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A COMPARISON OF THE RECURRENT INHIBITION OF α- AND γ-MOTONEURONES IN THE CAT

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SUMMARY

1. The degree of recurrent inhibition of tonically firing α- and γ-motoneurones to triceps surae muscles was assessed in decerebrated cats by measuring the change in probability of firing caused by an antidromic volley in other motoneurone axons.

2. In nine cats 91 % (thirty-one out of thirty-three) of α- and 54 % (twenty-five out of forty-six) of γ-motoneurones could be inhibited by antidromic volleys in α-motoneurone axons.

3. The degree of recurrent inhibition, expressed as the average reduction in probability of firing during the response, was typically in the range of 50–95 % for α-motoneurones compared to 20–85 % for γ-motoneurones.

4. The duration of recurrent inhibition was 20–50 msec for α-motoneurones and 5–40 msec for γ-motoneurones. The duration was dependent upon the frequency of firing of a neurone, being shorter at high frequencies than at low frequencies. When α- and γ-motoneurones had similar frequencies of discharge the durations of their recurrent inhibition were comparable.

5. Raising the strength of electrical stimulation to elicit an antidromic volley in γ- as well as α-motoneurone axons never produced or increased recurrent inhibition in either type of motoneurone.

6. The quantitative differences in recurrent inhibition of α- and γ-motoneurones are discussed in relation to the control of firing frequency.

INTRODUCTION

Renshaw cells mediate recurrent inhibition from motoneurone axon collaterals, not only to α-motoneurones (Renshaw, 1941; Eccles, Fatt & Koketsu, 1954) but also to other Renshaw cells (Ryall, 1970), Ia inhibitory interneurones (Hultborn, Jankowska & Lindstrom, 1971) and γ-motoneurones to muscle spindles (Ellaway, 1971). Since these neurones are all involved in determining the final output signal to skeletal muscle it is relevant to determine the relative degree to which they are inhibited by the recurrent loop when functioning under similar conditions. This report compares α- and γ-motoneurones.

Recurrent inhibition of γ-motoneurones is not found as frequently as that of α-motoneurones (Brown, Lawrence & Matthews, 1968; Ellaway, 1971; Grillner, 1969;

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Noth, 1971) although no direct comparison of numbers of motoneurones has ever been made. Neither has it been possible to gauge the relative potency of recurrent inhibition of the two types of motoneurone since different methods of assessing the inhibition have been used. For α-motoneurones monosynaptic testing and the change in frequency of firing caused by repetitive antidromic volleys (Granit, Pascoe & Steg, 1957), or size and duration of i.p.s.p.s in response to single antidromic volleys (Eccles *et al.* 1954) have largely been used, whereas Ellaway (1971) expressed the recurrent inhibition of γ-motoneurones in terms of changed probability of firing following single antidromic volleys. The present study was carried out to examine the relative strength of recurrent inhibition on the tonic firing of α- and γ-motoneurones when studied under closely similar conditions in decerebrated cats.

A supplementary problem that has been investigated is whether impulses generated in γ-motoneurone axons give rise to recurrent inhibition. It was implicit in earlier work that, for α-motoneurones, maximal α-efferent antidromic volleys elicited the maximum degree of inhibition (Granit *et al.* 1957) or the largest i.p.s.p.s (Eccles *et al.* 1954). It has now been confirmed that recruiting impulses in γ-motoneurone axons to an antidromic volley in α-axons does not increase the size of recurrent i.p.s.p.s in α-motoneurones (Westbury, 1980). Whether γ-motoneurones can receive recurrent inhibition from impulses in γ-axons has now been investigated. This question was left unresolved in previous studies (Ellaway, 1971; Noth, 1971) but is thought to be unlikely since few Renshaw cells appear to be influenced by antidromic volleys in γ-efferent axons (Kato & Fukushima, 1974) and preliminary studies suggest that γ-axons lack recurrent collaterals (Cullheim & Ulfhake, 1979; Westbury, 1979).

Preliminary findings of our work have been published (Ellaway & Murphy, 1980).

METHODS

The experiments were performed on twelve cats, decerebrated intercollicularly under halothane in oxygen anaesthesia. A lumbar laminectomy was performed to expose spinal roots L6–S2. Cats were fixed firmly to a myograph stand by clamps on the pelvis, 3rd lumbar vertebral spine, femur and tibia. After decerebration cats were allowed to breathe freely from the atmosphere for 1–2 hr. To prevent any spontaneous movements they were then paralysed with gallamine triethiodide (Flaxedil) and respired artificially. Blood pressure, rectal temperature and the temperatures of pools of paraffin oil covering nervous structures were monitored throughout the experiment and maintained within physiologically desirable ranges.

Intact ventral roots L7 and S1 were split longitudinally into two approximately equal parts. One part of each root was then cut and the central end mounted on bipolar platinum wire stimulating electrodes. Electrical stimulation of these cut ventral rootlets was employed to produce antidromic volleys in motoneurone axons. The volleys were monitored by recording from a more central recording position on the cut ventral root. The other parts of these ventral roots were retained intact to preserve a number of triceps surae γ- and α-motoneurones from which recordings could be made in the periphery. In these experiments the dorsal roots were left intact.

In four of the twelve cats antidromic volleys were elicited by stimulation of the cut nerve to either the gastrocnemius medialis (g.m.) or the combined gastrocnemius lateralis and soleus (g.l./sol.) muscles. In such experiments dorsal roots L6–S2 were cut, all ventral roots were retained intact and recordings of motoneurone activity made from the gastrocnemius nerve which was not being stimulated.

Recording and identification of motoneurones

Impulses in γ-motoneurones and a few α-motoneurones were recorded from small fasicles of either the g.m. or g.l./sol. muscle nerves. Fasicles were cut and split until single unit activity could be

recorded. The method and criteria for identification of α- and γ-motoneurones have been described recently (Ellaway & Trott, 1978). The conduction velocity of each motoneurone axon was established by noting the latency of the direct response to a stimulus applied to the intact part of the parent ventral root.

In the preparations described above, a large number of gastrocnemius soleus (g.s.) γ-motoneurones showed a background discharge. Relatively few α-motoneurones, however, were spontaneously active and few could be excited to discharge by stretch of the homonymous muscle since part of the muscle nerve was cut and the muscle paralysed. Thus an alternative means of recording α-motoneurones was employed: monitoring the electromyographic activity of single motor units in the muscle 1–2 hr after decerebration but before paralysis with gallamine triethiodide or dissection of the muscle nerve. Activity in thirty of the thirty-three α-motoneurones studied was recorded electromyographically with concentric needle electrodes and most motoneurones were made to discharge by stretch of the muscle.

Analysis of nerve and muscle impulse activity

Nerve impulse and e.m.g. unit activity were recorded by conventional means and displayed on an oscilloscope. Shaped pulses were used to signal events to a programmable computer (LINC-8, D.E.C.) for further analysis. The computer formed peri-stimulus time histograms (p.s.t.h.s) which give the probability of firing of a cell in relation to a stimulus. The computer was also programmed to form the cumulative sum (cusum) of the p.s.t.h. (Ellaway, 1977, 1978). A cumulative sum is formed by subtracting a reference level from the contents of each bin of the p.s.t.h. in turn and adding these differences together. A cusum plot is the sequential display of the accumulated differences. In this work the reference level was the mean count of a control period of 250 msec before the stimulus was given.

Abbreviations

G.m., gastrocnemius medialis. g.l./sol., gastrocnemius lateralis and soleus. g.s., triceps surae (i.e. g.m. + g.l./sol.).

RESULTS

Measurement and form of recurrent inhibition

Fig. 1*A* shows the effect of an antidromic volley in α-motoneurone axons on the spontaneous discharge of an α- and a γ-motoneurone. The volley was elicited by a stimulus applied to the central end of a cut half (see Methods) of ventral root L7 at a strength twice that of α threshold. It was generally found that shocks applied to the ventral root at a strength of 2–2·5 times α threshold elicited maximal α potentials in axons conducting at velocities above 50 m/sec as recorded at a more central site. Both the α- and γ-motoneurone (Fig. 1*A*) are inhibited and this is evident as an increase in duration of the interspike interval occurring at the time of the antidromic volley. Later in the experiment the discharge of the α-motoneurone ceased while that of the γ-motoneurone continued but at a lower frequency (Fig. 1*B*). The inhibition of the γ-motoneurone is again recognizable.

The difficulty of quantifying such inhibition is met by constructing p.s.t.h.s of the discharge. Fig. 2 presents histograms from the same two neurones seen in Fig. 1. The mean frequencies of background discharge at the time the p.s.t.h.s were constructed were 7 impulses/sec for the α-motoneurone (Fig. 2*A*) and 14 impulses/sec for the γ-motoneurone (Fig. 2*B*). The latency of inhibition is clearly indicated by the p.s.t.h.s and is longer for the γ- (8 msec) than the α-motoneurone (5 msec). This difference simply represents the longer conduction delay to the peripheral recording site for the slower γ-axon. The estimated central delays between arrival of the

antidromic volley and onset of inhibition were 2·1 and 2·0 msec respectively. The maximum systematic error in these measurements is +0·1–0·2 msec. This is the time taken for a spike to rise to the trigger level signalling an event to the computer. Other errors involve the conduction time of α- and γ-impulses in the ventral root since estimates of these are based on peripheral measurements of velocity. In comparing

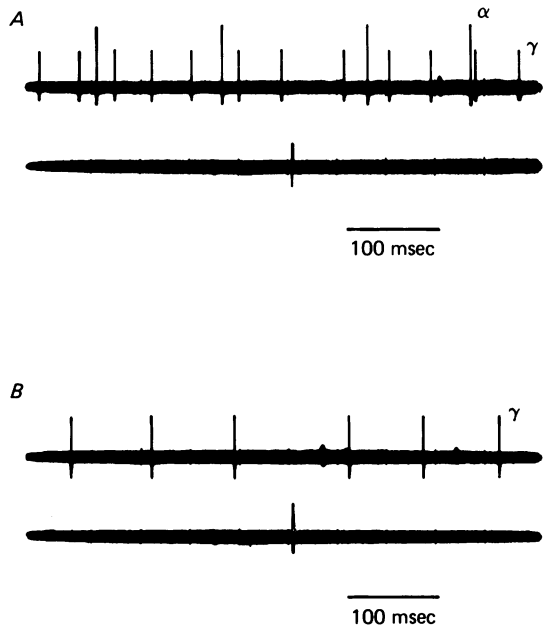

Fig. 1. Recurrent inhibition of α- and γ-motoneurones to g.l./sol. muscles. *A*, upper trace, discharge of an α- (axon conduction velocity 67 m/sec) and a γ- (axon conduction velocity 33 m/sec) motoneurone. Lower trace, recording of the antidromic volley entering the spinal cord in response to a shock to the central end of a cut half of ventral root L7 at a strength of twice α threshold. Stimulus applied 275 msec after the start of the sweep. *B*, As in *A*, but recording at a time when the γ-motoneurone alone was discharging.

α- and γ-efferents these errors are not likely to be greatly different over a short length of ventral root (approx. 15 mm). The mean value of estimated central delays for inhibition of five α-efferents was 1·8 msec ($\pm$0·2 msec, S.E.) and this did not differ significantly from the delay for γ-efferents (mean 2·2$\pm$0·14 msec, S.E.). The latter measurement confirms earlier work in which the mean central delay of recurrent inhibition of the triceps surae γ-motoneurones was found to be 2·3 msec (Ellaway, 1971). This short delay suggests that the inhibition of γ-efferents is mediated by a single interneurone, the Renshaw cell, in the same manner as α-motoneurones. The proposal (Noth, 1971; Kemm & Westbury, 1978) that additional interneurones are involved in the recurrent inhibition of γ-motoneurones is thus rendered unlikely.

The duration of inhibition can be measured more accurately from the cumulative sum derivatives (Fig. 2*C*) of the p.s.t.h.s and is indicated by the duration of the negative-going component. The onset of inhibition was usually clearly defined as a

sudden negative swing in the cusum. The end of the inhibition was more difficult to assess due to random fluctuations in bin counts of the histogram. We thus referred to a number of histograms in order to determine the best point of termination of the effect.

As well as measuring duration of inhibition, the change in probability of firing was assessed by noting the average reduction in number of spikes over the total duration

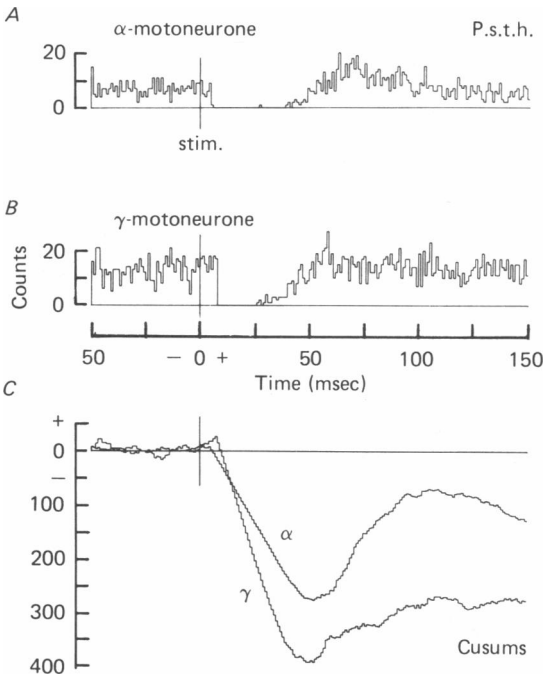

Fig. 2. Measurement of the degree of recurrent inhibition of an α- and γ-motoneurone. *A*, the α-motoneurone; p.s.t.h. of 1024 trials employing stimulation of part of ventral root L7 at twice α threshold. *B*, the γ-motoneurone; p.s.t.h. of 768 trials using the same stimulus; stimuli applied at time zero. *C*, cusums of the p.s.t.h.s in *A* and *B* above. These motoneurones from triceps surae are the same as those in Fig. 1.

of the inhibition. Thus, for the α-motoneurone in Fig. 2, the total duration of inhibition is 43 msec. During that time 275 fewer spikes occurred than expected, an average of 6·4 per msec bin below the control count in the histogram of 6·7 spikes/bin. The inhibition was thus expressed as an average reduction in the probability of firing of, in this case, 95 %. The inhibition of the γ-motoneurone in Fig. 2 lasted 36 msec, with an average reduction in probability of firing of 88 %. This manner of measuring inhibition, i.e. duration and intensity, was considered as providing the best basis for comparing the relative degree of recurrent inhibition of the two types of motoneurone. It may, however, have underestimated the potency of inhibition of those neurones where the probability of firing is actually zero for any length of time. This happened more frequently for α- than for γ-motoneurones (see next section).

Periods of raised probability of firing are present in the histograms of Fig. 2. They

are evident as positive-going phases of the cusums and occur after the inhibition. They were not seen for all neurones but, when present, were due to two factors. In certain instances the positive swing was the first phase of a continuing oscillation in the histogram having the same period as the background discharge of the motoneurone. This occurred for the α-motoneurone of Fig. 2, although the oscillation is cut short in the Figure, and it represents a tendency of the discharge to be reset or rephased by the period of inhibition. In other cases the increased count was not followed by such an oscillation and presumably indicated a period of late facilitation or disinhibition (Wilson & Burgess, 1962). Such late facilitation was always weaker than the preceding inhibition.

Relative strength of recurrent inhibition

When antidromic volleys in α-motoneurone axons were elicited by applying shocks to the cut central ends of approximately half of ventral root L7 or S1 the tonic discharge of thirty out of thirty-three (91 %) α-motoneurones and twenty-five out of forty-six (54 %) γ-motoneurones could be inhibited. The inhibition of most of the α-motoneurones was studied against a background discharge induced by stretch of the homonymous triceps surae while the remaining α- (six) and nearly all the γ-motoneurones showed a spontaneous background discharge. Five γ-motoneurones were excited to discharge by manual stimulation of the skin and pinna.

The number of γ-motoneurones receiving recurrent inhibition varied from animal to animal but the actual number tested in an individual experiment was small (two to fourteen, average 6·4). Since as few as one in seven and as many as five in six neurones were observed to be inhibited, a χ^2 test was carried out to determine whether the ratio varied more than could be expected by pure chance. Applying Yate's correction for small samples, the value of χ^2 was 7·6, showing no evidence of heterogeneity since the 10 % point is 17·3.

To compare the relative potency of recurrent inhibition of α- and γ-motoneurones several neurones of each type were studied in each of a number of individual cats. In the experiment illustrated by Fig. 3, seven out of eight α- and five out of six γ-motoneurones could be inhibited by antidromic volleys in α-axons. Examples of the p.s.t.h.s from the neurones receiving the weakest and strongest inhibition are presented for both γ- (Fig. 3*A*) and α- (Fig. 3*B*) motoneurones. In Fig. 3*C* the potency of inhibition of all the neurones is collated and expressed both in terms of duration of inhibition (abscissa) and average reduction in probability of firing (ordinate). Three of the γ-motoneurones received weaker inhibition than any of the α-motoneurones. Two others, however, received inhibition as potent as that to the α-motoneurones. One γ-motoneurone in particular (firing rate 12 impulses/sec; axonal conduction velocity, 34 m/sec) had an average reduction in probability of firing of 83 % lasting 37 msec. The inhibition of this γ-motoneurone was almost as powerful as that of the most strongly inhibited α-motoneurone studied in the same cat.

Such a distribution of recurrent inhibitory action from antidromic volleys in ventral root axons to α- and γ-motoneurones was quite typical of the five experiments in which similar numbers of both types of neurone were studied.

In three other cats recurrent inhibition of γ-motoneurones alone was studied by applying electrical shocks to one g.s. nerve branch (g.m. or g.l./sol.) while recording

motoneurone activity from the other. Maximal antidromic volleys in α-motoneurone axons were elicited and judged to be maximal from recordings taken at the ventral root. All ventral roots were intact and dorsal roots L6–S2 cut in these three cats. The proportion of γ-motoneurones (seventeen out of thirty-one, 55%) inhibited was similar to that found when ventral roots were stimulated in order to elicit antidromic volleys. Neither did the duration of inhibition or average reduction in probability of firing of γ-motoneurones differ in the two types of experiment.

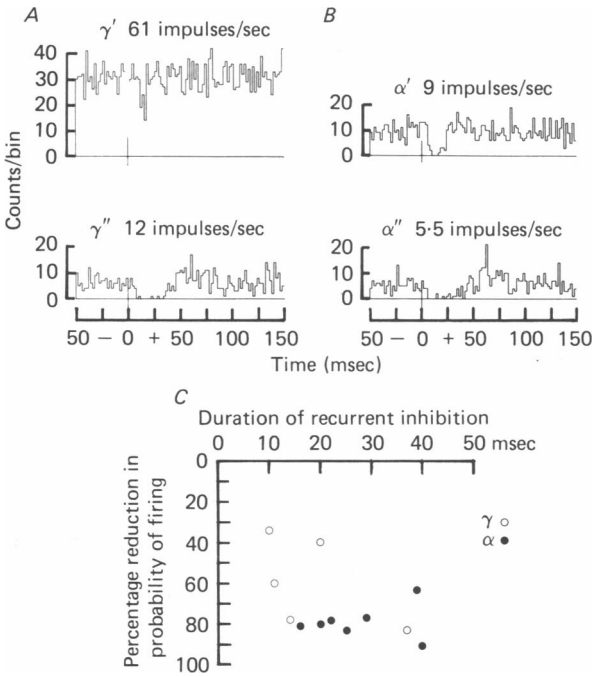

Fig. 3. Relative potency of recurrent inhibition for g.s. α- and γ-motoneurones. These results were obtained from one cat. *A*, p.s.t.h.s (256 trials) for the least inhibited (upper) and most inhibited (lower) γ-motoneurones in response to single shocks to part of ventral root S1 at three times α threshold. *B*, p.s.t.h.s (512 trials) for the least inhibited (upper) and most inhibited (lower) α-motoneurones responding to the same stimuli. The mean frequency of firing for each neurone is indicated above the p.s.t.h. *C*, a plot of the potency of the inhibition for all the neurones in terms of both duration and decreased probability of firing. Each point represents a single motoneurone.

Recurrent inhibition related to frequency of motoneurone firing

It was noticeable (see Fig. 3) that, for γ-motoneurones, the duration of recurrent inhibition was dependent upon the firing frequency of the cell. The inhibition of neurones firing at high frequencies tended to be shorter than for those having a low frequency of discharge. This held for an individual γ-motoneurone both for spontaneous and induced changes in firing rate. The data in Fig. 4*A* is from a g.l./sol. γ-motoneurone whose spontaneous rate of firing was close to 8 impulses/sec (mean interspike interval of 125 msec). Higher maintained rates of discharge were induced

by stimulation of the skin of the contralateral heel and of the belly, and by twisting the pinna. The relation between the duration of the recurrent inhibition and mean interspike interval was best fitted by a linear regression line. The relation was invariably present for γ-motoneurones and was observed either on lowering or raising the frequency of discharge by reflex stimulation.

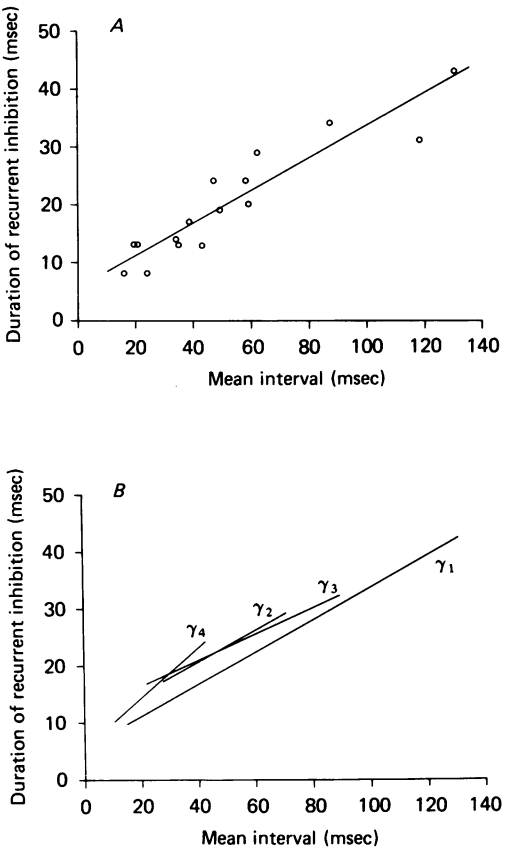

Fig. 4. Relationship between duration of recurrent inhibition and the mean interval of background discharge for γ-motoneurones. *A*, data from a single g.l./sol. neurone (axon conduction velocity, 21 m/sec). Each point assessed from a p.s.t.h. of 512 trials in response to a maximal α antidromic volley in the g.m. muscle nerve. A regression line has been fitted to the points. *B*, linear regressions for all four γ-motoneurones studied in the same cat.

The phenomenon was also seen when the frequency of discharge of a γ-motoneurone was lowered by intravenous injection of pentobarbitone sodium (Sagatal). Before administering the anaesthetic a g.l./sol. γ-motoneurone (conduction velocity 22 m/sec) had been firing at 67 impulses/sec. The duration of recurrent inhibition in response to a muscle nerve antidromic volley was 8 msec. After a total dose of 11 mg pentobarbitone/kg the frequency of firing had fallen to 7 impulses/sec and the duration of inhibition had increased to 51 msec. Using an intermittent infusion of

pentobarbitone, intermediate points had been obtained and a linear relation was found between duration of inhibition and frequency of firing which was indistinguishable from that described below. There was no change in the potency of the inhibition as measured by the average reduction in probability of firing.

Fig. 4*B* shows the regression lines computed for all four γ-motoneurones studied in one experiment. In general γ-motoneurones did not show sustained firing rates higher than 80 impulses/sec or lower than 5 impulses/sec. The regressions appeared linear over this range except that occasionally there was a tendency for the duration of inhibition to plateau for mean intervals longer than 100 msec. In contrast to the changes in duration of inhibition, the average reduction in probability of firing during the response was largely unaffected by changes in firing frequency.

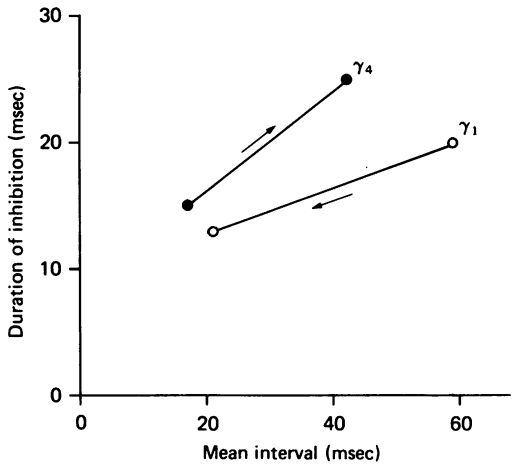

Fig. 5. Correlation of duration of recurrent inhibition with mean interval of discharge for two γ-motoneurones whose frequency of firing was altered by pinna stimulation. The arrows indicate the direction of change caused by twisting the ipsilateral pinna. Same neurones (γ_1 and γ_4) as in Fig. 4. See text for explanation.

In the experiment illustrated in Fig. 5 the discharges of two γ-motoneurones were monitored. Twisting the ipsilateral pinna caused the frequency of one γ-motoneurone to rise and the duration of its inhibition to decrease. The same stimulus lowered the frequency of the other γ-motoneurone and the duration of its inhibition increased. Clearly the duration of the recurrent inhibition is dependent upon the mean interval of discharge of the individual neurones rather than whether or not the peripheral stimulus to the pinna is present. Such changes are thus unlikely to be due to selective alteration by the stimulus to the excitability of recurrent loops controlling the two neurones. The effect is more likely to be a consequence of the biophysical interaction between concurrent facilitatory and inhibitory inputs to individual cells (see Discussion).

A similar dependency of duration of recurrent inhibition on firing frequency was also observed for six α-motoneurones. Inhibition could be as short as 20 msec for mean interspike intervals of 100 msec while at lower rates of firing (mean intervals

approaching 250 msec) this increased to 50 or 60 msec. The relationship was probably a general feature of α-motoneurones since it was evident for six out of eight other motoneurones although these could only be excited to discharge at two preferred frequencies of firing.

Lack of recurrent inhibition from antidromic volleys in γ-motoneurone axons

The degree of inhibition of a γ-motoneurone could not be increased by raising the stimulus strength to a ventral root or muscle nerve above maximum for α-motoneurone axons. This was confirmed on six γ-motoneurones in four cats. Fig. 6 shows that both

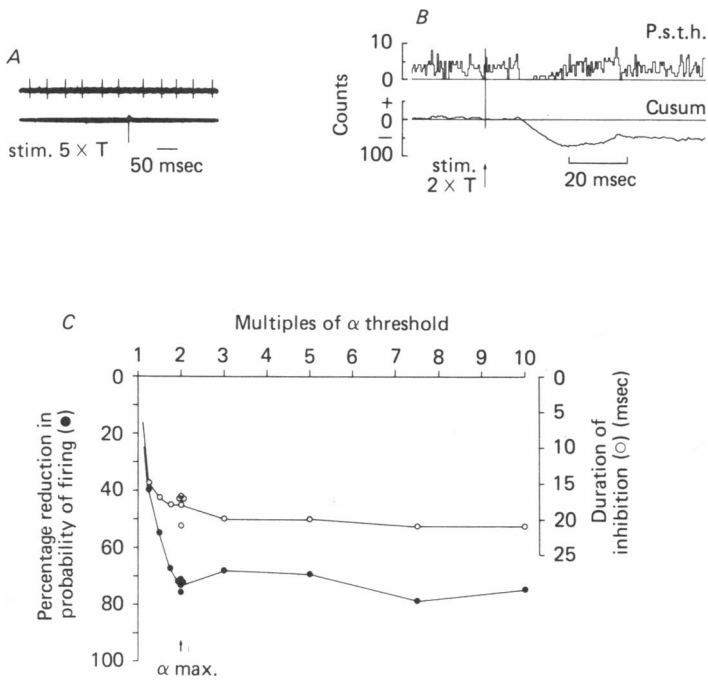

Fig. 6. Dependency of degree of recurrent inhibition upon strength of stimulus used to generate an antidromic volley. G.l./sol. γ-motoneurone (axon conduction velocity, 24 m/sec). *A*, γ discharge (upper trace) showing inhibition caused by an antidromic volley (lower trace) in response to a stimulus applied to the g.m. nerve at 5 times α threshold. *B*, a p.s.t.h. and cusum using a stimulus of twice α threshold. *C*, reduction in probability of firing (●) and duration of inhibition (○) plotted against strength of stimulation of the g.m. nerve. Each point has been calculated from a p.s.t.h. of the form seen in *B*.

the duration of inhibition and reduction in probability of firing reach a maximum at approximately 2–2·5 times threshold for α-motoneurone axons. This strength of stimulus was sufficient to excite all α-motoneurone axons in the muscle nerve and most motoneurone axons conducting at velocities greater than 50 m/sec when the stimulus was applied to a cut ventral root.

In all of these experiments the size of the α volley was monitored from ventral root fibres close to their entry to the spinal cord. To gauge the number of γ-motoneurone axons excited by a peripheral muscle nerve stimulus ventral root L7 was cut shortly

after one experiment in which the degree of inhibition of a γ-motoneurone to stimuli of different strengths had been determined. The peripheral end of the ventral root was split into filaments fine enough so that potentials could be detected from all γ-motoneurone axons responding to shocks to the g.m. muscle nerve up to 20 times α threshold. At this strength we found, in agreement with Boyd & Davey (1968), that all of the γ-axon population was recruited. Fig. 7 shows how the number of γ-axons

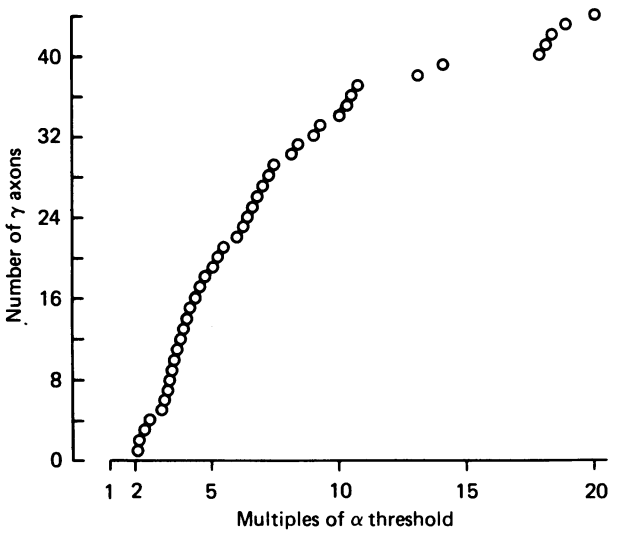

Fig. 7. Number of γ-motoneurone axons in which an antidromic impulse was elicited by stimuli of different strength applied to the g.m. muscle nerve. Recordings made from ventral root filaments. In the same cat a previous experiment had determined that the recurrent inhibition of a g.l./sol. γ-motoneurone reached a maximum at a stimulus strength of only twice α threshold.

excited by the stimulus increases as the strength of shock was raised up to 20 times α threshold. Clearly no γ-axons were recruited with shocks below 2 times α threshold and there was a progressive increase in the number of γ-axons recruited up to 20 times α threshold. In this experiment the duration and intensity of recurrent inhibition of a g.l./sol. γ-motoneurone (31 m/sec) were found to be maximal at strengths of stimulus applied to the muscle nerve of twice α threshold.

In experiments where part of ventral root L7 had been cut, stimulation of the central end of this root increased the recurrent inhibition of a γ-motoneurone seen in response to stimulation of a muscle nerve (dorsal roots cut). However, no increase occurred on raising the stimulus strength applied to the muscle nerve to elicit an antidromic volley in γ-axons. This suggests that the lack of a response to a γ-axon volley was not a result of saturation of the recurrent inhibitory pathway.

In similar experiments we have confirmed Westbury's finding (1980) that volleys in γ-motoneurone axons do not increase the recurrent inhibition of α-motoneurones caused by shocks maximal for α-motoneurone axons.

DISCUSSION

Recurrent inhibition of γ-motoneurones in the decerebrated cat is known to be caused by impulses travelling in α-motoneurone axons (Ellaway, 1971). This work makes it unlikely that impulses generated in γ-motoneurones also contribute recurrent inhibition to their own motoneurone type. In a preparation in which recurrent inhibition from α-axons is clearly evident it seems unlikely that a similar reflex from γ-axons would have been suppressed. There remains the possibility that an effect from an antidromic γ volley could have been occluded by an α volley which had arrived a few milliseconds earlier. This is improbable since 100 % occlusion would have had to have been present on every occasion that a γ-axon volley was tested. Moreover, additional α volleys always produced an increase in recurrent inhibition showing that the inhibition on a single γ-motoneurone from an α-motoneurone volley in its own nerve was not maximal.

We have also confirmed that antidromic volleys in γ-motoneurone axons do not contribute to the recurrent inhibition of α-motoneurones. This lack of effect had been stated in previous studies (e.g. Eccles *et al.* 1954) but, until recently (Westbury, 1980), had not been supported with direct evidence. The lack of participation of γ-axons in the recurrent inhibition of both α- and γ-motoneurones agrees with evidence that impulses in γ-axons do not give any appreciable excitation of Renshaw cells (Kato & Fukushima, 1974) and that γ-axons probably lack recurrent collaterals (Cullheim & Ulfhake, 1979).

The main aim of this work was to examine the relative strength of recurrent inhibition of α- and γ-motoneurones in the same preparation. We consider that the different approaches used to record γ- and α-motoneurone activity in the study did not affect the degree of recurrent inhibition received by the two types of neurone. Our reasons for believing this are as follows. Cats were paralysed when recording from γ-motoneurones whereas the majority of α-motoneurones were studied in the unparalysed preparation. However, we found that intravenous injection of gallamine triethiodide did not affect the inhibition received by γ-motoneurones and the gallamine has no effect on Renshaw cell discharge elicited by antidromic volleys (Eccles, Eccles & Fatt, 1956). The innervation of the homonymous muscle was largely intact when recording from α-motoneurones (as e.m.g. signals) but it is unlikely that afferent muscle activity elicited by stretch would have affected the efficacy of recurrent inhibition. Orthodromic excitation of Renshaw cells by discharges of motoneurones could have occurred when α-motoneurones were excited by muscle stretch. However, to facilitate single unit recording of motor units only low levels of α-motoneurone activity were evoked by stretching the g.s. muscles and this is considered to cause little or no discharge of Renshaw interneurones (Renshaw, 1946). Furthermore, the size of the antidromic volley recorded at the spinal cord was not noticeably diminished by collision with orthodromic impulses when stretching the muscle. With regard to direct effects on Renshaw cells by muscle afferents only the pressure/pain endings connected to Group III axons are thought to have an action (Ryall & Piercey, 1971). Since there is no background discharge and no discharge elicited by stretch in Group III afferents in the cat (Paintal, 1960; Bessou & Laporte, 1960; Ellaway & Trott, unpublished observations) their involvement may be discounted.

Most of the α-motoneurones studied (91 %) received recurrent inhibition whereas the proportion of γ-motoneurones affected was only 54 %. It should be borne in mind that in this study we have recorded from α-motoneurones with low thresholds for tonic firing in response to stretch (Henneman, Somjen & Carpenter, 1965). These low threshold α-motoneurones are the small tonic units which are found to be inhibited more frequently than large, phasic motoneurones (Granit *et al.* 1957; Henatsch & Schulte, 1958). Although some γ-motoneurones were inhibited as strongly as α-motoneurones they generally received less inhibition. But this was only true with respect to the degree to which their firing rate was suppressed at any instant in time following an antidromic test volley. Irrespective of the type of motoneurone, the duration of the inhibition has been shown to be dependent upon the frequency of firing. α- and γ-motoneurones tend to have the same duration of recurrent inhibition if their discharge rates are comparable. Since γ-motoneurones are able to fire tonically at higher rates (up to 80 impulses/sec) than α-motoneurones (generally restricted to below 20 impulses/sec) it does mean that γ-motoneurones frequently exhibit a relatively short duration of recurrent inhibition.

The relation between firing frequency and duration of inhibition is probably of general significance in the central nervous system. Monosynaptic testing has shown that the excitability of a nerve cell is not a linear function of its membrane potential but depends more closely upon the time course of induced conductance changes (Coombs, Eccles & Fatt, 1955). An inhibitory input produces a conductance change which ends shortly after the peak voltage change of an i.p.s.p. If a concurrent excitatory input causes a cell to fire, the impulse will abolish any remaining i.p.s.p. providing the conductance change due to the inhibitory input is over. Thus in a cell where randomly timed synaptic inputs are producing a tonic discharge the time course of a p.s.t.h. during inhibition is likely to be shorter than the intracellularly recorded i.p.s.p. If the rate of depolarization of a neurone increases due to increased excitatory input then the curtailment of an i.p.s.p. due to firing of the cell will occur earlier and would contribute to the relationship observed in the present study.

In cats anaesthetized with pentobarbitone sodium doubt was expressed as to whether activation of the recurrent loop produced significant i.p.s.p.s in γ-motoneurones (Kemm & Westbury, 1978). A slight depressant action of pentobarbitone sodium on Renshaw cell discharge has been reported (Eccles *et al.* 1956) and it is possible that the anaesthetic selectively depressed the recurrent inhibition of γ-motoneurones in these experiments. The present study makes this unlikely. When pentobarbitone sodium was administered in a dose of up to 11 mg/kg no depression of recurrent inhibition of a γ-motoneurone was seen. In fact the inhibition increased in duration, with no change in intensity, but this was concomitant with a fall in discharge frequency caused by the anaesthetic. This result presumably reflects the general relationship that we have found between the two parameters and not an action of the drug on the recurrent pathway.

The distribution and degree of recurrent inhibition of γ-motoneurones is clearly extensive. As has been suggested for α-motoneurones (Granit, Haase & Rutledge, 1960) a function of recurrent inhibition may be to control the frequency of γ-motoneurone discharge. This work shows that the control via Renshaw cells will come from α-motoneurone discharges rather than γ-motoneurones themselves. This is consistent with the findings of Fromm & Noth (1976) who showed that synergist

γ-motoneurones are inhibited during a tonic vibration reflex but that the inhibition is restricted to those γ-motoneurones which are found, by antidromic testing, to receive recurrent inhibition. It is unlikely that in motor acts α discharges could completely suppress γ-motoneurone activity. Repetitive antidromic volleys do not completely silence γ-motoneurones (Noth, 1971; Fromm, Haase & Noth, 1974) even when the volley is maximal for α-axons in a muscle nerve (P. H. Ellaway & P. R. Murphy, unpublished observation). Regulation of the frequency of γ-motoneurone discharge via the recurrent loop will be expected during co-activation of α- and γ-motoneurones. The control will clearly be proportional in that the duration of inhibition will depend upon the frequency of firing of the γ-motoneurone. Recurrent inhibition is likely to control the level of firing rather than the regularity of γ-motoneurone discharge which is adequately carried out by interaction between after-hyperpolarization (Gustaffson & Lipski, 1979) and synaptic input (Ellaway, 1972) together with supraspinal control of segmental reflex connections (Ellaway & Pascoe, 1965).

The significance of the recurrent inhibition of γ-motoneurones relates to the wider question concerning the function of recurrent inhibition of α-motoneurones and interneurones (Hultborn *et al.* 1971; Ryall, 1970). Hultborn, Lindstrom & Wigstrom (1979) propose that during weak contractions the recurrent loop would be facilitated by supraspinal action (Holmqvist & Lundberg, 1959; Haase & Vogel, 1971) in order to augment the low degree of activation of Renshaw cells caused by a small number of active α-motoneurones. Conversely, as drive to the motoneurones increased, the recurrent loop gain could be reduced. We have shown that there could be a further inherent element of control which is independent of supra-spinal influence on the gain of the recurrent loop and related solely to neuronal firing frequency. Thus a shortening of inhibitory effect will be expected as drive increases the frequency of firing. This inherent element will, however, be of more importance to the control of γ-motoneurone discharges which exhibit a wide range of firing frequencies.

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REFERENCES

Bessou, P. & Laporte, Y. (1960). Etudes des recepteurs musculaires innervés par les fibres afférentes du groupe III (fibres myelinisées fines) chez le chat. *Archs ital. Biol.* **99**, 293–321.

Boyd, I. A. & Davey, M. R. (1968). Composition of peripheral nerves. Edinburgh: Livingstone.

Brown, M. C., Lawrence, D. G. & Matthews, P. B. C. (1968). Antidromic inhibition of presumed fusimotor neurones by repetitive nerve stimulation of the ventral root in the decerebrate cat. *Experientia* **24**, 1210–1211.

Coombs, J. S., Eccles, J. C. & Fatt, P. (1955). The inhibitory suppression of reflex discharges from motoneurones. *J. Physiol.* **130**, 396–413.

Cullheim, S. & Ulfhake, B. (1979). Observations on the morphology of intracellularly stained gamma motoneurones in relation to their axon conduction velocity. *Neurosci. Lett.* **13**, 47–50.

Eccles, J. C., Eccles, R. M. & Fatt, P. (1956). Pharmacological investigations on a central synapse operated by acetylcholine. *J. Physiol.* **131**, 154–169.

ECCLES, J. C., FATT, P. & KOKETSU, K. (1954). Cholinergic and inhibitory synapses in a pathway from motor-axon collaterals to motoneurones. *J. Physiol.* **126**, 524–562.

ELLAWAY, P. H. (1971). Recurrent inhibition of fusimotor neurones exhibiting background discharges in the decerebrated and the spinal cat. *J. Physiol.* **216**, 419–439.

ELLAWAY, P. H. (1972). The variability in discharge of fusimotor neurones in the decerebrate cat. *Exp. Brain Res.* **14**, 105–117.

ELLAWAY, P. H. (1977). An application of cumulative sum technique (cusums) to neurophysiology. *J. Physiol.* **265**, 1–2*P*.

ELLAWAY, P. H. (1978). Cumulative sum technique and its application to the analysis of peri-stimulus time histograms. *Electroen. Neurophysiol.* **45**, 302–304.

ELLAWAY, P. H. & MURPHY, P. R. (1980). A quantitative comparison of recurrent inhibition of α- and γ-motoneurones in the cat. *J. Physiol.* **301**, 55–60*P*.

ELLAWAY, P. H. & PASCOE, J. E. (1965). Discharge of semitendinosus fusimotor neurones in the decerebrated and spinalized rabbit. *J. Physiol.* **181**, 200–213.

ELLAWAY, P. H. & TROTT, JUDY, R. (1978). Autogenetic reflex action on to gamma motoneurones by stretch of triceps surae in the decerebrated cat. *J. Physiol.* **276**, 49–66.

FROMM, C., HAASE, J. & NOTH, J. (1974). Length dependent autogenetic inhibition of extensor gamma motoneurones in the decerebrate cat. *Pflügers Arch.* **346**, 251–262.

FROMM, C. & NOTH, J. (1976). Reflex responses of gamma motoneurones to vibration of the muscle they innervate. *J. Physiol.* **256**, 117–136.

GRANIT, R., HAASE, J. & RUTLEDGE, L. T. (1960). Recurrent inhibition in relation to frequency of firing and limitation of discharge rate of extensor motoneurones. *J. Physiol.* **154**, 308–328.

GRANIT, R., PASCOE, J. E. & STEG, G. (1957). The behaviour of tonic α- and γ-motoneurones during stimulation of recurrent collaterals. *J. Physiol.* **138**, 381–400.

GRILLNER, S. (1969). The influence of DOPA on the static and dynamic fusimotor activity to the triceps surae of the spinal cat. *Acta physiol. scand.* **77**, 490–509.

GUSTAFSSON, B. & LIPSKI, J. (1979). Do gamma motoneurones lack a long lasting afterhyperpolarisation? *Brain Res.* **172**, 349–353.

HAASE, J. & VOGEL, B. (1971). Direkte und indirekte Wirkungen supraspinaler Reizungen auf Renshaw-Zellen. *Pflügers Arch.* **325**, 334–346.

HENATSCH, H. D. & SCHULTE, F. J. (1958). Reflexerregung und eigenhemmung tonischer und phasischer α-motoneurone wahrend chemischer dauererregung der Muskelspindeln. *Pflügers Arch ges. Physiol.* **268**, 134–147.

HENNEMAN, E., SOMJEN, G. & CARPENTER, D. O. (1965). Excitability and inhibitibility of motoneurones of different sizes. *J. Neurophysiol.* **28**, 599–620.

HOLMQVIST, B. & LUNDBERG, A. (1959). On the organisation of the supraspinal inhibitory control of interneurones of various spinal reflex arcs. *Archs ital. Biol.* **97**, 340–356.

HULTBORN, H., JANKOWSKA, E. & LINDSTROM, S. (1971). Recurrent inhibition from motor axon collaterals of transmission in the Ia inhibitory pathway to motoneurones. *J. Physiol.* **215**, 591–612.

HULTBORN, H., LINDSTROM, S. & WIGSTROM, H. (1979). On the function of recurrent inhibition in the spinal cord. *Exp. Brain Res.* **37**, 399–403.

KATO, M. & FUKUSHIMA, K. (1974). Effect of differential blocking of motor axons on antidromic activation of Renshaw cells in the cat. *Exp. Brain Res.* **20**, 135–143.

KEMM, R. E. & WESTBURY, D. R. (1978). Some properties of spinal γ-motoneurones in the cat, determined by micro-electrode recording. *J. Physiol.* **282**, 59–71.

NOTH, J. (1971). Recurrente Hemmung der Extensor-Fusimotoneurone? *Pflügers Arch.* **329**, 23–33.

PAINTAL, A. (1960). Functional analysis of Gp III afferent fibres of mammalian muscles. *J. Physiol.* **152**, 250–270.

RENSHAW, B. (1941). Influence of discharge of motoneurones upon excitation of neighbouring motoneurones. *J. Neurophysiol.* **4**, 167–183.

RENSHAW, B. (1946). Central effects of centripetal impulses in axons of spinal ventral roots. *J. Neurophysiol.* **9**, 191–204.

RYALL, R. W. (1970). Renshaw mediated inhibition of Renshaw cells: patterns of excitation and inhibition from impulses in motor axon collaterals. *J. Neurophysiol.* **33**, 257–270.

RYALL, R. W. & PIERCEY, M. F. (1971). Excitation and inhibition of Renshaw cells by impulses in peripheral afferent nerve fibres. *J. Neurophysiol.* **34**, 242–251.

WESTBURY, D. R. (1979). The morphology of four gamma motoneurones examined by horseradish peroxidase histochemistry. *J. Physiol.* **292**, 25–26*P*.

WESTBURY, D. R. (1980). Lack of a contribution from γ-motoneurone axons to Renshaw inhibition in the cat spinal cord. *Brain Res.* **186,** 217–221.

WILSON, V. J. & BURGESS, P. R. (1962). Disinhibition in the cat spinal cord. *J. Neurophysiol.* **25**, 392–404.