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SYNAPTIC ACTIONS ON MOTONEURONES IN RELATION TO THE TWO COMPONENTS OF THE GROUP I MUSCLE AFFERENT VOLLEY

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It was first suggested by Hoffmann (1922) that the central pathway for tendon reflexes (Eigenreflexen) was monosynaptic, being formed by the large reflexomotor collaterals of dorsal root fibres (cf. Cajal, 1909, figs. 113, 115). With the development of electrical techniques it was possible not only to confirm the monosynaptic nature of tendon reflexes, but also to show that they are mediated by very large afferent fibres conducting as fast as 116 m/sec (Lloyd, 1943*a*, *b*). It has further been shown that the afferent impulses from a muscle exert a monosynaptic excitatory action on a very restricted group of motoneurones, namely the motoneurones of that muscle and of muscles synergic with it (Lloyd, 1946; Laporte & Lloyd, 1952). Subsequent investigations have largely been concerned with the attempt to determine the type of receptor organ connected with these large afferent fibres, i.e. to decide between annulo-spiral endings and Golgi tendon organs, which together contribute almost all of the large afferent fibres (the so-called group I fibres) in muscle nerves (Sherrington, 1894; Matthews, 1933; Barker, 1948; Hunt, 1954). Attempts by Granit (1950, 1952), McCouch, Deering & Stewart (1950), Granit & Ström (1951), Hunt (1952) and Job (1953) specifically to excite Golgi tendon organs have uniformly failed to reveal any synaptic excitatory action that conforms with the pattern of group I monosynaptic excitation. On the other hand, with activation of muscle spindles and hence presumably of annulo-spiral endings by tetanization of the small motor fibres, i.e. the gamma efferents, Hunt (1952) obtained excitatory action on homonymous as well as on synergic motoneurones.

The effects exerted on motoneurones by impulses from Golgi tendon organs have been investigated, both with the use of adequate stimulation and with group I volleys evoked in the severed muscle nerve. Laporte & Lloyd (1952) carried out an extensive study utilizing the latter technique, and found that

group I fibres could reflexly inhibit homonymous and synergic motoneurones as well as facilitate antagonists. They associated this 'inverse myotatic reflex mechanism' with activity in tendon organ afferents. Adequate activation of Golgi tendon organs (Granit, 1950, 1952; McCouch *et al.* 1950; Granit & Ström, 1951; Hunt, 1952; Job, 1953) provided evidence that impulses from these receptors in a muscle inhibited motoneurones supplying that muscle, i.e. that they exerted an autogenetic inhibitory action (cf. Granit, 1955).

It has been reported (Bradley & Eccles, 1953) that, when afferent volleys were set up in the quadriceps and hamstring nerves of cat and recorded from the appropriate dorsal root, the group I spike was almost invariably double. By making use of the relatively large threshold differential between the afferent fibres contributing respectively to these two spike components, it was further shown that the fast spike was approximately proportional both to the monosynaptic facilitatory action on a synergic reflex and to the direct inhibitory action on reflexes to antagonist muscles. On the basis of this finding, it was suggested that the fast low-threshold component is composed of the fibres from annulo-spiral endings, whereas the slow high-threshold component comprises the fibres connected to Golgi tendon organs.

Intracellular recording from motoneurones provides the most direct method of evaluating synaptic action (Brock, Coombs & Eccles, 1952; Coombs, Eccles & Fatt, 1955*a–d*). It has been used in this present investigation, which has been designed to discover how far components revealed by velocity and threshold measurements on group I fibres correspond to the two functional subdivisions, those afferents from annulo-spiral endings and those from Golgi tendon organs. The basic assumption for the present work has been the generally accepted hypothesis, as outlined above, that the large afferent fibres from annulo-spiral endings (group Ia) are the only group I fibres responsible for monosynaptic activation of motoneurones, and that, with the exception of the direct inhibitory action of group Ia impulses on motoneurones to antagonistic muscles, all inhibitory actions of the group I impulses are due to the Golgi tendon organ afferents (group Ib). In order to avoid confusions in terminology, we will provisionally refer to the fast (low-threshold) and the slow (high-threshold) components of group I as the F and S components respectively. The relationship of these two components to groups Ia and Ib will be examined in the Discussion.

METHODS

The experiments have been performed on cats under light pentobarbital anaesthesia.

The micro-electrode technique for intracellular recording from motoneurones has been described by Brock, *et al.* (1952) and by Eccles, Fatt, Landgren & Winsbury (1954). For recording of inhibitory potentials the micro-electrodes were filled with a solution of 0·6 M-K_2SO_4 (cf. Coombs, *et al.* 1955*b*). All records are formed by the superposition of about 25 faint traces.

RESULTS

The double spike response of group I afferent volleys

The spike potentials produced by group I afferent volleys from quadriceps and hamstring nerves have shown a wide range of variation in different preparations. Occasionally only a simple group I spike is found, but sometimes the separation of the group I spike into the two components, F and S, has been as extreme as in Fig. 1, where there was even a double spike in the monophasic

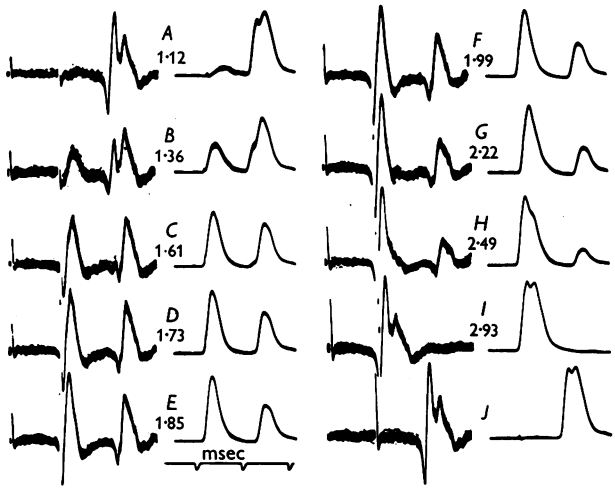

Fig. 1. Dorsal root recording of group I afferent volleys from biceps posterior and semitendinosus (BST) nerves (superimposed tracings). The corresponding records show, to the left, the triphasic spike and, to the right, the monophasic spike from the severed dorsal root, negativity being upwards. In each record two stimuli were applied to the nerve at an interval of 1·0 msec so that the fibres excited by the conditioning stimulus were refractory to the testing stimulus. The testing stimulus was supramaximal and kept constant throughout the series (response to testing stimulus alone shown in record *J*). For the conditioning volleys the stimulus strengths relative to threshold strength are indicated on the records.

record (*J*) as well as in the triphasic recording by a surface electrode. The complete series of triphasic responses that is partly illustrated in Fig. 1 *A–J* were recorded and then the dorsal roots were divided and mounted for recording the series of monophasic responses that is also partly illustrated in *A–J*. For ease of comparison the corresponding members of the two series have been mounted as pairs.

In this series of Fig. 1 the usual procedure was adopted of applying double stimuli to the muscle nerve (cf. Bradley & Eccles, 1953, figs. 2, 3 and 5), the first or conditioning stimulus being varied in strength while the testing stimulus was at least 50 % supramaximal for group I (*J* gives control response to second alone) and at an interval (1·0 msec) which was just sufficiently brief

to prevent the testing stimulus from exciting any fibres that had already responded to the conditioning, as is shown with a maximum conditioning stimulus in record *I*. Thus, with each of the complex spike potentials of Fig. 1, every group I fibre will have been excited once, if not by the conditioning then by the testing stimulus. It will be seen that, with increasing strength of the conditioning stimulus from *A* to *D*, there was a progressive diminution in the F spike of the testing response, so that it was barely detectable in *D*, while with *E* no trace remained. Correspondingly, the conditioning stimulus set up an F spike of progressively increasing size, though in *E* it still was a little lower than the control sizes (*J*) both for triphasic and monophasic recording. In contrast, the S spike of the testing response (triphasic recording) suffered very little change over the range of stimulus intervals from *A* to *E*. Actually there was a small increase from *A* to *C*, but this was attributable to the diminution in the terminal positive phase of the F spike, which would be superimposed on it. The virtual equivalence of *C* and *D* indicates that the conditioning stimulus had not excited any of the S fibres until its strength was increased beyond that of *D*. Already in *E* the S spike of the testing response was very slightly diminished, both with triphasic and monophasic recording, and this effect continued with increase in the stimulus until none survived in *I*. *Pari passu*, in records *F* to *I*, the conditioning stimulus was exciting progressively more S fibres. Evidently the most interesting responses occurred in *D* and *E*, where the conditioning stimuli excited virtually all of the F but none of the S fibres.

The effectiveness of the conditioning stimulus in setting up F impulses cannot simply be assessed by the size of the F spike, for it will be diminished by two overlapping factors. One operates with conditioning stimuli that are not considerably stronger than the maximum for F fibres. The other operates only with triphasic spike potentials and with conditioning stimuli that are strong enough to excite S impulses. This second factor is due simply to the superposition of the initial positive phase of the S spike on the summit of the F spike. The first factor depends on the well-attested observation that, when a stimulus is applied to a nerve fibre at just above threshold strength, the initiation of an impulse occurs after an abnormally long delay, up to 0·3 msec (Blair & Erlanger, 1933). Hence, until the stimulus is considerably above threshold for all fibres of one group, i.e. at well above maximum strength, there will be a considerable asynchronism in the initiation of impulses. Consequently the size of the F spike will give too low an index of the number of F fibres excited by the conditioning stimulus in records *A–E*. As would be expected, the asynchronism has less effect in depressing the size of the relatively longer monophasic F spike, though it is still considerable. For example, with the monophasic testing responses of *A–D*, direct measurements of the F spikes can be made and added on to the corresponding F spikes of the conditioning responses to give the total size of the F spike potential. With *A*, *B*, *C* and *D* the respective totals were only 90, 80, 87 and 90% of the F spike in the control response (*J*). In *E* no F spike was detectable in the testing response, yet in the conditioning response it was only 93% of the control, and it was still a little depressed even with the stronger conditioning stimuli in *F* (96%) and *G* (98%). This detailed analysis of the afferent spike potentials will be applied later in an attempt to evaluate the synaptic actions by the F and S volleys.

The relationship of the group I volley to monosynaptic excitatory action

When, as in Figs. 1 and 4, it was possible to adjust the strength of stimulation applied to a muscle nerve so that it was maximum for the F fibres and yet subthreshold for the S, conditions were particularly favourable for discrimination between the synaptic actions of the F and S volleys. Fig. 2 is chosen for special consideration from a large number of similar investigations, because it was recorded from the same muscle nerve as Fig. 1, but a few hours earlier when conditions were even better for separation of the two components.

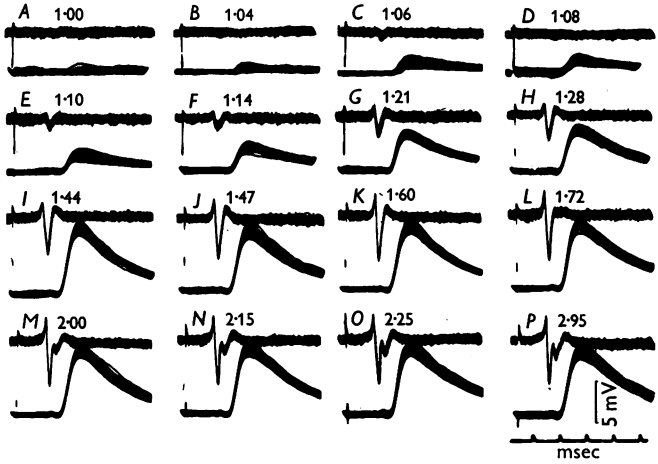

Fig. 2. Relationship of monosynaptic EPSP to the two components of the group I volley. Simultaneous recordings (superimposed tracings) of triphasic volley at the S1 dorsal root entry zone and intracellular potential of a BST motoneurone. Observe that, in the triphasic recording, negativity unconventionally is signalled by a downwards deflexion. Stimulus strengths relative to threshold strength are indicated on the records.

It will be seen that a very small excitatory post-synaptic potential (EPSP) was generated by the smallest afferent volley that could be recorded from the dorsal root (*B*), and that with increasing strengths of stimulation there were parallel increases in the F volleys and the EPSP's. The monosynaptic nature of the EPSP was established by its brief latent period (0·5 msec) and simple time course. It can also be seen that in record *I* the EPSP reached a maximum size with a strength of stimulus that was subthreshold for the S fibres, record *K* being just at threshold for the latter. The detailed relationships of the F and S volleys to the monosynaptic EPSP's are shown in the plotted measurements of Fig. 3. Several features call for comment. With each stimulus in Fig. 2 the superimposed traces of EPSP's showed an appreciable range of variation, and correspondingly in Fig. 3 each EPSP is plotted not by a point, but by a line corresponding to the range of variation. The EPSP is seen to have a threshold identical with that for the F volley and to increase to a maximum height with

a stimulus strength of 1·44 times threshold (Record *I*, Fig. 2). More complex behaviour was exhibited by the F spike in the upper graph of Fig. 3, as measured from the triphasic surface potentials (cf. upper traces of Fig. 2). It continued to increase in size with stimulus strengths up to 1·8, and then declined again during a further increase in stimulus from 2·3 to 2·5 times threshold. This decline was also observed for the F spike in Fig. 1 and was sufficiently explained by the superposition of the initial positive phase of the triphasic S spike.

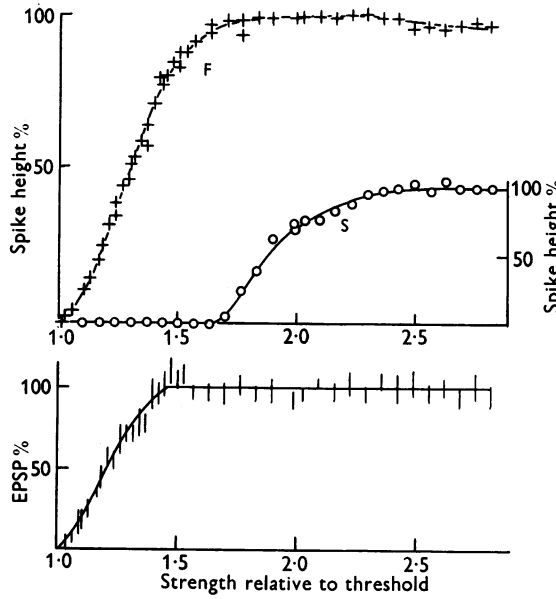

Fig. 3. Lower graph shows the height of the monosynaptic EPSP and upper graph the size of the F (+) and S (○) spike potentials all plotted against stimulus strength in multiples of the threshold strength. Measurements from the same series that is partially illustrated in Fig. 2. Every point is measured from a record composed of about twenty-five superimposed sweeps. Further description in text.

Though in Fig. 3 the thresholds for the EPSP and the F spike were identical, there was a considerable discrepancy in the strengths for their respective maxima, 1·44 and 1·8 respectively, However, this discrepancy is probably attributable to the diminishing asynchronism of the maximum F volley, as revealed in the analysis of Fig. 1*E*, where likewise with just maximum stimulation the F spike only attained 89% of size observed with supramaximal stimulation.

A more rigorous investigation of the relationship of afferent volley to maximum EPSP is illustrated in Fig. 4. As in Fig. 1, there was an initial conditioning stimulus of varying strength followed 1 msec later by a testing stimulus that was above maximum for group I. This testing stimulus excited all the group I fibres not excited by the conditioning. The control responses to

the conditioning stimulus alone are shown immediately following the responses evoked by the double stimuli. In *A* the tracing of the afferent spike potentials shows that the testing stimulus excited a considerable fraction of the F fibres, and correspondingly the testing volley evoked a considerable addition to the EPSP (cf. control response, *B*). In *C* the testing stimulus apparently evoked an S spike uncontaminated by F, yet it still caused a late hump to appear on the EPSP record, as may be seen by comparison with the control response (*D*) evoked by the conditioning stimulus alone. There is even a trace of this late hump in record *E* as compared with the control in *F*. Still further increase of

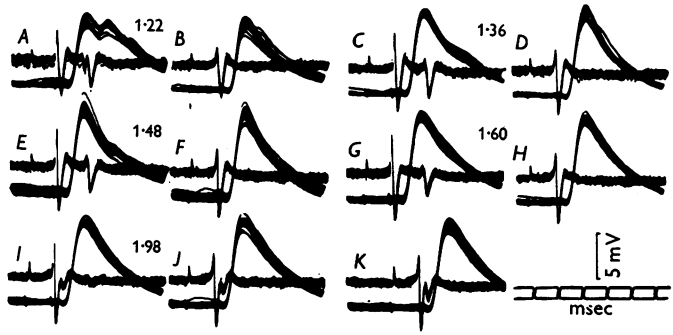

Fig. 4. Contribution by the S volley to the monosynaptic EPSP as tested by the double volley technique. Simultaneous recording from the S1 dorsal root entry zone and intracellularly from a BST motoneurone. Observe that, in the triphasic records, negativity is unconventionally signalled by a downwards deflexion. The strength of the testing stimulus was supramaximal for the group I fibres and kept constant throughout the series (response to testing stimulus alone shown in record *K*). For the conditioning volleys in records *A*, *C*, *E*, *G* and *I* the stimulus strengths relative to threshold strength are indicated on the records. Following each of these double volley records, there is shown in *B*, *D*, *F*, *H* and *J* the response at the same stimulus strength as used for conditioning in the preceding record.

the conditioning stimulus finally eliminated all trace of this addition by the testing stimulus (compare *G* with *H*, and *I* with *J*). As judged by the height of the EPSP, the conditioning stimuli in *C* and *D* were already producing a maximum response. Actually the humps added by the S volley in *C* and *E* had such a long latent period that, if evoked by the conditioning stimulus, they would not appreciably have added to the summit of the EPSP. However, their latent periods were still sufficiently short to identify the humps as due to a monosynaptic EPSP. The monosynaptic EPSP thus contributed by the S volley in record *C* (Fig. 4) was 8% of the total monosynaptic EPSP. It should be noted that the higher threshold range of the S volley (cf. records *G* and *H*) is ineffective monosynaptically. On the basis of our original assumption, the conclusion may be drawn that some Ia fibres from annulo-spiral endings are interspersed among the lowest threshold fibres giving the S spike potential.

Similar series of observations have been made in many experiments with

other thigh muscle motoneurones, particularly biceps-semitendinosus and quadriceps motoneurones, but also with semimembranosus and gracilis motoneurones. The motoneurones to thigh muscles are particularly suited to this investigation because the separation between the F and S spikes occurs frequently. The contamination of Ia fibres in the S volley was investigated in six cats in which the F spike in nerves from thigh muscles reached a maximum with a stimulus that was subliminal for the S spike. In each animal a number of neurones were investigated with the use of the double-volley technique as illustrated in Fig. 4. In one the maximal addition of monosynaptic EPSP by the S volley in percentage of the total was 10%. In the remaining animals the corresponding values were 0, 3, 5 and 6%.

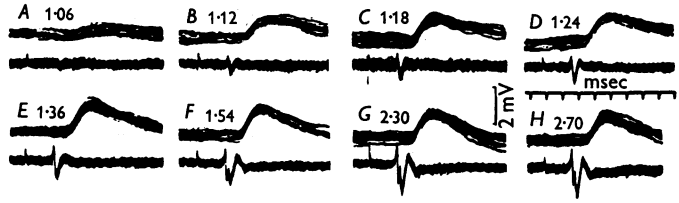

Fig. 5. Relationship of the intracellularly recorded monosynaptic EPSP to the F and S components in the plantaris nerve. Observe that, in the triphasic recording, negativity unconventionally is signalled by a downwards deflexion. Stimulus strengths for single volleys in records *A–H* relative to threshold strength are indicated on the records.

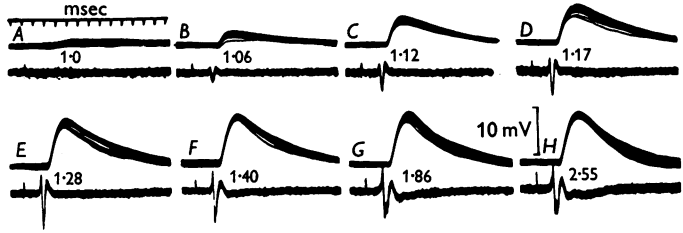

Fig. 6. Relationship of the intracellularly recorded monosynaptic EPSP to the two components of the afferent volley in the gastrocnemius-soleus nerve. The stimulus strengths relative to threshold strength are indicated on the records. Observe that, in the triphasic recording, negativity is unconventionally signalled by downwards deflexion.

The separation of the group I volley into two components has also been found, although not so frequently, in nerves to muscles lying more distally in the limb. (Sherrington referred to the portion of the hind limb between the knee joint and the ankle joint as the leg, and this term will henceforth be used.) The records in Fig. 5 show that the volley in the nerve to plantaris displays the separation into the F and S components. These series closely resemble the records in Fig. 2, the S component likewise failing to increase the monosynaptic EPSP. Similarly, with gastrocnemius volleys in Fig. 6 the monosynaptic EPSP increased from *A* to *E* but not with the further increase of stimulus that added the S components of the group I volley.

Fig. 7 illustrates one of several experiments in which separation in two components was found in all the nerves to hind-limb muscles that were tested. There are shown monosynaptic EPSP's in motoneurones receiving excitatory action from the muscle nerves to plantaris, flexor digitorum longus, lateral gastrocnemius plus soleus, medial gastrocnemius and tibialis anticus plus extensor digitorum longus. In each horizontal row the records on the right

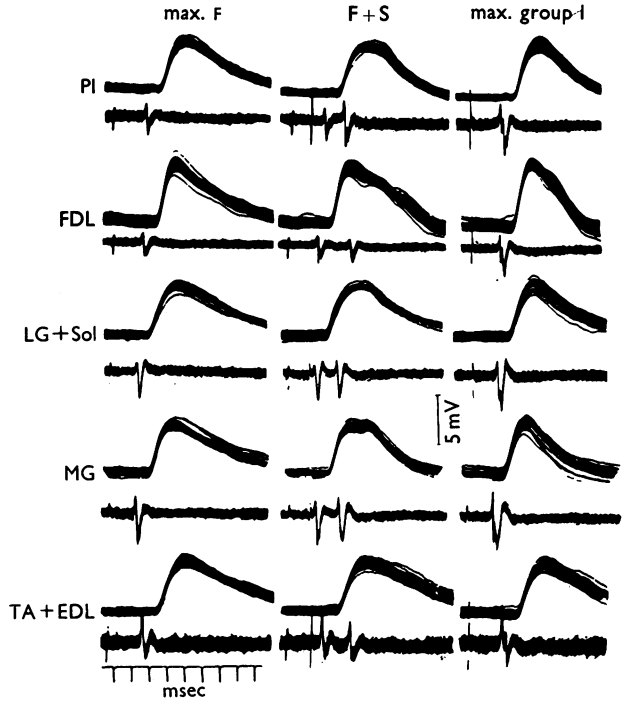

Fig. 7. Contribution to the intracellularly recorded monosynaptic EPSP by the F and S components in the nerves to plantaris, flexor digitorum longus, lateral gastrocnemius + soleus, medial gastrocnemius and tibialis anticus + extensor digitorum longus. Each horizontal row of records was obtained from a motoneurone receiving large monosynaptic innervation from the nerve indicated. Records to the right show the response to a maximal group I volley and to the left are seen the responses to stimuli just maximal for the fast volley. The middle column shows double volleys, the strengths of stimulation for conditioning and testing being the same as used to obtain the records to the left and right respectively. Observe that, in the triphasic recording from the dorsal root entry zone, negativity is unconventionally signalled by downwards deflexion.

are evoked by a stimulus that gives a maximum group I volley and monosynaptic EPSP. To the left is the response with a stimulus that just gives a maximum F volley. In the middle column the double volley technique was used, the strengths of stimulation for the conditioning and testing volleys being the same as used to obtain the records to the left and right respectively.

It appears from the records in Fig. 7 that in all these nerves the s volley contributed some monosynaptic innervation to the motoneurones investigated, but the contribution was in all cases smaller than 15% of the total EPSP. The fibres giving this additional contribution were always within the low threshold range of the s volley.

It should be noted that this quantitative evaluation of the monosynaptic excitatory action of the s volley is based on the recording from a number of motoneurones of each type. It is important to have multiple sampling for each type of motoneurone because even in one preparation there is a significant range of variation in the percentage of total monosynaptic EPSP that is contributed by the s volley. In the experiment of Fig. 7 three cells of each type were investigated in addition to those illustrated. With these the contribution made by the second volley in relation to the total monosynaptic EPSP was the same as, or less than, in the illustrated records.

The measurements of the monosynaptic EPSP's contributed by the s volley may be used to calculate the proportion of group Ia fibres that are contained in the s volley. When making this calculation allowance should be made for any departure from a simple arithmetical addition in the summation of superimposed EPSP's. It has been shown that, when a motoneurone is depolarized, the size of the monosynaptic EPSP produced by a given volley is reduced in proportion to the depolarization (Coombs *et al.* 1955*c*). There is experimental evidence that EPSP's sum in accordance with these expectations (Eccles, Eccles & Lundberg, unpublished observations). With a membrane potential of 70 mV, we should expect that the EPSP added by the highest threshold group I afferents, when the cell is depolarized by about 7 mV, would have 10% less depolarizing effect than that of the low threshold fibres. Thus, when the s volley contributes 5% of the maximum EPSP, somewhat less than 5·5% of the Ia fibres are contained in the s volley; hence for all practical purposes the figures given for the addition to the EPSP by the s volley can be used to describe the proportion of Ia fibres in it, it being assumed that group Ia fibres over the whole threshold range are approximately equivalent. In accordance with the assumptions made it can therefore be concluded that in thigh muscle nerves the group I fibres giving the s spike contain 0–10% of the Ia fibres, whereas with the nerves to leg muscles the proportion is 0–15%.

Figs. 8–10 illustrate the frequently observed condition with the nerves from the leg muscles, where there is no clear separation of the group I volley into two components with different conduction velocities.

In Fig. 8 the monosynaptic EPSP and the afferent volley had the same threshold and with increasing stimulus both increased from *A* to *F*. With response *E* (stimulus 1·18 times threshold) the EPSP had attained 94% of its maximum and it was maximal in *F*. The single afferent spike, however, continued to increase, the spikes in *E* and *F* being only 45 and 63% respectively

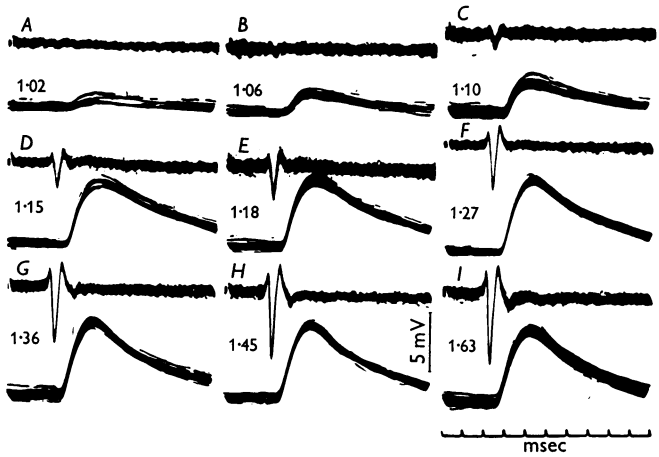

Fig. 8. Relationship of the intracellularly recorded monosynaptic EPSP to the group I afferent volley from the medial gastrocnemius nerve. The afferent volley shows no sign of separation into two components with different velocity. Observe that in the triphasic spike recorded at the S1 dorsal root entry zone negativity is unconventionally signalled by a downwards deflexion. Stimulus strengths relative to threshold strength are indicated on the records.

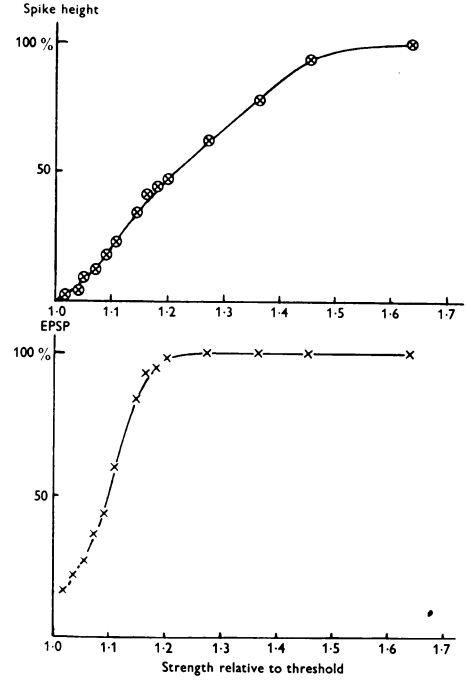

Fig. 9. The upper curve shows the size of the group I spike as recorded by a surface electrode at the dorsal root entry zone and the lower curve the size of the monosynaptic EPSP, both being plotted against stimulus strength expressed in multiples of the threshold strength. The measurements are from the same series as the records in Fig. 8 (medial gastrocnemius nerve).

of the maximum in *I*. The plotted curves for EPSP and afferent spike size (Fig. 9) resemble those of Fig. 3 except over ranges of stimulus strength above the maximum for the EPSP. In Fig. 9 the afferent spike was doubled in size over this range as against an increase of 19% in Fig. 3. Presumably this larger increase is attributable to the addition of I b impulses which were not sufficiently delayed in Fig. 8 to give a separate S spike.

In Fig. 10 there is again no velocity separation in the plantaris nerve, but nevertheless only the low-threshold fibres contribute monosynaptic excitatory action to the motoneurone. The small volley in record *A* is seen to give approximately 80% of the total EPSP. As tested with the sensitive double-volley technique it is apparent from the lowest records in Fig. 10 that hardly any monosynaptic innervation is contributed by those 50% of the group I afferents with the higher thresholds.

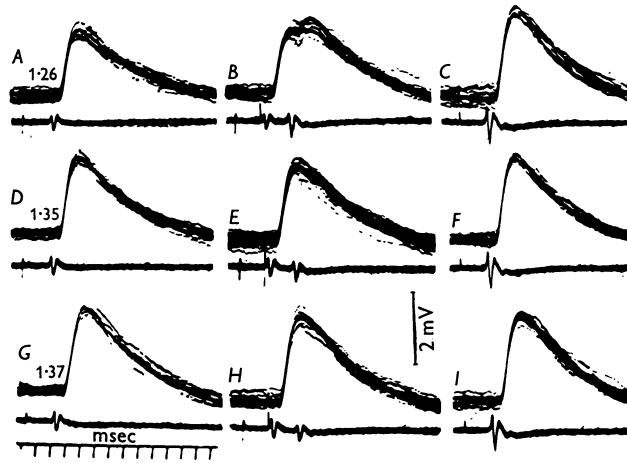

Fig. 10. Intracellular recording from a motoneurone receiving monosynaptic innervation from the nerve to plantaris. Records *C–F* and *I* all show the response to a stimulus, slightly supramaximal to group I and without any sign of separation into F and S components in the spike. Observe that, in the triphasic recording, negativity is unconventionally signalled by a downwards deflexion. To obtain records *A*, *D* and *G* the stimulus strengths relative to threshold strength were: 1·26, 1·35 and 1·37; and, in records *B*, *E* and *H*, these stimuli were used to evoke the conditioning response, the testing stimulus in *B*, *E* and *H* being the same as in the records to the right.

The relationship of the group I volley to inhibitory actions

Bradley & Eccles (1953) reported that the direct inhibition of monosynaptic reflexes was entirely caused by fibres of the fast component of the group I volley. Intracellular recording of the inhibitory post-synaptic potential (IPSP) of motoneurones provides a rigorous method of re-examining this observation. In Fig. 11 *A–E* single afferent volleys in the quadriceps nerve have evoked in a semitendinosus motoneurone the IPSP's which have been shown to be

responsible for direct inhibitory action (Brock *et al.* 1952; Coombs *et al.*1955*d*). The composition of the afferent volleys has been tested by the double-volley technique in the corresponding records *F* to *J*. The *F* volley is thus shown to attain its maximum at the same stimulus strength (1·43 times threshold) that gives the maximum IPSP (records *C* and *H*). Records *H* and *I* illustrate further the importance of using the double-volley technique in these experiments. In record *I* almost half of the fibres of the S volley were excited, but, on account of the delay and asynchronism in initiation of the impulses by threshold stimuli, the S spike potential merely caused a decrease in the terminal positive phase of the triphasic F spike. With direct inhibitory action it has been a constant finding that the IPSP appears with the F spike. Even with the use of the double-volley technique, there has never been any addition to this IPSP by the S volley. With the mono-synaptic EPSP evidence was often found for a small proportion of Ia fibres in the S volley. Possibly the existence of an interneurone in the inhibitory pathway is the explanation for the failure of these Ia fibres to produce any detectable direct IPSP.

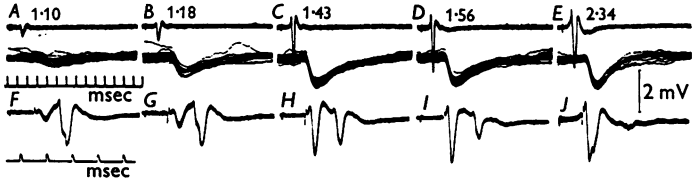

Fig. 11. Intracellular recording from a semitendinosus motoneurone showing direct inhibitory action by the F component of the group I volley in the quadriceps nerve. The stimulus strengths relative to threshold are indicated on records *A–E*. In records *F–J* the double volley was recorded at the root entry zone. The testing stimulus was supramaximal for group I fibres and the stimulus strengths used to evoke the conditioning volleys were the same as in the corresponding records *A–E*. Observe that, in the triphasic records, negativity unconventionally is signalled by downwards deflexion.

Fig. 12 serves to illustrate an inhibitory action by group I afferents, which is attributable to afferent impulses arising from Golgi tendon organs (Ib inhibition). The afferent volley in the nerve to flexor digitorum longus muscle showed a separation into F and S spikes which is best seen in record *M*. With a stimulus strength 1·17 times threshold the volley was submaximal for the F spike (record *H*) and evoked a monosynaptic EPSP in a plantaris motoneurone (lower trace of record *A*). When the conditioning stimulus was increased to 1·21 times threshold, the testing volley (record *I*) had no F component, but was still maximum for S. Thus there was complete threshold discrimination between the F and S components. It can be observed in Fig. 12 that the monosynaptic EPSP was caused only by the F volley, since an increase of the stimulus strength beyond 1·21 failed to bring about any increase in its magnitude. Increase of the stimulus strength to 1·25 times threshold excited some S impulses, as evidenced by a slight decrease of the S spike of the testing

response in *J*, but there was no change in the monosynaptic EPSP (record *C*). However at a strength of 1·31 times threshold (record *D*) a small IPSP was superimposed on the declining phase of the EPSP, while the deficit in the testing S spike response in record *K* shows that about one-fourth of the S spike fibres had been excited. Further slight increases in the stimulus caused a large increment in the IPSP, which reached a maximum in record F with a stimulus strength of 1·47 times threshold that was also maximum for the S fibres. It should be noted that doubling of the stimulus strength (record *G*) now neither increased the magnitude of the IPSP nor changed its time course.

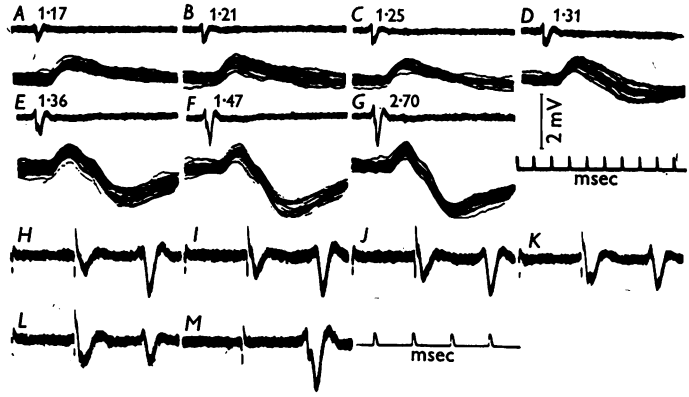

Fig. 12. Synaptic action in a plantaris motoneurone contributed by F and S volleys in the nerve of flexor digitorum longus. The stimulus strengths relative to threshold strength are indicated on records *A–G*. The lower set of records show double volleys recorded at the dorsal root entry. Stimulus strength for testing volley (shown alone in record *M*) was supramaximal for group I fibres. Stimulus strengths for conditioning volleys in *H–L* were the same as used to obtain the corresponding records *A–E*. Observe that, in the triphasic records, positivity unconventionally is signalled by an upwards deflexion.

The intracellular records shown in Fig. 12 correspond well with the findings by Laporte & Lloyd (1952). In fig. 3 of their paper the facilitatory effect of a conditioning volley in the flexor digitorum longus nerve on monosynaptic reflexes evoked by a plantaris nerve volley reached a maximum at a strength that was subthreshold for the production of group I inhibition.

The functional organization of the synaptic action by Golgi tendon-organ afferents will be dealt with in a later paper (Eccles, Eccles & Lundberg, unpublished observations). For the present purpose it is enough to show that these effects appear with activation of the S component of the group I volley. With one exception, dealt with below, there has been no trace of synaptic action characteristic of Golgi tendon organ activity with the F component of the group I volley. This does not necessarily imply that the F spike potential is entirely uncontaminated by Ib impulses. Presumably some spatial summation is needed for excitation of the interneurones that are interposed on the

inhibitory pathway, hence a small group Ib volley would not be expected to evoke an IPSP response in a motoneurone. It may, however, be concluded that the afferent fibres from Golgi tendon organs very predominantly belong to the S component of the group I volley.

Only in one cat have we observed that F impulses produced an IPSP which, according to our original assumption, would be attributable to Ib impulses from Golgi tendon organs. In Fig. 13 *J* the double-volley technique revealed that a stimulus 1·39 times threshold evoked a volley in quadriceps nerve that was practically maximum for the F fibres and yet included no S stimulus.

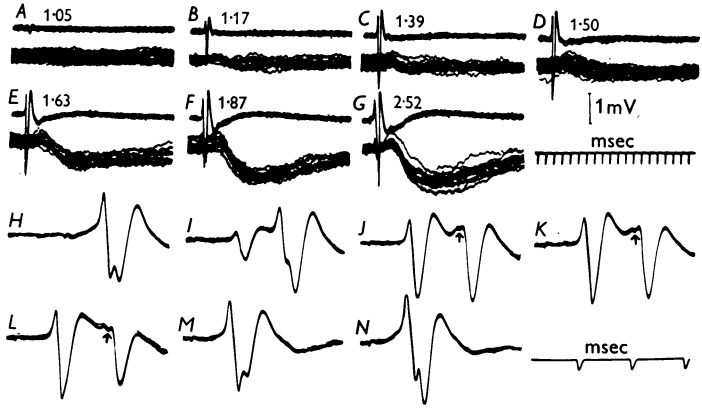

Fig. 13. Intracellular recording from a medial gastrocnemius motoneurone showing inhibitory action by F and S volleys in the nerve to quadriceps. For records *A–G* the stimulus strengths relative to threshold strength are shown in the figure. In records *H–N*, showing the double volleys recorded at the L6 dorsal root entry zone, the testing stimulus was supramaximal to group I and the stimulus strengths used to evoke the conditioning response were the same as in the corresponding records *A–G*. In record *J* the conditioning stimulus is subthreshold for the S and supramaximal for the F fibres, except for the few fibres giving the small spike marked by an arrow. This small spike remains in *K* and *L*, and probably its high threshold was caused by injury to a small filament of the quadriceps nerve. Observe that, in the triphasic records, negativity unconventionally is signalled by a downwards deflexion.

Nevertheless, this volley produced a small IPSP in a gastrocnemius motoneurone (record *C*). This IPSP was also evoked by the volley set up by a still weaker stimulation (1·17 times threshold in *B*), and was almost unchanged when the stimulus was increased to 1·50 times threshold in *D*. However, with further increase in the stimulus to 1·63 and 1·87 times threshold a large increase in the IPSP (*E* and *F*) paralleled an increase in the S spike to maximum (*L* and *M*). In *G* still further increase in the stimulus added slightly to the IPSP. The size of the IPSP attributable to the F volley alone (records *B* and *C*) was approximately 20% of the IPSP produced by a maximum group I volley (records *F* and *G*).

In a number of other gastrocnemius motoneurones from the same cat the

F volley of quadriceps nerve contributed approximately the same proportion of an IPSP that our original assumptions would exclude from the group Ia category. Hence it seems that in occasional animals a substantial number of Ib fibres may be interspersed amongst the Ia fibres making up the F volley.

Discussion

The present investigations confirm and extend the original report of Bradley & Eccles (1953) that the group I afferent impulses from muscles of the hind limb are frequently separable into two subgroups by virtue of differences both in conduction velocity and threshold. This separation is most often found with the nerves from thigh muscles, but also occurs not infrequently with the nerves to leg muscles.

The existence of fast (F) and slow (S) components of the group I volley would not in itself justify their identification respectively as due to afferent fibres from annulo-spiral endings (group Ia) and Golgi tendon organs (group Ib). Bradley & Eccles (1953) based this identification on a good correlation between the F component and both the monosynaptic excitatory and the direct inhibitory actions of a group I volley. It was assumed that these two central actions were entirely due to impulses from annulo-spiral endings, i.e. to Ia impulses as defined by Hunt (1952, 1954) and by Laporte & Lloyd (1952). The present investigation has likewise taken advantage of these assumptions in identifying the Ia afferent impulses, but has also employed a positive identification of the group Ib afferent impulses from Golgi tendon organs, namely, the disynaptic inhibitory action on motoneurones other than those of antagonist muscles (cf. Granit, 1950, 1952, 1955; Laporte & Lloyd, 1952).

On the basis of these assumptions and of the intracellular recording of the EPSP and IPSP of motoneurones it has been possible to evaluate quantitatively the distribution of group Ia impulses in the F and S components. Evaluation by monosynaptic EPSP's has shown that with the nerves to thigh and to leg muscles 90–100% and 85–100% respectively are included in the F component. The remaining group Ia fibres are in the lowest threshold fraction of the S component. The rather larger proportion of group Ia fibres in the S component of leg muscles may be correlated with the less clear separation between the F and S spikes in the nerves to leg muscles. Evaluation by the direct IPSP has differed from the evaluation by the monosynaptic EPSP in that it has failed to reveal any group Ia fibres in the S component. Possibly this discrepancy is attributable to the interneurone on the central inhibitory pathway.

Intracellular recording from motoneurones has shown that the IPSP which is assumed to be produced by group Ib impulses is almost entirely correlatable with the S component of the group I afferent volley. It may therefore be concluded that the group Ib afferent impulses occur very predominantly in

the S volley. However, in those experiments where the F volley gives no IPSP of the Ib type, it should not be concluded that it is uncontaminated by Ib impulses. A few impulses in the F volley could be ineffective because they failed to activate any of the interneurones on the inhibitory pathway. On account of synaptic relays on the inhibitory pathways, it is not permissible to calculate the percentage of Ib fibres in the F component in the way that was possible with Ia fibres on the basis of the monosynaptic EPSP's produced by the S volley.

It should be noted that, even when there is no separation with respect to conduction velocity, there is, nevertheless, often a high degree of threshold discrimination between the Ia and Ib afferent fibres. For example in Figs. 8 and 10 only the low-threshold half of the group I volley contributes to the monosynaptic EPSP. Similar observations have been made by Laporte & Lloyd (1952) and Rall (1955).

By recording from dorsal root filaments Laporte & Bessou (1957) have directly identified the receptors responsible for adequate activation of individual afferents in the F and S components of the nerves to thigh muscles. Of 30 fibres discharged by Golgi tendon organs 3 were in the F component. On the other hand, out of 70 fibres originating in muscle spindles, 10 were in the S component. This would give a higher contamination of group Ia fibres in the S component than the one derived above, varying between 0 and 10% with a mean value of about 5%. Laporte & Bessou (1957) have suggested that their higher percentage may be due to the fact that the spindle afferents found by them in the S component possibly are composed not entirely of fibres with annulo-spiral endings, but also of group II fibres from flower-spray endings, which may overlap in conduction velocity with the group I fibres. There is thus no inconsistency between the proportions observed by Laporte & Bessou and in the present investigation.

Since these two different investigations have shown that the group Ia and Ib fibres are respectively almost entirely in the F and S components of the group I spike, it is justifiable to discard the nomenclature, 'F' and 'S' components, that was provisionally adopted at the beginning of this paper and to refer to the two components of the group I spike by the symbols, Ia and Ib, that were introduced originally to refer to their origins from annulo-spiral endings and Golgi tendon organs respectively (Hunt, 1952, 1954; Laporte & Lloyd, 1952).

The separation into Ia and Ib volleys has already been used extensively for analysis of central connexion of the group I afferents (Eccles, Fatt, Landgren & Winsbury, 1954; Eccles, Fatt & Landgren, 1956; Lundberg & Oscarsson, 1956; Oscarsson, 1956). The present findings justify the use of the separation in attempts to disclose the central connexions of afferent fibres from annulo-spiral endings and of Golgi tendon organs. The small intermixture of the two

groups of afferents would complicate interpretation only under very special circumstances. However, it cannot be excluded that it may account for the convergence of excitatory action by Ia and Ib volleys that was observed with the use of the double-volley technique on neurones of the dorsal spino-cerebellar tract (Lundberg & Oscarsson, 1956). In this case the action of the later incoming Ib volley probably was superimposed on excitatory action that survived the impulse generated by the earlier Ia volley, and possibly the admixture of a few Ia fibres in the Ib volley would be sufficient to discharge the cell.

The investigation by Laporte & Bessou (1957) and the present results give direct and conclusive evidence concerning the receptor organs responsible for the various actions of group I afferent volleys on motoneurones. In agreement with the generally accepted hypothesis, it can be stated: first, that both the monosynaptic EPSP and the direct IPSP, that is, the IPSP evoked by group I impulses from antagonist muscles, are caused by afferent fibres with annulo-spiral endings; and secondly, that the IPSP produced by impulses from muscles other than antagonists (Granit, 1950, 1952, 1955; Laporte & Lloyd, 1952) is caused by afferent fibres with Golgi tendon endings.

SUMMARY

1. Intracellularly recorded synaptic actions on motoneurones have been correlated with the fast and slow components of the group I spike in afferent volleys from muscle nerves.

2. The double spike, although most often encountered in nerves from thigh muscles, is sometimes also found in nerves from leg muscles.

3. In motoneurones of thigh muscles 90–100 % of the monosynaptic EPSP occurs with the fast component as against 85–100 % in nerves from leg muscles. It is concluded that these figures give the percentage of group Ia fibres, that is, fibres with annulo-spinal endings, contained in the fast component.

4. In nerves that do not give double spike responses it has frequently been observed that only the 50 % of the afferents with lower threshold contribute monosynaptic EPSP.

5. The fast component is entirely responsible for direct inhibitory action on motoneurones of antagonistic muscles.

6. Inhibitory action by group I muscle afferents on motoneurones other than antagonists was found to be caused by the slow component. It is concluded that Golgi afferents, that is group Ib fibres, to a very predominant extent occur in the slow component of the group I spike.

7. It is proposed to refer henceforth to the fast and the slow group I volleys as the Ia and the Ib volleys respectively.

REFERENCES

BARKER, D. (1948). The innervation of the muscle-spindle. *Quart. J. micr. Sci.* **89**, 143–186.

BLAIR, E. & ERLANGER, J. (1933). A comparison of the characteristics of axons through their individual electrical responses. *Amer. J. Physiol.* **106**, 524–564.

BRADLEY, K. & ECCLES, J. C. (1953). Analysis of the fast afferent impulses from thigh muscles. *J. Physiol.* **122**, 462–473.

BROCK, L. G., COOMBS, J. S. & ECCLES, J. C. (1952). The recording of potentials from motoneurones with an intracellular electrode. *J. Physiol.* **117**, 431–460.

CAJAL, S. R. (1909). *Histologie du système nerveux de l'homme et des vertébrés.* Vol. I. Paris: Maloine.

COOMBS, J. S., ECCLES, J. C. & FATT, P. (1955*a*). The electrical properties of the motoneurone membrane. *J. Physiol.* **130**, 291–325.

COOMBS, J. S., ECCLES, J. C. & FATT, P. (1955*b*). The specific ionic conductances and the ionic movements across the motoneuronal membrane that produce the inhibitory post-synaptic potential. *J. Physiol.* **130**, 326–373.

COOMBS, J. S., ECCLES, J. C. & FATT, P. (1955*c*). Excitatory synaptic action in motoneurones. *J. Physiol.* **130**, 374–395.

COOMBS, J. S., ECCLES, J. C. & FATT, P. (1955*d*). The inhibitory suppression of reflex discharges from motoneurones. *J. Physiol.* **130**, 396–413.

ECCLES, J. C., ECCLES, R. M. & LUNDBERG, A. (1957). The convergence of monosynaptic excitatory afferents on to many different species of alpha motoneurones. *J. Physiol.* (in the Press).

ECCLES, J. C., FATT, P. & LANDGREN, S. (1956). The central pathway for the direct inhibitory action of impulses in the largest afferent nerve fibres to muscle. *J. Neurophysiol.* **19**, 75–98.

ECCLES, J. C., FATT, P., LANDGREN, S. & WINSBURY, G. J. (1954). Spinal cord potentials generated by volley in the large muscle afferents. *J. Physiol.* **125**, 590–606.

GRANIT, R. (1950). Reflex self-regulation of muscle contraction and autogenetic inhibition. *J. Neurophysiol.* **13**, 351–372.

GRANIT, R. (1952). Reflexes to stretch and contraction of antagonists around ankle joint. *J. Neurophysiol.* **15**, 269–276.

GRANIT, R. (1955). *Receptors and Sensory Perception.* New Haven: Yale University Press.

GRANIT, R. & STRÖM, G. (1951). Autogenetic modulation of excitability of single ventral horn cells. *J. Neurophysiol.* **14**, 113–132.

HOFFMAN, P. (1922). *Untersuchungen über die Eigenreflexe (Sehnenreflexe) menschlicher Muskeln.* Berlin: Springer.

HUNT, C. C. (1952). The effect of stretch receptors from muscle on the discharge of motoneurones. *J. Physiol.* **117**, 359–379.

HUNT, C. C. (1954). Relation of function to diameter in afferent fibres of muscle nerves. *J. gen. Physiol.* **38**, 117–131.

JOB, C. (1953). Über autogene Inhibition und Reflexumkehr bei spinalisierten und decerebrierten Katzen. *Pflüg. Arch. ges. Physiol.* **256**, 406–418.

LAPORTE, Y. & BESSOU, P. (1957) Distribution dans les sous-groupes rapide et lent du groupe I des fibres Ia d'origine fusoriale et des fibres Ib d'origine golgienne. *C.R. Soc. Biol., Paris* (in the Press).

LAPORTE, Y. & LLOYD, D. P. C. (1952). Nature and significance of the reflex connections established by large afferent fibres of muscular origin. *Amer. J. Physiol.* **169**, 609–621.

LLOYD, D. P. C. (1943*a*). Reflex action in relation to pattern and peripheral source of afferent stimulation. *J. Neurophysiol.* **6**, 111–119.

LLOYD, D. P. C. (1943*b*). Conduction and synaptic transmission of the reflex response to stretch in spinal cats. *J. Neurophysiol.* **6**, 317–326.

LLOYD, D. P. C. (1946). Facilitation and inhibition of spinal motoneurones. *J. Neurophysiol.* **9**, 421–438.

LUNDBERG, A. & OSCARSSON, O. (1956). Functional organization of the dorsal-spino-cerebellar tract in the cat. IV *Acta physiol. scand.* **38**, 53–75

McCouch, G. P., Deering, I. D. & Stewart, W. B. (1950). Inhibition of knee jerk from tendon spindles of crureus. *J. Neurophysiol.* **13**, 343–350.

Matthews, B. H. C. (1933). Nerve endings in mammalian muscle. *J. Physiol.* **78**, 1–53.

Oscarsson, O. (1956). The functional organization of the ventral spino-cerebellar tract. I. *Acta physiol. scand.* **38**, 145–165

Rall, W. (1955). Experimental monosynaptic input-output relations in the mammalian spinal cord. *J. cell. comp. Physiol.* **46**, 413–437.

Sherrington, C. S. (1894). On the anatomical constitution of nerves of skeletal muscles; with remarks on recurrent fibres in the ventral spinal nerve-root. *J. Physiol.* **17**, 211–258.