A TEN YEAR EXPERIENCE

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THE ORIGINAL EFFECTS OF THE POLIO VIRUS

To more easily understand the late effects of polio it is important to understand the original effects of the poliomyelitis virus. The portal of entry for the polio virus was oral, and the infection in its first stage was in the gut cells lining the intestine. Many people infected with the virus at this stage thought they simply had gastrointestinal flu with symptoms of diarrhea and nausea. For a significant percentage of the people infected, the disease ended at this point.

But for some the virus continued to multiply and spilled over into the blood stream, signaling the viremia stage of the infection. Symptoms then were not unlike a more generalized influenza, with fever, chills, malaise and achiness. Once again for a significant percentage of patients, the disease process ended there. However, in a small percentage of the patients, the virus crossed over into the central nervous system and infected the anterior horn cells. These individuals contracted "paralytic" polio.

The distribution of weakness or paralysis depended on which anterior horn cells were involved. Spinal anterior horn cell involvement resulted in weakness or paralysis in the arms, legs, and trunk to one degree or another. Bulbar (brainstem) anterior horn cell involvement resulted in visual, swallowing, or breathing difficulties in any combination.

It is this period of fever, weakness, paralysis, and muscle pain that many polio survivors (or their parents) remember. This was the time when infected motor neurons were dying, leaving all the muscle fibers they innervated orphaned (or denervated). If all the motor neurons that supplied a particular muscle died, the result was complete paralysis of that muscle. If only a percentage of the motor neurons died, the muscle was only weakened, but not completely paralyzed.

Many polio survivors remember having muscles completely paralyzed that started to move again, although weakly, after a period of a few weeks. That occurred because some of the anterior horn cells were simply shocked when neighboring anterior horn cells died. These nonfunctioning anterior horn cells were not dead but stunned by the swelling caused by adjacent nerve cell destruction. As days and weeks passed, the swelling subsided and the surviving anterior horn cells revived. Kenny hot packs and stretching helped control the pain and contracture of the denervated muscle. With time and exercise, the muscle got stronger.

Over the course of months and years, many polio survivors got stronger in a previously weakened or paralyzed muscle and were able to discard braces or wheelchairs. This was not only because of hard work and exercise but also because of a phenomenon called "sprouting". Denervated or orphaned muscle fibers within the same muscle sent out a chemical distress signal that caused tiny sprouts to grow from the terminal nerve branches of neighboring, surviving motor neurons. Very slowly these sprouts grew over to the orphaned muscle fibers and reinnervated them, causing the motor unit to grow in size. In fact some motor units sprouted to innervate three to four times as many muscle fibers as Mother Nature had originally intended. As a result polio survivors felt stronger, and this pattern of strength and weakness

remained stable for the next twenty to thirty years.

THE LATE EFFECTS OF POLIO

Since 1981, four hundred eighty-three (483) post-polio patients have been seen at St. Mary's Hospital Post-Polio Clinic. When the data collected by Barbara L. Bammann, M.D., at Alta Bates-Herrich Rehabilitation Hospital and by Michael Berlly, M.D., at Santa Clara Valley Medical Center are added, there is a wealth of statistics and information. In all, seven hundred eleven (711) patients have been examined in the Bay Area Post-Polio Clinics. The ages range between thirty and seventy-six. the following material is based in large part on these statistics.

FATIGUE: The most frequent symptom was unaccustomed fatigue (79%). During the past decade, a number of investigators have looked at the problem using a variety of research tools. The picture that has emerged from this data suggests that after thirty years, the metabolically overburdened surviving motor neurons appear to have become incapable of supporting the integrity of all distal nerve terminals, resulting in defective neuromuscular transmission.

To better understand this, visualize what happens at the junction of the nerve and muscle under the usual circumstances. The electrical impulse travels from the anterior horn cell, down the nerve fiber to the tiny terminal nerve endings, which contact the individual muscle fibers. The arrival of the electrical i pulse at the terminal nerve ending causes the release of a chemical (acetylchloine), which in turn causes the electrical impulse to penetrate into the muscle fiber, resulting in contraction of that tiny muscle fiber. In polio survivors, repeated discharges of the large reinnervated motor unit results in a failure of some of the muscle fibers to fire, thereby not contributing to the force of contraction. Polio survivors experience this as fatigue or a loss of stamina.

WEAKNESS: A significant number of the patients had new or increasing weakness for reasons not directly related to their polio residuals. Having stated that, we note that there were a large number of patients who did have increasing weakness which was directly attributable to the late effects of polio. Some clinicians would argue that 'acute' overwork weakness and post-polio muscular atrophy are one and the same. We have chosen to separate them for record keeping purposes and for prognostic reasons.

Clinically, polio survivors who have engaged in excessively strenuous activities like tennis, weight-lifting, stair climbing, etc., have noted the sudden onset of weakness in particular muscle groups overused in that particular activity. Even though it may take months and with proper rest, the strength may return nearly to the level it was prior to the strenuous activity. This reversible weakness is what we refer to as 'acute' overwork weakness.

Cumulative research suggests that the old overburdened motor neuron, which has been driving many more muscle fibers than it was meant to drive, is so metabolically exhausted by the strenuous activity that some of the terminal nerve endings die back, leaving many of the muscle fibers of the same motor unit orphaned again. If the weakened muscle group is allowed to rest (by stopping the offending activity or bracing, for example), new sprouts will grow and recapture the lost muscle fibers.

'Chronic' overwork weakness, or what has been called post-polio muscular atrophy (PPMA), differs inasmuch as there is no singular event of strenuous activity or exercise that signals the onset of increasing weakness. Rather there is a very slow and gradual loss of strength with accompanying atrophy of a particular muscle group. And no amount of rest will reverse the weakness. Rather there is a very slow and gradual loss of strength with accompanying atrophy of a particular muscle group. And no amount of rest will reverse the weakness. the overburdened motor neuron is unable to send out new sprouts to capture the orphaned muscle fibers, which have become denervated slowly from "pushing" in order to complete ordinary day-today activities.

RESPIRATORY PROBLEMS: Of the total number of patients seen in the Bay Area's Post- Polio Clinics, only one hundred fifteen (16%) were classified as having worsening respiratory problems.

Recurrent bronchitis was a frequent enough problem to warrant mentioning, even though the patients for whom it was a problem had a perfectly adequate cough and no difficulty handling secretions. The problem seems to be more of a chemical bronchitis (as opposed to infection) caused by night-time aspiration of tiny amounts of stomach acid, which refluxes from the stomach into the esophagus because of hiatus hernia. Fully one hundred sixty-seven polio survivors (23%) have symptomatic indigestion secondary to hiatus hernia. Despite reports of sleep apnea in polio survivors, we have seen only one documented case.

JOINT AND MUSCLE PAIN: Various musculoskeletal aches and pains were a problem for five hundred fifty-nine polio survivors (74%). Most polio survivors suffer from chronic pain, which for some can be quite disabling.

Nearly all polio survivors (99% of our patients) had scoliosis. Scoliosis per se does not hurt, but the long standing effects of rotation and curvature of the spine have been associated with conditions that do hurt. We found that 34% had facet arthropathy or wear and tear arthritis of the little joints of the spine; 41% had degenerative disc disease; 12% had pain in the back and legs or in the neck and arms; 11% had lumbar spinal stenosis, all of whom were over the age of sixty. The other joints in addition to the ones of the spine showed signs of wear and tear arthritis. The shoulder was affected in 22%, while the knee was affected in 21%. Degenerative arthritis was present in the hip in 11%, in the ankle/foot in 6%, and in the wrist in 4%. Interestingly, if a leg was braced because of weakness, the arthritis generally developed on the unbraced side, which had been the side of major weight bearing for many years.

COLD INTOLERANCE: Forty one percent of the polio survivors complained of worsening cold intolerance. For nearly everyone it was not a new problem, but one that had been present since the onset of polio to a less bothersome degree. It appears that some of the sympathetic nerve fibers were affected originally, and these fibers are the ones that constrict the blood vessels of the skin in response to cold. Normally, when we are exposed to cold, the blood vessels of the skin constrict to shunt blood away form the surface of the skin down to the underlying bone and muscle where heat can be conserved and core body temperature maintained at 98.6 degrees. Some polio survivors have lost that ability to shunt blood away from the skin, and so heat radiates away from the affected limb and it cools down. The best treatment is an elastic stocking and good woolly socks.

The other part of the problem is decreased muscular performance in the presence of cold. Many polio survivors find they are unable to swim in an unheated pool, or they find that they have more trouble walking in cold winter weather. This is explained by the fact that when muscle is cooled, defective neuromuscular transmission is made worse. If you suffer from cold intolerance, you can only be advised to try to avoid cold exposure and "button up your overcoat."

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