Post Polio Information

By J. Ann Singleton

Post polio syndrome was first mentioned during the civil war. One of the earliest articles in a major medical journal was in 1887. Polio is a viral disease. It was contracted the same way as any viral disease such as the common cold. It usually presented with high fever sometimes lasting a month or so and severe body and head pain and cramping.

There are three kinds of damage to the body, which were called “types”. These are, Non paralytic, paralytic, and bulbar. In the days before viral typing, this was the easiest way to categorize what happened during and after a case of polio. In non-paralytic polio, damage to the body was thought to be minor to non-detectable. In paralytic polio, parts of the body were paralyzed to a greater or lesser degree. In bulbar polio, the breathing and swallowing muscles were damaged.

Since sixty percent of the neurons have to be damaged before weakness is identified in an area of the body, it was very imprecise to diagnose the “type” of polio by the damage demonstrated at the time of illness. This is also the reason why in post polio, areas thought to be unaffected previously can show weakness or symptoms today.

There are also three major strains or **“**viral types” of the polio virus, Brunhilde (type I), Lansing (type II), and Leon (type III). Today there are over one hundred variations of these three types identified. Even after viral typing was available, very few of the total cases were typed at the time of illness to identify the strain. Typing was more important for the researchers trying to find a vaccine than for a family doctor to deal with the consequences of the illness. With three strains of the virus, it is theoretically possible to have more than one polio illnesses at the same time or to have a polio illness more than once.

The most common strain was the Brunhilde (type I), which caused arm, leg and sometimes breathing damage. This strain was identified in 1945. The Lansing (type II) was least likely to cause paralysis but did damage the brain stem. This is the kind that caused non-paralytic polio that was referred tot by a variety of names such as summer flu etc. It is thought by researchers today to cause many cases of Chronic Fatigue Syndrome (CFS) in persons living before the vaccines for polio were in use.The third, Leon (type III) was very rare. It caused arm and leg paralysis and severe brain stem damage. All three viral types caused damage to both the brain and spinal cord. The use of the term “type” when used in post polio literature should always be defined as much confusion can arise between the results of the illness and the viral strain responsible.

The first part of the body to be affected is the brain. In every case of polio they have autopsied and tested they have found the same pattern of damage. Today specific imaging can find the residual scars in the brain resulting from the original polio attack. These areas of scaring do not get worse with age, but can be of concern leading to unnecessary testing if the patient and doctor are not aware of their presence.

These findings indicate that chronic fatigue is associated with impairments of attention and information processing speed but not of verbal memory or higher level cognitive processes both in patients with Chronic Fatigue Syndrome and in polio survivors. Given the histopathology documentation of frequent and severe poliovirus lesions in the brain's activating system, it was hypothesized that damage to the Reticulating Activating system and basal ganglia is responsible for both fatigue and impaired attention in polio survivors.

Brain stem centers were found to be "involved in even mild cases" of polio (5). The midbrain reticular formation was "always severely altered" (6), being "heavily peppered throughout" (711) with lesions that were "very common and often severe" (7). Hypothalamic, thalamic and caudate nuclei, the putamen and globus pallidus were also lesioned by the poliovirus (11,12). Neurons in the periaquiductal gray, locusceruleus, median raphe nuclei and especially the substantia nigra were also damaged or destroyed by poliovirus infection (5, 811). Dopamine production is impaired

Recent studies that relate the symptoms of post polio fatigue and Chronic Fatigue Syndrome (CFS) to clinically significant deficits on neuro-psychologic tests of attention, histo pathologic and neuro radiologic evidence of brain lesions, impaired activation of the hypothalamic pituitary adrenal axis, increased prolactin secretion and EEG slow wave activityThis damage is found in the hypothalamus, the brain stem and the reticulating activating system. To over simplify things when we are stressed or fatigued the ability of our muscles to function is markedly reduced.

The hypothalamus also helps regulate our body temperature. Most post polio's bodies are stuck on air conditioning. We radiate heat at all times which can lead to difficulty when the temperature around us drops below 70˚. We can suffer from hypothermia in the mid to high 60S. Our ability to think and move our muscles drops very fast when we are chilled and it may take hours for polio damaged body to regain a normal temperature. Our normal temperature is usually a degree or two below the national average of 98.6˚. We should take a wrap or jacket when sitting in a cold office or restaurant. It is usually wise to dress for a temperature ten degrees cooler than the actual temperature and dress in layers on cool days and the reverse in hot weather.

Chilling can be very dangerous after surgery and lead to shock if the recovery room staff are not aware of the dangers Post polio patients should make sure that the surgeon and anesthesiologist are familiar with post polio. We also react differently to anesthesia, which can cause problems. Many post polio patients have difficulty with

lying flat on the back or with legs higher than the head due to chest wall muscles and diaphragm weakness.

The hypothalamus regulates the pituitary gland and damage changes our production of neurotransmitters, which can include serotonin, norepinephrin, and dopamine.It also regulates the thyroid gland and it is common for hypothyroid ism to slowly manifest over time.

Fatigue is the most commonly reported and most debilitating PostPolio Sequelae (PPS) affecting the more than 1.8 million North American polio survivors. In two national surveys, 91% of polio survivors reported new or increased fatigue, 41% reported fatigue significantly interfering with performing or completing work and 25% reported fatigue interfering with self care activities (1,2). Fatigue was reported to be triggered or exacerbated by physical overexertion in 92% and by emotional stress in 61%.

Importantly, polio survivors differentiate between the physical tiredness and decreased endurance they associate with new muscles weakness and a "brain fatigue" that is characterized by problems with attention and cognition. Between 70% and 96% of polio survivors reporting fatigue complained of difficulty with concentration, memory, attention, word finding, maintaining wakefulness and thinking clearly, with 77% percent reporting moderate to severe difficulty with these symptoms (3). Stress either physical or mental exacerbates this.

These findings indicate that chronic fatigue is associated with impairments of attention and information processing speed but not of verbal memory or higher level cognitive processes both in patients with CFSand in polio survivors. Given the histo pathologic documentation of frequent and severe poliovirus lesions in the brain's activating system, it was hypothesized that damage to the Reticulating Activating System and BG is responsible for both fatigue and impaired attention in polio survivors.

These reports are reminiscent of the symptoms associated with nearly two dozen outbreaks during this century of Myalgic Encephalomyelitis (ME) and Chronic Fatigue Syndrome (CFS), conditions that can be related historically, clinically, anatomically or physiologically to poliovirus infections. These relationships will be described in an attempt to suggest a possible common pathophysiology for all post viral fatigue syndromes (PVFS). For example, Parkinson's disease (PD) patients demonstrate not only an impaired ability to "transfer attention" (37) but also marked fatigue (38). In one survey 33% of PD patients reported that fatigue was their "most disabling symptom" (39). "Excessive fatigue" was reported in another study by 48% of PD patients (40), fatigue that was associated with abnormal glucose metabolism or blood flow in the putamen and supplementary motor area (cf. the Brain Fatigue Generator Model, below). It is noteworthy that one of the first descriptions of cognitive dysfunction in PD (41) could serve as a definition for Post Viral Fatigue Syndrome,

i.e., syndromes "characterized by a diminution of voluntary attention, spontaneous interest, initiative and the capacity for effort and work, with significant and objective fatiguability, and a slight diminution of memory."

"Atypical" Poliomyelitis and Chronic Fatigue. Beginning in Los Angeles in 1934 and continuing for more than twenty years, there were over a dozen outbreaks of a disease that was at first diagnosed as poliomyelitis, then as "abortive" or "atypical" poliomyelitis and finally named " " (ME) (26,42). Like poliomyelitis, initial symptoms of ME included headache, neck pain, low grade fever and myalgia that were often followed by paresis. Irritability and anxiety, symptoms typical of the encephalitis accompanying bulbar polio (cf. 22), and even a few cases of post acute parkinsonism (42) were noted. Patients demonstrated hyper-somnolence and "conspicuous changes in their levels of concentration" that persisted for months after the acute illness (26). Slowing of the EEG with the emergence of theta activity, similar to that documented in polio survivors, was also noted (4446,
cf. 23).

Unlike poliomyelitis, there were frequent complaints of numbness or paresthesias, usually no respiratory involvement, infrequent paralysis or muscle atrophy and almost invariably no fatalities. CSF protein was usually normal and poliovirus was never isolated from ME patients. Also unlike poliomyelitis, recovery from the acute symptoms of ME sometimes required months or years (43). Most patients were left with a marked "exhaustion and fatiguability" that were "always made worse by exercise (and) emotional stress" (26). Patients continued to demonstrate fatigue, hyper-somnolence, impaired concentration, and reported "an inordinate desire to sleep," anomia, that they were "not as quick or incisive in thought as before, (had) a decreased ability to learn and a decline in their short term memory" for years after the acute episode (26).

Despite the differences between poliomyelitis and ME, an association with the poliovirus was suggested by the fact that, of the more than one dozen ME outbreaks before the introduction of the Salk vaccine, nine occurred during or immediately after outbreaks of polio and several involved hospital staff who cared for polio patients (42,4753).

The reticulating activating system manages our ability to sleep and wake. It is located in the brain just above the brain stem and the upper brain stem. It routs signals from the spinal cord and muscles up to the brain. Damage to this area can also affect the regularity of respiration when sleeping and ability to concentrate when awake.

Some people had only the gastrointestinal form or non paralytic type of polio. It is a mystery how many of these people have had silent central nervous system involvement and are still subject to post polio. Sixty percent of the neurons must be dead before function problems are noted on most physical tests. It has become clear that individuals who were diagnosed as having had non paralytic Poliomyelitis also can develop new symptoms. Between 1933 and 1983, a total of 484,862 cases of paralytic

and non paralytic poliomyelitis were reported to the Centers for Disease Control. The CDC admits that this number is an under stimulation of the true incidence of polio and it is now thought that for each case of reported polio there were 200 more cases not diagnosed. or reported because reporting requirements were not instituted until the late 1950's. There are over1.5 million persons alive today who are at risk for developing symptoms.

Bulbar polio involved the brain stem where the centers for the cranial nerves are located. The cranial nerves involve smell, vision, and eyeball movements. The trigeminal nerve and facial nerve service cheeks, tears function, gums, and muscles of the face, etc. The auditory nerves provide hearing, the glossopharyngeal nerve controls (in part) swallowing and functions in the throat, taste, and tongue movement. The vagus nerve involvement sends signals to the heart, intestines, lungs, and the accessory nerves that control upper neck movement. Thus bulbar polio can affect any or all of these functions.

One of the symptoms of bulbar involvement is a loss of breathing function later in life. The main cause of respiratory difficulties in polio survivors is the inability to take a deep breath due to weakness of the respiratory muscles and to cough effectively to clear secretions and mucus plugs. Sometimes when we cough our ability to bring air back into the lungs is a problem. The lack of a good cough can lead to further pulmonary complications such as pneumonia, including aspiration pneumonia and respiratory insufficiency. Most doctors are unaware of a common post polio problem, namely **Alveolar Hypoventilation *Without* Primary Bronchopulmonary Disease and Positional Apnea**. How many of you are not able to sleep while laying flat on your back? I imagine that you probably sleep with your head elevated on a couple of pillows and curled up on your right side. Why is this? Your lungs are not damaged by the polio but the muscles that make them function usually are. It is very easy to cough the air out of your lungs but be unable to pull the air back in. This can result in a cough that many doctors interpret as asthma and treat with inhaled steroids, which can damage the nerves causing further loss of function. This reflex involves vagus nerve irritability and miss diagnosis can lead to a round of erroneous treatment. Steroids are among the class of drugs that can cause serious problems to post polio patients. If you were or are a smoker these problems are magnified and your life will be much shorter.

Individuals who used an iron lung, or barely escaped one, during the acute phase should be aware of potential problems to avoid under ventilation and possible respiratory failure. Those survivors who had high spinal polio and who have upper body weakness and/or diaphragm weakness may also be at risk. Under ventilation (hypoventilation) begins during sleep and results in an elevation of carbon dioxide levels and a decrease in oxygen levels in the blood. Causes include respiratory muscle weakness, and sleep apnea, (central, obstructive, or mixed).

Other contributing factors include diminished vital capacity (VC). A diminished vital capacity is common in everyone who is aging. Polio survivors who have

impairment of the diaphragmatic and/or intercostal (rib) muscles combined with normal changes due to aging may lose vital capacity at a rate of 6090% greater than normal, thus exacerbating the development of under ventilation.

Signs and symptoms include: daytime sleepiness, morning headaches, feeling unrested in the morning, need to sleep sitting up, sleep disturbances (including dreams of being smothered, nightmares, restless sleep, interrupted sleep). Other signs may be snoring, fatigue or exhaustion from normal activities, poor concentration and impaired intellectual function, shortness of breath on exertion, fewer words per breath, use of accessory muscles to breath. A weak cough increases susceptibility to respiratory infection and pneumonia.

An individual experiencing a combination of any of the above should immediately seek a respiratory evaluation by a Pulmonologist who is familiar with post polio problems, preferably one experienced in neuromuscular disease, and possibly a sleep study. When vital capacity declines from examination to examination to a range under 1 L (liter), assistance with ventilation must be considered,

Under ventilation is a very serious condition, which if ignored and left untreated can lead to death. Under ventilation can be aggravated by the use of oxygen therapy. Oxygen treatment is not indicated to treat these problems. Our oxygen sensing ability is compromised and without proper carbon dioxide levels we will cease to breathe altogether. This is very important when we are not fully conscious as during surgery, accidents resulting in shock, and being under the influence of depressive type drugs such as opiates. Emergency personnel need to know how to administer a cough assist, as it is known in respiratory therapy. If an individual's cough is weak (less than 4.5L. [Liters] per sec.), the onset of a cold must be dealt with as a serious and potentially life threatening situation. Manually assisted coughing techniques can provide deep breaths to augment cough flows. Techniques include postural drainage, percussion, and abdominal thrusts administered by a therapist or a caregiver who places their hands on the individual's abdomen and deliver thrusts timed just before the person coughs. For an adult with less than 1.5L of vital capacity, the individual should be given the greatest volume of air that he or she can hold in the lungs, either with an Ambu bag or volume ventilator, before the thrust In an emergency situation we should not be strapped flat on our backs by emergency personnel, but rather they should keep us in an upright position or be ready to breath for us with an Ambu bag or positive pressure respirator. In an emergency situation a Heimlich maneuver can restore breathing, but it will take quite a few minutes to allow our regular breathing rhythm to reassert itself. As most emergency personnel and doctors are completely unaware of these problems, it is very important that you carry a one page synopsis of your medical history with you at all times.

Our immune system tried to fight off the virus that was attacking our body. Unfortunately the protein signature of the outer covering of the virus is sixty percent identical to the myelin sheath, which forms the insulation on our nerves. To oversimplify

again, this has left us with frayed insulation on all of our nerves. The virus traveled all over our body and it did not just target one or two parts of the body such as a leg or arm, but damaged all parts of the body to a greater or lesser degree.

As our body tried to fight off the virus and recover, its prime directive was to restore function. In order to do this, it put into use all the reserve tissues that are needed to repair the injuries and declining function of aging. This is why those who contracted polio prior to the age of 10 years old had a higher chance of a more normal recovery. The nerve tissue had not differentiated, that is to say it had not decided what form it would take when it matured and could be patched into whatever use was needed. Once a nerve cell is differentiated it can seldom be reprogrammed to switch functions. One of the systems usually used for spare parts is the system that the muscles use to tell the brain that they are being overused. Simply, we cannot always tell when our muscles are being overused. One of the first symptoms of overuse is irritability. Since the muscles cannot contact the brain directly the signals are sent via the sympathetic nervous system resulting in irritability. The resulting irritability can be hard on those around us.

If this is ignored, the signals activating the muscles are degraded, the muscle starts to twitch, and then signals of pain are felt. The major problem in post polio is to be aware of our limits and cease the activity at the first sign of irritability. Once the pain hits, the muscles and nerve pathways are being damaged, sometimes permanently. Sometimes my family and friends see the signs before I do, and tell me that I need to take a break.

Due to the damage brought about by the poliovirus, many muscle cells were orphaned. When nearby nerve cells sprouted out to adopt the orphans the sprouts are never as strong as the original equipment. In my case, my legs, the muscles only have ten percent of the nerve pathways and mass that they originally did. Since each nerve can activate up to 1000 muscle cells. This means that each nerve is driving 1000 muscle fibers. Each remaining muscle fiber has to do ten to 100 times the work for which it was designed. If I walk one block, my muscles have to do the same work as a normal person walking over ten blocks. The cells are using many times the oxygen and cellular nutrition. In effect, the pain I experience is a type of runners burn caused by the buildup of lactic acid and waste products. In my case they routinely build up to tens of times the normal level. This is why it takes ten or more times the normal rest to get that muscle group back to normal. These over used muscles become inflamed and the resulting inflammation can set off many disease processes in the body. This is why tylenol and opiates are of little use in treating the cause of pain.

We must think of our body as a rechargeable battery. The battery will die if it is not recharged fully. Another way to think of it is to liken your mobility time to a checking account. If you do not replace what you have used you will overdraw your account and the more times you do that the more likely it is that the bank will no longer honor your

checks. If you chronically over use any muscle group, it will cease to be able to function.

There are many drugs that can cause problems for people with polio damage. We live in a time when most patients want an instant fix for their ailments, an antibiotic for an illness or a pill or shot for pain. Our doctors are conditioned to respond to these expectations. As we age it is not unusual for patients to go to a different doctor for each problem. This can be especially dangerous as one doctor may be unaware of what other doctors have prescribed or the interactions and contra indications for other conditions. One doctor simply can't be current on everything. Insurance companies and HMO's rely on the primary care physician to keep everything straight. The doctor may not be as well trained in specific drugs as the other doctors or specialists. Many doctors simply do not have time to fully research the drugs they prescribe and rely on information supplied to them.

This leaves us with a potentially serious problem. We as patients may innocently take a medication that may cause further problems or even give us a new ailment completely. As we age we can't remember everything and many doctors do not care or have the time to listen carefully to what we have to tell them. Dr. Paul D. Stolley, M.D. (Professor of Medicine, University of Pennsylvania School of Medicine) wrote the Preface for the book "Worst Pills Best Pills". In it he states, " For older Americans, nothing less than the closest attention to their health problems is adequate. Although many older adults can benefit from drugs which lower blood pressure, reduce the pain and discomfort of angina or arthritis, or treat other common ailments, they are extraordinarily sensitive to adverse effects of medicines, such as drug induced Parkinsonism, confusion, decreased coordination, falls and hip fractures, and mental deterioration. Thus the best medicine may be no medicine. A medicine that is fine for one person may not be good for another.

If a drug is needed, the choice could be made by the active and informed participation with a sympathetic and unhurried physician. Furthermore, the continual monitoring of the patient is necessary after drug treatment is initiated. Is the desired therapeutic effect occurring? Are there adverse effects? When should the drug be stopped? Is there the possibility of an undesirable drug interaction because of the other drugs the patient is taking?" (Ed. note. I recommend you read this book and refer to it or one like it when new prescriptions come your way.)

Many doctors do not realize that post polio carries with it drug interactions and consequences that the normal population does not have to deal with and therefore not many doctors are familiar with them. They may linger in affect long after their stated duration.

1. **Beta Blockers** Inderal, Propandolol, Cardinol, Corgard, Inderide, Lopressor, Tenormin, etc. (Block beta cells which make the muscles work)
2. **Benzodiazapams**e.g. Valium, diazepam (Muscle relaxants reduce the ability to control our muscles)

3. **Opiates** (central nervous system depressants) e.g. Darvon, Demerol, Morphine, Tylenol 3 (Our central nervous system controls our muscles)
4. **Steroids** Prednisone, Cortisone, etc.(Our mylin sheath is frayed and degraded and these chemicals can act as an acid on our raw nerves.)
5. **Muscle Relaxants** e.g. Norgesic, Norflex, (includes also Bensodiazapams Valium) also drugs similar to them in chemical structure such as Quinine, Quinidine and Procaniamide
6. **Cholesterol lowering medications in the "statin" family** as well as Baycol, Lopid® (gemfibrozil) and Mevacor® (lovastatin)
7. **Medications to increase bone density** Miacalcin, Menasor, Zocot, Fozamax, etc.
8. **Some Antibiotics** e.g. Tetracyclines

Body mechanics become very important to the continuing normalcy of our movements. Many of us have scoliosis or curvature of the spine from long years of sitting in a chair that was not fitted to our body. In chairs, one size does not fit all. With any chair we sit in, it is very important that the arm of the chair be exactly under our forearm when our elbow is bent at 90˚. This keeps our back straight and allows our lungs to hold the maximum amount of air. It will also allow us to use the arms of a chair to assist us in standing and to pivot in and out of a car. The back of our seat should touch our lower back when our knees are bent at a 90˚ angle. This should be the case in all chairs that we routinely sit in. I found my ideal easy chair, a Chippendale wing chair where the arms are the correct height is and the back is high enough to support my head. My sofa is adapted by keeping a matching pillow on the arm to raise the armrest to a correct height.

Choosing a vehicle
Make sure that your car is also ergonomically correct for you. **If your seat is lower than your knees, do not buy it.** The muscles in the front of the thigh wear out first in most post polio's, If you have to lift your legs over a threshold of more that two or three inches, you will have to expend precious muscle use to get in or out. In my case, getting in and out of such a car is thirty minutes of walking time that I will loose. Most Asian and European cars have armrests that are much too short to be of use as a pivot point

Do not purchase a car or van with the thought of getting a hoist or ramp installed. Most of the cars and vans today **cannot**l be adapted because of in floor storage and crumple zones. I drive a Dodge Grand Caravan with a VMI conversion. It was purchased with the conversion in place and is similar to a Braun "Entervan". It has a lowered floor, kneels and has automatic door and ramp in the passenger side. There are conversions for many makes of cars with both side and back entry. The conversion added $17,000 to the price of my car and is not covered by medicare or insurance. If you are still working, your insurance or vocational rehab may pay part of the price. However without my scooter the van hauls a tremendous amount and this has come in handy.

Get a handicapped parking permit if you can. If you have upper arm problems after a long drive or use hand controls, apply for the state toll permit that will allow you to not have to pay tolls on any road, bridge or tunnel. Just give your number to the toll taker instead of your coins. You can also get a transponder that goes into a zero account so that you can use the fast lane. **The distance you walk out side of a building is subtracted from the distance you can walk inside.**

Safety Equipment

When we were young, nobody thought of using helmets, elbow and knee padding, and wrist splints to go rollerskating. Helmets are required in our state for bike riding children. The use of safety equipment is being taken for granted. Few of us think of a cane, walker, or crutches as safety equipment, but it is. One of the first things that we notice when post polio makes itself known is our lack of balance and our difficulty staying erect if we are bumped. The small muscles in our lower legs and ankles assist our balance and wear out very early on in our condition. I may not use my cane in the house where I can usually grab a piece of furniture, and thresholds to trip over have been eliminated. I do not leave the house or car without my cane. I use it to change direction, on my weakest side, for those times when my leg gives out without warning. I use a wrist strap so that I don't have to hold on to it when using that arm for other things.

Canes must be fitted. The best type of cane to use is one where the handle is at 90˚ to the shaft or one that has just a small knob on the top. The top of the cane should come to the first wrinkle in the wrist above the base of the thumb. A cane that is too long or with a crook handle can cause CarpelTunnel syndrome, tennis elbow, and rotor cuff degeneration. If it is too short, your back will not be straight, your lungs will not be able to inflate properly, and you may develop lower back problems.

As our ankle muscles weaken, our ankles tend to pronate or no longer be straight over the heel. This will throw out our knee alignment, hip alignment, and cause lower back problems. Orthotics fitted by a knowledgeable doctor and worn as much as possible can keep everything in line and reduce their related problems. It is best to have the casts made while you are sitting or lying on a table so that the joints can be held in the proper alignment when the cast is made. Those made by standing in a tub of casting material will not be of any use as they can only replicate an already existing problem not prevent it. Proper alignment is vital and can make your moving and walking much more efficient. You will walk with less effort and pain.
Bracing and orthotics should be used before further damage is done *no*t after the fact. The idea is to use assistive devices to prevent further loss of function, not as the result of injury by bad use of available muscle recourses. They are safety equipment not badges of disability.

Think about using a chair or scooter while it is still an option *not* your only alternative to staying in bed. I use mine when I shop alone or to attend outside activities such as art

festivals and theme parks. That saves my energy for enjoying the activity and allows me to not fall apart when it is over. With the scooter, I can carry the packages and grandchildren. I hate having to leave early because I am tired and so do the people with me because they have to leave too.

Range of motion exercises are very important. Muscle control and strength can be regained to some degree with patterning or putting the muscles through full extension and contraction multiple times starting out very gently and being careful to never over-stress the muscle group. If the patient is not able to move the muscle group it can be done by a therapist. Joints will stiffen to the point us uselessness without motion

One of the major problems we have as we age is weight gain. As a rule of thumb, a person must briskly walk for half an hour to use up 100 calories. We do not have the muscle mass or ability to exercise and therefore we gain weight. As a rule of thumb you have to walk a mile to use up 100 calories. This weight gain leads to further loss of function as the more of us there is for our weakened muscles to move the shorter the distance they can move it. As Dr. Bruno suggests, the metabolism of our nerve cells is abnormal due to the damage done by the polio. It is so easy to fall into the trap of having a little caffeine and a carbohydrate snack to give us a little energy, when what we really need is a smaller amount of caffeine and a low carbohydrate, high protein snack that is part of a carefully balanced diet. According to Dr. Bruno “If you want to treat your PPS by mouth, loose weight, and have less fatigue and try the proven dietary “supplement” of taking 16 grams of protein at breakfast”. We must limit our intake of simple carbohydrates, also known as the white foods, including bread, rice, potatoes, and sweets. Many post polio people feel better and have fewer symptoms when their weight is on the low side of normal and their protein intake is higher than their intake of carbohydrates.

Almost all post polio persons are type A overachievers. There is some controversy whether this is a result of the illness and the brain changes inherent in it or the result of therapy and social pressure not to be different. Whatever the cause; we are resistant to anything that makes us seem different than the average person and reluctant to use devices because,we think that using such devices marks us as less valuable than others. Rest is not being lazy, but rather an important part of our recovery of function after use. We normally work harder than the average person and far longer than our bodies should. We are our own worst enemies in refusing to acknowledge what is taking place or to cause a fuss. We are very vain when it comes to using assistive equipment. We are also usually five percent more intelligent than the normal population so start using that extra five percent where it will do the most. Take care of yourself, so that you have as much function as possible, for as long as possible, and have fun doing it

Post Polio Bibliography

1**.“The Merck Manual”** 14th Edition, Merck & Co Inc

2**.“Worst Pills Best Pills”** 1988 Ed. Author Sidney M Wolfe, M.D., Lisa Fugate, Elizabeth P. Hulstrand, Laurie E. Kamimoto and the Public Citizen Health Research Group

3**. "A Warning for Polio Survivors",** (Revised from Jan. 1995), Ruth WylerPlaut, N.J. PostPolio Support group
4. **"Physicians Desk Reference**" 1998 ED, Montvail NJ, Medical Economics Company, Inc.
6.**The Polio Paradox, 2002**, Dr. Richard L. Bruno, Director, The PostPolio Institute Englewood Hospital and Medical Center, Englewood, New Jersey USA 07631
Email PPSENG@AOL.COM Phone (201) 8943724 or TOLL FREE 1877POST.

REFERENCES

1 Parsons PE National Health Interview Survey Washington, DC: National Center for Health Statistics, 1989
2 Bruno RL, Frick NM Stress and "Type A" behavior as precipitants of PostPolio Sequelae In LSHalstead and DO Wiechers (Eds.) Research and Clinical Aspects of the Late Effects of PoliomyelitisWhite Plains: March of Dimes Research Foundation, 1987.
3 Bruno RL, Frick NM, Cohen J. Polioencephalitis, stress and the etiology of PostPolio Sequelae.
Orthopedics 1991;14(11):12691276.
4 Trojan D, Gendron T, Cashman NR. Electrophysiology and electrodiagnosis of the postpolio motorunit Orthopedics 1991;14:13531361.
5 Bodian D. Poliomyelitis: Neuropathologic observations in relation to motor symptoms. JAMA1947;134: 11481154.
6 Guizetti HU. Betrachtungen zur poliomyelitis des hirnstammes. Deutsch Ztschr f Nervenh 1933;131:2942.
7 Bodian D. Histopathological basis of clinical findings in poliomyelitis. Am J Med 1949;6:563578.
8 Barnhart M, Rhines R, McCarter JC, HW Magoun. Distribution of lesions of the brain stem inpoliomyelitis. Arch Neurol Psychiatry 1948;59: 368377.
9 Matzke HA, Baker AB. Poliomyelitis: A study of the midbrain. Arch Neurol Psychiatry 1951;65:115.
10 Peers JH. The pathology of convalescent poliomyelitis in man. Am J Pathol 1942;19:673695.
11 Luhan JA. Epidemic poliomyelitis: Some pathological observations on human material. Arch Pathol1946;42: 245260.
12 Howe HA, Bodian D. Neural Mechanisms of Poliomyelitis. New York, NY: The Commonwealth Fund, 1942.
13 Morruzi G, Magoun HW. Brain stem reticular formation and activation of the EEG. EEG ClinNeurophysiol 1949;1: 455473.
14 Bentivoglio M, Steriade M. Brainstemdiencephalic circuits as a structural substrate of the ascendingreticular activation concept. In M Mancia and G Marini (Eds.) The Diencephalon and Sleep. New York,
NY: Raven Press, 1990.
15 Sakai K, El Mansari M , Lin JS , et al. The posterior hypothalamus in the regulation of wakefulnessand paradoxical sleep. In M Mancia and G Marini (Eds.) The Diencephalon and Sleep. New York, NY:
Raven Press, 1990.
16 Mesulam MM. Attention, confusional states, and neglect. In MM Mesulam (Ed) Principles ofBehavioral Neurology. Philadelphia, PA: Davis, 1985.
17 Pribram KH, McGuinness D. Attention and paraattentional processing: Eventrelated brainpotentials as tests of a model. Ann NY Acad Sci 1992;658: 6592.
18 Jones BE. Influence of the brainstem reticular formation, including intrinsic mono aminergic andcholinergic neurons, on forebrain mechanisms of sleep and waking. In M Mancia and G Marini (Eds.)
The Diencephalon and Sleep. New York, NY: Raven Press, 1990.
19 Robbins TW. Psychopharmacological and neurobiological aspects of the energetics of informationprocessing In GRJ Hockey, AWK Gaillard, MGH Coles (Eds.) Energetics and Human InformationProcessing. Dordrecht: Martinus Nijhoff, 1986.
20 Mandell AJ. Toward a psychobiology of transcendence. In JM Davidson and RJ Davidson (Eds.) ThePsychobiology of Consciousness. New York, NY: Plenum, 1980.
21 Brown JR, Baker AB, McQuarrie I , et al. The Bulbar form of poliomyelitis. JAMA 1947;135: 425 428.
22 Baker AB, Neurologic signs of bulbar poliomyelitis. In Poliomyelitis Philadelphia, PA: Lippincott, 1949.
23 Holmgren BE. Electroencephalography in poliomyelitis In Poliomyelitis Philadelphia, PA: Lippincott, 1952.
24 Meyer E. Psychological considerations in a group of children with poliomyelitis. J Pediatrics 1947;31:
25 Bruno RL, Galski T, DeLuca J. Neuropsychology of PostPolio Fatigue. Arch Phys Med Rehabil1993;74: 10611065.
26 Behan PO, Behan WMH. Postviral fatigue syndrome. CRC Critical Reviews in Neurobiology 1988;4: 157178.
27 Buchwald DPR, Cheney PR, Peterson DL, et al. A chronic illness characterized by fatigue neurologicand immunologic disorders and active human herpesvirus type 6 infection. Ann Int Med 1992;116:103 113.
28 Hyde BM. Myalgic Encephalomyelitis (Chronic Fatigue Syndrome): An historical perspective. In BMHyde J Goldstein, P Levine (Eds.) The Clinical and Scientific Basis of MyalgicEncephalomyelitis/Chronic Fatigue Syndrome . Ottawa, Ontario: The Nightingale Research Foundation, 1992.
29 Bickerstaff ER, Cloake PCP. Mesencephalitis and rhombencephalitis. Brit Med J 1951;2:7781.
30 Barrett AM, Gardner D, McFarlan AM. An outbreak of encephalitis, possibly due to poliomyelitisvirus. Brit Med J 1952;1:13171322.
31 Magoun HW. In Poliomyelitis Philadelphia, PA: Lippincott, 1949, p 250.
32 Duvoisin RC, Yahr MD. Encephalitis and parkinsonism Arch Neurol 1965;12:227239.
33 Heilman KM, Voeller KKS, Nadeau SE. A possible pathophysiologic substrate of attention deficithyperactivity disorder. J Child Neurol 1991; 6 (Suppl):S74S79.
34 DennyBrown D, Yanagisawa N. The role of the basal ganglia in the initiation of movement. In MDYahr (Ed.) The Basal Ganglia. New York, NY: Raven, 1976.
35 Owen AM, Roberts AC, Hodges JR, et al. Contrasting mechanisms if impaired attentional setshiftingin patients with frontal lobe damage or Parkinson's disease. Brain 1993;116:11591175.
36 Echiverri HC, Tatum WO, Merens TA, Coker SB. Akinetic mutism. Pediatric Neurol 1988;4:228230.
37 Bowen FP. Behavioral alterations in patients with basal ganglia lesions. Research Publications: AssocRes Nerv Ment Dis 1976;55:169180.
38 Brown RG, Marsden CD. Cognitive function in Parkinson's disease. TINS 1990;1:2128.
39 Hilten JJ van, Hoogland G, van der Velde EA , et al. Diurnal effects of motor activity and fatigue inParkinson's disease. J Neurol Neurosurg Psychiatry 1993;56:874877.
40 Friedman J, Friedman H. Fatigue in Parkinson's disease. Neurology 1993;43:20162018.
41 Naville F. Encephale 1922;17:369375
42 Acheson ED. The clinical syndrome variously called benign myalgic encephalomyelitis, Iceland Diseaseand epidemic neuromyasthenia. Am J Med 1959;26: 569595.

43 Hyde BM, Bergman S. Chronic aspects of Akureyri Disease. In R Jenkins and JF Mowbray (Eds.) PostViral Fatigue Syndrome. Chichester: John Wiley & Sons, 1991.
44 Ramsay AM, O'Sullivan E . Encephalomyelitis simulating poliomyelitis. Lancet i: 1956;762 767.
45 Galpine JF, Brady. Benign myalgic encephalomyelitis. Lancet 1957;i:757758.
46 Daikos GK, Garzonis S, Paleologue A, et al. Benign myalgic encephalomyelitis. Lancet 1959;i: 693 696.
47 Macrae AD, Galpine JF. An illness resembling poliomyelitis observed in nurses. Lancet 1954;ii: 350 352.
48 Gilliam AG. Epidemiological study of an epidemic, diagnosed as poliomyelitis, occurring among thepersonnel of the Los Angeles County General Hospital during the summer of 1934. US Public Health Bull1938; (No 240): 190.
49 Gsell VO. Abortive poliomyelitis. Helvetica Medica Acta 1949;3/4:169183.
50 Fog T. Vegetative (epidemic?) neuritis. Ugeskr Laeg 1953;115: 12441248.
51 Pellew RA. Clinical description of a disease resembling poliomyelitis seen in Adelaide. Med J Aust1951;1: 944946.
52 White DN, Burtch RB. Iceland Disease: A new infection simulating acute anterior poliomyelitis. Neurology 1954;4: 506516.
53 Deisher JB. Benign myalgic encephalomyelitis (Iceland Disease) in Alaska. Northwest Med 1957;56: 14511456.
54 Sigurdsson B, Sigurjonsson J, Sigurdsson HJ, et al. A disease epidemic in Iceland simulatingpoliomyelitis. Am J Hyg 1950;52:222238.
55 Sigurdsson B, Gudmundsson KR. Clinical findings six years after outbreak of Akureyri Disease. Lancet 1957;i: 766767.
56 Sigurdsson B, Gudnadottir M, Petursson G. Response to poliomyelitis vaccination. Lancet 1958;i: 370 371.
57 Hart RH. Epidemic neuromyesthenia. N Eng J Med 1969;281:797.
58 Henderson DA, Shelokov A. Epidemic neuromyesthenia. N Eng J Med 1959;260:757 764.
59 Hyde BM, Jain A. Clinical observations of central nervous system dysfunction in post infectious, acute onset ME/CFS. In BM Hyde J Goldstein, P Levine (Eds.) The Clinical and Scientific Basis ofMyalgic Encephalomyelitis/Chronic Fatigue Syndrome . Ottawa, Ontario: The Nightingale ResearchFoundation, 1992.
60 Altay HT, Toner BB, Brooker H, et al. The neuropsychological dimensions of postinfectiousneuromyesthenia. Int'l J Psychiatry in Med 1990;20:141149.
61 Jamal GA. Evidence for organic disturbance in Post Viral Fatigue Syndrome. In BM Hyde JGoldstein, P Levine (Eds.) The Clinical and Scientific Basis of Myalgic Encephalomyelitis/ChronicFatigue Syndrome . Ottawa, Ontario: The Nightingale Research Foundation, 1992.
62 DeLuca J, Johnson SK, Natelson BH. Information processing efficiency in chronic fatigue syndromeand multiple sclerosis Arch Neurol 1993;50: 301304.
63 Sandman CA, Barron JL, Nackoul K , et al. Memory deficits associated with chronic fatigue immunedysfunction syndrome. Biol Psychiatry 1993;33:618623.
64 Lonnberg F. Late onset polio sequelae in Denmark. Scand J Rehab Med 1993;Suppl 28: 132.
65 McDonald E, Cope H, David A. Cognitive impairment in patients with chronic fatigue. J NeurolNeurosurg Psychiatry 1993;56:812815.
66 Graffman J, Schwartz V, Dale JK, et al. Analysis of neuropsychological functioning in patients withchronic fatigue syndrome. J Neurol Neurosurg Psychiatry 1991;56:684689.
67 Bruno RL, Cohen J, Galski T, Frick NM. The neuroanatomy of postpolio fatigue. Arch Phys MedRehabil 1994;75:498504.
68 Wasserstrom R, Mamourian AC, McGary CT, Miller G. Bulbar poliomyelitis: MR findings withpathological correlation. AJNR 1992;13:371373.
69 Parasuraman R, Nestor P. Energetics of attention and Alzheimer's Disease In GRJ Hockey, AWKGaillard, MGH Coles (Eds.) Energetics and Human Information Processing. Dordrecht: MartinusNijhoff, 1986.
70 Howe HA, Bodian D. Neuropathological evidence on the portal of entry problem in humanpoliomyelitis. Bull Johns Hopkins Hosp 1941;69:183214.
71 Bodian D, Howe HA. Neurotropism and the genesis of cerebral lesions in poliomyelitis. Bull JohnsHopkins Hosp 1941;68: 5876.
72 Kirkpatrick JB, Hayman LA. Whitematter lesions in MR imaging of clinically healthy brains ofelderly subjects: Possible pathological basis. Radiology 1987;162:509511.

73 Daugherty SA, Henry BE, Peterson DL, et al. Chronic fatigue syndrome in northern Nevada. RevInfect Dis 1991;13 (Suppl 1):S3944.
74 Almkvist O, Wahlund LO, AndersonLundman G, et al. Whitematter hyperintensity andneuropsychological functions in dementia and healthy aging. Arch Neurol 1992;49:626632.
75 Kertesz A, Polk M , Carr T. Cognition and white matter changes on magnetic resonance imaging in dementia Arch Neurol 1990;47:387391.
76 Austron MG, Thompson RF, Hendrie HC, et al. Foci of increased T2 signal intensity in MR images ofhealthy elderly subjects: A followup study. J Am Geriatr Soc 1990;38:11331138.
77 Boone KB, Miller BL, Lesser IM , et al. Neuropsychological correlates of whitematter lesions inhealthy elderly subjects. Arch Neurol 1992;49:549554.
78 Junque C, Pujol J , Vendrell P, et al. Leukoaraiosis on magnetic resonance imaging and speed ofmental processing. Arch Neurol 1990;47:151156.
79 Roelcke U, Kappos L, LechnerScott J, et al. Reduced glucose metabolism in the frontal cortex andbasal ganglia of multiple sclerosis patients with fatigue. Neurol 1997;48:15661571.
80 Gupta KL, Shetty KR, Rudman IW, Rudman D. Impaired nocturnal growth hormone secretion withage in postpolio syndrome. Clin Res 1992;40:688.
81 Neubuerger KT. Bulbar poliomyelitis. In Poliomyelitis Philadelphia, PA: Lippincott, 1949.
82 Petrusz P, Merchenthaler I. The corticotropinreleasing factor system In CB Nemeroff (Ed.) Neuroendocrinology . Boca Raton, FL: CRC Press, 1992.
83 Meyerhoff JL, Mougey EH, Kant GJ. Paraventricular lesions abolish the stress induced rise inpituitary cyclic AMP and attenuate the increases in plasma levels of proopiomelanocortinderivedpeptides and prolactin. Neuroendocrinology 1987;46:222230.
84 Dallman MF, Strack A , Akana S, et al. Feast and famine: Critical role of glucocorticoids with insulinin daily energy flow. Frontiers in Neuroendocrinology 1993;14:303347.

85 Bruno RL, Sapolsky R, Zimmerman JR, Frick NM. The pathophysiology of a central cause of post polio fatigue. Ann NY Acad Sci 1995;753: 257275.
86 Moodradian AD. Geriatric Neuroendocrinology. In CB Nemeroff (Ed.) Neuroendocrinology . BocaRaton, FL: CRC Press, 1992.
87 Miller LH, Kastin AJ, Sandman CA, et al. Polypeptide influences on attention, memory and anxiety inman. Pharm Biochem & Beh 1974;2: 663668.
88 Reinberg A, Briere L, Fraboulet G, et al. Clinical chronobiology of ACTH 117. Chronobiologia1981;8:101115.
89 Strand FL, Cayer A , Gonzalez E, Stoboy H. Peptide enhancement of neuromuscular function. PharmBiochem & Beh 1976;5 (Suppl 1):179187.
90 Van Wimersma Greidanus Tj B, de Wied. Effects of systemic and intracerebral administration of twoopposite acting ACTHrelated peptides on extinction of conditioned avoidance behavior. Neuroendocrinology 1971;7:291301.
91 Demitrack MA, Dale JK, Straus SE, et al. Evidence for impaired activation of the hypothalamic pituitaryadrenal axis in patients with chronic fatigue syndrome. J Clin Endocrinology Metabolism1991;73: 12241234.
92 Majeed T, Dinan TG, Behan PO. Ipsaprione induced ACTH release Evidence of impaired activationof the HPA axis in patients with CFS. Proceedings of the First World Congress of Chronic FatigueSyndrome and Related Disorders: Brussels, 1995, page 103.
93 Bruno RL, Frick NM, Creange SJ, et al. Polioencephalitis and the brain fatigue generator model ofpostviral fatigue syndromes. Journal of Chronic Fatigue Syndrome 1996;2:527.
94 Costa DC, Tannock C, Brostoff J. Brainstem perfusion is impaired in chronic fatigue syndrome. Q JMed 1995;88:767773.
95 Tavio M, Chierochetti F, Bianchini B, et al. Brain positron emission tomography (PET) in chronicfatigue syndrome: A useful tool for differential diagnosis. Proceedings of the American Association forChronic Fatigue Syndrome Conference . San Francisco: AACFS, 1996.
96 Rowe PC, BouHolaigah I, Kan JS, Calkins H. Is neurally mediated hypotension an unrecognizedcause of chronic fatigue syndrome? Lancet 1995; 345:623624.
97 BouHolaigah I, Rowe PC, Kan JS, Calkins H. The relationship between neurally mediatedhypotension and the chronic fatigue syndrome. JAMA 1995;274:961967.
98 Lapp CW. NMH, SOTS and CFS. Proceedings of the American Association for Chronic FatigueSyndrome Conference. San Francisco: AACFS, 1996.
99 Bruno RL. Chronic fatigue, fainting and autonomic dysfunction: Further similarities between post polio Fatigue and Chronic Fatigue Syndrome? J Chronic Fatigue Syndrome 1997; 3:107117.
10
101 McQuarie I. The evolution of signs and symptoms of poliomyelitis. In Poliomyelitis . Philadelphia, PA: Lippincott, 1949, pp.5761.
102 Bakheit A. Abnormal argininevasopressin secretion and water metabolism in patients with post viral fatigue syndrome. Acta Neurol Scand 1993;87:234238.
103 Pan JT. Neuroendocrine functions of dopamine. In T W Stone (Ed.) CNS neurotransmitters andneuromodulators: Dopamine. Boca Raton: CRC Press, 1996:213232
104 Valentino DA, Arruda JE, Gold SM. Comparison of QEEG and response accuracy in good vs poorperformers during a vigilance task. Int J Psychophysiol 1993;15:123134.
105 O'Hanlon JF, Beatty J. Concurrence of electroencephalographic and performance changes during asimulated radar watch and some implications for the arousal theory of vigilance. In RR Mackie (Ed.) Vigilance. New York: Plenum, 1977, pp.189201.
106 Bruno RL, Creange SJ, Zimmerman JR, Frick NM. Elevated plasma prolactin and EEG slow wavepower in postpolio fatigue: Implications for a dopamine deficiency underlying chronic fatiguesyndromes. J Chronic Fatigue Syndrome 1998 (in press).
107 Bruno RL The psychology and psychology of the postpolio brain Keynote address, InternationalPostPolio Conference, Calgary: October, 1997.
108 Marcel B, Komaroff AL, Fagioli LR, et al. Cognitive deficits in patients with chronic fatiguesyndrome. Biol Psychiatry, 1996; 40:535541.
109 Bruno RL, Zimmerman JR, Creange SJ, Lewis T, Molzen T, Frick NM. Bromocriptine in thetreatment of post polio fatigue: A pilot study with implications for the pathophysiology of fatigue. Am JPhys Med Rehabil 1996; 75 (5): 340347.
110 Fagan D, Scott DD, Mitchell M, Tiplady B. Effects of remoxipride on measures of psychologicalperformance in healthy volunteers. Psychopharmacology 1991;105:225229
111 Gear JHS. Non polio causes of polio like paralytic syndrome. Review of Infectious Diseases 1984; 66:379382.
112 Shindarov LM, Chumakov MP, Voroshilava MK, et al. Epidemiological, clinical, andpathomorphological characteristics of epidemic poliomyelitislike disease caused by enterovirus 71. JHygiene Epidemiol Microbiol 1979; 3:284295.
113 Dowsett EG. Human enteroviral infections. J Hospital Infections 1988;11:103115.
114 Heathfield KWG, Pilsworth R, Wall BJ, Corsellis JAN. Coxsackie B5 infection in Essex, 1965, withparticular reference to the nervous system. Q J Med 1967; 36:579595.
115 Vorashilova MK, Chumakov MP. Poliomyelitislike properties of ABIV coxsackie A7 group ofviruses. Prog Med Virol 1959; 21:106110.
116 Grist NR. Type A7 Coxsackie (Type IV poliomyelitis) viral infection in Scotland. J Hygiene(Cambridge) 1962, 60: 323325.
117 Tuteja U, Pandya G, Bharagava R, et al. Enterovirus specific IgM responses in children with acuteand chronic paralytic syndrome. J Tropical Med and Hygiene 1995; 98: 367372.

Dr. Richard L. Bruno is chairperson of the International PostPolio Task Force and director of The PostPolio Institute andThe International Centre for PostPolio Education and Research at Englewood (New Jersey) Hospital and Medical Center.

Send Comments to J. Ann Singleton, 481 Meadowood BLVD, Fern Park FL 32730.