J. Physiol. (1987), **383**, *pp.* 301–326
With 6 text-figures
Printed in Great Britain

RECURRENT INHIBITORY CONNEXIONS AMONG NECK MOTONEURONES IN THE CAT

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(*Received 19 February 1986*)

SUMMARY

1. Intracellular recordings were made from motoneurones innervating neck muscles in the cat. Dorsal roots were cut and muscle nerves electrically stimulated to activate α motor axons.

2. Recurrent inhibitory post-synaptic potentials (i.p.s.p.s) evoked by antidromic volleys in homonymous or heteronymous nerves were found in the majority of motoneurones studied, including those to dorsal neck muscles (biventer cervicis, splenius and complexus) as well as to occipitoscapularis and levator scapulae ventralis.

3. Central latencies of the recurrent i.p.s.p.s indicate disynaptic transmission. Amplitudes ranged from 100 μV (criterion level) to 2·2 mV. Average amplitudes were less than 0·6 mV.

4. The recurrent i.p.s.p.s were distributed to non-synergistic as well as to synergistic motoneurones. Analysis of relative strength of recurrent inhibition indicates influence of proximity of motoneurone pools, functional relatedness of muscles, as well as other factors. Variation in intrinsic motoneuronal properties probably underlies positive correlations (independent of variation in resting potential) between recurrent i.p.s.p.s evoked from different sources in motoneurones of a single pool.

5. Recordings (mainly extracellular) were also made from interneurones (Renshaw cells), located in the C3 and C4 segments of the spinal cord, that were excited by antidromic volleys in muscle nerves. The response varied from a single action potential to a burst of up to nineteen action potentials. Central latencies to the first response indicate monosynaptic transmission. Many Renshaw cells were excited by antidromic volleys in several muscle nerves, though this was restricted to nerves of the same segmental level as the Renshaw cell. All the muscle nerves studied were effective in activating Renshaw cells.

6. The results indicate that in many ways the recurrent i.p.s.p.s and the responses of Renshaw cells recorded in the neck segments resemble those in the hind-limb segments. Thus, the basic organization of recurrent inhibition in the neck segments resembles that occurring elsewhere in the spinal cord. A difference is the tendency for recurrent i.p.s.p.s in neck motoneurones to be relatively small in amplitude and Renshaw cell responses to be less strong than those recorded in the hind-limb segments. It is suggested that this is related to the segmentation of neck muscles and their motoneurone pools.

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7. It is concluded that recurrent inhibition is a prominent feature of spinal organization governing neck muscles. It can therefore be expected to participate in control of head movements.

INTRODUCTION

Recurrent inhibition is a means of controlling motor output from the spinal cord. This mechanism has been studied extensively and is mediated by axon collaterals of motoneurones projecting to Renshaw cells, inhibitory interneurones which in turn project back to motoneurones, as well as to a few select types of interneurones (see Willis, 1971; Haase, Cleveland & Ross, 1975; Baldissera, Hultborn & Illert, 1981 for review and references). Recurrent inhibitory actions have been described in spinal cord segments controlling hind-limb (e.g. Renshaw, 1941; Eccles, Fatt & Koketsu, 1954; Wilson, Talbot & Diecke, 1960; Eccles, Eccles, Iggo & Ito, 1961*a*; Hultborn, Jankowska & Lindström, 1971*c*), forelimb (Thomas & Wilson, 1967), tail (Jankowska, Padel & Zarzecki, 1978), back (Jankowska & Odutola, 1980) and respiratory (Kirkwood, Sears & Westgaard, 1981; Hilaire, Khatib & Monteau, 1983; Lipski, Fyffe & Jodkowski, 1985) muscles. However, there are some differences in organization within the different regions of the spinal cord, which would suggest functional specialization of recurrent inhibition. For example, among hind-limb motoneurones, motoneurones innervating muscles acting on the toes receive little recurrent inhibition and produce little or none (H. Hultborn, R. Katz & R. Mackel, personal communication; also indicated in Eccles *et al.* 1961*a*, and anatomically in Cullheim & Kellerth, 1978*a*), while those motoneurones innervating muscles acting on more proximal joints receive and distribute more recurrent actions. It would therefore appear, as summarized by Baldissera *et al.* (1981), that recurrent inhibition is involved in control of muscles which position the limb rather than of muscles controlling movements of the digits. For muscles tightly coupled in antagonistic action, namely limb flexors and extensors that act on the same joint (Hultborn *et al.* 1971*c*) and, presumably, some ipsi- and contralateral muscles of the tail (Jankowska *et al.* 1978), the distribution of recurrent inhibition is correlated with that of reciprocal inhibition (see Discussion). Furthermore, recurrent inhibition is not ubiquitous. In the spinal cord, it appears to be lacking for perianal sphincters (Jankowska *et al.* 1978; Mackel, 1979) and very weak for the diaphragm (Hilaire *et al.* 1983; Lipski *et al.* 1985). Until recently (Kirkwood *et al.* 1981; Hilaire *et al.* 1983; Lipski *et al.* 1985) it was thought to be absent for respiratory muscles (thoracic: Sears, 1964; diaphragm: Gill & Kuno, 1963), although interneurones behaving like Renshaw cells were found (thoracic: Sears, 1964).

Similarly, there is conflicting evidence concerning the occurrence of recurrent inhibition in the neck segments of the spinal cord. In the initial study of it, recurrent inhibition appeared to be very rare for neck muscles acting on the head, while frequent for a neck muscle with primary action on the shoulder (trapzius: Rapoport, 1979). It was suggested that recurrent inhibition might be relatively unimportant in control of head *versus* control of limb movements. However, motor axons of neck muscles acting as head movers (as well as those of trapezius) were effective in activating putative Renshaw cells (Rapoport, 1979). Furthermore, motoneurones

innervating dorsal neck muscles, as well as those innervating trapezius, give off axon collaterals (at least one), with about forty to ninety swellings (probable presynaptic terminals) per axon collateral system (Keirstead, Rose & Vanner, 1982). The number of collaterals and swellings of neck motoneurones are comparable to those of a variety of hind-limb motoneurones (Cullheim & Kellerth, 1978*a*). Thus, the structural basis for recurrent inhibition (axon collaterals, Renshaw cells) is certainly present in the neck as in the hind-limb segments. Unless the axon collaterals and Renshaw-like cells of the neck segments serve some different function, there is no anatomical reason for recurrent inhibition to be much rarer in the neck than in the hind-limb segments.

To resolve this problem, reinvestigation of the occurrence of recurrent inhibition in the neck segments was necessary. In fact, there is a brief report by Jankowska & Odutola (1980), in their work on back motoneurones, that recurrent inhibition of neck motoneurones is more frequent than previously thought: there is no need to invoke a different function for the circuitry. The present work extends these observations, in documenting the occurrence of recurrent inhibition among neck motoneurones and in investigating features of its organization and distribution. Some of the results have been briefly described previously (Brink & Suzuki, 1985).

METHODS

Preparation

Experiments were performed on thirteen adult cats anaesthetized with chloralose (60–70 mg/kg I.V. initial dose with 10–20 mg/kg supplements given during the course of the experiments). The cats were paralysed with gallamine triethiodide (Flaxedil) and artificially respirated; ventilation was adjusted to keep end-tidal CO_2 near 4 %. Blood pressure was monitored from the femoral artery and maintained at or above 100 mmHg by infusion of dextran or aramine (metaraminol bitartrate) if necessary. Body temperature was kept at 37–38 °C by a servo-controlled heating lamp.

During surgery, the cats were anaesthetized with a halothane–nitrous oxide mixture, which was later withdrawn after administration of chloralose. As during all stages of surgery and experimentation, blood pressure, reflexes, and breathing were closely monitored during the transition from halothane to chloralose anaesthesia, to be certain that the cats remained deeply anaesthetized. Surgery involved tracheotomy, nerve dissection, and laminectomy to expose the second to fifth cervical segments of the spinal cord. Dorsal roots were cut from C2 to C5. That transection of dorsal roots was complete was confirmed at the end of the experiment by inspection through a dissecting microscope after widening the laminectomy.

Muscle nerves were dissected, cut, and mounted on bipolar platinum electrodes for stimulation. The nerves dissected were: the C3 and C4 segmental nerves to biventer cervicis (b.c.); C3 complexus; C3 and C4 splenius; occipitoscapularis (o.s., a C4 nerve); and C3 and C4 levator scapulae ventralis (l.s.v.). The innervation of l.s.v. is usually from C3 (Reighard & Jennings, 1963; Crouch, 1969): however, in a few cats it was from C4. Because some branches to complexus are very short, it was usually not possible to dissect the entire nerve. Thus, the full set of C3 complexus motor axons was not stimulated, and effects may be underrepresented. Complexus, b.c. and splenius are innervated by branches of the dorsal rami; o.s. and l.s.v. by branches of the ventral rami (Reighard & Jennings, 1963; Crouch, 1969). Complexus and b.c. are head extensors; splenius, a lateral rotator which, acting bilaterally, extends the head. The action attributed to l.s.v. and o.s. (also called levator scapularis dorsalis) is pulling the scapula forward. However, the patterns of origin and insertion of these muscles on head and scapula make action in moving the head also likely. Indeed, o.s. is activated with other neck muscles during vestibular stimulation (Baker, Goldberg, Wickland & Peterson, 1985) and during free head movements (Richmond, Loeb & Reesor, 1985). In a few experiments, the contralateral C3 b.c. and complexus nerves were dissected and mounted together for stimulation, and contralateral dorsal roots cut.

Recording

Intracellular recording from motoneurones and Renshaw cells, and extracellular recording from Renshaw cells, were done in the same experiments, using glass micro-electrodes filled with 1 M-potassium acetate. The electrodes had outer tip diameters of 1·5–2 μm, and resistances of 3–5 MΩ. For intracellular records, signals were amplified without filtering and displayed on an oscilloscope, as well as recorded on a chart recorder, to monitor resting potential and height of the action potential. The signals were also amplified at high gain and filtered (bandpass 0·3 or 3 Hz to 3–5 kHz) to record post-synaptic potentials. Varying the high-pass filter had little or no effect on the duration or sizes of the potentials recorded. These signals were displayed on the oscilloscope and photographed; they were also led to a PDP-11/44 computer (Digital Equipment Corp.) for averaging with on-line display and storage. Bin width was 0·05 ms. The number of trials contributing to an averaged record varied from about 10–120 for very small potentials, typically about 40–80. Extracellular field potentials were recorded after withdrawing the electrode from a motoneurone.

Extracellular responses from Renshaw cells were displayed on the oscilloscope after filtering (bandpass usually 100 Hz to 2–5 kHz) and amplification, and were photographed, usually as single sweeps.

Antidromic volleys were recorded after advancing the micro-electrode tip deep into the spinal cord, past the location of the best antidromic fields (by about 1 mm or more). At such locations, presumably in the white matter, the antidromic potentials recorded on stimulation of muscle nerves were small, growing smoothly with increasing stimulus strengths, and had the shortest latencies with respect to the onset of the stimulus artifact (Fig. 1*D*). Central latencies, and the stimulus strength needed to obtain maximal volleys, were determined from these potentials. Thresholds for the antidromic volleys or fields were monitored throughout the course of the experiment.

Stimulation

Nerves were stimulated with rectangular constant current pulses 0·1 ms in duration. Stimuli were delivered usually at rates of 2/s, sometimes at 0·5/s to collect single-sweep data from Renshaw cells. To detect recurrent inhibitory post-synaptic potentials (recurrent i.p.s.p.s), the stimuli used were supramaximal for the antidromic potentials recorded deep in the spinal cord. Typically, stimuli were about 3–5 times threshold (3–5 *T*) for the antidromic volley. Stronger stimuli were sometimes used, but were usually avoided to preclude any possibility of activating unmyelinated afferents that might run in the ventral roots (Coggeshall, Coulter & Willis, 1974). Although not systematically tested, data on the growth of recurrent i.p.s.p.s, or e.p.s.p.s in Renshaw cells (see Fig. 5), in addition to monitor of the growth of antidromic volleys, indicated that maximal effects were attained with stimuli of strength about 2 times threshold for the antidromic volley and less than 3 *T*. This is similar to effects from a variety of hind-limb nerves: mean stimulus strength for maximal antidromic volleys was 2·2 *T* for α-motor axons (Boyd & Davey, 1968).

Single stimuli were used to elicit the recurrent i.p.s.p.s or to excite Renshaw cells. That single maximal stimuli were appropriate to test for presence of effects was suggested by previous work on lumbosacral Renshaw cells (Eccles, Eccles, Iggo & Lundberg, 1961*b*). When tested, double stimuli were no more effective than single ones.

Data analysis

Central latencies and amplitudes of intracellular potentials were measured mainly from computer-averaged records after subtraction of or comparison with extracellular records; the values corresponded well with values measured from photographs of oscilloscope tracings. Peak amplitudes were measured with respect to a 1 mV calibration pulse. Central latencies were measured from the positive peak of the antidromic potential recorded as described previously and illustrated in Fig. 1*D*. Accuracy of the latencies was estimated to be to the nearest 0·1 ms.

In spatial facilitation tests, the test and conditioning stimuli were presented each alone, and then together. The tests were repeated to establish reliability. For spatial facilitation of Renshaw cell activation, a series of single-sweep records were obtained under each condition, and various parameters measured. For spatial facilitation of recurrent i.p.s.p.s, the response to the combined stimulation was compared to the computer sum of the responses to each alone.

Non-parametric statistical tests were used to compare strengths of actions and look for

correlations between effects. For the first, actions of different inputs to the same motoneurones were compared, to minimize influence of other factors such as resting potential or type of motoneurone (Friedman, Sypert, Munson & Fleshman, 1981), and the tests are therefore for matched pairs. Non-parametric statistics were used because some sample sizes were small, and to avoid the assumptions necessary for use of parametric statistics. The specific statistical tests used are listed in the Results. The criterion for non-significance was set at $P > 0{\cdot}05$; two-tailed in all tests where appropriate.

RESULTS

Recurrent inhibition in motoneurones

Data were obtained from 119 motoneurones located in the C3–C4 region of the spinal cord. The sample includes 17 unidentified motoneurones as well as those antidromically activated by stimulation of the muscle nerves listed in Table 1. The unidentified neurones were identified as motoneurones by the shapes of their action potentials and by their location in areas where large antidromic field potentials were recorded. The large majority of motoneurones had action potentials of 50 mV or more (mostly 50–60 mV, although up to 80 mV) or resting potentials of 40 mV or more at the time of recording; all data on amplitude of responses are from such motoneurones. Some of the results (on detecting presence of recurrent inhibition, e.g. Table 1) include positive results from a few motoneurones with poorer resting potentials.

Of the identified motoneurones, recurrent inhibitory potentials of 100 μV (criterion level) to 2·2 mV were evoked from some source in the majority of them: in 33/38 b.c., 6/6 complexus, 14/20 splenius, 22/25 o.s. and 6/12 l.s.v. Examples of recurrent inhibitory potentials are illustrated in Fig. 1.

Recurrent i.p.s.p.s were evoked by both homonymous and heteronymous nerves (see below). They were easily reversed by passage of hyperpolarizing current and enhanced by depolarizing current (Fig. 1*Cf*; Eccles *et al.* 1954; Burke, Fedina & Lundberg, 1971). In a number of cases, passage of depolarizing current was necessary for their detection. The recurrent i.p.s.p.s sometimes showed the ripples characteristic of those recorded in lumbar motoneurones (hind-limb: Eccles *et al.* 1954; back: Jankowska & Odutola, 1980). These are especially clear in Fig. 1*D* (and are also present in 1*A* and *B*). The frequency of the ripples corresponds to the frequency of firing of Renshaw cells (e.g. Fig. 5). The duration of the i.p.s.p.s was 20–30 ms at the longest; smaller i.p.s.p.s were of shorter duration. When studied, thresholds for evoking the recurrent i.p.s.p.s were low: at or near threshold for the antidromic volley; as already mentioned, maximal recurrent i.p.s.p.s could be evoked with stimuli of 2 *T*.

Latencies. Latencies (Fig. 2) of homonymous and heteronymous recurrent i.p.s.p.s ranged from 1·0 to 2·5 ms when the i.p.s.p.s were evoked from nerves of the same segmental level as the recorded motoneurone: smaller i.p.s.p.s had longer latencies. These latencies are compatible with disynaptic transmission (Eccles, Fatt & Landgren, 1956; Eccles, Eccles & Lundberg, 1957; Fetz, Jankowska, Johannisson & Lipski, 1979). Indeed, the distribution of latencies corresponds well to that obtained if 0·5 ms as an estimate for central delay (0·3–0·4 ms for synaptic delay (Jankowska & Roberts, 1972; Munson & Sypert, 1979) and 0·1–0·2 ms for minimum conduction time in Renshaw cell axons (Jankowska & Smith, 1973)) is added to the latencies for activation of Renshaw cells (see Fig. 6). Central latencies were slightly longer for

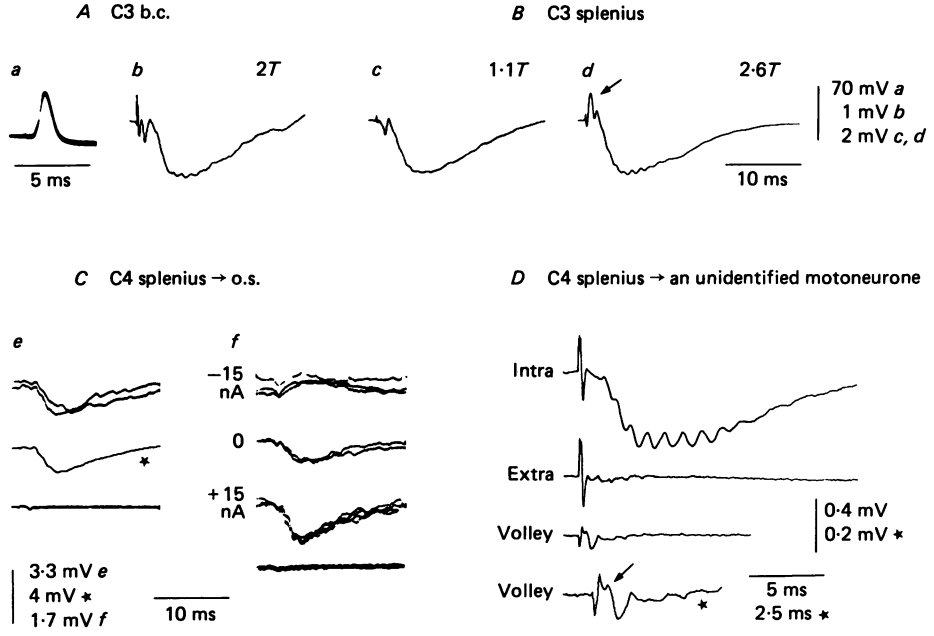

Fig. 1. Recurrent i.p.s.p.s. in neck motoneurones. *A* and *B*, homonymous recurrent i.p.s.p.s. in a biventer cervicis (b.c.; *A*) and a splenius (*B*) motoneurone. The recurrent i.p.s.p.s were detectable with stimuli below threshold for the antidromic spike (compare *Aa* and *b*); arrow in *Bd* indicates an M spike. *C* and *D*, heteronymous i.p.s.p.s produced in occipitoscapularis (o.s.; *C*) and an unidentified motoneurone (*D*) by stimulating splenius motor axons. In *Ce*, both the averaged record (middle trace) and records photographed from the oscilloscope (top trace) at the time of averaging are shown. Bottom trace is the extracellular field. In *Cf* (photographed from the oscilloscope), polarizing currents were applied. *D* illustrates the ripples characteristic of recurrent i.p.s.p.s. Top trace is the intracellular record, second is the extracellular, and bottom traces are records of the antidromic volley. Arrow indicates positive peak of the antidromic volley, for measurement of latencies. Further explanation given in text.

i.p.s.p.s evoked from nerves of adjacent segments (e.g. from C4 nerves in a C3 motoneurone), ranging from 1·2 to 2·6 ms. Probably both increased conduction time and (generally) weaker activation contribute to the lengthening.

Homonymous recurrent inhibition. Homonymous recurrent i.p.s.p.s were usually detected by using stimuli below threshold for antidromic activation of the motoneurone action potential (Fig. 1*A* and *B*), sometimes combined with depolarization of cells to enhance detection. In some cases, the mechanism generating the IS–SD spike inactivated, leaving only the M spike (e.g. Fig. 1*B*), so that stronger stimuli could be used to detect the i.p.s.p.s. In a few cases, the soma action potential was blocked by a short preceding hyperpolarizing pulse (Eccles *et al.* 1961*a*). As a systematic study of homonymous recurrent i.p.s.p.s. at maximal strength stimuli was not carried out, the frequencies and amplitudes of homonymous i.p.s.p.s. are certainly underrepresented. Even with these limitations, homonymous recurrent inhibition was detected in twenty-one b.c., eleven splenius, one complexus, eight o.s. but no l.s.v.

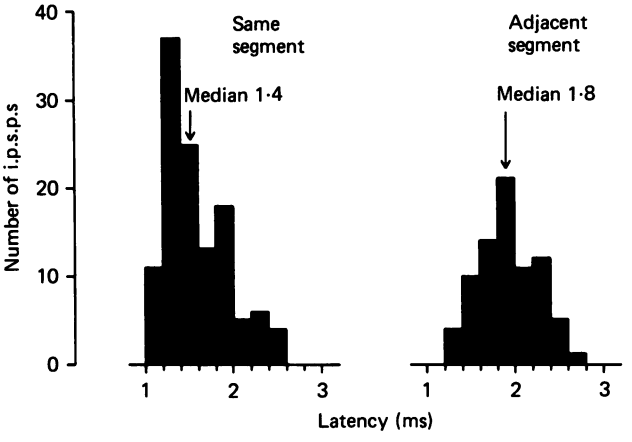

Fig. 2. Latencies of recurrent i.p.s.p.s. Left, i.p.s.p.s. evoked by antidromic volleys in nerves of the same segmental level as the recorded motoneurone. Right, i.p.s.p.s evoked from nerves of the adjacent segment (e.g. from C3 nerves in C4 motoneurones).

motoneurones. For b.c. and splenius, this represents about half the sample of neurones.

Since most homonymous recurrent i.p.s.p.s were evoked with stimuli below threshold for the antidromic action potential, it is evident that thresholds for the recurrent i.p.s.p.s were quite low; the potentials evoked with submaximal stimuli could be quite large, up to 1·9 mV. When stimuli of about 2 T or more could be used ($n = 7$), recurrent i.p.s.p.s recorded at resting potential ranged from 220 μV to 2·2 mV (for b.c., $n = 5$, median = 490 μV, mean = 560 μV, S.D. = 350 μV).

Many neck muscles are innervated by several segmental nerves; both C3 and C4 nerves to b.c. and splenius were studied in the same experiments. For motoneurones to such segmental muscles, the extrasegmental nerves to the same muscle as well as the homonymous nerves were quite effective in producing i.p.s.p.s: such recurrent i.p.s.p.s were seen in nineteen out of twenty-six b.c. and in six out of fifteen splenius motoneurones.

Distribution of recurrent inhibition across different species of motoneurones. Aside from the homonymous inhibition, recurrent i.p.s.p.s were often evoked by stimulation of other muscle nerves, of both segments. Examples of heteronymous recurrent i.p.s.p.s in o.s. and b.c. motoneurones are illustrated in Figs. 3 and 4. The results are summarized in Table 1, which emphasizes the functional distribution of recurrent inhibition. In this tabulation, a negative result means that no effects were produced by either the C3 or C4 nerve, or, in a few cases, by the nerve at the segment of the motoneurone tested (in effect, at C3). Positive results mean that effects were produced by one or the other or both nerves. The majority of b.c. and splenius motoneurones were recorded at C3. Since there was no evidence for statistically significant differences in inputs tabulated as frequency of occurrence for motoneurones located at C4 *versus* C3, data from motoneurones at both locations are included. Indeed, as seen in Fig. 4, effects in motoneurones located at the two levels could be quite similar.

From Table 1 it can be seen that, for the set of muscle nerves tested, l.s.v. is the

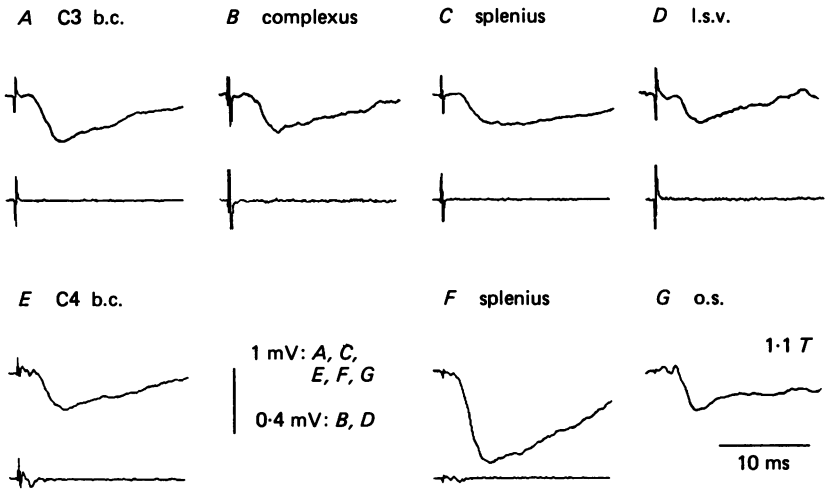

Fig. 3. Examples of recurrent i.p.s.p.s of different origin evoked in an occipitoscapularis (o.s.) motoneurone. Lower traces are extracellular records. C3 nerves were stimulated in *A–D*, and C4 nerves in *E–G*. Stimuli were supramaximal for the antidromic volleys, except in *G*, which shows the homonymous recurrent i.p.s.p. evoked with near-threshold stimulation. All records are averaged.

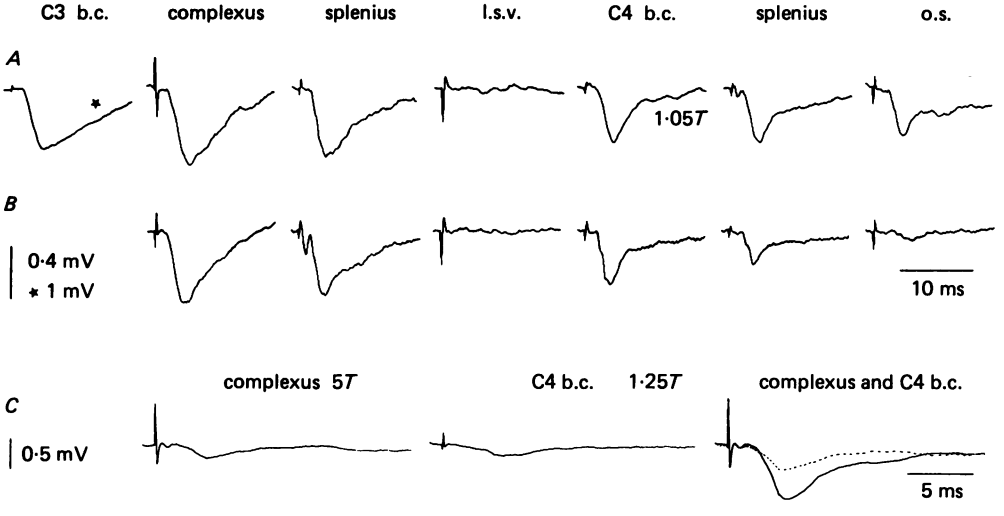

Fig. 4. Examples of recurrent i.p.s.p.s of different origin evoked in biventer cervicis (b.c.) motoneurones. Records in *A* are from a b.c. motoneurone located at C4 (homonymous recurrent i.p.s.p. is submaximal); in *B* and *C* are from two different C3 b.c. motoneurones. *C* illustrates spatial facilitation of recurrent inhibition occurring when two nerves are stimulated together; the dotted line represents the sum of the responses to each stimulus alone.

TABLE 1. Recurrent inhibitory connexions between different species of neck motoneurones. Data are summarized as frequency of occurrence of the recurrent i.p.s.p.s. Homonymous i.p.s.p.s are excluded as the stimuli used to evoke them were usually submaximal

	Nerve stimulated				
Motoneurones	b.c.	Complexus	Splenius	o.s.	l.s.v.
b.c.	*	29/36	22/32	5/20	0/16
Complexus	6/6	*	5/6	2/3	0/3
Splenius	8/16	3/14	*	2/8	1/9
o.s.	14/16	8/13	21/23	*	1/11
l.s.v.	0/8	0/4	6/11	0/6	*

least effective, rarely producing i.p.s.p.s. It is also the least affected, receiving recurrent i.p.s.p.s only from the splenius nerve. The next in effectiveness is o.s., which nevertheless does produce i.p.s.p.s in b.c., complexus, and splenius. In contrast, o.s. motoneurones frequently receive i.p.s.p.s from all three, b.c., complexus and splenius. The synergists b.c. and complexus (Anderson, 1977) frequently receive recurrent i.p.s.p.s from each other as well as from splenius; they appear to be less effective in producing recurrent inhibition in splenius. In general, splenius is the most effective in producing recurrent i.p.s.p.s: it appears to produce more than it receives.

In the few cases tried (three b.c., one complexus and one splenius motoneurone) there was no evidence for contralateral recurrent inhibition evoked from b.c. and complexus axons. While the data are few, they are in line with observations on back muscles innervated by the dorsal rami (Jankowska & Odutola, 1980) and in contrast to the crossed recurrent inhibition of tail motoneurones (Jankowska *et al.* 1978) and unidentified motoneurones (Jankowska & Odutola, 1980) of the upper lumbar region.

Several examples of spatial facilitation of effects from different nerves were seen (e.g. Fig. 4*C*), indicating convergence on the intermediary interneurones (see below), as described for Renshaw cells in the hind-limb segments of the spinal cord (Eccles *et al.* 1954, 1961*b*; Ryall, 1970, 1981).

Comparative strengths of recurrent inhibitory connexions. To examine functional distribution, the tabulation of Table 1 deliberately sacrificed information on spatial distribution of recurrent inhibition, and on amplitudes of effects. In this section, effects produced by and in spatially different motoneurone pools are treated separately, to examine relative strengths of actions. Since the sample of complexus motoneurones was small, and recurrent inhibitory connexions to l.s.v. rare and those to splenius rather weak, the most detailed analysis was carried out for C3 b.c. and for o.s. motoneurones.

In previous studies, average amplitude of recurrent i.p.s.p.s has been used as an indicator of relative strength (Eccles *et al.* 1954, 1961*a*; Hultborn *et al.* 1971*c*). Average amplitudes of (non-zero) i.p.s.p.s in C3 b.c. and o.s. motoneurones are listed in Table 2, together with frequency of occurrence of the i.p.s.p.s. (If negative responses (zero amplitudes) are included in the averages, values for C3 b.c. motoneurones are reduced by about 0·05 mV; for o.s. motoneurones, by about 0·1 mV for

TABLE 2. Relative strength of heteronymous recurrent i.p.s.p.s produced in biventer cervicis (b.c.) or occipitoscapularis (o.s.) motoneurones. Different means of measuring strength of i.p.s.p.s were used, as detailed in the text. The matched samples are a subset of motoneurones, ones for which data from all nerves were obtained while resting potential remained stable. The full samples include motoneurones with action potentials greater than 50 mV

			Nerve stimulated				
Motoneurones	Sample	Measure	C3 b.c.	C3 Complexus	C3 Splenius	C4 b.c.	C4 Splenius
C3 b.c.	Matched ($n = 9$)	Mean rank	—	1·9	2·8	1·9	3·4
	Full	Frequency of occurrence	—	19/23	16/22	10/15	7/14
		Mean amplitude ±S.D. (μV)	—	300±180	250±140	200±80	160±50
		(n)		(18)	(15)	(10)	(6)
o.s.	Matched ($n = 10$)	Mean rank	2·3	4·5	2·9	4·0	1·3
	Full	Frequency of occurrence	10/13	7/12	12/14	7/13	15/18
		Mean amplitude ±S.D. (μV)	290±230	210±80	290±130	280±200	560±520
		(n)	(7)	(5)	(7)	(5)	(11)

C3 nerves and 0·2 mV for C4 nerves.) The results would indicate that the recurrent i.p.s.p.s are generally rather small (less than 0·6 mV). Amplitudes of other recurrent inhibitory connexions not listed in Table 2 were similar. As reflected by large standard deviations, and as indicated in other studies (Friedman *et al.* 1981), recurrent i.p.s.p.s evoked in motoneurones of a single pool were often quite variable in size (on sources of variability, see the next section of the Results and also the Discussion). This large variability diminishes the usefulness of average amplitude as a measure; however, it remains a means of roughly comparing the recurrent i.p.s.p.s produced in neck motoneurones with those recorded in motoneurones of other regions of the spinal cord.

In order to control for the variability *between* motoneurones in examining relative strength of recurrent inhibition among neck motoneurones, comparisons were restricted to effects produced by different inputs to the *same* motoneurones. The statistics chosen make use of relative (larger or smaller) rather than absolute size of effects, so that data obtained from polarized cells could be included. Only i.p.s.p.s obtained while resting potentials were stable were included in the analysis. In the most extensive examinations, for ten o.s. and nine b.c. motoneurones, amplitudes of effects from all heteronymous nerves could be compared. For C3 b.c., since l.s.v. was not effective and o.s. rarely so, comparison was restricted to C3 complexus, C3 splenius, C4 b.c., and C4 splenius, with effects ranked from 1 (most effective) to 4. For o.s. motoneurones, comparison was made for all but o.s. and l.s.v. (ranks 1–5).

The means of the ranks obtained in this way are listed in Table 2. Were there consistent differences between inputs, the mean ranks would be 1, 2, etc. This is nearly so for o.s. motoneurones. If there were no differences between inputs, the mean ranks of all inputs should be near the middle of the range of possible ranks. This is approximately the case for C3 b.c. motoneurones. Friedman two-way analyses of variance (Siegel, 1956) indicate the presence of significant differences between inputs for both C3 b.c. ($\chi_r^2 = 10{\cdot}26$, d.f. $= 3$, $P < 0{\cdot}02$) and o.s. ($\chi_r^2 = 19{\cdot}8$, d.f. $= 4$, $P < 0{\cdot}001$) motoneurones. For C3 b.c. motoneurones, this probably reflects the tendency of C4 splenius to be the least effective in producing recurrent i.p.s.p.s (as suggested from the frequency of occurrence and average amplitude of this connexion). When only C3 complexus, C3 splenius, and C4 b.c. recurrent i.p.s.p.s are compared, there is no significant effect (although significance persists when other combinations of three inputs including C4 splenius are compared). This tendency is confirmed by sign tests for matched pairs (Siegel, 1956) conducted on larger samples of the motoneurones: only C4 splenius is significantly different from (less effective than) C3 complexus (used as a standard for comparison) in producing recurrent i.p.s.p.s ($P = 0{\cdot}008$, $n = 12$). In fact, the most effective nerve was the homonymous C3 b.c., even though the stimuli used were usually submaximal: effects were significantly greater than those produced by all other nerves ($P = 0{\cdot}01$–$0{\cdot}03$, $n = 6$–12) except C3 complexus ($n = 12$). (Correspondingly, the average amplitude of homonymous recurrent i.p.s.p.s, though usually submaximal, was largest: $0{\cdot}4 \pm 0{\cdot}3$ mV, $n = 12$). Thus, a general interpretation of the strength of recurrent inhibition in C3 b.c. motoneurones would be the homonymous strongest; followed by a synergist (complexus) and non-synergist (splenius) of the same segment and an extrasegmental nerve to the homonymous muscle, all of roughly comparable strength; then the extrasegmental (C4) splenius; then o.s.; and lastly l.s.v. (both local and extrasegmental).

For o.s. motoneurones, the ranking procedure summarized in Table 2 clearly indicates differential effectiveness of heteronymous motor axons in producing recurrent inhibition. C4 splenius is the most effective, with a mean rank of 1·3 (nearly 1, and with the largest average amplitude), followed by C3 b.c., C3 splenius, C4 b.c. and C3 complexus. Sign tests for matched pairs would suggest grouping C3 b.c. and C3 splenius together, and C4 b.c. with C3 complexus. Specifically, effects from C4 splenius are significantly different from (greater than) effects from C3 splenius ($P \leqslant 0{\cdot}004$, $n = 17$) which is not different from C3 b.c. ($n = 11$); C3 b.c. is different from C4 b.c. ($P = 0{\cdot}04$, $n = 11$), which is not different from C3 complexus ($n = 12$). At the end of this list would come the ineffective l.s.v. The sample of homonymous o.s. recurrent i.p.s.p.s was too small to permit comparison.

The effect of distance between giving and receiving motoneurone pools on the strength of recurrent inhibition can be seen in Table 2 and further tested by supplementary sign tests for matched pairs, by comparing effects produced by the C3 and C4 nerves to a single muscle, on motoneurones located at C3 and C4. The sign tests performed for o.s. motoneurones (which are in C4) showed that the C4 splenius actions were significantly greater than those from C3 splenius; evidence for spatial influence. In C3 b.c. motoneurones, the C4 splenius tended to be less effective than the C3 splenius, but this did not reach significance ($n = 14$). Other evidence for a spatial influence came from l.s.v. motoneurones: six out of eleven received recurrent i.p.s.p.s from the splenius of the same segment, while only one out of six received i.p.s.p.s from the splenius nerve of the adjacent segment.

The remaining obvious test was to compare effects of C3 *versus* C4 b.c. stimulation. It was anticipated that the nearby b.c. nerve would be more effective in producing recurrent inhibition in o.s. motoneurones; in fact, contrary to expectation, the more distant C3 nerve was the more effective. Thus, some other factor(s) are influencing the strength of recurrent inhibition. A possibility is the number of α motor axons activated: b.c, with two large branches innervating it at C3, has many more at C3 than at C4 (Richmond, Anstee, Sherwin & Abrahams, 1976).

Correlations between inputs. It has been mentioned that amplitudes of recurrent i.p.s.p.s produced by a given nerve in a given motoneurone pool could show considerable variation. Additionally, the response pattern ranged from no recurrent inhibition from any heteronymous nerve (even with depolarization) to responses from one to up to all six heteronymous nerves. Among other possibilities, such variation could come about if the motoneurones of a pool were functionally subdivided, with some inputs effective in a certain subgroup of the motoneurones and other inputs in a different group. This could be expected to result in negative, or no, correlations between some pairs of inputs. To look for possible patternings of recurrent inhibition, tests for correlation were carried out for the inputs to C3 b.c. and to o.s. motoneurones. The test used was the Kendall rank correlation (Siegel, 1956), for which a partial correlation, factoring out possible co-variation due to a third influence, may be calculated. The results of the correlation tests are listed in Table 3.

To avoid redundancy, all other inputs to C3 b.c. were compared to C3 complexus effects; for o.s., all were compared to C4 splenius effects. In fact, positive correlations were seen in all cases. For C3 b.c. motoneurones, when recurrent inhibition from C3 complexus was large, so was that from C3 splenius, C4 b.c., and C4 splenius.

TABLE 3. Correlations between recurrent i.p.s.p.s of different origin. The i.p.s.p.s examined were evoked either in biventer cervicis (b.c.) or in occipitoscapularis (o.s.) motoneurones. A refers to the source of the heteronymous i.p.s.p. used as a standard. X designates the various other sources to be tested for correlation with the standard. *n* indicates the number of motoneurones contributing to the correlation (here limited to motoneurones for which i.p.s.p.s at resting potential, and resting potentials (rather than action potentials), were measured). The Kendall rank correlation coefficients (τ) are listed in the next three columns: first, for correlation between amplitudes of recurrent i.p.s.p.s (standard with test), then for correlation between amplitude of recurrent i.p.s.p.s and resting potential (first for the test (X) i.p.s.p.s. and then for the standard, (A) i.p.s.p.). The final column lists the Kendall partial rank correlation coefficients (correlation between amplitudes of recurrent i.p.s.p.s of different origin when effect of resting potential is removed)

Motoneurones	A	X	*n*	τ_{AX}	$\tau_{X\cdot r.p.}$	$\tau_{A\cdot r.p.}$	$\tau_{AX\cdot r.p.}$
C3 b.c.	Complexus	C3 splenius	18	0·39*	−0·25 n.s.	−0·06 n.s.	0·39
		C4 b.c.	13	0·47***	−0·32*	−0·26 n.s.	0·47
		C4 splenius	11	0·67***	−0·64***	−0·24 n.s.	0·69
o.s.	C4 splenius	C3 b.c.	8	0·89**	−0·38 n.s.	−0·23 n.s.	0·89
		Complexus	8	0·70*	−0·08 n.s.	−0·11 n.s.	0·70
		C3 splenius	10	0·81***	−0·22 n.s.	−0·40 n.s.	0·90
		C4 b.c.	11	0·84***	−0·42 n.s.	−0·38 n.s.	0·81

* $P < 0·05$, two tailed. ** $P < 0·01$. *** $P < 0·005$. n.s. $P > 0·05$. r.p. resting potential.

Relatedly, there was a significant positive correlation ($r_s = 0{\cdot}72$, $n = 13$, $P < 0{\cdot}01$; Sperman rank correlation (Siegel, 1956; Hays, 1981)) between amplitude of recurrent inhibition from C3 complexus and number of other heteronymous effects (this could range from 0 to 4, including o.s.). Similarly, for o.s. motoneurones, there were significant positive correlations between the amplitude of the recurrent i.p.s.p.s from C4 splenius on the one hand, and those from C3 b.c., C3 complexus, C3 splenius, C4 b.c., or with number of heteronymous inputs (Spearman rank correlation = 0·94, $n = 11$, $P < 0{\cdot}001$) on the other. The positive correlations held whether amplitudes were measured with the cells at resting potential or polarized. The positive correlations between inputs were not simply secondary to variations in resting potentials: for the most part, there was no significant correlation between resting potential (range 40–70 mV, mostly 40–60 mV) and the amplitude of the recurrent i.p.s.p.s. In a few cases, there was a significant negative correlation between the two (see Table 3). However, in all cases, calculating the partial correlations, where co-variation in recurrent i.p.s.p.s associated with variation in resting potential is controlled for, gave coefficients of the same value (see Table 3).

These positive correlations would suggest that the target motoneurones are not subdivided by inputs, but have a fairly homogeneous set of potential inputs. The amount of recurrent inhibition received, in terms of amplitude and number of inputs, would seem to shift up or down across individual motoneurones. Such a shifting could be caused by factors extraneous to the motoneurones, such as variations in the preparations, leading to excitability differences. However, since wide variations in recurrent inhibitory amplitudes could occur in the same preparation, other factors contribute. Very probably, these include factors intrinsic to the motoneurones. Variability in the amount of recurrent inhibition, in association with the type of motoneurone, has long been noticed (Granit, Pascoe & Steg, 1957; Kuno, 1959; Eccles *et al.* 1961*a*). More recently the amount of recurrent inhibition has been shown to be correlated with membrane properties, positively with input resistance, negatively with rheobase, as well as with motoneuronal type *per se* (slow > fatigue-resistant > fast fatigable) independent of co-variation of membrane properties (Friedman *et al.* 1981). Correlation of recurrent inhibition with membrane properties would lead to the positive correlations seen between recurrent i.p.s.p.s: thus, the positive correlations may well reflect variations between motoneurones in the receiving pool.

Renshaw cells

Nature of the response. As has been reported previously (Rapoport, 1979; Keirstead *et al.* 1982) neurones (Renshaw cells) activated by stimulation of the motor axons to neck muscles were commonly encountered. All the muscle nerves dissected were effective in exciting Renshaw cells. The responses of twenty-seven Renshaw cells were studied during the course of experiments on recurrent inhibition in motoneurones; systematic tracking for Renshaw cells was not carried out and the sampling cannot be considered unbiased. Neurones chosen for study had distinctive, identifiable shapes. None was spontaneously active. Thresholds for Renshaw cell activation were near threshold for the incoming antidromic volleys: in more than 50 % of the cases (twenty-five out of forty) stimuli of 1·1 *T* or less were effective, and in 90 %, stimuli

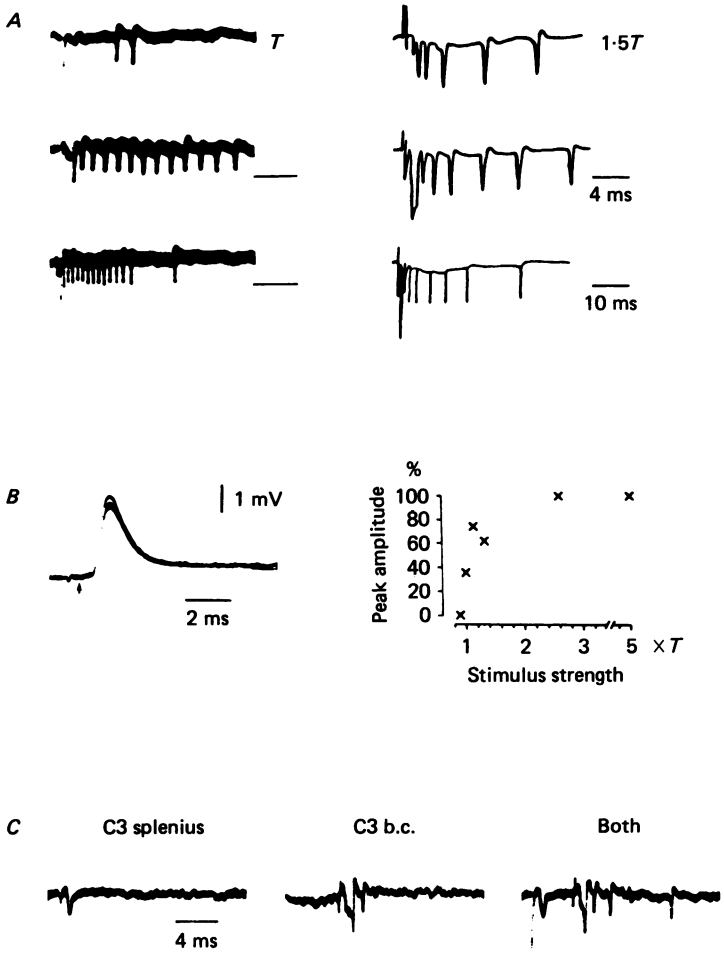

Fig. 5. Responses of cervical Renshaw cells to stimulation of motor axons. *A* and *C* are extracellular records, with negativity down. In *A*, records on the left were photographed from the oscilloscope; on the right, they were retraced from photographs. Record in *B* is intracellular, photographed from the oscilloscope. Stimuli were supramaximal for the antidromic volleys unless otherwise indicated. *A*, responses of two C3 Renshaw cells to stimulation of C3 b.c. axons at submaximal (top traces) and supramaximal strengths. *B*, e.p.s.p. in a C4 Renshaw cell. Arrow indicates time of antidromic volley. On the right, growth of the e.p.s.p. with increase in stimulus strength. *C*, convergence onto a C3 Renshaw cell. In this example, stimulation of C3 b.c. evokes two action potentials, the first on the antidromic field. Stimulation of C3 splenius alone evokes only an antidromic field and no response of the Renshaw cell. Stimulating both leads to a facilitated response. Further explanation in text.

of 1·5 *T* or less were effective. Examples of responses of Renshaw cells are shown in Fig. 5. In Fig. 5*A*, left, submaximal stimulation of the C3 b.c. nerve evokes two action potentials at a long and rather variable latency; stimulation at a strength maximal for the antidromic volley leads to a high-frequency burst of responses, and shortening of latency. In Fig. 5*A*, right, again increasing stimulus strength up to maximal leads

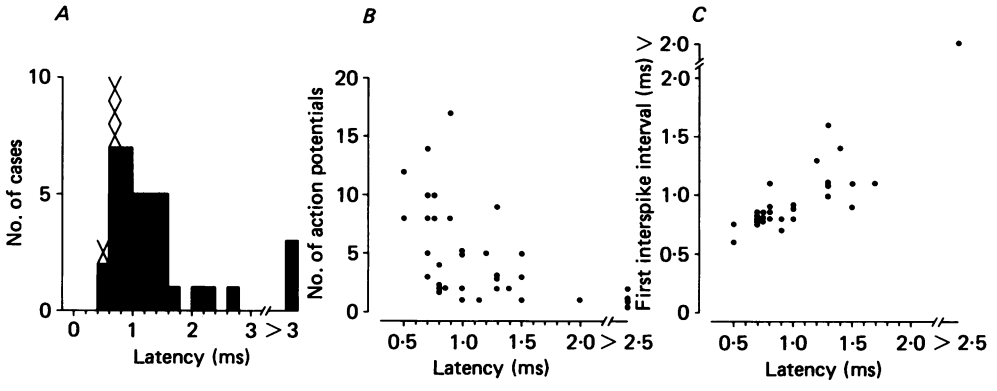

Fig. 6. Latency of Renshaw cell responses to stimulation of motor axons. In *A*, crosses represent latencies of intracellular e.p.s.p.s; remainder are of extracellular action potentials. *B* illustrates the negative correlation of latency of the Renshaw cell response with (median) number of action potentials in the response. *C* shows the positive correlation of latency with the interval between the first two action potentials of the response. Further explanation in text.

to increased number of responses and shorter intervals between spikes. This type of response pattern is characteristic of Renshaw cells described elsewhere (Renshaw, 1946; Eccles *et al.* 1954, 1961*a*; Ryall, 1970, 1981; Jankowska & Odutola, 1980). With maximal-strength stimuli, the response was usually a burst of two to up to nineteen action potentials, with a very short interval between the first two responses, the burst lasting up to 64 ms after the stimulus. More typically, median latency to the last response ranged from 2 (for responses of two action potentials) to 52 ms, with an over-all median of 20 ms.

Intracellular recordings were made for three of the twenty-seven Renshaw cells studied. An example of an intracellularly recorded e.p.s.p. is given in Fig. 5*B*. The cell rapidly deteriorated after penetration: resting potential declined from 60 to 30 mV at the time of the records. The e.p.s.p. shows the characteristic shape described for Renshaw cells in the lumbar spinal cord (Eccles *et al.* 1961*b*; Jankowska & Odutola, 1980): a large initial component which declines rapidly to a more slowly decaying depolarization. The e.p.s.p. appeared with stimuli at threshold strength for the antidromic volley and increased rapidly to maximum amplitude with increasing strength of stimulation.

Latencies (Fig. 6) to the first extracellularly recorded response ranged from 0·5 to 12·4 ms. About half (sixteen out of thirty-eight) of the extracellularly recorded responses had latencies of 0·9 ms or less, within the monosynaptic range for intracellularly recorded responses. (Most monosynaptic e.p.s.p.s in motoneurones and interneurones have central latencies of 0·5–0·9 ms, whereas most disynaptic effects have latencies greater than 1 ms (Eccles *et al.* 1956, 1957; Fetz *et al.* 1979; Jankowska, Johannisson & Lipski, 1981); 0·9 ms is taken as a dividing point between mono- and disynaptic effects since it does not allow enough time for two synaptic delays plus central conduction.) That most longer-latency extracellular responses also reflect monosynaptic activation is suggested from intracellular recordings. One Renshaw cell

was first studied extracellularly and responded with central latencies of 1·3 and 1·4 ms, respectively, on stimulation of (C4) splenius and b.c. motor axons. Intracellular recordings revealed e.p.s.p.s with central latencies of 0·7 ms (monosynaptic) in both cases (latencies of e.p.s.p.s: crosses in Fig. 6). The median time-to-peak of the e.p.s.p.s was 0·8 ms (range 0·65–1·0, $n = 4$). It would seem that the action potentials in this cell were initiated only when the cell was nearly maximally depolarized by the underlying e.p.s.p.s. This would be expected in cases of weak activation. By this reasoning, allowing central latencies for monosynaptic e.p.s.p.s of up to 0·9 ms, and 0·8 ms to e.p.s.p. peak, latencies for monosynaptic activation of an action potential might range up to 1·7 ms, representing nearly the full sample of latencies encountered (see also Eccles *et al.* 1956).

That latency of action potentials reflects strength of activation is also suggested by the previously mentioned tendency for latency to shorten with increasing stimulus strength, which is paralleled by shortening interspike intervals and increasing numbers of action potentials. The relationship between these three indicators of excitation was examined for responses to supramaximal stimuli (Fig. 6). There was a significant negative correlation between latency to the first response and number of spikes in the burst (Fig. 6*B*, $r_s = -0{\cdot}65$, $n = 37$, $P < 0{\cdot}002$, Spearman rank correlation), and a significant positive correlation between latency to the first response and interval between the first two responses, for bursts of two or more spikes (Fig. 6*C*, $r_s = 0{\cdot}75$, $n = 29$, $P < 0{\cdot}002$). Of these three indicators the number of spikes may be the least reliable, or, at least, it is somewhat disassociated from the other two. Specifically, while none of the responses consisting of only a single action potential had the shortest latencies (less than 1 ms), some responses consisting of two action potentials did have such short latencies, and short (less than 1 ms) interspike intervals. It would seem that some additional factor(s) contributes to determining the number of spikes. One possibility is Renshaw cell inhibition of other Renshaw cells (Ryall, 1970, 1981; Ryall, Piercey & Polosa, 1971; suggested by Rapoport, 1979). It may also be pointed out that while latency and first interspike interval will depend largely or entirely on the fast component of the underlying e.p.s.p., the number of spikes will depend also on the slow component and anything that might influence it independently.

Convergence. For twenty Renshaw cells, the responses to all or nearly all of the dissected nerves were studied. Table 4 summarizes the results, which represent the median number of responses evoked by a single stimulus at strength to activate maximally the α motor axons of each muscle nerve. Since sampling of Renshaw cells was not systematic (not unbiased), Table 4 does not indicate the relative frequency of convergence *versus* non-convergence. This would in any event be precluded by the limited variety of nerves tested at any one segmental level.

In Table 4 the Renshaw cells are arranged by location, from rostral to caudal through the C3 and C4 segments. It is immediately apparent that the input to Renshaw cells is spatially restricted. Those in C3 are activated by nerves of the C3 segment and not those of C4; Renshaw cells located in C4 are activated by C4 and not C3 nerves. The only exception was one cell located in caudal C3, bordering on C4, in an area dominated by antidromic fields from C4 nerves. This Renshaw cell was excited by the C3 b.c. nerve as well as all the C4 nerves. Convergence onto Renshaw

TABLE 4. Convergence on to cervical Renshaw cells. The columns list the (median) number of action potentials discharged by each Renshaw cell to single stimuli supramaximal for α-motor axons of the nerves indicated. The second column on the left indicates rostrocaudal location (segmental level) of each Renshaw cell

Renshaw cell		Nerve stimulated							
		C3				C4			
No.	Level	b.c.	complexus	splenius	l.s.v.	b.c.	splenius	o.s.	l.s.v.
1	C3 r.	.	.	3	.	.	.	.	
2	C3 r.	2	.	1	.	.	.	.	
3	C3 r.m.	2	1	*					
4	C3 m.	.	.	2	.		.	.	
5	C3 m.	12	.	.		.	.	.	.
6	C3 m.	3	5	1		.	.	.	.
7	C3 m.	8	.	*	.	.	.	.	
8	C3 m.	14	.	.	.	.	.	.	
9	C3 m.	8	3	3		.	.	.	.
10	C3 m.	.	.	2	.	.	.	.	
11	C3 m.	5	.	10	1	.	.	.	
12	C3 m.c.	8	.	1	.	.	.	.	
13	C3 m.c.	2	.	4	.	.	.	.	
14	C3 c.–C4 r.	1	.	.	.	8	1	5	
15	C4 r.	.	.	.	.	.	5	.	
16	C4 r.m.	.	.	.	.	1	.	1	
17	C4 m.		.	.	.	.	1	5	
18	C4 m.		.	.	.	8	.	3	
19	C4 m.	.	.	.	.	.	.	17	
20	C4 m.	.	.	.	.	2	2	1	

Dots, no response. * Convergence seen as spatial facilitation only. r., rostral. m., mid. c., caudal. Blank indicates not tested at supramaximal strength.

cells in the border areas between segments would account for the spatial facilitation of recurrent inhibition evoked from C3 and C4 nerves (Fig. 4*C*).

Table 4 illustrates that, for the nerves tested, a variety of patterns of convergence occur. Thus, of Renshaw cells at C4, some were activated by all three of the C4 nerves, b.c., splenius, and o.s., and others by different combinations of two inputs. Similarly, different combinations of two or three inputs were seen for neurones at C3. It would seem from Table 4 that neurones activated by the C3 dorsal ramus nerves tend not to receive convergent input from l.s.v., which would parallel the relative lack of recurrent inhibition from l.s.v. in motoneurones with axons in the dorsal rami. However, convergence from l.s.v. did occur in one example listed and in another (not listed because not extensively studied), in both cases being rather weak (in terms of number of spikes).

In some examples, convergence was seen only as spatial facilitation of the Renshaw activation by another input. Spatial facilitation was demonstrated in four cases. It was apparent as increased frequency of response (to 90 or 100 % of trials) when the test stimulus (at threshold or just below, 0–50 % responding) was preceded by the conditioning stimulus (by itself ineffective). For example, a test stimulus of C3 b.c. evoked a single action potential in three out of fourteen trials. When a conditioning stimulus preceding the test stimulus by 3 ms was delivered to C3 splenius (itself ineffective in evoking spikes) the neurone responded in sixteen out of eighteen trials with one to three spikes (median two). In other cases, the facilitation wrought by the conditioning stimulus was manifested as an increase in the number of action potentials to a suprathreshold test stimulus, together with a shortening of the latency to the first response by 0·2–0·4 ms. An example is illustrated in Fig. 5*C*. No evidence for inhibition of Renshaw cells was found.

In this sample of Renshaw cells, when a cell was activated by two or more nerves it was generally dominated by one of the inputs, showing a greater number of action potentials and having a short (less than 1 ms) latency and short interspike intervals. For example, for cell no. 9 of Table 4, b.c. evokes the strongest response by all three criteria: number of spikes, latency (0·9 ms *vs.* 1·3–1·4 ms), and initial interspike interval (0·8 *versus* 1·1–1·2 ms). In a few cases (e.g. Nos. 16 and 20 of Table 4), the responses to all nerves had longer (greater than 1 ms) latencies and longer (greater than 1 ms) interspike intervals; presumably the nerves stimulated represent only the fringe of nerves exciting those Renshaw cells, with the predominant nerve or nerves not tested.

DISCUSSION

The results indicate that recurrent inhibition is prominent within the neck segments of the spinal cord, as it is in other regions. The basic features of recurrent inhibition in the neck segments, and of responses of the Renshaw cells, resemble those in other regions of the spinal cord. As in the hind-limb region, the recurrent inhibition within the neck segments is distributed to a variety of motoneurone species. Spatially, recurrent inhibitory connexions extend at least to the adjacent segment. In contrast to the wider spatial distribution of recurrent inhibition, the input to Renshaw cells is restricted, in the neck as in the hind-limb region of the spinal cord (Eccles *et al.* 1961*b*; Ryall *et al.* 1971). Spatial restriction of the input can be explained by the short

length of the motor axon collaterals that project to Renshaw cells (Cullheim & Kellerth, 1978*a*). As studied in the hind-limb region, Renshaw cells are funicular (Jankowska & Smith, 1973; van Keulen, 1979; Lagerback & Kellerth, 1985) and can project to distances over 12 mm (Jankowska & Smith, 1973), at least over one segment, which would result in a wider spatial distribution of recurrent inhibition in motoneurones. The similarities of effects in neck and hind-limb regions of the spinal cord suggest that the same basic anatomical organization holds in the neck region.

In showing recurrent inhibition of homonymous and heteronymous origin to occur frequently in identified dorsal neck motoneurones, the present results are in apparent disagreement with previous observations of only scarce homonymous and no heteronymous recurrent i.p.s.p.s (Rapoport, 1979). To some extent, differences in preparation (use of sodium pentobarbitone *versus* chloralose for anaesthesia) might contribute. However, some homonymous recurrent i.p.s.p.s were reported in the earlier study and, for reasons discussed by Jankowska & Odotula (1980), were probably underestimated. The greater apparent disparity concerns heteronymous recurrent i.p.s.p.s. However, most of the heteronymous connexions studied by Rapoport (1979) involved trapezius or sternocleidomastiod as giving or receiving motoneurones (S. Rapoport, personal communication), combinations not tested in the present work, and for which recurrent inhibition may well be lacking. Therefore, the disparity in results between the studies is not as great as it would seem.

Factors affecting distribution of recurrent inhibition

Studies on recurrent inhibition in other regions of the spinal cord have defined several factors that influence strength of recurrent inhibition. These include function of, and functional relations between, giving and receiving motoneurones (Wilson *et al.* 1980; Thomas & Wilson, 1967; Hultborn *et al.* 1971*c*), proximity of motoneurone pools (Eccles *et al.* 1961*a*; Kirkwood *et al.* 1981) and type of motoneurone receiving (Granit *et al.* 1957; Kuno, 1959; Friedman *et al.* 1981) or producing (indicated in Eccles *et al.* 1961*a*, *b*) recurrent inhibition. A factor related to the number of α motor axons contained in the nerve stimulated has been explicitly stated by Kirkwood *et al.* (1981). The present results suggest that all these factors influence recurrent inhibition of neck motoneurones.

Spatial factors. In early observations on the distribution of recurrent inhibition among hind-limb motoneurones, it appeared that the distribution could be explained largely on the basis of proximity of motoneuronal pools (Renshaw, 1941; Eccles *et al.* 1954, 1961*a*). However, since location of motoneuronal pools within the spinal cord is not unrelated to muscle function (Romanes, 1951), the two factors of proximity and functional relations between muscles are compounded. Subsequent work (Wilson *et al.* 1960; Thomas & Wilson, 1967; Hultborn *et al.* 1971*c*) has shown enough exceptions to the proximity hypothesis to emphasize functional relationships. That spatial factors *per se* do influence recurrent inhibition is indicated in the work of Eccles *et al.* (1961*a*), but is more readily seen with muscles having rostrocaudal segmentation (of the motoneuronal pools), where spatial and functional aspects may be separated out. Thus, for motoneurones with axons in the intercostal nerves, recurrent inhibition was found up to three segments away, but diminishing in strength with distance (Kirkwood *et al.* 1981). In the present work, segmental nerves

(of splenius) were more effective on motoneurones located at the same level than on those located one segment distant. That proximity is not the predominant factor is evident in that not all nerves at one segment are effective in motoneurones at that level (e.g. l.s.v. has no effect in C3 b.c. or complexus). Furthermore, as seen in the case of the segmental nerves to b.c., other factors may override spatial influences.

As a possible explanation for the greater effect of C3 than of C4 b.c. motor axons on motoneurones at C4, it was pointed out that there are probably many more α motor axons at C3 than at C4. Counts of efferent fibres, though highly variable, support this (Richmond *et al.* 1976). However, the counts suggest the same for splenius, with about the same ratio of C3 to C4 fibres (averaging about 100 to 10). That so few C4 splenius axons can produce larger recurrent i.p.s.p.s. in C4 motoneurones strengthens the case for proximity. However, that C3 to C4 ratios are similar for b.c. and splenius suggests that something other than simply numbers of axons must account for the difference between b.c. and splenius.

Considering transversal locations of motoneurone pools, there is some evidence in the hind-limb segments of the spinal cord that motoneurone pools located very dorsally and laterally (supplying the digits) give and receive little or no recurrent inhibition (Eccles *et al.* 1961*a*; H. Hultborn, R. Katz & R. Mackel, personal communication) and give off few or no axon collaterals (Cullheim & Kellerth, 1978*a*). In the upper cervical cord, transversal distances are small: there is no evidence that laterally located motoneurones might be less effective in producing recurrent inhibition and less affected by it. The laterally located trapezius (Rapoport, 1978), l.s.v., o.s. (E. E. Brink & I. Suzuki, personal observations) motoneurones give and receive recurrent inhibition, and excite Renshaw cells (Rapoport, 1979; present data) as do more medially located motoneurones (splenius, complexus, and b.c.: Richmond, Scott & Abrahams, 1978). Indeed, axon collaterals have been described for trapezius as well as for dorsal neck motoneurones (Keirstead *et al.* 1982).

Functional influences. Among hind-limb motoneurones, recurrent inhibition is typically largest in homonymous motoneurones, occurs between synergists as well as between other motoneuronal species, but is conspicuously absent between strict antagonists acting on the same joint (Wilson *et al.* 1960; Hultborn *et al.* 1971*c*; this also holds for the forelimb: Thomas & Wilson, 1967). Instead, the recurrent inhibition is distributed to the Ia inhibitory interneurones projecting to the antagonists (Hultborn, Jankowska & Lindström, 1971*a*, *b*). Thus, to a large extent, recurrent inhibition among limb motoneurones is correlated with the distribution of 1a reflexes: positively with 1a synergism, negatively with reciprocal inhibition (Hultborn *et al.* 1971*c*). Distribution of recurrent inhibition in the neck segments shows similarities in that (a) at least for C3 b.c. motoneurones, homonymous axons are the most effective, (b) recurrent inhibition occurs between synergists (b.c. and complexus; there are occasional synergistic connexions from b.c. to splenius and from splenius to complexus (Anderson, 1977)), (c) recurrent inhibition extends beyond synergist connexions. However, it may not be possible to extend the comparison to correlation with reciprocal inhibition: observations so far have shown no evidence that Ia reciprocal inhibition occurs among neck motoneurones (Rapoport, 1979).

Since neck muscles are innervated by several nerves, and recurrent inhibition, generally, is largest for the homonymous motoneurone pool, it might be suspected

that other nerves to the same muscle would exert the most powerful effects. Were this so, then C4 b.c. for example, should be more effective than C3 complexus or C3 splenius in C3 b.c. motoneurones. In fact, while C4 b.c. is effective, it is not more effective, presumably because of its greater distance from the target motoneurones. (It is, however, more effective than other C4 nerves: o.s. is rarely effective; sign tests for matched pairs indicate a significant difference between C4 b.c. and C4 splenius effects, $P < 0{\cdot}03$, $n = 12$.)

In the distribution of recurrent inhibition described in the Results, l.s.v. was the least effective and the least affected of the motoneurone pools tested: only splenius axons inhibited these motoneurones. The l.s.v. was the only ventrally located muscle that was tested: possibly its recurrent inhibitory connexions are with other ventral muscles. Because the relationships and reflex linkages of l.s.v. and o.s. are unknown, it is difficult to interpret the data on recurrent inhibition involving these muscles in terms of function. The connexions that do occur are not related to whether innervation is by dorsal or ventral rami: both l.s.v. and o.s. are innervated by branches of ventral rami, but their connexions are with muscles innervated by dorsal rami rather than with each other. The splenius nerve does inhibit l.s.v.; assuming that occurrence of recurrent inhibitory connexions signals some functional relation (Hultborn *et al.* 1971*c*), it is possible splenius and l.s.v. are active together in lateral or rotatory movements of the head. Similarly, the frequent recurrent inhibitory connexions between o.s. and the dorsal neck muscles would suggest that these muscles work together at times. It may be anticipated that some reflex linkages between these muscles occur; however, it appears that at least splenius and o.s. are not linked in Ia synergism (E. E. Brink & I. Suzuki, unpublished observations).

Aside from functional relations, variations in muscle composition probably also influence distribution of recurrent inhibition across motoneuronal species. In the hind-limb segments, recurrent inhibition is largest in slow motoneurones, intermediate in fast fatigue-resistant, and smallest in fast fatiguable motoneurones (Friedman *et al.* 1981) when measured at resting potential. Additionally, it appears that motor axons to predominantly fast muscles are more effective in eliciting recurrent actions (from data of Eccles *et al.* 1961*a*, *b*: effects from lateral or medial gastrocnemius are stronger than those from soleus). Morphologically, axon collaterals systems of fast fatigable motor units have greater numbers of swellings (presumably presynaptic terminals) (Cullheim & Kellerth, 1978*b*). In the neck segments, the frequency of occurrence of recurrent inhibition indicates that splenius is the most effective in eliciting recurrent inhibition but receives the least. The o.s. motoneurone pool is the least effective in producing recurrent inhibition, but receives widely; b.c. and complexus are intermediary, both giving and receiving. Of these muscles, splenius has the greatest percentage of fast fatigable motor units, while o.s. the greatest percentage of slow fibres (Richmond & Abrahams, 1975; l.s.v. has not been studied). Thus, the relative effectiveness in producing and receiving recurrent inhibition might be expected on the basis of muscle composition.

In fact, it might be anticipated that if segmental muscles show systematic variation in muscle fibre composition, the strength of recurrent inhibition should also vary between muscle compartments. For example, if the C4 compartment of splenius contains relatively more slow fibres than C3, recurrent inhibition would be expected

to be larger in C4 than in C3 motoneurones, and actions from C4 weaker or fewer. Possible segmental variation in recurrent inhibition has not been thoroughly studied in the present experiments.

Is there a difference between recurrent inhibition in neck compared with hind-limb motoneurones?

Recurrent i.p.s.p.s produced in neck motoneurones on stimulation of single segmental nerves tend to be, on average, relatively small (average amplitudes less than 0·6 mV and therefore 'weak' according to Hultborn *et al.* 1971*c*) and short in duration (20–30 ms at the longest, *versus* about 50 ms in hind-limb motoneurones, Eccles *et al.* 1954, 1961*a*). This is so even where 'strong' actions (greater than 0·6 mV, Hultborn *et al.* 1971*c*) might be expected, i.e. for maximal homonymous recurrent i.p.s.p.s or i.p.s.p.s between synergists. Correspondingly, the responses of Renshaw cells in the neck regions of the spinal cord did not reach the maximal numbers of spikes (Eccles *et al.* 1961*b*) or lengths of bursts (Eccles *et al.* 1954) seen for some cells in the hind-limb region. Recurrent i.p.s.p.s and Renshaw cell responses in the neck regions are within range of responses in the hind-limb region, but limited to the lower side of that range. On the other hand, recurrent actions in neck motoneurones appear to be comparatively greater than those in respiratory motoneurones. For motoneurones with axons travelling in the internal or external intercostal nerves, amplitudes of homonymous and heteronymous recurrent i.p.s.p.s in individual motoneurones measure 0·1–0·2 mV, with maximal Renshaw cell responses usually consisting of two to three spikes (Kirkwood *et al.* 1981). Similarly, recurrent i.p.s.p.s average 0·1 mV in diaphragm motoneurones (Lipski *et al.* 1985).

A tendency for recurrent effects to be weaker in the neck than in the hind-limb region of the spinal cord might occur for a number of reasons. One factor that may be expected to affect the strength of recurrent inhibition is the segmentation of muscles, their innervation and their motoneurone pools. The total number of α motor axons innervating a neck muscle (roughly estimated at about 200, from Richmond *et al.* (1976)) is roughly comparable to the numbers innervating the hip, knee and ankle muscles studied for recurrent inhibition (Boyd & Davey, 1968). However, the number per segmental neck muscle nerve is less (Richmond *et al.* 1976) than the number which would be activated by stimulating a hind-limb muscle nerve, and represents only a fraction of the neck motoneuronal pool. Since the present results indicate that recurrent effects are exerted from at least one segment distant and data from intercostal nerves (where a similar organization pertains) indicates that effects may be exerted from up to three segments distant (Kirkwood *et al.* 1981), the total recurrent action may be estimated by summing effects from the several segmental nerves. For example, from the present results, by summing the average amplitudes of the recurrent i.p.s.p.s produced by splenius in o.s. (and assuming that the C1 and C2 nerves also contribute, to a lesser extent), the resultant recurrent i.p.s.p. certainly reaches 0·6 mV and may reach about 1 mV. Thus, while effects from individual segmental nerves may be weak, recurrent inhibition from the total motoneuronal pool of a neck muscle might in fact be strong, and comparable to recurrent inhibition in hind-limb motoneurones.

The prevalence of recurrent inhibition in the spinal cord emphasizes its function

as a basic mechanism controlling motor output. The present results show that recurrent inhibition does occur among neck motoneurones, and will therefore be involved in the control of head movements. As has been discussed, there are differences in the organization of recurrent inhibition as it occurs in different regions of the spinal cord, particularly in the distribution of recurrent effects. It may be anticipated that more extensive examination of recurrent inhibition, coupled with further examination of reflex linkages between muscles to aid interpretation of muscle function, will reveal features of spinal organization unique for the neck muscles and the particular movements and functions they subserve.

The authors wish to thank Professor V. J. Wilson and Dr R. Mackel for much helpful discussion and support, and Ms S. Wong and Ms M. Harvey for technical assistance. This work was supported in part by grants from the National Science Foundation (BNS-8315908) and from the National Institutes of Health (NS02619).

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