Nature and Significance of the Reflex Connections Established by Large Afferent Fibers of Muscular Origin

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A centripetal impulse volley traversing the myelinated afferent fibers in the nerve to a given muscle, or fraction thereof, is known to provoke a variety of reflex effects (4). By direct, or monosynaptic impingement upon motor nuclei it promotes the discharge of homonymous motoneurons, which is to say motoneurons supplying that given muscle or muscle fraction; it facilitates the response of motoneurons that supply the muscle remainder or synergists; it inhibits the response of motoneurons that supply antagonists. Such direct actions are mediated by the group of largest afferent fibers (group I) peculiar to muscle nerves, and are exerted in the service of, and in association with, myotatic reflexes.

By internuncially relayed impingement upon motor nuclei of action engendered in the medium (group II) and smaller (group III) afferent fibers, an afferent volley brings about reflex discharge predominantly to flexor muscles; facilitation of response in flexor nuclei; and inhibition of response in extensor nuclei. These polysynaptic reflex effects, widespread throughout the limb, are manifestations of flexor reflexes.

A systematic re-examination of the conditioning potentialities of afferent volleys arising in muscle nerves has been made, and the results herewith presented, because considerable reason existed for supposing that the list of established reflex actions enumerated above was far from complete. In a recent study on the actions of muscle afferent fibers, however, Brock, Eccles and Rall (5) found no evidence for postulating actions not listed.

METHOD AND PRELIMINARY CONSIDERATIONS

Decapitate preparations maintained by artificial respiration have been employed. In each experiment several muscle nerves were prepared for stimulation. These were severed distally and arranged upon stimulating electrodes in a manner designed to avoid vicarious excitation of nearby structures. Appropriate ventral roots were severed to block antidromic volleys in motor fibers and to provide for the recording of reflex discharges. Ready access to nerves and roots without continued exposure was secured by the use of paraffin pools saturated with 95 per cent O₂ and 5 per cent CO₂.

In experiments of the sort to be described the procedure is to measure excitability change induced in a given motor nucleus by stimulation of a given muscle nerve. Average excitability in a motor nucleus is gauged by the magnitude of monosynaptic reflexes discharged therefrom in response to constant single shock stimulations of the homonymous muscle nerve, the standard for comparison being average magnitude attained in the absence of specific conditioning stimulation. Temporal course
of excitability change in a motor nucleus is found by determining the average magnitude of conditioned monosynaptic test reflexes at each of a variety of intervals between conditioning and test stimulations. To represent the experimental findings graphically magnitude of conditioned test reflexes is plotted in percentage of control value on the ordinates against time interval between conditioning and test volleys on the abscissae. The origin represents that circumstance in which the *group I* impulses of conditioning and test volleys are synchronous in the spinal cord at the level of the ventral root from which test responses are recorded. A correction for converting from stimulus interval to volley interval must be found for each series of observations by direct recording of the afferent volleys at the proper level of the spinal cord. These things having been done it is possible to compare directly the results of individual experiments.

![Fig. 1](https://via.placeholder.com/150)

**Fig. 1 (left).** *Conditioning of semitendinosus monosynaptic reflexes by afferent volleys in nerve to extensor longus digitorum to illustrate nonspecific effects exerted by traveling impulses.* In this and all subsequent figures magnitude of test reflex is expressed on the *ordinates* in percentage of the control values.

**Fig. 2 (right).** *Conditioning of extensor longus monosynaptic reflexes by afferent volleys in the knee flexor nerves.*

The resting spinal cord is a thing of fluctuating responsivity. Variability cannot be reduced, as by narcosis, without producing other and highly undesirable consequences. To improve accuracy with which measurements of conditioning action can be made in the face of fluctuation larger numbers of conditioned and control responses must be recorded at each stimulation interval, and the number of stimulus intervals at which tests of conditioning are made multiplied. A by-product of the effort to improve accuracy has been the finding that not all divergences are of the random sort to be expected. Some of the ‘errors’ are related systematically to the fact of a conditioning stimulus having been applied, and yet bear none of the marks of synaptic orthodoxy. On the contrary they belong in the category of vicarious impulse interactions. Such ‘nonspecific’ interactions between afferent impulses are seen to best advantage when the sources of conditioning and test volleys are the nerves to muscles that possess no monosynaptic reflex interconnection, although they may appear also as distortions of the early course of monosynaptic reflex conditioning effect. Certain of the most striking encountered instances are presented in figures 1 and 2. Triphasic course of interaction during the first millisecond clearly stamps the causal agency as being the presynaptic impulses of the conditioning volleys. In figure 1 the sequence of enhancement-depression-enhancement is that to be expected of interaction between structures orientated at 90° each to the other (6). The contrary sequence, illustrated in figure 2, accords with interaction between structures orientated in parallel (7, 8). Further analysis would require far more exhaustive study than has been attempted, and would be irrelevant to the present discussion.

Effective magnitude of vicarious or nonspecific conditioning effects is deter-
mined in large measure by intensity of the testing monosynaptic reflex. Whereas a ‘large’ monosynaptic reflex still may be doubled or trebled by synaptic impingement of facilitating impulses, or diminished to a quarter of its resting size by convergent inhibiting impulses, it is relatively insensitive to vicarious conditioning by passing or neighboring activity (9). ‘Small’ monosynaptic reflexes fall easy prey to passing disturbances: they should be used only with considerable caution and suitable reservations concerning the interpretation of observations. The smallest attainable monosynaptic reflex, of course, is that discharged by a single motoneuron.

A large reflex response to conditioning stimulation appearing in the same ventral root as does the test response may cause difficulty of measurement merely by the fact of its presence. Monosynaptic reflexes traversing independent pathways but the same ventral root, if closely related in time of inception, may become synchronized in the manner described by Katz and Schmitt (7). Errors of subtraction when two reflexes overlap in time are notoriously great, particularly if synergist paths are involved, and an opportunity arises for the test volley to condition the response to the conditioning volley. In short, measurement of conditioning action may be less than satisfactory when conditioning and test volleys are in near synchrony.

Many of the effects to be described take place when monosynaptic reflexes pertaining to a given muscle are played upon by afferent volleys arising in the nerves to its synergists and antagonists. In such instances the known direct effects, facilitatory or inhibitory as the case may be, inevitably are exerted upon the monosynaptic reflex pathway, and the course of the conditioning so imposed creates the base line from which other forms of action must be gauged.

**Disynaptic Group I Inhibitory Linkage Between Synergists**

**Extensor Nuclei.** Figure 3 illustrates the manner in which the monosynaptic reflexes of an extensor muscle may be conditioned by afferent volleys in the nerve to a synergist. Frequently, as was the case in this experiment, it is possible, by the use of the feeblest conditioning volleys, to limit conditioning action to facilitation mediated through monosynaptic reflex connection (curve 3A). After slight increase in the strength of conditioning stimulation the course of monosynaptic reflex facilitation was truncated abruptly, at approximately 0.5 msec., by an inhibitory action of considerable intensity (curve 3B). Still further increase in strength of conditioning stimulation caused the appearance, at approximately 1.8 msec., of an inhibitory action that is attributable to the stimulation of group II afferent fibers (curve 3C).

**Flexor Nuclei.** In figure 4 are the results of another but similar experiment performed upon a flexor nucleus. An uninterrupted curve of monosynaptic reflex facilitation was not obtained. Curve 4A shows how the monosynaptic reflex facilitation, the anticipated course of which is extrapolated by the broken line, was interrupted by an inhibitory action. Monosynaptic facilitation was powerful and the superposed inhibitory action was insufficient to bring the overall effect to the side of depression. To obtain curve 4B conditioning volleys were increased to group II strength with the result that a second facilitatory action was added to those already present actions which determined the form of curve 4A.

The observations of figures 3 and 4 serve to distinguish the short latency inhibition of synergists, flexor and extensor alike, from the known group II effects that have opposite direction in flexor and extensor nuclei. But to characterize the inhibitory action itself poses the demand for observation in greater detail utilizing conditioning volleys in more finely graded series. Figures 5 and 6 illustrate experi-
ments designed to fulfill the requirement. Observations therein, again dealing with an extensor nucleus (fig. 5) and a flexor nucleus (fig. 6), were not extended in range of time intervals to include the known group II effects.

In figure 5 curves A, B and C chart a progressive development with increased conditioning stimulation of the inhibitory process. It took place in the absence of progressive increase in the degree of monosynaptic facilitation. Smaller afferent conditioning volleys would have produced less facilitation, probably without significant divergence, at 0.5 msec., from the established course of facilitation. Latency of the inhibitory action did not shift as the action itself increased in intensity. Figure 6 depicts a slightly different situation in that 1) undistorted monosynaptic facilitation was secured by the use of the weakest conditioning volleys (curve 6A), 2) increase

in monosynaptic facilitation was accompanied by the first appearance of deviation at 0.5 msec. in the direction of inhibition (curve 6B), and 3) still further increase in monosynaptic facilitation was associated with an inhibitory action of unchanged latency, but then so powerful that curve 6C crosses both curves 6A and 6B to reveal a net result brought down to the side of absolute inhibition.

Results comparable to those exemplified in figures 3 to 6 have been obtained in experiments utilizing other pairs of synergist nerves for conditioning and testing stimulation. Named in pairs according to the muscles of origin the combinations of conditioning and testing volleys that have been employed with similar result are: the two nerves of triceps surae; soleus and gastrocnemius medialis, which provides the first recorded observation of the pattern of interaction between the reflex paths of a red and a pale head of a muscle; the two heads of flexor longus; two branches of
Concerning the Postulation of an Inhibitory Action of Relatively Brief (0.5 to 0.6 msec.) Latency. Brooks and Eccles (10) studying the conditioning of monosynaptic reflexes by afferent volleys arising in the nerves of synergists have described facilitation curves containing a discontinuity, which they ascribe not to the superposition of inhibitory action, but rather to "special features of the synaptic excitatory process." According to their analysis of the experimental observations, a rapidly decaying facilitation led, at the point of discontinuity, into a slowly decaying facilitation. More detailed examination shows that, in fact, the opposite is true: the slow decay precedes the steep decline.

Fig. 5 (left). Conditioning of plantaris monosynaptic reflexes by flexor longus afferent volleys of three different magnitudes, increasing in the order A, B, C, which designations identify the individual curves.

Fig. 6 (right). Conditioning of extensor longus monosynaptic reflexes by afferent volleys in the nerve of tibialis anterior. Curves A, B and C together plot the action by volleys of increasing magnitude.

Postulation of a facilitatory process endowed with special features might have been thought sufficient explanation for the form of curve B in figure 6 considered in isolation, but such a postulate cannot account for the absence of early discontinuity in curve 6A, nor for the steeper decline of curve 6C in the face of intensified monosynaptic facilitation. Appearance (curve 6B) and intensification (curve 6C) of an inhibitory action superimposed upon progressively increasing monosynaptic group I facilitation will account for the manner in which the curves of figure 6 differ. Since the observations of figure 6 were obtained in examination of a flexor nucleus it is concluded, in disagreement with Brock, Eccles and Rall (5), that certain afferent impulses engendered in the nerve to a flexor muscle do act in a manner such as to reduce excitability in the motor nucleus of a synergist. Mere inspection of curve 3B makes self-evident the fact of inhibition exerted with brief latency in an extensor nucleus by afferent impulses arising from a synergist.

Concerning Latency. Differential latency between reflex effects at a motor nucleus might be ascribed to differential afferent conduction velocity, or to difference
in the number of neurons serially placed in the executant pathways. Both circumstances could be operative in conjunction. Facilitation in a synergic nucleus is mediated monosynaptically by the highest velocity afferent fibers (11). The latency differential between facilitation and the newly described inhibition, being approximately 0.5 to 0.6 msec., is equivalent to the time required for negotiating an internuncial relay. This being so, an attempt to account for differential latency on the basis of differential afferent conduction velocity must include the postulate of a monosynaptic inhibitory connection. Alternatively an accounting based upon the assumption of a disynaptic inhibitory pathway presupposes approximate equivalence in conduction velocity of the afferent fibers mediating the opposed influences. Latency differential due to differential afferent conduction velocity in two monosynaptic pathways should vary with afferent conduction distance, that due to intercalation of a neuron in one of otherwise similar pathways could be independent of afferent conduction distance. Since the latency differential is relatively fixed, whether conditioning stimulation is applied to the knee flexor nerves close to the sciatic notch, or to the pretibial flexor nerves well below the knee, there is good reason for supposing that the inhibition of synergists is mediated by a disynaptic pathway from high velocity (i.e. group I) afferent fibers.

Concerning Threshold. Monosynaptic reflex facilitation of synergists and the newly described inhibitory action upon them are dissociated with ease from the known group II effects by variation in strength of afferent conditioning stimulation. They cannot so easily be dissociated one from the other, and when they are it is only in the sense that facilitation can be obtained free from interruption by the inhibitory process. The reverse presumably could not be achieved in nerve stimulation experiments, for the lowest threshold fibers facilitate the response of synergic motor nuclei by direct impingement thereupon (11). Clearly threshold to brief shocks of the afferent fibers mediating the inhibitory action is closely similar to that of the afferent fibers mediating monosynaptic facilitation. In a monosynaptic pathway threshold for influence upon the motoneurons is that of the afferent fibers exerting the influence. In a disynaptic pathway threshold for influence upon the final elements, the motoneurons, will depend not only upon that of the afferent fibers, but also upon response threshold of the intermediary elements, the interneurons. Presence in the inhibitory pathway of an internuncial relay requiring a degree of summation for response is sufficient basis for the finding that monosynaptic facilitation can be isolated by the use of sufficiently small afferent volleys. However, a slight difference in threshold between the fibers responsible for the two effects cannot be excluded. Furthermore it might be said that some fibers mediating the inhibitory action are of higher threshold than those mediating facilitation, for in some experiments inhibitory action will continue to grow in intensity with increased volleys after monosynaptic facilitation has reached a ceiling (cf. fig. 5).

Observations on threshold for monosynaptic facilitation of synergists and for the newly described inhibition are compatible only with the conclusion that the group I band of afferent fibers contains the afferent paths for both actions.

Disynaptic Group I Inhibitory Linkage Between Muscles Not Possessed of Monosynaptic Reflex Interconnection

Systematic survey of the reflex connections between afferent fibers of hindlimb muscles and heteronymous motor nuclei reveals a number of instances in which no monosynaptic pathway exists (4). Frequently, however, one finds such nuclei in-
hibited by a process that first manifests itself when test volleys follow conditioning volleys by an interval of 0.5 to 0.6 msec. Figure 7 exemplifies the finding and characterizes the time course of that inhibitory action up to onset, during the third millisecond, of group II inhibition. Inhibition in the present situation is indistinguishable from that exerted upon the monosynaptic reflex paths of synergist muscles, for which reason extended discussion concerning latency and threshold is unnecessary. It may be concluded that disynaptic group I inhibitory connection links certain muscles that are not linked by monosynaptic reflex connections.

Inhibitory action comparable to that illustrated in figure 7 has been found utilizing the following couplings of conditioning and test volleys named in reference to the muscles of origin: quadriceps, or a fraction thereof, and triceps surae (fig. 8); quadriceps and plantaris; knee flexors and triceps surae; extensor brevis and triceps surae; triceps surae and a) quadriceps (fig. 9), b) plantaris, c) flexor brevis; flexor longus and a) quadriceps (fig. 16), b) the knee flexors, c) triceps surae (figs. 7 and 10);

![Fig. 7](image1)

**Fig. 7.** (left). Inhibition of monosynaptic reflexes of triceps surae by afferent volleys in the nerve to flexor longus.

**Fig. 8.** (right). Onset of inhibitory action by afferent volleys in the nerve of vastus lateralis upon monosynaptic reflexes of triceps surae (curve A) and upon monosynaptic reflexes of the proper antagonist biceps femoris posterior (curve B).

plantaris and quadriceps; anterior hamstrings and pretibial flexors. In brief, disynaptic group I inhibitory action is rather widespread throughout the hindlimb musculature.

The relations between quadriceps and triceps surae are of particular interest and demand more detailed consideration. Figure 8 presents the result of an experiment in which the actions of vastus lateralis afferent volleys were tested by means of monosynaptic reflexes pertaining to triceps surae (curve 8A) and to the proper antagonist biceps femoris posterior (curve 8B). The latency differential between monosynaptic inhibition and disynaptic inhibition is apparent. In an earlier paper (4) the latency of inhibition in the situation depicted by curve 8A was given as 1.5 msec., and Brooks and Eccles described the latency as being in excess of 1.5 msec. (12). Obviously in both those observations the inhibition was due to the action of group II fibers. This seeming conflict of result is one illustration of the fact that negative findings are not reliable when internuncially relayed paths are involved. The earlier findings still can be repeated in some preparations.
When conditioning and test volleys are coupled in reverse a new question arises, for Brooks and Eccles (12) have described the inhibition of quadriceps by afferent volleys in the nerve of triceps surae in such a manner as to imply a pathway entirely comparable to that concerned in the inhibition of quadriceps by knee flexor afferent volleys. Figure 9 illustrates an experiment that demonstrates the difference between the two inhibitory pathways to quadriceps; that from triceps surae being disynaptic (curve 9A), that from the knee flexors direct (curve 9B). The onset of group II inhibition during the third millisecond can be seen in curve 9A.

Since afferent volleys from a given muscle nerve through disynaptic connection inhibit the motor nuclei of certain other independent muscles, search was made, without success, in an attempt to find a comparable and reciprocal facilitating action of such volleys upon the motor nuclei of proper antagonists to the muscles. Figure 10 illustrates a typical result. The facilitation evident in curve 10B was due to the action of group II fibers and is in no way reciprocal to the disynaptic inhibition in curve 10A.

**Disynaptic Group I Facilitatory Linkage Between Antagonists**

Frequently, when the monosynaptic reflexes of a given muscle are conditioned by afferent volleys engendered in the nerve to its proper antagonist the anticipated course of direct inhibition is interrupted by a facilitatory action that compares, except in the direction of effect, with the inhibitory actions that have been discussed. Figures 11 and 12 exemplify the experimental results obtainable in observation upon extensor and flexor nuclei. Abrupt descent of each curve (11 and 13) marks the onset and development of direct inhibition which, if uninterrupted by other
happenings, would decay along the course described by the broken lines (9). Instead
the curves return sharply in the direction of facilitation. After approximately 2
msec. further change, in the pattern of the flexor reflex and the result of action by
\textit{group II} afferent impulses, supervenes.

Comparable result has been obtained in a variety of situations in which the
nerves of antagonists were employed for conditioning and testing. Specifically,
disynaptic facilitatory action has been encountered in experiments upon these com-
binations: quadriceps volleys conditioning knee flexor monosynaptic reflexes and
vice versa; volleys from tibialis anterior conditioning triceps surae; extensor longus
volleys conditioning \textit{a}) triceps surae, \textit{b}) flexor longus (fig. 14), \textit{c}) plantaris (fig. 13),
and \textit{d}) flexor brevis (fig. 11); extensor brevis volleys conditioning flexor longus; vol-
leys from triceps surae conditioning the ankle flexors (fig. 12); flexor longus volleys
conditioning extensor longus; plantaris volleys conditioning the combined pretibial
flexors.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure11}
\caption{Fig. 11 (left). \textbf{CONDITIONING OF FLEXOR BREVIS MONOSYNAPTIC REFLEXES} by afferent volleys
in the nerve to its antagonist extensor longus.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure12}
\caption{Fig. 12 (right). \textbf{CONDITIONING OF MONOSYNAPTIC REFLEXES} pertaining to the combined pre-
tibial flexors by afferent volleys in the nerves to triceps surae.}
\end{figure}

By careful adjustment in strength of afferent conditioning volleys it is possible
in some instances to dissociate disynaptic facilitation of antagonists and monosynap-
tic inhibition. For instance, in figure 13, \textit{curve A} represents the early course of mono-
synaptic inhibition uncomplicated by secondary actions. \textit{Curve 13B}, charting the
effect of increased afferent conditioning volleys, shows the monosynaptic inhibition
increased in intensity. If no other change had occurred \textit{curve 13B} would have fol-
lowed the course indicated by the broken line, whereas in fact \textit{curve 13B} diverges,
at approximately 0.5 msec., in the direction of facilitation and crosses \textit{curve 13A}.
In the circumstance there is no doubt that a facilitatory action indeed was exerted.
Further increase in strength of the conditioning volleys (\textit{curve 13C}) brought no
further change during the initial 1.75 msec., but thereafter the inhibitory action of
\textit{group II} impulses, an expression of the flexor reflex, was added.

Secondary facilitation in an extensor nucleus, the effect of small volleys in the
nerves of proper antagonists, is not an unknown phenomenon (9, p. 432; 12), but the
proper time relations of the action have not been appreciated, nor has its significance.
Influence of Afferent Fibers Not Arising in Muscle

Cutaneous nerves contain aggregations of myelinated fibers (14) that resemble superficially the group II and group III aggregations in muscle nerves. At the elementary level of analysis the reflex result of stimulating these several aggregations is not dissimilar (13), despite the wide variety of afferent functions subserved by the constituent fibers. When nerve trunks are employed for conditioning stimulation fibers arising in tissues other than muscle may contribute to the result, and change of the type depicted in figure 14 may appear. Curve 14A plots the conditioning of flexor longus monosynaptic reflexes by afferent volleys restricted to the nerve of its proper antagonist, extensor longus. The sequence of monosynaptic reflex inhibition, disynaptic reflex facilitation, and group II inhibition is to be found therein. Curve 14B plots the change of result consequent to the addition of peroneal cutaneous afferent impulses to the conditioning volleys. The added cutaneous afferent impulses act as do the group II impulses in the nerve to extensor longus, but with diminished latency. An important reason undoubtedly is that alpha cutaneous afferent fibers in range of diameter, and hence of velocity, extend into the group I range without however displaying therein any peak of numerical preponderance (cf. reference 14, figs. 5 and 8).

On the Lability of Disynaptic Group I Conditioning

Passing mention has been made of the fact that disynaptic group I effects on occasion are not apparent. In the original studies on the conditioning actions of group I impulses (4, 9) very little effect attributable to relayed group I activity could be found even in the presence of obvious group II activity. In some preparations, and in circumstances that are not fully predictable, the earlier observations can be repeated.

Figure 15 illustrates the result of an experiment that was designed to display

![Fig. 13](image_url)  ![Fig. 14](image_url)
the conditioning potentialities of quadriceps group I afferent volleys upon monosynaptic reflexes of the antagonist knee flexors (curve 15A) and upon those of triceps surae. Monosynaptic inhibition was found, but the disynaptic facilitation of the knee flexor test reflexes, seen in other experiments, was absent, as was the disynaptic inhibition of triceps surae, otherwise documented in figure 8.

Not all disynaptic effects are necessarily present or absent in a given preparation. In the experiment of figure 16 monosynaptic reflexes of quadriceps were conditioned by afferent volleys from three sources. Curve 16A proves the direct inhibitory action of knee flexor afferent volleys to have been present. Curve 16B shows the disynaptic inhibitory action of volleys from triceps surae, noted in figure 9, to have been absent, the initial change being that referable to the action of group II impulses. Despite that fact the quadriceps monosynaptic reflexes were inhibited in the disynaptic group I pattern by afferent volleys from flexor longus (curve 16C).

Fig. 15 (left). CONDITIONING OF KNEE FLEXOR MONOSYNAPTIC REFLEXES by quadriceps afferent volley (curve A). In this experiment the monosynaptic test reflexes of triceps surae were unaffected by quadriceps afferent volleys (curve B).

Fig. 16 (right). ONSET OF CONDITIONING OF QUADRICEPS MONOSYNAPTIC REFLEXES by afferent volleys in the nerves to the knee flexors (curve A), to triceps surae (curve B) and to flexor longus (curve C).

Either members of a species differ widely in the finer structure of the spinal cords, or connections that can be demonstrated in a number of individuals are present, but inactive, in the remainder. Adopting the latter alternative, and accepting the postulate of disynapticity made for reasons earlier stated, a sufficient explanation for lability shall be that afferent volleys reaching the internuncial relay upon occasion and for certain reasons do not reach threshold therein and secure discharge therefrom. Variation of stimulus strength is one maneuver by means of which one frequently may control the response of the internuncial relay, and so the appearance of the motoneuron of the disynaptic effects (figs. 3, 6 and 13). Upon occasion cooling of the preparation has caused a reversible loss of disynaptic effects, the monosynaptic effects being retained. But temperature change is not an obligate determinant, for the disynaptic effects upon other occasions have not been lost.

FUNCTIONAL IMPLICATIONS

In no place has the present paper dealt with conditioning of monosynaptic reflexes by homonymous afferent volleys, for which reason it provides no evidence concerning the inhibition of homonymous motoneurons, which is to say ‘autogenetic inhibition.’ Autogenetic inhibition in this strict and proper sense has been shown to
occur (15, 16), and evidence is forthcoming (17) that tendon organs are the point of origin for the action. From this one may conclude that group I afferent fibers are concerned (18), although one is not yet in a position to define the executant pathway. Nevertheless it is likely that the pathway for autogenetic inhibition compares with that responsible for inhibition of the muscle remainder or of a synergist.

As the matter presently stands it may be said that fibers within the group I band can bring about the following seven effects: 1) A monosynaptic reflex discharge of homonymous motoneurons. 2) Monosynaptic reflex facilitation of synergists. 3) Monosynaptic reflex inhibition of antagonists. 4) Inhibition of homonymous motoneurons through an undefined pathway. 5) Disynaptic reflex inhibition of synergists. 6) Disynaptic reflex inhibition of certain other motor nuclei. 7) Disynaptic reflex facilitation of antagonists.

With this variety of actions chargeable to the group I fibers, the older implicit notion of unity of function is untenable. Fortunately there is no need to resort to assumption and argument for Hunt and Kuffler (18) have demonstrated that both A-type and B-type receptors are represented by afferent fibers in the group I band. Duality of function, therefore, is established.

Myotatic Reflex Mechanism. Monosynaptic reflex discharge, facilitation of synergists, and inhibition of antagonists by group I fibers are associated actions in the pattern of the myotatic reflex (2, 4). Stretch origin of monosynaptic reflexes is proven. Hunt (17) has shown that afferent discharges from muscle spindles can produce discharges of homonymous motoneurons, and facilitation both of homonymous and synergist monosynaptic reflexes. Stretch of a muscle is known to inhibit a myotatic reflex in its antagonist, although there is no proof as yet that muscle spindles are the point of origin. The myotatic reflex mechanism pre-empts to its service one of the two known functional subgroupings of group I fibers, that which, it will be conceded generally, is associated with muscle spindles.

Inverse Myotatic Reflex Mechanism. It is proposed to associate the inhibition of homonymous motoneurons, disynaptic reflex inhibition of synergists and disynaptic reflex facilitation of antagonists, for these actions together represent a precisely reciprocal mechanism within which the direction of action at any given motor nucleus is precisely inverse to that in the monosynaptic system. The inverse myotatic reflex mechanism as proposed would require for its afferent limb the remaining functional subgrouping of the group I fiber band, that associated with tendon organs. Recently, Hunt (17) has shown that the response of homonymous and synergist motoneurons is inhibited, and that of antagonist motoneurons facilitated in circumstances that are known to precipitate tendon organ activity at the expense of muscle spindle activity. One cannot avoid the implication that the effects described by Hunt were mediated through the inverse myotatic reflex mechanism as here described. Nevertheless it is well to remember that the chain of evidence, however impressive, is circumstantial.

Inhibition of Neighboring Muscles. The finding of disynaptic inhibitory connection between muscles that are not partners in a common myotatic unit raises a rather special problem in functional correlation. Unless one is to suppose the existence of a third as yet unknown functional category of group I fibers, that form of inhibition must be associated by common origin in the peripheral receptor apparatus either with the myotatic reflex, or with its inverse, the lengthening reaction. Plausible arguments can be advanced in favor of either alternative, but the statement of them, without experimental basis for preference, would be idle discursion.
Disynaptic inhibitory connection between independent muscles forms a suitable mechanism for the silent period that may appear in muscles other than that subjected to brief stretch (19). Recently Granit (15) found that stretch or contraction of quadriceps may depress the response of gastrocnemius motoneurons and the reverse, and presented his evidence that large afferent fibers were concerned. Those muscles are known (figs. 8 and 9) to be linked by disynaptic inhibitory connection.

SUMMARY

An afferent volley arising in the nerve to a given muscle, or muscle fraction, and traveling in group I afferent fibers has, in addition to the previously known monosynaptic reflex effects (9) the following actions: 1) It inhibits the motoneurons of supply to that muscle or fraction thereof (15-17). 2) Through a disynaptic central pathway it inhibits the response of motoneurons of supply to the muscle remainder or synergists. 3) Through a disynaptic central pathway it facilitates the response of motoneurons that supply antagonists.

Those actions are precisely inverse to the monosynaptic reflex actions which represent the myotatic reflex mechanism. In association they represent an 'inverse myotatic reflex mechanism,' or mechanism of the lengthening reaction.

In addition volleys of the sort described inhibit, through disynaptic connection, the response of motoneurons of certain muscles that are not related in synergism or antagonism. It is not known whether inhibition in these instances is to be associated with the myotatic reflex, or with its inverse, the lengthening reaction. The finding however does reveal a mechanism for silent periods that appear in one muscle when another is involved in a tendon jerk response.

REFERENCES