IS ANKLE CONTRACTURE AFTER STROKE DUE TO ABNORMAL FORCE TRANSMISSION BETWEEN THE GASTROCNEMIUS AND SOLEUS MUSCLES?

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SUMMARY
Contracture, the loss of joint range of motion, is a common complication after stroke but the mechanisms of contracture remain unclear. It has been hypothesised that contracture could be caused by increased inter-muscle force transmission (tethering of adjacent muscles). This study compared force transmission between the gastrocnemius and soleus muscles of people with ankle contractures after stroke and control subjects. Inter-muscle force transmission was examined by measuring changes in soleus muscle fascicle length during passive knee motion. There were no between-group differences in change in soleus fascicle length and pennation during knee extension, after adjusting for ankle motion. Ankle contracture after stroke is not due to abnormal inter-muscle force transmission between gastrocnemius and soleus.

INTRODUCTION
Contracture is a common complication after stroke and other neurological disorders. Contracture is thought to be due to changes in passive mechanical properties of muscle fascicles or tendons, or both. Another potential mechanism is that inter-muscle force transmission, the lateral transfer of force between adjacent muscles, could reduce muscle extensibility and joint mobility [1, 2]. This study examines whether ankle contracture after stroke in humans is due to abnormal inter-muscle force transmission between the gastrocnemius and soleus muscles.

METHODS
Data were obtained from five subjects with stroke who had ankle contractures and six able-bodied control subjects. All subjects with stroke had a left hemiplegia.

Subjects were seated on an isokinetic dynamometer with the thigh supported and the shank strapped firmly to the dynamometer input arm. The ankle was firmly fixed in a range of motion brace locked at 90 degrees of dorsiflexion (ankle plantargrade). The dynamometer passively extended the knee from 90 degrees knee flexion to near-full extension at an angular velocity of 1 degree per second. To determine if there was any movement of the ankle joint within the range of motion brace ankle angle was monitored using clusters of three reflective markers mounted on stalks firmly attached to the foot and shank. An image-based 3D motion analysis system (Optitrack) was used to track movement of the marker clusters. The relative motion of the two marker clusters provided a measure of ankle motion within the brace.

Ultrasound images of soleus muscle fascicles during knee extension were recorded using transducers from two portable ultrasound units coupled side-by-side to obtain a wide field of view. Images from both transducers were cropped and stitched to obtain a composite video sequence. The length and pennation of soleus muscle fascicles were tracked using Matlab cross-correlation techniques. For each subject, data from the best of three trials were analysed. The slopes of changes in soleus fascicle length and pennation as the knee was extended were adjusted for ankle motion using regression models. Between-group comparisons of slopes were made using t-tests.

RESULTS AND DISCUSSION
Subjects with stroke had significant ankle contracture (mean between-group difference in ankle range of motion = 14 degrees, p = 0.028) as well as ankle dorsiflexor and plantarflexor muscle weakness.

On average approximately 5 degrees of ankle joint motion occurred within the range of motion boot, but the amount of motion did not differ between groups (mean between-group difference in ankle joint motion = 1.0 degrees, p = 0.53). Graphs of soleus muscle fascicle length, pennation and ankle angle during knee extension are shown in Figure 1. Some subjects showed small increases in fascicle length or pennation with knee angle while others showed slight decreases, but these were always small. Confidence intervals of slopes of change in fascicle length and pennation with knee angle for individual subjects were narrow and did not cross zero for all but two subjects (data not shown).

There were no differences between subjects with stroke who had contracture and control subjects in change in fascicle length (between-group difference in slopes = 0.044 mm/degree, 95% CI -0.108 to 0.195 mm/degree, p = 0.53).
and pennation (between-group difference in slopes = 0.045 degree/degree, 95% CI -0.052 to 0.141 degree/degree, p = 0.32) during knee extension adjusted for ankle motion. That is, the change in soleus fascicle length and pennation that would have occurred during knee extension if the ankle had not moved was not different between groups.

This is the first study to compare inter-muscle force transmission between the gastrocnemius and soleus in subjects with stroke who had ankle contractures and control subjects who did not have ankle contracture. It was important to monitor ankle motion within the range of motion brace as small changes in soleus fascicle length, if they occurred, could have been due to small changes in ankle joint angle. Ankle joint motion was quantified using 3D motion analysis and analyses of change in soleus fascicle length and pennation during knee extension were statistically adjusted for changes in ankle angle. Change in soleus fascicle length and pennation during knee extension while adjusting for ankle angle was not different between groups.

CONCLUSIONS
Ankle plantarflexor muscle contracture after stroke is not due to abnormal inter-muscle force transmission between the gastrocnemius and soleus muscles. Further research is needed to better determine the mechanisms of contracture after stroke.

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REFERENCES

Figure 1. Graphs of (a) soleus muscle fascicle length, (b) pennation and (c) ankle angle during knee extension. Knee angles of -90 and 0 degrees indicate knee flexion and full knee extension respectively.