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RECURRENT ACTIONS ON γ-MOTONEURONES MEDIATED VIA LARGE AND SMALL VENTRAL ROOT FIBRES IN THE CAT

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SUMMARY

1. Effects on single lumbar γ-motoneurones, mediated via fibres running in the ventral roots, were studied by micro-electrode recording in cats anaesthetized with chloralose. Graded electrical stimulation of ventral roots or of peripheral nerves was used.

2. The cells were identified as γ-motoneurones by antidromic stimulation and by measurement of their axonal conduction velocity. Some of the cells were classified as static or dynamic.

3. The findings confirm the previously demonstrated existence of low-threshold, presumed recurrent, inhibition of both static and dynamic γ-motoneurones.

4. Strong evidence for the occurrence of high-threshold recurrent inhibition of γ-motoneurones is also presented.

5. In addition, excitatory effects on γ-cells, also mediated via fibres in the ventral roots, are described.

6. The low-threshold effects from ventral root fibres are attributed to recurrent α-collateral activity and the high-threshold effects to γ-collateral activity.

7. The significance of recurrent inhibition of γ-motoneurones is discussed in relation to the 'gain regulator' concept proposed by Hultborn, Lindström & Wigström (1979).

INTRODUCTION

The activity of lumbar γ-motoneurones is influenced by a complex pattern of excitatory and inhibitory reflexes from skin, joint and muscle afferent fibres. This was recently shown in experiments using electrical stimulation of peripheral nerves (Appelberg, Johansson & Kalistratov, 1977; Appelberg, Hulliger, Johansson & Sojka, 1983*a–c*). Both excitatory and inhibitory muscle afferent reflexes to the γ-cells came from wide receptive fields and could be conveyed by group II as well as higher threshold muscle afferents. In contrast, activation of group I muscle fibres very rarely had excitatory or inhibitory effects (Appelberg *et al.* 1983*a*). As group I inhibitory

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† Where all the experimental work was done.

effects were regularly elicited from non-autogenetic and non-synergistic nerves, the risk that some of them could be of recurrent nature was considered negligible. Nevertheless, since stimulation of both autogenetic and heteronymous muscle nerves at higher intensities often had powerful inhibitory effects on γ-cells, it was deemed important to examine whether such stimuli could at times act via recurrent collaterals.

Previous work on recurrent inhibition of γ-motoneurones (Brown, Lawrence & Matthews, 1968; Ellaway, 1968, 1971; Grillner, 1969; Noth, 1971) indicates that such effects are usually weak and that they are mediated via Renshaw cells activated from α-collaterals, acting on γ-cells in a way identical to that well known for recurrent inhibition of α-motoneurones. On the other hand, it is still a matter of debate whether, in addition to the contribution from α-motoneurones, recurrent inhibition via γ-cell axon collaterals is at all possible. Two lines of evidence suggest that such γ- to γ-cell recurrent inhibition should at best be rather weak. First, morphological studies using labelling of individual cells, with horseradish peroxidase, suggested that recurrent collaterals from γ-motoneurones are very rare (Westbury, 1979; Cullheim & Ulfhake, 1979). Secondly, electrophysiological investigations of recurrent inhibition of γ-motoneurones failed to demonstrate any further enhancement of inhibition when the intensity of antidromic stimulation of muscle nerves was increased to include γ-axons also (Ellaway, 1971). Clearly the interpretation of such findings is complicated by the possibility that the recurrent inhibitory pathway was more or less saturated by a maximum input from α-collaterals and that any γ-collateral contribution was thus hidden.

The present experiments confirm the view that recurrent inhibition of γ-motoneurones is less common and usually weaker than that of α-motoneurones. Occasionally, however, surprisingly potent effects from α-motoneurone collaterals were found. Furthermore, new information concerning three separate aspects of recurrent control of γ-motoneurones was obtained. First, with the aid of the classification method, based upon mesencephalic stimulation (see recent review by Appelberg, 1981), half of the γ-cells were classified as static or dynamic, and it was shown that either type of cells could receive recurrent inhibition (see also Ellaway, 1971). Secondly, clear evidence is presented showing that γ-cells could also receive recurrent inhibition from γ-fibres. Thirdly, in a few cases antidromic activity in ventral roots elicited excitation of γ-cells similar to the phenomenon of recurrent excitation described for α-motoneurones (Hultborn, Jankowska, Lindström & Roberts, 1971).

METHODS

All experiments in this series (Appelberg, Hulliger, Johansson & Sojka, 1983*a–c*; Johansson, 1981) were performed on cats anaesthetized with chloralose and paralysed with gallamonium iodide. The animals were artificially ventilated. The surgery comprised dissection of left hind-limb nerves for their electrical stimulation and laminectomy to provide access to lumbar motoneuronal nuclei for micro-electrode recording of single γ-cells. Further, craniotomies were made for stimulation with two grids of platinium–iridium electrodes in the right rubral area. The first grid was used for classification of γ-cells as static or dynamic via stimulation in the mesencephalic area for dynamic control. The second grid was used for indirect stimulation of rubro-spinal tract via interposito-rubral fibres (Appelberg, 1981; Johansson, 1981).

Methodologically, the present experiments differed only marginally from the study of Appelberg *et al.* (1983*a*) where the full account of the general methods used for single unit recording from γ-cells in the spinal cord has been given. Therefore this section is largely confined to the specific aspects of the investigation of actions on identified γ-motoneurones mediated by fibres passing through the ventral roots.

The present paper is based upon six successful experiments on cats weighing 2·4–4·4 kg. Four of these experiments were only concerned with the investigation of actions mediated by ventral root fibres. In these animals the ipsilateral dorsal roots of the L_5 (L_4 in one experiment) to S_1 segments were cut. In the two remaining animals these roots were left intact, since the experiments were also used for other tests with orthodromic stimulation of afferent fibres entering the spinal cord through the dorsal roots (Appelberg *et al.* 1983*a–c*).

Operation and denervation. In all six experiments a widespread denervation of the left hind limb was performed (Appelberg *et al.* 1983*a*), and a number of hind-limb muscle nerves were dissected free and cut, so that their central ends were available for electrical stimulation. In all animals except one the sensory-motor cortex was removed.

Stimulation. Generally the same procedures were used as in the reflex studies referred to. However, particularly careful attention was paid to the accurate measurement of the intensities of stimulation which were employed for the antidromic activation of α- and γ-motor fibres in peripheral muscle nerves and in ventral roots. In particular, the absolute thresholds for activation of the most excitable α-fibres were regularly checked. Appropriate measures were taken to keep the stimulating conditions as constant as possible (maintenance of good contact between nerve tissue and electrodes, and removal of any tissue fluid which might have short-circuited the nerve fibres and caused spread of excitation). The intensities of stimulation were always expressed as multiples of the most recently determined value of α-threshold intensity. When ventral roots were stimulated, the threshold for α-fibres was determined by monitoring the antidromic α-field potential in a motor nucleus whose efferent axons were passing through this particular ventral root. With stimulation of peripheral muscle nerves the threshold for α-fibre activation was determined by recording either the compound action potentials in ventral roots or the α-field potentials in the appropriate motor nuclei.

Recording. Nearly all observations on recurrent effects in the present study were made with extracellular recordings. As the search was for inhibitory effects, spontaneous activity of the cells was a necessary prerequisite. Peripheral nerves or ventral roots were stimulated at a repetition rate of 1–2/sec and the responses of the cells were superimposed for a number of cycles, usually ten to fifty. An inhibitory effect then revealed itself as a pause or a manifest reduction of the spontaneous discharge of the cell.

Abbreviations used in the text

D.p., deep peroneal nerve (pure muscle nerve supplying extensor digitorum longus and tibialis anterior); g.s., gastrocnemius and soleus (triceps) (nerves or muscles); HRP, horseradish peroxidase; L, lumbar; p.b.s.t., posterior biceps and semitendinosus (nerves or muscles); S, sacral; *T*, threshold; v.r., ventral root.

RESULTS

Single neurone activity was recorded, usually extracellularly, with micro-electrodes placed in the motor nuclei of hind-limb muscles in the L_6–S_1 segments of the left spinal cord. The cells were identified as motoneurones with conduction velocities in the γ range by antidromic stimulation of their parent nerve. Usually an attempt was also made to classify the cells as static or dynamic using electrical stimulation in a particular mesencephalic area which is known to influence only dynamic cells (Appelberg, 1981).

Recurrent actions mediated through the ventral roots were studied by the use of electrical stimulation of either various hind-limb muscle nerves or of individual ventral roots of the L_6–S_1 segments. In the two animals with intact dorsal roots only the latter form of stimulation was used.

The present sample of responsive cells consists of thirteen γ-motoneurones (four dynamic, two static and seven non-classified) which could be influenced by electrically evoked activity reaching the spinal cord through ventral roots.

Low-threshold inhibition from ventral root fibres. Fig. 1 gives an example of a dynamic p.b.s.t. γ-motoneurone which was markedly inhibited by antidromic stimulation

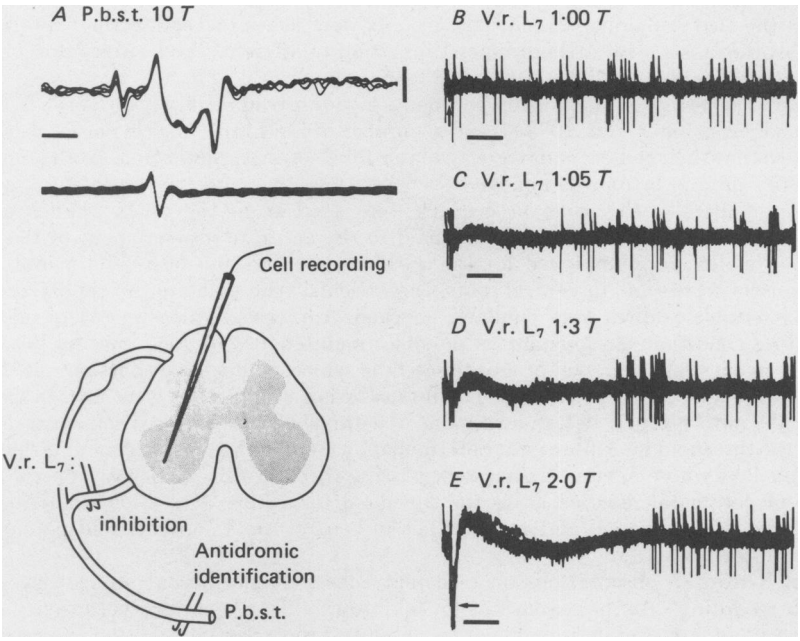

Fig. 1. Inhibition of a dynamic p.b.s.t. γ-motoneurone by low-threshold stimulation of axons passing through the ventral root L_7 (v.r.L_7). The cell was identified by recording the antidromic action potential (*A*, upper trace) elicited by stimulation of the p.b.s.t. nerve (*A*, lower trace, incoming volley recorded from dorsal root) at 10 times α-threshold (latency 2·8 msec; conduction velocity 36 m/sec). In *B*, absence of effects with stimulation at α-threshold (1·00 *T*, stimulation strength eliciting a just noticeable α-field potential; see arrow). In *C* and *D*, inhibition caused by ventral root L_7 stimulation at 1·05 and 1·3 times α-threshold. In *E*, antidromic invasion of the cell (arrow) following ventral root L_7 stimulation at 2·0 times α-threshold. All records show extracellular recordings of spike activity with the micro-electrode placed in the p.b.s.t. nucleus at the L_7 segment. Twenty superimposed sweeps in all records, except *A* (five sweeps). Preparation (see inset) with intact dorsal roots. Horizontal calibrations: bar in *A* = 1 msec; bars in *B*–*E* = 10 msec. Vertical calibrations: bar in *A* (applicable to all recordings) = 0·2 mV.

within the α range of intensities. The cell was recorded in a preparation with intact dorsal roots. Therefore, antidromic stimulation had to be performed on ventral roots rather than on peripheral nerves (see inset of Fig. 1). Using a collision technique with paired stimulation of individual ventral roots and the p.b.s.t. nerve, it could be established that the axon of the cell left the cord through the L_7 ventral root. This was also the root from which the most pronounced inhibitory effects could be elicited. The cell, which was spontaneously active, did not react to stimulation of ventral root L_7 at 1·00 times the threshold for α-cells (Fig. 1*B*; the antidromic field potential was

just noticeable and is marked by the arrow). However, already at 1·05 times the α-threshold a clear inhibition of the spontaneous discharge was elicited. The inhibition lasted for about 40 msec (Fig. 1*C*). The size of this inhibition increased further to about 55 msec, by stimulation of the ventral root L_7 at 1·3 times threshold (Fig. 1*D*). Finally, at 2·0 times threshold the axon of the γ-motoneurone itself was antidromically activated in the ventral root (Fig. 1*E*, γ-spike seen as a large downwards deflexion superimposed on the α-field potential, see arrow). At this intensity the pause was further prolonged. However, this can no longer be attributed with certainty to a further enhancement of the inhibition, since the antidromic impulse most probably caused a resetting of the spontaneous discharge of the cell.

As always in preparations with intact dorsal roots, the α-threshold was determined by monitoring the antidromic field potential in the motor nucleus while stimulating the associated ventral root (Fig. 1, inset and Methods). With the γ-cell of Fig. 1 the first weak inhibition could be elicited at a stimulation intensity as low as 1·05 times α-threshold. In these situations special care was taken with ventral root stimulation, to ensure that the root was not short-circuited by tissue fluid. It seems very unlikely, therefore, that the inhibition shown in Fig. 1 was brought about by spread of stimulation and orthodromic activation of afferent axons reaching the spinal cord via dorsal roots.

Similar inhibitory effects on lumbar γ-motoneurones from low-threshold ventral root fibres were found in eight cells. Of the four cells classified as dynamic, all showed such low-threshold inhibition, either from ventral roots (one cell) or from muscle nerves in preparations with cut dorsal roots (three cells). The same type of low-threshold recurrent inhibitory action was seen also in one of the two cells classified as static and in three of the seven non-classified cells.

High-threshold inhibition from ventral root fibres. Fig. 2 shows responses to muscle nerve stimulation of a γ-motoneurone which was identified from the p.b.s.t. nerve (Fig. 2*A*) but not further classified. The cell was studied in a preparation with cut dorsal roots. Therefore, responses to stimulation of muscle nerves, as well as of ventral roots, could be relied on for the investigation of antidromic reflex action. It was found that autogenetic (from p.b.s.t., Fig. 2*D–F*) but not heteronymous (from g.s., Fig. 2*B*) inhibitory actions could be elicited. However, inhibitory effects were not evoked at low threshold (within the α-range), as for the cell in Fig. 1. In Fig. 2*C* the p.b.s.t. nerve was stimulated at 2·5 times α-threshold. At this intensity all the α-fibres were probably excited, as is also indicated by the size of the α-field potential, which was the same with 2·5 as with 50 times threshold (see arrows in Fig. 2*C* and *F*). Yet, at 2·5 times threshold the cell did not show any sign of being influenced. However, when the intensity of stimulation of the p.b.s.t. nerve was raised to 5 times threshold, an inhibition lasting some 25 msec was evident (Fig. 2*D*). At 25 times threshold (Fig. 2*E*) the period of silence was increased to about 30 msec. However, as the cell was now also antidromically activated the prolongation of the silent period could not safely be ascribed to a further increase of inhibition. Nevertheless, since with stimulation at 50 times threshold (Fig. 2*F*) the period of silence was still further increased (40 msec), a further enhancement of a true inhibitory action must have occurred. The contribution to the duration of the silent period from resetting must have been the same at 25 and 50 times threshold.

High-threshold recurrent inhibitory effects were seen in seven cells altogether; three of these were classified as dynamic, four belonged to the non-classified category. Pure high-threshold effects, with no effect whatsoever from low-threshold ventral root fibres, were seen in four of these seven cells. In the remaining three cells the duration of the inhibition clearly increased once high-threshold fibres were also activated (as with the cell of Fig. 2).

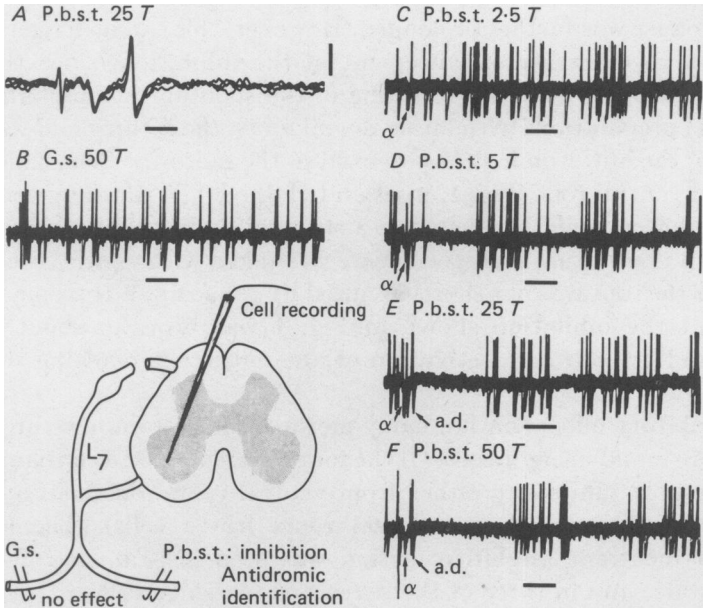

Fig. 2. Autogenetic inhibition of a p.b.s.t. γ-motoneurone mediated exclusively by high-threshold ventral root fibres which were activated in the muscle nerve. The cell was identified by recording the antidromic action potential (*A*), evoked by stimulation of the p.b.s.t. nerve at 25 times α-threshold (latency 4·3 msec; conduction velocity 23 m/sec). This cell was not further classified by means of stimulation of the mesencephalic area for dynamic control. In *B*, absence of effects with stimulation of the g.s. nerve at 25 *T* and, in C, with stimulation of the p.b.s.t. nerve at 2·5 *T*. Stimulation of the p.b.s.t. nerve at 5 times α-threshold (*D*) revealed inhibition with a silent period lasting about 25 msec. With stimulation at 25 times α-threshold (*E*) the silent period lasted 30 msec, and at 50 *T* (*F*) it was 40 msec (see text). The arrows (α) in *C*, *D*, *E* and *F* indicate the α-field potential. Note that the α-field potential is of about of the same size with 2·5 as with 50 times threshold. The arrows (a.d.) in *E* and *F* point out the antidromic spikes. All records show superimposed responses (fifteen sweeps, apart from *A*: five sweeps) to single-shock stimulation. Preparation (see inset) with dorsal roots L_5–S_1 cut. Horizontal calibrations: bar in *A* = 2 msec; bars in *B*–*F* = 10 msec. Vertical calibrations: bar in *A* (applicable to all recordings) = 0·2 mV.

Mixed low- and high-threshold inhibition from ventral root fibres. The evidence that inhibitory effects were elicited solely from either low-threshold or high-threshold fibres was not as clear for all cells as in Figs. 1 and 2. In some cases inhibitory actions from high-threshold fibres were demonstrated while low-threshold effects could not be wholly excluded. In other instances genuinely 'mixed' effects were encountered, when it could be demonstrated that contributions to maximum inhibition came from

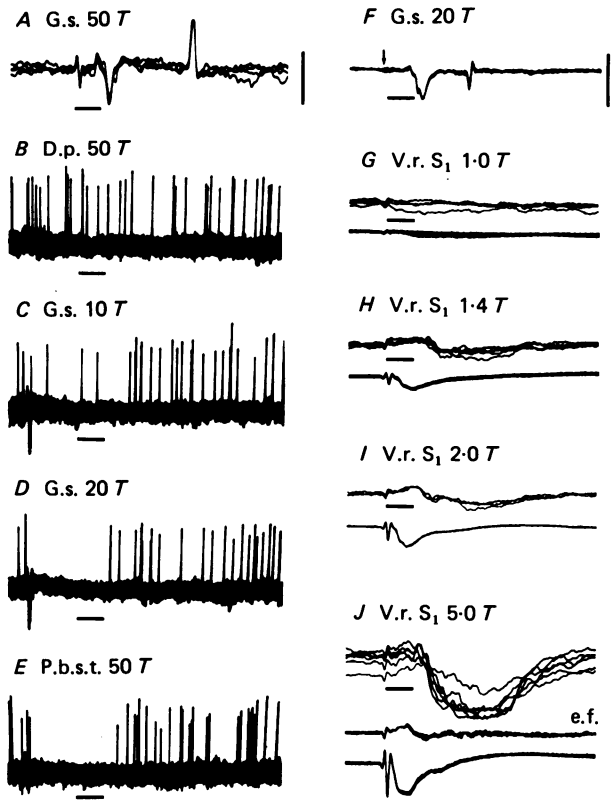

Fig. 3. Mixed low- and high-threshold inhibition of γ-motoneurones mediated via fibres passing through the ventral roots. Responses of two non-classified cells. *A–E*. In *A*, antidromic identification by stimulation of the g.s. nerve at 50 times α-threshold (latency 8·4 msec; conduction velocity 16 m/sec). In *B*, absence of effect with stimulation of the d.p. nerve at 50 *T*. In *C* and *D*, inhibitory responses elicited by stimulation of the g.s. nerve at 10 and 20 times α-threshold (both stimulations below the threshold for antidromic activation of the cell). In *E*, inhibition caused by stimulation of the p.b.s.t. nerve at 50 *T*. Preparation with dorsal roots L_5–S_1 cut. *F–J*. In *F*, antidromic identification (arrow at stimulus shock), prior to the penetration of the cell, by stimulation of the g.s. nerve at 20 times α-threshold (latency 6·2 msec; conduction velocity 29 m/sec). In *G–J*, responses to stimulations of the ventral root S_1 (v.r.S_1). Upper traces: cell recordings; lower traces: ventral root volleys; e.f.: extracellular field. In *G*, no effect can be seen at 1·0 times α-threshold (α-field potential just noticeable; see also ventral root recording). In *H*, an i.p.s.p. appeared with stimulation of the ventral root S_1 at 1·4 times α-threshold. In *I*, the i.p.s.p. increased only slightly when the stimulation strength was raised to 2·0 times α-threshold, but with stronger stimulation (5 *T*, in *J*) the i.p.s.p. was greatly enhanced. The intracellular recording was interrupted before an exact figure of the membrane potential could be obtained. Preparation with intact dorsal roots. In all records superimposed responses to single-shock stimulation (*B–E*: thirty sweeps; *A* and *F–J*: three to six sweeps). Horizontal calibrations: bars in *A* and in *F–J* = 2 msec; bars in *B–E* = 10 msec. Vertical calibrations: bars in *A* and *F* (applicable to all recordings) = 1 mV.

both large and small ventral root axons. Examples of these two possibilities are demonstrated in Fig. 3. The g.s. γ-cell to the left (*A–E*) was not tested autogenetically below 10 times α-threshold. However, at this stimulus strength a weak inhibitory effect was observed (Fig. 3*C*). Nevertheless, an autogenetic high-threshold effect was

clearly demonstrated by the marked increase in inhibition caused by stimulation at 20 times threshold (note that this still was below the threshold for antidromic activation of the axon). This particular cell, recorded in a preparation with cut dorsal roots, was also inhibited by antidromic activation of axons in the p.b.s.t. nerve (Fig. 3*E*) but not in the d.p. nerve (Fig. 3*B*).

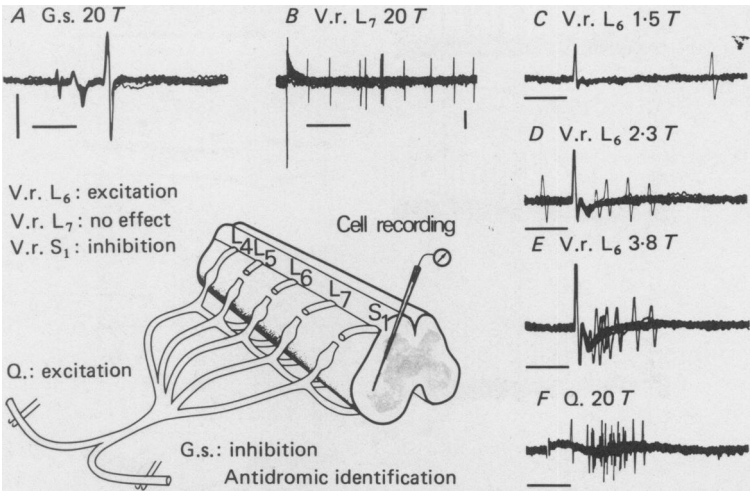

Fig. 4. Excitation of a dynamic g.s. γ-motoneurone by stimulation of the quadriceps nerve (Q.) and the ventral root L_6 (v.r.L_6). The cell was identified by antidromic activation of the g.s. nerve, stimulated at 20 times α-threshold (*A*, latency 4·3 msec; condition velocity 31 m/sec). In *B*, absence of effect with stimulation of the ventral root L_7. In *C–E*, responses to stimulation of the ventral root L_6. In *C*, no effect with stimulation at 1·5 times α-threshold. In *D*, weak excitation at 2·3 *T*. In *E*, clear-cut excitation at 3·8 *T*. In *F*, excitation elicited by stimulation of the quadriceps nerve at 20 *T*. Preparation (see inset) with dorsal roots L_5–S_1 cut. In all records superimposed responses (ten sweeps), to single-shock stimulation. Horizontal calibrations: bars in *A*, *C*, *D* and *E* = 4 msec; bar in *B* = 20 msec; bar in *F* = 10 msec. Vertical calibrations: bar in *A* = 0·4 mV; bar in *B* (applicable to *B–F*) = 0·4 mV.

The g.s. γ-cell to the right in Fig. 3 (*F–J*) was recorded intracellularly. As the dorsal roots were intact in this preparation recurrent effects could only be tested by stimulation of ventral roots. The cell was antidromically identified before penetration (*F*). At α-threshold stimulation of the sacral ventral root the cell was not affected (*G*). On the other hand, at 1·4 times threshold a clear inhibitory effect was evoked, with a latency of 2·8 msec (*H*). A further increase in stimulation intensity to 2·0 times threshold seemed to add little to the inhibitory effect (*I* compared with *H*). However, at still higher strength, i.e. 5 times threshold (*J*), a very marked increase of the inhibitory effect occurred, furnishing another example of a high-threshold effect, in this case superimposed upon the low-threshold action.

Excitation from ventral root fibres. The cell illustrated in Fig. 4 was identified as a triceps γ-cell (Fig. 4*A*). It was classified as dynamic, and its axon passed in the S_1 ventral root. In this particular experiment dorsal roots were cut between L_5 and S_1 and even proximal hind-limb muscles could therefore be assumed to be deafferented. In the cell of Fig. 4 the occurrence of both low-threshold inhibition and high-threshold

excitation could be demonstrated. Although the cell exhibited a rather weak spontaneous activity, *ventral root-mediated inhibition* could nevertheless be ascertained in extracellular recordings by stimulation of the g.s. nerve. This effect already appeared at 1·5 times α-threshold for the nerve and was, therefore, a clear-cut low-threshold effect. A similar inhibitory action was evoked from the S_1 ventral root. On the other hand, stimulation of the L_7 ventral root did not exert any effect on the cell, not even at 20 times α-threshold (Fig. 4*B*). In contrast, when the ventral root L_6 was stimulated at 2·3 times threshold or more, a clear-cut *excitatory effect* was seen in the cell (Fig. 4*D* and *E*). As no response was elicited by stimulating the ventral root L_6 at 1·5 times threshold (Fig. 4*C*), this was a high-threshold rather than a low-threshold effect. The genuine character of the observed excitation was corroborated by the finding of a similar excitation, evoked from the quadriceps nerve (Fig. 4*F*).

Equivalent findings of recurrent excitatory effects were obtained in five different cells. In three cases they were only evoked at high threshold. In the remaining two cells only high-threshold stimulation was used and a possible contribution to the effect from low-threshold fibres could not be excluded. Two of the γ-cells in which these effects were observed were classified as dynamic, one as static; the remaining two cells were not classified.

DISCUSSION

This study provides new information on the types of excitatory and inhibitory responses that can be elicited by activity in ventral root fibres. Clear examples are described of both low-threshold and high-threshold effects on γ-motoneurones, that may be ascribed to activity in recurrent collaterals of α- and γ-motoneurones, respectively.

Usually the effects evoked in motoneurones by stimulation of ventral roots or of muscle nerves, in preparations with severed dorsal roots, are attributed to an action of motor-axon collaterals. This rests on the assumption that all the fibres activated in the ventral roots are efferent rather than afferent. Partly on this basis, the concept of the recurrent inhibitory pathway, with α-motoneurone collaterals projecting to Renshaw cells, which in turn inhibit α-motoneurones, Ia inhibitory interneurones and other Renshaw cells, has come to be widely accepted (for review see Lindström, 1973). However, recent evidence indicates the existence of a certain proportion of myelinated sensory axons in ventral roots (for review see Coggeshall, 1980). Such fibres seem to comprise about 4% of the whole population of afferent nerve fibres (Loeb, 1976). However nothing is known about the central connexions of these ventral root afferent fibres. For the time being we therefore accept the conventional interpretation of effects caused by excitation of ventral root fibres as being mediated by activity in motor axon collaterals. Low-threshold recurrent effects are therefore attributed to α-collateral activity, and high-threshold effects to γ-collateral activity.

Over and above the mere confirmation of the existence of recurrent effects in γ-motoneurones, the present study also furnished evidence for the occurrence of recurrent inhibition of γ-motoneurones mediated via recurrent collaterals from other γ-cells. Also, in a few cases activity in ventral root fibres was shown to elicit excitatory

effects in γ-cells. The majority of these findings are based on extracellular recordings from γ-motoneurones. Apart from one exception, precise estimates of the latencies of the evoked effects could not therefore be obtained. Hence it is at present not possible to decide whether the observed, presumably recurrent, inhibitory effects were mediated via the same type of disynaptic inhibitory pathway as is known for α-motoneurones, i.e. by Renshaw cells projecting onto α-motoneurones, or whether other types of interneurones and polysynaptic pathways were responsible. The only case for which a reliable estimate of the i.p.s.p. latency was available gave a figure of 2·8 msec. This is clearly longer than the latency values reported for recurrent inhibition of α-motoneurones (Eccles, Fatt & Koketsu, 1954) but it agrees reasonably well with the estimates of latency made by Ellaway (1971).

No attempts were made in the present experiments to obtain a quantitative estimate of the occurrence of recurrent inhibition in γ-motoneurones. Nevertheless, our impression during the experiments was that strong effects, comparable to those regularly seen in α-motoneurones, were rare. The method used for the detection and assessment of recurrent effects (superposition of single-shock responses) is not particularly sensitive. Weak inhibitory effects may therefore have been overlooked. Ellaway & Murphy (1981), using a more sensitive method to detect inhibitory responses, recently reported that 54 % of their sample of γ-motoneurones, compared with 91 % of the α-motoneurones, received recurrent inhibition. They also stated that reductions in the probability of firing as low as 20 % were seen in γ-cells, compared to the lowest value of 50 % in α-cells. Thus, recurrent inhibitory effects in γ-motoneurones often seem to be small enough to be detectable only with highly sensitive methods (Ellaway, 1971; Ellaway & Murphy, 1981).

Ellaway (1971) used DOPA (see Bergmans & Grillner, 1968, 1969) in order to differentiate between static and dynamic γ-motoneurones. He concluded that both functional classes could receive recurrent inhibition. This finding was supported in the present experiments in which, though, another method of γ-cell classification was used. However, our findings are at variance with those of Ellaway (1971) with respect to the origin of the observed inhibitory effects. Whereas Ellaway considered it unlikely that the inhibitions were ever caused by impulses in recurrent collaterals of γ-motoneurones, firm evidence has now been obtained for the occurrence of such *γ to γ recurrent inhibition*. It was even demonstrated that a particular γ-cell may lack recurrent inhibition at low threshold, i.e. from collaterals of α-cells, but that it may be strongly inhibited from γ-axon collaterals. The action of these may be restricted to γ-cells, since both Westbury (1980) and Ellaway & Murphy (1981) were unable to demonstrate such γ-collateral recurrent inhibition in α-motoneurones. Experiments indicating that a population of Renshaw cells was excited by antidromic activation of γ-axons were reported by Kato & Fukushima (1974). These cells may constitute a class acting only on γ-motoneurones.

Thus there seem to be differences between the recurrent control of α- and γ-motoneurones, which add to the arguments against the concept of stereotyped α–γ linkage (see Appelberg, Hulliger, Johansson & Sojka, 1981, 1982, 1983*a–c*; Johansson, 1981).

The observation of facilitatory effects on γ-motoneurones caused by activation of ventral roots or of muscle nerves, in preparations with cut dorsal roots, is puzzling

and not easily interpreted. It is well established that the phenomenon of recurrent facilitation in α-motoneurones (Wilson, 1959; Wilson, Diecke & Talbot, 1960; Wilson & Burgess, 1962*a*, *b*) is caused by Renshaw cells feeding inhibition to group Ia inhibitory interneurones, resulting in disinhibition of α-cells (Hultborn *et al.* 1971). Since group Ia inhibitory effects seem to be rare or absent in γ-motoneurones (Appelberg *et al.* 1983*a*), the findings of recurrent facilitatory actions must be interpreted in another way. Renshaw cells are known to inhibit each other (Ryall, 1970; Ryall & Piercey, 1970). Provided that Renshaw cells were spontaneously active in our preparations, disinhibition of γ-cells by recurrent inhibition of Renshaw cells might have been the cause of the observed recurrent facilitation.

The functional role of recurrent inhibition has been a matter of discussion for a long time. Among other things it has been proposed that it constitutes a mechanism for lateral inhibition in the motor nuclei (Haase, Cleveland & Ross, 1975), that it is a stabilizing negative feed-back system, or that it operates as a switch for the activity in agonistic and antagonistic muscles (Wand & Pompeiano, 1979). Hultborn, Lindström & Wigström (1979) drew attention to the fact that all these hypotheses were narrowly focused onto α-motoneurones and could hardly serve as general explanations of the functional significance of recurrent inhibition in the motor 'output stage'. In particular, these proposals failed to take into account the recently discovered recurrent effects on γ-cells (Ellaway, 1968, 1971; Noth, 1971).

Hultborn *et al.* (1979) introduced the hypothesis that the recurrent pathways, both to α- and γ-motoneurones, function as a 'variable gain regulator' for the total motor output. They argued that during 'weak and finely tuned' movements a low input–output gain for the motoneurone pool (including both α- and γ-cells) would be desirable. Conversely, gross movements that demand strength would be performed with less recurrent inhibition and thus with a higher motoneuronal gain.

As a sufficient and necessary basis for their general hypothesis on recurrent inhibition in the motor-output system, they forwarded the concept of an 'output stage' of the motor system, consisting of α- and γ-motoneurones and of Ia inhibitory interneurones, operating together as a functional unity. The concept presupposes that α- and γ- motoneurones are 'usually activated in parallel' and that 'recurrent inhibition controls in a parallel fashion all neurones participating in the output stage'.

However, recent findings of considerable differences between α- and γ-motoneurones with regard to reflexes from muscle, skin and joint inputs (Appelberg *et al.* 1981, 1982, 1983*a–c*) make it less likely that a parallelism in α- and γ-activity is a general rule. Therefore, if the recurrent inhibitory system acts as a gain regulator, it seems likely that the recurrent control of γ-motoneurones should, at least in part, be specific.

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