Architecture of the medial gastrocnemius in children with spastic diplegia

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Ultrasound images were obtained of the medial gastrocnemius at different ankle joint positions with the knee extended. Fascicle length and deep fascicle angle were measured in five normally developing adults (mean age 33 years, age range 24 to 36 years) and in five normally developing children (mean age 7.8 years, age range 7 to 11 years), and in seven children with spastic diplegia (mean age 10 years, age range 6 to 13 years). These architectural variables were similar in the groups of normally developing adults and children. Importantly, no statistical difference could be found between the normally developing children and those with diplegia for fascicle length. Deep fascicle angles were reduced significantly in the clinical group at a particular ankle joint angle but not at the resting angles. The difference in deep fascicle angles is explained as a function of resting muscle length and is not attributed any clinical importance. Our results do not explain the structural origin of muscle contracture explicitly. However, they do indicate that most of the fixed shortness in the medial gastrocnemii of ambulant children with spastic diplegia is not due to reduced muscle fascicle length. We suggest that muscle contracture may be better explained in terms of shortness of the aponeuroses of pennate muscles, such as the medial gastrocnemius, through reduced muscle fascicle diameter.

Ambulant children with spastic cerebral palsy (CP) have abnormal gait patterns. Their altered joint positions may be due to hypertonia, spasticity, and muscle weakness initially, with fixed shortening of the musculotendinous units developing as they grow. In many children this muscle contracture results in immobility before they reach adulthood. A detailed understanding of the development of fixed muscle shortening, as well as direct measurements of its extent, may allow optimal selection and timing of treatment that would improve the quality of life for these children.

There is a widely held belief in the clinical community that the muscle contractures observed in spastic CP are due to a reduction in muscle fascicle length as a consequence of hypertonus and poor joint position (O'Dwyer et al. 1989). It is well documented that muscle fascicles adapt to imposed mechanical conditions in adult animals by changing length (Tabary et al. 1972). Specifically, there is an increase in the number of sarcomeres in series when a muscle is immobilized in the lengthened position and a decrease when it is fixed in the shortened position. This adaptation may preserve sarcomere length and optimal sarcomere force production during contraction (Williams and Goldspink 1978). Concurrent with the change in sarcomere number are alterations in fascicle diameter, morphology, and biochemistry (for review, see Gossman et al. 1982). When immobilization casts are removed, these adaptations reverse over a period of weeks (Williams and Goldspink 1971).

In the infant rabbit, immobilization of the musculotendinous unit in the lengthened position results, initially, in an increase in the number of sarcomeres in series (Tardieu et al. 1977). However, after a few days, Tardieu and colleagues found that the number of serial sarcomeres had reduced. The authors suggest that a proportionate increase in the length of the tendon had occurred. As in adult animals, immobilization of young muscle in the shortened position results in significant sarcomere loss.

Few histological studies of muscle in spastic CP exist. Two groups have reported an increased variation in fascicle diameter in the gastrocnemius of ambulant individuals (Rose et al. 1994, Ito et al. 1996) and a predominance of fascicle type. It is difficult to draw conclusions about gross changes in muscle morphology in spastic CP from these studies as differences in mean fascicle area or diameter were not found.

Indirect measurements of muscle length in children have been made by Tardieu and colleagues (Tardieu et al. 1982). These studies measured the passive and active torque-angle relations of the ankle in children with spastic CP and in normally developing children. The group with CP was characterized by a steeper passive torque-angle relation with the onset of a small passive torque and the range of active torque occurring at more plantarflexed positions. The authors inferred that the muscle bellies of these children were short and that their tendons were of normal, or greater than normal, length. Unfortunately, few direct measurements of the dimensions of the musculotendinous unit components have been made in patient groups. Halar and coworkers used an X-ray technique to distinguish gastrocnemius belly and tendon length (Halar et al. 1978). They found that muscle belly length was reduced in adult individuals with acquired hemiplegia due to stroke.

A number of authors have stated that muscle contracture may be prevented by regular stretching of the affected muscle though there is conflicting evidence as to the quantity of stretching required (Tardieu et al. 1988, Williams 1990). Conservative treatment of fixed shortening in children involves stretching the musculotendinous unit through serial casting, splinting, and physiotherapy. While these may result in improved range temporarily, there is some evidence from animal studies that only the tendons experience significant changes in length (Tardieu et al. 1977).

The identification and measurement of the components of the muscle belly that become contracted in children with spastic CP have not been reported but non-invasive measurements of fascicle length and superficial and deep fascicle angles are possible. The relation of fascicle length and pennation angle to joint position has been made clear in vivo by investigations in adults with ultrasound imaging (Narici et al. 1996) and MRI (Scott et al. 1993). Fascicle length of the medial and lateral heads of the gastrocnemius was shown to increase and deep fascicle angle to decrease with increasing dorsiflexion (Maganaris et al.1998). Narici and colleagues (1998) reported a decrease in the deep fascicle angle of the medial head in athletes whose knee joint had been immobilized subsequent to knee ligament injury, using ultrasound imaging. Conversely, Kawakami and colleagues (1993) have shown that adults with hypertrophic calf musculature exhibit increased pennation angles compared with normally developing individuals.

The aim of this study was to compare the muscle architecture of the medial gastrocnemius in normally developing children and adults, with children who have spastic diplegia and plantarflexion contractures, using ultrasound imaging. Our initial hypothesis, based on the literature cited above, is that muscle fascicle length is reduced in children with spastic diplegia.

Method

Sixteen individuals agreed to participate in this study. There were three groups: group 1, five normally developing adults (three males, two females; mean age 33 years, age range 24 to 36 years); group 2, five normally developing children (three males, two females; mean age 7.8 years, age range 7 to 11 years); group 3, seven ambulant children with spastic diplegia (three males, four females; mean age 10 years, age range 6 to 13 years). These children had plantarflexion contractures of greater than 10° with the knee extended.

Each participant lay prone on the examination couch with the distal portion of their legs off the plinth. In normally developing participants, the musculature of the calf of the left leg was the object of measurement. For those with CP, the limb with the most severe plantarflexion contractures was chosen, as determined by clinical examination. The positions of the most prominent points of the lateral malleolus and the fibula head (leg length, nominally) were marked with a fascicle-tip pen and the distance between them measured with an anthropometer (Holtain, Crymych, Wales). B-mode ultrasound images (AU4 Idea; EsaoteBiomedica, Milan, Italy) were obtained from the distal portion of the medial gastrocnemius (Fig. 1) with a linear array probe (7.5 or 10 MHz) proximal to the distal toe of the muscle. Care was taken to align the probe so that the muscle fascicles were in the image plane. Images were obtained at several angles throughout the participant's ankle joint range, including maximum dorsiflexion and maximum plantarflexion. The ankle angle (made by the lateral border of the hindfoot and the principal axis of the leg) was measured by manual goniometry. An assistant maintained the required ankle position during image collection.

All images were recorded on videotape and the video sequences were downloaded to an IBM compatible personal computer using a framegrabber (Broadway Pro; Data Translation, Marlboro, MA, USA). Individual frames were selected for analysis and saved in Tagged Image File Format. An image analysis program (Scion Image 3b; Scion, Maryland, USA) was used to measure the deep fascicle angle and muscle thickness. Fascicle length was computed from deep fascicle angle and muscle thickness by trigonometry (see Fig. 1 and equation below):

$$F_l = \frac{T_g}{\sin \theta}$$

where F_i is fascicle length, T_g is the local thickness of the medial gastrocnemius, and sin θ is the deep fascicle angle.

Values of fascicle length were normalized to the length of the leg by dividing them by the distance between the fibula head and the lateral malleolus.

DATA ANALYSIS

Mean values and standard deviations of measurements obtained at ankle joint angles common to members in each group were tabulated. The dependence of deep fascicle angle and fascicle length on ankle joint angle was determined on the pooled data in each group by linear regression. Dependence could be demonstrated by values of significance for the *F* value in the ANOVA of less than 0.05. Student *t*-tests were performed on measurements made between the different groups at angles common to both groups and at resting ankle angles. Again, we assumed that significant differences of the means were obtained at the 5% level (p < 0.05).



Figure 1: Typical ultrasound image of a portion of medial gastrocnemius belly illustrating measurements of deep fascicle angle and fascicle length.

Table I: Anthropometric and passive range of motion data from participants expressed as mean (SD)

Group I	Resting ankle angle, °	Max. dors., °	Max. plant., °	Tibial length, mm ^a	Foot length, mm	Ages, y
Adults, Mean (SD)	-28.8 (5)	10.6 (4.7)	-49(7.4)	364 (16)	255 (10)	33(5.1)
ND children, Mean (Sl	D) -23.8 (5)	9 (4.2)	-45(7.1)	288 (38) ^b	211 (22)	7.8 (2.2) ^b
Children with CP, Mea	n (SD) -46 (8.5) ^c	-24.4 (9.4) ^c	-59.3 (11.7) ^c	271 (38)	194 (25)	10(3.3)

^aMeasured as distance between fibula head and lateral malleolus; ^bIndicates a significant difference between means of adult and ND child groups; ^cIndicates a significant difference between means of the CP and ND child groups (*t*-test, unpaired, unequal variances, p < 0.05); Max. dors, maximum dorsiflexion; Max. plant, maximum plantarflexion.



Ankle angle°

Figure 2: Architectural variables vs ankle joint angle for normally developing children and adults; (a) deep fascicle angle; (b) un-normalized fascicle length; (c) normalized fascicle length.



Figure 3: Architectural variables vs ankle joint angle for normally developing children and those with CP; (a) deep fascicle angle; (b) un-normalized fascicle length; (c) normalized fascicle lengthg.

Results

Table I contains anthropometric and passive range of motion data from the participants in this study. Resting ankle angle, maximum dorsiflexion, and maximum plantarflexion were significantly different between normally developing children and children with spastic diplegia.

EFFECT OF ANKLE JOINT ANGLE ON MEDIAL GASTROCNEMIUS ARCHITECTURE

Figures 2 and 3 depict the relation of the architectural variables to ankle joint angle. In the groups of adults and normally developing children, fascicle length, normalized fascicle length, and deep fascicle angle were found to be dependent on ankle angle (ANOVA *F*, p<0.05). With increasing plantarflexion, deep fascicle angles increased and fascicle lengths decreased. In adults, our results are similar to those values reported previously by Narici and coworkers (1996) and Maganaris and colleagues (1998).

In the group with CP, these variables could not be shown to be dependent on ankle angle.

DIFFERENCES BETWEEN STUDY GROUPS

Measurements at ankle joint angles common to the different groups were analyzed and tabulated (see Table II). Significance tests were performed on the difference of the means of the architectural variables at common joint angles (Student *t*-test, unpaired, unequal variance).

Deep fascicle angle appeared smaller in the group of normally developing children than in the adult group at each ankle position, but the difference was significant at a 30° ankle angle only. Mean fascicle length was also smaller in the group of normally developing children (significant differences were found at 15 and 30° of plantarflexion). However, no significant differences between normalized fascicle length in these two groups could be found.

At an ankle angle of 30° of plantarflexion, the deep fascicle

angle in the group with CP was significantly smaller than in the group of normally developing children. However, no significant difference in deep fascicle angle could be found at the resting joint angle. Significant differences could not be found at 30° of plantarflexion or at the resting joint angle for fascicle length or normalized fascicle length between normally developing children and those with spastic diplegia.

Discussion

Ultrasound imaging has been used previously to investigate muscle architecture in normally developing adults. In this study we report architectural variables of the medial gastrocnemius in normally developing adults and children, and in a group of children with spastic diplegia. Data from our adult group is comparable with that reported in the literature (Narici et al. 1996, Maganaris et al. 1998). As in those studies, we were able to show a dependence of deep fascicle angle (increasing with increasing plantarflexion) and fascicle length (decreasing with increasing plantarflexion) on ankle joint angle.

This study has demonstrated that normalized fascicle length and deep fascicle angle in normally developing children and adults are similar throughout the ankle joint range (Fig. 2). We noted that mean values of deep fascicle angle were slightly smaller in the child group, but this result was significant at 30° of plantarflexion only. Heslinga and Huijing (1990) showed that fascicle angles increased in the rat gastrocnemius with normal growth, and a number of studies in animals and humans have identified an increase in fascicle angles with hypertrophy (Kawakami et al. 1993).

Our results show that architecture of the medial gastrocnemius in normally developing children and children with spastic diplegia does not differ greatly (see Fig. 3). Deep fascicle angles appear consistently smaller in those with spastic diplegia (see Fig. 3a), at any particular ankle joint angle. We confirmed this observation by performing significance tests

Table II: Fascicle lengths (normalized and un-normalized) and deep fascicle angles of medial gastrocnemius at particular ankle joint angles and at resting ankle angle for three study groups expressed as a mean (SD)

	Ankle joint angle, °						
	0	-15	-30	Resting			
	Mean (SD)	Mean (SD)	Mean SD	Mean (SD)			
Deep fascicle angle, °							
Adults	18.5 (2.8)	21.5 (2.2)	26.8 (2.1)	N/A			
ND children	16.8 (2.9)	19.2 (4.2)	21.4 (3.4) ^a	21.7 (4.6)			
Children with CP	N/A	N/A	15.6 (3.4) ^b	19.7 (4.5)			
Fascicle length, mm							
Adults	58.9 (4.8)	56.3 (4.9)	44.6 (3.7)	N/A			
ND children	49.9 (14.4)	$41(10.2)^{a}$	36.1 (6.3) ^a	37.4 (10.1)			
Children with CP	N/A	N/A	38.3 (11.5)	31.8 (9.8)			
Normalized fascicle length, mm							
Adults	0.162 (0.010)	0.154 (0.011)	0.122 (0.012)	N/A			
ND children	0.171 (0.035)	0.142 (0.032)	0.126 (0.022)	0.130 (0.036)			
Children with CP	N/A	N/A	0.148 (0.042) ^c	0.118 (0.031)			

^aIndicates a significant difference between mean values of ND children and adult groups.

 b Indicates a significant difference between means of groups with CP and ND children (*t*-test, unpaired, unequal variances, p < 0.05).

 $^{c}n=6$. For the adult and ND children group n=5. For the group with CP n=7 except here.

N/A indicates that data were not available for the calculations of means or standard deviations.

at a common ankle angle of 30° of plantarflexion (see Table II). However, over the range of motion of the ankle joint, inspection of the data suggests that deep fascicle angles in the CP group are of similar magnitude. Indeed, with no external forces applied to the foot (i.e. at the resting ankle angle), significant difference between deep fascicle angle could not be demonstrated. To emphasize the similarity of deep fascicle angles in the group of normally developing children and those with spastic diplegia, the data in Figure 3c was replotted relative to the resting ankle angle (Fig. 4). It is clear from the figure that there is little if any difference between these groups when the data are normalized in this way.

Importantly, significant differences in fascicle length between normally developing children and those with diplegia could not be detected at a common angle or at the resting ankle angles, and we reject our initial hypothesis that fascicle lengths are reduced in ambulant children with spastic diplegia.

CLINICAL IMPLICATIONS

Much of the rationale for conservative treatment in ambulant children with spastic diplegia is based on animal studies of



Figure 4: Deep fascicle angle vs ankle position relative to resting angle in normally developing children and those with CP. (Fig 3c replotted relative to resting ankle angle).



Figure 5: Simple planar representation of morphology and architecture of medial gastrocnemius belly. Diagram depicts two muscles with an identical number of fascicles (20), same fascicle length, and same superficial and deep aponeurosis angles. In (a) fascicles are 33% greater in diameter than in (b). Muscle belly in (a) is clearly longer. Figure illustrates that, in pennate muscle, muscle fascicle diameter is a determinant of whole muscle belly length.

adaptation of muscle fascicle length through serial sarcomere addition or loss due to joint immobilization in lengthened or shortened positions. There are a number of treatments aimed at maintaining or increasing muscle length including physiotherapy, splinting, and serial casting yet, to-date, quantitative measurements of whole muscle- or fascicle-lengths have not been made. The results presented here suggest that medial gastrocnemius fascicles in this group are of normal length. We conclude that reduction in muscle fascicle length does not account for the fixed shortening of the medial gastrocnemius in ambulant children with spastic diplegia. This conclusion seemingly contradicts clinical observations that muscle bellies in these children are short. Clearly, one or more elements of the musculotendinous unit must be short for those participants with spasticity in this study to have ankle joint angles at rest 20° more plantarflexed than normally developing children (see Table I). One explanation is that most of the fixed shortening of the medial gastrocnemius is due to a decrease in the mean muscle fascicle diameter. An appreciation of how the length of the medial gastrocnemius can be regulated by fascicle diameter can be gained from an understanding of its morphology and architecture. Data from cadaveric studies (Wickiewicz et al. 1983, Frederich and Brand 1990) show that the ratio of fascicle length to muscle length in these muscles is approximately 1:4. It follows that the primary determinant of muscle length in the medial gastrocnemius is the length of the deep and superficial aponeuroses (the internal tendons of the muscle). Change in the length of the aponeurosis should be proportional to change in the mean diameter of muscle fascicles inserting or originating from it, as the number of fascicles probably remains unchanged. There is some evidence in the literature to support this view. Heslinga and Huijing (1992) found similar fascicle lengths, and decreased aponeurosis and whole muscle lengths in the gastrocnemii of rats with their plantarflexors immobilized in the shortened position. They concluded that the reduction in muscle length was due solely to a reduction in aponeurosis length because of muscle fascicle atrophy.

A small number of histological studies have been performed on muscle biopsies obtained during surgery from young individuals with spastic CP. Though variation in fascicle diameter was observed (Rose et al. 1994, Ito et al. 1996), none of these studies measured a difference in mean fascicle diameter between spastic and normal muscle. In some individuals, type-I fascicle predominance in gastrocnemius muscles was also reported: a feature associated with spastic muscle in other conditions (Edstrom et al. 1973, Dattola et al. 1993) and in muscle subject to chronic low frequency electrical stimulation (Salmons and Vrbova 1969). Interestingly, chronic low frequency stimulation has been reported to result in decreased fascicle diameter (Brown et al. 1976). Rose and McGill (1998) suggested in their review that the increased variation in fascicle size and type-I fascicle predominance may be due to the low-frequency prolonged firing rates observed in spastic CP.

If contraction of the aponeurosis due to reduced muscle fascicle diameter is responsible for fixed contracture, preventing and reversing these changes in muscle through strength training and/or electrical stimulation could be more valuable in improving joint position than attempting to increase fascicle length by stretching and serial casting.

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References

- Dattola R, Girlanda P, Vita G, Santoro M, Roberto ML, Toscano A, Venuto C, Bardello A, Messina C. (1993) Muscle rearrangement in patients with hemiparesis after stroke: an electrophysiological and morphological study. *European Journal of Neurology* **33**: 109–14.
- Edstrom L, Grimby L, Hannerz J. (1973) Correlation between recruitment order of motor units and muscular atrophy pattern in upper motorneuron lesion: significance of spasticity. *Experientia* **29:** 560–1.
- Freiderich J, Brand R. (1990) Muscle fiber architecture in the human lower limb. *Journal of Biomechanics* 23: 91–5.
- Gossman MR, Sahrmann SA, Rose SJ. (1982) Review of lengthassociated changes in muscle. *Physical Therapy* 62: 1799–808.
- Halar EM, Stolov WC, Venkatesh B, Brozovich FV, Harley JD. (1978) Gastrocnemius muscle belly and tendon length in stroke patients and able-bodied persons. *Archives of Physical Medicine and Rehabilitation* 59: 476–84.
- Heslinga JW, Huijing PA. (1990) Effects of growth on architecture and functional characteristics of adult rat gastrocnemius muscle. *Journal of Morphology* 206: 119–32.
- — (1992) Effects of short length immobilisation of medial gastrocnemius of growing young rats. *European Journal of Morphology* **30**: 257–73.
- Ito J, Araki A, Tanaka H, Tasaki T, Cho K, Yamazaki R. (1996) Muscle histopathology in spastic cerebral palsy. *Brain and Development* 18: 299–303.
- Kawakami Y, Abe T, Fukunaga T. (1993) Muscle fibre pennation angles are greater in hypertrophied than in normal muscles. *Journal of Applied Physiology* **74:** 2740–4.
- Maganaris CN, Baltzopoulos V, Sargeant AJ. (1998) In vivo measurements of the triceps surae complex architecture in man: implications for muscle function. *Journal of Physiology* **512:** 603–14.
- Narici MV, Binzoni T, Hiltbrand E, Fasel J, Terrier F, Cerretelli P. (1996) In vivo human gastrocnemius architecture with changing joint angle at rest and during graded isometric contraction. *Journal of Physiology* **496**: 287–97.
- Capodaglio P, Minetti AE, Ferrari-Bardile A, Maini M, Cerretelli P. (1998) Changes in human skeletal muscle architecture induced by disuse-atrophy. *Journal of Physiology* **506**: 59P.
- O'Dwyer NJ, Neilson PD, Nash J. (1989) Mechanisms of muscle growth related to muscle contracture in cerebral palsy. *Developmental Medicine & Child Neurology* **31:** 534–7.
- Rose J, Haskell W, Gamble J, Hamilton R, Brown D, Rinsky L. (1994) Muscle pathology and clinical measures of disability in children with cerebral palsy. *Journal of Orthopaedic Research* 12: 758–68.
- Rose J, McGill K. (1998) The motor unit in cerebral palsy. Developmental Medicine & Child Neurology 40: 270-7.
- Salmons S, Vrbova G. (1969) The influence of activity on some contractile characteristics of mammalian fast and slow muscles. *Journal of Physiology* **201**: 535–49.
- Scott SH, Engstrom CM, Loeb GE. (1993) Morphometry of human thigh muscles. Determination of fascicle architecture by magnetic resonance imaging. *Journal of Anatomy* 182: 249–57.
- Tabary JC, Tabary C, Tardieu C. (1972) Physiological and structural changes in the cat's soleus muscle due to immobilization at different lengths by plaster cast. *Journal of Physiology* 224: 231–44.
- Tardieu C, Tabary JC, Tabary C, Huet de la Tour E. (1977) Comparison of the sarcomere number adaption in young and adult animals. Influence of tendon adaptation. *Journal de Physiologie* 73: 1045–55. (In French).
- Huet de la Tour E, Bret MD, Tardieu G. (1982) Muscle hypoextensibility in children with cerebral palsy. I: Clinical and experimental observations. Archives of Physical Medicine and Rehabilitation 63: 97–102.
- Lespargot A, Tabary C, Bret MD. (1988) For how long must the soleus muscle be stretched each day to prevent contracture? *Developmental Medicine & Child Neurology* **30**: 3–10.

- Wickiewicz TL, Roy R, Powell P, Edgerton VR. (1983) Muscle architecture of the human lower limb. *Clinical Orthopaedics* and Related Research 179: 275–83.
- Williams PE. (1990) Use of intermittent stretch in the prevention of serial sarcomere loss in immobilised muscle. *Annals of the Rheumatic Diseases* 49: 316–7.
- Goldspink G. (1971) Longitudinal growth of striated muscle fibres. *Journal of Cell Science* **9:** 751–2.
- — (1978) Changes in sarcomere length and physiological properties in immobilized muscle. *Journal of Anatomy* 127: 459–68.