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SOME PROPERTIES OF MAMMALIAN SKELETAL MUSCLE FIBRES WITH PARTICULAR REFERENCE TO FIBRILLATION POTENTIALS

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Fibrillation potentials recorded from denervated muscle are generally believed to originate in single muscle fibres (Denny-Brown & Pennybacker, 1938). Eccles (1941) suggested that some of the fibrillation potentials recorded from denervated skeletal muscles of the cat may arise at any point along the muscle fibre, whereas Hayes & Woolsey (1942) concluded from their experiments on denervated rat diaphragm that all fibrillation potentials take origin from the end-plate zone. These views were based on observations by means of relatively large extracellular recording electrodes, and the origin of the potentials could not be determined with absolute certainty.

With intracellular electrodes Nicholls (1956) measured the resting potentials and membrane resistance of denervated muscles of the frog, but no account was given of spontaneous fibrillation potentials, probably because fibrillation is infrequent in amphibia (Fatt & Katz, 1952). Membrane resting potentials of denervated mammalian skeletal muscles were also studied with intracellular electrodes by Ware, Bennett & McIntyre (1954). These authors likewise made no report on the spontaneous fibrillation potentials.

The experiments described below were designed primarily to study the fibrillation potentials recorded with intracellular electrodes from denervated muscle fibres. This study also permitted observations on the responses to motor nerve stimulation in mammalian skeletal muscles *in vivo*.

METHODS

Twenty-five albino rats weighing between 200 and 250 g were used. The sciatic nerves of fourteen of these rats were cut in mid-thigh under ether anaesthesia 3–22 days before recording. Study of the response to electrical stimulation was carried out in the eleven remaining rats. Some of the rats were curarized with an intravenous dose of tubocurarine chloride (1·4 mg/kg).

At the time of the recording the trachea and right external jugular vein were cannulated under ether anaesthesia. The animal was then maintained at a surgical anaesthetic level by thiopental

sodium administered intravenously. The tracheal cannula was attached to a Plexiglass chamber equipped with a flutter valve, through which pure oxygen flowed. Intermittent positive-pressure artificial respiration was administered when needed.

The denervated muscles (anterior tibialis, extensor digitorum longus, peroneus longus, soleus and gastrocnemius) were exposed by reflexion of the skin and fasciae. The entire limb, with its blood supply intact, was kept immersed in mammalian Krebs's solution (NaCl, 118·5; $NaHCO_3$, 24·85; $CaCl_2$, 2·54; KH_2PO_4, 1·118; $MgSO_4$, 1·118; KCl, 4·74 mM; and dextrose 1 g/l.; the solution was equilibrated with $O_2 + CO_2$ mixture). The cut sciatic nerve was exposed for stimulation. In some experiments the muscle used was reflected to facilitate insertion of the microelectrode into the under surface, thereby avoiding the fibrous tissue on its outer surface.

The recording electrodes were glass micropipettes, measuring less than 0·5 μ in outside diameter filled with 3M-KCl solution. The glass micropipette electrode was connected to the input grid of a cathode follower, which in turn was connected to a direct-coupled amplifier. A condenser-coupled amplifier was also available for high gain records. A calibration signal could be sent through the indifferent electrode in the bath to the preparation as desired. A battery was used to balance the 2 V above ground presented by the floating grid of the cathode follower. The frequency response of the recording system was flat from 0 to 6000 c/s, falling to 50% at 15,000 c/s.

The micropipette was attached to a micromanipulator, and the initial contact of the micro-electrode tip with the muscle could be observed under a microscope. Further insertion of the micro-electrode was carried out blindly because it was necessary to search for fibrillating fibres in the depth of the muscle. Penetration of the muscle fibres was indicated by a sudden shift of steady potential to negativity recorded at the tip of the microelectrode. These changes were striking and offered reliable criteria for fibre penetration; however, little confidence could be placed in the absolute values of the recorded membrane resting potentials. This will be discussed later.

RESULTS

Innervated muscles

The muscles in the control animals showed no fibrillary movements under the microscope. As the microelectrode entered a muscle fibre, signalled by a sudden shift of steady potential, stimulation of the motor nerve elicited a spike action potential and a muscle twitch. If the electrode was not dislodged by the muscle movement, the spike potential, in response to a subsequent stimulus, would decrease in amplitude and show an inflexion in its rising phase. As the amplitude of the spike was further reduced, the inflexion would become more conspicuous, appearing as a 'notch', and eventually the spike would split into two components. This change, presumably due to a delay in impulse conduction along the impaled fibre, is similar to that observed in damaged nerve fibres (Tasaki, 1952; Woodbury, 1952).

As one would expect, when stimulation was directly applied to the muscle, no prepotential was recorded, but prepotentials were frequently observed following nerve stimulation (Fig. 1). On some occasions the spikes evoked by repetitive stimulation were initiated at a constant level of potential, as shown in Fig. 1. In this experiment the first two stimuli were followed by depolarization of the membrane to the same potential level where spike discharges occurred, although the membrane resting potentials were not the same in the two cases. Depolarization of the fibre following subsequent stimuli did not reach a critical level and failed to initiate spike discharges. In most cases,

however, responses to successive stimuli were unpredictable in their configurations, probably because of the rapid deterioration of the impaled fibre consequent on contraction.

In curarized rats nerve stimulation elicited responses such as those illustrated in Fig. 2 (these experiments were carried out in collaboration with

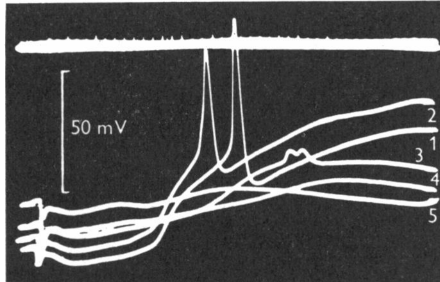


Fig. 1. Single muscle fibre responses to nerve stimulation. The first line of the record represents zero membrane potential with time marks 1 msec superimposed; the numbers at end of each response indicate the order in which the stimuli were applied to the nerve; note the critical level for the spikes to rise and the inflexion in the rising phase of the second and 'splitting' of the third response, presumably due to a delay in impulse conduction. In this and subsequent figures positivity is upwards unless stated otherwise.

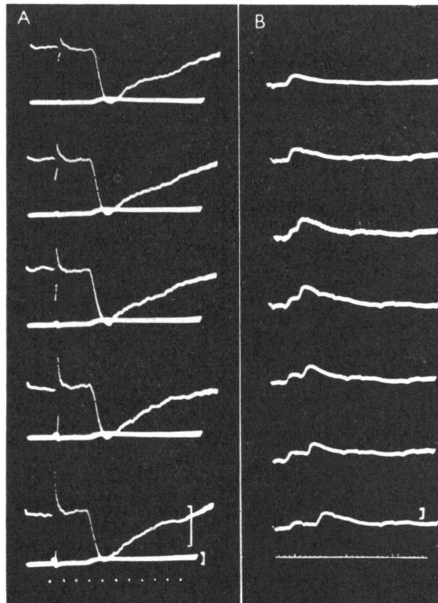


Fig. 2. Small potentials in response to nerve stimulation after the animal had been curarized. A, responses to stimulation of increasing strength from 0·5 to 5·0 V with a pulse duration of 0·2 msec; the first line of each record was taken through a condenser-coupled amplifier in which downward deflexion represents positivity; B, responses to twin shocks of variou s intervals. Time marks, 1 msec; voltage calibration, 2 mV.

Dr Paul Fatt). The size of these responses recorded from different experiments varied considerably, but their time course was fairly consistent and comparable to the measurements given by Fatt & Katz (1951) for the end-plate potentials of the frog's sartorius muscle. These potentials appeared to be 'all-or-none'; the size of the potential recorded from a given fibre was not affected by altering the strength of the nerve stimulation (Fig. 2A). Further, summation of two potentials evoked by successive stimuli was observed. With deep curarization the summated potentials failed to generate a spike discharge, although the potential in response to the second stimulus tended to be greater than the first (Fig. 2B). This is consistent with the observations of Eccles, Katz & Kuffler (1941).

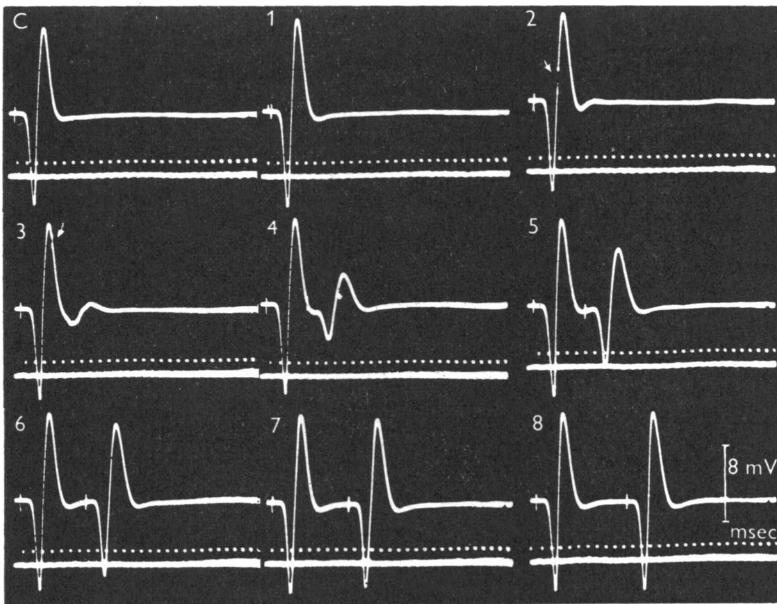


Fig. 3. Responses of a motor unit to twin shocks at various intervals; time marks, 1 msec; calibration, 8 mV.

The absolute refractory period of these potentials was found to be about 2 msec (Fig. 2B). This observation was substantiated by the experiments in which the excitability of motor units was tested. Since the discharge of a motor unit results from synchronous firing of many muscle fibres, it cannot be detected with intracellular electrodes. In these experiments an extracellular microelectrode in the muscle recorded a diphasic potential in response to application of stimulus to the motor nerve (Fig. 3): the diphasic potentials might be as large as 25 mV. When twin shocks were applied to the nerve within an interval of 2 msec no response to the second shock was recorded.

As the interval between the two shocks was lengthened, the second response began to grow. The gradation of the response of a motor unit probably depends upon the number of responding muscle fibres. Fig. 4 shows the recovery of a motor unit following a single stimulus to the motor nerve.

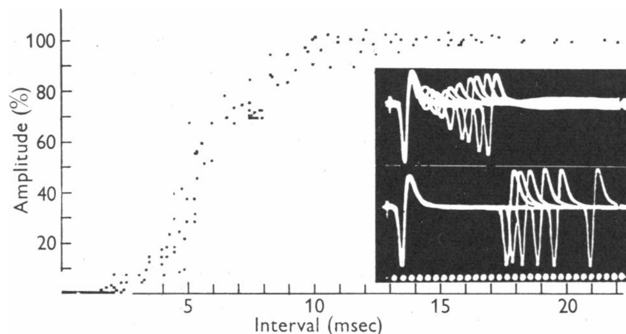


Fig. 4. Recovery of a motor unit following single stimulation to motor nerve. The inserted superimposed records illustrate the gradual increase in size of the responses to test stimuli.

Denervated muscles

Two days after a muscle was denervated no fibrillary movement of the exposed muscle could be observed under the microscope, nor were any spontaneous potentials detected. On the third day both visible and electrical fibrillation began, but the number of fibres involved was small. Stimulation of the nerve distal to the division failed to elicit any movement of the muscle. From the fourth to the twenty-second day extensive fibrillary movements were observed, which disappeared about 30 min after the muscle had been exposed, but fibrillation could still be detected electrically up to more than 3 hr after exposure.

Fibrillation potentials recorded with an extracellular microelectrode were diphasic. Their amplitude varied considerably, but commonly fell between 1·0 and 3·0 mV when recorded with the same microelectrodes used for intracellular recordings. Spikes of different amplitudes were often recorded simultaneously. The multiplicity of unit discharges recorded with an extracellular electrode can be ascribed to overlapping of the potential fields generated by several muscle fibres (Lundervold & Li, 1953). The gradient of potentials in the fields must be steep since a slight movement of the microelectrode would cause a disproportionate increase in the size of one unit in comparison to another.

Frequently a diphasic unit would suddenly become monophasic with an enormous increase in amplitude. In these instances the spikes were positive and were not intermingled with spikes of various amplitudes. The frequency of discharge remained unaltered, and there was a shift of the steady potential

to negativity, indicating that the recording electrode probably had entered a muscle fibre and was recording what had previously been an extracellular diphasic potential from the other side of the surface membrane. The transformation of a diphasic spike into a monophasic one as the recording electrode impales the surface membrane has been demonstrated in cortical neurones (Li, 1955).

The spontaneous fibrillation potentials, recorded intracellularly, were distinguishable from injury potentials because they were found only in denervated muscles; they also maintained a constant amplitude and frequency for long periods. Fig. 5 illustrates this by showing that the frequency of discharge of a fibre was fairly regular until signs of deterioration of the resting and action potentials developed. Usually this happened from 1 to 20 min following the impalement of the fibre by the microelectrode.

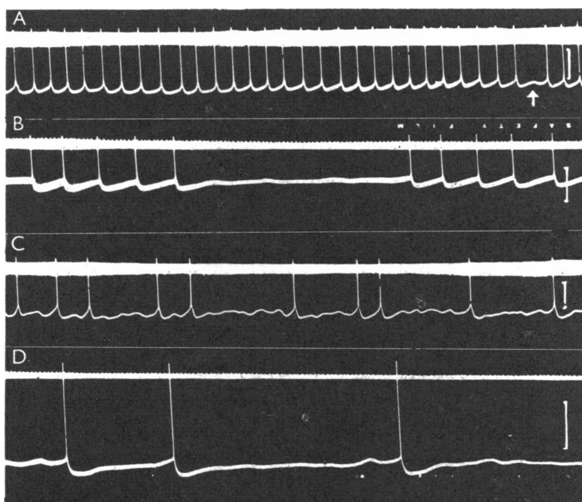


Fig. 5. Fibrillation potentials. The marks on the zero membrane potential line are 10 msec apart; vertical bars represent 50 mV. Note the different frequencies of the spontaneous discharges recorded from different fibres and the instability of the membrane resting potential. In A rhythmic oscillation of membrane potential is illustrated by absence of spike indicated by arrow.

Fig. 5 also illustrates that in some cases fibrillation potentials were associated with regular oscillations of membrane resting potentials similar to those recorded by other authors in other rhythmically firing structures (Weidmann, 1951; Eyzaguirre & Kuffler, 1955; Frank & Fuortes, 1955). In agreement with these authors it was observed that spike discharges occurred when oscillation of the underlying slow potential reached a given depolarization level, which was constant in a given unit. The requirement for a critical level of depolarization for initiation of spike discharges is comparable to that observed in innervated muscle fibres (see Fig. 1).

It was also noted (Fig. 5 A) that rhythmic oscillations of potentials could be recorded in the absence of spike discharge, suggesting that the spike is not a necessary link in the events leading to rhythmicity. This is consistent with the conclusions obtained from experiments with nerve fibres in reaction to chemical agents (Brink, Bronk & Larrabee, 1946).

In some experiments oscillation of the underlying slow potential was not present, thus the record appeared flat in the interval between the spike discharges. The spike was usually preceded by a prepotential. The size of the prepotentials recorded from a muscle fibre at a given electrode position was constant, although there were variations in different experiments (Fig. 6).

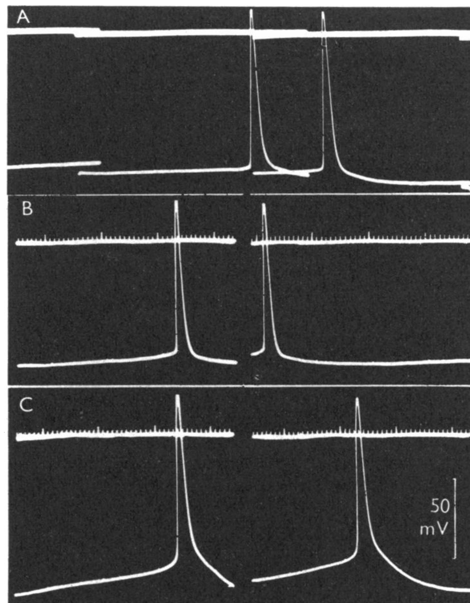


Fig. 6. Prepotentials of various sizes recorded from three different fibres. Time marks, 1 msec; voltage calibration, 50 mV.

It is conceivable that the spike potential was propagating from an active focus and the size of the prepotential was determined by the distance between the active focus and the site of recording.

Frequently spontaneous rhythmic small potentials were recorded from a denervated fibre. Size and frequency of these small potentials recorded from different fibres varied, but their configuration and time course were rather constant (Fig. 7). In the experiment shown in Fig. 7 A the rising phase of a small potential took 1 msec to reach its crest of 10 mV, followed by a slow decline which lasted for 24 msec. In B the amplitude of the small potential was 5·5 mV with a rise time of 1 msec and total duration of 30 msec. A possible

explanation derived from these measurements is that these small potentials might be end-plate potentials.

In many experiments small potentials were recorded from a muscle fibre concomitant with spike potentials (Fig. 8). Usually the frequencies of small potentials and spikes were different and unrelated. In these cases, when the onset of a spike happened to occur in the rising phase of a small potential, the discharge of the spike falsely appeared as though initiated by the small potential identical to an end-plate potential. Conversely, if a small potential occurred in the falling phase of a spike, the small potential would appear as a 'hump'.

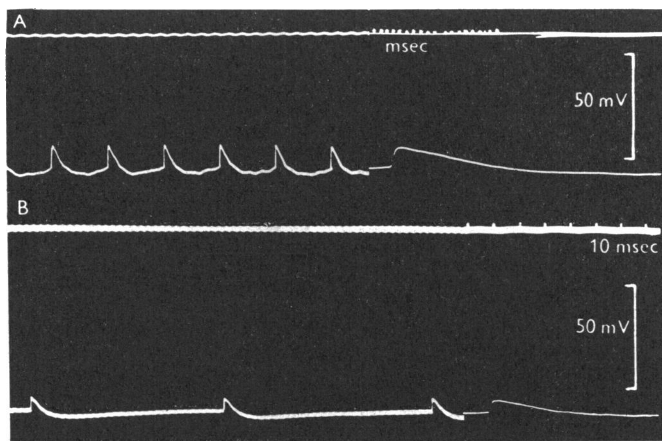
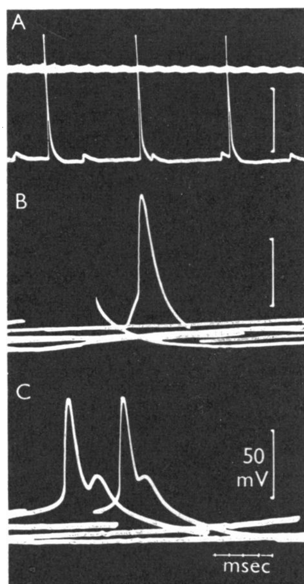


Fig. 7. Spontaneous discharge of small potentials recorded from two denervated muscle fibres with slow and fast sweep speeds. Zero membrane potential line marks 1 msec in A and 10 msec in B; voltage calibration 50 mV.

In other experiments the spike discharges were always preceded by small potentials. In these cases, identification of these two potentials as two different events became difficult (Fig. 9). The record taken with an expanded time base showed that there was a distinct 'step' amounting to 25 mV with a duration of 1·5 msec. The configuration of this record bears a remarkable resemblance to those obtained following nerve stimulation in innervated muscles (see Fig. 1).

In innervated muscles deterioration of action potentials in response to repetitive nerve stimulation was observed. Similar observations were also made in spontaneous discharges of action potentials in denervated muscle fibres, except that in the latter case the process of deterioration could be observed in its entirety and was found to be reversible (Fig. 10). In the experiment shown in Fig. 10 the spike potential, after its repetitive occurrence for several seconds, gradually evolved into two small potentials of identical shape and size. For a period the record appeared to consist of paired discharges.

Later the pair began to merge into a large potential with a 'notch' at its peak. With the disappearance of the notch the potential resumed its original size and shape. No significant change in the resting potential was detected. These observations would indicate that deterioration of a spike action potential is not irreversible and the amplitude of a deteriorating spike action potential may not be associated with a change in the resting potential.



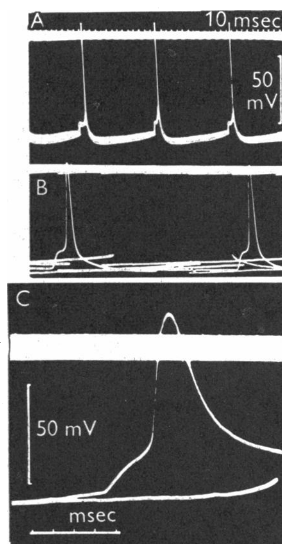


Fig. 8 Fig. 9

Fig. 8. Small potentials and spike potentials recorded from three different muscle fibres. A shows the independent occurrence of these two potentials; B, occurrence of spike potential in the rising phase of small potential; C occurrence of small potential in the falling phase of spike.

Fig. 9. Small potentials preceding spike potentials. A, slow sweep speed; B and C recorded from two fibres with different sweep speeds; time marks, 10 msec in A and 1 msec in C; voltage calibration, 50 mV.

Membrane resting potentials

In the course of this study resting potentials of 325 denervated and 111 innervated muscle fibres were measured. Resting potential in fibrillating muscle fibres appeared to remain unchanged after the muscle had been denervated. Thus, the mean value was found to be $61 \pm 16{\cdot}4$ mV, while the mean value in normal controls was $61 \pm 4{\cdot}2$ mV. There was no appreciable difference in resting potential measurements obtained from experiments on different days after denervation. In view of the wide range of these measurements, which accounted for the high standard deviations ($\pm 16{\cdot}4$ and $\pm 4{\cdot}2$ mV), and of the fact that these measurements were relatively low compared to those,

reported by Trautwein, Zink & Kayser (1953) in guinea-pigs and cats, Bennett Ware, Dunn & McIntyre (1953), Ware *et al.* (1954) in mice, and Nicholls (1956) in frogs, questions were raised of a possible error resulting from junction potentials created at the electrode tips (del Castillo & Katz, 1955; Adrian, 1956). In fact, some of our records occasionally showed a small resting potential (20 mV) with action potentials exceeding the zero membrane potential by 50–60 mV, or a large resting potential (130 mV) with an 'overshoot' of the

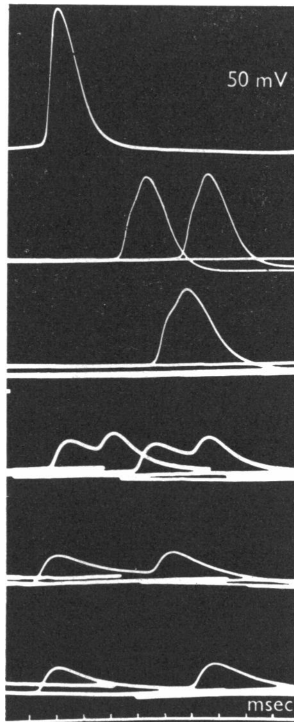


Fig. 10. Progressive change of spontaneous fibrillation potentials. This process was found to be reversible; during recovery the potentials followed a similar sequence of changes, in the reverse order.

action potential of less than 5 mV. In these examples it is conceivable that the apparent resting potentials were substantially decreased or increased by a 'tip potential' effect.

Consequently, using electrodes with a junction potential (measured in Ringer's solution with a symmetrical recording system) of less than 5 mV and resistance of 3–10 MΩ, experiments for the determination of membrane resting potentials were repeated. It was frequently found that when the electrode was brought into contact with the muscle the resistance increased with a concomitant change in potential at the tip of the recording electrode.

If the electrode was then inserted further and was located inside a muscle fibre, the resistance of the microelectrode would again decrease to its original value. In some experiments fluctuation of the electrode resistance would occur even though the electrode was constantly kept inside a fibre, suggesting that the 'tip potentials' might be continuously changed as long as the micropipette was in an electrolyte medium with constituents carrying electrical charges, as pointed out by del Castillo & Katz (1955) and Adrian (1956). After taking precautions against this error, the membrane potentials determined again showed no significant difference in the muscles before and after denervation, as shown in Table 1. It has been noted (Nastuk & Hodgkin, 1950; del Castillo & Katz, 1955; Frank & Fuortes, 1955) that when a micropipette is inserted into the depth of living tissues, large potentials may develop at its tip and therefore greatly obscure the measurements of membrane potentials.

TABLE 1. Membrane resting potentials of rat skeletal muscle *in vivo*

	No. of fibres examined	Resting potential	
		Range (mV)	Mean (mV)
Innervated muscle	55	64–99	72·2 ± 5·3
Denervated muscle	75	55–98	73·0 ± 10·3

It is perhaps for this reason that the values of membrane potentials measured in this research, which required blind searching for fibrillation potentials from the depth of muscle, presented a much wider distribution than those obtained by others who selected fibres from only the superficial layer of muscle.

DISCUSSION

Since most penetrations were made in the depth of the muscle, it was impossible to locate end-plates; nevertheless, when the animal was curarized and the nerve stimulated, the relatively small potentials sometimes recorded within a muscle fibre would suggest that occasionally the recording microelectrode was located close to the terminal axon of the motor nerve. Since the refractory period for the small potentials could be established by twin shocks, it would appear that the responses arose from the same source. Thus, the small potentials of mammalian skeletal muscle fibres seem similar to end-plate potentials of the frog's sartorius muscles. This is in agreement with the observations of Boyd & Martin (1956).

However, in some of our experiments with denervated muscle fibres large spike potentials were frequently seen to be intermingled with small potentials. Their occurrence was independent and the discharge of the one could occur during the refractory period following the other. The small potentials intermingling with large spikes should not be interpreted as delayed propagation of action potentials of the muscle fibre, for such a potential was characterized by a 'notch' and paired discharges before evolving into two small potentials

of identical shape and size. If conduction of the surface membrane was interrupted, excitation of one segment of the fibre did not necessarily affect the activity of the other and the microelectrode recording from the blocked region was capable of detecting potential changes on the two sides. This implies that there were multiple foci in a single muscle fibre from which the fibrillation potentials could be initiated, although at any given time the activity of many of these foci might have been subthreshold.

Whether or not these foci corresponded to the end-plate regions is undetermined; however, multiple end-plates in a single muscle fibre which were derived from the same parent axon have been demonstrated by anatomical methods (Kulchitsky, 1924; Denny-Brown & Pennybacker, 1938; Feindel, Hinshaw & Weddell, 1952), and it is possible that denervation leads to their becoming hyperexcitable or spontaneously active.

It was demonstrated that the membrane resting potentials of denervated muscle fibres tended to oscillate. The instability of membrane potential, which was not observed in innervated muscle fibres, may indicate metabolic changes in denervated muscles, as evidenced by the studies of Humoller, Griswold & McIntyre (1950) and Humoller, Hatch & McIntyre (1951).

Membrane resting potentials of skeletal muscle of mice were determined *in vivo* by Bennett *et al.* (1953). The measurements given by these authors do not agree with those of guinea-pigs and cats reported by Trautwein *et al.* (1953). These discrepancies could be attributed to the difference in experimental animals, but if the criteria for the selection of measurements are not consistent, the results inevitably will be different. Because of the possible error produced by junction potentials at the electrode tips, it is inadvisable to consider the high values obtained as necessarily nearest to the true membrane potential measurements (del Castillo & Katz, 1955). For similar reasons it seems equally inadvisable to reject measurements of relatively low values. Until the technique and criteria for the determination of membrane resting potentials are standardized, different results reported from different laboratories are to be expected. In the meantime, our figures are closer to those of Trautwein *et al.* (1953) than to those of Bennett *et al.* (1953).

SUMMARY

Attempts to determine the origin of fibrillation potentials recorded from rat's skeletal muscle with microelectrodes have led to the following results and conclusions:

1. In curarized animals stimulation of the motor nerve elicited small potential responses from innervated muscle fibres similar to end-plate potentials. These small potentials were found to be 'all-or-none', and to have an absolute refractory period of about 2 msec.

2. The excitability of motor unit discharges was determined and the recovery of a motor unit subsequent to a stimulus to the motor nerve was found to be of the order of 10 msec.

3. Fibrillation potentials were sometimes recorded intermingling with small potentials, suggesting that there are multiple foci from which fibrillation potentials could be initiated, although at any given time the activity of many of these foci might be subthreshold.

4. That the origin of fibrillation potentials is in denervated single muscle fibres was established by means of intracellular recording. Whether or not these potentials are initiated by isolated foci other than the end-plates remains to be determined.

5. The membrane resting potentials of denervated muscle fibres tended to oscillate, frequently in a rhythmic manner, which might not precipitate an action potential. Spike discharge was initiated only when the underlying potential reached a critical level.

6. Membrane potentials of the rat skeletal muscle *in vivo* were found to be $72{\cdot}2 \pm 5{\cdot}3$ mV. There was no significant statistical change after denervation.

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