A method for simulating the reflex output of a motoneuron pool

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An analysis of the reflex output of a motoneuron pool in response to a Ia-afferent input is presented. The analysis is based upon a model of the motoneuron pool which includes the subthreshold behavior of motoneurons (integration of synaptic inputs) and the statistical distribution of the motoneurons according to their resting conductance. The latter feature allows for the orderly recruitment of the motoneurons in the order of low resting conductance to high resting conductance. The number of active motoneurons (i.e. the excitation level) is determined by the balance of the excitatory and inhibitory conductances acting on the pool. The reflex output in response to a Ia-EPSP is computed at various excitation levels and with different amounts of presynaptic inhibition. The reflex output is the same for a given excitation level, regardless of the mixture of excitatory and inhibitory postsynaptic conductances used to produce that excitation level. In contrast, presynaptic inhibition markedly affects the relation between reflex output and excitation level.

Introduction

Since the first intracellular recordings from spinal motoneurons (Brock et al., 1952), much has been learned about the synaptic circuits impinging on these final common pathways for motor output. Yet, such recordings have only rarely been possible in normal, behaving animals and only then in quiet states such as sleep (Glenn and Dement, 1981). To study reflex function in active animals and in human studies of both normal and pathological states, we must still rely on reflex testing, generally of whole motor pools with electrical or mechanical stimuli.

The results of these studies are often interpreted qualitatively in terms of a 'circuit diagram' derived from intracellular recording studies, without much attempt to validate the predictions against some model of the neural network. One reason

for this lack of validation is that single motoneurons have a complex mixture of intrinsic and synaptic currents that is far from being fully understood (Burke and Rudomin, 1977). In addition, to model the response of a motor pool, the way in which these currents vary over the entire population of motoneurons must be known, an even more formidable task.

What we would like to propose in this paper is a simple model of a motor pool, but one which catches enough of the flavor of the biological system that it may be useful to a number of individuals engaged in reflex studies in animals or humans. The model can be programmed on a sufficiently powerful microcomputer and even allows for some results of interest to be derived analytically. By way of example, we will give a few computer simulations and analytical results relevant to recent studies on the modulation of the H-reflex during locomotion in normal human subjects (Capaday and Stein, 1986, 1987).

The H-reflex is the electrical analog of the tendon jerk, and is routinely used for reflex testing. The H-reflex often changes with the excitation level of the motor pool, as assessed experimentally from the mean level of the rectified surface EMG. We will concentrate on the mechanisms that may lead to a change in the slope and y-intercept of the H-reflex vs EMG relation. Both these changes occur in going from a standing posture to walking and running (Capaday and Stein, 1986, 1987).

We also show that a change in the slope of the line relating the reflex output to the level of EMG represents a change in the gain (in the formal sense of the output divided by the input) of the pathway from the Ia afferents to the α-motoneurons (central gain). The change in gain as well as threshold is counter to the suggestion made by several authors that only the threshold of this reflex changes (Feldman and Orlovsky, 1972; Houk, 1976).

Description of the model

The H-reflex represents the synchronous discharge of α-motoneurons to Ia-EPSPs. Only the interaction of the monosynaptic EPSP current with the combination of excitatory and inhibitory currents acting across the motoneuron membrane is considered here. Either the combination of these currents will depolarize the motoneuron to threshold, which means that the motoneuron will contribute to the reflex output, or the membrane potential will remain subthreshold and the neuron will not contribute to the reflex output.

For this purpose therefore, a classical, subthreshold model of a motoneuron is sufficient as shown in Fig. 1. It includes a resting conductance (G_r), excitatory (G_e) and inhibitory (G_i) conductances which represent in effect descending control of the motoneurons, the monosynaptic EPSP conductance (G_{epsp}), the fast potassium current (G_{K_f}) which repolarizes the motoneuron after a spike (Barrett et al., 1980), and finally the afterhyperpolarization conductance (G_{ahp}) which regulates the rate of motoneuron firing (Granit, 1972). For simplicity, we will ignore subthreshold membrane non-linearities and the cable properties of the dendritic tree. These
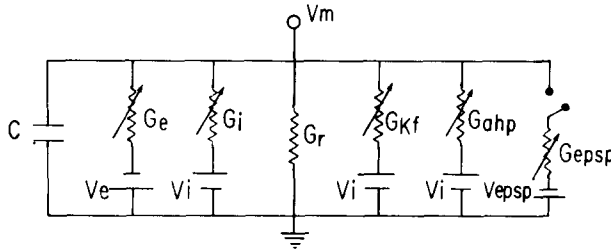
Fig. 1. An electrical model of the subthreshold behavior of α-motoneurons. The excitatory conductance (G_e) depolarizes the motoneuron toward threshold, the inhibitory conductance (G_i) hyperpolarizes the motoneuron and the resting conductance (G_r) determines the resting membrane potential. The fast potassium current which flows through the conductance (G_{K_f}) serves to repolarize the motoneuron after an action potential and the conductance of the afterhyperpolarization (G_{ahp}) limits the firing rate of the motoneuron. Closing the switch produces an EPSP in the motoneuron. More details are given in the text.

simplifications are justified if we consider that, in the end, it is the integration of synaptic currents at the axon hillock that matters.

Increasing the G_e conductance will depolarize the motoneuron to threshold, an increase in the G_i conductance will hyperpolarize the motoneuron, and closing the switch shown in Fig. 1 will produce an EPSP in the motoneuron. The conductance G_{K_f} and G_{ahp} are only activated if the membrane potential (V) reaches threshold (voltage dependence) and are time dependent, with an approximately exponential decay (Barrett et al., 1980). The total current (I_{tot}) flowing across the membrane is given by:

$$I_{tot} = C\, dV/dt + G_e(V - V_e) + G_i(V - V_i) + G_{epsp}(V - V_{epsp}) + G_{K_f}(V - V_{K_f})$$

$$+ G_{ahp}(V - V_{ahp}) + G_r V = 0 \qquad (1)$$

where: $C\, dV/dt$ is the capacitative current, V_e, V_i, V_{epsp}, V_{K_f}, and V_{ahp} are the reversal potentials of the respective currents. Notice that all changes of membrane potential are referenced with respect to a resting potential of zero. Once activated the conductances G_{K_f} and G_{ahp} decay exponentially with respective time constants obtained from the literature on cat motoneurons (Barrett et al., 1980; Burke and Rudomin, 1977). The size of the EPSP will depend on the EPSP current and the values of all the conductances which are active:

$$EPSP = \frac{G_{epsp}(V_{epsp} - V)}{G_r + G_e + G_i + G_{K_f} + G_{ahp} + G_{epsp}} \qquad (2)$$

The range of values for the EPSP conductance was obtained from Eccles's classic book (Eccles, 1964). The range of values of the various conductances used in the computations as well as those of other parameters are listed in Table I. The equilibrium potential of the membrane (V_m) will depend on the sum of all currents flowing across the membrane and the V of all the active conductances. If we

TABLE I

VALUES OF VARIOUS PARAMETERS USED IN THE COMPUTATIONS DESCRIBED IN THE TEST

The mean value of the resting conductance (G_r) used was 0.69 μS. The actual statistical distribution of the resting conductances is given by Eqn. 6, where the parameter a in that equation is equal to the minimum value of G_r given above.

Parameter	Value
G_{K_f}	1–2 μS
G_{ahp}	1–2 μS
G_{epsp}	0.015–0.06 μS
Time constant of G_{K_f}	3–5 ms
Time constant of G_{ahp}	50–70 ms
Threshold (V_t)	10–12 mV
Membrane capacitance	4.2 nF
Mean value of G_r	0.69 μS
Minimum value of G_r	0.17 μS

take the reversal potential of all the inhibitory currents to be approximately the same, then:

$$V_m = \frac{G_e V_e + V_i \left(G_i + G_{K_f} + G_{ahp} \right)}{G_r + G_e + G_i + G_{K_f} + G_{ahp}} \tag{3}$$

Because of the membrane capacitance (C) the membrane potential will not change instantaneously to its steady-state value but will be a function of time given by the solution to the differential Eqn. 1 which can be rewritten as:

$$\tau \frac{dV}{dt} = (V_m - V) \tag{4}$$

where: $\tau = C/G_{tot}$ is the membrane time constant ($G_{tot} = G_r + G_e + G_i + G_{K_f} + G_{ahp} + G_{epsp}$) and V_m is given by Eqn. 3.

The solution of Eqn. 4 is composed of the sum of several exponential processes, but it cannot be obtained analytically because G_{tot} and hence τ are changing with time. It can, however, be evaluated numerically for example by a fourth order Runge-Kutta type algorithm (Hornbeck, 1975). Once a motoneuron is active in the model it discharges repetitively as long as the excitatory drive to it is maintained (slow processes of adaptation are ignored). An EPSP induced by electrical stimulation of the muscle nerve can occur at any point along the membrane potential trajectory. If the sum of the EPSP and the value of the membrane potential at the time the EPSP occurs is large enough to reach threshold (V_t) the neuron will discharge an action potential and contribute to the reflex output. Unless the EPSP is very large the motoneuron will not fire an action potential at all times during its membrane potential trajectory.

A novel aspect of the present approach is the development of simple methods for determining the fraction of motoneurons in a pool which respond to each stimulus. To determine this fraction two points along the depolarization trajectory are
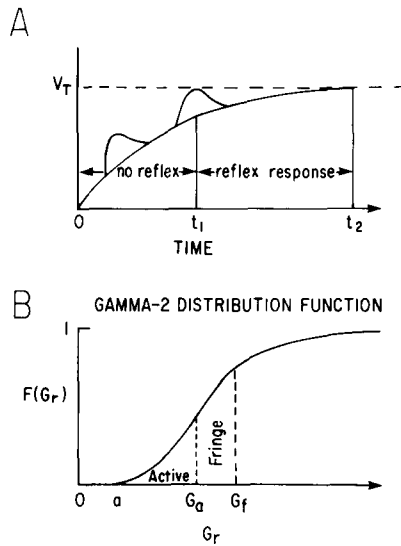
Fig. 2. A: schematic illustration of a motoneuron's membrane potential trajectory towards threshold showing how its probability of firing in response to an EPSP is calculated. In the interval between time zero and t_1 the EPSP will not bring the membrane potential to threshold, in the interval between t_1 and t_2 the EPSP will make the motoneuron fire. The firing probability is equal to the proportion of the time t_2 during which the EPSP will make the motoneuron fire. B: an example of the Gamma-2 distribution function with a starting value of a. Neurons with a resting conductance $G_r \le G_a$ are active (i.e. firing repetitively). Those with a resting conductance $G_a < G_r \le G_f$ are not active but will be reflexly recruited by the Ia-EPSP (i.e. they are in the subliminal fringe).

important (Fig. 2A). First, the time (t_1) when the sum of the membrane potential and the EPSP are just large enough to reach threshold. Second, the time (t_2) when the depolarization of the membrane reaches the threshold on its own. Therefore, the proportion of the time that the motoneuron can be brought to threshold by the EPSP, or equivalently the firing probability is given by:

$$P = \frac{t_2 - t_1}{t_2} \tag{5}$$

If the EPSP is large enough to bring the membrane potential to threshold at its most hyperpolarized point (i.e. just after the motoneuron fired) then $t_1 = 0$ and $P = 1$; the motoneuron will always fire in response to such an EPSP. If there is no EPSP, then $t_1 = t_2$ and $P = 0$. For intermediate values of the EPSP the probability of firing in response to that EPSP will be between 0 and 1. Thus, among the motoneurons that are active (i.e. firing repetitively) only a proportion of these (n_a), given by the product of P and the fraction of active motoneurons, will be discharged.

Not all motoneurons are equally excitable; in fact, Gustaffsson and Pinter (1985) have reported an approximately eleven-fold difference in the intrinsic excitability (rheobase current/membrane capacitance) of the triceps surae motoneurons. This can be accounted for by an asymmetric distribution of resting membrane conduc-

tances (Gustaffsson and Pinter, 1985). The distribution is skewed such that a large proportion of the motoneurons have a low resting conductance (high resistance) and a relatively small proportion have a high resting conductance (low resistance).

Another novel aspect of our approach is inclusion of a simple probability density function consistent with the skewed experimental observations:

$$f_2(G_r) = \frac{2}{b}(G_r - a)\exp\left[-(G_r - a)^2/b\right] \tag{6}$$

This function is a gamma distribution of order 2 (Peebles, 1982). Note that Eqn. 6 applies for $G_r > a$, the minimum resting conductance; the value of b is determined from the mean value of the distribution which is $a + 2b$. This skewed statistical distribution of the motoneurons according to their resting conductance allows for their orderly recruitment in the direction of low resting conductance to high resting conductance.

The next issue to address is the distribution of the excitatory, inhibitory and EPSP conductances across the motoneuron pool. Based on the theoretical analysis of Stein and Bertoldi the EPSP conductance may be related to the square root of the motoneuron's resting membrane resistance (Stein and Bertoldi, 1981). Very little is known about the distribution of descending excitatory and inhibitory inputs to the motoneurons. One of the few findings on this issue is that the size of the corticomotoneuronal EPSP is correlated to that of the Ia-EPSP in the same motoneuron (Clough et al., 1968). In the absence of exact information on the relations between these variables and the motoneuron's resting conductance we made the assumption that the parameters G_e, G_i, and G_{epsp} are independent of the resting conductance (G_r). In other words, in the absence of detailed information, we chose the null hypothesis that all motoneurons in the pool are excited or inhibited to the same extent. These simplifying assumptions also allow for the derivation of some simple analytical relations to be presented below.

Several examples where the conductances G_e, G_i, and G_{epsp} were made functions of G_r were also simulated. For instance, we simulated the situation where G_e is inversely related to the resting conductance G_r, G_i is directly related to G_r, and G_{epsp} is related to the square root of $1/G_r$ (i.e. the square root of the resting membrane resistance). These assumptions did not qualitatively affect the results, although small quantitative differences were found. Therefore, for simplicity, the presentation given below is based on the null hypothesis as discussed above.

The motoneurons which are active are those whose steady-state membrane potential is equal to or greater than the threshold (V_t). Using Eqn. 3 this requires that the resting conductances G_r be less than or equal to a value G_a given by:

$$G_a = \left[G_e(V_e - V_t) + G_i(V_i - V_t)\right]/V_t \tag{7}$$

A fraction of the population will not be excited, if their $V_m < V_t -$ EPSP. This requires that the resting conductance be greater than a value G_f where:

$$G_f = \left[G_e(V_e - V_t) + G_i(V_i - V_t) + G_{epsp}(V_{epsp} - V_t)\right]/V_t \tag{8}$$

Thus, the motoneuron pool can be divided into 3 groups (Fig. 2B): (a) a fraction with $G_r \leq G_a$ will have $V_m \geq V_t$, and therefore will fire repetitively and be activated

by the EPSP on a proportion of the trials given by Eqn. 5, where t_1 and t_2 are obtained from the solution to Eqns. 2 and 4; (b) a fraction with $G_a < G_r \leq G_f$ will have $V_t - \text{EPSP} \leq V_m < V_t$. They will not be firing tonically but are part of the subliminal fringe which will be discharged by the EPSP; (c) and finally a fraction with $G_r > G_f$ will be subthreshold for both tonic firing and activation by the EPSP. In terms of the probability distribution function $F_2(G_r)$, which is the integral of the density function, the proportion of active motoneurons is $1 - F_2(G_a)$, and the proportion in the fringe is given by $F_2(G_a) - F_2(G_f)$.

The computations of the equations described above were done on a PDP 11/34 computer by a program written in the DESCTOP system simulation language (Korn, 1985). The simulation package is also available for IBM compatible PCs. The program can be obtained from the authors on request.

Results

Some simulated results using the model described above and suitable values obtained from the literature for cat motoneurons are shown in Fig. 3. At rest (no external excitatory or inhibitory inputs) a small reflex response is elicited. As the level of excitatory input is increased, the reflex response is increased even before any overt activity of the motoneurons is observed, reminiscent of the well known Jendrassik manoeuvre. Also shown in Fig. 3 are the effects of adding a constant postsynaptic inhibition and increasing the amount of presynaptic inhibition (decreasing the G_{epsp}). The postsynaptic inhibition shifts the curve to the right, without much change in its form, so that more excitation is required to obtain the same reflex output, whereas the presynaptic inhibition reduces the reflex at all levels of the excitatory conductance.
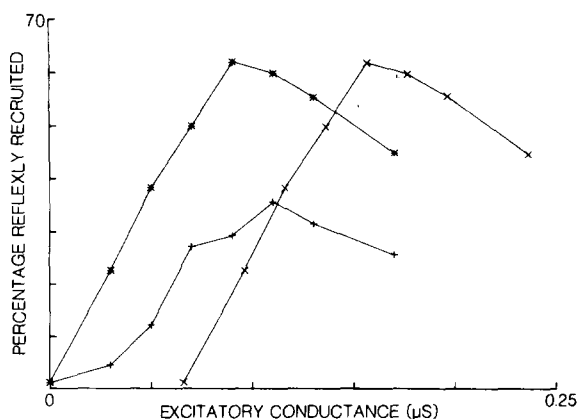

Fig. 3. Relation between the excitatory conductance G_e and the percentage of reflexly recruited motoneurons. The EPSP conductance was 0.04 μS for the curves marked with the symbols ($\times$) and ($*$), and 0.02 μS for the curve marked with ($+$). The rightmost curve represents the reflex response when the inhibitory conductance G_i was increased to 0.2 μS. The most excitable motoneurons begin to discharge when G_e is equal to 0.03 μS in the absence of postsynaptic inhibition and 0.09 μS in its presence.
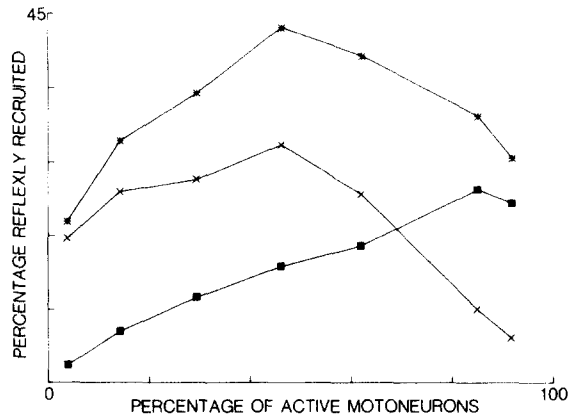
Fig. 4. Relation between the percentage of active motoneurons (the excitation level of the motoneuron pool) and the percentage of reflexly recruited motoneurons. The topmost curve represents the total proportion of motoneurons reflexly recruited and is the sum of the two curves below it. The curve marked with the symbol ($\times$) represents the contribution of motoneurons in the subliminal fringe, that marked with ($*$) represents the contribution of the motoneurons that are both active and reflexly recruited by the Ia-EPSP ($G_{epsp} \approx 0.04 \ \mu$S).

Notice also that, as the level of excitatory input increases, the number of reflexly recruited motoneurons increases to a maximum and then begins to decrease. The reason for this is shown in Fig. 4 where the number of reflexly recruited motoneurons is plotted against the number of active motoneurons. The number of active motoneurons is used as the x-axis because in many experimental situations the level of excitatory drive is not known and one can only relate the reflex to the mean level of EMG activity, which is related to the number of active motoneurons. When there is activity in the motoneuron pool the total number of reflexly recruited motoneurons is the sum of the motoneurons which are reflexly recruited and active (n_a), as well as those that are in the subliminal fringe (n_f) (i.e. inactive but reflexly recruited). It can be seen in Fig. 4 that the number of motoneurons that are active and reflexly recruited increases over most of the excitation level of the pool, whereas the number in the subliminal fringe increases over the first fifty percent of the excitation level and decreases thereafter. Since the number of motoneurons in the fringe decreases faster than the number that are active and reflexly recruited increases, the total number decreases. Two factors contribute to the decrease of the subliminal fringe. First, as the excitation level increases, fewer motoneurons are available to be recruited and second, the EPSP is increasingly shunted by the excitatory conductance and the large resting conductance of the remaining motoneurons.

When the EPSP is small the reflex output increases over a much greater range of the motoneuron pool excitation level than when it is large (Fig. 5). This is due to the way the subliminal fringe motoneurons are recruited by the EPSP. As the size of the EPSP increases, the contribution of the subliminal fringe motoneurons reaches a maximum at a relatively lower excitation level than when the EPSP is smaller (Fig. 5) and declines rapidly thereafter. Thus, a large EPSP recruits, initially, a very large

60

TOTAL

0

60

% IN SUBLIMINAL FRINGE

0

60

% ACTIVE & REFLEXLY RECRUITED

0   100

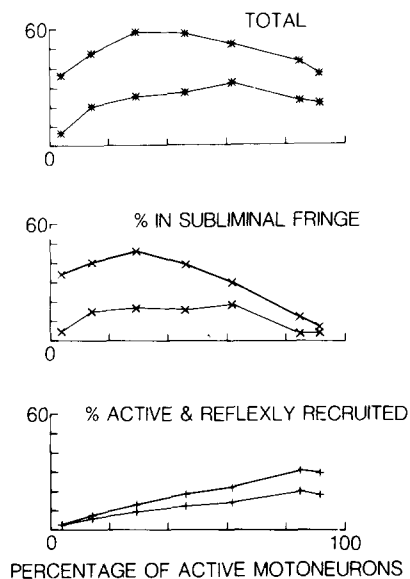PERCENTAGE OF ACTIVE MOTONEURONS

Fig. 5. Dependence of the percentage of motoneurons reflexly recruited on the size of the EPSP. The total percentage of motoneurons reflexly recruited is plotted in the topmost graph. The contribution of those in the subliminal fringe and those that are active and reflexly recruited are shown respectively in each of the two lower graphs. The topmost plot of each pair represents the respective response when the G_{epsp} was 0.06 μS and that of the lower plot when the G_{epsp} was 0.03 μS. Note that the proportion of the active and reflexly recruited motoneurons (+) increases over essentially the whole range of excitation level, whereas the proportion in the subliminal fringe (×) and hence the total recruited (*) begin to decrease at a much lower level of the excitation level when the EPSP is large.

proportion of motoneurons, and as the excitation level increases fewer motoneurons are left in the subliminal fringe. Note that the contribution of the motoneurons that are active and reflexly recruited increases over essentially the whole range of the excitation level in each case (Fig. 5).

In plotting the percentage of motoneurons excited by the EPSP all neurons are given equal weight, whereas those with larger resting conductances produce larger amounts of EMG and tension. The simplest weighting is to scale the contribution of a neuron to the reflex according to its resting conductance. This is justified if we assume that a mixed muscle has an approximately ten-fold range of motoneuron resting conductances (Kernell, 1966; Gustaffsson and Pinter, 1985) and that the range of motor unit potentials in such a muscle is also ten-fold (Milner-Brown and Stein, 1975).

The effects of various combinations of postsynaptic excitation and inhibition and of presynaptic inhibition on the weighted reflex output are shown in Fig. 6. In one case the motoneurons were depolarized by increasing the excitatory conductance and setting the inhibitory conductance to zero. In the second case, the motoneurons were depolarized by increasing the excitatory conductance and setting the inhibitory conductance to a constant value. This was done to determine whether the effects of
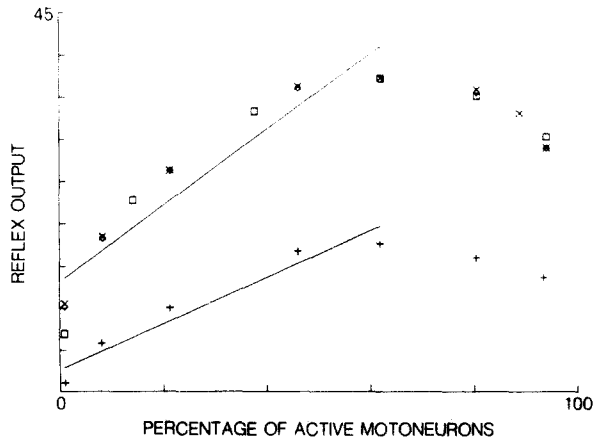
Fig. 6. Size of the reflex output (in arbitrary units) as a function of the number of active motoneurons in 4 different conditions. The topmost points represent the response when the motoneurons were depolarized by: (1) an increase of the excitatory conductance ($G_i = 0$) ($\Diamond$), (2) by an increase in the excitatory conductance with a constant amount of inhibitory conductance ($G_i = 0.2$ μS) acting on the motoneurons ($\times$), and finally (3) by an increase of the excitatory conductance and a removal of an equal amount of inhibitory conductance ($\square$). The lowermost points represent the reflex response when the G_{epsp} was reduced from 0.04 to 0.02 μS. The straight lines were fitted to the points on the increasing portion of the reflex output.

postsynaptic inhibition could be distinguished from those of presynaptic inhibition. Finally, the situation in which motoneurons are depolarized by an increase of the excitatory conductance and a reduction of the inhibitory conductance such that the total membrane conductance remains approximately constant was also simulated. This mechanism of motoneuron depolarization was reported to occur during fictive locomotion in the cat (Shefchyk et al., 1984). Since the total membrane conductance remains approximately constant, the EPSP is not shunted by the increasing excitatory conductance. Regardless of the particular combination of G_e and G_i used to depolarize the motoneurons, the size of the reflex remains essentially tied to the excitation level (Fig. 6). This is because each of the two subgroups (those active and recruited and those in the subliminal fringe) remain tied to the excitation level. The basis for this result can be shown analytically, and may have been anticipated from Fig. 3. First, the proportion of motoneurons that are active and reflexly recruited is considered.

Clearly, if the inhibitory current is increased, the excitatory current must also be increased by the same amount if the membrane potential is to reach threshold. At the threshold potential the added inhibitory current will be equal to:

$$\Delta I_i = G_i (V_i - V_t)$$

and the excitatory current must therefore be increased by an amount:

$$\Delta I_e = G_e (V_e - V_t)$$

Because the sum of the two must be zero, it therefore follows that:

$$\frac{\Delta G_e}{\Delta G_i} = \frac{(V_t - V_i)}{(V_e - V_t)} \tag{9}$$

Since the V_t, V_e, V_i are constants, the amount by which the G_e must be increased is a constant proportion of the increase in G_i and is independent of the motoneurons' resting conductance. The consequence of Eqn. 9 is that the added inhibitory current is exactly counteracted by an increase of the excitatory current. This has the effect of keeping the firing probability of a motoneuron the same regardless of the combination of G_e and G_i acting across its membrane. It can also be verified that by increasing the inhibitory conductance by an amount ΔG_i and the excitatory conductance by an amount $K\Delta G_i$ in Eqn. 7 (where K is equal to the expression on the right hand side of Eqn. 9), the value of G_a and hence the proportion of active motoneurons does not change. Since neither the probabilities of firing of the active motoneurons nor their number changes, the proportion of motoneurons that are active and reflexly recruited must remain the same.

The number of motoneurons in the subliminal fringe is also tied to excitation level for the following reason. The motoneurons in the subliminal fringe are those whose resting conductance is $G_a < G_r \leq G_f$. From Eqns. 7 and 8 it follows that G_f is given by:

$$G_f = G_a + G_{esp}(V_{esp} - V_t)/V_t \tag{10}$$

Thus, the extent of the subliminal fringe depends only on the EPSP conductance and the excitation level and is independent of the particular mixture of excitatory and inhibitory conductances used to reach that level. The EPSP conductance also determines the probability of firing of a motoneuron and hence the number of reflexly recruited motoneurons that are active. Therefore, presynaptic inhibition, by decreasing the size of the EPSP, decreases the size of the reflex at all excitation levels as well as the steepness and y-intercept of the relation between reflex output and excitation level, as shown in Fig. 6.

Note that Eqn. 10 applies only when the excitation level is above zero (i.e. there are some active motoneurons in the pool). If the excitation level is zero, then the extent of the subliminal fringe is given by Eqn. 8. This equation incorporates the intuitive notions that the size of the reflex output of a quiescent motoneuron pool is directly related to how close the motoneurons are to threshold and on the size of the EPSP.

Discussion

The simulations have revealed that the size of the monosynaptic reflex (H-reflex, or short latency stretch-reflex) depends only on the exitation level of the motoneuron pool (i.e. the number of active motoneurons) and is independent of the way the motoneurons are depolarized by postsynaptic mechanisms. This result was also demonstrated analytically.

The H-reflex is relatively independent of peripheral effects, such as the level of fusimotor drive to the muscle spindles. It thus reflects the state of the central component of the stretch-reflex, namely the effectiveness of the synaptic transmission from the Ia-afferents to the α-motoneurons. The theoretical importance of the H-reflex vs EMG curves is that a change in the slope of this relation is due to a change in the gain of the pathway from the Ia-afferents to the motoneurons (central gain). Hence, it is important to determine the possible neural mechanisms that can alter the central gain of the monosynaptic reflex.

The following arguments demonstrate that a change in the slope of the H-reflex vs EMG curve represents a change in the gain of this pathway. The H-reflex is a linearly increasing function of the background EMG for a fixed stimulus strength (Gottlieb and Agarwal, 1971, 1979; Capaday and Stein, 1986). This has been termed 'automatic gain compensation' which means that the gain of the reflex increases with the excitation level of the motoneuron pool (Marsden et al., 1972; Matthews, 1986). We have shown that the basis for this may be a recruitment of progressively larger motor units rather than an increase of the number of motoneurons recruited (Fig. 4) and this conclusion is consistent with the experimental observations of Harrison and Taylor on cat triceps surae motoneurons (Harrison and Taylor, 1981). It is clear that if the reflex output is linearly related to the excitation level, then the gain must also be an increasing function of the excitation level and this has been experimentally verified (Gottlieb and Agarwal, 1979). Therefore, a change in the slope of the H-reflex vs EMG curve must be due to a change in the central gain of the monosynaptic reflex.

It was shown in this paper that presynaptic inhibition can change both the slope and y-intercept of this relation. Presynaptic inhibition is therefore a possible mechanism for changing the central gain of the monosynaptic reflex. It was also shown that postsynaptic factors do not markedly affect the relation between these two variables (Fig. 6). The curve parameters are essentially independent of the particular mixture of excitatory and inhibitory conductances acting on the motoneurons. This gives a rationale for the existence of presynaptic inhibition in a monosynaptic pathway. In the absence of an interposed interneuron the reflex input–output properties of a motoneuron pool would be tied to its excitation level and therefore not independently controllable.

These observations may also be used to explain, for example, our recent findings of changes in the relation between the H-reflex and the EMG in going from the standing posture to walking and running (Capaday and Stein, 1986, 1987). The very large value of the y-intercept during standing means functionally that forward body sway during quiet standing will be opposed by a relatively large reflex output. This must be due to a large EPSP resulting from a removal of either, or both, presynaptic and postsynaptic inhibition. Moreover, the very large EPSP would saturate the reflex output at a low level of motoneuron pool activity and thus produce a relatively flat relation between reflex output and EMG activity which is observed experimentally.

The zero or slightly negative values of the y-intercept observed during walking and running are due to strong hyperpolarization of the motoneurons during their

'off' phase (zero activity), probably also in conjunction with increased presynaptic inhibition at zero and low levels of activity. As both of these inhibitory influences are removed during the active phase of motoneuronal activity in walking a relatively steep relation between reflex output and EMG would result, again as observed experimentally. Finally, the difference between walking and running is largely a difference in the central gain of this reflex (Capaday and Stein, 1987). The lower gain during running could only result from an increase in presynaptic inhibition according to the present analysis.

These examples illustrate the utility of the present approach in understanding the mechanisms underlying changes in reflex characteristics during functional motor tasks. The method should be applicable to a variety of such studies and can be used to design invasive animal experiments to verify the predicted mechanisms.

Acknowledgement

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