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CENTRIFUGAL DORSAL ROOT DISCHARGES INDUCED BY MOTONEURONE ACTIVATION

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SUMMARY

1. It has been confirmed that antidromic stimulation of motoneurones in the cat lumbar cord can induce, when properly conditioned, a centrifugal discharge in dorsal root afferent fibres.

2. The effective conditioning can be (*a*) an orthodromic volley to the same or an adjacent dorsal root, (*b*) a volley to the dorsal column one or two segments above the tested level, or (*c*) a natural stimulus applied to the ipsi- or contralateral hind limb.

3. The conditioning stimulus acts by increasing presynaptic excitability; the peak of its effect (maximum presynaptic depolarization) occurs 7–10 msec after the arrival of the conditioning volley to the cord and then quickly decays.

4. A large antidromic field potential in the ventral horn is not necessary for the production of a centrifugal dorsal root discharge. Activation of a ventral root filament of approximately 100 μ in diameter can still induce such a discharge in a single dorsal root fibre. Furthermore, antidromic stimulation of the remaining fibres of the same ventral root cannot affect the terminals activated by the thin ventral root filament.

5. The phenomenon of motoneurone–presynaptic interaction was obtained in different types of experimental preparations: acute and chronic spinal, anaemic and midcollicular decerebrate, animals with intact supraspinal centres, and one animal without acute laminectomy.

INTRODUCTION

It is well known that a long-lasting depolarization, the dorsal root potential, is produced in cat primary afferent fibres as a result of activity in the same or neighbouring fibres (Barron & Matthews, 1938*a*). During the rising phase of this primary afferent depolarization, centrifugal dorsal root

discharges (the dorsal root reflex) have also been observed in both cutaneous and muscle afferents (Toennies, 1938; Brooks & Koizumi, 1956). It has been proposed that these discharges are directly related to the level and rise time of the depolarization occurring in the presynaptic terminals (Barron & Matthews, 1938*b*; Tregear, 1958; Eccles, Kozak & Magni, 1961).

The current hypothesis concerning the generation of this presynaptic depolarization postulates the presence of chemical, axo-axonic synapses located somewhere near the endings of primary afferent fibres (Eccles, 1964). It has also been suggested that the depolarization could be due to some unspecified electrical mechanism reflecting the activity of spinal interneurones (Bonnet & Bremer, 1938, 1952; Eccles & Malcolm, 1946; Lloyd & McIntyre, 1949). Implicit in this proposition is the possibility that activity in post-synaptic neurones can affect the membrane potential of presynaptic elements. Wall (1958) tested this hypothesis by studying the effect of antidromic firing of motoneurones on presynaptic terminals in the ventral horn of the cat, but found no evidence to indicate that motoneurone discharge could influence the excitability of presynaptic terminals. Recently, however, it has been reported that antidromic activation of spinal motoneurones in the cat can induce, when preceded by an orthodromic volley to a dorsal root, a centrifugal discharge in dorsal root fibres (Decima, 1969; Decima & Goldberg, 1969*a*).

It is the purpose of this paper to study this motoneurone-presynaptic interaction further in a variety of experimental situations, to analyse some of the conditions necessary for this interaction to occur, and to elucidate the nature of the facilitatory influence of the orthodromic dorsal root stimulation. A brief communication concerning some of these results has been published elsewhere (Decima & Goldberg, 1969*b*).

METHODS

All experiments were performed in adult cats and the following preparations were used.

Spinal preparation. Under ether anaesthesia a tracheotomy was performed and the carotid arteries ligated bilaterally. Ephedrine (2–3 mg/kg) was injected I.M. and the brain destroyed anaemically by compression of the vertebral arteries. The ether was discontinued, artificial respiration begun, and the spinal cord sectioned at C1. A laminectomy from L5 to S1 exposed the spinal cord; the dura was opened and the cord covered with warm mineral oil. The appropriate dorsal and ventral roots were sectioned as far from their entry into the cord as possible and mounted in the oil pool on bipolar silver electrodes. Rectal and pool temperature were maintained between 37 and 39° C throughout the experiment. Gallamine triethiodide (Flaxedil) was administered I.V. after spinal section in all cases.

Decerebrate preparation. Three decerebrate preparations were used. In one case the entire brain rostral to the inferior colliculis was removed by suction. In the other two cases the decerebration was obtained anaemically by ligating the branches of both

carotid arteries and then clamping the basilar artery at the mid-pontine level (Pollock & Davis, 1923). The decerebrations and the subsequent lumbar laminectomy were performed under ether anaesthesia.

Other preparations. In three cats the operation and recording were made under sodium pentobarbitone anaesthesia (Nembutal, 35 mg/kg I.P.). In a fourth cat the spinal cord was sectioned at the level of Th 9 under Nembutal, and 40 hr later the acute phase of the experiment was performed as described above in the spinal preparation section. In a fifth cat the experimental procedures were performed without opening the spinal dural sac as will be described in the text.

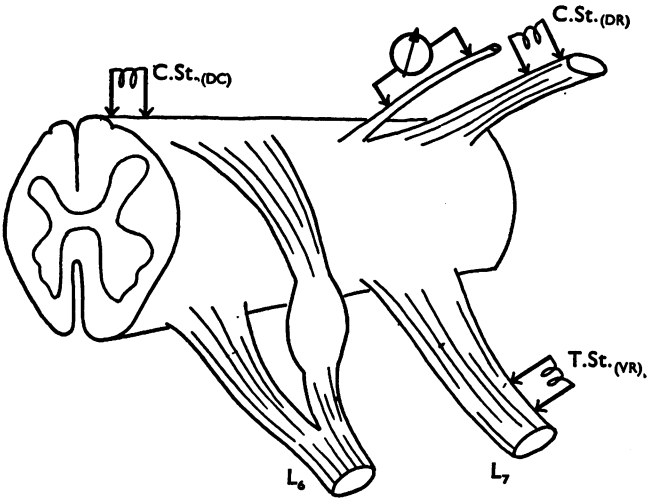

Fig. 1. Diagram of the usual recording and stimulating arrangement. The conditioning stimulus (C.St.) was applied to the main portion of the dorsal root (DR) or to the dorsal columns (DC) one or two segments above the recording level; the test stimulus (T.St.) was applied to all or part of the ventral root (VR). The recording electrodes were placed on a dorsal root filament; in cases where the dorsal root potential has to be recorded the active electrode was positioned near the filament entrance into the cord and the other electrode at the dead end of the filament.

Stimulation and recording. The test stimulus (T.St.) designates an electrical shock delivered to the central stump of cut ventral roots (L7 or S1 in most cases) or the central end of deafferented nerves in the hind limb, e.g. peroneal, branches of the medial plantar, lateral gastrocnemius, posterior tibialis and sciatic (in the latter experiments the dorsal roots L5 to S2 were cut on the ipsilateral side). The conditioning stimulus (C.St.) usually designates one or a train of electrical shocks delivered to the dorsal roots (L6, L7 or S1), to the cord dorsum (L5 to L6), or to a de-efferented sciatic nerve 4–40 msec before the T.St. In some cases the C.St. was a natural stimulus, e.g. muscle stretch, rubbing of the skin. Frequency of stimulation (C.St.-T.St. pair) was from 0·2 to 5 c/s. To reduce the possibility of current spread from ventral root stimulation, the roots were cut and stimulated as far from the cord as possible. The proximal electrode was always the cathode and the duration and intensity of stimulation were kept at a minimum (10–50 μsec; 0·2–1 V).

Recordings were made from filaments of dorsal roots L6, L7 and S1 (central

stump) or from the central ends of de-efferented muscle and mixed nerves in the hind limb. Ventral roots L 5 to S 2 were cut on the ipsilateral side in the latter cases. In the experiments in which the dorsal root potential was recorded, the proximal recording electrode was placed 2–3 mm from the point where the dorsal root fibres enter the cord. However, in most animals the recording was made far from the cord to reduce the possibility of trauma to the dorsal root–spinal cord junction. Figure 1 is a diagram of the experimental arrangement.

In some experiments presynaptic excitability was measured using Wall's technique. The electrode used for focal stimulation was a glass micropipette (15–20 μ tip diameter) filled with 0·9 % (w/v) NaCl. Electrical stimulation was delivered from pulse generators (Tektronix type 161 and Grass stimulator S 8) through isolation units. Responses were displayed on a cathode ray oscilloscope (Tektronix type 565) and photographed with a Grass kymograph camera. In seven experiments signals displayed on the oscilloscope were fed from the signal out jacks at the back of the oscilloscope into a Sanborn 3917 A tape recorder and were subsequently played back into the oscilloscope and photographed. Amplification of the responses were obtained with AC-coupled Tektronix type 122 preamplifiers (1 sec time constant) and type 3 A 3 and 2 A 63 Tektronix DC differential amplifiers.

RESULTS

Motoneurone-presynaptc interaction. The basic phenomenon with which this paper is concerned was studied mainly in spinal preparations and is presented in Fig. 2. When the stimulus was applied to the ventral root (T.St.) the only electrical event consistently observed in the dorsal root filament was a complex wave form probably signalling the arrival of the motor volley to the cord. This potential variation proved to be a 'field' effect since it persisted after the dorsal root was crushed between the cord and the recording electrode. The effect of a volley to the main part of the dorsal root (C.St.) is shown in Fig. 2*B*, where both the dorsal root potential and the dorsal root reflex are clearly seen. It should be noted that 20 msec after the stimulus, at a time when the dorsal root potential has reached its peak, there is practically no dorsal root reflex activity present in the filament. If now both the C.St. and T.St. are delivered together but with the dorsal root volley preceding the ventral root stimulation by 21 msec, the antidromic activation of the motoneurones now drives a large centrifugal discharge in the dorsal root fibres (Fig. 2*C*).

The same phenomenon is seen with the use of a fast time base in Fig. 3. This Figure shows the absence of activity in the filament 20 msec after the conditioning stimulus to the dorsal root (Fig. 3*A*). Figure 3*B* clearly shows the biphasic wave ('field' potential) usually observed immediately after the ventral root stimulus. However, the use of many superimposed tracings reveals that in a few sweeps an action potential follows the first complex deflexion 0·9 msec after the shock. The effect of a conditioning volley preceding the ventral root stimulus by 21 msec is shown in Fig. 3*C*; a large synchronous discharge is present 0·8 msec after the ventral root

artifact. It should be noted that this response has a fixed latency since even at this fast sweep practically no jitter is present. The presence of a centrifugal discharge driven by the T.St. itself (i.e. no C.St.) as in Fig. 3*B*, was not altogether uncommon; however, it was always considerably smaller and of longer latency than that obtained with C.St. The dorsal root centrifugal discharge could be obtained by using the ventral root of the

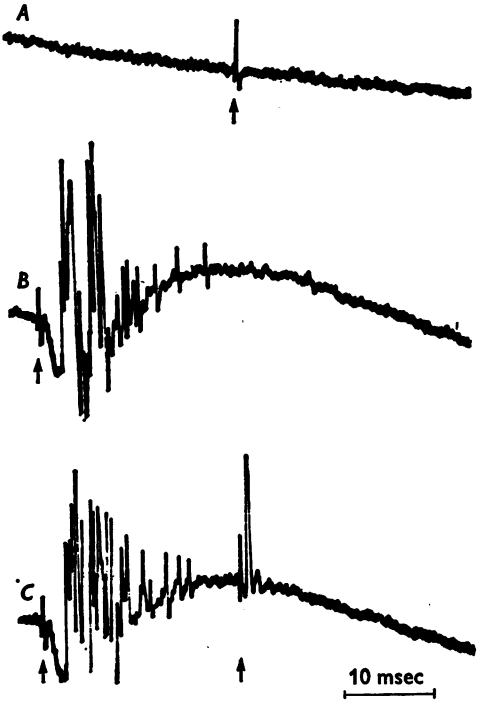

Fig. 2. Centrifugal dorsal root discharge triggered by motoneurone activation. All records were obtained from the central end of a thin dorsal root filament (S 1). *A*, the arrow signals the moment of stimulation of ventral root L 7 (T.St. alone). *B*, stimulation of the upper half of dorsal root L 7 (C.St. alone); the dorsal root reflex discharge is seen on the rising phase of a slow depolarization (the dorsal root potential). *C*, same as *B*, but with stimulation of the ventral root delivered 21 msec after the C.St. The large antidromic discharge driven by the T.St. is clearly seen.

same or an adjacent segment as the T.St. However, the size of the response as a function of the activated motor pool, i.e. same versus adjacent segment, was not specifically studied.

Type and nature of the conditioning stimulation. In one of the initial experiments, dorsal root L 7 was split into four filaments of approximately equal size. Each one of these dorsal root filaments was separately tested

for a ventral root-driven discharge while the remaining three filaments were stimulated together for delivery of the C.St. (the same T.St. to ventral root L 7 was used throughout). Under these experimental conditions, the T.St. triggered a centrifugal discharge in only one of the four filaments. However, if a dorsal column volley was used as the C.St. the situation

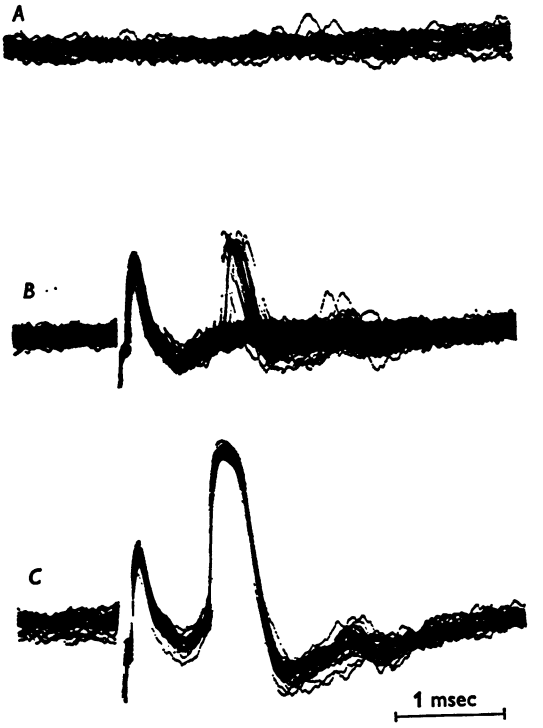

Fig. 3. Recording of the dorsal root centrifugal discharge with the use of a fast time base and a delayed sweep circuit. These records are from the same experiment as Fig. 2; recording was made from a dorsal root S 1 filament by the photographic superimposition of twenty-five sweeps in each tracing. *A*, delayed sweep begins approximately 20 msec after a conditioning stimulus (C.St.) was delivered to the upper half of dorsal root L 7. *B*, stimulation of ventral root L 7 (T.St.) alone. *C*, same as *B*, but with C.St. delivered 21 msec before the T.St.

changed completely: after a short period, an area of the dorsal funiculi was found, stimulation of which was capable of successfully conditioning a centrifugal discharge in all four L 7 dorsal root filaments. Similar results have been repeatedly obtained in other experiments by simply changing the dorsal root used for the C.St., e.g. L 6 instead of L 7, L 6 plus L 7. These results obviously indicate that the size and location of the conditioning

volley are important factors for obtaining results in a given group of dorsal root fibres.

Another factor related to the C.St. was the type of afferent volley needed to obtain an effective conditioning. The experiments so far reported (Figs. 2 and 3) have been concerned only with the use of brief electrical shocks as C.St. i.e. synchronous afferent volleys. It is known, however, that natural stimuli can also produce depolarization of primary afferent fibres inside the C.N.S. (Barron & Matthews, 1938*a*). Therefore, one could ask if

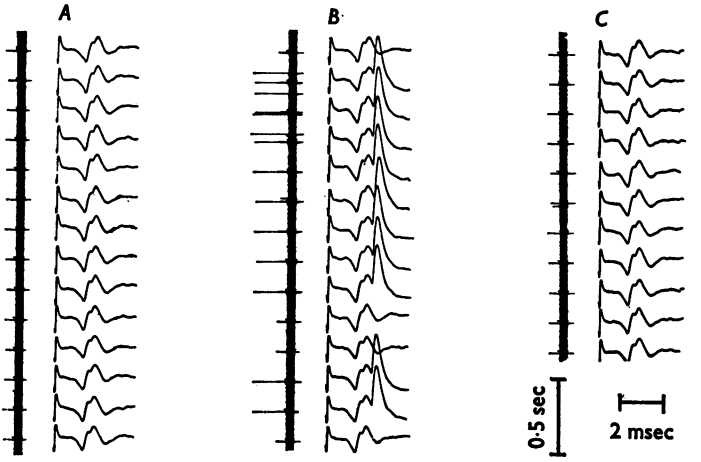

Fig. 4. Use of natural stimulation for conditioning motoneurone-presynaptic interaction. Recording was made from the central stump of a fine dorsal root filament (L 7). In each column, discharges in the dorsal fibres were recorded by a stationary beam on the left and with a fast time base on the right. The sweep was triggered at the moment when the T.St. was being delivered to the deafferented sciatic nerve in the thigh. *A*, stimulation of the deafferented sciatic nerve at 5 c/s; only a 'field' potential signalling the arrival of the sciatic motor volley to the cord is seen. *B*, pinching of the inner toe of the contralateral paw (C.St.) during sciatic stimulation; a single spike was driven in the dorsal root filament. *C*, control immediately after withdrawal of natural stimulation (C.St.).

natural stimuli could be used successfully as the conditioning stimulus. Fig. 4 shows that this is indeed the case. In this experiment the T.St. was applied to the deafferented left sciatic nerve in the thigh. The conditioning stimulation used was pinching of the contralateral paw (Fig. 4*B*). A single fibre action potential is clearly driven in the thin dorsal root filament by motoneurone excitation during the application of this natural, and conditioning, stimulus to the contralateral hind limb.

It was originally hypothesized that an increase of presynaptic excitability, induced by the conditioning stimulus, made it possible for the motoneurone–presynaptic interaction to trigger a centrifugal dorsal root

discharge (Decima, 1969; Decima & Goldberg, 1969*b*). This interpretation was based upon the fact that primary afferent depolarization, produced by different sensory inputs to the cord, is accompanied by an increase in presynaptic excitability (Wall, 1958; Eccles, Magni & Willis, 1962). A possible test of this hypothesis would be to compare the time course of the effect of the C.St. upon the motoneurone-presynaptic interaction vis-à-vis the excitability changes on primary afferent terminals. This was done in the experiment illustrated in Fig. 5. The tip of a glass micropipette was placed in the ventral horn at the level of L 7 and its position was determined by recording from the pipette while stimulating ventral root L 7 antidromically. The pipette was lowered into the cord until the typical negative field potential was obtained, indicating that invasion of the somadendritic membrane was being recorded and that the electrode tip was located in the motor nucleus. A negative current pulse (0·15 msec) was then injected through the pipette (the T.St.) and the centrifugal spike evoked in a dorsal root L 7 filament was recorded. Since the micropipette was in the motor pool, it can be safely assumed that the presynaptic terminals being tested, whichever they are, correspond to the type of fibre involved in the phenomenon of motoneurone-presynaptic interaction. A C.St. to 2/3 of dorsal root L 7 was delivered at various times before the pipette pulse (the T.St.), and the amplitude of the centrifugal spike was plotted as a function of the C.St.–T.St. interval (Fig. 5*A*). The same procedure was then repeated except that now the T.St. was a shock delivered to ventral root L 7 and the amplitude of the centrifugal spike, induced by the motoneurone-presynaptic interaction, was measured at various C.St.–T.St. intervals (Fig. 5*B*). It is obvious that there is a close correlation between the time course of excitability increase in the presynaptic terminals (Fig. 5*A*) and the time course of facilitation of motoneurone-presynaptic interaction (Fig. 5*B*). The peak of the effect in both cases usually occurs about 7–10 msec (6·5–7·0 msec after the C.St. in Fig. 5) and decays rapidly thereafter. In Fig. 5*C* a tracing of the dorsal root potential evoked by the C.St. is presented (recording made from the same dorsal root L 7 filament as in Fig. 5*A* and *B*). It should be noted that the peak of the dorsal root potential occurs at a time when both the presynaptic excitability and facilitation of the interaction have been reduced almost to control levels.

Under the condition of this experiment (extracellular recording from a fibre population), it is not possible to establish whether the dorsal root fibres involved in motoneurone-presynaptic interaction contribute to the production of the dorsal root potential observed. However, intra-axonal recordings of primary afferent depolarization, obtained at the dorsal root-cord junction, have a similar time course to the dorsal root potential shown

(Eccles, 1964). Therefore, it can be assumed that if the presynaptic depolarization producing the results of Fig. 5*A* can be recorded at the dorsal root entrance, its time course would correspond to those presented in Figs. 5 and 6. The fact that the excitability change in the presynaptic terminals has such a different time course from the dorsal root potential is indeed what would be expected if one considers the cable properties of the fibres. In other words, if the dorsal root potential is a depolarization

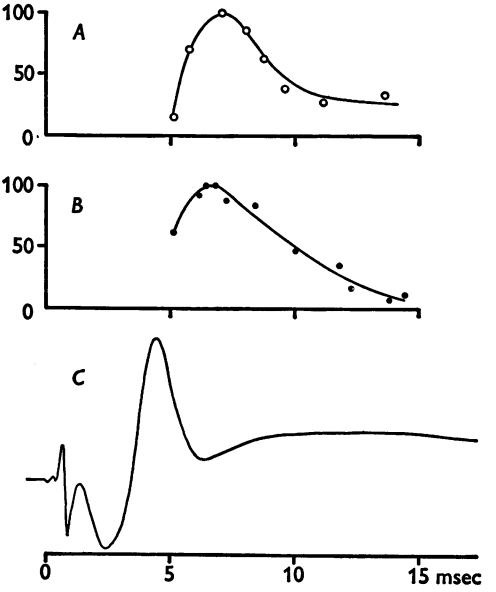

Fig. 5. Presynaptic excitability changes and conditioning of the motoneurone-presynaptic interaction. All records were obtained from the same dorsal root L7 filament. The C.St. was delivered to 2/3 of dorsal root L7 and was unchanged throughout the experiment. A micropipette was inserted in the ventral horn at L7 for focal stimulation. In *A*, the amplitude of the spike produced in the dorsal root filament by the pipette stimulus is plotted at various C.St.–T.St. intervals. The largest average amplitude was considered to be 100% and the average amplitudes of all other C.St.–T.St. intervals were related to it. *B* is the same as *A*, except that the T.St. was a shock delivered to ventral root L7 and the average amplitudes of the antidromic spike induced by motoneurone-presynaptic interaction are plotted. *C* is a tracing of ten superimposed sweeps of the response evoked in the dorsal root L7 filament by the C.St.; both the dorsal root reflex discharge and the initial part of the dorsal root potential are seen.

generated in the terminals in the ventral horn and propagated electrotonically from there to the dorsal root, the time course of the generator should have a sharper rise time and occur at a shorter latency when recorded at the terminals as compared with a recording made from the dorsal roots. This is indeed what the results of Fig. 5 indicate.

At the stimulus intensities usually utilized in these experiments, the C.St. produced a dorsal root reflex in the dorsal root filament from which the recording was being made (see Fig. 2*B*). It also evoked orthodromic action potentials in the ventral root used for the test stimulus. These two factors made it difficult to accurately determine the effect of the C.St. on the motoneurone-presynaptic interaction at short C.St.–T.St. intervals. These complicating factors were eliminated in the experiment illustrated

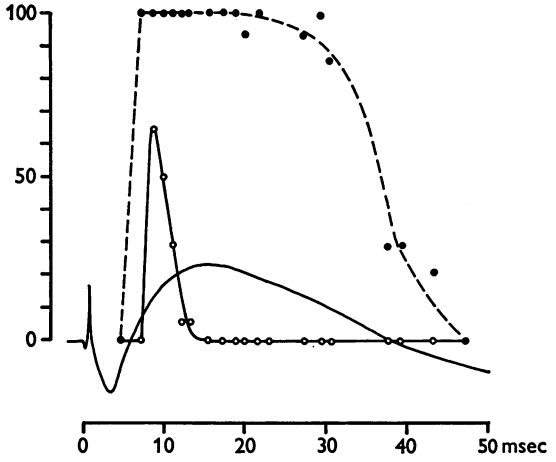

Fig. 6. Firing probability of two dorsal root fibres at different C.St.–T.St. intervals. C.St. was a single shock to 2/3 of dorsal root L 7. The T.St. was applied to ventral root L 7. Recording was made from a thin dorsal root filament (L 7). Closed circles: fibre no. 1. Open circles: fibre no. 2. The tracing at the bottom is the dorsal root potential recorded in the same filament. See text for further details.

in Fig. 6. In this experiment the C.St. delivered to 2/3 of dorsal root L 7 was subthreshold for the dorsal root reflex discharge and did not produce propagated potentials in ventral root L 7 which was the root used for the T.St. At this low C.St. intensity two single fibres could still be observed firing as a result of the ventral root L 7 stimulus. At each C.St.–T.St. interval fourteen trials were run and the firing probability for each fibre was determined. The results of Fig. 6 show that the duration and intensity of the facilitation was different for the two fibres; however, the latency for the highest firing probability was similar in both cases, namely 7–9 msec. Again, as in Fig. 5, this peak occurs during the rising phase of the dorsal root potential. It is clear, therefore, that the time course of the facilitation produced by the C.St. on motoneurone-presynaptic interaction does not depend on the presence of either the dorsal root reflex or orthodromic discharge of motoneurones.

Size of test stimulus. One of the important questions which must be asked is how many motoneurones need to be synchronously activated in order to drive a centrifugal action potential in a single primary afferent fibre. The experimental results shown in Fig. 7 are from one of the experiments designed to analyse this problem. The first step in this experiment was to find a dorsal root filament presenting the phenomenon (centrifugal driving of primary afferent fibres by motoneurone activation). After this filament was found, it was teased apart until only a single fibre driven by

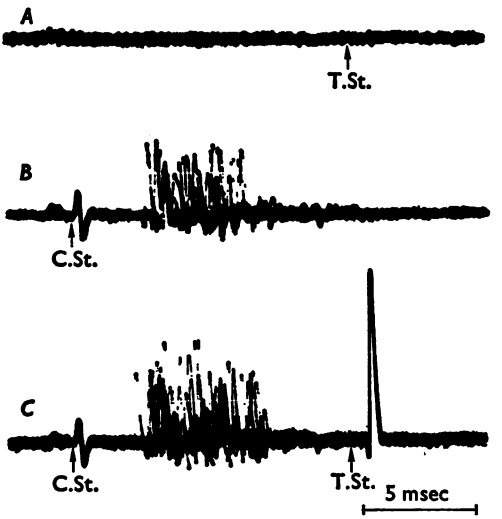

Fig. 7. Discrete motoneurone activation driving a single dorsal root fibre. Recording was made from a fine filament of dorsal root L 7; each record was obtained by photographic superimposition of twenty sweeps. *A*, the arrow indicates the position of a stimulus (T.St.) delivered to a very fine filament of ventral root L 7 (approximately 100 μ in diameter). *B*, stimulation of dorsal root L 6 (C.St.); dorsal root potential is not seen because the proximal recording electrode was placed far from the root entrance into the cord. *C*, same as *B*, but with the T.St. delivered 12·2 msec after the C.St. A single fibre action potential is driven by the T.St. Notice that this fibre (large spike) is not previously discharged in the dorsal root reflex.

the motoneurone activation was isolated. The dissection was then continued at the place of application of the test stimulus, i.e. the ventral root central stump. By progressive teasing out of the ventral root the point was reached where stimulation of a ventral root filament of approximately 100 μ in diameter was still capable of driving the dorsal root spike (Fig. 7*C*). It should be stressed that simultaneous stimulation of all the other remaining ventral root filaments was absolutely incapable of activating that single dorsal root fibre. Stimulation of fine branches of different muscle nerves in animals with dorsal roots L 5 to S 1 severed gave similar results. As in

the experiment with ventral root splitting, this procedure showed again that a single dorsal root fibre could be driven by a very small nerve filament. The small size of the ventral root filament explains the lack of field potential in Fig. 7*A* when only the test stimulus was being delivered. This picture also shows that the activated dorsal root fibre does not need to be discharged by the dorsal root reflex in order for the phenomenon of motoneurone-presynaptic interaction to be observed. The time-locked character of this centrifugal dorsal root discharge is also clearly seen and is in direct contrast to the variability of the discharges produced by the dorsal root reflex.

Different experimental preparations in which motoneurone-presynaptic interaction was observed. The preparation most often used in these experiments was a curarized animal with an acute high spinal section (made under ether anaesthesia). The mid-collicular and the anaemic decerebration were also used. The animals in this series were not curarized and were breathing by themselves. In all of these preparations antidromic motoneurone stimulation was consistently able to induce a centrifugal dorsal root discharge when properly conditioned.

The animal in which the spinal cord was cut 40 hr before the acute experiment, was used to test the possibility that acute surgical trauma to the C.N.S. by high spinal section, or decerebration, could be responsible for the appearance of the phenomenon. In this case again, antidromic stimulation of a ventral root, if properly conditioned, was capable ot driving a dorsal root centrifugal discharge.

Motoneurone-presynaptic interaction could be obtained in only two of the three cats anaesthetized with Nembutal (with their supraspinal centres intact). The effect of Nembutal was also studied on one of the standard high spinal animals. A subanaesthetic dose of the drug (15 mg/kg) in this preparation produced a considerable reduction (larger than 50 %) in the dorsal root discharge a few minutes after the injection.

The experiment illustrated in Fig. 8 was designed to test whether the centrifugal dorsal root discharge triggered by the ventral root stimulation was to any extent dependent on the somewhat abnormal conditions which may prevail in the spinal cord after an acute laminectomy (Barron, 1940). The experiment was carried out in two stages: the first was one of aseptic surgery during which a hemilaminectomy exposed the L 6, L 7 and S 1 segments and their corresponding roots on the left side. Under the dissection microscope, dorsal root S 1 and ventral root L 7 were severed extradurally. Two weeks after this operation the animal was in excellent condition except for a slight muscle weakness in the left hind limb. The final stage of the experiment was carried out at this time; its initial steps were the same as many of the acute experiments, i.e. high spinal section (C 1)

and anaemic destruction of the brain under ether anaesthesia. The L 6 and S 1 nerves were approached via a low mid line laparotomy and severed as far distally as possible (see diagram of Fig. 8). A bipolar stimulating electrode was then placed on the central stump of S 1 and fixed to the nearby muscles. Another stimulating electrode was put on the sciatic

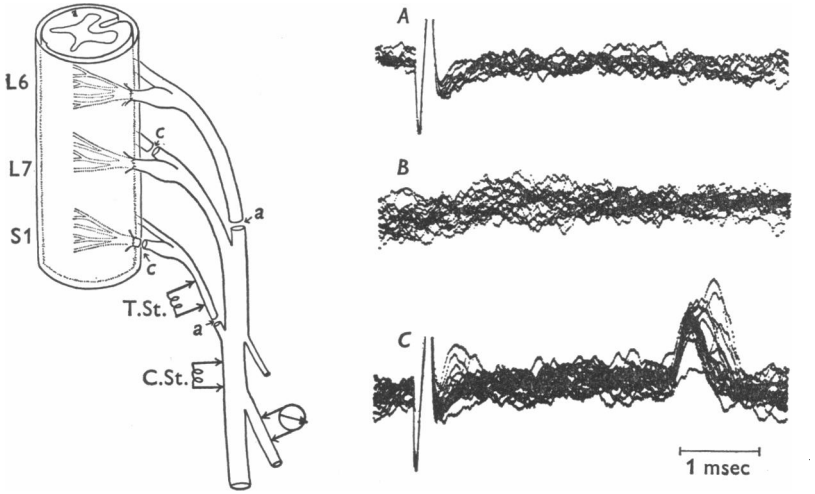

Fig. 8. Centrifugal dorsal root discharge in an animal without acute laminectomy. At the left is a diagram of the experimental preparation. The acute (*a*) and chronic (*c*) section of nerve fibres are marked. The T.St. was applied to the deafferented S 1 nerve; the C.St. was delivered to the de-efferented sciatic nerve. All recordings were made from the tibialis nerve with the use of a delayed sweep; trace in *A* was made by photographic superimposition of ten sweeps, and in *B* and *C* of twenty sweeps. *A*, stimulation of S 1 nerve (T.St.). *B*, stimulation of sciatic nerve (C.St.) delivered approximately 19 msec before the beginning of the sweep (burst of four shocks at a rate of 400 c/s). *C*, same as *B*, but with the T.St. delivered 20 msec after the C.St. Note the synchronous discharge driven by the T.St. after a delay of 3·2 msec. See text for further details of this experiment.

nerve, in continuity, and various sciatic nerve branches were dissected. The diagram at the left of Fig. 8 summarizes the experimental preparation and the stimulating and recording arrangements. Thus the T.St. was delivered to S 1 motoneurones by the stimulating electrode buried deep in the pelvis; the C.St. (dorsal root L 7) was given by the electrode placed on the de-efferented sciatic nerve in the thigh. The positive response obtained in the de-efferented tibialis nerve can be observed in Fig. 8*C*. This experiment, therefore, clearly demonstrates that the centrifugal dorsal root discharge triggered by motoneurone-presynaptic interaction does not depend on the surgical trauma to the cord likely to be produced by an acute laminectomy.

DISCUSSION

The observation (Decima, 1969; Decima & Goldberg, 1969*a*) that motoneurone activation, if properly conditioned, can induce a centrifugal dorsal root discharge has been confirmed. The possibility of an experimental error due to current spread from the ventral root has been ruled out by the experiment in which motoneurone activation was obtained by stimulation of a deafferented sciatic nerve (Fig. 4). It has also been established that the discharge recorded in the dorsal root was indeed a propagating action potential, and not an electrotonic or field potential, since it was recorded in a de-efferented peripheral mixed nerve as well (Fig. 8).

A criticism of these experimental results could be made perhaps on the basis of the large synchronous motoneurone excitation commonly used as the T.St. (i.e. antidromic stimulation of a whole ventral root). However, the type of experiment illustrated in Fig. 7 shows that the motoneurone-presynaptic interaction can easily be obtained with the use of a very discrete motoneurone activation. It should be stressed here that in both the ventral root splitting experiments and in the experiments using deafferented peripheral nerves, the stimulation of only a small number of particular motoneurones was capable of driving a given dorsal root fibre. On no occasion was it possible to induce firing of the same fibre by the simultaneous stimulation of the remaining motor axons belonging to the same ventral root. This evidence indicates that the phenomenon cannot be obtained simply by inducing large potential fields in the ventral horn. Rather, some special type of spatial arrangement between particular motoneurones and the presynaptic terminals of a given primary afferent fibre needs to be present in order to explain the results herein described.

The fact that the centrifugal dorsal root discharge could also be obtained when the C.St. was a natural stimulus (Fig. 4) clearly indicates that a massive synchronous afferent volley is not essential for motoneurone-presynaptic interaction to be observed. Although a dorsal root volley (C.St.) does produce changes in motoneurone excitability, the time course of these changes as reported by Renshaw (1942) and Brock, Coombs & Eccles (1953), are too short (2–4 msec) to account for the results illustrated in Fig. 5*B*. On the other hand, the change in presynaptic excitability induced by the C.St. (Fig. 5*A*) does have a similar time course to the facilitation of motoneurone-presynaptic interaction (Fig. 5*B*). We can conclude, therefore, that the effectiveness of the conditioning stimulus in facilitating motoneurone-presynaptic interaction lies in its ability to increase the excitability of the presynaptic terminals.

Antidromic stimulation of a ventral root in the frog can sometimes produce a centrifugal dorsal root discharge without a previous conditioning

volley (Katz & Miledi, 1963). Unlike the cat, however, ventral root stimulation in this animal induces a dorsal root potential (Barron & Matthews 1938*a*) and the centrifugal dorsal root discharge, when present, is observed riding on this depolarization. On the other hand, even in the absence of an experimentally induced dorsal root potential, ventral root stimulation in the cat could occasionally drive a centrifugal dorsal root discharge (Fig. 3*B*). This new experimental finding in the cat can be explained if one considers that the resting potential (i.e. the excitability) of presynaptic terminals is not constant but is continuously varying within certain limits (Barron & Matthews, 1938*a*; Wall, 1964; Rudomin & Dutton, 1969). This must be the logical assumption if one accepts the fact that (1) natural stimuli do depolarize primary afferent fibres and (2) a continuous barrage of impulses are reaching the cord at any instant in time. It is therefore not difficult to postulate that motoneurone activation will find, at any given moment, some presynaptic terminals at a resting potential low enough to reach firing level as a result of the motoneurone-presynaptic interaction occurring at the time of motoneurone firing.

The physiological significance of this motoneurone-presynaptic interaction is not clear at present. However, two main lines of evidence indicate that this interaction cannot be considered an experimental artifact. (1) Motoneurone-presynaptic interaction is present in a large variety of experimental preparations (acute and chronic spinal, midcollicular and anaemic decerebration, animals with intact supraspinal centres and one animal without acute laminectomy) and (2) neither massive activation of motoneurones nor large synchronous conditioning volleys are necessary for this interaction to be observed.

In conclusion, these experimental results clearly indicate that motoneurone activation does depolarize presynaptic terminals since such activation is capable of driving a centrifugal dorsal root discharge when properly conditioned. Thus, it could be said that changes in presynaptic excitability do occur every time a motoneurone fires. However, the actual discharge of a dorsal root fibre would depend on the membrane potential level of the presynaptic terminals at the moment of motoneurone activation.

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