Distribution of Effective Synaptic Currents in Cat Triceps Surae Motoneurons. VI. Contralateral Pyramidal Tract

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Binder, Marc D., Farrel R. Robinson, and Randall K. Powers. Distribution of effective synaptic currents in cat triceps surae motoneurons. VI. Contralateral pyramidal tract. J. Neurophysiol. 80: 241–248, 1998. We measured the effective synaptic currents (I_E) produced by stimulating the contralateral pyramidal tract (PT) in triceps surae motoneurons of the cat. This is an oligosynaptic pathway in the cat that generates both excitation and inhibition in hindlimb motoneurons. We also determined the effect of the PT synaptic input on the discharge rate of some of the motoneurons by inducing repetitive firing with long, injected current pulses during which the PT stimulation was repeated. At resting potential, all but one triceps motoneuron received a net depolarizing effective synaptic current from the PT stimulation. The effective synaptic currents (I_E) were much larger in putative type F motoneurons than in putative type S motoneurons [+4.6 ± 2.9 (SD) nA for type F vs. 0.9 ± 2.4 nA for putative type S]. When the values of I_E at the threshold for repetitive firing were estimated, the distribution was markedly altered. More than 60% of the putative type S motoneurons received a net hyperpolarizing effective synaptic current from the pyramidal tract stimulation as did 33% of the putative type F motoneurons. This distribution pattern is very similar to that observed previously for the effective synaptic currents produced by stimulating the contralateral red nucleus. As would be expected from the wide range of I_E values at threshold (−4.8 to +8.7 nA), the PT stimulation produced dramatically different effects on the discharge of different triceps motoneurons. The discharge rates of those motoneurons that received depolarizing effective synaptic currents at threshold were accelerated by PT stimulation (+1 to +8 imp/s), whereas the discharge rates of cells that received hyperpolarizing currents were retarded by the PT input (−2 to −7 imp/s). The change in firing rates produced by the PT stimulation was generally approximated by the product of the effective synaptic currents and the slopes of the motoneurons’ frequency-current relations. Our findings indicate that the contralateral pyramidal tract may provide a powerful source of synaptic drive to some high-threshold motoneurons while concurrently inhibiting low-threshold cells. Thus this input system, like that from the contralateral red nucleus, can potentially alter the gain of the input–output function of the motoneuron pool as well as disrupt the normal hierarchy of recruitment thresholds.

INTRODUCTION

The recruitment order and firing rate modulation of the constituents of a motoneuron pool depend on both the intrinsic properties of the motoneurons and on the distribution of synaptic inputs within the pool (Binder et al. 1996; Burke 1981; Heckman and Binder 1990; Henneman and Mendell 1981). Although most quantitative descriptions of the synaptic input to motoneurons are based on measurements of the amplitude of postsynaptic potentials (PSPs) (Binder et al. 1996), we have argued that the synaptic current reaching the somatic recording electrode (effective synaptic current; I_E) is a more functionally relevant measure of the magnitude of a synaptic input (Binder et al. 1996; Heckman and Binder 1988, 1990, 1991b; Lindsay and Binder 1991; Powers and Binder 1995; Powers et al. 1992, 1993; Westcott et al. 1995; see also Redman 1976). I_E can be measured readily under steady-state conditions using a modified voltage-clamp procedure (Heckman and Binder 1988; Lindsay and Binder 1991). This technique not only yields the value of the effective synaptic current at the motoneuron’s resting potential but further provides an estimate of its somatic voltage dependence, its somatic reversal potential, and the change it produces in the motoneuron’s input resistance measured at the soma. From these data, one can readily calculate the effective synaptic current at the motoneuron’s threshold for repetitive discharge. This value then can be combined with the slope of the steady-state frequency-current (f–I) relation to predict the effect of the synaptic input on motoneuron firing behavior (Binder et al. 1996; Powers and Binder 1995; Powers et al. 1992). Equivalent results recently have been obtained from layer 5 pyramidal neurons in the rat cortex (Schwindt and Crill 1996).

In our previous studies, we have measured the effective synaptic currents generated in cat hindlimb motoneurons by homonymous Ia afferent fibers (Heckman and Binder 1988), Renshaw interneurons (Lindsay and Binder 1991), and Ia-inhibitory interneurons (Heckman and Binder 1991b) and by stimulation of the contralateral red nucleus (Powers et al. 1992, 1993) and ipsilateral Deiter’s nucleus (Westcott et al. 1995). Descriptions of the amplitude and distribution of additional synaptic input systems are necessary to understand and ultimately model the input-output functions of motoneuron pools (Binder et al. 1996; Heckman 1994; Heckman and Binder 1991a, 1993a,b; Kernell and Hultborn 1990). To this end, we have examined the synaptic input from the contralateral pyramidal tract (PT) to cat triceps surae motoneurons.

Endo and colleagues (1975) demonstrated that stimulation of the contralateral motor cortex in cats generates predominantly excitatory PSPs in medial gastrocnemius motoneurons, whereas the PSPs evoked in soleus motoneurons are predominantly inhibitory. When they stimulated the contralateral red nucleus in the same motoneurons, the pattern of PSPs they observed was similar in almost all cases.

In the results reported here, we found that the magnitudes and distribution of the effective synaptic currents produced by PT stimulation were quite similar to those observed earlier for the red nucleus (Powers et al. 1993). Measured at rest, the effective synaptic currents were depolarizing in all

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but one motoneuron (a putative type S cell). However, the currents in the putative type F motoneurons were about five times larger, on average, than those in the putative type S motoneurons. When the amplitudes of the effective synaptic currents were estimated at the voltage threshold for repetitive firing, the distribution of PT input was markedly altered, as was the case for the red nucleus (Powers et al. 1993). More than 60% of the putative type S motoneurons received a hyperpolarizing current from the pyramidal tract, but among the putative type F cells, two-thirds received net excitatory effective synaptic currents and one-third net inhibitory effective synaptic currents.

Our findings indicate that the pyramidal tract may provide a powerful source of synaptic drive to some high-threshold motoneurons while concurrently inhibiting low-threshold cells. Thus was the case for the contralateral red nucleus, this input system potentially can alter the gain of the input-output function of the motoneuron pool, change the hierarchy of recruitment thresholds within the pool, and mediate rate limiting of discharge in low-threshold motoneurons (i.e., saturation of firing rates of low-threshold motoneurons as higher-threshold motoneurons are recruited and increase their discharge rates) (Binder et al. 1996; Heckman and Binder 1990, 1991a, 1993a,b).

METHODS

Experimental preparation

We obtained the data presented here from six adult cats. We induced anesthesia with an intraperitoneal injection of pentobarbital sodium (40 mg/kg) and gave intravenous supplements at regular intervals to maintain a deep level of anesthesia throughout all surgical and experimental procedures. Details of our animal maintenance and surgical procedures may be found in previous papers of this series (Heckman and Binder 1988, 1991b; Lindsay and Binder 1991; Powers and Binder 1995; Powers et al. 1993; Westcott et al. 1995). Only specific procedures germane to these experiments are presented in the following paragraphs.

A conventional laminectomy was performed (L₄–S₁). In the left hindlimb, the nerves to the medial gastrocnemius (MG) and synergist lateral gastrocnemius and soleus (LGS) muscles were dissected carefully but left in continuity with their muscles. The cat was then mounted in a rigid “Goteborg type” spinal cord recording frame with the head mounted in a stereotaxic holder. To access the contralateral (right) pyramidal tract, we opened a small craniotomy centered ~2 mm to the right of the midline on the nucal ridge over the cerebellum (Berman 1968) and opened a dural flap.

Pyramidal tract identification

We placed a monopolar stimulating electrode in the pyramidal track ventral to the trapezoid body and caudal to the pontine nuclei. We chose this site to avoid direct stimulation of reticulospinal neurons located dorsal and more caudal to the pyramidal tract. We made several penetrations starting at 1.5 mm to the right of the midline, angled 15° dorsocaudal to ventrorostral, and passing through anterior-posterior (AP) –6 at dorsal-ventral (DV) 0 (Berman 1968). To locate the optimal stimulation site, we recorded extracellular activity in the brain stem with epoxylite-coated etched tungsten microelectrodes. We looked for a site ventral to the trapezoid body, identifiable by large auditory potentials, at which we could record antidromic field potentials driven from electrical stimulation applied to the dorsolateral surface of the left lumbar spinal cord between L₄ and L₅. When we identified such a site, we replaced the recording electrode with a larger epoxylite-coated etched tungsten stimulating electrode with ~1 mm of its tip exposed. To place the stimulating electrode at the recording site accurately, we referenced both electrodes to the same reference point using a ×50 microscope on the zeroing stand. A monopolar electrode was placed on the L₅ lateral column to monitor the descending volleys produced by pyramidal tract stimulation.

At the end of each experiment, we confirmed the position of our stimulating electrode by making an electrolytic lesion on site with the stimulating electrode in the pyramidal tract (30 mA for 30 s; electrode negative). We then gave the cat a lethal dose of pentobarbital and perfused it through the left ventricle with normal saline followed by a buffered glyoxal/ethanol solution (Prefer, Anatech). We removed the brain stem between the superior colliculi and the dorsal column nuclei and put it in 10% neutral buffered formalin for 2 wk after which we soaked it in 30% sucrose until it sank. The right half of the block was cut into 50-μm parasagittal frozen sections, stained with cresyl violet, mounted, and examined to locate the stimulation site (e.g., Fig. 1).

Experimental protocol

We used potassium sulfate-filled, glass capillary microelectrodes to obtain intracellular recordings from MG and LGS motoneurons. The electrode tips were broken to yield in situ resistances of 2–8 MΩ. The motoneurons were identified based on antidromic action potentials evoked by muscle nerve stimulation. Only motoneurons with stable resting potentials of at least ~55 mV or greater and antidromic action potentials with positive overshoots were accepted for study. The data were stored on VCR tape via a pulse code modulated (PCM) digitizing unit. On-line display and off-line analysis of the recorded signals was performed using the MacAdios II data acquisition board (GW Instruments) and a Macintosh II microcomputer.

The initial motoneuron recordings consisted of antidromic action potentials and responses to 50-ms, injected current pulses to determine rheobase (Fleschner et al. 1981; Zengel et al. 1985). Next we used our modified voltage-clamp technique (Heckman and Binder 1988, 1991b; Lindsay and Binder 1991; Powers and Binder 1995; Powers et al. 1993; Westcott et al. 1995) to measure the effective, steady-state synaptic current (Iₛ) generated in the motoneuron by supramaximal PT stimulation at 200 Hz. We used 100-μs current pulses in all of the experiments. In five of the six cats, we used bipolar stimulation with strengths of 600–800 μA, and in the sixth cat we used monopolar negative stimulation with strengths of 1,000 μA. Subsequently, we attempted to elicit repetitive discharge with 1-s depolarizing current pulses. In those cells capable of sustained repetitive discharge, we injected a series of suprathreshold 1-s depolarizing current pulses of various amplitudes, during some of which we repeated the 200-Hz PT stimulation (Powers and Binder 1995; Powers et al. 1992). Finally, rheobase and action potential measurements were repeated before withdrawing the microelectrode from the cell.

Measurements and analysis

Figure 2 illustrates how we measured the effective synaptic currents (Iₛ) produced by pyramidal tract stimulation. Figure 2A shows intracellular recordings from a motoneuron in response to a series of depolarizing and hyperpolarizing injected current pulses and synaptic current produced by electrical stimulation within the contralateral PT at 200 Hz. The experimental records were divided into three 500-ms epochs: injected current alone, concurrent injected and synaptic currents, and synaptic current alone. The steady-state response of the neuron to injected current was desig-
nated as \( V_i \), that to injected and synaptic current as \( V_{i,s} \), and the difference between these two values, as \( \Delta V_s \). By plotting the change in membrane potential during concurrent synaptic and injected current \( (V_{i,s}) \) versus the amount of injected current \( (I) \), we could interpolate the effective synaptic current at rest \( (I_s) \), defined as the current required to clamp the membrane potential at the resting potential during the activation of the synaptic input \( (\text{Fig. } 2B, \bullet ) \) (Heckman and Binder 1988). \( I_s \) was calculated by multiplying the slope of the best-fit linear regression line of \( V_{i,s} \) versus \( I \), \(-1\) (i.e., the magnitude \( I_s \) is assumed to be equal in magnitude and opposite in sign to the injected current required to clamp the membrane potential at the resting value) (Lindsay and Binder 1991; Powers et al. 1992).

The slope of the linear relation between \( V_{i,s} \) and \( I \) provides a measure of the input resistance during synaptic activation \( (R_{\text{syn}}) \). We could determine whether or not the synaptic input produced any detectable conductance change in the motoneuron soma by comparing this slope to that of \( V_i \) \( (\square) \) versus \( I \). The slope of the linear fit to \( V_i \) versus the amount of injected current \( (I) \) yields the steady-state input resistance \( (R_{\text{ss}}) \) in the absence of synaptic activation. A significant difference between the slopes indicates that the synaptic input altered the conductance of the motoneuron measured at the soma (Heckman and Binder 1991b; Lindsay and Binder 1991; Powers et al. 1993). We also measured the relation between the peak voltage response \( (V_{\text{max}}) \) and injected current to obtain a measure of input resistance analogous to that reported by several other investigators, who have used relatively short \( (50-100 \text{ ms}) \) current pulses \( (e.g., \text{Zengel et al. } 1985) \). This measurement \( (R_{\text{syn}} \text{ in } \text{Fig. } 2B) \) subsequently will be referred to simply as input resistance, as opposed to the steady-state input resistance \( (R_{\text{ss}}) \) that is obtained from the voltage measured \( 400-500 \text{ ms} \) after current onset (Heckman and Binder 1988).

We used the relation between \( \Delta V_s \) \( (\text{the amplitude of the steady-}
\text{state synaptic potential}) \) and \( V_i \) \( (\text{the membrane potential relative to rest just before the onset of synaptic activation}) \) to determine the somatic reversal potential and the somatic voltage-dependence of the effective synaptic current \( (\text{Lindsay and Binder } 1991) \). A plot of \( \Delta V_s \) versus \( V_i \) reveals the dependence of steady-state synaptic potential on somatic membrane potential, and the x intercept of the linear fit approximates the somatic reversal potential for the effective synaptic current \( (\text{Fig. } 2, \text{C and D}) \). Determining the effect of somatic membrane potential of the effective synaptic current is particularly important in the analysis of inhibitory or mixed excitatory and inhibitory inputs, where some of the synaptic boutons may be close to the soma \( (cf. \text{Powers et al. } 1993) \).

We estimated the effective synaptic current flowing at threshold for repetitive discharge \( (I_{\text{thresh}}) \) in two ways. In those cells in which \( I_s \) was depolarizing at rest, and \( \Delta V_s \) was not correlated with \( V_i \) \( (e.g., \text{Fig. } 2C) \), the reversal potential \( (V_{\text{rev}}) \), in absolute voltage as measured at the soma was assumed to be \( 0 \text{ mV} \) (Coombs et al. 1955; Finkel and Redman 1983). The effective synaptic current at threshold \( (I_{\text{thresh}}) \) was determined by estimating the motoneuron threshold potential during repetitive firing and then calculating how that change in driving force would have increased or decreased the effective synaptic current measured at rest \( (I_{\text{thresh}}) \). The effect of the change in driving force is equal to \( I_{\text{thresh}} (V_{\text{rev}}) \) versus \( I_{\text{thresh}} (V_{\text{rev}}) \) where \( V_{\text{rev}} \) is the reversal potential for the net effective synaptic current \( (\text{Powers et al. } 1992; \text{Powers and Binder } 1995) \).

\[
I_{\text{thresh}} = (I_{\text{thresh}})(V_{\text{rev}})/(V_{\text{rev}} - V_{\text{rev}})
\]

This correction for the change in driving force generally resulted in a relatively modest decrease in the amplitudes of depolarizing effective synaptic currents \( (i.e., I_{\text{thresh}} \text{ is approximately equal to } 0.9I_{\text{rest}}) \). If the true value of \( V_{\text{rev}} \) was significantly more positive \( (e.g., +40 \text{ mV relative to the soma}) \) due to a remote dendritic location of the synapses, then \( I_{\text{thresh}} \) would be approximately equal to \( 0.95I_{\text{rest}} \).

When a significant linear correlation was found between \( \Delta V_s \) and \( V_i \) \( (\text{e.g., } \text{Fig. } 2D) \), we used this relation to predict \( \Delta V_i \) for any value of \( V_i \). First we estimated the voltage threshold for repetitive discharge \( (V_{\text{thresh}}) \) from the current threshold for repetitive discharge \( (I_{\text{thresh}} = 1.5 \text{ times rheobase}) \) (Kernell and Monster 1981) and the cell’s input resistance during synaptic current activation \( (R_{\text{syn}}) \), based on Ohm’s law: \( V_{\text{thresh}} = I_{\text{thresh}}/R_{\text{syn}} \). Second we estimated the steady-state synaptic potential \( (\Delta V_s) \) at threshold \( (V_i = V_{\text{thresh}}) \) based on the relation of \( \Delta V_s \) versus \( V_i \) \( (\text{cf. } \text{Fig. } 2D) \). \( I_{\text{thresh}} \) then was estimated by dividing this extrapolated value of \( \Delta V_s \) by \( R_{\text{syn}} \) \( (\text{Powers and Binder } 1995; \text{Powers et al. } 1992) \).

\[
I_{\text{thresh}} = \Delta V_s / R_{\text{syn}}
\]
FIG. 2. Measurement of effective synaptic current generated by supramaximal 200-Hz stimulation within the contralateral pyramidal tract. A: response of a lateral gastrocnemius-soleus motoneuron to injected currents (bottom set of traces) and synaptic currents (---). Experimental protocol consists of 3 500-ms epochs (injected current alone, injected + synaptic current and synaptic current alone), which are numbered and separated by vertical dotted lines. Mean resting potential (measured before current injection) has been subtracted from each trace and the traces have been digitally low-pass filtered (100-Hz cutoff) for clarity. Measurements are indicated for the bottom voltage trace. \( V_{i} \): steady-state voltage response to injected current alone; \( V_{max} \): peak voltage response to injected current; \( V_{i+s} \): steady-state voltage response to synaptic and injected current; \( \Delta V_s \): \( V_{i+s} - V_{i} \), change in voltage due to synaptic current. B: voltage responses vs. injected current \((I)\). ---, best linear fit to the data points \((V; \circ; V_{i+s}; \bullet; V_{max}; *)\). Effective synaptic current \((I_N)\) is taken to be equal in magnitude and opposite in sign to the current at which \( V_{i+s} = 0 \) (estimated from the 0 intercept of the fit to \( V_{i+s} \) vs. \( I \)). Slope of the linear fit to \( V_i \) vs. \( I \) gives the steady-state input resistance \((R_{Ns})\), that of the fit to \( V_{max} \) vs. \( I \) gives the maximum input resistance \((R_{Nmax})\), and that of the fit to \( V_{i+s} \) vs. \( I \) gives the steady-state input resistance during synaptic activation \((R_{Nsyn})\). C: relationship between steady-state synaptic potential \((\Delta V_s)\) and somatic membrane potential \((V_i)\). D: plot \( \Delta V_s \) vs. \( V_i \) for a different motoneuron in which the magnitude of the effective synaptic current \((I_N)\) was strongly dependent on the somatic membrane potential.

clamp trials (i.e., injected current alone, injected + synaptic current, synaptic current alone). To ensure that the same amount of effective synaptic current was generated during the repetitive firing trials, we compared the steady-state PSP after the injected current with that recorded earlier under subthreshold conditions. These trials were alternated with controls, in which the same amount of current was injected through the microelectrode, but the pyramidal tract was not stimulated. The change in firing rate \((\Delta F)\) produced by the synaptic input was calculated from the difference between the average firing rate during the last 300 ms of current injection in test and control (injected current alone) trials (Powers and Binder 1995; Powers et al. 1992). At least two control and two test trials were run at each level of injected current. When the recording conditions were optimal and repetitive firing was robust and consistent, the trials were repeated at one or more additional levels of injected current.

Sources of error

Inherent in all measurements made from intracellular recordings are errors due to initial artifacts from impalement injury, changes during the recording period due to current leak around the impalement site, and small signal to noise ratios. We previously have estimated the measurement errors associated with \( I_N \) and \( R_N \) at \( \sim 15\% \) (Heckman and Binder 1988, 1991b; Lindsay and Binder 1991; Powers and Binder 1995; Powers et al. 1993). Considerable effort was made
during recording to compensate for electrode resistance by carefully adjusting the bridge balance circuit, and we assessed this again offline. Any measurement error that occurred would be random and thus should not generate significant trends in the data.

Selective activation of descending motor systems is imperfect due to the difficulty associated with consistent electrode placement and activation of other axons or neurons in the vicinity of the electrode (Baldissera et al. 1981). Our histological results indicated that in each experiment, we had placed the stimulating electrodes securely within the pyramidal tract under the trapezoid body where the nearest reticular neurons were over 2 mm away (e.g., Fig. 1). Thus it is unlikely that current spread from our stimulation site to activate reticulospinal neurons. Further, it has been reported that in the cat, only ~16% of identified corticospinal neurons could be antidromically activated by stimulation of the medullary reticular formation (Endo et al. 1973). Thus it is unlikely that neurons activated by pyramidal tract collaterals made a significant contribution to the synaptic currents we measured in triceps motoneurons.

RESULTS

We measured the steady-state, effective synaptic currents produced by stimulating the contralateral pyramidal tract in 23 triceps surae motoneurons (19 MG and 4 LGS). In addition, we could measure the change in motoneuron discharge rate produced by the synaptic input in 17 of these cells. The values for input resistance [mean: 1.4 ± 0.63 (SD) MΩ; range: 0.5–2.8 MΩ] and rheobase (mean: 9.9 ± 4.7 nA; range 2.9–20.5 nA) indicate that our sample included nearly the full range of motoneurons within these pools (Fleshman et al. 1981; LaBella et al. 1989; Powers and Binder 1985).

Distribution of $I_N$ generated by pyramidal tract stimulation

The distribution of effective synaptic currents ($I_N$) within the triceps surae motoneuron pools evoked by stimulating the contralateral pyramidal tract is shown in Fig. 3. In Fig. 3A, the values of $I_N$ measured at the resting potential are plotted versus motoneuron input resistance. In Fig. 3B, the values of $I_N$ are those estimated at the motoneuron threshold potential. The motoneurons have been classified provisionally into unit type based on their rheobase and input resistance values (Zengel et al. 1985). At rest, a clearer difference in the magnitude of $I_N$ is apparent with respect to motor unit type: the effective synaptic currents in the putative type F motoneurons are about five times greater, on average, than those in the putative type S motoneurons (+4.6 ± 2.9 nA for type F vs. 0.9 ± 2.4 nA for putative type S; $P < 0.01$, unpaired t-test).

The distribution of effective synaptic currents from the pyramidal tract looks quite different when the estimated values of $I_N$ at threshold are plotted (Fig. 3B). At motoneuron threshold, >60% of the putative type S units received hyperpolarizing effective synaptic currents, as did a third of the putative type F units. Thus the change in driving force between rest and threshold had a dramatic effect on the small effective synaptic currents (Fig. 3A).

Effects of resting potential on $I_N$

Because $I_N$ can be clearly influenced by changes in somatic membrane potential in some motoneurons (e.g., Fig. 2D), it is conceivable that differences in resting potential between different cells could have contributed to the variance of $I_N$ values. If so, systematic differences in resting potential between putative type S and F cells could have contributed to systematic differences in their mean $I_N$ values measured at rest. Although our sample of cells exhibited a wide range of resting potentials (−74 to −58 mV, mean: −64.2 ± 5.0 mV), there were no significant differences between the resting potentials of our putative type S and type F motoneurons (−62.3 ± 5.8 mV for type S, −65.3 ± 4.7 mV for type F). Further, $I_N$ measured at rest was not correlated with membrane potential ($I_N$rest vs. $V_{rest}$, $r = −0.39$, $P = 0.064$).

Effects on PT input on repetitive discharge

We could assess the functional significance of the input from the contralateral pyramidal tract by examining its effect on repetitive discharge in 17 motoneurons. As detailed in METHODS, a series of 1-s depolarizing current pulses, above threshold for repetitive firing, was injected into the motoneuron. After 500 ms, the pyramidal tract was again stimulated.
at 200 Hz for 1 s. These trials were alternated with controls, in which the same amount of current was injected through the microelectrode, but the PT was not stimulated. As would be expected from the wide range of estimated effective synaptic current magnitudes at threshold (−4.8 to +8.7 nA; Fig. 3B), the PT input produced dramatically different effects on the discharge of different motoneurons. The discharge rates of those motoneurons that received depolarizing effective synaptic currents at threshold were accelerated by PT stimulation (+1 to +8 imp/s), whereas the discharge rates of cells that received hyperpolarizing currents were retarded by the pyramidal tract input (−2 to −7 imp/s). As illustrated in Fig. 4, the observed change in firing rate produced by the pyramidal tract stimulation (∆Fobs) was generally close to that predicted from the measurement of the effective synaptic current at threshold and the slope of the motoneuron’s f-I relation (i.e., ∆Fpr = IN * fI) (Powers and Binder 1995; Power et al. 1992).

**Conductance changes induced by pyramidal tract synaptic input**

Our standard experimental protocol provides a means of measuring the input resistance of the cell both with and without the synaptic activation (cf. Fig. 2B). The resting steady-state input resistance (RNss) is plotted versus the input resistance measured during the steady-state activation of the contralateral pyramidal tract (RNSyn) in Fig. 5. The linear regression analysis indicates that the PT synaptic input reduced motoneuron input resistance by 40% on average (RNss = 0.56 * RNSyn + 0.15; r = 0.89). The extent to which the synaptic conductance altered motoneuron input resistance was not related to putative motor unit type (type F vs. type S; unpaired t-test; t = 1.7; P > 0.1) nor was it correlated with the magnitude of the effective synaptic current induced (∆RN vs. IN; r = 0.17, P > 0.4).

**DISCUSSION**

We have found that activation of the contralateral pyramidal tract produces a wide range of effective synaptic currents in cat triceps surae motoneurons. Measured at resting potential, the effective synaptic currents produced by stimulating the contralateral pyramidal tract were much larger in putative type F motoneurons than in putative type S motoneurons (+4.6 ± 2.9 nA for type F vs. 0.9 ± 2.4 nA for putative type S). Further, when the current values were extrapolated to the threshold potential for repetitive firing, >60% of the putative type S motoneurons received a net hyperpolarizing effective synaptic current from the pyramidal tract stimulation as did a third of the putative type F motoneurons.

The data we obtained on the effects of the pyramidal tract stimulation on repetitive motoneuron firing were consistent with both the range and magnitude of the effective synaptic currents we measured under subthreshold conditions. The discharge rates of those motoneurons that received depolarizing effective synaptic currents at threshold were accelerated by from 1 to 8 impulses/s, whereas the discharge rates of cells that received hyperpolarizing currents were retarded by the pyramidal tract input by from −2 to −7 imp/s. As we have previously reported for other synaptic input systems (Powers and Binder 1995), these changes in firing rate were generally approximated by the product of the net effective synaptic current at threshold and the slope of the motoneuron’s frequency-current relation (i.e., ∆F = IN * fI) (Power et al. 1992).

**Comparison of synaptic input from pyramidal tract with those of other systems**

We now have studied the distribution of effective synaptic currents for six different input systems to cat lumbar motoneurons. All of these data have been collected under comparable conditions, namely acute experiments on cats deeply anesthetized with barbiturate. Thus it is likely that the level of excitability of interneurons and motoneurons in these experiments might be quite different from that in intact, awake behaving animals. As a consequence, the magnitude of the
The present results indicate that the contralateral pyramidal tract is capable of producing strong inhibition of low-threshold motoneurons, while providing a powerful excitatory drive to some high-threshold motoneurons. Thus like the rubrospinal pathway, this pyramidal input might be responsible in part for the distinctive pattern of rate limiting observed in the discharge of low-threshold motoneurons during sustained contractions (Heckman and Binder 1993a). In addition, the input from the pyramidal tract (as well as input from other systems exhibiting a similar effective synaptic current distribution) potentially can alter the gain of the input-output function of the motoneuron pool and may disrupt the normal hierarchy of recruitment thresholds within the pool (Heckman and Binder 1990).

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FIG. 6. Graphic representation of the magnitude and distribution of the effective synaptic currents (I_E) from 6 different input systems measured in cat lumbar motoneurons at rest. Stripped line represents I_E from the contralateral pyramidal tract from the present study; the dark, stippled band represents I_E from homonymous Ia afferent fibers (Heckman and Binder 1988); the stripped band represents I_E from Ia-inhibitory interneurons (Heckman and Binder 1991); the black band represents the I_E from Renshaw interneurons (Lindsay and Binder 1991); the thick lines outline the I_E from contralateral rubrospinal neurons (Powers et al. 1993); and the light stippled band represents I_E from ipsilateral Deiter’s nucleus (Westcott et al. 1995).

Effective synaptic currents we have measured, particularly those mediated through interneurons, may underestimate the actual strength of these inputs under more natural conditions. Nonetheless, these data certainly permit us to assess the relative strengths of these input systems as well as their patterns of distribution within the triceps motoneuron pools.

Our data on the distribution of effective synaptic currents from the contralateral pyramidal tract, along with those of five other synaptic input systems, are summarized graphically in Fig. 6. Both the range of amplitudes and the pattern of distribution of the effective synaptic currents from the pyramidal tract were most similar to those produced by stimulation within the contralateral red nucleus (Powers et al. 1993). This is not surprising given that pyramidal and rubrospinal tract neurons converge on common interneurons within the contralateral intermediate nucleus (Baldissera et al. 1981).

Both the rubrospinal and pyramidal tract neurons activate excitatory and inhibitory segmental interneuronal pathways that project to triceps motoneurons. These pathways share common elements with the segmental reflex pathways activated by muscle, cutaneous and articular afferents (Baldissera et al. 1981; McCrea 1996). As a consequence of this convergence, it is somewhat imprudent to speculate on exactly how these descending neurons will shape motor output. What is clear, however, is that motoneurons innervating a muscle do not receive identical patterns of synaptic input from these afferent systems.


