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For the Lincolnshire Post-Polio Network
Article on Manual Muscle Testing by Professor Murray Maitland, March 2993

The loss of muscle function following polio has been documented in several studies. It was found that symptomatic subjects had evidence of more severe original polio involvement by history (documented electromyographically), were weaker and capable of performing less work than asymptomatic subjects, and recovered strength less readily than controls.[1]

Dalakas and coworkers have performed several studies examining the histology and electromyography of people with chronic polio.[3-5 7] Dalakas et al., examined muscle weakness that developed a mean of 28.8 years after recovery from acute polio. The researchers concluded that new weakness was a dysfunction of the surviving motor neurons that causes a slow disintegration of the terminals of individual nerve axons. Dalakas also studied thirty-five muscle biopsies performed on 27 patients with postpoliomyelitis progressive muscular atrophy (PPMA) (8 patients had two biopsies) and 5 asymptomatic postpolio patients in an attempt to define diagnostic criteria for the newly weakening muscles and to provide insights into the mechanism of the disease. The newly weakened muscles show signs of recent denervation. [3]

Rekand et al. studied 148 people with post-polio syndrome and they found that while Although symptoms defined in the PPS are unspecific and may occur in the general population, the risk for developing such symptoms are higher among the polio victims. The difference in risk among nonparalytic and paralytic patients may depend on the extent of motor neuron damage in the acute stage.[8] Rekand et al. also studied 39 patients who had polio. Three patients (6.7%) had neurologic and neurophysiologic findings and development of symptoms consistent with motoneuron damage. The authors concluded that some nonparalytic patients may have subclinical acute motoneuron damage with subsequent development and manifestation of motor weakness and neuromuscular symptoms many years later. These symptoms should be considered a differential diagnosis in patients who have a history of nonparalytic poliomyelitis.[9]

The evaluation of muscle function (strength) using manual muscle tests is subjective from grades 3 through 5. Strong muscle contraction is rated as 4 or 5 but the definition of "normal" is not adequate in this subjective test to differentiate between weaker and stronger muscles. Bohannon and Corrigon found that manual muscle tests of grade 5 had enormous variation in force, up to 86% of all quantitatively measured forces. These findings help to explain the insensitivity of manual muscle testing at higher force levels and why it has such a profound ceiling effect. [2]

There can be many technical reasons for this effect including the fact that examiner strength limits detection of moderate quadriceps weakness with manual resistance.[6]

Sharrard studied polio patients by autopsy.[10] He found that muscle grades of 3 were given to individuals with 85% denervation of the muscles. In other words, profound muscle weakness must be evident before abnormal grades are given.

Quantifiable muscle strength tests are commonly carried out using an isokinetic dynamometer (Biodex, Cybex, Kincom). Using this technique we assess muscle function in a controlled manner. Endurance, fatique, maximal torque, pain free torque, power and work are some of the variables that I have measured, and are well documented in the literature. We use 5 definitions of normal depending on the condition we are examining:

1. Less than 10 percent difference between sides
2. Strength to body weight ratio
3. Minimal or no symptoms
4. Compared to normal, age-matched values
5. Sport or activity specific

At this time, I am attempting to develop more efficient, alternative lower extremity muscle tests but they have not been validated at this time.

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