SEQUENTIAL MUSCLE ACTIVATION IN THE HEMIPARETIC ARM

ABSTRACT: This study investigated the chronological activation sequence of multiple joint movements of the hemiparetic arm in patients with central hemiparesis compared to healthy test subjects.

Twelve patients with central hemiparesis and eight healthy control subjects were studied. First, in rapid abduction movement of the upper limb, the electromyographic activities of the middle part of the deltoid muscle, the brachial biceps muscle and the extensor muscles of the fingers, were registered. Second,

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in rapid flexion of the arm, the electromyographic activities of the ventral part of the deltoid muscle, the brachial biceps muscle and the superficial flexor muscles of the fingers, were measured.

From the EMG data registered, activation duration, activation latency and the innervation sequence were determined and compared between the patient group and the control group.

In the patient group, a significant prolongation of the activation duration was shown only in abduction. However, the activation latency was significantly prolonged in both movements compared to healthy test subjects. In the innervation sequences, a simultaneous activation was most frequently shown in healthy subjects. In healthy subjects, the deltoid muscle also usually functioned as leading muscle, whereas there was sometimes a shift distally to the brachial biceps muscle in the hemiparetic patients.

The speed of rapid multiple joint movements in hemiparetic extremities seems to be unaffected in certain movements (anteversion), in others (abduction) it seems to be significantly reduced. This, as well as the fact that the activation latency is significantly longer in the hemiparetic limbs should be taken into consideration when choosing rehabilitation exercises.

KEY WORDS: HEMIPARETIC ARM, POLYARTICULAR MOVEMENT, CHRONOLOGICAL ACTIVATION SEQUENCE.

INTRODUCTION AND RATIONALE

Everyday movements require sequential activation of the muscles involved. In grasping movements of the upper limb, limb muscles that are close to the trunk and proximal are activated first (Soechting and Flanders, 1989). According to Levin (1996), the level at which movement is planned and controlled is unknown, and most probably these functions are distributed throughout cortical and subcortical areas. Distal limb segments are mainly represented in the primary motor cortex. The premotor, the supplementary motor and the primary motor cortex are available to the cerebral cortex for the innervation of proximal limbs and trunk muscles (Colebatch et al, 1990). The chronological coordination of motor cortical activity in performing voluntary movements is controlled by areas of the secondary motor cortex. Changes in the activation sequence of arm muscles in damage to the premotor cortex have already been observed (Freund and Hummelsheim, 1985).

This study was designed to establish whether an altered chronological activation sequence in the muscles involved can be observed in patients with typical central pareses, i.e. patients with a paralysis gradient increasing to distal.

SUBJECTS AND METHODS

Twelve patients with central hemiparesis following a lesion in the middle cerebral artery territory and a typical pattern of paresis were investigated. A control group consisted of eight healthy test subjects of same sex and comparable age. There were no restrictions of the passive mobility in the tested arm in either group. All test subjects were right-handed.

From the upright standing position, the participants of both groups were asked to execute a flexion (anteversion) and an abduction of the glenohumeral joint with the straight upper limb in direction of the accelerance transducer at the level of the shoulder. Whereas the palm of the hand of the investigated arm was rotated to ventral in flexion movement, it was parallel to the axis of the body in abduction. The test subjects were requested to perform these movements at maximum speed.

Patients were included if they had sustained a single cebro-vascular accident 4-6 months before, as documented by their medical history and confirmed by computer-assisted tomography scan and nuclear magnetic resonance. Subjects with neglect, apraxia, subluxation or pain in their upper limb were excluded.

In the anteversion movement, surface electrodes were positioned on the ventral part of the deltoid muscle, the biceps muscle in the middle upper arm and the superficial flexor muscle of the fingers.

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Figure 1: Testing procedure.

In the abduction movement, the electrodes were placed on the middle part of the deltoid muscle, the brachial biceps muscle and the extensor muscle of the fingers. A weight of 600 g was fixed to the hand to intensify the electromyographic activity.

The EMG signals were entered into a commercial data system via an analog/ digital converter after rectification and filtration (3 Hz to 3 kHz). The scanning rate was 1 kHz, so that a temporal resolution of 1 ms was possible.

To determine the duration of movement, an acceleration transducer that marked the total duration of the movement was applied at the level of the shoulder (Fig. 1).

In the healthy control subjects, investigation of the dominant and nondominant limb did not reveal any differences in the electromyogram pattern. In the patients, the measurements were carried out on the paretic side.

From the EMG data registered, activation duration, activation latency and the innervation sequence of the muscles co-innervated in the movement were determined in anteversion and abduction of the arm.

The duration of activation is the time from the electromyographic activity first measured up to reaction of the acceleration transducer at the level of the shoulder. It correlates with the movement duration.

The activation latency is the relative time interval up to activation of all recorded muscles. It corresponds to the time interval from the beginning of activation of the "leading muscle" first innervated to the beginning of activation of the muscle innervated last.

In order to be able to compare the patient group quantitatively with the control group in respect of the innervation latencies determined from the individual recordings in anteversion and abduction movement, the first electromyographic activation was given the value 0. Starting from this value, the time up to the beginning of activation in the subsequent muscle groups could be measured. With the same beginning of activation of different muscle groups, the same numerical values consequently result.







The t-test was used for statistical calculation of the duration and latency of activation between the groups after the empirical variance had been determined, the F test carried out and the level of significance laid down as alpha = 0.05. The innervation sequences were presented descriptively in tables.

RESULTS

All patients had sustained a unilateral stroke. Seven patients had clinical signs of spasticity and weakness on the right

side, and five patients on the left side of the hemicorps. Their mean age was 54.5 (8.9 years and eight of them were male. In the control group the registered EMG-activity was compared of the sidematched arms.

Figure 2 shows as an example the electromyographic activity of the abduction movement of a healthy test subject. In the lower recording, the arrow shows the signal of the acceleration transducer in contact of the fingertips in the horizontal, which corresponds to the end of the the



Figure 3: Electromyographic example of the hemiparetic limb of a patient in the abduction movement.

Table 1: Mean duration of activation in anteversion of the arm.

test	activation [ms]	
groups	χ	± S
normal subjects	642,4	158,5
patients	799,0	283,0 J 11.3. (a = 0,03)

Table 2: Mean duration of activation in abduction of the arm.

test	activation [ms]		
groups	Х	± S	
normal subjects	540	81) * (ar = 0.05)	
patients	694	164 f ($\alpha = 0.05$)	

Table 3: Mean activation latencies in anteversion of the arm.

test	activation latency [ms]	
groups	χ	± S
healthy subjects	56,4	54,0 $\mathbf{j} * (\alpha = 0.05)$
patients	169,5	121,9) (a = 0,03)

Table 4: Mean activation latencies in abduction of the arm.

test	activation latency [ms]	
groups	χ	± S
healthy subjects	26	21,2 $\mathbf{j} * (\alpha = 0.05)$
patients	130,8	127,6 f ($\alpha = 0,05$)

activation of the three muscle groups is simultaneous, followed by a steep rise in amplitude up to maximum activation. This activation level is maintained after abduction of the arm to the horizontal. Figure 3 shows an electromyographic example of the hemiparetic limb of a patient in the abduction movement. It becomes evident here that the activation of the deltoid muscle and the brachial biceps muscle starts simultaneously, whereas that of the extensor muscle of the fingers only starts with a latency of 35 ms.

The duration of movement (activation) was compared first of all for each test subject. Comparison of the mean values for the duration of the anteversion movement up to the horizontal (Tab. 1) showed a time of 642.4 ms (158.5 ms in the normal subjects and 684 ms (136 ms in the patients. The difference is not significant at the significance level of alpha = 0.05.

The duration of movement in abduction (Tab. 2) to reaction of the acceleration meter of normal subjects averaged 540 ms (81 ms. In the patients, this movement took 694 ms (164 ms. The abduction movement in the patients is thus significantly slower than in the normal subjects, i.e. by an average of 154 ms.

Tables 3 and 4 show the mean activation latencies in the comparison groups in anteversion and abduction of the arm. The activation latencies of the hemiparetic patients are significantly longer than those of the healthy test subjects.

Tables 5 to 8 show the innervation sequences in anteversion and abduction of each individual test subject. In anteversion of the healthy test subjects (Tab. 5), it is apparent that the deltoid muscle is the sole leading muscle in four cases. Simultaneous activation of all three muscles is recorded in three cases. There is simultaneous activation of the deltoid muscle and brachial biceps muscle in one case.

In anteversion in the patients (Tab. 6), the brachial biceps muscle is the sole leading muscle in six cases and the deltoid muscle in four cases. There is simultaneous activation of the deltoid muscle and the brachial biceps muscle in one case, and simultaneous activation of the brachial biceps muscle and the superficial flexor muscle of the fingers in another case. The innervation sequences in abduction in normal subjects (Tab. 7) are solely led by the deltoid muscle in three cases. Simultaneous activation of the deltoid muscle and the brachial biceps muscle as well as the deltoid muscle and extensor muscle of the fingers was found in one case each.

Simultaneous activation of all three muscles was registered in three cases.

The innervation sequences during abduction in the patients (Tab. 8) was led by the deltoid muscle as sole muscle in five cases, and by the deltoid muscle together with the brachial biceps muscle in four cases. The brachial biceps muscle was the sole leading muscle in three cases. Simultaneous activation of all three recorded muscles did not occur.

The frequency of the leading musculature decreases from proximal to distal, especially in anteversion in normal subjects, however simultaneous activation of all three muscles was found most frequently in anteversion in normal subjects. A shift to the distal brachial biceps muscle was shown in the hemiparetic limb in the patients and simultaneous activation of all three muscles was found most frequently in healthy normal subjects in both anteversion and abduction. Evidently, this enables more fluid, rapid and synchronous performance of a movement.

DISCUSSION

The amplitude of the EMG activity registered with surface electrodes is influenced by a series of variables and factors which are difficult to determine (e.g. cutaneous resistance, characteristics of the connective tissue structure and the musculature between electrodes). Numerical data for the amplitudes of muscular activity was therefore dispensed with.

After lesion to the primary motor system, there are alterations in the recruiting characteristics in motor units as well as histological changes in the affected musculature. However, the literature is contradictory in this regard (Slager et al, 1985, Dietz et al, 1986, Jakobsson et al, 1991, Datolla et al, 1993). In some cases, there appears to be atrophy especially of the rapid ATPase-rich type 2 fibers, and in some cases a transformation of type 1 into type 2 fibers appears to predominate. The latter indicates that there may be a tendency to facilitate further rapid movements by transformation. However, if rapid movements nevertheless cannot be implemented, a reduced use of neuronal elements is likely to be

Table 5: Innervation sequences	in anteversion of	of healthy subjects.
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test subjects Nr.:	Deltoideus [ms]	Biceps [ms]	Flexor [ms]
1	0	15	20
2	0	50	85
3	0	0	150
4	0	55	65
5	0	20	55
6	0	0	0
7	0	0	0
8	0	0	0

Table 6: Innervation sequences in anteversion of hemiparetic patients.

patients Nr.:	Deltoideus [ms]	Biceps [ms]	Flexor [ms]
1	128	0	128
2	28	0	171
3	36	0	0
4	57	0	129
5	0	0	171
6	416	0	314
7	36	0	157
8	0	271	328
9	28	0	129
10	0	136	164
11	0	71	157
12	0	25	45

Table 7: Innervation sequences	in abduction of	f healthy subject	s.
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test subjects Nr.:	Deltoideus [ms]	Biceps [ms]	Extensor [ms]
1	0	20	0
2	0	50	50
3	0	0	20
4	0	50	50
5	0	50	50
6	0	0	0
7	0	0	0
8	0	0	0

responsible (Rosenfalck and Andreassen, 1980). Recent studies have documented that decreased recruitment of agonist motor units rather than increased antagonist co-contraction predominates in hemiplegia (Gowland et al, 1992, Fellows et al, 1994). These developmental differences may explain the reduced speed of the abduction movement found in this study in patients as well as the relatively unaffected duration of anteversion.

patients Nr.:	Deltoideus [ms]	Biceps [ms]	Extensor [ms]
1	0	0	20
2	0	186	43
3	43	0	100
4	0	0	36
5	28	0	20
6	0	428	357
7	0	0	186
8	0	171	20
9	70	0	114
10	0	357	43
11	0	0	35
12	0	80	80

 Table 8: Innervation sequences in abduction of hemiparetic patients.

Since multiple-joint movements were investigated in this study, in contrast to those published in the literature (Knutsson and Martensson, 1980, Bourbonnais et al, 1989, Dietz et al, 1991), compensatory muscular influences (e.g. brachial biceps muscle) cannot be ruled out.

The activation latency was significantly longer in the hemiparetic limbs compared to that of healthy test subjects. This result largely corresponds to that of Tsuji and Nakamura (1987) who found a prolonged latency time from the beginning of EMG activity up to the beginning of the rise in tension in contraction of the gastrocnemius muscle in patients with central hemipareses.

Simultaneous activation was found very much more rarely in the investigated patients than in the healthy test subjects. The coordination between the muscle groups of the paretic limb thus appears to be markedly altered. The pattern of muscle activation in the patients varied from that in healthy subjects, indicating that the patients have a changed strategy for executing the complex movements investigated. In some cases, the brachial biceps muscle played a special role, mostly for reasons of joint mechanics and evidently because of the particular distribution of the spasticity. The anterior serratus, infraspinatus, supraspinatus and deltoid muscles which regularly participate in the abduction of the shoulder joint frequently demonstrate paretic changes. The changed activation patterns are also reflected in the results of the present paper. They include the innervation sequences that show a shift to the brachial biceps muscle in hemiparetic patients, whereas in healthy subjects the deltoid muscle usually functions either as the leading muscle or is simultaneously activated.

To summarize, there are sometimes appreciable changes in the electromyographic activity and the interaction of the affected limb muscles in central hemiparesis after stroke in the region supplied with blood by the middle cerebral artery and with a typical distribution pattern of the paresis. The speed of certain rapid multiple joint movements of hemiparetic limbs is unaffected (anteversion) whereas it is significantly reduced in other movements (abduction). There is significant prolongation of the activation latency in both movements compared to healthy subjects.

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