Spontaneous electrical activity of human muscle☆

Activité électrique spontanée du muscle chez l'homme

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Abstract | References

Abstract

The spontaneous electrical activity of human muscle was studied in 197 normals, 67 patients with peripheral nerve involvement and 29 patients with progressive muscular dystrophy. "Noise" was observed in the end-plate zones of normal muscle; after minute displacements of the electrode the noise could be seen to consist of randomly occurring purely negative discharges 0.5–2 msec in duration and up to 100 μV in amplitude. This activity probably represents extracellularly recorded miniature end-plate potentials.

Outside the end-plate zones of normal muscle a single site was rarely encountered yielding a spontaneous discharge similar to the fibrillation potentials of denervated muscle. In normal muscle such a discharge may correspond to the propagated disphasic potentials superimposed on the "end-plate noise".

The fibrillation potentials in patients with lower motor neurone disease were found to have longer durations than usually stated (1–5 msec as compared with 0.5–2 msec), a significant proportion of triphasic potentials (30%) and voltages half of which were of the same order (100–300 μV) as those of motor unit potentials. The fibrillations found in 29 of 76 patients with progressive muscular dystrophy had the same average duration, amplitude and shape as in denervated muscles.

The fibrillation potentials were initiated in the end-plate zone as evidenced by the initial negative deflection of the potential recorded there and the initial positive deflection outside the end-plate zone. The initiation of the fibrillation potentials cannot be ascribed to mechanical stimulation by the intramuscular needle electrode since fibrillation potentials could also be recorded subcutaneously.

With the 50 μ diameter leads of a multi-electrode fibrillation potentials were recorded with peak-to-peak amplitudes as high as 8.5 mV. The decline in amplitude along the multi-lead electrode was the same for fibrillation potentials 1 mV or more in amplitude as for the spike components of motor unit potentials, the voltage falling to less than one-tenth of maximum within 0.45 mm. This suggests that "high voltage" fibrillation potentials represent the spontaneous discharge of a sub-unit. The amplitudes of 100–600 μV fibrillation potentials declined relatively less with distance, suggesting that they were recorded farther away from the generators (about 0.5 mm) than the high voltage potentials. The small amplitude and rapid attenuation of positive sharp waves suggest that they are derived from a single muscle fibre.

Cross talk in our multi-lead electrodes was insignificant and cannot explain the discrepancy between our findings and those of other investigators with regard to the voltage decline of spike potentials.

DEBUNKING THE MYTH: DENERVATED MUSCLE IS THE SOLITARY CAUSE OF MUSCLE SPONTANEOUS ELECTRICAL ACTIVITY

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Spontaneous Activity (figure 4) : Normal muscle cells do not discharge spontaneously. Muscles cells that are undergoing denervation hypersensitivity or are depolarizing for other reasons may show spontaneous activity. This activity may take one of two forms: fibrillation potentials or fasciculation potentials. Both of these are electrical activity (and contraction) of muscle in the absence of action potentials in the motor nerve innervating them. The difference between the two is a matter of degree - fasciculations are spontaneous contractions of enough muscle fibers that the twitch is visible under the skin. Fibrillations are of single muscle fibers and are not visible. As you might surmise, the electrophysiological appearance of these potentials is also a matter of degree - a fibrillation potential is a small amplitude, short duration potential (it is about 1 - 5% of the CMAP you get by nerve stimulation). Fasciculation potentials are larger (although smaller than the CMAP) and longer in duration. Both a graded qualitatively (see the table below.

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Abnormal Spontaneous Activity

When the needle is no longer being moved, normal muscle is electrically silent. Spontaneous electrical activity with the needle at rest is generally pathologic. Abnormal spontaneous activity arises when a muscle fiber loses its innervation. Clinical entities associated with abnormal spontaneous activity include denervation due to axonal injury to motor nerve fibers, and muscle fiber necrosis (because portions of individual muscle fibers separated by necrosis from their motor end plate become denervated). Motor axonal injury occurs with many neuropathies. Myofiber injury occurs in inflammatory myopathy, dystrophies, and metabolic disorders of muscle, such as hyperkalemic periodic paralysis. These same myopathic disorders often produce abnormal prolongation of insertional activity, as described earlier.

The most commons forms of abnormal spontaneous activity are *positive waves* and *fibrillation* potentials. Positive waves are potentials generated from injured single muscle fibers and represent the repolarization phase of the muscle action potential. Fibrillation potentials are bi- or triphasic potentials with an initial positive component and are thought to represent distant recording spontaneous discharge from a single denervated muscle fiber. Fibrillation potentials are of variable amplitude (20–200 μV) with a rhythmic discharge pattern (0.5–20 Hz). Fibrillation potential amplitude depends upon fiber size. Chronically denervated, atrophied muscle has small-amplitude fibrillation potentials, whereas recent denervation