Research Note

EMG Patterns in Antagonist Muscles During Isometric Contraction in Man: Relations to Response Dynamics*

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Summary. We studied the EMG activity of biceps and triceps in human subjects during isometric force adjustments at the elbow. Rapid targeted force pulses exhibited stereotyped trajectories in which peak force was a linear function of the derivatives of force and the time to peak force was largely independent of its amplitude. These responses were associated with an alternating triphasic pattern of EMG bursts in agonist and antagonist muscles similar to that previously described for rapid limb movements. When the instructions demanded rapid force pulses, initial agonist bursts were of constant duration, and their magnitude was strongly related to peak force achieved. The timing of EMG bursts in antagonist pairs was closely coupled to the dynamics of the force trajectory, and the rising phase of the force was determined by both agonist and antagonist bursts. When peak force was kept constant and rise time systematically varied, the presence and magnitude of antagonist and late agonist bursts were dependent on the rate of rise of force, appearing at a threshold value and then increasing in proportion to this parameter. It is proposed that antagonist activity compensates for nonlinearity in muscle properties to enable the linear scaling of targeted forces which characterizes performance in this task.

Key words: Triphasic EMG patterns – Isometric contraction – Muscle mechanics

Introduction

An alternating triphasic pattern of EMG bursts in agonist and antagonist muscles is known to characterize rapid limb movements (Wacholder and Altenburger 1926). An initial burst in the agonist (AG1) is followed by a silent period coinciding with a burst in the antagonist (ANT) and then by a second burst in the agonist (AG2). AG1 initially accelerates the limb, while ANT has generally been held to be specifically responsible for terminal deceleration (Hallett et al. 1975; Lestienne 1979; Marsden et al. 1983). Although the magnitude of ANT varies proportionally with limb velocity (Bouisset and Lestienne 1974) this relationship is subject to other complex dependencies (Marsden et al. 1983), particularly ones related to loads opposing movement (Lestienne 1979), intended torque (Ghez and Martin 1982), and instruction set (Waters and Strick 1981). The significance and mechanisms of these interactions are poorly understood, largely because the direct output of muscles is force, and the relationship between an intended position change and the associated muscle forces is not a unique one. To isolate the effect of intended torque from intended change in position, we have examined the relations between response dynamics and EMG activity in an antagonist pair over a wide range of forces and rise times under isometric conditions. The present results suggest that specific patterns of activation of opposing muscles at a joint are constrained by, and compensate for, frequency-dependent mechanical properties of muscles as well as the stereotyped features of motoneuron recruitment.

Methods

Experiments were conducted on three neurologically normal adult subjects, seated with right shoulder abducted to 90° and elbow flexed to 90°. The right forearm was fixed in a rigid cuff coupled to a strain gauge sensitive to flexion and extension torques generated at the elbow. The upper arm and shoulder were restrained to prevent movement of the elbow joint. The subject's force output
and a target level were displayed as vertically moving horizontal lines on an oscilloscope. Subjects were trained to align the displayed force with the target level and to produce accurate responses to changes in target level of varied amplitudes (40 to 200 Newtons). Two task conditions were examined. In the first (rapid pulse condition), the target was stepped and the subjects were instructed to produce a transient pulse of force with as brief a rise time as possible to just reach the new target level. In the second (matched trajectory condition), the force target was shifted to a different level with a one-half-cycle sinusoidal waveform (rise times 80–400 ms); the subject’s task was to then match both the height and rise time of the target shift, which thus served as a template. Performance of this task was greatly facilitated by presenting to the subjects, along with the visual display, a frequency modulated audio signal first of the target and then of their force trajectory. The audio signal, provided through earphones, enabled subjects to produce force trajectories that were both smooth and accurately matched to the contour and amplitude of the previously presented template.

Under both task conditions, subjects were instructed to aim their force pulses as accurately as possible, but not to correct the response once it had begun. Additionally, they were to allow force to return passively to baseline after peak force was achieved. Target shifts were presented in blocks of 25 trials in which amplitude and rise time (for the matched trajectory condition) were maintained constant. No reaction time constraints were imposed.

The experiments were controlled and data acquired (bin width 2 ms) by a digital computer (PDP 11/T03). EMG signals from biceps and triceps were recorded through pairs of surface electrodes and integrated according to a previously discussed method (Ghez and Martin 1982) which allowed the digitized signal to follow closely the rise and fall of the EMG envelope. Trials were excluded from analysis if the responses showed evidence of corrections during the rising phase of the force trajectory. The criterion for exclusion was two or more peaks in the first positive wave of the first derivative of force (dF/dt).

Results and Discussion

Our subjects’ rapid force pulses exhibited highly stereotyped trajectories over a wide range of
amplitudes: the peak force achieved was a tight linear function of the peaks of the first ($r = 0.90-0.98$) and second ($r = 0.8-0.95$) derivatives of force. The time to achieve peak force was largely independent of peak force ($r = 0.01-0.4$) and averaged 65-100 ms across subjects and sessions. Thus, different peak forces aimed to different target amplitudes were produced by proportional changes in the early derivatives of the force trajectory. This preferred strategy of amplitude rather than width (i.e. duration) modulation of an underlying stereotyped waveform has previously been described for rapid isometric contractions by human subjects (Freund and Budingen 1978) and by cats (Ghez and Vicario 1978). The fact that early dynamics of the trajectory (in particular, peak $d^2F/dt^2$, which occurs at about 25% of the force rise time) were highly predictive of the ensuing peak force and were scaled to the magnitude of the target displacements, indicates that response amplitude is largely preprogrammed and that descending commands specifically control the derivatives of force (Ghez 1979).

EMG patterns associated with rapid force pulses were also highly stereotyped. Alternating bursts in agonist and antagonist were present for all response amplitudes and had a fixed temporal relationship to the dynamic features of the force trajectory. This temporal relationship is illustrated in Fig. 1. In the agonist, an initial burst (AG1) terminates at the time of the peak $dF/dt$. Following a silent period, one (AG2) or more late bursts in the agonist occur during the falling phase of the force. The antagonist muscle exhibits a small amount of coactivation (ANT-Co) during AG1, but at or just prior to the peak $dF/dt$ a large reciprocal burst (ANT-R) occurs, which lasts throughout the falling phase of the $dF/dt$. The result of these relationships is that AG1 and ANT-R lead the positive and negative peaks of the second derivative of force ($d^2F/dt^2$), respectively (dotted line superimposed on the EMG in Fig. 1). In this and other subjects, the integrated value of the EMG signal comprising AG1 showed steep linear relations ($r = 0.70-0.80$) to the peak force. ANT-R showed a weaker correlation ($r = 0.30-0.40$) and a 50% lower
slope in relation to this parameter. However, ANT-R was correlated with the magnitude of the negative peak of $dF/dt^2$ ($r = 0.50-0.65$). When tonic activity was present in the agonist prior to the response, an early pause preceded AG1 and, as this initial tonic activity was increased (by increasing the initial force requirement), late agonist bursts increased in size and number.

The alternating character of the EMG burst pattern was highly dependent on the rate of rise of force. This specific relationship was investigated by using the matched trajectory condition to vary rise time of force independently of peak force. Figure 2 shows a typical series of responses of similar size but different rise times; the raster is sorted (from top to bottom) in order of decreasing time to peak force. Here, the integrated EMG has been transformed into a frequency modulated pulse train (Evarts 1974). For long rise times ($> 200$ ms, region ‘a’ in Fig. 2), agonist and coactive antagonist activity last throughout the rising phase of the force and there is neither a distinct silent period nor reciprocal bursts. For intermediate rise times (120–200 ms, region ‘b’ in Fig. 2), agonist activity becomes clustered into a distinct burst terminating at the time of peak dF/dt, and is followed by a silent period. It should be noted that in this region peak force (circles on Fig. 2) occurs well after the termination of the agonist burst. Only for the fastest rise times ($< 120$ ms, region ‘c’ in Fig. 2) does ANT-R appear, along with late agonist bursts. When peak force was kept constant and force rise time varied, ANT-R, when present, was strongly correlated with rise time ($r = 0.6$) while AG1 showed minimal dependence on this parameter ($r = 0.3$). As can be seen in Fig. 2, the duration of AG1 varied directly with the rise time of force in region ‘a’ and with that of dF/dt in regions ‘b’ and ‘c’.

These data suggest that the stereotyped dynamics of force trajectories of rapid force pulses are determined by a specific temporal pattern of commands to both agonist and antagonist muscles. In rapid pulses the rising phase of force is shaped by an alternating pattern of agonist and antagonist bursts temporally divided at the peak dF/dt. The subject modulates peak force by adjusting the amplitude of AG1. AG2 and other late agonist bursts appear to be unrelated to task demands since they actually retard the return of force to baseline. They are only apparent in the fastest pulses, and they may be the consequence of initial synchronization of motor unit firing in rapid contraction (Desmedt and Godaux 1977) coupled with descending facilitation of agonist motoneurons outlasting the initial burst. While ANT-R may contribute to returning force to baseline, both its consistent temporal lead over the negative peak of the second derivative of force ($d^2F/dt^2$) and its correlation with this parameter indicate that it functions to curtail the rising force. The emergence of ANT-R in the most rapid force pulses is appropriate to counteract low pass properties of muscles (Partridge 1965), since in the most rapid pulses the mechanical effect of the initial agonist burst will last well beyond the termination of the burst itself. It may also compensate for non-linear summation of muscle tension associated with high frequency activation (Burke et al. 1976) of motor units. Thus, as a population of motor units is recruited over a shorter duration, relatively more antagonist compensation would be required to maintain proportional scaling of force output. The fact that ANT-R shows little dependence on peak force in the most rapid pulses may be related to the observation that increasing peak forces are achieved by the recruitment of motor units with shorter twitch contraction times (Henneman and Mendell 1981). Thus, control strategies appear to be adapted to the stereotyped properties of motoneurons and muscles. Since limb deceleration does not occur under isometric conditions, the present results emphasize that the functions subserved by reciprocal antagonist activity must be considered in relation to how mechanical and segmental constraints affect the control requirements of the task.

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References


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