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# RECURRENT INHIBITION IN THE CAT'S SPINAL CORD

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It was shown by Renshaw (1941) that antidromic stimulation of motor nerves results in a central inhibitory action indiscriminately affecting motoneurones in proximity to the activated cells. Different explanations have been advanced for the mechanism of this 'antidromic' inhibitory action. First, the time course of the inhibition has been correlated with that of the flow of after-currents of active motoneurones, and it has been suggested that these current flows cause the inhibition (Lloyd, 1951). Secondly, Eccles, Fatt & Koketsu (1954) have suggested that impulses in axon collaterals in the ventral horn discharge special interneurones. These neurones, characterized by their high initial firing frequency, were first described by Renshaw (1946), and have therefore been designated as 'Renshaw cells'. Intracellular and extracellular recordings from the ventral horn, and pharmacological investigations (Eccles *et al.* 1954), have indicated that impulses in axon collaterals cause release of acetylcholine (ACh) at their synapses with Renshaw cells, whose subsequent discharges in turn hyperpolarize neighbouring motoneurones; and the evidence strongly suggests that this hyperpolarization leads to 'antidromic' inhibition. Antidromic facilitation has also been observed (Renshaw, 1941), and has now been shown to be mediated by cholinergic axon collaterals (Wilson, 1958). The excitatory junction between axon collaterals and Renshaw cells shares most pharmacological properties with the neuromuscular junction, provided the diffusional barriers peculiar to the central nervous system are overcome. This is true for drugs acting post-synaptically such as anticholinesterases and curare (Eccles, Eccles & Fatt, 1956; Curtis & Eccles, 1958*a*, *b*), as well as for botulinum toxin that acts presynaptically (V. B. Brooks & D. R. Curtis, 1956, unpublished observations). The inhibitory junction between Renshaw cells and motoneurones is pharmacologically indistinguishable from other inhibitory junctions; it can be blocked by strychnine (Eccles *et al.* 1954) and by tetanus toxin (Brooks, Curtis & Eccles, 1957). While 'antidromic' inhibition has been studied by means of antidromic stimulation, it is also brought into play when motoneurones are stimulated orthodromically (Renshaw, 1946). For that

reason Granit, Pascoe & Steg (1957) and Brooks & Wilson (1958), in a preliminary communication to this paper, have substituted the term 'recurrent' inhibition for 'antidromic' inhibition.

Since the inhibition is not restricted to any particular group of motoneurones, but rather affects all nuclei close to the active cells (Renshaw, 1941), it has been thought unlikely (Eccles *et al.* 1954; Eccles, 1955) that it has a co-ordinating function, but instead that such a negative feed-back mechanism has an indiscriminate anticonvulsant action. The present experiments further link Renshaw cell discharge with recurrent inhibition and exclude after-currents between motoneurones as a significant cause for the inhibition. The main purpose of the work, however, is to demonstrate a co-ordinating function for recurrent inhibition, which has been suggested independently by Burns (1958). It will be shown that a general inhibitory action, that damps the discharge of nuclei in close proximity to an active nucleus, decreases or prevents spread of reflex responses from the nuclei of activated muscles. Such spread does occur, and it has been studied most closely by Alvord & Fuortes (1953). The inhibitory feed-back of recurrent inhibition thus helps to confine stretch reflexes to their paths of afferent origin.

## METHODS

These experiments have been carried out in unanaesthetized spinal or decerebrate cats. In the spinal preparations the cerebral circulation had been shut off by tying off the common carotid arteries and clamping the vertebral arteries. The animals were prepared under ether, and experiments were begun not less than 2 hr after cessation of ether administration.

Some experiments were concerned mainly with the action of dihydro-beta-erythroidine (DHE; kindly supplied by Merck and Co., U.S.A.) on antidromic inhibition, Renshaw cell discharge and antidromic electrotonus. For this purpose, laminectomy was performed on cats whose spinal cords had been divided at the upper cervical level. The dorsal and ventral roots of the lumbosacral segments were cut on one side. The animal was immobilized by means of intravenously injected Flaxedil (gallamine triethiodide, American Cyanamid Co., U.S.A.), a drug which, when given I.V. exerts no effect on Renshaw cell discharge or antidromic inhibition. Paralysis during control runs was essential in order to make the levels of excitation before and after injection of DHE comparable. Maximal monosynaptic test reflexes were evoked by stimulation of a cut dorsal root and recorded in a fraction of the corresponding ventral root. This reflex was inhibited by a conditioning volley in the remainder of the ventral root. The experimental arrangement is shown diagrammatically in Fig. 1*A*. Renshaw cell discharge was recorded at the dorsolateral surface of the cord (Eccles *et al.* 1954); antidromic electrotonus was recorded in the manner described by Lloyd (1951).

The recovery curve of motoneurones was studied in similar preparations. A maximal test reflex was elicited by stimulating a dorsal root and recorded in the corresponding ventral root. The test stimulus was preceded at various intervals by a conditioning antidromic volley in the whole ventral root.

The bulk of the experiments dealt with the effect of DHE and of strychnine on homonymous and heteronymous reflex discharge. For this purpose the roots on one side were left intact. The pair of nerves used for study (e.g. medial and lateral gastrocnemius) were cut, and both a recording and a stimulating electrode pair were placed on the central end of each. This type of experimental arrangement is illustrated in diagram *B* of Fig. 1. Reflexes were then elicited by repetitive stimulation at frequencies of 50–200/sec. It is possible to evoke both homonymous and

heteronymous discharges with this type of stimulation, as was shown by Alvord & Fuortes (1953). Furthermore, each response falls into the period of inhibition caused by the preceding one. Recurrent inhibition of the heteronymous reflex is brought about in two ways. First, antidromic stimulation of the motor axons in the homonymous path results in activation of the inhibitory mechanism, and secondly, the inhibition is again brought into action by orthodromic activation of homonymous, and possibly heteronymous, motoneurones.

In some of the animals one side was prepared for study of homonymous and heteronymous reflex discharges; the other side was prepared as described previously for the study of antidromic inhibitory curves. The effects of drugs on inhibitory curves and high frequency reflex discharge could be compared in the same animal. The temperature of the animals and of the oil pool was maintained at 36–37 and 35–36° C, respectively.

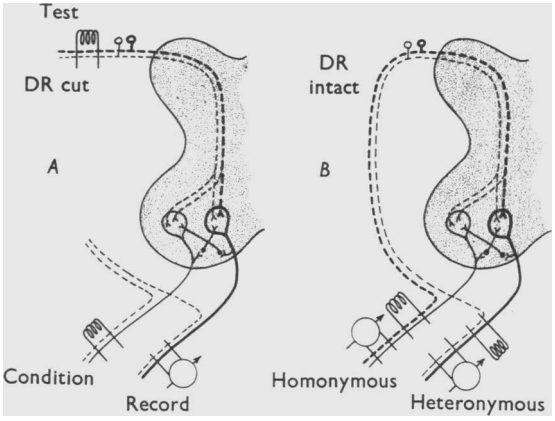


Fig. 1. Diagrams of the experimental arrangements. For description see text.

## RESULTS

### *The relation between recurrent inhibition, Renshaw cell discharge and ventral root electrotonus*

A monosynaptic reflex, evoked by stimulating a dorsal root and recorded in part of the corresponding ventral root, can be inhibited by a preceding antidromic volley in the remainder of the ventral root (see Fig. 1*A*). A typical inhibitory curve obtained in such a manner in a decerebrate preparation is illustrated in Fig. 2*A*. Ventral root electrotonus (Lloyd, 1951) was recorded in the same experiment (Fig. 2*B*); and an electrode on the surface of the cord was used to record the rhythm of synchronized discharges of Renshaw cells activated by the antidromic volley (Eccles *et al.* 1954) (Fig. 2*C*). Intravenous injection of dihydro-beta-erythroidine (DHE) 0·4 mg/kg greatly reduces antidromic inhibitory action. The effect of DHE on the inhibitory curve, as shown in Fig. 2*A*, is typical of the result of many experiments. Inhibition during the first few milliseconds is not affected, but the peak is usually reduced; the later inhibition is substantially diminished. In many preparations the duration of inhibition is shortened, but it is never removed completely. The effect of the same injection of DHE on ventral root electrotonus and on Renshaw cell

discharge can be seen in Fig. 2*B* and *C*. On the one hand, ventral root electrotonus is not significantly diminished; the small difference between the control and the electrotonus recorded after injection of the drug is no greater than differences between controls recorded at various times during the experiment. On the other hand, it is clear that the duration of Renshaw cell discharge is greatly shortened, as previously described by Eccles *et al.* (1954).

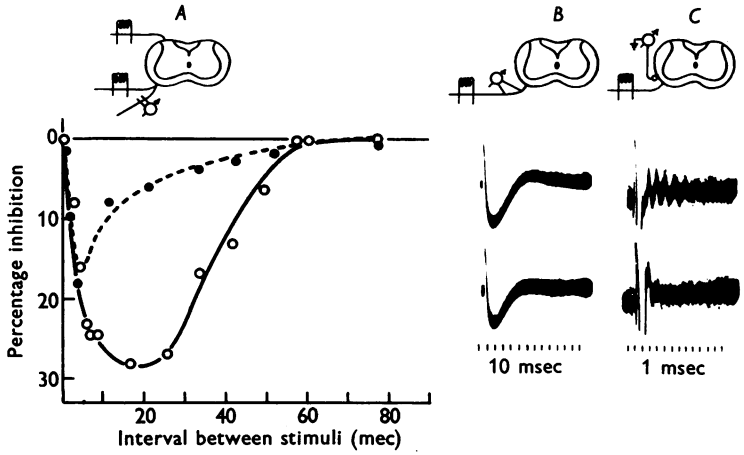


Fig. 2. *A*: Recurrent inhibitory curves obtained in a decerebrate preparation. Circles show: ○, inhibition before; ●, after, intravenous injection of DHE 0·4 mg/kg. Each point was determined from 20 superimposed reflex responses. For procedures see text. *B*: Effect of the same injection of DHE on ventral root electrotonus. For *B* and *C*: upper traces, controls; lower traces, after injection of DHE; diagrams show the experimental arrangements. *C*: Effect of the same injection of DHE on Renshaw cell discharge recorded from the surface of the cord.

The remaining initial synchronous discharge of Renshaw cells after the action of DHE is not due to insufficient dosage: Curtis & Eccles (1958*b*, fig. 6) found that the initial discharge of individual cells remains after close application of DHE. This cellular resistance thus probably accounts for the remaining inhibition (Fig. 2*A*) and for the remaining Renshaw cell discharge (see lower trace, Fig. 2*C*).

The effect of DHE on recurrent inhibition is further illustrated in Fig. 3, which also shows the action of strychnine on the inhibition. In this preparation the lateral and medial gastrocnemius nerves were employed, the reflex in one branch being inhibited by antidromic stimulation of the other. Injection of DHE 2·0 mg/kg reduced the inhibition, but a considerable amount remained.

Subconvulsive doses of strychnine do not affect cell discharge but they reduce antidromically evoked hyperpolarizations in motoneurones (Eccles *et al.* 1954). This effect on the inhibitory junction is similar to the reduction of direct inhibition by strychnine (Bradley, Easton & Eccles, 1953). In preparations anaesthetized with Nembutal (pentobarbitone, Abbott Laboratories)

this is accomplished with doses of strychnine in the order of 0·1 mg/kg. Much smaller doses are required to obtain a similar effect on recurrent inhibition in spinal and decerebrate preparations. Figure 3 shows the effect of strychnine 0·03 mg/kg on inhibitory curves previously diminished by DHE: the inhibition is decreased further, but not completely abolished.

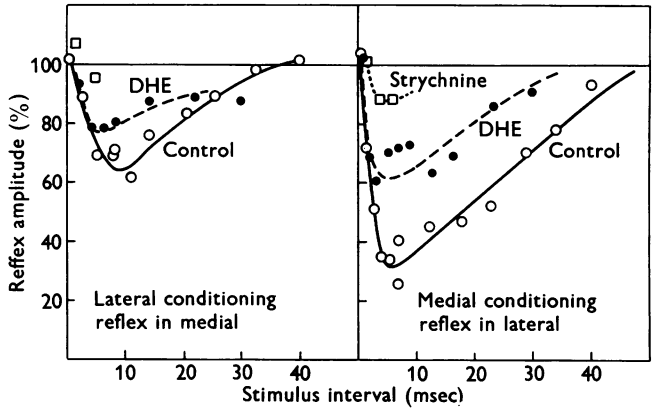


Fig. 3. Inhibitory curves obtained by conditioning a monosynaptic rest reflex, elicited by dorsal root stimulation and recorded in either branch of the gastrocnemius nerve, by antidromic stimulation of the other branch. Decerebrate preparation. ○, control; ●, after intravenous injection of DHE 2 mg/kg; □, after a subsequent intravenous injection of strychnine sulphate 0·03 mg/kg. Each point was determined from approximately 20 superimposed reflex responses.

*Recurrent inhibition and motoneurone recovery*

Following invasion of a motoneurone pool by a maximal antidromic volley, monosynaptic reflexes set up in this pool by subsequent orthodromic volleys are depressed for periods as long as 120 msec (Lloyd, 1951). Stimulation of the ventral root will not only result in invasion of the motoneurones by antidromic impulses but also in activation of the recurrent inhibitory mechanism (see Eccles *et al.* 1954, fig. 2). Therefore, part of the depression is due to recurrent inhibition. The contribution of recurrent inhibition to the recovery curve is illustrated in Fig. 4. At stimulus intervals greater than 50–60 msec the control recovery curve and the one obtained after injection of DHE are essentially similar. However, the drug removes a considerable amount of the early part of the depression. The depression which remains must still contain a contribution from recurrent inhibition, since DHE never completely blocks the inhibitory mechanism.

*Recurrent inhibition of homonymous and heteronymous reflexes*

Threshold stimulation of the nerve to one head of a muscle with repetitive shocks at frequencies of 100/sec or higher elicits a reflex in the stimulated nerve

(homonymous reflex) and, in favourable preparations, in the nerve to the other head of the muscle (heteronymous reflex) (Alvord & Fuortes, 1953). Reflexes elicited by stimulation in the range of approximately 40–175/sec were studied in the present experiments. Heteronymous reflexes were observed in half the cases, usually appearing even at frequencies as low as 40/sec. Lloyd (1957) has described temporal summation of monosynaptic reflexes in the frequency range of 60 and 100/sec in spinal animals under Nembutal anaesthesia. Similar

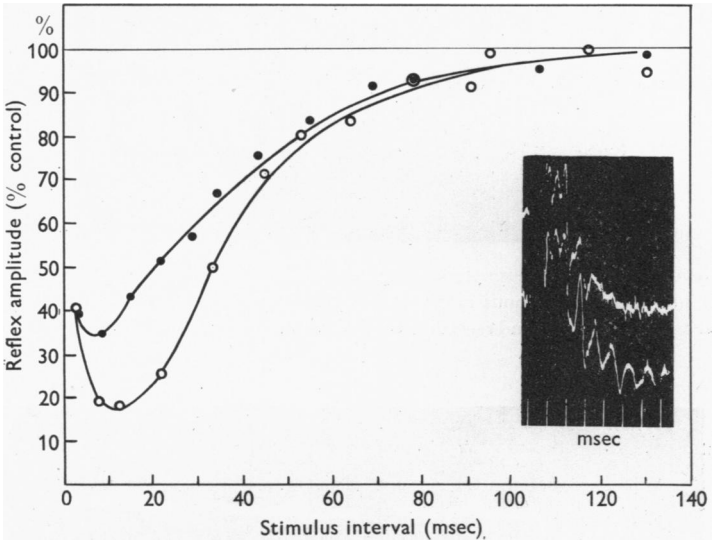


**Fig. 4.** Plots of amplitudes of monosynaptic reflexes evoked by stimulation of the 7th lumbar dorsal root (ordinate) when conditioned by preceding antidromic volleys in the whole 7th lumbar ventral root: abscissa, stimulus interval. Circles show: ○, motoneurone recovery curve before, ●, after, intravenous injection of DHE 0·7 mg/kg. Inset shows records of Renshaw cell discharge recorded at the cord surface before and after injection of DHE (lower and upper traces, respectively). Spinal aninal.

results were obtained in this study: increases of both homonymous and heteronymous sustained reflexes were observed as the frequency of stimulation was increased from 50 to about 100/sec. At higher rates of stimulation reflex amplitudes usually decreased. In a few animals no temporal summation was seen at the frequencies studied. In the experiments described above, DHE has been shown to reduce recurrent inhibition. The following experiments have been performed to study the effect of this drug on homonymous and heteronymous reflex discharges.

Figure 5 shows the result of an experiment in which two branches of the nerve to quadriceps were used for stimulating and recording. In the control, stimulation of either branch produced homonymous responses, but no heteronymous response could be seen. Injection of DHE 2 mg resulted in an increase

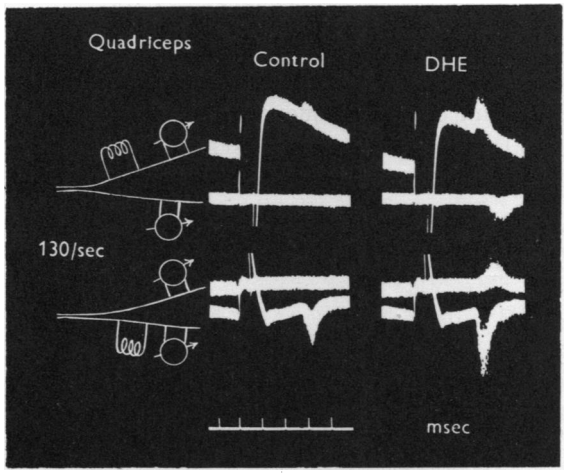


Fig. 5. Homonymous and heteronymous responses in branches of the quadriceps nerve are shown before and after intravenous injection of DHE 0·9 mg/kg. About 20 superimposed traces per record. Stimulating and recording conditions are shown in the diagrams. Decerebrate cat.

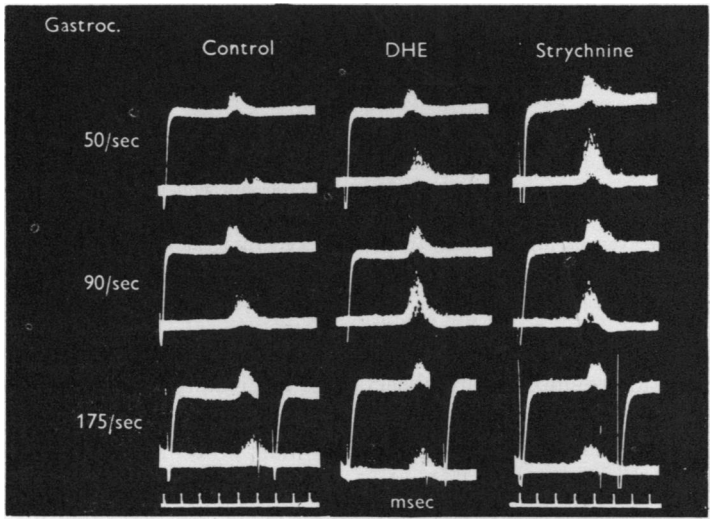


Fig. 6. Homonymous and heteronymous reflexes obtained by stimulating the medial branch of the gastrocnemius nerve, in a decerebrate cat. In each case the upper beam, with the large stimulus artifact, shows the homonymous response. For the different frequencies illustrated the three columns show the control responses, the response after intravenous injection of DHE, 1 mg/kg, and the response after a subsequent intravenous injection of strychnine 0·03 mg/kg. About 20 superimposed traces per record.

in homonymous reflexes, and also brought about the appearance of heteronymous responses.

Similar results have been obtained in preparations in which both homonymous and heteronymous responses were present. Such an experiment, carried out with stimulation of the medial gastrocnemius nerve, is illustrated in Fig. 6. Intravenous injection of DHE 1 mg/kg had little effect on the homonymous response, but caused considerable increase in the heteronymous reflex, particularly at frequencies of stimulation up to 90/sec. This can be seen in Fig. 6, and also in Fig. 7, where the absolute size of the response before and

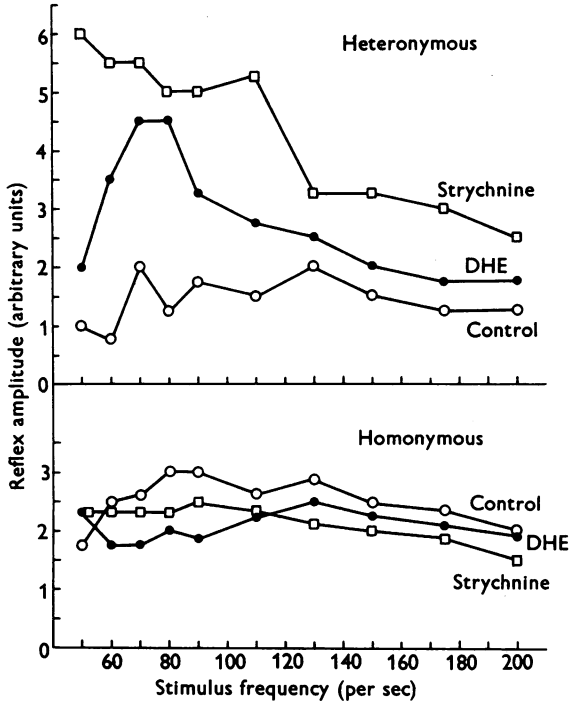


Fig. 7. Plots of reflex amplitudes (ordinates) against stimulus frequency (abscissae) for controls, after intravenous DHE and after intravenous strychnine; same experiment as Fig. 5. Upper set of graphs, heteronymous reflexes; lower set, homonymous reflexes.

after injecting DHE is plotted as a function of stimulus frequency. This type of result has been obtained frequently. Homonymous and heteronymous responses have been studied in seventeen cases in nine preparations. Heteronymous reflexes were increased by DHE in ten cases, unchanged in seven; homonymous reflexes were increased in five cases, unchanged in twelve. The lack of change in heteronymous reflexes, seen in several cases, may be due to several factors, among them the fact that DHE removes only part of the recurrent inhibition (Figs. 2, 3). After recurrent inhibition has been decreased

by DHE it can be diminished further by subconvulsive doses of strychnine (Fig. 3). Similarly, such doses of strychnine, following an injection of DHE, further enhance heteronymous reflexes (Figs. 6, 7).

While all these results have been obtained in extensor nuclei in decerebrate preparations, similar effects have been seen with flexor nuclei (biceps-semitendinosus) in spinal animals. In those experiments the effect has been weak, possibly owing to the lower level of excitability of spinal animals as compared with decerebrate ones.

## DISCUSSION

The experiments described in the first part of Results provide further evidence for the cholinergic nature of the recurrent inhibitory pathway: the recurrent inhibitory curve is modified by DHE and strychnine in the same way as are Renshaw cell discharge and antidromically evoked hyperpolarization of motoneurones (Eccles *et al.* 1954). It also has been shown that eserine, which lengthens the duration of Renshaw cell discharge (Eccles *et al.* 1954) increases the duration of the recurrent inhibitory curve (Wilson, 1958). In contrast, ventral root electrotonus is not modified by DHE. Furthermore, two tests of electrotonus on roots adjacent to active segments have shown it to be very small, although recurrent inhibition there is very pronounced. All the evidence therefore indicates that recurrent inhibition follows activation of Renshaw cells, as described by Eccles *et al.* (1954) and that after-currents between motoneurones do not contribute markedly to production of the inhibition.

The myotatic reflex originates from stretch of muscle fibres. The afferent path of this reflex is conducted from muscle spindles to the cord through large Group I fibres (Ia) (Lloyd, 1943; Hunt, 1954) that make synaptic connexions not only with motoneurones of the muscle or fraction of a muscle which they supply (homonymous connexions) but also, through afferent branches, with motoneurones of synergistic muscle groups (heteronymous connexions). Notwithstanding these connexions, the myotatic reflex, evoked by muscle stretch usually 'remains confined not only to the particular muscle stretched, but to that *portion* of it which stretch affects' (Liddell & Sherrington, 1924, authors' italics). Similarly, electrically evoked monosynaptic reflexes normally reflect only into the stimulated muscle nerve (Lloyd, 1943).

However, there are many examples of involvement of heteronymous motoneurones, although often such action manifests itself only as facilitation between synergic units (Lloyd, 1946). Under appropriate conditions, heteronymous motoneurones can be made to discharge. Monosynaptic testing with single shocks has shown that cells normally fired by one of a pair of synergists can be discharged by another following application of a background of steady stretch (Granit & Ström, 1951); after cooling of the spinal cord (Lloyd, Hunt & McIntyre, 1955); and after post-tetanic potentiation (Beswick & Evanson, 1955; Lloyd *et al.* 1955). Similarly, repetitive stimulation at frequencies of

50 and more per second is often sufficient to start heteronymous firing (Alvord & Fuortes, 1953). Clearly, in the normal course of events the stretch reflex maintains a high degree of localization, which can, however, be overcome under conditions of strong facilitation. The present experiments show that recurrent inhibition plays a part in the preservation of this localization.

Some of the experimental conditions must be considered in the interpretation of the results. The demonstration of heteronymous reflexes is made difficult by the necessity of performing the experiment on paralysed animals: reduction of muscle tension reduces muscle spindle activity and thus also the central excitatory state. On the other hand, the discriminatory power of recurrent inhibition between homonymous and heteronymous reflexes is increased by dealing with weak heteronymous reflexes. Furthermore, in the experiments as performed, the motoneurones of the homonymous pathway were activated orthodromically as well as antidromically. Therefore the homonymous nucleus is in a state of depression from continuous antidromic activation. This depression (Fig. 4), which is only slightly affected by DHE, may partially account for the finding that homonymous reflexes are less influenced by DHE than are heteronymous ones. The recurrent inhibitory system is inoperative in preventing spread when test reflexes are evoked by single shocks, as was pointed out by Lloyd (1943); the recurrent volley then arrives too late. In that case localization rests on the relatively greater power of homonymous connexions. However, in the case of natural reflexes originating with muscle stretch, where motoneurones fire asynchronously at frequencies of 5–100/sec (Adrian & Bronk, 1929), the same conditions apply as in the present experiments, carried out with repetitive synchronous nerve stimulation in that frequency range.

Thus there do not appear to be serious obstacles to the conjecture that localization of stretch reflexes is a function of recurrent inhibition in the normal animal. Presumably such localization subserves execution of fine movements which are known to depend upon accurate perception of the extension of muscles, mediated by muscle spindles, and adjusted by stretch reflexes. The reduction of the functional field of spindle receptors in the cord by recurrent inhibition may also be considered to be related to analogous afferent phenomena. Inhibitory systems that prevent spread to neighbouring units are known for many kinds of sensations (Brooks & Wilson, 1958). This problem is discussed in a consideration of interaction of cortical motor cells (Brooks, 1959).

Granit *et al.* (1957) have suggested another function for recurrent inhibition: 'stabilizing the sustained output of impulses in the stretch reflex at a slow rate, thereby serving as an efferent antagonist of cumulative, presynaptic excitatory states produced by the gamma-driven nuclear bag afferents'. It is indeed to be expected that structures as numerous as axon collaterals would serve more than one function.

SUMMARY

1. The intensity and duration of recurrent inhibition is reduced by intravenous injection of dihydro-beta-erythroidine (DHE) in doses sufficient to reduce discharge of Renshaw cells; but ventral root electrotonus is unaffected by DHE. From this it is concluded that current flow between motoneurones does not contribute significantly to recurrent inhibition, which is produced by activity of Renshaw cells.

2. Reduction of recurrent inhibition is accompanied by increase of heteronymous reflexes, and also sometimes by increase of homonymous reflexes. From this it is also concluded that it is a function of recurrent inhibition to limit stretch reflexes to their stimulated paths. The significance of these findings is discussed.

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