# Polio Survivors' Page

# ACUTE ANTERIOR POLIOMYELITIS, SOME OBSERVATIONS

*by Edward Snapp, R.P.T.*

## INTRODUCTION

My opinion concerning polio, the disease process and its aftermath of paralysis, pain, weakness, incoordination, deformity, and fatigue is a result of my experiences. I have had the opportunity to work with thousands of patients with polio during the epidemics in the late 1940's and 1950's. I followed many of these people through years of rehabilitative efforts. In recent years, I have again come in contact with a number of these patients and have observed the development of this latent complex of symptoms and disabilities called post polio syndrome.

What you read in this discourse is the product of my clinical observations and conclusions. I do not ask you to believe what I say without question. Rather, I hope you can add your thoughts to mine and develop logical conclusions which may benefit you as well as others.

Presently, my work involves the progressive development of the body and its neuromuscular processors. It offers a different view of polio-related disabilities and places them in developmentally, rather than nerve root, correlated categories.

In effect, the observable pattern of neuromuscular failures seems to be related to certain sequences of developmental relationships, rather than the specific destruction of nerve tissue by the infection. Coupled with the previously mentioned connective tissue disruption, a more logical picture of polio-related disabilities is produced. Also, it allows for a projection of the effects and a possible treatment approach for Post Polio Syndrome.

## BACKGROUND

Acute Anterior Poliomyelitis has been a major concern in the field of medicine from time to time. Polio was annually epidemic in the United States in the 1940's and 50's, until the Salk and Sabin vaccines. During those years, I was first a survivor of the disease and second, a physical therapist in charge of the Polio and Rehabilititation Center in Houston,Texas. After years of working with many patients who had polio, I came to some conclusions about the nature of the disease, which I believed merited further investigation.

Early treatment of polio consisted of casting, splinting and bracing to limit deformities; even so, they persisted. The most notable change in treatment was initiated by Sister Elizabeth Kenny, who advocated the use of very hot packs to the affected areas during and after the acute phase. She also stressed extensive muscle stretching and a pattern of specific muscle "reeducation" which required the patient to contract isolated muscles in an attempt to retain function. These treatment modalities were more effective than previous methods, although the paralysis and deformities persisted.

Specific symptoms and other related factors were consistent among patients with polio. The involved

muscles tended to be exquisitely painful when compressed. Attempting to lift with support under the shoulders and thighs could cause great discomfort which might persist for hours. Stretching the muscles produced excruciating pain first in tendonous areas, then into muscular areas as the area being stretched increased. Residual pain, which might last for hours, could frequently be relieved by applying hot packs.

Reasonable coordination by patients could be expected, if the involved muscles were sufficiently strong and there was no limitation of range of motion within the scope of activity. Inadequate muscle strength and/or range of motion, coordination and response to postural stresses, load factors, etc., were major factors in creating and enhancing deformities.

During the acute phase, many patients experienced severe pain and "muscle spasm," especially in posterior trunk muscles. This might be expected in any spinal cord inflammatory process. While I do not argue against the term "muscle spasm," I believe it fails to encompass the real cause of discomfort and dysfunction. Many patients retained a significant amount of symptomatic discomfort for many years, regardless of the amount of stretching, exercise, massage, etc. used to alleviate the pain. Involved muscles were still painful when pressed; tendonous areas were still painful when stretched. Additionally, they did not feel the same as uninvolved muscles when palpated.

People with polio feel as though they can move as they did before their disability occurred. They can still relate to such feelings as running, stepping up high steps, and swimming fast, even though the level of involvement prohibits these activities.

For those who have incurred significant involvement, the surviving muscles are used as substitute or secondary muscles, and may be subject to rapid fatigue. Because many survivors of polio use maximum effort to maintain movement and/or posture, fatigue becomes a major factor in the patient's ability to accomplish routine daily activities.

During and after the epidemic, it was accepted that the terminal motor nerves were destroyed in the anterior horn. Patients who had demonstrated total zero function in an upper extremity still rated total zeros after seven years. Muscle reeducation efforts, total pattern exercise efforts, reflex efforts, hot packs, cold packs, electrical stimulation, etc. produced no reaction. However, on one occasion, the electrical stimulation unit was inadvertantly raised to the maximum intensity. While using tetanization current, we observed a solid contraction of a paralyzed deltoid section. Although painful, by using these maximum intensity, intermittent applications, various muscles contracted at each impulse.

However, the same results were not achieved when used on the lower extremities, perhaps due to the depth of the surface tissues. While this should have been an effective treatment, patients were unable to recover any function. The muscle showed the ability to contract in sufficient bulk to affect movement which meant the terminal nerves were intact; yet the patient was unable to produce any voluntary contraction.

As stated above, the intensity of the electrical current had to be extremely high to elicit a contraction. Other unaffected muscles in the near vicinity required far less intensity of contraction and showed violent and painful contraction when stimulated with the higher intensity. The same proved true when stimulating the same muscle groups on the opposite and uninvolved upper extremity.

We knew that nerves of greater diameter carry impulses more easily, and damaged nerves may have more resistance to impulse transmission. In general, we believed that the disease was more or less specific to the anterior horn calls. Skin sensations were little, if any, affected by the disease. However, the intensity of the pain in the muscular and tendonous areas, both during and after the disease process, seemed to indicate sensory involvement. The acute and post-acute phases displayed the symptoms of connective tissue inflammation. This inflammation probably included cell membranes, the membranes covering the axions of the nerve and the many parts of the muscles.

Polio did not follow the expected pattern in nerve root involvement, especially if the infection was centered in tracts or contiguous areas of the spinal cord. Polio was famous for its "scattered" involvement. We became aware of many similarities in our patients. Total extremities were frequently involved and rotation patterns were often seen. Possibly the most common pattern we observed was the weakness of the quadriceps, failure of the tibialis anterior, tightness of the tensor fascia lata, inward rotation of the thigh and outward rotation of the leg. This pattern was the cause of many surgical procedures. There were other correlated dysfunctions which appeared to be scattered paralysis and frequently intractable contractures.

This research took place around the time the polio epidemic ended. With the discovery of the Salk and Sabin Vaccines, the interest in polio ended. Therefore, nothing was published and further research was discouraged. Hence, the post polio symdrome epidemic was far advanced before medical science acknowledged its existence.

## POST POLIO SYNDROME

It has become obvious that a significant percentage of nervous system dysfunctions result from an apparent "deprogramming" of specific portions of the central nervous system memory. This appears to be due to a reaction to either physical of chemical shock. "Deprogramming" is very like the reaction when a computer's power plug is inadvertently disconnected. The machine and its circuits remain intact and undamaged, but the program is lost. For the program to be reinstated, it must be returned to the proper file from which it was lost, or it cannot be retrieved or accessed.

It is possible, indeed likely, that a considerable amount of paralysis from polio is the result of this type of mechanism. It is also likely that continued stress and chronic severe fatigue produces similar trauma, causing post polio syndrome. If this is true, there is a possible avenue to reinstate function, at least to the segments that were functioning before the onset of post polio syndrome. In addition, there is a possibility of reinstating function in some of the previously involved areas affected by the initial disease process.

Since 1989, we have used a very comprehensive treatment program for patients with post polio syndrome. The treatment's original intent was to relieve pain and try to slow the decline of strength and/or function. The response of our patients has been more than gratifying.

The treatment regime is based on the concept that multiple factors affect progressive pain and weakness. To be truly effective, it appears necessary to address all these factors simultaneously. Present indications are that the treatment format may both stop the immediate progression of post polio syndrome and simultaneously improves strength, range of motion, endurance and comfort in a high percentage of patients with post polio syndrome. The methods and modalities used are all basic physical therapy procedures, but the programs must be individually structured for both clinical and home use.

The results to date have been positive and promising. Patients who completed 2 weeks or more of therapy, have maintained improved function through simple, individualized home programs:

All patients report increased energy, less fatigue, and faster recovery from fatigue.

Incidences of post-treatment pain were caused by exceeding home program guidelines. Each occurance was corrected without medication by following advice given in telephone consultation.

Most patients reported additional improvement of function after discharge from clinical treatment.

Some patients have reported the ability to contract muscles which have not functioned for years, including some that had not been active since the acute disease period.

There have been:

NO NEGATIVE REACTIONS FROM TREATMENT

NO SUSTAINED FATIGUE

NO INCREASED WEAKNESS

NO INCREASED PAIN.

Those of us who have had the privilege of working with these patients are pleased and excited by the results observed to date. We are continuing to make every effort to improve the effectiveness of our treatment methods, to enhance post treatment home programs, to preserve and increase improvements and to provide consultation which may be of value in maintaining the best quality of life possible.

**SYMPTOMS AND SEQUELAE OF POST POLIO SYNDROME:**

1. The progressive debilitation appears to be related to chronic progressive fatigue.
2. The fatigue patterns follow specific lines of effort dictated by previous patterns of paralysis and weakness.
3. Failure of primary patterns, and lack of redundant functional ability results in total functional breakdown and inability to recover from fatigue in day to day routine.
4. Patterns of dysfunction observed in polio patients do not follow nerve root distribution as should be expected in an infection or inflammation of the spinal cord tissue.
5. Patterns of dysfunction do follow developmental patterns as defined in CCDT developmental chronology.
6. It is therefore assumed that at least part of the failure of function may occur due to traumatic shock to the developmentally based computer function.
7. It is similarly postulated that near total fatigue in a developmental pattern may cause that pattern to be evaluated by the nervous system as incapable of function and thus removed from the sequence of available function. Thus, even the rested tissues would not be recalled for functional effort.
8. Muscle tone in involved muscles is frequently so poor that the sensation if loading, effort or resistance are either poorly equated, so the stress and load factors which would normally dictate the specific muscles, positions and efforts necessary for function totally fail to initiate effort or shift posture and motor patterns to favor secondary or substitute pattern efforts. These secondary patterns are invariably less efficient, require much more effort and intent by the patient, and fatigue much more quickly.
   Patients who lack sufficient tone to relate to position or loading are most likely to: (a) progressively fail to initiate contraction in a required muscle, (b) have insufficient strength for functional activity, (c) fail to maintain contraction unless intentionally applying thoughtful effort to that specific muscular area. In any case, these areas remain in functional failure from sensory deprivation either from or, in addition to, motor nerve deprivation.
9. To attain reasonable function, a developmental sequence, lost through trauma to the CNS, must be reprogrammed in its proper position relative to its original genetically specified point in the embryonic or fetal developmental process. It is, therefore, impractical and frequently, impossible to "teach" these functions through aware efforts.
10. Re-establishment of lost or erased information in any computer requires that it be replaced in its original and proper file to be accessed automatically through its original program. This is similarly true in our human computer system.
11. Trunk, hip, and shoulder failure commonly precedes peripheral failure.
12. Destruction of nerve bodies on the anterior horn incapacitates a motor nerve permanently.
13. Scarring of an efferent axon reduces both the rate and intensity of the motor stimulus.
14. Scarring of connective tissue surrounding muscle fibers and muscle bundles intensifies muscle pain, sensation, limiting muscle function and range of motion, increasing fatigue.

15. Shock from disease, toxicity, fever and/or near total fatigue may have deprogrammed areas during the disease process.
16. Severe chronic fatigue of motor tracts or initiating or controlling sensory tracts may have deprogrammed areas.

## FACTORS WHICH AFFECT PARALYSIS, WEAKNESS, FATIGUE AND PAIN IN POST POLIO SYNDROME

*Anterior horn cell loss* - irrecoverable loss of nerve-muscle function forcing change of coordination, pattern, balance, primary muscle.

*Incoordinate contracted areas* - multiple mini spasms within a muscle. Severe pain in rest and activity.

*Trigger point pain* - resulting from posture and stress fatigue. Remotely radiated or distributed pain, fatigue or sensory disruption.

*Scarred fascia* - limits range of motion, produces pain on stretching.

*Scarred neural sheath* - inhibits nerve impulse transmission.

*Developmental muscle sensation failure* - produces hypotonia or atonia causing difficulty in initiation or sustaining muscle contraction.

*Computer failure* - loss genetically programmed pattern movements or inability to properly respond to stimulation.

*Complexing failure* - inability to maintain multifactor coordination.

*Transition failure* - loss of function or loss of balance when changing from extension loading to flexion loading or any change of balance.

*Chronic fatigue* - progressive non-recovering fatigue producing increasing feelings of weakness or exhaustion.

*Aging* - probably the true trigger mechanism for Post Polio Syndrome. Genetically initiated changes in activity levels, postures, responses, etc.

I FIND LITTLE EVIDENCE THAT "OVERUSE" IS A SIGNIFICANT FACTOR IN POST POLIO DYSFUNCTION.

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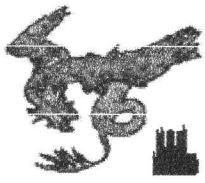

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