Antagonist activation of triceps brachii is greater than biceps brachii muscle

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Introduction
Muscle coactivation is the simultaneous activation of agonist and antagonist muscles acting on the same joint and has, among others, the mechanical effect of making a joint stiffer. Joint stiffness, in turn, is influenced by the velocity of movement being higher over the course of faster movements (1). Because coactivation increases the stiffness and the stability of a joint, it is likely that during fast movement coactivation could be greater but very few studies (2, 3) have been conducted on verifying this hypothesis. Coactivation of muscles surrounding joints such as the elbow has been poorly investigated in comparison to the knee, and there are no reports on the differences between elbow extensors and flexors when they act as antagonist. The aims of this study were: 1) to verify if the amount of antagonist activation is influenced by the speed of movement; 2) to investigate differences in antagonist activation between biceps brachii (BB) and triceps brachii (TB).

Methods
8 male and 4 female young healthy subjects (age 25.8 ± 4.4 yr; stature 175.2 ± 9.5 cm; weight 67.5 ± 13.2 kg) volunteered for this study. The elbow flexion and extension torques of the dominant limb were measured with an isokinetic dynamometer. The sEMG signals from the BB and from the TB of the dominant limb were recorded by means of a two linear array of 4 electrodes to enhance the spatial resolution of the sEMG and thus to reduce crosstalk (4). Participants were requested to perform 3 different tasks: (a) 3 maximal voluntary isometric contractions (IMVC); (b) 3 maximal isokinetic concentric contractions at 15, 30, 60, 120, 180, 240° s⁻¹ and (c) 3 maximal isokinetic eccentric contractions at 15° s⁻¹ (used for normalization). For both BB and TB, antagonist RMS amplitudes were calculated as the percentage of the average RMS amplitude obtained at the selected contraction with respect to the average RMS value of the maximal eccentric contraction at 15° s⁻¹. A repeated-measures ANOVA was used to assess statistical differences (P<0.05).

Results
No statistical differences were found between the two muscle groups and among the different angular velocities in agonist activation (fig.1). Antagonist %RMSmax for BB ranged from 5.7 ± 5.2 at IMVC to 18.6 ± 8.6 at 240° s⁻¹ whilst TB showed an antagonist %RMSmax ranging from 26.0 ± 19.0 at IMVC to 37.8 ± 13.9 at 240° s⁻¹ (fig.1). Statistical differences were present between the two muscles at each angular velocity (P<0.01). BB and TB antagonist activation was not velocity dependent even if activation of both muscles showed a trend to slightly increase as the velocity of movement increased. Torque values ranged from 70.5 ± 22.7 Nm at IMVC to 32.3 ± 12.5 at 240° s⁻¹ for EE and from 79.1 ± 32.1 Nm at IMVC to 42.4 ± 15.0 at 240° s⁻¹ for EE (fig.2). Despite the EE torque/velocity curve was above the EF curve, no statistical difference was found between the two data sets.

Discussion/Conclusion
In this study, we compared the EMG activity of BB and TB during isokinetic concentric exercises and we investigated the influence of angular velocity on their activation. The antagonist activation of BB was on average 16.2% lower than TB antagonist activation and this difference was statistically significant at each of the examined angular velocities. The greater coactivation of TB during EF could be responsible for the tendency of the EF torque/velocity curve of being below the EE curve. Contrary to our expectation, antagonist activation did not result velocity-dependent for both muscles. These findings suggest that a different specialization of the two muscles can be responsible for different levels of antagonist coactivation and that the elbow extensors may play a crucial role in the control of the forces generated around the elbow joint in order to guarantee the stiffness of the whole joint.

References