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# Different Types of Fibrillation Potentials in Human Needle EMG

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## 1. Introduction

Rhythmic fibrillation potentials are the hallmark of denervated muscle fibres in needle EMG of a striated muscle (Conrad et al. 1972, Heckmann & Ludin 1982). They are readily activated by the insertion of an EMG needle electrode (Kugelberg & Petersén 1949). Irregular fibrillation potentials may also be present (Buchthal & Rosenfalck 1966, Purves & Sakmann 1974). There is, however, an obvious difficulty to discriminate between true irregular fibrillation potentials of a denervated muscle and end plate spikes, which occur in a normal muscle. This may lead to the conclusion that only rhythmic fibrillation potentials matter (Stöhr 1977). We have pointed out that fibrillation potentials, whether regular or irregular, have longer minimum interpotential intervals than end plate spikes (Partanen & Danner 1982). During long sequences, the mean interval between successive end plate spikes and rhythmic fibrillation potential tends to increase, whereas irregular fibrillations do not show this type of “self-inhibition” (Partanen & Danner 1982, Partanen & Nousiainen 1983). The aim of this chapter is to describe fibrillation potentials of different categories in either completely or partially denervated human limb muscles, or after a muscle injury. We also compare the characteristics of fibrillation potentials to neurally driven sequences, such as “myokymic” fibrillation potentials and end plate spikes (Brown & Varkey 1981, Partanen & Nousiainen 1983, Partanen 1999). “Myokymic” fibrillation potentials are a rare phenomenon of innervated muscle fibres. They have not been described earlier and are readily confused with end plate spikes. The term “myokymic” fibrillations is descriptive. The pathophysiology of “myokymic fibrillation” is different from true myokymia of whole motor units (see Willison 1982, Stålberg & Trontelj 1982).

## 2. Material and methods

Spontaneous activity was recorded with concentric needle electrodes (DISA 13L58) and a 4-channel Disa 1500 EMG machine interfaced with a 4-channel Teac R 61 D cassette recorder. Amplification was set at 50  $\mu\text{V}/\text{div}$ , and high-pass and low-pass filters at 20 and 2000 Hz, respectively. 94 sequences of spontaneous activity were divided into the following categories by audiovisual analysis: random fibrillations, slightly irregular fibrillations with occasional pauses, regular fibrillations (Partanen & Danner 1982), "myokymic" fibrillations, and end plate spikes (Partanen 1999). 10- or 20-second samples of the given sequences were digitized (sampling frequency 10 kHz) and analyzed in a Hewlett Packard 340 computer for interpotential intervals and wave forms.

"Myokymic" discharges of partially denervated muscles usually exhibited short sequences and they were found to be either "fibrillation-like" or "motor unit potential-like". These sequences were occasionally studied with another EMG needle inserted a few millimeters from the primary electrode in parallel with the muscle fibres. In "fibrillation-like" myokymic discharges it was difficult to find a synchronous discharge of the same muscle fibre in the second EMG channel whereas in "motor unit potential-like" sequences a synchronous discharge was readily observed indicating a sum potential of several muscle fibres of the motor unit. In such a case the sequence was omitted.

The raw EMG data were processed with an automatic analysis system (Partanen 1999). The rise rate, computed using a simple "low pass differentiator" (Usui & Admiror 1982) with a user definable threshold level was applied for potential recognition. Each potential recognized as belonging to the given sequence was marked with a cursor. Thereafter the sequence was checked visually on a large screen, potential by potential in order to correct possible misclassifications of the automatic program. In case of uncertainty, caused, for example, by superimposition of potentials the data were discarded and a new sample of the given sequence was taken from the tape and the procedure was repeated.

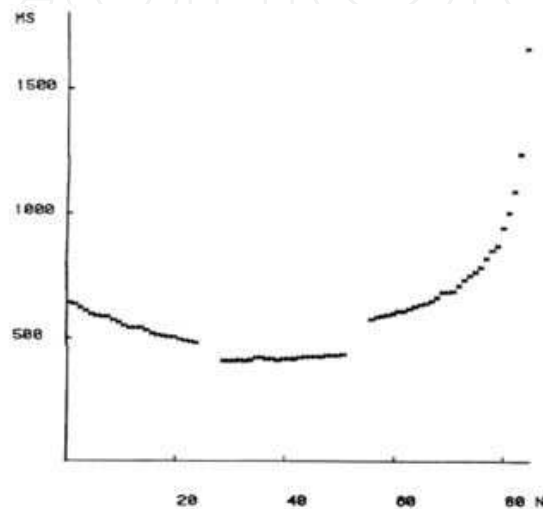
Subsequently the analysis program computed the number of intervals, mean, standard deviation, median, minimum, maximum, and interval range of the intervals, as well as the amplitude, and the spike duration of the averaged potential. The initial positive deflection of the potential could also be measured. In order to assess the regularity of firing, also the mean consecutive difference (MCD) (Stålberg et al. 1971) and the average proportional consecutive interval difference (APCID) (Conrad et al. 1972) were calculated.

The different potential categories were analyzed using Student's unpaired t-test.

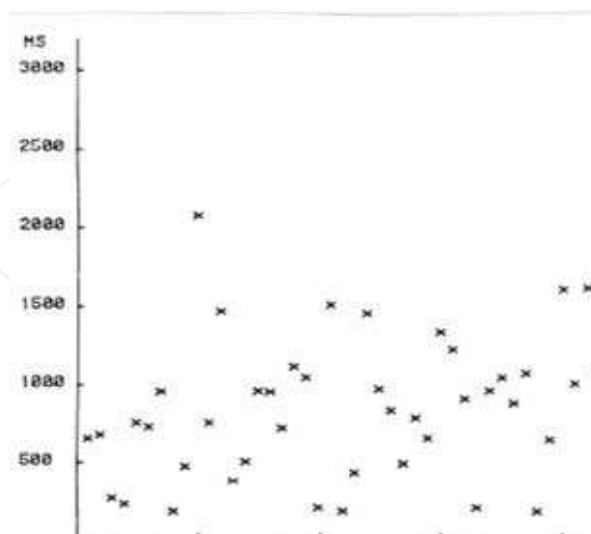
## 3. Results

Figures 1-3 show the typical firing pattern of different spontaneous fibrillation categories. Random and slightly irregular fibrillations with pauses could usually be recorded for several minutes. In many cases we collected several samples of a sequence, one of which was chosen

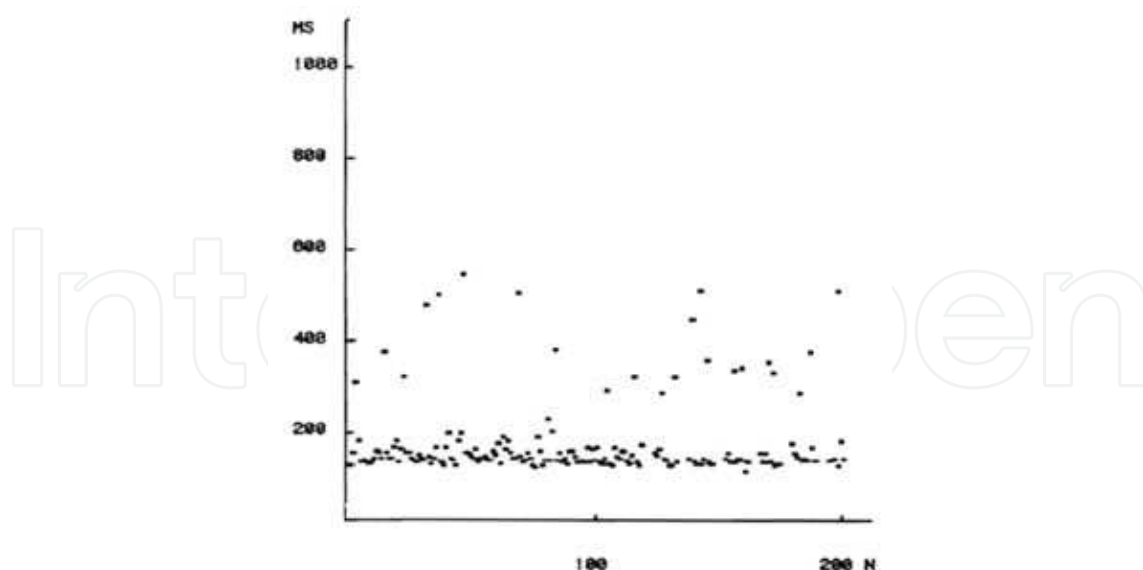
for the analysis. This was possible because these fibrillation sequences were persistent with the same firing pattern. Also a sequence of rhythmic fibrillation potentials had to proceed several seconds in order to be accepted. Rhythmic fibrillations were usually elicited by needle insertion and they showed a gradual shift of interpotential intervals during the recording time (Conrad et al. 1972). A short-lasting burst of insertion activity was not accepted. Slightly irregular fibrillations with pauses did not show a gradual shift in the basal interval, nor were they affected by needle insertion.



**Figure 1.** A regular sequence of fibrillation potentials. Interruptions in line indicate interruptions in recording. On the vertical axis is the interpotential interval of fibrillation potentials. On the horizontal axis is the number of successive intervals. There are 20 intervals per division. From Partanen, J.V. & Danner, R. (1982), Author's own work.



**Figure 2.** Randomly occurring fibrillations 33 days after muscle biopsy. Vertical axis is the interpotential interval. Horizontal axis is the number of successive intervals; there are 10 intervals per division. From Partanen, J.V. & Danner, R. (1982), Author's own work.



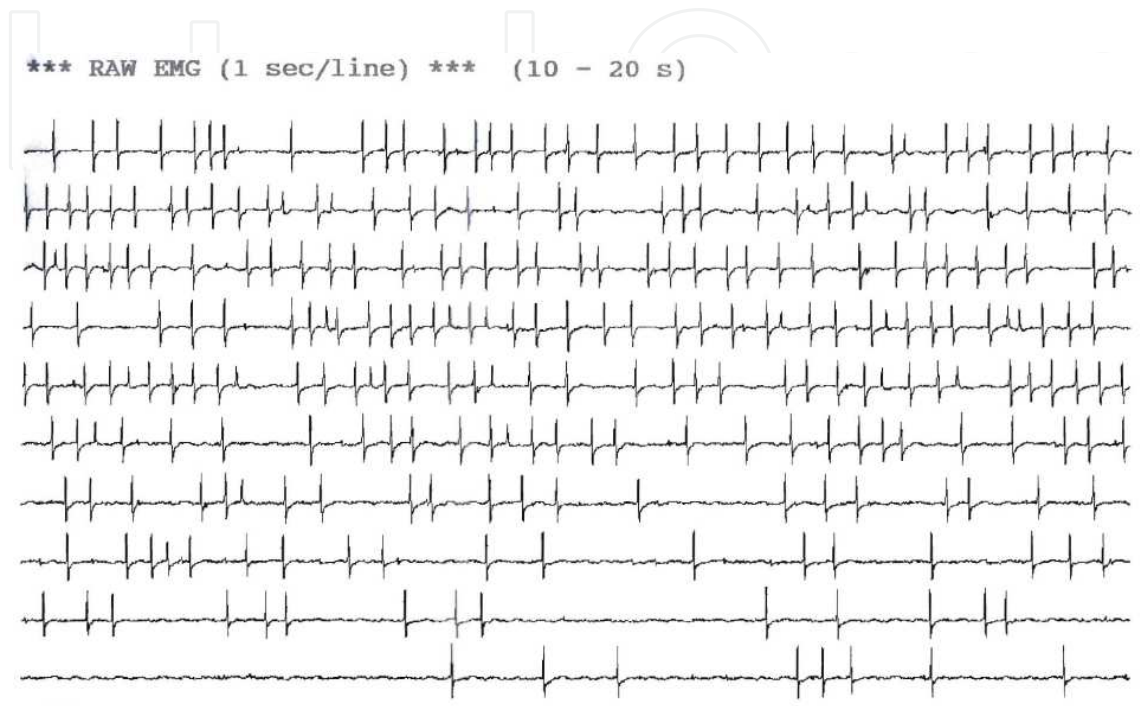
**Figure 3.** Slightly irregular fibrillations with occasional pauses (long intervals); 51 days after muscle biopsy. Note the slight irregularity in the basal (short) intervals. From Partanen, J.V. & Danner, R. (1982), Author’s own work.



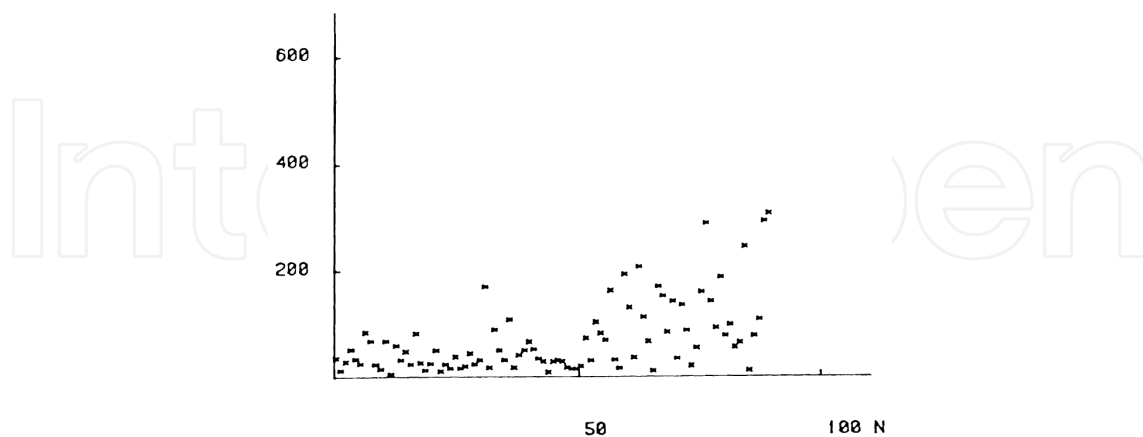
**Figure 4.** “Myokymic” fibrillations. Note the doublets and triplets and short rapid bursts of potentials.

“Myokymic” fibrillations were found in chronically partially denervated muscles, polymyositis and after chemotherapy. The duration of the bursts was short. There were also single fibrillation potentials, doublets and triplets and independent potentials from several different

muscle fibres (Fig. 4). The bursts were spontaneous, not elicited by needle insertion. End plate spikes were found when the needle insertion hit an “active spot” of the muscle (Fig. 5-6). They were most readily found at the end plate zone, but they were not confined to it (Partanen, 1999). In several instances we could simultaneously record two unsynchronous foci of end plate spikes at completely different sites of a normal muscle.



**Figure 5.** A sequence of end plate spikes. Observe the gradual slowing of the firing frequency.



**Figure 6.** The firing pattern of a single sequence of end plate spikes (about 10 s, 78 intervals) recorded from the gastrocnemius muscle. Horizontal axis: number of successive intervals. Vertical axis: interpotential interval in ms. Note the variability in interpotential intervals, numerous short intervals and the gradual increase of the mean interval. APCID 176.4 ms, MCD 48.0 ms and the minimum and maximum intervals 9 ms and 292 ms, respectively. From Partanen, J. & Nousiainen, U. (1983), Author’s own work.

We also performed an analysis of the initial positivity on 39 different end plate spikes and 33 different fibrillation potentials. 14 out of the 39 end plate spikes had an initial positive deflection, with the mean duration 0.5 ms, SD 0.17, and range 0.3-0.9 ms. The rest had a negative onset. All 33 fibrillation potentials had an initial positive deflection, with mean duration 1.6 ms, SD 0.4, and range 0.6-2.4 ms. Thus, when an initial positive deflection was observed in end plate spikes, it was significantly ( $t=9.9$ ;  $p<0.001$ ) shorter than that of fibrillation potentials. The 95 % confidence interval for difference was 0.8 to 1.2 ms.

Table 1 presents the mean characteristics in different fibrillation potential categories and the significance of difference of the variables compared to end plate spikes. Table 2 presents the differences between variables of different fibrillation categories.

	Fibrillations						Neurally driven sequences			
	Random		With pauses		Regular		"Myokymic"		End plate spikes	
N	41		13		13		12		15	
Intervals (ms)	mean	SE	mean	SE	mean	SE	mean	SE	mean	SE
mean	510***	43	223	37	556*	191	142	37	130	42
median	387***	37	209*	39	548*	190	104	30	95	31
minimum	159***	14	148***	25	513***	186	25*	5	12	3
maximum	1406***	127	412	49	637	199	736	204	578	163
<b>Regularity</b>										
APCID	151	7	52*	6	5***	1	128*	14	175	13
MCD	358***	42	47	7	13*	5	111	36	116	42
<b>Potential</b>										
Ampl ( $\mu$ V)	145	13	120	14	117	19	169	36	128	14
Spike duration	2.3	0.1	3.1	0.4	2.4	0.1	2.5	0.2	2.8	0.4
(ms)										

\*\*\*  $p \leq 0.001$

\*\*  $p \leq 0.01$

\*  $p \leq 0.05$  compared to end plate spikes

**Table 1.** Interval, regularity and potential variables of different spontaneous activity categories. "With pauses": slightly irregular fibrillation potentials with pauses.



	Random/With pauses	Random/Regular	With pauses/Regular
<b>Interval</b>			
Min	NS	NS	NS
Max	***	**	NS
Mean	***	NS	NS
APCID	***	***	***
MCD	***	***	***
Amplitude	NS	NS	NS
Spike duration	NS	NS	NS

\*\*\*  $p \leq 0.001$ , \*\*  $p \leq 0.01$ , \*  $p \leq 0.05$

**Table 2.** The significance of differences between various fibrillation categories. “With pauses”: slightly irregular fibrillation potentials with pauses.

## 4. Discussion

### 4.1. Firing patterns of fibrillation potentials

Denny-Brown & Pennybacker (1938) described the periodic contractions of denervated muscle fibres as true fibrillations and differentiated fibrillation from fasciculations and myokymia. Jasper & Ballem (1949) found positive sharp waves, often in combination with fibrillation potentials and claimed that they may represent local potentials set up at the needle point by the injury. They stated that positive sharp waves do not occur in a normal muscle. Kugelberg & Petersén (1949) also described positive sharp waves, “synchronized activity” in totally denervated muscles as well as fibrillation potentials of both constant frequency (regular fibrillations) and “repetitive fibrillary activity”, i.e. slightly irregular fibrillations with pauses (see Results). It was claimed that only rhythmic, regular fibrillation potentials have clinical significance (Stöhr 1977). However, also irregular fibrillations do exist, and in fact there are several types of them. Irregular fibrillations do not usually change their firing pattern during the time of an EMG recording. The incidence of irregular fibrillations reported in literature is very variable. Heckmann & Ludin (1982) pointed out that even in totally denervated muscle irregularly firing potentials may be found (in canine muscle), and Buchthal & Rosenfalck (1966) stated that half of the fibrillation potentials whose discharge pattern was examined appeared irregular. In fact, the period of time after nerve or muscle injury seems to be essential. Approximately half of fibrillation sequences were irregular 30 days after muscle injury, while in more recent injuries the sequences were mainly regular (Partanen & Danner 1982). My experience in clinical ENMG work is that irregular fibrillations are most common 1 – 3 months after axonal injury and in extreme cases only a number of irregular fibrillation sequences may be present with no regular fibrillations at all (unpublished personal observation). There may



also be mixed forms of fibrillations, with mainly regular rhythm but sudden changes of the interval (Partanen & Danner 1982, Conrad et al. 1972).

#### **4.2. End plate spikes**

Jasper and Ballem (1949) were the first to describe end plate spikes in clinical EMG: "Action potentials comparable to those described by Snodgrass and Sperry (mammalian muscle action potentials of less than a millisecond) were sometimes seen from limb muscles were thought to have been derived from nerve filaments since they were usually associated with particularly acute pain (as though the needle tip were penetrating a nerve) and were of the same form as those obtained when the needle was deliberately inserted in nerve". Kugelberg & Petersén (1949) described end plate spikes as "protracted irregular activity". "Such discharge was mostly irregular, might be ordinary motor unit potentials as in fasciculation or little amplitude and duration as in fibrillation. The activity in question cannot be voluntarily controlled. It does not disappear in relaxation, nor does it increase in frequency on slight voluntary contraction. A slight pressure or bending of the needle may increase the frequency while a discharge is going on, start new ones or reactivate potentials which had stopped".

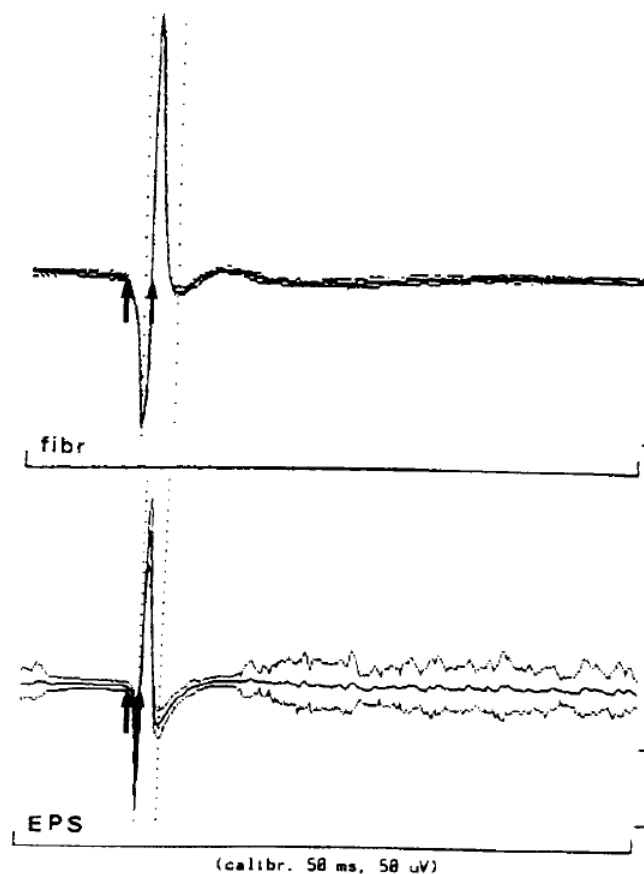
Jones et al. (1955) further studied the origin of end plate spikes as "nerve potentials" with iron marks at sites of their appearance and found most of these iron dots close to peripheral intramuscular nerve twigs. Buchthal & Rosenfalck (1966) observed that miniature end plate potentials (MEPPS) were often associated with end plate spikes, "spontaneous diphasic potentials". They conjectured that these potentials originated in the muscle fibres, "several synchronized miniature potentials attaining an amplitude sufficient to elicit a propagated response". Finally, Brown & Varkey (1981) proved "nerve potentials" to be postsynaptic potentials, recorded from muscle fibres. End plate spikes show very irregular firing pattern with numerous short intervals less than 30 ms and gradual slowing of the firing (Partanen 1999).

The prevailing hypothesis concerning end plate spikes states that they are elicited by nerve irritation caused by the needle electrode and recorded postsynaptically by the same needle electrode (Brown & Varkey 1981). However, injury potentials of peripheral motor nerve fibres present a different firing pattern (Wall et al. 1974, Macefield 1998). Thus there is an obvious discrepancy between the sustained firing pattern of end plate spikes and experimentally observed real firing patterns of injured or irritated motor axons. Firing of end plate spikes differs also from abnormal firing patterns of motor nerve fibres or motor units (see Willison, 1982, Stålberg & Trontelj 1982). On the other hand, there is evidence that end plate spikes actually represent action potentials of intrafusal muscle fibres and beta motor units (Partanen & Nousiainen 1983, Partanen 1999, Partanen et al. 2010). The propagation patterns of intrafusal nuclear bag and nuclear chain muscle fibres (Barker et al. 1978) are similar to those of end plate spikes (Partanen & Palmu 2009, Partanen 2012).

#### **4.3. Wave form of fibrillation potentials and end plate spikes**

It was emphasized that end plate spikes, ("spontaneous diphasic potentials"), which have a negative onset at the end plate zone, may show positive onset phase as fibrillation potentials do

when they are propagated outside the end plate zone and thus the form of the potential is indistinguishable from that of a fibrillation potential (Buchthal & Rosenfalck 1966). We note that there is a distinct difference between the wave forms of end plate spikes and fibrillation potentials. The former show either a negative onset or a short positive onset, whereas fibrillation potentials show a positive onset which always is longer than that of end plate spikes (see Results). However, the amplitude and spike duration of fibrillation potentials and end plate spikes are similar (Table 1). We have observed fibrillation potentials with negative onset, (mainly as “negative sharp waves”, obviously cannula-recorded positive sharp waves with inverted polarity), but these are rare and do not happen to be present in the material collected for this work. This fact is not in concert with the data published earlier, which state that a considerable number of fibrillation potentials may have a negative onset (Buchthal & Rosenfalck 1966, Heckmann & Ludin 1982). In any case fibrillation potentials and end plate spikes may be distinguished both by the firing pattern and the wave form at the onset of the potential (Fig. 7).



**Figure 7.** The positive deflection before the main spike component (pair of arrows) is shorter in averaged end plate spike (EPS) than in averaged fibrillation potential (fibr). The averaged mean potential is shown with  $\pm 1$  SD curves. From Partanen, J. (1999), Author's own work.

The formation of the shape of end-plate spikes was extensively studied by Dumitru (2000) according to the needle irritation hypothesis of peripheral nerve branch or nerve terminal (tip or shaft irritation of the terminal nerve). He explained the formation of biphasic and triphasic

form of an end plate spike and considered that triphasic end plate spikes are rather common. However, he could not differentiate the shape of triphasic end plate spikes from that of triphasic fibrillation potentials. The spreading of an ectopic nerve irritation potential to the other nerve branches of the motor unit was not considered. Ectopic nerve action potential will spread to both directions from its place of origin, and thus a motor unit or fasciculation potential should be formed instead of an end plate spike. Dumitru (2000) also describes the formation of "atypical" biphasic/monophasic end plate spike configuration (resembling positive sharp waves). First, the electrode may completely compress the muscle fibre, preventing action potential propagation past the electrode ("sealed end effect"). Second, a "compressed end" may occur; following crushing or compression of tissue, the membrane retains no functional sodium channels and, therefore, can only sustain a passive current flow, but not an active current flow. However, Pickett & Schmidley (1980) explained end plate spikes with positive sharp wave form, "sputtering positive potentials" elegantly. These potentials represented cannula-recorded potentials of the concentric needle electrode and changed their form from positive waves to usual end plate spikes when the electrode was withdrawn. Sputtering positive potentials could not be recorded with a monopolar needle electrode.

#### **4.4. Origin of regular and irregular fibrillation potentials**

Based on the pattern of discharges, two classes of spontaneously active fibres were found in experimental study of rat diaphragm: rhythmically discharging fibres, and fibres in which action potentials occur at irregular intervals (Purves & Sakmann 1974). The majority of the sites of origin in both regular and irregular fibres were at the former end plate zone; however, there was no region along the length that could not be a site of origin. Regularly occurring action potentials were associated with oscillations of the membrane potential. Irregularly discharging fibres were brought to threshold by discrete non-propagated depolarizations called fibrillatory origin potentials (f.o.p.s.) (Purves & Sakmann 1974). F.o.p.s. are generated at the T-tubuli, since detubulation with glycerol abolishes the spontaneous activity (Smith & Thesleff 1976). Thus, the integrity of the transverse tubular system is a prerequisite for the presence of irregular spontaneous activity. It was also observed, that these discrete depolarizations are caused by regenerative increase in the Na conductance of the membrane, similar to that associated with the normal action potential (Purves & Sakmann 1974, Smith & Thesleff 1976).

We may presume that in humans, fibrillations with regular rhythm also derive from the membrane potential oscillations of denervated muscle fibres (Thesleff 1982a) or the denervated part of a muscle fibre, as in muscular injury (Partanen & Danner 1982). Irregular fibrillations are accordingly caused by f.o.p.s reaching the firing threshold of an action potential. Immediately after a f.o.p. there is a period during which the probability of a second f.o.p. occurring is very low (Purves & Sakmann 1974). In denervated muscle fibres there are newly synthesized potassium channels, and they produce a longer duration of the hyperpolarization of the intracellular action potential compared to normal tissue. This hyperpolarization may last up to 100 ms and more (Thesleff 1982a, Dumitru 2000). Thus the refractory period after which a second action potential may occur is increased in denervated muscle fibres, compared to normal muscle fibres. Thus slightly irregular fibrillations with pauses may be fired by a muscle

fibre eliciting a large number of f.o.p.s., which mainly reactivate the fibre immediately after the refractory period of a spontaneous potential. An occasional failure of a f.o.p. to occur may be seen as a pause in the fibrillation sequence. We have rarely observed a slightly irregular fibrillation sequence even without pauses, evidently representing a muscle fibre with a large number of f.o.p.s. On the other hand, random fibrillations may be associated with very infrequently occurring f.o.p.s. In any case, regular fibrillations are the first to be present also in experimental studies and irregular fibrillations arise later on (Purves & Sakmann 1974, Smith & Thesleff 1976).

#### 4.5. "Myokymic" fibrillations

"Myokymic" fibrillations have not been categorized as an entity of its own earlier. They may be distinguished from true myokymia by the single fibre potential pattern. True myokymia exhibits a motor unit potential pattern, and was not studied in the present work. The high firing frequency of "myokymic" fibrillations shows that these potentials are not elicited by denervated muscle fibres with a prolonged refractory period. We attribute these potentials to spontaneous large acetylcholine release (giant or slow-rising MEPPs) to the synaptic cleft. This type of transmitter release may occur spontaneously in regenerating nerve terminals or after botulin toxin injection or application of 4-aminoquinoline, without any motor nerve action potential and depolarization of the motor nerve terminal (Thesleff 1982b, Sellin et al. 1996). Evidently large spontaneous transmitter release may cause a short burst of postsynaptic potentials of a single muscle fibre, recorded as "myokymic" fibrillations. It is conceivable that no antidromic spreading of the potential to the rest of motor unit takes place without depolarization of the nerve terminal, as in peripherally originating fasciculation potentials (see Stålberg & Trontelj 1982).

"Myokymic" fibrillations and end plate spikes can be distinguished by their firing pattern. "Myokymic" fibrillations fire in short high-frequency bursts, doublets and triplets and they may be found at any region in the muscle. Needle insertion does not activate them. End plate spikes show sustained firing with a very irregular rhythm with numerous short but also long intervals, and the mean interval lengthens if the needle is not moved. End plate spikes are found in the active spots of the muscle being studied, often associated with miniature end plate potentials and pain (Wiederholt 1970).

## 5. Comments

It is of utmost importance that a clinical neurophysiologist performing ENMG studies recognizes different types of spontaneous activity. Confusing end plate spikes with fibrillation potentials may cause false positive findings of axonal damage. Even the difference between rhythmic and irregular fibrillation potentials may be difficult to grasp and there are differences between individual examiners in this respect (Trillenbergh & Spencer 2010). False classification of potentials is a frequent error, especially among resident-level examiners (Kendall & Werner 2006). We studied parameters by which it could be possible to distinguish between different

types of fibrillation potentials: rhythmic, random, and slightly irregular fibrillations with pauses, “myokymic” fibrillations, and end plate spikes. The most effective differentiating variables in this respect proved to be the minimum interval, APCID and MCD. Interval analysis of the activity of a single motor unit also shows an entirely different discharge pattern compared to spontaneous potentials (Conrad et al. 1972). The functionality to calculate these variables for sequences of spontaneous EMG potentials should be included in future ENMG devices. The lack of tools for editing and interval analysis of EMG potential sequences can be considered to be a major shortcoming in the present ENMG machines.

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