THE ETIOLOGY OF POST-POLIO PROGRESSIVE MUSCULAR ATROPHY

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The etiology or cause of the Post-Polio Syndrome (PPS) and PPMA (new weakness with or without atrophy) is not known. Numerous theories or hypotheses have been proposed. None have been proven, four of these theories have been thought by most researchers to be more likely.

The leading theory is that there is drop-out of newer nerve terminals from the reinnervating motor nerve cell. When the acute poliomyelitis occurred many motor nerve cells died. The remaining motor nerve cells had to send out new nerve terminals to supply signals to the muscle fibers that had lost their nerve cells. Many years later (30-40 years after the original polio) these new nerve terminals may be contracting and dropping out. This theory is supported by single fiber electromyographic (SFEMG) studies and by some muscle biopsy studies. Why these nerve terminals would drop out is also not known although theories have been suggested for this also.

An alternative theory is that the motor nerve cells themselves are dying. Possibly after supplying many more muscle fibers than normal, the motor nerve cells remaining after the acute polio can no longer maintain this increased metabolic demand (of supplying too many muscle fibers) and begin to die earlier in life than normal. A few muscle biopsy studies support this theory.

A third theory is that PPMA is an auto-immune problem, i.e., a person's immune system (antibody, white blood cells) attacks his or her own tissues. This theory could overlap with the two above as immune system could attack either nerve terminals or the motor nerve cells or both. This auto-immune reaction might occur because proteins in the nervous system were permanently altered by the original poliovirus infection. This theory is supported by the finding of unknown antibodies in the spinal fluid and the finding of white blood cells (inflammation) in the spinal cords of patients with PPMA.

The fourth hypothesis is that the poliovirus may have come back or reactivated in some way. There is no convincing data to support this theory but whenever there is inflammation (see previous paragraph) the possibility of an infection is suggested. However, poliovirus antibodies are not increased in PPMA patients and poliovirus has not been isolated from the throat, stool or spinal fluid. Thus, if poliovirus is present at all, it could only be in the spinal cord in a very altered form since infectious virus cannot be recovered from anywhere else. This would be a very atypical and unusual poliovirus infection but this theory cannot be excluded for sure until spinal cords are examined. In any case, PPS and PPMA patients are in no way an infectious risk to others. This is not a problem or worry.

In this brief review, I've only mentioned the most frequently discussed possible causes. Maybe another will turn out to be the actual one. As you can see, we have a lot of work to do to solve the question of the Post-Polio Syndrome. Again, let me emphasize that there is no hard evidence to support an active poliovirus as the cause and PPS patients are not infectious or contagious.

Return to PPS Index