Polio Biology VII
Holistic Polio

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On reflection, it is quite obvious that everything in the body works together. Put another way: nothing can happen inside the body without an effect on all parts of the system. After reading some recent articles on nerve and muscle function I started thinking about Post-Polio Syndrome and how closely a skeletal muscle fiber and attached nerve work together. The relationship is so close that it is quite impossible, at times, to tell what the primary cause of neuromuscular weakness is. This is particularly true when we are focusing on the synapse, the gap between nerve and muscle through which stimulant chemicals like acetyl-choline act. Without a nerve connection a muscle is an orphan and incompetent. Without a muscle a motor-nerve is useless. It is commonly understood that in Post-Polio Syndrome the end fibers of an axon (long fiber which is part of a nerve cell) break down leaving a small but effectively insulated moat between muscle and nerve. Since each nerve end fiber innervates only one muscle fiber the muscle cell is then cut from stimulation. Without stimulation we know that the receptors of stimulation on the surface of the muscle fiber are forever lost.*[1]*

I think most of us assume that the loss of end fibers in PPS is due to overuse. And we have a tendency to extrapolate to say that if this could only be remedied we would defeat our foe. However, during the increased neuromuscular load of exercise the earliest response of the neuromuscular system is to allow for increased efficiency of nerve stimulation. A later response is the production of muscle protein and the consequent hypertrophic growth of muscle fibers. Also, even in normal neuromuscular systems there is only one end fiber per muscle fiber. During synaptogenesis several fibers may innervate one muscle but eventually all lose touch but one. This evolutionary competition for muscle fibers by nerves is believed to result in efficient and varied innervation of muscle because different kinds of fibers have different effects on the contraction of muscle.*[2]* In normals there would be a tendency toward a diversity of muscle tension strength along the length of a muscle which would be produced by the natural competition of early nerve type synapses. Polio obviously alters this process by killing neurons. This allows for secondary synaptogenetic events.

What happened to skeletal muscle fiber receptors while they were left without stimulation for months during acute polio? We know now that they break down. People hoping for cures to spinal injury must exercise their muscles with electrical stimulation to keep muscle cell receptors alive. The old maxim "Use em or lose em" applies here. We with polio are in a position of slow loss of both end fibers and muscle receptors. How can we say that polio is a nerve disease when it is also, obviously, a muscle disease? And, by the way, I'm not convinced that the "cause" of PPS is overuse. There could be a host of other reasons why these end fibers of nerves, and/or muscle receptors, lose function. When analyzed there may be more than one cause. With some of the advances appearing now in measuring and differentiating antibodies, along with similar advances in DNA and RNA cloning techniques, which have been used to identify polio virus fragments in PPS, it may not

be long until we have more definitive answers. Until then we should understand that we are a system and that whatever proximately caused our PPS, reversing secondary and tertiary problems will likely prove very difficult, because we are whole.

References.

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Top of Article

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Reception | Library | Networking | Directory | What's New?

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