literature more often relates new muscle weakness to the metabolic failure of poliovirus-damaged neurons. An inability to document “progressive neuromuscular failure” by measuring the number of motor units would be expected, because it is the size of motor units that has been correlated with new muscle weakness.

Further, surveys of more than 1,000 polio survivors have found an association between PPS, physical overexertion, and emotional stress, not aging. The lack of progression of symptoms in “unstressed” subjects also should have been expected.

The suggestion that chronic fatigue syndrome (CFS) and fibromyalgia—disorders for which there is no known etiology, effective treatment, or even agreement as to their existence—as causes of PPS in the 20% of subjects with “no alternative explanation” for new symptoms is far from parsimonious and allows the inference that PPS does not exist. Such an inference is evidenced by the Lancet summary of the authors’ paper headlined “Post-Polio Syndrome Called into Question.” Post-polio fatigue has actually been suggested as a model for the pathophysiology of CFS, because poliovirus lesions in the reticular activating system are well documented, and recent studies have shown lesions on MRI of the brain, neuroendocrine and neuropsychological abnormalities in polio survivors with fatigue that are identical to those in CFS.

Polio survivors must be assured that their symptoms are “real,” do have a physiological basis, and that a decade of clinical research has identified “rational therapeutic approaches,” based on decreasing physical and emotional stress, that do indeed “produce substantial benefits” to those with PPS.

Richard L. Bruno, PhD
West Orange, NJ

Reply from the Author: The comments of Dr. R.L. Bruno are important. Our study identified a population-based cohort of individuals who had paralytic polio. As Dr. Bruno mentioned, these were individuals who had paralytic polio but were not “stressed” other than by the average stresses of life and aging. We found that it was very reassuring that these individuals as a group did not have any evidence of progressive neuromuscular failure.

We thought that it was also important that those individuals who had symptoms compatible with “post-polio syndrome” also showed no evidence of progressive neuromuscular failure. We would agree that aging or interval since onset of polio are not risk factors for developing subsequent difficulties. This was reported in our first study. We agree entirely with the conclusion that it is important to evaluate patients on an individual basis to identify specific causes for their difficulty. Most patients in the study had new symptoms, but most of these could be accounted for by factors not directly related to post-polio neuromuscular failure.

Quantitation of fatigue is challenging because fatigue was not a major symptom in this population-based group of polio survivors.

As neurologists who treat many patients who come for evaluation of post-polio symptoms, we would agree that most patients have symptoms that are “real.” One of the major points that we made in our first manuscript was that the psychological profile of these individuals was completed normal. Overall, therefore, we would agree with Dr. Bruno’s comments: there is no doubt that polio survivors are subject to many different types of problems. However, as a population they do not appear to be at risk for developing progressive neuromuscular failure in significant numbers. We were impressed and very reassured that in this population-based study, polio survivors as a group did not have any evidence of progressive neuromuscular failure.

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References

Correction
In the September issue’s Book Review section, a review of “Clinical Neurology for Psychiatrists,” by D.M. Kaufman (Neurology 1996;47:856) was inadvertently credited to Robert B. Daroff. The review was actually authored by Robert B. Daroff, Jr.
Correction
Neurology 1996;47:1360-1360-a
DOI 10.1212/WNL.47.5.1360-a

This information is current as of November 1, 1996

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