In the nearly 40 years I have been studying and treating polio survivors, I have never seen a patient with abnormal vision that could be explained by having had polio. And I wasn’t surprised. In the 1940’s, polio savant David Bodian performed scores of human autopsies on those who died after having had polio. He stated, “All available evidence shows conclusively that every case of polio exhibits damage in the brain.” However, as part of his research into how the poliovirus found its way into and damaged the brain and spinal cord, he injected poliovirus directly into monkeys’ vision neurons at the back of the brain and absolutely nothing happened; the vision neurons were not infected with the poliovirus and just kept working! The poliovirus’ lack of interest in other than motor neurons prevented polio survivors from having any vision impairment.

But, Bodian did make clear that poliovirus could severely damage motor neurons in the brain stem swallowing, breathing and blood pressure control neurons and cause what was clinically diagnosed as “bulbar” polio. Although the numbers vary from year to year, epidemic to epidemic, approximately 15% of patients had clinical bulbar polio. But if the poliovirus could kill the above-mentioned motor neurons, could it attack other brainstem motor neurons, for example those controlling eye movements? The answer: sometimes.

Abnormal Eye Movements and Polio

Going back to my well-thumbed copy of the 1948 proceedings of the First International Poliomyelitis Conference, I could find only two sentences buried in the discussion section on bulbar polio that mentioned eye movements. It referred to polio expert A. B. Baker’s description of one of the largest polio outbreaks, the 1946 Minnesota epidemic, during which 23% of children had clinically diagnosed bulbar polio: 90% had damage to the vagus nerve, causing problems with breathing and swallowing, while 6% had cardiovascular collapse, 83% of whom died. But among these bulbar polio patients 12% were found to have loss of eye muscle control and even eye muscle paralysis.

It turns out that Baker’s finding of eye movement abnormalities in bulbar polio patients actually was 41 years old. Ivan Wickmann, in his famous 1907 paper on Heine-Medin’s disease (the original name for what in 1840 Heine himself called “infantile spinal paralysis”) mentioned cases of bulbar polio where there was damage to the brain stem’s sixth cranial nerve that controls the muscles that move your eyes outward, away from your nose, causing “crossed eye(s).” But Baker added to Wickmann’s finding, reporting that the most common eye movement abnormality in bulbar polio wasn’t crossed eyes but nystagmus, the back-and-forth or up-and-down “twitching” movement of the eyes.

But if eye movement abnormalities had been seen with bulbar polio as far back as 1907, why have they been given such short shrift in the medical literature? In a paper describing the extensive polio epidemics in Israel from 1949 to 1954, the authors explained why abnormal eye movements in bulbar polio survivors were so infrequently documented. Eye movement abnormalities might indeed occur more frequently than was reported, “but being often transient (would) hardly be included in clinical statistics. Since most of the cases involving the muscles of the eye are accompanied by other serious, often dangerous symptoms…the eyes tended to frequently be overlooked.”

A 1952 paper documented that cranial nerve damage causing abnormal eye movements fortunately was transient. The authors reexamined 59 Swiss polio survivors, all of whom had had symptoms of
cranial nerve damage, 2 to 18 years after their acute polio. At follow-up 39 patients (66%) had no cranial nerve damage symptoms, 18 (31%) of patients had minor symptoms and 2 patients (3%) -- one having the sixth and another the seventh cranial nerve affected -- had more severe symptoms. However, none of the symptoms in any of the bulbar polio survivors “affected the life of the patients.”

**Could Abnormal Eye Movements be PPS?**

Yes, abnormal eye movements could be PPS. If someone had clinically-diagnosed bulbar polio, the remaining, poliovirus-damaged brain stem neurons that control eye movements could be “browning out” and causing eye movement abnormalities. But, although small muscles are constantly moving our eyes, it takes about 10,000 times more force to walk on a level surface than it does to move the eyes. Muscles moving small “weights” (the eyes, eyelids, facial muscles) are less likely to experience “overuse abuse” than are muscles used to lift bags of groceries or to walk, which is probably why we’ve never seen abnormal eye movements in polio survivors. Based on the Minnesota and Swiss data, abnormal movements of the eyes would be very rare, seen in less than 2% of bulbar polio survivors. But seen they could be.

However, as with all PPS, other causes of eye movement abnormalities must be ruled out before PPS is accepted as the culprit. What’s more, any abnormal eye movements must be evaluated without delay since they may be symptoms, not of PPS, but of a neurological disease that requires immediate treatment.

More information by Dr. Richard L. Bruno, HD, PhD in *The Encyclopedia of Polio and Post-Polio Sequelae* on our website. It contains all of Dr. Richard Bruno’s newly published and updated articles, monographs, commentaries, videos and “Bruno Bytes” (Q & A articles) and links to his book. You can easily access it from Dr. Bruno’s website as well: [www.pospolioinfo.com](http://www.pospolioinfo.com)