Weakness in Patients With Hemiparesis

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Clinical and experimental results are reviewed concerning muscle weakness in patients with hemiparesis after a stroke. The discussion includes the important role that alterations in the physiology of motor units, notably changes in firing rates and muscle fiber atrophy, play in the manifestation of muscle weakness. This role is compared with the lesser role that spasticity (defined as hyperactive stretch reflexes) of the antagonist muscle group appears to play in determining the weakness of agonist muscles. The contribution of other factors that result in mechanical restraint of the agonist by the antagonist (e.g., passive mechanical properties and inappropriate cocontraction) is discussed relative to muscle weakness in patients with hemiparesis.

Normal Force Production

A review of the physiology of normal force production provides the necessary background to understand the possible mechanisms of muscle weakness in patients with hemiparesis. (See Brooks, 1986, and Evarts, Wise, & Bousfield, 1985, for a more detailed review.)

The force produced by a normal muscle contraction depends on the number and type of motor units recruited and the characteristics of that motor unit discharge. In motor control, the motor unit represents the smallest functional unit. A motor unit comprises the alpha-motoneuron, its axon, and the muscle fibers it innervates. (The term muscle unit may be used in reference to the muscle component of the motor unit.)

Muscle force or tension is increased when either the absolute number of active motor units is increased, or the firing rates of already-active motor units are increased, or both (see Burke, 1981; Henne-man & Mendell, 1981). Usually, both processes occur simultaneously. Motoneurons are organized in such a way that the excitability of the motoneuron is responsive to the required muscle unit force. Thus, an orderly recruitment pattern of motor units on the basis of Cerebrovascular accident, or stroke, is a common condition in patients seen by occupational therapists (Trombly & Scott, 1977; Walker, 1981). Initially, stroke results in hypotonia and areflexia. The motor behavior usually progresses to a stage of spastic hemiparesis, which may include (a) spasticity, characterized by hyperactive stretch reflexes, clonus, and the clasp-knife reflex; (b) alteration of cutaneous reflexes (e.g., Babinski sign); (c) disturbance of other sensory functions (i.e., vision, proprioception, pain, temperature); and (d) paresis. Paresis is a partial paralysis that is manifested by decreased muscle strength or weakness.

Clinically, muscle weakness has been recognized by patients and therapists as a limiting factor (Duncan & Badke, 1987) in the motor rehabilitation of patients after a stroke. Muscle weakness is reflected by the inability of patients with spastic hemiparesis to generate normal levels of muscle force. Deficits in “muscle strength” would reduce “the capacity of a muscle to produce the tension necessary for maintaining posture, initiating movement, or controlling movement during conditions of loading of the musculoskeletal system” (Smidt & Rogers, 1982, p. 1283). Earlier reviews (Chan, 1986; Chapman & Wiesendanger, 1982; Pierot-Deseilligny, 1983) have focused on the issue of spasticity and its role in the motor control of patients with spastic hemiparesis. This paper reviews physiological changes in the nervous system of patients with hemiparesis that may contribute to muscle weakness.
of their force-producing characteristics exists in movement. According to this principle, low-force-producing motor units are the first to be recruited, and as force requirements increase, higher force-producing motor units are recruited. For example, in the task of putting on a shirt, a certain number of motor units in the agonist would be active. If, however, resistance was encountered, as in trying to move the hand through a small armhole, more and larger units would be recruited to meet the increased force demands. As force requirements for a movement decrease, motor units are derecruited in the reverse order, with the higher force-producing units being the first to stop firing and the low-force-producing units the last to stop firing. The central nervous system appears to match the amount of muscle force produced with the demands of the task. In general, low-threshold motor units (those recruited first) have smaller motoneurons and axons and show less terminal branching than do high-threshold motor units.

Considering the full range of motor units innervating a given muscle, the motoneurons show a range of intrinsic properties and the muscle units show a range of histological and metabolic properties. The unique properties of both motoneuron and muscle components of motor units are adapted to the orderly recruitment of motor units. Low-threshold motor units, which develop less active force, are more resistant to fatigue than are high-threshold units. Motor units are classified as type FF, FR, or S, on the basis of the fatigue resistance and twitch tension of the motor units (Burke, 1981). Type FF refers to fast-contracting, fast-fatiguing motor units that can generate high-twitch tension. Such units are prevalent in muscles that subserve fine movements of the hands and face. Type FR refers to fast-contracting but fatigue-resistant motor units, and type S refers to fatigue-resistant, slow-contracting (meaning the twitch is long-lasting) motor units that generate low-twitch tensions. Type S motor units are most prevalent in postural muscles. The succession of recruited motor units, from type S to FR to FF, helps to provide for a smooth increase in the force development.

Besides changes in the absolute number of active motor units and the recruitment order of these units, muscle force is adjusted by changes in the firing rates of the active motor units. Each time the motoneuron fires, its muscle fibers contract, producing a single twitch contraction. As the motoneuron firing rate increases, the individual muscle twitches summate to produce more force output. This process continues until the motor unit's level of maximal force output is achieved. The relationship between the stimulus rate of the muscle nerve and the force production in the muscle (i.e., a sigmoid, or S-shaped, relationship [Rack & Westbury, 1969]) defines an optimal range of firing rates for motor units, over which an increase in the firing rate results in an increase in the force production of the motor unit. Below this optimal range, the decreased firing of the motor unit will generate unfused twitches resulting in a small proportion of the motor unit's force-generating capability. Above this optimal range, the increased firing of the motor unit cannot produce more tension because the twitches had fused previously at an earlier rate to produce the unit's maximal tension. As with the order of motor unit recruitment, modifications in the firing rates of motor units can provide fine gradations of force, as observed in movements of the fingers or eyes that are produced by muscles with a small number of motor units (Brooks, 1986). In these muscles that do not have a large number of motor units to recruit and derecruit, force development is graded more often by changes in the firing rates of the available motor units.

Physiological Changes in the Hemiparetic Motor System

Specific changes at the motoneuron or muscle level can decrease a person's ability to produce force, which will be observed, measured, and documented clinically as muscle weakness. These changes, summarized below, have been observed in stroke patients:

1. Motoneuron Changes:
   - Loss of motor units
   - Changes in recruitment order of motor units
   - Changes in firing rates of motor units

2. Nerve Changes:
   - Changes in peripheral nerve conduction

3. Muscle Changes:
   - Changes in morphological and contractile properties of motor units
   - Changes in mechanical properties of muscles

Reductions in the level of force generated by the agonist muscles (i.e., the prime movers) in patients with hemiparesis may result from direct changes (e.g., changes in the physiology of agonist motor units) or from indirect changes (e.g., an increased mechanical restraint, active or passive) that are imposed by antagonist muscles which oppose the action of agonists. In this section, we will review evidence from clinical studies as to how such physiological changes may contribute to the phenomenon of weakness in patients with hemiparesis.

Direct Changes of Agonist Motor Units

Decreased number of agonist motor units. The number of active motor units participating in small, controlled movements has been examined in patients...
with hemiparesis. McComas, Fawcett, Campbell, & Sica (1971) developed a method to estimate the number of functioning motor units in normal subjects. McComas, Sica, Upton, & Aquilera (1973) then used this method with hemiparetic subjects. Between the 2nd and 6th month after a stroke, the number of functioning motor units was reduced by approximately half. One explanation for this loss is that degeneration of the corticospinal tract after a stroke results in transynaptic changes in the motoneurons (Dietz, Ketelsen, Berger, & Quintern, 1986; McComas et al., 1973). Changes in motoneuron properties could reduce the overall activity of motor units as well as alter the function of their respective muscle unit components.

Denervation potentials (e.g., fibrillation and positive sharp waves) have been reported in hemiparetic limbs, indicating that some muscle fibers or motor units have lost connection with their motoneurons. Fibrillation potentials have been observed in about two thirds of patients with hemiplegia and are recorded more often in distal muscles (Goldkamp, 1967). This latter observation coincides with a possible role for the corticospinal tract in the denervation process, because this pathway projects more heavily onto motoneurons supplying distal limb muscles (Kuypers, 1981). Other electromyographic studies have confirmed the presence of a denervation process in patients with hemiparesis (Bhala, 1969; Cruz-Martinez, 1984; Johnson, Denny, & Kelley, 1975; Krueger & Waylonis, 1973; Segura & Sahgal, 1981; Spaans & Wilts, 1982). Alternatively, the appearance of denervation potentials in hemiparetic muscles could also be explained by chronic pressure being exerted on peripheral nerves (Chokroverty & Medina, 1978). However, pressure blocks would also result in a decrease in the motor conduction velocity (maximum) of peripheral nerves in patients with hemiparesis. Most studies have not found such a result (Goldkamp, 1967; McComas et al., 1973; Namba, Schuman, & Grob, 1971; Segura & Sahgal, 1981; Young & Mayer, 1982); however, a few have (Cruz-Martinez, 1984; Panin, Paul, Policoff, & Eson, 1967). Indirect evidence supports the theory that some degenerative changes of motoneurons may occur after a stroke; however, anatomical evidence for degenerative changes in motoneurons (e.g., cell shrinkage, loss of synapses) after a stroke has not yet been demonstrated in humans.

Changes in the properties of agonist motor units. Muscle atrophy is a common clinical finding in patients with hemiparesis. Evidence on fiber measurements in biopsied muscle supports the idea that the presumptive fast-contracting fibers belonging to high-threshold motor units (type FF or FR, higher force producing) are atrophied in patients with hemiparesis (Brooke & Engel, 1969; Dietz et al., 1986; Edstrom, Grimby, & Hannerz, 1973; Scelsi, Lotta, Lommi, Poggi, & Marchetti, 1984). Hypertrophy of presumptive slow-contracting muscle fibers belonging to low-threshold (type S, low-force-producing) motor units has also been reported (Edstrom, 1970). Such modifications of the morphological characteristics of muscle in general, and of muscle units in particular, could alter the force-producing capability of motor units.

Studies on the contractile properties of motor units in patients with hemiparesis have shown that the overall contraction time is prolonged in hemiparetic muscle (McComas et al., 1973; Visser, Oosterhoff, Hermans, Boon, & Zilvold, 1985). In particular, the mean twitch contraction time is increased in fast-contracting motor units (Young & Mayer, 1982). Furthermore, a unique class of motor units—slow-contracting and fatigable—not present in normal muscle, have been found in long-term hemiparetic muscle. Young and Mayer found, in general, that all motor units recorded from the paretic side of patients with hemiparesis were more fatigable, as judged by a fatigue index (i.e., the ratio of twitch tension before and after a repetitive stimulus train). In the clinic, the increased fatigability of the hemiparetic motor system would be reflected by a patient’s poor endurance in performing repetitive tasks or movements.

Disrupted recruitment order of agonist motor units. To date, there is conflicting evidence as to whether the recruitment order of motor units is disrupted in patients with hemiparesis. An early observation of abnormal recruitment order of motor units in patients with hemiparesis (Grimby, Hannerz, & Ranlund, 1972; Grimby & Hannerz, 1973) has not been confirmed (Rosenfalck & Andreasen, 1980). Such studies, which require the sampling of motor units across the full range of activation thresholds, have been limited by the recording technology. In the future, tungsten electrodes (Bigland-Ritchie, 1983), which offer selective recording of high- and low-threshold motor units in human muscle, may allow for a more definitive evaluation of the recruitment pattern of motor units in patients with hemiparesis.

Decreased agonist motor unit firing rates. If the overall firing rates of motor units are decreased in patients with hemiparesis, then the active motor units will generate less effective tension (Rack & Westbury, 1969). Consequently, for a patient to reach a given force level or to produce a particular movement, additional motor units would have to be recruited. This explanation would account for the increased levels of electromyographic activity per unit of force observed in elbow flexor muscles of paretic limbs, compared with contralateral nonparetic limbs in a subpopulation of patients with hemiparesis (Tang & Rymer, 1981). Similar results have been reported for other
muscle groups of upper and lower limbs (Visser & Aanen, 1981). Also, Freuden, Dietz, Wita, & Kapp (1973) showed a decreased firing rate in motor unit recordings from intrinsic hand muscles of patients with hemiparesis. A similar decrease was observed (Rosenfalck & Andreassen, 1980) in the mean firing rate of motor units recorded from the tibialis anterior muscle of patients with hemiparesis. However, in another study (Dietz et al., 1986), no difference was found between the mean firing rates of gastrocnemius motor units recorded from affected and unaffected sides of patients with hemiparesis. More studies are needed to elucidate this important point, because an overall decrease in the firing rates of motor units in paretic muscle would directly contribute to muscle weakness (Rosenfalck & Andreassen; Tang & Rymer).

Peripheral feedback may help to maintain a constant force level in the modulation of motor unit firing frequencies. When a patient with hemiparesis tries to maintain a constant force level, the force record shows irregularities not observed in normal subjects (Rosenfalck & Andreassen, 1980). Difficulty in maintaining a constant force level could be important for the performance of finely controlled movements such as writing or eating. In patients with hemiparesis, the inability to maintain a constant force level has been related to a problem in the modulation of motor unit firing frequencies. Rosenfalck & Andreassen proposed that this deficit may indicate a disturbance in the usage of peripheral feedback for the control of hemiparetic movement. Overall, changes observed in hemiparetic muscles support the idea that the agonist motor units themselves have been altered. An understanding of the specific mechanisms responsible for these changes will require further research.

**Passive or Active Restraint of Agonist Activation**

Any alteration in the motor system that constrains, either by a passive or active process, the contraction of the agonist limits the patient's ability to produce efficient voluntary movements. This concept was recognized in the treatment of patients with spastic hemiparesis by Bobath (1978), who suggested that weakness in the agonist results from spasticity (defined by hyperactive stretch reflexes) of the antagonist muscle. Considering the possible link between agonist muscle dysfunction and antagonist muscle spasticity, Bobath proposed that the normalization of muscle tone is a priority for the rehabilitation of these individuals. The idea is that a reduction or elimination of the spasticity would unmask the voluntary capabilities of the patient for movement.

Recent evidence in patients with hemiparesis, however, does not support a major role for the spasticity of the antagonist in limiting voluntary activation of the agonist. Bohannon, Larkin, Smith, & Horton (1987) reported that a measure of decreased muscle strength in the upper limb musculature of patients with hemiparesis correlated with the level of spasticity assessed in these same muscles, but not with the level of spasticity in the antagonists. The covariation between the level of spasticity and the measure of strength for the agonist muscle group would not be expected if the spasticity of the antagonist muscles were responsible for the observed decrease in agonist muscle strength. However, in Bohannon et al. (1987), the low incidence of spasticity in the antagonist muscle group across subjects does not provide a definitive test of its role in limiting voluntary activation of the agonist.

**Passive mechanical restraint of agonist activation.** Studies that examine the increased resistance of spastic-paretic muscle to externally imposed stretch provide a means to assess contributions from reflex and mechanical (structural) factors. In a study by Lee, Broughton, & Rymer (1987), four patients with spastic hemiparesis who demonstrated increased resistance in elbow flexor muscles to externally imposed stretch (i.e., spasticity) on the paretic side as compared with the contralateral side also showed low electromyographic activity in the paretic muscles (i.e., flexors and extensors). Therefore, the measured resistance of the limb to passive stretch was not due to enhanced stretch reflexes. Lee et al. concluded that the increased resistance to stretch was due to altered mechanical properties of the elbow musculature in these patients with hemiparesis.

Other studies (Dietz, Quintern, & Berger, 1981; Hufschmidt & Mauritz, 1985) supported the importance of mechanical properties over stretch reflex enhancement in limiting voluntary activation of muscles. During the swing phase of gait, Dietz et al. (1981) found that difficulty in dorsiflexing the spastic-paretic foot was accompanied by a significant increase in the electromyographic activity in the tibialis anterior muscle, as compared with normal subjects. Because no coactivation of the triceps surae muscles (antagonists) was observed, Dietz et al. (1981) proposed that the excessive activation of the tibialis anterior muscle was necessary to overcome a resistance of nonreflexive origin (e.g., some alteration in the passive mechanical properties of the triceps surae muscle group). Likewise, results of Hufschmidt and Mauritz, in characterizing the passive mechanical properties of spastic ankle muscles in patients with hemiparesis with long-term spasticity, supported the hypothesis that structural changes occur in spastic muscle.

**Active restraint of agonist activation.** Inappropriate activation of antagonist muscles during a movement that requires selective activation in the agonist
strength measurement testing to monitor motor status. To set up such a program, baseline data must be obtained to assess the patient's initial level of muscle dysfunction in patients with spastic hemiparesis and considered as potential predictors of functional independence. For example, Bohannon (1986) reported that improvement in muscle strength in patients with hemiparesis was related to clinical improvement. Andrews, Brocklehurst, Richards, & Laycock (1981) reported that for patients 3 months after a stroke, improvements in muscle strength coincided with improvements in the performance of activities of daily living. Bohannon & Smith (1987) found that their strength measures for paretic muscle groups reflected an improvement in strength (estimated at 20%) after 1 month of a rehabilitative program. Presumably, this improvement in strength was the net result of any spontaneous central nervous system recovery (e.g., after decreased edema) or motor recovery due to the therapeutic exercise regime. More studies on patients with hemiparesis are required to assess what role muscle strength training should play in rehabilitating patients after a stroke.

The assessment of true muscle strength can be confounded by severe spasticity, abnormal passive mechanical properties of muscles, or abnormal muscle activation patterns (e.g., cocontraction). However, if the ability to produce an efficient muscle contraction is the basis for any movement (Duncan & Badke, 1987), then as therapists, we must rethink the potential merit of strengthening exercises for our patients with hemiparesis. We must consider conditions in which paretic muscle can be strengthened to help rather than to hurt the patient. Clinical studies can help us with this consideration.

Inefficent muscle contractions reduce force output in patients with hemiparesis (Bourbonnais, Vanden Noven, Carey, & Rymer, 1987, in press), thereby affecting all movements and limiting functional performance. Overall, the maximal force produced by a given muscle group on the paretic side is reduced in patients with hemiparesis (Rosenfalck & Andrasson, 1980; Visser et al., 1985). Bohannon (1987) examined strength deficits (i.e., ratio of paretic to nonparetic muscle strength) in patients with hemiparesis and found that parameters such as gender, weight, and age, which correlated with strength measures for normal or nonparetic muscles, did not correlate with strength measures for paretic muscles. The impact of such disrupted relationships on the functioning of the hemiparetic motor system has not been determined.

In summary, studies to date do not support a major role for the spasticity of the antagonist in limiting the production of agonist muscle force. Agonist muscle dysfunction in patients with spastic hemiparesis may be due more to restraint factors imposed by the passive mechanical properties of antagonist muscles and surrounding tissues and/or by inappropriate, ungraded cocontractions of antagonists may limit agonist force production.

In clinical practice, the terms 'muscle weakness' and 'muscle strength' are often used interchangeably. However, muscle weakness usually refers to less-than-normal muscle strength. If so, then a patient with hemiparesis who demonstrates weakness of the upper limb musculature could benefit from a regimen of strengthening exercises. To set up such a program, baseline data must be obtained to assess the patient's initial level of muscle strength. However, strength training and strength measurement testing to monitor motor status and recovery after a stroke are controversial issues.

Clinical Significance of Muscle Weakness in Hemiparesis

It is questioned whether the terms muscle weakness and muscle strength apply to people with spasticity. Muscle weakness usually refers to less-than-normal muscle strength. If so, then a patient with hemiparesis who demonstrates weakness of the upper limb musculature could benefit from a regimen of strengthening exercises. To set up such a program, baseline data must be obtained to assess the patient's initial level of muscle strength. However, strength training and strength measurement testing to monitor motor status and recovery after a stroke are controversial issues.
Conclusion

Physiological changes in the motor system of adult patients with hemiparesis, which might account, in part, for muscle weakness, have been reviewed. Such changes are often interrelated, and their effects summate, along with other factors, to contribute to the overall severity of the motor disturbance. For example, a patient's ability to produce muscle force can be limited by specific changes at the motoneuron or muscle level or by changes in the organization of the nervous system. For a more in-depth discussion of the motor control of stroke patients as well as for examples of how our present knowledge of motor control, on the basis of research findings, can be melded with the clinical treatment approach, see Duncan and Badke (1987).

Further clinical research is required to sort out the relative contributions of the above-mentioned factors to the phenomenon of muscle weakness in patients with hemiparesis. Both occupational and physical therapists have considerable expertise to contribute to the study of motor control problems. This is exemplified by the growing number of therapist-researchers contributing to our knowledge base in normal and abnormal motor control.

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References


