STUDY OF GAIT CYCLES AND MUSCLE ACTIVITY PATTERNS IN PATIENTS WITH PROGRESSIVE NEUROMUSCULAR DISEASES.

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SUMMARY

The gait of 12 patients with progressive neuromuscular disorders have been studied with footswitches and EMG. In comparison with matched controls the patients walked with low velocity, a shortened duration of the time for heel on to ball on, and a prolonged duration of heel up to ball up. EMG:s tended to have long duration compared to the controls.

INTRODUCTION

One of the primary characteristics of progressive neuromuscular diseases is the continous impairment of gait, which often leads to wheel chair confinement. There are several valuable clinical descriptions of gait in these conditions (1,2,3,4).

Recently there has in addition been published a quantitative study on the pathomechanics of gait in Duchenne Musc Dystrophy (5). There is indeed a need of quantitative studies not so much for diagnosis, but rather because of the need to evaluate the results of orthopedic treatment instituted to prolong the period of ambulation (6,7,8).

To evaluate the results of treatment it is necessary to do several measurements over a long period of time, and a simple method of gait recording is therefore necessary. Recording with footswitches is both simple and fast, and should therefore be useful, provided it proves to be sufficiently sensitive to the changes in gait that are characteristic of progressive neuromuscular disorders. We have previously used footswitch recording to study gait in children, and grown up subjects (9,10), and we have the necessary controls for a study of the matter. We have therefore recorded the gait with footswitches in twelve patients with progressive neuromuscular diseases. In addition surface EMG was recorded from six muscles.

Patients

Twelve patients with a diagnosis of progressive neuromuscular disorder have been investigated. Ten were from Yugoslavia (University Hospital, Ljubljana, and University Hospital, Zagreb), and two were from Sweden (University Hospital, Linköping). The age, sex, and

diagnosis of the patients are presented in Table I. Fig. 1 shows profile drawings of the patients photographed in standing position. The ordinate behind each patient coincides with the plumbline, and the histograms behind summarizes the muscle strength (scale 0-5). From left to right the bars correspond to 1/ neck, 2/ shoulders, 3/ elbows, 4/ wrist and fingers, 5/ trunk, 6/ hips, 7/ knees, and 8/ ankles and toes.

All patients have been followed clinically for several years. The diagnostic procedures have included electromyography, motor conduction velocity determination, and muscle biopsy.

Table I. Age and sex of the patients. The diagnosis is indicated by SA (benign spinal muscular atrophy), and by DM (muscle dystrophy).

PAT No.	AGE (years)	SEX	DIAGNOSIS
SA 1	23	M	Spinal muscular atrophy
SA 2	12	M	"
SA 3	13	F	"
SA 4	17	F	"
DM 1	27	F	Muscle dystrophy 'limb girdle'
DM 2	10	F	"
DM 3	40	F	"
DM 4	17	F	"
DM 5	18	M	"
DM 6	25	M	"
DM 7	16	M	Muscle dystrophy Becker
DM 8	11	M	Muscle dystrophy Duchenne

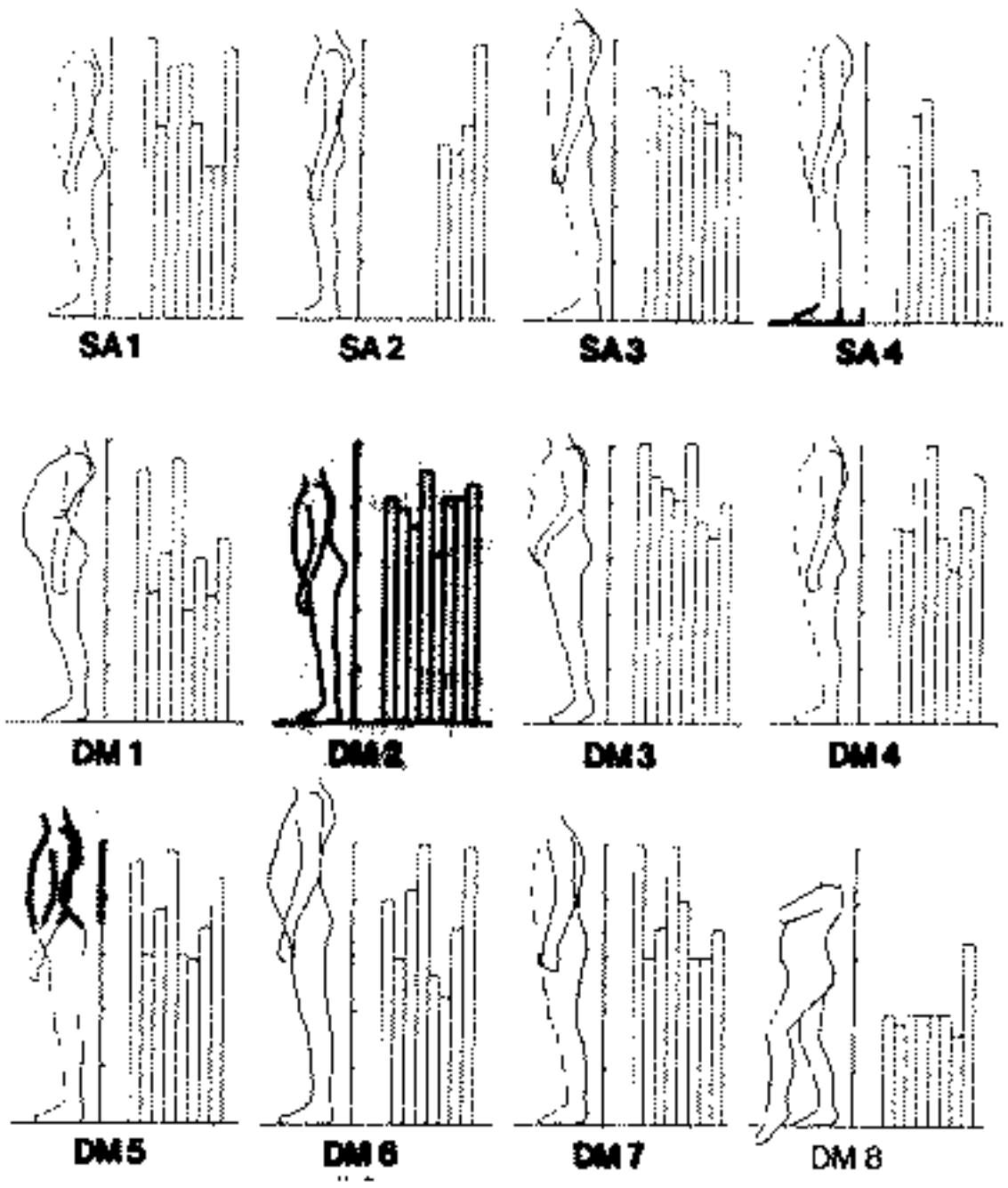


Fig. 1.
Profile drawings of the patients.

Controls

Matched controls have been selected from two groups of in all 253 subjects previously collected (9,10), and covering an age range from 3-65 years. In ten of the control subjects electromyographic gait recording was added to the recording of the switches.

Method

The gait recordings were performed both in Ljubljana and in Linköping and taped for later processing in Linköping. The procedure has been described elsewhere (9), but a short summary will be given.

The patients were requested to walk at five different velocities on a walkway consisting of a 10 m metal net, covering an ordinary carpet. The velocities corresponded to what he or she considered to be very slow, slow, ordinary, fast, and very fast. Four trials were made at each of the requested velocities.

The footswitches consist of electrically conductive tape placed on each foot under the heel and the methatarsophalangeal joint. They are called the heel- and ballswitches respectively. The patients wore thin socks with rubber soles on which the tape was placed.

Surface EMG was simultaneously recorded from six muscle groups on one side, (tib ant (TA), triceps sur (TS), rectus fem (RF), biceps fem (BF), gluteus med (GM), paraspinal (PS). We used silver-silverchloride electrodes (Beckman), and the myoelectric signals were amplified by a miniaturised preamplifier placed on the skin near the electrodes.

All signals were recorded by an analogue tape recorder (Racal Thermionic Store 14). Simultaneously or afterwards the footswitch signals were processed by a program in a computer (PDP 11/34). The EMG signals were integrated, rectified, AD-converted, and sampled. The signals were then adjusted for correct amplification and displayed on a graphic terminal display. All data were stored on disk for later work-up.

Relations between phases

With the aid of footswitches the stride (S) can be divided in several phases, (Fig. 2); stance (ST); swing (SW); and double support (DS). Stance may be divided in the time for heel on to ball on (HOBO), the time for foot flat (FF), and the time for heel up to ball up (HUBU).

A more complete description of the phases is found in another paper at this symposium (see Larsson, Odenrick and Sandlund, Clinical use of gait analysis with footswitches).

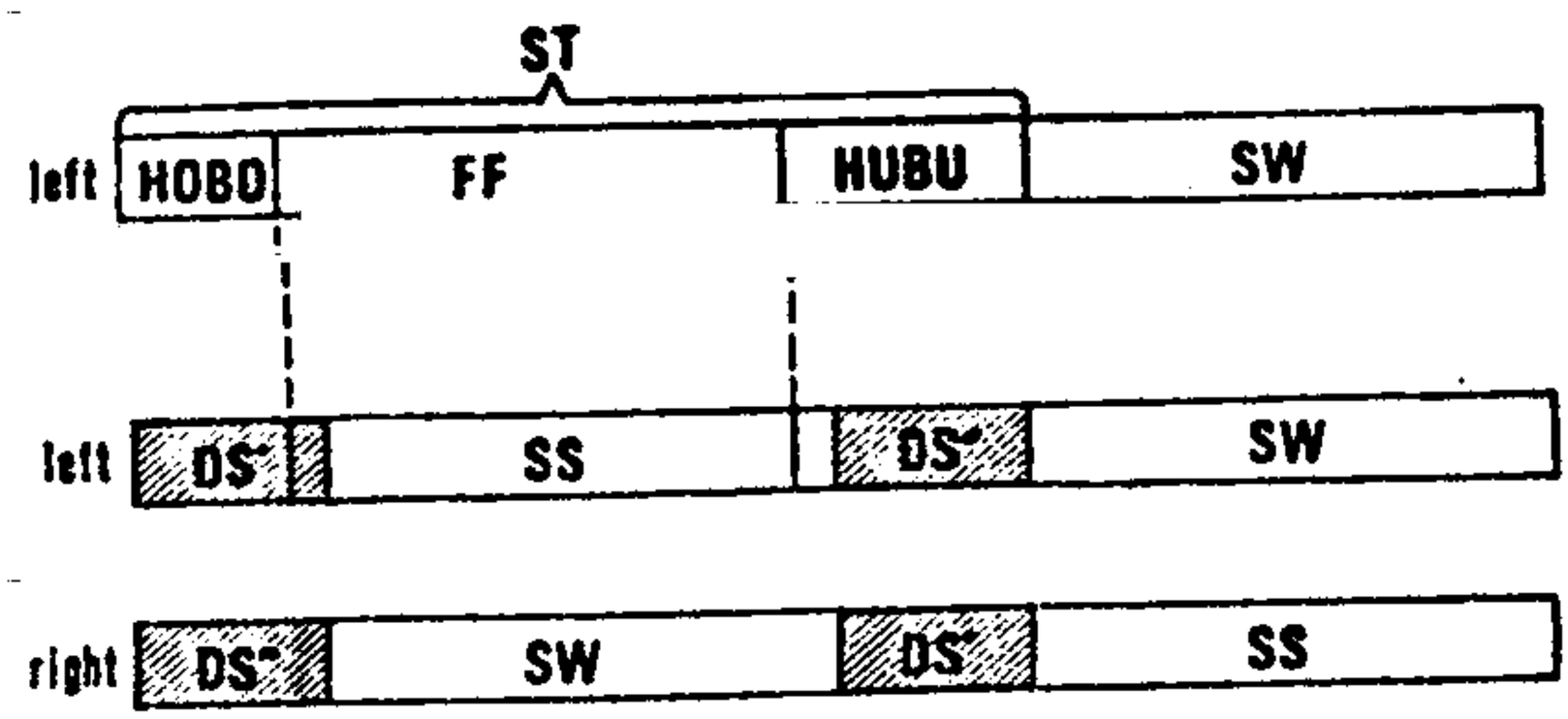


Fig. 2.

The phases of the stride as defined by the status of the footswitches.
Stance (ST), Swing (SW), Double support (DS' + DS" = DS)
Heel on to ball on (HOBO)
Foot flat (FF)
Heel up to ball up (HUBU)

RESULTS

Clinical observations

Patients with progressive spinal atrophy

The spinal atrophy-patients walked with a decreased velocity, their strides were comparatively short and the stride frequency accordingly somewhat increased in relation to velocity. The trunk generally did not move much, in one of the patients (SA2) it moved moderately to the same side as the stance.

They had a slight kyfosis and a slight lordosis which tended to increase when they walked. The pelvic movements were not increased except in SA2, where they were increased in all planes.

They did not walk on a broad base. They tended to have a valgus position in the knee and ankle joints. Foot on was flat.

Patients with muscular dystrophy

The walking pattern of the muscular dystrophy-patients was less homogenous than that of the patients with spinal atrophy.

In two patients, DM4 and DM7, the gait disturbance observed was very slight, except for a slight increase in lumbar lordosis when walking (DM4), and a slight increase of pelvic movements especially in the sagittal plane (DM7).

Three patients, (DM1, DM5, DM8), had a very obvious lordosis and they lurched the body to the same side as the stance phase. The pelvic movements were increased in frontal, sagittal, and transversal planes. Patient DM6 had the same disturbances, but less so. Three (DM1, DM5, and DM6) threw the lower leg forward from the knee in a pendulous way and foot on was flat in a slightly supinated position. In DM1 the toes struck before the heel, and DM8 walked on his toes. Both of them walked on a broad base.

The arms tended to hang passively behind the body also when they tried to walk fast, except in DM8 who held them out from the body with balancing movements.

Two patients, DM3 and DM2, mother and daughter, walked differently. They were leaning slightly forward and used comparatively short, fast strides. The trunk did not move so much, the lordosis was slight if any, and the pelvic movements were not increased. Foot on was flat, sometimes with the toes before the heel. The arms seemed to move ordinarily at the shoulders.

Footswitch recording

Velocity

In comparison with normal subjects velocity was low in all the ranges requested (Table II).

Table II.

Requested velocity	Velocity [m/s] Patients n = 12	Controls n = 170
Very slow	0.35±0.11	0.54±0.13
Ordinary	0.86±0.32	1.23±0.21
Very fast	1.33±0.29	2.34±0.44

Relation between stride frequency and stride length

In some of the subjects the stride frequency was high in relation to stride length, in some the relation was normal. In no case was the stride length longer in relation to stride frequency than in normal cases.

A diagrammatic representation of the results in the patient with the Becker myopathy (DM7) is shown in fig. 3.

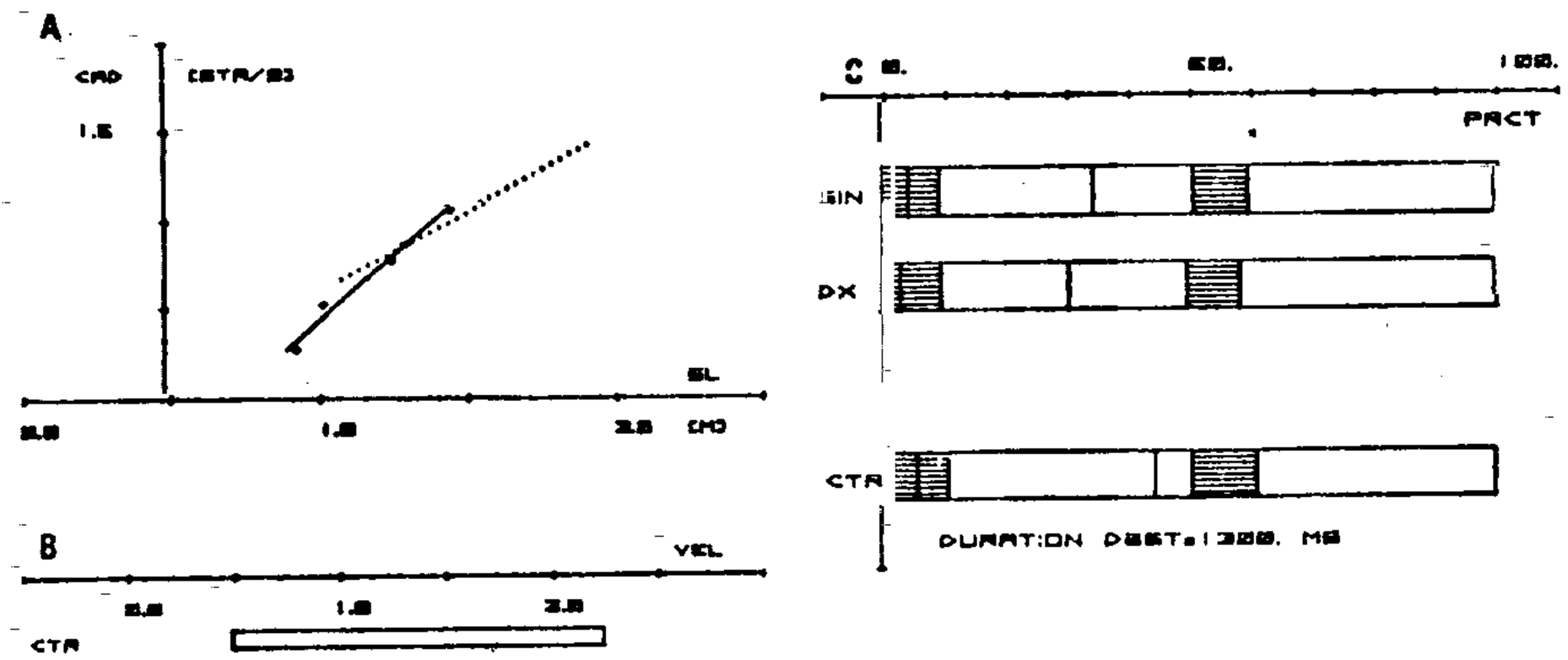


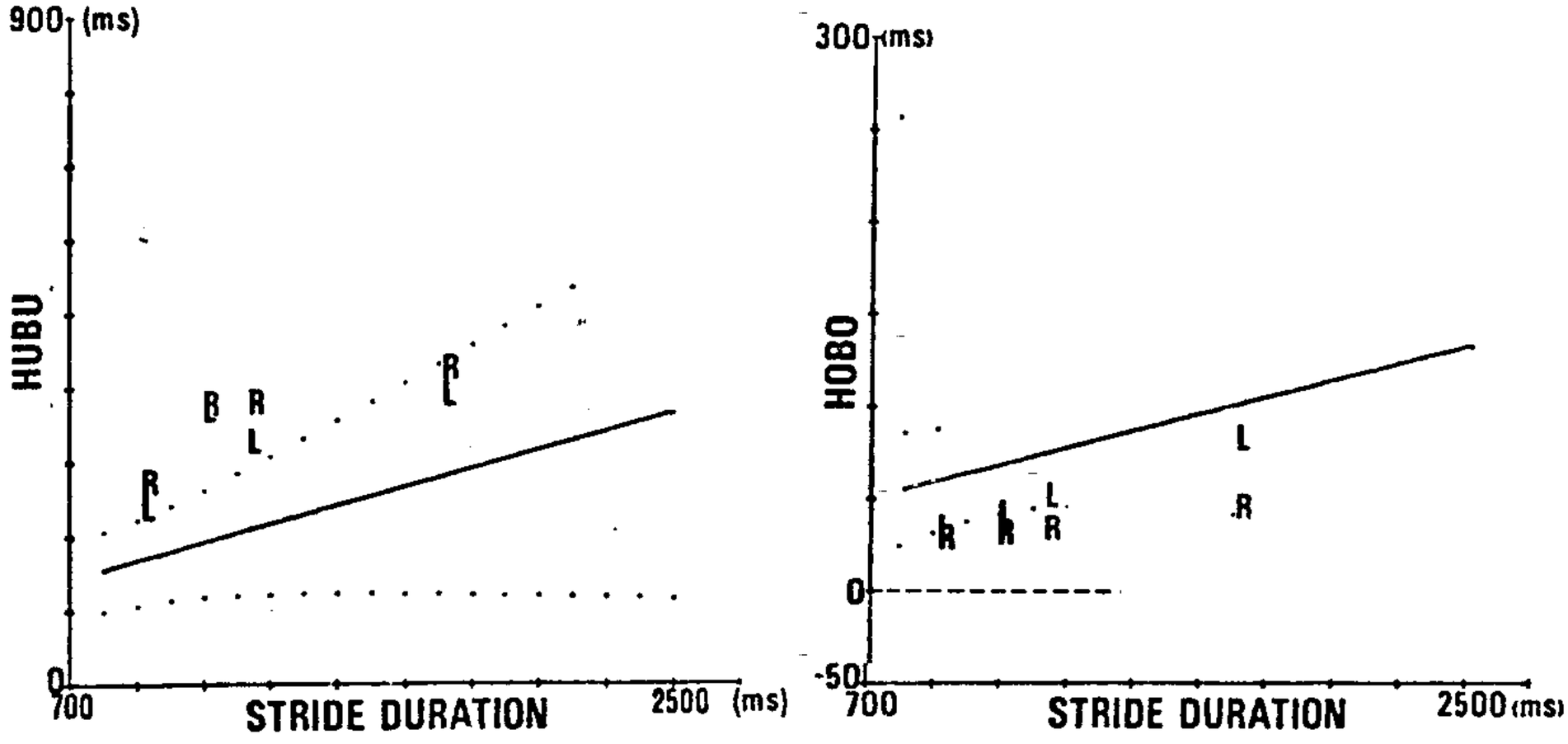
Fig. 3.

Gait diagrams from patient DM7.
A. Relation between stride frequency (CAD) and stride length (SL).
B. Velocities.
C. Phase diagrams. Hatched parts correspond to double support.

See also fig. 2.

Clinically his gait disturbance was slight, mostly consisting of slightly increased pelvic movements. The velocity was low (3B), the maximum not exceeding 1.5 m/s. The relation between stride frequency and stride length is normal (3A).

The phase diagram (3C) shows that stance and swing correspond to the control values. Double support (hatched parts) is normal. The most obvious difference to the control diagram is the prolonged duration of heel up to ball up (HUBU), which is also demonstrated in fig. 4A. The duration of heel on to ball on tends to be short (see also fig. 4B).



A B

Fig. 4. Patient DM7.

A. Heel up to ball up (HUBU), and B. Heel on to ball on (HOBO) at different duration of the stride. Mean values and 95% prediction intervals as calculated from matched controls are indicated.

The patient above was chosen for illustration because of his slight gait disturbance. In spite of that the duration of heel up to ball up was prolonged, a trait which was observed in all the myopathic patients.

A prolonged duration of HUBU was also seen in the spinal atrophy patients. In these cases it was, however, primarily seen only in high velocities as illustrated by the patient SA2 (fig. 5). In addition this patient demonstrates a shortening of HOBO on the left side (fig. 5C, and fig. 6A and 6B). More or less clear asymmetries were in fact common in both groups of patients.

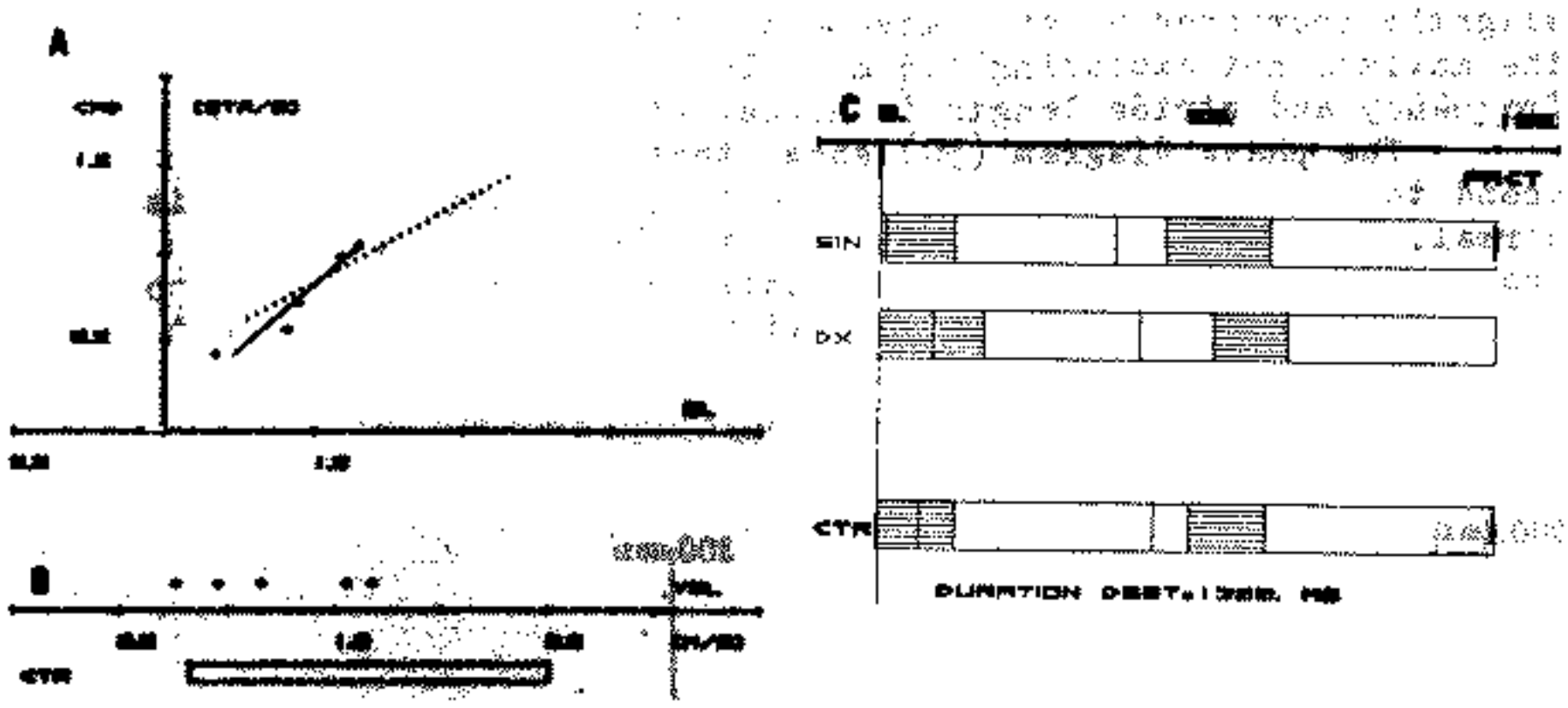
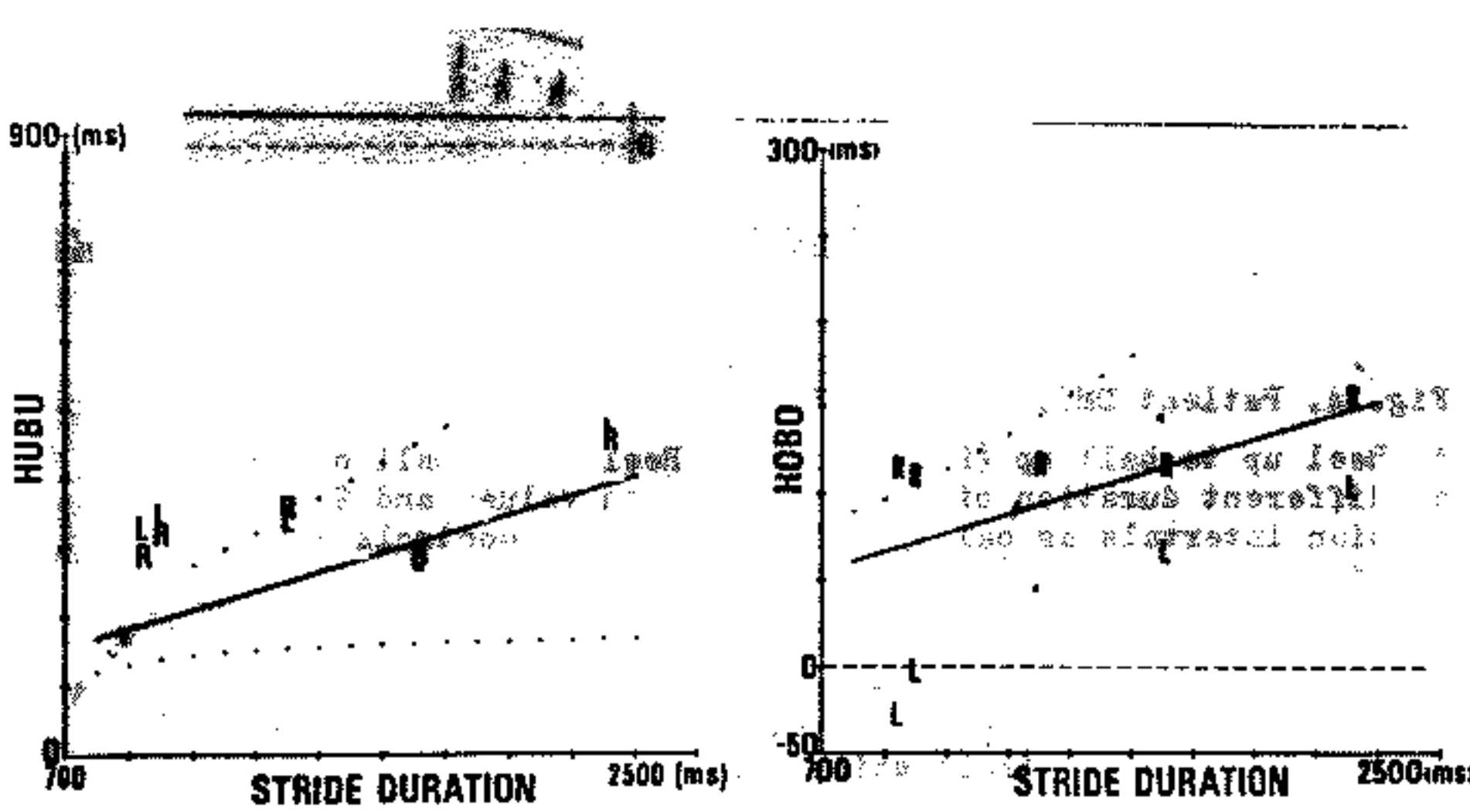


Fig. 5.

Gait diagrams from patient SA2, A, B, and C as in fig. 3.



A. B.

Fig. 6.

Patient SA2. A and B as in fig. 4.

Electromyography

The EMG:s from the patients were compared to a group of ten controls of different age and sex. When duration and amplitude were scored in relation to age and velocity in the control group it was found that duration increased with lower age and lower velocity, while amplitude increased with lower age and higher velocity. These factors were therefore accounted for in the scoring.

The patient scores then showed a clear tendency towards increased duration of the EMG in relation to the controls. The increase was unrelated to diagnosis. (Fig. 7).

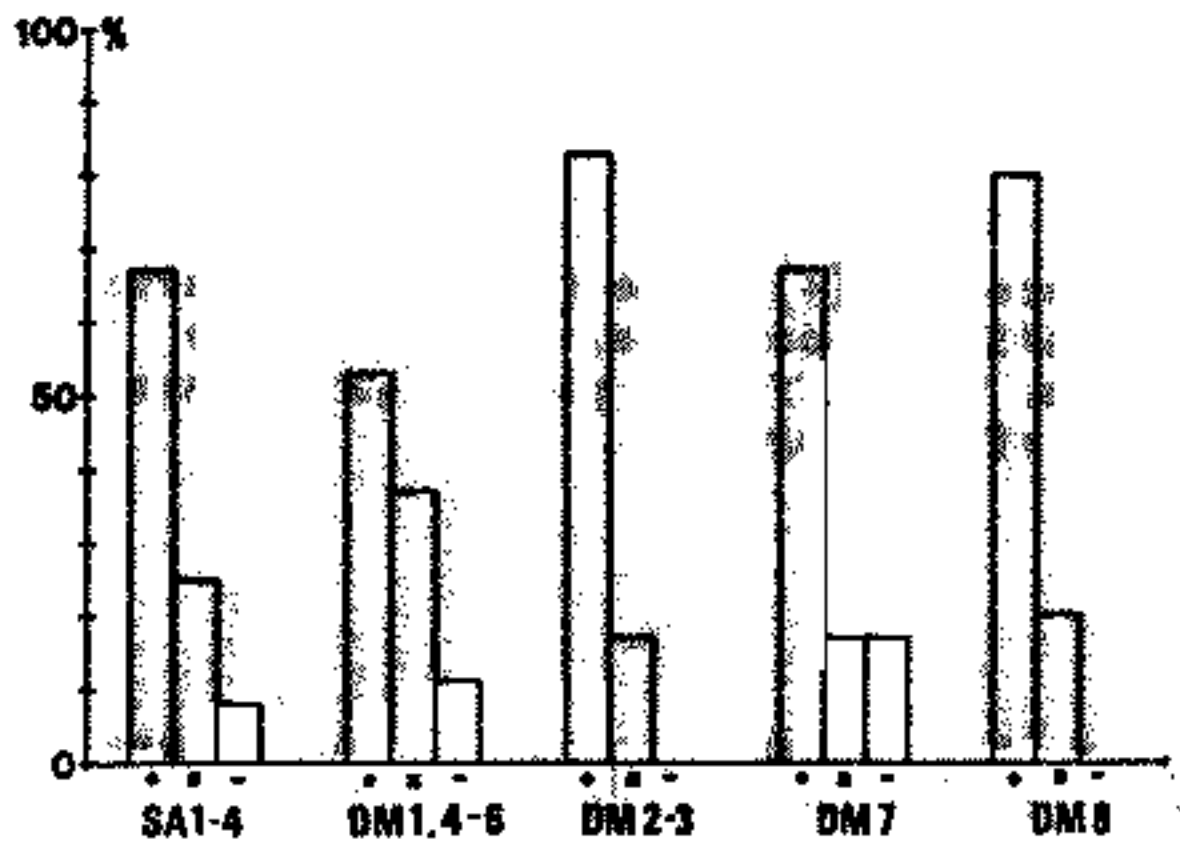


Fig. 7.
The relative number of muscles with increased (+), same (=), and decreased (-) duration of the EMG in relation to controls. For diagnostic symbols see fig. 1 and table I.

When the scores were grouped in respect of muscle, it is seen that the increased duration did not include triceps surae (TS), (fig. 8).

There were no certain trends in the scores for amplitude.

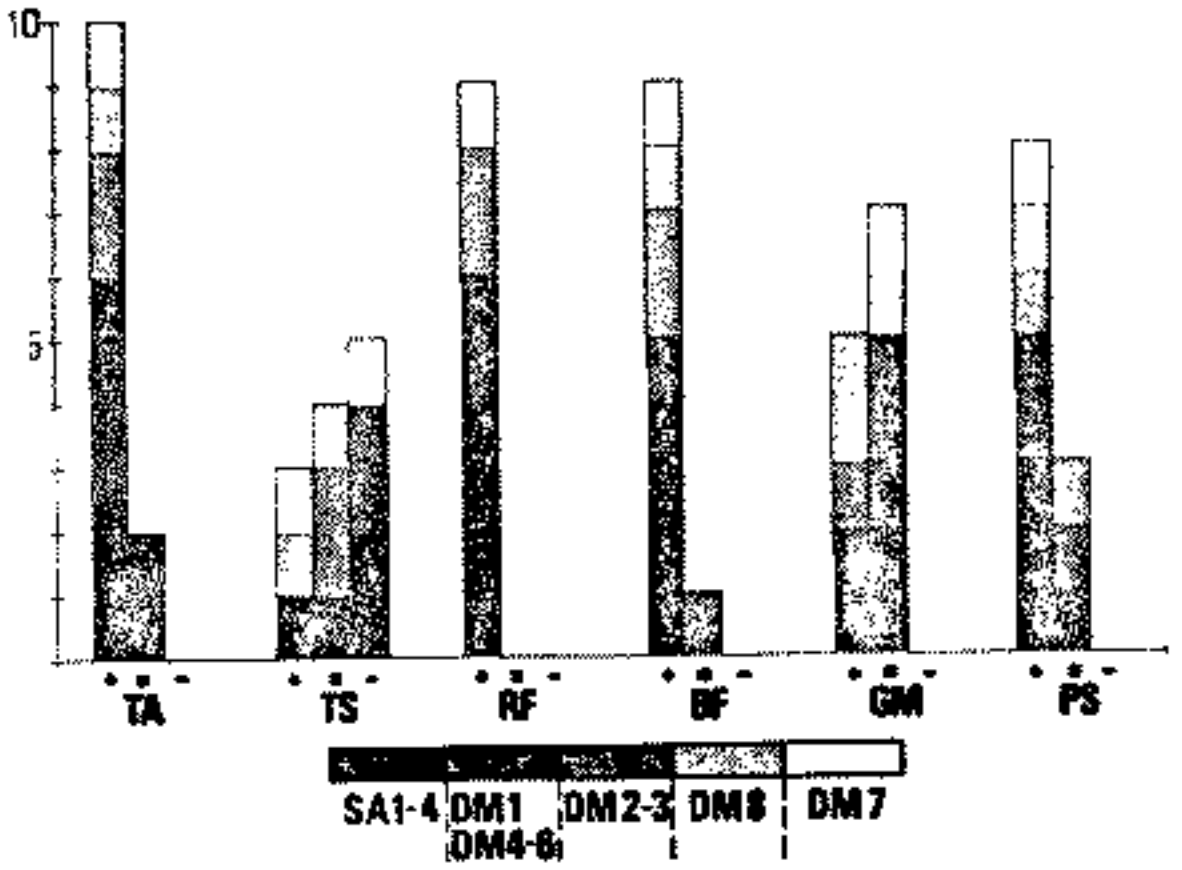


Fig. 8.

The number of patients with increased (+), same (=), and decreased (-) duration of the EMG in relation to controls. The scores are grouped with respect to muscles. The different patient groups are indicated by different degrees of shading.

DISCUSSION

The alterations in gait observed in neuromuscular disorders are to be explained 1) by the primary defects in the peripheral nerves and muscles, and 2) by its compensation as effected by a normal CNS (11). The assignment of a gait alteration to one or other of these two mechanisms may result in different therapeutic approaches and is therefore of practical interest.

The shortened duration of heel on to ball on as observed in many patients is for instance probably mostly explained by a peripheral paresis in the dorsiflectors of the foot, which is an early defect both in the dystrophic and in the spinal atrophy patients.

The prolonged duration of the EMG signal observed in most of the muscles may on the other hand be explained as compensation to a peripheral paresis. Increasing the duration of the contraction may be the only way to act when the muscle is too weak for its ordinary function.

The prolonged duration of heel up to ball up, an expression of a tendency to walk on the toes, may have different explanation in different patients. The patients DM2 and DM3, daughter and mother, leaned forward when walking. Normal subjects who are requested to lean slightly forward when walking will have an obvious increase of heel up to ball up in this position (unpublished observations). The increase in these cases seems to be a passive effect of the change in position. Normal subjects - and both these patients - compensated for their impaired balance with shorter and faster

strides. In patients who are not leaning forward when walking this explanation cannot be used. In these cases we suggest that the increased duration of heel up to ball up expresses a compensatory lengthening of the leg by extension of the ankle, and in this way a lengthening of the stride. The primary defect in this case could be a paretic inability to lengthen the stride by extension in the hip (5).

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