	9	4	2	1	2	3	4	50
1940	5	5	3	..	1	..	2	2	1	4	..	..	23
1941	..	1	1	..	..	1	..	1	..	..	..	..	4
1942	..	1	4	3	4	5	1	..	3	3	5	2	31
1943	..	10	38	59	23	7	2	3	15	14	4	4	179

[1] These figures include Maori notifications where these have been listed separately

Table 1 (continued)
Poliomyelitis, 1915 - 61: New Cases Notified, by Months

Month Year	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Totals
1944	19	8	11	5.	1	..	..	..	..	..	..	1	45
1945	..	1	2	..	1	2	1	..	2	1	2	4	16
1946	26	23	9	22	14	8	6	4	..	1	..	..	113
1947	3	1	2	..	1	0	1	..	..	1	17	109	135
1948	55	43	76	96	117	63	85	85	117	77	80	69	963
1949	94	66	84	33	18	10	11	7	5	8	9	10	355
1950	6	9	18	5	5	9	3	2	4	1	7	3	72
1951	4	1	1	5	3	..	..	1	1	4	3	3	26
1952	1	4	7	12	6	15	50	75	121	198	192	209	890
1953	179	103	48	21	17	4	3	3	6	5	11	3	403
1954	2	3	9	2	3	5	9	1	2	1	2	4	43
1955	16	17	2	1	6	4	6	31	51	105	246	218	703
1956	226	218	171	106	42	20	16	10	9	11	17	11	897
1957	12	14	7	12	7	..	..	6	..	1	1	3	63
1958	5	7	3	1	2	2	4	1	9	8	10	5	57
1959	2	4	1	4	..	..	..	2	1	1	..	1	16
1960	..	1	..	..	2	..	..	..	..	..	1	..	4
1961	3	6	12	37	76	54	12	5	2	3	1	3	214

Source: : Department of Health Annual Reports, 1915 - 1961, *AJHR* H-31.

Table 2
Poliomyelitis 1908 - 61: Deaths Notified, by Years[2]

Year	Number of Deaths			Year	Number of Deaths		
	Males	Females	Totals		Males	Females	Totals
1908	1	2	3	1935	..	1	1
1909	1	2	3	1936	3	2	5
1910	3	1	4	1937	12	27	39
1911	1	..	1	1938	2	2	4
1912	..	2	2	1939	2	2	4
1913	..	2	2	1940	3	..	3
1914	16	9	25	1941	2	..	2
1915	2	2	4	1942	1	3	4
1916	76	47	123	1943	18	6	24
1917	6	4	10	1944	2	3	5
1918	2	2	4	1945	..	1	1
1919	1	..	1	1946	2	2	4
1920	1	1	2	1947	6	3	9
1921	8	3	11	1948	25	27	52
1922	6	3	9	1949	7	6	13
1923	2	..	2	1950	2	..	2
1924	8	14	22	1951	1	2	3
1925	91	82	173	1952	37	20	57
1926	7	4	11	1953	21	5	26
1927	6	1	7	1954	..	..	..
1928	10	7	17	1955	19	10	29
1929	5	2	7	1956	33	17	50
1930	2	3	5	1957	1	1	2
1931	4	1	5	1958	1	5	6
1932	12	7	19	1959	..	1	1
1933	6	2	8	1960	..	..	..
1934	..	2	2	1961	4	3	7

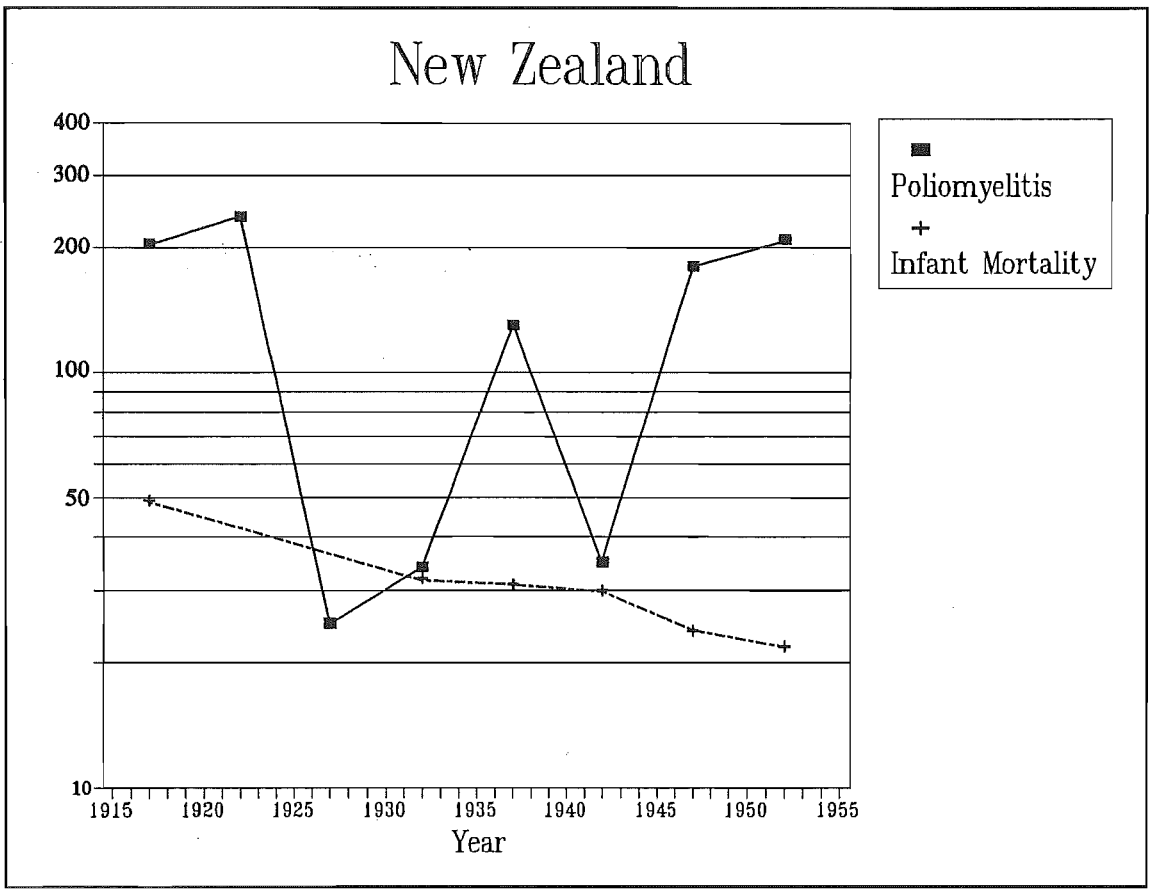
Source: : Department of Health Annual Reports, 1915 - 1961, *AJHR* H-31.

[2] These figures include Maori notifications where these have been listed separately

APPENDIX 3

FIGURES SHOWING THE INCIDENCE OF POLIOMYELITIS RELATIVE TO INFANT MORTALITY[1]

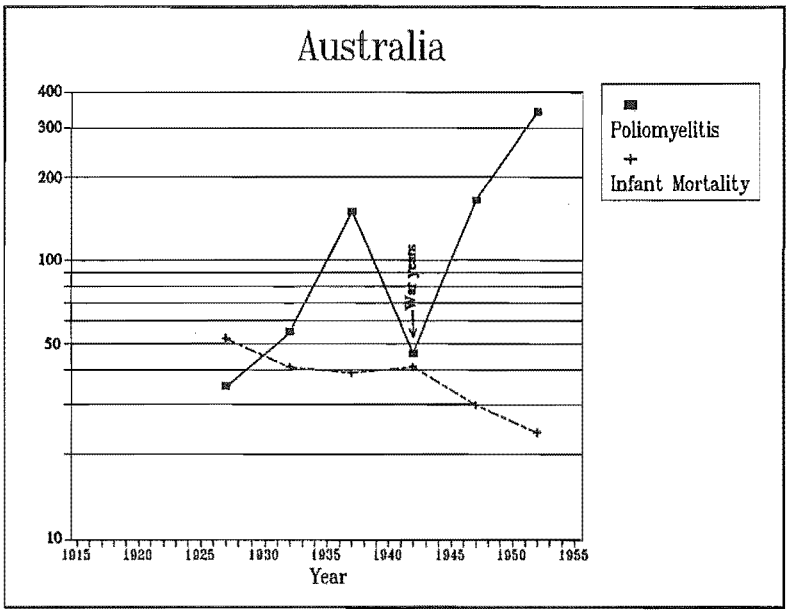
Figure 1
Poliomyelitis and Infant Mortality in New Zealand, 1925 - 1955



Infant mortality rate per thousand live births - 5 year medians
Poliomyelitis cases per million population - 5 year medians

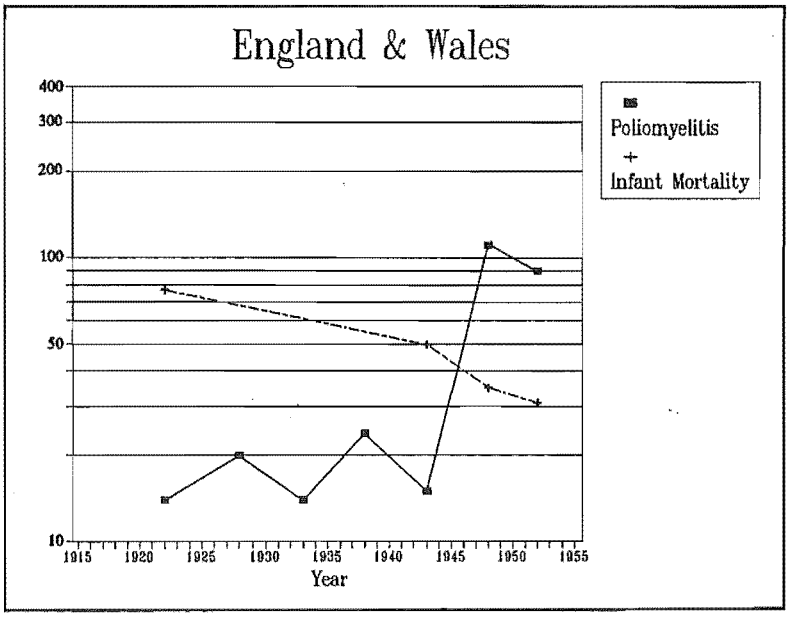
[1] These graphs are taken from Caughey et al., 'A Study of Immunity to Poliomyelitis in New Zealand', *NZMJ,* Volume LV #308, August 1956.

Figure 2
Poliomyelitis and Infant Mortality in Australia, 1925 - 1955



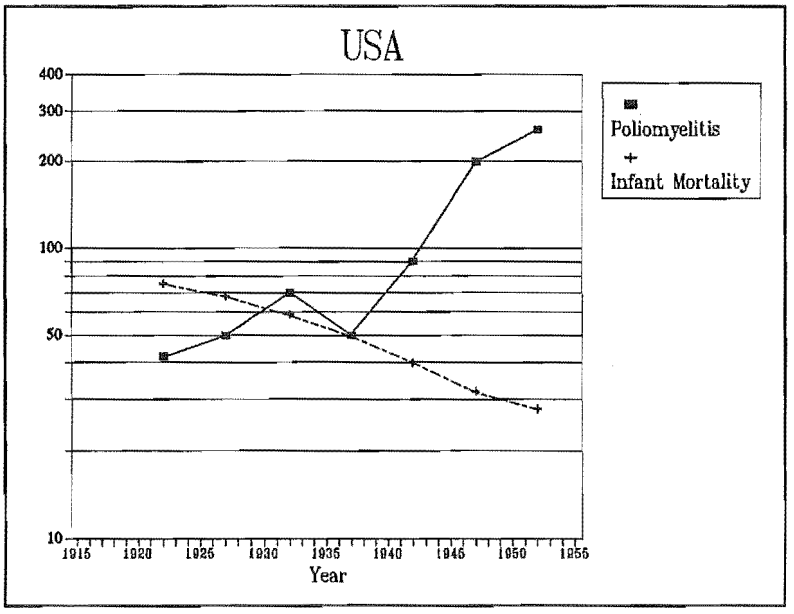
Infant mortality rate per thousand live births - 5 year medians
Poliomyelitis cases per million population - 5 year medians

Figure 3
Poliomyelitis and Infant Mortality in England and Wales, 1925 - 1955



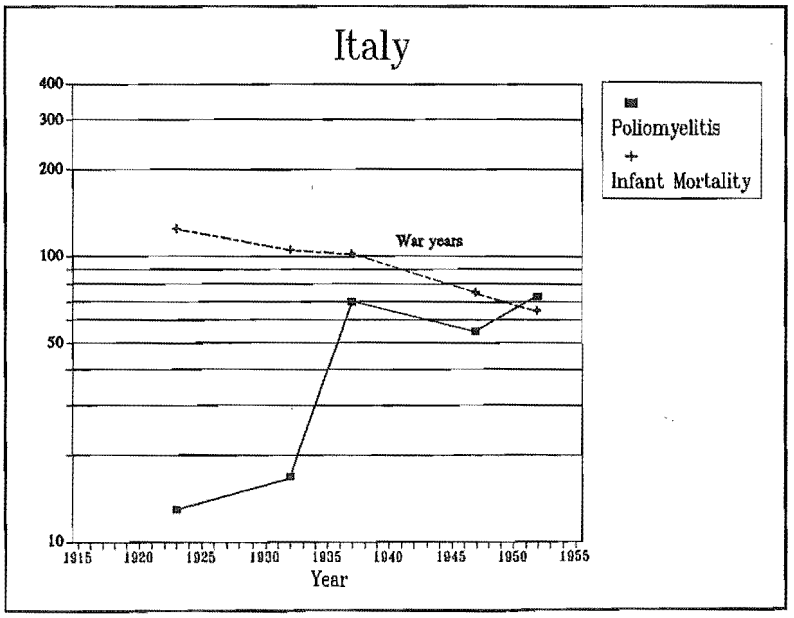
Infant mortality rate per thousand live births - 5 year medians
Poliomyelitis cases per million population - 5 year medians

Figure 4
Poliomyelitis and Infant Mortality in United States of America, 1925 - 1955



Infant mortality rate per thousand live births - 5 year medians
Poliomyelitis cases per million population - 5 year medians

Figure 5
Poliomyelitis and Infant Mortality in Italy, 1925 - 1955

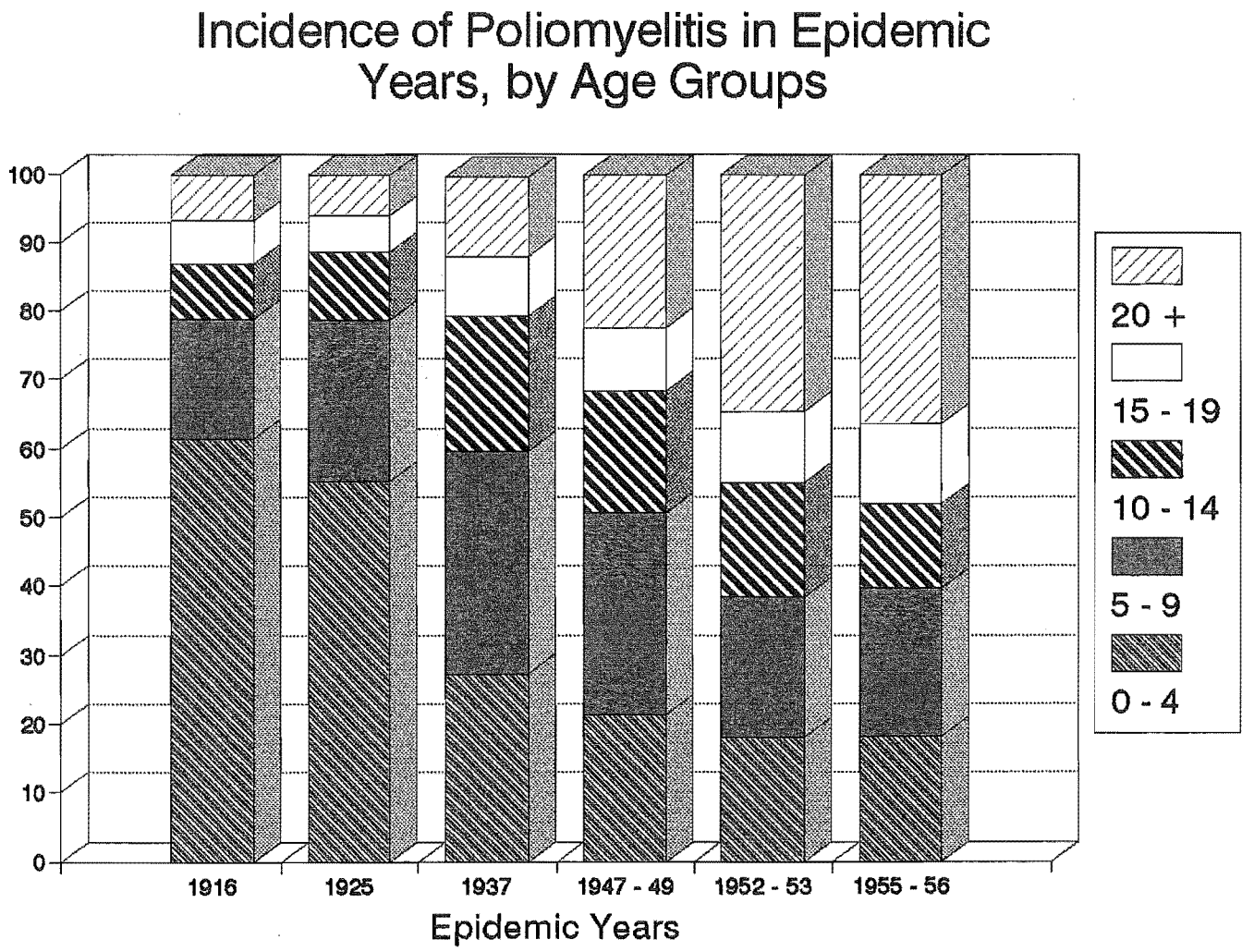


Infant mortality rate per thousand live births - 5 year medians
Poliomyelitis cases per million population - 5 year medians

APPENDIX 4

FIGURE SHOWING CASES OF POLIOMYELITIS IN EPIDEMIC YEARS BY AGE GROUPS

Figure 6



Source: F.S. Maclean, *Challenge for Health*, p. 327.

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131/9 B89-90	General 3711	1931 - 32
131/9 B89-90	General 3700	1932 - 35
131/9 B89-90	General 3701	1935 - 36
131/9 B89-90	Travelling 3702	1936 - 37
131/9 B89-90	General (incl reports) Dec 36 - Jan 37 3703	1936 - 37
131/9 B89-90	General (incl reports) Mar 37 - Apr 37 3704	1937
131/9 B89-90	General (incl reports) Apr 37 - Jun 37 3705	1937
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131/9/17A B92	Statistics	1925
131/9 9275	General	nd
131/9A 9276	Clippings	nd
131/9 9277	Suggestions by A Kent, Auckland	nd
131/9 9279	In Australia	1937
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