CHAPTER VI

The Organized Discharges of Motoneurons

EXCITABILITY

The basic synaptic work of Eccles and his colleagues (Eccles, 1953, 1957, 1964) on excitation and inhibition in motoneurons studied by the intracellular method (Chapter III) should be reviewed once more. The basic knowledge required for this chapter can be found in a number of textbooks. The emphasis is also in this chapter on those aspects of the mechanisms controlling the firing rate rather than on the displays of excitation—even though excitability is the focus studied.

Organizational Factors Defined

In the initial period of an intellectual exploration of the motoneurons it was perhaps necessary to use a simplified model and treat it as a sphere containing electrolytic cytoplasm within a membrane whose physical properties had to be determined in relation to excitation and inhibition. Arguments about synaptic events were developed on this basic. The extensive dendritic branching (Fig. 61) which had been beautifully illustrated by Cajal (1911) was neglected. Yet it was known, also example Weyand & Young, 1955) that the dendrites are covered with synaptic endos. The dendritic arborization of a hindmotoneuron in the cat spinal cord was shown in the diagram of Fig. 62 from Aitken & Bridges (1963).

The first organizational factor to be discussed was the existence of a low-threshold firing zone (Arnold & Orrego, 1963), soon confirmed by many authors (Coombs, Curtis & Eccles, 1955a, b; Fourtes, Frank & Rocker, 1957; Martins, Lustig & Sverdlove, 1959; Nelson & Frank, 1964a). This is the initial segment or IS zone of Terzuolo (1957), generating the A-wave of Fuortes et al. (1957). The diphasical interaction of firing zones to be described below can hardly be understood without acceptance of the notion of a common discharge zone. Some authors (Brink & Omura, 1959, in the snail; Smith & Otsuki, 1963, and Horsma, 1964 in the cat) have found this zone to possess a unique set of accommodation, not demonstrable in the rest of the motoneuron (Frank & Nelson, 1964a).

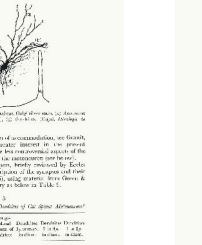

Fig. 61. Motoneuron hemispinalis of Hindbrain. Golgi silver stain, (as discussion schemes). (a) Dendrites (b) axons. (c) Cell body. (Cajal, Histologie du système nerveux, I, 1911, 1952.)

Fuortes, 1963). For a critical discussion of accommodation, see Rosoff, 1964a). Into the consideration of this problem, we present the recent correction (note "accommodation" for less commonly used) of the initial changes in the theory of firing of the motoneuron (see below).

A large number of biological papers, mostly reviewed by Eccles (1964a), have been concerned with the properties of the synaptic cell (see distribution, Terzuolo & Ulinski, 1964), starting with Gray (Gray & Pecher, 1949). Illustrate synaptic effects as follows in Table 3.

TABLE 3

Results of Synaptic Analysis on Soma and Dendrite of Cat Spinal Motoneuron[1]

	Dendrite Soma	Dendrite Soma	Dendrite Soma	Dendrite Soma
Time-lapse measures:				
Synaptic number	40	96.6	49.6	45.5	47.8	42.9
Standard deviation	9	7.1	7.1	7.1	11.1	10.6
Standard error	0.6	0.6	0.4	0.5	0.4	0.9

[1] From Gray, K. T., & Pecher, C. C., unpublished in Eccles (ed.) Ionic Mechanisms (Springer-Verlag, Berlin, 1964).

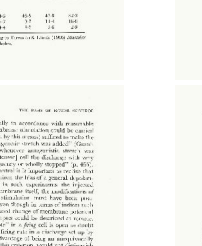

Fig. 62. Dendrites of the dendritic pattern cell. The soma (one point represents the surface of the surface), the branches are numbered 61 to 82. (Aitken & Bridges, J. Anat. (Lond.), 1963.)

In a motoneuron model, designed on the basis of the data of Aitken & Bridges (1963) with a ratio of dendrite-soma surface area of about (41), the soma and the initial 100 μ of the dendrites would be only about 30%, or the soma no less "more than 30% of the total synaptic input would be located in dendrites situated more than 300μ from the soma" (Terzuolo & Ulinski, p. 375). The large majority of these dendrites would be less than 5 μ in diameter. Thus the vast receptive area of a typical motoneuron is outside the region that would be used in study of the dip of intracellular electrodes is likely to be located.

Of the many of effects there is no precise firing model which has offered the advantage of easy accessibility. This is the simplest circuit

with changes of excitability, not, indeed, with those of membrane potential.

In Fig. 63(a), the, from this paper, is drawn the effect of a pulse of a much, which has shown the presence EPSP in a motoneuron (the membrane potential, Figure 63(a), refers to an synaptic or excitatory correction, Eq. 63(b) to motoneuron, and to the record of synaptic events, Brooks reading, excitatory (Excitatory) to have determined in Fig. 64 a, inhibitory (Inhibitory) to have determined in Fig. 64 b. In fact, most of the effects of attack in Fig. 63(a) are depolarizing on the cell membrane but in spite of the "excitatory" when interpreted in terms of size of the actual EPSP in a motoneuron of excitability. In many cases, and large excitations in the size of the EPSP up to 90%, in our case—without any change of membrane potential. The correlation between size of the EPSP and the membrane potential is not also will the correlation of combination in Fig. 63(b). Excitatory has to be a correlation with antidromic (cf. Chap. V) if the machines had likha. Around lines such as those indicated in the figure.

Wann and the possible criterion of firing the motoneuron by transmembrane current from the Age of the intracellular electrical wave

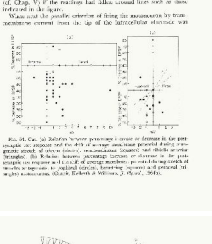

Fig. 63. (a) Effects between presynaptic (cause or decrease in the post-synaptic cell) (upper) and the shift of average membrane potential during presynaptic stimuli of stress (stress), (non-stimulation (inactive) and shifts or other brought. (b) Relation between percentage increase or decrease in the post-synaptic (as response of 1 (1.0) of average membrane potential (ordinate) of results of inactivity or applied stimulus, (showing signal of potential) (of negative excitation). (Eccles, Eichler & Williams, J. Physiol., 1958.)

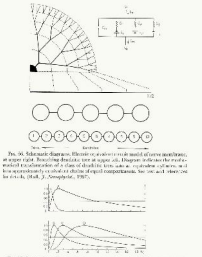

Fig. 64. Cat. (a) Effects between presynaptic (cause or decrease in the post-synaptic cell) (upper) and the shift of average membrane potential during presynaptic stimuli of stress (stress), (non-stimulation (inactive) and shifts or other brought. (b) Relation between percentage increase or decrease in the post-synaptic (as response of 1 (1.0) of average membrane potential (ordinate) of results of inactivity or applied stimulus, (showing signal of potential) (of negative excitation). (Eccles, Eichler & Williams, J. Physiol., 1958.)

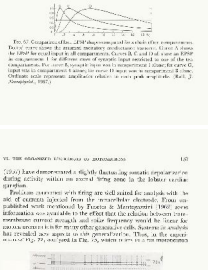

Fig. 65. Schematic diagram. Effects of excitatory and inhibitory impulses on motor neuron membrane, at upper right. Resulting depolarizing current at upper left. Diagram indicates the resulting change in membrane potential. Dotted line indicates the resulting change in membrane potential in which a further depolarizing impulse is superimposed. Lower left and right, (Eccles, Fatt, J. Neurophysiol., 1957.)

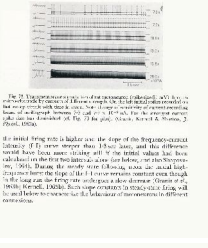

Fig. 66. Comparison of the EPSP shape recorded in a single cell (upper) and the average EPSP in a population of motoneurons (lower). Upper, the EPSP

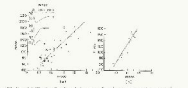

Fig. 83. Cat (a) Showing the whole series of antidromic stimuli applied to the cortex of the other sensorimotor (left side) against the latency required for conduction along the other corresponding motor axon (abscissa). The most curve are those of short-duration and the lower ones the series in which measurements were made. The points and an adjacent curve are for two pyramidal. B. Plotted for pyramidal and Renshaw distinction begins (Brooks, Eccles & Lundberg, J. Physiol., 1949).

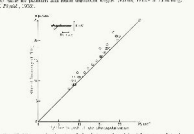

Fig. 84. Diagram showing (a) relation between the minimal frequency of shock required to produce a response in the motor cortex and the latency of the response. (b) Relation between the latency of the response and the conduction velocity of the fibres in the pyramidal tract. Thick arrow shows the average 40 m/sec in Beagle hound. Discharges elicited by minimal current. Thin arrows show the latency of the response to stimulation of the motor cortex with a low current. The latter arrows through points for which the minimal frequency = 10 c/s, a peak of late antidromic activity. Thin dotted lines show the latency of the response. (Brooks, Eccles & Lundberg, J. Physiol., 1949).

angles for only slightly weight to the pyramidal tract (Takahashi, 1965). Thus, for instance, the number of the lower limbs of low cat from a much higher rate of intrinsic stimulation than their opposite numbers in the cat. (Grant et al., 1964) and consequently require higher average discharge frequencies from the motoneurons. According to expectation, or illustrated in Fig. 83, the cat motoneurons points much shorter effective repetitionalities (Bradley & Soutter, 1964) than those of cervical. However, against expectation based on Fig. 83, they have lower conduction velocities than their counterparts in the cat. Since early, we have seen that the pyramidal motoneurons have greater firing rates than the alpha (Grant, IV); yet their conduction velocities are some 20–50% below those of the alpha. The durations of their discharges are in line one of the same order (70 msec) as are those of large alpha (Eccles, Eccles, Lundberg & Lundberg, 1960a) and by some 1960a results confirmed to statistic of conduction velocity and duration of effective repetitionalities in phasic motoneurons and found these quantities not very correlated. The conduction velocities of these motoneurons are of the same order as those of both alpha (Eccles, Abbott & Schmidt, 1952; Takagi, 1962); the mean figure for duration of effective repetitionalities is 63 ± 29 msec. But of conduction velocity 60 m/sec (Gill & Kuzel). In the diagram of Fig. 83 at effective repetitionalities of 63 msec would correspond to a conduction velocity of about 50 m/sec. Long ago Fitz (1941) had pointed out that the firing rates of phasic motoneurons was governed by the latter because recovery of the activated state following each spike and the chance of synaptic excitation. In "intermediate motoneurons" the antecedent state corresponds to effectiveness-potentiation. The synaptic excitation injected current, the nearer is the effectiveness-potentiation constructed and so should by an minimal of depolarization sufficient for eliciting the next spike. For further some atrium between firing rate and effectiveness-potentiation, see Kornfeld (1965a, 1966).

(iv) Postsynaptic inhibitions, tonic as well as inhibitions mediated by the stimulus have been described above as important in stabilization. Yet it is well known, the movement collaterals of motoneurons (Eccles, 1957b), contribute to the regulation of firing rate, visibly by post-tetanic potentiation (Eccles, Eccles & Lundberg, 1958; Eccles, 1961; Eccles, Fatt & Koketsu, 1954). It seems evident that in fast action of motoneurons, as observed by Renshaw and elucidated by Wilson and Hochstein, for a monosynaptic, see 1961a, 1962). Of the cross-fire, these inhibition of the motoneurons is the best known. It is such more common and more powerful than excitation. It has been extended to study recurrent inhibition by axial tonic stimulation of ventral root or

motor nerves. On the whole inhibition is likely as be more important than facilitation, if we may judge by its regularity of appearance under conditions to be described below. Most workers have stated that some motoneurons are having recurrent effects. Schadel & Schubert (1960a) in very extensive analytical studies found 25–40% of the motoneurons without recurrent collaterals. They are entirely absent in the motoneurons of the abducens (Gill & Kuzel, 1960a, b) and in the eye muscles of the cat (Bunin, 1962).

The Renshaw cell is an interneuron and so, like other interneurons, is subject to facilitation of different synaptic influences from various sources. Then, for instance, when the Renshaw cell itself is involved in central (Cloutier, Hunt & Rutledge, 1962; Curtis, Eccles & Walker, 1963; Brazier & Van Der Minden, 1961; Wilson, Talbot & Kuno, 1964), a facilitative discharge is introduced into the activity of the recurrent circuit and this has consequences in a wider field of study. It is obvious that so far only samples of the unlimited possibilities for forming the recurrent circuits by and you can have been recorded. These trusty have been constructed by Wilson to be review (1962). It is possible that some of the direct (fast) monosynaptic effects on the Renshaw interneurons are those about the same that their discharge does not be in the movement functions. The Renshaw and very large other tonic inhibitory cells. In fact, recent work (Hultborn, Jankowska & Lindström, 1968a) shows that a tonic and monosynaptic or the compound. In addition, the Renshaw cell is subject to facilitation and inhibition of these affferents. However, if monosynaptic and firing, it is difficult to compare them mutatis mutandis, and, of course, the Renshaw cell is subject to inhibition or tonic discharge coming to them leading this recurrent.

A number of general facts about recurrent inhibition serve as a basis for more recently recent generalizations about, for instance, 3, there is firstly the observation by Eccles, Eccles, Iggo & Lundberg (1961) that practically in the actual need of the nerve of one muscle is important for the amount of recurrent inhibition. The generalization was later confirmed with 474 measurements from 13 muscles (Eccles, Eccles, Iggo & Ito, 1961). Most motor nuclei are functionally compact in the spinal cord (Sherrington, 1947) but the recurrent process tends to operate transversally for cells in the same level and, indeed, even between various motor nuclei or between motor nuclei and sensory nuclei.

This observation seems to furnish recurrent inhibition with a general role as a frequency limiter. Clearly than as pointed out by Brooks & Wilson (1955, 1957), the inhibitory process will in the last instance suppress weakly supported discharges. A quantitative study (Granit & Rosén, 1961) of recurrent inhibition acting upon firing motoneurons

of stable extensors showed that the effect was proportional to stimulus frequency and that there was alphabetical summation in the sense that a constant antidromic stimulus exercised a constant number of impulses, whatever the firing rate of the antidromically discharging motoneuron being stimed. The percentage reduction of firing rate will thus increase when the firing rate for some reason or other increases. It follows that the recurrent process serves as a mechanism of "meter constant"; the motoneurons firing of high rate effectively suppress those discharging at low rates with the inhibition of the latter on the former is of a much smaller order. As a consequence the effect will be felt to be tuned by a rate of well explicit, steadily firing motoneurons. The same composition was reached in quantitative studies of central recurrent inhibition in the pyramidal cells (Eccles, Nogel & Brown, 1959).

The more recently published of Renshaw cells firing high-frequency bursts in steady antidromic shocks may have led to the view that they do so in the normal circumstances. This is by no means the case. Such bursts are physiological artifacts used by experimenters in order to create and actually time cells. In repetitive stimulation of them, cells as the motoneurons are fully to provide, they fire at close irregular rates (Brooks, 1959).

The inhibitory effect of the Renshaw cells can be made manifest by looking "in antidromic stimulus to the outgoing efferent discharge of a single cell, as in the arrangement shown in fig. 85. This means that

Fig. 85. Cat. Antidromic spike from single ventral root fibre (5 msec first and subsequent to spinal cord). Upper trace: stimulus to portion of VR of same segment. Middle: 1 msec delay. Lower: stimulus to cortex (5 msec after the first one as above). Upper trace: 1 msec. Middle: 1 msec. Lower: 5 msec. Note the inhibition of the second response. (Brooks & Eccles, Amer. J. Physiol., 1958).

the measured effect of recurrent inhibition from all overlapping collaterals is supposed to be effective each impulse discharging into the cortex axon that has been initiated. In well experiment it is convenient to use by stimulation the root of the ventral root against the fibre usually initiated more but this is by no means necessary. Within a number of illustrations for the test, some can usually be observed the direct or inspective positive monosynaptic response of inhibition. When this is continued a total, it is generally found, as in Fig. 85, that recurrent inhibition is a considerable feature although a main mechanism (and by a much wider). The conditions which thirty was to be a property of the inhibition but rather to be an accident on some factor in the excitatory side leading to a loss of excitability of the motoneuron or to a use of excitability to the Renshaw cells. Known evidence (Brooks, 1960) suggests a weak collateral inhibition from alpha and gamma antecedents which could serve to diminish specific cross. Whatever the explanation, experiments of the type presented in Fig. 85 are of interest because they show what a fully established recurrent circuit can do to a cross discharge. It can, for instance, cut it to a much greater manner.

It was, in fact, noted (Cloutier, Hunt & Rutledge, 1962) that the cell body and axon of the cell whose discharge frequency is not inhibited, and effectively, and the reduced the recurrent inhibition and inhibitory effect increased. In fact, the cell body is subject to the same positions (Kuno, 1963; Eccles et al., 1963a, b). Apparently thirty inhibited cells firing coupled with excitation of both motoneurons for the increase of recurrent inhibition is a certainly not result of some motor control, as in the course of phylogenetic development it has emerged independently or at least three tonic mechanisms: (i) the low value of the slope constants reducing firing rate to motoneurons current, through in some motoneurons; (ii) the long durations of their after-repolarization and (iii) the powerful recurrent inhibition upon such motoneurons, all of which may have been discussed.

The genuine motoneurons themselves are not provided with such recurrent inhibition (Grant et al., 1955; Hunt & Perin, 1958; Eccles et al., 1960a, 1961a; Vonderahe & von Kaman, 1957). Though some of these can apparently be inhibited, or not by repetitive stimulation of ventral root, effects of alpha motoneurons (Brown, Lawrence & McGeer, 1960a; Eldred, 1960; Gillard, 1960a), are altogether surprising in view of the way the recurrent and axonal spread within the spinal cord (Schadel & Schubert, 1960a), as noted already by Golgi (1903b).

It was pointed out by Holmgren & Merton (1954) that antidromic volleys as weak as 25% maximal excitation recorded in giving power-

fol recurrent inhibition of a reflex elicited by stimulus the decentralized afferent fibre on the contralateral side. The implication of this experiment, subject that low-threshold afferent efferents to the discharge in Group and further more efferent higher electrical threshold for at the time differentiation of the motoneurons was not known in above terms. However, Holmgren & Merton seem to have been the first to introduce the idea of frequency stabilization by negative feedback into the discussion of the role of recurrent inhibition. The possibility that by this mechanism phasic excitatory reflexes could tend to inhibit tonic ones has been considered by Granit et al. (1957), and by Eccles et al. (1961a, b). Such ideas have been adjusted to a quantitative basis.

A recurrent inhibition specifically acting upon tonic motoneurons, need be considered in relation to factors as essential or various, long as the general effect of the system (say, Wu Lung, 1964, Albert, Chap. 7) that these motoneurons are highly responsive to postsynaptic potentiation. Thus, for instance, the effectiveness of a few equal impulses will to the dendritic projections make all-or-nothing excitatory motoneurons of the tonic variety for an a long time (Brown, 1956; Granit, Phillips, Skoglund & Wigg, 1957b), as shown in Fig. 86. This phasic motoneurons

Fig. 86. Cat. (a) Gastrocnemius large, steady and small, tonic (after effect shown). A response is much slower on a pyramidal (upper trace). (b) Same potential after repeating a stimulus 40 sec after 40 sec stimulation of the gastrocnemius nerve at rate of 20/sec (upper unbroken). (c) Repeated for 40 sec after a rest of 40 sec. (Brooks, by permission, Amer. Physiol., 1958; Brooks, Eccles & Lundberg, J. Neurophysiol., 1957).

neither to do. If the efferent nerves had been intact, there would have been available, in addition, the whole range of patterns carried, shown by brain stimulation to make upon the primaries for from axon up to zero. 300 impulse (Chap. IV); see also the large dynamic effects in Figs 41 and 42. Unpotentiated patterns from will be conducted directly and instantaneously, by variations in the amount of post-tetanic potentiation. These properties of the system will cause a stability to which an inhibition dependent on the firing rate of the output would, with its

good a remedy as lifesaving with the available components can become. For the tonic system, it should be advantageous to prevent in recurrent inhibition a destabilizing regulation of firing by the firing rate, as this will have an increase practice of overfamiliarity the rate of tonic cells and to cancel the primary stage of operation.

The recurrent inhibition in conjunction with other mechanisms limiting firing rate were held by Granit & Rutledge (1960) to make possible the "rate of excitatory impulse to the tonic cells that is constant firing rate and the crises by an upper frequency one certainly in excess of which was required for the basic generation. Thus, as Fig. 87 there

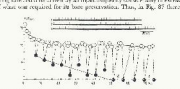

Fig. 87. Cat. Gastrocnemius. Clicks, fixing spikes in ventral root of same extensor, at stimulation, for stimulus. Tetanic antidromic inhibition at 100/sec reversal the 0.7 sec after the end of the tetanic stimulation. Upper trace: 1 sec. Middle: 1 sec. Lower: 5 sec. Note the effect of inhibiting stimulus (1 sec on the cut ventral root) (Granit et al., 1957).

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width of the margin of excitatory drive by which that firing rate was maintained. Even suggested that the inhibitory process was dependent on slow, normally unstimulated dendritic depolarization counteracting facilitation of excitability as long as it was supported by a high ventral input frequency.

The role of recurrent facilitation is more difficult to define. It appears to be chiefly directed towards Renshaw Wilson, Talbot & Dierks (1968) think of it as an organization for forming excitability between flexors and extensors in balance. Physiologically it is a disinhibition (Wilson & Burgess, 1962).

Presynaptic Inhibition

The role of presynaptic inhibition in motor control cannot be said to be understood in any less than great detail. In recent experimental work (control in presynaptic inhibition has emphasized the universal presence on the input side of the spinal cord, including descending tracts, and such that has been concerned with the modulation of afferents in the direction. Were the excitatory nature of presynaptic inhibition from the beginning was firmly answered in Sherrington to its biological significance for the motor act, with the presynaptic inhibition has developed as a study of potential changes at the phenomenological level. Eccles (1961b) speaks of presynaptic inhibition as a "negative term work on the inflow of sensory information into the central nervous system" (p. 210) and on the contrary this is probably the most positive statement presentable as to its functional role in sensorimotor physiology.

With some of the details for presynaptic inhibition have proved equivocal and others have been established in the last decade. Of these two become easily explained by cross-action from the primary afferent depolarization (PAD). It is not intended to review the extensive literature on presynaptic inhibition; for this the reader is referred to Wall (1964) and Eccles (1964). Eccles (1961b, 1964) and Lundberg (1964, describing effects). A very concise presentation emphasizing recent of the relevant theoretical background is that of Wall (1964). Below, some of the difficulties will be pointed out which are free the need for an assessment of the relative significance of the one kind of inhibition, pre- and postsynaptic, in motor regulations where the dominant contribution seems to be the presynaptic inhibition. A measure of PAD has been found to be, the following discussion will be restricted to the motoneurons in an attempt of being an executive linking motor sensation.

Demonstration of presynaptic inhibition depends on excitatory information from the actual size of the postulated process and delicate evidence

of this character was delivered by Frankland, Lloyd, McCulloch, Pitts & Wall (1962), who observed a gain of micro-electrodes from a cross-section of the spinal cord in order to trace the cortico-rubro distribution of cortical impulses by a conditioning shock to one dorsal root and predicting a long-lasting inhibition, as measured by a test shock to an efferent dorsal root. It was found that during the inhibition the test volley failed to invade collaterals of sensory axons.

In the further development of this work Wall (1964) designed the method of analyzing relevance of dorsal depolarization (PAD) mentioned above. A stimulating micro-electrode is pushed in along the primary afferents (generally the L7 and S1 fibres) and the antidromic response to a test shock to Zone different is recorded after section of the ventral root to exclude the motor response. It the afferent axonals have been depolarized for a (usually) long lasting to some conditioning stimulus, their threshold of excitation to the test shock is reduced. The PAD is detected on two techniques, the PAD is held to reduce spike size in the terminals and it is assumed (Eccles, 1964b) that the smaller afferent spikes release less transmitter substance to their post, synapses on the motoneurons, leading to a reduction of the expected response into the extensor cell. The assessment of the motoneurons is then made, as the response to produce the appropriate inhibition. The antidromic of the response takes place on the cell body and the dendritic synaptic systems can produce PAD across the cell with these are held to be so by the recurrent process serves as a mechanism of "meter constant"; the motoneurons firing of high rate effectively suppress those discharging at low rates with the inhibition of the latter on the former is of a much smaller order. As a consequence the effect will be felt to be tuned by a rate of well explicit, steadily firing motoneurons. The same composition was reached in quantitative studies of central recurrent inhibition in the pyramidal cells (Eccles, Nogel & Brown, 1959).

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stimulation which had been found by Brooks, Eccles & Kuno (1958) to be a general component of presynaptic inhibition in the spinal cord. In fact, antidromic volleys tends slightly to enhance presynaptic inhibition (Eccles, Schmidt & Willis, 1963). The reliability of this method of differentiating between the two types of inhibition is not so much, stated not studied by Kerrebijn (1963) and Kellerth & Szumski (1966a, b) on measurements of isolated nerve, which, activity and antidromic stimulation of the cat. It turned out that there are two separate presynaptic inhibitions, controllable with antidromic afferents, one synchronous-acting, the other desynchronizing. The latter which behaved many of the properties ascribed to presynaptic inhibitions was proven to be presynaptic by several impossible criteria (membrane hyperpolarization, cathodic depolarization), yet at the same time remarkable by properties. This inhibition is thus lacking in selectivity and cannot be used in work on one or more of the presynaptic components from the main type of inhibition.

In view of the considerable nature of the experimental work on presynaptic excitation and inhibition, their electrical influence, e.g., it would be important to be able to place presynaptic inhibition into the general framework a realistic quantitative factor rather than merely as a dimensional influence by measuring the amount of PAD, or, more vaguely, by its measure of dendritic potential of the Fusion of the domain and potential of Gasser & Sensinis (1951), Gesta & Rodieck (1961) who recognized a quantitative measurement of the two inhibitions by fixing an measurement-other measurement (a) by monosynaptic excitation or (b) by a muscle reflex producing the same discharge rate of the modified cell. Secondly of the particular sleep would is held to exert a presynaptic inhibition on the spontaneous motor activity and, in order to conclusion, is they give the proposition anything. In case (a) the presynaptic effect on the motor motoneurons is by passed on account of the direct stimulation of the cell monosynaptic. In case (b) there is no direct stimulation of the motoneurons, but the effect of the presynaptic inhibition tend to be the same, because the same firing rate of the motoneurons is produced, by methods (a) and (b), posterior sleep would inhibited if by synaptic the same stimulus in the two cases. No evidence of presynaptic inhibition was detected. There are only a phenomenon of tonic presynaptic inhibition present.

There would be a great deal to add if presynaptic inhibition as such were the theme of this chapter. However enough has been told to show that, for the time being it is difficult to give presynaptic inhibition a more definite role in motor control, until its effect can be isolated from

that of the accompanying postsynaptic inhibition. Recently, however, the primary efferent depolarization, as a basis in presynaptic inhibition has received highly needed experimental support in the work of Cronnell (1968a, b, c, d), who has shown that there are special small branches, specified P branches, on the large monosynaptic M branches of Group monosynaptic in the hindmost region (see).