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Muscle weakness in cerebral palsy

Beyin felcinde kas zayıflığı

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Over the last two decades, muscle weakness has been shown to be a major component of cerebral palsy (CP) pathology. Caused by multiple etiologies including variations in the muscle fiber type, pathologic motor unit function, co-contraction of agonists and antagonists, and muscle size and rigidity, weakness interferes with function and leads to limited function and participation. Muscle strength was found to be associated with walking ability and with functional scales. Children with CP were found to be weaker than typically developing children, and differences were found with respect to muscle groups in children with CP. Muscle weakness should be evaluated as objectively as possible to improve the quality of diagnosis and treatment. Manual muscle testing is not sufficient for evaluation, and instrumented muscle testing is validated in CP. Muscle strengthening is an important part of treatment of CP. Several methods of strengthening have been described. Muscle lengthening and other spasticity-modifying therapies have been shown to have a positive effect on muscle strength. Children who participated in muscle strengthening programs had a better quality of life and improved function.

Key words: Cerebral palsy/rehabilitation; child; exercise; muscle strength; muscle weakness.

Son yirmi yılda, kas zayıflığının beyin felci (BF) patolojisinde ana bileşenlerden biri olduğu gösterilmiştir. Kas lifleri tipindeki değişiklikler, patolojik motor birim fonksiyonu, agonist ve antagonistlerin karşılıklı kontraksiyonu, kas boyutu ve rijiditesi gibi birçok nedenden kaynaklanan zayıflık, fonksiyonu bozarak sınırlı fonksiyon ve katılıma yol açmaktadır. Kas gücünün yürüme kapasitesi ve fonksiyonel ölçeklerle ilişkili olduğu gösterilmiştir. Beyin felçli çocukların, gelişimi normal çocuklardan daha zayıf oldukları görülmüş ve BF'li çocuklarda kas grupları açısından farklılıklar olduğu belirlenmiştir. Tanı ve tedavi kalitesini artırmak için, kas zayıflığı mümkün olduğu kadar objektif değerlendirilmelidir. Manuel kas testi değerlendirme için yeterli değildir; aletle yapılan kas testinin BF'de geçerliliği gösterilmiştir. Kas güçlendirme BF'de tedavinin önemli bir parçasıdır. Güçlendirmeyle ilgili çeşitli yöntemler tanımlanmıştır. Öte yandan, kas uzatma ve spastisiteyi azaltıcı diğer tedavilerin de kas gücü üzerine olumlu etkisi olduğu görülmüştür. Kas güçlendirme programlarına katılan çocuklarda yaşam kalitesi artmakta ve fonksiyonlarında düzelme görülmektedir.

Anahtar sözcükler: Beyin felci/rehabilitasyon; çocuk; egzersiz; kas gücü; kas zayıflığı.

For a long time, the common opinion among health professionals was that muscle weakness was not a major problem in children with cerebral palsy (CP). Many therapists in the past held the opinion that muscle strengthening could lead to increase in spasticity, and was therefore contraindicated in CP. Another common belief was that children with CP would not be able to gain from resistance training due to low selective muscle control and that weakness was not a major component of motor dysfunction in CP.[1]

Muscle weakness in CP is multifactorial, due to changes in muscle fiber type variation, pathologic motor unit recruitment, agonist-antagonist co-contraction, reduced selective control, reduced muscle cross-section, volume and rigidity due to collagen infiltration, and other causes.[1,2] The American Physical Therapy Association (APTA) published recommendations in 2007 regarding fitness training in children with CP. The research committee of the APTA section on pediatrics reported that children with CP were weaker, had

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less endurance, and had limited physical activity levels. They recommended that physical fitness and muscle strengthening should be made an important component in the treatment of children with CP.[3]

The aim of this paper is to review three aspects of muscle weakness in CP: etiology and scientific basis; evaluation of muscle strength and methods for muscle strengthening.

Muscle function

Muscle strength is proportional to muscle fiber velocity and to muscle fiber tension. Muscle fibers are divided into three groups based on their metabolic characteristics.[4] There are fast twitch and slow twitch fibers, and the fast twitch fibers are divided between fast oxidative and fast glycolytic fibers, on the basis of their energy utilization characteristics.[2,4] The fast glycolytic and fast oxidative fibers can produce higher muscle tension and force than slow fibers. However, when discussing muscle strength, one should consider endurance as well as tension and strength. Endurance depends on the type of work the muscle performs, and the difference between muscle fibers is not evident under low loads. The difference becomes evident under higher workload. The fast glycolytic fibers are more fatigable followed by the fast oxidative fibers, and the slow oxidative fibers have the largest endurance for prolonged activity. Specific muscles demonstrate different types of muscle fibers, suiting the muscle action and load.[2,4,5]

Muscle strength was found to be proportional to the cross-sectional area of the muscle, which is a parameter of muscle size. Thus, the common simplification that bigger muscles are stronger can be used for most purposes in clinical discussions.[4] As will be shown, some studies on muscle properties and strengthening in CP have used size as an outcome measure.

Motor units are the basic functional units of muscle movement, defined as one axon and the muscle fibers innervated by this specific axon. A muscle is comprised of several motor units with variable numbers of motor units per muscle and variable number of muscle fibers per motor unit.[2,4] Motor units with high tension rates usually have a fast contraction time and motor units with low tension have a low contraction time. Slow motor units were found to have a small number of slow oxidative muscle fibers, while fast motor units were found to have either a large number of fast glycolytic fibers or a smaller number of fast oxidative fibers. When the muscle tension decline during repetitive contraction was compared to the muscle fiber type, the fast glycolytic motor units were found to fatigue fast, the fast oxidative fibers were found to fatigue slower, and the slow oxidative fibers were the least fatigable ones.[4]

Motor units are important for muscle activation and for control of motion and muscle function. When a muscle contracts against a force, the force exerted by the muscle is controlled by recruiting motor units until enough force is exerted to perform the task. Motor units are recruited at a sequence which is specific to the muscle, when the muscle is activated. Low threshold units are recruited first, and then higher threshold units are recruited. Once all motor units are active, further modulation of muscle activity is done by an adjustment of the firing rate of individual motor units. This rank-order pattern of regulation is aimed towards minimization of fatigue, and ensures that the fatigable motor units are recruited last.[2,4]

Muscle activity is controlled by the central nervous system via peripheral nerves. When a muscle contracts, the antagonist muscle should not contract at the same time in order to achieve maximal force. The prevention of agonist and antagonist co-contraction is important to normal motion. However, studies have shown that co-contraction can also be found in typically developing (TD) children. Muscle co-activation can be part of a mechanism to increase stability in children, but the degree of co-contraction can differ between physiologic and pathologic conditions.[6-8]

Muscle weakness in cerebral palsy

Patients with CP demonstrate muscle weakness in the involved limbs as was shown by Wiley and Damiano.[9] Maximum voluntary contraction was measured, and patients with diplegic type CP were weaker than age-matched TD children. Patients with hemiplegic type CP were weaker on the involved side, but the uninvolved side was also weaker than the control group. Hip and ankle flexors were found to be stronger than their antagonists in all involved extremities, but the difference was not statistically significant.[9] Elder et al.[8] identified weakness in ankle plantar flexors in diplegic and hemiplegic type CP patients, more significant on the involved side of the hemiplegic group. The uninvolved side of the hemiplegic group was also

weaker than TD children. They identified lower cross-sectional area of the muscles, partial recruitment of motor units, and co-activation of antagonists as causes of muscle weakness in both diplegic and hemiplegic type CP.[8] Stackhouse et al.[7] compared children with CP (diplegic type; 7-13 year old) to TD children and examined maximum voluntary isometric contraction (MVIC), antagonist co-activation, and fatigability. They found that the children with CP were weaker, had a lower MVIC and a higher rate of antagonist co-activation than TD children. Interestingly, though there was no difference in the fatigability of the triceps surae between the two groups, the TD children had a higher fatigability in the quadriceps. The finding may suggest that the tendency towards crouching in diplegic type CP patients of this age group may cause increased work to the quadriceps muscle, thus strengthening it.[7]

Eek and Beckung[10] found that children with diplegic type CP were weak and that there were differences between muscle groups, with the quadriceps being stronger than other groups, and among different Gross Motor Function Classification System (GMFCS) levels. They also identified a correlation between the muscle strength and the GMFCS level of their patients.[10]

Rose and McGill[11] examined torque and motor unit recruitment in CP patients and controls. They found that CP patients developed less torque at the ankle joint than controls. They also found that, at a low level of activation of motor unit recruitment, firing rate was similar in both groups, but at MVIC, recruitment in the CP group was lower. They concluded that CP patients could not recruit the higher threshold motor units necessary for maximal contraction, and that they were not able to modify the firing rates of low threshold motor units.[11]

As was mentioned, co-activity of agonists and antagonists is found in TD children. However, children with CP exhibit a higher level of co-activity. Elder et al.[8] found doubled co-activation in the CP group compared to the TD group, and the level was higher in diplegic children than in hemiplegic children. Tedroff et al.[6] reported similar results in antagonistic muscles, as well as in agonistic muscles. They raised the possibility that the lower torques found in CP patients might be due to the dispersion of the voluntary contraction to other, non-prime mover muscles. This dysfunction of the central nervous system and the muscles necessitates more research in order to quantify the effect of agonist co-activity and antagonist co-activity, as well as other possible causes on muscle weakness in CP.[6]

Muscle size was the subject of several research projects. Ohata et al.[12] examined the thickness of the quadriceps femoris muscles in children and adolescents with CP. They identified a positive correlation between the muscle thickness and functional level as measured by the GMFCS. Spasticity was not found to correlate with muscle size, but knee flexion contracture correlated positively with spasticity level and negatively with the muscle size. The study showed associations between the different measurements, but could not prove any causative effect of muscle size on activity limitation or of activity level on muscle size. The authors suggested the use of muscle size as a vicarious mode of muscle strength in CP.[12]

Spasticity is common in children with CP, and may be one of the causes of muscle weakness. Ross and Engsber[13] showed that CP patients were weaker than controls in all muscle groups, and that they were weaker distally than proximally. They also proved that there was no correlation between the level of spasticity and antagonist muscle weakness. Thus, the theory that muscles in CP patients are weak because their antagonists are spastic was not sustained.[13] Damiano et al.[14] reported that voluntary torque was lower in children with increased spasticity and this finding correlated with a lower functional level. The association they found between weakness and stiffness explained only 20-40% of muscle weakness, and therefore cannot be called a causative relationship. They suggested several explanations for the correlation, either the use of stiffness as a compensatory mechanism to weakness, or both weakness and stiffness were the results of inactivity.[14]

Structural changes were investigated by several authors. Booth et al.[15] examined the distribution of collagen in the vastus lateralis muscle of children with CP in various levels of severity. They identified increased amounts of collagen in patients with moderately severe and severe involvement, but there was no association with muscle atrophy. This finding correlated with the modified Ashworth scale and balance, and showed a positive trend with other clinical findings such as clonus, selective control, and ambu-

latory status.[15] Friden and Lieber[16] found that spastic muscles had shorter sarcomere length and higher elastic modulus compared to healthy muscle cells. They suggested that cellular components might be modified by spasticity, especially titin and collagen. Olsson et al.[17] examined the molecular weight and sequence of titin in spastic muscle and found it to be unchanged. They reported a change in the myosin heavy chain molecule compared to controls. They identified an intracellular amorphous material as well as increased levels of connective tissue and reduced number of mitochondria. They suggested that passive tension in spastic muscles increased due to extra cellular and intracellular changes in the muscles, but not due to a change in titin structure.[17]

Vaz et al.[18] examined muscle strength and stiffness in the upper limbs of children with CP compared to TD children. They found that TD children were stronger than CP patients both in wrist extensors and flexors. The CP patients had stronger flexion when the wrist was in 30 degrees of flexion than in extension. They suggested that this finding was due to a change in muscle tissue resulting from spasticity. Poor performance of manual tasks correlated to extensor weakness and flexor stiffness.[18]

An abnormal fiber type distribution was also reported in CP patients Decreased number of type I fibers and increased number of type II fibers were reported, compatible with higher fatigability.[15,17] Previous studies reported both muscle atrophy and hypertrophy with either type I fiber dominance or type II fiber dominance and with increased variation of muscle fiber size as well as variable dominance of fiber types.[2,4] Rose et al.[19] reported that the type I fiber dominance was in the ambulatory patients and the type II fiber dominance was in nonambulatory patients, similar to spinal cord injury patients.

Evaluation of muscle strength

Evaluation of muscle strength can be done in various ways. The most common and easy method is manual muscle testing. This method is based on the patient performing a MVIC of a muscle against the examiner's hand and is easy to perform but inaccurate. The patient needs to cooperate with the examiner and perform a maximal contraction of one muscle group only, without causing a stretch reflex. This is a task that many children with CP find very difficult due to cognitive limitation or co-contraction of antagonists or agonists.[20,21] Common knowledge among clinicians is the inter-rater difference in manual muscle testing evaluation. Furthermore, the strength measured in a certain muscle length cannot be extrapolated to other lengths. This evaluation method will remain an important part of the clinical evaluation of CP patients, but its use in research should be considered as doubtful.

Isometric and isokinetic muscle testing proved to be reliable in the CP population. These methods can be used in most age groups, and when performed properly can yield reliable results.[3,21-23] These methods are more time consuming but more reproducible. All measurement methods require patient cooperation and understanding, and low selective control is a hindrance to all measurement methods.

Hand-held dynamometers are less expensive, portable, and easier to use. The force transducer of the instrument is placed on the examined limb and the patient is asked to perform a maximum contraction. The caveats of the instrument are the inability to stabilize the patient, the possibility of the examiner pressing on the instrument and adding to the measured force, and the need to measure the exact distance from the joint center to calculate torque. The isokinetic testing machines offer good stabilization of the patient, accurate and repeatable measurement and no examiner bias. Various types of muscle contraction can be measured (concentric, eccentric and isokinetic). This is probably the best method for research, but its clinical use is limited due to the time required, the necessary cooperation and understanding of the patients, cost, and lack of portability.[3]

Muscle strengthening in cerebral palsy

For years the common belief was that muscle strengthening can increase spasticity and should not be part of the therapies CP patients receive. In the last decade, however, many papers have shown the opposite. I will review part of them in this section.

Damiano et al.[1,24] used free ankle weights to strengthen the quadriceps in children with CP. Before this short-term intervention, the CP group was weaker than normal controls who participated in the same program, but at the end of the training period, the strength in the quadriceps of the CP group was normal. This training program improved the terminal

swing knee extension of the patients and reduced the degree of crouch gait.[1,24] This research showed that children with CP could strengthen their muscles using a simple exercise program without any increase in spasticity.

Rose and McGill[11] suggested that, because of the deficient excitatory drive and partial recruitment of motor units, treatments aimed at increasing muscle strength could improve movement and gait. Dodd et al.[25] reported similar results using a home-based exercise program. The patients were in teen age and all were GMFCS 1-3. The patients increased their lower limb strength within six weeks compared to a control group and the improvement sustained after 18 weeks. There was also a trend for improvement in the GMFM (Gross Motor Function Measure) dimensions D and E (standing, running and jumping) but it was not statistically significant.[25] In another study, a twice weekly task-specific exercise improved the strength in hip, knee and ankle flexors and extensors as well as some spatio-temporal parameters and functional tests in a group of eight children with CP.[26] These patients were 4-8 years old, showing that muscle strengthening can be achieved even in this young age group. A group of patients with CP, 6-12 year old participated in a free weight strengthening program, three times a week for six weeks. Muscle strength, GMFM dimensions D and E, and spatio-temporal parameters improved, and the improvement continued four weeks later. Furthermore, muscle tone was reduced in this patient group.[27]

Unger et al.[28] examined the effect of a muscle strengthening program in a school setup, targeted to fit the individual needs of each participant, 1-3 times per week, for eight weeks. They used 3-D motion analysis to evaluate the participants' gait and identified a reduction in crouch compared to an increase in crouching in the control group. They recommended the use of similar programs with inexpensive equipment to encourage muscle strengthening in adolescents with CP.[28] Eek et al.[29] examined the effect of muscle strengthening in 16 patients with spastic diplegic CP. They participated in an exercise program with free weights, rubber bands and body weights for resistance during a period of eight weeks. Three-dimensional motion analysis, GMFM, muscle strength evaluation using a hand-held dynamometer, and other clinical evaluations were performed. The patients were GMFCS I-II and were weak before the intervention. There was a significant increase in the strength in several muscles and a significant improvement in the GMFM. Most spatio-temporal parameters were within normal before the intervention, and there was an increase in hip extensor moment and plantar flexor power generation at push off.[29]

McBurney et al.[30] examined the effect of a home-based training program aimed at strengthening the major support muscles of the lower limbs using simple exercises and free weights in a backpack as resistance. The participants found the intervention beneficial, without any negative outcome. They reported feeling stronger, having improved range of motion, and feeling better mentally. Activities and participation were improved as well. Participants felt that the incorporation of a fitness program had an ongoing influence on their lives.[30]

Children with CP usually have a low cardio-respiratory fitness. Before beginning any muscle strengthening program, these children need to have a thorough evaluation of their cardiac and respiratory condition. A balanced nutrition program should be established, and bone density should be evaluated before any youth with CP begins resistance exercises.[2]

Other interventions can affect muscle strength. Spasticity reduction can improve patients' function and cooperation, and enhance muscle strengthening interventions. Fry et al.[31] examined the effect of gastrocnemius recession on the muscle volume. They found that the muscle volume increased significantly in the year following gastrocnemius recession. The authors concluded that improved joint position after the operation enabled increased loading of the muscle leading to hypertrophy. Since we know that muscle strength is correlated to its size, we can deduce that the muscle would be stronger following the intervention. On the other hand, van Doornik et al.[32] gave oral baclofen to children with spastic CP and found an increase in ankle plantar flexors torque, which they attributed to reduced spasticity, increasing the ability to exercise and muscle strength.

In conclusion, muscle strength is becoming more and more important for health practitioners treating children with CP. More research is necessary on muscle strength in CP, but several practical conclusions can be drawn now. The common practice of letting

the physical therapist take care of muscle strength is wrong, and physicians should consider muscle strength when treating children with CP. Adequate strength is an important prerequisite for any orthopaedic intervention, and it should be carefully evaluated before surgery. Muscle strengthening should be a major component of any rehabilitation program. Modern methods of muscle strengthening should be developed, and access to muscle strengthening equipment and sporting activities for children with CP should be set as a target for every community. Parents and health professionals should be instructed about the importance of strengthening and educated on the advantages of strengthening of CP patients.

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