Motor unit behavior in Parkinson’s disease.

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This article reviews the literature related to motor unit behavior in Parkinson’s disease (PD). The focus is on bradykinesia, or slowed movement. There is sparse literature on muscular performance in PD, as PD is regarded as a disease of higher motor centers. Nevertheless, a decrease in muscle activation has been demonstrated, and motor unit behavior is altered so that (1) the discharge patterns of motor units are irregular and intermittent, (2) a greater number of motor units are recruited at low thresholds as compared with the findings for age-matched control subjects, and (3) antagonist muscles are abnormally coactivated. Possible reasons for these changes include imbalances in excitatory and inhibitory inputs to motor neurons, adaptations in motor neurons secondary to disuse, or deviations in the normal aging process. For the physical therapy of persons with PD, we propose a greater emphasis on strength-training exercises.

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Parkinson’s disease (PD) is a degenerative condition of the central nervous system that is clinically classified under the rubric of movement disorders. The disease affects mainly older persons and is sometimes thought of as accelerated aging because the symptoms and pathology associated with PD closely resemble those of normal aging.[1] Experimenters study PD for two reasons. First, medical research seeks to find a cure for PD, which is a crippling and progressive disease that worsens with time and as the efficacy of pharmacological treatment wanes. Second, PD is studied for the purpose of understanding the functions of the basal ganglia, as PD is considered to be the prototypical disease of these nuclei.

The main feature of PD is decreased movement. Both reflexive and voluntary movements are diminished, and are usually described as “negative” features of PD, based on the belief that they result directly from damage to motor centers. Tremor and rigidity constitute the “positive” symptoms of PD because they are thought to be caused by release, or disinhibition, of different (intact) motor centers that normally receive input from the damaged areas. This review focuses on the negative features of PD, or more generally, bradykinesia (slowed movement).

This article describes what is known about the behavior of motor units (MUs) in PD. An MU comprises a single motor neuron and the muscle fibers it innervates, and can be studied using intramuscular electromyography (EMG). Because motor neurons are the last cells to receive central commands from spinal and supraspinal motor centers, their behavior provides a window into central motor processes that are otherwise inaccessible in studies involving human subjects. By examining intramuscular EMG records, it is also possible to determine whether MUs are themselves affected by the disease process. Motor units are known to alter their properties as a result of damage to the central nervous system, even if the spinal cord is untouched.[1-4] Thus, even though PD is typically considered to influence movement through cortical circuits,[5] it is possible that cells distal to the cortex are affected by the disease.

This article begins with a general description of the motor symptoms caused by PD, with a focus on bradykinesia. The proposed mechanisms for bradykinesia are described, followed by a review of the literature describing MU activity in persons with PD. Some recent data collected in our laboratory are included in this section. Taken together, the observations suggest that there is normal recruitment of MUs in PD, but that the overall population of MUs in individuals with PD includes more lower-threshold MUs compared with those of healthy age-matched control subjects. Motor unit discharge patterns and the degree of antagonist coactivation also differ between subjects with PD and healthy subjects. We discuss the implications of these findings to physical therapy in the last section.

Overview of Parkinson’s Disease

Parkinson’s disease was described originally as a “shaking palsy,” with tremor at rest considered as the pathognomonic sign. It is now clear, however, that other motor problems, such as rigidity, bradykinesia, and postural instability are often more prominent features of the disease.[6,7] The manifestations of PD can differ widely among individuals with the disease. In addition to varying degrees of bradykinesia, rigidity, and tremor, individuals also differ in the degree to which sensory, autonomic, and cognitive functions are affected by the disease.[8] Changes in these non-motor systems could also contribute to the movement abnormalities in PD. Indeed, several authors[9-11] have suggested recently that motor disturbances in PD are sensorimotor in origin.

Tremor and Rigidity

Before describing the characteristics of bradykinesia, we will briefly describe the other prominent symptoms of PD -
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tremor and rigidity - as these symptoms are usually present to some extent in individuals with PD. The tremor of PD is distal and is typically considered to be a “resting tremor,” although it can also occur during active movement.[12] The frequency of the tremor is low (4-6 Hz) as compared with that of physiological tremor, and increases to 8 to 12 Hz with movement.[8] Surface EMG recordings have revealed that agonist and antagonist muscles are activated alternately during the tremor of PD.[13]

The mechanisms underlying parkinsonian tremor are poorly understood. Parkinsonian tremor does not appear to be related to afferent feedback or hyperactivity in the stretch reflex. This is demonstrated by the failure of deafferentation to abolish tremor and by the absence of an increase in muscle afferent activity preceding single tremor beats.[12] One suggestion is that tremor is generated by cells within the ventral intermediate nucleus that receive abnormal inhibitory inputs from the globus pallidus in PD.[14] Surgical lesions of the ventral intermediate nucleus in the thalamus abolish resting tremor.[15]

Patients with PD also commonly have muscular stiffness, or rigidity. Clinicians identify the rigidity of PD as “lead pipe” because of the constant resistance of the muscles to passive displacement throughout the entire range of joint motion. The rigidity of PD is also sometimes characterized as “cogwheel,” in reference to the repetitive (5-6 Hz) stops that can occur as the limb is passively moved.[15] Cogwheel rigidity is believed to be caused by an underlying tremor that is usually masked by rigidity, but uncovered when the muscle is stretched.

As with tremor, the mechanisms that underlie rigidity are unclear. Three possibilities are currently being considered. First, there is evidence that tile gm of long-latency stretch responses is abnormally high in parkinsonian rigidity.[16] These responses are elicited by muscle stretch and occur later than the segmental stretch reflex. The circuitry underlying the long-latency responses is the subject of debate. Some authors hold that afferent signals reach the cortex to activate corticospinal neurons,[17] whereas others believe the reflexes are spinal, with the longer latency attributable to delayed afferent input.[18] Irrespective of the mechanism underlying these responses, if they are increased in gain, the expected result would be an overall increase in muscle activation, and hence rigidity. A second suggestion to explain rigidity is that the activity of inhibitory interneurons in the spinal cord is decreased, which would increase the excitability of motor neurons.[19] A third proposed mechanism for rigidity is that the elastic properties of muscle are changed in PD secondary to disuse, causing the muscle to stiffen independent of neural activation.[20] This view is supported by the observation in persons with PD that range of motion can be limited and muscles can be stiff in the absence of any EMG signal.[20,21]

Bradykinesia

Bradykinesia is the hallmark and most disabling symptom of PD. Early in the disease, the most notable manifestation of bradykinesia is difficulty with walking, speaking, or getting into and out of chairs.[22] Individuals might fail to swing an arm during walking, or they may be lacking in facial expression.[22-24] Later, the bradykinesia affects all movements and, at its worst, can result in a complete inability to move. Patients require intense concentration to overcome the apparent inertia of the limbs that exists for even the simplest motor tasks. Movement initiation is particularly impaired when unnatural or novel movements are attempted,[25] or when combining several movements concurrently.[26,27]

The causes of bradykinesia are not known. The most popular view is that cortical motor centers are inadequately activated by excitatory circuits passing through the basal ganglia.[28,29] As a result, motor-neuron pools are not provided with adequate facilitation, and movements are small and weak.[28,29] The implication of this view is that cells in the motor cortex and spinal cord are functioning normally.

Support for the notion that bradykinesia is caused by insufficient excitation of the motor cortex derives partly from reports that patients with PD will move normally under some extreme circumstances.[30] “Kinesia paradoxica” was described in the classic papers on PD and refers to normal movement by otherwise akinetic patients in certain situations. For example, when a building was on fire or when a ball was thrown to them, some patients were able to move with apparently normal speed.[30] If bradykinesia is caused by inadequate activation of otherwise normal cortical and subcortical centers, normal movement would be expected when these centers were activated by other routes not involving the basal ganglia.

Actually, kinesia paradoxica is poorly documented and it is unclear whether “normal” movements have really been produced by persons with PD.[31] Although there is some recent evidence that speech movements can be normal in some contexts,[25] experimenters have generally not been able to reproduce kinesia paradoxica. Even with medication, or when movement cues are changed, bradykinesia seems to persist to some extent in patients with PD.[32-35] Thus, the phenomenon of kinesia paradoxica alone is insufficient evidence of normal motor centers in the cortex and spinal cord of individuals with
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PD.

Whether rigidity contributes to bradykinesia is unclear. We will present data that demonstrate increased coactivation during isometric contractions in subjects with PD as compared with healthy older subjects. Other investigators,[36-39] however, have used surface EMG to measure antagonist activity during dynamic movements and have reported no coactivation in subjects with parkinsonian bradykinesia. Also, rigidity and bradykinesia have been reported to occur independently,[8] suggesting that rigidity does not always cause bradykinesia. One resolution to these discrepancies is that antagonist muscles are activated abnormally in PD only under some conditions; rigidity “at rest” may differ from rigidity that occurs during dynamic or isometric contractions.

Muscle and Motor Unit

Activation Patterns

Associated With Bradykinesia

Hallet and Khoshbin[35] have described the EMG patterns of muscle activity in persons with parkinsonian bradykinesia. Figure 1 shows surface EMG records of a healthy 83-year-old subject (traces A-C) and a patient with PD (traces D-F) attempting to move as quickly as possible. The temporal patterns of muscle activity were similar in both individuals and exhibit a triphasic EMG pattern (three bursts occurring in the order of agonist-antagonist-agonist). Thus, the patients with PD seemed to select the appropriate muscle groups and activation patterns to perform a simple movement. The EMG patterns in the muscles of the patient with PD, however, differed from those of the healthy subject in that the bursts of EMG activity in the agonist muscle did not increase in magnitude for the larger amplitude movements (compare traces A and C with traces D and F). Hallet and Khoshbin interpreted their data as indicating that patients with PD are unable to sufficiently “energize” agonist muscles during movements made as quickly as possible. This view was also put forth in 1978 by Wiesendanger, who observed that the surface EMG signal was less synchronized and more temporally dispersed in subjects with PD.[40] An apparent compensation for the decreased muscular activation was to evoke more “cycles” of activity to complete movements. This pattern is illustrated clearly in Figure 1, which shows that the triphasic EMG pattern occurred once in the healthy subject but was repeated several times in the patient with PD.

For a given electrode arrangement, the size of a surface EMG signal depends on the number of MUs recruited as well as the discharge rate and size of these MUs. Therefore, the underlying cause for the diminished surface EMG signal can be better understood by examining the behavior of single MUs. A decreased number of MUs, a reduction in the rate at which each MU discharges action potentials, and a failure to recruit large MUs are three possible factors that could contribute to a decrease in the magnitude of the surface EMG signal. Large MUs are believed to be necessary to make fast movements[41,42] and might be unavailable to patients with PD, given that fast-twitch muscle fibers appear to atrophy selectively in the disease.[43]

To record the EMG signal from single MUs, either needle or fine-wire electrodes are inserted directly into the muscle belly.[44] When this technique is combined with force recordings of an isometrically contracting muscle, it is possible to determine the force at which an MU is recruited and its behavior over the course of a contraction. Averaging techniques can also be used to estimate the contribution of the single MU to the overall force.[45]

There are limitations to using intramuscular electrodes to monitor activity in single MUs. Typically, subjects must perform contractions that are isometric and have a low force. The reason for these restrictions is that, as muscle fibers shorten during anisometric or high-force contractions, the inserted electrode can move relative to the active muscle fibers. The movement alters the geometrical relationship between the recording surface(s) of the electrode and the MU, resulting in a change in the shape or amplitude of the recorded action potential. Because these characteristics of single MU records are used to identify individual MUs, it is difficult to isolate a single MU when the shape of the action potential changes. Another reason that high-force contractions cannot be studied is that an increase in contractile force involves the recruitment of more MUs. As more MU action potentials are recorded, the signal from the MU of interest is obscured in the interference pattern and is difficult to isolate. Because of these limitations, MUs sampled with intramuscular electrodes tend to be those recruited at lower forces.

In a typical MU experiment, subjects are asked to maintain low-force contractions over several minutes. Their ability to control discharge rate, recruit new units, or maintain a constant force is then assessed. Several authors[46-49] have examined MU behavior in persons with PD using these techniques. Their reports are mostly descriptive, but nevertheless reveal aberrant patterns of discharge in MUs. For example, MUs are only activated after long delays and often stop discharging for several seconds as the subjects maintain low-force contractions.[46,47]

Several authors[46,48,49] have also demonstrated that the
discharge rate of single MUs is highly variable in PD. In these experiments, subjects were asked to maintain either a constant MU discharge rate or a constant force and were unable to do so. Figure 2 shows some results, in the form of instantaneous discharge rates (left panels) and joint-interval histograms (right panels), for single MUs. The graph of instantaneous discharge rate reveals a steady rate in a healthy subject (panel A), but a highly variable rate in a subject with PD (panel B). The joint-interval histogram, in which the time between consecutive MU action potentials is plotted, provides another method of examining the regularity of MU discharge patterns. Again, a comparison between the joint-interval histograms (panels C and D) reveals more variability in the discharge pattern of MUs in the subject with PD than in the healthy subject.

The lowest constant rate of MU discharge in PD has been described as abnormally low (3-5 Hz) in subjects who were able to learn to maintain a regular rate in some MUs.[47,48] Petajan and Jarcho[46] have reported that this rate could not be raised even with increased effort. It is now clear, however, that healthy subjects can also maintain such low discharge rates.[50] It could be that a larger proportion of MUs are activated at these low discharge rates in persons with PD than in healthy subjects. Thus, in experiments that require activation of single, low-threshold MUs, a larger fraction of those recorded have low discharge rates. These low rates are related to the lower tremor frequencies (4-6 Hz) in patients with PD,[47,51] which is to be expected given that MUs discharging at the lowest (unfused) rates probably provide the greatest contribution to tremor.[52]

To date, patterns of MU recruitment have not been examined quantitatively in subjects with PD. According to the size principle, MUs are recruited in a fixed order that proceeds from slower MUs (tubes S) to faster MUs (tubes FR, FL, and FF).[53,54] This recruitment order can be evaluated by examining the relationship between the force at which a single MU is activated and the force exerted by that single MU, because slower MUs generally produce smaller forces than faster MUs.[55] Thus, MUs that exert smaller forces are normally recruited when a muscle contracts at low forces, and larger MUs are recruited as the force increases. When a muscle is activated to make any movement, whether the movement is fast or slow, voluntary or involuntary, recruitment should generally follow an orderly progression according to the magnitude of the force exerted by the MU.[54]

Only one study has addressed the issue of MU recruitment in patients with PD. Young and Shahani[12] examined whether smaller MUs were consistently activated before larger MUs within single bursts of EMG activity produced during resting tremor. They derived MU size from the amplitude of the MU action potential and observed that smaller action potentials always preceded larger action potentials in subjects with PD. These data are suggestive of recruitment by MU size, but cannot be taken alone as evidence for normal recruitment for two reasons. First, the magnitude of the recorded action potentials could have been influenced by the proximity of the recording electrode to the recorded MUs (and not the size of the MU). Second, only involuntary movements were examined, and it is possible that recruitment patterns differ for voluntary movements.

We have recently begun experiments to evaluate patterns of recruitment during voluntary contractions in subjects with PD. To do this, we have adopted the methods of Milner-Brown et al.[45] in which the force contribution of single MUs is estimated from the magnitude of the spike-triggered average force (see the article by Clamann[56] in this special series). Our records are from the first dorsal interosseous muscle (FDI), which abducts the index finger around the metacarpophalangeal joint, and the second palmar interosseous muscle (SPI), which is the antagonist muscle to the FDI. Records from the SPI allow us to address the possibility that antagonist muscles are coactivated in PD. Coactivation would result in a decrease in the measured recruitment threshold and spike-triggered average force of MUs in the FDI.

Preliminary results from these experiments are depicted in Figure 3, which shows recruitment patterns in subjects with PD, healthy elderly subjects, and healthy young subjects. Single MUs from the FDI are plotted according to their spike-triggered average force and recruitment force. For this comparison, we defined recruitment force as the average force at which each MU maintained the slowest steady discharge rate. The spike-triggered average force provides an index of MU size. A comparison among the three groups revealed no differences in overall patterns of recruitment; MUs with smaller forces had lower recruitment thresholds than those that produced larger forces. The PD and healthy young groups differed from the healthy elderly group, however, in that fewer MUs were recorded at lower forces (<1% maximal voluntary contraction [MVC]) in the healthy elderly subjects. The reason for this finding appears to be an age-related reorganization of MUs that results in fewer but larger MUs innervating a muscle.[57]

Another observation from these early experiments is depicted in Figure 4, which shows the patterns of MU activity in the SPI and FDI during isometric contractions of the FDI. The subjects were asked either to slowly increase the muscle force over 2 seconds or to move as quickly as possible to a target that was 20% of their MVC force. The top panels show the records from a healthy older subject, and the lower panels show those taken from a typical...
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subject with PD. During both the graded contraction (records A and B) and the quick contraction (records C and D), both subjects recruited MUs in the FDI. In the healthy subject, the SPI was silent for both types of contractions, except for a small amount of activity as the subject released the graded contraction. In contrast, the antagonist muscle in the subject with PD was active for both the graded and quick contractions. Often the antagonist muscle appeared to be activated in bursts of approximately 6 Hz during the dynamic contractions. Low-frequency bursts of activity in agonist muscles has been described previously in subjects with PD and attributed to long-term synchronization of MUs - even in cases of no overt tremor.[12,46] It is unclear why such activity would be present in antagonist muscles. More subjects need to be examined to determine whether coactivation occurs in all patients with bradykinesia.

The observation that coactivation was greater in PD contradicts previous reports that antagonist muscles were not activated during movements in PD.[36-39] Three possible factors might explain the discrepancy. First, coactivity may not have been detected because surface electrodes were used in previous experiments. Surface electrodes are further away from muscles than intramuscular electrodes, and are therefore less likely to pick up small amounts of activity in the muscles. Second, coactivation may only occur in distal muscles, and not in the more proximal muscles studied in these earlier experiments. Third, antagonist muscles may only be coactivated during isometric contractions in PD, and not during the dynamic contractions that were previously examined.

Taken together, these data suggest that MU behavior is altered in PD. The three main differences in MU behavior in persons with PD as compared with healthy aged subjects were (1) inconsistent discharge rates, (2) activation of more MUs at low forces of contraction, and (3) increased coactivation. The first two of these changes might contribute to the decreased magnitude of surface EMG activity that has been observed in persons with bradykinesia (Fig. 1). Overall, there has been little work in this area, which probably reflects the view that the motor deficits in PD are related to abnormal processing at cortical levels. Whether the changes in MU activity reflect altered descending input, or actual changes in the physiological properties of MUs, requires further testing.

Mechanisms for Abnormal Motor Unit Behavior

Why should PD produce changes in MU behavior? One possibility is that the described changes in MU activity are caused by abnormalities in the descending commands sent to motor neurons. These abnormalities might originate in the motor cortex or in other supraspinal centers receiving input from the basal ganglia. For example, it has been suggested that a route through the nucleus gigantocellularis plays a role in altering spinal cord neurons in PD.[19] This nucleus receives inputs from the substantia nigra pars compacta,[57] which is damaged in PD, and projects to the spinal cord. Delwaide et al[19] suggested that reticulo-spinal pathways originating in the nucleus gigantocellularis were disinhibited in PD, resulting in abnormal descending influences on spinal cord interneurons. Unbalanced influences on interneurons could change the gain of circuitry mediating reciprocal activation or alter the tonic state of motor neurons, and thus the ability to maintain constant discharge.

Motor neurons may also be affected directly by neuronal degeneration at sites other than the basal ganglia. Parkinson's disease attacks not only the basal ganglia cells in the substantia nigra, it also causes cells to degenerate in the locus coeruleus, thalamus, brain stem, autonomic nuclei, and spinal cord.[58-60] The spinal cord also receives dopaminergic projections from the thalamus and hypothalamus.[61] Given that motor neurons receive numerous inputs from descending brain-stem and cortical pathways as well as from propriospinal and sensory afferents, damage to many of these areas in PD could disturb the normal balance of excitatory and inhibitory influences onto motor neurons. The result of such an imbalance would be a change in the function of motor neurons. If, for example, motor neurons receive more inhibition than facilitation, activation by descending systems would need to be increased to bring these cells to threshold.

There is evidence to suggest that such imbalances occur in spinal cord neurons. Delwaide et al[19] have demonstrated that activity in the Ib interneuron is decreased in patients with rigidity secondary to PD. This interneuron is excited by Ib primary afferents (from Golgi tendon organs) and inhibits homonymous motor neurons (see review by McCrea[62]). It also receives inputs from other afferent types and descending tracts. Delwaide et al demonstrated that reflexive inhibition of the soleus muscle H-reflex was decreased in PD when the gastrocnemius medialis nerve was stimulated. Because this inhibition is believed to be mediated by the Ib interneuron, these results were interpreted as evidence that the Ib inhibitory interneuron functions abnormally in PD, perhaps due to changes in its activation by long descending pathways. Decreased Ib inhibition is expected to result in increased muscle stiffness during active movement or when a muscle is stretched.[63]
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Other researchers,[64-66] using similar techniques, have demonstrated increased Ia reciprocal inhibition in patients with PD, both with and without rigidity. Reciprocal inhibition refers to the inhibition of antagonist muscles that occurs when an agonist muscle is excited by a stretch.[62] A disynaptic circuit, utilizing the Ia inhibitory interneuron, mediates the response. Increased reciprocal inhibition could conceivably function in bradykinesia by decreasing the excitability of motor neurons during active movements. Increased Ia inhibition could also contribute to increased muscle stiffness in persons with PD, as experiments with animals have shown that reciprocal inhibition can reduce the response of a limb to a perturbation.[67,68]

These findings suggest that spinal cord circuits behave abnormally in PD, and could alter motor-neuron behavior. These changes most likely influence active movements given that, at rest, motor-neuron excitability is unchanged. Experiments utilizing the H-reflex to examine motor neuron pools in the relaxed muscles of persons with PD have clearly demonstrated no differences in the magnitude of this reflex.[69,70] Because the H-reflex represents the number of motor neurons activated by the monosynaptic reflex, the H-reflex would be increased if motor neurons were tonically facilitated. Thus, the effect of increased or decreased inhibitory interneurons might only be realized when those neurons are activated during movement.

Another explanation for the changes that have been observed in MUs is that they occur secondary to disuse. After movements have been slowed by disease, adaptations could occur in either motor-neuron pools or muscle fibers. Both fast- and slow-twitch muscle types are affected by disuse, although slow fiber types appear to be more susceptible to atrophy.[71-73] Disuse has also been shown to increase the proportion of MUs recorded at high recruitment thresholds and to decrease the maximal firing rates of MUs.[74] Except for weakness, the changes resulting from disuse differ from those described for PD, suggesting that disuse is not an important factor altering MU behavior in PD.

The final possibility regarding the changes in MU behavior described in this article is that they reflect variations on the process of cell death and reinnervation that occur in normal aging. As mentioned earlier, PD is often thought of as accelerated aging, given that bradykinesia is a prominent feature of movement in aging and that the amount of dopamine and the number of dopamine receptors progressively decline with aging.[69,75-77] In normal aging, motor neurons - particularly the fast types - appear to die.[78] Some of the muscle fibers left denervated by this neuronal loss can be reinnervated by surviving motor neurons. The functional result of this process is an increase in the force contribution of MUs and an impaired ability to maintain a constant force contraction.[79] It may be that cell death occurs in PD, but without the normal reinnervation. Alternatively, PD may represent a process that occurs normally at much older ages (after age 80 years).[80] As aging progresses, it is thought that neurons that once expanded in response to neuronal death begin to shrink. Either of these scenarios might explain the observation that subjects with PD both recruited small MUs at lower forces and were weaker than age-matched control subjects.

Implications for Physical Therapy

The goal of physical therapy intervention for PD is to maximize a patient’s ability to function independently. For example, therapy should focus on preventing musculoskeletal deformities and on maintaining the ability to balance, walk, and perform daily activities for as long as possible.[81-83] Ideally, therapy would delay or minimize the need for drug treatment, as long-term levodopa use can produce unwanted dyskinesias.[84,85] To these ends, we believe that an argument can be made for a program of muscle strengthening for persons with PD. The major deficits described in this article - weakness, coactivation, and a decreased EMG signal - contribute to the diminished function of persons with PD and are amenable to change through strength training. A program of resistive exercise aimed at strengthening the muscles might also prevent the aggravation of symptoms secondary to disuse atrophy.[73]

To date, physical therapy approaches to PD have not emphasized strengthening. Instead, therapy has focused on improving motor control and preventing unwanted movements. For example, a treatment program recommended recently for patients with PD begins with relaxation, passive muscle stretching, and positioning.[82,86] It is thought that these treatments will prevent excess muscle activity and decreased range of motion from contributing to abnormal movement.[87] Treatment then progresses to exercises for active range of motion, postural alignment, balance, and gait. Other recommended approaches include swteching,[83] light marching to music,[24,83] moving to sensory triggers,[81,87,88] electrical stimulation,[81] and biofeedback.[81] Weakness is not considered to be a problem that needs to be addressed by physical therapists, even though it is a widely recognized sequela of PD.[39,89,90]

The few controlled trials of physical therapy procedures have produced equivocal results. One study[91] showed some improvement in symptoms (eg, tremor), coordination, and strength following 12 weeks of karate...
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training or stretching. Another study[83] showed that movement therapies such as marching to music or light exercise improved gait, but not balance or posture. Whether neuromuscular therapies such as neurodevelopmental treatment and proprioceptive neuromuscular facilitation benefit persons with PD has not been shown conclusively. In one study, these treatments had no effect on the movement of subjects with PD.[92] whereas another study demonstrated that these methods combined with movement training and flexibility exercises improved the movement of two subjects with advanced PD.[82]

It is not clear why resistive exercises have not been attempted with persons with PD. The literature generally suggests that resistive training is beneficial to other patient populations in which the muscles have been denervated. For example, persons with post-polio syndrome have been trained successfully using weight training, without any long-term deficits.[93,94] Resistive training also benefits elderly persons,95 who also have partially denervated muscles.[78,96] Strength training could complement more traditional treatment approaches to PD. Exercise has been shown to be associated with decreased reaction time in elderly individuals.[97,98] Thus, it is possible that strength training could improve akinetic and bradykinetic symptoms. Programs of strength training have also been shown to decrease coactivation in young people, suggesting they might be useful for decreasing unwanted coactivation of muscles.[92] Strengthening would represent a new approach to reducing the overactivity of antagonist muscles - a problem usually treated with relaxation.[82,87] Strengthening muscles in patients with PD may also help to prevent falling, which is a tremendous problem for these patients. Decreased strength is a factor contributing to increased falling in elderly persons.[99,100]

Even very old subjects can increase their muscle strength with weight training programs.[88,95] Thus, it is reasonable to believe that persons with PD can make similar improvements. The increased strength in elderly individuals has been attributed to both increased MU recruitment and muscle hypertrophy.[95,101] Because strength training is associated with neural adaptations,[102] it is conceivable that the EMG activity pattern produced by trained subjects with PD will improve in both consistency and “energy.” We are currently designing experiments to determine whether some of these beneficial effects can be produced with strength training of subjects with PD.

Summary

This article described observations on MU activity in persons with PD. These persons are unable to maintain constant discharge rates in MUs, particularly for extended periods of time. Other observations included a possible shift in the MU population to lower recruitment thresholds, increased coactivation, and muscle weakness in persons with PD as compared with healthy control subjects. These observations alone do not indicate mechanisms underlying these changes. They might be produced by altered descending commands to motor neurons or spinal cord interneurons. Alternatively, they may reflect changes that have occurred in motor neurons due to either disuse or accelerated aging. Current physical therapy approaches to the management of PD have produced equivocal results. Instead, we advocate the prescription of strength-training programs for patients with PD.

References

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