Muscle Function, Muscle Structure, and Electrophysiology in a Dynamic Perspective in Late Polio

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The muscular impairment in patients with a history of polio varies from none to severe. The relationship between the degree of initial involvement and the effect of various compensatory mechanisms determines the clinical picture, which changes dynamically. Early and late recovery after poliomyelitis depend on a number of factors. Clinical improvement that appears within a *few weeks* after the acute phase is probably due to recovery in the excitability of functional, but not degenerated, motor neurons. Degeneration of neurons, causing peripheral denervation, is compensated by collateral sprouting, i.e., by nerve twigs branching off from surviving motor units overlapping with the denervated ones. This is most likely the main factor explaining recovery within the *first 6-12 months*. Another late compensatory process is the increase in size of the muscle fibers. As a result of these processes, normal muscle strength and presumably normal muscle volume can be seen despite a calculated loss exceeding 50% of the number of motor neurons.

Besides the need for a certain muscle volume and, thus, potential for force development, the metabolic adaptation to endurance activity is also a key issue. It is well known that capillarization and the activity of mitochondrial enzymes adapt to a level dependent on the physical activity.*[2,·13]* With immobilization or reduced physical activity, these factors, of basic importance for aerobic capacity and, thus, performance in endurance activities, are reduced. In polio patients with low general physical activity, reduced aerobic muscle capacity may occur, but it is combined with marked muscle fiber hypertrophy, demonstrating different adaptation patterns for resistance and endurance activities.

In this chapter, we first describe the basic morphologic and electrophysiologic adaptations in muscle function and then review the changes that can be seen over time in persons with polio sequelae. By combining data on muscle strength, muscle structure, and electrophysiologic recordings of the size of the motor units, further insight can be obtained on these compensatory processes.

MUSCLE STRENGTH

For strength measurements, the reader should be reminded of the need for objective measurements of muscle strength in polio patients, either by special dynamometers (such as Cybex[9], Kin-Com,[10] and Lido[1] or by hand-held manual measuring devices (such as Myometer[8]). Good reliability of repeated dynamometer measurements of knee muscles has been reported by Kilfoil and St. Pierre[14] and by Grimby et al.[10] Manual muscle testing does not give reliable information in muscles with strength levels of fair (being able to move the extremity against gravity) or above, as shown already in the 1960s by Beasly[3] and illustrated in chapter 4 by Agre in this book. Isometric measurements are usually quite adequate, as isometric and isokinetic strength values correlate significantly[7] and change in parallel over time.[11]

Muscle strength can be reduced by various degrees and obviously quite differently in different muscle groups, depending on the distribution of the original polio involvement. Whether the dynamics of the compensatory processes differ between muscle groups, however, has not been studied. The stimulus for muscle fiber hypertrophy may vary between muscle groups, depending on their activity pattern.

It is possible to demonstrate a relationship between the perception of new or increased muscle weakness and measurements of reduction in muscle strength. In follow-up studies of persons with poliomyelitis sequelae, it has been possible to demonstrate significant reduction in muscle strength in those who acknowledge new weakness but not in those who do not acknowledge new muscular weakness.[10,11] Thus, in a group of 44 Swedish patients,[11] a 9% (p <0.01) reduction in strength for knee extension at 60°/s angular velocity during a 4-5 year period was demonstrated. When the patients were divided into two groups, the strength reduction was significant (16%, p <0.01) only among those who reported new muscle weakness. As a comparison, the normal age-dependent reduction in muscle strength in the age ranges of 30-70 years (mean age, 53) can be estimated to be around 2-5% during a similar time period and thus not more than about 1% per year.[5,15]

In the following chapter, we use the terms *unstable* and *stable* muscle function for those who acknowledge and do not acknowledge, respectively, new or increased muscle weakness in the tested extremity. Such a division is better than using the post-polio syndrome classification in studies comparing various muscle parameters.

MUSCLE STRUCTURE

Morphological changes in the post-polio muscle are discussed further in chapter 4 in this volume by Borg and Edström. Here, information is given mainly on the adaptive process with changes in fiber size after loss of motor neurons.

Markedly increased muscle fiber areas are seen in the polio-affected muscles,[4,9] provided that muscle strength is only slightly reduced and there is not severe muscle atrophy. According to the report by Grimby and coworkers[9] on the vastus lateralis muscle, mean fiber area was 8 x 10^3 µm² in the post-polio muscles compared to 4.4 and 4.1 x 10^3 µm² for men and women, respectively, in the control groups. There was an increase in type I as well as type II fibers but a somewhat less increase in type IIB fibers. Similar results were found by Borg and coworkers[4] in the tibialis anterior muscle in polio subjects who were excessive users as defined from electromyography (EMG).

In a 4-5-year follow-up study, no systematic changes in mean muscle fiber area could be seen, but when the changes were analyzed in individual polio subjects, certain patterns were noted.[10] Subjects who acknowledged increased muscle weakness (unstable) did not, as a group, show a significant reduction in muscle fiber area. However, some subjects who had average fiber areas close to three times the control

values showed a reduction in size during the follow-up period, usually combined with a decrease in muscle strength, but proportionally less. Obviously, compensation by reinnervation could partially compensate for the reduction in fiber size. Other subjects (stable as well as unstable), on the other hand, showed further increase in fiber size during the follow-up period. These findings illustrate the dynamic process in which increased motor unit use due to the parallel loss of motor units can be a stimulus for further fiber hypertrophy, but also indicate that an optimal size for fiber hypertrophy may be reached. The reduction in size of these very large muscle fibers may be caused by several factors, such as basic biological mechanisms leading to reduced capacity to maintain fiber hypertrophy or fiber size reduction due to disease.

NEUROPHYSIOLOGICAL METHODS

A review of the different methods of testing in the post-polio patient is given to explain their differences as well as to highlight the chief parameters measured by each.

SINGLE-FIBER EMG (SFEMG)

The method of SFEMG is described in detail elsewhere.*[19]* Two parameters are usually measured. One is *fiber density*, a measure of the number of muscle fibers of a motor unit within a hemisphere within a radius of about 300 µm from the small recording electrode. This measure increases with changes in the topography of muscles fibers within the motor unit, typi cally in cases of reinnervation. It may also increase in myopathy, but other EMG parameters differentiate these two conditions.

The other parameter is *neuromuscular jitter*, an indicator of neuromuscular transmission. When neuromuscular transmission is disturbed, e.g., in myasthenia gravis, the jitter is increased. With more pronounced disturbance, there is also occasional impulse blockings due to impulse failure. Jitter, however, is also increased in conditions with ongoing reinnervation. This is usually due to immaturity of nerve endings and neuromuscular junctions. There may be both pre-synaptic and post-synaptic reasons. Increased jitter and intermittent neuromuscular blocking are seen in some of the recordings from polio patients, but to such a low degree that neuromuscular failure could only explain part of the muscular symptoms.

CONCENTRIC OR MONOPOLAR ELECTRODE EMG (CONVENTIONAL EMG)

In the most commonly used EMG technique, an EMG electrode, with a recording surface somewhat larger than the SFEMG electrode, records from 0.5-2 mm within the motor unit that normally has a diameter of 5-10 mm. The investigation consists of three phases. First, the muscle is studied at rest. Normally, no activity is recorded. Denervated muscle fibers, not yet reinnervated, usually discharge spontaneously, and so-called fibrillation potentials and positive waves are recorded. These are often signs of recent denervation. In the second phase of examination using weak voluntary muscle activation, motor unit potentials (MUPs) are studied regarding various shape parameters. Long-duration and high-amplitude MUPs reflect reinnervation and are typical of a neurogenic disease. Finally, in the third phase, the patient is asked for maximal contraction of the muscle. Normally, a large number of independently firing motor units create a "noise" signal. In cases of a neurogenic condition with loss of some motor units and reinnervation of others, the pattern during maximal contraction shows higher amplitudes of the peaks and overall a less dense signal. All these parameters can be quantitated.

In every patient with polio, at least some muscles show neurogenic changes on EMG, i.e., MUPs with long duration and high amplitudes. Such changes are often found even in muscles that are clinically

normal in strength and volume. By complete reinnervation, a full functional restoration can be achieved.

In some muscles denervation activity is seen, usually as fibrillation potentials. This is often due to denervation after recent loss of neurons as an effect of age. Sometimes, this finding may be due to local reasons, such as radiculopathy or nerve entrapment, unrelated to polio (not uncommon in nonpolio patients) or is due to unfavorable situations related to sequelae of polio. If the reason is due to local and definable causes, these should be treated if possible. If no obvious cause is found, the signs of denervation may indicate progressive neurodegeneration, which may lead to muscle symptoms.

MACRO-EMG

Macro-EMG is a relatively new method, described in detail elsewhere.*[7,19]* It is used to study the total electrical strength of a motor unit. The amplitude of the obtained signal depends on the number and size of muscle fibers in one motor unit. The recording electrode consists of a modified SFEMG electrode with the cannula insulated except for a 15-mm tip. The SFEMG recording surface is exposed 7.5 mm behind the tip. Recording is made on two channels using a two-channel EMG machine connected to a personal computer for analysis (Intersoft software). On one, the signal from the cannula (using a surface electrode as reference) is recorded and fed to an averager. On the other, the SFEMG recording is obtained between the small surface and the cannula. This signal serves as a trigger for the averaging process. Amplifier filters are set to 5 Hz to 10 kHz and to 500 Hz to 10 kHz for macro and SFEMG, respectively. The electrode is inserted into the voluntarily activated muscle, and a position is sought where an acceptable SFEMG potential is seen. At this moment, the averaging process starts and continues until a smooth baseline and a constant macro MUP is obtained on the "cannula" channel.

ELECTROMYOGRAPHIC FINDINGS

In a recent study*[18]* of 18 patients with two examinations 4 years apart, macro-EMG recordings from vastus lateralis muscle were used to quantify muscle changes. Muscle biopsies were taken in this muscle, and maximal isokinetic and isometric torque measurements of knee extension were performed. The results are briefly summarized as follows: At the initial evaluation, the macro MUP amplitudes were increased, compared to age-matched controls, by 10 times in the stable group and 16 times in the unstable group, an intergroup difference that is significant (p <0.01). There was no correlation between EMG findings and strength values during the initial examination. Four years later, the strength was unchanged in the stable group but decreased in the unstable, as described earlier, whereas the macro MUP amplitude had increased by 67% and 35% in the stable and unstable groups, respectively. This increase could not be explained by a change in fiber area, which on an average was unchanged.

In two other macro-EMG studies, no increase over time was demonstrated. In one of them,*[21]* this conclusion was based on the lack of correlation between macro-EMG size and time from acute polio, although the possibility of changes with time was not excluded. In the other study,*[16]* two examinations were performed 1 year apart with no consistent change in macro MUP.

GENERAL DISCUSSION

The dynamics of late changes after polio are summarized briefly.

FIBER SIZE

Muscle fiber hypertrophy seems to be a characteristic feature of the muscles in polio patients. The upper

limit for this compensatory factor is probably reached earlier than that for reinnervation. This is indicated by the fact that during the 4-year follow-up period, there was no further average increase in muscle fiber area in the studied group, whereas the size of the motor unit due to reinnervation continued to increase. Muscle fiber size depends on muscle activity, as already discussed, but also influences muscle strength. Thus, increased activity of motor units as a consequence of the reduction in number of motor units may be a stimulus for muscle fiber hypertrophy. In that way, muscle strength will be better maintained than it would have been without utilization of that compensatory process.

REINNERVATION

As seen from our macro-EMG studies, there is an on-going denervation/reinnervation years after the acute stage of polio. The motor units successively contain increasing numbers of muscle fibers, to a great extent reflecting the degree of reinnervation. Collateral sprouting is highly effective as a compensatory mechanism after denervation. In patients with acute polio more than 20 years ago, the motor unit size due to reinnervation is often 10-15 times the normal size.

There was no correlation between strength and Macro MUP amplitudes in our study.*[18]* Such a correlation was reported, however, in another study of 10 patients with L5 rhizopathy or history of polio. *[20]* Lack of these correlations, however, is not surprising. Strength depends on the varying combinations of the number of motor units, fiber size, and the number of muscle fibers. Furthermore, the contractile properties of reinnervating motor units may be abnormal, with less mechanical output for a given electrical signal.*[6]* Extramuscular factors, such as joints and connective tissue, may also play a functional role, influencing, for example, the degree of maximal muscle activation and force output. The disturbed neuromuscular transmission, typical of reinnervation, could influence the functional output, as earlier reported,*[21]* although this was not an important factor in our study.

DECOMPENSATION

Decompensation of the muscle changes in late polio occurs during life due to two phenomena. One is the reversion of the factors discussed earlier. With decreased daily activity and less training, decompensation related to muscle fiber size and oxidative metabolism will occur. The other factor is the continuous loss of motor neurons, as indicated by the increase in macro-EMG changes. This compensatory mechanism may already be utilized more or less completely. In such a situation, further neuronal loss leads to a functional impairment that is proportional to reduction in neurons.

The reinnervation is limited both by central and peripheral factors. The peripheral factors set the limit when a denervated motor unit is no longer overlapping with other motor units, i.e., when all muscle fibers within certain fascicles belong to one motor unit due to previous reinnervation cycles. Overlapping with another motor unit is a prerequisite for reinnervation.

The central factors are related to the status of the motor neuron. In post-polio subjects, the number of muscle fibers losing their innervation with the degeneration of each anterior horn cell is much larger than typically occurs with normal aging, where the motor units usually are only slightly increased in size. This implies a greater strain on reinnervation mechanisms in patients with earlier enlarged motor units who then have additional loss of neurons. Furthermore, the physiological aging process may be exaggerated due to increased demands on the remaining reduced motor neuron pool. For every movement involving weak muscles, a larger portion of the motor neuron pool is utilized to produce the necessary force. In addition, mechanical strains occurring with reduced muscle mass may damage the muscle at a myofibrillar level.

COMBINATION OF FACTORS

Although some statistical characteristics could be found to separate the stable and unstable groups, none of the measured morphological parameters, strength, or neurophysiological findings could be used to predict post-polio syndrome (as defined by Halstead and Rossi*[12]*) in the individual patient. Most EMG studies have failed to depict EMG changes that may be used to diagnose or predict post-polio syndrome. *[21]* Even in studies where functional tests concern individual muscles, the EMG changes may be similar in patients with unstable and stable muscle function.

Thus, muscle strength in post-polio patients reflects the dynamic changes in degeneration, compensation, and decompensation. The progressive enlargement of macro-EMG signals, as a compensatory response to loss of neurons, is only one factor in the dynamic change in muscular strength, since the change in strength is the combined effect of the number of available motor neurons, number and size of muscle fibers in each motor unit, neuromuscular transmission, and the mechanical properties of reinnervated muscle fibers. There are a number of possible combinations of denervation-reinnervation and strength that make interpretation of their relationship difficult:

1. If there is a loss of functioning motor units, not leading to axonal degeneration and therefore not reinnervation, the macro-EMG will not change while strength decreases. This stage of inexcitable neurons may be seen in the acute phase of polio but not later in life.
2. If reinnervation is successful, but the strength developed by each individual motor unit is decreased compared to normal motor units, macro MUP amplitude increases but strength will still be maintained. It has been demonstrated that reinnervated motor units in amyotrophic lateral sclerosis are weaker than expected from their electrical size as measured with macro-EMG,*[6]* and our patients may show a similar situation.
3. Finally, and probably most importantly, reinnervation may compensate for new denervation until a maximal capacity for reinnervation is reached. After this stage, additional loss of motor units cannot be compensated. A continued loss of motor units will then present clinically as a new or accelerating decrease in strength and activity level over time and may, as a result, also be combined with reduced stimulus to maintain the marked fiber hypertrophy.

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