The blue component did not extend farther radially than the green. Because the highly yellow lens of the ground squirrel's eye strongly absorbs those wavelengths shorter than about 500 nm, there is the possibility that the intensity of the blue light was insufficient to stimulate an existing, but very insensitive, surround. Nevertheless, because of evidence to be described in a paper in preparation, it seems quite certain that these latter receptive fields represent a second class of opponent color units.

The opponent color receptive fields of the ground squirrel are similar to those of some retinal ganglion cells in the goldfish (*6*) and to those of some cells in the lateral geniculate nucleus of the monkey (*7*). A comparision of the opponent color receptive fields of these three animals will be made in a paper now in preparation.

The present report, together with the previous one (*3*), further substantiates the statement that highly sophisticated neural integrations occur within the retina of the ground squirrel. No other mammalian retina is known to process both movement and color information to such a great extent. It is important to remember that all of this complex neural coding takes place before any visual information is transmitted through the optic nerve to the brain.

CHARLES R. MICHAEL*
Biological Laboratories, Harvard University, Cambridge, Massachusetts

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* Present address: Department of Biophysics, Johns Hopkins University, Baltimore, Md. 21218.

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Postsynaptic versus Presynaptic Inhibition in Antagonistic Stretch Reflexes

Abstract. *Motoneurons of the cat gastrocnemius-soleus muscle were studied intracellularly with conventional glass micropipettes. Each of these motoneurons was made to fire repetitively by stretch of its own muscle (gastrocnemius-soleus), and by current injected through the impaling microelectrode. By comparing the amount of inhibitory influence from antagonistic stretch of posterior biceps on the repetitive firing in these two different situations, an estimate could be obtained of the relative contribution of postsynaptic inhibition in this type of antagonistic stretch reflex. Even when the experimental conditions were such as to favor presynaptic inhibition, only strong postsynaptic inhibitory effects were seen; presynaptic inhibition was not found.*

Presynaptic reduction in the potency of the excitatory inflow to spinal motoneurons from muscle afferents was reported by Frank and Fuortes (*1*). They found that the monosynaptic excitatory postsynaptic potential recorded intracellularly from gastrocnemius motoneurons was sometimes reduced in size by a volley in the hamstring afferents. Moreover, the inhibitory hamstring volley alone produced no hyperpolarization or change in excitability of the postsynaptic membrane. Later, Frank (*2*) proposed two possible explanations of these findings: (i) the excitatory inflow had been influenced by the hamstring volley before arriving at the motoneuron postsynaptic membrane, or (ii) interaction between the excitatory and inhibitory volleys occurred within the motoneuron but at a distant site where the effect of the inhibitory volley alone could not be detected by the microelectrode that impaled the soma of the cell.

Eccles and co-workers (*3*) have used the similarities between depression of excitatory postsynaptic potentials and the amount of depolarization at the primary afferent terminals to argue for the existence of presynaptic inhibition. The depolarization of the presynaptic terminals is postulated to decrease the amplitude of the action potentials propagated into these terminals and thus to diminish the amount of transmitter substance liberated. Presynaptic inhibition has also been reported to differ from the previously known spinal postsynaptic inhibitions in its pharmacological properties (*4*). However, postsynaptic inhibitions which, like presynaptic inhibition, are resistant to strychnine have recently been described in spinal motoneurons (*5, 6*). In addition, the peripherally activated, postsynaptic, strychnine resistant inhibitions are removed by picrotoxin (*6, 7*) held to be a specific antagonist of presynaptic inhibition (*4*).

This study was undertaken to gain information about the relative contribution of postsynaptic inhibition in spinal motoneurons when muscle stretch is used as a stimulus.

A motoneuron, which is fired repetitively by autogenetic muscle stretch (meaning stretch of its own muscle), reduces its rate of firing in response to antagonistic muscle stretch (*8*). This inhibition may reflect a combination of events occurring at the motoneuron postsynaptic membrane, at the presynaptic excitatory terminals, and at interneuronal relays. The excitability change occurring at the postsynaptic membrane may be caused by true postsynaptic inhibition or by a removal of background excitation, and can be assessed from the reduction in motoneuron discharge rate when firing is produced solely by passing a constant depolarizing current through the impaling microelectrode tip, thus bypassing the primary afferent terminals. By comparing the amount of inhibition in these two different situations, that is, synaptically induced firing and firing induced by injected currents, an estimate can be obtained of the relative contribution of postsynaptic excitability changes.

The measurements were collected from 18 cats, which were anesthetized with pentobarbitone (35 to 40 mg/kg) and immobolized by gallaminetriethiodide (Flaxedil, Abbott). The posterior biceps and gastrocnemius-soleus muscles in the left hind limb were freed and strings were tied to the cut distal tendons so as to be able to stretch the muscles by weights. The lumbar cord was exposed and transected at L_2. Except for the ipsilateral L_7 dorsal root, the spinal cord was bilaterally de-afferented from L_5 and below. The ipsilateral L_7 (often also S_1) ventral root was cut, and the peripheral stump was stim-

ulated in order to localize contractions from unwanted muscles and thus to complete the denervation peripherally. In the cat many blood vessels enter the spinal canal together with the spinal nerves. With this particular preparation the condition of the cord was improved if some roots were left intact. Any possible spontaneous afferent activity through the remaining small spinal nerves L_2, L_3, and L_4 should not affect the conclusions of this study, since these inputs are of minor importance in the reflex actions under investigation. Thus, this spinal preparation may be more or less regarded as consisting of only two afferent inputs (from the gastrocnemius-soleus and the posterior biceps muscles) with their central connections. Gastrocnemius-soleus motoneurons were impaled in the L_7 segment with conventional potassium citrate (or sometimes potassium chloride) glass micropipettes having resistances of 4 to 10 Mohm. A bridge circuit was arranged for passing polarizing currents through the recording microelectrode.

The animals on barbiturate anesthesia were often also strychninized (0.08 to 0.15 mg/kg), both of which are factors described as potentiating presynaptic inhibition (*4*); here, postsynaptic inhibition should be depressed, if anything. Furthermore, with this combination of muscles the upper flexor (posterior biceps) is supposed to give prominent presynaptic inhibition (*1, 3*) but only small postsynaptic effects, at least from the group I afferents (*9*), to the gastrocnemius-soleus motoneurons.

The general recording procedure is illustrated by the results in Fig. 1. This gastrocnemius-soleus motoneuron (monosynaptic spike shown in Fig. 1*G*) was impaled with a potassium citrate microelectrode in a cat treated with strychnine (0.08 mg/kg). Monosynaptic excitatory postsynaptic potentials (EPSP's) were produced by electrical stimulation of the nerve of the gastrocnemius-soleus muscle and several recordings were averaged to obtain the size of the EPSP and its half decay time. The control EPSP (Fig. 1*A*) had a peak amplitude of 10.0 mv and a half decay time of 5.5 msec. During 1000-g stretch of the posterior biceps muscle, the EPSP diminished in size by 26 percent to 7.4 mv and the half decay time was shortened by 29 percent to 3.9 msec (Fig. 1*B*). By using the time mark as a reference line, it is seen that no shift of the membrane potential occurred during this posterior biceps stretch; the EPSP started from the same d-c level in the two different situations (Fig. 1, *A* and *B*, respectively). This particular neuron was not excitable enough to be fired by muscle stretch alone, but with a weak subthreshold depolarizing current of constant strength continuously passing through the microelectrode, stretch of the gastrocnemius-soleus muscle by a 500-g weight caused the neuron to fire repetitively (Fig. 1*C*; onset of stretch is indicated by downward deflection of myograph at arrow 1). When a 1000-g weight was added to the posterior biceps muscle (arrow 2 in Fig. 1*C*) the discharge was inhibited. Upon release of the muscle the firing was resumed. As seen at the end of the record in Fig. 1*C*, the firing terminated when ultimately the weight was removed from the gastrocnemius-soleus muscle. In Fig. 1*D* approximately the same frequency of firing was then produced by the injected current alone. Loading the posterior biceps muscle with a weight of 1000 g again reduced the firing rate. These testing procedures were repeated several times so as to obtain multiple records with which to make comparisons. In this neuron the synaptically induced firing was, on the average, reduced by 5.71 spikes per second, while the corresponding value for the firing from direct stimulation was 5.63 spikes per second. The insignificant difference between these two values may mean that if presynaptic inhibition was acting at the afferent gastrocnemius-soleus terminals of this cell it had no functional

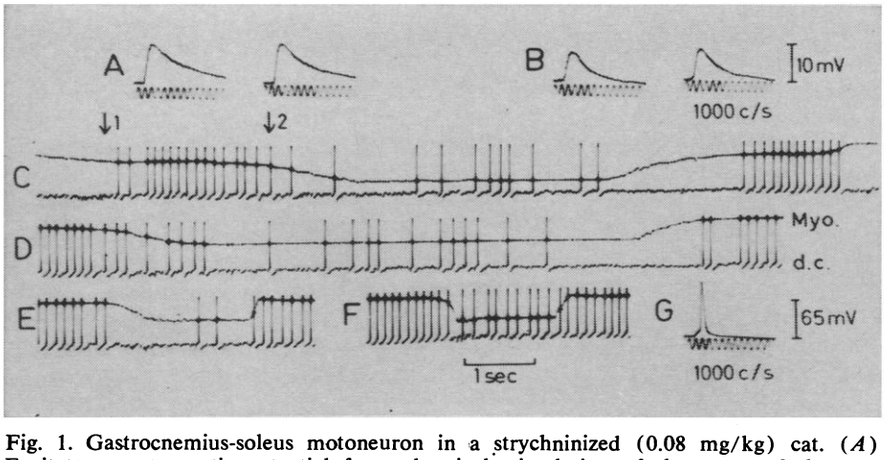

Fig. 1. Gastrocnemius-soleus motoneuron in a strychninized (0.08 mg/kg) cat. (*A*) Excitatory postsynaptic potential from electrical stimulation of the nerve of the gastrocnemius-soleus muscle. (*B*) The same potential during 1000-g stretch of posterior biceps. (*C*) A subthreshold depolarizing current was injected through the impaling microelectrode; 500-g stretch of the gastrocnemius-soleus muscle (indicated by downward deflection of myograph at arrow 1) fired the neuron repetitively. Adding a 1000-g weight to posterior biceps (arrow 2) inhibited the discharge. When the weight was removed from posterior biceps, the firing was resumed, only to terminate when ultimately the load was taken off gastrocnemius-soleus. (*D*) The neuron was fired repetitively by injected current alone; 1000-g stretch of posterior biceps inhibited this discharge also. (*E*) Repetitive firing from injected current and 1000-g stretch of posterior biceps. (*F*) Same as (*E*), but recorded after the intravenous administration of picrotoxin (1.0 mg/kg). (*G*) Monosynaptic spike from stimulation of the nerve of the gastrocnemius-soleus muscle.

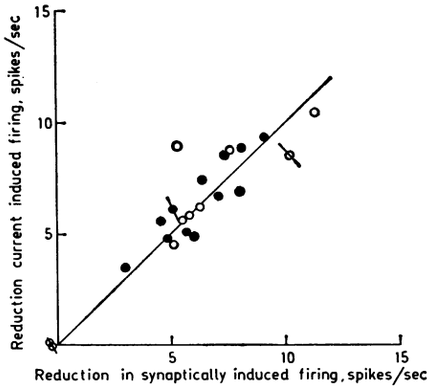

Fig. 2. Results from the 22 gastrocnemius-soleus motoneurons investigated. Open circles, neurons from strychninized (0.08 to 0.15 mg/kg) cats. Filled circles, neurons from nonstrychninized cats. For each motoneuron is shown the amount of inhibition from posterior biceps stretch on firing induced by autogenetic muscle stretch (plotted on the *x*-axis) and on firing induced by injected current (plotted on the *y*-axis). A line of unit slope is drawn in the graph. The diagonal bars (to be projected on the *x*- and *y*-axes) crossing three circles indicate the calculated standard errors for these randomly selected cases, respectively.

importance. To test the possibility that spontaneous afferent inflow was affecting the postsynaptic membrane of this particular motoneuron, the nerve of the gastrocnemius-soleus muscle was furthermore blocked by the application of procaine. However, the threshold for direct stimulation as well as the inhibition from posterior biceps stretch remained unchanged, again demonstrating the postsynaptic character of this inhibition. Such postsynaptic inhibitions by muscle stretch without effect on the average membrane potential have earlier been described (*10*). In Fig. 1*E* is again shown the inhibitory effect of a 1000-g stretch of posterior biceps on the firing induced by injected current. Between *E* and *F* of Fig. 1, picrotoxin was administered intravenously in a dose of 1.0 mg/kg. A few minutes later most of this strychnine-resistant postsynaptic inhibition had been removed by picrotoxin (Fig. 1*F*, to be compared with *E* of the same figure). This result is in agreement with what has been found in a previous study (*7*).

In the cases where KCl microelectrodes were used, it was possible to demonstrate the postsynaptic nature of these stretch-induced inhibitions in an additional way: by injecting chloride ions from the impaling microelectrode into the motoneurons the inhibitory effects could be reduced or even reversed into excitatory responses, showing that the reduction in excitability was not caused by a removal of background excitation (see also *7, 11*).

Figure 2 summarizes the results of the 22 gastrocnemius-soleus motoneurons investigated in this study. For each motoneuron several recordings were made in order to obtain an average estimate of the amount of inhibition from posterior biceps stretch on repetitive firing induced by injected depolarizing current (plotted on the *y*-axis) and on repetitive firing induced by autogenetic muscle stretch (plotted on the *x*-axis). Therefore, in a situation where, for example, only presynaptic inhibition is involved, the points would fall along the *x*-axis. On the other hand, if only postsynaptic inhibition is activated, the points would fall along a line with unit slope. A line of unit slope is drawn in Fig. 2 and the points are seen to be more or less randomly distributed around this line. The ratio $\Sigma y/\Sigma x$ was calculated and found to be 1.037, indicating only an insignificant deviation from unity.

It may therefore be concluded that, even when the experimental conditions are chosen so as to favor presynaptic inhibition (*3, 4*), postsynaptic inhibition is by far the more powerful mechanism of the two in determining motoneuron activity during maintained stretch reflexes. Therefore, it may not be possible to use primary afferent depolarization and changes in the size of the excitatory postsynaptic potential produced by synchronous nerve stimulation (*1, 3*) to assess the importance of presynaptic inhibition in normal reflex activity.

D. G. GREEN*
J.-O. KELLERTH
Nobel Institute for Neurophysiology, Karolinska Institutet, Stockholm 60, Sweden

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* Postdoctoral fellow of the U.S. Public Health Service.

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Perception of Temporal Order and Relative Visual Latency

Abstract. *Judgments of temporal order to monocular pairs of flashes of equal luminance delivered at various onset asynchronies to the light-adapted fovea and periphery show that uncertainty of temporal order results when the onset of the foveal flash is delayed. Relative latencies vary as a function of peripheral (nasal vs. temporal) locus stimulated.*

When a person is stimulated by two spatially discriminable flashes of light and is asked to report which flash appeared first, his choice of one or the other alternative is generally determined by the temporal relationships between the onsets of the two stimuli. Physical changes in the direction and magnitude of the onset asynchrony between two flashes are likely to produce concomitant changes in perceived onset asynchrony, thus influencing judgments of temporal order. Maximal uncertainty about temporal order is reflected by an equal probability of a subject's choosing between the two alternatives under a given condition of stimulation. A report by Hirsh and Sherrick (*1*) presents data to the effect that maximal uncertainty regarding the temporal order of two flashes at different eccentricities obtains when they are physically simultaneous, regardless of the retinal positions of, and spatial separation between, the two stimuli. These authors concluded that a relatively fixed onset asynchrony of 20 msec is required for 75-percent-correct detection of the temporal order of two events, independent of sense modality employed and stimulus conditions.

Table 1. Onset asynchronies required for maximal uncertainty of temporal order judgments (50 percent response) and corresponding probable errors (PE). Negative asynchrony indicates that the peripheral flash occurred first.

Subject	Foveal—30° nasal pair		Foveal—30° temporal pair	
	50% (msec)	PE	50% (msec)	PE
LR	−12	16	−34	20
JR	−25	14.5	−34.5	16
ER	−60	27	−75	16
DH	−46	15.5	−57.5	17
DD	−52.5	18.5	−31	21

This study was conducted on the initial assumption that the temporal interval between the onsets of two visual stimuli which yields maximal uncertainty about their temporal order represents an estimate of the average amount of latency difference to the two flashes. The experiment was designed to investigate the dependency of judgments of temporal order on the location of the flashes on the retina. Within the framework presented here, Hirsh and Sherrick's generalization is questioned in the light of both existing reaction time (*2*) and psychophysical (*3*) measures of latency differences across the retina, and evidence that judgments of temporal order are a function of attributes of the stimulus (*4*).

The stimuli were pairs of light flashes generated by Sylvania R1131C glow modulator tubes. Each target sub-