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INTERACTION BETWEEN MUSCLE FIBRES IN A TWITCH

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It is simplest to suppose that with inertialess isometric recording the mechanical twitch of a whole muscle to a single maximal shock is the sum of all the elementary twitches to be recorded if each motor nerve fibre were excited individually one by one. In this paper, however, it is shown that this expectation is not exactly obeyed; when the muscle is excited synchronously by a single shock the resultant twitch is stronger and lasts longer than the sum of its components taken separately. This augmentation of the twitch only occurs if all the motor units are excited within a millisecond or so of each other; thus it is found that even slightly asynchronous excitation gives smaller and briefer twitches. These observations are of interest because all explanations in terms of known phenomena apparently fail.

A preliminary account has already appeared (Merton, 1951*a*).

METHODS

The arrangements for the human experiments have been described in detail in a previous paper (Merton, 1951*b*). The muscle was the adductor pollicis, action potentials led-off by surface electrodes and amplified, tension recorded by a strain gauge and direct-coupled amplifier. The ulnar nerve was stimulated at the wrist or the elbow by condenser discharges delivered through pulse transformers.

In the cat experiments the animal was under chloralose with its tibia fixed by drills through the bone. The tendon of tibialis anterior was connected to the strain gauge by a wire. Its nerve was dissected out for stimulating, unwanted branches being cut.

RESULTS

The effects of precise synchronization of the motor volley on the muscle twitch were brought to light by the observation that maximal twitches of the adductor pollicis muscle were different in shape when the stimulus was given at the wrist instead of the elbow. Investigation of this phenomenon showed that there is a sufficient length of nerve in the human forearm to allow the differences in conduction velocity among the fibres to disperse the volley

when it comes from the elbow, and this asynchronous volley gives rise to a smaller and briefer twitch. In animal experiments such lengths of nerve are not commonly employed, which is one reason why the phenomenon has previously escaped notice.

As compared with that from the wrist the twitch elicited from the elbow constantly has an earlier peak and the falling phase is more hollow (Fig. 1). The peak tension may be slightly greater or less than with stimulation at the wrist, consistently greater or less in any one experiment if care is taken that the position of the limb, etc., remain unchanged and the muscle is not fatigued. As will be proved, it is the asynchrony which causes the changes in

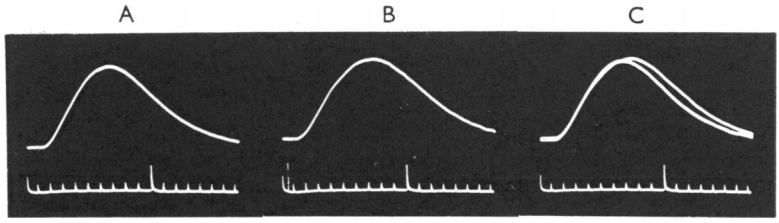

Fig. 1. Twitches of the adductor pollicis with a maximal shock to the ulnar nerve. A, stimulus at elbow; B, at wrist; C, the two superimposed; shock latencies adjusted to give coincident rising phases. The smaller and briefer twitch is with the volley from the elbow. Twitch height about 1 kg. Time markers, 10 and 100 msec.

shape and also reduces the size of the twitch. The reason the two twitches are nevertheless about the same height is that the elbow stimulus causes contraction of the ulnar muscles of the forearm, which jerks the hand in the splint and makes a small and inconstant contribution to the tension usually about enough to bring the asynchronous twitch up to size. These actions are revealed by experiments with double stimulation at wrist and elbow. To see the contribution of the forearm muscles the nerve is stimulated at the wrist and the effect noted of adding simultaneous stimuli at the elbow. Provided the wrist shock is maximal, changes in the twitch can only be due to muscles contracting in the forearm. Conversely, the synchronization effect accounts for changes in the twitch which occur when, during stimulation at the elbow, simultaneous stimuli at the wrist are thrown in. With both stimuli the twitch is bigger than with either separately which is because both factors tending to increase it are present, contraction of the forearm muscles and a synchronous volley from the wrist. The experiments which justify these statements will now be described in detail.

Contraction of forearm muscles. Maximal shocks are applied rhythmically at the wrist, one every 10 sec approximately. When a stimulus is applied simultaneously at the elbow (or a few milliseconds before or afterwards) the

mechanical response increases in size (Fig. 2A). There is no increase in the muscle action potential so the increase in the twitch cannot be accounted for by the wrist stimulus being unintentionally submaximal. The effect is absent unless the elbow electrode is over the ulnar nerve and unless the stimulus causes a large contraction of flexor carpi ulnaris. In a severe case of myasthenia gravis in whom the forearm muscles could be fatigued selectively a state was reached in which, with stimulation at the elbow, the adductor was contracting well but the twitch in the forearm muscles was practically impalpable. At that time the effect in question was absent—with double stimulation at wrist and elbow the contractions were identical with those from the wrist alone.

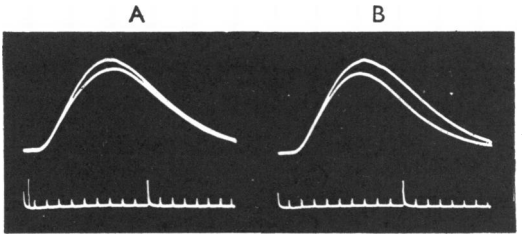

Fig. 2. Simultaneous double stimulation at the wrist and elbow. Double stimulation (the larger twitch in each case) compared with, A, the wrist only, B, the elbow only. Time markers, 10 and 100 msec.

This evidence strongly suggests that the increase in the twitch produced by switching in the elbow stimulus is due to contraction of forearm muscles, notably the flexor carpi ulnaris. It will be seen from Fig. 2A that the increase affects the different parts of the twitch similarly; the rate of rise, peak tension and rate of fall are all increased. There is little change in the shape of the response except that the peak may occur slightly earlier.

Synchronization of the motor volley. If contraction of the flexor carpi ulnaris were the only relevant influence introduced by moving the shock from wrist to elbow, we should expect the twitch elicited from the wrist always to be smaller than that from the elbow and to increase up to elbow size, but no further, with double stimulation. Double stimulation, however, gives a twitch that is bigger than with either separately, and it follows from this that there must be two factors tending to increase the twitch tension. One is the contraction of flexor carpi ulnaris, only present with the stimulus at the elbow. The other, which acts only with the stimulus at the wrist, remains to be investigated. For this purpose shocks are applied, at the usual 10 sec interval, to the elbow. Switching in a simultaneous shock at the wrist increases the tension and changes the twitch shape (Fig. 2B). This effect is absent if the electrode is not over the ulnar nerve; the shock must be nearly maximal for the adductor pollicis before it appears and does not increase if it is made

supramaximal. (When the wrist shock is half maximal the size of the twitch is actually reduced. It will be shown later that this surprising effect can also be accounted for in terms of a change in the synchrony of the volley but one independent of the increase under consideration at the moment.) These tests indicate that the action of the wrist shock in increasing the twitch is on the motor fibres to the adductor pollicis. The *action potential* following a motor volley from the wrist is slightly taller and briefer than when the nerve is excited maximally at the elbow. Both the larger action potential and the larger twitch could be explained if the wrist shock excited a few more motor fibres which the elbow shock did not. But great care was always taken to ensure that the elbow shock was maximal and, further, such an explanation could offer no reason for the change in duration of the action potential. The alternative is that all the motor fibres were in fact stimulated at the elbow, but owing to the greater distance they travel to the muscle all the impulses do not arrive at the same instant, the action potentials in individual motor units are therefore slightly out of step and do not sum so effectively; as a result the action potential of the whole muscle would be smaller and, owing to the dispersion, slightly prolonged. This would explain the differences in the action potentials, but could dispersion of the motor volley produced in this way also give rise to the changes observed in the mechanical response? An experiment was designed to answer this question. The principle is that if a shock at the elbow is followed by one at the wrist timed to fall just after the elbow volley of impulses has passed, but within the refractory period, the wrist shock should be quite ineffective, however strong, if it is true that exactly the same motor fibres are involved at both sites of stimulation. In the experiment illustrated in Fig. 3 the elbow shock was triggered by the same pulse that started the cathode-ray tube sweep. The wrist shock was set off after a variable delay. Either shock could be switched off. With each shock separately it was arranged by adjusting the delay that the early rising phases of both action potentials coincided on the tube face (Fig. 3A). With both shocks in circuit the potential was similar to that with the wrist shock alone. The action potential record shown in Fig. 3B has indeed the appearance of a single trace but inspection of the stimulus artifacts shows that there are two traces, both with delayed artifacts (wrist) and only one with an artifact at the start of the sweep (elbow). On increasing the delay about 1 msec the action potential with both shocks became indistinguishable from the elbow type. In Fig. 3C the action potential with double stimulation is compared with that from a single volley from the elbow. They are identical. On this record both traces carry the elbow shock artifact, but only one has the wrist artifact. At this critical interval the wrist shock, even if made as strong as the subject could bear, does not give rise to an action potential any larger than with the elbow shock alone. With slightly longer intervals some of the fibres have recovered

from the absolute refractory period and double stimulation results. So far, then, it has been confirmed that the greater height of the action potential with the wrist stimulus is due to its greater degree of synchrony. In the first experiment with the latencies equal, the fronts of the fastest action potentials from the elbow have just reached the wrist when the shock is sent in there.

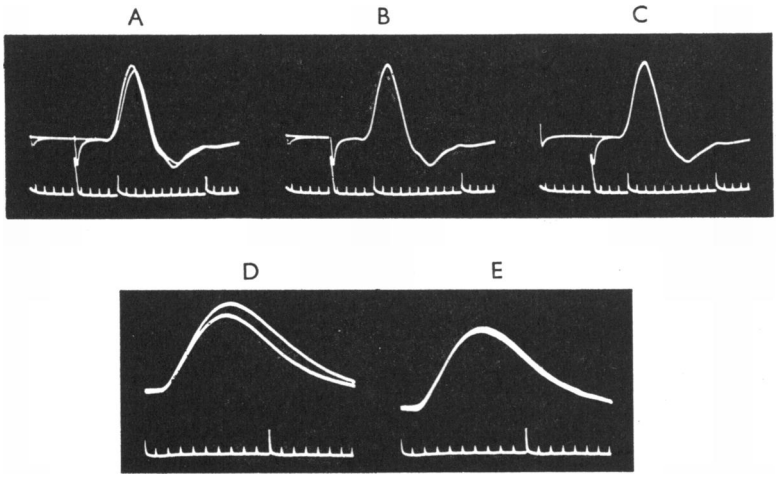

Fig. 3. Double stimulation at two intervals. A, action potentials from single stimuli at the wrist and elbow superimposed, with the early rising phases arranged to coincide (shock interval 5·2 msec). B, double stimulation with timing as in A compared with the wrist alone. C, double stimulation at the critical interval (6·0 msec, see text) compared with the elbow alone. Time markers in A, B and C, 1 and 10 msec. Action potentials approximately 10 mV peak to peak. D, twitch from double stimulation with interval as in A, compared with twitch from elbow stimulus alone. E, twitch from double stimulation at the critical interval also compared with that from the elbow alone. Time markers in D and E, 10 and 100 msec.

These fibres are unaffected but all the others are stimulated so as to come into synchrony. Thus the wrist type of volley is sent to the muscle. At the slightly longer critical interval the fronts of all action potentials have reached or passed the wrist. The shock thus falls on fibres which are either active or refractory and has no action. The asynchrony of the volley is unaffected and the muscle action potential is of the elbow type.

Turning now to mechanical twitches resulting from these various modes of stimulation, Fig. 3D compares the twitch due to a volley from the elbow with that from double stimulation at the shorter interval, i.e. with the wrist type action potential. Double stimulation, as previously observed, gives a larger twitch. (Fig. 3D is in fact a repetition of Fig. 2B.) With the shocks at the critical interval, however, i.e. with the elbow type action potential, the twitch reduces to the elbow type (Fig. 3E). Thus at the critical interval when the wrist shock cannot alter the synchrony of the volley it is also unable to

increase the mechanical twitch. It appears, then, that all the effects of adding the wrist stimulus are due to changes in synchronization, for if the shock is so timed that synchronization is unaffected, the mechanical response as well as the action potential is of the elbow type. The precise changes produced by improving synchronization are a higher and also a later peak, and flattening out of the hollow falling phase. The rate of rise of the first half of the rising phase is unaltered. Such effects are quite contrary to ordinary expectations, for whereas it is commonplace that a dispersed volley results in a smaller and broader action potential there seems no very good reason why the passage of slightly asynchronous action potentials over the fibres of a muscle should cause a considerable alteration in the mechanical twitch they trigger off.

Artificial desynchronization

The truth of the conclusions just reached can be confirmed in a simple and quite different way. Two stimulating electrodes are placed about 4 cm apart on the ulnar nerve above the wrist. Stimuli are applied simultaneously, that to the proximal electrode (furthest from the muscle) being maximal and always in circuit. Mechanical and electrical responses are the same, apart from a latency difference of about 1 msec, when there is either a maximal stimulus or no stimulus at all through the distal electrode. With a half maximal stimulus to the distal electrode a partial volley descends to the muscle. A similar volley ascends and meets a maximal volley coming from the proximal electrode. Mutual cancellation occurs except in those fibres which were not stimulated by the distal electrode. In those fibres, i.e. about half the total, a volley descends to the muscle and arrives about 1 msec after the first. The number of impulses in the motor volley is not altered, but by this manoeuvre they have been desynchronized into two half-volleys separated by 1 msec. The mechanical response is reduced in height and changed in shape, the reduction being greatest when the volley is divided into equal parts. The reduced twitch has an earlier peak and a quicker falling phase, but the initial rate of rise is the same (Fig. 4A). When a reduction is brought about by stimulating with a single submaximal shock (Fig. 4B) the shape of the twitch is little changed, the various parts being reduced in about the same proportion. At times the reduction in the twitch by desynchronizing has amounted to 30%. It is certainly very striking that an apparently so trifling alteration in the timing of impulses in a volley can have so large an effect on the mechanical outcome. The previous conclusions on volleys dispersed by traversing long stretches of nerve are thus fully substantiated, for it is extremely difficult to see how the present mode of stimulation could act except by desynchronizing, and the resulting alterations in the twitch are of the same nature as those found when the volley travels from the elbow instead of the wrist.

The same experiment has been performed on the tibialis anterior of the cat after all other muscles had been put out of action by nerve section. The stimulating electrodes were silver wires on which the nerve was laid under paraffin, the cathodes being 3–4 cm apart. The records obtained demonstrate more clearly than the human records the difference in shape of the twitch when it is reduced by desynchronizing, as compared with the roughly equal reduction of all parts with a submaximal shock (Fig. 5).

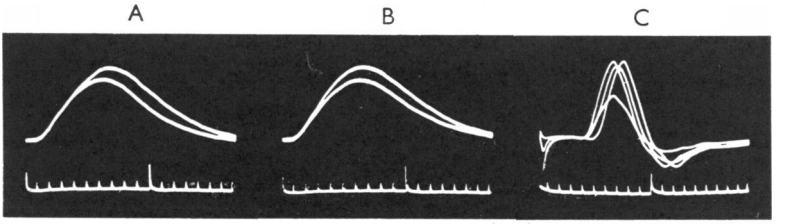

Fig. 4. Artificial desynchronization. A, twitch due to a single maximal shock compared with that following a desynchronized volley. B, maximal twitch compared with one reduced by making the single shock submaximal. Time markers for A and B, 10 and 100 msec. C, action potentials during artificial desynchronization, namely: maximal stimulus at distal electrode only, at proximal only, at proximal together with half-maximal at distal, this half-maximal stimulus alone. Time markers in C, 1 and 10 msec.

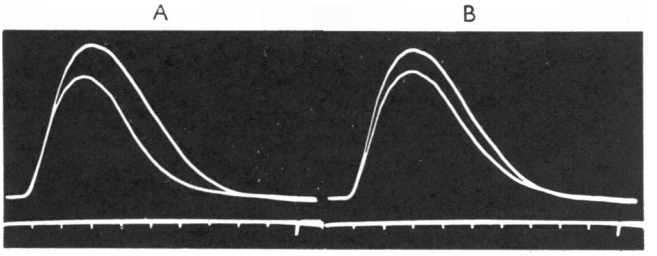

Fig. 5. Artificial desynchronization in the cat's tibialis anterior. A, maximal twitch and one reduced by desynchronization; B, maximal and submaximal twitches from single shocks. Time markers, 10 msec (faintly) and 100 msec.

The electrodes do not necessarily have to be close together in order to perform artificial desynchronization. With one at the elbow and one at the wrist the same effect is obtained by making the wrist stimulus half-maximal. This observation has already been referred to. Double stimulation at wrist and elbow can thus be a complex affair; during maximal stimulation at the elbow as the (simultaneous) wrist stimulus is increased in voltage the twitch at first decreases in size due to artificial desynchronization, then regains its original size and finally becomes larger than with the elbow shock alone because a maximal wrist shock improves the synchrony. It was consideration of this easily demonstrated but somewhat baffling sequence that first turned conjecture to the dispersion or otherwise of the motor volley.

If the two shocks are not put in simultaneously, but with the stimulus nearer to the muscle first lagging and then leading, it is possible to alter the

interval between half-volleys from zero up to a little more than twice the conduction time between electrodes, before double stimulation of some fibres occurs. When this is done it is found that the reduction of the twitch due to desynchronization is at its greatest when the volleys are separated by only 0·8 msec or even less. With electrodes at wrist and elbow the largest possible separation of half-volleys is about 10 msec, but as the interval is increased from 0·8 to 10 msec there is no alteration in the twitch that cannot be accounted for by mechanical dispersion. From this it is clear that the phenomenon is concerned with synchronization rather than desynchronization. The two half-volleys behave quite independently until they are within 0·8 msec of each other; at shorter intervals they interact to give an unwontedly large twitch.

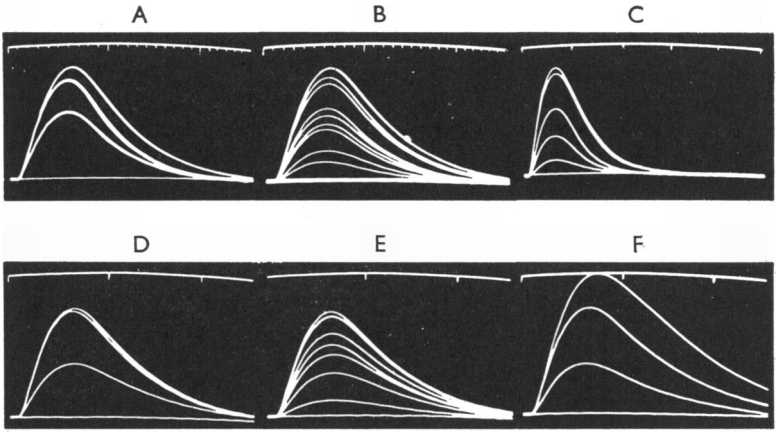

Fig. 6. The effect of exercise on desynchronization and on twitch form; A, B and C before, D, E, and F after, exercise. A, D and F, twitches with and without desynchronization; A, before exercise; D, immediately after 20 sec exercise, circulation occluded; F, shortly after removal of the occluding cuff. In each the smallest twitch is the response to the half-volley from the electrode nearer to the muscle, given alone. B, C and E, series of twitches with progressively decreasing shock strength from maximal down to threshold; B, before exercise; C, a similar series from another experiment taken on a slower time base to show more clearly the changes in form; E, after exercise, circulation occluded (taken just after D). Time markers, 100 msec.

The effect of exercise. If the muscle makes a strong voluntary contraction lasting more than 10 sec the effect of artificial desynchronization temporarily disappears. To record this clearly it is convenient to prevent recovery by arrest of the circulation with a blood-pressure cuff. Such an experiment is shown in Fig. 6A and D. After maximal voluntary efforts of 10–30 sec duration the twitch height is increased by post-tetanic potentiation; more prolonged contraction leads to a reduction due to fatigue. In Fig. 6 the

duration of contraction was chosen so that the twitch tension should be approximately the same before and after, but this is in no way necessary to the demonstration. In this experiment after removal of the cuff considerable post-tetanic potentiation was uncovered and at that time the synchronization effect was (relatively and absolutely) greater (Fig. 6F). Some part of post-tetanic potentiation as ordinarily elicited by single synchronous volleys (Brown & von Euler, 1938), may therefore be an augmentation of the synchronization effect, but a large potentiation may be obtained (e.g. immediately after a 15 sec contraction) at a time when no synchronization effect can be detected.

Tetani. It had been suspected that the reputed weakness of voluntary contractions as compared with tetani excited electrically might be due to the asynchronous nature of voluntary excitation. It is found, however, that a rapid tetanus of desynchronized volleys does not differ, except initially, from one made up of synchronous volleys. Furthermore, if measured carefully, the tension exerted in voluntary efforts and in maximal tetani turns out to be the same (Merton, 1954).

Interaction in single submaximal twitches

If it is true, as stated above, that the two half-volleys in an artificial desynchronization experiment behave independently when they are more than 0·8 msec apart, it can be inferred that the twitch response to a single half volley should be just half the desynchronized twitch. In the human it is not possible to be certain that the two half-volleys are exactly equal, so it cannot be confirmed directly that the peak tension to a single half-volley is half that due to the two. However, it can readily be seen that the peak *time* and the total duration of the half-twitch are effectively the same as in the (maximal) desynchronized twitch. The comparison is shown in Fig. 6A and F. In Fig. 6A the small twitch is rather more than half-maximal and so lasts a little longer than the desynchronized twitch; in Fig. 6F the reverse has occurred; but the general proposition that peak times and durations of these two types of twitch are the same is sufficiently established. These observations on the shape of a single half-twitch reinforce the view that it is merely one independent half of a desynchronized twitch.

The shorter rising time and total duration of a half-maximal twitch is only one stage of a continuous decrease in both these parameters that can be observed if a single (synchronous) maximal shock is progressively reduced in strength (Fig. 6B, C). For several years I have attributed these changes to inadequacies in the technique of recording human mechanical responses, but it is now apparent that they are in reality due to a diminishing degree of synchronous interaction between fibres as fewer are excited. Interaction can be prevented either by stimulating groups of fibres asynchronously, or by not stimulating some groups at all.

After exercise sufficient to abolish the effect of desynchronization (Fig. 6D) the changes in rising time and duration of single submaximal twitches also

vanish (Fig. 6E). This is strong confirmatory evidence that such changes are due to a varying degree of interaction and not to inferior recording conditions.

DISCUSSION

A reduction of the mechanical response by asynchronous stimulation was investigated by Rushton (1932) in the frog's sartorius muscle. The effect was first observed in experiments in which some of the muscle fibres were stimulated directly, and the others a few milliseconds later through the nerve. But it could also be obtained after curare when direct stimuli were applied to the two ends of the muscle. The mechanical response was smaller when the stimuli were not simultaneous than when they were. The explanation was thought to be that many of the muscle fibres in the sartorius do not run the whole length of the muscle but are joined to others in the belly of the muscle. If all the fibres are not excited at once, some of the short fibres when they contract will at first find the fibres in series with them are passive and offer little resistance to stretching. A muscle fibre which is allowed to shorten, even if the shortening is soon arrested, does not develop its full tension; as a result of this many fibres will give a smaller contribution with asynchronous excitation.

A similar explanation could obviously account for the present results, but there are strong grounds against it. An effect due to waste in passive stretching should increase continuously as asynchrony increases, but, as has been shown, the reduction in these experiments is at its greatest when the two half-volleys are separated by only 0·8 msec or even less. Such behaviour is patently inconsistent with an explanation in terms of passive stretch of other fibres, but a modified version could be made to work in which the two ends of a single fibre are supposed to be supplied by different motor nerves. In this case passive stretch occurs in the active fibre itself when one end is excited before the other. The contraction wave as it propagates will stretch the part of the fibre it has not yet reached. The interval of 0·8 msec would represent the time taken for the action potential to travel from one end-plate to the other, for clearly no improvement in performance would result unless the motor impulse arrived at the second end-plate before the muscle action potential from the first. An objection, although not perhaps a conclusive one, can be found to this suggestion also. If the suggestion were true then at long intervals between the two half-volleys the muscle fibre should have recovered from the action potential set up by the first half-volley by the time an impulse in the second arrives at the other end-plate 10 msec later. The fibre would then be excited again. In this way it should be possible to excite some of the muscle fibres twice, although all the motor fibres were stimulated only once. But no sign of a rise in the twitch tension with long intervals between half

volleys was ever observed, although 10 msec (less 0·8 msec for conduction) is ample time for the muscle to recover from the refractory period. The proposed explanation therefore fails.

This experiment also makes it most unlikely that individual muscle fibres are supplied (in significant numbers) from two different motor nerve fibres, a question recently reviewed by Tiegs (1953). The evidence in this case is, however, less compelling, for it might be argued that, owing to some peculiarity of anatomy or excitability, the pairs of nerve fibres supposed to be engaged in multiple innervation were always stimulated together. If they cannot be segregated into the two half-volleys the present method would fail to detect them. This objection cannot apply to the reduction of the twitch by mechanical interaction, for the very essence of this proposal is that each end-plate is excited by a different half-volley. Mechanical interaction is therefore more rigidly excluded than is double innervation.

An alternative possibility is that neuromuscular block normally exists in a proportion of the fibres as Adrian & Lucas (1912) demonstrated in the frog. It could be supposed that when the muscle is stimulated by a maximal asynchronous volley some of the fibres do not contract, but for some reason a highly synchronous volley can break through the block and a bigger contraction results. Such a hypothesis will have difficulty explaining the changes in form of the twitch, for we have seen that slightly reducing the number of fibres taking part in a twitch does not greatly change its form. Study of the action potentials seems to rule out the possibility altogether, for the smaller size of the asynchronous potential can be accounted for quantitatively. In the action potential illustration, Fig. 4C, of the artificial desynchronization experiment the two largest potentials with a latency difference of about 1 msec are the responses to maximal stimulation at each electrode separately. The desynchronized potential lies between them, and is the result of a maximal shock at the proximal electrode given together with a half-maximal shock at the distal electrode. The latter is also recorded alone (the smallest potential). Assuming that action potentials sum algebraically and that the distal shock is exactly half-maximal, the asynchronous potential should be the same as the sum of two half-maximal potentials separated by the conduction latency; that is, it should be the algebraic mean of the two maximal potentials recorded. It is obvious that it is approximately so. The possibility can thus be excluded that 15–20% of the fibres fail to respond to an asynchronous volley. Neuromuscular block does not therefore appear to play a significant part in the phenomenon.

To summarize the position reached, it has been shown that a synchronous volley gives rise to a longer and a larger twitch. It might be that some of the muscle fibres are not excited by an asynchronous volley, but this has been shown to be most improbable. Since all the fibres are excited in each case it follows that in a synchronous twitch they must individually develop a larger tension. The first possibility is that in both cases the contractile response is set going in each fibre in the ordinary way but in an asynchronous twitch

mechanical interaction occurs in the muscle in such a way as to waste the contraction and reduce the tension. Half a millisecond is a very short time for sufficient interaction to occur between twitches lasting more than 100 msec. Furthermore, the only simple way of wasting the contraction, namely by stretching passive series elements, cannot be fitted in with the facts. If these conclusions are correct, that all the fibres contract and do not waste their contraction by mechanical interaction, it follows that in the synchronous twitch the contraction is not set going in just the ordinary way, but is larger from the start. Synchronous action potentials apparently trigger larger mechanical responses in each fibre than asynchronous potentials, a conclusion that contradicts certain incautiously worded statements of the all-or-none principle.

Electrical interaction. The only apparent difference between synchronous and dispersed action potentials which might account for their varying potency in triggering the contractile mechanism is conduction velocity. Synchronous potentials should be conducted more slowly (Katz & Schmitt, 1940). This is because their action currents are attempting to circulate simultaneously in the extracellular fluid, and this is equivalent from the point of view of the individual fibres to raising the external resistance, so that they conduct more slowly (Hodgkin, 1939). Such a slowing is only to be expected if the interval between action potentials in neighbouring fibres is of the order of the spike duration. The hypothesis therefore offers a very satisfactory explanation of why the synchronization effect is only apparent if the dispersion is less than 0·8 msec.

If synchronous potentials are conducted more slowly the form of their recorded action potential should be modified. The artificial desynchronization experiment (Fig. 4C) shows a discrepancy that may be due to this cause. We have already seen that if the two half-volleys are equal (and do not interact) the asynchronous potential should be the mean of the two synchronous potentials. In general, whatever the relative sizes of the two 'half-volleys', the asynchronous potential should always lie between the other two, even if not in the mean position. In the limit where the two maximal potentials meet or cross, the asynchronous potential should also pass through the meeting or crossing point. It can be seen in Fig. 4C that it does not do so, passing slightly above the first crossing and somewhat less above the second. Such discrepancies are magnified by exercise and have regularly been observed in more than twenty experiments. They can only mean that the component potentials which sum to give the synchronous potential are individually smaller in the first phase and larger in the second than when excitation is asynchronous. With surface leads such a difference is consistent with slower conduction. (The discrepancies noted are not such as can be attributed to changes in the number of fibres responding—in that case both phases increase or diminish together.)

Hill's 'active state'. The alterations in the shape of the twitch caused by more synchronous excitation suggest that the underlying change is a prolongation of the intrinsic 'active state' (Hill, 1949). In this theory the contractile process ('active state') is switched on fully at the start of a twitch but soon begins to fall off. The muscle, which cannot shorten instantaneously

owing to its viscous properties, does not have time to stretch the series elastic element (tendon, etc.) fully, before the active state decays. The twitch tension is therefore much smaller than the tetanic tension (which is equal to that of the fully-switched-on active state). If the series elastic component is increased the twitch is further reduced in height, its rising phase slowed and the peak delayed. Hill's theory thus provides additional grounds for rejecting the suggestion that in desynchronized twitches the contraction may be wasted by stretching passive series elements, for in these twitches the rising phase is not slowed and the peak comes earlier and not later (Fig. 5A). Rather, the fact that the early rising phases of both synchronous and desynchronized twitches are identical implies that in both the active state is fully switched on at the beginning, but in the synchronous twitch lasts longer at its full value before falling off. Similar arguments have been advanced by Goffart & Ritchie (1952) in the potentiation of the twitch by adrenaline, which is of just the same type. Why slower action potentials should trigger off a longer active state is quite unknown, but it is of interest that in adrenaline potentiation also the conduction of muscle action potentials is slowed (Brown, Bülbring & Burns, 1948). A causal relationship between reduced conduction velocity and prolongation of the twitch was considered and rejected by Goffart & Ritchie. They did not, however, entertain the possibility that a slower action potential might, as it went along the fibre, call up a longer active state. This hypothesis is attractive not only because it brings together the synchrony and adrenaline results, but because it offers also to make some sense of the correlation which seems to exist in the animal kingdom between the speed of action of a contractile tissue and the velocity of its action potential.

SUMMARY

1. The mechanical twitch of adductor pollicis elicited by a maximal volley from the wrist is larger and lasts longer than when the volley is set up at the elbow. This is shown to be because the wrist volley is more synchronous, the elbow volley suffering temporal dispersion in its journey down the forearm.

2. A maximal volley can be split into half-volleys by a technique of double stimulation; such an artificially desynchronized volley has the same effects as one dispersed by conduction from a distance.

3. When the time relation of two half-volleys is altered it is found that no increase in effect occurs when they are more than 0·8 msec apart; at longer intervals they behave quite independently. Hence the phenomenon is one of interaction occurring only in highly synchronized volleys.

4. Interaction diminishes not only during asynchronous excitation, but also when the total number of fibres involved in a twitch is reduced. Thus submaximal twitches are briefer than maximal twitches.

5. A few seconds' exercise of the muscle temporarily abolishes both the synchronization effect and the differences in duration between large and small twitches.

6. The very short times involved, taken with other evidence, are thought to rule out a mechanical explanation of the synchronization effect.

7. Measurement of action potentials excludes the presence of neuromuscular block, but it reveals an interaction between individual action potentials. The velocity of propagation along the muscle fibres is thought to be decreased with synchronous stimulation.

8. The taller and later peak of the synchronous twitch implies that Hill's 'active state' is prolonged. A causal relation between slower propagation and longer active state would explain the results.

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