Rancho Los Amigos Post-Polio Support Group

Newsletter - October 2015

Acute Polio and Post-Polio - A Review

By Richard Lloyd Daggett

In our August 2015 newsletter we reported on research studies of several drugs and treatments for post-polio (PPS). These treatments and medications were thought to alleviate some of our post-polio symptoms, but the current research indicates that they are of little or no benefit.

In this newsletter we are taking a basic look at polio, both during the initial attack and the causes of our current post-polio issues. This might be a repeat to some readers, but this review will be helpful to many. This review is based on the best and most comprehensive medical literature available.

1600 BC to 20th Century

Although cases of poliomyelitis had been described as early as 1600 BC, it was the 20th century that witnessed regular polio epidemics in North America. These epidemics peaked in 1952, with more than 57,000 reported new cases in the United States. Thanks to the introduction of the Salk injectable vaccine in 1955 and the Sabin oral polio vaccine in 1961, these epidemics were essentially brought to a halt.

Poliomyelitis results from infection by one of three subtypes of this single-stranded RNA enterovirus. It is transmitted by fecal–oral spread and is extremely infectious. The virus is replicated in the gut and lymphoid tissue. Most (95%) infected individuals have no visible symptoms or may report mild flu-like symptoms. If the virus enters the bloodstream, the

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central nervous system is susceptible to invasion. Symptoms become more pronounced as fever and meningeal irritation develop. These individuals may later demonstrate neurologic symptoms and signs of acute poliomyelitis infection. Most common is asymmetric flaccid paralysis. The overall risk of outwardly visible paralytic polio in infected persons is 1–2%.

Surprising Effects

The polio virus ultimately causes destruction of anterior horn motor neurons, resulting in limb paralysis. However, David Bodian, MD, PhD, and others in the late 1940s, showed that under microscopic examination, all cases had some "encephalitic" changes in addition to the typical anterior horn cell destruction. The centers most severely affected were in the brainstem and cerebellum and include the reticular formation, vestibular nuclei, and roof nuclei of the cerebellum. In addition, Bodian reported that, "***There was hardly an individual who did not have lesions, sometimes of a fairly severe degree, of most of the motor nuclei of the cranial nerves as well as in the surrounding reticular formation***."

Bodian also reported that more than 50% of motor neurons had to be damaged before there was any visible loss of strength. This appears to indicate that many additional people had a significant polio infection, but these cases were not reported because the symptoms (fever, aches, and listlessness) were attributed to some other heath issue.

The clinical manifestations of poliomyelitis vary widely. Patients may report weakness in only one limb or may have rapid progression of complete paralysis and loss of respiratory function. Paralysis is more often found in the legs than the arms. Brainstem symptoms (bulbar polio myelitis) occur in at least 10–15% of patients, manifesting as involvement of any of the cranial nerves; facial weakness, swallowing, and speaking. Reticular formation involvement produces impaired respiratory control, and cardiovascular instability. Occasionally, patients have lack of muscular control or, in the pre-paralytic stage, become agitated, mentally dulled, or display upper motor neuron signs.

Then and Now

Recovery begins after two to three weeks and ranges from complete recovery to major residual dysfunction (*e.g.*, permanent respiratory difficulties, paralysis). Younger patients who have paralytic poliomyelitis have better recovery than older patients. Recovery is said to plateau at approximately seven to ten months. Treatment is mainly supportive, ranging from breathing assistance to casts, braces, and crutches. Three factors contribute to recovery: **1.** number of recovered motor units that resume function, **2.** number of motor units that develop "sprouts" to re-innervate "orphaned" muscle fibers (graphic B and C on page 5), and **3.** muscle enlargement induced by strenuous exercise.

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Many polio survivors are now experiencing a renewal of weakness and other polio related problems, commonly referred to as post-polio syndrome or PPS. There has been considerable debate over the underlying cause of PPS. The most commonly accepted explanation for the late effects of polio is that of overuse or premature aging of polio-affected motor units. The so-called giant motor units that develop on recovery are presumed to be unable to sustain the increased metabolic demands. As such, the sprouts begin to fall off, and motor unit function deteriorates (graphic D). Measurements of the electrical activity of neurons, and their action potential, and muscle biopsy data support this theory. They suggest disintegration of function of the motor units and the terminal sprouts themselves 30–40 years after the acute poliomyelitis infection. Other explanations include musculoskeletal disuse and normal age-related loss.

Persistent "low-grade" poliovirus infection or reactivation has been proposed by a few studies using a variety of tests trying to isolate the virus' genetic material, or evidence of virus residuals in the cerebral spinal fluid of PPS patients. However, there are at least as many studies providing evidence against this persistent virus theory as there are supporting it.

Common Symptoms

The most common symptoms reported by PPS patients include fatigue and weakness, joint and muscle pain, respiratory difficulties, cold intolerance, plus speech and swallowing difficulties. Fatigue is the most commonly reported symptom in PPS. This includes central fatigue (sleepiness, difficulty concentrating) and peripheral fatigue (muscular weakness).

Central fatigue is a nonspecific symptom given the numerous potential causes, such as sleep apnea or depression, in addition to PPS. Previous reports described extensive lesions in the reticular activating system in acute poliomyelitis patients. Investigators found very active signals in the high-fatigue group but none in the low-fatigue group.

Peripheral (muscular) fatigue has also been examined using functional studies, electromyography, and muscle biopsies. All of these methods suggest that there is constant remodeling of the giant motor units, with sprouts "dropping off". The most frequent symptom of PPS is progressive muscular weakness. The progression is slow and may occur in muscles previously affected by polio or, less commonly, in muscles previously assumed to be unaffected by the original polio attack.

The extent of new weakness seems to correlate with the severity of the acute polio infection and with the amount of recovery, *i.e.*, individuals with greater recovery seem to have a greater chance of developing new weakness.

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Treatment

Treatment of fatigue, both central and peripheral, primarily involves lifestyle changes. These include regularly scheduled rest periods as well as individually tailored exercise programs, depending on the current functional level of the patient.

Pain is almost as common as fatigue in PPS patients. Rehabilitation medicine specialists have proposed three types of pain in PPS patients. Type I pain is post-polio muscle pain. It is an aching, deep or superficial muscle pain described as similar to the pain experienced during the acute polio infection. It can be precipitated by strenuous activity, stress, or cold temperatures. Type II pain is part of an "overuse" syndrome, which includes bursitis, tendinitis, myofascial, and soft tissue injuries, secondary to poor biomechanics or posture. Type III pain includes degenerative joint disease, low back pain, and nerve compression syndromes. It is the result of chronic over-use, unequal loading of joints, and asymmetric muscle function secondary to weakness. For example, wrist and shoulder pain may develop in some patients secondary to long-standing use of crutches.

Treatment of pain in PPS is similar to the management of chronic pain in general. Lifestyle modifications, physiotherapy, assistive devices, analgesics, and joint or trigger point injections are the most commonly used options.

One of the hallmarks of the treatment of acute poliomyelitis was the negative-pressure ventilator, or iron lung. When respiratory failure was present, it was the major cause of morbidity and mortality. Respiratory symptoms occur in up to 40% of PPS patients. Symptoms range from mildly decreased pulmonary function to acute respiratory failure and the need for assisted ventilation. Contributing to these symptoms are restrictive chest wall changes (scoliosis, kyphosis), altered chest wall strength (decreased maximum inspiratory/expiratory pressures), recurrent infections, and sleep-related disordered breathing.

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Some of the resources used in this report:

Bodian D: Histopathological basis of clinical findings in poliomyelitis.

Paul JR: History of Poliomyelitis. Yale University Press,

Dalakas MC, Elder G, Hallett M, Ravits J, Baker M, Papadopoulos N, Albrecht P, Sever J: A long-term follow-up study of patients with post-poliomyelitis neuromuscular symptoms.

Halstead LS, Rossi CD: New problems in old polio patients: Results of a survey of 539 polio survivors.

Fischer DA: Sleep-disordered breathing as a late effect of poliomyelitis.

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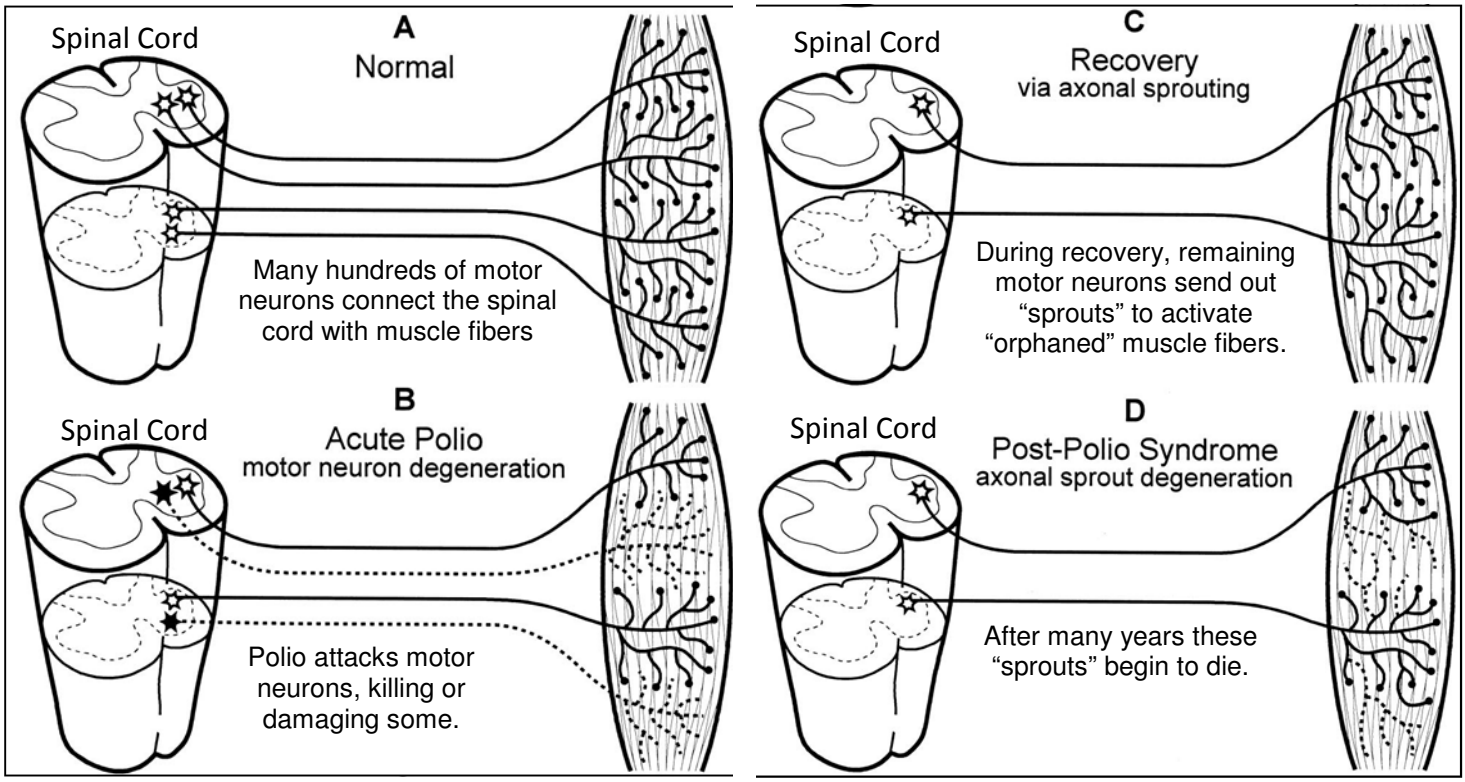

Probable mechanism of post-polio syndrome:

(A) Under normal conditions, a healthy lower motor unit is composed of a motor neuron cell body (located in the anterior horn of the spinal cord gray matter), the motor axon, and muscle cells (fibers) innervated by the axon.

(B) The polio virus infects some motor neuron cell bodies, which subsequently die, while others survive (or were never infected). The loss of lower motor units results in muscle fiber denervation and the weakness that occurs as a result of acute poliomyelitis.

(C) Motor axon terminal sprouting reinnervates previously denervated muscle fibers, creating a "giant" motor unit. This is associated with improvement in strength in the weeks and months after an acute attack of polio.

(D) However, after many years, abnormally enlarged motor units are no longer able to maintain the extensive sprout pattern. Sprouts start to degenerate, producing new denervation and muscle weakness (post-polio syndrome).

Meeting Notices

Rancho Los Amigos Post-Polio Support Group:

Saturday, October 24, 2015 - 2 p.m. to 4 p.m. - Present concerns and future needs.

For additional information, please call Diane at (562) 861-8128

Post-Polio Support Group of Orange County:

Saturday, November 14, 2015 - 2 p.m. to 4 p.m. - HICAP

For information Contact Marilyn at (714) 839-3121 or prisofoc@aol.com

Please remember, both groups encourage your family and friends to join you at our meetings.

Rancho Los Amigos Post-Polio Support Group
Meets on the 4th Saturday of the month,
except as listed in the newsletter.
Please call Diane at 562-861-8128
or e-mail ranchoppsg@hotmail.com