EMG AND CINEMATIC PATTERNS ON ELBOW EXTENSION DURING A THROWING TASK PERFORMED AT DIFFERENT SPEEDS: THE MODULATION OF THE INITIAL AGONIST BURST (AG1)

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INTRODUCTION

A lot of experimental research has been done on in the past few years to understand how the Central Nervous System (CNS) programs ballistic movements performed at different speeds. Special emphasis has been given to the changes on initial agonist burst (AG1), the main responsible for the initial limb acceleration. The "dual-strategy" hypothesis (Corcos et al., 1989; Gottlieb et al., 1989, 1990a,b; Gottlieb, 1993) defends that the speed regulation is dependent on a "Speed-Sensitive Strategy" which actuates changing the excitation pulse height to MN, producing different initial rates of agonist EMG rise. The element that allows to identify this strategy is the initial slope of EMG agonist signal, and the integrated EMG of the first 30 ms of AG1 was proposed as an adequate measure of this element. The experimental support for the "dual-strategy" hypothesis was obtained in single-joint movements, performed on laboratorial environments. On one hand it became more attractive as a paradigm for testing hypothesis but, on the other hand, it didn't allow direct applications to more complex and "natural" movements. The aim of the present study is to investigate the neuromuscular mechanisms of agonist modulation which are responsible for the speed regulation of a forearm extension in the dart throwing to a target.

METHODS

Experiments were performed on thirteen right-handed males (four skilled dart throwers and nine untrained subjects. 20-35 years old). The task was to throw a dart to a concentric target at a distance of 2,37 m and 1.72 m of centre height. All subjects performed 30 trials on each of the three different conditions, after 30 training trials. Subjects were instructed before each training period: condition P "try to reach the target centre", condition C "try to reach the target centre throwing as fast as possible" and condition V "try to throw as fast as possible". 20 trials on each condition were selected for analysis.

To control if the goal of each condition was accomplished, the accuracy was measured by the target punctuation (1 represents the target centre) and a laser system was used to determine dart speed. Two electrogoniometers (EGN), on shoulder and elbow joints, measured the amplitude. time and velocity movement parameters on each joint. The shoulder EGN was used only to control changes on the arm participation. Surface EMG were recorded from the triceps (lateral head) and biceps brachii, with active bipolar electrodes. The EMG signals were digitised, together with the EGN signals. with a sample rate of 1000 Hz, and stored on a hard disk for later analysis. After acquisition, EGN signals were low pass filtered with a cut-off frequency of 16 Hz. Raw EMG were digitally filtered (20 to 500 Hi), full wave rectified and smoothed. The angle, velocity and EMG signals were normalised and averaged in blocks of 20 trials representative of each condition and subject.

The cinematic and EMG parameters were measured on the averaged signals of each subject. From the cinematic parameters taken, special attention was paid to the peak velocity of elbow extension (PV), and to the duration of the forearm acceleration phase (TA). The initial agonist EMG burst (AG1) was isolated to determine time and amplitude parameters: the burst duration (DAG1), the latency between movement onset

and time of maximum EMG peak (TPMAG1), the maximum peak value (PMAG1), the integrated EMG of the burst (iEMG AG1) and the integrated EMG of the first 30 ms of AG1 (iEMG30).

RESULTS AND DISCUSSION

The elbow movements were accomplished by a three- or biphasic EMG similar to the pattern normally found on rapid single-joint movements. The magnitude parameters of AG1 (iEMG and PM) increased with velocity. In the majority of cases, this increase was obtained without changes of DAG1, in agreement with the findings of invariance on AG1 duration on single-joint movements performed with different movement speeds (Freund & Budingen, 1978; Brown & Caoke, 1981; Brown & Gilleard, 1991). However, increases of DAG1 were seen in about 30% of the cases, confirming previous works with elbow movements (Wadman et al., 1979; Berardelli et al., 1984; Brown & Cooke, 1984) and supporting that the prolongation of the agonist activation represents a possible additional mechanism that can be used by the CNS to increase speed on fast movements.

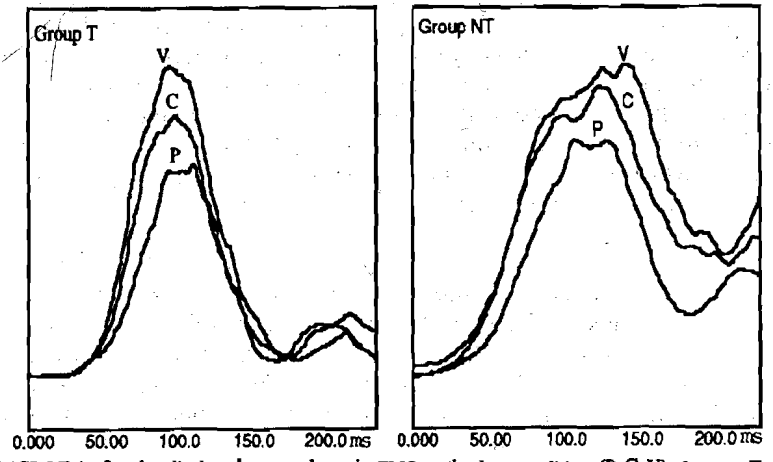

FIGURE 1 - Overlap display of averaged agonist EMG on the three conditions (P, C, V) of groups T and NT.

The comparison between different conditions shows that the closer relation with the increase of PV was the increase of the initial rate of agonist rise. This is evidenced by the qualitative analysis of the agonist EMG envelope (Figure 1) and by the increase of iEMG30 values (Figure 2), when subjects changed from condition P to condition C. However, when subjects passed to condition V, this increase on the initial slope of AG1 was not always present, suggesting the exhaustion of this mechanism in condition C for some subjects. This was specially evident in the NT group and was reflected on the averaged EMG signals representing this group, with a total superposition of C and V on the first 50 ms. The incapacity to produce an additional increase in the initial rate development seems to be compensated on these subjects by the prolongation of high intensity levels of agonist activation. The capacity to increase motor unit recruitment in a short period seems to be saturated, and insufficient to' increase the acceleration impulse necessary to perform faster movements. Thinking this way, the increase of AG1 duration represents a secondary mechanism that can be used by CNS.

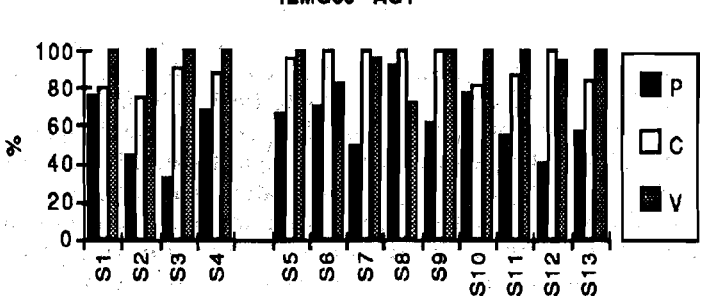

FIGURE 2 - Parameters of AG1 on the three conditions (P, C, V) of each subject: duration (DAG1), and percentage values of maximum EMG peak (PM AG1), integrated EMG (iEMG AG1), and integrated EMG of the first 30 ms (iEMG). The maximum reference for each subject was the highest value of each parameter on the three conditions.

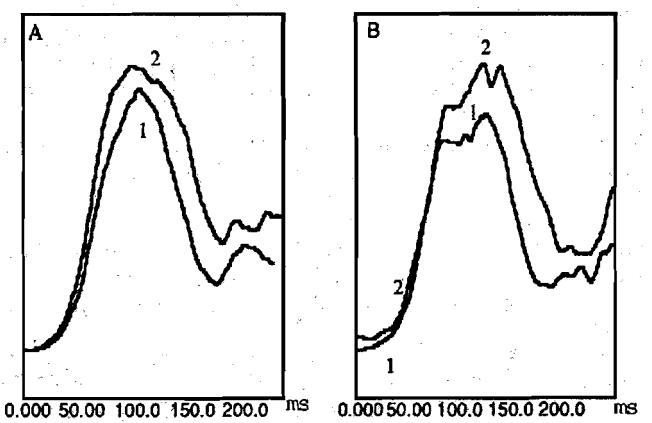

FIGURE 3 - Averaged EMG signals representing two different profiles of elbow adaptation (A, B). The curve 1 is the mean signal of the first moment (P or C) and the averaged EMG signal of the second moment (C or V) is the curve 2.

The relation between these neuromuscular mechanisms and the elbow cinematic can be more clearly understood combining the signals in a different way. The EMG signals were grouped, normalised and averaged according to the elbow cinematic adaptation profile (Figure 3): increase in PV with identical TA (A), and increase in PV and TA (B). Group A showed a different inital EMG development and a clear increase in the iEMG30 with elbow velocity. On the contrary, in the group B the iEMG30 was found to remain constant and the fundamental mechanism was the AG1 prolongation. The motor system modulated the duration of the pulse to the agonist motoneurones, without modifications of its amplitude. The initial rate of EMG rise did not change and the prolongation of the agonist activation is propably related to the larger acceleration phase. According to the "dual-strategy" model, group A reflected the choice of a "Speed-Sensitive Strategy", and the changes on group B agreed with a "Speed-Insentive Strategy".

CONCLUSIONS

The increase in the initial rate of agonist EMG rise, measured by the integrated EMG of the first 30 ms of AG1 (iEMG30), revealed to be the most related variable to the elbow movement speed. This finding agrees with the "dual-strategy" model (Corcos et al., 1989; Gottlieb et al., 1989, 1990a, b; Gottlieb, 1993). which recently described the intensity of the excitation pulse to agonist MN pool as the fundamental variable controlled by CNS to produce an increase in movement speed. By a low-pass filtering effect, the MN pool transforms changes in excitation pulse amplitude into different initial slopes of EMG. As a secondary mechanism, the prolongation of AG1 was also used when the increase in the initial agonist rate was not sufficient. This mechanism was more evident on untrained subjects, and it was associated with the increase in the duration of the acceleration phase. Besides the differences due to the level of practice. the results showed that the neuromuscular regulation of velocity has a certain degree of inter-individual variability, with individual differences among the use of the mechanisms for agonist modulation.

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