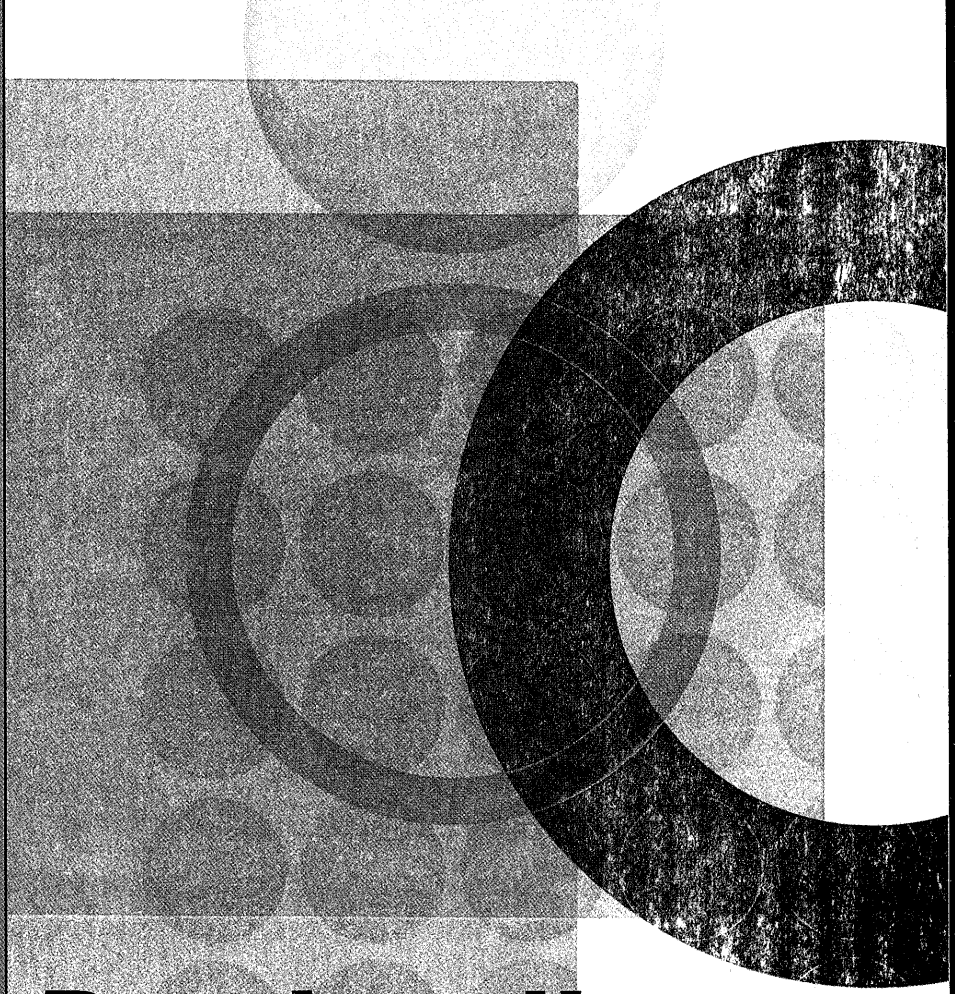

Postpolio Syndrome

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CHAPTER 5

Joint and Muscle Pain

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Postpolio syndrome (PPS) was initially defined as the clinical syndrome of new weakness, pain, and fatigue in patients who have recovered from acute polio.[12] More recently, new criteria for PPS have been developed, but fatigue and new weakness are essential elements.[13] Table 1 lists the most common symptoms recorded from a number of studies.[4,5,14,15,21] Muscle and joint pain are two of the most prevalent symptoms seen in PPS. Studies have found that the prevalence of muscle pain ranges from 38%[21] to 79%[15] and the prevalence joint pain ranges from 42%[21] to 80%.[15] In both these reports, either joint pain[21] or muscle pain[15] was the most prevalent complaint found in participants.

A correlation between muscle pain and weakness is believed to be either a measure of overuse or disuse that falls into a vicious cycle (Fig. 1). When musculoskeletal overuse occurs, pain develops. Rest and immobilization can relieve this pain, but this leads to decreased use of certain muscles, with development of disuse atrophy and further weakness. After this, relatively normal use of the muscle leads to pain and further disuse. Sometimes pain and subsequent weakness do not occur with activity. The fact that recent physical activity was not always associated with PPS may indicate that the intensity of the activity that is performed is more important. Frequent periods of activity with alternating periods of work and rest resulted in less evidence of local muscle fatigue, increased capacity to perform work, and increased ability to recover strength after activity in symptomatic postpolio patients,[1] Therefore, treatment of polio-related pain includes gentle paced exercises, preferably in water, bracing of unstable joints and limbs, and resting of muscles that are being overused.

Therefore, these authors suggest a causal relationship between symptoms of new weakness and musculoskeletal pain syndromes. This chapter covers research studies that offer various perspectives and varying support for this hypothesis. Possible causes for pain in polio survivors are examined in depth. The chapter also, reviews specific diagnoses for pain in polio survivors and examines outcome studies of useful treatments for specific pain syndromes.

Table 1. COMPARISON OF MOST COMMON NEW SYMPTOMS IN SUBJECTS WITH A HISTORY OF PARALYTIC POLIO REPORTED IN SIX STUDIES

SYMPTOM	CODD ET AL.[5] (*N* = 28)	HALSTEAD AND ROSSI[14] (*N* = 132)	CHETWYND AND HOGAN[4] (*N* = 694)	AGRE ET AL.[2] (*N* = 79)	RAMLOW[21] (*N* = 474)	HALSTEAD AND ROSSI[15] (*N* = 539)
Fatigue,%	59	89*	48	86	34	87*
Joint pain,%	74*	71	60*	77	42*	80
Muscle pain,%	48	71	52	86*	38	79
New weakness,%	71	N/A	47	69	38	N/A
In affected muscle,%	66	69	N/A	80	N/A	87
In unaffected muscle,%	15	50	N/A	53	N/A	77
Cold sensitivity,%	46	29	N/A	N/A	26	N/A

*Most frequent symptom.
Adapted from Gawne AC, Halstead LS: Post-polio syndrome: Pathophysiology and clinical management in CRC. Clin Rev Phys Med Rehabil 7:147–188, 1995.

CAUSE OF PAIN

The clinical finding of pain is correlated with the defining PPS symptom of new weakness. Trojan and colleagues used a case control study in order to determine which factors predispose polio survivors to develop PPS.[27] They evaluated weight gain; joint pain; muscle pain, either at rest or with exercise; degenerative joint disease (DJD); degenerative disc disease; age at presentation of acute polio; and degree of weakness initially, after recovery, and at presentation. Risk factors for the development of new weakness were determined by using an odds ratio. Factors predictive for PPS included variables consistent with chronic overuse such as length of time since polio (odds ratio [OR] = 1.6), recent weight gain, (OR = 6.4) muscle pain occurring with exercise (OR = 5.0), and joint pain (OR = 2.3). In addition, whereas the initial amount of weakness and present age were also correlated to PPS, gender and age of polio onset were not. The recent amount of physical activity and extent of recovery after acute polio were not correlated with the development of new weakness.

Maynard et al. reported a correlational analysis on secondary conditions associated with decreasing functional abilities among 120 postpolio subjects who participated in a clinical study, noting the prevalence of secondary conditions associated with PPS.[16] Significant correlations were found between new functional limitations and the following individual characteristics: (1) diagnosis of non–polio-related comorbidities, (2) reduced cardiovascular fitness, (3) obesity, (4) and elevated ratio of cholesterol to high-density lipoprotein level. Musculoskeletal problems of the upper and lower limbs were also notably prevalent (Table 2). Lower extremity weakness with gait de-

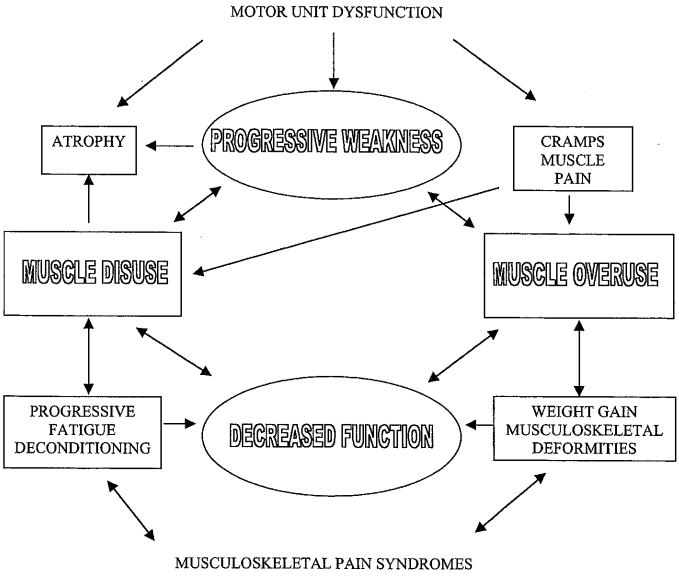

Figure 1. Vicious cycle of overuse or disuse leading to weakness and pain.

viations were significantly ($P < 0.05$) correlated with the presence of new functional limitations associated with declining functional capacity.

Gawne and colleagues studied the prevalence of postpolio muscle pain (PPMP) in 150 polio survivors using a pain questionnaire.[10] Participants answered questions regarding the intensity of the pain, temporal pattern, and relieving and exacerbating factors, and they completed the McGill Pain Questionnaire. Eighty percent of par-

Table 2. PREVALENCE OF SECONDARY MUSCULOSKELETAL PROBLEMS AMONG 120 POLIO SURVIVORS

SECONDARY CONDITION	PATIENTS, %
Sensory loss in the hand	79
Median neuropathy at the wrist	58
Carpal tunnel syndrome (symptomatic)	24
Ulnar neuropathy at the wrist	31
Hand or wrist arthritis (radiograph)	48
Hand weakness	56
Impaired hand dexterity	52
Upper limb joint pain	56
Lower limb joint pain	49
Spinal pain	50
Gait abnormality	59

Adapted from Maynard FM: Managing the late effects of polio from a life-course perspective. Ann NY Acad Sci 753:354–360, 1995.

ticipants complained of pain. Of these, 64 patients (44%) met the criteria for PPMP. There were 23 men (36%) and 41 women (64%) with PPMP and 36 men (45%) and 44 women (55%) without PPMP. There were no significant differences between the groups with or without pain with respect to age or gender. These patients were significantly ($P < .0001$) more likely to have new weakness (90.2%) as opposed to the group with no pain (48.3%). Both polio groups with and without PPMP were statistically similar to each other with respect to exacerbating and relieving factors (Tables 3 and 4) and on the remaining responses on the McGill Pain Questionnaire.

The strongest evidence favoring musculoskeletal pain and a causative relationship to PPS is the prospective population based cohort study of polio survivors reported by Windebank et al.[35] Repeated detailed quantitative and electrophysiologic studies were done at 5-year intervals 30–55 years after patients experienced acute polio. These patients demonstrated no new weakness, but 60% of them were symptomatic. Among 20 of the 30 symptomatic patients, 16 had a musculoskeletal pain problem believed to explain their symptoms. Other medical conditions such as diabetes, obesity, alcoholism, and depression likely explained new weakness in 4 symptomatic patients. All symptomatic subjects had stable or improving neuromuscular function. Chronic overuse of muscles or muscles acting in a compensatory manner were suggested as predisposing factors for pain.

Smith et al. demonstrated further support for chronic musculoskeletal overuse in a study of 111 consecutive polio patients.[24] Gait deviations were seen in 100% of all patients. These included 53% who had an uneven pelvic base and 43% with major trunk oscillations. While standing, 69% of these patients had an absent lumbar curve and 38% of them were weight bearing on their stronger leg. Pain was the most prevalent complaint in this group of patients, occurring in 85% of those who walked unaided and 100% of those who used an assistive device or wheelchair.

Chronic overuse of weak muscles or weak muscles acting in a compensatory fashion was demonstrated by Perry et al., who evaluated the dynamic electromyography during gait analysis on 21 polio survivors with strength in the quadriceps greater than or equal to 4/5 and strength in the soleus of 0–3/5.[20] All patients had symptoms of PPS including fatigue, pain, and new weakness in the thigh or calf. They studied the gluteus maximus, long head of the biceps femoris, quadriceps, and soleus muscles. Overuse during walking (a total effort of greater than two standard deviations more than normal) was found in all four muscle groups. It was most common in the quadriceps followed by the gluteus maximus, and it was least common in the weaker soleus muscles. In addition to overuse, gait variations (e.g., genu recurvatum or flexion of the knee during the gait cycle) and equinus contractures were observed.

Additionally, joints of the upper limbs, especially the wrist and shoulder, are prone to DJD when assuming a weight-bearing role. This commonly occurs with the use of assistive devices, such as wheelchairs, crutches, and canes. Werner et al. studied 61 polio survivors ages 49 $\pm$ 6 years with a mean duration of disability of 35 $\pm$ 4 years.[33] They obtained radiographs of both hands and wrists in addition to a physical examination.[33] The prevalence of severe to moderate DJD of the hand or wrist was 13%, and mild DJD was present in 68% of patients. Risk factors associated with osteoarthritis

Table 3. FACTORS THAT RELIEVE PAIN

GROUP	MASSAGE	BRACE	SLEEP	WEIGHT LOSS	STRETCH	HEAT	ICE	REST	EXERCISE	INJECTION
PPMP,%	47	23	47	33	36	78	1	90	1	1
No PPMP,%	50	16	34	16	27	58	6	84	6	11*

* Significant $P < .001$

Table 4. FACTORS THAT EXACERBATE PAIN

GROUP	COLD	REST	EXERCISE	WEIGHT	STRESS	ACTIVITY
PPMP,%	68	3	61	39	53	78
No PPMP,%	56	10	51	32	46	78

Significant $P < .001$.

included advanced age, lower limb weakness, use of an assistive device, and severity of the disability.

Nerve compression syndromes, including carpal tunnel syndrome (CTS), ulnar mononeuropathy at the wrist or elbow, brachial plexopathy, and cervical or lumbosacral radiculopathy, are syndromes that can cause pain as well as neurologic deficits in postpolio individuals.[32] These neuropathies can be detected on electromyogram (EMG) and nerve conduction studies (NCS). These tests can also detect subclinical neuropathies (i.e., before the individual has the characteristic symptoms of CTS). To assess the prevalence of these conditions, Gawne et al. conducted a retrospective study of 100 consecutive patients seen in a postpolio clinic.[9] EMG and NCS, including routine studies of the median nerve, were performed on all patients. A total of 49% of the patients had an abnormal peripheral nerve study result. CTS, either alone or in conjunction with an ulnar nerve neuropathy, was the most prevalent finding in a total of 38% of all patients. Ulnar neuropathy at the wrist was seen in 22% of patients, and CTS and ulnar neuropathy together were found in 3% of patients. A peripheral neuropathy was seen in 3% and either cervical or lumbosacral radiculopathy was present in 4% of all patients (Table 5).

In a similar study, Werner et al. looked at risk factors for the development of CTS in a retrospective study of 148 consecutive postpolio patients.[32] Electrodiagnostic tests were done only on the patients who had classic signs and symptoms consistent with CTS such as nighttime numbness or abnormal sensory findings in the median nerve distribution, or a positive Tinel's or Phalen's sign. Of the 148 subjects, only 50% underwent sensory NCS. CTS was found in 33 patients (22%). There was no difference in age, gender, work status, or duration of disability in those with or without CTS. Risk factors for the development of CTS included use of an assistive device (cane, crutch, or wheelchair), with use of more than one device increasing the risk even further.

Muscle pain may be a sign of overuse, causing breakdown products such as creatinine kinase (CK) to accumulate. Waring et al. found elevated CK levels in PPS patients;[30] however, other studies failed to demonstrate this relationship. Nelson found that the incidence of an elevated CK level in a group of polio patients with delayed

Table 5. ELECTRODIAGNOSTIC FINDINGS IN 100 CONSECUTIVE POSTPOLIO PATIENTS

FINDING	PATIENTS, *N*	PATIENTS,%
Carpal tunnel syndrome (CTS)	35	35
Ulnar neuropathy at the wrist	2	2
CTS and ulnar neuropathy	3	3
Peripheral neuropathy	3	3
Brachial plexopathy	1	1
Tibial neuropathy	1	1
Radiculopathy	4	4
Total	49	49

weakness (15 of 29) did not differ from polio patients without delayed weakness.[16] Trojan et al. also failed to demonstrate a relationship between muscle pain and elevated CK levels.[26] Further study is required in order to demonstrate a correlation between new weakness, pain, and measurable laboratory values of CK.

In another study by Willen and Grimby, 32 consecutive postpolio subjects were evaluated for pain.[36] Twenty-two patients fit the criteria for PPS, and 10 did not. Pain was assessed using a visual analog scale (VAS) on a scale of 0 (no pain) to 100 (worst possible pain). Of these individuals, 29 (91%) reported pain from muscles used for ambulation, 11 had joint pain at rest, and 12 reported muscle pain at rest. Pain symptoms were not related to whether the patient had PPS (new weakness) or to the presence of weakness in that limb; rather, they were correlated to the level of activity. The examiners found that the weaker patients experienced less pain. This study suggests that the more active an individual is, the more pain he or she experiences, as measured by the VAS. It also demonstrates that characteristics of the pain varied by site, with polio survivors describing a cramping, aching pain in the lower limbs and an aching pain in the trunk and non–polio-affected upper limbs.

In another study of 875 patients seen at a community postpolio clinic, Yarnell reported that pain was the most common complaint, occurring in 79% (693) of those studied.[39] Of the participants who complained of pain, 99% complained of pain associated with scoliosis. Yarnell proposed that scoliosis together with gait deviations could lead to DJD (44%), facet arthropathy (32%), mechanical low back pain (20%), spinal stenosis (10%), radiculopathy (5%), or sacroiliac joint dysfunction (4%) (participants could choose more than one option, so the numbers add up to 100%). In addition, causes of articular pain included DJD of the shoulder (19%) knee (16%), hip, ankle, and wrist. Nonarticular causes of pain included tendonitis, bursitis (28%), CTS (19%), myofascial pain (13%), tension headaches (11%), ulnar compression neuropathy (25%), and lateral femoral cutaneous neuropathy (25%). Wear and tear of the peripheral joints, especially the knees and shoulders, was a frequent cause of pain. Biomechanically, the shoulders are not suited to weight bearing, yet many polio survivors use their shoulders to walk with crutches, transfer, and push wheelchairs. Biomechanical abuse of unbraced knees, especially in those with genu recurvatum or valgus, commonly results in significant abnormalities, especially degenerative joint changes and pain.

Trojan et al. used a cross-sectional design with 126 polio survivors to identify predictive factors and correlates of muscle and joint pain.[28] They found that muscle pain was correlated with female gender, longer duration of general fatigue, and poorer physical and mental health. Predictive factors for joint pain were female gender, older age, higher body mass index (obesity), longer duration of stability after acute polio, greater weakness after acute polio, weaker lower limbs at the time of examination, and poorer general and mental health. Again, greater weakness, especially in the lower limbs, appears to be correlated with greater joint pain. The finding of a significant female prevalence has not been found in other studies, but both of these studies had a nonsignificant prevalence of women.[10,36]

In 1989, Waring et al. did a retrospective study of 104 ambulatory postpolio patients (43 men and 61 women) seen at the University of Michigan with clinical and EMG evidence of previous polio and the ability to ambulate.[31] Of these patients, 77% reported pain as a symptom. Associated medical conditions included 26 participants with genu recurvatum, 26 with DJD of the knee and 10 with DJD in other locations, 19 with CTS, and seven with a history of a knee injury. The average walking distance had decreased considerably at the time of the evaluation compared with the time of their initial recovery from polio.

Although all these studies demonstrate a significant relationship between musculoskeletal pain and PPS (i.e., the presence of new weakness and declining function), causation is not established because either problem could lead to the other through the vicious cycle described previously. However, these studies do show that pain and, in particular, PPMP and joint pain are common problems in the postpolio population

DIAGNOSIS

Because PPS is a diagnosis of exclusion, nonmusculoskeletal and neurologic pain syndromes must first be ruled out. Imaging studies and electrodiagnostic studies are usually necessary. For example, thyroid disease, heart disease, and rheumatologic syndromes must often be excluded. Frequently, studies demonstrate one or more comorbidity.[8]

In order to facilitate diagnoses and treatment of pain in polio survivors, a pain classification has been proposed by Gawne and colleagues.[11] **Type I** pain, or PPMP, occurs only in muscles affected by polio. It is described as a superficial aching pain that many patients say is similar to muscle pain they experienced during acute polio. It is characterized by muscle cramps, fasiculations, or a crawling sensation. It typically occurs at the end of the day or at night when the patient tries to relax. Physical activity, stress, and cold temperatures exacerbate the pain.

Type II, or overuse, pain includes injuries to soft tissue, muscles, tendons, bursa, and ligaments. Common examples are rotator cuff tendinitis; subacromial and greater trochanteric bursitis; and myofascial pain, especially in the upper and lower back. Myofascial pain in postpolio patients is similar to myofascial pain in other patients and is characterized by bands of taut muscles and discrete trigger points that elicit a jump response when palpated. These occur because of poor posture or improper body mechanics.[6]

Type III, or biomechanical, pain presents as DJD, low back pain, or pain from nerve compression syndromes. Weakness induced by polio-affected muscles and by poor body mechanics makes the joints, especially of the lower limbs, more susceptible to the development of DJD. In addition, years of ambulating on unstable joints and supporting tissue increase the stress and the energy expenditure to perform a given task. These costs accumulate slowly until they cross a critical threshold. In a study of 40 consecutive patients seen in a multidisciplinary postpolio clinic, 95% of all patients had pain complaints.[11] Of these patients, 17% had PPMP, 47% had overuse pain, and 77% had biomechanical

pain. (Some patients had more than one type of pain; therefore, totals add up to > 100%.) In a larger study of 150 patients, 44% met the criteria for PPMP.[10]

Finally, another pain syndrome seen in polio survivors is fibromyalgia, which is pain similar to postpolio muscle pain and myofascial pain. It is classified according to the following criteria[38]: widespread pain in all four quadrants of the body for a minimum of 3 months and at least 11 of the 18 specified tender points (Fig. 2).

Although these criteria focus on tender point count, a consensus of 35 fibromyalgia experts have recently determined that a person does not need to have the required 11 tender points to be diagnosed and treated for fibromyalgia syndrome. This criterion was created for research purposes, and people may still meet criteria with < 11 of the required tender points as long as widespread pain and many of the common symptoms associated with fibromyalgia are present. Commonly associated symptoms include:

- Fatigue
- Temporal mandibular joint dysfunction
- Skin sensitivities
- Chronic headaches (tension type or migraines)
- Morning stiffness
- Dizziness or impaired coordination
- Sleep disorder (or sleep that is unrefreshing)
- Postexertion malaise and muscle pain
- Numbness and tingling sensations
- Irritable bowel syndrome
- Cognitive or memory impairment
- Menstrual cramping and/or premenstrual syndrome

Trojan and Cashman studied the prevalence of fibromyalgia in polio survivors by evaluating 95 patients seen at a university-affiliated postpolio clinic.[26] Using the diagnostic criteria for fibromyalgia listed previously, they found that 10.5% of all patients had fibromyalgia. In addition, another 10.5% met the criteria for borderline fibromyalgia. In summary, a fibromyalgia-like syndrome was present in 25% of all patients who met the criteria for PPS and in 21% of all polio patients who attended that clinic.

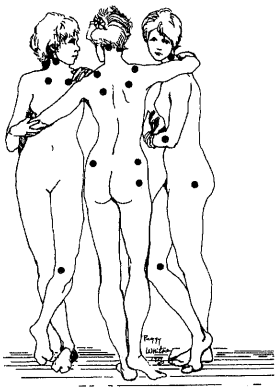

Figure 2. Tender points for diagnosis of fibromyalgia. (From Wolfe F, Smythe HA, Yanus MB: American College of Rheumatology clinical criteria for the diagnosis of fibromyalgia. Arthritis Rheum 33:160–172, 1990, with permission.)

In the Gawne et al. study of 150 polio survivors, pain descriptors were measured using the McGill Pain Questionnaire.[10] A total of 114 individuals with fibromyalgia and 94 persons with rheumatoid arthritis served as a comparison group. Both polio groups were statistically similar in their responses to this questionnaire to a group of patients with fibromyalgia, ($P < 0.01$), but they were different than patients with rheumatoid arthritis. This study suggests that the quality of pain in polio survivors is similar to fibromyalgia. This raises the possibility that fibromyalgia may be more prevalent in polio survivors than previously believed or that some of the other symptoms (e.g., fatigue, sleep disorders) that polio patients experience may be caused by fibromyalgia.

Windebank et al. did another study that demonstrated a significant incidence of fibromyalgia.[34] They examined 30 symptomatic patients and found causes for pain and fatigue in 20 patients. Among the 10 who did not have another condition to explain the symptoms of fatigue, pain, and new weakness, the symptom pattern was similar to chronic fatigue syndrome or fibromyalgia.

Although the causes of PPS and fibromyalgia are unclear, a reduction of growth hormone secretion with a resultant disruption of normal muscle repair can contribute to the coexistence of these conditions and to symptoms of pain, fatigue, and weakness in patients with PPS. It has been postulated that 80% of growth hormone is secreted during stage 4 sleep and that disruption of sleep results in a reduced level of the secretion of growth hormone and somatemedin C, or insulinlike growth factor (IGF-1).[26] This hormone stimulates the synthesis of protein and nucleic acids in muscle cells as well as neurons and may possibly stimulate regeneration of peripheral nerves after injury. This reinnervation includes sprouting, which occurs to strengthen muscles and assist recovery.[23] Somatemedin C has been found to be low in patients with fibromyalgia as well as those with PPS. In 1991, Shetty et al. reported that the serum IGF-1 level was low in 10 postpolio subjects.[22] Later, they also found reduced levels of IGF-1 in 124 polio survivors compared with a group of healthy age-matched controls.[23] However, they found that there was no difference in IGF concentrations among those with functional decline compared with those who were stable. Sunnerhagen and colleagues found no significant difference in IGF levels in 87 patients with a history of polio compared with a reference group.[25] There is clearly a need to further explore the relationship between fibromyalgia, IGF-1 levels, and the effects of growth hormone.

TREATMENT OF MUSCULOSKELETAL PAIN

Pain management of patients with PPS is based on a few basic principles, supplemented by class-specific recommendations. These basic principles include making efforts to improve abnormal body mechanics, mechanically correct and minimize postural and gait deviations, relieve or support weakened muscles or joints, promote lifestyle modifications, and decrease the abnormally high work load of muscles relative to their limited capacity.[6]

Treatment of patients with PPMP includes decreasing activity throughout the day. Pain can be relieved with rest, application of moist heat, and gentle stretching. Stretching has a role in maintaining the extensibility of muscle and connective tissue; however, it must be performed judiciously because there are situations in which a polio survivor may derive greater functional benefit and move about more safely with tighter tendons and reduced joint range of motion. A variety of medications are used to treat postpolio muscle pain, including nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen, tramadol, benzodiazepines, and (rarely) narcotics. The use of tricyclic antidepressants (TCAs), especially amitriptyline, can help with pain and fatigue.

Treatment for overuse pain includes modification of extremity use, followed by modalities such as ice, heat, ultrasound, transcutaneous electrical nerve stimulation, and NSAIDs. Treatment for myofascial pain consists of myofascial release techniques, including spray and stretch and trigger-point injections. Rest is often not possible because many patients rely on their upper extremities for both locomotion and self-care. In rare cases, corticosteroid injections or surgery may be needed.[7]

Treatment for biomechanical pain includes posture and back care education as well as decreasing weight bearing through the use of assistive devices such as braces, crutches, wheelchairs, and scooters. Abnormal biomechanics can often be modified with fairly simple and practical interventions such as cervical pillows, lumbar rolls, gluteal pads, dorsal-lumbar corsets, and heel lifts.[6] Biomechanical pain is usually improved by conservative measures aimed at reducing mechanical stress such as supporting weakened muscles, stabilizing abnormal joint movements, and improving body biomechanics. In particular, efforts should be directed at improving posture and body mechanics during routine daily activities such as sitting, standing, walking, and sleeping, as well as any repetitious activities at work. Anti-inflammatory agents are used commonly to supplement conservative measures, and joint injections can also be helpful. Weight bearing with the wrist hyperextended and radially deviated should be avoided.[7]

For those with CTS who must use a cane or crutch, an ergonomically designed grip such as an Ortho-ease (Lumex) or a "gel grip" (Superlite) is prescribed to place the wrist in a more neutral position and distribute weight-bearing on the palm. Superlite also has a Tornado Tip, which absorbs shock and can be placed on canes or crutches. The use of aluminum or "chromy steel" reduces the weight further, decreasing pressure on joints. Providing adequate support for weakened muscles and unstable joints can often be a difficult challenge; however, the basic orthotic principles are similar to those used in the management of patients with other neuromuscular diseases. For individuals with low back pain, lumbosacral corsets, a shoe lift, or pelvic lift can help improve biomechanics. For genu recurvatum (back knee) or genu valgus (knock knee) caused by quadriceps weakness or ligament instability, a knee ankle foot orthosis (KAFO) with a free ankle and an extension stop at the knee is used. Townsend has recently developed a "polio brace" specifically for this purpose. Constructed as a custom-made laminated knee orthoses, it controls hyperextension at the knee while allowing

free movement at the ankle. Polio survivors with dorsiflexor weakness or ankle instability can benefit from an athletic ankle splint, high-top shoes, or an ankle foot orthosis (AFO). Many individuals need an orthosis that combines strength and lightness. The new plastics and lightweight metals can often be used alone or in combination. Some polio survivors prefer to repair and use their old braces rather than start over with new ones. Others may resist using any kind of brace for cosmetic and psychological reasons. Orthotics are recommended to improve safety by reducing the risk of falls, to reduce pain, and to decrease fatigue by improving gait speed and symmetry.

In Perry et al.'s study of muscle overuse, they concluded that the most expedient way to reduce symptoms such as pain that occurred through overuse was the prescription of an appropriate AFO or a KAFO.[20] An AFO should preferably be a solid shell, allowing 15–20° of plantar flexion to allow the foot to reach foot flat during the stance phase of gait, reducing the demand for the use of the quadriceps. In this study 11 of 21 subjects wore AFOs, with 9 of the 11 reporting subjective improvements with symptoms.

In Waring et al.'s study of 104 patients, 19% were using braces at the time of their initial evaluation.[31] An additional 37 patients were prescribed braces, including AFOs (31.4%), KAFOs with free offset knee joints (25.7%), and KAFOs with drop lock knees (42.9%). Patients who were prescribed orthoses to correct their ambulation and those who used them daily reported improved ability to walk, improved walking safety, and reduced knee and overall pain ($P < 0.05$) compared with those who did not wear orthoses. Those who did not use orthoses on a daily basis showed fewer improvements than those who did. Reasons for not wearing braces included fit, not enough training, and that they "don't help." Reasons for not using a prescribed cane or crutch included "don't want to," not enough training, pain, and "doesn't help." This uncontrolled clinical observation study provides case series evidence for the effectiveness of a biomechanically oriented musculoskeletal intervention on PPS symptom reduction.

Many times, an assistive device is necessary to help with either pain or deformity. In a recent study of 27 polio survivors (15 women and 12 men ages 46–82 years), Bernstein and Gawne found that 74% reported foot pain and 89% reported foot deformity, ranging from limb length discrepancies to hammertoes, cavus feet, and equinus deformity.[7] All of the patients reported using some type of appliance, assistive device, special shoe, or brace for ambulation.

Peach and Olejnik studied 77 polio survivors, dividing them into compliers (complied with all recommendations), partial compliers (accepted some recommendations), and noncompliers.[18] At the time of follow-up (mean, 2.1 years later), the complier group reported resolution of muscle pain in 28%, resolution of joint pain in 41%, and improvement in symptoms of muscle pain in 72% and joint pain in 53%, but none were unchanged or worse. The noncompliers showed worsening or progression of muscle pain in 29% and joint pain in 18%; only 14% had some improvement in muscle pain, and none had resolution of muscle or joint pain or improvement in joint pain. The partial compliance group demonstrated a wider range

of responses, either no change or some improvement but little resolution (3–4%). Muscle strength increased in compliers, but decreased in the other groups. Reasons for noncompliance included lifestyle and resistance to orthotics and other assistive devices.

Alternative treatments have been suggested for polio pain; they were recently tested by Valbano et al. in a study of 50 polio survivors with myofascial pain.[29] Biomagnets were placed over palpable "trigger points," and examiners found a significant difference between the treatment group and the control group. Whereas only four patients (19%) in the placebo group reported relief, 22 patients (76%) in the magnet group reported relief. Whether the pain was of a myofascial nature or arthritic nature, the patients responded well to the static magnetic field. The effect was noticed within 45 minutes from the onset of the application.

In their study of 150 polio survivors, Gawne et al.[10] found that the majority reported that pain-relieving strategies included rest, heat, massage and stretching. Braces were helpful in 16–23% of all those studied. Local injection was found to be significantly more effective in patients who had overuse or biomechanical pain, relieving pain in 11% of those patients but only 1% of those with PPMP (see Table 3). Things that were of little use or actually exacerbated pain included ice, exercise or activity, stress, and weight gain (see Table 4).

In a study that evaluated the effects of dynamic water exercise in individuals with PPS, Willen et al. compared both the effects of training on cardiovascular function as well as general well-being.[37] Pain was measured on the VAS using a 1-to-100 scale and the Nottingham Health Profile (NHP) that measures pain, sleep energy level, and other measures. They found that the group who participated in an 8-month period of water exercises 40 minutes long three times a week had a no significant change in the amount of pain on the VAS; however, there were significant differences on the NHP (37 pretraining, 18 posttraining) compared with the control group (41 pretraining, 46 posttraining). In addition, they reported increased well-being and improved fitness on the NHP as a positive aspect of the water exercise.

Medications that are used to treat pain associated with PPS include NSAIDs (e.g., ibuprofen, naproxen) and cyclooxygenase-2 (COX-2) inhibitors (e.g., rofecoxib or celecoxib).[19] These all raise the risk of gastrointestinal effects such as ulcers and renal side effects, but these are seen less frequently with COX-2 inhibitors. Less frequent side effects include edema, diarrhea, and cardiovascular complaints such as hypertension. Injectable corticosteroids (Celestone and Kenalog) can also be used. Muscle relaxants such as cyclobenzaprine or baclofen may be useful, especially when there is muscle spasm. However, they carry the risk of sedation and further fatigue. Nerve stabilizers such as carbamazepine and gabapentin are occasionally helpful, but they also carry the risk of sedation and dizziness. Substance P blockers such as capsaicin can be used for localized pain, but they may cause burning and stinging. Narcotics such as codeine, hydrocodone, and even oxycodone can be used in cases of severe pain, but they can cause constipation, tolerance, and sedation. TCAs such as amitriptyline or trazodone and minor tranquilizers such as diazepam and alprazolam can assist with sleep as well as depression and anxiety, but they also cause drowsiness.

Other antidepressants such as fluoxetine have been tried, and tramadol is also sometimes successful.

Trojan and Cashman recommend the use of low-dose amitriptyline, cyclobenzaprine, fluoxetine, NSAIDs, relaxation, heat, massage, injections, and aerobic exercise for the treatment of patients with fibromyalgia.[26] They found that low-dose amitriptyline was helpful in controlling symptoms in approximately 50% of those with fibromyalgia or borderline fibromyalgia. If this was insufficient, most patients benefited from an alternative therapy.

The medication and modality used depend on the type of pain the patient is experiencing as well as the intensity and duration of the pain. It is recommended that when a true analgesic is required, it should be given in moderate amounts and on a schedule, not just when the pain is so severe that a higher dose is necessary.[19] If taken together, mild muscle relaxants or anxiolytics may make painkillers work better and at a lower dose, but they do have their own side effects. Nonpharmacologic measures such as hot packs, ice packs, ultrasound, hypnosis, massage, acupuncture, and relaxation can be used to avoid the side effects medications may cause.

CONCLUSION

Physical disability secondary to polio causes increased energy rates while patients perform tasks such as ambulation and ADLs, as well as reduced movement economy compared with nondisabled individuals. Reduced movement economy may contribute to increased fatigue. In addition, polio patients commonly develop musculoskeletal overuse syndromes, such as muscle strains. This leads to pain, which is treated by and relieved by rest, leading to disuse weakness and atrophy. Weakness can further exacerbate pain, perpetuating that cycle. A vicious cycle can occur in which fatigue, weakness, pain, and loss of physical function restrict physical activity, which in turn leads to further muscle and cardiorespiratory deconditioning and further reduction in function. When patients were prescribed orthoses (e.g., braces) to correct their ambulation, those who use them daily reported improved ability to walk, improved walking safety, and reduced knee and overall pain. Additional measures that have been found to helpful for treatment of both pain and disability are water exercises; heat; rest; and a number of medications, particularly those that assist with sleep in cases of fibromyalgia and PPMP.

Pain can be reduced by altering biomechanics and by changing to a lifestyle that reduces physical activity. These strategies may be difficult to accomplish, however, because they often require developing behaviors that are different than old familiar ones. Altering the pace and intensity of discretionary activities and learning new ways to gain more control over when and how activities are performed are essential. Education in the proper use of orthotic devices is important to improve compliance with the recommendations. Restoration of function as well pain relief can be accomplished by an interdisciplinary team that includes the polio survivor, physical therapist, occupational therapist, psychologist, orthotist, rehabilitation engineer, and physician.

The available research favors a causal relationship between symptoms of new weakness and musculoskeletal pain syndromes. Commonly accepted clinical thinking and practice

regarding musculoskeletal pain syndromes and syndromes of fatigue and new weakness further support this relationship. Demonstrated effectiveness of treatments for patients with musculoskeletal pain syndromes offers further support for a likely causal relationship.

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