Polio Survivors' Page

"Getting Social Security Benefits"

from the Nebraska Polio Survivors Assn.
with thanks to POGOS New Mexico Easter Seal Society Newsletter

If you have worked 20 out of the past 40 quarters and have contributed to the Social Security fund during that time, you may be eligible for the Social Security disability program. Apply at any Social Security office as soon as you become disabled. Ask SSA for their pamphlet called "Disability." (05-10029)

In order to qualify, you must show that you are unable to engage in any significant gainful activity due to a medically determinable impairment (physical or mental) which has or is expected to last 12 continuous months or is expected to lead to death.

This is very difficult to do. The Social Security disability program is extremely tough to get into, so it is important to give the government exactly what it needs to qualify you.

Whether or not you are accepted will probably depend upon your ability to get your doctors to provide the information SSA requires for a favorable decision. They must tell the truth-and that truth must include **EXTENSIVE DETAILS** from your medical records. They must also **ACT QUICKLY**. Each doctor you list (and you will need to name every doctor who has information on your post-polio condition) will have ten days to respond to the inquiry from SSA.

WHAT YOU SHOULD DO

It is in your best interest to contact your doctors ahead of time and discuss what you are doing. Then continue to check with each of them until they have sent the required info to SSA. Keep after them to get it done within the time limit.

Unsubstantiated opinions from your doctor won't do. **THE SSA ISN'T INTERESTED IN THE IDEA THAT YOUR DOCTORS BELIEVE YOU ARE DISABLED AND UNABLE TO WORK.** They require a mountain of **FACTS** to substantiate your claim. These should be very strong, detailed letters. The doctors must make SSA understand your situation through what they write.

SSA wants to hear about lab findings and test results. They want to know exactly how your problem limits your ability to work. They want to know precisely what it is that you are unable to do. Doctors should remember that it is SSA that determines whether or not you are disabled (not the doctors) and SSA makes this determination, in part, based on specific information provided by your doctors. It is up to your doctors to **SHOW HOW** you are disabled.

FOR EXAMPLE:

- What did your EMG show?
- What are the results of your muscle tests?
- What are the numeric degrees of range of motion of involved joints?
- What are the specifics of your pulmonary tests?

- What do your swallowing tests show?
- On what dates were specific treatments started and stopped?
- What X-rays and further studies need to be referenced to illustrate details of your case?
- How can the doctors demonstrate your fatigue:

- Exactly how much of a certain activity can you do before wearing out?

- How can your doctors demonstrate your weakness?

> Copy will be sent once this error sorted.

Transfer interrupted!

or carry?
How long can you stand and/or walk?
How long can you sit?
How are you at climbing, balancing, stooping, kneeling, crouching, crawling, reaching, pushing?

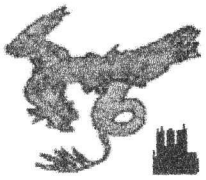
- How can your doctors demonstrate your pain?
- What are your other limitations?

- How are you at handling objects. fingering, feeling?
- How is your eyesight, your hearing?
- What speech difficulties do you have?
- What swallowing difficulties do you experience?
- What environmental restrictions do you experience, such as reactions to temperature extremes, dust, fumes?
- Specifically how do the limitations you have affect what you do?

- How long have you had these impairments and how long are they expected to last?
- Will any of them result in death?

Remember, if you need assistance with Social Security Disability, call toll free 1-800-772-1213. SSA can answer most questions over the phone. You can also write or visit any Social Security office in the state. Request to speak to a disability counselor if your SSA office has one.

See the Social Security Manual on Post Polio Claims for the specific requirements.



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Polio Survivors' Page

The Late Effects of Polio: From Taming a Mysterious Syndrome to Managing the Ravages of Time

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Good morning. It is a real pleasure to be here today and see so many old friends, even a patient from Michigan. I am really thrilled to be here in Southern California and participate in this Conference on Aging with a Disability. I have been communicating with Dr. Campbell for a couple of years now, sharing some of our different work, and becoming aware of this study's goals. I am delighted to hear the results and learn what has been found by comparing two major kinds of disability groups. I have also been involved with conferences on spinal cord injury and aging. There was a large one last spring in Denver. It is striking to note the number of similarities between the health issues faced by aging people with any type of long-standing disability from a motor impairment; i.e., quadriplegia, paraplegia, polio with paraplegia or arm weakness, stroke, and hemiplegias. As people live many years with these conditions, a number of similar new problems often develop.

Dr. Campbell asked me to speak about some of the implications of recent research for our thinking about the late effects of polio. First, however, I should give some explanation for why I came up with the subtitle for my talk: "From Taming a Mysterious Syndrome to Managing the Ravages of Time." This title addresses a changing perspective over the last ten years on the questions: What are the causes of the late effects of polio? How should we view them? To what do we attribute them? What can we do about them?

Before I address the question of causes for the late effects of polio, I need to say a few words about "languaging." I have found that many of the apparent disagreements between physicians, and sometimes between polio survivors and their physicians, about the late effects of polio or what to do about them relate to the labels that people use for what they are experiencing. I would like to begin by sharing with you the labels that I will use in this talk and their definitions.

New Health Problems Among Post-Polios

Table 1 shows several terms that have been used to describe the late effects of polio. One way you can think about them is as new health problems among people with a past history of polio. I have tried to adopt some of the Institute of Medicine's new terminology, which includes the notion of secondary conditions. Secondary conditions are any new health problems that occur in people with another primary disabling condition. In this instance, if you have had paralytic polio, then any other new medical condition that may develop in your life can be correctly labeled a secondary condition, since it comes afterward in time. This has often been confused in the literature, which uses the term secondary complication. Actually,

"secondary complication" is redundant because all complications are secondary, since they follow the primary condition in time. A complication, as we normally think about it, would fit into the second category in Table 1, a late effect of polio (the primary condition). Late effects of polio are specific new health problems, or disabilities and rehabilitative problems, which result from chronic polio-caused impairments (typically muscle weaknesses). If you have a muscle weakness or a paralysis after your polio, you have a polio-related muscle impairment, which may or may not result in some loss of function in your life (a functional limitation). If the functional limitation prevents you from performing an important life activity or role, then you have a disability.

Post-polio syndrome is a term that has become very confusing, and sometimes very frightening, which is why I used "mysterious syndrome" in the subtitle of this talk. When people first started talking about post-polio syndrome it was in this context: No one understands what is happening to polio survivors; something has to be done about these new problems; why won't anyone acknowledge that these problems are real? Dr. Lauro Halstead has offered a definition of post-polio syndrome. It includes new muscle weakness, fatigue, and pain, which are the most common new symptoms that develop in a group of people with long-standing polio. I believe that the new disability or handicap that occurs as a consequence of these new disabling symptoms is the critical issue for defining who has post-polio syndrome. If there are two, or even only one, of the three symptoms, I would say a person has post-polio syndrome if he or she is having new functional limitation or disability as a result of the symptoms.

I have often been asked the question, "If you have post-polio syndrome once, can you recover from it and have it again?" The answer relates to how you define the term post-polio syndrome. I have the viewpoint that a person could become newly impaired but then recover from the disability. Even if the new symptoms do not totally disappear, if the person is rehabilitated and newly accommodated to a degree of impairment, has figured out how to cope with it, and is no longer disabled from it, then the person would have recovered from the post-polio syndrome. Of course, this means that later in life a new problem could come along and again cause greater impairment and disability. So a person could have post-polio syndrome a second or third time.

The other term that has been used in reference to new problems among polio survivors is post-polio progressive muscular atrophy. This term is more of a medical diagnosis which allows people to be classified in a lexicon of neurological diseases. It refers to the pathology of abnormal changes in motor neurons which result in new muscle weakness. Atrophy, which is a shrinkage of muscle, may or may not accompany the new weakness. Slowly progressive weakness and atrophy probably occur in all muscles that have either clinical or subclinical signs of chronic partial denervation and reinnervation from previous damage to motor nerve cells, such as the damage from old polio.

The Need to Understand "Why"

The major question I want to address this morning is "why?" Why are people developing the late effects of polio? Do the symptoms really result from some type of new muscle condition, such as post-polio muscular atrophy? Are they a result of some yet unknown triggers in the body which affect the nervous system? Or are the majority of the new problems being experienced by polio survivors really due to the cumulative effects of aging? While I pose this question as a dichotomy, my own opinion is that there is truth to both positions.

You may ask: "Why is it worthwhile explaining to consumers, as well as professionals, why people develop the late effects of polio?" I believe there are good reasons for both professionals and consumers to understand why. One reason is that from a professional perspective, how we treat a condition, a disease, or a disability will relate to what we understand to be causing it. Different causes for symptoms will necessitate different treatment approaches, even if the severity of a symptom such as weakness is exactly the same. Understanding the cause of symptoms should guide management strategies because it may give us different expectations for the person's future. This relates to the expected life course of a person's

disease, including expected new functional limitations and disabilities, as Dr. Campbell discussed so well.

From a lay person's perspective, the important reason for discussing "why" is what some psychologists have called "the human need to understand." Any time something happens to us, there is an inevitable response: Why did that happen to me? We always ask ourselves that question afterward. A number of researchers are finding out that how you answer that question will have a great deal to do with what happens afterward. How you respond and experience something that happens to you -- for example, a disease or injury -- may be independent of what the physical reality is.

But, as another famous philosopher said, "What is reality?" A study was done with accident victims. It found that a person's outcomes did not relate to whether most other people believed that an accident was the victim's fault or another person's fault. What mattered was how the injured person himself explained what happened to him. The people who did the best after a disabling injury were not the ones who primarily blamed others or who blamed themselves for what happened. They were the people who could accept some responsibility for their own part in causing the accident, even if everyone else in the world would have said, "It was all the other person's fault." I certainly enjoyed the remarks of the stroke survivor who wrote the book titled Stroke: An Owner's Manual (see Josephs, 1992, under Plenary #3). This title implies that you have to take ownership and responsibility for what has happened to you, to take charge of the experience of post-stroke living that you are going to have.

Recent Post-Polio Research

Now I want to share with you, briefly, some of the research about what is causing the late effects of polio. The leading theory for the last ten years is that most new weakness occurring in polio survivors is due to a disintegration of the motor units. The words disintegration and/or deterioration may not be the best words because they sound very negative. Nevertheless, there is a shrinking in size of the motor unit: the nerve cell, its attached nerve axons, and supplied muscle fibers. The leading theories for why this disintegration is going on have included: immunological factors, aging itself, chronic overuse from too much exercise or abuse, environmental factors, and other metabolic or circulatory factors affecting muscle and nerve.

In order to understand these theories, it is necessary to review nerve and muscle physiology related to polio. Within the spinal cord are nerve cells that connect through an axon, which is like a long wire that goes out through the body, to eventually reach muscle. Once nerve fibers reach muscle, they branch in various directions in smaller and smaller branches until they finally reach an end plate. The end plate forms the connection between nerve and muscle.

An electrical impulse starts in the nerve cell and runs down the axon like a little electric current. When it reaches the end plate, it causes a small release of chemical that makes the muscle fiber contract. Individual muscle fibers interdigitate so a muscle is not really one long contracting band, but many intertwining short fibers. Each muscle fiber is connected to its nerve by one end plate. All of the muscle fibers that are connected to the same nerve will contract at the same time. Normally, when an electrical impulse comes down an axon, all the muscle fibers connected to it will contract.

After polio, some nerve cells die from the viral infection. If all the nerves that reach one muscle are lost, then the muscle will completely atrophy and there will be no voluntary movement. However, more commonly, after polio not all, but only some, of the nerves to a particular muscle died. It could be 10%, 50%, or perhaps 75% of the cells that died. If even 10% of the nerves that normally reach a muscle survive, useful movement in the muscle will remain. The muscle will be weak, but it will probably be strong enough to be quite useful. This fact leads to some of the problems that occur in polio survivors.

There are several mechanisms by which people recovered strength after acute polio. One was that some cells temporarily didn't work and then recovered. This often happened in what was called "nonparalytic polio." These people had transient paralysis. Within several weeks, they were essentially fully recovered in

terms of their strength. In these instances, cells probably did not die, but they temporarily didn't work. A second mechanism for regaining strength after acute polio was muscle hypertrophy -- survivors worked hard in therapy programs with exercise. What exercise does is make each little muscle fiber a little fatter. It gets more protein in it. When all the larger muscle fibers contract, you get more force. The person becomes stronger.

The third mechanism for becoming stronger is more interesting and relates to what we think accounts for the late effects of polio. It is called "nerve sprouting." This is a compensation of the surviving nerve cells. New axon branches grow out from axons of surviving nerve cells within the muscle, so that the new axon sprouts reach some of the muscle fibers which had lost their original nerve supply. Thus, in the post-polio muscles with mild or moderate weakness, surviving motor units are much larger; that is, there are more muscle fibers connected to the same nerve than is the normal case. Research has shown this to be about two and one-half to three times as many muscle fibers connected to an individual nerve cell as would occur normally.

Another issue is that newer research has shown that sprouting is not a one-time event. Muscle with partial loss of nerve supply will constantly be showing some muscle fiber breakdown and some new muscle fiber growth. Muscle tissue is actually much more dynamic than most people think. Everyone is aware that bone, for example, is constantly being broken down and built up new in the body. This happens at a very fast rate when you are young and your bones are growing and shaping. When you get older, it is only gradual. When you become very old, the dissolution becomes much faster than the building up of new bone, and the bones become thinner, a condition known as osteoporosis.

Muscle metabolism is similar. There is a constant building up of new muscle fiber and constant breaking down of some existing muscle fiber. If the balance between the muscle build-up and the muscle breakdown shifts to greater breakdown, then the person becomes weaker. A very strong stimulus to muscle breakdown is disuse. Breakdown also accelerates with aging and with many medical illnesses that result in decreased activity.

In post-polio syndrome, disintegration of motor units results from a dying-off of the previous nerve sprouts. Whereas post-polio nerve cells after full recovery and rehabilitation were much larger than normal nerve cells and supplied two and one-half to three times as many muscle fibers, in the later stages of post-polio these nerves shrink back to their original size. Therefore, the post-polio person experiences his muscles becoming weaker again, because he is losing the compensation mechanism, so effective in his younger years, that immediately followed the original loss of nerve cells from polio.

On a microscopic level, we know that this dying-back of giant motor nerves is not an all-or-nothing phenomenon. There are many stages of degeneration during which some of the nerve fiber branches are sick or in the process of slowing down but are not yet unable to function. At this stage there is often a defect in the release of acetylcholine, the chemical that connects the nerve impulse to muscle contraction. This fact probably explains why sometimes replacing acetylcholine will help some polio survivors (see Trojan & Cashman, 1989).

Another problem can be what is called "conduction blocking." This occurs when the electrical impulse comes down the nerve, reaches the branch point, and goes only one way. Thus the impulse bypasses some of the nerve fiber branches. This phenomenon is also thought to occur when you have slow deterioration of the metabolism in some of the nerve fiber branches.

Immunological Studies

Now let us consider some of the recent research about what triggers nerve and muscle deterioration in some post-polios. Immunological theories about the late effects of polio have been proposed because polio was a viral infection, and viruses affect the immunological system. The immunological system is our

body's primary defense against infection. As the public knows since the AIDS epidemic, viral infections can lead to later changes in the immunologic system. There have been studies that have shown the antibody abnormalities in symptomatic post-polios, particularly antibodies in the cerebrospinal fluid.

Recently a great deal of interest was generated by a study published by Sharief in 1991 in the New England Journal of Medicine. This study looked at oligoclonal immunoglobulin bands in cerebrospinal fluid and found they were predominant and elevated. Particularly the antipolio virus IG-M antibodies were elevated in 56% of the post-polio survivors, but in none of the asymptomatic post-polios or controls studied. This certainly suggests something very specific to the post-polio condition.

Other research studies have contradicted this finding, and the jury is still out as to whether there is a strong correlation between these abnormal antibodies in the cerebrospinal fluid and the late effects of polio or post- polio syndrome. To me, it is very intriguing that post-polio syndrome may be associated with immunological abnormalities because the immune system is greatly affected by general health, by your emotional health, and by life style. These are issues that a person can control. Dr. Bernie Siegel has written a great deal about what you can do as a cancer survivor to control and influence your immunological system, and what this can do to cancer cell growth. This issue should be remembered by post-polios.

Electrophysiological Studies

The next area of research is electrophysiologic studies, or EMG studies, of nerve and muscle functioning in post-polios. To summarize the work done by a variety of people, weakened post-polio muscles with partially denervated and reinnervated fibers have increased fiber density and increased jitter on single fiber electromyographic studies. Additionally, increased motor unit size has been found on macro-EMG, and its presence correlates with increased jitter (Trojan, Gendron, & Cashman, 1991). What do these findings mean? Essentially, they confirm the earlier theories that proposed these abnormalities would result from a shrinking back in the size of enlarged motor units that are typical of partially paralyzed post-polio muscles.

Animal Studies

A number of animal studies and some clinical studies have shown progressive impairments of neuromuscular transmission with age and years since onset of nerve loss. This confirms a problem with the release of acetylcholine at the connection between nerve and muscle in post-polios. Dr. Cashman labels this problem "a syndrome of delayed failure of reinnervation." In other words, rather than calling it a disintegration or a deterioration of the nerve cells, he calls it a delayed failure of reinnervation, which emphasizes that the growth of new nerve fibers doesn't keep pace with the rate of loss of nerve fibers.

I want to share with you -- especially the professional group in the audience -- one recent animal study by Dr. Pachter in 1991. It is exciting because it is the closest thing yet to an animal model for the post-polio syndrome. He took a group of mice and cut one of the nerve roots at the level of the spinal cord to one of the major leg muscles. What that means is, he created a leg muscle that had lost half its normal nerve supply and he had created a partially denervated muscle. He then tested the muscle for twitch and tetanic tension. Twitch tension means that you give one electrical shock to a nerve, and measure how much strength is produced by the contracting muscle that responds to that electrical impulse. In tetanic tension you give a long volley of repetitive electrical shocks to the muscle so that all the muscle fibers eventually contract and contribute their amount of strength to the force that is measured.

When these tests were done at one month after root section, as you would expect after half the nerves to a muscle are lost, both the twitch and the tetanic tensions or forces in the muscle were reduced. Three months later, however, only the tetanic tension was reduced. This means that now one twitch of the muscle was just as strong as it was before the muscle lost half its nerve supply, or as in the control mice. However, if you did the repetitive electrical shock, which would be more similar to an endurance phenomenon of repetitive muscle use, the tetanic tension was still reduced. At six months there was no difference between

the control animals and the ones with nerve damage.

Essentially, the remaining nerve roots to the muscles had completely compensated and were successful in taking over, from the standpoint of producing force or tension, both individual twitch tension and the tetanic twitch tension.

Next, the researchers studied the mice after nine months. By then, both the twitch and tetanic tensions were again reduced. When they sacrificed the mice and looked at their muscles under the microscope, changes of chronically denervated and partially reinnervated muscles were found, and these changes were very similar to what is typically seen in mice that are considered old at 20 months of age. This kind of mouse normally has a life span of 24 months. Therefore, the changes that are associated with normal aging in mice were already seen at nine months in those muscles which had been used actively after partial nerve loss. The mice had been able to run around easily during the time when they had normal strength in the partially denervated muscles.

I describe this study as a concurring piece of animal research evidence to support the idea that the post-polio syndrome may result from a deterioration of hyperfunctioning motor units, which may result from metabolic failure of nerve cell bodies that have had to compensate for the loss of nerve cells supplying the same muscle over a long time.

Hormonal Studies

Next, I would like to describe some new hormonal studies. They were done in Milwaukee, and some of you may have read about them in the lay press. They involve the role of growth hormone and aging. Growth hormone is secreted by the pituitary gland. After adolescence, a very low, but constant, level of growth hormone is still produced. Recently people have begun to measure these very small amounts of growth hormone and look at the result in relation to typical events associated with aging. Dr. Rudman tested growth hormone levels in a group of polio survivors, both those with symptoms of post-polio syndrome and those who were not complaining of any new difficulty (Rudman & Shetty, 1991). He was measuring somatomedin C, the active part of growth hormone, which stimulates nerve regeneration and sprouting and muscle protein synthesis. It has been shown in animal studies that growth hormone does play an essential role in allowing the reinnervation process to continue on a constant level in muscle.

Recent studies have shown that by the age of 40, some people show an arrest of growth hormone production and low levels of somatomedin C. If you first look not at polio patients but at an aging population in general, gradually rising percentages of people over age 40 will show an arrest of growth hormone. It is believed that this relates to what we think of as signs of aging, such as baldness, muscles becoming smaller, arthritis, difficulty walking.

When Dr. Rudman looked at post-polio syndrome patients, he found that nine out of ten with new symptoms had low levels of somatomedin C, whereas all of those who were stable post-polios still had normal levels. So he postulated that in some polio survivors with post-polio syndrome, there is an arrest of growth hormone. This provides another interesting piece of theoretical knowledge about post-polio syndrome. It suggests that late effects of polio may largely be a result of aging.

There are a number of changes in the body's function that researchers in geriatrics have found to be associated with "normal" aging, as opposed to being a result of new diseases that may occur as you become older. One is a decline in motor nerve cell counts. It turns out that only a very small percentage of motor nerve cells are lost until people are well over 60 or 70 years of age. We don't know if nerve cells that survive acute polio have suffered a metabolic insult and may have a shortened life span. This has still not been ruled out. Decreasing cardiopulmonary capacity occurs steadily with age. As you become older, there is a steady decline of vital capacity and breathing ability, as well as some changes in your heart muscles' capacity. Collagen tissue (the connective tissue in your body that holds your joints and muscles and bones

together) becomes stiffer, again pretty much in a straight-line relationship with age.

Another issue is that chronic diseases occur statistically at a greater frequency as a population becomes older. These other conditions will have a great deal of influence on who develops new post-polio syndrome problems. Lastly, muscle disuse atrophy, as a result of general slowing down, often occurs in older people. It probably isn't healthy, but many older people simply lose interest in or ability to perform vigorous activity.

Exercise Studies

The next type of research I want to discuss concerns exercise studies. Dr. Agre in Wisconsin has done a great deal of exercise work in post-polios and has shown that post-polios who are weaker and show greater evidence for initial polio involvement are the ones who most commonly develop new symptoms (Agre & Rodriquez, 1991). Endurance time is the same in these weaker post-polios, even though total work capacity is less. He found that recovery of strength from exhaustion, however, was prolonged.

This finding adds support to the notion that interval training, or taking frequent short rest periods, and pacing are indeed good ideas. Perceived fatigue was the same in weak and strong post-polio subjects. This finding is important because some people said, "Post-polio people are just complaining more about getting tired easily." In fact, Dr. Agre showed that there was no research evidence to support this idea. He also did serial isometric strength testing in post-polios. A fairly large number of post-polio subjects, I think 30 or 40, were tested for maximum strength without exercise intervention over a period of three years. Also a control group of people without a history of polio were tested.

What is interesting to me is that the study was actually able to measure a statistical decline in the neighborhood of about ½% per year in the total maximum muscle force. It was a small decline, but the slope of the loss of strength was exactly the same in the people who were weak from their long-standing polio and in the group of people who never had polio. This suggests that a very small loss of muscle strength is perhaps going on with normal aging. However, this study was not able to show that the loss was more accelerated in polio survivors.

The results of many exercise studies indicate that some polio muscles certainly can increase in strength, some patients certainly can improve their fitness, and interval training and pacing are critical to the success of any exercise program. In other words, one could summarize the results of exercise studies by saying that you cannot make broad generalizations. Exercise is not always bad. Exercise can be helpful. Certainly stretching exercises are commonly helpful. Conditioning training to improve cardiopulmonary capacity is very helpful, if you can devise a way of doing it. If you have extensive paralysis, it may be difficult to do enough work to stimulate your heart to pump harder and therefore become more fit, or to breathe harder. However, strengthening exercise in some muscles, in the right patients, can be very helpful. In other patients, it is probably futile and a waste of time to try strengthening exercises. That is why it takes individual evaluation.

Stress

The next area of investigation about why some polio survivors become more disabled is the role of stress. In a number of studies a high correlation is shown between stress levels and symptoms of post-polio syndrome; that is, complaints of weakness, pain, and fatigue. Dr. Richard Bruno has hypothesized that polio survivors have a reduced capacity to secrete cortisone in response to stress (Bruno, Frick, & Cohen, 1991). He has suggested that this may relate to loss of cells in the area of the brain controlling the body's response to stress. Autopsy studies of people who died after acute polio do show reduced numbers of cells in the reticular formation of the brainstem. Exactly what these changes mean and how much they relate clinically to post-polio syndrome is not clear at this point. Dr. Bruno's theory certainly adds some theoretical support to the common wisdom that too much stress is not healthy, especially for polio

survivors, and that learning to control and reduce stress can be health enhancing.

I also need to remind everyone that experiencing progressive disability is in fact stressful. You must be very much aware of this issue when you are doing studies; for example, in the studies that Dr. Campbell and her colleagues have reported here. In order to make generalizable conclusions about polio or stroke survivors, it is important to be sure that the group of people you are studying in detail are not the atypical ones with many new difficulties, because they are more likely to come into a clinic or a support group, and may be more likely to volunteer for a study than post-polios whose health is good and whose functional abilities are stable. Post-polios coming to clinics are more likely to be distressed and looking for answers; therefore their problems at the time of being studied may be different. Figure 1 illustrates how new health problems and functional limitations in post-polios are related. Functional limitations can be locomotor, such as how we walk around, or they can involve how we take care of ourselves. Musculoskeletal pain problems may commonly be a result of chronic muscle weakness and unusual ways of performing functional activity. However, reduced cardiovascular fitness or general loss of health from heart disease, diabetes, or cancer (conditions known as co-morbidities) probably has nothing to do with the fact that you had polio. Nevertheless, if you are a polio survivor with long-standing weakness and you develop these conditions, you may well experience a great deal more new weakness or fatigue than other people without a history of polio would experience.

An unfortunate truism for polio survivors that has been learned from a number of studies about aging with a disability is that the functional abilities that required the greatest effort during rehabilitation, that took the longest, and that depended on the greatest personal determination to ultimately achieve and accomplish are the very same abilities that will be lost first as a person becomes older and weaker, whether from a condition unrelated to the polio or from the late effects of polio.

Loss of functional abilities impacts greatly on people's life style, including how they support themselves, what they do for themselves, and what they may need other people to do for them. If a person has a new functional loss, there are three generic choices for responding to it. First, you may give up the activity; for example, you may have to quit skiing if your legs become too weak. A lot of people do this when they get older. They give up tennis, although they may keep golfing until they're well into their 80s. Second, people can learn to do the lost functional activity differently. They may need to develop new skills to be able to still do it independently in a different way, or they may require new equipment to do it independently. This choice for responding is the typical kind of thing done through rehabilitation.

Third, but important, a person may choose to get help. This means getting someone else to do what you cannot do for yourself, or what you cannot do for yourself without risk of injury, or risk of wasting too much energy so you can't do other things you would really like to do more. It may take some patient explanation to family members or friends who are needed for providing the help in order for this to be possible. Everyone in rehabilitation believes that some governmental financial help should be made available for people with severe disabilities who could live independently from the standpoint of making decisions, if they could only have the extra hands to help them.

Epidemiologic Studies

The last area of research I would like to discuss is epidemiologic research. Population-based studies of polio survivors in Minnesota have shown that 24% to 40% are having new problems (Windebank, Litchy, Daube et al., 1991). My colleagues and I also did a large study on the late effects of polio among polio survivors in Michigan (Maynard et al., 1990). We found our sample also had very high rates of symptoms of weakness, fatigue, and pain. They were generally lower than what has been reported in the literature because most other studies have sought out people volunteering to participate in questionnaires. We used a more selected sample, although it still relied on a statewide polio survivors' group. We particularly chose for study people who were nonsymptomatic, in addition to those with symptoms, so that we could look for correlations between subject characteristics and new problems.

We used the concept of "period of physical best" in our interviews and questions. The period of physical best is the time in a person's life after acute polio and subsequent rehabilitation when they had the greatest physical functioning, such as walking endurance or strength for strenuous work. For people who acquired polio during childhood, this usually occurs in early adulthood, when normal growth and development is complete. We used this concept because it is very difficult retrospectively to know what is a new symptom. For example, a loss of walking ability may have been present in one person for 30 or 40 years, but it may be new in another person who never had previous walking difficulty. If a researcher did not examine the person 30 years ago and then also today, it is hard to be certain about what is new. Therefore, we let the post-polios define this time period for themselves.

Table 2 shows what we found in our 120 people. Fifty-six percent reported current difficulty with activities of daily living (ADL), up 25% from the time of physical best. Difficulty with intermediate ADL, such as getting around in the community for shopping, etc., rose 25% from the time of physical best, to a current total of 81% of our subjects. Certainly, people were becoming more disabled. As for the kinds of disabling conditions, hand and wrist problems were probably the most common. Our results support the notion that overuse of the upper limbs is particularly common in people who have long-standing weakness problems in their legs. Greater use of the arms and hands is an important compensation, but it does lead to shoulder and hand arthritis, nerve compression syndromes, and other wearing-out problems of the musculoskeletal system in the upper limbs.

If we looked for correlations as to which conditions were found on examination of post-polio subjects who had the greatest losses in functional independence, we again found upper limb problems common, as well as spinal pain, lower limb weakness, and gait deviations. Many of these new problems are related to the severity of the original polio, but the other strong association was with having secondary conditions, or co-morbidities. Obesity was strongly correlated with greater loss of functional abilities. Reduced exercise capacity was also strongly correlated. People who were less fit from a cardiopulmonary standpoint were in fact reporting greater functional decline. Elevated cholesterol ratios, interestingly, were also higher in those losing function. I'm not sure that the average cholesterol level for all of our subjects was higher than normal, but high levels were certainly prevalent, particularly in men. There was a strong correlation between decline and having any non-polio related condition, such as heart disease, emphysema, stroke, etc. Prevalences for anemia and hypertension were probably very close to what occurs in the non-polio population; perhaps they were even lower than in an age-matched able-bodied population.

Implications for Management

About 35% of the 120 people had at least one other co-morbid condition. Fifteen percent of the sample had elevated depression scores on the Brief Symptom Inventory. These scores were unassociated with the severity of polio weakness, but were associated with having other non-polio related medical conditions. They were also associated with some personality factors, including a pessimistic expectation for life in general and a resentful style of anger coping; that is, a tendency to direct anger in on oneself.

To conclude my talk, I want to address what we can do about these new problems. I like to emphasize the concept of re-rehabilitation, which is going through rehabilitation efforts and evaluation the second or third time when new problems develop. There is usually a great deal that can be done to improve remediable or correctable conditions as well as figure out how to best compensate for what may not be remediable. An important balance must be found between "use it or lose it" on the one side (the motto known well to polio survivors when they were young) and "conserve it to preserve it." The body has to be much more gingerly taken care of when you are older than when you are young and can better afford to abuse yourself because the body is more adaptable. This can mean that you may need an electric cart at the same time that you can still walk. I know a lot of people in this room who have learned this.

People in a declining phase of physical capacity need to adopt energy-saving techniques. They have to be

flexible and creative, to invent and figure out how to get around problems. This approach allows answers to be found. For example, exercise can be helpful, as I mentioned before. However, there are all types of exercises. For example, aquatic exercises are particularly helpful. Back care and joint protection flexibility exercises are often crucial to prevent pain and preserve function.

There are many community resources for health promotion, including physical therapy that can teach you how to do your own exercises; health clubs; information on nutrition. Many of these health and wellness resources need to be adapted specifically for people with long-standing motor disabilities. Also, there is the whole issue of mental attitude and life style enhancement. Support groups can be particularly helpful to address this in one way or another. Attitudes and outlook on life can have major consequences and can influence the immunological system.

Sunny Roller and I recently put a book together, which I have referenced (Maynard & Roller, 1992). It presents a modular program that is designed to establish an accessible and convenient community location for health promotion efforts of exercise, nutrition, and life style enhancement. It is designed to enable local people in your area to teach and facilitate activities because people can't travel a long way to attend health promotion activities. These are the goals of the program. It is crucial to find local facilitators with professional expertise to lead these programs. The manual basically provides a curriculum guide for them. The Stay Well manual is dedicated to preventing the unnecessary progression of disability and encouraging the participants to get well and stay well for years to come.

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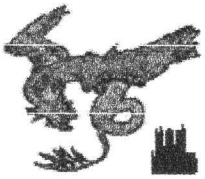

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