Spatial zones for muscle coactivation and the control of postural stability

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Abstract

It is hypothesized that, depending on the motor task, the angular range of a joint may be subdivided into zones in which agonist and antagonist muscles are coactive, only one group of muscles is active or neither group is active. It is further hypothesized that central commands may change the size and location of these spatial zones. By studying responses in patients, we sought to determine whether the specification of zones of agonist/antagonist muscle coactivation ("coactivation zones") may be essential for postural stability. At an initial elbow angle (130°; full extension is 180°), flexors were pre-activated by compensating an initial load which was equal to approximately 30% of the subject’s maximal isometric voluntary contraction effort. Subjects were instructed not to correct the arm displacement elicited by a sudden decrease in the load. Data from 10 trials were collected at each of 4–6 final load levels separated by 1.5–2 Nm in order to map out the relationship between torque and angle in each subject. The procedure was repeated from a more flexed initial position of the elbow (100°). EMG activity from two elbow flexors and two elbow extensors, as well as torque, velocity and joint position were recorded. Healthy control subjects and patients with mild clinical symptoms had coactivation zones or small silent zones around the final positions established after unloading. In these subjects, final positions of the limb were stable. Voluntary movement, i.e., transition of the limb from one initial position to another, was associated with a change in the location of the zone in articular space. The presence of large silent zones in patients with moderate or severe symptoms was correlated with postural instability and oscillations about the final position of the arm after unloading. The comparison of results from healthy and hemiparetic subjects implies that the central specification of the size and the location of a coactivation zone may be fundamental for the control of posture and movement.

Keywords: Motor control; Co-contraction; Posture; Movement; Hemiplegia

1. Introduction

The description of reciprocal and simultaneous patterns of agonist and antagonist muscle activation is considered a fundamental way of understanding motor function. Muscle activity patterns are commonly studied in terms of the temporal aspects and magnitudes of electromyographic (EMG) bursts during many different types of movements from fast single-joint movement to locomotion (e.g. [6,66]). EMG patterns have also been found to correlate with different spatial characteristics of movement such as direction and magnitude [22,44].

A theoretical framework for the description of a mechanism underlying the specification of spatial characteristics of muscle activation has been proposed in the \( \lambda \) model of motor control [17,18]. We hypothesize that depending on the motor task, the angular range of a joint may be subdivided into zones in which agonist and antagonist muscles are coactive, only one group of muscles is active or neither group is active. According to this hypothesis, central commands may change the size and location of these spatial zones in the angular coordinates defined for each joint (single-joint movements) or group of joints (multi-joint movements) [18].

In the \( \lambda \) model for single-joint movement, at least two central commands have been defined which regulate the spatial characteristics of agonist and antagonist muscle activation (Fig. 1). These central commands may be associated with independent components of mono- or polysynaptic influences from descending systems onto flexor and extensor motoneurons. In other words, descending influences may, although not necessarily always, be specified independently of current events in the periphery. The
range in which agonist and antagonist muscles may be coactive (C) which may occur with R, specifies an angular transition of agonist to antagonist activity or vice versa occurs Fig. 1, upper panel. A second command (C) which may occur with R, specifies an angular range in which agonist and antagonist muscles may be simultaneously active (coactivation zone) if C > 0 (Fig. 1, middle panel) or silent (silent zone) if C < 0 (Fig. 1, lower panel). In other words, the C command separates agonist and antagonist thresholds such that angle R occurs between them. In the case when C > 0, if no change in position is to occur, the C command should separate the thresholds so that the activity of agonist and antagonist muscles produces equal and opposite torques. In other words, at position R, the joint would remain motionless if the C command changes from zero to a positive value. Under constant central commands, if the joint is deflected from position R, active muscle torque elicited by external forces would tend to bring the joint back to R. The sign of the net joint torque produced by each muscle group to counteract external forces changes at angle R (Fig. 1, middle panel). Thus, although the R command when C ≠ 0 no longer represents the threshold angle for the pure transition of activity from one group of muscles to the other, it still represents the referent angle which influences muscle recruitment and sets the location of the coactivation or silent zone in the angular range. Control inputs to motoneurons may shift the point (R) and/or change the width of zone (C).

A given combination of R and C commands is associated with a single-valued relationship between static joint torque and angle, called the invariant characteristic (IC) of the joint (Fig. 1, compare solid curves in different panels). The term “invariant” implies that for all combinations of muscle torques and angles for this IC the values of the R and C commands are the same. It does not imply that the shape of the IC is constant when central commands are changed. Similarly, it does not imply that tonic EMG levels are the same for different points on the IC [18]. Since R and C commands have the dimension of position (angle), the understanding of motor control processes in the λ model is different from the traditional one in which control processes are considered in terms of reciprocal and coactivation EMG patterns for agonist and antagonist muscles during different motor tasks. R and C commands are, in essence, independent of EMG patterns (but not vice versa [18]). For example, when C = 0, the R command defines the angle at which the transition from agonist to antagonist activity occurs. Whether or not the EMG transition actually occurs depends on the spatial relationship between the actual joint position (θ) and R. For example, for a given IC, passive extension from an angle less than R to one greater than R (Fig. 1, upper panel) will result in the transition of activity from the extensor to the flexor muscle group. On the other hand, passive extension in ranges less than R will result only in modulation of activity in one muscle group without any switching between muscle groups. With increasing speed of extension, phasic reflexes may modify this behavior (see [18]). At the same time, muscle activation also depends on the C command which specifies the width of the coactivation zone. An example of the latter can be seen in Fig. 1 (middle panel). If the final position of the joint is
such that the load is balanced inside the coactivation zone (filled circles), both agonist and antagonist muscles will be tonically active. In contrast, although the coactivation zone may be present, the load may be balanced outside this zone (open circles) so that only one muscle group will be active. These examples illustrate a general notion of the $\lambda$ model: EMG patterns are not programmed but emerge from the interaction of the central control signals, proprioceptive feedback, intrinsic muscle structures and external forces [1].

Both R and C commands influence the net joint stiffness defined as the slope of the IC at a given operating point. The wider the coactivation zone defined by the C command, the steeper the slope of the torque/angle characteristic in that zone (compare solid lines in upper and middle panels, Fig. 1). Since the shape of the IC is non-linear and the slope increases with increasing muscle torque depending on the difference between the actual angle and angle R (Fig. 1), the R command may also affect stiffness. Imagine that the threshold angle of the flexor muscles, $\lambda_r$, is shifted to the left by an R command when the joint is in an initial position, $\Theta_i$ (Fig. 1, middle panel). After the shift, a new operating point on the IC is attained in which the amount of torque and, as a consequence, stiffness associated with position $\Theta_i$ is greater. Thus the regulation of stiffness may result from the modification of the operating point on the IC by the R command. Stiffness influences the stability of posture and movement [57]. Stability also depends on parameters influencing velocity-dependent characteristics of sarcomeres and proprioceptive feedback.

The framework of the model can be used for the understanding of the control of stability by comparing motor behavior of healthy subjects and patients with sensory-motor dysfunction. In particular, among other sensorimotor disturbances resulting from hemispheric stroke, the ability to produce smooth movement is impaired [49]. This occurs along with enhanced agonist/antagonist muscle co-contraction [36,38] and considerable slowing of movement [49]. Abnormal co-contraction during goal-directed movements such as reaching and locomotion may be associated with diminished agonist motor unit activation in these patients [10], impaired antagonist inhibition [38] or both [36]. In addition, weakness [5], altered mechanical properties of motor units [39], improper spatial and temporal muscle recruitment [20] and disruption in the organization of segmental reflex activity [9] may play a role in the appearance of abnormal coactivation during movement.

Spastic muscles in hemiparetic subjects may be characterized by significantly increased stretch reflex activity [3,55] which may be due to a decrease in the stretch reflex threshold and to limitations in its central regulation [40,48,51]. These findings have led to the hypothesis that the regulation of reciprocal muscular activation and muscle coactivation may also be affected in these subjects.

Based on the suggestion of the $\lambda$ model regarding the spatial zones for different patterns of muscle activation, we investigated how such zones were used in the postural control of the elbow joint in normal subjects and in those with postural control deficits due to unilateral stroke. Such data may improve our understanding of normal motor control and impaired control following lesions in the central nervous system. Some of the data have been presented in abstract form [52].

2. Materials and methods

2.1. Experimental procedures

The forearm was placed on a horizontal manipulandum and the hand and forearm were stabilized in the neutral position between pronation and supination in a bi-valve splint adjusted by velcro straps. The flexion/extension axis of the elbow joint was aligned vertically with the axis of rotation of a torque motor (Mavilor Motors, MT 2000). Each initial position (approximately 130° and 100° flexion of the elbow; full extension being defined as 180°) was achieved by lining up a vertical cursor within a 3° target window on the computer screen in front of the subject. Thereby the subject resisted the load created by the torque motor opposing elbow flexion. Load torques (L) opposing elbow flexion are considered positive. The initial load torque ($L_i = 4$–$10.9$ Nm) corresponded to 20–30% of the subject’s maximal isometric voluntary contraction (MVC) which was measured at the beginning of the testing session. After a variable delay period (1–2 s), the screen was blanked and the load torque was suddenly decreased in a single step during less than 20 ms to a final load, $L_f$. The load could be decreased to a final positive load ($L_i > L_f > 0$; partial unloading), to a zero load ($L_i = 0$; complete unloading) or to a load assisting flexion ($L_i < 0$; assisting unloading). For example, if the initial load was 10 Nm, unloading resulted in final load levels of 8, 6, 4, 2, 0 and $-2$ Nm in different trials. The first four levels represented partial unloadings, the fifth a complete and the last, an assisting unloading. For the assisting unloading, the initial 10 Nm load resisting flexion was decreased by 12 Nm resulting in a final load of $-2$ Nm that assisted flexion.

The decrease in the load torque caused the elbow to flex to a new position at which the arm torque balanced the final external torque for about 1 s (total recording time was 1.7 s). Subjects were instructed not to correct the natural displacement of the arm (“do not intervene”). A critical assumption underlying the use of this paradigm is that in following the instruction, subjects are able to maintain the same pattern of control signals regardless of load perturbations. The feasibility of this assumption has been tested in other studies [2,18]. Static torque/angle characteristics of the arm recorded using the unloading paradigm appear to remain invariant in spite of variations in the unloading procedure. Both double-step decreases in the load torque...
with variable inter-step time and the use of position-dependent, elastic loads with positive or negative stiffness have yielded similar results. In contrast, when loading instead of unloading paradigms have been used, it was reportedly difficult for subjects not to intervene (Fig. 4 in [18]; see also [12,58]). Therefore, only unloading stimuli were used in the present study.

The unloading stimuli were randomly presented and a total of 10 trials were collected at each final load level for a total of 40–60 trials per experiment. The initial position of the elbow was then changed to a more flexed posture and the experiment was repeated. Starting angles of 130° and 100° were used in order to avoid an extreme flexion position of the elbow following unloading. In addition, these two starting angles were chosen since some hemiparetic subjects could not adequately stabilize their arms against the initial load at more extended initial positions of the elbow joint. For safety reasons, a mechanical device on the apparatus arrested the movement when the elbow angle reached 30°.

Table 2

<table>
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<tr>
<th>130° initial torque (Nm)</th>
<th>Slope (Nm/deg)</th>
<th>Zone (deg)</th>
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<tr>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S1</td>
<td>6.4</td>
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</tr>
<tr>
<td>S2</td>
<td>10.0</td>
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<td>10.9</td>
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<td>10.5</td>
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<tr>
<td>S5</td>
<td>8.4</td>
<td>0.135</td>
<td>31.0 *</td>
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<tr>
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Table 2

<table>
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<tr>
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<tr>
<td>H10 b</td>
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<td>13</td>
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<td>S.D.</td>
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For hemiparetic subjects, clinical spasticity and arm function (Fugl–Meyer) scores are also listed.

a indicates that the coactivation or silent zone extended beyond the range of joint angles investigated.

b indicates those subjects in whom silent instead of coactivation zones were present.
2.2. Subjects

Seven healthy control and 10 hemiparetic subjects participated in the study after being informed of the experimental procedures and giving their written consent according to the procedures approved of by the local Hospital Ethics Committee. For the hemiparetic subjects, the type and location of the brain lesion were identified from results of NMR or CAT scan tests (Table 1). Hemiparetic subjects were recruited from the Rehabilitation Institute of Montreal and met the following inclusion criteria: (1) had sustained a single stroke at least 6 months previously leading to arm paresis; (2) were less than 65 years old; (3) had no other neurological disorders; (4) had full range of joint motion at the elbow; (5) were able to understand simple commands (no receptive or expressive aphasia); (6) had no perceptual or visual field problems (Bell’s test [28]); (7) had some control of isolated muscles in the upper limb (Brunnstrom stages 4–6 [7]); and (8) were able to give their informed consent. In addition, subjects were excluded if they were non-ambulatory, had shoulder subluxation or had pain in the arm. Control subjects were excluded if they had a medical history of: pain or previous orthopedic or neurological problems affecting the shoulder, elbow or wrist. Spasticity and sensorimotor function in the arm of hemiparetic subjects were evaluated clinically by an experienced physiotherapist before physiological testing began.

Clinical spasticity in the elbow flexors was measured with a valid [35] and reliable [54] three-parameter assessment. Biceps tendon jerks were evoked by a maximal tap on the biceps tendon by a reflex hammer. Responses were scored on a 5-point scale. Resistance to full-range elbow extension was evaluated by passively extending the arm from the fully flexed position at a moderate speed and scored on a modified 5-point Ashworth scale [4]. The third parameter, wrist clonus, was evoked by rapidly extending the hand. The number of clonic beats in response to the stimulus was rated on a 4-point scale. Composite spasticity scores ranging from 0–5, 6–9, 10–12 and 13–16 correspond to “no”, “mild”, “moderate” and “severe” spas-
ticity respectively. In previous studies, the scale has been shown to be significantly inversely correlated with the threshold of the stretch reflex in the elbow flexor muscles in spastic hemiparetic subjects \( (r = -0.65, \ P < 0.05 \) [51]) and with clinically measured residual functional ability of the affected arm [49].

The Fugl–Meyer test [27] was used to assess volitional movements in the upper limb of the hemiparetic subjects. This assessment measures the ability of the subject to make isolated movements within and out of pathological synergy patterns. In addition, the scale assesses sensory function, reflexes, hand function and coordination. A maximum score of 66 corresponds to normal function.

2.3. Data acquisition and analysis

EMG activity of two elbow flexors (biceps brachii, BB, and brachioradialis, BR) and two elbow extensors (lateral head of triceps brachii, TB, and anconeus, AN) were recorded with active (gain = 10; band-pass filter = 45–500 Hz) bipolar surface electrodes (1 mm silver chloride strips, 1 cm long and 1 cm apart). EMG signals were further amplified, filtered and sampled at a rate of 1000 Hz before being stored on computer disk.

Torque was recorded with linear strain gauges mounted on the common shaft of the torque motor and manipulandum having, besides inertia, negligible resistance. Position and velocity were measured with a high precision hybrid electromagnetic resolver (RDC-1920) aligned with the shaft of the torque motor. For each subject, torque and position data were analyzed trial-by-trial on an interactive computer display. Pre-unloading values were measured in the 200 ms period prior to unloading. Post-unloading values were measured once the arm had reached a stable position during the approximately 1.4 s holding period. These values were measured in the final 400 ms of each trial (between 1 and 1.4 s after unloading; see solid horizontal bar in Fig. 3 below). In normal subjects, stationary position and torque values could be obtained earlier in the trial (see Fig. 3). However, values were measured late in the trial in order to compare data from the arms of normal subjects with those from hemiparetic subjects which displayed oscillations after unloading and which only reached a steady state after several hundred milliseconds. The final torque and position values were then averaged and used to construct torque versus angle diagrams.

Final positions were easily identified by examination of velocity versus angle phase diagrams (Fig. 2A), which were also used to identify those trials in which the subject did not comply with instructions and intervened voluntarily (Fig. 2B). Voluntary interventions or movement corrections were identified as inflections in the phase diagram. In addition, corrective movements could also be identified from the analysis of individual position and velocity traces. Phase diagrams integrating information about position and velocity, however, were generally more helpful than individual traces in the identification of inflection points associated with movement corrections. Since the unloading
was rapid (in less than 20 ms) and the magnitude and timing of the unloading were unpredictable, there was little chance of voluntary intervention during unloading although the possibility that subjects made undetected smooth partial corrections cannot be ruled out. Later corrections could be identified by the presence of additional movements following or slightly before the establishment of the new final position as a result of unloading (Fig. 2B, thin arrow). Late corrections exceeding 3° were present in less than 1% of trials for all subjects. These trials were excluded from the analysis.

The degree of postural stability was measured in control and hemiparetic subjects by determining the logarithmic decrement of decay of the oscillations of the arm about the final position: \( D = (\ln P_1/P_2)/T_{12} \) where \( P_1 \) and \( P_2 \) are the peak velocities of, respectively, the first overshoot and the following undershoot of the arm position; \( T_{12} \) is the time between the two peaks. The inverse of the decrement, \( 1/D \), is the time required for the velocity oscillations to decrease in amplitude by a factor \( 1/e \approx 0.37 \). For example, \( 1/D = 0 \) if the transition from one position to the other is produced aperiodically, i.e., without terminal oscillations, and \( 1/D = \infty \) if oscillations are self-sustained, i.e., proceed without decay. In normal subjects, this coefficient was below 0.23 s (see Table 2 and Fig. 13). Values above 0.23 s were considered to be abnormal.

Decrement \( D \) is related to the system’s damping (d) defined as a coefficient or a gain factor influencing the dependency of force or torque on velocity. For some physical systems, the relationship between d and D may be linear (e.g., \( d = 4 \) mNs for a system consisting of a mass, \( m \), suspended on a damped spring). Measurements of d in neurally controlled musculoskeletal systems is technically difficult and usually based on simplified, linear models of the system. In contrast, decrement D can be measured regardless of any model and therefore is a preferable measure of the system’s damping properties.

Coefficient D is invariant for linear systems so that its value remains the same if it is calculated using other velocity peaks than those indicated above. In the present study, coefficients \( 1/D \) were calculated for the first two \( (P_1, P_2) \) and the second two \( (P_3, P_4) \) velocity peaks and the difference between the two values of \( 1/D \) were used as an estimate of the linearity of the system.

2.4. Coactivation zones

EMG signals from trials corresponding to the same final torque level for each starting angle were grouped, rectified, high-pass filtered at 40 Hz and averaged for the analysis of the presence or absence of tonic agonist and antagonist activity in the final 400 ms of the trial, during which time the arm had stabilized in a new static final position as a result of unloading. Activity in a muscle was classified as “present” when, for the 400 ms period, the EMG signal surpassed and remained above 1 S.D. of the baseline.
activity recorded at rest. The coactivation zone was defined as the range of final positions after unloading in which both flexor and extensor muscles were simultaneously active (see solid horizontal bar in Fig. 3 below).

2.5. Statistical analysis

We tested whether final positions for each unloading level were significantly different from each other for the two torque/angle characteristics recorded in each subject (a 2-way ANOVA in which the factors were level of unloading and final position with repeated measures on position). Final torques and positions were then used to reconstruct the ICs for each subject.

Second order polynomial functions were used to fit each torque/angle characteristic and the $r^2$ values of these relationships were used as estimates of the correlation between torque and angle.

Positions associated with two final torque levels (6 Nm and 2 Nm) were determined for each IC. Stiffness was computed as the ratio $\frac{\Delta T}{\Delta \theta}$ of the change in torque ($\Delta T = 4$ Nm) to the change in the position associated with these two torque levels. The estimation of stiffness was confined to this part of the curve because it was the most linear, data from both healthy and hemiparetic subjects were available, and it was above the level at which there were substantial contributions to the slope from passive stiffness.

Finally, the slope of the torque/angle relationship and the size of the coactivation zone were correlated with lesion location, clinical spasticity, functional scores and the logarithmic decrement of decay using Pearson’s Product Moment statistics in order to describe the relationship between clinical and physiological parameters which characterize the neurological deficit in the hemiparetic subjects.

3. Results

3.1. Healthy subjects

3.1.1. Kinematics, EMG patterns and torque/angle characteristics

In healthy subjects, unloading resulted in a silent period and an after-volley in the agonist muscles (BB and BR) and a stretch response in the antagonist muscles (TB and AN; Fig. 3). Following the dynamic phase of unloading, a new final combination of joint position and muscle torque was established (see torque and position traces) with correspondingly new levels of agonist and antagonist EMG activity. Mean tonic EMG levels for flexors and extensors before and after unloading were measured in the periods indicated by the open and solid horizontal bars respectively (Fig. 3). A lower level of EMG activity was established in the flexors after unloading while extensor activity was either absent or increased (see Section 3.1.2 “Coactivation and silent zones” below). An examination of arm position traces following unloading suggests that subjects complied with the instruction not to correct the arm deflections (see Section 4).

Fig. 4 (third panel from the top) shows the mean arm positions for one subject for different amounts of unloading. It may be seen that following unloading, the arm position stabilized within approximately 400 ms and then remained relatively constant until the end of the trial. For all subjects, after complete unloading, final positions differed from initial ones by as much as 28–61° ( > 20% of the articular range of the elbow). In spite of these substantial differences, subjects complied with the instruction not to intervene so that final positions after unloading remained stable.

The patterns of kinematic responses changed systematically with the size of the unloading step (Fig. 4). The final joint position was a monotonic function of the size of the step (change in torque) as was the magnitude of and the time to the peak velocity. Reaching the final joint position was associated with rapidly decaying oscillations (Fig. 4, velocity traces) characterized in particular with an initial positional overshoot related to the size of the step (Fig. 4, angle traces, third panel; velocity/angle traces also called phase diagrams, Fig. 4, bottom panel).

The reconstruction of two final torque/angle characteristics from a healthy subject (S6) is shown in Fig. 5. One characteristic was recorded from the initial elbow angle of 130° (right curve) and the second from the initial elbow angle of 100° (left curve). The initial combination of torque and position is shown by a filled circle for each
series of unloadings and represents initial equilibrium points (EP) of the system. Partial unloadings from each starting position resulted in the stabilization of the arm at new EPs (open circles: mean ± S.D. for position; the S.D. of the torque did not exceed the size of the symbol). In all subjects, for each characteristic, the final EPs after separate unloading levels were significantly different from each other (ANOVA, \( P < 0.05 \)).

Torque/angle characteristics were measured for all healthy subjects. In all but one case, the characteristics were non-linear. Torque/angle relationships were approximated by second order polynomials (see Section 2). For the characteristics shown in Fig. 5, these polynomials were: \( y = 13.45 - 0.444x + 0.003x^2 \), \( r^2 = 0.992 \) for initial position 130°; and \( y = 21.33 - 0.795x + 0.007x^2 \), \( r^2 = 0.993 \) for initial position 100°. The high \( r^2 \) values imply that there may be a separate single-valued relationship between torque and angle for each characteristic. Voluntary action (the transition to the more flexed initial position of the arm) was associated with a shift in the torque/angle characteristic implying a change in the initial control variables. For the group, the mean stiffness for both characteristics, measured in the linear range between 2 and 6 Nm (see Section 2), was 0.273 ± 0.069 Nm/deg (Table 2).

![Fig. 8. Single unloading trial in a hemiparetic subject (H7). The torque step (complete unloading of the limb from an initial torque of 7.5 Nm) resulted in substantial terminal oscillations associated with the reciprocal phasic bursts and the lack of tonic EMG in the antagonist muscles (TB and AN). Oscillatory behavior is also evident in the velocity and position traces. In this subject, the magnitude of terminal oscillations exceeded the limits of motion of the manipulandum so that the subject’s arm hit the edge of the apparatus as reflected in the three peaks in the torque trace. Abbreviations as in Fig. 3.](image)

![Fig. 9. Averaged \((n = 10)\) kinematic responses to four different levels of unloading in a hemiparetic subject (H7) from an initial load level of 7.5 Nm. As in normal subjects, responses changed systematically with the level of unloading (see Fig. 4). Note that the amount of oscillations increased with the level of unloading.](image)

### 3.1.2. Coactivation and silent zones

The patterns of tonic agonist and antagonist muscle activity were analyzed for each torque/angle characteristic allowing us to determine the angular range in which muscle coactivation occurred. Tonic agonist (BB) and antagonist (AN) EMG levels associated with each EP for a torque/angle characteristic in one healthy subject (S4) are shown in Fig. 6. At the initial combination of torque and position of the limb before unloading (point A), tonic flexor activity was high, corresponding to about 30% of MVC (see Section 2) whereas tonic antagonist activity was absent. In the steady state after unloading, tonic agonist EMG decreased with muscle shortening and decreasing final torque (points B to F) until it was absent at point G. At the same time, antagonist activity increased with muscle lengthening from point C and was highest at point G.
Fig. 10. Two invariant characteristics (ICs) measured in two hemiparetic subjects in whom no (A) or substantial (B) terminal oscillations in response to unloading were observed. In spite of the differences in kinematic behavior, each subject produced an IC distinct from the first when the initial position was changed.

In this example, a coactivation zone where both agonist and antagonist muscles were simultaneously active, was identified in the positional range between 113.9° (point C) and 82.0° (point F). This, in fact, may be an underestimate of the actual extension of the coactivation zone, since we only investigated discrete points in the continuum of the angular range.

Several levels of partial unloading brought the arm into the coactivation zone where both agonist and antagonist muscles were tonically active. The negative torques were balanced (point G in Fig. 6) only by antagonist muscles.

Coactivation zones were analyzed in 6 out of the 7 healthy subjects in whom both characteristics were recorded. In all but two cases, coactivation zones were present for both characteristics (Fig. 7). The shift in the position of the characteristic was associated with a spatial shift in the coactivation zones. In one characteristic for each of two subjects (S5 and S6), our discrete-point estimation did not allow us to observe a coactivation zone. In these two cases, there were small silent zones (width 9° and 9.7° respectively). In other words, there was a small gap between the activation zones of the agonist and antagonist muscles.

3.2. Hemiparetic subjects

3.2.1. Kinematics, EMG patterns and torque/angle characteristics

Muscles of hemiparetic subjects were considerably weaker than healthy subjects. Flexor MVCs were generally

Fig. 11. The absence of a coactivation zone in a hemiparetic subject (H8). An IC recorded from the initial position of 100° (filled circle) is shown. Final EPs following partial unloadings are shown as open circles (mean ± S.D.). Tonic EMG levels in agonist (top) and antagonist (bottom) muscles associated with each equilibrium point, EP (A–F), were measured as described in Fig. 6. The vertical arrow indicates the threshold of the agonist EMG occurring at point E. Until this point, tonic agonist EMG activity decreased and there was no appreciable antagonist EMG even when the joint was completely unloaded or assisting (negative) loads were applied. Thus, there was no coactivation zone.
less than 25 Nm in stroke patients and greater than 25 Nm in healthy subjects. Thus, lower initial torques compared to healthy subjects were used which, nevertheless, still correspond to about 30% MVC. However, there was an overlap in the level of initial torque between the two groups (see Table 2).

The most remarkable difference in the response to unloading in hemiparetic subjects was that the majority of subjects (6 out of 10) displayed long duration decaying oscillations at the end of unloading (Fig. 8, compare with Fig. 3) accompanied by reciprocal bursting activity in agonist and antagonist muscles (Fig. 8, top four traces). In four of these six subjects, the oscillations diminished before the end of the trial making it possible to measure stable final joint position, torque and EMG values in the final 400 ms of the trial. In one subject (H7), oscillatory behavior continued until the end of the trial for each level of unloading and in one other, H10, oscillations continued until the end of the recording period only for the complete unloading level. In these cases, final torques and positions were estimated as the mean values over the final 400 ms of the trial (see Section 2). In all cases, tonic EMG activity tended to be stable in this period of time. Examination of the ensemble of 10 trials at each level of unloading showed no systematic change in the oscillatory behavior with repeated trials. In spite of this, there was still a systematic dependency of final position, peak velocity and time to peak velocity on the final torque (Fig. 9).

Torque/angle characteristics from both starting positions were recorded in all of the hemiparetic subjects, examples of which are shown in Fig. 10. As in healthy subjects, for each characteristic, the final torques and positions for each unloading level were significantly different from each other (ANOVA, $P < 0.05$). The data in Fig. 10A are from a subject (H2) whose terminal oscillations were in the limit of normal (for definition, see Section 2) while those shown in Fig. 10B are from a subject (H5) who had abnormal oscillations (Table 2). A coactivation zone was present in the former case and a silent zone was present in the latter.

A torque/angle diagram for one hemiparetic subject with a silent zone instead of a coactivation zone (H8) is shown in Fig. 11. Silent zones occurred in six out of the ten hemiparetic subjects. These six subjects are indicated in Table 2. All subjects with silent instead of coactivation zones showed abnormal terminal oscillations. For the other four, as in healthy subjects, a stable position was reached after small and rapidly decaying terminal oscillations. Thereby tonic agonist activity systematically decreased whereas antagonist activity increased with joint flexion.

Stiffness, measured as the slope of the torque/angle characteristic in the range between 2 and 6 Nm (see Section 2), was significantly higher in stroke patients compared to healthy subjects. For both characteristics, stiffness in stroke patients was $0.368 \pm 0.145$ Nm/deg and that in normal subjects was $0.273 \pm 0.069$ Nm/deg ($P < 0.05$). However, some stroke subjects had stiffness values equal to or less than normal (see Table 2). Table 2 lists stiffness, decrement of decay and coactivation zone data for all subjects for the characteristic recorded from the initial elbow position of 130°. Data for the other characteristic were similar since the two characteristics were similar in shape (parallel; Figs. 5 and 10).

### 3.2.2. Coactivation and silent zones

In the initial arm position, hemiparetic subjects compensated the load with only agonist activity as was the case in healthy subjects. In three subjects (H1, H2, H4), the width of the coactivation zone was in the limits of the normal range (Table 2; Fig. 12). One subject demonstrated large coactivation zones (H3) for both torque/angle characteristics. In the remaining six hemiparetic subjects (H5–H10), instead of a coactivation zone, a large silent zone was found which was substantially larger than that observed in healthy subjects (Table 2; see S5 and S6 in Fig. 7).

We were unable to find a relationship between the size of the coactivation or silent zone and the location of the brain lesion in our patients. Of the four patients with coactivation zones, two had cortical, one had a subcortical and one had both cortical and subcortical involvement. Of the six subjects without coactivation zones, four had subcortical and two had cortical lesions (Table 1). On the other hand, the size of the coactivation or silent zone was

![Fig. 12. Coactivation and silent zones in hemiparetic subjects. Coactivation zones (black bars) were present in four of the ten subjects (H1–H4). In H3, the ends of the coactivation zones were unknown since agonist EMG as well as antagonist muscle activity was present, even at the highest level of unloading (when the final torque was $-2$ Nm). Six subjects (H5 to H10) had decreasing levels of tonic agonist activity with increasing levels of unloading and no tonic antagonist activity. Thus, in these subjects, silent zones instead of coactivation zones were observed.](image)
Fig. 13. The inverse of the logarithmic decrement of decay (1/D) characterizing the oscillations about the final arm position (see Section 2) were correlated with the size of the coactivation (C > 0) or silent (C < 0) zone for healthy (filled circles) and hemiparetic (open circles) subjects. The values of 1/D in subjects whose arms oscillated about the final position are denoted by open circles with diagonal lines. The value of 1/D in subject H4, who had a coactivation zone of indeterminable size, is denoted as an open circle with a horizontal and vertical line. Data shown are those calculated from the first (left panels; P1 and P2) and the second two peaks of oscillation (right panels; P3 and P4) for complete (upper panels) and partial (half of the complete; lower panels) unloading from the initial position of 130° for each subject. The inset in the left upper panel shows the data from which the value 1/D was calculated.

significantly correlated with the clinical severity of the sensorimotor deficit in these subjects (r = −0.79) as well as with the amount of oscillatory behavior (r = 0.92).

Abnormal terminal oscillations were observed in all subjects who demonstrated large silent zones. For complete unloading, the value of 1/D (inverse of decrement of decay, see Section 2) for the first two velocity peaks ranged from 0.259 to 0.400 s (mean = 0.331 ± 0.061 s) for those subjects with terminal oscillations which exceeded the values for healthy subjects (Table 2; Fig. 13, open circles with diagonal line). The values of 1/D measured for the second two velocity peaks ranged from 0.296 to 3.293 s (mean = 1.135 ± 1.094 s). The difference between the two values of 1/D is a measure of the system’s non-linearity (see Section 2).

The mean value of 1/D for subjects with no oscillations was 0.113 ± 0.037 s (open circles) which was not significantly different from 1/D for healthy subjects (Table 2; 0.145 ± 0.05 s; filled circles).

4. Discussion

4.1. Basic results

We recorded EMG signals and torque/angle characteristics from two initial positions of the arm in healthy and hemiparetic subjects. In healthy subjects and in hemiparetic subjects with mild symptoms, agonist muscle activity systematically decreased whereas antagonist muscle activity increased with joint flexion implying length-dependent regulation of muscle activity associated with a tonic stretch reflex [19,56,59]. Stable final positions were associated with coactivation or small silent zones in healthy subjects and in four hemiparetic subjects with mild symptoms. For all subjects, a voluntary change in the initial position of the limb was associated with a shift in the torque/angle characteristic and a change in the location of the coactivation or silent zone in joint angular coordinates. Hemiparetic subjects with moderate to severe symptoms had silent zones instead of coactivation zones. Postural stability was quantified in terms of the decrement (D) of decay of the terminal oscillations. In subjects with coactivation zones, unloading resulted in the arm reaching the final elbow position after minimal terminal oscillations as indicated by low 1/D values while in those with large silent zones, the final position was reached after long terminal oscillations associated with high 1/D values. Considering both groups together, postural stability could also be quantified in terms of 1/D and correlated with the size of the coactivation or silent zone (Fig. 13; Table 2). In addition, the relationship between the size of the coactivation zone and the severity of clinical signs in hemiparetic subjects was significant.
4.2. Coactivation zones: a feedforward mechanism for postural control

Is the coactivation zone established in response to unloading or is it a consequence of central commands established during the specification of the initial position to prevent instability in the anticipated final position? The latter case may be regarded as a feedforward mechanism. The answer to this question depends on whether or not subjects modified their central commands in response to the perturbation.

The response to unloading of an actively contracting muscle may stem from three sources: a change in central commands, reflex-, position- and velocity-dependent modifications of EMG activity, and mechanical muscle reactions. In this study, we attempted to ensure that central commands were held constant by instructing the subject not to correct the arm displacement due to the load perturbation. Both healthy and hemiparetic subjects complied with the instructional paradigm during the dynamic (unloading) and static (holding) phases of the trial as evidenced by the lack of inflection points in the phase diagrams (see Figs. 2, 4 and 9) and the stable mean final positions during the long holding periods of each trial even though these positions were substantially different from the initial ones (Figs. 3, 4, 8 and 9). While healthy subjects had to make several practice trials before they were able to not intervene to the perturbation, hemiparetic subjects complied more easily since their voluntary responses were too slow or too limited to react to the sudden perturbation as indicated by the inability of the subjects to rapidly suppress the terminal oscillations in the ongoing trial (Fig. 8) or to diminish them from trial to trial.

Indeed, it has been suggested that the do not intervene paradigm does not guarantee that central commands remain unchanged in all types of perturbations. Specifically, Feldman [16] (reproduced in Fig. 4 of [18]) found that the system is most sensitive to perturbations which stretch contracting muscles sometimes triggering coactivation of agonist and antagonist muscles (see also Fig. 1B of [34]). The stretch (loading) stimulus could lead to changes in central commands (see also [12,58]) and thus the recorded torque/angle characteristic could not be called “invariant”. On the other hand, the assumption on the invariance of central commands in response to unloading stimuli has been supported by additional tests in previous studies [2,18] (see Section 2). For this reason, we limited our paradigm to unloading responses. For the same reason, our data are not directly comparable to those of Gottlieb and Agarwal [34] who used a loading/unloading paradigm.

Consider the possibility that our subjects, contrary to instructions, changed central commands in response to the unloading stimulus. Recall that central commands, according to the definition (see Section 1), are variables which may influence net joint torque independently of other variables characterizing the motor output (i.e., joint angle, velocity and muscle torque itself). A change in such independent variables is shown by the shift in the static torque/angle characteristic when subjects specified a more flexed initial position of the elbow (Figs. 5 and 10). In the $\lambda$ model, the shift is associated with a change in the R command. It is unlikely that subjects triggered changes in the R command in response to unloading from a given initial position. Triggered reactions substantially vary from trial to trial both in terms of latency, amplitude and direction [12]. In our experiments, triggered reactions would have caused changes in R commands leading to variations in the positions of the torque/angle characteristics. Then, the EPs resulting from unloading from a given initial position would have been dispersed in the area between the two extreme positions of the characteristics. The single-valued relationship that we regularly observed between the net torque and joint angle would have been destroyed.

It is also unlikely that subjects changed C commands in response to unloading. A characteristic property of the C command is that the joint can be held at a constant angle while varying the amount of agonist and antagonist coactivation (implying the maintenance of a steady R command while the C command varies; see [2,18]). To preserve the joint position, the C commands should result in equal and opposite changes in the torques of agonist and antagonist muscles at this position (see Fig. 1 in Section 1). In other words, C commands may not affect the final EPs [53] and the relationship between the static torque and joint angle would be the same even if C commands were triggered in response to unloading. However, if a C command was applied after the end of movement in the present study, the agonist and antagonist EMG activity would simultaneously increase while the final joint angle would remain stable, a situation never observed in our experiments (e.g., Fig. 3). Thus, changes in C commands could only be triggered before the end of movement. In this case, subjects would have had to anticipate the appropriate final position and to specify a C command which was balanced in terms of agonist and antagonist torques at this position. However, in our experiments, unloading steps were unpredictable and this strategy would have inevitably led to an error in the anticipation of the final position. The inappropriately specified C command could not have produced balanced agonist and antagonist torques in the actual position and would have resulted in movement to a new position. This would have led again, to the observance of a random rather than a monotonic relationship between the final net joint torque and position. Thus, the monotonic relationship between torque and angle for each initial position (Figs. 5, 6, 10 and 11) observed in our study suggests that no factors (change in central commands) independent of position and torque contributed to the unloading response. Our finding of high $r^2$ values of the equations characterizing the torque/angle relationships indicating low variability in the unloading responses also
supports the conclusion that triggered and voluntary reactions were unlikely. Therefore, we consider the torque/angle relationships recorded in the present study as invariant characteristics (ICs) each associated with constant central commands. Invariance of the central commands implies that subjects specified spatial coactivation zones prior to movement onset. This may be illustrative of a feedforward mechanism allowing the system to prepare an appropriate level of stiffness in specific angular ranges of the joint to prevent large joint excursions in response to sudden perturbations and to provide joint stability. This feedforward mechanism may be impaired in some hemiparetic patients as evidenced by the observation of large silent zones and long terminal oscillations in response to unloading in these patients.

4.3. Control of postural stability

The difference in the stability of the final position of the arm in the two populations was not related to the differences in the initial loads used in the two groups. The size of the initial loads was comparable in the two subject groups since it represented 30% of the individual subject’s MVC. In addition, in six out of ten hemiparetic subjects, even small partial unloadings led to terminal oscillations around the final position. This response was never seen in healthy subjects. In a study of elbow loading and unloading in normal subjects [34], no long-lasting oscillations were recorded when using initial loads generally smaller (1–5 Nm) than those we used in stroke patients (4–10.2 Nm). On the other hand, in the present study, the initial torque was similar in several subjects belonging to different groups (for example S1 and S5 compared to H8 and H5; Table 2). Nevertheless, long-lasting terminal oscillations were observed in hemiparetic (Fig. 8) but not healthy subjects.

ICs have previously been measured both in postural and movement tasks for healthy subjects [2,31,34,53,64] and in children with Down’s syndrome [13]. To our knowledge, they have not previously been described in the upper limb of hemiparetic subjects. In static conditions, the stiffness of the system is defined by the slope of the IC at a given operating point. In our study, these slopes were higher in hemiparetic subjects compared to healthy subjects. This may not be associated with an increase in passive stiffness since passive stiffness is reportedly unchanged in hemiparetic arms compared to normal [32]. The finding that stiffness was greater in hemiparetic subjects, despite the lack of muscle coactivation, may partly be explained by changes in muscle elasticity due to altered mechanical properties of motor units in these patients [39].

The significance of coactivation of agonist and antagonist muscles in the regulation of joint stiffness and the control of stability of posture and movement has been widely discussed (for recent review see [62]). For example, normal subjects use a coactivation strategy to transiently increase stiffness in order to stabilize unpredictable and unstable loads [41,57]. In hemiparetic subjects, the use of coactivation strategies to stabilize the limb in different parts of the articular range of the elbow have not previously been described, although changed coactivation patterns have been reported during maximal voluntary isometric efforts at the elbow [14]. Results of the present study imply that coactivation is associated with specific zones in angular coordinates and that this spatial aspect of coactivation may play a substantial role in stability of posture and movement.

Despite the general increase in stiffness of individual ICs, positional instability was observed in those subjects with absent or only phasic recruitment of antagonist muscles (subjects H5–H10). The abnormal arm oscillations may be related to hyperactivity in stretch reflex loops [8,15], a common finding in hemiparesis. However, aside from stretch reflex thresholds, other aspects of hyperactive stretch reflex activity have not been correlated with the type or magnitude of the functional deficit [11,49,61]. Since the size of the coactivation zone was significantly negatively correlated with the degree of the clinical sensorimotor deficit, it is likely that the main factor responsible for arm instability may have been the absence of a coactivation zone in an appropriate part of articular space. In this case, after unloading, the limb would flex into a silent zone in which extensor muscles would not provide adequate deceleration until the extensor zone of activation is reached. Eventually, extensor muscles would be activated and return the limb to the silent zone. Then, flexor muscles would not be activated in time to prevent a large extension of the limb and another cycle of oscillation would result. Thus postural instability may be related to the presence of a silent zone in the articular range even though stiffness of individual muscle groups outside of this zone may be high.

In hemiparetic subjects, a fundamental motor impairment is likely the inability to modify control patterns according to the task requirement in addition to the general limitation of the set of available control patterns. Several studies have demonstrated that hemiparetic subjects generally show increased coactivation even during slow purposeful movements (e.g., pointing [49] and locomotion [45]). On the other hand, in the present study, most subjects were not able to prepare a coactivation zone in anticipation of unloading. These demonstrations of inappropriately increased or decreased coactivation indicate that the ability to regulate coactivation commands according to the motor task may be impaired in some hemiparetic subjects.

The patterns of behavior seen in hemiparetic subjects may not be considered entirely abnormal [47,50]. It is likely not difficult to train healthy subjects to produce prolonged decaying oscillations in response to unloading with characteristic reciprocal bursts of activity in agonist and antagonist muscles. Assuming that this is the case, the role of the coactivation command in postural stability in
both populations of subjects can be described by the relationship between the logarithmic decrement of decay and the width of the coactivation zone (Fig. 13). Two states can be distinguished. A stable response to unloading is observed when \( C > -10^p \). This was the case for all but one healthy and four of the ten hemiparetic subjects. When \( C \leq -10^p \), the system was unstable, as was the case in one healthy and six hemiparetic subjects. Healthy subjects are likely able to voluntarily regulate C commands in order to select desirable stability states, while this would not be possible in some hemiparetic subjects.

The C command, however, may not be the only factor contributing to postural stability and movement. Other factors are the velocity-dependent properties of muscles and proprioceptive feedback. A more in-depth analysis of the problem may require the use of a dynamical model (see for example [63]).

4.4. Localized or distributed control of coactivation zones?

The presence of ICs in different initial arm configurations in hemiparetic subjects implies that the mechanisms for the production of ICs and shifts in ICs (in the limited range investigated) are generally preserved following discrete brain lesions involving mainly the parietal lobe and/or the internal capsule and subcortical structures [51]. The generalizability of this finding is, however, necessarily limited to the subset of patients and the articular ranges investigated in this study. In our patients, the findings of instability and the lack of coactivation zones imply that the ability to adequately specify coactivation commands may be lost or impaired.

Although the level at which movement is planned and controlled is unknown, these functions are likely distributed throughout cortical, subcortical and cerebellar structures. The parietal cortex may coordinate attention with goal-directed movements and may play a role in the production of the movement itself [30,33,43]. For example, cellular activity in both motor and parietal cortices is selectively tuned to the direction of a goal-directed movement [30,43]. Kalaska [42] has suggested that the parietal cortex may form part of the central control mechanism coding the location of the target as well as the kinematic parameters of the limb during goal-directed movement. The supplementary motor and premotor cortices have also been implicated in the formation of the motor command based on lesion studies in monkeys in which disturbances in visually guided reaching movements were demonstrated [37,46].

The cerebellum also appears to play a fundamental role in the regulation of reciprocal and coactivation muscle patterns during movement (see [62] for recent review). In particular, Purkinje cell discharge decreases during tasks requiring coactivation of the forearm and displays reciprocal activity during alternating flexion and extension movements [26]. Averaged activity of cerebellar neurons during single-joint movements in monkeys (Fig. 3 in [23]) may be interpreted as a superposition of ramp-shaped R and C commands [18]. However, the cerebellum may not be the only structure involved in the regulation of coactivation and stiffness. Humphrey and Reed [41] identified two distinct populations of neurons in the primary motor cortex reminiscent of R and C command patterns that were activated when monkeys performed tasks requiring reciprocal inhibition or coactivation of the forearm respectively. Thus the origin of descending reciprocal and coactivation control may be distributed such that lesions in any one structure may result in only partial movement disruption.

Anatomically, it is known that selective basal ganglia lesions do not affect voluntary movements of the extremities while lesions of the anterior or posterior limb of the internal capsule lead to initially severe motor impairment followed by recovery [25]. Lesions least likely to allow full sensorimotor recovery are those in the posterior limb of the internal capsule combined with damage to the lateral thalamus. In the monkey, axons of the primary motor cortex, premotor cortex and the supplementary motor area pass through the middle third of the posterior limb of the internal capsule, the capsular genu and the anterior limb respectively. Therefore, small capsular lesions can disrupt the output of functionally and anatomically distinct motor areas selectively. However, in the clinical literature, the anatomical association with the functional deficit following stroke is less distinct [29]. Unilateral hemispheric damage results in deficits in, among others, the ability to make smooth and accurate visually guided arm movements in external space [21,49,60]. Distinct aspects of movement (speed, inter-joint coordination, precision) are differentially impaired depending on the side of the hemispheric lesion [24,65]. In our study, the lack of coactivation zones was not related either to the laterality or the location of the lesion favoring the hypothesis of distributed control of coactivation. A more complete understanding of the relationship between localization and function may be gained from studying specific aspects of motor control in different groups of patients (i.e., stroke, Parkinson and cerebellar patients).

In conclusion, the spatial characteristics of coactivation may be essential determinants of behavior allowing the intact system to chose between a stable position or oscillatory movements. Our results imply that, in healthy subjects, coactivation zones are prepared in advance of unloading and may involve a feedforward mechanism. Disruptions in motor control following hemispheric stroke may result in part from a deficit in the specification of spatial zones for muscle coactivation.

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