General Information Letter for Polio Survivors

Why are “old polios” who were stable for years now losing function? What should they do about it?

Jacquelin Perry, MD, DSc (Hon), Rancho Los Amigos National Rehabilitation Center, Downey, California

The basic problem is that polio destroyed some of the nerve cells that activate the muscles. To the extent possible, the neurological system responded by having the remaining nerves adopt the muscle fibers that had lost their original nerve supply. This meant that nerve cells now had a demand much greater than normal. While this was an effective solution initially, the passage of time (30+ years, usually) has taught us that overuse can be destructive. As a result, these secondary nerves are wearing out with resulting muscle loss, i.e., post-polio syndrome.

Post-polio muscle strength is commonly overestimated as the usual test depends on manual resistance by the examiner. In addition, polio survivors mask their disability by clever use of their normal control and normal position sense to substitute for missing musculature. The post-polio muscle graded “normal” (5) averages 25% less than “true” normal (only 50% normal for the quadriceps). Similarly, the muscle graded “good” (4) is only 40% of normal strength. These strengths are adequate for a person to carry on customary activities in a typical manner, but at a demand that is 2-21/2 times the usual intensity; hence, the muscle nerves have been experiencing strain for years.

The apparent abrupt loss in function relates to two functions. One is the buffer zone present in all of our physiological systems which enables them to accept strain for a considerable time, but once the buffer limits are exceeded, the loss is very prominent. Secondly, activities such as walking or lifting objects present fixed mechanical demands. As long as the person’s muscle strength exceeds that demand, he/she can continue to perform as usual but with earlier fatigue. When the strength goes below the essential limit, suddenly that function is lost.

The answer is redesigning your lifestyle to avoid those activities that cause muscle strain, cramping, persistent fatigue, and, consequently, weakening. This means to very carefully look at how you are using your arms, legs, and back, and to avoid those tasks that cause the symptoms of persistent fatigue, muscle soreness, and/or a sense of weakness after use. At times, this requires the employment of special devices to take the load off of the arms. If the changes are made early, strength can be recovered. It will not be sufficient to prepare the muscles for excessive strain again, but it does bring the muscles up to a more useful level. Other ways of reducing strain is by using self-care devices, walking aids, braces, and corrective surgery to lessen the stress.

Once the strain has been reduced, then cautious exercise may be of value. We have been using short duration (5 repetitions) or moderate intensity (50-70% of one’s maximum capability). Let me caution you not to take on the exercises, however, until you have worked out a lifestyle that avoids the strain. Also, if the exercises cause any pain, persistent fatigue, or increased weakness, STOP! This means just the mechanics/activities of daily living (ADL) are sufficient exercise for your muscles.

Recent research on the course of muscle strength over time in persons over age 50 years showed a normal average decline of 1% per year, but for post-polio survivors the rate was 2% per year. The rate of change is so subtle that a four-year study was needed for a measurable change. Also, the weaker “polios” experienced greater functional loss. This latter...
fact appeared to indicate strength training by exercise would deter the process. However, retesting this group of polio survivors at eight years and adding muscle analysis told a different story. The muscle fibers were hypertrophied, twice normal size, not atrophic. The person with the greatest strength loss also had the greatest hypertrophy.* MRI recordings showed areas of muscle loss and fatty replacement. The source of the visible muscle atrophy is muscle fiber loss secondary to nerve fiber overuse failure. These findings confirm the need for a saving program rather than challenging exercise.

The advantage of having had polio rather than another disability is that it allowed one to resume a very active and profitable life for many years. Now it is necessary to recognize that excessive strain was being experienced and that lifestyles must be changed to accommodate this situation.

Be an “Intelligent Hypochondriac” – Listen to your body and adopt a program that avoids the strain. ■

* “Several histologic studies have shown that the myofibers of polio survivors can be twice the size of those found in other persons. A few studies have provided indirect evidence for a possible transformation of some of the surviving type II (fast-twitch fibers) to type I (slow-twitch fibers). The few studies performed have shown a preponderance of type I muscle fibers in very weak muscles that were constantly being used in daily activities. It has been postulated that a person would have to utilize all motor units in these very weak muscles to perform all daily activities and that, over time, the type II fibers are transformed to type I fibers.”


**Selected Post-Polio Bibliography**

  This self-directed learning module briefly highlights the differential diagnosis for acute weakness in patients with acute respiratory failure requiring prolonged mechanical ventilation.


  This study from Slovenia found that the new symptoms in post-polio survivors, which may be classified as post-polio syndrome, increased their walking and climbing stairs disability, increased their disability to perform daily activities, and also decreased their satisfaction with life.

  This paper describes the establishment of a post-polio clinic and the principles adopted in quantitative muscle testing using twitch interpolation. Peripheral endurance and/or voluntary drive to muscles is impaired in about 30% of prior-polio patients attending the clinic. Progression of these deficits is slow and not easily predicted by factors associated with the original illness.