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Management of Postpolio Syndrome

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Recent research has shed light on the pathogenesis of the postpolio syndrome and has helped explain its symptoms and the rationale for management. The aim of this article is to familiarize physicians with this syndrome. The history, acute infection, definition, and diagnosis are discussed, as well as the various symptoms and their management. People with postpolio syndrome can educate health professionals about this condition and can help others inflicted with this syndrome. Thus far, no cure is available. A correct diagnosis is important, and the physician must realize that severe comorbidities tend to afflict people with this syndrome. Numerous management options are available to help these people enjoy a high quality of life.

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The term "postpolio syndrome" was first used by a group of people with a history of acute poliomyelitis who were trying to interest the medical profession in their symptom complex and understand it themselves.*[1]* The medical profession and patients with postpolio syndrome have since gained a

substantial understanding of this disorder; however, some physicians still do not recognize it as a medical condition, and previous victims of polio still need to obtain information on their new symptoms and understand the manner in which these symptoms are related to the previous active polio (see Appendix for a list of recent publications). In this review, the current understanding of postpolio syndrome and its management is summarized.

The first known description of postpolio syndrome was published in the French medical literature in 1875 and included a hypothesis for this condition.*[2]* Occasional reports continued to appear in the medical literature until the early 1980s. Some of these caused a considerable amount of fear among previous victims of polio, especially one report that indicated a link with amyotrophic lateral sclerosis.*[3]* The basis for the understanding of the syndrome expanded during the early 1980s with a petition from patients with postpolio syndrome to the National Institutes of Health;*[4]* the subsequent pioneering work of Dalakas,*[4]* Halstead,*[5]* and their colleagues; and the Palm Springs Conference. More recently, international conferences *[4,6]* have further expanded the understanding of this syndrome, and consumer conferences at national levels (Seventh International Post-Polio and Independent Living Conference, St. Louis, Missouri, May 1997) and local levels have helped consumers and medical professionals to understand the cause, evaluation, and management of postpolio syndrome. Patients with postpolio syndrome recently qualified for Social Security disability benefits.

ACUTE POLIOMYELITIS

Summer epidemics of acute poliomyelitis afflicted the Western industrial world during the 1940s and 1950s.*[6]* Recreational activities were discontinued, traffic was diverted from villages affected by the epidemics, and some families hid or denied cases.*[6]* Research to understand this condition was not initiated until a postpolio syndrome developed in victims of these epidemics and they sought medical help.*[4]*

With the development of an effective vaccine by Salk,*[7]* the subsequent use of orally administered attenuated vaccine, and the substantial effort by the World Health Organization, polio has been eradicated from the Americas. The last reported incidence of paralysis due to the poliovirus was identified in Peru in September 1991.*[8]* Although acute polio is endemic in many parts of the world, major strides toward eradication have been made by national poliomyelitis immunization days.*[9]* The goal of the World Health Organization is global eradication of poliomyelitis by the year 2000.*[9,10]*

Three types of poliomyelitis viruses cause the infection: enteroviruses 1, 2, and 3.*[11,12]* The infection is spread by contact with infected secretion from the gastrointestinal or respiratory tract. The virus first enters the gastrointestinal tract and then the bloodstream. The method in which the virus then enters the central nervous system was unknown for a long time, but recent research*[11,13]* demonstrated that the virus receptor is concentrated mostly in the motor end plate and from there ascends through the motor neuron nerves into the central nervous system, where it causes complete or partial damage to the nerve cell, mainly the motor neuron cell. Investigators have increasingly realized that involvement of the central nervous system is much more diffuse than simply the motor neurons*[14-17]* (Table 1).

Table 1. **Distribution of Pathologic Changes Within the Central Nervous System**

Spinal cord
Motor neurons of the anterior horn cells, especially lumbar and cervical
Neurons in the intermediate, intermediolateral, and posterior horns
Dorsal root ganglia
Cortical neurons
Precentral motor cortex
Hypothalamus
Thalamus
Cerebellum
Cerebellar roof nuclei and vermis
Brain stem
Nucleus ambiguus
Facial, hypoglossal, vestibular, and trigeminal nuclei
Reticular formation in medulla, pons, and midbrain

From Dalakas.[17] By permission.

Recognizing the criteria needed for the diagnosis of acute polio, as emphasized by Windebank and associates,*[18]* is important when patients are being enrolled in studies of postpolio syndrome. These criteria include, in addition to demonstrable weakness or paralysis, a history compatible with polio, fever, stiff neck or back, 10 to 500 leukocytes per milliliter of cerebrospinal fluid, and increased protein in cerebrospinal fluid. Patients who have three or more of these criteria, along with paralysis, are appropriate candidates.

The three variants of acute polio are the abortive type (mildest), the nonparalytic type, and the paralytic type (most severe). An unresolved issue is whether the postpolio syndrome can later develop in people who had the abortive or nonparalytic type of acute polio. Every year, full-blown paralytic polio develops in several children as a complication of orally administered attenuated vaccine. The Centers for Disease Control and Prevention is addressing the issue of which type of vaccination should be used--the killed virus or the attenuated.*[19,20]*

Pronounced recovery after initial acute poliomyelitis is not well recognized. Recovery depends on the percentage of motor neurons affected.*[1,17]* People who have had 50% of their motor neurons partially or completely damaged can have "full recovery." Recovery begins at 2 months and is continuous, but the decompensation of this balance of recovery and remodeling is the reason for the onset of postpolio syndrome.*[17]*

POSTPOLIO SYNDROME

The pathogenesis of the postpolio syndrome (best described by Dalakas*[17]*) consists of decompensation of a chronic denervation and reinnervation process to the extent that the remaining motor neuron can no longer maintain new sprouts, and thus denervation exceeds reinnervation (Fig. 1, 2, and 3) (see Pathogenesis). The initial symptoms are fatigue, weakness, pain, cold intolerance, muscle atrophy, and new problems associated with activities of daily living. These symptoms have been evaluated in four major studies.*[5,21-23]* (Table 2). The frequencies of the symptoms are as follows: fatigue, 62 to 89%; weakness in previously affected muscles, 54 to 87%, and in previously unaffected muscles, 33 to 77%; muscle pain, 39 to 86%; joint pain, 51 to 79%; cold intolerance, 29 to 56%; atrophy, 28 to 39%; new difficulties with walking, 52 to 85%; new problems with climbing stairs, 54 to 83%; and new difficulties with dressing, 16 to 62%. Of note, new problems with activities of daily living are associated with the

lower extremities to a much greater extent than with the upper extremities.

Diagnosis is by exclusion because no test is specific for postpolio syndrome. Halstead[25] summarized the criteria for the diagnosis of postpolio syndrome as follows: (1) history and findings on physical examination and electromyography compatible with previous poliomyelitis; (2) electromyographic findings that demonstrate anterior horn cell disease; (3) history of neurologic recovery with a definite period of stability; (4) gradual or overt onset of weakness and fatigue, muscle pain, joint pain, and decreased endurance and function; and (5) exclusion of other conditions that could cause the symptoms listed in criteria 4.

Table 2. **New Health Problems and Complaints Associated With Activities of Daily Living***

	Study			
Symptom	Halstead & Rossi[5] (N = 539)	Halstead & Rossi[21] (N = 132)	Agre et al[22] (N = 79)	Lønnberg[23] (N = 3,607)
New health complaints				
Fatigue	87	89	86	62
Weakness				
Previously affected muscles	87	69	80	54
Previously unaffected muscles	77	50	53	33
Muscle pain	80	71	86	39
Joint pain	79	71	77	51
Cold intolerance	...	29	56	42
Atrophy	...	28	39	...
New complaints associated with activities of daily living				
Walking	85	64	...	52
Climbing stairs	83	61	67	54
Dressing	62	17	16	17

*Expressed in percentages.
From Agre.[24] By permission.

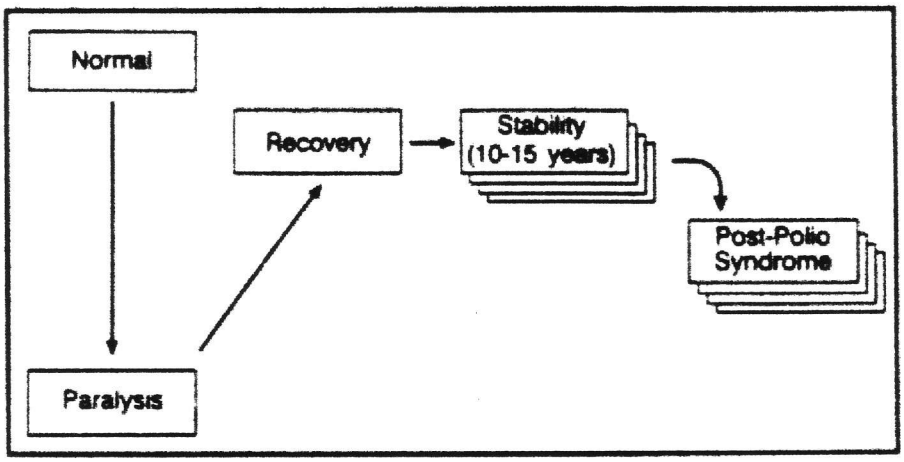

Fig. 1. Summary of clinical course of postpolio syndrome, beginning with acute paralysis and continuing through partial recovery, stability, and new weakness (postpolio syndrome). *Overlapping boxes* during periods of "stability" and "postpolio syndrome" indicate continuing subclinical instability of motor units despite clinically stable condition. Postpolio syndrome occurs when motor neurons in "stable" postpolio state can no longer maintain all distal axonal sprouts. (From Dalakas.*[1]* By permission.)

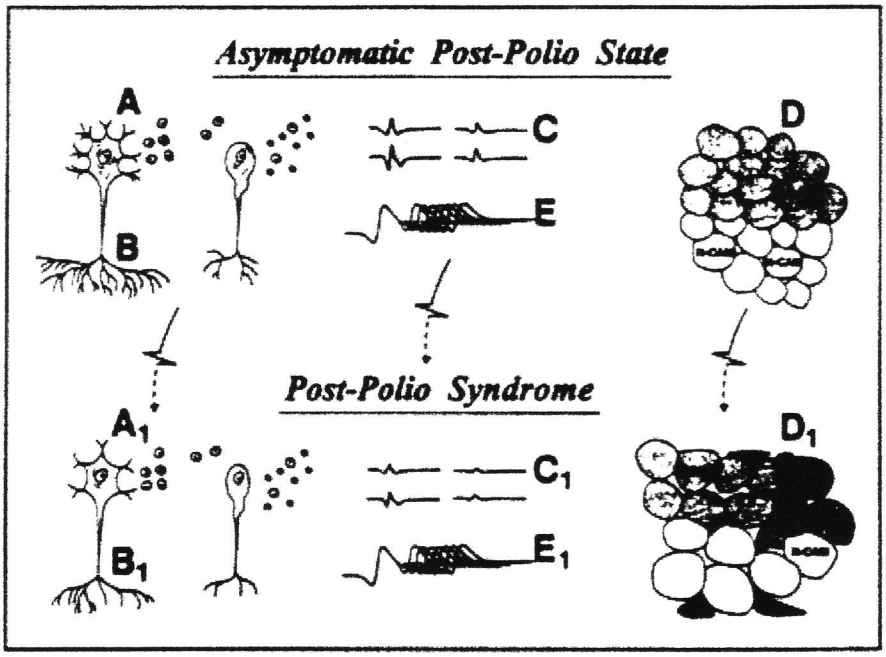

Fig. 2. Summary of mechanism of postpolio syndrome, from asymptomatic postpolio state to development of syndrome. Asymptomatic postpolio state is characterized by (A) mild inflammation of spinal cord motor neurons; (B) oversprouting of distal axons, resulting in effective reinnervation; and (C and D) enlarged motor units with high-amplitude macroelectromyographic potentials (C) and grouping of very large fibers histochemically (D). In addition, denervation continues with effective reinnervation, resulting in abnormal jitter (E), and muscle fibers are positive for neural cell adhesive molecules (N-CAM) and are normal in size (D). A1-E1, Postpolio syndrome occurs when remaining motor neurons can no longer maintain all distal sprouts and degree of denervation

exceeds that of reinnervation. Consequently, some distal nerve terminals are lost (B1), resulting in atrophy of individual muscle fibers (seen in biopsy specimen as small, angulated fibers (D1) and reduction in size of motor unit, represented by decrease in macroelectromyographic amplitude (C1). Many of the chronically denervated-reinnervated and often hypertrophic muscle fibers appear "moth-eaten " on enzyme histochemical staining because of impaired oxidative staining (D1) that corresponds to impaired oxidative metabolism on magnetic resonance spectroscopy. (From Dalakas,*[17]* By permission.)

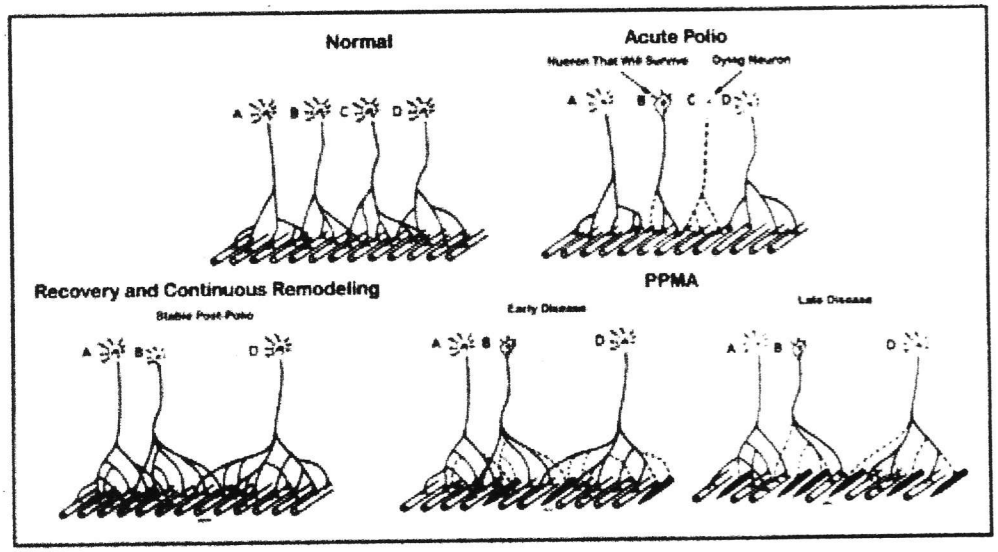

Fig. 3. Neurons remaining after acute polio and during chronic postpolio state. In stable postpolio, continuing remodeling of motor unit occurs by effective reinnervation of newly denervated fibers. In early postpolio muscular atrophy (PPMA), only small, scattered angulated fibers (dark) are present, representing disintegration of distal sprouts corresponding to early new muscle weakness. As PPMA progresses, more muscle fibers can become denervated, and hence there is possible development of atrophic fibers in small groups, represented as two contiguous dark fibers. *Dots* represent nuclear clumps remaining after acute polio attack. (From Dalakas.*[17]* By permission.)

The differential diagnosis includes conditions that can cause fatigue and weakness, such as multiple sclerosis, neuropathies, amyotrophic lateral sclerosis, other anterior horn cell diseases, anemia, myasthenia gravis, chronic infection, hypothyroidism, collagen disorders, depression, and side effects from various medications. If pain is present, osteoarthritis, bursitis, tendinitis, myalgias, polymyalgia rheumatica, fibromyalgia, neuropathies, and poliomyositis must be considered in the differential diagnosis. The onset of the postpolio syndrome occurs approximately 30 years after acute poliomyelitis; therefore, patients are often elderly and may have other medical concerns that can easily complicate the diagnosis.

Evaluation. Because the diagnosis of postpolio syndrome is by exclusion, a detailed history and thorough physical examination are essential. If the acute episode occurred during infancy, gestational and delivery histories are necessary. Some people with postpolio syndrome have no knowledge of previous polio because a sporadic acute case was not diagnosed or because the family denied that any member had active polio during epidemics. Records from the time of the acute infection may be difficult to obtain but are helpful. The physician should note whether other family members were afflicted, whether treatment

during the acute phase was administered at home or in a hospital, and whether the patient received subsequent rehabilitation. Of importance, the physician should also note the extent of recovery and whether braces , ambulatory aids, wheelchairs, or other assistive devices were used and whether they were later discarded. Patients often say that parents or medical professionals told them to discontinue using the devices despite severe functional difficulty. During elicitation of the history, the physician should establish the onset of the stability phase, inventory the patient's peak functional status, note the conclusion of the stabilization phase, and determine whether any exacerbating factors arose in conjunction with a change in occupations, psychosocial stressors, or other medical problems.

A thorough physical examination of the neuromusculoskeletal system is necessary. The evaluation may indicate a different medical condition or a concurrent medical condition--for example, sensory deficits, spasticity, or joint disease.

No laboratory tests are specific for the diagnosis of postpolio syndrome, but laboratory tests are necessary to exclude other medical conditions. These tests could include complete blood cell count, erythrocyte sedimentation rate, electrolytes, blood glucose, and liver, kidney, and thyroid function tests. An increase in the creatine kinase concentration in patients with postpolio syndrome has been found to be related to the intensity of immediately preceding activity.*[26]* Electromyographic and nerve conduction studies are indicated to confirm the sequelae of acute polio, extent of involvement, and activity of denervation or to exclude conditions such as myasthenia gravis and neuropathies. Five electrophysiologic phases have been recognized: acute myelitis (first month), early recovery (2 to 12 months), late recovery, functional stability, and late changes during functional stability.*[27]* With late changes, fasciculations, positive wave, fibrillations, and increased amplitude and duration of motor unit potential are evident. The late changes in postpolio syndrome are nonspecific and are related to the three types of motor units: normal motor units unaffected by polio, stable reinnervated units, and unstable reinnervated units. The changes are a reflection of the damage to the whole motor unit rather than an isolated abnormality of the neuromuscular transmission.*[27]*

Muscle biopsies, spinal fluid studies, immunologic studies, and viral assays do not provide specificity for the diagnosis of postpolio syndrome.*[4]*

Psychosocial assessment is important. Bruno and associates*[14]* demonstrated that clinically significant psychosocial factors and manifestations are related to the acute polio experience and the experience during recovery and stabilization. These findings point toward certain personality traits as well as depression, anxiety, and problems with concentration, memory, and thinking clearly--"attention deficit."*[28]*

Patients with postpolio syndrome tend to have a particular way of describing their symptoms and a particular reaction to assessment, or they put up "screens." Their behavior is, in many ways, similar to but also different from that in patients with chronic pain syndrome. The physician must remember the patient's psychosocial experience, which was often traumatic and occasionally contradictory to the individual's well-being. The physician should look beyond the screens that the patient puts up; otherwise, he may conclude that the patient has "only" a psychologic problem rather than a sequela of the postpolio syndrome. Polio victims frequently underwent extreme measures of physical therapy, surgical treatment, and other nonproven therapeutic interventions with no apparent benefits but were expected to show some satisfactory signs of "improvement." Typically, patients with postpolio syndrome report that they were told to discard ambulatory aids and other assisted devices and to perform in a "heroic fashion"*[6]* and that family, friends, and medical professionals had a perceived notion that this was the only acceptable means to show a positive attitude toward obtaining the desired result. The medical profession and patients with postpolio syndrome are increasingly realizing that the current rehabilitation philosophy of using available function without abuse and overuse is the correct management of this condition. Often, patients with postpolio syndrome report that they were told that their symptoms were in their head, probably a reflection of the issues previously mentioned and the screens that patients have learned to put

up to survive in this "normal" world. Therefore, the physician can expect dramatizations of symptoms, augmentation, and giveaway on a physical examination, but *these are part of the psychosocial picture and not the cause of the syndrome.[29]*

Special tests are indicated if a progressive decline in cardiovascular, pulmonary, or swallowing function is evident. If cognition problems or memory seems to be a major issue, neuropsychologic testing is indicated. Often, patients with postpolio syndrome are in the age-group that experiences cardiovascular disorders, and therefore a thorough cardiovascular evaluation is necessary, including analysis of blood lipids. A frequent conflict is between the principles of intensive cardiovascular rehabilitation and those of energy saving to avoid devastating fatigue.

The National Rehabilitation Hospital Limb Classification[30] (Table 3) and corresponding muscle classification[31] (Fig. 4) are helpful in the evaluation, research, and treatment of postpolio syndrome.

Table 3. **National Rehabilitation Hospital Postpolio Limb Classification**

Class	Description
I	No clinical polio
II	Subclinical polio
III	Clinically stable polio
IV	Clinically unstable polio
V	Severely atrophic polio

From Halstead and associates.[30] By permission.

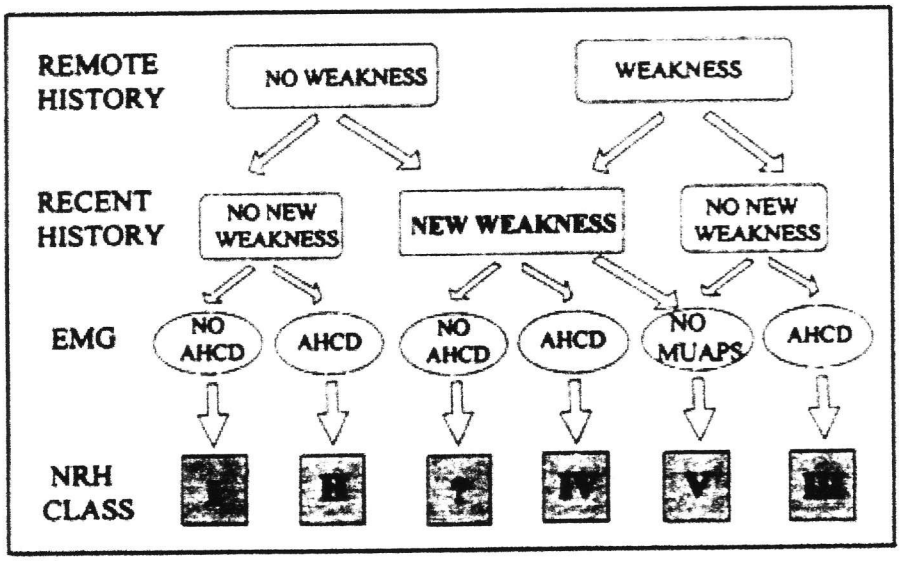

Fig. 4. National Rehabilitation Hospital (NRH) postpolio muscle classification. AHCD = anterior horn cell disease; EMG = electromyogram; MUAPS = motor unit action potentials. (From Gawne.[31] By permission.)

Pathogenesis. The exact pathogenesis of postpolio syndrome is still controversial, but the general opinion is that the mechanism is decompensation of the continuous process of denervation and reinnervation until the remaining motor neurons can no longer maintain new sprouts, and thus denervation exceeds reinnervation[17] (Fig. 1, 2, and 3). Concern persists, however, about the role of the poliovirus and immunologic responses.[32,33] The syndrome is the result of generalized neuronal disease, and neurons with various functions and at various sites are involved in the "scattered"

destruction of neurons[17] (Table 1). The neuronal population is variably involved (Fig. 2). There may be a normal neuron at a distance from the affected neurons, a normal neuron near an affected neuron and therefore stressed, a recovered neuron chronically stressed or moderately affected but surviving as a small neuron, or an incompletely recovered and "scarred" neuron.[17] Increased jitter and blocking are evident on single-fiber electromyography as well as neural cell adhesive molecules[17] (Fig. 3). Neuromuscular junction impairment or impairment of oxidative phosphorylase is debatable, but evidence shows that the energy mechanism of the muscle is impaired.[17,34] Loss or attrition of axonal sprouts occurs. The effects of normal aging must be considered because neuronal loss is evident after age 60 years. Evidence exists of some immune dysregulation in the spinal cord, muscle, and lymphocytes; of response to the poliovirus RNA by IgG interthecal synthesis; and of interthecal IgM poliovirus-specific antibodies and increased serum IgM poliovirus-specific antibodies;[35] however, the relationship of these findings to the development of postpolio syndrome is not completely clear.[33]

Although no definite risk factors exist for postpolio syndrome, evidence shows several associated factors.[17,36] Symptoms appear first in the weakest limbs, first in patients with the most severe residual paralysis and early bulbar-respiratory difficulty, and earlier in patients who were older when they had acute polio.

The onset of postpolio syndrome is usually 28.8 years after the acute episode. The reported range is 15 to 54 years, but probably any person who has had acute polio will eventually have postpolio syndrome.[17] Progression is slow, with a slight increase in muscle weakness, as demonstrated in a recent study.[37,38] The greater the postpolio residual effects, the greater the susceptibility for the syndrome because the neurons have much less reserve. When progression is discussed in postpolio syndrome support groups, all members fear that they will be equally affected, and this fear is understandable because, for some people with severe residual effects, their lives change from independence in daily activities to total dependence.[39]

The incidence of the development of postpolio syndrome in patients who have had acute polio varies considerably - from 22 to 68%.[18] The syndrome is estimated to occur in 28.5% of persons who have had paralytic polio. Patients with minimal residue have minimal changes, and patients with minimal neuronal reserve have a high incidence, especially 25 to 30 years (or earlier) after acute polio. The prevalence estimate has changed during the years since the first national survey and is now about 1.6 million.

Manifestations. **Fatigue.** Fatigue is a symptom in 62 to 89% of patients with postpolio syndrome. In the assessment and treatment of patients for fatigue (as in pain management), the placebo effect is about 43%.[40] The two types of fatigue are central and peripheral. The symptoms with central fatigue are difficulty in concentrating and remembering and somnolence. Many theories have been proposed for the pathogenesis of central fatigue, including chronic pain, type A personality, depression, dysfunctional reticular activation system , sleep disorders, and respiratory dysfunction.[16,28,41] Probably, more than one factor is present, and therefore a detailed assessment is important.

The symptoms of peripheral fatigue are decreased muscular endurance and muscle fatigability. The causes are metabolic exhaustion of the enlarged motor units, neuromuscular junction transmission defect, and, probably, overuse myopathy.[42,43] Electromyographic evidence of neuromuscular junction defect in patients with postpolio syndrome consists of a decrement on repetitive stimulation and increased jitter, but this defect is also seen in patients with myasthenia gravis, amyotrophic lateral sclerosis, and postpolio sequelae without the syndrome and therefore is nonspecific.[27,44]

The most important aspect of treatment of fatigue is educating the patient; thus, an appropriate evaluation of the patient's activities is necessary.[45,46] If the patient fails to adopt the basic management principles of fatigue - that is, energy saving, conservation, and pacing one's activities - other measures will not be helpful. Frequently, the patient lives by the doctrine of "not giving in," and multiple visits with the

physician and other health-care professionals are needed to convince the patient of the importance of changing activities; this doctrine may also be the reason the patient does not complete follow-up.*[47]*

Cashman and Trojan*[42,43]* evaluated pyridostigmine bromide (Mestinon) for the treatment of fatigue in postpolio syndrome. The results showed that systemic fatigue decreased from 20 to 88% in 53% of patients. Decreased muscle fatigability and increased activity were evident. On single-fiber electromyography, decreased jitter was evident in the responders but not in the nonresponders. The medication seemed to be most effective in those who were most fatigued, but little change was evident in isokinetic strength. In another study of pyridostigmine, static strength and dynamic power were significantly improved, and improvement was evident in dressing and walking.*[48]*

The recommended dosage of pyridostigmine is 30 mg daily, which is increased gradually to about 60 mg three times a day. Side effects are common,*[43]* including increased muscle twitching, nausea, loose stools, frequent urination, headaches, and facial flushing. Contraindications to the use of anticholinesterases are cardiac arrhythmias, bronchospasms (asthma), increased bronchial secretion, and urologic and intestinal obstruction. Another medication that has been used to control fatigue is amantadine hydrochloride. This drug is mainly aimed at facilitating arousal but has not proved to provide better results than placebo in patients with the postpolio syndrome.*[40]*

As previously noted, other factors may contribute to fatigue - for example, pain, muscular weakness, depression, and various medications. Interest in substances with anabolic or cellular reparative properties has increased substantially. One of these is serum insulin-like growth factor I, which was found to be low in patients with postpolio syndrome in initial studies, but more recent studies did not find this factor to be low*[49]* and therefore did not support its use in the treatment of postpolio syndrome. Other therapeutic agents that have been considered are deprenyl, dehydro-epiandrosterone, ciliary neurotropic factor, acetyl-L-carnitine, and L-carnitine, but their benefits in postpolio syndrome have not been proved.

Naps have proved to be beneficial in the management of postpolio syndrome fatigue.

Weakness. The second most common symptom mentioned by patients with postpolio syndrome is weakness - 54 to 87% of patients note it in previously affected muscles and 33 to 77% note it in previously unaffected muscles.*[24]* Windebank and colleagues*[18]* found no evidence that previously unaffected muscles later become weak; rather, they discovered that the patient was unaware or had not been told that the particular muscle had been affected during the acute episode. This evidence is supported by detailed initial muscular strength testing during acute polio, subsequent review of these records, and comparison with symptoms of currently weak muscles. Education on this issue is important because the patient may otherwise perceive this weakness to be a progression of the disease. Evidence of new muscle atrophy has been noted in 28 to 39% of cases.*[24]* Muscle twitching or fasciculation has also been reported and can be observed during physical examination.

The treatment of weakness consists of exercises (if indicated),*[50]* relief of associated pain, treatment of compression neuropathies, and education on overuse of muscles. Other causes of weakness that should be considered are weight gain, aging,*[34]* and recent illnesses leading to immobilization (bed rest decreases muscle strength by 25% in 2 weeks). Muscle strength in patients with postpolio syndrome may be rated as normal on manual muscle testing, but such patients have a deficit of up to 50% with quantitative measurement on isokinetic strength evaluation (Cybex).*[24]* The reason for overuse weakness may be metabolic fatigue or anatomic disruption caused by recent strenuous activity or excessive exertion.*[24]*

Reports on strengthening exercises are conflicting, but the key factor seems to be intensity. The principal strengthening exercises for patients with postpolio syndrome should be nonfatiguing. The exercise should be done every other day, and the perceived rate of exertion should be less than very hard. The loads should be held for only 4 or 5 seconds, and there should be a 10-second rest between bouts and a

5-minute rest between sets. The patient should do about three sets of 5 to 10 repetitions.[24,31,51-55]

The results of strengthening exercises have varied in postpolio syndrome and depend on the type of exercise program: 60% increase in isokinetic strength and 17% increase in citrate synthase, no decline in strength in 6 to 12 months, 8.4% increase in maximal torque, mean increase of 78% in 10 ranges of motion, and 5% increase in isometric strength.[34,52,56]

The effects of prednisone on strength have been studied. The justification for the use of prednisone was the possibility that an immunologic disorder causes the postpolio syndrome.[35] Only a modest increase in strength was noticeable after 3 months of high doses of prednisone, and the results were short-term.

Electrical stimulation can be used to strengthen weakened muscles or to reeducate muscles inactive because of disuse.[24] Electrical stimulation can also be used to decrease pain, as in myofascial pain syndrome and fibromyalgia. Electrical stimulation can facilitate the initiation of a strengthening exercise program and decrease disuse because of pain. The same principle applies with electrical stimulation exercises as with regular exercises - avoid fatiguing stimulation.

Finding the balance between intensity of exercise and avoidance of increased fatigue is important. Therefore, the exercise program must be realistic, with a definite selection of a limited number of specific muscles to improve function. People who maintain exercise programs built on these principles have a decrease in fatigue, an increase in strength, and a decrease in pain.[47]

Pain. Pain, the third most common symptom in postpolio syndrome, has been noted in the muscles in 39 to 86% of persons and in the joints in 51 to 79%.[24] The peripheral or muscle pain (myalgic pain) is perceived as a deep ache and may be a symptom of muscle disuse or overuse (as previously explained) or muscle dysfunction manifested by cramps.[1] Possibly, postpolio syndrome pain is a part of myofascial pain syndrome or fibromyalgia. Recent studies have found that fibromyalgia is extremely prevalent in patients with postpolio syndrome.[57]

Treatment of pain is usually combined with treatment of other postpolio syndrome symptoms - for example, fatigue and deconditioning. The treatment of pain, however, depends on the type of pain being managed. Antidepressants or similar medication used for myofascial pain problems should be considered. For myalgic pain, measures such as heat, either superficial or deep; electrical stimulation, either high-voltage galvanic stimulation or trigger-point injections; and stretching exercises may be indicated,[58] as well as biofeedback and muscle relaxation exercises. Of importance, posture and body mechanics should be evaluated, and the patient should be told how to reduce or avoid pain during activities of daily living, household tasks, and work.

For joint pain, heat should be used. Acetaminophen or nonsteroidal anti-inflammatory drugs can be of substantial help. Specific exercises to strengthen the muscles surrounding the arthritic joint can be beneficial. Bracing may be necessary to alleviate symptoms and prevent further acceleration of joint overuse.

Narcotic medications should be avoided but may be necessary if extensive trials of other measures fail.

Deconditioning and Obesity. Patients with increasing fatigue, pain, and weakness become progressively more inactive and gain weight.[34] The downhill course may, however, begin with progressive weight gain, and patients with postpolio syndrome in the United States are heavier than those in Sweden.[24] An estimated 60% of people with postpolio syndrome are overweight. With total bed rest, cardiovascular fitness decreases by 25% in a 3-week period. Therefore, prolonged bed rest for pain or any other medical reason may create a vicious cycle. Various aerobic exercise programs modified from the usual intense cardiovascular rehabilitation programs have been described.[24,31,53,59,60] Intensities have been stated to be less than 70% of maximal heart rate, with exercise bouts of 2 to 5 minutes and 1 minute of rest for a

total of 15 to 30 minutes per session. Other investigators have prescribed less intensive programs, with perceived exertion at a comfortable level and pain maintained near zero. The activity consists of bicycle ergometer, submaximal walking, mobility, strengthening and stretching exercises,*[58]* and treadmill walking. In all studies, the benefits have been positive, with increased work capacity of 80%, increased aerobic power of 15%, decreased energy cost of walking, increased walking duration, and improved efficiency of movement, but some studies have also reported no change in cardiorespiratory conditioning despite benefits. Weight reduction programs should be instituted because they can help in managing comorbidities such as diabetes, hyperlipidemia, hypertension, and cardiovascular disorders.

Gait Disturbance. New walking difficulties are reported in 52 to 85% of patients with postpolio syndrome and new stair climbing difficulties in 54 to 83%.*[24]* Commonly, gait disturbance occurs in patients who previously used ambulatory aids, such as a cane, crutches, or braces, and then discarded them. The cause of the gait disturbance is progressive weakness, pain, osteoarthritis, and joint instability resulting from weakened muscle, strain on ligaments, and malalignment. Although the obvious solution is to have the patient resume use of ambulatory aids, convincing the patient to do this is often difficult, if not almost impossible, because of the ingrained philosophy of "not giving in." The usual approach is to allow independent short-distance walking without further compromise of involved joints, especially the knees. Of note, the shoulders can easily be "worn out" if the patient is bearing considerable weight on them by using crutches or a cane. The most common bracing problem at the knee is genu recurvatum (back kneeing occurs to avoid collapse of the knee during standing because of weakened quadriceps). This problem can usually be solved by a free-moving knee joint with a stop that prevents recurvatum but allows enough extension to lock the knee. At the ankle, the problem is usually ankle instability and footdrop (or heel drop), usually solved by a plastic ankle-foot component with an ankle joint that allows dorsiflexion and therefore a smoother gait pattern but at the same time a plantar flexion stop to prevent footdrop.

Before a cane or crutches can be prescribed, the function of the shoulders must be evaluated, and any evidence of weakness or joint pain necessitates that the distance of walking be limited in an attempt to preserve the shoulders for other functions of mobility, such as transfers and independent activities of daily living. If the weakness of the shoulders is to the extent that further use would compromise these functions, an electrical wheelchair should be considered for mobility.

Usually, the most difficult suggestion for a new patient with postpolio syndrome is a wheelchair, but one is often necessary for those who had the least neuronal reserve after the initial acute polio episode and the least recovery. Often, a power-operated vehicle (for example, an electrical scooter) is necessary, with goals of conserving as much of the patient's energy as possible, avoiding fatigue from ambulation, and preserving the shoulders for other essential functions of daily living. Several options are available for transporting a motorized wheelchair by automobile or van, including attachments for platforms.

Activities of Daily Living. New problems with activities of daily living, such as dressing, were reported by 16 to 62% of patients with postpolio syndrome.*[24]* The most common problems are related to weakness around the shoulders, often with associated pain. Other impairments can involve elbow, wrist, or hand function. The shoulder derives its stability from muscles and ligaments, and severe weakness can cause traction pain to develop. Cuff slings that wrap around the arm and over the shoulder girdle can be of assistance. Patients who are able to use their hands for various activities, such as computer work and piano playing, may tire easily because of shoulder weakness. Detachable mobile forearm supports can be of substantial help in these situations. In the management of wrist and hand problems, the muscular and joint functions that are involved and in what combination determine the type of splint that may help (static or dynamic orthosis).

Patients may benefit considerably from an assessment by occupational therapists, who can provide suggestions on energy saving, joint protection, assisted devices, and modification of activities, as well as safety issues related to these activities.*[45]*

Neuropsychologic Concerns. Patients with postpolio syndrome have reported increased symptoms from emotional stress, a feeling of anxiety, and difficulty falling asleep because "my mind is racing." They have been found to be depressed, to have type A personality traits, and to be oversensitive to criticism.*[14,61]* Among questions raised are the following:*[62]* (1) Does polio affect cognition and mood? (2) Is depression causing tiredness? (3) Is a complaint of pain used to gain attention?

Windebank and associates*[18]* found no evidence of depression in their patients with postpolio syndrome. Tate and colleagues*[63]* reported that depressed patients with postpolio syndrome had more pain, poorer medical status, worse coping mechanisms, and less satisfaction. Women were found to have more somatic complaints and men to be more isolated, but these studies found no new weakness related to psychopathology.*[62]* Bruno and Frick*[64]* found that patients were more sensitive to criticism and failure and less compliant with therapy and, in a subsequent study, that emotional stress led to fatigue, 48% of patients reported anxiety, and 58.2% had difficulty falling asleep. Therefore, patients with postpolio syndrome have increased stress and anxiety symptoms, but no relationship has been found between psychopathology and physical disability. When evaluating cognition, Freidenberg and coworkers*[65]* found no abnormality, and Bruno and associates*[28]* found that 70% of patients had problems with concentration, memory, and thinking clearly because of "attention deficit." If neuropsychologic symptoms are interfering with the patient's function, the patient should be referred to a psychiatrist or psychologist. Treatment considerations are medication to improve sleep or to decrease anxiety or depression and psychologic counseling for cognitive therapy. Social workers can be of substantial assistance if family disharmony or other social stress exists.

Respiratory Problems. New breathing problems were reported by 42% of patients with postpolio syndrome,*[66]* and 87.5% of those who needed assistance during the acute phase needed assistance again in 3 to 59 years. Breathing problems are especially likely to occur in patients with residual respiratory muscle weakness. Other cofactors, which can contribute substantially, are emphysema, scoliosis, and cardiovascular problems. The respiratory characteristics of patients with postpolio syndrome and late onset of chronic ventilatory failure that necessitates assisted ventilation are (1) muscle weakness, (2) decreased pulmonary volume, (3) decreased maximal inspiratory pressure, (4) decreased maximal expiratory pressure, and (5) decreased air flow.*[66]*

These changes in respiratory function cause chronic microatelectasis, decreased pulmonary compliance, increased chest wall tightness, chronic alveolar hypoventilation, decreased cough and expiratory flow, and decreased clearing of secretions.*[66]* The management of respiratory sequelae in patients with postpolio syndrome includes early detection to prevent further compromise due to decreased pulmonary function. Therefore, early assessment and management by a thoracic disease specialist are indicated for appropriate treatment and regular follow-up. Early noninvasive treatment consists of intermittent positive pressure ventilation, assisted coughing, and blood gas monitoring, especially during the night to detect possible hypoxemia.*[66]* These patients should also be assessed by respiratory and physical therapists for instruction in assisted coughing and stretching of tight accessory respiratory muscles, posture principles, fitness, and muscle relaxation. If scoliosis is a contributing factor, the patient may benefit from a back brace (see subsequent discussion) and, if in a wheelchair, a seating orthosis. Referral to a dietitian is indicated if obesity is a concern.

Swallowing Problems. New symptoms of swallowing problems occur in patients with bulbar and nonbulbar postpolio syndrome.*[67]* Sonies and Dalakas*[67]* found that oral intake was not threatened and that aspiration was extremely infrequent. If aspiration does occur, especially in patients with progressive respiratory problems, it is a concern. Sonies and Dalakas found that tongue function, swallowing initiation, and oral stasis impaired transfer and that the hyoid bone was abnormally elevated. In the pharyngeal phase, there was evidence of unilateral bolus flow, pooling at the cricopharynx, and pharyngeal stasis. Problems during the esophageal phase of swallowing indicated gastric reflux and delayed mobility in the lower esophagus. Of interest, the swallowing studies seemed to be a more

sensitive test of decline in motor function than skeletal muscle testing.

Patients with postpolio syndrome and previous and new swallowing problems should be referred for evaluation of swallowing. Management consists of education in appropriate food preparation, positioning at meals, and enhancement of individual swallowing phases as indicated.*[67]*

Autonomic Nervous System Dysfunction. Intolerance to cold was reported by 29 to 56% of patients with postpolio syndrome.*[24]* The cause is unclear but is probably peripheral. The peripheral component could include muscular atrophy and therefore decreased heat production. Often, the vascular system in the affected limb is hypoplastic when muscular atrophy is severe; thus, circulation is not well developed. The hypoplastic vascular bed may suggest atherosclerotic peripheral vascular disease, an inaccurate diagnosis. Because of weakness, there may be stasis in the limbs from lack of action of the muscle pump. Usually, cold intolerance is managed by adding layers of clothes, and patients with postpolio syndrome generally wear warm socks in bed.

Scoliosis and Abdominal Weakness. Truncal muscle weakness involving the erector spine or the abdominal muscles may lead to the development of paralytic scoliosis during the recovery phase after acute polio. Fortunately, this condition was usually aggressively treated, but scoliosis may later progress with increased weakness as part of the postpolio syndrome. Scoliosis can be a cofactor in cardiopulmonary problems. If the patient is ambulatory, the abdominal muscle weakness should be managed by a back brace that provides abdominal support, and a cane or crutch should be used for further stability. Progressive paralytic scoliosis may necessitate evaluation by a surgeon specialized in scoliotic surgical procedures for consideration of fusion. If the scoliosis does not seem to be progressive and is not compromising other functions, bracing should be considered. For patients confined to wheelchairs, the same principles apply, but a seating orthosis in addition to the back brace further improves posture in the wheelchair.

Patients with postpolio syndrome who have scoliosis or abdominal muscle weakness should definitely see a physical therapist for instruction in back posture principles and body mechanics, back care, and safe and appropriate (avoiding overuse) mobility, including bed mobility, transfers, and gait, if indicated. Strengthening, stretching, and respiratory exercises may also be needed.

Insomnia. Bruno and associates*[14]* reported that 69% of persons with postpolio syndrome who had fatigue frequently had difficulty falling asleep because of "mind-racing." This problem seems to be related to stress and anxiety, but patients with postpolio syndrome may have other severe comorbidities (as previously mentioned), including fibromyalgia syndrome,*[57]* which is associated with a high frequency of insomnia and poor quality of sleep. Patients with chronic pain (as noted previously) may also have pronounced insomnia because of the pain.

People with postpolio syndrome who have bulbar muscle weakness, respiratory compromise, or obesity may have sleep apnea.*[66]* Patients with severe depression may have insomnia. Therefore, patients with insomnia and postpolio syndrome should be referred to sleep disorder centers for accurate evaluation and appropriate management, and they should receive assistance from other specialties as indicated (pulmonary medicine, psychiatry, and dietetics).

CONCLUSION

The understanding of acute poliomyelitis and postpolio syndrome has increased substantially during the past 1½ decades. An estimated 1.6 million people in the United States are survivors of polio. Although we are on the verge of eradicating acute polio, it still exists, and travel is increasing; thus everyone should be vaccinated. The protocol of vaccination is still unresolved because several cases of polio occur each year after use of attenuated vaccine. The pathophysiology of postpolio syndrome is usually thought to be decompensation of the balance between denervation and reinnervation after a generalized neuronal

disease; the remaining motor neurons can no longer maintain new sprouts, and denervation exceeds reinnervation. The exact role that the poliomyelitis virus and immunologic processes have in the postpolio syndrome is unresolved. The postpolio syndrome is a set of symptoms, including fatigue, new weakness, and pain. It is also frequently associated with deconditioning, obesity, gait disturbance, disturbance of activities of daily living, neuropsychologic concerns, respiratory problems, swallowing problems, autonomic dysfunction, scoliosis, and insomnia. Thus, patients with the syndrome must undergo extensive detailed assessment, thorough examination, and, frequently, extensive investigation. Multiple secondary or comorbid medical factors can contribute considerably to symptom development or aggravation. No specific test exists for postpolio syndrome. Management demands a team approach: physiatrists, pulmonologists, psychiatrists, psychologists, orthopedists, cardiovascular specialists, physical therapists, occupational therapists, speech pathologists, speech therapists, medical social workers, dietitians, and orthotists. The extensive patient education that is needed from these medical professionals can be conducted one-on-one, but participation in educational programs with postpolio syndrome support groups has also proved to be essential. The postpolio syndrome support groups function as peer visitors by helping new patients comply with suggestions from medical professionals and by describing their experiences as polio victims and what they have done in order to maximize their quality of life.

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APPENDIX

Dalakas MC, Bartfeld H, Kurland LT, editors. The post-polio syndrome: advances in the pathogenesis and treatment. Ann N Y Acad Sci 1995;753:1-409

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