J. Physiol. (1938) 93, 285–300 612.817:598.6

NEURO-MUSCULAR CONDUCTION IN THE FOWL

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(*Received* 26 *May* 1938)

Avian muscle has been known for many years to differ in several respects from mammalian muscle, particularly in its high sensitivity to substances having a nicotine-like action. In the course of experiments on these reactions, we found that it differed from mammalian muscle also in its responses to nerve stimulation. We have been unable to find any comprehensive account of the normal behaviour of the avian nerve-muscle preparation, especially in connexion with its action potentials, with which this paper is mainly concerned. The chief differences from mammalian muscle have been found in the effects of initial tension on the twitch response, and in the behaviour of the muscle excited with two nerve volleys. We believe that we have obtained satisfactory evidence to show that certain fibres of an avian muscle require the peripheral summation of two nerve impulses before a propagated disturbance is set up in them. A preliminary account of these experiments has already appeared in this *Journal* [Brown & Harvey, 1938].

Methods

In all the experiments we have used domestic fowls. In most respects white Leghorn hens of 1·5–2 kg. have proved to be most satisfactory, especially for experiments of long duration, as they appear to suffer less from respiratory difficulties. In all experiments anæsthesia was induced and maintained by intravenous injection of 10 p.c. "Pernocton" solution. The initial dose of 0·3–0·5 c.c. per kg. was injected slowly, usually over a period of 5 minutes into a wing vein. Anæsthesia comes on during the injection, and care must be taken to stop the administration as soon as the neck muscles become relaxed. The margin between full anæsthesia and a fatal stoppage of respiration is narrow, and the respiratory movements must be watched with care during the administration of the anæsthetic. The initial dose maintains anæsthesia for about $\frac{3}{4}$ hr., after

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which further injections of approximately 0·1 c.c. per kg. may be made into a cannula in the jugular vein whenever necessary. Capons are to be avoided, as they are extremely sensitive to the anæsthetic. In the experiments in which curarine was employed, the animals were first anæsthetized with "Pernocton" and the muscles prepared for recording. Immediately before the administration of curarine, a string was tied tightly round the neck, just below the head, artificial respiration was started, and the brain and medulla were destroyed by thrusting a bradawl into the foramen magnum.

The muscle used for recording in the majority of the experiments was the lateral head of gastrocnemius, which was prepared as follows. The feathers were plucked from the leg and thigh, and the skin was incised along the posterior aspect of the leg from the tarsus to the upper end of the femur. The tendon of gastrocnemius was detached just proximal to the tibio-tarsal joint, and the external head of the muscle was separated from the middle head and from the muscles covering the tibia, as far as the knee joint. The vastus externus and the semimembranosus and semitendinosus were retracted, exposing the sciatic nerve and blood vessels which lie on the posterior surface of the femur. The transversely lying muscle fibres, which cover the nerve and vessels, just behind and above the knee joint, were carefully incised and the nerve and vessels dissected from each other. The nerve was cut some 5 cm. proximal to gastrocnemius, and all branches, other than the twig supplying the lateral head of gastrocnemius, were cut.

The muscle and nerve were now covered with hot moist cloths, and drills were inserted in the upper end of the femur, the condyles of the femur and into the lower end of the tibia. These provided a means of fixing the leg rigidly to the myograph base. The preparation was completed by tying a steel hook, with an insulating segment, into the tendon.

The methods used for stimulation and recording were the same as those previously described for cats [see Brown & Euler, 1938, and previous papers from this laboratory]. In a number of experiments, we have recorded the electrical responses of a muscle excited by directly applied induction shocks. For this purpose, the gastrocnemius could not be used, as we found it impracticable to excite such a large muscle maximally with a single shock. We therefore used the flexor perforatus indicis secundus pedis, a flattened muscle about 3 cm. long, which lies beneath the origin of the external head of gastrocnemius, and receives a branch from the nerve to gastrocnemius. The stimuli were break induction shocks from coreless coils, and were applied to the muscle by silver pins

thrust into its substance. The action potentials were led by small calomel half cells to a two-stage D.C. amplifier and thence to a cathode-ray oscillograph.

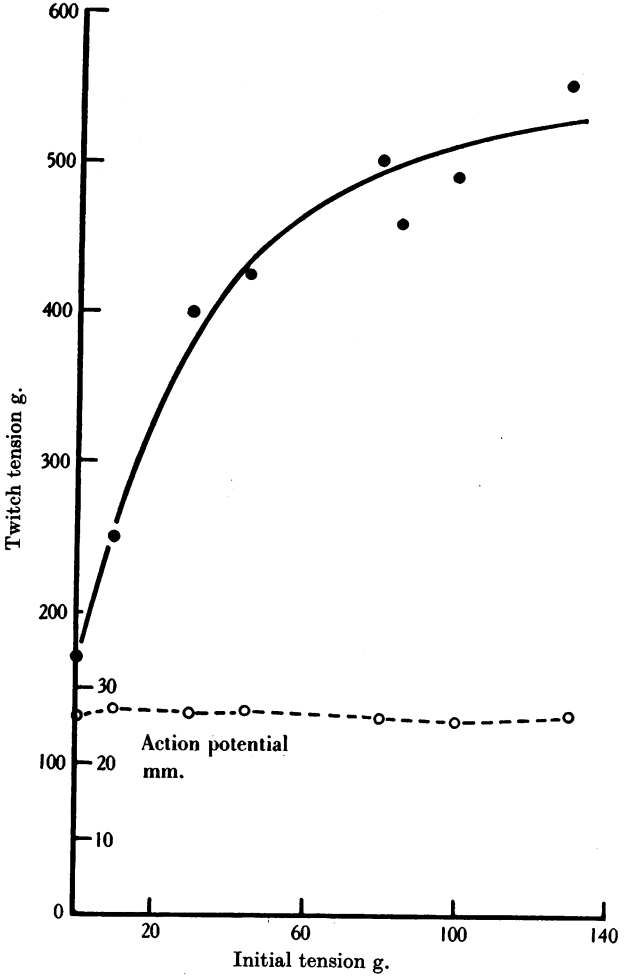

Fig. 1. Curves showing effect of initial tension on twitch tension (●) and action potential (○) of fowl's gastrocnemius.

RESULTS

(*a*) *The effect of initial tension on the isometric twitch*

The tension response of the fowl's gastrocnemius to single maximal motor nerve volleys is profoundly influenced by the initial tension applied to the muscle. Fig. 1 illustrates this phenomenon. With the initial

tension zero, the twitch tension was 170 g. Increase of the initial tension to 130 g. caused an increase in twitch tension to the high value of 550 g. These changes are many times greater than those produced in frog's and mammalian muscle by alterations in initial tension. Fig. 1 also shows the amplitude of the action potentials simultaneously recorded. It will be noted that there is a small random variation, which does not exceed 5 p.c. This finding is of importance in connexion with the observations we have made on the amplitude of the action potentials of the responses to two successive nerve volleys (*vide infra*), since Fulton [1926] described increase in the action current of muscle, recorded with the string galvanometer, when the initial tension was increased. It is clear that, with the methods of recording we have used, the action potential of the fowl's muscle is not significantly affected by the initial tension, in spite of the great effect on the tension of the mechanical twitch.

(*b*) *Response of the muscle to two nerve volleys*

The muscular summation curve. When two volleys are set up at the same point in the motor nerve, muscular summation first appears when the stimuli are 0·8–1·0 msec. apart. As the stimuli are separated in time

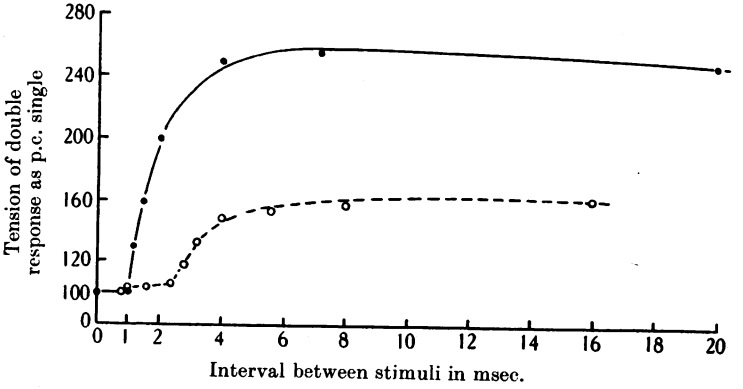

Fig. 2. Curve showing summation of muscular response to two nerve volleys in the fowl's gastrocnemius (●) and cat's tibialis anterior (○).

the tension curve rises very steeply, and at 1·2 msec. the combined tension may be 250 p.c. of the single response (Fig. 2). Further separation of the stimuli yields only a very small increase in tension, the maximum tension being reached with an interval of 4–5 msec. This description applies only to fresh muscles; as an experiment proceeds, the

curve relating summated tension to stimulation interval is flattened at the shorter intervals, and slowly reaches its maximum at an interval as long as 10 msec. In the same figure is shown a similar muscular summation curve derived from the cat's tibialis anterior. The ratio of the tension of a double response of the bird's muscle to the tension of a single response has varied in different experiments between 2·4 and 3·7. These values are greater than those normally found in mammalian muscle, with the exception of the internal rectus of the eye [Cooper & Eccles, 1930].The difference is probably due largely to the effects of initial tension, described above, since the second volley excites fibres which are already beginning to develop tension. We believe, however, that this does not entirely account for the high ratio, and that the entry into contraction of additional fibres, which have not responded to the first volley, as described in the next section, also contributes to the effect.

Action potentials accompanying the responses to two volleys. When the muscle is excited by two maximal nerve volleys set up at intervals less than 150 msec., the action potential accompanying the second muscular response is regularly greater than the first. Fig. 3 shows a series of responses at intervals of 18, 40, 90 and 200 msec., and Fig. 4 shows the results of a similar experiment plotted graphically. As soon as the action potentials become discrete, i.e. at about 20 msec. interval, the second may be as much as 25 p.c. greater than the first. With further separation, the disparity declines along a smooth curve, and, at intervals greater than 150 msec., the two become and remain equal. This difference between the first and second responses can be seen with tetanic stimulation at suitable frequencies.

There appear to be four possible explanations of the increased amplitude of the second action potential: (*a*) increase in action potential due to change in initial tension of the muscle; (*b*) closer synchronization of the response of the individual fibres of the muscle as a result of previous activity; (*c*) a property inherent in the muscle fibre, analogous to "Treppe" whereby a second excitation wave is accompanied by a greater action potential; and (*d*) the contraction of fibres additional to those excited by the first volley. The possibility that local effects in the nerve are responsible for the phenomenon has been excluded by delivering the stimuli, in some experiments, through two separate pairs of electrodes, some 2 cm. apart on the nerve. The first volley was set up by the pair distal to the muscle and the second through the proximal pair. Under these conditions the phenomenon occurs just as well as when both volleys are set up through the same electrode.

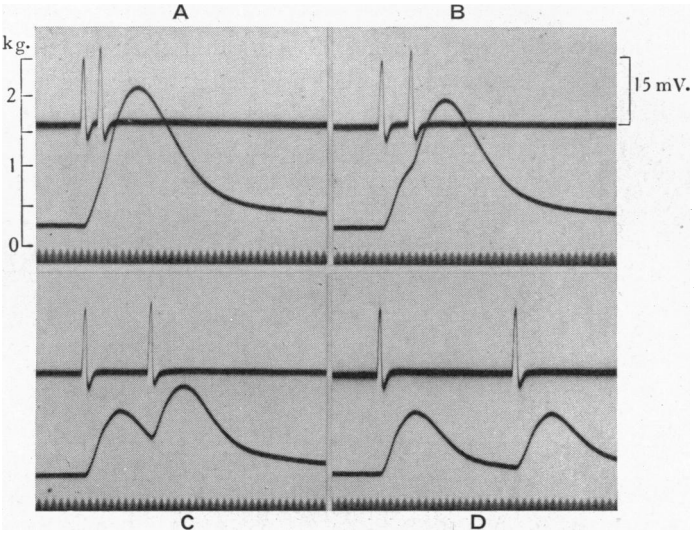

Fig. 3. Hen 1·9 kg. Myograms and action potentials of gastrocnemius in response to two maximal nerve volleys (A) 18, (B) 40, (C) 90 and (D) 200 msec. apart. Time 10 msec.

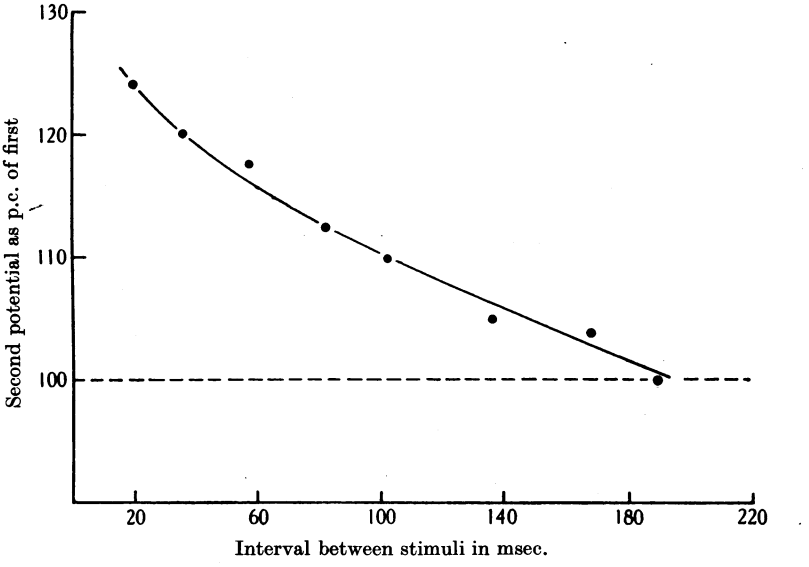

Fig. 4. Curve showing relation between size of second action potential and interval at which it follows the first potential.

(*a*) This explanation is adequately excluded by the experiments described above, in which increases of initial tension, nearly as great as the tensions evoked by a single nerve volley, were accompanied by changes in action potential not greater than 5 p.c.

(*b*) We have no evidence that the second response shows less temporal dispersion than the first; in fact, the second potential wave is often of longer duration than the first. Fig. 5 shows two responses of the flexor perforatus muscle to two nerve volleys 10 msec. apart; the second response is 21 p.c. greater in amplitude than the first and 17 p.c. greater in duration.

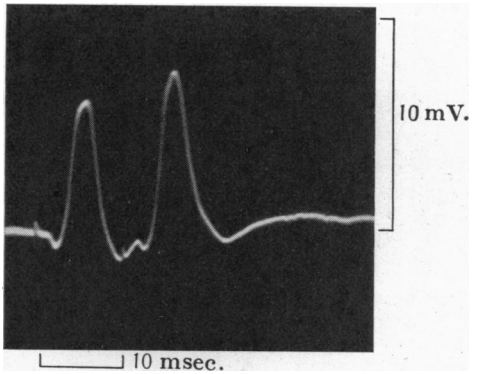

Fig. 5. Hen 2·5 kg. Action potentials of flexor perforatus muscle in response to two maximal nerve volleys 10 msec. apart. D.C. amplifier and cathode-ray tube.

(*c*) When avian muscle is excited by directly applied induction shocks, the second response is not greater than the first. Fig. 6 shows (A) the responses of the flexor perforatus muscle to two stimuli through the nerve; (B) to two stimuli applied directly to the muscle 1 cm. distant from the recording electrodes; and (C) to two stimuli applied 2 mm. from the recording electrodes. The response to nerve stimulation shows the characteristic difference in the amplitude of the action potentials, the second response being 12 p.c. greater than the first. When the stimuli are applied directly to the muscle, but at some distance from the recording electrodes, the fibres from which the action potential arises are mainly excited through the nerve, since the fibre length in this muscle is not more than 8 mm. Under these conditions, the difference between the first and second action potentials is still evident. In Fig. 6 B the second response is still 6 p.c. greater than the first. When, on the other hand, the stimuli are applied sufficiently close to the recording electrodes for

the excitation to include all the fibres from which the recording electrodes are leading, the second response is only 93 p.c. of the first, Fig. 6C. Since the stimulation artefact is of almost equal magnitude in B and C it is unlikely that any essential differences in size between the first and second responses have been obscured by it.

(*d*) We are therefore left with the probability that the increase of the second action potential is due to the contraction of additional muscle fibres. It would appear that certain fibres of the bird's muscle cannot be excited by a single nerve impulse, and that such an ineffective impulse leaves behind it a period of raised excitability of the muscle end plate, which enables a second incident impulse to act as an effective stimulus.

In connexion with the experiments on direct stimulation of muscle,

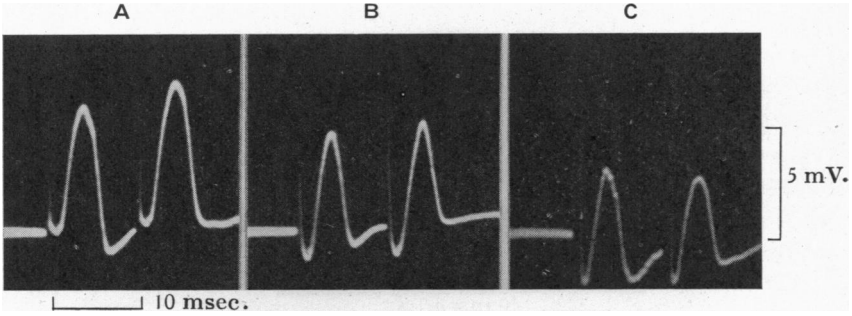

Fig. 6. Hen 2·5 kg. Action potentials of flexor perforatus in response to two stimuli 10 msec. apart applied A to the nerve, B to the muscle 1 cm. from recording electrodes, and C to the muscle, 2 mm. from recording electrodes. D.C. amplifier and cathode-ray tube.

we attempted to record the electrical response of directly stimulated muscle after degenerative section of its motor nerve. The mechanical response of such a muscle is noticeably slower, in all its phases, than that of the innervated muscle when directly stimulated; this slowing is associated with a very great temporal scattering of the electrical response. So great is this dispersion, that the action potential consists of an irregular and inconstant wave, many times longer in duration than the normal potential, and consequently completely unsuitable for accurate measurement of its amplitude.

In most of the experiments on the effects of two volleys, the muscle has been covered with a cellophane bag and warmed by an electrical heater with a bowl reflector, placed about 1 metre away. Under these conditions, and with the muscle in the vertical position for recording, its temperature is materially lower than in the body. In one experiment,

therefore, we enclosed the whole bird in a heating cabinet, and measured the muscle temperature with a thermocouple. Increasing the muscle temperature from 29 to 36° C. did not produce any significant change in the ratio of the first to the second electrical response to two volleys 25 msec. apart. The normal temperature of the leg muscles in the unæsthetized hen is, indeed, approximately 40° C., and we did not succeed in raising the temperature of the exposed, experimental muscle to this level. But we regard the fact that, with increase of the muscle temperature from 29 to 36° C., the disparity between the electrical responses to the two stimuli remains unaltered, as a strong indication that it represents a normal characteristic of the muscle, under natural conditions.

Response to tetanic stimulation. The frequency of tetanic stimulation producing a fully fused tension response in the fowl's muscle is of the order of 60 per sec., a frequency not greatly different from that required by corresponding muscles of the mammal. As might be expected from the summation curve with two volleys, the ratio of twitch tension to full tetanic tension is high, in the region of 8. Cooper & Eccles [1930] give values between 3·26 and 4·95 for this ratio in mammalian muscles. The internal rectus of the eye, with a twitch-tetanus ratio of 10·7, is the only mammalian muscle which has a ratio resembling that found for avian muscle.

As mentioned above, the action potentials of avian muscle during a tetanus show the disparity between the first and second responses observed with two volleys. At frequencies of stimulation of 50 per sec., when the potentials are discrete, the second action potential is regularly larger than the first; the third and subsequent responses are of the same size as the second, until the decline develops, which normally ensues in a prolonged stimulation. The diminished responses at the end of a long tetanus are considerably prolonged, and their small amplitude is probably attributable to temporal dispersion of the responses of the individual fibres.

Effect of a preceding tetanus on the response to one and two volleys. In the bird, as in the mammal [cf. Brown & Euler, 1938], a tetanus evokes a long lasting increase in the twitch tension Fig. 7. It will be noted in this figure that the responses to the single volleys which precede the tetanus are somewhat irregular. This phenomenon we have observed in most experiments, and figures given by van Dijk [1934] show the same effect. A similar irregularity has been seen in the tension response optically recorded, and in the action potential with single volleys. Such an irregularity is to be expected if a single volley fails to excite all the fibres of the

muscle, since very slight variations in the thresholds of individual fibres will throw them in or out of action, in the responses to successive volleys. The potentiated twitches immediately succeeding the tetanus show, on the other hand, a regular decline, the usual irregularities of twitch tension being absent during the period of potentiation. This suggests that a tetanus is followed by a period of raised excitability of the motor end plates, like that observed in the partially curarized mammalian muscle, which enables a single nerve volley to excite all the muscle fibres. It was, accordingly, to be expected that the normally observed increase in the

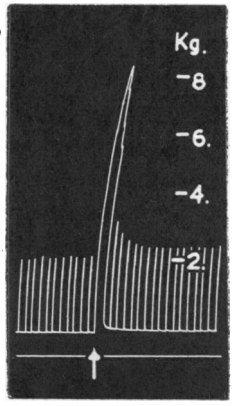

Fig. 7.

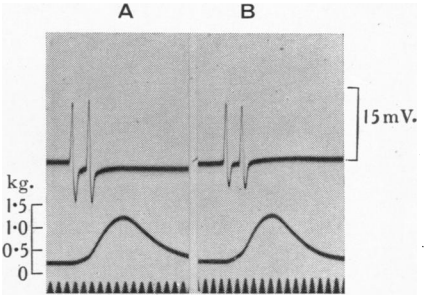

Fig. 8.

Fig. 7. Hen 1·6 kg. Tension response of gastrocnemius to maximal nerve volleys at intervals of 10 sec. At arrow, motor nerve tetanus at 50 per sec. for 10 sec.

Fig. 8. Hen 1·5 kg. Myograms and action potentials of gastrocnemius in response to maximal nerve volleys 20 msec. apart, A, before and B, 280 msec. after a tetanus at 30 per sec. for 1·0 sec. Time, 10 msec.

second response to a pair of stimuli would, after a tetanus, be absent. This proved to be the case. Fig. 8 shows the electrical responses of the fowl's gastrocnemius to two maximal volleys, 20 msec. apart, A before, and B 280 msec. after a tetanus at 30 per sec. for 1·0 sec. In the original record, the negative deflexion of the first response before the tetanus was 12·7 mm., and of the second 14·7 mm., a difference of 16 p.c. After the tetanus, the first response was 12·8 mm. and the second 12·3 mm. The tetanus has, therefore, abolished the normal enhancement of the second response, and replaced it by a small (4 p.c.) diminution.

We stated above that the increase size of the second response was probably attributable to the entry into contraction of additional fibres, which did not respond to the first volley, and we suggested that a tetanus

would lower the threshold of all the muscle end plates for a longer period, so that the first volley, following within a few seconds after a tetanus, should excite all the muscle fibres. On this assumption, it might be expected that the first electrical response to a pair of volleys after a tetanus should have the same amplitude as the second to the pair immediately preceding the tetanus. In the experiment quoted above, however, the first action potential of the post-tetanic pair is no greater than the first of the pre-tetanic pair, suggesting, at first sight, that the first post-tetanic volley is still, in part, subliminal, exciting only about the same number of muscle fibres as the first pre-tetanic volley. We are convinced that such an interpretation would be incorrect, and that the amplitude of the electrical response to the volley following the tetanus is reduced, not because all fibres are not responding, but by the interference of another effect. Brown & Euler [1938] have recently shown that the potentiated twitches, which follow a tetanus in mammalian muscle, are accompanied by smaller action potentials than the normal twitches preceding the tetanus, and that this diminution in action potential is the result of a depolarization of the muscle, and not a consequence of a fall in the number of muscle fibres responding. It could in any case be regarded as probable that this reduction of the post-tetanic electrical response by depolarization would occur in avian as in mammalian muscle. There is, moreover, evidence that it does so. Table I shows the changes

TABLE I

Action potentials before tetanus (mV.)				Action potentials after tetanus (mV.)		
1st	2nd	2nd as p.c. 1st	Frequency tetanus per sec.	1st	2nd	2nd as p.c. 1st after tetanus
22·7	25·8	114	3	21·7*	24·2*	112*
21·6	25·7	119	30	22·1	21·1	96
23·1	26·3	114	50	21·7	21·7	100
21·2	25·7	122	240	21·3	22·1	104
22·4	25·6	114	600	21·7	21·7	100
22·2	25·8	117		21·7	21·7	100

Diminution of 1st produced by tetanus 2 p.c.
,, 2nd ,, 16 p.c.

* These figures are omitted from mean values of post-tetanic pairs as the tetanus was of too low a frequency to produce its characteristic effect.

produced by tetanus in the electrical responses to pairs of stimuli. We have reason to believe, as stated above, that the second response of a pair represents a contraction of all the fibres of the muscle; if, therefore, the second responses of pairs are compared before and after a tetanus they should give a clear indication of the degree of depression of the total action potential by the tetanus, uncomplicated by any change in the number of

fibres involved in the response. Thus, in Table I, the second responses of post-tetanic pairs are 16 p.c. less than the second responses of the pre-tetanic pairs. At the same time, the *first* responses of the post-tetanic pairs are only 2 p.c. less than the corresponding first pre-tetanic responses. This suggests that the tetanus has increased the number of fibres responding to the first volley by 14 p.c. Before the tetanus, the second responses were 17 p.c. greater than the first responses; after the tetanus

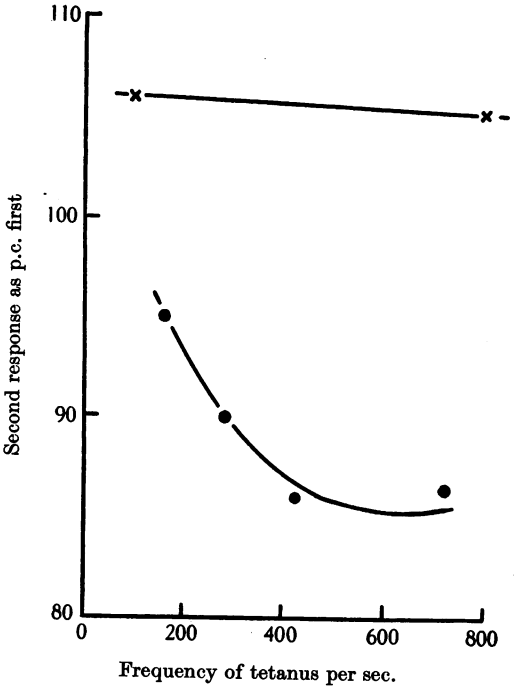

Fig. 9. Curve showing relation between frequency of tetanus and size of second response of a subsequent pair of responses 45 msec. apart (●). The size of the second responses before the tetani are shown above (×).

the two responses are approximately equal. It would appear, therefore, that the tetanus has so increased the first response that it involves nearly all the fibres of the muscle, but the increased action potential which would normally accompany the entry into activity of these additional fibres has been masked by the 16 p.c. reduction in the total action potential, produced by the post-tetanic depolarization. We conclude, therefore, that a tetanus is followed by a short period of raised excitability of the motor end plate, which enables a single nerve impulse to excite fibres which

previously required the incidence of two impulses to bring them into activity.

It will be noted in Table I that the second of the pair of responses after the tetanus at 30 per sec. is only 96 p.c. of the first. In certain experiments this effect has been very prominent and the second response after the tetanus has been regularly less than the first. There appears, moreover, to be a relationship between the frequency of the tetanus and the degree of depression of the second response. Fig. 9 shows graphically the results of one experiment. Before tetanic stimulation, the second response of a pair 40 msec. apart was between 5 and 7 p.c. greater than the first. After tetanization at 180 per sec. the second response was 5 p.c. less than the first and the difference between the two increased as the frequency of the tetanus increased. The significance of this finding will be considered in the discussion.

Effect of drugs on response to two volleys. On the supposition that adrenaline might exert some influence on muscle end-plate excitability, we studied in a few experiments the effect on the response to two volleys of adrenaline administered by intravenous injection. In doses up to 150 γ per kg., adrenaline has very little effect on the disparity between the action potentials in response to two volleys, 25 msec. apart. There was a suggestion that the responses approached a little nearer to equality through increase in the size of the first response, but the change was inconstant and of doubtful significance. It is possible that stimulation of the sympathetic supply of the muscles might have yielded more conclusive results, but we found that such stimulation was not feasible in the bird.

As might be expected, from its effects in the mammal [Brown, 1938], curarine, in doses insufficient completely to block neuro-muscular conduction, greatly exaggerates the disparity between the action potential responses to a pair of nerve volleys. Thus, in one experiment, on the flexor perforatus muscle, the second action potential of a pair set up 10 msec. apart was 9 p.c. greater than the first before curarine; after intravenous injection of 1 mg. of curarine the second potential was 68 p.c. greater than the first.

Discussion

The experiments on fowl muscle described above have revealed certain very characteristic differences between its responses to nerve stimulation and those of the muscle of mammals. The large effects of initial tension on the twitch tension are probably associated with the particular postural and phasic functions of the muscle we have examined, and we are

unaware whether they apply to other muscles of the bird. The initial tension effect can introduce a very serious source of error into experiments on these muscles, and must be controlled with exactitude if reproducible responses are desired.

The most striking difference between avian and mammalian muscle is seen in their electrical responses to two closely following maximal nerve volleys. In mammalian muscle such responses are equal; in other words, no more muscle fibres are excited by the second volley than by the first. In the bird, the second response is greater, not only in amplitude, but in duration. The neuro-muscular transmitting apparatus is certainly involved in this effect, since the electrical responses of muscle directly excited with two maximal shocks are equal, or, when a difference occurs, the second is smaller in amplitude than the first. We have excluded other possible causes of the phenomenon, and are left with the probability that the increase in the second action potential is due to the fact that the second volley has excited additional muscle fibres, which failed to respond to the first volley. Complete proof of this assumption could be provided only by showing that the additional fibres of the muscle could respond to a directly applied stimulus, during the refractory period of the nerve and of the fibres responding to the first volley. The difficulty of detecting a small increment of potential, masked by a large stimulus artefact and by the temporal dispersion among the fibres of a fairly large muscle, which alone precludes accurate timing of the second stimulus, has, however, so far prevented us from demonstrating this.

The phenomenon of the augmented second action potential has been observed by Samojloff [1908] and by Adrian & Lucas [1912] in freshly isolated frog's muscle, stimulated through its nerve, and it appears to differ only in its time relations from the effects we have observed. In the fowl, as in the frog [Bremer, 1927], the effect is greatly exaggerated by small doses of curarine, the use of which substance is necessary in the mammal to elicit the effect at all [Brown, 1938].

The effect of a tetanic stimulation in producing a longer increase in end-plate excitability, causing the first response to a post-tetanic volley to approach maximality, is obviously very closely allied to the relief of a partial curare paralysis by a similar tetanus [Boyd, 1932; Brown & Euler, 1938].

In the description of the experiments on the effects of tetani we have referred to the difficulties of interpretation of the size of the action potentials following a tetanus, on account of the reduction in the amplitude of a maximal electrical response, accompanying the post-tetanic

mechanical potentiation. In the experiments shown in Table I this effect of a tetanus could be controlled by comparing the sizes of the second responses before and after the tetanus, on the assumption that they were maximal responses both before and after. A further complication is, however, introduced in some experiments by the finding that the second response after the tetanus is significantly smaller than the first, as shown in Fig. 9. This suggests, at first sight, that the second response has now become submaximal, in comparison with the now maximal first response. The validity of such an interpretation is, however, made doubtful by the fact that the second response of *directly* excited muscle is often less than the first (Fig. 6C). A similar reduction in the size of a second directly excited response has been described for frog's muscle by Adrian & Lucas [1912]. It would appear then that in amphibian and avian muscle, at least, the electrical change of a second response is less than the electrical change of a first response, *if the first response is maximal.* In the case, therefore, of the experiments referred to in Fig. 9, we must conclude that the depression of the second responses after a tetanus is attributable to the fact that they follow first responses, which have approached closer to maximality as the frequency of the preceding tetanus has increased. The cause of this depression of the action potential of a second response, following a maximal first response, is open to question. It is unlikely that it can be due to persisting refractoriness of fibres excited by the first stimulus, since in frog's muscle the depression may still be present at an interval of 50 msec., and in the fowl at an interval of 40 msec. A prolonged negative after-potential has been found by Brown & Euler to follow a tetanus, and this is accompanied by a reduction in subsequent single action potentials. The single response of frog's muscle is accompanied by a slow negative after potential which lasts, according to Schaefer [1936], some 50–80 msec. Adrian & Lucas [1912] show that the reduction in second response of directly stimulated muscle also lasts about 50 msec. (their Fig. 10). It seems, therefore, not unreasonable to attribute the reduction in second action potential to its falling in the period of negative after-potential following the first response. Normally in avian muscle, this reduction is completely obscured by the failure of the first volley to excite all the muscle fibres and by the increment of fibres participating in the second response.

We conclude that about 25 p.c. of the muscle fibres of the fowl's leg muscles fail to respond by contraction to a single nerve impulse. The ineffective impulse leaves behind it at the motor end plate a period of raised excitability which lasts some 150 msec., and a second impulse

reaching the motor end plate during this period is now effective in setting up a contraction. A period of tetanus leaves behind it a similar, but longer lasting period of enhanced excitability, during which single nerve volleys can excite all the fibres of the muscle, though the interference of the prolonged depolarization due to the tetanus prevents a corresponding enhancement of the electrical response.

SUMMARY

1. Optical isometric myograms and action potential records have been taken from the leg muscles of the domestic fowl anæsthetized with pernocton.

2. The initial tension to which the muscle is subjected has a much greater effect on the maximal motor nerve twitch tension than in the mammal.

3. When two maximal motor nerve volleys follow one another at intervals less than 150 msec., the action potential in response to the second is greater than the first.

4. This effect has been shown to be due to the fact that a single nerve volley fails to excite all the fibres of the muscle, but leaves behind it a period of raised excitability of the motor end plates. A second volley arriving during this period can thus excite the fibres which failed to respond to the first volley.

5. Brief tetanic stimulation of the motor nerve is followed by a long lasting increase in twitch tension and an increase in excitability, which enables a single subsequent volley to excite the muscle maximally.

6. Certain difficulties in the interpretation of changes in the amplitude of action potentials are considered in relation to the depolarization which follows a tetanus, and the slow potentials accompanying a single twitch.

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