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MU firing characteristics in human dystrophic muscle

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Abstract. During isometric contractions of constant force surface EMG as well as intramuscular MU potentials from extensor digitorum communis and biceps brachii muscles were recorded on magnetic tape for further off-line analysis. Surface EMG power spectra were computed and transformed so as to reveal low-frequency peaks which might correspond to MU firing rates. From intramuscular recordings, single MU action potentials were identified with an aid of semi-automatic recognition program. For each single MU action potential train (MUAPT) statistical parameters of interspike intervals (ISIs) were determined and related to the measured muscle force level. Sixty four MUAPTs from 9 patients and 55 MUAPTs from 4 normals were analysed so far. The estimates of MU firing rate from surface and needle EMG corresponded well to each other. The MU firing rates were higher in muscular dystrophy and this difference was more pronounced for higher levels of muscle force. The tendency towards mean MU firing rate increase is stronger for the patients with more advanced disease.The typical dependency of standard deviation of ISIs on their mean value may be approximated by two lines of different slope. There were reported experimental data indicating that the breaking point of this dependency may be an estimate of AHP duration in motoneurones. Our results for dystrophic muscle showed a shift of this point towards shorter ISIs, as compared to normals. This suggests that in muscular dystrophy also motoneurones may be altered, either by the disease itself or as a compensation for changes in muscular part of a MU.

Key words: motor unit activity, muscular dystrophy

INTRODUCTION

The activity of motor units (MUs) and its relation to the force output of a skeletal muscle since long has attracted an attention of scientists and clinicians. Now it is well known that in normal muscle an increase in force is achieved by recruitment of more MUs and increase in firing rate of those already active. The relative contribution of both mechanisms is rather difficult to assess although it is generally agreed that during weak contractions the MU recruitment is more relevant whereas the contribution of firing rate modulation increases on the higher force levels.

In typical neurogenic diseases of muscle, the number of recruited additional MUs is reduced and the neuromuscular system compensates for this loss with increased MU firing rate. Enhanced firing rate correlates with high-grade paresis. In demyelinating neuropaties however, as was recently shown by Reiners et al. (1989), MU firing rate was decreased over whole range of voluntary contraction which corresponded well with increased percentage of type I fibres determined by histopathological investigation.

Much less obvious are the data on mechanism of recruitment in myopathic muscle, in particular in muscle dystrophy, since in myogenic diseases it is much more difficult to evaluate and follow up the order of recruitment, the number of active MUs and the possible increase of firing rate of individual motor units.

Some authors, as Dietz et al. 1975, Halonen 1981, Freund et al. 1983 described increased firing rate in dystrophic muscles, some others, as Daube 1986, Fuglsang-Frederikssen et al., 1987, Kimura 1989 reported the same range of firing rates as in normal muscle. This discrepancy between the results of various studies might depend on different conditions of testing and various severity of investigated cases.

We decided therefore to investigate MU activity in more detail to find out which characteristics of this activity may be changed in dystrophic muscles.

METHODS

Experiment

During isometric muscle contractions the muscle potentials were picked up, amplified by DISA electromyograph and stored on magnetic tape for off-line analysis. During each experimental session, two kinds of bipolar electrodes were used: (1) intramuscular fine wire electrodes, made from tungsten wire of 90 mm diameter, introduced into the muscle by means of disposable hypodermic needle and (2) surface electrodes of 12 mm diameter, separated by 22 mm.

The subject was comfortably seated in the armchair with the arm and forearm placed on the appropriate support. Two series of experiments with different groups of subjects were performed for the two muscles: extensor digitorum communis (EDC) and biceps brachii (BB).

The subject was asked to press a lever attached to tensometric strain gauges and maintain a constant tension for about 20 seconds. The output of force transducer was fed into the electromyograph display together with the reference level. The levels of tension investigated were 100%, 75%, 50%, 25%, 20%, 10% and a few levels below 10% of maximal voluntary contraction (MVC). The tension signal was recorded on the other channel of tape recorder.

Analysis of intramuscular recordings

The potentials were introduced into an IBM PC by an A/D converter "AMBEX" with the sampling rate 30 kHz. The potentials of individual MUs were semi-automatically identified with help of a specialized software. The following statistical parameters of single motor unit potential trains (MUAPTs) were computed: mean value of interspike interval (ISI) and mean firing rate; standard deviation of ISIs; skewness and curtosis of ISI distribution; serial correlation coefficient. The last four parameters were also computed after the subtraction of a moving average. Finally, the cross-correlation histo-

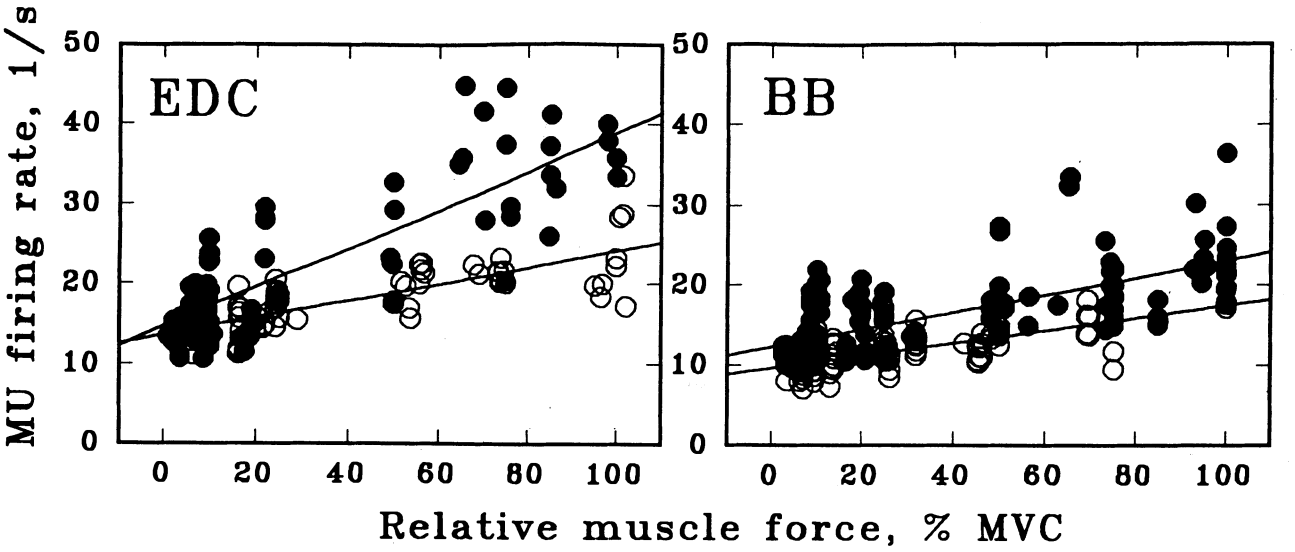

Fig. 1. Firing rates of individual MUs plotted vs relative muscle force level (left-hand plot - EDC, right-hand plot - BB). Open circles, normal MUs, filled circles, dystrophic MUs. For each set of data the regression line is shown.

grams of the pairs of single MUAPTs were constructed.

Analysis of surface recordings

The surface EMG was sampled with the rate 2 kHz. The power spectra were computed from stationary fragments of recordings. The spectra from several fragments were summed up to decrease the error of estimation. The spectra were thereafter subjected to special transformation aiming towards revealing peaks corresponding to mean MU firing rates.

RESULTS

The results are shown in Figures 1-4. We have concentrated only on those characteristics of MU firing which illustrated most clearly the difference between normal and dystrophic muscle.

In Figure 1 the firing rate of individual MUs is plotted vs relative muscle force level. It can be clearly seen that the average firing rate of dystrophic MUs is higher and its rise with the force level is steeper than that of normal MUs. These differences are more pronounced in EDL than BB. The average MU firing rate increases with the progress of the disease, as illustrates Fig. 2.

In Figure 3 the interdependence between standard deviation of ISIs (SDI) and their mean value (MI) is shown. The data for dystrophic MUs are also shifted towards shorter ISIs. Figure 4 presents a comparison between mean MU firing rate determined from the analysis of intramuscular (needle) recordings and that calculated from the power spectra of surface EMG.

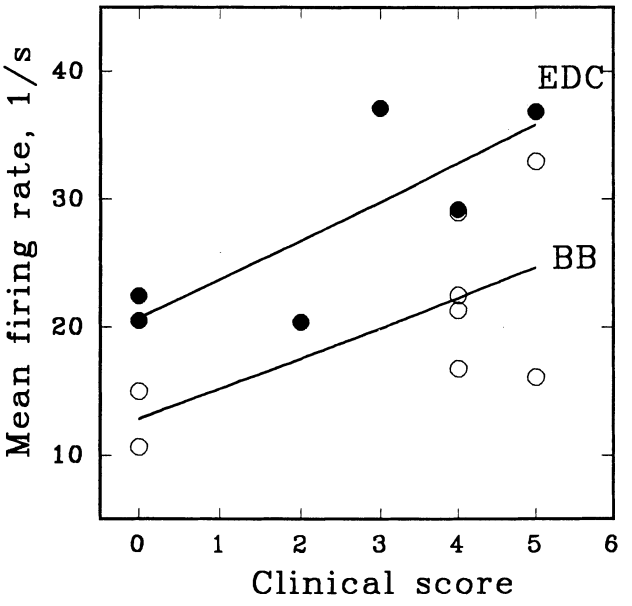

Fig. 2. The mean firing rate calculated for each subject from all the MUs analyzed at the force level 75% MVC, plotted vs his (or her) clinical score (0 for normals). Filled circles, EDC, open circles, BB.

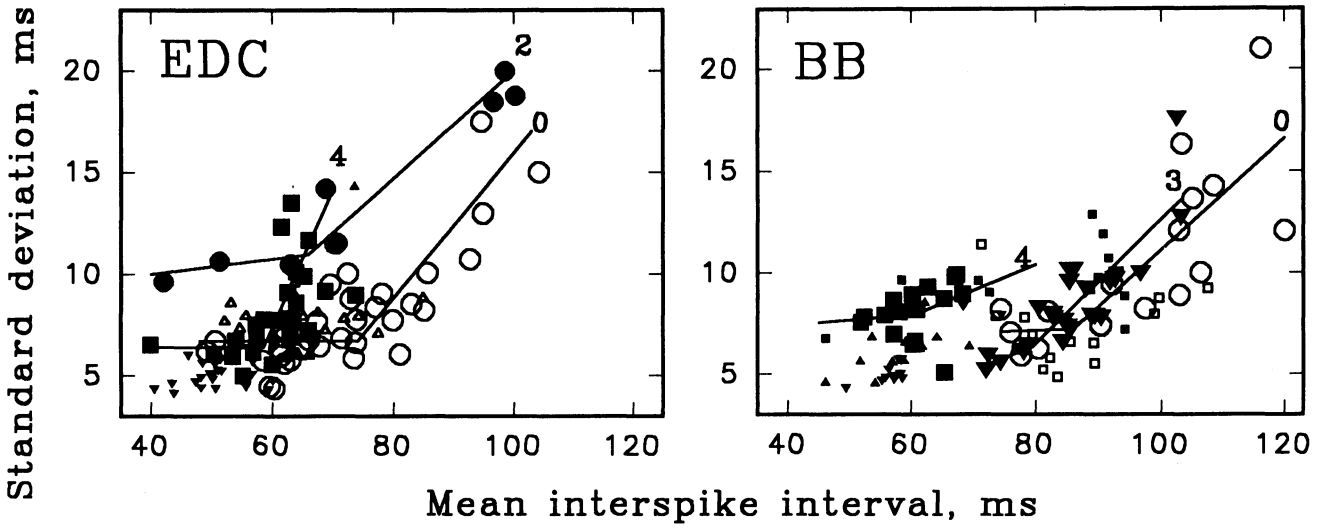

Fig. 3. The dependence of the standard deviation of ISIs on their mean value (left-hand plot, EDC, right-hand plot, BB). Each subject is represented on the plot by the different symbol (open symbols, normals, filled symbols, patients). The lines show the approximation of the results for the individual subjects (marked by larger symbols); numbers signify the scores assigned by clinical evaluation (0 for normal).

The cross-correlation histograms did not reveal any significant correlation between individual MUAPTs, except for several dystrophic MU potentials which were strongly linked together. These cases represented evidently complex potentials whose individual components were separated by such a long interval (comparable with ISI) that they were initially recognized as generated by two separate MUs. The jitter of late component was the more pronounced, the longer was its delay. These observations are in agreement with those of Hilton-Brown and Stålberg (1983) and Zalewska and Hausmanowa-Petrusewicz (1991).

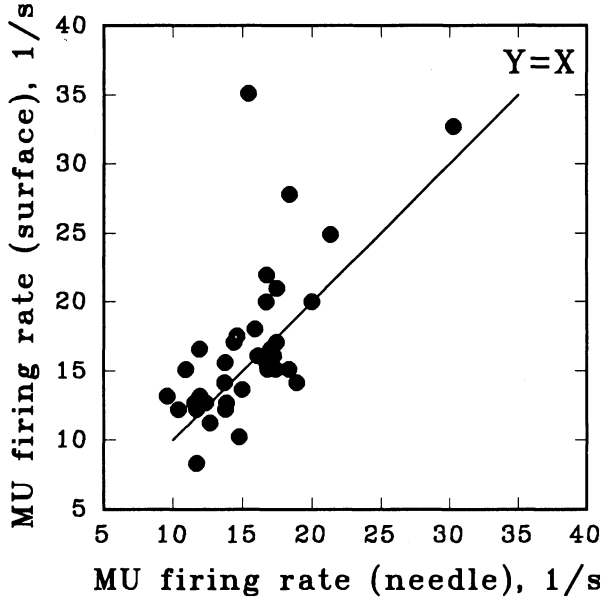

Fig. 4. The average firing rate calculated for each subject at each force level: a comparison between data from intramuscular (X-axis) and surface (Y-axis) electrodes.

DISCUSSION

All our results show that most of MUs from a dystrophic muscle tend to fire with higher rate than normal ones. This tendency is more pronounced at advanced stages of the disease. Also the increase of the firing rate with increasing muscle force is steeper in diseased muscle, which is clearly seen in EDC (cf. Fig. 1). This effect is not so clear on the plot for BB, probably because for some of the patients the data for higher force levels could not be obtained because of technical reasons. In both experimental series, there were MUs from dystrophic muscles whose firing rates fell into the range of normal MUs. Probably even in diseased muscle some MUs can be found which are not yet affected. The probability of finding them decreases with the advance of the disease. Therefore, it's well possible

that someone investigating MU activity at low levels of muscle force, from the patient at the early stage of muscle dystrophy, might not find those MUs whose firing rate was already increased. This may explain the discrepancy in the experimental results, mentioned in the Introduction.

The dependence of SDI on MI was investigated by several authors. Tokizane and Shimazu (1964) presented such plots for numerous human muscles. For each muscle, the authors distinguished two groups of points which they ascribed to two types of MUs, tonic (slower) and kinetic (faster). So far, no one of the other authors who were dealing with this subject, confirmed this dychotomy. Person (1985) also presented the plots of SDI vs MI. The general character of the dependencies was the same as of those from Tokizane and Shimazu and many others, with certain characteristic range of ISI durations above which the SDI became strongly dependent on MI. In this range also other characteristics of MU activity, such as ISI distribution and the value of serial correlation coefficient, changed their character. This characteristic ISI duration was shorter in muscles with higher content of fast MUs and corresponded well with the duration of AHP determined recently in human motoneurones by Kudina (1989). These data indicate that the value of ISI length above which all these changes occur, may be a rough estimate of an average of AHP durations in motoneurone pool of a given muscle. Our results show that this ISI duration in dystrophic muscle is shifted towards shorter values; this tendency seems to be more pronounced in more affected muscle (cf. Fig. 2). This may suggest that AHP in the motoneurones supplying dystrophic muscles has shorter duration than that in normal ones.

The evaluation of our data is still not completed. We don't yet have the quantitative results to confirm the observation that there is no substantial increase in the number of MUs recruited on subsequent force levels in dystrophic muscle, as compared with the normal one (at least in BB). However, it is our feeling that the higher density of the electromyographic recordings in muscle dystrophy is caused mainly by the two factors described above: the increasing firing rate of individual MUs and the presence of satellite potentials.

The comparison between the results obtained from surface and intramuscular recordings show quite a good correspondence between the mean firing rate determined by both methods. The scatter of points on the plot in Fig. 4 could well be expected, since the MU samples represented in both cases were not the same. This result encourages us to further studies implementing better methods of the evaluation of surface EMG power spectra.

To summarize, all the results presented above seem to support a view that in dystrophic muscle the MUs are faster in average than those in normal one, either by selective affection of slow MUs by the disease or by changes in motoneurones induced by changes in their muscle units. The comparison between the results obtained by intramuscular and surface electrodes indicate that also this latter method may be useful in further investigations.

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