FROM
The Post-Polio Institute
and
The International Centre for Post-Polio Education and Research
postpolioinfo@aol.com

Cincinnati's Stealth Polio Epidemic< A Medical Whodunnit with Meaning for Polio Survivors.

Dr. Richard L. Bruno

Cincinnati, 1947. The summer arrives bringing heat, humidity ... and mounting fear. Polio is coming again. In 1946, America's worst epidemic ever had brought the death rate from polio to an all-time high. But as July became August and then September in Cincinnati, there was an eerie calm. While there had been 167 cases of polio during the previous summer, only 40 cases were reported by the end of August. Why were there so few cases? Had the "The Summer Plague" miraculously come to an end? Or was it attacking Cincinnati in disguise?

For at least four weeks during August and early September, pediatricians saw a new illness they called the "summer grippe." Its symptoms--headache, fever, stomach pain, nausea, sore throat and generalized aching--came on suddenly in children from 1 to 10 years old. The kids didn't get sick enough to go to the hospital, and saw their doctor once if at all. Albert Sabin, then a doctor at The Children's Hospital of Cincinnati, who was to develop the oral polio

vaccine a decade later, reported that there were at least 10,000 cases of "summer grippe" in Cincinnati and that "in some parts of the city hardly a child escaped."

Why was Sabin, a preeminent polio researcher, interested in the "summer grippe?" Because many of the children had a stiff neck, the red-flag symptom that required immediate hospitalization and often resulted in the terrifying diagnosis "rule out poliomyelitis." But since "summer grippe" symptoms did not lead to paralysis and disappeared within a week, pediatricians were not interested in hospitalizing children.

But Sabin was. He remembered that there had been an unusually mild polio outbreak in Denmark in 1934 when, although only 27 children were paralyzed, 600 more reported a "slight fever." He also recalled the unusual increase in influenza in Copenhagen during August and September, 1934, and the 100 cases of polio that came with it. Sabin wondered if a mild form of the poliovirus could have caused the "slight fever" and flu in Denmark in 1934 and the "summer grippe" in Cincinnati in 1947.

A Medical Detective Story

Sabin decided to find out. From August 22 to September 9, he admitted 13 children with "summer grippe" symptoms to The Children's Hospital. The kids had fevers of about 103 degrees, almost all were listless and had headaches, many had sore throats and most had stomach pain. These fluish,

feverish and sometimes fussy children had spinal taps and bodily fluid specimens taken. Eight were diagnosed with "summer grippe." Two had stiff necks and were diagnosed with "non-paralytic polio," one had "dysentery," another had a sinus infection and yet another had pneumonia. None of the children was seriously ill and they all left the hospital in about nine days.

With the children gone, Sabin returned to his lab to see if a poliovirus caused the "summer grippe." Remarkably, he found antibodies (cells produced by the immune system that render a virus harmless) to the Type II poliovirus (also known as the "Lansing strain") in the blood of five of the eight children with "summer grippe," in the child with sinus infection and in one of the two children who had non-paralytic polio. But Sabin knew that antibodies could have been present because the children had been exposed to poliovirus during the epidemic of 1946. So he followed the accepted procedure to prove that the Type II poliovirus had indeed been present in the children he exposed monkeys to specimens collected from his patients and watched the monkeys to see if weakness or paralysis developed. Then he performed autopsies on the animals to look for the damage to the spinal cord and brain that was the hallmark of poliovirus infection.

Specimens from "summer grippe" patients did indeed damage monkeys specimens from one patient paralyzed a monkey, specimens from three damaged motor neurons in

the spinal cord, and specimens from four damaged the brain stem neurons responsible for keeping the brain awake. Specimens from only one of the children who had non-paralytic polio paralyzed a monkey in spite of the child's lack of Type II antibodies. However, when Sabin exposed monkeys to specimens from seven different patients diagnosed during August with non-paralytic polio, only one monkey became paralyzed while the other animals had no evidence of nerve damage.

A Kinder, Gentler Poliovirus?

Sabin concluded that a mild or "low-virulence" Type II poliovirus caused the flu-like symptoms of the "summer grippe." Although his mild poliovirus did not cause even muscle weakness in humans, it did nearly twice the damage to monkeys' nervous systems than did the virus causing non-paralytic polio in Cincinnati that summer. What's more, Sabin's low-virulence poliovirus did something even the most virulent paralytic poliovirus did not do sicken at least 10,000 kids. At its worst, the paralytic poliovirus in Cincinnati felled only 167. Some mild virus!

Yet not everyone agreed that a kinder, gentler poliovirus caused the "summer grippe," not even David Bodian, editor of The American Journal of Hygiene, that published Sabin's findings. Bodian told Sabin in a letter that the evidence supporting Sabin's conclusion was "very far from being satisfactory" and that the paper would be "subject to serious criticisms."

Bodian, a Johns Hopkins pathologist, is the unsung hero of the polio vaccine. He had performed dozens of postmortems on humans who had contracted polio and scores of autopsies on monkeys intentionally infected with poliovirus. Through this work, Bodian discovered that the poliovirus entered the blood before it entered the CNS, making feasible the future Salk and Sabin blood-borne-antibody vaccines. Bodian also discovered that damaged to specific neurons in the brain stem and brain stem and spinal cord was the calling card of the poliovirus, and that as many as 60 percent of spinal cord motor neurons had to be killed by the poliovirus for any muscle weakness, let alone paralysis, to occur. Bodian should have been the one scientist to readily accept Sabin's claim that a low-virulence poliovirus not only caused "summer grippe" symptoms but also killed neurons, although not enough neurons to cause weakness or even a stiff neck. Bodian did not. Bodian wrote to Sabin that a "causal relationship" between the poliovirus and the "summer grippe" had not been proved, saying, "It is equally plausible to assume that the [polio] virus was found in accidental relationship with the illness." Sabin wrote back that bodily fluid collected during the same period from 24 additional patients with non-paralytic poliomyelitis did not cause nerve damage in monkeys, supporting his claim that he had isolated a poliovirus unique in that it caused "summer grippe" symptoms and damaged neurons in monkeys at a much higher rate than did the non-paralytic poliovirus floating around Cincinnati that summer. But Sabin could

not actually prove a causal relationship between his "stealth" poliovirus and the "summer grippe." "It is, in fact (a) matter of probable guilt by association," he admitted to Bodian.

Sabin was missing two important cards needed to trump Bodian's criticisms and support his claim that two different polioviruses were circulating simultaneously one card Sabin wouldn't hold until 1952; the final card wouldn't be dealt until 1955.

In 1949, Bodian himself discovered that it was not just the Type II poliovirus that damaged human neurons, but that there were three types of poliovirus that caused illness in humans, each requiring a unique antibody to render it harmless. So in 1952, Sabin tested bodily fluid saved from his 1947 paralytic and non-paralytic polio patients--but, unfortunately, not from the "summer grippe" kids--for the other types of poliovirus. He discovered Type I antibodies and concluded that the Type I poliovirus--not Type II--was responsible for paralytic and non-paralytic polio in 1947.

So Sabin was right there were two different types of poliovirus circulating simultaneously in Cincinnati that summer. But did the "high-virulence" Type I poliovirus cause "typical polio" while a "low-virulence" Type II virus caused the "summer grippe?" That answer would come years later and from thousands of miles away.

From the Heartland to Iceland

In September, 1948, three cases of paralytic polio were diagnosed in Akureyri, Iceland. And although no other cases of polio were reported, over the next few months more than 1,100 Icelanders came down with typical polio symptoms (fever, neck pain, muscle weakness and even some paralysis) as well as symptoms not associated with polio (tingling, numbness and "general tiredness." Many Icelanders who became I'll in 1948 still have fatigue and trouble with attention today.) Although fluid samples from four patients were sent to Bodian's laboratory for testing, neither poliovirus nor antibodies were found. Yet doctors in Iceland concluded that there were only two possible causes for what has come to be called Iceland Disease "Either a strain of polio [virus] of low virulence was responsible for this epidemic" or "some unknown [neuron-damaging] virus has been present."

Hard evidence for a low-virulence poliovirus did not come for six more years. 1955 brought an extensive polio epidemic to Iceland caused by the Type I poliovirus, along with two new outbreaks of Iceland Disease. Incredibly, not one case of polio was reported in towns with Iceland Disease in spite of the fact that only 7 percent of the children had antibodies to Type I poliovirus. Equally surprising, 100 percent of the children in the Iceland Disease towns had antibodies to Type II poliovirus. Just as Sabin thought happened in Cincinnati, children in Iceland had been exposed to a low-virulence Type II poliovirus that damaged their nervous systems and

caused symptoms of Iceland Disease. But, somehow, just the presence of Type II poliovirus prevented infection by Type I poliovirus without protective antibodies.

How could an infection by one type of poliovirus prevent infection by another? That answer came during the 1959 Singapore study of Sabin's own oral polio vaccine. Children were given all three live attenuated (totally non-virulent) polioviruses developed for the vaccine. Unexpectedly, the Type II poliovirus was found to be "dominant." Just as a flock of dominant blue jays stops less aggressive robins from roosting in your back yard, the dominant Type II poliovirus blocks all other polioviruses--even the naturally-occurring Type I poliovirus that was causing Singapore's 1959 polio epidemic--from entering the blood stream.

So children in Iceland and Singapore were protected from paralytic polio by a Type II poliovirus that blocked a high-virulence Type I poliovirus from entering their blood. And a Type II poliovirus protected the children of Cincinnati from paralysis by blocking a Type I poliovirus from entering their blood but at the price of the "summer grippe," a very much milder version of Iceland Disease. When the "summer grippe" epidemic was at its peak by the end of August, there were not more than 40 reported cases of poliomyelitis. Only after the "summer grippe" had left town by mid-September did polio cases start to increase, reaching a total of 170, the highest number in Cincinnati history.

The PPS Connection

Knowing that there are relatively "mild" polioviruses, that damage the spinal cord and brain without causing paralysis or even weakness, is important as we try to explain the cause of post-polio sequelae (PPS) today. Many doctors, some of whom actually treat PPS, believe that those who had non-paralytic polio cannot have new symptoms today because non-paralytic polio is thought not to be associated with nerve damage. For example, it is the failure and death of polio-damaged nerves that has recently been linked to new muscle weakness in paralytic polio survivors.

However the "summer grippe" proved that a "mild" poliovirus can damage and even kill neurons without causing paralysis. Nerve damage in "mild" cases of polio explains why studies by the National Institutes of Health and the U.S. Public Health Service found that new symptoms--fatigue, muscle weakness and pain--occur in 28 percent to 40 percent of non-paralytic polio survivors. It is also important to note that three recent studies found that at least 76 percent of paralytic polio survivors report new muscle weakness in limbs that were not weakened during the original poliovirus infection. Further, a 1953 study found that 39 percent of those (mis)diagnosed with "non-paralytic polio" actually had paralytic polio, as shown by their having weakness in at least one muscle. At the height of the epidemics, non-paralytic polio may have been a "diagnosis of convenience," given to mildly affected children by physicians overwhelmed by the sheer numbers of patients with severe paralytic polio.

So the story of the poliovirus and its effects on the body may be like the fable of the elephant and the blind men everyone infected by the poliovirus is left holding a slightly different piece. Doctors must remember that polio survivors' symptoms today depend, not on a diagnosis given 40 years ago, but on how many neurons were killed by the poliovirus in any given area of the spinal cord and brain. The more than one million North Americans who had "non-paralytic" polio must be assertive and advocate for themselves, showing doctors the research describing the varied effects of poliovirus damage to nervous system and making clear that non-paralytic polio survivors do develop PPS.

What is more, we have seen many patients in their 40's and 50's, with a "questionable" diagnosis of polio or non-paralytic polio, who report fatigue and trouble with attention today who have been diagnosed with CFS and told "there's nothing to be done." Further, there may be as many as 158,430 Americans around 50 years old who had "Summer Grippe," never knew it, and are now experiencing with CFS-like symptoms when they actually have PPS. So, the one to six million North Americans who had "non-paralytic" polio or "Summer Grippe" must be assertive and advocate for themselves, showing doctors the research describing the varied effects of poliovirus on the nervous system and making clear that "non-paralytic" polio survivors do develop PPS. "Summer Grippe" and "non-paralytic" polio survivors must follow the same prescription as survivors of paralytic polio to manage their new

symptoms listen to your body, stop activities before symptoms start and discard the "use it or lose it" philosophy and begin to "conserve to preserve" your remaining, poliovirus-damaged neurons.

References for the research studies described can be found at ftp://members.aol.com/harvestctr/Library/stealth.html.

See Bruno RL. "Paralytic Versus Non-Paralytic Polio A Distinction without a Difference?" (American Journal of Physical Medicine and Rehabilitation, 1999; 79 4-12) for a detailed explanation of the relationship between silent poliovirus episodes and late-onset fatigue symptoms.