SEPARATE CONTROL OF AGONIST AND ANTAGONIST MUSCLES DURING FATIGUE

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Our recent observation (1) that the amplitude of the H-reflex in the antagonist muscles did not follow the same time course or pattern as the enhancement of the antagonist EMG activation, suggests that coactivation is controlled by supraspinal mechanisms.

The purpose of this study was to investigate the control mechanisms at the cortical and spinal (motor neurone) levels of antagonist coactivation during a submaximal fatiguing contraction of the elbow flexors at 50 % of maximal voluntary contraction (MVC). We recorded motor evoked potentials in the biceps brachii and triceps brachii muscles in response to magnetic stimulation of the motor cortex (MEP) and corticospinal tract (cervicomedullary motor evoked potentials – CMEP), as well as maximal M-wave (Mmax), before, during and after the fatigue task (3).

The results showed that although the coactivation ratio did not change at task failure, the MVC torque produced by the biceps brachii declined by 48% (P<0.01) with no change in MVC torque for the triceps brachii. In contrast to the MEP and CMEP areas (normalized to Mmax) of the biceps brachii that increased (50%) over the first 40% of the time to task failure and then plateaued, both responses in the triceps brachii increased (150-180%) gradually throughout the fatigue task. The lengthening of the silent period, that follows the MEP, increased to a similar extent in both agonist (53%) and antagonist (43%) muscles during the fatiguing contraction. However, at task failure, when the antagonist muscles acted as an agonist during an elbow extension MVC, the alteration of the silent period was not present and its duration was comparable to the pre-fatigue conditions.

In conclusion, a sustained contraction held at 50% MVC torque with the elbow flexors is accompanied by a progressive increase in activation of the antagonist muscle (triceps brachii). Because the coactivation ratio was the same at the onset and end of the fatigue task, there is no evidence that task failure is due to the opposite action of the antagonist muscles. Collectively, these results suggest that the level of coactivation is mediated by supraspinal rather than spinal mechanisms (1) and the agonist and antagonist muscles are controlled by independent descending inputs (2).