The Cerebellum and Initiation of Movement:
The Stretch Reflex

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Studies of the stretch reflex in decerebrate cats indicate a phase advance of peak sinusoidal
tension in steady-state cycles between 0.1 and 10 Hz. This phase advance is reduced in acute and
chronic cerebellotomy, as shown in previous investigations. Also, the augmentation of muscle
peak tension in initial sinusoidal stretch cycles at 0.5–5 Hz has been found to be reduced during
the time of reflex and motor instability in the several months following cerebellar ablation.

This report shows the increased amplitude and phase lead of integrated electromyographic
activity in initiating sinusoidal stretch cycles in the decerebrate cat. These reflex aspects are
demonstrated in relation to the discharge of neurons in the dorsal spinocerebellar tract and of
cerebellar cortical Purkinje cells in initial sinusoidal cycles. The intensity and phase advance of
the discharge in dorsal spinocerebellar tract neurons is altered little, but these features are usually
increased in Purkinje cells during initial stretches compared to continuous cycling.

In terms of overall motor control, these findings are compatible with concepts of movement
control, modulated by the cerebellum, in which the discharge of antagonist motor neurons is
regulated in concert with that of agonist muscles upon initiation and termination of movement.

INTRODUCTION

During the investigation of dynamic stretch reflex responses in the decerebrate cat
in the Neurology Laboratory at Yale in the 1960s, Higgins, Partridge, and Glaser
observed that reflex sinusoidal tension was greater in initial stretches of a series than in
later steady-state cycles at a particular stretch frequency [1]. The peak tensions and
sometimes the phase lead were increased in initiating stretch cycles from a standing
start. The effect was seen to be present over several cycles at 1 Hz, and sometimes
extending to ten or more sinusoidal stretches. This augmentation of reflex muscle
tension was best developed in the frequency band of .5–5 Hz, and it could be abolished
by acute or chronic removal of the cerebellum in the cat. Chronic decerebellation was
attended by several months of instability of posture, gait, grooming activity, and
feeding followed by steady recovery [2]. This type of recovery has been termed
"cerebellar compensation" and presumably occurs because motor centers in the
forebrain, including motor cortex, assume some aspects of cerebellar control in posture
and movement.

As shown in animals decerebrated weeks or months after total cerebellotomy, the
most severe motor instability was associated with the loss of augmentation of reflex
sinusoidal tension in initial cycles. The period of compensation was accompanied by
a gradual and partial return of the augmentation of peak force. Glaser reported these
findings at the Nobel Symposium, Muscular Afferents and Motor Control, Stock-
holm, in 1965 [3]. This symposium also included a presentation by Van Der Meulen et

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al., concerning the reduction or loss of fusimotor drive on muscle spindles and reduction of spindle sensitivity in the course of chronic cerebellar removal [4]. This reduction of spindle sensitivity was subsequently shown by Gilman and Ebel [5], to be due to dis-facilitation of gamma efferent neuronal discharge associated with loss of cerebellar function. Granit et al. had reported a decade earlier on the subservience of the alpha and gamma motor neurons to cerebellar control [6]. While there has been a great deal of interest in physiological investigations of cerebellar control of voluntary movement, there has been only infrequent reference to effects on spinal centers, particularly the stretch reflex.

METHODS

These experiments were carried out over a number of years on the classic Sherringtonian decerebrate cat preparation following a suitable period of general anesthesia to perform craniotomy, transect the midbrain at the rostral superior colliculus, and remove all brain rostral to that point. The care and preparation of the animals conformed to the guidelines of the American Physiological Society. The methods of recording and preparing the experimental controls have been outlined in previous reports [1, 2, 7]. In most experiments, muscle stretch was applied by a servomotor linkage, under electronic control, at the tendon of triceps surae in the hind limb. Electromyographic (EMG) recordings were made with monopolar electrodes inserted into the muscle being stretched, while neuronal unit recordings were made either from single axons of the ipsilateral dorsal spinocerebellar tract or from individual neurons in the ipsilateral cerebellar cortex (Larsell lobule IV, anterior lobe). The recordings from the spinocerebellar tract were mostly intra-axonal, while those in the cortex were extracellular, using a potassium chloride-filled glass microelectrode. Neuronal units were identified by their characteristic discharge at rest and with muscle stretch and by their responses to volley stimulations at the periphery or in central tracts. Neuronal discharge data, as well as recordings of muscle length, force, and EMG activity, were made on FM magnetic tape. The data were then analyzed with a small averaging computer over repetitive stretch cycles. This technique permitted study of accumulated impulse occurrence within certain time bins related to the stretch cycle and the observation of histograms of interimpulse intervals at rest and during sinusoidal stretch. EMG activity was more easily correlated with the stretch cycle if electronically integrated to indicate more clearly the onset of peak response.

RESULTS

The onset of sinusoidal stretch is accompanied by an increase in reflex tension, as shown in Fig. 1 (the vertical calibration line indicates 1,000 grams). This increase is preceded by a burst of EMG activity in each stretch cycle. The EMG is shown as a rectified and integrated millivolt signal at B and D of Fig. 1 (the vertical calibration line indicates 500 millivolts per second). These representative sequences are shown for stretches at 1 and 3 Hz and peak amplitudes of 6 mm. The initial cycles show an augmentation of tension at the peak compared to cycles which have attained a steady state. The integrated EMG activity, obtained by monopolar needle electrode in gastrocnemius muscle, shows a sharp rise in early cycles, especially at 3 Hz. This reflex response appears early in each stretch cycle and is very much phase-advanced over both length and tension. For example, in the initiating cycles, the phase advance of most rapidly rising EMG activity in each cycle is usually in excess of 120 degrees
ahead of the length peak. The tension peak is usually around 20–24 degrees in advance of length. This phase advance for tension or EMG in initiating cycles is, however, not appreciably increased over that of later cycles. The magnitude of peak tension and phase advance is shown in the graphs at Fig. 2 for the later, continuous (steady-state) cycles at several frequencies. In Fig. 2 the gain magnitude at A is shown as a logarithmic expression of the ratio of peak output tension to peak input length (g/mm) in decibel units. As shown in the previous studies [1,2,3], the gain magnitude for muscle peak tension and phase advance are both reduced in steady cycles after cerebellectomy. Therefore, it was recognized that not only was there a loss of augmentation of tension in initiating cycles of stretch following removal of the cerebellum, but there was also a continuous reduction in the responsiveness of the stretch reflex to dynamic input.

In Fig. 2, the tension notation on the y-axis refers to the gain of the stretch reflex. The gain curve is drawn for a 1 mm stretch. The other points are for larger stretches, as indicated. The gain curves cannot be superposed at these several amplitudes, reflecting a degree of non-linearity in stretch reflex responses. While data for initiating cycles in a stretch sequence are limited, they imply a greater gain and a slightly greater phase advance overall, particularly during stretching at rates exceeding 1 Hz. It is probable that the reflex tension developed during initiating cycles of stretch reflects some of this non-linear stretch reflex behavior.

In the course of recording neuronal impulse trains in response to muscle stretch in
FIG. 2. Harmonic analysis of stretch reflex responses. Muscle force (log grams at peak) vs. stretch frequency at 1, 2, 5, and 10 mm amplitudes. Steady-state conditions for gain (A) and phase (B) of reflex responses (see text). Solid lines drawn through 1 mm data points.

the dorsal spinocerebellar tract or at the cerebellar cortex, occasional detailed observations were made of initial cycles in a series of stretches. Figure 3A shows the discharge pattern of a neuron in the dorsal spinocerebellar tract in initial cycles. This neuron, electrically identified as to its cerebellar terminus and its response to peripheral stimulation, had a major input from spindle primary afferents. The discharge pattern varied little over the course of ten initial stretch cycles and was found consistent with that in multiple steady-state cycles in the same preparation.

On the other hand, the discharge of a Purkinje neuron shown in Fig. 3B, while much more irregular than that of the secondary afferent neuron, was modified in early stretch cycles compared to the steady state. The irregularity of the simple spike inter-impulse intervals and the occurrence of complex spikes (climbing fiber discharge, olivocerebellar projection) in Purkinje cells make it difficult to locate the center of the most intense discharge. However, Fig. 3 does show an increased phase advance of Purkinje neuron discharge, especially in the first through the fourth cycles. The solid circles indicate an initial shift of response intensity toward the 90-degree phase-lead point in the stretch cycle, followed by a shift away from it.

In Fig. 4, the discharges of a neuron of the dorsal spinocerebellar tract and those of a Purkinje neuron have been collected into computer time bins in relation to a 1 Hz continuous sinusoidal stretch of the ipsilateral triceps surae. Numerous cycles usually have to be collected in order to see a relation between impulse occurrence and the sinusoidal input because of the variability in the discharge of many of these neurons. In the lower part of Fig. 4, impulse interval histograms, obtained during initial sinusoidal stretches for the same neurons shown above, indicate that the discharge intensity and pattern of the dorsal spinocerebellar spike train remain essentially unchanged at the
peaks. However, the intensity of peak discharge of the Purkinje neuron is increased in initial cycles (shown in Fig. 4D).

The harmonic analysis of steady-state cycles for a Purkinje neuron reveals a pronounced responsiveness at stretch frequencies above 1 Hz (Fig. 5). In Fig. 5, the logarithmic expression in decibels on the y-axis represents the gain of the response. The gain has been derived from measurement of the impulse frequency at the peak of neuronal discharge divided by the amplitude of the sinusoidal muscle stretch. It is difficult to measure much effect below about .1 Hz and this fact is shown as an approximation by the nearly vertical dotted lines. The phase lead of the peak Purkinje cell discharge is greater than that seen with neurons of the dorsal spinocerebellar tract which receive a principal input from spindle primary endings. The velocity sensitivity of muscle spindle primary endings is closely reflected in the discharge properties of neurons in the dorsal spinocerebellar tract [7]. These neurons often show a peak discharge with an approximate 45-degree phase advance over length input. However, the Purkinje cell discharge can attain a peak phase lead that is even greater. This lead is enhanced especially during initial stretch cycles from a standing start.

DISCUSSION

In connection with studies of clinical cerebellar ataxia, Holmes [8,9] and, more recently, Marsden et al. [10] and Hallett et al. [11] demonstrated a delay in the onset
or the termination of human voluntary movement on command. Literature pertaining to animal experiments, mainly in the monkey, has stressed the importance of cerebellar discharge over the dentato-rubrothalamic pathway in the co-activation of alpha and gamma motor neurons in the voluntary control of movement [12,13]. While the precise control of movement is disrupted by breaking the alpha-gamma linkage maintained by neocerebellar structures, muscular reflex control may also be impaired by a similar disturbance in the paleocerebellum and its regulation of brain stem and spinal reflexes. This aspect of cerebellar regulation is emphasized in animal experiments, such as treadmill studies of gait function in the mesencephalic cat preparation [14,15]. Not only is the afferent input to the cerebellum from spinal centers closely linked to actions of various muscle groups in gait [16], but cooling or removal of part of the anterior lobe has been found to affect the orderliness of gait motion [17,18]. In addition, the gait-modulated discharges of static and dynamic gamma motor neurons in premamillary cat preparations reveal extensive changes in the excitability of these neurons apart from any consideration of alpha-gamma linkage at the level of cerebral cortex [19]. It is probable that the red nucleus and other brain stem structures are directly involved in the cerebellar modulation of decerebrate motor response [14,15] and also in setting the status of spinal motor reflexes preparatory to maintained posture and movement.

Studies of the initiation of movement in animals have largely reported the behavior
of lateral cerebellar neurons (Purkinje cells) in response to learned movement patterns in the intact and unanesthetized monkey [13,20]. These and similar observations have confirmed the general theoretical interpretation of cerebellar function as principally occurring in the thalamo-cortical integrating systems for voluntary motor function (dentato-rubrothalamic pathway). However, investigations of intermediate cortex Purkinje neurons in the monkey have shown a linkage with motor performance and learned movement patterns [20]. Furthermore, it has recently been shown that neurons of the dentate and interpositus nuclei have a bidirectional linkage of their discharge with learned movement patterns, the dentate discharges preceding those of the interpositus [21]. In terms of stretch reflex activity, it has been proposed that the alpha-gamma motor neuron linkage occurs at the motor cortex, at least in man and primates. In this view, the co-activation of various fusimotor and extrafusal motor neurons can occur in several ways to produce accurate, voluntary movement.

There has been little attention given to probable descending cerebellar influences on brain stem and spinal centers involved in initiating movement. However, evidence for cerebellar control of the gain of stretch reflexes has appeared in connection with several studies [22,23,24]. There is some evidence that the cerebellum regulates the gain of stretch responses in antagonist muscles so as to assist in the initiation and termination of movements led by agonist groups. Other evidence for cerebellar regulation of gain in simple reflexes has been presented for the pupillary light reflex and the optokinetic response [25,26]. Finally, the discharge of Purkinje neurons and other cerebellar cortical elements to sudden velocity inputs to muscle has been reported.

FIG 5. Harmonic analysis of peak discharge of a Purkinje neuron excited by sinusoidal stretch of ipsilateral triceps surae. Vertical axes: Gain (A) and phase angle (B) of average peak instantaneous impulse frequency (IPF) over several muscle stretch cycles. Horizontal axis: Sinusoidal stretch frequency in Hz (see text).
These studies indicate that the discharge of certain Purkinje cells in the vermis and intermediate cortex is consistently modified by either simple spikes or complex spikes, in response to velocity input.

The studies summarized here indicate a specific cerebellar response to muscle stretch and a regulation of lower centers in the course of the initiation of movement. Rubrospinal, reticulospinal, and vestibulospinal tracts are probably the principal pathways over which these effects on dynamic stretch responses occur. While the cerebello-thalamo-cortical projection from the massive lateral cerebellar structure is probably the major component in the voluntary regulation of movement, the outflow from vermis and intermediate cortex of the anterior lobe appears to have a major ancillary role in the control of the reflex substrate upon which voluntary action occurs. The degree to which this occurs and its importance in initiating movements remains to be investigated further in relation to the course of events in the stretch reflex and the associated neuronal discharges which modify the cerebellar outflow to brain stem and spinal centers. In particular, cerebellar regulation of the antagonist motor neuron reflex discharge in concert with that of the agonist muscles has a direct relation to the studies described here for the initiation and termination of movement.

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