



Bruno “Bytes” – December, 2016

(Bits and Tidbits from the Post-Polio Coffee House)

Available through a “link” from www.postpolioinfo.com

(or) directly through <http://www.papolionetwork.org/bruno-bytes.html>

On the topic of Protein, Polio Survivors and getting too much (12/6/2016)

Dr. Bruno’s Original Post: A Coffee House member asked about using protein powders as part of THE POST-POLIO “DIET” (postpolioinfo.com/library/prodiet.pdf) to increase the protein intake polio survivors need to decrease fatigue and muscle weakness. THE POST-POLIO DIET isn't a high-protein diet. It's designed for you to get the amount of protein the A.D.A. says is necessary for your desired body weight...and not have just a donut for breakfast!

The formula is easy: Divide your desired body weight by 2. So a 160# man should have 80 grams of LOW FAT protein/day, 20 grams at each meal and a 20 gram snack. (One ounce of meat or dairy is about 7 grams of protein.)(100 grams = 3.5 ounces)

What does 20 grams of protein look like?

<http://www.fitnessreals.com/2012/04/what-does-20-grams-of-protein-look-like/>

Here's some new information:

Can You Get Too Much Protein?

<http://www.nytimes.com/2016/12/06/well/eat/can-you-get-too-much-protein.html? r=0>

On the topic of Cardiac & Blood Pressure Drugs and Polio Survivors (12/7/2016)

Dr. Bruno’s Original Post: CARDIAC DRUGS, HIGH BLOOD PRESSURE AND POLIO SURVIVORS

Available here - <http://www.papolionetwork.org/living-with-post-polio-syndrome.html>

Additional Post: My doctor is sending me to a neurologist for my my hand/arm/head tremors and he mentioned propranolol for help with both... he is waiting for the neurologist's recommendation. What do you think?

Dr. Bruno’s Response: The neurologist should be aware that propranolol has been known to cause fatigue in polio survivors.

Additional Post: Before prescribing my new treatment (for newly diagnosed high blood pressure) my physician read the entire above article. Appointment went overtime, she wasn't worried and I know I'll have a good outcome.

On the topic of our “Polio Foot” turning color (12/7/2016)

Original Post: Why does my “polio” foot turn blue/red at times.

Dr. Bruno’s Response: I’ll refer you to this <http://postpolioinfo.com/library/gcold.pdf>

Polio survivors are extremely sensitive to changes in temperature. At merely cool temperatures, most polio survivors report that their feet have always been cold to the touch, their skin a purplish color. However, as polio survivors have aged, 50% report "intolerance to cold" and that their limbs have become more sensitive to pain as the temperature decreases (Owen,1985). Cold was reported to cause muscle weakness in 62% of polio survivors, muscle pain in 60%, and fatigue in 39%. (Bruno & Frick, 1987). When polio survivors were cooled in the laboratory from 86o F to 68o F, motor nerves functioned as if they were at 50o F and polio survivors lost 75% of their hand muscle strength. (Bruno, et al., 1985a) But, although polio survivors are twice as sensitive to pain as those without polio, no increase in pain sensitivity was found at lower temperatures. (Bruno, et al., 1985b)

The reason polio survivors have such trouble with cold is that the parts of the central nervous system that should control body temperature were damaged by the poliovirus. In the brain the hypothalamus (the "automatic computer" that controls the inner bodily environment) was damaged by the poliovirus, including the body's "thermostat" and the brain area that tells your blood vessels to constrict. (Bodian, 1949)

In the spinal cord, the nerves that carry the message from the brain that tells the capillaries in the skin to contract when it's cold were also killed by the poliovirus. (Bodian, 1949) Thus, polio survivors are unable to stop warm blood from flowing to the surface of the skin as the outside temperature drops. This allows loss of heat from the blood near the surface of the skin and causes the limbs to cool. When the limbs cool, arteries carrying blood to the skin and veins that should carry blood out of the skin narrow passively as they get cold, trapping blue venous blood in the capillaries and causing the feet to look blue and to become even colder. The cold skin chills the motor nerves, causing them to conduct more slowly and to be less efficient in making muscles contract. The cold also chills tendons and ligaments (like putting a rubber band in the freezer) making movement of weak muscles more difficult. As polio survivors know, it takes hours under an electric blanket or a long, hot bath to warm cold legs and regain strength.

However, when polio survivors take a hot bath, blood vessels do exactly the opposite of what they do in the cold. Polio feet and legs become bright red as arteries and veins relax and blood rushes to the skin. Then, when polio survivors stand to get out of the tub, they can feel dizzy or even faint as blood pools in their legs and causes their blood pressure to drop (see Bruno,1997). The pooling of blood in the feet also explains why polio survivors' feet swell, swelling that increases as they get older. And polio survivors' easily losing body heat explains why they have an increase in symptoms, especially cold-induced muscle pain, as the seasons change.

Polio survivors need to dress as if it were 20o F colder than the outside temperature. They need to dress in layers and wear heat retaining socks or undergarments made of polypropylene (marketed as Gortex or Thinsulate) that should be put on immediately after showering when the skin is warm.

Polio survivors need to remind doctors that EMGs or nerve conduction tests must be performed in a room that is at least 75o F to prevent false abnormal readings and that a heated blanket is necessary in the recovery room after polio survivors have surgery (Bruno, 1996).

Bodian, D. (1949) Histopathological basis of clinical findings in poliomyelitis. American Journal of Medicine, 6, 563-578.

Bruno, R.L., Johnson J.C., Berman W.S. (1985a). Motor and sensory functioning with changing ambient temperature in Post-Polio subjects: Autonomic and electrophysiological correlates. In L.S. Halstead and D.O. Wiechers (Eds.), Late Effects of Poliomyelitis. Miami: Symposia Foundation.

Bruno, R.L., Johnson, J.C., Berman, W.S. (1985b). Vasomotor abnormalities as Post-Polio Sequelae: Functional and clinical implications. Orthopedics,

Bruno, R.L. (1996) Preventing complications in polio survivors undergoing surgery. PPS Monograph Series. Volume 6 (1). Hackensack: Harvest Press.

Bruno R.L. (1997) Chronic fatigue, fainting and autonomic dysfunction:

Further similarities between post-polio fatigue and Chronic Fatigue Syndrome? Journal of Chronic Fatigue Syndrome, 3, 107-117.

Owen, R.R. (1985). Polio residuals clinic and exercise protocol: Research implications. In LS Halstead and DO Wiechers (Eds.): Late Effects of Poliomyelitis. Miami: Symposia Foundation.

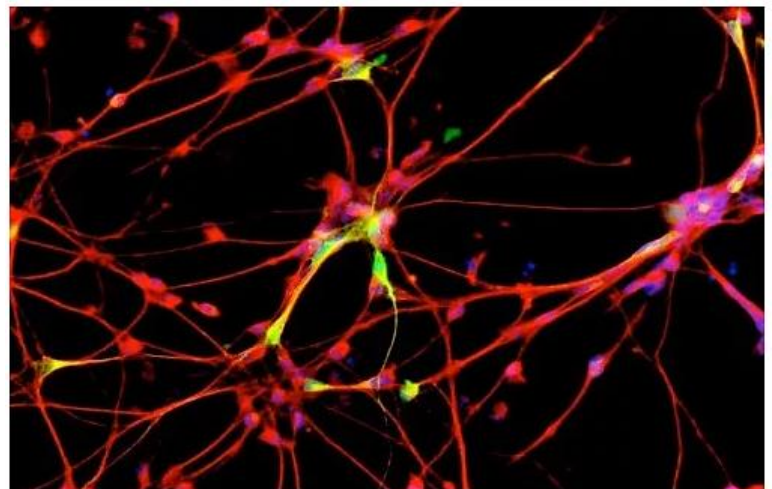
On the topic of an experimental drug for PPS "Brain" Fatigue (12/8/2016)

Dr. Bruno's Original Post: In a few years this experimental drug may be of help to polio survivors with brain fatigue. It "prevented the decline in...brain glucose (blood sugar) utilization," which our research found is a big problem for polio survivors with fatigue and brain "brownout."

A new investigational drug originally developed for type 2 diabetes is being readied for human clinical trials in search of the world's first treatment to impede the progression of Parkinson's disease following publication of research findings today in the journal Science Translational Medicine.

Newswise — GRAND RAPIDS, Mich. (Dec. 7, 2016)—

"We hope this will be a watershed moment for millions of people living with Parkinson's disease," says Patrik Brundin, M.D., Ph.D., director of Van Andel Research Institute's Center for Neurodegenerative Science, chairman of The Cure Parkinson's Trust's Linked Clinical Trials Committee, and the study's senior author. "All of our research in Parkinson's models suggests this drug could potentially slow the disease's progression in people as well."



Credit: Photo courtesy of the Patrik Brundin Laboratory, Van Andel Research Institute.

Dopamine-producing brain cells after treatment with MSDC-0160.

Until now, Parkinson's treatments have focused on symptom management. If successful in human trials, MSDC-0160 would be the world's first therapy to treat the underlying disease and slow its progression—potentially improving quality of life and preventing the occurrence of falls and cognitive decline. It may also reduce or delay the need for medications that can have debilitating side effects, says Brundin.

Parkinson's disease afflicts between 7-10 million people worldwide, including an estimated 1 million Americans, and these numbers are expected to increase dramatically as the average human lifespan increases. There is currently no cure, and first-line treatment has remained relatively unchanged since the introduction of levodopa in the 1960s. Tom Isaacs, a co-founder of The Cure Parkinson's Trust who has lived with Parkinson's for 22 years, says MSDC-0160 represents one of the most promising treatments the Trust's international consortium has seen to date. "Our scientific team has evaluated more than 120 potential treatments for Parkinson's disease, and MSDC-0160 offers the genuine prospect of being a breakthrough that could make a significant and permanent impact on people's lives in the near future," says Isaacs. "We are working tirelessly to move this drug into human trials as quickly as possible in our pursuit of a cure."

MSDC-0160 was developed by Kalamazoo, Michigan-based Metabolic Solutions Development Company (MSDC) to treat type 2 diabetes. In 2012, Brundin recognized it as an exciting drug candidate because of its mode of action, proven safety in people, local availability and the start-up company's interest in collaborating on drug repurposing initiatives. After four years of work, the effects of the drug in the laboratory exceeded Brundin's expectations.

The novelty of MSDC-0160 stems from a recently revived revelation that Parkinson's may originate, at least partially, in the body's energy metabolism. The new drug appears to regulate mitochondrial function in brain cells and restore the cells' ability to convert basic nutrients into energy. Consequently, the cells' ability to handle potentially harmful proteins is normalized, which leads to reduced inflammation and less nerve cell death.

"Parkinson's disease and diabetes may have vastly different symptoms with unrelated patient outcomes; however, we're discovering they share many underlying mechanisms at the molecular level and respond similarly to a new class of insulin sensitizers like MSDC-0160," says Jerry Colca, Ph.D., co-founder, president and chief scientific officer of MSDC.

While Brundin says he is eager to see MSDC-0160 launched into a clinical trial in Parkinson's disease, he's equally excited about the possibility of testing the drug in Lewy body dementia and other cognitive decline conditions, such as Alzheimer's disease. "This is an immensely promising avenue for drug discovery," says Brundin. "Whatever the outcome of the upcoming trial for Parkinson's, we now have a new road to follow in search of better treatments that cut to the root of this and other insidious diseases." The Cure Parkinson's Trust and Van Andel Research Institute are currently working with MSDC to address regulatory issues and obtain funding to organize the clinical trial, which Brundin hopes can begin sometime in 2017.

Funding for the research was provided by Van Andel Research Institute, The Cure Parkinson's Trust, the Campbell Foundation, and the Spica Foundation.

The paper's authors include Anamitra Ghosh, Trevor Tyson, Sonia George, Erin N. Hildebrandt, Jennifer A. Steiner, Zachary Madaj, Emily Schulz, Emily Machiela, Martha L. Escobar Galvis, Jeremy M. Van Raamsdonk and Patrik Brundin, all of Van Andel Research Institute; William G. McDonald and Jerry R. Colca, both of Metabolic Solutions Development Company; and Jeffrey H. Kordower, of Rush University Medical Center and Van Andel Research Institute.

Watch this piece...

<http://www.newswise.com/articles/view/666015/?sc=mwhn>

On the topic of Having PPS and Anesthesia (12/15/2016)

Original Post: My husband has PPS and CODP. Recently he has started very rapidly losing the use of his hands. His neurologist did several nerve conduction test, etc., today and is going to do surgery (after the first of the year) on both wrists to relieve compressed nerves in his upper arms. Since he will not be completely asleep, is there anything special, other than the usual PPS warnings about anesthesia and oxygen that the anesthesiologist needs to know?

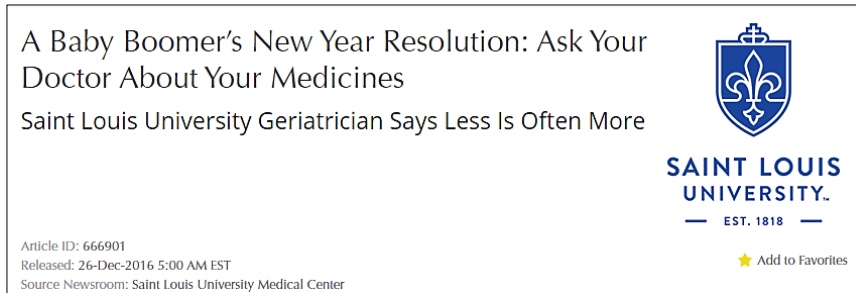
Dr. Bruno's Response: Typically, polio survivors need half the amount of general anesthesia and at least twice the amount of local anesthesia. We also recommend using Marcaine instead of Xylocaine as the local. This is covered in the surgical article and in the dental article in [Post Polio Library](#). Please take a look about the information in both on local anesthetics.

Additional Post: We met with the surgeon and printed out the info you suggested (above) and he understands about the anesthesia and oxygen. He was very interested in getting this info. I have it printed for the anesthesiologist when we meet with him/her as well. Thank you.

**Editor's Note: The articles referenced above, and an updated version of the Anesthesia Warning Card, with a "scan" code is available [HERE](http://www.papolionetwork.org/-anesthesia-warning.html). <http://www.papolionetwork.org/-anesthesia-warning.html>
Not online? Feel free to email us.

[On the topic of Asking our Physicians about Medications](#) 12/26/2016)

Dr. Bruno's Original Post: A Polio Survivor's (Baby Boomer) New Year's Resolution – because Less is often More !



<http://www.newswise.com/articles/a-baby-boomer-s-new-year-resolution-ask-your-doctor-about-your-medicines>

We have to take an active role and know the "why and what" before taking or changing meds. What's most important is that *discussion with our physicians is VITAL*.

[On the topic of Swallowing and Choking issues](#) (12/27/2016)

Original Post: I read *The Polio Paradox* two years ago. Everything that's happening to my body is exactly what you wrote about. Everything is worse, including difficulty swallowing. I have also realized that running out of breath when speaking is pretty frightening.

Dr. Bruno's Response: Poliovirus-damaged brain stem neurons that control the vagus nerve that carries commands from the brain stem to activate muscles in your throat, esophagus, stomach and intestines, are the cause of swallowing problems. Vagus damage disrupting the normal functioning of the gut may explain our 2000 Post-Polio Survey findings that swallowing difficulty, diarrhea, colitis, ulcers and constipation are as much as six times more common in polio survivors than in non-polio survivors.

Polio survivors have also been reporting another problem: Food sticking in the upper esophagus. We think this is due to the vagus not stimulating esophagus muscles to move the food downward. When food gets stuck, irritation triggers a painful esophagus muscle spasm that also stimulates the vagus nerve.

Polio survivors having frequent or severe trouble swallowing problems should see an ENT doctor and may need a video swallow study to find their cause and make sure something other than PPS isn't causing the trouble. Usually, slowly eating small bites of food, drinking water after each bite, tucking your chin or turning your head to one side when you swallow, swallowing several times, and eating your big meal when you're most rested is all that's needed to treat swallowing problems. A speech therapist can help you learn tricks to beat your unique problems.

On the topic of PPS and CFS/ME and was it Really Polio (12/29/2016)

Original Post: Does anyone know if it is possible to have ME/CFS or Fibromyalgia as well as PPS? The reason I'm asking, is that my health has deteriorated dramatically over the last three years - gradual for 10 years and then Wham! I seem to have symptoms that tick nearly every box of both other conditions, including seizures, which began a few years ago. I have seen my PPS consultant and an Epilepsy specialist, but neither has had the time or inclination to look at my symptoms as a whole package. Only recently have I read that seizures can be a symptom of ME. My gut instinct is that I may have more than one problem, but I don't know if there are recognized relationships between the three (PPS/ME/Fibromyalgia) conditions.

Dr. Bruno's Response: Diagnostically, PPS is ALWAYS a diagnosis of exclusion; but so is ME/CFS. If you have ME/CFS fatigue you can't have PPS fatigue. If you had polio and develop late-onset fatigue, you have post-polio fatigue.

Unfortunately, Fibromyalgia has become an "anything goes" diagnosis for muscle pain.

The term Fibromyalgia = muscle pain.

Seizures are not known to be a symptom of chronic fatigue, but fatigue often follows a seizure.

Additional Post: So if ME/CFS and PPS is always a diagnosis of exclusion how do you know which is the right diagnosis? What about people who were never diagnosed or even knew they had polio? They could possibly have PPS and not know it.

Dr. Bruno's Response: If you had polio, develop fatigue and nothing else is going on, you have PPS. The statistics are that if one child in a household had paralytic polio another child had "non-paralytic" polio. Often the paralyzed kid got all the attention and even obvious muscle weakness was ignored (as in two studies showed at the time), in about 40% of the so-called "non-paralytic" kids.

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<http://www.papolionetwork.org/bruno-bytes.html>

Scroll down the page (through the Current Month posts).

Previous months are located there, and are available by "clicking" on them, in easily printable PDF format