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NET DEPOLARIZATION AND DISCHARGE RATE OF MOTONEURONES, AS MEASURED BY RECURRENT INHIBITION

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Sherrington, struck by the way reflex excitation and inhibition could be graded against each other, often used the simile that this took place as if by algebraical summation. To-day it is possible to translate this general notion into the simplest kind of algebra and find out how well it works in suitable experiments. Such an attempt forms the subject of the present paper. Recurrent inhibition (Renshaw, 1946) of tonically discharging extensor motoneurones in mammals has been used because this acts by the antidromic route and hence can be made maximal and constant with less risk of stirring up accessory circuits than is present with orthodromic inhibitions.

We have previously reported (Granit & Rutledge, 1960; Granit, Haase & Rutledge, 1960) that the normal tonic firing frequency F_n of an extensor motoneurone in the decerebrate preparation is depressed to a slower rate F_i under maintained recurrent inhibition. F_i was found to be linearly related to F_n. In those experiments the antidromic stimulus was locked to the discharging spike and thus was forced to act at the low average frequency of the latter. Therefore it varied with the discharge rate of the motoneurone to whose action it was tied, the experiments being designed to study 'frequency limitation' of a discharge. The present problem requires that antidromic stimulation should be constant in rate and strength and of high frequency while the rate of firing of the motoneurone (F_n) is varied by tetanizing extensor muscle nerves of de-efferented animals at different rates and strength. It will often become necessary to draw upon the results of the two papers mentioned above, which will then for convenience be referred to respectively as (I) and (II).

Since the experiment is run in terms of discharge frequency it is necessary to know how this is related to depolarizing current across the cell

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membrane. Direct measurements by K. Frank and M. G. F. Fuortes (personal communication) prove the relationship to be linear over a range greatly exceeding the normal firing frequency of the motoneurones. Thus the concept 'generator potential' not only applies to the motoneurones but, just as in several sense organs (Katz, 1950; MacNichol, 1956; Fuortes, 1958, 1959; Loewenstein, 1960; Wolbarsht, 1960), impulse frequency is likely to be proportional to magnitude of generator potential, even though at the moment the evidence for this view is based merely on artificial stimulation of motoneurones by the electrical current.

Motoneurones differ from sense organs in being simultaneously bombarded by both excitatory and inhibitory impulses. For the purpose of the test we shall accept the now well-supported conclusion from the work of Eccles and his colleagues (see e.g. Eccles, 1957, 1961) that excitation and inhibition have their equivalents in depolarizing and polarizing potentials across the cell membrane corresponding to depolarizing and polarizing currents. This by no means excludes other forms of inhibition, but these at the moment do not concern us since the recurrent variety is of the polarizing type (Eccles, Fatt & Koketsu, 1954). From this would follow (cf. I, II) that the discharge frequency

$$F = k(P_{\text{dep}} + P_{\text{pol}}), \tag{1}$$

where F is the discharge frequency of the motoneurone, k a constant, P_{dep} the excitatory and P_{pol} the inhibitory current, the two being of opposite sign. Their difference $(P_{\text{dep}} + P_{\text{pol}})$ is defined as 'depolarizing pressure' (cf. I; and Phillips, 1959) or, more strictly, as net depolarizing current.

If now a motoneurone is excited by maintained orthodromic tetanization of the muscular afferents to discharge at various rates F_{n}, an antidromic tetanus of constant rate and strength, causing a constant inhibition (P'_{pol}), should, since

$$F_{\text{n}} - F_{\text{i}} = -kP'_{\text{pol}}, \tag{2}$$

reduce the discharge frequency by a constant number of impulses.

The derivation of eqn. 2 is based on the effect of current applied to the motoneurone and on what has been found by studying motoneurones with the intracellular technique during recurrent inhibition (cf. above). It is not evident that it is valid for a reflex in which, for instance, P'_{pol} may be influenced by the orthodromic stimulus (cf. paper II, and Haase & Van Der Meulen, 1961). The aim of the present work is to find out by experimentation whether the generalization made in eqn. 1 (and its corollary eqn. 2) is legitimate.

Observations have also been made on the relation between $F_{\text{n}} - F_{\text{i}}$ and antidromic tetanus rate. Finally, the results have been considered

from the point of view of the tasks assignable to the recurrent collaterals which are common in all nervous centres.

METHODS

The decerebrate cat was used and single ventral root fibres were functionally isolated in $L7$ or $S1$. Both legs were denervated and on the experimental side the ventral roots were severed intradurally from $L5$ to end of cord. Sometimes the tail end of the cord was cut across (when the tail tended to move). Stimulating electrodes (Fig. 1) were placed on the central ends of the severed gastrocnemius nerves to elicit a maintained tonic reflex by tetani varied in rate and strength (cf. I, II). Another pair of electrodes was placed on the

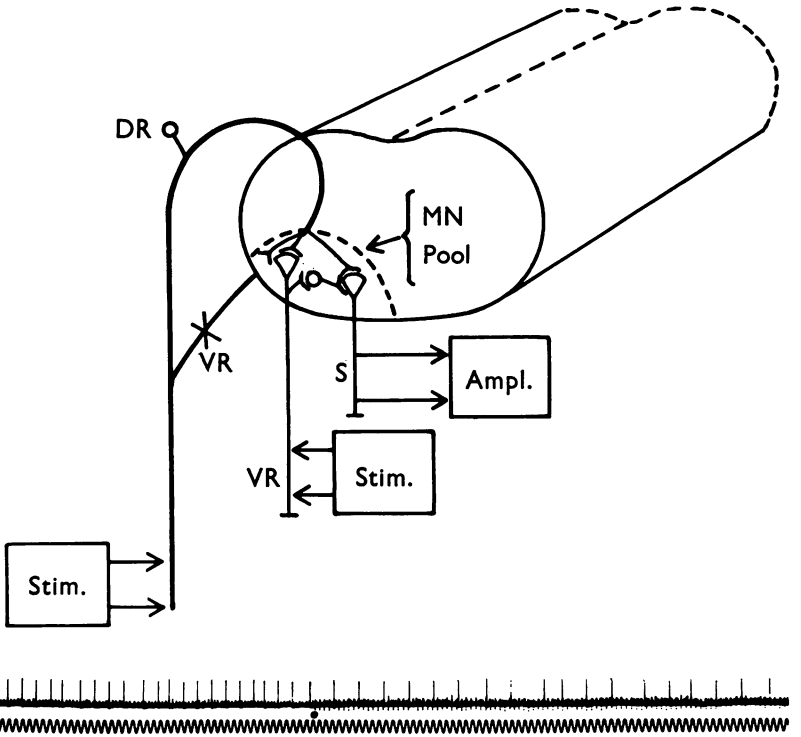

Fig. 1. Experimental arrangement. Stimulating electrodes on severed gastrocnemius nerves and on ventral root (VR) from which single fibre (S) has been isolated. DR, dorsal root. Within motoneurone pool (MN) recurrent circuit diagrammatically shown. Below, record of spike discharging in response to repetitive stimulation of gastrocnemius nerves; antidromic stimulation at 48/sec inserted at moment marked by dot; time marker, 100 c/s.

rest of the ventral root from which the fibre was isolated (see Fig. 1) in order to stimulate its fellow motoneurones antidromically so as to induce recurrent inhibition. In any one experiment the rate of antidromic tetanization was of constant and maximal strength, its rate being not less than 42/sec and generally between 55 and 65/sec. This reduced the firing frequency of the motoneurone from F_n to the inhibited discharge rate F_i.

The decerebrate animal, being 'tonic' in itself, offers the advantage of making it possible to stay within low stimulus strengths in trying to increase F_n. This is of some importance because repeated use of strong tetani tends to decrease stability of the recurrent effect (I, II). Such stimuli might influence the Renshaw cells as proved for dorsal root stimulation by Haase & Van Der Meulen (1961). Eccles *et al.* (1954) have found no effects on Renshaw cells from large muscular afferents. Our problem required finding motoneurones which could be made to discharge over a large range. The great majority of tonic cells is heavily stabilized in rate of discharge with respect to muscular afferents (Denny-Brown, 1929; Granit, 1958; I; II). To obtain a number of cells varying in F_n over a large range it became necessary to study some less well stabilized cells during the adaptive phase characterized by gradually diminishing discharge rate.

In this paper we have called 'Procedure I' the earlier type of experiment (II) in which the recurrent inhibition is inserted at a definite moment, in this case 20 sec after initiation of stimulation. F_n is measured for 4–5 sec before and after the period of 5 sec antidromic stimulation to give F_i. The two F_n values are averaged. Such measurements are single tests repeated at intervals of 1 min. The disadvantage of Procedure I is that it takes a long time to accumulate a sufficient number of values, thus giving systematic changes in the state of the animal a chance to influence the measurements.

'Procedure II' consists in maintaining the afferent tetanus for some time and by automatic regulation inserting 2 sec antidromic tetani at intervals of 7 sec. Between each run 2 min were allowed to elapse. Procedure II gave access to 'adapting' motoneurones whose initial frequency accordingly was high relative to the final stable value. The risk with this procedure is that available surplus excitation in many such cells is reduced while stimulation goes on, as analysed in (I), and that high rates of F_n are more favourably located with respect to this time factor than are low rates. To some extent this systematic error can be overcome by taking runs at low stimulus strengths also, giving low values of F_n a chance to appear in the beginning, and also by multiplying the number of observations. For F_n, 2–4 sec measurements before and after antidromic stimulation were averaged and some three to five values were obtained for each run. Independently of discharge rate, time of maintenance of a discharge will in many cells influence $F_n - F_i$. A description of such effects was given in (I). Some cells do not possess this complication, which makes it necessary to restrict number of tests in each run by Procedure II. We have also made combined experiments applying both Procedures I and II to the same motoneurone. This should be a good check on systematic errors.

RESULTS

Testing equation 2. A graphic presentation of our findings is obtained by plotting F_i against F_n in impulses per second and studying some individual experiments before summarizing the results. If there were no recurrent inhibition acting upon the selected motoneurone, F_n would be identical with F_i and the plot of one against the other a straight line with an angular coefficient of 45°. This line is drawn in all the records of Fig. 2. Equation 2 then states that, in every case where recurrent inhibition is present, the curve obtained must be parallel to this line and shifted downwards by an amount dependent upon the degree of recurrent inhibition in that particular motoneurone. It follows from eqn. 2 that the regression line, as calculated for each motoneurone by the method of least squares, in an ideal case should be at 45° from the x-axis, i.e. possess a regression coefficient $b = 1{\cdot}00$. The coefficients for the samples (= all observations

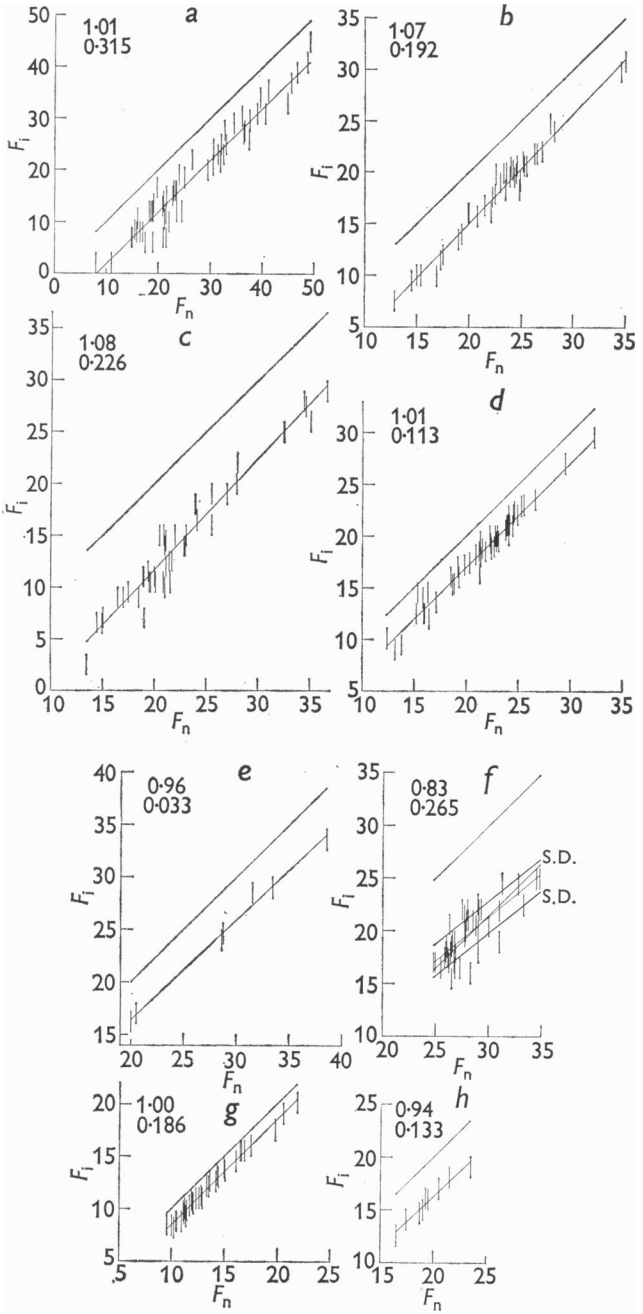

Fig. 2. Eight different experiments aiming at measuring $F_n - F_1$. A 45° line drawn in each diagram to show $F_1 = F_n$. Regression line, regression coefficient and standard error shown in each graph, the two latter on the left; standard deviation shown in the graph (*f*) in which range is small and variability large. In this case second line crossing regression line is drawn to regression coefficient 1·00. *a*, Procedure I+II; *b*, same; *c*, Procedure II; *d*, Procedure I+II; *e*, Procedure I; *f*, Procedure II; *g*, same; *h*, Procedure I.

on any one spike) illustrated in Fig. 2 are shown above the curves in each graph together with the standard error $s/\sqrt{N}$ in measuring $F_n - F_1$; N here is the number of observations, s the standard deviation. These examples have been chosen to illustrate experimental variations such as large range (F_n max $- F_n$ min), small range, large and small values of the difference $F_n - F_1$, Procedure I, Procedure II, Procedure I+II, and number of observations (N). One 'bad' experiment (Fig. 2(f)) is also included, bad in the sense that the range was small and s (marked S.D in the graph) large. The value of b was 0·83 in this case, but, considering s the alternative line with $b = 1{\cdot}00$ can hardly be said to be excluded by the experimental results. While deviations of individual regression lines from $b = 1{\cdot}00$ do not necessarily invalidate eqn. 2, it is nevertheless of some interest to know that while only 2 cells fell outside the limits $0{\cdot}9 < b < 1{\cdot}1$, 16 cells fell inside it. The total number of cells was 18, the total number of observations 470.

Particularly interesting is the rare type of motoneurone (cf. Fig. 2a) which covered a range from 9·0 to 49·5 impulses/sec with a recurrent inhibition of the order of 8 impulses/sec, because this neurone could be studied by Procedures I and II for low and high-range values; 58 measurements were obtained. In spite of the scatter, the standard error being 0·315, the number of observations allowed the coefficient b ($= 1{\cdot}01$) to be determined with considerable accuracy. The scatter arose from the values by Procedure II taken during 'adaptation' of the discharge frequency to the small range obtainable by Procedure I (which generally is more reliable). However, range being important for the problem, half the number of cells was studied by Procedure II and three in that group by both Procedures I and II. The influence of time of maintenance of the discharge and loss of surplus excitation was discussed in (I). When in spite of constant F_n the resistance to recurrent inhibition changes rapidly, the cell is useless for the present purpose. It is not known whether this depends upon the cell or upon something in the state of the preparation. Of 20 cells only 2 were left out for this reason. The beginning and end of every discharge offer problems of their own, which will not be considered in the present work.

Now all the experiments with the 18 motoneurones, regardless of range and frequency of discharge, may be treated as one single experiment testing validity of eqn. 2. The regression coefficients for individual cells should then be weighted with respect to the number N in order to obtain the true average value for b. When this is done it is found that

$$\Sigma(bN)/\Sigma(N) = 0{\cdot}996$$

which is an unexpectedly good approximation to the theoretical, $b = 1{\cdot}00$.

For each cell or sample the standard error ($s/\sqrt{N}$) for measuring $F_n - F_i$ was calculated (see below and Fig. 2). The average is 0·240. The mean $F_n - F_i$ is 4·86, the weighted mean 5·45. This means that in the averages

$$F_n - F_i = 5{\cdot}5.$$

Still regarding the 470 observations as members of one single experiment devoted to examining the constancy of $F_n - F_i$, it is of considerable interest to study the deviations from the mean $F_n - F_i$ for each motoneurone. The average standard deviation was 1·14 and experimental experience shows that the values always can be measured to 1·0 spike/sec.

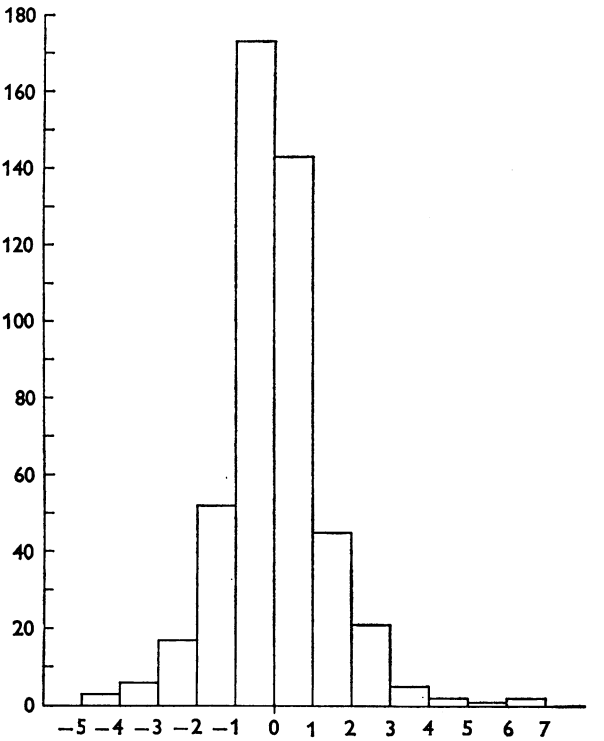

Fig. 3. Histogram of 470 deviations from $F_n - F_i$ = constant. Unit variate for the classes is 1·0 impulse/sec.

Therefore, in plotting a histogram in order to demonstrate the distribution of the deviations from the sample means, the unit variate is chosen to be 1·0. Figure 3 illustrates the result. There is no reason to believe that the distribution of variates is anything but normal. Apparently systematic errors are of a small order and have cancelled out. Thus the results of the analysis of Fig. 3 agree with the findings based on the idea of an average regression coefficient of the order of 1·0.

The normal distribution in Fig. 3 means that it is legitimate to calculate the limits of error of the sample means for their various degrees of freedom. This calculation has been carried out for the probability level 0·95 and consists in multiplying the standard error by the appropriate factor t. It is believed that after giving for each experiment the value so obtained together with N (in brackets), further elaboration of the results by statistical methods yields nothing new, even though for some of the best experiments the variance around the regression line was also calculated. For the 18 cells $ts/\sqrt{N}$ is: 0·15 (12), 0·19 (7), 0·23 (22), 0·59 (18), 0·52 (9), 0·63 (10), 1·34 (8), 1·26 (14), 0·31 (9), 0·63 (58), 1·02 (24), 0·45 (44), 0·23 (48), 0·39 (39), 0·31 (42), 0·54 (41), 0·18 (30), 0·38 (35), mean = 0·519.

Variation of rate of antidromic stimulation. The problem of $F_n - F_1$ as a function of antidromic stimulus frequency is not as well defined as that of the previous section because the discharge pattern of the Renshaw cells changes from the well-known bursts at slow rates to single spikes per shock at high rates of tetanization and the transitional region is not precisely known *in casu*. Parallel work in this laboratory by J. Haase & J. Van Der Meulen (1961 and personal communication) on single Renshaw cells established that the probable range within which this change takes place is from 5 to 15 shocks/sec. From our point of view rates below 5/sec are of little physiological interest, because motoneurones do not discharge rhythmically below 5/sec and many of them have still higher thresholds for regular repetitive firing (see statistical distribution curves in paper II as well as the actual measurements shown above in Fig. 2). An effect of stimulus frequency on the value of $F_n - F_1$ can generally be traced up to 30/sec, sometimes to 40/sec (see Fig. 5); this range is also the characteristic range of variation of the discharge frequencies of motoneurones.

The physically well-defined stimulus frequency is thus changed by the intercalated Renshaw cell into a function which for low rates cannot increase in proportion to the number of antidromic shocks per second. A simple calculation also exemplifies this: let slowly repeated antidromic shocks be followed by silent periods of the order of 40 msec—a value typical of many motoneurones. If these silent periods could be maintained up to stimulus rates of the order of 25/sec and if F_n were of the same order, then recurrent inhibition ought to be practically permanent, i.e. $F_1 = 0$. This is never the case.

Figure 4 should be studied from these points of view. As antidromic stimulation rates increase, irregular silent periods are gradually replaced by an 'inhibitory state' of reasonably regular firing at some rate lower than that of the control. The two experiments have been chosen to demon-

strate one case with, and one without rebound. Clearly, at low rates of antidromic stimulation, the momentary increase of discharge frequency during rebound compensates for the preceding loss of spikes in the silent

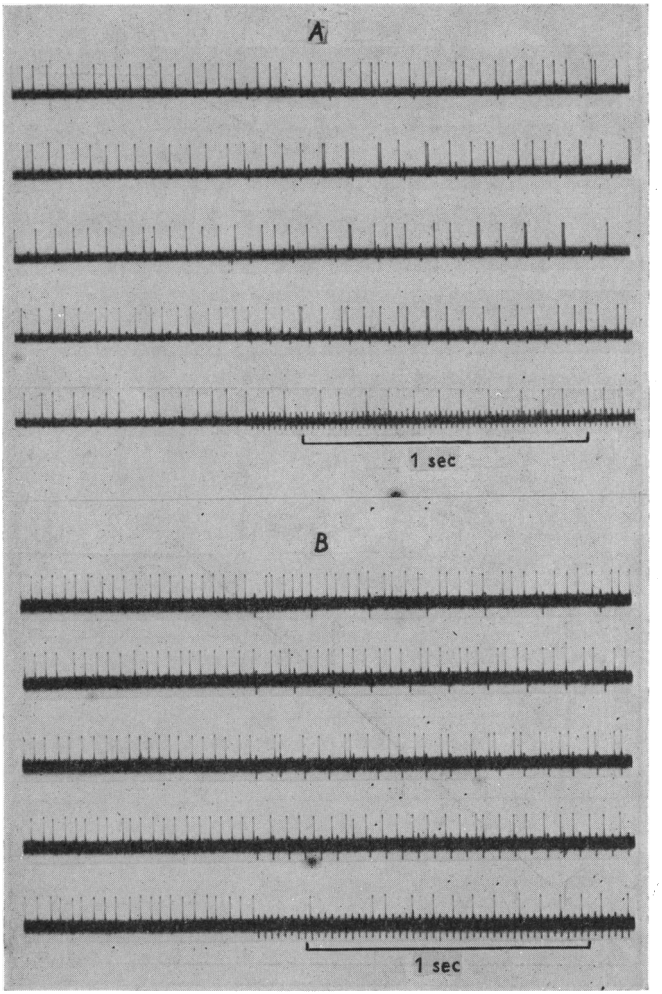

Fig. 4. Two different experiments in which rate of antidromic stimulation is varied from record to record. Stimulus frequencies from above downwards are: (shocks/sec) *A*, 3·4, 5·5, 6·7, 14·5, 48·0; *B*, 4·8, 7·4, 11·6, 17·4, 42·0.

periods. We believe this rebound to be a useful index (when present) of the transformation of the pattern of discharge of the Renshaw cells, as discussed above. Rapidly occurring antidromic shocks cut short incipient rebound and replace it by a fresh wave of recurrent inhibition.

After these remarks, which show that a variation of stimulus rate introduces one more variable into an already difficult experiment, it may seem surprising that in the few experiments in which complete curves were obtained the results suggested that (beginning from such low rates as

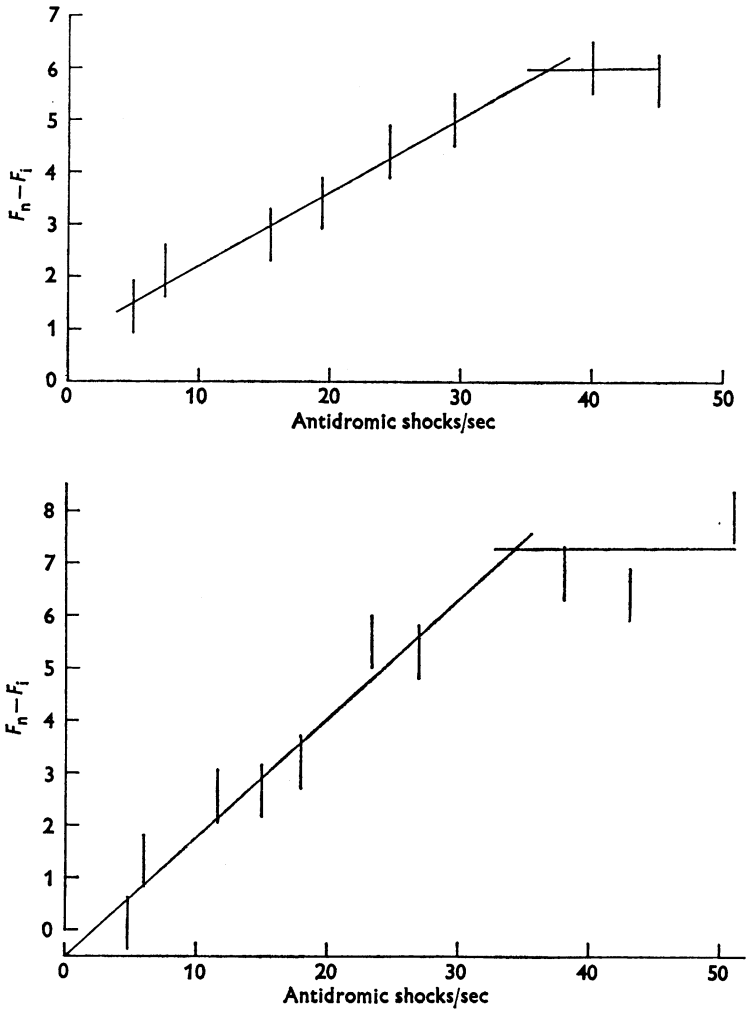

Fig. 5. Two experiments showing effect of antidromic stimulus rate (abscissa) on $F_n - F_i$. The horizontal line shows final value of the latter.

5–10 shocks/sec) up to the maximum $F_n - F_i$, efficacy of recurrent inhibition actually increased in direct proportion to frequency of antidromic stimulation. This is illustrated in Fig. 5. The curves had a flat portion at very slow stimulation rates (not illustrated) which represents the threshold for the effect of stimulus frequency, when only irregular silent periods

and rebounds are produced by the antidromic shocks. These experiments also show why it was necessary to use an antidromic tetanus of high frequency.

It should be pointed out that the bursts by which Renshaw cells discharge at slow rates of antidromic stimulation are very likely artifacts from the physiological point of view, except with well synchronized single contractions, convulsions etc. Some motoneurones (see Fig. 4*B*) show signs of this initial burst by having a long initial pause. Others do not. The Renshaw cells are naturally stimulated by the asynchronous firing of the many motoneurones which converge upon them and not by slow synchronous shocks. For this reason the linearly rising portion of the curves of Fig. 5, falling as it does within the range within which motoneurones discharge, is probably a better approximation to something normal than the effects seen at low rates in Fig. 4.

In our previous experiments (II) when the antidromic shock was tied to the firing spike its frequency varied with the low discharge frequency of the motoneurone investigated. As a consequence one would have expected $F_n - F_i$ always to increase with F_n, as it actually did in many experiments and on an average (33 motoneurones), confirming the results of Fig. 5. But in those experiments there were several individual cases in which $F_n - F_i$ was maximal at low rates of discharge (= low rates of antidromic stimulation) and decreased for higher rates. At the time it was suggested that this was due to competition with 'natural' recurrent inhibition. The present results suggest, however, that owing to the low discharge rates and small frequency range of the previous experiments (12 impulses/sec on an average; F_n min. forming a peak between 6 and 10 impulses/sec), several tests must have fallen within the transitional range where the discharge pattern of the Renshaw cells undergoes large alteration, and that this is a more likely explanation of the exceptions mentioned, inasmuch as at slow rates bursts from the Renshaw cells occur (cf. above) and extend duration and thereby efficacy of inhibition. In the first set of experiments of this paper the rate of antidromic stimulation was constant, generally around 55/sec, and so the experiments cannot very well be compared with the earlier ones in which rate of stimulation was at the low and variable firing frequency of the motoneurones.

DISCUSSION

The deduction tested in this paper (eqn. 1) extended to reflex firing what seemed a likely consequence from work on direct electrical stimulation of motoneurones (K. Frank & M. G. F. Fuortes, 1960, and personal communication) and on intracellular recording of changes of membrane potential during recurrent inhibition (Eccles *et al.* 1954). The results have been in good agreement with our deduction. The test carried out would seem to be a necessary preliminary to any quantitative work on motoneurones enagaged in balancing out the net effect of natural excitatory and inhibitory influences converging upon them. In a general way our conclusions are also supported by the qualitative findings of Phillips (1956) and Terzuolo (1959). They may further be said to extend the realm of applicability of the concept of 'generator potential' (Granit, 1947).

The biological law established here—like all such laws—can hardly be valid in the extremes. At the low rates or strengths of stimulation needed in many motoneurones for small values of F_n, there may not be enough surplus excitation to support the discharge. This problem was studied in detail by Granit & Rutledge (1960). At the highest rates of F_n the output from the fellow motoneurones, clashing as it does with the antidromic stimuli, may succeed in effectively reducing the rate of antidromic stimulation. Animals in feeble condition may never be able to support maintained, tonic discharges well enough for the test to be applied. Too strong afferent stimuli may alter the quantity P'_{pol} (cf. II; and Haase & Van Der Meulen, 1961) and thus invalidate the conditions under which the trial must be conducted. It is also possible that the motoneurone possesses degrees of freedom with respect to facilitation and depression which are not reflected by changes of membrane potential, such as effects of the electrical field (Fatt, 1957*a*, *b*) and unfathomed factors such as those described for invertebrates by Hagiwara & Bullock (1957), Bullock (1959) and Tauc (1960). However, the fact that the experimental test can be carried out at all, i.e. with cells remaining in reasonably steady states for the 1–2 hr required for making 30–50 observations—proves that our deduction contains an essential element of truth.

Of considerable interest is the detailed agreement with the results of Hartline (1949) and Hartline & Ratliff (1956) concerning the so-called lateral inhibition in the *Limulus* eye. This is linear with discharge frequency in the sense that one inhibiting ommatidium influences another adjacent one in proportion to its own rate of firing whilst the rate of firing of the inhibited ommatidium is unimportant as long as the inhibiting ommatidium is fired at constant rate. These are the very results we have had above with recurrent inhibition, which probably fulfils similar tasks, as suggested by Brooks (1959) and Wilson, Talbot & Diecke (1960). In our case the plan of work for the first experimental section was deducted from a hypothesis that possibly is valid for lateral inhibition also. In both cases frequency of discharge rather than spike interval gives simple linear relations. Hartline & Ratliff (1957) have designed interesting experiments calculated to throw light on visual mechanisms of contrast. In our case it is necessary to think of what the results signify for tonic reflexes.

To this end, let us recall that muscular contraction is by no means linear with frequency of efferent stimulation. On the contrary, low rates of stimulation (around 5/sec) have but little influence on the tonic soleus and then, as stimulus rate is increased, isometric tension rises rapidly towards a maximum reached asymptotically around rates of 30/sec. This is shown by the curve in Fig. 6 (black dots) taken from the work of Matthews (1959), which refers to soleus stimulated electrically through its nerve at

different rates. This is a muscle for which the natural frequency of efferent discharge in tonic reflexes is from 5 to 25 impulses/sec (Denny-Brown, 1929; Granit, 1958) and fairly independent of extension. Let us now assume that the scale on the abscissa represents a variation in firing rate from edge to centre (containing the most active neurones firing at 20–25/sec) of the soleus motoneurone pool. Let a constant recurrent inhibition, largely maintained by the centre of the pool, be distributed across this pool in accordance with our findings. This means that if from each abscissa 6 impulses/sec (average $F_n - F_1$ from p. 466) be subtracted, the upper curve (1) should illustrate how much the discharge across the pool would contribute to tension in the absence of recurrent inhibition. The difference

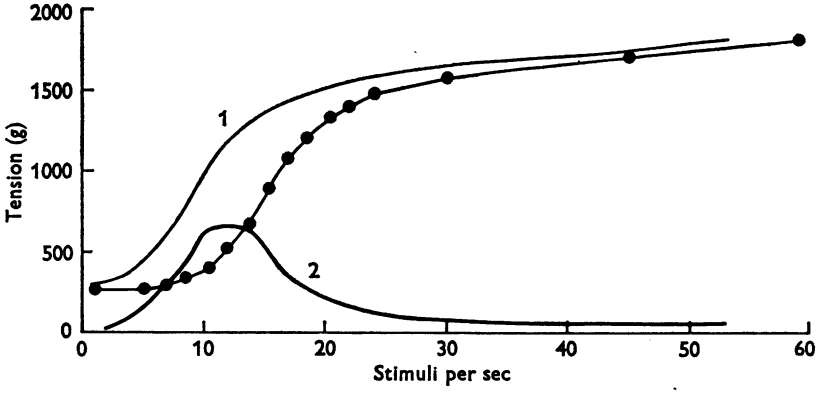

Fig. 6. Curve with observations in filled circles from P. B. C. Matthews (1959) illustrates isometric tension (ordinates) plotted against stimulus frequency to muscle nerve (abscissae) of soleus at initial length determined by tension value below 50 g.

It is assumed (see text) that abscissa also represents natural firing frequencies across the soleus motoneurone pool, the range being from 25 to 5 impulses/sec from centre to edge of pool. An approximate idea of what the experimental curve would have looked like in the absence of recurrent inhibition is obtained by subtracting for all points in the curve 6 impulses/sec. This is the upper curve (1). The lower one (2) is the difference between the two others.

between the two curves, drawn below them (2), shows the range of operation of recurrent inhibition on this assumption. Clearly its contribution to control of muscle tension has been most powerful in the very range in which the soleus motoneurones operate effectively. Furthermore, recurrent inhibition has served to diminish the contribution to muscular tension of the fringe neurones which fire at very slow rates. To this should be added that recurrent inhibition must be mutual, so that, on our results, the rapidly firing motoneurones will inhibit the slowly firing ones far more effectively than the latter can inhibit the fast ones. With an organization of the pool as implied in Fig.6, this means that the fringe neurones will

be further suppressed by their rapidly firing neighbours. Low discharge frequencies can on the whole never be maintained unless the corresponding motoneurones are very powerfully driven from the orthodromic side and thus capable of quickly replacing loss of depolarizing pressure (II). These factors also aid in concentrating the activity in the pool to its centre whose neurones are likely to be firing at rates capable of contributing decisively to muscle tension. Since recurrent inhibition is controlled from supraspinal stations (II; and Haase & Van Der Meulen, 1961) and since its effective range in soleus, according to Fig. 6, is also the critical range for changes of tension, recurrent inhibition should be a very potent instrument of control for tonic muscles. In the two previous papers (I, II) the significance of recurrent control has been discussed in detail from other points of view.

SUMMARY

1. In decerebrate de-efferented cats single-fibre reflexes in response to maintained tetanus of the gastrocnemius nerves have been recorded from appropriate ventral roots and their discharge rate has been suppressed by antidromic tetanization of the rest of the root at constant rate and strength thereby inducing a constant amount of recurrent inhibition.

2. If under such circumstances F_n be the normal frequency of discharge and F_i the inhibited value, both given in impulses per second, it is found that $F_n - F_i =$ constant. This relation was deduced from present knowledge of properties of motoneurones and the experiments were designated as a test of a hypothesis on these lines formulated in equations 1 and 2.

3. With respect to rate of antidromic tetanization at constant strength, it is found that $F_n - F_i$ is directly proportional to it from around 5–10/sec up to a maximum around 30–40 shocks/sec.

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