Abnormal force–EMG relations in paretic limbs of hemiparetic human subjects

A TANG AND W Z RYMER

From the Biomedical Engineering Program and Departments of Physiology and Neurology, Northwestern University, Chicago, USA

SUMMARY The relations between surface EMG and isometric force generated by elbow flexor muscles were compared in normal and paretic limbs of 17 hemiparetic human subjects. Similar analyses were performed on both arms of 11 normal subjects. In almost half of the hemiparetic subjects examined (8/17), the slope of the relation between elbow flexion force and surface EMG, measured over the biceps-brachialis and brachioradialis muscle groups was increased in the paretic limb. A mechanism based on anomalous reductions in mean motor unit discharge rate in paretic muscles is advanced as the most likely cause of the findings.

The muscular weakness that occurs in the syndrome of the “upper motor neuron” is attributed largely to a loss of descending excitation to the spinal segment.¹ The relevant descending excitatory projections are interrupted, characteristically, by compressive or ischaemic lesions either of the relevant neurons, or of the fibre pathways as they traverse the basal ganglia or brainstem. While there is little doubt that loss of descending excitation is an important component of the weakness, there are now several observations suggesting that some disorganisation of the motor output at the segmental level arises, and that this disorganisation may also contribute to the muscle weakness. For example, Andreassen² and Rosenfalk and Andreassen,³ have reported that motor unit discharge rates are abnormally low in spastic muscles, and abnormal patterns of discharge have also been reported in a number of other supraspinal neurological syndromes.^{4,5}

Our own interest in a possible intrinsic segmental contribution to muscular weakness in supraspinal lesions was aroused by studies performed in an animal model of spasticity, the decerebrate cat with an added dorsal hemisection of the spinal cord.^{6,7} This model displays a number of the cardinal features of the upper motor neuron syndrome below the spinal lesion, including mus-

cular hypertonia and a prominent clasp-knife reflex. In addition, the slope of the relation between mean rectified intramuscular electromyogram (EMG) and isometric force was increased by a factor of four to sevenfold after the spinal lesion was introduced.⁷ This change in force-EMG slope resulted from a substantial fall in mean motor unit discharge rate, which caused the force output for each motor unit to drop substantially. As a consequence, many more motor units had to be recruited in order to produce a given level of force, thereby altering the force-EMG relations. In view of these animal findings, we attempted to determine whether anomalous reductions in motor unit discharge rate could contribute to the weakness accompanying many supraspinal lesions in man. We chose to investigate muscles of hemiparetic subjects, because the presence of an ostensibly normal contralateral limb allowed us to make detailed quantitative comparisons between normal and paretic sides of the one subject. However, rather than immediately attempting an evaluation of recruitment and discharge rate patterns of isolated single motor units, we reasoned that the relation between surface EMG and isometric force might provide a better global measure of motor unit performance, in an analogy with the studies in the animal model of spasticity (see refs 8 and 9 for evaluation of force-EMG test procedures). Our findings were that in almost half of the hemiparetic subjects examined, the slope of the relation between biceps-brachialis and brachio-

Address for reprint requests: Dr WZ Rymer, Department of Physiology, Ward 5-319, Northwestern University Medical School, 303 E Chicago Avenue, Chicago, IL 60611, USA.

Accepted 16 June 1981

radialis EMG and elbow flexion force (measured at the wrist) was abnormally increased on the paretic side. The reasons for this finding, including the possible contributions of abnormally low motor unit discharge rates are discussed. Portions of this material have appeared previously in abstract form.¹⁰

SUBJECT CHARACTERISTICS

The relations between isometric force and EMG were examined in biceps-brachialis (BB) and brachioradialis (BR) muscle groups of 11 normal and 17 hemiparetic human subjects. The hemiparetic subjects were chosen largely from the outpatient clinics of the Rehabilitation Institute of Chicago (RIC). The normal controls were selected from students and faculty of the Northwestern University Medical Center and the RIC.

Patients

The hemiparetic subject group consisted of 10 males and seven females ranging in age from 25 to 69 years. Of these 17 subjects, hemiparesis was attributed to stroke in nine, to craniocerebral trauma in five and to demyelinating disease in two. The cause was uncertain in one case. The duration of the hemiparesis ranged from 4 months to 23 years. The hemiparesis was right sided in eight and left sided in nine subjects. Sixteen hemiparetic subjects were right handed, and one was left handed. The 11 normal subjects were selected so as to provide a roughly comparable age (22-63) and sex distribution (eight male, three female). All of the normal subjects were right handed. The hemiparetic subjects were chosen on the basis of an *exclusively unilateral motor disorder* with little or no associated sensory, affective or cognitive disturbance. A subject also had to have sufficient power and co-ordination of the affected arm to maintain a stable isometric flexion force of at least 2 kg weight at the wrist for 10 seconds. Subjects with tremor, ataxia, dense hemiplegia, or contractures were excluded from our study. Each hemiparetic subject was given a detailed neurological examination by one investigator (WZR). The power, tone and tendon reflexes in both arms were quantified using an arbitrary scale of 0 to 5. For power, this scale was ordered so that normal power equalled 5, and complete paralysis was 0. For tone, marked hypertonia registered as 5, and normal tone as 0 or 1. Hyperreflexia was also quantified with a similar scale, in which marked hyperreflexia with clonus registered as 5, and absent reflexes as zero. In addition, power was quantified by estimating the greatest elbow flexion force that the subject could reliably reproduce against the load cell. While the values for power certainly underestimated maximum force output, they still served to provide a more objective standard than the clinical scaling procedure. We also assessed the range of active and passive movement, and the circumference of each arm was measured at a fixed point above each elbow flexure.

Methods

EMG recordings

Bipolar surface EMG recordings were made from biceps-brachialis and brachioradialis muscle groups on each side using either 1 cm diameter silver plated electrodes, or pregelled, disposable 2 cm electrodes (3M company). For biceps-brachialis recordings, electrodes were placed on each arm at a specified distance from the elbow flexure, precisely in the mid-line of the limb. The brachioradialis electrodes were placed over the muscular prominence of the muscle on each arm, at equivalent distances below the elbow skin flexure. Electrodes were aligned in the longitudinal axis of the limb, with their centres 50 mm apart on each arm. Large ground electrodes were placed at the wrist. Recording electrodes were also placed over the triceps brachii muscle groups on each side, so that we could detect any cocontraction.

EMG signals were first processed by a small peripheral preamplifier with a gain of 120, band-pass filtered (30-10 KHz), full wave rectified and further amplified by a factor of 40. Additional low-pass filtering, selected according to the A-D sampling interval, was used to prevent aliasing of digitised data. The roll-off of the band-pass filters was 12 dB/octave.

Potential errors in EMG analysis

Although the band width of our EMG recording system was substantial (10 Hz to 10 kHz) our computer sampling rate was limited to 250 Hz. In order to prevent aliasing, the EMG was subjected to further low-pass filtering, which had the effect of reducing power within the physiological range of the EMG power spectrum. The power reduction, however, occurred in that portion of the spectrum which is determined largely by the shape of the motor unit action potential, rather than by the level of recruitment and rate modulation. There are no established alterations in the shape of motor unit action potentials in hemiparesis (although see later in Discussion), so that we have no particular reason to believe that our choice of filter cut-off would produce any artifact in our mean rectified EMG estimates.

Testing procedures

As shown in fig 1, measurement of EMG and isometric force output of elbow flexors was made with a subject seated comfortably so that his shoulders lay parallel to the axis of the load cell. The wrist was inserted through a rigid ring attached to the load cell and for the hemiparetic limb, the wrist was held firmly within the ring with the help of a strap. The elbow of the arm to be tested was flexed to 90°, and the shoulder abducted to about 70°; the forearm was horizontal. A rigid splint attached to the frame of the device was used to stabilise the upper arm and to prevent forces arising from shoulder or axial motion from being transmitted to the wrist and load cell. We took considerable pains to ensure that the axis of force production at the wrist was in line with

Fig 1 Positioning of subject during the test. Subject flexes wrist against an immovable handle attached to a load cell. Force magnitude is fed back to an oscilloscope in front of subject, and he uses this force signal to follow a computer controlled target. The test is repeated in the same fashion for the other arm.

the axis of the load cell, and that rotational and off-line forces were minimised. While the latter objective was realised readily in the normal subject population, there was often a tendency for the paretic limb to pronate with increasing force, thereby inducing rotational forces. In order to prevent this, we allowed the hemiparetic subject to establish the most stable wrist position of the paretic limb in terms of pronation-supination. Because the orientation of the wrist is an important factor in determining the balance of EMG in the various elbow flexor muscles,¹¹ we then replicated this position precisely in the non-paretic limb.

The protocol required that the subject exert a specific force against the handle of the apparatus, to match the position of a target window displayed on an oscilloscope screen. The position of the target window and sequence of targets was regulated by a laboratory minicomputer (Digital Equipment Corp PDP11/03). After each matching period, the target window reverted to a zero force level, allowing the subject to rest, usually for at least 9 seconds. Approximately nine different force levels were displayed in a random sequence in a given experimental run, and each force level was repeated up to four times in a given experiment. The range of forces examined also varied with different subjects depending upon the degree of paresis. For this reason, we uniformly examined the responses of the paretic arm first, and then used an identical range of forces for the normal limb. The maximum force at the wrist reached 9 kg in some subjects, however, most of our subjects were required to produce 5 kg or less. The data collected consisted of 5 second epochs of EMG from biceps-brachialis and brachioradialis electrode pairs, together with the force measured at the wrist. These data were usually sampled every 5 ms although some data were collected using 2ms samples. Force and EMG

records were digitally converted and stored on digital disks for latter analysis.

While the best general measure of muscle force is undoubtedly net torque at the wrist (rather than linear force), for the sake of convenience, we continued to use the level of force measured at the load cell as the unit of measurement. This is because the force generated at the wrist by elbow flexor muscles should have the same moment arm in each limb, provided that the forearms are of equal length (which they were), and that position of the wrists in the holder was precisely matched (which we were careful to ensure).

Choice of joint angle

Ideally, we would have preferred to examine force-EMG relations of a particular muscle at several different muscle lengths. Because of the possible onset of fatigue in paretic muscles and our need to document force-EMG relations precisely at a given length, we opted to examine only one joint angle on any given experimental day. Accordingly, we chose a rather flexed state of the elbow (90°) since effects of rate modulation of motor units (which we believe to be involved in any EMG abnormality) is maximal in shortened muscles.¹²

Data analysis

Force and EMG records were displayed and a standard one or two second interval designated over which mean rectified EMG and force values, standard deviations and coefficients of variation were calculated for all records. These mean force and EMG values were then plotted on appropriate coordinates, and straight lines fitted using least-squares regression. In order to provide a reference standard for comparing the level of EMG in the paretic and non-paretic limbs, we also estimated the mean regression slopes and their standard deviations for our whole population of normals and for the normal (non-paretic) limbs of our hemiparetic subjects.

Results

Our major finding was that in almost half of the hemiparetic subjects examined, the amount of EMG produced per unit force was substantially increased in elbow flexor muscles of paretic arms. As an illustration, we will describe the force-EMG relations for one particular hemiparetic subject, and then document the patterns or response for the total population.

Comparison of EMG responses on normal and paretic sides

Figure 2 shows examples of the rectified surface EMG recordings from the biceps-brachialis muscle groups on the paretic (2a) and non-paretic sides (2b) of one subject. While the isometric force level

Fig. 2 Two records of force and surface EMG data taken from the biceps-brachialis (BB) muscle groups of hemiparetic subject 14. Force and EMG were recorded simultaneously and sampled at 5 ms intervals. EMG records shown here were additionally low passed filtered at 50 Hz to prevent aliasing. (a) Taken from paretic muscle group. (b) Taken from normal muscle group.

is essentially identical on each side, the level of rectified EMG is greatly augmented in the paretic muscles. This apparent alteration of force-EMG relations in the paretic muscles did not result from cocontraction of antagonist muscles—there was no concurrent excitation of the triceps musculature.

The increased irregularity of the rectified EMG trace depicted in fig 2a suggests that the variance of EMG might well be abnormally increased in paretic muscles. In order to describe the EMG variability precisely and to quantify further the relation between EMG and force, a number of statistical measures of EMG and force output were derived, including the mean, standard deviation (SD) and the coefficient of variation (SD/mean) of both the EMG and isometric force. Both mean and coefficient of variation were calculated for 1–2 second data segments (exemplified in fig 2), at a number of different force levels in both arms of normal and hemiparetic subjects. Because the magnitude of this EMG variability could depend upon the mean background EMG level, and also to permit direct comparisons of EMG and force variability at different forces, we calculated the coefficient of variation of both the EMG and force at several different isometric loads in both normal and paretic limb.

Our finding was that the coefficient of variation of the EMG showed limited force dependence, and was usually not augmented in paretic limbs. For the subject depicted in fig 2, the coefficient of variation for BB EMG in the paretic limb was 0.99 at 2 kg and 0.91 at 3 kg force; an insignificant change. The nor-

mal BB muscles of the same subject showed a coefficient of variation that was broadly comparable (0.97) at 2 kg with only modest increase at the higher force level (1.07 at 3 kg). The coefficient of variation of the force was much smaller, being 0.06 at 2.0 kg. When assessed across the hemiparetic population as a whole, the mean coefficient of variation of BB EMG in the paretic limbs was 0.915 at low force and this increased slightly to 1.003 at the higher force level ($n=17$). For the normal population, the coefficient of variation for BB varied from 0.99 at 2 kg up to 1.08 at the higher force level (3–4 kg); again a modest force dependence. Similar observations were made for BR EMG responses. In sum, for both normal and hemiparetic subjects, the coefficient of variation of EMG remain relatively constant or increased slightly as forces were increased and showed no significant difference between paretic and normal limbs. It follows that comparisons of force-EMG relations between normal and paretic sides can be made quite directly.

Quantification of force-EMG relations

For each subject, the mean EMG and isometric force levels were measured over a range of forces, providing a series of paired force and EMG values for each muscle group in both arms. Each data set consisted of approximately 40 pairs of force and EMG points, which were then plotted on to appropriate axes (see Methods).

Figure 3 shows a series of points derived from the paretic and normal BB muscles of the same subject depicted in fig 2 (subject 14). While the scatter of the data points collected from the paretic side is somewhat greater, it is clear that the force-EMG relations are well fitted by straight lines and

Fig. 3 Mean force-EMG data and corresponding regression lines from biceps-brachialis (BB) muscle groups of hemiparetic subject 14. Force is measured at the wrist. Open circles and solid line represent data and regression line from paretic BB muscle group. Closed circles and dashed line represent data and regression line from normal muscle group in same subject. Note that the slope derived for the paretic limb is greater than that of the normal limb (0.172 mv/kg paretic, versus 0.054 mv/kg normal).

that the two data sets are entirely separable. Given the straight line relation, the slope and intercept constants can serve to characterise the force-EMG relations quite precisely. In this instance, the slope of force-EMG relation is approximately *three times* greater on the paretic than on the normal side (0.172 mv/kg vs 0.054 mv/kg). These slopes are also statistically separable (*t* test slopes, $p < 0.05$).

For the range of forces that could be examined, force-EMG relations proved to be linear in all 17 hemiparetic and 11 normal subjects, with ordinate intercepts usually close to 0. Regression slopes were uniformly significantly different from zero, with correlation coefficients reaching statistical significance (at $p < 0.01$) in all but one subject. We attempted to quantify further the difference between force-EMG relations in the paretic and normal limbs by deriving a ratio of regression slopes, expressed as slope/weaker limb divided by slope/stronger limb. This approach is based on the premise that patterns of motor unit activation in the non-paretic limb of the hemiparetic subject are essentially normal (see Discussion), and that the force-EMG relations of the non-paretic limb are likely to provide more appropriate controls than any more broadly based population measure. We depend also on the fact that the form of the force-EMG relation was consistently linear on normal and paretic sides. The interpretation of these slope ratios is that ratios greatly exceeding 1

suggest a substantial augmentation of force-EMG slope on the paretic side. For the case illustrated in fig 3, the slope ratio was 3.169.

Ideally, the slope of the force-EMG relation should be expressed in terms of millivolts of surface or intramuscular EMG per unit of force at the muscle tendon, and compared against some specified range of normal. However, we are at present unable to estimate the force output of individual muscles directly. Moreover, the range of normality for our force-EMG relations proved to be very wide, with substantial overlap between normal and hemiparetic groups, supporting previous observations of this type.¹³ For example, the mean BB slope in hemiparetic limbs was $0.05 \text{ mv/kg} \pm 0.04 \text{ SD}$ ($n=17$) with range of 0.005 to 0.165 mv/kg, while the mean slope for normal subjects was $0.07 \text{ mv/kg} \pm 0.03$ with range of 0.04 to 0.155 mv/kg ($n=11$). It is likely that slope differences between different subjects could have arisen partly out of differences in muscle bulk, or from variations in the electrical properties of intervening soft tissue (see Discussion). The latter effects are less likely to provide major sources of error in data collected from both arms of the same subject.

Population summary

Figure 4 summarises the slope ratios for hemiparetic subjects, and compares them with the equivalent ratios for normal subjects examined under identical conditions. (In the normal subjects, the ratios were calculated with the slope of the non-dominant limb in the numerator.) These plots reveal that the slope ratios for hemiparetic elbow flexor muscles are much more dispersed than in normal subjects, resulting in a somewhat larger mean value (BB 1.71 paretic vs 1.19 normal, BR 1.59 paretic vs 0.98 normal) and an even larger standard deviation (BB 1.37 vs 0.38; BR 0.84 vs 0.58). While there is clearly a substantial clustering of slope values near 1 in both normal and abnormal subject populations, there appear to be a number of hemiparetic subjects in whom the slope ratios for BB and BR muscles are greatly increased (8/16 for BR, 7/16 for BB). For the BB muscles these subjects had slope ratios ranging from 1.6 up to 5.2, and the slopes on the two sides of the same subject were statistically separable in all but one case. The exceptional case illustrated, in which BB and BR ratios exceeding 3.5 did not reach statistical significance is derived from a subject in whom the range of forces traversed was too small to permit accurate regression analysis. However, for the same subject, the mean EMG values calculated at a particular force were quite different on each side (for example 0.04 mv paretic vs 0.02 mv normal at 2 kg), suggesting that the regression lines may have reached statistical signifi-

Fig 4 Distribution of slope ratios for hemiparetic and healthy subjects. Closed circles indicate the slopes of the two muscles of a pair were different from each other at a 5% level of significance (t test). Open circles indicate the slopes were not significantly different. The "X" and bar above each graph denote the mean and standard deviation of the slope ratios. The 16 circles on BB and BR plots of hemiparetic results do not represent identical groups—one subject differs in each panel because of defective EMG recordings on one set of electrodes.

cance with a larger data base. Triceps brachii EMGs were recorded in all subjects, and showed no evidence of sustained cocontraction.

Clinical correlations

If abnormal augmentation of EMG in paretic muscles results from a substantial lowering of motor unit discharge, we might expect to find a strong clinical correlation with the degree of muscle weakness (although see Discussion). This expectation was sustained, but only quite loosely. Figure 5 relates the maximum force achieved in the various experiments to the slope ratios of BB and BR muscle groups. While it is clear that the largest ratios arose in the subjects with the lowest force output, there were several subjects in whom higher ratios were associated with relatively good muscle power. The slope ratios were also plotted against the degree of spasticity, the subject's age, the duration of illness, the existence of stroke vs non-stroke etiology, the presence of flexion deformity, the handedness of the subject (right or

Fig 5 Relation between force-EMG slope ratio and maximum level of maintained isometric force generated by paretic arm. Slope ratios (slope weak/slope strong) are shown for 11 BB pairs and for 14 BR pairs. There is a rough correlation between very high values of the slope ratio and marked weakness.

left), and the degree of pronation supination of the limb, all without significant correlation. We were also unable to establish any relation between the magnitude of the slope ratio and the presence of muscle wasting (as estimated by arm circumference) although we found very little evidence of wasting in our subjects.

Discussion

Our results have shown that for almost half of the hemiparetic subjects examined, the EMG produced per unit force is augmented in elbow flexor muscles of the paretic limb as compared with the contralateral normal limb. Before we evaluate possible explanations for this EMG augmentation, we should first qualify the notion of normality as it is applied to the non-paretic limb. While it is well known that motor performance of the non-dominant arm may deteriorate after a dominant hemisphere lesion,¹⁴ (presumably because of a disturbance of information transfer from dominant to non-dominant hemisphere via commissural connections), there is no reported evidence of any alteration in patterns of muscle or motor unit activation in these non-paretic muscles. The non-paretic arm should therefore serve as an excellent standard for comparison.

Relation between motor unit discharge rate and surface EMG activity

Here we review the mechanism by which abnormally low motor unit discharge rates might

Fig 6 Diagrammatic representation of the relation between stimulus rate and tetanic force of muscle. Figure is based on results reported for the cat soleus.¹² However, all known muscles and motor units show such sigmoid relations.

give rise to abnormal force-EMG relations (see also ref 7). As shown in fig 6, the force produced by a typical muscle is a sigmoidal function of the rate of stimulation of the muscle nerve.¹¹ At very low stimulus rates (here less than 6/s), the force response is largely unfused, and small rate increases produce little augmentation in force. The relation between stimulus rate (or discharge rate) and tension is known to be comparable for single motor units. If discharge rate of single motor units is severely depressed in spastic-paretic states, then each individual motor unit contributes relatively little force so that many more motor units must be recruited to produce a given level of muscle force. However, while the force output of each motor unit is low under these conditions, the electrical activity of each unit would still be comparatively normal, thereby producing a net EMG augmentation per unit force increase. Although the locus and shape of the relation between discharge rate and motor unit force will certainly vary in different muscles, the sigmoidal form undoubtedly holds quite generally and would apply to motor units of the biceps brachialis muscles. As a consequence, the mechanism of severely reduced motor unit discharge rate can also be advanced to explain EMG augmentation in paretic human elbow muscles.

Our explanation for EMG augmentation in paretic muscles depends on the premise that motor unit discharge rate is significantly lower than it ought to be for any given level of descending excitatory command. While any reduction of motor unit discharge rate would mean that additional motor units would have to be recruited to

achieve some criterion force level, the most significant augmentation of motor output would arise if motor unit discharge rate fell onto the initial flat part of the rate-force relation (cf fig 6). We have not yet attempted to establish the relevant rate-force relations of individual motor units directly, largely because they would have to be assessed on the appropriate set of motor units. Such an experiment poses substantial technical problems in the biceps-brachialis muscles.

Factors responsible for EMG augmentation

A number of mechanisms other than anomalous reductions of discharge rate may have been responsible for the EMG augmentation. For example, wasting of muscle would be expected to lead to a relatively modest alteration in the electrical activity of the individual muscle fibre, but to a rather more substantial reduction in force output. This differential effect could arise because the amplitude of the action potential varies in proportion to fibre diameter, whereas fibre tension varies in proportion to the fibre cross sectional area, (a squared relation).¹⁵ The outcome would be that for a given force level, more motor units would have to be active, producing an augmentation in EMG output per unit force. However, while we cannot exclude subtle changes in muscle mass, our measurements of limb circumference did not reveal any significant wasting, and there was no correlation of EMG augmentation with any differences in limb circumference. It seems unlikely that wasting of paretic muscles was responsible for our findings.

Unexpected EMG augmentation could also have arisen as a result of a reduction in the conduction velocity of muscle fibres in the paretic muscles. Such a reduction would be manifested as a slowing in the time course of the motor unit action potential, and would induce a shift in frequency of the power spectrum of the EMG, with more power being distributed over lower frequencies.^{16, 17} This decline in conduction velocity would of itself augment total EMG power¹⁷ producing augmentation of rectified EMG. In addition, since the skin acts as a band-pass filter, a substantial shift of power to frequencies falling below the low-pass cut-off might induce an additional increase in power measured at the skin surface. Reductions in fibre conduction velocity (and the associated changes in the EMG power spectrum) could arise as a result of muscle fibre wasting, as a result of muscular fatigue in the paretic limb,^{17, 19} or from neuromuscular pathology. In each of these cases, we would anticipate

significant alterations in the shape and time-course of the motor-unit action potential. With the exception of occasional instances of lower-motor neuron type disorder (for example ref 18) no such alterations have been described in hemiplegic subjects. Moreover, while some degree of fatigue may have arisen in the course of our experiments, fatigue was probably not primarily responsible for our observations, because EMG augmentation was evident in the earliest recordings from the paretic limb, and its magnitude did not correlate with the temporal order of collected trials. The contribution of wasting has been assessed in the previous paragraph.

Anomalous EMG augmentation could also be caused by contraction of antagonist muscles, which would reduce net force output without altering agonist EMG. However, as we reported in Results, EMG recordings from triceps brachii muscles did not reveal any sustained cocontraction. A related possibility is that the distribution of activity in different flexor muscles responsible for force development at the wrist is modified in the hemiparetic subjects. This modification might produce an altered relation between surface EMG and *net* force, simply because one particular muscle assumed a greater role. Under these conditions, augmentation in BB EMG should be accompanied by a reduction in EMG of synergists (such as brachioradialis). No such reductions were recorded. Although we sampled rather few muscles, we found no evidence of any major disruption of agonist muscular synergy.

By exclusion, it appears that a reduction of mean motor unit discharge rate is the most likely cause of the EMG augmentation, a prediction supported by recent reports describing such rate reductions in hemiparetic human subjects.^{2,3} However, it is acknowledged that the EMG changes will probably have multiple causal factors, including some degree of atrophy, fatigue and alteration of muscle biophysical characteristics.

Clinical correlations: power or effort?

Reductions in force output for each motor unit would mean that many more motor units would have to be activated if the muscle were to reach some required level of force. This situation could give rise to weakness when all readily accessible motor units are recruited, and to an increased sense of effort at lower levels of activation, since a stronger "central command" would be required to generate a given level of force. This weakness and increased sense of effort might also be accentuated if higher threshold motor units are

more readily fatigued, especially if the total number of available motor units were depleted.²⁰ In sum, there is good reason to anticipate that the EMG abnormalities would correlate well with the effort expended by our subjects, but this prediction was not tested directly.

A number of studies have reported on the increased sense of effort that is associated with force output by paretic limbs (for example ref 21). In the absence of quantitative EMG measurements, we cannot estimate any segmental contribution to the weakness in these particular subjects, nonetheless, our findings in similar subjects suggest that anomalous reductions in motor unit discharge may well have been important.

The authors are grateful to the staff physicians of the Chicago Rehabilitation Institute for referring patients for this study. They are also grateful to Dr J Schmeil for her advice on statistical procedures.

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