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LITERATURE REVIEW

Is There Muscular Weakness in Parkinson's Disease?

ABSTRACT

Cano-de-la-Cuerda R, Pérez-de-Heredia M, Miangolarra-Page JC, Muñoz-Hellín E, Fernández-de-las-Peñas C: Is there muscular weakness in Parkinson's Disease? Am J Phys Med Rehabil 2010;89:70–76.

Controversy exists as to whether muscle weakness is present in Parkinson's disease (PD). Computerized literature searches identified clinical trials and reviews about muscular strength assessment in patients with Parkinson's disease, using the following databases: PubMed, Ovid MEDLINE, Ovid EMBASE, the Cochrane Database of Systematic Reviews, Cumulative Index to Nursing and Allied Health Literature, and Physiotherapy Evidence Database. Seventeen articles fulfilled all criteria for selection. These studies suggested that isokinetic muscle strength was decreased in patients with Parkinson's disease and that muscle weakness was not specifically related to tremor or rigidity. Bilateral asymmetrical muscle weakness was present in Parkinson's disease when presenting with clinical unilateral hemiparkinsonism. Recent studies using sensitive mechanical devices have provided evidence that muscle strength is reduced in patients with Parkinson's disease compared with age-matched controls. The specific cause of this weakness is not known. Questions under debate were whether this weakness was of central or peripheral origin and whether it was intrinsic to the disease or a secondary phenomenon.

Key Words: Muscular Strength Assessment, Isokinetic Measurement, Isometric Measurement, Quantitative Measurement, Muscle Weakness, Parkinson's Disease

Parkinson's disease (PD) is the second most frequent neurodegenerative disease after Alzheimer's disease. There is a debate that muscle weakness is present in PD.[1] Weakness in patients with neurological diseases is a frequently found nonspecific symptom, and studies showing objective quantification of weakness in neurological diseases have been rarely presented.[2] Recent studies have provided objective evidence that muscle strength has been reduced in patients with PD compared with age-matched controls.[3,4] Weakness is reported to be present bilaterally,[1] and muscle weakness in patients with PD has been reported to increase with performance velocity, especially as the disease progresses.[5] There is a debate about strength testing in the context of movement disorders that has clinical features, such as bradykinesia and rigidity, as confounding motor control during strength testing.

Weakness may be an early sign of PD, and it may be an early indicator of bilateral disease. The specific cause of reported weakness is not known, and questions such as whether it is of central or peripheral origin, and intrinsic to the disease

or a secondary phenomenon, remain matters of debate.[4] Diminished performance caused by weakness may be a primary sign of PD[1,3] and may be explained by disturbed motor programming in the basal ganglia. Peripheral changes in nerve or muscle are unlikely to explain the weakness. There is some debate as to whether there may be mild changes in the neuromuscular apparatus in patients with PD; however, these studies cannot explain the difference in strength between the on and off medication conditions found in some studies, presumably related with the dopamine receptors location.

We present a literature review about the evidence of the isokinetic and isometric muscle weakness present in patients with PD and the relationship between this lack of muscle strength and functional performance in persons with PD.

METHODS

Data Sources

Computerized literature searches were performed to identify clinical/controlled trials and reviews of muscular strength assessment in PD, making use of the following databases: PubMed (from 1975), Ovid MEDLINE (from 1975), Ovid EMBASE (from 1975), the Cochrane Database of Systematic Reviews, the Cochrane Collaboration Trials Register, Cumulative Index to Nursing and Allied Health Literature, and Physiotherapy Evidence Database.

Medical Subject Headings and the main key words for search were muscular strength assessment, isokinetic measurement, isometric measurement, quantitative measurement, muscle weakness, and PD. The search strategy followed the guidelines described by Greenhalgh.[6] This search was supervised by an expert librarian scientist who helped us during each stage of the procedure. When database facilities allowed search limits, searches were restricted to clinical or controlled trials. We also checked the reference lists of the articles that were identified in the database searches.

Selected articles were reviewed independently by two authors. The reviewed articles included only those published in the English language. Published proceedings and abstracts were excluded.

Data Extraction

As with article selection, data from each study were extracted independently by two authors. A standardized, data-extraction form that contained questions on population, interventions, methodology, results, and outcome measures was used, according to the Consolidated Standards of Reporting Trials statement.[7] For each article, the following data were recorded: inclusion and exclusion criteria, design, randomization, description of study subject attrition and subject/investigator blinding, outcome measures, details of the therapeutic intervention, and results. Finally, both authors had to achieve a consensus on each item on the data-extraction form.

RESULTS

Selected Articles

Twenty potentially relevant articles were identified. Of those 20 articles, all were identified on MEDLINE. Three articles were subsequently excluded because the exact methods of strength testing were not described. Seventeen articles fulfilled all criteria for selection.

Isokinetic strength evaluation was investigated in eight studies,[1,3–5,8–11] and isometric strength was measured in nine studies.[1,12–19] The relationship between muscle strength and functional performance was observed in six studies.[10,14,20–23] The influence of pharmacological treatment on muscle strength was observed in three of these studies.[2,21,24]

Description of Reviewed Articles

Isokinetic Measurement in PD

Koller and Kase[1] performed a quantitative assessment of muscle strength in 21 male patients with PD. Isokinetic strength of the wrist and knee flexors and extensors was measured bilaterally using a Cybex II isokinetic machine. They found that mean isokinetic strength was decreased in early stage PD as measured by the Hoehn and Yahr Scale (Table 1) when these subjects were compared with age-matched normal subjects. These investigators com-

TABLE 1 Hoehn and Yahr staging of Parkinson's disease[29]

Stage I
Signs and symptoms on one side only
Symptoms mild
Symptoms inconvenient but not disabling
Usually presents with tremor of one limb
Friends have noticed changes in posture, locomotion, and facial expression
Stage II
Symptoms are bilateral
Minimal disability
Posture and gait affected
Stage III
Significant slowing of body movements
Early impairment of equilibrium on walking or standing
Generalized dysfunction that is moderately severe
Stage IV
Severe symptoms
Can still walk to a limited extent
Rigidity and bradykinesia
Stage V
Cachectic stage
Invalidism complete
Cannot stand or walk

pared both the more obvious parkinsonian side and the clinically less affected or unaffected side in the subjects with PD and the control subjects. Within-subject with PD comparisons of strength between the more and less clinically affected sides were not statistically different at 5 rpm. The subjects with PD who had little to no clinical evidence of parkinsonism on their unaffected side did indeed have strength deficits on the relatively unaffected side which were comparable statistically but not clinically with the strength deficits observed on the clinically affected side. The strength deficits seen in the subjects with PD on both sides were worse than what was observed in the control subjects.

Nogaki and Morimatsu[4] studied the isokinetic strength of knee extension and flexion at two speeds of movement (30 and 90 degrees per second) using a Cybex II dynamometer in 23 patients with PD to observe whether muscle weakness is inherent to the disease. To counteract normal variation among subjects, they selected patients with symptoms completely or largely confined to one side, according to the Japan PD Rating Scale and compared sides for each patient. In all patient groups considered together, the mean maximum peak torque of the more affected side was significantly less than for the less affected side for both extension and flexion at both velocities. Although significant differences between sides were observed at both 30 and 90 degrees per second, the differences between sides were larger at 90 degrees than at 30 degrees per second. In the mildly affected group, maximum peak torque of the more affected side also was significantly less than for the less affected side for both extension and flexion at both velocities. In contrast, in the moderately affected group the maximum peak torque in both extension and flexion at 30 degrees per second showed no significant difference between sides, whereas at 90 degrees per second this value showed a significant difference between sides in both extension and flexion, with the more affected side being weaker.

They observed strength reduction on the more affected side even in the early stage of disease, and their results showed that at faster speeds the strength difference between sides increased.[3] Similar conclusions were observed in another study by the same authors.[8]

In another study,[5] isokinetic muscle strength of knee extension and flexion in 18 patients with PD who showed marked laterality in clinical signs was measured, using a Cybex II dynamometer, and compared strength between the sides in the same patient. In all patient groups, the maximum peak torque of the more affected side was significantly less than for the less affected side at 15 and 30 rpm with the difference between the sides being larger at 30 than 15 rpm, whereas at 5 rpm there were no significant differences between sides. In the Hoehn and Yahr stage I group, the maximum peak torque in both extension and flexion at each velocity showed no significant difference between the sides. In contrast, in the stage II and III groups the maximum peak torque at 5 rpm showed no significant difference between the sides, whereas at 15 and 30 rpm these values showed a tendency and a significant difference between the sides, respectively, with the more affected side being weaker.

With the availability of sensitive new equipment, several studies have shown that muscle strength was reduced in patients with PD. More research is needed to clarify whether tremor and rigidity disallow a valid assessment of isolated motor control and to answer whether there is muscular weakness in PD.

Isometric Measurement in PD

Using an isometric dynamometer, Jordan et al.[15] found no difference in the maximal isometric hand grip force between control subjects and individuals with PD. In contrast, Stelmach and Worringham[16] reported, in a similar study, longer times to reach the peak torque and contraction durations, larger impulses and lower rates of force development, and force-time profiles with many more irregularities in patients with PD than in aged-matched controls. They also initiated lower force contractions with longer latencies, unlike controls. There was a significant correlation between slower torque relaxation time and a more impaired motor score on the Unified PD Rating Scale.[17]

Using a dynamometer calibrated in pounds, Koller and Kase[1] reported no significant difference between groups for isometric grip strength compared with controls. In hemiparkinsonism, hand grip strength did not differ between the affected and unaffected sides. Muscle strength was slightly less on the affected side, but there was no statistical difference between the two sides. The hand grip strength was significantly decreased in patients with unilateral tremor compared with unilateral rigidity. The fact that the hand grip strength was impaired on the same side that some patients had unilateral tremor seems to confound strength testing in this hand.

Two studies[12,14] have shown that isometric torque in patients with PD was less than that in control subjects. Kunesch et al.[18] found that the time required to release force to baseline was substantially lengthened, and the ability to maintain constant force over time was impaired in patients with PD. Patients with PD had more irregular force-time curves and changes in the rate of force production compared with control subjects, and patients were also substantially slower in initiating

a force production, and the delay was localized in the premotor reaction time.[19]

Most studies of strength in PD have compared performance of patients and normals. Some authors found greater changes comparing force produced in isokinetic contractions rather than conventional isometric contractions. The lack of statistical agreement among studies may reflect the very wide range of strength that can be found among different individuals, especially when age and gender are considered. It seemed that the reduced ability to release isometric force was reflective of the force control difficulties in PD when measuring isometric strength.

Muscle Strength and Functional Performance in PD

Few studies have examined the relationship between muscle strength and functional performance in persons with PD. For example, rising from a chair is a physically demanding function required for independent living. In a survey of 101 individuals with PD, 81% of respondents reported having difficulty with rising from chair.[20] The ability to rise from a chair in PD has been studied by Inkster et al.[10] These authors compared lower-limb strength between individuals with PD and healthy sex- and age-matched controls. Both performed maximal concentric, isokinetic knee and hip extensor torques on an isokinetic dynamometer to quantify and compare muscle strength. Subjects also rose from a chair at their comfortable pace without the use of their arms, and the duration of this task provided a measure of sit-to-stand ability. Subjects with PD were tested in an on and off medication state on different days. Mean hip and knee extensor torques were lower in subjects with PD, with greater deficits found at the hip. Greater hip strength was related to better sit-to-stand ability in subjects with PD, whereas greater knee strength was related to better sit-to-stand ability in controls.

Only one study[14] has studied the relationship between isokinetic muscle strength in patients with PD and gait parameters. Gait, concentric isometric, and eccentric strengths in the ankle dorsiflexors were investigated in 25 patients with PD (14 men and 11 women) and 37 control subjects (19 men and 18 women) that were age matched. In all subjects with PD, mean concentric torque was significantly lowered. However, mean eccentric torque was significantly lowered in male subjects only. Patients walked with significantly lower maximum and ordinary velocity, compared with controls. At constant velocity, stride length was shorter, and single support had a shorter duration. The duration for heel strike on to ball in men was shorter, reflecting the flat foot strike of patients with PD. In male patients, there were relationships between concentric strength and gait velocity and between stride strength and stride frequency. Generally, female patients had fewer symptoms and less deviation from their respective controls in the measurements.

Previous studies have shown stride length and velocity to be good indicators of the patient's current medication efficacy.[21] Single-support safely is a sensitive measure of gait function in many gait disturbances.[22,23]

Further research is required to evaluate whether improving muscle strength would lead to improved functional performance in patients with PD. Although we were able to detect a reduced ability to generate force in subjects with PD, the relative contribution of the central and peripheral system could not be assessed. In support of a strengthening program, however, studies with healthy, elderly subjects have shown that such a strengthening program can prevent weakness secondary to disuse atrophy.

Pharmacologic Treatment and Muscle Strength in PD

Some studies[2,21,24] have examined the influence of pharmacological treatment on muscle strength in PD. Pedersen and Öberg[2] evaluated the peak torques in isometric, concentric, and eccentric contraction at 30, 120, and 180 degrees per second in ankle dorsiflexors recorded before and after the withdrawal of medication. Although clinical scores were significantly increased, peak torques in isometric, concentric, and eccentric contractions decreased at almost all velocities after withdrawal of medication, even in the unilaterally affected patients. The peak torque at slow and fast angular velocities was affected. The reduction in strength was 8%–10% at the eccentric velocities measures, and from 15% to 30% at the concentric velocities measured.

Corcos et al.[24] have showed that isometric strength can be impaired in PD even with the administration of antiparkinsonian medication. They have shown that medication influences both strength and rate of force development, and those changes in strength correlate significantly with changes in contraction time. However, some patients can still generate similar levels of force in an on and off medication, although the speed at which they do so is much slower when off.

Strength seems to be influenced by medication in patients with PD. The withdrawal of antiparkinsonian medication produces muscle weakness especially in extensor muscles in patients with PD.[24] Removing medication also leads to a decrease in the rate of force generation. Strength measurement may be used in the evaluation of pharmacological therapy.

DISCUSSION

Isokinetic Strength Measurements

Parkinsonian patients often complain of weakness. Although frequently used to measure strength in other disease processes, isokinetic strength measurement has not often been used to measure strength in neurological diseases,[2] possibly because involuntary movements may confound valid assessment of motor control.

Although isokinetic muscle strength is likely to depend on movement velocity in the early stages of PD, it may be influenced by bradykinesia, as the disease progresses,[9] and so the speed-force correlation seen in these patients may give clues to the understanding of the pathophysiology of bradykinesia. Hallet and Khoshbin[25] demonstrated that bradykinesia resulted from the inability to energize the appropriate muscles to generate force at a sufficient rate. In that sense, the speed dependency of weakness found in the study of Nogaki et al.[9] may certainly represent bradykinesia itself. If bradykinesia is an inability to produce "force" sufficient enough to generate the speed required, the force could be the primary factor afflicted in patients with PD.

Koller and Kase[1] have showed that isokinetic muscle strength is decreased in early PD compared with age-matched normal subjects and was present both on the affected and unaffected sides. Muscle torque is known to decline with increasing isokinetic velocity throughout the range of joint motion in normal subjects, but results suggest that muscle weakness in patients with PD increases with performance velocity, especially as the disease progresses,[3,5,8] and this is a distinct manifestation of the disease itself.[4]

Isometric Strength Measurements

Koller and Kase[1] reported that isometric strength was not reduced in patients with PD. If reduced speed is the consequence of reduced power, isometric power should be decreased in these patients, but this was not the case. A possible explanation is that isometric strength measurement is not sensitive enough to detect reduced power.[9]

Results suggest that subjects with PD are deficient in the regulation of force and time parameters rather than simply in force production.[15] Several factors may contribute to the observed disturbance of isometric force control when instructed to maintain it: a defect in processing visuospatial information, an impairment of proprioceptive delivery and sensorimotor integration, an incorrect computation of the required force, and the possibility of a noisy motor output system disturbing the precision of force production in relation to the target force level in PD.[18] Some authors found greater changes when comparing force produced in isokinetic contractions rather than conventional isometric contractions. The lack of agreement among studies may reflect the very wide range of strength that can be found among different individuals, especially when age and gender are considered. It should be emphasized that muscle weakness in PD can be very difficult to detect clinically on a single evaluation of a patient, although Yanagawa et al.[12] did estimate that voluntary muscle strength was reduced in 6 of 15 patients evaluated by standard manual muscle strength testing.[24] Using the same strength evaluation, other authors[1] did not observe the same results. Regardless, manual muscle strength testing is usually considered when evaluating muscular weakness in PD.

Muscle Strength and Functional Performance in PD

Inkster et al.[10] showed that individuals with mild PD generate smaller lower-limb forces compared with controls, and this reduced strength, particularly at the hip, may be one factor that contributes to the difficulty of persons with PD to rise from a chair. Although patients with PD may be more subject to deconditioning effects and these would more likely be seen in the large proximal muscle groups, it is possible that the reduced extensor strength may be a contributing factor to the flexed posture observed in later stages of PD.[10] The suggestion that extensor muscles were relatively weaker than flexor muscles as the disease progresses have been documented in three studies.[9,24,26] The extensor weakness seems to be a primarily caused by decreased tonic activation of the extensor muscles and not by muscle coactivation. These results suggest that there is an abnormal relationship between agonist *vs.* antagonist muscle weakness in patients with PD.[24]

Dietz[27] has showed how impaired motor control and performance in PD are related to impaired proprioceptive delivery to the brain. The use of external sensory cueing improves motor control in PD,[28] although the problem may not be peripheral per se, more research is needed to determine these questions.

Pharmacologic Treatment and Muscle Strength in PD

Although there is some debate as to whether there may be mild changes in the neuromuscular apparatus in patients with PD, these cannot explain the differences between strengths in an on and off medication found in studies because antiparkinsonian medication is not known to affect peripheral neuromuscular function.[24]

A bilateral decrease in static as well as concentric and eccentric peak torques (even in the uni-

laterally affected patients) has been found after withdrawal of medication. The peak torque at slow and fast angular velocities seems to be affected. The reduction in strength was 8%–10% at the eccentric velocities measured and from 15% to 30% at the concentric velocities measured in the study of Pedersen and Öberg.[2]

The observed torque limb difference seems to disappear after withdrawal of medication in the concentric contraction, but not in the eccentric contraction. The reduction in strength seems equal in both limbs and not connected to the most clinically affected side. Clinical ratings may initially be sufficient in the evaluation of the patient; but for evaluation of additional medications and when introducing new drugs, quantitative measurements could be useful.

There are several clinical implications about this review. Isokinetic measurements can be helpful in strength evaluation in patients with PD. Further studies are needed to resolve the question of whether muscle testing is a good indicator of strength in the context of movement disorders that have clinical features, such as bradykinesia, tremor, and rigidity, and the relationship with functional performances. The inability of standard clinical muscle testing to detect true weakness in PD seems an important statement to make.

There are limitations of the current review. We have only included articles written in English, so it is likely that we have missed some relevant studies written in other languages, and the findings of the current review are limited by study heterogeneity and methodological flaws of the included trials.

CONCLUSION

Recent studies using mechanical devices seem to have provided evidence that muscle strength is reduced in patients with PD compared with age-matched controls even at early stages of the disease and in the unaffected side. Isokinetic strength measurements have been shown to have high reliability in patients with PD, especially at faster speeds, and in combination with clinical assessment may be used as a supplement in the evaluation of symptoms and therapy in these patients.

Further studies are needed to resolve the question of whether muscle testing is a good indicator of strength in the context of movement disorders that have clinical features, such as bradykinesia, tremor, and rigidity, and the relationship with functional performances.

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