

THE ANTAGONIST MUSCLE PATTERN IN ELBOW EXTENSION OF A THROWING TASK

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INTRODUCTION

The myoelectric activity of agonist and antagonist muscles in fast human movements is characterised by a triphasic pattern. The most important events are the first agonist activation (AG1) responsible for the initial limb acceleration, and the antagonist activation (ANT), coincident with the electrical silence of the agonist muscle, and related with movement braking. If the role of the first agonist burst to the initial limb acceleration is obvious, the accurate definition of the antagonist impulse function is not clearly defined on the literature. The origins of this EMG event have been attributed to both peripheral and central sources, but the relative participation of each one has also carried out controversy (Angel, 1977, Marsden et al., 1983). The purpose of the present study was (1) to characterise the antagonistic pattern on the elbow extension when subjects had to perform an overarm throw onto a target, and (2) to analyse the antagonist changes when subjects throw with different accuracy/speed requirements.

METHODS

Experiments were performed on 13 subjects: 4 skilled dart throwers and 9 untrained subjects. The task was a dart throw to a concentric target at a distance of 3 meters. Each subject performed on three conditions with different constraints: (P) "try to reach the target centre", (C) "try to reach the target centre as fast as possible" and (V) "just try to be as fast as possible". Electrogoniometers on shoulder and elbow joints measured position and velocity movement parameters on each joint. Surface EMGs were recorded from triceps and biceps brachii with active bipolar electrodes. The angle, velocity and EMG signals were averaged in blocks of 20 trials representatives of each condition.

RESULTS

The antagonist EMG contained a phasic burst which began between the movement onset and the end of the agonist activation, with an usual co-contraction duration of less than 30 ms. The time interval between the movement onset and the beginning of ANT (Fig. 1) was normally higher than the EMG latency necessary for spindle influences, considered to be less than 20 ms (Tarkka, 1986), indicating that this burst could be influenced by the muscular response to stretch, although together with other peripheral influences and modulated by higher central commands. While the agonist activation is initially produced only by a central generator pattern, the antagonist burst is probably dependent on the interplay of a central program responsible for its beginning, with the participation of the peripheral

ifferences on duration and amplitude modulation processes. Because it is impossible to identify clearly the relative role of each factor on antagonist EMG patterns, the identification of the antagonist regulation mechanisms is more complex.

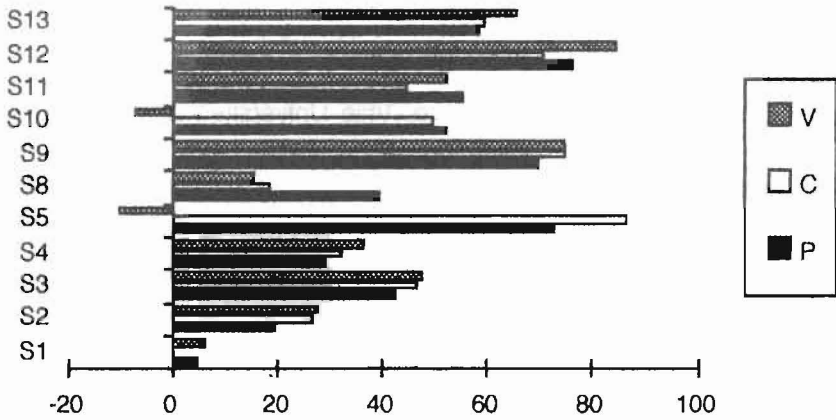


Fig. 1 - Time interval between movement initiation and the onset of antagonist phasic burst; on subjects S6 and S7 it was not possible to determine this parameter.

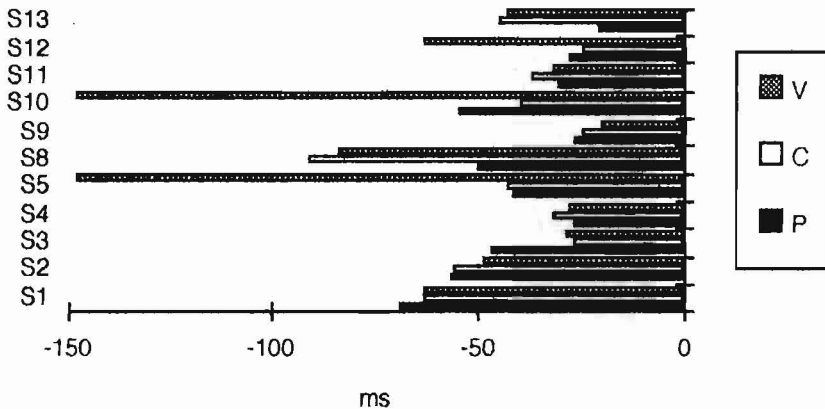


Fig. 2 - Time interval between the onset of antagonist phasic burst and the end of the acceleration phase; on subjects S6 and S7 it was not possible to determine this parameter.

The antagonist pulse always began during the acceleration phase and, in most cases, developed its maximum intensity around the moment of elbow peak velocity (Fig. 2). Two subjects (S5, S10) that showed a modification on the antagonist pattern on condition V, were exceptions. So, the antagonist burst seems to represent more than an impulse braking, preventing full extension, and we must admit its participation in the control of the end of the acceleration phase. These findings could be related to the hypothesis of

Wierzbicka et al. (1986) who postulated that, in very fast movements, the main role of the antagonist activation is to control movement time. In the task we have studied, the total movement time is meaningless, as there were no time or spatial requirements to interrupt the elbow extension. The possibility of a strong relation between the antagonist burst and the duration of the acceleration phase should be accepted. This idea is reinforced by the recent study of Jaric et al. (1995), that found that stronger antagonists, conditioned by a training program, could improve the performance of rapid elbow movements, since it facilitates the arrest of the movement in a short time, providing a longer time for acceleration and an increase in movement velocity.

Antagonist modifications with velocity indicated an amplitude increase, measured by the integrated EMG (Fig. 3), with the increase of the throwing velocity, maintaining the temporal structure of antagonist activation among conditions (Fig. 4).

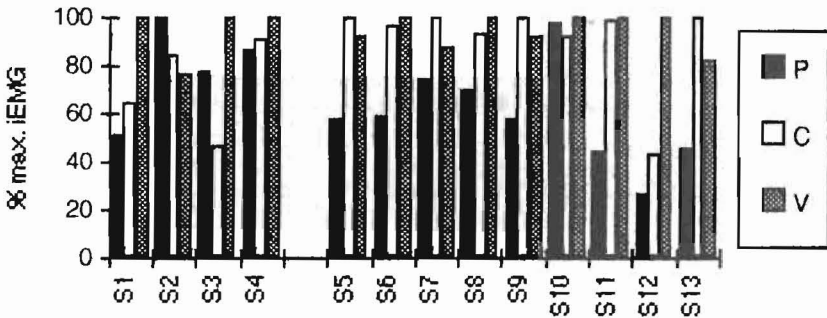


Fig. 3 - Integrated EMG of antagonist phasic burst (ANT) during the acceleration phase on the three conditions (P, C, V) of each subject.

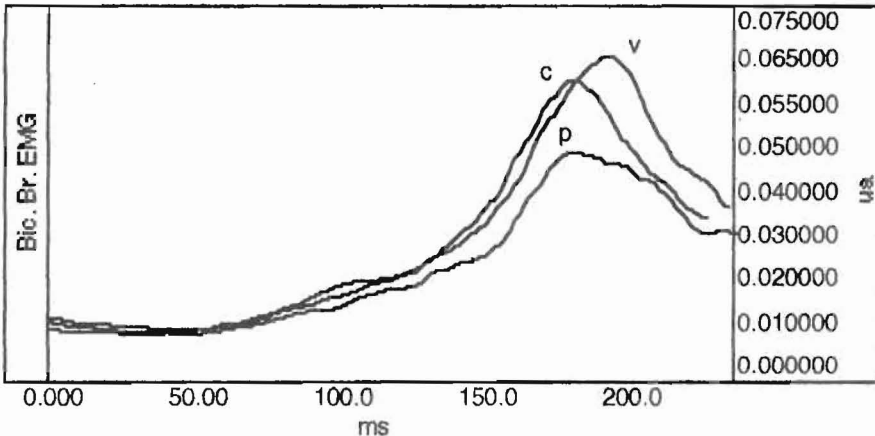


Fig. 4 - Averaged antagonist EMG of all subjects in the conditions P, C and V.

Two subjects (S5, S10) presented an alternative way of braking the movement, when the accuracy constraints were absent (condition V), based on the increase of the joint stiffness through the co-contraction of agonist

and antagonist muscles (Fig. 5). This kind of braking mechanism was proposed by Ghez and Martin (1982) and has a reduced timing accuracy demand when compared with the active braking, produced by the antagonist phasic burst.

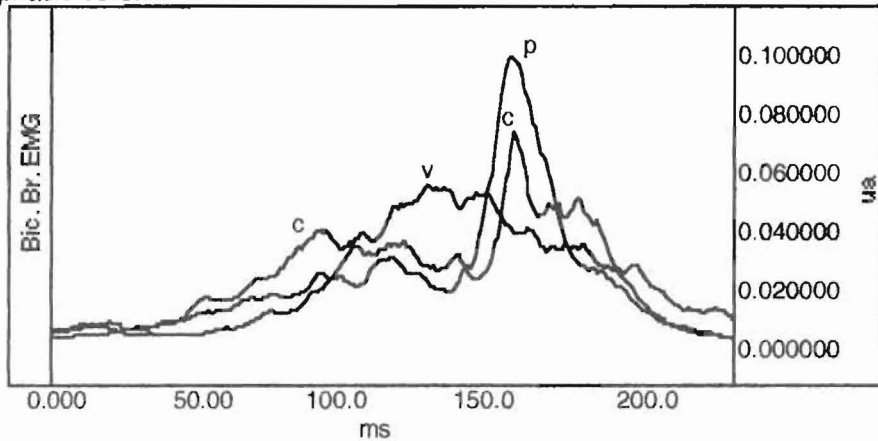


Fig. 5 - Averaged antagonist EMG of subject S10 on conditions P, C and V.

CONCLUSION

The timing of the antagonist phasic burst invite us (1) to associate it with the control of the end of the acceleration phase and (2) to accept that it could be influenced by the muscular response to stretch, although modulated by higher central commands. (3) The general tendency was to maintain the temporal structure of antagonist EMG among conditions and to increase its intensity with the increase of the throwing velocity. (4) Two subjects presented an alternative way of braking the movement when the accuracy constraints were absent.

REFERENCES

- Angel, R. (1977). Antagonist muscle activity during rapid arm movements: Central versus proprioceptive influences. *Journal of Neurology, Neurosurgery, and Psychiatry*, 40, 683-686.
- Ghez, C., & Martin, J. (1982). The control of rapid limb movement in the cat. III - Agonist - Antagonist coupling. *Experimental Brain Research*, 45, 115 - 125.
- Jaric, S., Ropret, R., Kukulj, M., & Ilic, D. (1995). Role of agonist and antagonist muscle strength in performance of rapid movements. *European Journal of Applied Physiology*, 71, 464-468.
- Marsden, C., Obeso, J., & Rothwell, J. (1983). The function of the antagonist muscle during fast limb movements in man. *Journal of Physiology*, 335, 1-13.
- Tarkka, I. (1986a). Short and long latency reflexes in human muscles following electrical and mechanical stimulation. *Acta Physiologica Scandinavica*, 128, Supplementum 557, 1-32.
- Wierzbicka, M., Wiegner, A., & Shahani, B. (1986). Role of agonist and antagonist muscles in fast arm movements in man. *Experimental Brain Research*, 63, 331-340.