

ROLE PLAYED BY THE ANTAGONISTS IN THE CONTROL
OF A VOLUNTARY MOVEMENT

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Summary

The role played by the antagonistic muscles in the braking of voluntary movements is considered in connection with the proportional control of prostheses and orthoses. An experimental investigation of this role in the case of elbow flexion in normal subjects is described.

Results showed consistent patterns of muscular activity. Depending on the value of force exerted by the agonist muscles, two different mechanisms appear to be involved in the limitation of amplitude of the movement. For lower force values, visco-elastic forces arising from the passive lengthening of the antagonist are sufficient to brake the movement. For higher force values, contraction of the antagonist is required, and this requires an increasing stiffening of this muscle.

These results suggest a method of control movement of prostheses and orthoses by providing stiffness based on the level of EMG activity of the antagonist.

Introduction

The limitation of amplitude of a voluntary movement in a normal subject involves the operation of braking forces to oppose those which initiate the movement. According to the conditions in which the movement is carried out, in particular the velocity of the movement and the nature and intensity of the opposing resistances, these braking forces may be mediated either by the contractile forces exerted by the antagonist muscles, or simply by the visco-elastic forces exerted by these muscles /1,2/. The need for the same sort of a braking system appropriate to the movement concerned must be considered in the case of proportional control of prostheses and orthoses. If the principle of myoelectric control is to be used, the braking system should, as much as possible, be based on the electromyographic activity of the muscles which play the role of antagonist in the natural movement /3,4/. A better understanding of the role played by the antagonists in the braking of a voluntary movement would thus seem to be desirable.

Research supported by a grant from the Délégation Générale à la Recherche Scientifique (No. 7270251)

The aim of the present study was to examine this role in the case of flexion of the elbow in normal subjects. Such a single-joint movement can be considered as a functional unit of movement, and as such permits a precise analysis of patterns of muscular activity. Furthermore, the results of such an investigation should lend themselves to potential application to both prostheses for the elbow and orthoses for the forearm. The movements studied in this investigation were carried out against light inertia loads thus corresponding to a range of normal movements.

Methods

The subjects were required to perform unidirectional flexions of the right elbow, the arm and forearm being situated in the same horizontal plane. The hand was maintained in semi-pronation and the wrist immobilized. The subjects were asked to limit the amplitude of their movements by aiming approximately at a reference point situated at an angle of 110 degrees to the fully extended forearm; this provided for an amplitude of approximately 40 degrees, i.e. plus or minus 20 degrees (± 0.349 rad.) on either side of the orthogonal position of the arm and forearm.

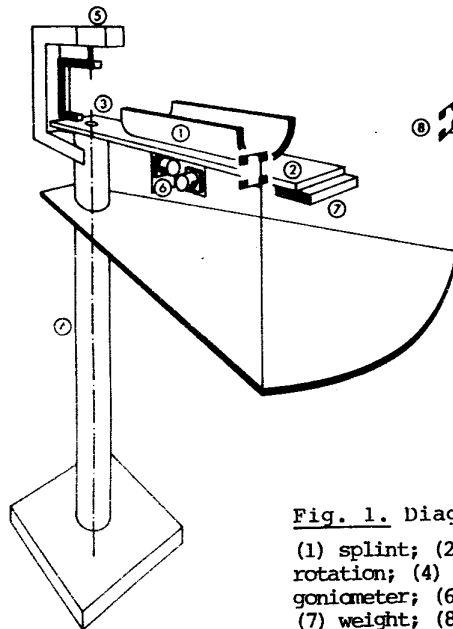


Fig. 1. Diagram of apparatus.

(1) splint; (2) plank; (3) axis of rotation; (4) support column; (5) goniometer; (6) accelerometer; (7) weight; (8) reference point.

As in previous study /5/, the forearm was held in a splint, fixed to a plank which rotated about a vertical axis coinciding with the rotation axis of the elbow; this mechanical system was fitted with a tangential accelerometer and goniometer (Fig. 1). The instantaneous values of angular acceleration (θ'') and angular displacement (θ) were thus directly obtained. The angular velocity (θ') was obtained by continuous differentiation of the signal θ . The EMG of an agonistic muscle, the biceps brachii (EMG-B), and its antagonist, the triceps (EMG-T), were recorded by means of bipolar surface electrodes placed on the belly of the biceps and the long head of the triceps. EMG "envelopes" for each muscle (EMGeB and EMGeT) were obtained by rectification and filtering of muscle potentials by means of a device consisting of three RC circuits in series /6/. Integrated EMG's for the biceps (Q_B) and triceps (Q_T) were obtained by means of an analogue-converter which delivered a number of impulses proportional to the surface EMG for each muscle /7/. The instantaneous values of the force (F) of the agonistic muscle group (represented here by a single muscle of the same anatomical dimensions as the biceps (Fig. 2) and termed the "Equivalent Flexor" /8/) were determined by means of an on-line analogue computer. After suitable amplification, these different variables were recorded by means of moving magnet oscillographs with an appropriate

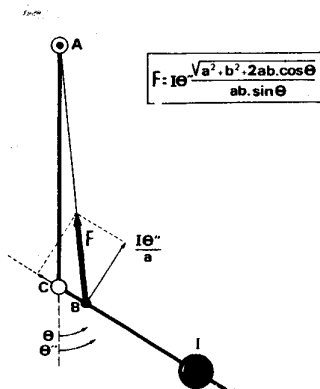


Fig. 2. Scheme for calculating force F of the "Equivalent Flexor".

C - rotation axis of elbow; A,B - upper and lower insertions of the biceps; a,b - distances CB, CA; F - force developed by "Equivalent Flexor"; I - moment of inertia of the forearm plus hand, together with moveable part of the mechanical system; θ'' - angular acceleration; θ - angle of forearm in relation to its position in complete extension. "a" and "b" were taken from classical measures; "I" was determined experimentally /20/.

frequency range.

Three series of experiments were carried out on five, presumably normal, male adults. Subjects and inertia values were varied between series. Inertia was varied by means of light weights attached to the plank (Fig. 1) at a distance of 26 cm. from the rotation

axis. In all, four inertia values were used, corresponding to 0, 1, 2 or 3 kg. additional weights. For any given inertia condition, each subject was first required to perform the movement with his own natural velocity and then with slower or faster rates according to the direction of the experimenter. Five separate movements were carried out in each case.

The subjects were asked to make sure that their movements were continuous and that they should respect the amplitude limits indicated by the reference point. They were also requested to relax their muscles as much as possible before each movement.

Results

Three distinct types of muscular activity patterns were found. This classification was made on the basis of a simple temporal characteristic of the EMG - a period during which the biceps and triceps were simultaneously relaxed - which constituted a regular feature of one of the three types of patterns (Fig. 3). This type

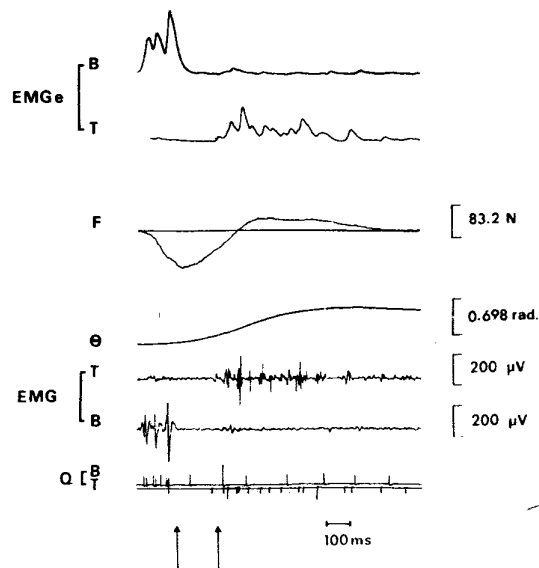


Fig. 3. Experimental tracings for a Type-S movement.

From top to bottom: EMGe B, T - EMG envelopes for biceps and triceps; F - force of "Equivalent Flexor" (in Newtons); θ - angular displacement (in radians); EMG-T, EMG-B - surface EMG activity for triceps and biceps; Q_B , Q_T - integrated EMG's for biceps and triceps.

Distance between arrows at bottom of figure indicates period of common electromyographical silence. Calibrations shown on right-hand side and bottom of figure.

(Type S) corresponded to the movements performed at the subject's own natural velocity. It consisted of discrete successive phases of EMG activity from the biceps and triceps respectively, separated by the common relaxation period referred to above.

This period of electromyographical silence is not present in either the slower (Type L) or faster (Type R) movements. Type L is characterised by continuous agonistic activity (Fig. 4). It should be noted that the level of this activity, although very low, is considerably higher than the background noise ($5 \mu\text{V}$), with which it should not be confused. Type R is characterised by a partial overlap of phasic activity in the agonist and antagonist muscles.

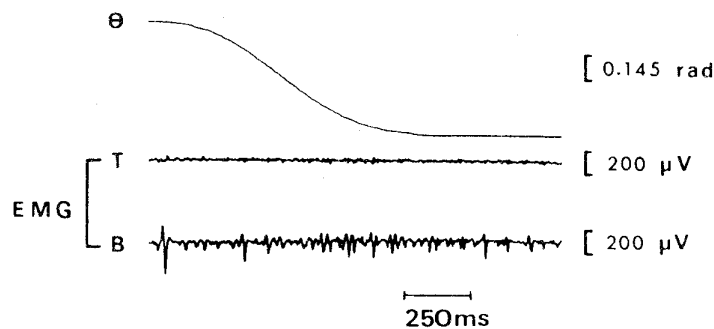


Fig. 4. Experimental tracings for a Type-L movement.

From top to bottom: θ - angular displacement (in radians); EMG-T, EMG-B - surface EMG activity for triceps and biceps. Note that the θ tracing is reversed in relation to that shown in Fig. 3. Calibrations shown on right-hand side and bottom of figure.

These three types of patterns of muscular activity were found to be independent of the subject or the inertia value. They occurred successively at increasing values of maximal force F exerted by the "Equivalent Flexor" during a movement (Fig. 5) and at correspondingly increasing levels of EMG activity of agonist muscle.

Moreover, since an F value could be assigned to each movement, it was possible to quantify upper and lower limits for the Type-S movements. These limits were consistent for each subject, and independent of the inertia value used. For each of these upper and lower limits, one and only one F value was found. In other words, the pattern of reciprocal EMG activity of agonist and antagonist was related to the force value given by the "Equivalent Flexor".

According to whether the force value F is above or below the lower limit of Type S, two different physiological mechanisms appear to be involved in the limitation of the amplitude of movement. In effect, if the value of F is lower than F_0 ($20 \text{ N} < F_0 < 30 \text{ N}$), no contraction of the antagonist muscle is required to limit the amplitude of the movement. This is the case for Type L. By contrast, antagonistic activity comes into play in movements where F values higher than F_0 are involved (Types S and R). Furthermore, in these two types, the higher the F value the sooner the EMG activity of the antagonist will appear and the higher will its level be.

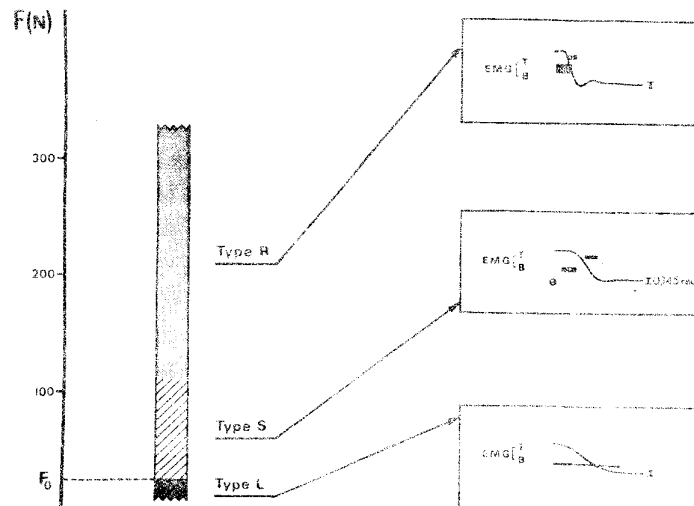


Fig. 5. Types of muscular activity pattern in relation to force F .

The three types of muscular activity pattern are shown schematically in relation to the force value exerted by the "Equivalent Flexor". For each type of pattern the angular displacement (θ) is shown, together with EMG activity, represented by the horizontal lines (level of activity indicated by thickness of line). For F values below F_0 (the lower limit for Type-S movements), passive antagonist forces alone are sufficient to brake the movement. (Diagram based on data taken from a single subject).

On the basis of the coefficients of elasticity and viscosity measured in inactive muscles (Lestienne and Pertuzon, to be published), it can be shown that the passive forces F' , which oppose the movement, cannot exceed an approximate value of 25 N. These

passive forces are essentially the opposing elastic forces due to the lengthening of the antagonist muscle. From the correspondence between F_0 and F' it follows that Type-L movements can be braked by passive forces alone; in this case the angular velocity is never higher than 1.5 rad/s for the lowest inertia value.

Discussion

From the physiological aspect, the first point to be emphasized is the consistency of the patterns of muscular activity. As already pointed out, the limits for Type S were consistent both for the subjects and the tests. This consistency indicates the representative character of the muscles examined in this investigation - that is, the biceps can be considered as a representative flexor, and the long head of the triceps as a representative extensor. This fact has already been stressed by Wachholder and Altenburger /2/, who regarded the biceps-triceps activity patterns as general representatives of agonist-antagonist patterns, at least for the type of movements investigated. This point is of some importance since it justifies the investigation of a single muscle in each group, and in particular, the use of the notion of an "Equivalent Flexor".

Movements of Type R and Type L should be considered separately. The main characteristic of Type-R movements is the partial overlapping of EMG activity recorded from the biceps brachii and triceps - a clear exception to the law of reciprocal agonist-antagonist inhibition. (Indeed, as certain authors, notably Person /9/ have pointed out, this law does not inevitably apply in cases of natural movement). From the present results it would appear that the beginning of antagonist EMG activity depends on the precise biomechanical conditions in which the movement is carried out.

In the case of Type-L movements, there is, in fact, no EMG activity from the antagonist muscle. It is here that we find evidence of the part played by passive forces, visco-elastic in origin, in the limitation of the amplitude of voluntary movements. In Type-L movements the active forces exerted by the "Equivalent Flexor" are low ($F < F_0$), and are balanced almost continuously by the compensating visco-elastic forces developed in the lengthened antagonist. The important role of visco-elastic or rheologic factors has similarly been emphasized by Long et al. /10/ in the control of hand-movements.

When higher levels of force are exerted by the agonist muscles ($F > F_0$), the braking of a movement at the required amplitude now depends on the activation of the antagonist. This activation will occur more or less rapidly according to the F value. This phenomenon is accompanied by an increase in the integrated EMG of the antagonist and hence in the force which is exerted /11/. As the increase in force developed by a muscle is accompanied by an increase in its stiffness /12, 13, 14/, it could be said that the limitation of amplitude of a movement is brought about by the increasing stiffness of the antagonist. Furthermore, the latter is proportional to the force developed by the agonist.

With regard to the potential application of these discoveries to the control of prostheses and orthoses, certain possibilities are suggested by the good reproducibility of patterns of muscular activity /15/. In the case of prostheses, amputation above the elbow is frequently followed by degeneration of the triceps /16/. Even if no such degeneration takes place, it is evident that the visco-elastic properties of this muscle cannot be used; furthermore, it is likely that the EMG signal from the triceps may not be sufficiently clear to permit a limitation of the movement amplitude comparable to that achieved by a normal subject. However, bearing in mind the relationship noted above concerning the electrical activity of the biceps and the stiffness of the triceps, we should perhaps consider the possibility of controlling the amplitudes of movements of artificial limbs by means of a program of variable stiffness, based upon the level of EMG activity of the biceps.

In the case of orthoses, it is necessary to consider the fact that the stiffness of the paralyzed antagonist muscles is likely to be markedly different from that of normal subjects. As far as EMG activity is concerned, this is in any case at a reduced level in normal subjects, since it occurs simultaneously with the lengthening of the antagonist muscles /17, 18, 19/; with weakened muscles the level of activity is of course even further reduced. Here again, the possibility of variable stiffness program appears to provide an attractive solution to the problem of movement braking.

These tentative suggestions concerning the braking of movements of prostheses and orthoses would apply in the case where the muscles which normally control the movement are still at least partially functional. They also appear to be relevant in cases where

other muscles have to be used for providing myoelectric control. Finally, the emphasis which has been placed on the role of viscoelastic forces should not exclude consideration of the important role of positional feed-back in the precise control of movement /4/. It is, however, probable that such feed-back control would itself be subject to modification by the braking system proposed above.

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