NONISOMETRIC BEHAVIOR OF ANTAGONIST FASCICLES DURING AGONIST ISOMETRIC CONTRACTIONS

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INTRODUCTION
The majority of human activities require muscle coactivation, which is defined by the activation of the antagonist muscle during a voluntary muscle group’s contraction. By contracting, the antagonist muscle produces a force whose direction is the opposite of the agonist's. Consequently, the external torque developed around the joint is the result of the agonist torque minus the one generated by the antagonist muscle [1, 2, 3]. Besides, during voluntary contractions at varying strengths, it has been shown that antagonist muscle activity increased in parallel with that of agonist muscle [1]. This might indicate that antagonist muscle activity is generated to maintain or improve joint stability [4, 3, 5, 6]. The current investigation was designed to analyze the effect of different voluntary contractions at varying strengths on the mechanical changes in the antagonist muscle-tendon unit in parallel with its coactivation.

METHODS
Measurements were taken on twelve volunteers who participated in this investigation. The subject was secured on an adjustable chair in a slightly reclined position with the right foot strapped onto the footplate of an ankle ergometer. The isometric torques exerted by the plantarflexor or the dorsiflexor muscles were recorded by a strain-gauge transducer. Voluntary EMG activities in the MG and TA were obtained by means of bipolar surface electrodes. The MVC torque and associated average EMG (aEMG) activities were determined for a 2s-period during the torque plateau at the different levels of force. The architectural changes of the agonist and antagonist muscles during isometric contraction were investigated by ultrasonography (Mindray DP-6600). Probes were fixed firmly onto the right leg over the mediolateral center of MG muscle, the myotendinous junction and on the central region of the TA muscle. The clearly visible fiber bundle lying between superficial aponeurosis and deep aponeurosis was considered as fascicle length (Lf). The pennation angle (θ) was defined as the angle between the aponeurosis and the tangent of fascicles at the points of attachment onto the aponeurosis. These parameters were measured by using a public domain image program (Scion image). The cross point (P) of ultrasonic echoes was defined as the position where the fascicle attached to the deep aponeurosis. The longitudinal displacement (Δx) performed by the P point was considered as the length change of the respective aponeurosis as well as the free tendon during contraction. The longitudinal displacement travelled by MTJ was also measured (ΔMTJ) for antagonist muscle.

RESULTS AND DISCUSSION
The aEMG activity for the TA muscle increased linearly (P<0.001; r²=0.99) throughout the different levels. Similar to the agonist muscle, the aEMG of the MG antagonist muscle increased but the evolution fits to a third-degree polynomial (r²=0.99; y=0.3191 + 0.03373.x + 0.001634.x² + 1.943e⁻⁶.x³). Our results are supported by the effects reported in literature regarding coactivation [4, 1]. The agonist-to-antagonist ratio increased for the MG muscle to 11.5 ± 5.1% (P < 0.001) at 20% of the MVC and remained constant during the remaining levels of force. This evolution could mean that antagonist MG muscle increases the joint stability and couldn’t act as a brake for high levels of force. When the dorsiflexion torque increased, Lf of agonist muscle (TA; Figure 1 and 2) decreased from 56.1 ± 8.4 to 42.2 ± 8.0 mm (P < 0.001), θ increased from 14.2° ± 1.8 to 21.2° ± 3.9 (P < 0.001) and Δx increased up to 16.3 ± 3.1 mm (P < 0.001) at maximum. Consistent with previous TA muscle in vivo studies, our results show a fascicle length reduced and pennation angle increased [7]. With the enhancement of the contraction strengths ranging from 0 to 100% of MVC, Lf of the antagonist muscle (MG; Figure 1 and 2) increased from 47.0 ± 6.9 to 53.4 ± 6.8 mm (P < 0.001), θ (MG) decreased from 26.4° ± 3.7 to 23.9° ± 2.8 (P < 0.001). The changes in Lf and θ variables were curvilinear. There is no significant difference for pennation angle and fascicule length between 40% and 100% of the MVC and 65% and 100% of the MVC, respectively. When the dorsiflexion torque increased, the MTJ displacement increased linearly up to 7.5 ± 2.1 mm (P < 0.001) at maximum. [8] reported that when the agonist shorts actively, the antagonist will have to lengthen either passively or in an eccentric contraction. As [7] have reported, the antagonist muscle, we also observed a nonisometric behavior of the antagonist muscle. The similar evolution of coactivation ratio and architectural parameters indicated that the antagonist muscle was stiffer according to the applied force and thus improved joint stability.
**Figure 1:** Changes in $L_f$ (■, □) and $\theta$ (▲, Δ) of the antagonist$^1$ (MG) and agonist$^2$ (TA) muscles during graded force development up to maximal voluntary contraction. Significant differences from initial value: *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

**Figure 2:** Typical sonographs of agonist (A,C) and antagonist (B,D) muscles at rest (A,B) and 100% MVC (C,D). The white arrow in each ultrasonic image corresponds to the P point which is defined as the cross section between muscle fascicle and deep aponeurosis.

**CONCLUSIONS**

The knowledge of the mechanical muscle parameters behavior and parallel electrical activity can’t be limited to the agonist muscle. The antagonist muscle clearly influenced the movement and contributed to maintain or improve joint stability. As [7] have reported on the agonist muscle, we have also observed a nonisometric behavior of the antagonist muscle. The similar evolution of coactivation ratio and architectural parameters indicated that the antagonist muscle was stiffer according to the applied force and improved joint stability. The increased coactivation ratio up to ~20% MVC and the isometric behaviour of the architectural parameters up to ~40% MVC reflect presumably the need to enhance progressively the stability of the joint because of a greater compliance of the muscle-tendon unit at low torque levels. We expect that this study will led to a better understanding of force transmission and muscular function.

**REFERENCES**