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Simulation of EMG in pathological situations

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Abstract

Objective: A mathematical model for simulation of the EMG from a muscle with its motor units is used. The study aims at correlating EMG findings (single-fiber EMG and concentric-needle EMG) with various induced morphological changes.

Methods: Reinnervation has been simulated by removing motor units randomly followed by a complete reinnervation from adjacent surviving motor units. Fibre type grouping and grouped atrophy can be seen. Myopathy is simulated by increased fibre diameter variation, loss of fibres and muscle fibre splitting.

Results and conclusion: The simulation gives quantitative aspects of the importance of each of these factors. It indicates the relative sensitivity of various EMG parameters. The model can be used both for education and for research.

Keywords: Simulation; Nerve-muscle disease; Electromyography; Motor unit, Concentric needle EMG

1. Introduction

It is difficult to establish the relationship between various kinds of pathological changes of the motor unit and the shape of the EMG signal. This is due to lack of practical methods to identify one motor unit in a muscle biopsy. It is also very time-consuming to collect data for the study of the influence of one selected morphological factor on the EMG signal because of the co-variation of parameters in pathology. Mathematical models may give us unique possibilities to study in detail the influence on the EMG signal of individual morphological and physiological parameters that are known to change in neurogenic and myopathic disorders.

The aim of this study was to analyze the effect on the motor unit potential (MUP) of some typical changes in a muscle that are known to occur in nerve-muscle disorders.

2. Methods

2.1. Model for EMG recording

The single muscle fibre action potentials are simulated with a line source model described earlier (Nandedkar and Stålberg, 1983). The obtained EMG signals are generated by the summation of single-fibre action potentials with respect to the recording electrode that has been chosen for the simulation. Volume conduction characteristics and effect of the reference electrode are taken into consideration (Nandedkar et al., 1988a).

The model as well as the various parameters discussed in this study have been described in detail previously (Stålberg and Karlsson, 2001)

A number of individual parameters can be varied in the muscle in order to allow the analysis of the effect on the MUP of these discrete parameters. Two types of EMG recordings were simulated, single-fibre EMG (SFEMG) and concentric-needle EMG (CNEMG). In the simulations of SFEMG recordings, the fibre density (FD) was measured as described for FD measurements in live recordings. The electrode was placed close to one muscle fibre and the number of fibres belonging to the same motor unit within 300 μm radius of the recording hemisphere were calculated. An average of 20 recording sites was calculated and called FD. In CNEMG simulation the motor unit potentials (MUPs) from active motor units were calculated. These signals were exported to commercial EMG equipment (Keypoint, Medtronic) for analysis according to the principles used in EMG studies performed in the laboratory. The following parameters were analyzed: amplitude, duration, area, number of phases and turns, size index (area/amplitude, normalized for amplitude) according to definitions given elsewhere (Stålberg et al., 1996).

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Table 1
Characteristics of standard muscle in the model

No. of motor units in muscle	489
No. of fibres in motor unit	175 ± 125
Density of fibres (/mm^2)	5 ± 1
Fibre diameter (μm)	50 ± 5
End-plate position (mm from muscle centre)	0 ± 1
Jitter (μs)	20 ± 10

2.2. Model for denervation-reinnervation

In order to investigate the neurogenic EMG changes in a more realistic model than by just increasing the number of muscle fibres randomly in a motor unit, a simulation of reinnervation was performed with certain assumptions about reinnervation anatomy and efficacy. When a motor unit is denervated the nerve terminals belonging to the most adjacent muscle fibres will reinnervate its fibres. Animal studies (Kugelberg et al., 1970) and studies in human reinnervated muscle (Stålberg and Dioszeghy, 1991) indicate that the surviving nerve terminals only reinnervate muscle fibres within their own fascicle. This will ultimately limit the reinnervation. When only one motor unit is represented in a given fascicle, denervation of that motor unit will not be compensated in this area but the fibre will remain denervated and undergo atrophy.

In the model of denervation-reinnervation we have therefore made the following assumptions.

For the individual motor unit, the default values stated in Table 1 have been used. The muscle model contains type 1 and type 2 fibres, but no subtypes. The fibres within the motor units are randomly distributed (Edström and Kugelberg, 1968). The motor units are randomly distributed in the muscle cross-section. When muscle fibres become atrophic after definite denervation, no compression of other fibres is simulated.

2.2.1. Denervation process

There is a random loss of neurones, without respect to their size. The number of neurones is decreased one by one starting with 0% neurone loss, ending with 100% loss.

2.2.2. Reinnervation process

A denervated muscle fibre is only reinnervated by one nerve fibre innervating muscle fibres less than 50 μm away.

The reinnervation only occurs within a given fascicle.

The reinnervation is taken randomly from the most adjacent nerve fibres without relation to the size of the motor unit.

The reinnervation is complete, i.e. all muscle fibres that fulfil the criteria for reinnervation will be.

No muscle fibre atrophy occurring during the time a muscle fibre stays denervated, is simulated.

Jitter will increase to 90 ± 20 μs immediately after reinnervation and then go back to 50 + 20 μs after some time.

The model displays all muscle fibres for an individual motor unit with the same colours, but different for different motor units. It also shows the two fibre types with different colours (Fig. 1) similar to pictures obtained in ATPase staining of a muscle biopsy.

2.3. Model for myopathy

The finding of increased fibre diameter variation, often with abnormally small and large muscle fibres, is one of the early signs of myopathy (Dubowitz and Brooke, 1973)

This was the first step of simulation. Another typical change is loss of muscle fibres, a feature which was added to the previous. Finally, the effect of longitudinal muscle fibre splitting, a common feature in myopathies, was added. Other typical morphological changes such as fibrosis, perifasicular atrophy or focal muscle-fibre necrosis were not simulated.

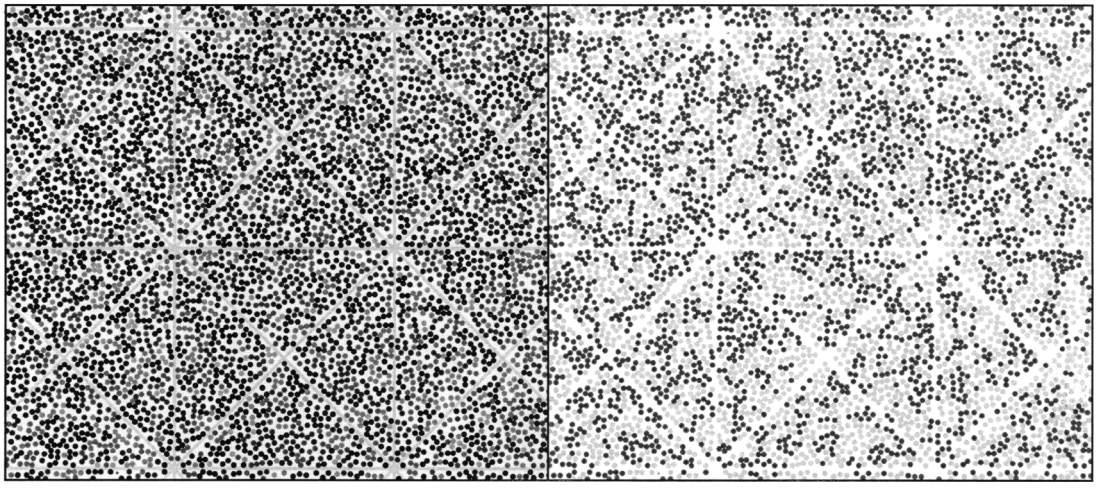

Fig. 1. Two types of display in the simulation of the muscle fibres and motor units in reinnervation. (A) Individual motor units, where all fibres in a given motor unit have the same colour (grey scale). (B) Fibre type 1 and 2 and simulating ATPase staining.

The following situations were simulated.

1. Increased number of fibres: general homogenous increase corresponding to large normal motor units
2. Increased number of fibres arranged in groups
3. Reinnervation after loss or neurones
4. Change in fibre diameter variation corresponding to general hypertrophy and atrophy
5. Decreased number of fibres and increased fibre diameter variation corresponding to myopathy

3. Results

3.1. Effect of number of muscle fibres in a motor unit

Motor units contain a different number of muscle fibres. In the normal situation the distribution is random, in pathology the fibres are usually scattered in a non-homogeneous fashion. In a previous study (Stålberg and Karlsson, 2001) two situations occurring in the normal muscle were studied. Firstly, a varying number of muscle fibres with constant density (same number of fibres/mm^2), i.e. corresponding to different territory size, and secondly, a varying number of randomly distributed muscle fibres within a given territory, i.e. different fibre density. In the first case, there was no systematic relationship between any of the parameters and territory size, except for the duration parameter, which increased when the motor unit size was increased from 50 up to 200 fibres, e.g. for motor unit diameter less than 7 mm. In the second situation the following parameters increased: amplitude (linearly), duration (between 50 and 250 fibres), area (linearly), size index (fastest in the range of 50–250 fibres/mm^2) (Fig. 2). The other parameters did not show any direct relationship to fibre density.

3.2. Effect on MUP parameters of fibre type grouping due to denervation-reinnervation

In this study we increased the number of muscle fibres in the motor units in a way that simulates the situation seen in reinnervation with so-called collateral sprouting. This gives increased number of muscle fibres organized in groups within a constant or slightly increased territory. It may increase somewhat since a reinnervating motor unit extends to the borders of the fascicles where it was originally represented. The question is whether a given EMG parameter is different for random and grouped distribution, respectively,

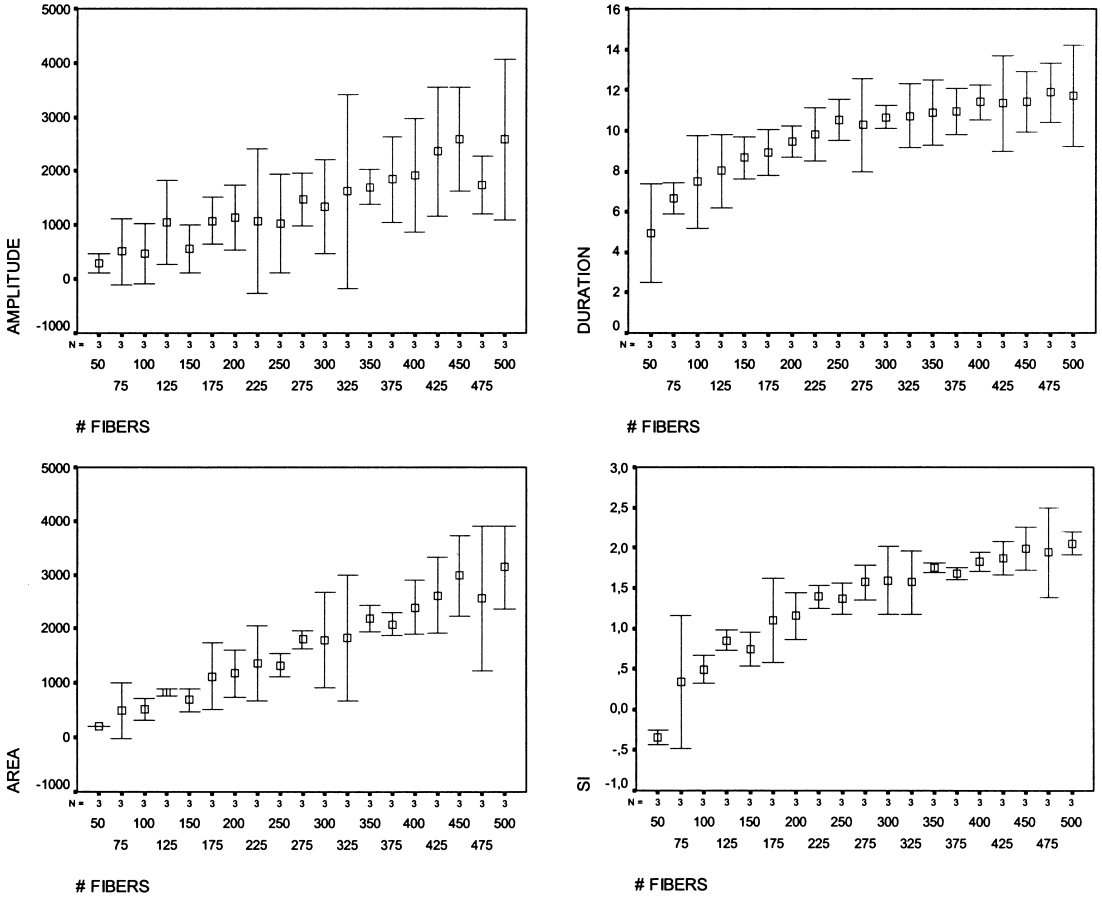

Fig. 2. from a previous simulation (Stålberg and Karlsson, 2001,with permission). Mean values with 95% confidence interval for amplitude, duration, area and size index for motor units with different density of muscle fibres, i.e. number of muscle fibres within a given territory (6 mm diameter in this case).

when comparing the same total number of muscle fibres in the motor unit.

The normal muscle in the model contains 489 motor units.

In this study we have iterated the denervation process and analyzed each level of 10% intervals.

A visual inspection was performed, similar to the analysis of a muscle biopsy (Fig. 3). The change in motor unit organization was seen visually in the ATPase staining when about 30–40% of the motor units is lost. The organization of muscle fibres of a given motor unit becomes very heterogeneous. Large grouping was seen with about 60–70% loss of neurones. When about 80–90% of the neurones were lost, grouped atrophy is seen in many areas of the muscle.

For each step of 10% loss of neurones, EMG was performed (CNEMG and SFEMG). The CNEMG was obtained 10–30 mm from the end-plate. Twenty to 25 motor units were collected for each situation with random electrode insertion, and a mean value of the parameters was calculated. Three complete CNEMG studies were performed for each degree of denervation. As can be seen in Table 2, MUP parameters show significant changes for different degree of axonal loss (Table 2 and Fig. 4).

Table 2
Degree of neuronal loss (in percent) necessary to give a significant change in parameters[a]

Amplitude	Rise	Duration	Area	Thickness	SI	Phases	Turns	FD
70	50	30	40	40	40	30[b]	30[b]	30

[a] Thickness (area/amplitude) SI, size index (area/amplitude, normalized for amplitude); FD, fibre density.
[b] Not significant for 90% loss of neurones.

In order to compare with earlier simulation with a random increase in number of muscle fibres (normal small and large motor units), the following information is necessary. In the

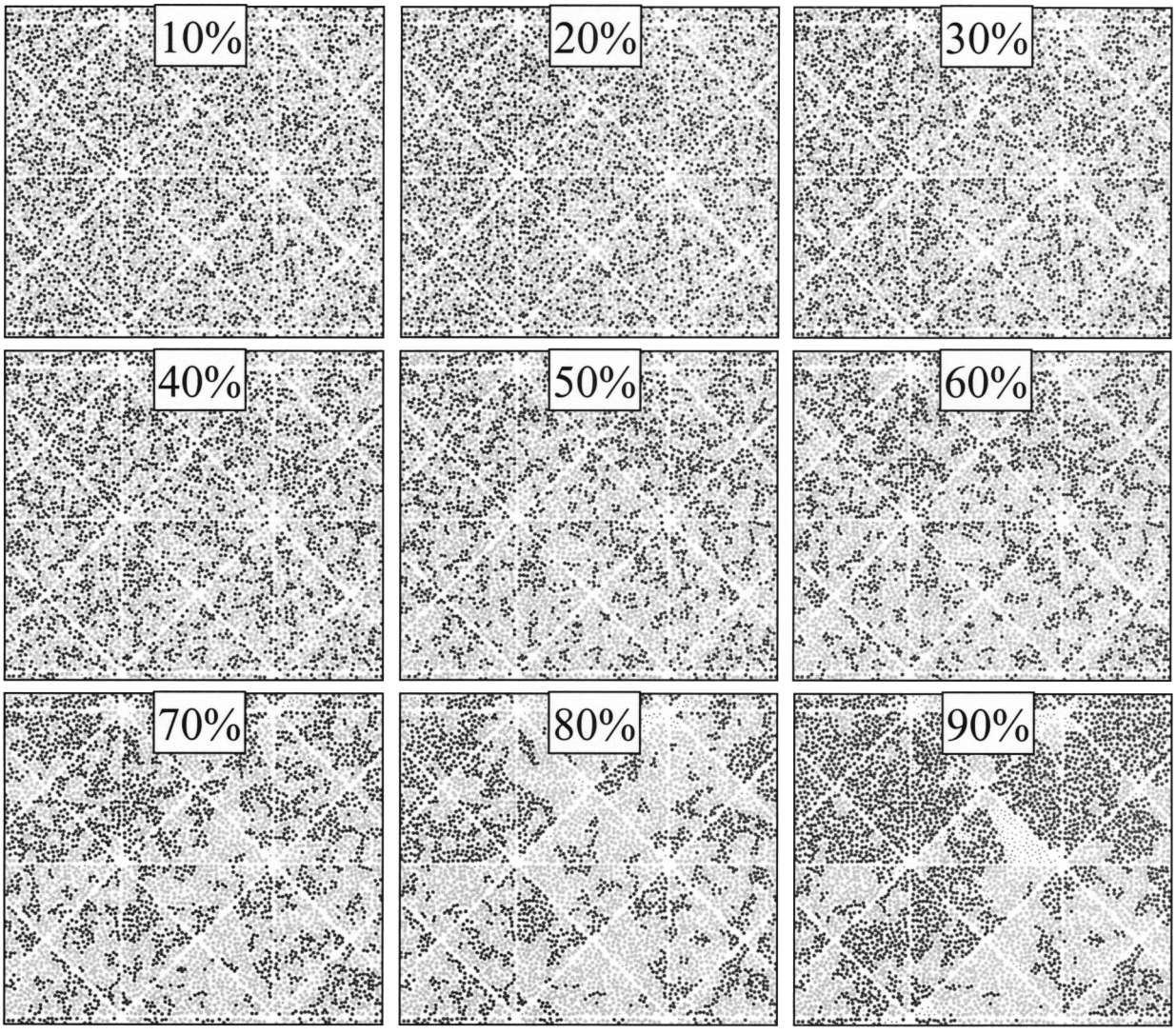

Fig. 3. Visual inspection of simulated muscles at different degree of axonal loss (10–90% loss). Note small grouping in APTase staining when about 40% of the motor units are lost. Grouped atrophy is clearly seen at 90% loss of motor units.

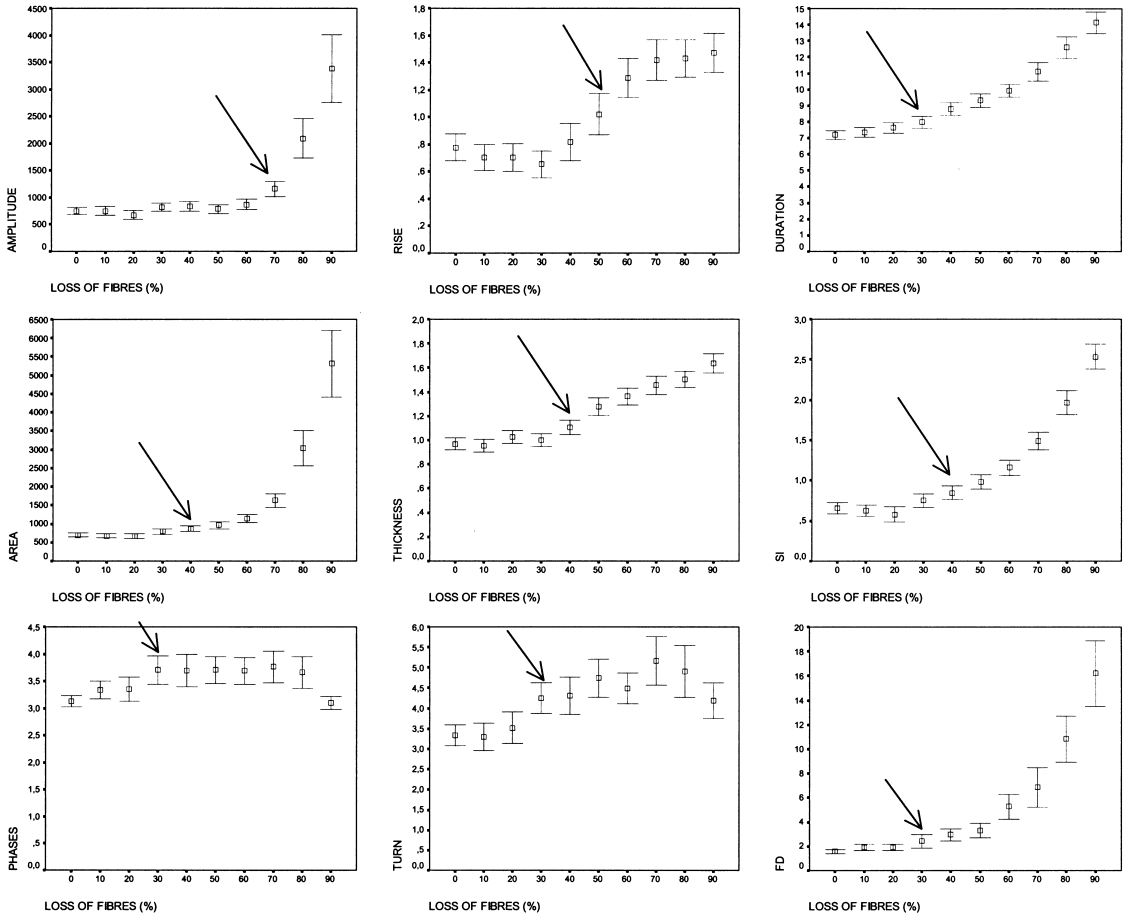

Fig. 4. Change in EMG parameters in relationship to loss of neurones. Arrows indicate significant changes ($P < 0.01$).

situation of 25% loss of motor units, the number of muscle fibres in each motor unit has increased by 33%. Similarly, 50% loss of motor units means an increase by 100% of fibres in surviving motor units and 75% loss means an increase by 300%. The change in EMG parameters obtained in motor units with a different number of fibres, either distributed randomly or grouped due to reinnervation, respectively, differs. As seen in Fig. 5, the effect of number of muscle fibres is more pronounced for random distribution, particularly for amplitude and area.

3.3. Effect of grouping on fibre density

The SFEMG fibre density, i.e. number of fibres within a hemisphere of 300 μm radius, was recorded in the same motor units as described above. Fibre density was recorded in 20 positions 3 times for each degree of abnormality. FD is the earliest EMG parameter to change in reinnervation with values becoming significantly abnormal with about 10% ($P < 0.05$) or 30% ($P < 0.01$) loss of axons (Fig. 4). The fibre density increases linearly from 1.8 to 15 in these motor units. For comparison, the FD was measured in motor units when the number of muscle fibres was increased randomly. Note that the FD increases somewhat faster in grouping, than in random distribution, comparing the same number of fibres/mm^2 (Fig. 6).

3.4. 'Myopathy' with increased fibre diameter variation

Four muscles were generated, each with 100 motor units. The 4 muscles were given different fibre diameter variation 50 ± 5, 50 ± 10, 50 ± 20 and 50 ± 30 μm to simulate the situation in myopathy. In these 4 muscles, 20–24 motor units were recorded 20 mm from the end-plate zone. As seen in Fig. 7, there were only minor changes in the amplitude and duration parameters but the MUPs became more complex with increasing number of phases and turns for higher diameter variation. The parameters became significantly abnormal ($P < 0.01$) as follows: turns 10 μm, phases at 20 μm and the others not significant at 30 μm.

3.5. 'Myopathy' with increased fibre diameter variation and loss of fibres

Five muscles were constructed, each with 150 motor units. Each motor unit had an increased fibre diameter variation 50 ± 20, compared with normal 50 ± 5. Within the same territory the number of muscle fibres was different for the 5 muscles, containing 5, 4, 3, 2, 1 fibres/mm^2,

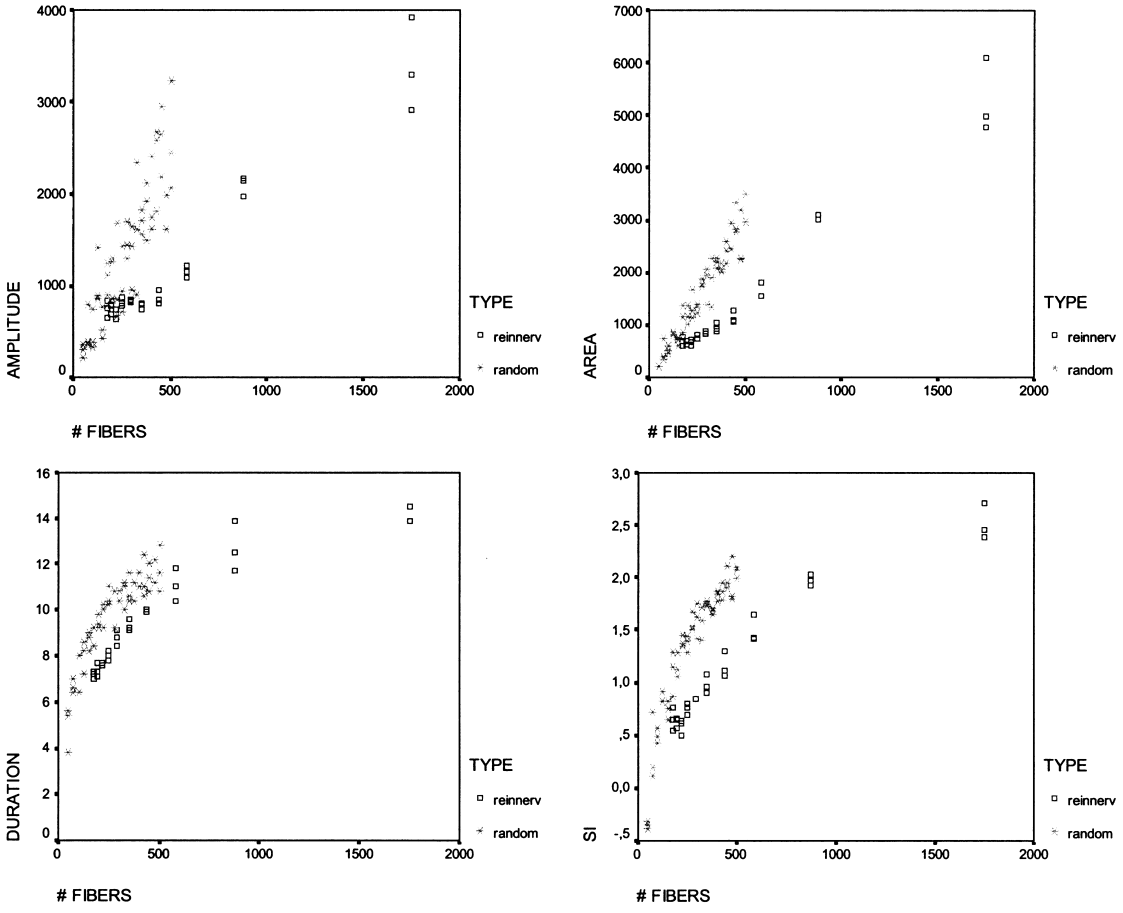

Fig. 5. Relationship between number of muscle fibres and EMG parameters for random distribution and grouped distribution (as in reinnervation). Note the more pronounced changes when the fibres are randomly distributed rather than grouped as in reinnervation.

respectively, where 5 fibres/mm^2 is normal. This was obtained by using motor units with 100 ± 10, 80 ± 10, 60 ± 10, 40 ± 10 and 20 ± 10 muscle fibres, respectively. For each such muscle, 5 insertions were made giving 20–25 MUPs for each muscle. The results showed an abrupt decrease in amplitude and area when the number of muscle fibres fell below 4 fibres/mm^2. Duration, thickness, size index, number of phases and turns showed a great scatter but the relationship between these parameters and number of fibres seemed to decrease linearly from 100 down to 20 fibres (Fig. 8). Statistically the change became significant ($P < 0.01$) as follows: area and size index at 80 fibres, duration at 60 fibres, thickness and turns at 40 fibres, phases at 20 fibres. Amplitude showed a drop when the number was decreased from 100 to 80, but due to the large scatter of data this value was not significant and did not continue with further loss of fibres.

3.6. 'Myopathy' with increased fibre diameter variation tested for muscle fibre splitting

One muscle was generated with increased fibre diameter variation, set to 50 ± 20. Initial MUP values were obtained

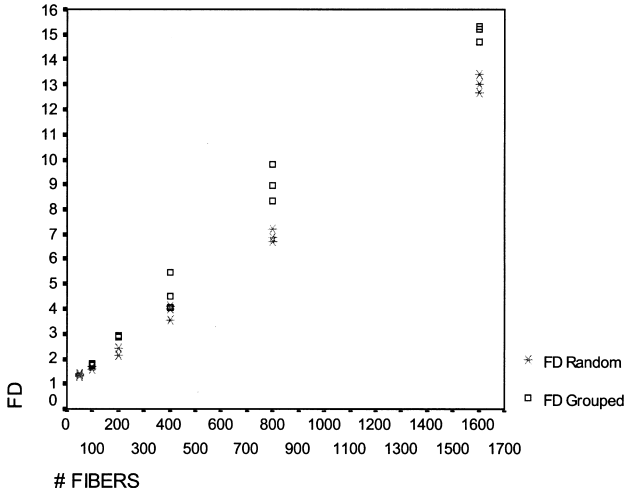

Fig. 6. Fibre density for different number of fibres. Two situations are compared; when the additional muscle fibres were neighbouring the fibres in the original normal motor unit and when the fibres were distributed randomly. The fibre density increases somewhat more in the case of grouping.

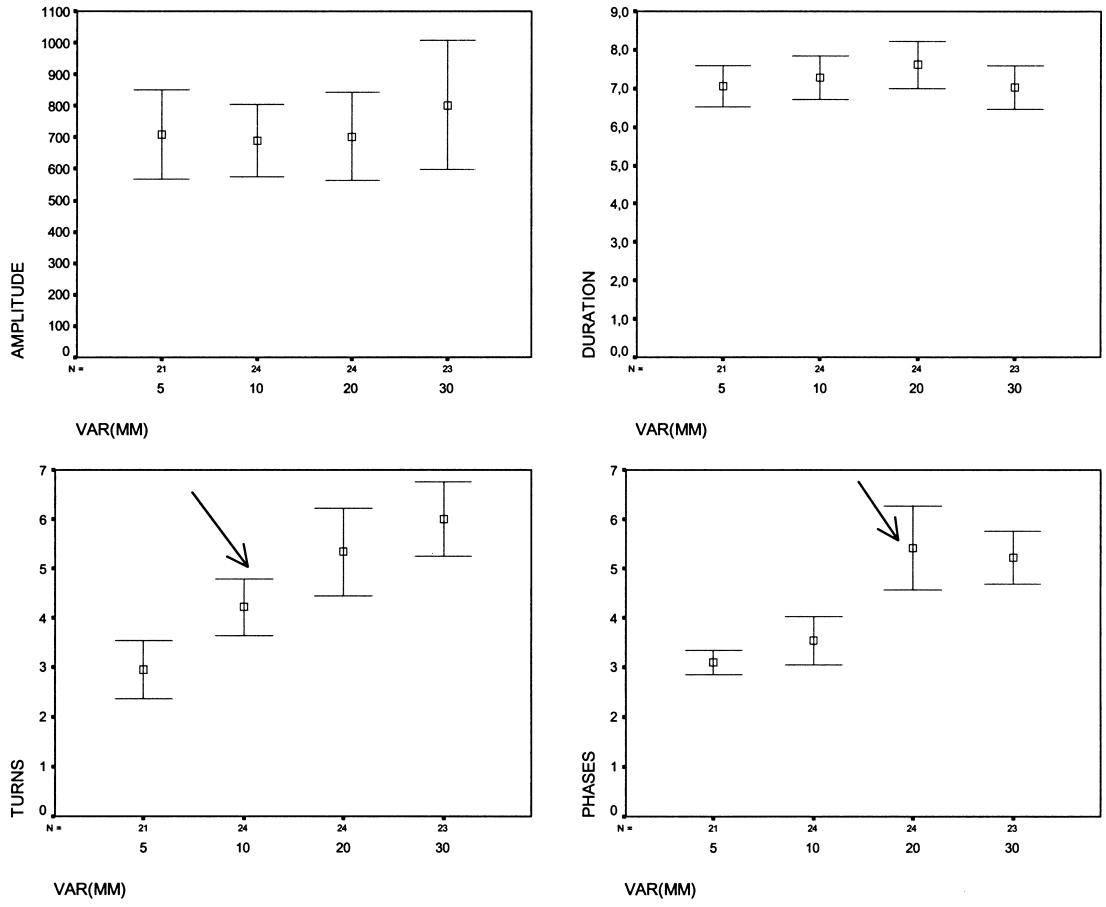

Fig. 7. Mean values with 95% confidence interval for amplitude, duration, phases and turns with different fibre diameter variation. Significant changes ($P < 0.01$) are indicated with arrows.

from 8 recording positions performed 20 mm from the end-plate zone giving 30 MUPs. In the second phase, the largest fibres, $>$ 60 μm in diameter, were split longitudinally into 4 fibres, each with a randomized diameter of 20–50 μm. Small groups were therefore formed (Fig. 9). Thirty MUPs were now recorded from identical positions as used for recordings in the first step.

The amplitude decreased somewhat and the duration increased but not significantly, Complexity for the MUP (turn, phases) increased a little. In general the changes caused by splitting were of moderate degree (Table 3 and Fig. 10).

4. Discussion

For the interpretation of the EMG signal used in daily routine, it is of great value to know the relationship between motor unit morphology and its generated signals. Since the introduction of EMG in the late 1940s, such relationships have been sought after. The literature reflects the great interest and importance of this knowledge and a number of publications are available (Buchthal et al., 1954; Nandedkar et al., 1988b; Stålberg et al., 1996; Kimura, 1989; Dumitru, 1995). The approach is usually to compare the EMG with muscle biopsy data, sometimes as group statistics, sometimes by comparing data from the same muscle (Buchthal and Kamieniecka, 1982; Warmolts and Engel, 1973; Barkhaus et al., 1990; Bertorini et al., 1994). However, it has not been possible to make a detailed correlation between morphological and neurophysiological parameters from a given motor unit. The co-variation between different types of pathology has made it impossible to extract the influence and importance of each individual factor. In this study we have used a model of a muscle including the electrical field of each active muscle fibre. The MUPs have been generated based on knowledge about individual muscle fibre action potentials, volume conduction, and electrode characteristics using data from the literature (Stålberg and Karlsson, 2001). This approach may be used for the general understanding of EMG, it may be used for education and it may create a number of questions and ideas worthwhile testing in real recordings.

The presented simulation has shown the effect on MUP of individual morphological parameters that typically change in nerve-muscle disorders. It has shown that the MUP parameters do not necessarily covariate, but reflect abnormalities in different ways, with different relationships to pathology. A given abnormality in one parameter may not

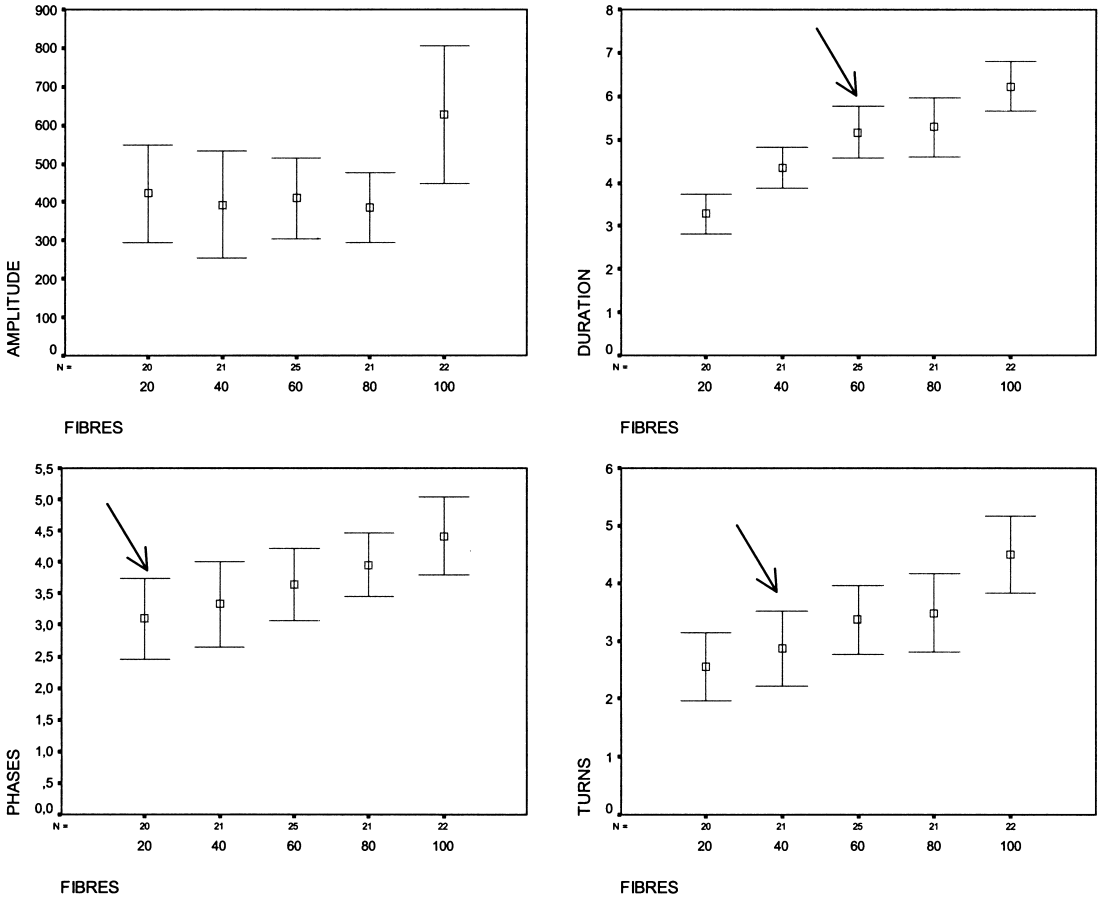

Fig. 8. Mean values with 95% confidence interval for all MUP parameters with different number of fibres. Significant change ($P < 0.01$) is indicated with arrows.

be correctly interpreted until information from other parameters is available.

It is certainly true that many of the findings reported here have been correctly predicted from live recording and empirical observation over the years. The quantitative relationship and dependency on individual morphological parameters has not been known.

The situation of reinnervation by means of collateral sprouting with increasing number of muscle fibres in groups was simulated. It is known (Kugelberg et al., 1970; Karpati and Engel, 1968) that in the progression of denervation-reinnervation the groups of fibres belonging to one motor unit increase in size, and form so-called small and, later, large fibre type grouping. When such a dense group of fibres finally becomes denervated, its central fibres are far from surviving reinnervating motor units and therefore cannot become reinnervated. In this situation the fibres become atrophic and are no longer activated by the axon. In the extreme case of a motor unit filling a fascicle, the entire fascicle becomes atrophic, grouped atrophy. This process should lead to decrease in the motor unit size towards the end of the process. Such fractionation has been discussed, e.g. in amyotrophic lateral sclerosis and late poliomyelitis.

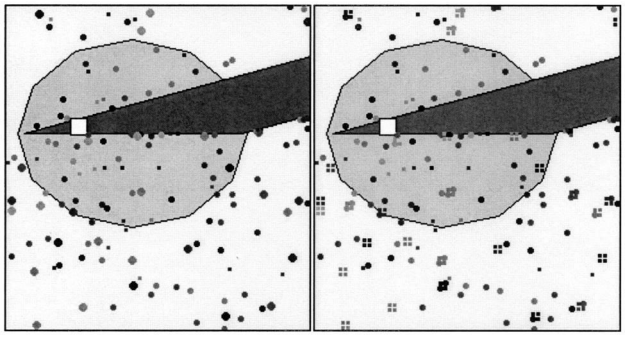

Fig. 9. The largest fibres, >60 μm, were split longitudinally into 4 fibres, each with a randomized diameter of 20–50 mm, and small groups were therefore formed.

The simulations showed the difficulty in revealing these changes by visual inspection. Not until 40% of the neurones were lost did small grouping occur. Also, EMG changes appeared relatively late. The changes occurred after 30% loss of neurones for FD, at 30% for duration and not until at 70% loss for amplitude changes. It should be noted that in real recordings, the effect of a changed mean diameter, seen in pathology, may be superimposed. In some slow processes of denervation-reinnervation, increased fibre diameter may be seen as a compensatory phenomenon. In others, fibre

Table 3
Mean ± 1SD for all parameters and 30 MUPs before and after split

	Amplitude	Rise	Duration	Area	Area/ampl.	SI	Phases	Turns
Before	549 ± 274	0.60 ± 0.49	7.28 ± 1.37	495 ± 193	1.00 ± 0.31	0.36 ± 0.34	4.73 ± 1.76	5.20 ± 1.86
After	481 ± 246	0.72 ± 0.57	8.64 ± 1.93	492 ± 187	1.12 ± 0.32	0.38 ± 0.37	5.20 ± 2.14	5.57 ± 2.14

atrophy may occur. The effect of fibre diameter is separately examined in a previous study (Stålberg and Karlsson, 2001). With increasing diameter the amplitude of the MUP increases, but the duration is relatively unchanged. Thus, in reinnervation with simultaneous muscle fibre hypertrophy the amplitude parameter may become abnormal at an earlier stage than indicated in the present study. As seen, other parameters such as percent polyphasic motor units was increased but were not related to the degree of denervation.

For comparison, dense motor units with randomly scattered muscle fibres but with the same number of fibres/mm^2 were simulated. In the generally more dense motor unit the motor units showed expected changes with increasing amplitude and duration. It was surprising for us to see that the parameter dependency on number of fibres was more pronounced for a random distribution of fibres than grouped, as in reinnervation, when comparing the same total number of fibres within a given territory. This may be explained on a statistical basis. When the generators are located in few but separated groups (giving a certain number of total fibres in the motor unit) it may be more likely to record from 'distant' generators more often than when the same number of fibres are evenly distributed over the area. This difference is seen mainly for the amplitude parameter and to a lesser extent for the duration. For SFEMG the situation is the opposite: changes are more pronounced for grouped distribution. Since the FD estimation in SFEMG is based on measuring number of adjacent muscle fibres (within 300 μm) by approaching the recording electrode close to one fibre, it will increase earlier in reinnervation than with random increase in muscle fibres. This is one of the reasons why FD in clinical routine has been shown to be useful as an early indicator of reinnervation (Henriksson and Stålberg, 1978).

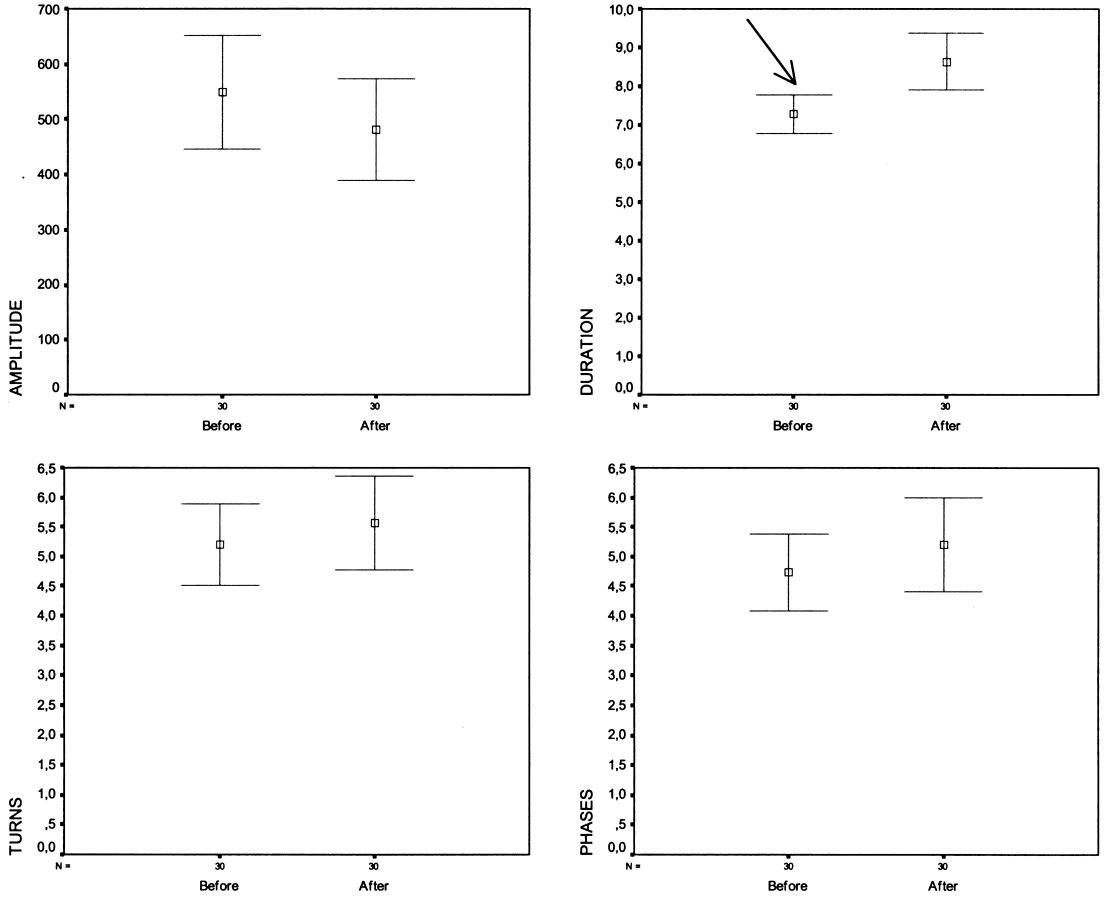

Fig. 10. Mean values with 95% confidence interval for amplitude, duration, phases and turns before and after muscle fibre splitting. Only duration is changed significantly.

The simulations of myopathy were made in a few steps. Only fibre diameter variation, an early sign of myopathy, caused changes in the shape of the MUP with increased number of phases and turns. This EMG abnormality has sometimes been considered to be a sign of loss of muscle fibres. The effect of fibre diameter on the shape is more pronounced the longer the distance from the end-plate the recording is performed (Stålberg and Karlsson, 2001). This factor can be used to advantage in routine EMG. Myopathic changes may be more pronounced in more distal parts of the muscle. In most simulations, the recordings are performed at a given distance from the end-plate zone, often 10–30 mm.

The next step included loss of muscle fibres with the same abnormal distribution of fibre diameters. The amplitude changed somewhat, but more so the area and duration. Turns and phases decreased with loss of muscle fibres, contrary to what has been considered to explain the increase in these parameters in myopathy.

The final step is the effect of muscle-fibre splitting. Since each muscle fibre was made thinner, it also generated action potentials of lower amplitudes for a given distance from the fibre. This was not compensated by the fact that the number of muscle fibres increased, probably due to the effect of phase cancellation, making summation inefficient.

Already this small number of presented combinations of pathology has given suggestions regarding the relationship between the EMG signal and its generators. It has reinforced the general concept in some aspects, but pointed to new relationships not easily obtained from other studies. We are aware of the fact that these studies only give a schematic picture of pathological muscle.

The model has certain shortcomings. The length of the fibres is not simulated, nor the amount of fibrosis that may be of importance in myopathies. Nevertheless, it seems that the model gives sufficient similarities with real recordings and support reasonably well earlier hypothetical explanations of EMG. We are convinced that the model can be used for further studies of various combinations of abnormalities. At present it has been used for teaching but it seems also to have potential use in research, with the mentioned shortcomings and other factors taken into consideration.

The obtained results will hopefully help us understand the underlying type of muscle pathology from assessment of the correct sets of MUP parameters, i.e. by using the relationship between morphology and the generated EMG signal in the opposite direction from what is presented in this study.

Acknowledgements

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