The coactivation of antagonist muscles

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Since Sherrington’s convincing demonstration of the reciprocal innervation of opposing muscles, it has generally been thought that antagonist muscles are inactive during most voluntary movements. However, more recent evidence suggests that excitation of Renshaw cells may facilitate antagonist coactivation whereas excitation of la inhibitory neurons can induce reciprocal inhibition. A body of evidence has accumulated to indicate some of the circumstances which particularly favour the co-contraction of antagonist muscles. Isometric prehension, either in the precision grip or the power grip, can be shown to be one of the most important examples of antagonist coactivation. Studies of the discharge of single Purkinje cells of the intermediate cerebellar cortex in awake monkeys during performance of a maintained grip revealed that the majority of these neurons are deactivated during antagonist co-contraction. In contrast, other, unidentified neurons of the cerebellar cortex were as a group activated during grasping. It is suggested that the Purkinje cells act to inhibit antagonist muscles during reciprocal inhibition but are themselves inhibited during antagonist coactivation. These results support a suggestion made by Tilney and Pike in 1925 that the cerebellum plays an important role in switching between the coactivation and reciprocal inhibition of antagonist muscles.


Depuis la démonstration convaincante par Sherrington de l’innervation réciproque des muscles antagonistes, on a généralement pensé que ces muscles sont inactifs pendant la plupart des mouvements volontaires. Cependant des évidences plus récentes suggèrent que l’excitation des cellules de Renshaw peut faciliter la coactivation des antagonistes alors que l’excitation des interneuromes inhibiteurs la peut induire une inhibition réciproque. Un nombre de faits se sont accumulés pour indiquer les circonstances qui favorisent particulièrement la cocontraction des antagonistes. La préhension isométrique, soit pendant la pince de précision ou la pince de force, apparaîtra être un des exemples les plus importants de cocontraction d’antagonistes. Les études de décharge de cellules isolées de Purkinje dans le cortex cérébelleux intermédiaire chez des singes élevés pendant la pince maintenue ont révélé que la majorité de ces neurones réduisent leur décharge pendant la cocontraction des antagonistes. Par contre, d’autres neurones non identifiés du cortex cérébelleux étaient activés pendant la pince. Il est suggéré que l’activité des cellules de Purkinje participe à l’inhibition des muscles antagonistes pendant l’inhibition réciproque mais que ces cellules sont elles-mêmes inhibées pendant la coactivation des antagonistes. Ces résultats vont dans le sens d’une suggestion faite par Tilney et Pike en 1925 à l’effet que le cervelet joue un rôle important dans le passage de la coactivation à l’inhibition réciproque des muscles antagonistes.

In certain circumstances the central nervous system may independently vary both the phase and the force of contractions in mechanically opposing muscles. However, since the publication of Sherrington’s 14 classic papers on the reciprocal innervation of antagonist muscles, the cerebral capacity to shift between reciprocal and coactive control has been either denied, neglected or ignored. Moreover, the exact parameters establishing these conditions have proved elusive and the neural mechanisms have yet to be fully explained. As a consequence there has been a widespread tendency to accept the general idea that antagonist muscles cease to function when agonists contract although this is clearly a gross oversimplification of muscular activity in voluntary movements. The problem has been further compli-

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cated by the fact that several neurophysiological investigations into the neural control of antagonist muscles have become embroiled in the unproductive controversy over whether “muscles” or “movements” were represented in the motor cortex. Both Evarts and Phillips have suggested that this polemic should be laid to rest, first because it arises essentially from a false antithesis and second because it represents a general confusion over structure and function in the nervous system (Evarts 1967; Phillips 1975; Phillips and Porter 1977).

Reciprocal inhibition

It is interesting to note that prior to the publication of Sherrington’s numerous papers on reciprocal innervation, two French neurophysiologists, Beaunis (1889) and Demeny (1890), arrived independently at the similar conclusion that in most movements antagonist muscles co-contract to some extent (see also review by
prehensile force as possible. Forearm muscle activity was recorded from surface electrodes fixed in an elastic cuff over the flexors (EMGF) and extensors (EMGE) of the wrist and fingers.

Neurons in the cerebellar cortex were classified as Purkinje cells if at any time during the recording a complex action potential typical of the climbing fiber afferents was evident. All other neurons were grouped together as unidentified units, although they are likely to represent a heterogeneous category. A total of 208 cerebellar neurons were recorded, of which 91 changed discharge frequency during task performance.

The majority of the unidentified units (61 of 72) increased their discharge frequency during grasping. Eighty-six percent of the neurons were more active during the dynamic phase than during the application of static force. For these neurons, discharge frequency decreased an average of 16 spikes per second from the force ramp to the last 500 ms of maintained force, although the mean prehensile force was higher during this static period. For 13 unidentified neurons significant correlation coefficients ($p < 0.05$) were found between discharge frequency and rate of force change ($dF/dt$) measured over the dynamic phase. For 17 unidentified neurons significant correlation coefficients ($p < 0.05$) were found between the discharge frequency and force measured over the last 500 ms of the static period. Three unidentified neurons had significant correlations between discharge frequency and both prehensile force and the rate of force increase. An example of an unidentified neuron which was significantly correlated with both $dF/dt$ ($r = 0.41$, $N = 60$) and force ($r = 0.36$, $N = 60$) is shown in Fig. 2. A nonparametric statistic, the Kolmogorov—Smirnov ($K-S$) test was adapted from Mano (1979) to determine within 20 ms when the neuronal discharge frequency increased.

The $K-S$ test was used first to evaluate when each of the unidentified cerebellar units increased activity in relation to the onset of prehensile force. The distribution of the times of change for 81 cerebellar cortical neurons was widely dispersed about a mean of 250 ms before the onset of grasping. However, this observation is consistent with the scattered onset of contractions in the forearm flexor and extensor muscles of the wrist and fingers recorded from surface electrodes simultaneously with the activity of the majority of units. A computer program realigned each unit’s discharge about the onset of activity in the earliest muscle group to begin contracting, the forearm extensors. The $K-S$ test was again used to determine when the neuronal activity changed compared with the onset of forearm extensor activity. Thirty of 49 neurons changed firing frequency after the onset of activity in forearm extensor muscles and the average latency was 41 ms after the initial muscular activity. This observation raises the possibility that proprioceptive afferents may be partly responsible for changing the discharge frequency of neurons in the intermediate cerebellar cortex.

The most important single finding of the present study was that the majority of Purkinje cells (15 of 21 or about 71%) were inactivated during the application of prehensile force. This contrasts with only 15% (9 of 70) of the unidentified neurons which were similarly inactivated during grasping. An example of this reduction in Purkinje cell activity during prehension is shown in Fig. 3. This particular neuron shows an increase in complex spike activity at the same time as the simple spike response is reduced but it was the only neuron in which a statistically significant change in complex spike activity could be found. The activity of this neuron aligned about the onset of major surface activity in the forearm flexor and extensor muscles is also shown in Fig. 3. Although it appears that the decreased discharge frequency coincides best with activity in the flexor muscles, closer inspection shows that the alignment is not so much on the onset of flexor activity as it is with the period of strong co-contraction of flexors and extensors during application of prehensile force.

A significant ($p < 0.05$) negative correlation between discharge frequency and $dF/dt$ was found for three Purkinje cells and the activity of one of these was also significantly and inversely related to prehensile force (Fig. 4). The activity of this particular cell decreased proportionately as the rate of force change or force increased. The activity of this neuron aligned about the onset of force change is shown in Fig. 5. The activity histogram shows a brief excitation immediately prior to prehension. This excitation coincides with an isotonic extension of the fingers which frequently preceded the isometric grasping. Further proof was provided by inserting a pair of intramuscular electrodes into the extensor digitorum communis (EDC) muscle to record its activity with the unit discharge. Correct placement of the EMG electrodes was verified by electrical stimulation. The lower portion of Fig. 5 shows the activity in the forearm flexor muscles or the onset of activity in EDC during a first burst of activity which corresponds to an isotonic extension of the fingers. The burst of excitation in the neuron is simultaneous with the first burst of activity in EDC as the muscle contracts and stretches its antagonist. However, during isometric prehension and antagonist co-contraction, the excitation turns to a deactivation even though EDC continues to contract. Moreover, this inactivation has the same duration as the period of co-contraction.

**Discussion**

The results appear to show a clear difference in the discharge pattern among the different neurons of the
(probably incomplete) of situations that potentiate either reciprocal inhibition or antagonist cocontraction. The following conditions appear to favour reciprocal inhibition or at least antagonist alternations. (1) When external resistance prevents displacement or muscle shortening by the prime movers, the antagonists relax. However, isometric prehension is one important exception. (2) In rhythmic motor processes, such as locomotion, mastication, and respiration, agonist activity, in general, alternates with that of the antagonist. (3) In low velocity voluntary limb displacements without load, activity in antagonist muscles is usually alternated with that of the agonist.

In contrast antagonist co-contraction seems to occur most often in the following circumstances. (1) When muscular tension or limb position must be precisely monitored without load, the antagonists co-contract. The initial phase of learning a new motor skill may be an example of this co-contraction. (2) In higher velocity limb displacements or under loaded conditions, the antagonist muscles, after a short period of inactivity, contract strongly to decelerate the limb. (3) Isometric prehension of the hand in either the precision or power grip requires antagonist co-contraction to stabilize the wrist and to add stiffness to the carpal and metacarpal—phalangeal joints.

**Somatic afferents essential for prehension**

Thus far no mention has been made as to what the somatic afferents needed to elicit and sustain prehension really are. Evidence from Taub and his collaborators on the effects of limb deafferentation in primates suggests that animals that have learned to make grasping movements before rhizotomy will spontaneously relearn to grip objects with the hand (Taub 1976) and that these animals can develop as much prehensile force as a normal animal (Taub et al. 1966). In contrast, for animals deafferented prenatally prehension does not appear spontaneously and instead these monkeys must be taught grasping by a progressive shaping process (Taub 1981). It appears that although somatic afferents are especially needed for the acquisition or learning of prehension skills, under very special conditions the motor systems can be trained to produce the synergism needed for grasping without the benefit of this topographic feedback (Taub and Berman 1968).

Both in the healthy neonatal primate and in adulthood after certain lesions of the frontal lobe, particularly the supplementary motor area and lateral area 6, an involuntary reflex known as forced grasping can be elicited. This reflex involves the claspign of any object that contracts the palm or fingers of the hand. As a pathological sign, it is not yet clearly established whether this forced grasping is a focal or diffuse sign of cortical damage (Adie and Critchley 1927; Penfield and Welch 1951; Rushworth and Denny-Brown 1959; Bourbonnais et al. 1979). Rushworth and Denny-Brown (1959) showed that cutaneous sensation is of crucial importance to eliciting the grasp by abolishing the reflex with local anesthetic injected into the volar surface of the hand. However, it was necessary to apply stretch to the finger flexor muscles to maintain the grasping. Goldberger (1972) and Bourbonnais et al. (1979) both found that monkeys trained to make isometric grasp responses not only increased the rate of force application and the strength of prehension after lesions of the frontal cortex but also the same animals failed to release after initiating voluntary grasp. Although in this instance no imposed stretch was applied to the agonist muscles and the increase of muscle tension was centrally triggered, it is nevertheless likely that the spindles are activated by fusimotor drive in isometric contractions of finger muscles (Valbo 1971). Taken together these particular observations suggested that feedback from muscle tension and stretch receptors may play an important feedback role in controlling forces between co-contracting antagonists muscles in prehension.

**Cerebellar cortical activity in prehension**

The experiments I am about to describe were conducted in collaboration with Mr. Daniel Bourbonnais. In these studies three monkeys (Macaca fascicularis) were trained to exert an isometric precision grip of the thumb and forefinger on a hand-held strain gauge. The monkeys were required to maintain a constant force between an upper and lower threshold, indicated by a tone, for a 1-s duration. A dynamic phase of force application was defined as the period from the onset of force change until the crossing of the lower threshold and a static phase was selected as the last 500 ms of the maintained force period. Further details about this task have been published elsewhere (Smith et al. 1975; Smith 1979).

For two of the three monkeys as well as a fourth animal not used for cerebellar recording, extensive EMG recordings were made from the individual intrinsic and extrinsic muscles of the hand. The activity of 24 of these muscles on 20 rewarded trials of maintained prehension is shown in Figs. 1a and 1b. It can be seen that the precision grip in the macaque involves approximately the same degree of antagonist cocontraction as prehension in man.

Recording of single neurons in the cerebellar cortex began 1 or 2 days following the surgical preparation. A search was made for that region of the intermediate cerebellar cortex along the border between the anterior and posterior lobes which contains units with receptive fields on the wrist and fingers. Whenever possible single cell discharge was recorded at as many levels of
continued to precede activation of the agonist. This proved that the stimulus instigating the inhibitory reflex chain originated in the central nervous system, not from spindles or tendon organs in the contracting agonist muscle. It is important to note, however, that none of the neurons recorded in the motor cortex after deafferentation and cerebellctomy showed a clear reciprocal discharge pattern with flexion and extension movements.

Co-contraction of antagonist muscles

Tilney and Pike (1925) were the first to generally oppose Sherrington’s concept of reciprocal innervation as a general reflex model for voluntary movements. Their objection was based on an analysis of muscle tension changes in tibialis and gastrocnemius muscles evoked by stimulation of motor cortex as well as during spontaneous movements in the lightly anesthetized animal. They concluded that “muscular co-ordination depends primarily on the synchronous co-contraction relation in the antagonist muscle groups.” The following year Wachholder and Altenberger (1926) found that when subjects were told to “loosely” flex and extend the elbow only agonist activity was recorded but when the subjects “stiffened” the limb activity appeared in the antagonist muscle as well. These conditions are very similar to those used some years later by Hammond (1954) who instructed subjects to “resist” or “let go” to a stretch of the biceps muscle. Hammond’s data provided the first indication of what we recognize today as the long loop reflexes (Desmedt 1978). Levine and Kabat (1952) pointed out that silence in the antagonist is not synonymous with inhibition and that reciprocal inhibition is a relative phenomenon resulting from the modifying influences of supraspinal centers on segmental reflexes. Barnett and Harding (1955) specifically designed an experiment to measure antagonist activity during both active and passive extension of the elbow. Surprisingly they found that both active and passive conditions at higher velocities produced some contraction in the biceps muscle due, they presumed, to activation of the stretch reflex. Moreover, in general, slow voluntary movement of the unloaded limb produced reciprocal inhibition of antagonists whereas at moderate or high speeds against resistance the antagonists either briefly relaxed then contracted strongly or, in over 50% of their recordings, both opposing muscles contracted simultaneously (Barnett and Harding 1955). The simultaneous co-contraction of elbow antagonist muscles during voluntary movements in man has now been confirmed by others to augment in proportion to both increased movement velocity (Bouisset and Lestienne 1974; Lestienne and Bouisset 1968) as well as with higher displacement loads (Patton and Mortensen 1971). This contraction of the antagonist should not be considered paradoxical as it is nothing more than a lengthening contraction which has been frequently observed to increase muscular force in the cat hind limb in preparation for jumping (Walmsley et al. 1978; Zomlefer et al. 1977).

More recently Bizzi and Polit (Bizzi and Polit 1979; Polit and Bizzi 1979) have demonstrated that deafferented animals may correctly preprogram movements to a given limb position by specifying the final equilibrium tensions between antagonist muscles. Although this programming of position could be achieved by an alternate activation of agonist and antagonist muscles it could be more easily controlled by simultaneous co-contraction of antagonists.

Antagonist co-contraction in prehension

In primates the isometric clasping functions of the hand are among the best examples of coactivation of antagonist muscles. Napier (1956, 1960) has suggested that the prehensile repertoire of both monkeys and man may be divided into either a class of power grips or precision grips. The former is associated with the exertion of greater forces in which all the fingers serve to grasp an object against the palm. In the precision grip, by contrast, the compression forces are generated between the thumb and fingertip. Landsmeer (1962) and Long et al. (1970) have further distinguished between precision handling used in fine manipulatory finger movements and pinches in which control of the clasp force is the primary purpose. Muscular contractions in both the power grip and pinching are static and isometric as opposed to the dynamic and isotonic actions in fine finger movements (Long et al. 1970). Extensive electromyographic (EMG) recording from the forearm and the hand during both the power grip and pinching have shown that almost all the extrinsic and most of the intrinsic muscles are active during the exertion of higher compression forces (Basmajian 1978; Long et al. 1970; Rasch and Burke 1974). Of course not all of these muscles are acting as prime movers. Some, such as the extensors of the wrist, provide important postural stabilization to increase the mechanical advantage of the long flexors of the fingers whereas other muscles contract to provide the appropriate stiffness at the carpals, metacarpals, and phalangeal joints. These complex patterns of synergic coactivation of antagonist muscles probably evolved in parallel with the changes in hand morphology as an adaptation to the arboreal environment of early primates (LeGros-Clark 1959).

Conditions favouring either reciprocal inhibition or antagonist co-contraction

From this brief survey of various experiments examining the behaviour of antagonist muscles in voluntary motor control, I have attempted to extrapolate a list
Tilney and Pike (1925). However, much of our current thinking about the inhibitory relations between antagonist muscles can be attributed directly or indirectly to the ideas and experiments of Sherrington. Apart from the examples taken from the autonomic and ocoulomotor systems and the examples of direct inhibitory action on muscles found only in invertebrates, Sherrington studied essentially three instances of simple (i.e., not successive or alternating) reciprocal innervation in skeletal muscles. Two of those were segmental reflexes, the limb flexion and the knee jerk, which were studied exclusively in either spinal or decerebrate preparations. It was observed that the same stimulus which elicited agonist muscles activity also relaxed and inhibited the antagonist. When the antagonist tonus was particularly great as in decerebrate rigidity, the extensor inhibition was sometimes found to precede activity in the agonists of the limb flexion reflex (Sherrington 1906).

Contemporary neurophysiology can now explain in considerable detail how these reflexes are mediated within the spinal cord. The existence of inhibitory neurons receiving monosynaptic excitation from primary muscle stretch receptors and exerting a direct inhibition on motoneurons of the antagonist muscles had been postulated for some time but the first unequivocal identification of these neurons was achieved by Jankowska and Roberts (1972a, 1972b). These studies clearly provided the anatomical and physiological substrate for the reciprocal relation that Sherrington had observed many years earlier. Further experiments by Hultborn (Hultborn 1972, 1976; Hultborn et al. 1976) have added considerably to our knowledge about the interconnections of the Ia inhibitory interneurons both with segmental as well as supraspinal elements. Renshaw cells, for example, which are inhibitory interneurons driven in part by motoneurons, have now been shown to be at least partly under supraspinal control. Hultborn (1972, 1976) has suggested that the Renshaw and Ia inhibitory neurons are in balance and that excitation of the Renshaw cells will favour co-contraction of the antagonists whereas excitation of the Ia inhibitory interneurons will inhibit the antagonist in proportion to the intrafusal excitation of the agonist.

Sherrington’s third example of reciprocal innervation was derived from electrical stimulation of the “appropriate focus” of the contralateral motor cortex which produced an “immediate relaxation of the biceps with active contribution of the triceps” (Sherrington and Hering 1897). This assertion led to some considerable controversy about how electrical stimulation of the motor cortex evokes muscular activity and it was even felt by some to provide support for Hughlings Jackson’s statement that “the central nervous system knows nothing of muscles, it only knows movements” (quoted by Phillips 1973). In addition, a more specific debate centered about, first, the more tangible question as to whether punctate liminal electrical stimulation applied to motor cortex elicits either reciprocal inhibition or co-contraction in mechanically opposing muscles or simply single muscle activation (Chang et al. 1947; Hines 1944; Leyton and Sherrington 1917; Tilney and Pike 1925). However, more recently a second controversy has arisen as to whether corticospinal neurons are depolarized synthetically or directly by electrical stimulation (Anderson et al. 1975; Asanuma and Rosen 1972; Jankowska et al. 1975a; 1975b; Phillips and Porter 1977).

In spite of his conviction that reciprocal innervation was the important reflex mechanism underlying movement, Sherrington did admit that “under some certain forms of cerebral action true antagonistic muscles can be thrown synchronously into contraction” (Sherrington 1906). This antagonist coaction he called “double reciprocal innervation” (Sherrington 1909). The muscular contractions, he postulated, would generate less muscular force because of the double action of the reciprocal inhibitory reflexes, an interesting hypothesis which to my knowledge has never been tested. Nevertheless it is precisely these “certain forms of cerebral action” leading to either reciprocal inhibition or coactivation of muscles that shall be the principal theme of this paper.

Lashley (1951) pointed out some years ago that there are numerous examples of motor skills involving very high speed movements which are too rapid to be triggered in serial order by exteroceptive or proprioceptive afferent cues from the moving limb. Instead, some internal program must generate the pattern of excitation and inhibit needed to produce these movements. This implied that in order to execute some fast movements a central, probably cortical, source of reciprocal inhibition as suggested by Sherrington would be indispensable. Experimental evidence for Sherrington’s central inhibition has been shown for both man (Hallett et al. 1975a; Hufschmidt and Hufschmidt 1954; Simoyama and Tanaka 1974) and monkey (Evarts 1972a, 1972b). These studies all demonstrated that inhibition of agonist muscle activity can precede activity in the agonist. In each of these investigations the subjects were instructed or conditioned to contract the antagonist muscle against a tonic load and, after the presentation of a conditioned stimulus, they were required to activate the agonist as quickly as possible. It may be that these particular conditions are especially optimal for a brief reciprocal inhibition of central origin. The central origin of this inhibition has been shown by Lamarre et al. (1978) who found that after complete upper limb deafferentation the inhibition of the antagonist con-
Fig. 1. (a and b) Activity of 24 muscles of the hand during 20 maintained precision grips. All traces have been aligned on the reward.
Fig. 2. Illustrative data from unidentified unit in cerebellar cortex No. 163. (Upper left) Three photographed records of unit discharge and finger force traces. The onset and duration of the light (a “go” signal), tone, and reward are also shown. Immediately below is a computer-generated histogram of activity on 30 trials aligned on delivery of reward. K–S indicates the time of occurrence of statistically significant change in activity. (Upper right) Force traces of 30 finger pressures have been aligned about the onset of force change. Immediately below is the similarly aligned unit activity raster, and below that the summed raster is shown as a histogram. (Lower left) Surface activity of flexors on the same 30 trials aligned on the onset of activity in this muscle group. Below is shown the unit activity raster and summed histogram similarly aligned. (Lower right) A similar alignment on the onset of surface activity in the forearm extensor muscles.
FIG. 3. (Upper left) Three photographed trials with force traces and stimulus events for Purkinje neuron No. 148; climbing fiber discharges are shown by dots. Immediately below is the histogram of unit activity on 26 trials aligned on the reward delivery. (Upper right) Finger pressure traces aligned about the onset of force change. Immediately below is the neuron activity raster and summed histogram aligned similarly. (Lower left) Surface forearm flexor EMG activity aligned about the onset of major flexor activity. Immediately below is the unit activity raster and summed histogram activity raster and histogram.
cerebellar cortex. Purkinje cells, identified by the characteristic climbing fiber discharge, were, in general, inactivated during performance of the maintained precision grip. In contrast, the majority of the remaining unidentified neurons increased discharge frequency during prehension. Although identification of this group of neurons will be the focus of further studies, a small number of the unidentified neurons might be Purkinje cells for which a climbing fiber discharge was not seen due to the position of the electrode near the distal dendrites (Houngsgaard and Yamamoto 1979). However, most of the action potentials of cells in this group had conspicuous inflections (i.e., "notches") suggesting that the initial segment and somatodendritic portions of the spike were both visible.

If it is assumed that recordings from granule cells would be unlikely to give large unitary potentials, the basket, outer stellate, and Golgi cells are the most probable candidates for the unidentified neurons. Moreover, as the basket and stellate cells are known to inhibit Purkinje cells and the Golgi neurons disfacilitate Purkinje cells through glomerular inhibition the contrast between the two different discharge patterns appears reasonable.

It was a consistent finding that almost all the intrinsic and extrinsic muscles of the hand were activated during performance of the maintained precision grip. Virtually no agonist or synergistic pair of muscles was seen to contract reciprocally during isometric prehension. This somewhat unique aspect of the present motor task, coupled with the fact that most other studies of Purkinje cell discharge have examined activity during movements associated with the reciprocal inhibition of antagonist muscles (Harvey et al. 1977; Llinas and Wolf 1977; Mano 1979; Mano and Yamamoto 1980; Thach 1970), suggests the following hypothesis about the intermediate zone of the cerebellar cortex.

It is postulated that the intermediate zone acts to inhibit antagonist muscles when reciprocal inhibition of muscles is required. Activation of the Purkinje cells is thought to occur, in part, as a result of muscle afferent excitation from either the antagonist stretch during movement or fusimotor activity in isometric contractions. The Purkinje cell hyperpolarization of nuclear cells and subsequent disfacilitation of neurons projecting to the spinal cord (Toyama et al. 1968; Tsukahara et al. 1964) are hypothesized to ultimately remove excitation from antagonist muscle motor-neurons. In isometric prehension, or any instance of co-contraction of antagonist muscles, it is suggested that afferents from each member of the antagonist pair converge on basket and outer stellate cells to remove the Purkinje cell inhibition of the deep nuclei. A diagram based on a schema from Eccles et al. (1967) has been modified to illustrate how this mechanism might function (Fig. 6). This diagram is intended to explain both the burst of excitation from the Purkinje neuron illustrated in Fig. 5 during isotonic contraction of EDC and stretch of antagonist finger flexors, as well as the marked deactivation of discharge frequency during the antagonist co-activation phase of isometric prehension.

The idea that the cerebellum plays an important role in switching between reciprocal activation and antagonist co-contraction is neither new nor original. It was very explicitly stated by Tilney and Pike (1925) that following "deep lacerations of the cerebellum... The character of the disturbance determined by a cerebellar lesion is especially worthy of note. These disturbances are primarily in the nature of actual dissociations in the operations of the synergic unit. Such dissociations declare themselves by alterations in the degree of contractions and the timing of contractions... In either event the disproportion in the degree of tension increment in the two [antagonist] muscles of the synergic unit is at once obvious..." Unfortunately, this idea met with early, and in my opinion, misdirected opposition from various people in attendance at the combined meeting of the Neurology Section of the Royal Society of Medicine and the American Neurological Association Symposium on the cerebellum in 1927. Pollock
Fig. 5. Activity of Purkinje neuron No. 112 shown in Fig. 4. (Upper left) Three photographed trials with force traces and stimulus events; climbing fiber discharges are indicated by dots. Immediately below is a histogram of summed activity on 18 trials aligned on the delivery of reward. (Upper left) Force traces, unit activity raster, and summed histogram aligned on force onset (Lower left) Surface forearm flexor activity aligned about onset of activity, in this muscle group. Immediately below is similarly aligned unit activity raster and summed histogram. (Lower right) Twenty-two reward trials on which intramuscular activity from EDC was recorded. Alignment is about onset of activity in this muscle. Immediately below is unit activity raster and summed histogram. Note that unlike previous figures, flexor and extensor activity were not recorded simultaneously.
and Davis (1927) reported that cerebellar ablation in the decerebrate cat had no effect on the co-contraction of antagonist ankle muscles elicited by electrical stimulation of the foot, an observation they apparently felt conflicted with Tilney and Pike (1925). Kinnier Wilson in attendance at the Symposium noted later (Wilson 1928) that Tilney and Pike ‘‘... do not appear on the clinical side to have covered all the possibilities of antagonist action, since no investigation, seemingly, either of voluntary movement against strong resistance or of antagonist co-operation, has been made.’’ This criticism incidentally applies equally to Sherrington’s 14 classic studies of reciprocal innervation as well. Walshe (1927) was more openly critical, objecting that ‘‘the essential difficulty we have in accepting Tilney’s hypothesis as it stands is the statement that the relations of agonists and antagonist... are a function of the cerebellum and are profoundly upset when this organ is out of action,’’ although he offered no evidence to substantiate his opinion. Finally Holmes (1927) stated ‘‘I have examined, [the effects of more than eighty gunshot wounds of the cerebellum] especially since the appearance of Tilney and Pike’s paper, the relations of various groups of agonists and antagonists both by direct observation and by graphic methods but have been unable to detect any definite disorder in these relations.’’ Unfortunately the general consensus of this international opinion consigned the Tilney-Pike hypothesis to general disfavour and it has seldom been mentioned ever since.

More modern studies, however, may have differed about the details of the motor deficits following cerebellar lesions but, in general, the essence of the Tilney-Pike hypothesis, i.e., that the cerebellum is essential for the smooth transition between antagonist co-contraction and reciprocal inhibition, has been vindicated. For example, Terzuolo, et al (1973a, 1973b) have demonstrated that in ballistically initiated movements performed by monkeys the agonist-antagonist reciprocity is abolished after cerebellar ablation. In man, Hallett et al. (1975b) have replicated these results in studies of patients with cerebellar lesions. Moreover, a similar delay in timing of alternating agonist-antagonist EMG activity has been seen in forelimb muscles of both cat and monkey subjected to reversible cerebellar cooling (Burton and Englehardt 1977; Vilis and Hore 1980).

A more direct test of the hypothesis that Purkinje cells are inhibited during the co-contraction of antagonist muscles and excited during reciprocal inhibition comes from Murphy et al. (1973a, 1974b). These authors found that separate stretches of each muscle in an antagonist pair produced an excitatory burst of activity in Purkinje cells. This would suggest that there may be at least as much convergence of antagonist muscle afferents upon Purkinje cells as for other neurons of the cortex. However, only one neuron in these studies (Fig. 3 of Murphy et al. 1973b) was shown to be excited by simultaneous stretch of two antagonist muscles. Moreover, although this neuron was called a Purkinje cell it was not clear whether the identification was made on the basis of an apparent climbing fiber discharge or simply inferred on the basis of appropriate depth within the cerebellar cortex, a difficult distinction because Purkinje and basket cells lie very close to one another. Nevertheless, these experiments on antagonist muscle response to stretch are extremely valuable and it is hoped that further studies will continue to investigate these important functional relations.

Although it may be premature to propose a specific function for the intermediate cerebellar cortex at least two recent extensive reviews of cerebellar function
(Brooks 1979; Massion and Sasaki 1979) have emphasized that the intermediate and neocerebellum play an important role in the selection of either antagonist reciprocity or co-contraction in voluntary movements. The data from the present study suggest that further investigation of this hypothesis may well be profitable.

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