The polio virus is in a class of virus called enterovirus. Three different types of enterovirus can cause paralytic polio. Type III, or Leon, was discovered in Los Angeles in 1937; Type II, Lansing, was discovered the following year in 1938; Brunhilde was discovered in Baltimore in 1939. People can get polio more than once, and, theoretically, it is possible to acquire all three strains of polio and have paralytic polio from each one.

For the majority of polio survivors, the primary portal of entry was through the mouth thus the name enterovirus. One of the three types invaded either the lymph glands, the respiratory system, or primarily, the gastrointestinal tract. During the first period of infection, some people experienced clinically flu-like symptoms such as diarrhea, a low-grade fever, or malaise.

For a large segment of the population, the polio infection ended at this point of only invading the gastrointestinal tract.

A certain segment of the population exposed to the polio virus went on to the viremia stage where new enteroviruses that duplicated in the gut cells spilled out into the blood. At that time, people had flu-like symptoms or even a stiff neck and achiness all over. But again, for a segment of the population the infection ended there. Some epidemiologic studies done before the vaccines show that 80% of the population had been exposed to one or more of the three types of enterovirus but did not have paralytic polio. In a small segment — about 20% — the virus crossed the blood brain barrier and entered the central nervous system.

The central nervous system includes the brain and the spinal cord, the connection between the brain and virtually every part of the body. On top of the spinal cord is the brain stem. It is the primitive part of the central nervous system that every animal has, including lizards, for example, and is responsible for the primitive day-to-day functions for living, including pulse rate, blood pressure, breathing rate, and the state of being awake or asleep.

Collections of nerve cells called cranial nerves supply special sensations primarily in the head. There are 12 cranial nerves that are responsible for motor, sensory, and autonomic functions such as coordinating vision, facial muscles, the tongue, swallowing. They provide the autonomic functions of the lungs, heart, kidneys, and intestine.

In the brain stem, the reticular activating system, or reticular formation, turns on to keep one awake and alert. The deactivating system functions while one is asleep and resting.

The cortex is that part of the brain which is overdeveloped in humans compared to other animals. The outside part of the cortex is “gray” because it is where the nerve cell bodies are congregated. The inside part of the brain is “white” because it is where all of the axons extend from the cortex (or the gray part) down through the brain stem to the spinal cord. These axons or connections send signals to other spinal nerves, and then to muscle fibers, for reflexes and movement. (See top panel, page 3.)

In those individuals in which the virus crossed the blood brain barrier, it infected the anterior horn cells. Anterior horn cells lie in the spinal cord running from the base of the skull all the way down to the tip. In the spinal cord, it was only the anterior horn cells that the polio virus infected. (See middle panel, page 3.)

A motor unit is a single anterior horn cell or motor neuron and all the muscle fibers it excites. For example, one anterior horn cell located at the bottom part of the spinal cord going to a calf muscle innervates about 1,900 individual muscle fibers. In the biceps, one anterior horn cell or motor neuron innervates
about 500 individual muscle fibers. For small facial muscles, the innervation ratio is smaller — one nerve cell to 100 muscle fibers. In little eye muscles, one motor neuron innervates about five individual muscle fibers. So there is quite a spectrum of how many muscle fibers are innervated by a single motor neuron.

For individuals who had the infection in the spinal cord, clinical signs showed in legs, trunk, arms, and sometimes diaphragms. If the infection was high up in the spinal cord, it affected the neck or arms, and the day to day breathing muscles, including chest muscles. During the infection individuals experienced fever, paralysis, weakness, and, frequently, considerable muscle pain.

The cranial nerves could also be involved resulting in double vision, weakness of the face muscles, difficulty in swallowing, moving the tongue, difficulty breathing, and even coma stupor, due to the reticular activating system being affected. In the cortical form of polio, usually characterized by high fever and seizures, people did not survive.

In the bottom panel on page 3 (Figure A), two anterior horn cells or motor neurons have been infected by the polio virus and the third is temporarily stunned. At this point the onset of weakness occurred.

Figure B shows that after a period of weeks two of the anterior horn cells affected by the polio virus have died and left some muscle fibers orphaned or denervated.

There were at the same time white cells cleaning up the dead neurons and releasing toxins that temporarily stopped other anterior horn cells from working. With these cells stunned, or neurapraxic, and some completely destroyed, individuals experienced complete paralysis. After a few weeks, the neurapraxia would begin to wear off and individuals who had complete paralysis of the muscles or muscle groups started to get some flickers of movement when the neighboring nerve cells that were stunned started to function again.

Additionally, after a period of rest, Kenny packs, stretching to avoid contracture and tightness, and depending on how much denervation occurred, one got stronger. This strength was due to the surviving motor unit getting a little stronger.

Just as importantly, over a period of months and even up to several years, a phenomenon called "sprouting" occurred. Surviving muscle fibers sent out some sort of chemical distress signal that caused the adjacent surviving motor neurons to send out tiny little sprouts to innervate orphaned muscle fibers. From studies done on monkeys with polio, we know, depending on how many anterior horn cells were lost, the surviving cells could re-innervate up to three to four times as many muscles fibers as originally. And that is shown in Figure C.

Many individuals got stronger, not just from exercise alone, but from the sprouting process or re-innervation process which did seem to remain fairly stable for about 20 to 30 years.

Stanley K. Yarnell, MD

After the acute episode, polio survivors went through rehabilitation, multiple surgeries, wheelchairs, bracing. With hard work survivors became very productive individuals, raising families and holding jobs. Then 20, 30, 40 years later many noticed they were not functioning as well as before.

Some survivors reported inordinate fatigue. At the end of a work day, they were often exhausted. Recurrent aches and pains often developed in different parts of their bodies. Some survivors noted functional decline such as trouble mowing the lawn, doing heavy household chores, or even meeting some of their basic self-care or mobility needs.

Some people began to notice trouble breathing, more respiratory infections, trouble sleeping, increased irritability, decreased concentration, and trouble swallowing.

To illustrate this state, I would like to read a letter that was sent to Gazette International Networking Institute (G.I.N.I.) in 1979 by Larry Schneider. His comments were the catalyst for G.I.N.I. and others to realize this was not an isolated occurrence.

"As everyone grows old it is natural that physical limitations increase. With polios, the limitations come much earlier and, in these days, it is necessary to cope to the best of our ability and without much assistance from the 'medical profession.'"
The central nervous system is composed of the brain and the spinal cord.

The polio virus infected the anterior horn cells, thus affecting the skeletal muscles causing partial or complete paralysis.

A motor unit is composed of a nerve cell and all the muscle fibers it innervates. The neuromuscular junction is the junction between the nerve cell and the muscle fiber.

**Figure A**  
*During infection*  
Two of the five nerve cells have been infected by the polio virus. The middle one has temporarily stopped functioning.

**Figure B**  
*A few weeks later*  
The middle nerve cell has recovered. Two nerve cells

**Figure C**  
*Months, even years later*  
The surviving nerve cells "sprouted" to innervate the
"It’s now 40 years since I contracted polio at 17 and I was fortunate in being able to go to Warm Springs, Georgia. Later, whenever I had a problem or question it was the place to turn to. I could obtain a complete check-up to include special considerations given to the after-effects of polio. But with the switch of the March of Dimes, this is no longer true.

"During the past few years, I find myself being able to do less and less and tire far too easily. This led to my early retirement when I was no longer able to stand from my wheelchair by myself, and no longer able to walk with crutches or get out of bed alone.

"Since my last examination over 10 years ago, I have visited different doctors, wherever I happen to be living, with only very minor ailments. To each doctor, I was a strange and different individual because he was totally unfamiliar with polio. They were all more interested in learning from me than with giving me any worthwhile information. Most of them looked at me and said I would be a good ad for polio shots.

"As far as I know, there is no longer one center like the old Warm Springs Foundation which has answers to polio after-effects and aging.

"I would, therefore, like to suggest that readers of Rehabilitation Gazette, which seems to be the last polio link, submit names of doctors they know in their home town who can easily relate medical problems, (perhaps heart strain, low blood pressure, too much or too little exercise, etc.), to polio problems. In this way, a national directory could be established listing those experienced and ‘sympatico’ doctors to whom we could relate and turn for genuine and honest advice to ease our transition in these passing years."

Martin B. Wice, MD is assistant medical director of the department of rehabilitation medicine at St. John’s Mercy Medical Center, St. Louis, MO. Dr. Wice is president of the Board of Directors of G.I.N.I.

In addition, the medical community took a cue from Larry Schneider who was very critical of health professionals for ignoring the needs of individual polio survivors. Studies were begun on the changes taking place in some polio survivors. One survey was done by Lauro Halstead, MD, now at National Rehabilitation Hospital in Washington, DC.

Dr. Halstead defined post-polio syndrome in the following manner. An individual must have had polio. This may seem like a minor point, but in actuality I have had many people come to my clinic who never had polio. It is a very difficult situation because they are often quite adamant about it. Ideally the way to prove an individual had polio is to obtain the original hospital record. Also, we need to talk to the individual, but many times he or she was too young to remember the original polio episode. The next option is to review this with his or her parents, but quite often they have already died. If a history of a febrile episode, asymmetrical paralysis with sensory sparing, and some type of motor return can be documented, the individual most likely has a history of polio. Electromyogram findings, by showing the presence of a chronic neuropathic process, also can give an indication that someone may have had polio.

The next part of the definition requires neurologic stability for 15 years after which one experiences two of the following health problems: unaccustomed fatigue, a period of muscle aches and pains, new weakness in the muscles previously affected, or even in what were thought to be unaffected muscles functional loss, cold intolerance, or new muscle atrophy. It is also critical that there be no other medical diagnoses to explain these new problems.

Who gets the post-polio syndrome? Dr. Halstead's survey found that those who had more severe polio, those that were hospitalized, had four limbs paralyzed, spent time on a respirator, and were older when they contracted polio, tended to get the post-polio syndrome earlier.

There are other studies which also suggest that people who had more serious polio and were older when they had their polio are more prone to actually get the post-polio syndrome. As a clinician, on an individual
What is the incidence of the post-polio syndrome? It partially depends on how you define it. A study done by Anthony Windebank, MD, of Mayo Clinic, of Olmstead County, the county where Mayo's is located, found a 22% incidence. Another study showed a 41% incidence. Typically a 20 to 60% incident is quoted. Although, again, depending on how you define it, it could be as high as 89% or even 100%.

What is the cause of the post-polio syndrome? This is not known at this time. However, different theories will be discussed next by Frederick M. Maynard MD.

How does one assess an individual for the post-polio syndrome? The first thing I do when I examine polio survivors is to get a very detailed medical history from the time they had polio through their entire recovery process and then their new decline. I conduct a detailed physical examination, including a neuromuscular and functional evaluation. Tests are performed not so much to prove one has the post-polio syndrome (no test can do this), but to rule out other problems which can masquerade for it. Tests include blood work (including a CBC, chemistry profile, thyroid screen, CPK, and ANA), and a vital capacity (a breathing screen).

If I have cause for concern about breathing, I request a blood gas, which checks for oxygenation and for CO₂ retention. I may request a sleep study. I may also do a special barium “cookie” swallowing study, if swallowing is a problem. I may do electrodiagnostic testing of the arms and legs, not to prove that someone has the post-polio syndrome, but to look for other problems — carpal tunnel syndrome, radiculopathy — which may explain someone's symptoms. I also may request appropriate radiological testing such as x-rays of joints that are hurting to look for arthritis or for fractures. I may request an MRI scan of the spine to make sure nothing else is masquerading for the post-polio syndrome.

Other tests can be requested depending on what is found during the physical examination. Depending on the results of the above-mentioned tests, I will arrange appropriate referrals.

What is the course of the post-polio syndrome? Many individuals are terrorized that they will re-live their original polio episode or will end up in a nursing home. Neither is the case. With proper management polio survivors can live long, productive lives.

How does one manage the post-polio syndrome? This will be discussed in detail during the remainder of the conference.

Martin B. Wice, MD

There is now a clear consensus from electrophysiological studies that post-polio muscles in people that had any residual weakness do show signs of partial denervation and reinnervation. “Innervation” means that there is a nerve supplying the muscle. “De” before the “nervation” would imply a loss of nerve supply to that muscle and the muscle is said to be denervated. “Reinnervated” implies that the nerve was destroyed and it has grown back again. Among post-polios there are usually some muscle fibers in a state of denervation and others in a state of reinnervation. Even in a person whose strength is stable, there is constantly some new denervation and reinnervation occurring.

Polio survivors have an increased motor unit size. The result is that any one nerve cell must supply two/three times more muscle fibers than normal. These giant motor units appear to be under great metabolic stress from the constant high rate of use and muscle fiber turnover (from denervation/reinnervation). Thus, they are thought to be fragile and subject to degenerative fragmentation. Additionally, some animal and clinical studies have shown problems at the neuromuscular junctions of those motor units as survivors grow older.

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Therefore, one theory proposes that much of new post-polio muscle weakness is a result of failure of reinnervation. In other words, the partially denervated and reinnervated muscles that have been in balance for many years may suddenly have something trigger a change in their balance. Reinnervation of muscle fibers may stop happening as fast as denervation occurs. This leads to a loss in total functioning muscle fibers and weakening.

Bruce R. Pachter, PhD, a neurophysiologist at New York University Medical Center, conducted a series of experiments on rats in which one of the two nerve roots supplying the rat's leg muscle was cut where it comes out of the spinal canal. This procedure does not kill the nerve cells like the polio virus did, but it does disconnect many muscle fibers from their nerve supply. The result is some of the rat's leg muscles lose half of their nerve supply. These muscles are then half innervated and half denervated. One month later the
twitch tension, which is a measure of strength, and the tetanic tension, which is a measure of endurance, were both reduced, as expected.

But at three months, the twitch tension had returned to what it was before the nerve damage. Because three months is too fast for a nerve cell to re-grow from the spinal canal, the conclusion is that reinnervation (or sprouting) by the remaining nerve cells must account for the recovery of strength. However, at three months the tetanic tension, or the endurance of the muscle, was still reduced.

At six months after nerve damage, there was no difference in the twitch tension or the tetanic tension between the muscles that had lost half of their nerve supply and the ones that had not. This parallels the experience of polio survivors when they seemed to make a full recovery of strength over six months to two years after acute polio.

At nine months after nerve damage, the strength of the rat's leg muscles started to decline. When the rats were then sacrificed, their muscle biopsies showed abnormalities that were similar to the appearance of 20-24 month old rat's muscles. In other words, the nine-month old rats with nerve damage had muscles that looked and responded like "old" rat muscles (this strain of rat becomes old and usually dies at 20-24 months of age). Pachter concluded from his experimental rat model that some late changes in post-polio muscles are similar to accelerated aging.

Another major theory for declining strength is chronic overuse damage. Overuse is a funny term. There is a normal amount of muscle use which maintains a stable level of strength. If one does less than this, muscles will atrophy. It happens to all of us in our 40s or 50s that when we slow down and become less active, we get a little bit weaker. On the other hand a lot of vigorous activity and pushing the maximum, makes us stronger. This is not considered overuse, it is considered strengthening exercise, which can be healthy. If one wants to maintain near maximum strength and endurance throughout their lifetime, one has to remain physically active. However, it is a fine line between doing the optimal vigorous activity, and the point where it is potentially damaging. In fact, some degree of microscopic damage to muscle fibers must occur after resistive exercise in order to stimulate and trigger the body's reparative processes and produce greater strength.

Overuse is a very tricky concept. High use may be very good, and overuse is only bad when it causes damage. It remains unclear to what degree overuse damage contributes to the typical symptoms of post-polio syndrome.

James Agre, PhD, MD, University of Wisconsin, Madison, WI, has done some sophisticated studies on responses to exercise among polio survivors. One of his findings was that symptomatic post-polio people were weaker and showed evidence of greater initial polio involvement compared to asymptomatic polio survivors. The worse the original polio and the greater the initial degree of residual weakness, the more likely post-polio persons are to become symptomatic later in their life. This idea is common sense, and studies support it.

He also observed that endurance time was the same for symptomatic and asymptomatic polio survivors when the actual degree of strength was accounted for. People who are weaker had less endurance if the work of the task was the same. Similarly people with the same amount of strength will have the same amount of endurance, even when comparing a non-polio person with a post-polio person.

However, Dr. Agre found that recovery of strength from exhaustion was a little prolonged for polio survivors. This supports the experience of polio survivors that it takes a little longer for them to recover from an exhausting bout of exercise.

Dr. Agre has now followed some people for three years, and the polio survivors did not show an accelerated loss of maximum isometric strength. He did find a slight decrease of strength over three years, but it was exactly the same rate as in the people without a history of polio as they became three years older. He concluded that there is no evidence for saying the loss of muscle strength in post-polios occurs at other than the normal age-related rate for loss of muscle strength.

Other conclusions from Dr. Agre's and other researchers' exercise studies are that some post-polio muscles can increase their strength with proper strengthening exercise, and that some patients can improve their fitness with cardiovascular fitness exercises. Interval training and pacing are particularly critical to the success of fitness exercise programs in post-polios. A post-polio person undertaking a fitness program should be sure to take frequent rests or "time-outs." For example, ride an exercise bicycle for three or four minutes; then take a half of minute or a minute rest; then exercise for another three-four minutes and rest again until at least 20 minutes of activity has occurred. This is the way to successfully achieve...
Jacquelin Perry, MD, from Rancho Los Amigo Medical Center in Downey, CA, has done some very elegant biomechanical studies on asymptomatic and symptomatic polio survivors. She studied polio survivors with some residual weakness in their legs and compared those who were reporting the same leg strength now with those who were reporting greater weakness. Using a gait laboratory, she found that the symptomatic people were, in fact, using their muscles at much closer to the muscle's maximum strength on a continuous and repetitive basis compared to the asymptomatic people. Her work supports the notion that people with symptoms are pushing themselves to use their muscles on a regular basis near the maximum limit of their peak strength and their peak endurance.

Anthony Windebank, MD, from Mayo Clinic in Rochester, MN, showed in an epidemiologic follow-up study that symptomatic post-polios had more extensive initial polio involvement than asymptomatic post-polios. This is essentially what Dr. Perry showed. The more muscles and limbs with severe polio involvement, the more likely a polio survivor will become symptomatic.

At this point, I would like to mention the work of Dr. James Beasley, a PhD physical therapist during the 1960s. He performed quantitative strength measurements on muscles of various grades using manual muscle testing. He found that the maximum strength of muscles tested as normal in polio survivors, as compared to non-polio survivors who also tested normal, was only half as great. His work showed that manual muscle testing is not capable of distinguishing weakness of this degree, and more than half of normal maximum strength must be lost before people notice weakness in daily living activities (e.g., walking). Many post-polios who thought themselves, or were told by their doctors and therapists, that they had normal muscle strength and full recovery, in fact did not.

Related to the underestimation of severity of chronic post-polio weakness is my "rule of thumb" or axiom for which polio survivors are likely to lose which functional ability. It says that the abilities requiring the greatest effort to achieve in the original period of post-polio rehabilitation are the first ones to be lost as a polio survivor becomes older.

Therefore, I believe the bottom line about exercise and overuse for polio survivors is that a balance must be maintained. If one does too little, one may experience disuse atrophy and become weaker. If one uses muscles in unwise ways and in an unwise fashion (e.g., too long and too hard), one can experience overuse damage with pain and loss of strength. Only with the proper balance of sufficiently strenuous exercise and prudent restraint can aging polio survivors avoid symptoms of post-polio syndrome.

Since the late '70s, immunological theories have been considered. Could weakness be triggered by a change in the immune system? The original studies found few immunological abnormalities in polio survivors. However, a study was published by Mohammad K. Sharief, MB, ChB, et al, in 1991, which caused considerable new controversy. The study found cerebrospinal fluid antibody abnormalities in symptomatic survivors, but not asymptomatic survivors. It concluded that reactivation of the virus could not be ruled out.

In April of 1994, the National Institutes of Health, in conjunction with the New York Academy of Sciences, sponsored a three-day symposium that included some of the best virologists, neurophysiologists, and pathologists from around the world. They concluded that, at this point in time, there is still no evidence that the polio virus becomes reactivated and produces new symptoms. They did review some intriguing, unexplainable immunological abnormalities that apparently occur in a few polio survivors. However, no one really knows what they mean, and it is unclear if they are more common among the people who are getting worse, let alone causing their symptoms.

Continued on page 8
Another theory about the cause of new weakness involves hormonal changes. Research, led by Kaup R. Shetty, MD, and D. Rudman, MD, (now deceased) at the Medical College of Wisconsin in Milwaukee, suggested that the lack of growth hormone is responsible for the rapid course of new weakness in some polio survivors. Growth hormone is normally secreted, even in adults, by a small gland at the bottom of the brain, the pituitary gland. Growth hormone is important for the health of muscles and may be particularly essential to the precariously balanced metabolism of denervated/reinnervated polio muscle.

The Milwaukee researchers identified a syndrome of “growth hormone arrest” in which 50-year-old people experience early aging and take on characteristics of 80-year-old people. When they studied polio survivors, they found low growth hormone levels in nine of ten symptomatic survivors compared to normal levels of growth hormone in asymptomatic stable polio survivors. Unfortunately, their research on growth hormone replacement in normal people has not shown a clear benefit although muscle hypertrophy was observed. Growth hormone replacement therapy also does carry some significantly dangerous side effects. Further study is planned in order to investigate the benefits of growth hormone replacement in the most symptomatic polio survivors. I am personally skeptical that this research will lead to any safe and helpful therapy within the next several years.

Another theory emphasizes the central role of stress for initiating post-polio symptoms. All clinicians recognize that too much stress can be harmful for everyone. However, some level of stress is actually good and can spur one on to productivity. Many polio survivors have always had to cope with unusually stressful life situations related to their disability. Life can then become especially stressful when one becomes weaker, acquires new medical problems, and experiences additional new disabilities. This kind of health-related stress can quickly compound other stresses in life such as in family and jobs. Prolonged high levels of stress then begin to produce new health problems.

Additionally, Richard Bruno, PhD, suggests that polio survivors are unusually susceptible to situational stress. He points out there is residual damage in brain tissue from the acute polio infection, specifically in the reticular activating system that is essential for coordinating arousal, alertness, and the body’s hormonal responses to stress. He believes that, at least in some polio survivors, extreme exhaustion and fatigue may be related to a reduced capacity of the reticular activating system to respond to what otherwise would appear as expected, typical daily stresses. Nevertheless, even if capacity to respond to stress is not altered, too much stress is still bad and polio survivors must keep stress within healthy limits.

Another theory about the cause of post-polio syndrome emphasizes normal aging processes. When post-polio syndrome was first labeled in the early ‘80s, many professionals and survivors alike attributed it to “getting older.” Of course, there is some truth in this, and it is a very unique and challenging thing to get older with the residuals of polio.

There are many changes in body functions that occur as people get older. For example, there is a steady loss of maximum breathing capacity related to chronological age; mean blood pressure, cholesterol, weight, and fasting blood sugar levels all increase with greater age; collagen tissue connecting muscles, tendons, bones, and joints become stiffer as age increases. However, one has to differentiate these changes of normative aging, which is what typically happens on average to a 100 or 1,000 people who are getting old, from genetically-determined biological aging that occurs if a person remains free of disease (healthy aging).

A study was presented in 1984 in Warm Springs, GA, that showed that people without a history of polio do not gradually lose anterior horn cells in the spinal cord until age 60. After 60, there does tend to be a relatively modest decrease in the average number of nerve cells at the rate of about 10% per decade. The 90 year old people in the study had about 30% fewer on average motor nerve cells in the spinal cord compared to those under 60. This amount of loss of nerve cells is not sufficient, in and of itself, to account for the weakness that is experienced by many polio survivors. However, no one has established a way to study the life expectancy of the anterior horn cells that survived an acute polio infection, particularly the ones which may have survived but perhaps had some residual damage. Because these nerve cells have functioned under conditions of increased metabolic stress for many years, it could be that their lifespan is shortened or they are subject to degenerative fragmentation as they age. Some new information suggests that this may be the cause, although it is difficult to prove.

A newer perspective on the “aging theory” of post-polio problems emphasizes a life-course perspective. People in Western societies during the late 20th century are now expected to live into their mid to late 70s. This is a completely new phenomena in human history, and therefore dealing with age-related problems among polio survivors is truly pioneering. When studying health care utilization needs that develop among people over 65 in this country, we find a three to four times higher need for medical services and, of course, much higher cost. It is these new medical problems that everyone, including non-disabled people, experience as they get older that have a compounding effect on post-polio residuals. Getting
another disease makes the original post-polio muscle weakness and functional limitation much worse. A new illness, or an injury which requires long bed rest, or long period of inactivity, can cause new weakness by completely upsetting the balance of the chronic partially denervated/reinnervated muscles. Inactivity can lead to severe disuse atrophy from which a post-polio person may never completely recover. Periods of inactivity also adversely affect cardiovascular fitness.

Medical “co-morbidities” are new medical conditions not directly related to polio that occur during the course of a person's life. We found in our studies at the University of Michigan that 35% of polio survivors had new medical conditions that were unrelated to their polio history, but that were capable of producing disability even in non-polio people. Having one of these co-morbidities had one of the strongest statistical correlations with having new functional losses or post-polio syndrome. We also studied the psychological effects of developing new symptoms among polio survivors. We found that polio survivors who were depressed were most likely to have experienced new symptoms. We also found that a person's attitude toward controlling the world around them also was correlated with the presence of new symptoms. Thus, the life-course perspective suggests people with a history of polio residual weakness are especially vulnerable to losses in functional capacity as they age because of an increasing statistical probability that they will develop non-polio related medical problems and experience an exaggerated impact on function for them.

Frederick M. Maynard, MD

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**Readers Write**

“In 1971 I was prescribed Premarin, manufactured by Ayerst Laboratory, after a complete hysterectomy. I had documented corn allergy and the medication (Premarin) contained corn but was not labeled for corn allergy. Consequently, my health deteriorated.

With encouragement from Dr. Theron C. Randolph, a noted expert on corn allergy, I have amassed considerable documentation regarding my situation. I am interested in hearing from post-polio physicians, attorneys, editors of newspapers, magazines, publications, and other persons allergic to corn.” Jacqueline A. Frost, 18 Washington St., #1, Ipswich, MA 01938 (508/356-1658).

“In 1992 after an IVP/renal ultrasound, I experienced severe leg cramps and progressive weakness in both of my legs which were originally affected by polio. My research has found that Conray dye was administered during the test. I would be interested in connecting with other polio survivors who are willing to share their experiences after similar tests, whether they be positive or negative.” Ellen M. Guilford, 64 Sherman St, RR 1, Belfast, NY 14711

“I would be interested in communicating with polio survivors who are also dealing with rheumatoid arthritis.” Ethel, MI

“I am looking for Elizabeth Davenport, a polio survivor who was a childhood friend of mine in Frederick, Oklahoma, in 1943-45. Our fathers were both officers serving in World War II at Frederick Air Force Base.” Bessie Ruth Brock Brewer, MO

“I am trying to find information about the McLain Orthopedic Sanatorium, 915 Aubert Ave. in St. Louis. I was a patient as a teenager in 1925 where I received treatments from Drs. Klein and Krug. Were any other readers patients at McLain?” Elsie Borch, KS

“I would like to hear from children who went to the Sunbeam School for Crippled Children in 1950-51 in the Berea, Ohio, area.” Darlene, GA

“Last winter I discovered a product I found extremely helpful living in very cold, upstate New York. I use a pair of Grabber® Toe Heaters every day in cold weather. I attach one at the toes and one on the arch to a high quality winter sock, and the heaters provide comfortable warmth throughout the day. The toe heaters are 2-1/2 inches by 3 inches and, according to the label, the ingredients are iron, water, activated carbon, and salt. The manufacturer’s number is 800/423-1233.” David, NY
RESEARCH ON POST-POLIO

The researchers at Montreal Neurological Institute define post-polio syndrome as follows: a clinical syndrome of new weakness, fatigue, and pain in those who have recovered from paralytic polio.

The research on post-polio syndrome (PPS) at Montreal Neurological Institute and Hospital involves both clinical and basic science research. The large post-polio clinic which is run by Dr. Neil Cashman, neurologist, and Dr. Daria Trojan, physiatrist, provides volunteers for many completed and ongoing studies.

Dr. Daria Trojan directs the clinical research, or the research which is being done by collecting data and investigating individuals who visit the clinic. Current studies are documenting the prevalence of fibromyalgia in the post-polio clinic and identifying risk factors for post-polio syndrome.

Studies on the possible association of increased serum creatinine kinase with post-polio syndrome are being conducted. Creatinine kinase (CK) is an enzyme found in muscle cells and can be a marker of neuromuscular disease and injury. Elevations of serum CK levels have been reported as a result of muscle damage. The research is designed to determine whether or not an increased CK is a marker for PPS.

Anticholinesterases are agents which can improve neuromuscular transmission or the communication between nerve and muscle. Deficits in neuromuscular transmission may be a cause of fatigue in post-polio syndrome. Their studies provide preliminary evidence that anticholinesterases (such as pyridostigmine or Mestinon) may be useful in the treatment of disabling fatigue.

Additional research is being conducted on the use of specialized electromyographic (EMG) tests to assist in diagnosing. Muscle function and mechanisms of muscular fatigue are being studied as well. Montreal Neurological Institute is attempting to develop clinical measures of function (or performance) which would be useful to assess the progression of post-polio syndrome and the effects of any treatment.

Collaborators in these studies include: Dr. Neil Cashman and Dr. Daniel Gendron at the Montreal Neurological Hospital; Dr. Diane St. Pierre, Dr. Charles Rice, and Ms. Monica Kilfoil (now in Edmonton) at the McGill School of Physical and Occupational Therapy; Dr. John Esdaile from the Montreal General Hospital; Dr. Stan Shapiro and Ms. Cathy Tansey (now in Toronto) from the Department of Epidemiology of McGill University; Ms. Lois Finch, Ms. Ruth Dannenbaum, Ms. Adriana Venturini from the Physiotherapy Department at the Montreal Neurological Hospital; and Dr. Benjamin Chen at the Royal Victoria Hospital in Montreal.

Potpourri

No indigenous wild polio virus has been detected in the Western Hemisphere for the last 160 weeks. The last wild polio virus was detected on 5 September 1991 in Peru. Pan American Health Organization

There has been one confirmed case of polio in the United States in 1994. Five suspected cases with onset in 1994 have not yet been confirmed. Three cases of polio were confirmed in 1993; two were vaccine associated, and one was classified as imported. Morbidity and Mortality Weekly Report

The Danish National Society of Polio and Accident Victims (PTU) has during the last two years run an education program for physiotherapists in treating polio survivors. A video was made about a 44-year-old woman who contracted polio as a child and who is now diagnosed with the late effects of polio. She describes her experiences and her methods of managing. It is translated into English and can be used in American video equipment. The price is $45 which includes shipping costs. It can be ordered by writing or faxing to: PTU, Tuborgejvej 5, 2900 Hellerup Denmark. Tel: 45 31 61 90 00 Fax: 45 31 62 54 39 Attn: Damgard and Laursen.

Bonnie Hatfield, polio survivor from Half Moon Bay, CA, has compiled an extensive post-polio syndrome bibliography. The articles in the 40-page bibliography are grouped according to topic, i.e., dysphagia, fatigue, drug therapy, personal stories, etc. The bibliography, which is updated every August, is available from Bonnie Hatfield, 2 Coral Way, Half Moon Bay, CA 94019-2348, for $10. It is also available on 5-1/4 or 3-1/2 inch disks.

International Polio Network updates its Post-Polio Directory annually. Those currently listed will receive a confirmation letter shortly after the holidays to be included in the 1995 directory. Upon receipt please check the information for accuracy and return it immediately. The Post-Polio Directory lists clinics, health professionals, and support groups knowledgeable about the late effects of polio. To be added to the 1995 directory please send your name, address, and telephone number to our office.

The 1995 directory will be available March 1, 1995 to polio survivors for $4 and to organizations for $8.

International Polio Network would like to hear from individuals who are interested in being a volunteer editor for Polio Network News.

Thanks to the many individuals who have renewed their subscription to Polio Network News. Your support is vital to our work. Additional donations to G.I.N.I., a 501(c)(3), are tax deductible.