Occupational Therapy and the Postpolio Syndrome

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Approximately 75,000 polio survivors are experiencing new weakness, pain, and fatigue that are related to their initial disease. These problems affect their functional ability; therefore, they are of concern to occupational therapists. Overwork of a weakened neuromuscular system is believed to be the cause of these late symptoms. This article reviews current writings on the late effects of poliomyelitis. An understanding of the symptoms, causality, and psychosocial ramifications of this phenomenon facilitates effective occupational therapy intervention. Guidelines for occupational therapy assessment and treatment are included.

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A variety of new symptoms are now affecting at least 75,000 persons who recovered from poliomyelitis many years ago. *Caspar's (1985) article* was the first to make occupational therapists aware of this problem. Because these new symptoms are causing serious health and functional problems, it is likely that occupational therapists will increasingly be called on to evaluate and treat these persons. This article discusses the nature of the acute disease, symptoms of the late effects, and current theories on causality, and makes suggestions for treatments. Because the coping mechanisms learned during the initial disease have had such a profound effect on polio survivors, the importance of the psychosocial ramifications of the postpolio syndrome is stressed.

Description of Acute Poliomyelitis

Acute poliomyelitis (polio) is a viral disease that affects the anterior horn cells, or motor neurons, in the spinal cord. Some of the motor neurons are destroyed, some are not affected, and some are affected but recover with residual scarring *(Wiechers, 1985)*.

The clinical symptoms include flaccid paralysis and a loss of reflexes, with resulting atrophy in the affected muscles. Although the lower extremities are affected more often than the upper extremities, the paralysis may be asymmetrical and patchy in character and may result in complete or partial monoplegia, hemiplegia, paraplegia, or quadriplegia. Sensation is not affected *(Hopkins & Smith, 1978)*. A loss of motor neurons in the brain stem (bulbar polio) may lead to respiratory involvement. Bulbar polio was the major cause of death for early polio victims.

At the turn of the century, the disease earned the name *infantile paralysis* because it tended to strike young children. However, over the years there was a steady increase in the age of onset. A survey of 201 persons by *Halstead, Wiechers, and Rossi (1985)* showed that the median age of the respondents who acquired polio before 1949 was 8 years compared with a median age of 16 years for those affected in 1949 or later. Older victims manifested more severe symptoms than younger victims did. Sixteen percent of those who acquired polio before the age of 10 years required breathing equipment compared with 35.7% who were 10 years of age or older at the onset of the disease *(Halstead et al., 1985)*. *Weinstein, Shelekov, and Seltser (1952)* reported that, out of 259 patients hospitalized with poliomyelitis, monoplegia occurred more often in persons under 16 years of age than in adults, but quadriplegia occurred 2.5 to 3 times more often in adults than in children.

The Salk vaccine, which consists of inactivated, or killed, poliovirus, was introduced in the mid-1950s, followed in 1960 by the Sabin oral vaccine, which consists of live attenuated poliovirus *(Laurie, Maynard, Fischer, & Raymond, 1984)*. The annual number of reported polio cases in the United States dropped from a high of 56,000 in 1952 *(Codd & Kurland, 1985)* to an average of 12 since the vaccine was created. From 1982 through 1983, the 21 cases reported were all related to inoculation with the live vaccine, and 3 of those cases occurred in immune-deficient persons *(Fischer, 1985)*. The scourge of poliomyelitis appeared to have become a chapter of medical history.

Description of the Late Effects of Polio

Until recent years, poliomyelitis was considered to be a stable disease, although occasional reports of late-occurring muscle weakness and atrophy have appeared in the medical literature since 1875 *(Codd & Kurland, 1985)*. Recently, however, it has become clear that some polio survivors may experience a gradual decline in health and in functional ability decades after the acute attack.

The question of appropriate nomenclature has not been resolved. *Late effects of polio, postpolio sequelae, and progressive postpolio muscular atrophy* (PPMA) are among the terms used. The phrase *post-polio syndrome* is used in a general sense to describe those symptoms or conditions that are directly or indirectly related to the late effects of polio.

Researchers divide health problems related to previous poliomyelitis into two categories *(Codd & Kurland, 1985; Dalakas, Sever, et al., 1985)*. The first group includes symptoms that have a definable cause and that are primarily musculoskeletal rather than neurological in origin. These include joint, limb, or trunk deformities that can cause degenerative arthritis, nerve entrapment, pain, decreased stamina, frequent falls, unstable gait, or increased bracing needs. The second category, often referred to as PPMA, involves symptoms without a readily identifiable cause. These include slowly progressive muscular weakness both in previously weakened muscles and in muscles apparently spared during the acute attack. This weakness is often accompanied by muscle pain, fatigue, cold intolerance, or new difficulties in breathing *(Codd & Kurland, 1985; Dalakas, Sever, et al., 1985; Halstead et al., 1985; Owen, 1985)*.

Of all these new health problems, fatigue can be the most distressing because it imposes limits on activity

but its existence is not obvious to others. The fatigue, a systemic, generalized exhaustion, may be so severe and so out of proportion to the level of activity that such persons feel an overwhelming need to "just stop dead in their tracks" *(Raymond, 1986, p. 1398)*. Dr. Lauro Halstead, a polio survivor, describes a phenomenon called the *polio wall*, which is a sudden onset of symptoms such as intense fatigue, headache, weakness, hot and cold rashes, sweating, or a feeling like "hitting a wall" *(Halstead et al., 1985, p. 1398)*.

Concomitant with the development of new symptoms is an increase in difficulties with activities of daily living. Most frequently reported were difficulties with walking and climbing stairs and with transfers (e.g., wheelchair to bed). Other activities of daily living affected were homemaking, driving, bathing, dressing, eating/swallowing, and bladder/bowel function *(Brooke, Stolov, Shillam, & Kelly, 1987; Halstead et al., 1985; Kaufert, Syrotuik, Kaufert, & Gilbert, 1985)*.

Etiological Theories

Several theories attempt to explain the progression of disability from a disease that was once believed to be stable.

Amyotrophic Lateral Sclerosis

At one time, some researchers thought that the late effects of polio might actually be a form of amyotrophic lateral sclerosis (ALS), which bears a clinical similarity to the postpolio syndrome. However, ALS is characterized by new, rapidly progressive, generalized muscle weakness and by the presence of bulbar signs, respiratory difficulties, and upper motor neuron signs such as increased deep tendon reflexes, spasticity, and abnormal Babinski signs. Patients with ALS die within an average of 3 years after the onset of the disease *(Mulder, Rosenbaum, & Layton, 1972)*. The postpolio syndrome progresses slowly, and upper motor neuron signs are rarely present *(Dalakas, Elder, et al., 1986; Mulder et al., 1972)*. No evidence, either from clinical or experimental studies, suggests that the two conditions are related *(Laurie et al., 1984)*.

The Dormant Virus Theory

Although some researchers have demonstrated that the poliovirus can persist in humans under very specific circumstances, there is no evidence linking viral persistence with the late effects of polio *(Cashman, Siegel, & Antel, 1987; Codd & Kurland, 1985; Dalakas, Elder, et al., 1986; Johnson, 1984)*.

The Aging Process

Decreased muscle strength due to a loss of anterior horn cells is a normal part of the aging process. Since polio survivors already have a decreased number of anterior horn cells, some researchers believe that the natural aging process quickly depletes the remaining anterior horn cells and leads to progressive weakness *(Holman, 1986)*. However, *Tomlinson and Irving (1985)* have shown that there is no significant loss of motor neurons before the age of 60, yet many postpolio individuals experience serious changes at a much younger age. This lessens the credibility of the theory that aging alone can explain the late effects of polio.

Overwork Weakness

A motor unit consists of an anterior horn cell or motor neuron, its axon, and all of the individual muscle

fibers innervated by that motor neuron. When a motor neuron dies, Wallerian degeneration occurs in the axon, which results in the loss of trophic influence to the muscle fibers *(Wiechers, 1985)*. Motor units that are still functioning send axon sprouts to the orphaned muscle fibers, and a new neuromuscular junction is formed. This process, called *reinnervation by terminal axon sprouting*, may continue for 2 years or more after the acute infection *(Wiechers, 1985)*. If there are no functioning motor units nearby, the orphaned muscle fibers fibrillate and atrophy until they die *(Wiechers, 1985)*.

Poliomyelitis causes an overall loss of motor units; therefore, the remaining units must innervate many more muscle fibers than they did originally. A normal motor neuron drives 200 to 500 individual muscle fibers, whereas a reinnervated motor neuron may drive 1,000 to 5,000 or more individual muscle fibers *(Wiechers, 1985)*.

Increased muscle strength during recovery from paralytic poliomyelitis can result from (a) recovery of motor units that were initially affected but that regained function; (b) strengthening of the remaining functioning muscles by exercise; or (c) reinnervation by terminal axon sprouting of those muscle fibers that were orphaned *(Wiechers, 1985)*.

Evidence indicates that the anterior horn cells that were initially affected but later recovered do not recover normally. *Bodian's (1985)* research with rhesus monkeys enabled him to study changes in the spinal cord from a few days to 1 year after inoculation with the poliovirus. An examination of 40 separate limb regions in 11 animals sacrificed 2 to 5 days after the onset of paralysis showed that only 3% to 4% of the anterior horn cells appeared normal even when the extremities were clinically normal or only mildly paralyzed. However, at the end of 1 month, 90% of the surviving cells had regained a normal appearance, even though the vast majority had been affected.

This scarcity of normal-appearing motor neurons early in the paralytic process, even in cases with mild paralysis, supports evidence that many cells are invaded by the virus and later appear to recover. These become the parent motor neurons for those muscle fibers that were orphaned initially.

Some researchers postulate that the surviving motor neurons carry permanent scars from the infection and are unable to keep pace with the metabolic demands of innervating their overload of muscle fibers. This results in a dying back of the axons that have been overused *(Dalakas, Sever, et al., 1985; Wiechers, 1985)*. *Tomlinson and Irving (1985)* found that many of these surviving neurons are smaller than normal. Other researchers have found evidence of ongoing denervation in the muscles of polio survivors who, at the time of the investigation, showed no late symptoms *(Cashman, Maselli et al., 1987; Dalakas, Elder, et al., 1986; Martinez, Conde, & Ferrer, 1983)*.

In summary, most researchers believe that PPMA is caused primarily by the overwork of abnormal motor neurons that must innervate an excessively high number of muscle fibers. After many years of excessive metabolic demands, this results in the death of the terminal axon sprouts, leading to a significant and ongoing decrease in muscle strength.

Scope of the Problem

The number of polio survivors in the United States has been estimated at 254,000. According to the National Center for Health Statistics, polio is the Number 2 cause of paralysis in America, second only to stroke *(Speier, Owen, Knapp, & Canine, 1987)*. The percentage of people experiencing the late effects of poliomyelitis is difficult to determine. Those on whom many of the epidemiological studies have been based have been contacted through recently formed networks of polio survivor groups or rehabilitation centers. These studies may tend to overrepresent those groups with the highest levels of disability and

those experiencing the more severe postpolio sequelae because these people have the greatest amount of contact with rehabilitation facilities and support programs. Conversely, these studies may tend to underrepresent those who recovered so completely that they are no longer counted within the polio-disabled population *(Kaufert et al., 1985)*.

A study of polio patients treated at the Mayo Clinic between 1935 and 1955 showed that 22.4% of the 125 respondents had developed new symptoms related to their previous poliomyelitis *(Codd, Mulder, Kurland, Beard, & O'Fallon, 1985)*. A similar study located 670 polio patients who had been treated at the Sister Kenny Institute in Minneapolis, Minnesota, between July 1, 1952, and June 30, 1953. Of 327 respondents, 41% reported late symptoms *(Speier et al., 1987)*. Because these surveys studied patients treated at specific facilities within a particular time frame, they may be more heterogenous than the studies previously mentioned. However, a bias may still exist because those persons currently experiencing late sequelae might have a greater tendency to respond to questionnaires. In summary, it appears that 75,000 or more persons may be experiencing serious changes in health and function as a result of their previous poliomyelitis.

Adaptations of Persons Disabled by Polio

Polio survivors are their own best advocates. They designed many of the equipment adaptations that minimized their dependence on others *(Raymond, 1986)*. They catalyzed the movement for the rights of disabled people *(Laurie, 1982)*. Various studies have shown that a high percentage of polio survivors, including those with severe residual disabilities, have successfully coped with disability as measured by education, employment, and marriage *(Affeldt, West, Landauer, Wendland, & Arata, 1958; Bailey, 1985; Halstead et al., 1985)*.

During recovery from the acute attack, polio patients were pushed to exercise and to wean themselves away from equipment. *Mailhot (1980)* stated, "Persistence in strenuous activity was the great rehabilitation virtue and the pursuit of ambulation second only to the quest for the Holy Grail" (p. 7). Restoration of function was achieved by focusing on a goal rather than on the effort needed to reach that goal. Pain and fatigue were disregarded. The continued use of equipment represented the lack of a cure. Some patients used their canes, crutches, or braces only when they were not with their parents out of fear their parents would destroy the aids *(Kohl, 1987)*.

Now many polio survivors have had to start using ambulation aids, change from crutches to wheelchairs, or exchange manual chairs for motorized chairs. Others have had to resume or increase the use of respiratory equipment. This need to reduce activity levels or increase equipment use means more to these persons than just a change of life-style. It represents becoming disabled by the same disease for a second time *(Frick, 1985)*. Polio survivors traditionally have not afffiliated with each other as a disability group. That fact, coupled with the physicians' lack of knowledge, has left most people experiencing postpolio sequelae bereft of emotional or medical support and with a heightened sense of isolation. The combination of isolation and the loss of physical abilities thought to be permanent has made the psychological effects of the postpolio syndrome devastating *(Frick, 1985)*.

It is important to understand the coping mechanisms used during the acute phase of the disease in order to understand the current psychological stresses. Denial was useful when one expected complete recovery from an acute polio attack. If complete recovery did not occur, denial changed to minimization. This reduced patients' overwhelming fear about the future and allowed them to believe that life could still be meaningful and that normal activities were still possible. Overcompensation was another helpful mechanism used by polio survivors. It contributed to their assertiveness and brought them to the highest

possible level of functioning. The fear of not being as good as others was a challenge that pushed many polio survivors to become overachievers. However, although these coping strategies worked well in the early stages of the disease, they could be detrimental if used by persons suffering from the late effects of polio *(Carsey & Tepley, 1986)*.

According to *Carsey and Tepley (1986)*, social workers who are also polio survivors, there are benefits to seeing oneself as disabled. It allows one to be pleased with what one has accomplished. It allows one to join support groups that provide an environment in which to express the painful feelings that accompany physical loss. It allows one to shed the false pride that has prevented equipment use and thus gain extra energy to meet the demands of life.

Frick (1985) describes a process that polio survivors can use to adjust to a second period of disability. The first step, *mourning*, is appropriate no matter how often disability occurs in a person's life. During this period, the person may reject equipment because it symbolizes the loss of physical ability. Succeeding stages, which Frick calls *devaluing physique*, *enlarging the scope of values*, and *upholding asset evaluation*, help the person to minimize the importance of physical achievement and appearance while maximizing feelings of self-worth by participating in study, work, political, leisure, and other nonphysical activities. At the same time, the person learns to value achievements within the limits of his or her disability instead of judging activities by normal standards that are inappropriate.

Occupational Therapy Intervention

The following guidelines are based on my experiences as a clinician and a polio survivor as well as on an extensive review of the literature.

Evaluation

A comprehensive evaluation should start with an interview that clarifies the patient's present occupational role (e.g., student, worker, homemaker, volunteer, retiree). The person should be asked which activities produce pain, weakness, or fatigue; at what times of the day these symptoms occur; and how these problems interfere with present occupational roles. Equipment that the patient currently uses, such as splints, mobile arm supports, and adaptive aids, should be checked for fit and function.

If muscles in either upper extremity are weak, a manual muscle test should be performed on both upper extremities. An important factor to consider is that a polio-weakened muscle is much weaker than its clinical appearance indicates.

Perry (1985) found that polio muscles that appear normal (Grade 5) or good (Grade 4) clinically, as evaluated by manual muscle testing, may in fact have significant denervation and weakness when tested with electromyography. Patients with good or normal strength on a manual muscle test are expected to function in a normal manner. However, quantitative testing showed that Grade 5 in a polio muscle is actually only 75% as strong as Grade 5 in a nonpolio muscle, and Grade 4 in a polio muscle is only 40% as strong as Grade 4 in a nonpolio muscle. Therefore, a polio muscle that tests as 4/5 on a manual muscle test actually has to work 2.5 times as hard as a normal muscle to do the same job *(Perry, 1985)*. Using an advanced technique, called macroelectromyography, *Wiechers (1985)* also observed that muscles that were of fair, or antigravity, strength (Grade 3) actually had a very low number of individual motor units.

While performing manual muscle tests, the therapist should keep in mind that upper extremity strength varies markedly throughout the range of motion. Elbow flexors that are Grade 4 at 90° flexion can be

Grade 2 when the elbow is fully extended; shoulders may have greater strength at a lower elevation than when raised above 45° *(Perry, 1985)*. This may indicate that the person can perform functional activities most efficiently with the elbow flexed and the arm at the side.

Treatment

The role of exercise. Whether exercise is beneficial or detrimental in PPMA is controversial. A Swedish study reported improvement in the quadriceps muscles of 12 postpolio subjects after a 6-week high-intensity strengthening program *(Einarsson & Grimby, 1987)*. Wiechers, in a commentary on the study of Einarsson and Grimby, suggested the possibility that this study would have long-range negative effects (i.e., that hyperfunctioning of residual motor neurons might lead to decreased strength and cost the subjects additional ambulation time in the future) *(Einarsson and Grimby, p. 283)*.

Feldman and Soskolne (1987) initiated a 6-month program of rigidly controlled, nonfatiguing exercises for 32 muscles in six patients. Fourteen muscles showed improved strength, 17 maintained strength, and 1 lost strength. At the end of the strengthening program, patients were placed on a maintenance program and given strict instructions on life-style changes and the need to avoid fatigue and keep within the limits imposed by their disability.

Maynard (1985) reported that efforts to increase strength by controlled strengthening programs often aggravate the pain problems. His approach is to maintain strength by continuing functional activities such as self-care, ambulation, and work or recreational activities. He questions the role of strengthening exercises for a muscle already too weakened by a degenerative process to meet its normal requirements, and eschews even gentle strengthening exercises for a weakened muscle that is being used, perhaps exhaustively, on a regular basis in normal activities of daily living (e.g., the quadriceps in walking). *Herbison, Jaweed, and Ditunno (1985)* also supported the hypothesis that overwork to muscles that have a decreased number of motor units may contribute to damage.

If muscles have weakened from disuse (e.g., from immobilization following surgery or a fracture), a nonfatiguing trial strengthening program may be instituted by the occupational therapist. However, most researchers believe that muscles that are already being used extensively on a daily basis should not be stressed further. Because the long-range effects of exercise are unknown, any exercise program must be undertaken with extreme caution. Exercise should never be performed to the point of pain or muscle cramps *(Cashman, Maselli, et al., 1987)*. *Perry (1985)* cautioned patients who are on home exercise programs to stop exercising immediately if they develop persistent pain, muscle spasms, chronic fatigue, or further weakness.

The role of equipment and life-style modifications. Patients experiencing the late sequelae of poliomyelitis must be taught life-style changes that will help them avoid overusing their muscles. The focus of occupational therapy for this stage should be on evaluation of and retraining in activities of daily living, use of assistive devices, modifications of the work and home environments, and education in work simplification and energy conservation.

Occupational therapy intervention with the postpolio population may challenge some of our profession's long-held beliefs, such as the importance of independence in self-care activities. The patient's occupational roles need to be prioritized. If the role of worker is the most meaningful to the patient but getting ready for work each day causes fatigue that interferes with job performance, then independence in self-care is not a realistic goal. In his book about President Franklin D. Roosevelt, *Gallagher (1985)* wrote

> Roosevelt was quite capable of dressing himself. With ingenuity and practice the paraplegic

> learns how. Dressing, however, can never be as easy and unconscious a business as it is for the nonhandicapped. It can be done from a wheelchair, but it takes effort, thought, and time. Roosevelt, like any other polio or, indeed, like any other person, had limits to his energy and his strength.... Over the seven years of his rehabilitation, FDR had shown that he had fully as much motivation to be independent as any other polio [survivor]. ... Now, as President, faced with virtually unlimited demands upon his energy and interests, he chose not to waste his limited muscle power on things like dressing. (pp. 163-164)

The occupational therapist's creativity will be challenged to help the patient modify his or her life-style to enable the continuation of the most valued occupational roles. This challenge is heightened when a patient must start using orthoses or adaptive aids or finds that equipment that has functioned well for many years is no longer adequate due to progressive muscle weakening. For example, a person's upper extremities may have weakened to the extent that a mobile arm support no longer can be used for both feeding and written communication. If writing, typing, or computer use is a more valued activity than self-feeding, the mobile arm support should be adjusted to facilitate performance of the former activity, and the latter may be delegated to a caretaker.

The key to successful life-style modification is patient education. If a person does not understand his or her disorder, it is very difficult to implement the necessary interventions and strategies.

Conclusion

As many as 75,000 polio survivors in the United States are suffering from debilitating symptoms related to their original disease. Muscle weakening and atrophy, pain, and severe fatigue are some of the late sequelae that are decreasing functional levels. Because overwork weakness is thought to be the precipitating factor, strengthening activities may be precluded. Life-style changes incorporating assistive or orthotic devices and education in energy conservation and work simplification are the focus of treatment. Occupational therapy intervention can help these patients modify their life-styles so they can continue to perform those activities they find most valuable.

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