Rapid elbow flexion in the absence of proprioceptive and cutaneous feedback

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Summary. Rapid goal-directed movements of elbow flexion were studied in normal human subjects and in patients deprived of proprioceptive and cutaneous feedback. All normal subjects showed a burst of electromyographic (EMG) activity in the extensor muscle (antagonist) that served to arrest the limb precisely in the target zone. The magnitude of this burst covaried with the magnitude of the initial accelerating burst in the flexor muscle (agonist). In patients, there was a small decelerating burst poorly correlated with the agonist activity. All patients had difficulty to control the amplitude of their movements due to improper adjustment of the size and time of onset of the decelerating burst. It is concluded that the central nervous system can generate a sequence of commands to accelerate and decelerate a limb in the absence of peripheral feedback. However, information from the moving limb is required to adjust the magnitude and time of onset of deceleration.

Key words: Muscle activity - Voluntary movement - Deafferentation - Proprioceptive and cutaneous feedback - Central motor command

The exact role played by peripheral feedback in motor control is still a matter of debate. Most of the studies involving surgical deafferentation in man and in experimental animals have reported different degrees of motor deficit following the lesion, thus emphasizing the importance of somesthetic afferent input for the execution of voluntary movements (Mott and Sherrington 1895; Lassek 1953; Twitchell 1954; Knapp et al. 1963; Taub and Herman 1963; Nathan and Sears 1960; Bossom 1974; Taub et al. 1975). The recent demonstration that trained monkeys can still perform correct elbow movements after surgical deafferentation of the limb (Polit and Bizzi 1979) has revived the idea that central brain activity can control movement adequately without the need for sensory feedback (Lashley 1917; Feldman 1974, 1980; Kelso and Holt 1980).

One of the simplest voluntary motor tasks is a rapid monoarticular movement towards a target. Such a movement requires active acceleration and deceleration of the limb and is produced by a sequence of EMG bursts in agonist and antagonist muscles sometimes followed by a second burst in the agonist, the so-called triphasic pattern. This pattern has been observed during rapid arm (Garland and Angel 1971), forearm (Wachholder 1928; Barnett and Harding 1955; Lestienne et Bouisset 1967; Terzuolo et al. 1973, Hallet et al. 1975; Lestienne 1979; Brown and Cooke 1981a; Benecke et al. 1985), wrist (Sanes and Jennings 1984) and finger movements in man (Hallet and Marsden 1979; Meinck et al. 1984). Some investigators have suggested that the somesthetic afferents play a major role in the generation of the antagonist burst (Barnett and Harding 1955; Terzuolo et al. 1974; Ghez and Martin 1982). Others have reported that the activity in the antagonist persists in the absence of sensory feedback thus indicating that it is generated by a central command (Hallet et al. 1975; Rothwell et al. 1982; Sanes and Jennings 1984).

We had the opportunity to study three patients deprived of proprioceptive and cutaneous peripheral afferents but with an intact peripheral motor system. We found that these patients can indeed generate an antagonist burst. However, a quantitative comparison of these results with those obtained in normal subjects revealed the importance of peripheral afferent feedback in regulating the size and timing of the antagonist activity. Preliminary accounts of some of these results have been presented elsewhere (Forget and Lamarre 1982, 1983).

Methods

We have studied three patients (GL, RV, and JD) with selective sensory neuropathy involving primarily the larger cutaneous and muscular afferents. These patients showed no signs of lesions of the peripheral motor system.

Patient GL, a 34-year-old woman, was the most severely affected. She had been functionally deafferented following a second episode of
Electromyographic activity was recorded from the biceps brachii and the lateral head of the triceps with surface electrodes (Beckman Ag-AgCl of 9mm diameter). In some experiments EMG activity was also recorded from the long head of the triceps and the anconeus. The EMG activity was bandpass filtered (100 to 3000 Hz), amplified in a conventional manner, full-wave rectified and converted from voltage to frequency (time constant of 1ms) as in the method described by Evarts (1974). Selected data were recorded on multichannel magnetic tape or directly on photographic paper. A laboratory computer was used for on-line data acquisition and control of the task. The pulse intervals generated by the EMG activity were measured with a resolution of 1 ms and the angular displacement signal was digitized at 200 Hz. Sampling of the data started 500ms before the presentation of the tone and lasted for 3s.

Movement onset was detected from the position signal by a simple algorithm that measured the occurrence of the first of 10 consecutive samples that changed in the direction of flexion. The end-point of flexion was determined as the occurrence of the first of 3 consecutive samples with either the same value or that changed in the opposite direction (i.e. extension). The end-point of a continuous movement thus determined the end-point of flexion. A continuous movement was defined, in our experiments, as a flexion with only one phase of acceleration and deceleration. Movement velocity and acceleration were computed by three-point numerical differentiation and appropriate digital filtering. The EMG activity was displayed in raster form and could be aligned with any of the behavioral events measured, including movement onset and the time of occurrence of peak velocity, acceleration and deceleration. Software routines allowed correlation studies between the magnitude of EMG activity, movement amplitude, and maximal values of velocity, acceleration and deceleration.

Results

Characteristics of the EMG activity in normal subjects

The rapid elbow flexion towards a target was, in all normal subjects, accompanied by a triphasic pattern of muscular activity which was characterized by two bursts in the agonist biceps and a burst in the antagonist triceps occurring between the two agonist bursts. This is illustrated in Figure 1 for 90° (A) and 40° (B) flexions. Since for both amplitudes of movement the target position was the same (between 110° and 120° of elbow flexion), the starting position of the 90° movement was located further in extension and required stronger tonic EMG activity in the extensor muscles to maintain this position. In most instances, the cessation of the tonic triceps activity was the first change observed and this occurred some 20 to 40ms before the onset of the first agonist burst.

The detailed features of the triphasic pattern are presented in Figure 2. EMG activity in the agonist biceps (A-C) and the antagonist triceps (D-F) is aligned with the onset of movement, peak velocity and peak deceleration. Trials have been arranged in order of increasing magnitude of peak velocity (range: 286-537 deg/sec in this series). The first agonist burst started 60 to 120ms before the onset of displacement. In this and in some other normal subjects, cocontraction of the triceps was evident at the beginning of the movement for higher velocity movements. At about the time of peak velocity, the biceps activity ended and the triceps became active.
Fig. 1A and B. Position and raw EMG of a normal subject performing A 90° and B 40° rapid elbow flexion towards a target. Each series of traces represents (from top to bottom): the forearm displacement, the biceps and the lateral triceps EMG. The dotted lines indicate the position of the starting (below) and target (above) zones (10° width). The target zones are situated at the same elbow position for A and B. The movement amplitudes were, respectively, 93° and 40° with peak velocities of 399 and 235 deg/s for A and B. The calibrations are the same in A and B.

The triceps activity increased in magnitude with faster movements but the duration of the burst remained constant (approximately 100ms). Although peak velocity occurred earlier as movement speed increased, the onset of the antagonist burst remained well timed with the occurrence of peak velocity (Fig. 2E). The onset of the second agonist burst and the end of the antagonist burst occurred precisely at peak deceleration (Fig. 2C, F).

The presence of an antagonist burst which characterizes the triphasic pattern was dependent on two factors: a high movement velocity and an active deceleration. When movements were passively terminated (e.g.: flexion into a pillow) or when they are very slow, no activity could be observed in the antagonist triceps.

The mean peak velocities of all the normal subjects was 250 ± 54 deg/sec (range: 176 to 344) for the 40° amplitude movements and 415 ± 73 deg/sec (range: 304 to 540) for the 90° amplitude movements. The duration of movement varied between 120ms for the fastest 40° movements and 570 ms for the slowest 90° movements. The graph of Figure 3A shows that there was no activity in the triceps when peak velocity was lower than 150 deg/s for the 40° movements and 250 deg/s for the 90° movements. This difference disappeared, however, when the same data was plotted as a function of peak acceleration (Fig. 3B). Hence, irrespective of the movement amplitude, the antagonist activity in this task appeared when the movement reached a peak acceleration of about 3,000 deg/s²; thereafter, the amount of activity increased linearly with increasing acceleration.

**Characteristics of the EMG activity in deafferented patients**

The first EMG recordings from patient GL, obtained in 1981, did not show a consistent triphasic pattern even though the mean movement velocity (278 ± 48 deg/s) was above the threshold for the appearance of an antagonist burst in the normal subjects (see Fig. 3A). Furthermore, two clear bursts of activity in the agonist were only rarely observed.

The recordings were repeated in patient GL in 1983. A small but unfailing burst of activity was then detected in the antagonist (Fig. 4A, B). By that time the patient stated that she could function much more effectively in her every day life despite the fact that the sensory deficit had remained stable. The clinical investigations and evoked potentials studies were repeated. The results of these tests were similar to those of 1981 and confirmed that there was no return of sensation mediated by the large myelinated fibers. A definitive improvement was observed in her handwriting although, in the absence of visual feedback, her signature was still illegible.

The burst seen in the triceps had a fairly constant duration of 100ms and was seen to end precisely at the time of peak deceleration, independently of the velocity or the amplitude of the movement (Fig. 5F). In the agonist muscle, a clear reduction of activity was seen at the time of occurrence of the antagonist burst and this was followed by a second agonist burst starting at peak deceleration (Fig. 5C). These timing relationships were similar to those observed in normal subjects. However, contrary to the normals, the antagonist burst in the patient did not show a strong timing relationship with peak velocity. This was particularly evident for the 90° movements (compare Fig. 5E with Fig. 2E). The antagonist activity was also inhibited 20 to 40ms prior to the first agonist burst (Fig. 5D). In all three patients, the tonic activity in the triceps during postural holds was greater than in normal subjects.
Fig. 2A-F. Triphasic EMG pattern in the biceps (agonist, above) and the lateral head of triceps (antagonist, below) of a normal subject during rapid elbow flexions towards a target. Thirty trials of a 90° flexion task are displayed. The following notes apply to this and all other figures where raster activity is displayed. The three movement traces (middle) represent the upper and lower range of the executed amplitudes and an example of a movement ending in the middle of the target zone. All trials are aligned on the onset of movement (left), peak velocity (middle) and peak deceleration (right) indicated by the vertical lines. The trials have been placed in order of increasing velocity (from top to bottom). The histograms are the summation of the rasters above; their count is in arbitrary units and the bin width is 10 ms. Mean peak velocity: 399 ± 89 deg s⁻¹; mean amplitude: 91 ± 4 deg. These measurements, reached at the end of the continuous movements, are ± 1SD.

Patients RV and JD were each tested on only one occasion and they readily showed a triphasic pattern of EMG activity. Figure 4C and D gives an example of the raw EMG associated respectively with a 90° and 40° movement in patient RV. Figure 6 shows the results from patient RV for 30 90° movements. As was the case for patient GL, the triceps burst in patient RV was well timed with peak deceleration (Fig. 6F) but poorly synchronized with peak velocity (Fig. 6E).

Movement accuracy

On the average, more than 90% of the movements performed by normal subjects terminated within the target zone. Despite the fact that the three patients generated a "triphasic" pattern, about half of their movements either undershot or overshot the target.

Table 1 summarizes the data on movement accuracy for the normal subjects and for the three deafferented patients in the 40° and 90° tasks.

Movement accuracy was also documented by measuring the elbow angle at the time of occurrence of peak deceleration and plotting this in relation with the target.
PATIENT G L (1983)

PATIENT R V

Fig. 4A-D. Position and raw EMG of patient GL (1983) and patient RV performing 90° (A and C) and 40° (B and D) flexion towards a target. Each series of traces represents the movement and muscle activity as in Figure 1. The movement amplitudes of patient GL were respectively 89° and 39° with peak velocities of 309 and 258 deg/s in A and B. The movement amplitudes of patient RV were respectively 89° and 43° with peak velocities of 470 and 370 deg/s in C and D. The calibration is the same for the 90° and 40° movements.

Quantitative study of EMG activity

In the normal subjects and in the three patients, the magnitude of peak deceleration was always well correlated with the magnitude of the antagonist triceps burst. Triceps activity of the 10 normal subjects was also always strongly correlated with peak velocity and peak acceleration, as well as with the magnitude of the first agonist burst (Fig. 8E, F). In the patients, such correlations were either absent (Fig. 8A, B) or present (Fig. 8C, D) with lower correlation coefficients than in normal subjects. Table 2 summarizes the values of the correlation coefficients obtained for these relations in all subjects.

zone. In normal subjects (Fig. 7E, F) peak deceleration occurred near the lower limit of the target zone and had a relatively narrow distribution. In patients GL and RV (Fig. 7A-D) the mean angle at which peak deceleration occurred did not differ significantly from the normals but their overall distributions were much wider. The Hartley's test for variance homogeneity (Keppel 1982) showed that both patients had a variance that is significantly different ($p < 0.001$) from the variance of the normal subjects in each task. There was no significant difference between the distribution of the two patients shown in Figure 7. In patient JD, close to half of the peak decelerations occurred as the limb had passed beyond the target zone.
Discussion

The triphasic EMG pattern in normal subjects

In accordance with other investigators, we found in all normal subjects that the antagonist burst occurs at about peak velocity (Barnett and Harding 1955; Garland and Angel 1971; Angel 1974; Burton and Onoda 1978). Thus, for movements of the same amplitude but of different velocity, the peak velocity and the antagonist burst occur progressively earlier with increasing speed (Lestienne 1979; Marsden et al. 1983). For movements of the same velocity but of different amplitude, the peak velocity and the antagonist burst occur progressively earlier with decreasing amplitude (Marsden et al. 1983). The occurrence of the antagonist burst is then variable in time and does not seem to reflect a simple segmental stretch reflex as was probably the case in the experimental situation of Ghez and Martin (1982) in the cat and Terzuolo et al. (1974) in the monkey. These authors observed a burst of constant latency in the antagonist some 10-30ms following a sudden external unloading of the agonist that produced very high peaks of acceleration (14,000-46,000 deg/s^2). In our experiments, the threshold for the appearance of the antagonist burst (active breaking) was found to correspond to peak acceleration of about 3,000 deg/s^2.

Our results also confirm previous reports that the magnitude of the antagonist burst in linearly related to the peak velocity of the movements (Bouisset and...
Fig. 6. Integrated EMG activity of patient RV performing 90° elbow flexions. Thirty trials are plotted as in Figure 2. Mean peak velocity: $450 \pm 79$ deg $\cdot$ s$^{-1}$; mean amplitude: $91 \pm 8$ deg

Lestienne 1974; Lestienne 1979) and that for large and small movements of the same peak velocity, the large movements have less antagonist activity than the small movements (Marsden et al. 1983; Flament et al. 1984; Benecke et al. 1985). This has been interpreted as a consequence of a decreased need for active breaking during large movements because their termination was closer to the end of the range of motion where increased visco-elastic parameters could provide passive deceleration (Marsden et al. 1983). Such an interpretation cannot apply to our results since large and small movements ended at the same elbow position. It is evident that for a large amplitude movement to be executed with the same peak velocity as a small amplitude movement, peak acceleration has to be smaller. Our results show that the magnitude of the antagonist burst is linearly related with the peak acceleration independently of the amplitude of the movements (Fig. 3B). Thus the magnitude of the antagonist burst can be regarded as a function of acceleration, independently of the starting limb position or of the amplitude of the movement.

**EMG activity and movement parameters in deafferented patients**

Our results confirm previous reports (Hallet et al. 1975; Rothwell et al. 1982; Sanes et al. 1984) that a triphasic pattern of muscular activity can be generated in patients deprived of proprioceptive and cutaneous feedback. The first recordings obtained in patient GL did not show any evident antagonist burst during movements performed at velocities comparable to the normal subjects. It is possible that the strong tonic EMG activity present in this patient has obscured the presence of a small phasic burst in
the antagonist. In fact, in the subsequent experiments in patient GL and in the other two patients, the antagonist burst was and remained very small compared to normal subjects. Sanes and Jennings (1984) also observed a decrease of the amplitude of the antagonist burst in normal humans after ischaemic deafferentation. Despite the presence of an antagonist burst in the three patients we have studied, they showed a poor rate of success in terminating the movement precisely in the target zone. This is in contrast to the patient studied by Rothwell et al. (1982) who showed normal accuracy for the rapid flexions of the distal joint of the thumb. In addition to the fact that a different joint was studied in the two experiments, the patient of Rothwell et al. appeared to be less severely affected than our patients since sensation was preserved above the elbows.

In the normal subjects, the magnitude of the antagonist burst was always strongly correlated with the magnitude of the agonist burst and with peak acceleration, independently of the amplitude of the movement. If the magnitude of the antagonist was entirely generated centrally prior to movement onset as a simple function of the magnitude of the initial agonist burst these relations should not be modified in the absence of peripheral feedback. In the patients, the magnitude of the antagonist burst showed either no correlation or weaker correlations than normal with the magnitude of the initial agonist burst and peak acceleration. It can thus be concluded that peripheral feedback contributes to the precise adjustment of the amplitude of the antagonist burst.

Movement accuracy must also depend on the precise
timing of the antagonist burst. In normal subjects, the antagonist burst appears at a rather fixed time with respect to peak velocity so that peak deceleration occurs within a narrow range near or within the target zone. In the three patients, the time of occurrence of peak deceleration was much more variable than in normals, resulting in a high percentage of undershoot and overshoot. Thus afferent feedback would seem to play a role in adjusting the timing of the antagonist burst as well as its amplitude.

The nature and the origin of the peripheral information used to control the amplitude and the timing of the antagonist burst is unknown. Since the mass of the limb was constant in all our experiments, we cannot dissociate the acceleration from the force developed by the agonist burst.

<table>
<thead>
<tr>
<th>Table 1. Movement accuracy</th>
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<tbody>
<tr>
<td><strong>90° Task</strong></td>
</tr>
<tr>
<td>Success</td>
</tr>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>n = 10</td>
</tr>
<tr>
<td>(70°-100°)</td>
</tr>
<tr>
<td>Patients</td>
</tr>
<tr>
<td>GL (1981)</td>
</tr>
<tr>
<td>GL (1983)</td>
</tr>
<tr>
<td>JD</td>
</tr>
<tr>
<td>RV</td>
</tr>
</tbody>
</table>

* Average and range of the normal subjects

Table 2. Correlation coefficients of triceps EMG as a function of the parameters of movement and biceps EMG

<table>
<thead>
<tr>
<th>DECE./TRIC.</th>
<th>TRIC./ACC.</th>
<th>TRIC./VEL.</th>
<th>TRIC./BIC.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>90° Task</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normals</td>
<td>n = 10</td>
<td>0.91 ± 0.05</td>
<td>0.80 ± 0.09</td>
</tr>
<tr>
<td>Patients</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GL (1981)</td>
<td>0.17</td>
<td>0.22</td>
<td>0.11</td>
</tr>
<tr>
<td>GL (1983)</td>
<td>0.70</td>
<td>0.20</td>
<td>0.21</td>
</tr>
<tr>
<td>JD</td>
<td>0.71</td>
<td>0.21</td>
<td>0.31</td>
</tr>
<tr>
<td>RV</td>
<td>0.67</td>
<td>0.29</td>
<td>0.68</td>
</tr>
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</table>

| **40° Task** | |
| Normals | n = 10 | 0.90 ± 0.05 | 0.85 ± 0.12 | 0.87 ± 0.09 | 0.83 ± 0.09 |
| Patients | |
| GL (1981) | 0.47 | 0.19 | 0.33 | 0.20 |
| GL (1983) | 0.59 | 0.25 | 0.40 | 0.54 |
| JD | 0.76 | 0.35 | 0.44 | 0.33 |
| RV | 0.69 | 0.53 | 0.56 | 0.61 |

VEL: peak velocity, ACC: peak acceleration, DECE: peak deceleration, TRIC: lat. triceps, BIC: biceps. * p < 0.01, b p < 0.001

Hence both parameters could play a role. However, the observation that the antagonist activity increases with increasing inertia of the limb even though acceleration and movement amplitude remain the same (Lestienne 1979; Meinck et al. 1984), suggests that sensory information related to the force developed by the agonist may be more important than information related to acceleration. The inhibition of the tonic antagonist activity preceding the first agonist burst (Hufschmidt and Hufschmidt 1954) was seen in all our deafferented patients, indicating that this phenomenon is centrally generated. The duration of the initial agonist burst also appears to be centrally determined since changes in duration with different movement amplitudes observed in normal subjects (Wadman et al. 1979; Brown and Cooke 1984; Berardelli et al. 1984; Benecke et al. 1985) were seen in the deafferented subjects as well (Cooke et al. 1985 and also Figure 4 in this paper).
Finally, it is also clear that the second agonist burst is not a response to peripheral events since it was observed in the deafferented patients. Garland et al. (1972) have already reported the presence of two agonist bursts after lidocaine block of the nerves supplying the antagonist muscles. Close examination of our data from the deafferented patients shows that the segmentation of the agonist activity into a well defined two bursts pattern coincided with the appearance of a well defined burst in the antagonist muscle. This raises the possibility that the two phenomena are generated by a common central mechanism even though, at times, each pattern of activity can be seen to occur independently. In conclusion, sensory feedback from the moving limb is not a necessary condition for the appearance of the basic agonist or antagonist activity seen in the triphasic pattern. However, sensory inputs (most likely acceleration and/or force sensitive) seem to be able to influence this centrally generated pattern to provide a fine control of the magnitude and timing of the antagonist burst. This is in agreement with the fact that externally applied perturbations can modify the activity of the agonist and antagonist burst (Angel 1975, 1977; Hallet and Marsden 1979; Brown and Cooke 1981b). Finally, the triphasic pattern can be suppressed voluntarily when active deceleration is not required (Waters and Strick 1981; Marsden et al. 1984)

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