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ISOKINETIC STRENGTH, MACRO EMG AND MUSCLE BIOPSY OF PARETIC FOOT DORSIFLEXORS IN CHRONIC NEUROGENIC PARESIS

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ABSTRACT. Ten ambulatory patients with chronic dorsiflexor paresis due to prior poliomyelitis or lumbar root (LV) lesion and without recent decline of the foot dorsiflexor strength were examined with isokinetic strength measurement, macro EMG and muscle biopsy. Isokinetic strength measurement showed peak torques at 30°/s angular velocity ranging 6–44 Nm and at 240°/s 1–10 Nm. Mean of individual median macro EMG motor unit potential amplitudes was 2020 μV (SD 1040) which was 5–10 times higher than expected values in healthy subjects. Muscle biopsies showed a mean type 1 fibre proportion of 91% (SD 14) and a mean type 1 fibre area of 8561 μm² (SD 2773) which was about 2 times larger than those observed in healthy subjects. Peak torque and both motor unit potential amplitude and area were inversely correlated at 30 ($p < 0.01$ and $p < 0.025$), 60 ($p < 0.025$ and $p < 0.025$) and 120 ($p < 0.05$ and $p < 0.05$) °/s angular velocity, as were peak torque and type 1 muscle fibre proportion at 30 ($p < 0.05$) and 60 ($p < 0.05$) °/s angular velocity. Peak torque or macro EMG parameters were not correlated to muscle fibre area. The data suggest that the remaining muscle strength was directly correlated to the degree of collateral sprouting and that about half of it could be attributed to compensatory muscle fibre hypertrophy.

Key words: isokinetic strength, macro EMG, muscle biopsy, post-polio, LV root lesion.

Compensation for loss of motoneurons by collateral nerve sprouting has been shown experimentally (16) and is utilized in healthy man and in neuromuscular disorders (4, 10, 17, 9, 6). In muscles of the upper extremity there may be an age-related loss of as much as 50% of the motoneurons without noticeable muscle weakness (4). Studies of patients with neuromuscular disorders suggest even larger capacity to compensate for motorneuron loss (18) to maintain muscle strength.

Compensation for loss of muscle fibres might include muscle fibre hypertrophy. In muscle biopsy studies of patients with muscle weakness due to prior polio or lumbar (LV) root lesion a predominance of significantly hypertrophic type 1 muscle fibres was observed (2, 11). The increased type 1 muscle fibre proportion was suggested to be due to transformation of type 2 to type 1 fibres in response to long-term excessive use of remaining muscle fibres during the step cycle (2, 12). Studies of isokinetic strength indicated that the contractile properties of these fibres did not correspond to the histochemical muscle fibre type as determined by conventional ATPase techniques (24).

The aim of the present study was to evaluate to what extent compensation by collateral sprouting and by muscle fibre changes is utilized to maintain muscle strength at different degrees of stable, chronic neurogenic foot dorsiflexor paresis due to prior polio or lumbar root lesion.

PATIENTS AND METHODS

Ten patients, 6 men and 4 women, with muscle weakness after prior poliomyelitis (9 cases) or chronic LV root lesion (1 case) with a mean age of 65 (range 48–76) years were studied. The mean latency between the acute illness and the investigation was 36 (range 23–53) years for the polio patients and 14 years for the LV root lesion patient. All had a history of lower leg involvement during the acute stage and all exhibited typical electromyographical signs in the anterior tibial muscle, indicating compensatory collateral sprouting due to loss of motorneurons. At clinical examination foot dorsiflexor strength ranged from 3 to 5 according to Kendall et al. (15). One polio patient used two crutches or a wheelchair, one used two sticks and another one stick; the other patients walked unaided. Only patients who had experienced a stable foot dorsiflexor strength during recent years were selected for the study. In all, isokinetic strength measurements two years

earlier and at the present investigation showed that peak torque values at 30°/s were unchanged during this period.

Strength measurement

Maximal voluntary foot dorsiflexor strength was measured in isokinetic concentric movements with a dynamic dynamometer (KIN-COM 500H, Chattecx Corp., 101 Memorial Drive, P.O. Box 42887, Chattanooga TN, 37405, USA) at five different angular velocities—30, 60, 120, 180 and 240°/s according to Tollbäck et al. (24). Peak torque was determined at each velocity.

Macro EMG

Electromyographic recordings were made by macro EMG needle electrodes (Medelec 17915) and displayed on a Medelec oscilloscope (MS92B no 67127) connected to a microcomputer (Victor VPCII). Recordings were from motor units recruited at slight to moderate voluntary activation. Macro EMG recordings were analysed according to Stålberg (22, 23) using Stålberg Intersoft program Macro 4.1 1992.

Muscle biopsy

Muscle biopsies were performed in the tibialis anterior muscle by the percutaneous chonchotome method originally described by Radner (21). Cross-sections of 10–15 μm were cut in a cryostat operating at −25°C.

For light microscopical morphology and histochemistry, sections were stained with hematoxylin and eosin, modified trichrome (9), myosin-ATPase at pH 9.4, 4.6 and 4.3 (3, 20). The muscle fibre nomenclature was based on the ATPase stainability according to Brooke & Kaiser (3). Thus, fibres with a high content of acid-stable ATPase were termed type 1 while fibres with the opposite staining pattern were termed type 2. All fibres in a muscle fibre cross-section were classified and the total number of each type was estimated. Cross-sectional areas of the muscle fibres were measured by a semi-automatic technique used on photographs (MOP Videoplan, Kontron, Bildanalyse GmbH, Munich). 200 fibers from each biopsy were measured.

Statistics

Statistical analysis for correlations between the groups was made by means of the Spearman two-tailed rank correlation test. $p < 0.05$ was considered statistically significant.

RESULTS

Peak torque data are summarised in Table I. The peak torques ranged from 6–44 Nm at 30°/s to 1–10 Nm at 240°/s angular velocity.

Macro EMG data are summarised in Table II. Macro EMG recordings were made from 20 motor units in each patient. Median motor unit potential amplitudes and areas of the 10 patients were significantly correlated ($r = 0.99$, $p < 0.0005$). Median amplitude values ranged between 556 and 3652 μV. All these values were above the suggested, age-related maximum limits for healthy subjects (22). The mean of the

Table II. *Macro EMG data*

Case	Median amplitude (μV)	Median area (μVxms)
1	556	1676
2	2969	9252
3	3339	10464
4	1396	4401
5	2539	6679
6	3652	10694
7	1553	5324
8	1191	3991
9	1191	3108
10	1816	5634
M (SD)	2020 (1040)	6122 (3110)

Table I. *Isokinetic data*

Case	30°/s (Nm)	60°/s (Nm)	120°/s (Nm)	180°/s (Nm)	240°/s (Nm)
1	18	14	11	8	6
2	8	7	4	3	1
3	11	9	6	4	2
4	24	18	13	5	—*
5	6	6	3	1	—*
6	14	12	10	6	6
7	24	17	8	0	—*
8	44	32	22	15	10
9	16	13	9	5	—*
10	13	11	7	3	—*
M (SD)	18 (11)	14 (7)	9 (5)	5 (4)	5 (4)

* These patients did not reach the preset angular velocity.

Table III. *Muscle fibre data*

Case	Type I fibre (%)	Mean fibre area type I (μm^2)	Mean fibre area type II (μm^2)
1	98	6883	2581
2	99	9923	6148
3	97	13249	9337
4	93	12380	14067
5	98	9913	4567
6	100	6440	0
7	86	5424	7457
8	83	8624	13860
9	53	5298	10412
10	99	7477	16442
M (SD)	91 (14)	8561 (2773)	8487 (5339)

individual median motor unit potential amplitude values was 2020 μV (SD 1040). Median motor unit potential area values ranged between 1676 and 10 694 μVxms. The mean of the individual median area values was 6120 μVxms (SD 3110). This was about 5 times higher than corresponding mean values from healthy subjects (22). There was no correlation between macro EMG parameters and time elapsed since the acute illness.

Muscle biopsy data are summarised in Table III. The mean type 1 fibre proportion was 91% (SD 14). The mean type 1 muscle fibre area was 8561 μm^2 (SD 2773). This was about twice as high as corresponding values in healthy subjects (14). Type 2 fibre area was 8487 μm^2 (SD 5339). There was no correlation between muscle biopsy parameters and time elapsed since the acute illness.

Fig. 1 shows peak torque values at 30°/s and median macro EMG motor unit potential amplitudes of the 10 patients. There was a negative correlation between peak torque values at 30 ($r = -0.72$, $p < 0.01$), 60 ($r = -0.69$, $p < 0.025$) and 120 ($r = -0.57$, $p < 0.05$) °/s angular velocity and mean macro EMG motor unit potential amplitude.

There was a negative correlation between peak torque values at 30 ($r = -0.67$, $p < 0.025$), 60 ($r = -0.65$, $p < 0.025$) and 120 ($r = -0.57$, $p < 0.05$) °/s angular velocity and mean macro EMG motor unit potential area. There was no correlation at the higher angular velocities.

There was a positive correlation between peak torque values at 30 ($r = -0.62$, $p < 0.05$) and 60 ($r = -0.61$, $p < 0.05$) °/s angular velocity and mean

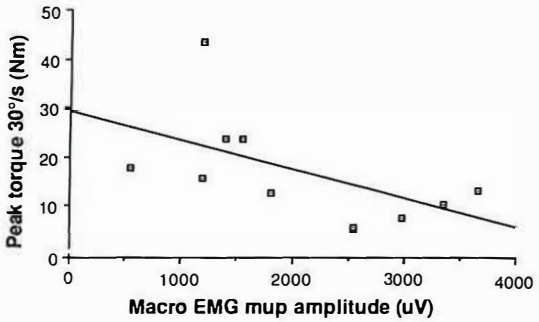

Fig. 1. Mean macro EMG mup amplitudes and peak torque values at 30°/s for the 10 patients.

type 1 muscle fibre proportion. There was no correlation at the higher angular velocities. There was a positive correlation between mean type 1 fibre proportion and mean macro EMG motor unit potential amplitude ($r = 0.68$, $p < 0.025$) and area ($r = 0.66$, $p < 0.025$). There was no correlation between the peak torque or macro EMG parameters and muscle fibre area.

DISCUSSION

This study shows that macro EMG motor unit potential amplitude and area and type 1 muscle fibre proportion are inversely correlated to strength in foot dorsiflexors of ambulatory patients with residual muscle weakness after prior polio or LV root lesion. It should be pointed out that the patients showed large interindividual variation of foot dorsiflexor strength and were selected with regard to a history or stable foot dorsiflexor strength.

Loss of motoneurons and compensatory collateral sprouting is probably the main reason for the observed increase of macro EMG potentials while increased muscle fibre size probably contributes to a limited extent. Macro EMG potentials were 5–10 times greater than those observed in healthy subjects (22). The muscle fibre hypertrophy has been considered never to increase the motor unit potential amplitude more than twice (22). The much larger increase of the motor unit potential may be due to an increased number of muscle fibres in the reinnervated motor unit. Considering the maximal increase in motor unit potential amplitude due to muscle fibre hypertrophy the present data indicate a loss of motor neurons of 60–80% of the anterior tibial neuron population, according to Stålberg (22). Macro EMG motor unit

potential amplitude and area were widely distributed within each patient. This probably reflects the heterogeneous distribution of the initial denervation and the restrictions of the collateral sprouting by the fascicle boundaries (16). There would remain a capacity for further collateral sprouting in some muscle areas but a higher mean macro EMG value probably indicates a lower such capacity.

The observed negative correlation between muscle strength and mean macro EMG motor unit potential would be expected if the original loss of mononeurons exceeded the compensatory sprouting capacity to maintain muscle strength. A further loss of motoneurons, due to age or other causes, compensated by collateral sprouting to maintain muscle strength, would not change the relation between strength and macro EMG motor unit potential if it occurred to a similar extent in all subjects. Our data showed no correlation between macro EMG parameters and time elapsed since the acute illness and thus no indication of significant time-related further increase of the macro EMG motor unit potential. Maselli et al. (18) followed macro EMG parameters in a group of late polio patients during "at least one year" and found no consistent increase or decrease of the macro EMG motor unit potentials. However, it was pointed out that the follow-up time was short. We suggest that some long-term increase of the macro EMG motor unit potentials during several years can not be excluded.

It has been suggested that increased muscle atrophy and weakness in the so called post-polio syndrome is due to inability to compensate for loss of motoneurons or to unstable neuromuscular connections (5, 6, 25); however, in several studies no correlation between new muscle weakness and electromyographical signs of ongoing denervation, impaired neuromuscular transmission or motor unit size could be established (5, 8, 18). The negative correlation between isokinetic strength and type 1 muscle fibre proportion probably reflects the effect of long-term excessive use of remaining muscle fibres on the muscle fibre composition. Previous studies have shown an increased type 1 fibre proportion in patients with electromyographically defined excessive use of the anterior tibial muscle fibres during walking probably due to transformation of type 2 to type 1 muscle fibres (2, 7).

The increased muscle fibre area as compared to healthy subjects (14) is in accordance with previous studies (2, 7). The muscle fibre hypertrophy is probably a significant compensatory mechanism to maintain muscle strength. The predominating type 1 muscle fibres had a mean area twice as large as that observed in healthy subjects (14). Since the type 1 fibre proportion was above 90% almost half of the muscle strength could be attributed to increased muscle fibre size provided that these muscle fibres have the same contractile properties as ordinary fibres. A previous study showed a discrepancy between the histochemical fibre type and relative isokinetic strength properties indicating that type 1 muscle fibres, which are transformed type 2 fibres (7), do retain their contractile type 2 fibre characteristics (24).

In conclusion, this study describes how motor unit size, muscle fibre size and types are related to muscle strength in chronic, neurogenic foot dorsiflexor paresis due to prior polio and lumbar root lesion. We suggest that further studies of these parameters are of interest to elucidate the pathophysiology of declining muscle strength in late post-polio.

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