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DORSAL ROOT REFLEXES OF MUSCLE GROUP I AFFERENT FIBRES

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Investigations on the interaction between afferent fibres have largely been concerned with cutaneous afferent fibres (Barron & Matthews, 1935, 1938; Toennies, 1938, 1939), which are particularly involved both on the input and output sides of the dorsal root reflex (DRR). Toennies (1939) reported that tapping the patellar tendon gave a DRR into the saphenous nerve, but it probably was not due to muscle stretch receptors, because a similar DRR was produced by tapping the skin over the tibia. Brooks, Koizumi & Malcolm (1955) and Brooks & Koizumi (1956) showed for the first time that there was a DRR from muscle afferents to muscle afferents, but only when the spinal cord was cooled. There was also a DRR from muscle nerve to cutaneous nerve and vice versa. So far as these types of DRR were investigated, they resembled the cutaneous-to-cutaneous DRR.

Further investigation of the phenomena of central interaction between muscle afferent volleys has been undertaken because it has seemed likely that such interaction forms the basis of the depression which muscle afferent volleys exert on the monosynaptic excitatory post-synaptic potential (EPSP) produced in motoneurones by other muscle afferent volleys (Frank & Fuortes, 1957; Frank, 1959). A preliminary report of this investigation on dorsal root reflexes has already been published (Eccles, Kozak & Magni, 1960).

METHODS

Throughout the whole experiment, the animal (cat) was lightly anaesthetized by pentobarbital sodium. The spinal cord was exposed by a laminectomy from L2 to S1. It was severed at the upper L2 level, and the left L4–S2 ventral roots were divided. The various peripheral nerves employed in the investigation were dissected on the left side and mounted on platinum electrodes (interpolar distance in excess of 10 mm) that could be used either for stimulating or recording. Approximate monophasic conditions of recording from peripheral nerves were secured by the application of procaine to the killed ends of the nerves, but with many of our records there was still much diphasic distortion. Both the

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cord and leg regions were covered by warmed mineral oil contained in pools formed by skin flaps. The animal was fixed in the rigid frame and the intracellular recording from afferent fibres was performed as described by Eccles & Krnjević (1959*a*). The temperature of the animal was registered by a thermometer inserted through the back of the neck so that it rested just outside the thoracic cage. The temperatures of the oil baths were also recorded frequently. The temperature of the interior of the spinal cord would lie between the temperatures of the body and of the oil pool. Since these temperatures rarely differed by more than 2 or 3° C, no large error would be introduced by assuming that the body temperature was a measure of the cord temperature. When cooling the spinal cord the whole animal was cooled by cold applications and the heating of the oil pool was also turned down so that the temperatures of the oil pool and of the body were approximately the same.

The following nerves were prepared, and their identification symbols are also given: posterior biceps plus semitendinosus (PBST); anterior biceps plus semimembranosus (SMAB); medial gastrocnemius (MG); lateral gastrocnemius plus soleus (LGS); plantaris (PL); flexor digitorum longus plus flexor hallucis longus (FDHL); peroneus longus, brevis and tertius (P); deep peroneal to tibialis anticus and extensor digitorum longus, the branch supplying skin and the extensor digitorum brevis muscle being eliminated (DP); sural (S); the purely cutaneous component of superficial peroneal (SP). When MG and LGS are combined the symbol is (GS), while P + DP = (PDP).

RESULTS

General investigations on dorsal root reflexes (DRRs) evoked by muscle afferent volleys

In the cooled spinal cord of the cat maximum Group I afferent volleys in the posterior biceps-semitendinosus (PBST) nerve always produced large dorsal root reflex (DRR) discharges in the afferent fibres of muscle nerves (Figs. 1E, 4, 5, 6, 7). Group I volleys in that part of the peroneal nerve supplying the anterior tibial muscles (PDP, see Methods) were also effective in producing DRRs (Figs. 1G; 6). Synchronous volleys in PBST and PDP were particularly effective (Figs. 1C, F, I; 3A, I; 4F; 8C).

In confirmation of Brooks & Koizumi (1956), these DRRs from muscle afferent fibres to muscle afferent fibres were usually slight at normal body temperature (Fig. 1A–C), and increased progressively as the cord was cooled (Fig. 1D–F), being always very large at the low temperature of 30° C. In many preparations the DRRs were very large when the body temperature was no lower than 35–33° C (Figs. 3; 4F–J; 5B). A DRR has been observed when the body temperature was as high as 40·5° C (Fig. 1G–I).

In general these DRRs from muscle afferent to muscle afferent have closely resembled the DRRs generated by cutaneous afferent volleys in cutaneous afferent nerves (Toennies, 1938, 1939). The central latent period was never less than 4 msec, and central latencies as long as 10 msec were observed with weak DRRs at low temperatures (about 30° C). Large DRRs were often up to 20 msec in duration, and there were sometimes distinct initial waves at 3–6 msec apart (Figs. 2E–M; 5, 6, 7), which evidently

were produced by an approximate synchronization of repetitive discharges in many fibres. Such preferred latencies for the discharges have been reported by Frank (1953) and will be illustrated later with intracellular recording.

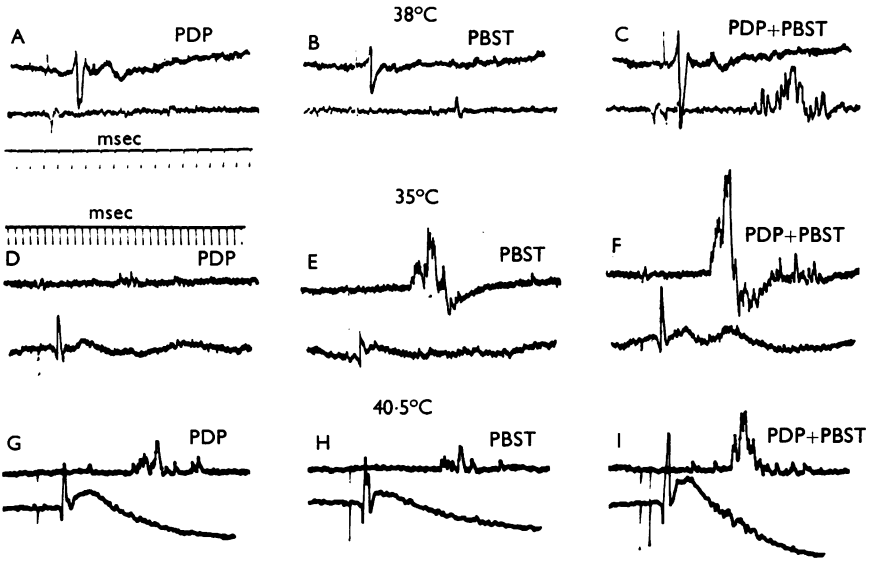

Fig. 1. Dorsal root reflexes (DRRs) produced by maximum Group I volleys in muscle nerves as indicated by the symbols above each record, and recorded in the FDHL nerve. In A–C the lower trace is from the FDHL nerve, the upper giving the potentials produced by the afferent volleys entering the cord through the L 7 dorsal root, negativity being downwards. D–F were recorded later in the same experiment after the temperature had fallen from 38 to 35° C. The monitored record from the L 7 dorsal root entry is now below the DRR record, and negativity is upwards. Note slower time scale for D–F. G–I are records as for D–F, but in another experiment with body temperature 40·5° C, and with time scale as for A–C.

When the DRR was small, a second volley at a brief interval (up to 5 msec) in the same afferent nerve caused a large increase in the DRR (Fig. 2A–D). With stimulus intervals longer than 10 msec there was depression of the DRR evoked by the testing volley, as was originally observed for cutaneous DRRs (Toennies, 1938). The depression gradually passed off as the testing interval was lengthened beyond some hundreds of milliseconds (Fig. 2E–I).

The prolonged depression that follows the production of each DRR by an afferent volley is well illustrated by observing the effects of repetitive stimulation. As is shown in the series of Fig. 2J–M, the DRR rapidly ceases, even during high-frequency tetanization. This complete suppression of the DRR is maintained so long as the tetanization is continued. Alternatively, the DRR can be observed at the steady state that is quickly

reached during sustained stimulation at relatively low frequency. As is shown in Fig. 3A–H, even a large DRR failed completely at a frequency of 5/sec, and almost no reflex survived at 2/sec. Over the frequency range 1/sec to 0·2/sec the DRR increased greatly, but only when the frequency was as slow as 0·14 to 0·1/sec was the DRR as large as with indefinitely long intervals between successive volleys. Figure 3I–L shows that even at a temperature as high as 40·5° C there is similar effect of suppression of the DRR as the frequency is raised to 0·2/sec and upwards. The small spike marked by an arrow is an example of a recurrent discharge, as discussed below. In the present investigation it has been a standard practice to evoke the DRRs at intervals of 7 sec, so as to avoid the depression.

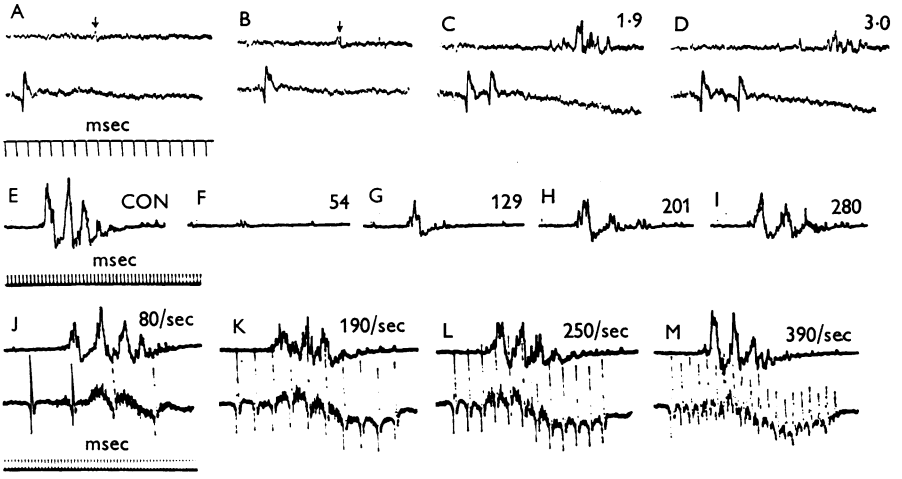

Fig. 2. Summation and depression of DRRs. A–D show DRRs (upper trace) and monitored dorsal root records produced by maximum Group I PBST volleys in MG nerve. Summed actions of two volleys at 1·9 and at 3·0 msec are seen in C and D, while A and B give control records for single volleys, there being DRRs in only one or two fibres. Body temperature 37° C. E shows control DRR discharged into the GS nerve in response to two maximum Group I PBST volleys at 3 msec interval. In F–I this double volley was employed as a test of the depression following conditioning by another similar double volley. The test intervals are indicated in msec. In J–M are DRRs produced in the GS nerve by repetitive Group I PBST volleys at the indicated frequencies, and as shown by the monitored records from the dorsal root in the lower traces. E–M are from the same preparation and at a body temperature of 31° C. Time scales are shown below each series.

Following a prolonged repetitive series of muscle afferent volleys there is rapid recovery from the depression (Figs. 2, 3) that occurs during the stimulation, and after several seconds a testing afferent volley usually evokes a larger DRR (Fig. 4B–D) than in the control responses before the tetanus (Fig. 4A). In every respect this potentiation is equivalent to the

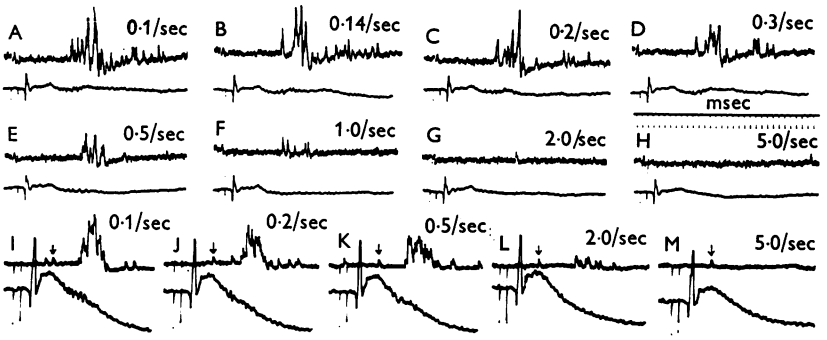

Fig. 3. Effect of frequency of stimulation on DRRs. A–H: Upper traces show DRRs produced by a single synchronized volley in PBST + PDP (maximum for Group I) and recorded in the FDHL nerve. The frequency of repetition is shown for each record, and below each record is the monitored record from L7 DR. Body temperature 33·5° C. I–M is a similar series in another preparation, but with a body temperature of 40·5° C. Arrows indicate a single impulse discharged with precisely the same latency at all frequencies. It is an example of a recurrent discharge (cf. Fig. 13). There is in addition an earlier spontaneous discharge in I and K.

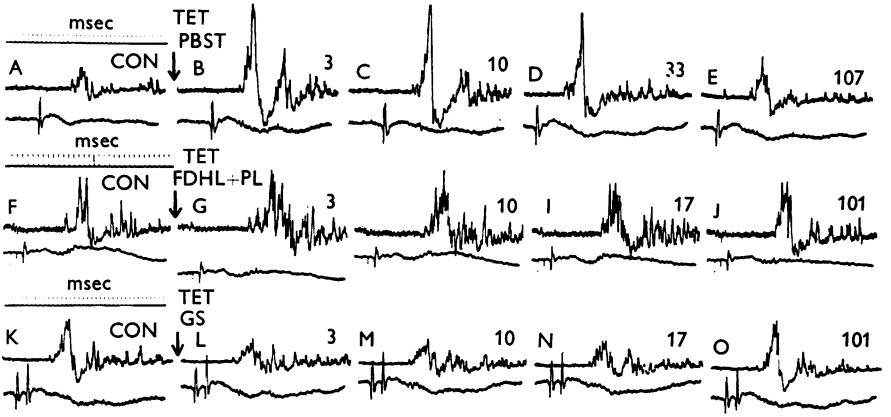

Fig. 4. Post-tetanic potentiation of DRRs. In series A–E a specimen control record is shown (A), evoked by a single PBST volley in GS nerve. Between A and B the standard conditioning tetanus of 300/sec for 15 sec was applied to the PBST nerve, and subsequently DRRs were evoked by testing PBST volleys at the times indicated in seconds after cessation of the conditioning tetanus. In F–J a synchronized PBST + PDP volley was employed to evoke a DRR into FDHL + PL nerve, F being control and G–J showing DRRs evoked at the indicated times in seconds after the end of a conditioning tetanus (300/sec for 15 sec) to the FDHL + PL nerve. K–O was from same preparation as A–E, but two PBST volleys evoked the DRR in GS nerve. The conditioning tetanus (300/sec for 15 sec) was applied as shown to the GS nerve and the times of the subsequent test volleys are shown in msec. Note time scales for each series. Body temperature for A–E and K–O was 31° C, for F–J, 33° C.

post-tetanic potentiation observed for conventional reflexes (Lloyd, 1949; Eccles & Rall, 1951; Wilson, 1955). After the standard conditioning tetanus of 300/sec for 15 sec, potentiation of the DRR persisted for at least 60 sec; no significant potentiation survived at 107 sec in Fig. 4E. The asynchronous nature of the discharge has prevented any accurate assessment of the degree of potentiation and hence no attempt has been made to plot the time course. The initial period of post-tetanic depression has been described for the DRRs evoked by cutaneous afferent volleys (Koketsu, 1956; Eccles & Krnjević, 1959*b*), but post-tetanic potentiation of cutaneous DRRs has not been reported.

When the DRR is large, post-tetanic potentiation is usually not observed with the DRRs from muscle afferents. In two experiments tetanization of a muscle nerve was not followed by potentiation of the DRRs evoked by another muscle nerve. However, further investigation is desirable before it can be concluded that DRRs never exhibit the cross-potentiation sometimes observed with the polysynaptic flexor reflex (Wilson 1955).

When the conditioning tetanus was applied to the muscle nerve in which the DRR was recorded, there was sometimes a fairly large post-tetanic potentiation of the DRR. As is illustrated in Fig. 4F–I, the potentiation was always largest immediately after cessation of the conditioning tetanus, even within 1 sec; all potentiation had disappeared in Fig. 4J at 101 sec. A more common post-tetanic effect has been a lengthening of the DRR, rather than a potentiation, which is seen by comparison between the control in Fig. 4F and the immediate post-tetanic records, G–I. Post-tetanic potentiation of the dorsal root potentials in cutaneous afferent fibres is a well attested phenomenon after tetanization of the recording afferent fibre (Woolsey & Larrabee, 1940; Lloyd, 1952; Eccles & Krnjević, 1959*a*), but no associated increase in the DRR has been described.

Types of muscle afferent fibres producing DRRs

Usually the PBST nerve exhibits an almost complete separation between the thresholds for the Ia and Ib afferent fibres respectively (Eccles, Eccles & Lundberg, 1957*a*; Laporte & Bessou, 1957). The best test of this separation is provided by the double-stimulation technique, where only those Group I afferent fibres not excited by the first stimulus can respond to a second stimulus that is just supramaximal for Group I and given about 1 msec later. Thus in Fig. 5A, B the first stimulus excited a large fraction (about two thirds) of the Group Ia fibres when at strengths of 1·38 and 1·64T respectively; yet, as shown in the corresponding records from the lateral gastrocnemius nerve, there was only a trace of DRR in both cases. The next strongest stimuli in Figs. 5A, B (1·70 and 1·85T respectively)

were maximal for the Group Ia fibres, but excited very few Ib fibres. There was correspondingly a large increase in the DRRs, which presumably was due to the considerable addition to the Ia volley, rather than to the few Ib impulses. However, the increase to a maximum Ib volley, with stimulus strengths of 2·0 to 2·46T in A and 2·06 ot 3·06T in B, further increased the DRR. Thus it can be concluded that in Fig. 5 the DRR was evoked by both the Ia and Ib afferent volleys. In Fig. 5A the large DRR at 1·70T indicated that the Ia volley was more powerful than the Ib, though the Ia volley had to be about two thirds maximal before it evoked any DRR. The relative potency of the PBST Ia volley in this same experiment is also illustrated by comparing the upper with the second row of records in Figs. 6 and 7. In other preparations (cf. Fig. 5B) the Ib volley appeared to be more powerful than the maximum Ia volley, the 3·06T response being compared with the 1·85T response. However,

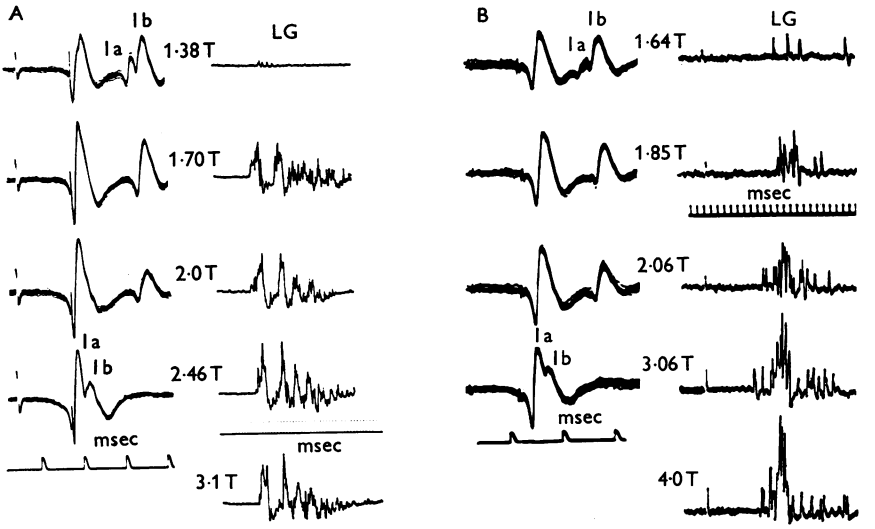

Fig. 5. Types of Group I impulses producing DRRs. In A single stimuli at the indicated strengths relative to threshold are applied to the PBST nerve and the dorsal root reflexes into LG nerve are in the right-hand column. In the left-hand column are, first, records produced by the PBST volley as it enters the cord through the L 7 and S 1 dorsal roots, and secondly, the responses evoked by a second stimulus applied 1·3 msec later through the same electrode on the PBST and at a strength just supramaximal for Group I. For that stimulus strength the interval is briefer than the refractory period, so it only excites fibres not excited by the first stimulus, which is thus shown to be maximal for Group I at 2·46T, and respectively submaximal and maximal for Group Ia at 1·38 and 1·70T. Time scales are shown for each column. Body temperature 31° C. B is a similar series from another experiment with body temperature 34° C. It is to be noted that in A and B the DRRs were evoked by the single stimuli that gave the first responses in the dorsal root records. These latter records were taken just subsequent to the DRR series.

in producing DRRs the requirement of summation is so dominant that the relative magnitudes of the Ia and Ib contributions cannot be derived from series such as those illustrated; nevertheless, they establish that both the Ia and Ib afferent impulses from muscle are concerned in the production of DRRs.

An invariable finding has been that increasing the stimulus beyond the maximal strength for Ib caused little or no further increase in the DRR, either in intensity or duration. For example, the stimulus strengths of 3·1T and 4·0T in Figs. 5A and B respectively would have excited many of the Group II afferent fibres, yet there was no appreciable increase in the DRRs. Evidently Group II and III afferent impulses from muscle are virtually ineffective in evoking DRRs in the large muscle afferent fibres.

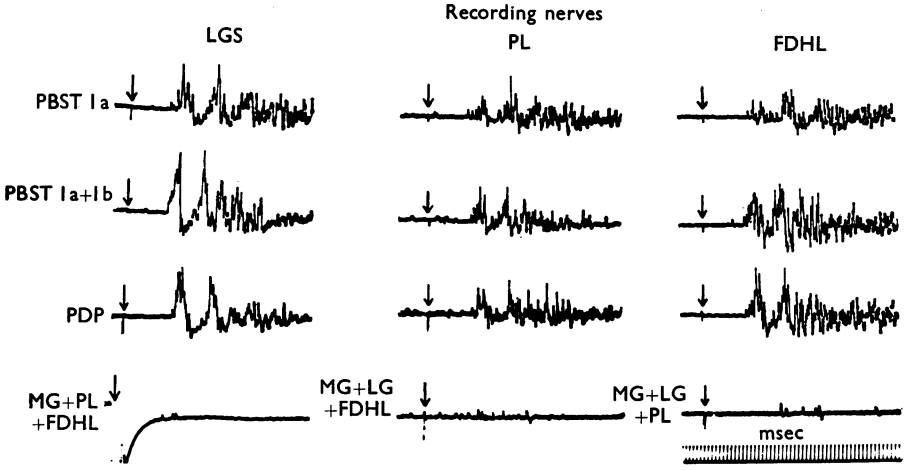

Fig. 6. DRRs into nerves to the post-tibial extensor muscles. The respective recording nerves are specified above the columns and volleys producing the DRRs are indicated to the left of the top three rows. In the lowermost row the volleys are different for each column, as shown, being applied in each case through the three nerves to the post-tibial extensor muscles that were not used for recording. Stimuli just maximal for Group I were applied to the PDP, MG, LG, PL and FDHL nerves. Body temperature 31° C.

Hitherto the general properties of the DRRs discharged into muscle afferent nerves have been illustrated with examples in which afferent volleys in the posterior biceps-semitendinosus (PBST) or peroneal (PDP) nerves evoked DRRs into the LGS, PL or FDHL nerves, as illustrated in the three upper rows of Fig. 6. Such DRRs are amongst the most readily evoked. When a systematic study was made of the DRRs discharged into many species of muscle nerves in response to many species of muscle afferent volleys, it was found that other muscle afferent volleys also could

produce DRRs, though they were always less effective than the PBST or PDP volleys. It was also found that any particular muscle afferent volley would produce DRRs in any muscle nerve with an approximately equivalent effectiveness, even into itself. For example, in Figs. 6 and 7 PBST volleys evoked large DRRs in LGS, PL, FDHL, P, and DP nerves. The smaller responses of the P nerve are at least in part attributable to the shunting by the superficial peroneal nerve which was left attached, but cut centrally.

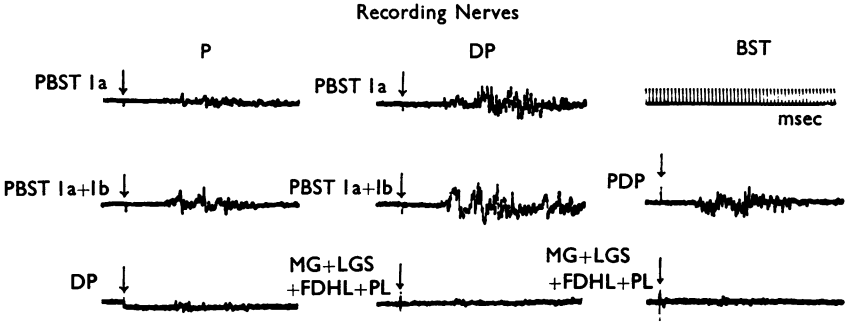

Fig. 7. DRRs into nerves to flexor muscles. The series are arranged as in Fig. 6, with recording nerves specified above each column, while the volleys producing the DRRs are specified just before each record. Except for the first two PBST volleys all volleys are evoked by stimuli just supramaximal for Group I. Body temperature 31° C.

The much lower effectiveness of Group I afferent volleys in the post-tibial extensor muscles (GS, PL and FDHL) is seen in the lowest row of Fig. 6, where there are barely detectable DRRs in contrast to the large DRRs produced in the same muscle nerves (LGS, PL or FDHL), by the PBST or PDP volleys. Similarly, in the lowest row of Fig. 7 a combined afferent volley from these three post-tibial extensor muscles produced an extremely small DRR into DP or BST nerves, which contrasted with the large DRRs produced by PBST or PDP volleys in the row above.

Since a considerable summation of excitatory actions is required in order to evoke a DRR, a more sensitive test for the effectiveness of an afferent volley would be to superimpose it upon a conditioning afferent volley that alone produced a DRR. For example, in the series of Fig. 8 a single PBST volley was employed for this purpose and superimposed thereon in C, G, K and O were single volleys in PDP, vastocrureus (VC), SMAB and GS nerves respectively. It is seen that each of these volleys increased the DRR above the PBST response in A, E, I, M, though with each alone (B, F, J, N) there was an appreciable DRR only to the PDP volley. It is further shown in D, H, L, P that brief repetitive stimulation (6 volleys at 280/sec) was much less effective in displaying the ability to produce

DRRs than was summation with the PBST volley. By this method of summation it has been shown that Group I afferent volleys from all tested hind-limb muscles share to some extent the ability to produce DRRs in the muscle nerves of that limb. It has further been found that this effectiveness was not appreciably diminished when there was segmental separation between the afferent volleys and the muscle nerve in which the DRR was recorded. For example, PBST volleys entering the cord at the L7–S1 level were very effective in evoking DRRs in the afferent fibres of vasto-crureus muscle, which have a segmental level of L5 to upper L6.

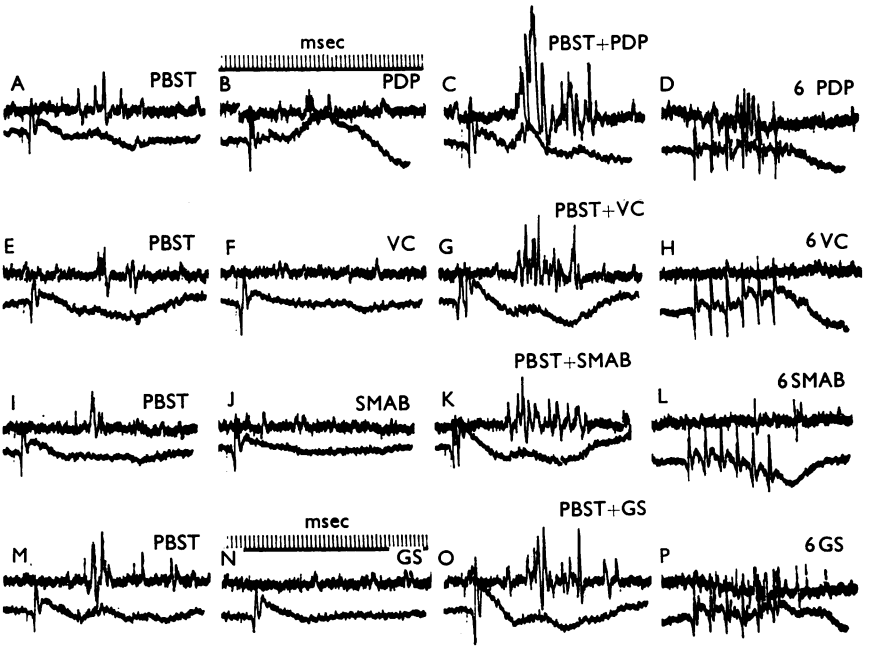

Fig. 8. DRR production by maximum Group I afferent volleys from different muscles. A, B and C show DRRs into the FDHL nerve produced by single PBST, PDP and combined PBST and PDP volleys respectively. There is a large DRR in C and in D a much smaller DRR is produced by 6 PDP volleys at 280/sec. Note the dorsal root records immediately below the DRR records. E–H, I–L and M–P are similar series for the VC, SMAB and GS nerves respectively instead of PDP. Body temperature 32° C. Same time scale for all records.

In the cool cord Brooks & Koizumi (1956) found that cutaneous afferent volleys caused the discharge of DRRs into muscle afferent fibres and reciprocally that muscle afferent volleys produced DRRs in cutaneous nerves (cf. Eccles & Krnjević, 1959*b*). The present investigation has confirmed these earlier observations. For example, in Fig. 9A, B volleys in the cutaneous afferents, superficial peroneal (SP) and sural (S) evoked

DRRs into the FDHL nerve, and, in Fig. 9C, SP + S evoked a large DRR, though not so large as that produced by the most effective combination of muscle afferents in Fig. 9D. Muscle afferent volleys were less effective in evoking DRRs into cutaneous afferent nerves, but in Fig. 9E, F there were small DRRs both from VC + FDHL + PL + GS, and from PBST + PDP, into sural nerve. In contrast, SP produced a very large DRR (G), which was not appreciably increased by summation with a FDHL + PL + GS afferent volley (H). Possibly the DRRs of E and F were due to Group II

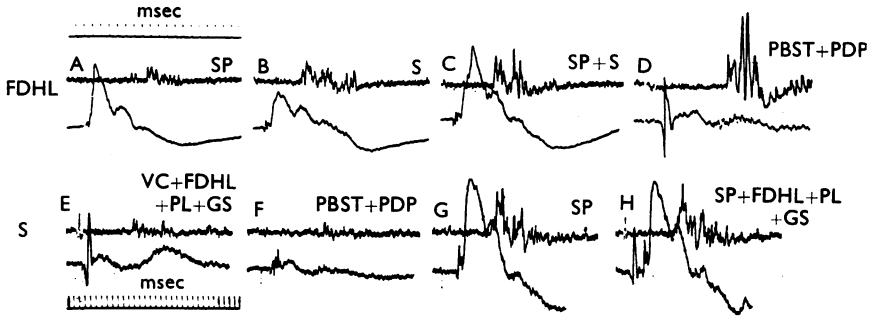

Fig. 9. Interaction between cutaneous and muscle afferent pathways in production of DRRs. A–D, recording of DRRs from FDHL nerve and stimulation of SP, S, SP + S and PBST + PDP as indicated. Time scale above A. E–H same preparations as A–D, but recording from sural nerve (S) and generating single volleys in the various cutaneous and muscle nerves as indicated: time scale below E. Temperature of body, 34·5° C.

afferent impulses from the muscles. Certainly, with DRRs into cutaneous nerves, PBST + PDP do not exhibit the much greater potency that obtains when their Group I afferent volleys produce DRRs in muscle nerves (cf. Figs. 6, 7, and 8). It is possible that it is the Group II components of the muscle nerves that evoke the small DRRs into cutaneous nerves (Fig. 9E, F); further investigation is required.

Type of muscle afferent fibre responding in the DRR

Toennies (1938) showed that DRRs occur in myelinated cutaneous afferent fibres ranging from the largest down to the delta fibres. Brooks & Koizumi (1956) also found that relatively small medullated fibres were concerned both in the inward and outward paths of the DRR. Fibres with conduction velocities below 30 m/sec were apparently not involved in DRRs into mixed cutaneous and muscle nerves such as peroneal or posterior tibial. Three different methods have been employed in attempting to identify the muscle afferent fibres conveying the impulses of the DRR.

(1) The outgoing impulses of the DRR will block afferent impulses travelling in the opposite direction in the same fibres. If an afferent volley

is set up peripherally in the muscle nerve at an appropriate time relative to the discharge of the DRR, there will be a deficit in its spike as recorded by an electrode in contact with the dorsal root. For example, in Fig. 10A the spike produced by a Group I afferent volley from GS nerve was only about half the control size when it was evoked by a stimulus applied 11·7–15·5 msec after a PBST afferent volley; hence the DRR must have discharged along about half of the Group I afferent fibres. When the stimulus to the GS nerve was weakened so that it would be exciting mainly Group Ia fibres (Eccles *et al.* 1957*a*), the spike in the dorsal root was almost completely abolished over the same range of intervals (Fig. 10B).

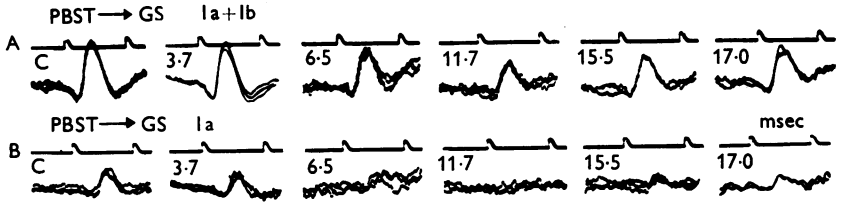

Fig. 10. Blockage of afferent volleys by DRR. In A a stimulus maximal for Group I was applied to the gastrocnemius nerve giving the control spike C in the L7 dorsal root as it enters the cord. In the other records it was preceded by a maximum PBST volley at the intervals indicated in msec. Note the depression of the GS spike at all testing intervals from 6·5 to 17·0 msec, with a maximum depression at 11·7 msec. B is a similar series, but the stimulus to the gastrocnemius nerve was weakened so that it would excite virtually only Group Ia fibres. Body temperature 33·5° C.

Evidently the surviving dorsal root spike in Fig. 10A was in the higher threshold component of Group I, i.e. the DRR was predominantly in the Group Ia afferent fibres of the GS muscle and to a lesser degree in the higher threshold Group Ib fibres. Similar results were invariably obtained in this type of experiment, but it must not, therefore, be concluded that DRRs do not occur in Ib afferent fibres. The threshold discrimination between Ia and Ib afferent fibres is not sufficiently good to justify any such exclusive statement (cf. Eccles *et al.* 1957*a*; Laporte & Bessou, 1957).

(2) An alternative method for identifying the afferent fibres conveying outgoing impulses in the DRR is to record the DRR in them by an intracellular electrode, as has been done previously for cutaneous afferent fibres (cf. Frank & Fuortes, 1955; Koketsu, 1956; Wall, 1959; Eccles & Krnjević, 1959*b*). The origin of the impaled muscle afferent fibre has been determined by stimulating the various muscle nerves and finding the one which directly generated a spike potential. This test invariably gave an unequivocal identification. There was no example of an afferent fibre in the spinal cord that was directly activated from two different muscle

nerves. Eight of the 16 Group Ia fibres sampled by this technique exhibited DRR discharges, as is seen in Fig. 11C–E. Usually there were only one or two discharges, which tended to occur at certain preferred intervals. For example, in Fig. 11E there was a single DRR discharge at the time of the second discharge in C and D. DRRs were also observed in 4 of the 23 muscle afferent fibres that were identified as being in Group Ib both by their high threshold and slightly slower conduction velocity.

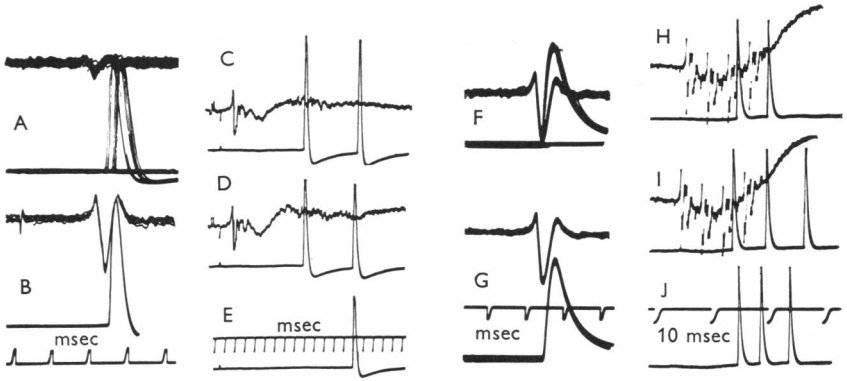

Fig. 11. Recording of DRRs by a micro-electrode inserted into primary muscle afferent fibres in the spinal cord. A–E, records from afferent fibre that was presumed to be Ia type on account of its low threshold as shown in record A, where stimulus just-threshold for the fibre (note failure of spike in many of the superposed tracings) excited also only a small afferent volley, as shown in upper record of A, negativity being recorded downwards. B shows a maximum Group I volley. In C–E are DRRs produced in that fibre by a combined PBST+PDP volley. Dorsal root records are shown, above the intracellular records with their two DRR spikes, in C and D. In E the same volley produced only a single DRR impulse at the time of the second impulses in C and D. Note separate time scales for A, B and for C, D, E. F–J represent a similar series to A–E, but for a presumed Ib afferent fibre from gastrocnemius, because as shown in F its threshold stimulus was almost maximal for Group I, as may be seen by comparing the dorsal root spikes in F and G. In H–J two or three impulses were generated in this fibre by a brief tetanus of 4 PBST volleys at 270/sec. Body temperatures were 31° C for A–E and 36° C for F–J.

For example, in Fig. 11, F and G provide evidence for the identification as a high-threshold gastrocnemius Ib fibre and in H–J repetitive DRRs are seen to be evoked by 4 PBST afferent volleys at 270/sec. In addition DRRs were observed in 5 of the 9 fibres that could have been either Ia or Ib. Three Group II muscle afferent fibres were also impaled and 2 of these showed DRR discharges. It can be concluded that DRRs may occur into any of the large afferent fibres of muscle, though the Group Ia fibres may be predominantly involved.

(3) If the DRR occurs in Group Ia afferent fibres, it would be expected that monosynaptic EPSPs would be produced in the appropriate motoneurones. For example, in Fig. 12B PBST volleys of the strengths and composition indicated in the three tests of Fig. 12A evoked the discharges of DRRs in the medial gastrocnemius nerve. Immediately above these DRRs are intracellular records simultaneously observed from a gastrocnemius motoneurone. It will be seen that motoneuronal depolarization began abruptly less than 1 msec before the onset of the DRR spikes in the peripheral nerve. This is precisely the time relation to be expected for the monosynaptic production of EPSPs by Ia impulses that are generated in the spinal cord close to the presynaptic terminals on the motoneurones.

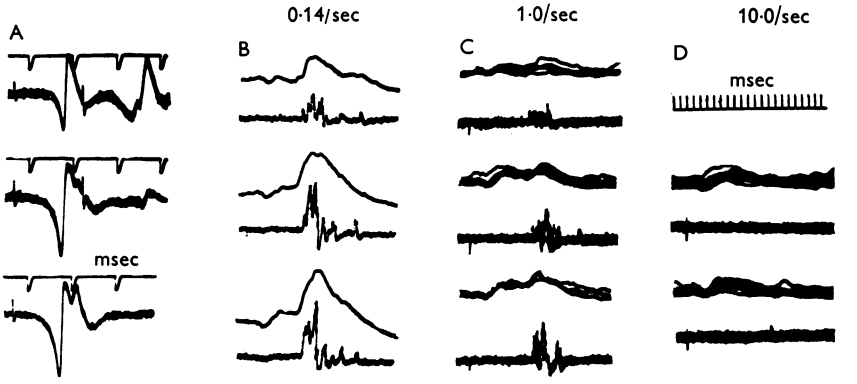

Fig. 12. EPSPs of motoneurones produced by DRRs. In column A are shown tests, as in Fig. 5, for the composition of PBST volleys generated by stimuli of strengths 1·5, 2·1 and 2·8 times threshold respectively. In columns B–D the upper traces are intracellular records from a LG motoneurone, while the lower traces are DRRs recorded from the MG nerve, for these three sizes of afferent volleys (cf. Fig. 5). Note that the principal wave of motoneurone depolarization begins less than 1 msec before the DRR appears in the peripheral nerve (see text). With column B the repetition frequency was 0·14/sec; while with C and D it was 1·0 and 10·0/sec respectively and the records consist of superposed sweeps. Further description in text. Body temperature 34·5° C.

This identification was confirmed by the tests at higher frequency of repetition in Fig. 12C and D. At 1/sec the DRR was much diminished and, correspondingly, so was the motoneuronal depolarization that occurred simultaneously. At 10/sec there was no DRR and the intracellular records show only the small earlier depolarization which also occurred in B and C, and which was probably due to the polysynaptic excitatory action of the PBST afferent volley. This method of testing has certainly confirmed that DRR discharges occur in Group Ia muscle afferents, but so far it has provided no evidence for or against the participation of Group Ib fibres in the DRR.

Recurrent impulse discharges into muscle nerves

When stimulating one muscle nerve and recording from another, it has sometimes been observed that a muscle afferent volley evoked the discharge of an impulse in a muscle afferent fibre by an entirely different mechanism from that producing the DRR. This discrimination was never in doubt, for it was made on the grounds both of the fixed latency of response and of the high frequency at which the discharge followed repetitive afferent volleys, even up to 300/sec or more, whereas the DRR fails even at 10/sec (Fig. 3). A further feature of recurrent discharges is that they often occur reciprocally, i.e. they still appear after reversal of the stimulating and recording nerves. This recurrent discharge may be attributed either to the existence of a branched axon, such as has been postulated by Barron & Matthews (1935) and Habgood (1953), or to ephaptic transmission in the spinal cord. With most of the recurrent discharges the delay in the spinal cord has been about 1·5–2 msec, which is just sufficient for conduction up to and down from the level of the cord section; hence such recurrent discharges are most likely attributable to ephapses made by the cord section (Renshaw & Therman, 1941). However, there have been three examples with central latencies too brief for such a pathway. For example, in Fig. 13 the conduction time was 3·1 msec whether it was measured for conduction from FDHL to PBST (D) or reciprocally (B). The threshold tests A and C show that the recurrent impulse was generated by Ib and Ia afferent impulses in the PBST and FDHL nerves respectively. The shortest conduction times up to the recording electrode on L7 DR at its cord entry were 1·7 msec for the FDHL Group I volley and 1·05 msec for the PBST Ib afferent volley; hence the central delay for the discharge of the recurrent impulse was not more than 0·35 msec. Transmission from one fibre to the other must have occurred close to their entry into the spinal cord, either by some ephaptic contact or by branching. As previously mentioned there was no example of an impaled fibre that could be activated directly from two muscle nerves, but only 51 fibres were impaled, whereas the few recurrent discharges were picked out from observations on many thousands of Group I muscle afferent fibres. The rarity of recurrent discharges with short central latency was also reported by Habgood (1953), there being only two examples in at least 2000 large afferent fibres. Possibly these recurrent discharges occur in collateral branches of primary afferent fibres, as postulated by Barron & Matthews (1935), but the existence of such branches has been doubted on histological grounds (Westbrook & Tower, 1940). A remote possibility that has not been excluded is that these brief-latency recurrent discharges are occurring not in afferent fibres, but in the motor fibres through ephapses that were

made by the ventral root section in the initial preparation (D. R. Curtis, personal communication). It would, however, be surprising if a motor fibre had the very low threshold exhibited in Fig. 13C.

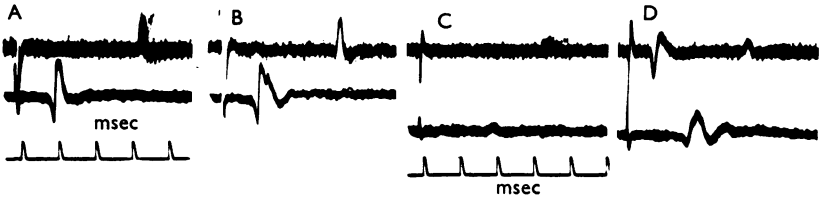

Fig. 13. Recurrent discharges between muscle afferent fibres. In A and B stimuli were applied to PBST nerve and the upper records are from FDHL nerve. With a maximal Group I volley there is a spike in FDHL nerve with a latency of 3·1 msec. As shown in the dorsal root record of A, the threshold stimulus for producing this spike was in the Ib threshold range. Immediately afterwards the stimulating and recording electrodes were reversed and reciprocal conduction with exactly the same latency is seen in D, while in C the threshold for the fibre in the FDHL nerve is seen to be in the low-threshold Ia range. Note that all records are formed by the superposition of many faint traces and there is a small latency range with the just-threshold stimulation in A and C, which is attributable presumably to variations in latency at the site of stimulation. Body temperature 38° C.

DISCUSSION

The demonstration of the widespread incidence of dorsal root reflexes in muscle afferent fibres reveals a hitherto unsuspected richness of interaction between these fibres in the spinal cord. Admittedly in the present investigation this could be fully demonstrated only in the cooled animal. However, the generation of impulses is a threshold phenomenon; in some manner not yet understood (cf. Brooks *et al.* 1955; Brooks & Koizumi, 1956) cooling lowers the threshold for the reflex discharge of impulses from the spinal cord along both ventral and dorsal roots. Thus the DRRs of the cooled preparation give evidence that at normal body temperature there is a subthreshold depolarization of the primary afferent fibres of muscle. Indeed, even at normal or raised body temperature this depolarization occasionally was adequate to generate the discharge of impulses (Figs. 1A–C, G–I; 2A–D; 3I–L). More direct evidence for this depolarization will be provided in later papers, but the present investigation is of value because it demonstrates the existence of the depolarization in the absence of experimental interference with the spinal cord.

The most interesting feature is the much greater effectiveness of the PBST and PDP volleys in producing DRRs. It is not possible to attribute this to the greater population of Group I fibres in the nerve to these flexor muscles. According to Rexed & Therman (1948), Group I fibres form a larger proportion of the afferent fibres of extensor muscles than of flexor

muscles (cf. Lloyd & Chang, 1948), and this preponderance extends also to the proportion measured relative to the total afferent plus efferent fibre population; hence the potency of Group I flexor afferents in producing DRRs must be due, not to numerical dominance, but to the more effective central action of the individual fibres. For example, the Group I afferent volley from the whole assemblage of post-tibial extensor muscles (GS + PL + FDHL) is much larger than that from PBST, and still larger than that from PDP, yet it was always much less effective in evoking DRRs (Figs. 6, 7, 8). Even at a cord temperature as low as 30° C a maximum Group I volley in the GS + PL + FDLH nerves produced either no DRR or a DRR in very few afferent fibres of any other muscle nerve, whether a flexor or extensor. Group I volleys in the nerves to the hip and knee extensor muscles (SMAB and Q respectively) were also relatively ineffective in generating DRRs (Fig. 8E–L). As an approximate estimate, it may be stated that for Group I volleys of the same size a volley from flexor muscles is more than three times as effective in evoking a DRR as that from extensor muscles. Possibly, this relative potency of Group I volleys from flexors is related complementarily to the relative impotence of flexor Group Ib volleys in acting on motoneurones through polysynaptic pathways (Eccles, Eccles & Lundberg, 1957*b*); their central pathway is channelled more to interneurones concerned in the DRR than to those acting on motoneurones. It should be pointed out that the production of DRRs from hip flexor nerves has not been investigated, though investigations of the potentials which such volleys generate in the cord and in the dorsal roots indicate that they would be less effective in evoking DRRs than the knee or ankle flexors (Eccles, Magni & Willis, unpublished observations).

Discussion of the present experimental results on interaction between afferent volleys in the same or different muscle nerves is best postponed until they can be correlated with experimental observations on depolarizations of the Group I afferent fibres in the spinal cord (Eccles, Magni & Willis, unpublished observations). The abrupt cessation of the DRR soon after the onset of a prolonged repetitive stimulation (Fig. 2J–M) is probably attributable in part at least to accommodation of the primary afferent fibre to a continuous depolarization. The depression of successive responses even at repetition rates as slow as 0·2–0·5/sec (Fig. 3) requires a more complex explanation. An important factor probably is the long time needed for full recovery of propagation through interneuronal pathways. It is expedient to defer further discussion to a later paper dealing with the interneuronal pathways. Discussion of the post-tetanic potentiations of DRRs, as illustrated in Fig. 4, will also be postponed to the later paper dealing with the depolarization of primary afferent fibres.

Finally, it can be stated that there is no evidence that DRRs are produced under normal physiological conditions by the discharges of the annulospiral endings or Golgi tendon organs. Their present significance derives from the evidence they provide for central interaction between muscle afferent fibres. It will be shown in subsequent papers that this central mechanism is of great interest and importance in relation to the control of reflex activity of the spinal cord.

SUMMARY

1. Particularly in the cooled animal (cat) muscle afferent volleys evoke reflex discharges into muscle afferents, which in general resemble the dorsal root reflexes (DRRs) of cutaneous afferents.

2. At brief intervals volleys sum in the production of DRRs, but beyond 10 msec there is a prolonged depression, so that rapid repetitive stimulation evokes only a transient DRR and DRRs are depressed when evoked at frequencies higher than 0·2/sec.

3. After prolonged high-frequency stimulation there is usually post-tetanic potentiation for at least 60 sec. Post-tetanic potentiation is also observed after tetanization of the afferent carrying the DRR.

4. Both Ia and Ib afferent volleys are effective producers of DRRs, Groups II and III having little or no action. Knee and ankle flexor afferents are much more effective than afferents from extensor muscles.

5. The DRRs occur equally well into the large afferent fibres of flexor and extensor muscles, but there is evidence that the Group Ia fibres are more involved than the Ib.

6. Preliminary observations show that in the production of DRRs there is also some reciprocal action between cutaneous and muscle afferent fibres.

7. The DRRs are readily distinguished from the very rare recurrent discharges from one afferent fibre to another. These discharges are due either to an ephapse or to branching.

8. The preponderant action of flexor afferent volleys is attributable not to numerical dominance of fibres, but to the more effective action of the individual flexor afferents. There is a preliminary discussion also of other features of the central interaction between muscle afferent fibres.

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