



POST-POLIOMYELITIS FATIGUE WHERE IS THE LESION?

THE CONTROVERSY - THREE POINTS OF VIEW

A REPORT OF A SPECIAL NEUROLOGY ROUNDS AT THE MONTREAL NEUROLOGICAL INSTITUTE AND HOSPITAL SEPTEMBER 26, 1997.

The origin of post-polio fatigue is controversial. Thanks to Dr. Cashman's initiative, there was a respectful discussion about the cause and source of Post-polio fatigue at the Neurological Grand Rounds of the Montreal Neurological Institute on the morning of September 26 attended by physicians, other health-care practitioners, students and a few members of Polio Quebec. The speakers were

[Dr. Richard Bruno](#) (Psychophysicologist and Director of the Post-Polio Rehabilitation Research Service, Kessler Institute for Rehabilitation, New Jersey Medical School) spoke about the role of cerebral abnormalities on post-polio fatigue.

Dr. Neil Cashman (Co-Director of the MNIH Post-Polio Clinic with Dr. Trojan)

Dr. Robert Miller (Chair of the Department of Neurology, California Pacific Medical Centre) who flew in for the occasion.

A lightly edited resumé delivered by Dr. Cashman at Polio Quebec's Annual General Meeting the following day appears below for those who were unable to be with us or who would like to "hear" it again.

"Post-polio fatigue is probably the most common and certainly one of the most disabling symptoms that occur after polio. There is a great deal of controversy in the field as to what is causing the fatigue. The stakes are very high, because if we were to understand what is causing the fatigue then we could design a treatment or therapy that would help counter it.

"Dr. Bruno has spent a great deal of time working on an idea prompted by early pathological studies done by David Bodean who did post-mortems and found that an encephalitis existed in the brains of people who had acute polio. Because about 10% of those who had polio actually died from the illness, their brains and spinal cords could be examined to try to figure out what the polio virus was doing to the nervous system. We all know that the polio virus affects and destroys motor neurons, or motor nerve cells in the spinal cord and in the brain stem. This is what accounts for weakness and muscle atrophy. What the original investigators were finding, and Dr. Bruno has capitalized on this, is evidence of destruction of nerve cells outside of the motor system. One area that is commonly affected in people who have had polio is the reticular formation, i.e., a group of nerve cells that seem to support consciousness, so to speak. Dr. Bruno has taken this and said - what if there is a post-polio syndrome of the reticular formation, and how would that manifest itself? Dr. Bruno's theory would present as a disorder in arousal, retrieval, attention, and it may account for some of the troubles people have with concentration, with memory and with

fatigue. This is the crux of Dr. Bruno's argument that, in fact the fatigue that people with PPS have is actually brain fatigue - nothing to do with the nerves and muscles. Rather it is a disorder in concentration and memory. He presented some data that suggests that medicines such as bromocryptine that increase the function of the reticular formation can actually improve fatigue and attention in people with this type of post-polio syndrome.

'Then I (Dr. Cashman) added that, yes, there is involvement of other parts of the brain and the brainstem, but the obvious and clear area of involvement by polio is the motor neurons, and when motor neurons die, remaining healthy motor neurons sprout extra branches to try to supply the muscle fibers that have lost their branches in the polio - a great recovery mechanism for say 10 or 20 years, but ultimately the motor nerve cell which is designed to support approximately 1,000 muscle fibers may, after polio, support 10,000 muscle fibers. Eventually the motor neuron starts to say this is too much. I can't keep this stability up forever, and these extra branches begin to degenerate. This is not a motor neuron disease like Lou Gehrig's disease. It's at the other end of the motor unit - it's a terminal axonal disease. The evidence we have of this is from muscle biopsies and a number of electrophysiological tests. We're certain - I'll put it that strongly - that there is degeneration of terminal axons. Everyone who has done these sorts of experiments in the world, agrees there is degeneration of terminal axons in PPS. But Dr. Trojan and I have tried to take this a step further. When you think about it, a terminal axon is not normal one day and gone the next. There must be an intervening period of poor function - of disfunction. And this may account for some waxing and waning symptoms of PPS, like fatigue. What Dr Trojan and I have done over the past five years is to collect evidence that there is a problem with the function of the terminal axons. One of the things we found is that one of the defects - one of the ways the nerve terminals are disfunctioning is that they have decreased release of a chemical called acetylcholine. This chemical accounts for the nerve talking to the muscle. If there isn't enough acetylcholine, the muscle doesn't get stimulated and doesn't work properly - doesn't generate a force. So we have tried to increase the communication between the nerve and the muscle by giving a medicine that prolongs the effect of acetylcholine in the junction between the nerve and the muscle. This is the basis for the use of Mestinon. We believe we have good preliminary results to show that Mestinon does work, and trust this will be demonstrated in the report, to be released in November, of a large multi-centred double blind Mestinon trial. [[See preliminary report in NewsBites](#)]

"Dr. Miller, well-versed and very knowledgeable about post-polio syndrome and other neuro-muscular diseases, defines fatigue very tightly as a decrease in muscle strength with exertion and a return of normal function with rest. He argued that the fatigue in PPS acts more like muscle fatigue than central or neuro-muscular junction fatigue. He has done studies demonstrating the problem with the role of calcium in the muscle. Calcium is used to stimulate a muscle contraction, and if the muscle depolarizes without the release of calcium, then a good force isn't generated for the effort the nerve has put into it. He believes that all of these ideas about fatigue, whether it be central from the brain, at the neuro-muscular junction, or whether it is at the level of the muscle fiber itself, fail to explain the severity of fatigue experienced by people with PPS. His major idea for therapy of this type of fatigue is to engage in exercise. Dr. Miller believes that a great deal of PPS fatigue is due to deconditioning, and that non-fatiguing exercise, or even fatiguing exercise, can lead to an increase in strength and a decrease in fatigue. I find this a bit hard to swallow, (said Dr. Cashman) but given Dr. Miller's love to run marathons and sailboard in San Francisco Bay at every opportunity, I can see where he's coming from.... If you're tired you go and exercise....

"So, there are at least three camps when it comes to understanding fatigue. People are paying attention and doing a great deal of work to understand the underlying physiology of fatigue and post-polio syndrome. The debate has now been formulated. We'll see which theory prevails over the next few years. It could be that we're all right. It could be that some people have one type of fatigue and others another type. It could

be that we are all wrong.

Video and Audio Cassettes (English only) are available of the Grand Rounds Contact Sally Aitken ([514 932 6092](tel:5149326092)) - or write [the Association](#). In UK contact the [Lincolnshire Post-Polio Network](#). Prices not available at time of going to press.

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The Secretary, Lincolnshire Post-Polio Network

PO Box 954, Lincoln, Lincolnshire, LN5 5ER United Kingdom

Telephone: [+44 \(0\)1522 888601](tel:+44(0)1522888601)

Facsimile: [+44 \(0\)870 1600840](tel:+44(0)8701600840)

Email: info@lincolnshirepostpolio.org.uk

Web Site: www.lincolnshirepostpolio.org.uk

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