Original Research

Electrophysiology and Electrodiagnosis of the Post-Polio Motor Unit

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Abstract

Post-poliomyelitis syndrome refers to new symptoms that may occur years after recovery from poliomyelitis. The most common of these symptoms are new weakness, fatigue, and pain. This article describes electrodiagnostic studies -- conventional electromyography (EMG), single fiber electromyography (SFEMG), and macroelectromyography (macro-EMG) -- that have provided information on the post-polio motor unit and on the possible etiology of some post-polio syndrome symptoms. Muscular fatigue, and indirectly, general fatigue, may be due to neuromuscular junction transmission defects in some post-polio individuals, as suggested by reduction of the compound motor action potentials on repetitive stimulation, and increased jitter and blocking on SFEMG. Progressive weakness and atrophy in post-polio syndrome is probably due to a distal degeneration of post-polio motor units with resultant irreversible muscle fiber denervation. Electrodiagnostic evidence of ongoing denervation includes fibrillation and fasciculation potentials on conventional EMG, increased jitter and blocking on SFEMG, and smaller macro-EMG amplitudes in newly weakened postpolio muscles. However, even though electrodiagnostic studies have provided insight into the possible causes of some postpolio syndrome symptoms, no specific electrodiagnostic test for the syndrome is currently available.

The late progression or appearance of neuromuscular symptoms in individuals who have previously recovered from paralytic poliomyelitis is now recognized as a major public health problem in North America. Although a wide variety of symptoms have been reported, weakness, fatigue, and muscle and joint pain are consistently the most frequently noted.*[1-9]* We favor the term post-poliomyelitis syndrome

for this constellation of new symptoms that often present simultaneously. Although some postpolio syndrome symptoms such as new weakness are almost certainly due to changes in the motor unit, it is unclear how or if pain, which is uncommon in the classical motor neuron diseases such as amyotrophic lateral sclerosis, is referable to disease of the motor unit. Thus, the post-poliomyelitis syndrome may be the most inclusive term, which encompasses symptoms that may or may not be due to motor unit dysfunction, and also refers to the frequent concurrence of these symptoms.

This article reviews the physiology of the post-polio motor unit, emphasizing electrodiagnostic studies, and relates these findings to three post-polio syndrome symptoms that may be explained by motor unit abnormalities: new fatigue, weakness, and atrophy.

Fatigue specific to post-polio patients can be either general or muscular.*[10]* General fatigue is usually described as a flu-like, diffuse exhaustion. Muscular fatigue (fatigability) can be defined as difficulty with muscular endurance, or increased weakness with exertion that improves with rest. Many causes have been proposed for general fatigue. These include depression, anxiety, malfunction of the brainstem reticular activating system from past involvement during acute polio, emotional stress, and even a subjective interpretation of diffuse muscular fatigability.*[9,11,12]* Possible causes of muscular or peripheral fatigue are neuromuscular junction transmission defects, overuse myopathy, and fiber type disproportion.*[3,13-15]*

New weakness can be reported as permanent, or transient, which is related to activity. We believe that transient weakness is actually muscular fatigue. New atrophy, or loss of muscle bulk, is also reported by post-polio syndrome patients. New weakness and atrophy have been theorized to be due to a number of causes including disuse, progressive denervation, and overuse myopathy.*[9]* Atrophy is not as frequent a complaint in polio survivors as new weakness and fatigue,*[2,5]* and may be a relatively late phenomenon in post-polio syndrome.*[16]*

THE NORMAL MOTOR UNIT

A motor unit, originally defined by Sherrington, consists of a motor neuron and all of the muscle fibers that it innervates or supports. Electrical excitation of a motor neuron in the central nervous system is followed by conduction of an impulse in the axon and its terminal branches, synaptic transmission at the neuromuscular junction mediated by the neurotransmitter acetylcholine, and depolarization and contraction of the muscle fibers it innervates. If enough motor units are stimulated, a clinically apparent muscle contraction will be produced. The ratio of muscle fibers innervated per motor neuron varies within a muscle and between muscles, and can range from six to 10 muscle fibers in extraocular muscles to 2000 muscle fibers in the gastrocnemius muscle.*[17]* In general, muscles that require greater specificity of action have smaller motor units.

A muscle is generally composed of two types of muscle fibers which differ in their enzymatic composition, speed of contraction, and fatigability. Type I fibers have more oxidative enzymes, are less susceptible to fatigue, and are also referred to as slow twitch fibers. Type II muscle fibers have a higher proportion of glycolytic enzymes, are more easily fatigued, and are also referred to as fast twitch fibers.

The muscle fiber type is believed to be determined by the motor neuron by which it is innervated. Thus, a specific motor neuron will innervate either Type I or Type II muscle fibers, but not both.*[17]* Motor neurons innervating Type II fibers are characterized by a higher threshold for activation, higher axonal conduction velocity, and a higher firing rate than motor neurons innervating Type I fibers.*[18]* Normally, the two types of muscle fibers are evenly interspersed within a muscle, producing a mosaic pattern.*[19]* During recruitment of muscle fibers for a muscle contraction, Type I fibers (within smaller motor units)

are usually excited first, followed by Type II fibers (within larger motor units). This method of recruitment allows the use of the less fatigable Type I fibers for continued lower levels of contraction, whereas the more easily fatigable, fast Type II fibers are used for short bursts of activity.*[18]*

The neuromuscular junction is the interface between a fine terminal nerve fiber of a motor neuron and a muscle fiber. The space between these two structures is known as the synaptic cleft. To produce a muscle fiber contraction, the depolarized motor neuron terminal releases acetylcholine into the synaptic cleft. Acetylcholine then binds to specific receptors on the muscle endplate. If enough acetylcholine is released and bound to receptors, the muscle endplate and muscle fiber sarcolemma will depolarize, calcium will be released intracellularly from the sarcoplasmic reticulum, and a calcium-dependent myofiber contraction will be produced. To allow endplate repolarization, acetylcholine is rapidly hydrolyzed by the enzyme acetylcholinesterase. The muscle fiber is then ready for another depolarization.*[17]*

THE MOTOR UNIT DURING AND AFTER ACUTE POLIOMYELITIS

During acute poliomyelitis, motor neuron invasion by polio virus can result in either motor neuron death or injury with partial or complete recovery.*[20]* Motor neuron death will cause denervation of the muscle fibers belonging to its motor unit with resultant loss of voluntary activation of those muscle fibers. If only a few motor neurons innervating a muscle are affected, no weakness may be perceived by the patient because of the normal reserve present in human muscle. More severe loss of motor neurons from acute polio results in partial or complete denervation of the muscles involved, and will be perceived as weakness or complete loss of voluntary muscle contraction by the patient.

Recovery of muscular force after acute poliomyelitis can occur by sprouting from intact motor neurons or by muscle fiber hypertrophy. If a motor neuron survives polio virus attack, within 3 to 4 weeks it will extend sprouts from its terminal axonal branches that will reinnervate locally denervated muscle fibers. *[21-24]* This process is limited by the number of remaining motor neurons, and by the ability of a motor neuron to form sprouts. It is also possible that the ability of a motor neuron to sprout and reinnervate is correlated with the severity of its own disruption by polio virus infection. As a result of these two limitations, some muscle fibers may not become reinnervated, and will undergo progressive atrophy over the ensuing months to years.*[25]*

Because terminal nerve sprouts are short (100 to 200 μ, or two to four muscle fiber diameters),*[26-27]* fiber type grouping of reinnervated muscle will result, and the normal mosaic interspersion of Type I and Type II fibers will be diminished or absent.*[28,29]* In addition, fiber type transformation from Type II to Type I fibers without selective loss of larger motor neurons (which innervate Type II fibers) can occur in weak, but still functional, post-polio muscles.*[30-32]* It has been estimated that even with a loss of 50% of the motor neurons supplying a muscle, the surviving motor neurons can achieve complete reinnervation resulting in normal muscle strength.*[33]* Muscle biopsy studies have shown that motor unit sizes after recovery from poliomyelitis can be up to seven-fold normal size.*[34]* In other words, a post-polio motor neuron may maintain up to seven times the number of muscle fibers as it would normally supply. In addition to sprouting with reinnervation, muscle fiber hypertrophy of innervated muscle may contribute to the recovery of motor strength after acute polio.*[30]*

The most likely etiology of new weakness many years after recovery from paralytic polio is a distal degeneration of these abnormally enlarged motor units, as hypothesized by Weichers and Hubbell.*[35]* Thus, new weakness decades after recovery from acute paralytic polio may be a result of the recovery process itself. Surviving motor neurons which may have permanent abnormalities from earlier polio and are now innervating many more muscle fibers than normal, may be unable to sustain such a great metabolic demand. Over time, terminal axonal sprouts may degenerate, with subsequent denervation of

individual muscle fibers. It is possible that some of these denervated muscle fibers may become reinnervated by sprouts from neighboring motor neurons, producing a continuous "remodeling" of the post-polio motor unit.*[29,36-38]*

However, some muscle fibers will become irreversibly denervated, causing a reduction in size of the motor unit, and will produce a clinically apparent weakness in the patient.*[35,36]* The normal aging process involves a dropout of motor units, predominantly after age 60,*[39-41]* and may have an additional effect on the already borderline or clinically weakened post-polio muscles.

ELECTROPHYSIOLOGY OF THE NORMAL AND POST-POLIO MOTOR UNIT

Conventional Electrolmyography. Electromyography (EMG) is a diagnostic technique used to evaluate the electrophysiology of a motor unit. It is performed by percutaneously inserting a small needle electrode into the muscle being evaluated. The concentric needle electrode can record activity of muscle fibers within a radius of about 1 mm. Activity is amplified and displayed on an oscilloscope. Normally, a muscle is silent at rest after insertional activity (produced by irritation from needle movement) has ceased. With muscle contraction, motor units are activated and motor unit action potentials (MUAPs) appear on the screen, usually as triphasic waves with an initial positive deflection.*[17]*

Because of the relatively small recording volume of an EMG needle in comparison to the diameter of a normal motor unit (in the biceps muscle motor unit, diameters range in size from 2 to 10 mm),*[42,43]* only five to 12 muscle fibers from a motor unit will contribute to the MUAP observed on the screen.*[44]* The amplitude of a MUAP is determined by the number of muscle fibers recorded with the needle. With increasing strength of contraction, there is an orderly recruitment of motor units with the number and size of MUAPs gradually increasing. At full contraction, separate MUAPs will be indistinguishable, resulting in a complete recruitment interference pattern.*[45]*

During acute polio, motor neuron loss can occur with denervation of muscle fibers. In the first few weeks, an EMG of a paretic muscle is silent at rest. After 2 to 4 weeks, a denervated muscle fiber will periodically depolarize spontaneously, producing fibrillation potentials and positive waves. A portion of or an entire motor unit may also become activated spontaneously, resulting in a fasciculation at rest which can be seen and felt by the patient. Fasciculations can also be seen occasionally in normal individuals, but are considered to be abnormal when accompanied by fibrillation potentials.*[46]* Motor neuron loss after poliomyelitis will produce a reduction in the number of motor units seen with recruitment.*[29,35]*

During the recovery process, motor units will enlarge. In addition, local sprouting will increase the number of muscle fibers innervated by the same motor neuron that are within the receptive field of the EMG needle. Motor unit action potentials will thus have abnormally large amplitudes and polyphasia. Decreased recruitment will remain, secondary to a reduction in the number of motor units available for activation during voluntary muscle contraction.*[28,29,35]*

Classically, it was believed that fibrillation and fasciculation potentials would disappear within 6 to 12 months of deneniration with maturation of the new terminal nerve sprouts.*[21,24,35]* However, it is now becoming evident that fibrillation potentials may never disappear in a post-polio survivor. The reason for this is unclear, but proposed mechanisms include ineffective or inefficient reinnervation of the very enlarged motor units, continuous remodeling with denervation and reinnervation of the enlarged motor units, and a metabolic abnormality in motor neurons affected by the polio virus that prevent them from providing normal innervation to all reinnervated muscle fibers.*[36]*

It is now known that long after recovery from acute poliomyelitis, many polio survivors will continue to

have abnormalities on conventional EMG regardless of whether or not they are having new symptoms. These include persistence of the spontaneous fibrillation and fasciculation potentials, enlarged MUAPs, and decreased recruitment.*[29]* Thus, abnormalities observed on conventional EMG do not allow distinction of symptomatic from asymptomatic individuals.*[29]*

Single fiber electromyography. Single fiber electromyography (SFEMG) is an electrodiagnostic technique that allows evaluation of single muscle fibers within a motor unit. Similar to conventional EMG, it is performed by percutaneous insertion of a small needle electrode into the voluntarily contracted muscle under study. However, the receptive volume of this needle (diameter of 25 μm) is smaller than that of a conventional EMG needle. If the motor unit is normal, this receptive volume includes up to one or two muscle fibers from the same motor unit. Muscle fiber depolarizations are recorded by the electrode, and are displayed on an oscilloscope.*[47]*

SFEMG allows the quantitation of the parameters of fiber density, jitter, and blocking. Fiber density is an indirect estimate of the number of muscle fibers per unit volume belonging to a single motor unit. In polio survivors, it can be regarded as an index of axonal sprouting, and consequently as a measure of denervation from the original poliomyelitis. Fiber density is measured by randomly inserting a needle into the muscle studied, and determining the number of muscle fibers that are depolarized by a single motor neuron within the receptive field of the electrode. The mean of 20 such measurements determines fiber density. Mean normal fiber density ranges between 1.3 and 1.7, and varies with different muscles. It is slightly higher in children under age 10 and in adults over 60.*[47]* As noted above, fiber density is increased in conditions which involve denervation and reinnervation by collateral sprouting, and in some myopathies.*[47]*

Jitter is the variability in the difference in the time of firing of two muscle fibers when both are innervated by the same motor neuron. In polio survivors, it provides an indication of the adequacy of terminal axonal impulse propagation and neuromuscular junction transmission. One jitter reading is obtained by calculating the mean consecutive difference (MCD) of 20 to 50 discharges of two muscle fibers. Usually, 20 jitter measurements are obtained by random insertions of the SFEMG needle electrode. Normal jitter values range from a lower limit of 5 μsec to an upper limit of 35 to 60 μsec for different muscles.*[47]* Usually, after age 70, jitter values increase slightly.*[47]*

Jitter is considered to be abnormal if more than one of 20 jitter values in a particular muscle exceed the normal upper limit for that muscle.*[47]* Blocking occurs when the pathology at any one of these sites is more severe and actual transmission failure is produced. Blocking can be expressed as a percent of muscle fibers within a muscle exhibiting this phenomenon. It is absent in normal muscle. Thus, SFEMG can provide information about neuromuscular transmission with determination of jitter and blocking, and about motor unit reorganization with fiber density.

Stimulation SFEMG is a modification of conventional SFEMG which differs in the method of motor unit activation. During conventional SFEMG, muscle fibers are activated voluntarily; during stimulation SFEMG, muscle fiber activation is produced by a stimulating needle electrode placed near the motor point of the muscle. Stimulation SFEMG also permits assessment of jitter and blocking. However, the jitter readings obtained with this technique will be lower since they reflect the neuromuscular transmission at only one muscle fiber, rather than two muscle fibers as in the more traditional voluntarily stimulated SFEMG.*[48,49]*

An advantage of stimulation SFEMG is that it allows jitter determination at variable stimulation frequencies chosen by the electromyographer.*[48,49]* Thus, rather than seeking motor units with different stimulation frequencies as in conventional SFEMG, the electromyographer has direct control over the

study of a single motor unit at a variety of stimulation frequencies. Because of the known dependence of jitter on activation frequencies in several disease processes involving the neuromuscular junction,[50-53] stimulation SFEMG may enable the electromyographer to gather more information on the function of the motor unit.

During the reinnervation process after acute polio, axonal sprouts are initially unmyelinated and conduction is slow and variable. The newly formed neuromuscular junctions may also be a site of transmission slowing or failure. These abnormalities will present as increased jitter and blocking on SFEMG (Fig 1A). In addition, fiber type grouping as a result of motor unit remodeling during reinnervation will result in increased fiber density on SFEMG in the involved muscle (Fig 1B).

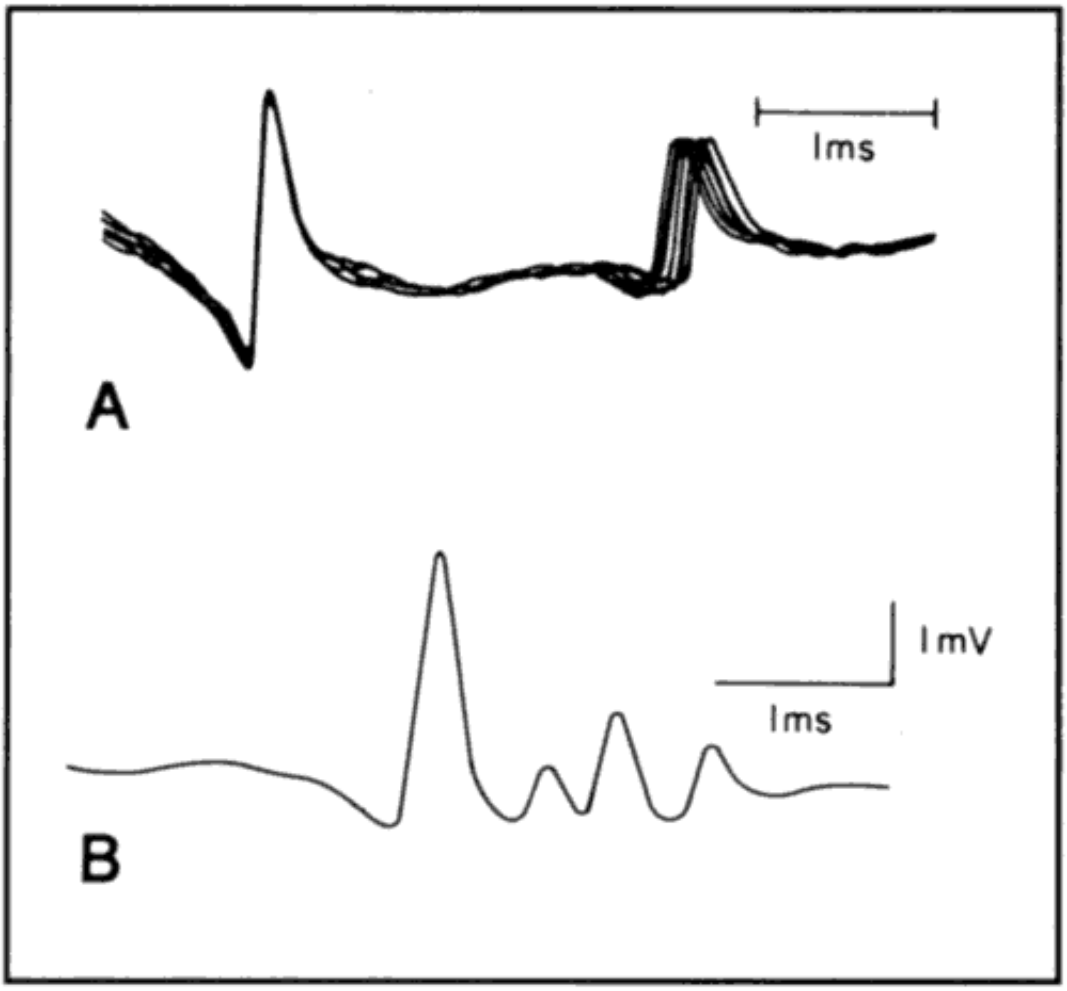

Fig 1: Single fiber electromyography of a postpolio muscle. Panel A illustrates increased jitter in 10 superimposed single muscle fiber action potentials (mean consecutive difference, 74 µsec; normal for muscle, <35 µsec). Panel B illustrates increased fiber density. Four single muscle fiber action potentials are observed in the vicinity of the SFEMG needle (mean ± SD normal value for age, 1.4 ± 0.11).[29]

Contrary to earlier hypotheses that the newly formed axonal sprouts and neuromuscular junctions would mature eventually,[21,35] increased jitter and blocking persist in the polio survivor.[29,36] Thus, abnormalities observed on SFEMG, as on EMG, are not helpful in distinguishing symptomatic from asymptomatic individuals.[29] However, a significant correlation between the percentage of fibers exhibiting jitter and fiber density has been found, suggesting that muscles with the most enlarged motor units as a result of sprouting are more likely to exhibit instability later in life (Fig 2).[29] Whether these abnormalities increase with the passage of time is not clear.[35,36] Certainly, a relationship of these

SFEMG abnormalities and the clinical evolution of post-polio syndrome has not consistently been found.

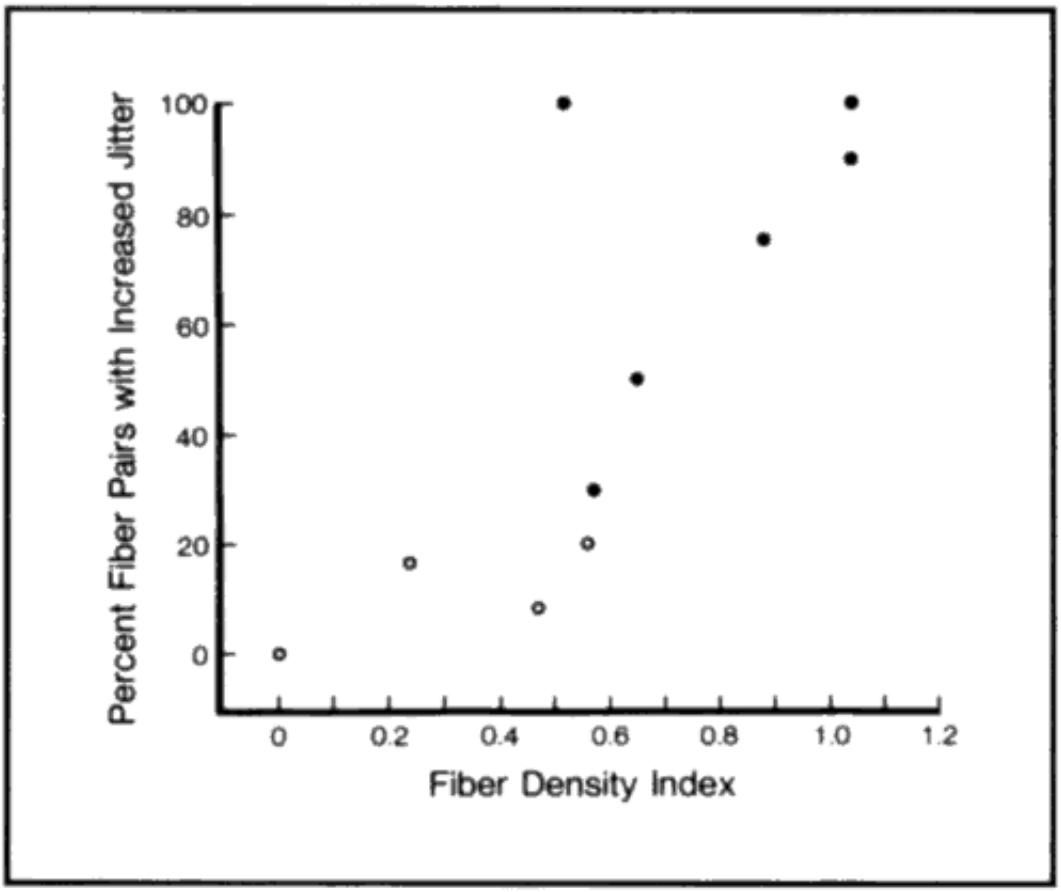

Fig 2: Graph of percentages of muscle fiber pairs exhibiting increased jitter versus fiber density index. A significant correlation is observed between percentage of fiber pairs exhibiting increased jitter and fiber density (Spearman correlation coefficient r = .77, $P<.05$). A significant association is also noted between fiber-type grouping on muscle biopsy (closed circles) and percentage and fiber pairs with increased jitter (Wilcoxon signed rank sum test, $P<.01$).*[29]*

We have performed stimulation SFEMG studies in symptomatic post-polio patients, and have confirmed the presence of increased jitter with this technique in polio survivors as compared to normal controls (Fig 3). We have also studied the neuromuscular junction in post-polio syndrome patients by evaluating jitter with different stimulation frequencies on stimulation SFEMG. Our preliminary studies suggest that jitter depends on stimulation frequency in a proportion of post-polio syndrome patients in a manner consistent with a presynaptic defect. A similar relationship between jitter and stimulation frequency is observed in amyotrophic lateral sclerosis, where exhaustion of acetylcholine stores at nerve terminals probably contributes to the neuromuscular defect.*[54]*

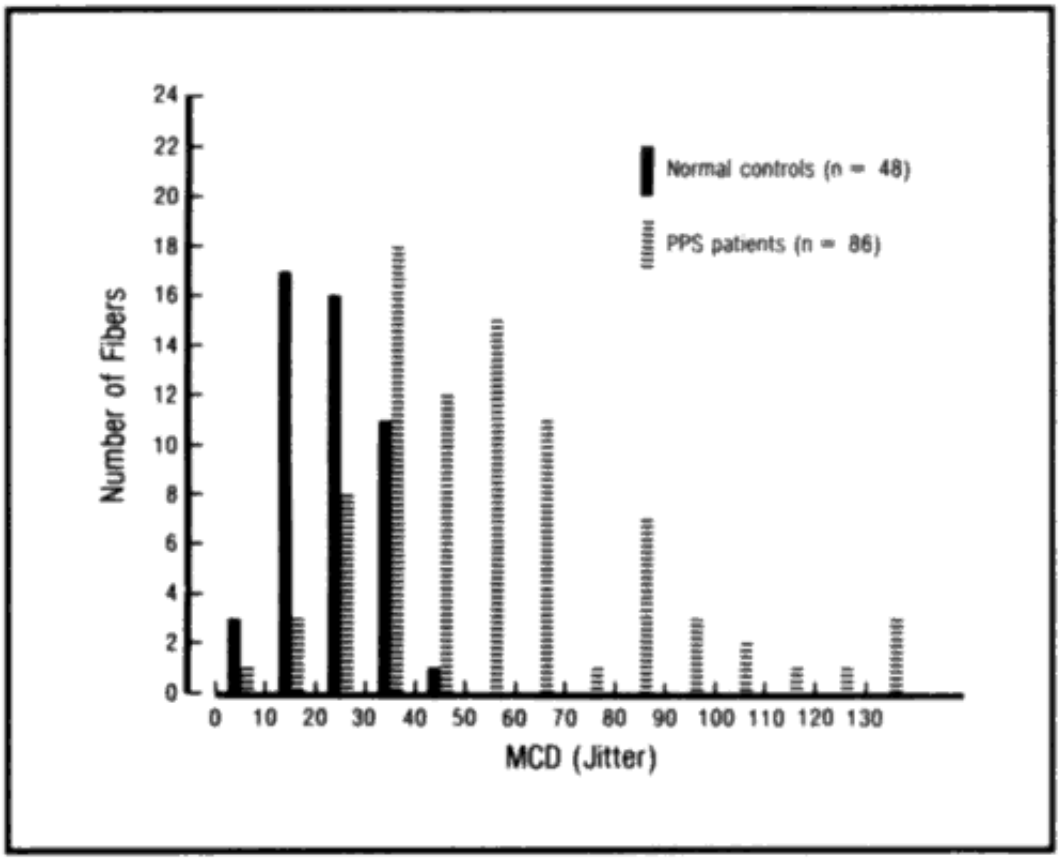

Fig 3: Distribution of jitter (mean consecutive difference) values in individual muscle fibers in post-polio syndrome and normal quadriceps muscles as determined by stimulation single fiber electromyography. Mean jitter for normal quadriceps was 23.17 ± SD 8.63 μsec (n = 48) as compared to mean jitter for post-polio syndrome quadriceps 56.48 ± SD 34.34 μsec (n = 86).

In addition, we have used stimulation SFEMG to probe the relationship between neuromuscular junction transmission defects and the postpolio syndrome symptoms of generalized fatigue and muscle fatigability. Evidence for neuromuscular junction transmission defects on SFEMG studies are seen in post-polio sydrome,*[28,29,35-37,55]* and also in such disorders as amyotrophic lateral sclerosis and myasthenia gravis.*[56-59]*

Clinically, all three disorders can present with muscle fatigability, defined as increased muscle weakness on exenion, improving with rest.*[55,58,59]* Since anticholinesterase agents can ameliorate clinical muscle fatigability and neuromuscular transmission defects in myasthenia gravis and amyotrophic lateral sclerosis,*[56,58-60]* we have studied their effect in post-polio syndrome.

Preliminary studies have shown that jitter, as measured by stimulation SFEMG, can decrease with edrophonium injection, indicating amelioration of the neuromuscular junction defect in a proportion of fatigued post-polio syndrome patients (Fig 4). We have also found that jitter response to edrophonium is related to the patients' subsequent subjective fatigue response to the oral anticholinesterase pyridostigmine. Therefore, fatigue in some patients may be due to an anticholinesterase-responsive neuromuscular junction transmission defect.*[61]* The fact that not all post-polio syndrome neuromuscular junctions responded similarly to an anticholinesterase agent and to high frequency stimulation helps support the notion for different neuromuscular transmission defects in post-polio syndrome.

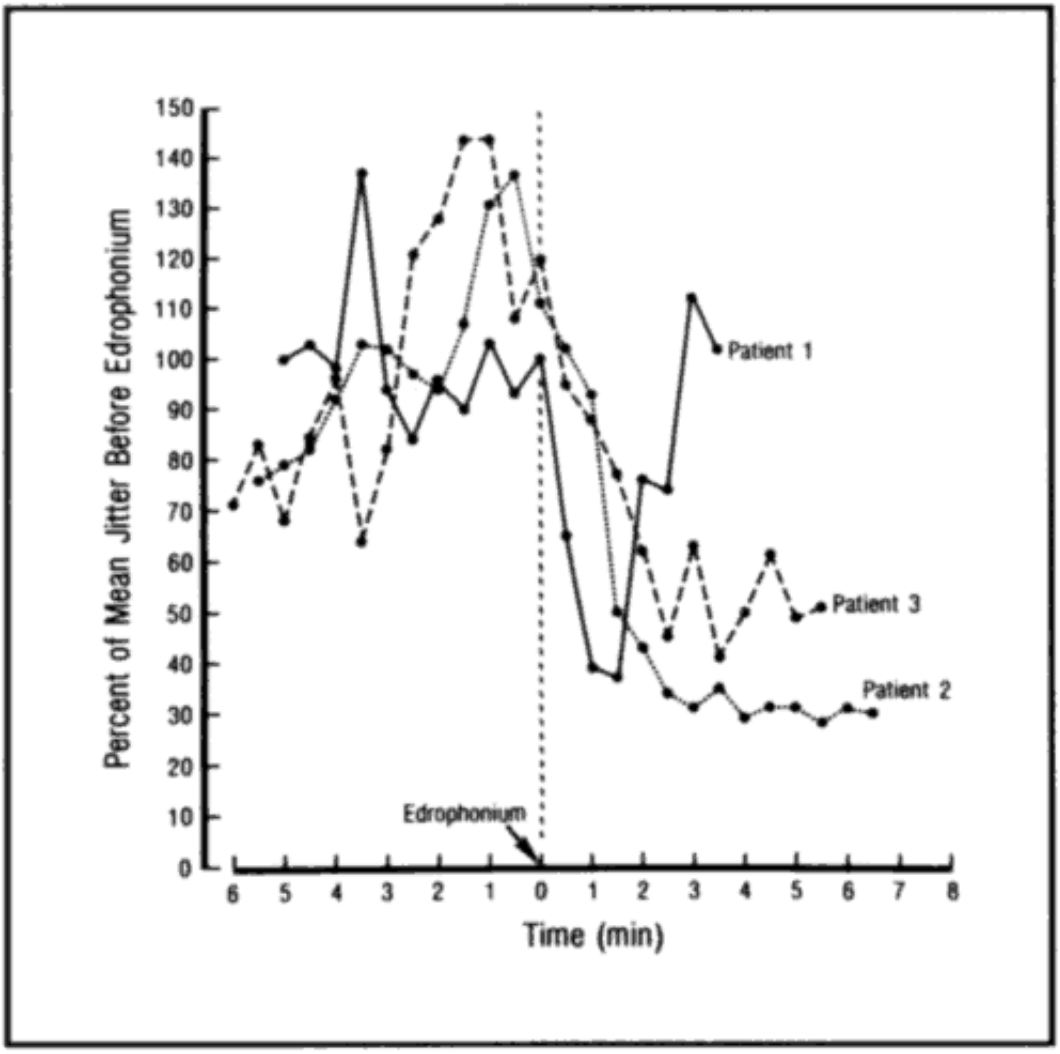

Fig 4: Jitter (mean consecutive difference) values obtained by stimulation single fiber electromyography before and after edrophonium injection in three patients. Jitter is expressed as percent of mean jitter before edrophonium injection.

Macroelectromyography. Macroelectromyography (macro-EMG) is a newer electrodiagnostic technique that can assess the size of the entire motor unit. Like EMG and SFEMG, it is performed by placing a needle electrode into the muscle being studied. Because the recording surface of the macro-EMG electrode is much larger than that of the EMG electrode, macroEMG can measure the summated action potential of most muscle fibers within a voluntarily activated motor unit. Either the area or amplitude of the macro-EMG motor unit potential (macro-MUP) can be used as an estimate of the motor unit size. *[62,63]* Simulation studies have shown that macro-MUP amplitude is positively correlated to the total number of muscle fibers and to the mean muscle fiber diameter in the motor unit under study.*[64]*

During the reinnervation process after acute polio, macro-EMG amplitudes will increase in size, reflecting the larger motor units found in these patients. The median macro-EMG amplitude can be up to seven times the normal limits for age, or 20 to 40 times the upper normal limit for individual motor units.*[63,65]* It is believed that with time macro-MUP amplitudes may decrease, reflecting the peripheral disintegration of the motor unit. However, a serial study of 11 post-polio patients showed no consistent trend in macro-MUP amplitudes measured at yearly intervals over 1 to 2 years.*[66]* This may reflect the slow progression of the disease process in the relatively short follow-up period.

Macro-MUP amplitudes have been shown to be smaller in post-polio muscles with new weakness and atrophy than in post-polio muscles of normal strength or in weak but stable post-polio muscles.*[65]*

However, there is no method as of now to distinguish between a small motor unit due to loss of axonal branches, and a small motor unit that was never enlarged by axonal sprouting. Thus, further study is needed of macro-MUP as a diagnostic technique for post-polio syndrome.

UTILITY OF ELECTRODIAGNOSTIC STUDIES IN POST-POLIO PATIENTS

Pathophysiology of the post-polio motor unit. Even though no specific electrodiagnostic abnormality has been observed in symptomatic post-polio patients,*[29]* electrodiagnostic studies have provided information on the possible etiologies of the post-polio syndrome symptoms of fatigue, weakness, and atrophy. It is possible that a combination of factors, including the degree of motor unit deterioration, reserve present in the muscle affected, the functional importance of the muscle involved, and the general activity level of the patient will determine whether or not new symptoms are perceived by the patient.

Evidence for neuromuscular junction deficits are frequently observed in polio survivors, and may be a cause of muscular fatigue. Earlier studies demonstrated a reduction of compound motor action potentials on supramaximal repetitive stimulation,*[55]* and numerous recent studies demonstrate increased jitter and blocking on SFEMG.*[28,29,35-37]* There appears to be a relationship between the degree of neuromuscular junction deficits and the degree of motor unit enlargement after polio. Jitter has been shown to be increased in patients with a higher fiber density (indicating greater motor unit reorganization),*[29]* increased macro-MUP amplitudes (suggesting larger motor units), and fiber-type grouping on muscle biopsy.*[66]* Therefore, the extent of initial recovery may predict the degree of electrophysiological deficits later in life.

New, permanent progressive weakness and atrophy in polio survivors is probably due to permanent denervation as a result of a distal degeneration of enlarged motor units.*[35]* Evidence for this theory includes smaller macro-MUP amplitude in newly weakened patients*[65]* and angular atrophic muscle fibers indicative of ongoing denervation on muscle biopsy.*[29]* Some evidence of denervation, including fibrillations on conventional EMG and widespread myofiber immunoreactivity for neural cell adhesion molecule, can be seen in motor units undergoing "remodeling" (ie, denervation/reinnervation without net loss in size of motor units), and thus cannot be regarded as unequivocal evidence for a progressive denervating syndrome in postpolio syndrome.

In addition to providing information on possible etiologies of post-polio syndrome symptoms, electrodiagnostic studies can be used to document changes in the motor unit that occur as a result of treatment. Our studies have revealed the amelioration of jitter with a shortacting intravenous anticholinesterase medication in a proportion of post-polio syndrome patients.*[61]* An uncontrolled, open trial of pyridostigmine in fatigued polio subjects has also revealed a subjective improvement in fatigue in approximately 60% of subjects.*[67]* Further controlled studies are warranted to better evaluate the benefits and risks of these medications in post-polio syndrome.

Clinical utility. At present, electrodiagnostic studies in post-polio patients can be used in clinical practice to confirm past poliomyelitis involving motor neurons, and as an aid to exclude other conditions that could be causing symptoms of increased weakness, fatigue, and atrophy. For example, nerve conduction studies that should be normal in polio survivors may be abnormal in those with a peripheral neuropathy. In addition, F-waves, H-reflexes, and somatosensory evoked potentials studies in combination with nerve conduction velocity studies can be helpful to exclude radiculopathies and myelopathies that may mimic post-polio syndrome.

Even though clinical electrophysiologic tests cannot be used currently for the diagnosis of post-polio syndrome,*[29]* it is possible that macro-EMG studies may prove useful in the future. Further carefully

controlled and clinically correlated electrodiagnostic studies may provide more information about the pathophysiology, diagnosis, and treatment of post-polio syndrome.

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