

H reflex studies in patients with cerebellar disorders

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The H reflex was described by Hoffman (1918) and it has been shown to have the characteristics of a monosynaptic reflex (Magladery, Porter, Park, and Teasdall, 1951; McLeod and Van Der Meulen, 1967). The time-course of recovery of the amplitude of the H wave after a conditioning volley was employed first by Magladery, Teasdall, Park, and Languth (1952) as a means of measuring motoneurone excitability in man. McLeod and Van Der Meulen (1967) studied the H reflex under experimental conditions in the cat, and concluded that if minimal stimulus intensities were employed the initial period of unresponsiveness in the recovery curve was an index of alpha motoneurone excitability.

The object of the present work was to investigate by means of the H reflex the state of alpha motoneurone excitability in the spinal cords of patients with cerebellar disorders. It has been shown in animals that the cerebellum influences alpha and gamma motoneurone activity at a spinal cord level (Granit, Holmgren, and Merton, 1955; Calma and Kidd, 1959; Van Der Meulen and Gilman, 1965) and it seems likely that disordered function of the alpha and gamma motor systems is partly responsible for the hypotonia and the incoordination of voluntary movements which are such striking clinical features of cerebellar disease in man.

METHODS

Twenty-two control subjects in whom there was no evidence of neurological disease, and 10 patients with cerebellar disorders were studied. None of the patients with cerebellar disorders displayed clinical evidence of pathological involvement of the brain-stem, corticospinal tracts, or peripheral nerves.

H waves were recorded from the calf muscles with surface electrodes using a method similar to that described by Mayer and Mawdsley (1965). The subject lay comfortably prone, in a relaxed position with the leg and thigh firmly supported; the foot hung freely, with its dorsum approximately at right angles to the tibia. Two silver recording electrodes, approximately 5 mm in diameter were strapped firmly to the calf. The proximal

recording electrode was positioned in the midline, at the junction of the gastrocnemius and soleus muscles, approximately 15 to 20 cm distal to the mid-popliteal point; the distal electrode was placed over the tendo Achillis. The medial popliteal nerve was stimulated through a bipolar electrode strapped very firmly to the popliteal fossa. A square-wave stimulus of duration 0.2 msec was derived from a Grass S4 stimulator or from a Disa Ministim. The action potentials were led off from the recording electrodes through amplifiers and displayed on the upper beam of an oscilloscope; a time scale was displayed on the lower beam. Photographic records were made on 35 mm film.

In each case the position of the stimulating electrodes was adjusted so that they were optimally placed for obtaining an H wave at the minimum stimulus intensity. In such a position, the H wave usually, but not always, appeared at a lower strength of stimulus than the M wave, or direct motor response. A conditioning shock of intensity less than 1.2 times threshold was followed at intervals which ranged from 1 to 1,000 msec by an identical test stimulus (Fig. 1). Ten seconds were allowed to elapse between each pair of stimuli. The ratio of the peak-to-peak amplitude of the H wave following the test volley to that of the H wave which followed the conditioning volley was calculated at each interval of time, and a recovery curve plotted.

Throughout the text, mean values are expressed with their standard deviations unless otherwise specified.

RESULTS

CHARACTERISTICS OF THE H WAVE The H wave was identified by the following characteristics (Magladery and McDougal, 1950; Mayer and Mawdsley, 1965; McLeod and Van Der Meulen, 1967). It was evoked at an intensity of stimulus below or near the threshold of the direct motor, or M response; its amplitude increased to a maximum level but subsequently decreased as the stimulus strength was raised progressively; its latency was reduced when the stimulating electrode was moved to a more proximal site.

The latency of the H wave in the 32 patients studied ranged from 26 to 31 msec. Investigations were conducted with stimulus intensities near threshold for the H response, and at such intensities of stimulus the amplitude of the H wave ranged from

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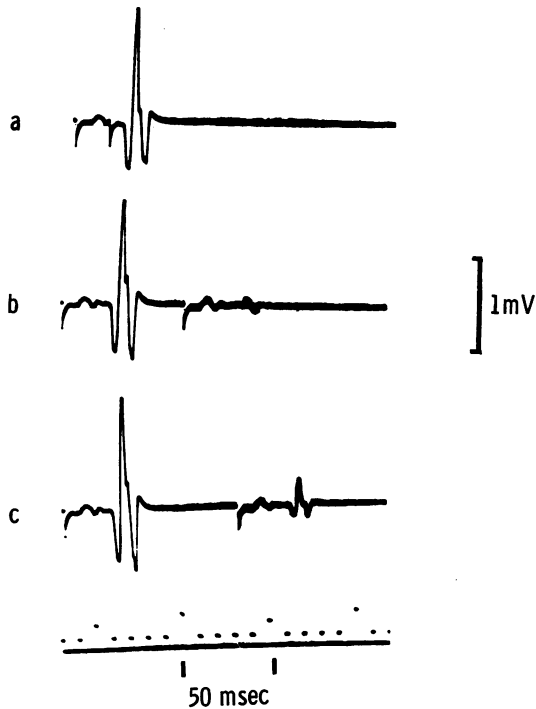


FIG. 1. *H* waves recorded in a control subject following paired stimuli. Stimulus intensity 1.06 times threshold of *H* reflex, for both conditioning and test shocks. A small *M* wave precedes the *H* response. (a) Interval between conditioning and test shocks, 20 msec. No *H* wave follows test shock. (b) Interval 70 msec. Ratio of amplitude of *H* wave following test shock to that following conditioning shock, 12%. (c) Interval 100 msec. Ratio of amplitude of *H* wave following test shock to that following conditioning shock, 24%.

150 μ v to 3.5 mV in control subjects, and from 250 μ v to 2.5 mV in patients with cerebellar disorders.

RECOVERY OF THE H REFLEX IN CONTROL SUBJECTS

Recovery curves of the *H* reflex following a condi-

tioning volley were plotted in 22 control subjects, in whom there was no clinical evidence of neurological disease, and whose ages ranged from 16 to 73 years (mean, 36.1). The intensity of the stimuli employed ranged from 1.04 to 1.16 (mean, 1.08) times threshold for the *H* reflex.

In 10 subjects there was an initial phase of facilitation at 5 to 15 msec after the conditioning stimulus, at which time the amplitude of the test response ranged from 16 to 212% of that of the conditioning response. The phase of early facilitation was followed by a period of unresponsiveness which ranged from 45 to 109 msec (mean, 72.6 ± 18.6 msec). The next phase in the cycle was a period of recovery of the test response, which reached its maximum amplitude at intervals of time which ranged from 125 to 275 msec after the conditioning volley. The maximum amplitude of the test response at this time was 25 to 175% of that of the conditioning response. Following this second phase of facilitation there was a late phase of depression, which was maximal at intervals of 400 to 700 msec. After this time there was a gradual recovery, but in only one case had complete recovery occurred by 1,000 msec. The results are summarized in Table I, and a mean curve plotted from the results obtained from all the control subjects is shown in Figure 2.

Stimulus intensity The effect of increasing the intensity of the stimulus from near threshold to maximal for the *H* reflex was studied in nine subjects. The mean stimulus intensity, when maintained near threshold for the *H* reflex, was 1.06 times threshold; when maximal for the *H* reflex the mean stimulus intensity was 1.27 times threshold. The observed changes in the recovery curve on raising the stimulus intensity were a reduction in the degree of early facilitation; a decrease in the duration of the period of unresponsiveness; a more pronounced second phase of facilitation; and a less pronounced late depression (Fig. 3). In the nine subjects studied, the mean duration of the period of unresponsiveness was reduced from 73.2 ± 18.4 msec at near threshold intensities, to 54.4 ± 17.3 msec at the higher intensities of stimulus.

TABLE I
PHASES OF H REFLEX RECOVERY CURVE

	Duration of initial facilitation (msec)	Duration of unresponsive period (msec)	Peak of second facilitation (msec)	Maximum of second depression (msec)
Control subjects (22)	5-15	45-109 (Mean 72.5 ± 18.6)	125-275	400-700
Cerebellar disorders (10)	5-25	15-43 (Mean 31.0 ± 8.8)	90-250	350-700

Mean figures are given with their standard deviations.

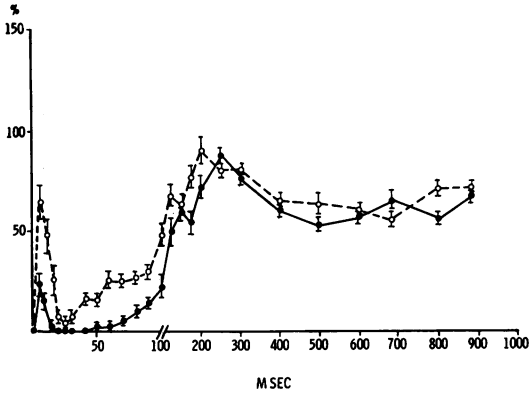


FIG. 2. Recovery curve of H reflex in control subjects (●—●) and patients with cerebellar disorders (○—○). Mean values from 22 control subjects and 10 patients with cerebellar disorders; vertical bars represent standard error of mean. Ordinate: ratio of amplitude of test response to amplitude of conditioning response, expressed as a percentage. Abscissa: interval between conditioning and test stimuli, in milliseconds. Note change in time-scale after 100 msec.

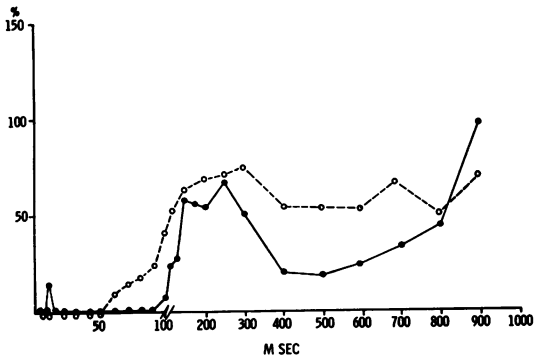


FIG. 3. Effect of increase of stimulus intensity on recovery of H reflex. Control subject. Stimulus intensity 1.07 times threshold for H reflex (●—●) and 1.25 times threshold for H reflex (○—○). Abscissa and ordinate same as in Figure 2.

Age of subjects In Fig. 4 the duration of the period of unresponsiveness has been plotted against the age of the subject; it can be seen that there is no apparent correlation in subjects below the age of 60. Beyond this age there may be a reduction in the duration of the unresponsive period, but there are not sufficient observations to permit a firm conclusion to be drawn.

RECOVERY OF THE H REFLEX IN PATIENTS WITH CEREBELLAR DISORDERS Ten patients with cerebellar

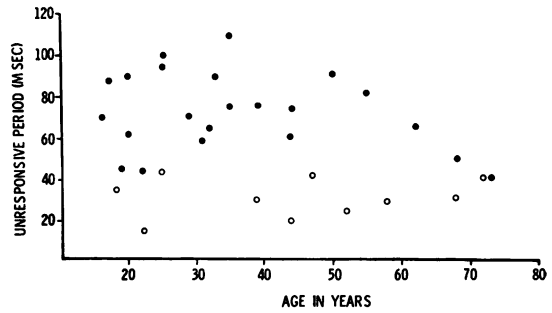


FIG. 4. Effect of age on unresponsive period in recovery curve of H reflex. ● = control subjects. ○ = patients with cerebellar disorders.

disorders, whose ages ranged from 18 to 72 years (mean, 44.5) were studied. The clinical diagnosis in each of the patients is shown in Table II. All of the patients had normal or decreased muscle tone in the limbs, impaired coordination of movements, ataxia of gait, normally brisk deep tendon reflexes, flexor plantar responses, and no impairment of sensation. In no case was there clinical evidence of cortico-spinal tract or peripheral nerve involvement. The stimulus intensities employed ranged from 1.05 to 1.18 (mean, 1.10) times the threshold for the H reflex.

TABLE II

CLINICAL DIAGNOSIS IN PATIENTS WITH CEREBELLAR DISORDERS

Patient	Age	Sex	Clinical diagnosis
A.P.	72	M	Metastatic tumour in cerebellum from carcinoma of bronchus
J.B.	22	M	Primary cerebellar degeneration
G.J.	46	M	Alcoholic cerebellar degeneration
J.D.	68	M	Late onset cerebellar degeneration ? alcoholic
H.W.	25	M	Cholesteatoma in left cerebello-pontine angle
G.T.	18	M	Post-traumatic cerebellar degeneration
A.R.	44	M	Cerebellar degeneration ? alcoholic
W.L.	58	M	Late onset cerebellar degeneration ? alcoholic
J.R.	39	M	Haemangioma of cerebellum

In seven of the 10 subjects there was an initial phase of facilitation at 5 to 25 msec after the conditioning stimulus, at which time the amplitude of the test response ranged from 44 to 175% of that of the conditioning response. It can be seen from Fig. 2, in which the curves are plotted from the mean values obtained from all the control subjects and from all the patients with cerebellar disorders, that the amplitude and duration of the initial phase of facilitation is increased in the patients with cerebellar disorders. Since early facilitation did not occur in all subjects, a comparison has also been made of the degree of facilitation in those 10 control subjects and seven

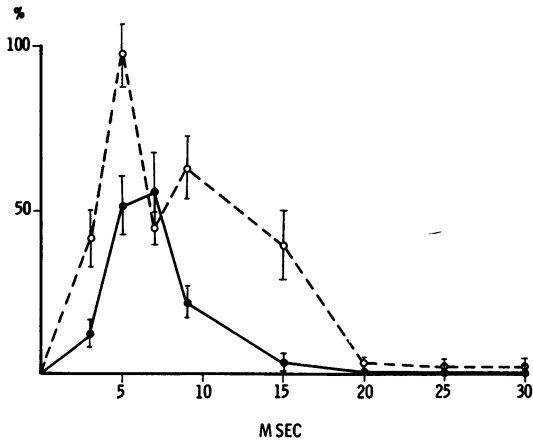


FIG. 5. Initial facilitation in recovery of H reflex in control subjects and patients with cerebellar disorders. Mean values for 10 control subjects (●—●) and seven patients with cerebellar disorders (○—○); vertical bars represent standard error of mean. Abscissa and ordinate same as in Figure 2.

patients with cerebellar disorders in whom facilitation was present. It can be seen from Fig. 5 that the degree of facilitation is more pronounced in the patients with cerebellar disorders.

The period of unresponsiveness in the patients with cerebellar disorders ranged from 15 to 43 msec (mean, 31.0 ± 8.8 msec), which is significantly shorter than that in the control subjects (t test, $P < 0.001$). The difference between the two groups can be seen in Fig. 6, and from the mean curves shown in Figure 2. The difference was not related to the age of the subjects (Fig. 4).

Recovery after the period of unresponsiveness occurred more rapidly, and the second peak of facilitation was achieved slightly earlier in the patients with cerebellar disorders than in the control subjects. There was no significant difference in the later phases of recovery (Fig. 2).

PATHOLOGY

One of the patients with cerebellar ataxia (W.L.) died nine days after electrophysiological studies had been performed and a significant reduction in the duration of the unresponsive period of the recovery curve (30 msec) had been demonstrated. Necropsy was performed.

CLINICAL HISTORY The patient, a retired male cook, aged 58, had complained of increasing difficulty in swallowing food for a period of two years, and unsteadiness of gait for a period of three months before admission to hospital.

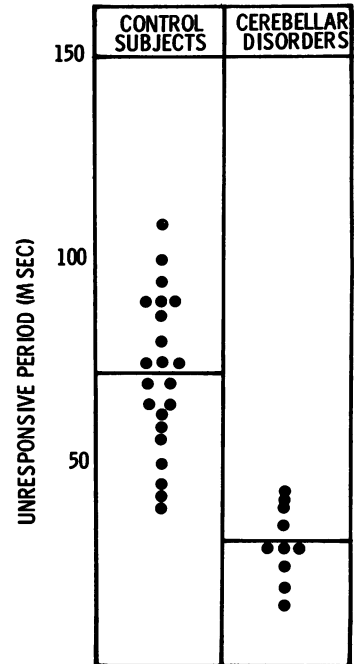


FIG. 6. Duration of unresponsive period of H reflex recovery curve in control subjects and patients with cerebellar disorders. Horizontal bars represent mean for each group.

He had been a heavy drinker in the past but had had little alcohol for the past two years. On physical examination, intellectual function was normal, speech was slurred. Cranial nerves were intact. There was no nystagmus. Tone was decreased in upper and lower limbs. There was no weakness. Incoordination of movements was more pronounced in lower than upper limbs. Deep tendon reflexes were brisk, and knee jerks were pendular. Plantar responses were flexor. Blood pressure was 140/90 mm Hg and no abnormality was found on general examination. Full blood count, serum electrolytes, liver function tests, serum proteins, and cerebrospinal fluid were all normal. Radiograph of the skull, electroencephalogram and cerebral scan were normal. Oesophagectomy was carried out for carcinoma of the oesophagus, which had been demonstrated radiologically, but the patient died three days post-operatively of broncho-pneumonia.

NEUROPATHOLOGICAL FINDINGS (Dr. Brian Turner)
Macroscopic features The cerebrum weighed 1,028 g and the cerebellum and brain-stem weighed 167 g. The basal vessels showed some mild slight congestive changes but no other significant abnormality. The mammillary bodies were normal. There was conspicuous atrophy in the anterior cerebellar vermis extending laterally into the hemispheres, and decreasing in extent in a lateral direction. The brain-stem showed no recognizable abnormality

Microscopic features Representative blocks of brain were embedded in celloidin and stained with Luxol fast blue—haematoxylin van Gieson, and the Nissl method. The cerebral cortex showed little abnormality apart from some increase in the number of astrocyte nuclei. The central white matter was normally myelinated. In the wall of the third ventricle there was a small discrete area of old tissue necrosis with marked glial proliferation. The mammillary bodies appeared normal. The thalamus and basal ganglia showed no abnormality. The cerebellum showed severe degenerative changes most marked in the anterior vermis. There was Purkinje cell loss with proliferation of the Bergmann astrocytes. The molecular layer was slightly thinned. In the pyramis more marked changes were apparent in the granular layer. In the brainstem some degenerative changes were apparent in the inferior olivary nuclei. No degeneration was noted in the medullary pyramids or in the corticospinal tracts in the cervical cord.

COMMENT The changes in the cerebellum are those of a recent degenerative process affecting the vermis predominantly. The changes are unlike in distribution and nature those found in carcinomatous degeneration and resemble more closely those found in alcoholic degeneration.

DISCUSSION

The H wave in man and in the cat has the characteristics of a monosynaptic reflex (Magladery *et al.*, 1951; McLeod and Van Der Meulen, 1967). The recovery of the H reflex after a conditioning volley has been studied in spasticity and in Parkinson's disease by several groups of workers who have concluded that in these conditions there is increased excitability of spinal motoneurons (Magladery *et al.*, 1952; Ioku, Ribera, Cooper, and Matsouka, 1965; Matsouka, Waltz, Terada, Ikeda, and Cooper, 1966; Olsen and Diamantopoulos, 1967; Takamori, 1967; Yap, 1967). However, the interpretation of H reflex recovery curves is complicated by the fact that the time course of recovery after a conditioning volley is influenced by the intensity of stimulus employed and by the effects of the muscle contraction (Paillard, 1959; McComas and Payan, 1966; Olsen and Diamantopoulos, 1967). In order to assess the importance of these factors, McLeod and Van Der Meulen (1967) studied the recovery of the H reflex under experimental conditions in the cat. It was found that the time course of recovery of the H reflex after a conditioning volley was almost identical with that of the monosynaptic reflex recorded from the ventral root. It was also found that the initial period of unresponsiveness or subnormality in the recovery curve was not influenced significantly by the reflex muscle contraction, but that the later phases of the curve were so influenced. It was concluded that if the effects of muscle contraction were

minimized by employing a low intensity of stimulus' the duration of the initial period of unresponsiveness provided an index of the state of excitability of the spinal motoneurone pool.

The duration of the initial period of unresponsiveness in the H reflex recovery curve in man was shortened by increasing the stimulus intensity to levels which resulted in the appearance of a significant direct motor response or M wave; similar observations have been made by other workers (McComas and Payan, 1966; Olsen and Diamantopoulos, 1967). Since it has been shown in cats that the excitability of spinal motoneurons is profoundly influenced by muscle contraction (Hunt, 1952; Bianconi, Granit, and Reis, 1964; Granit, Kellerth, and Szumski, 1966), it seems likely that the effect is a consequence of the contraction of the calf muscles which occurs when a significant number of motor fibres is excited directly by higher stimulus intensities. In the present study, the stimulus was therefore maintained at an intensity close to threshold for the H reflex; at such an intensity, the M wave was small or absent.

The time course of recovery of the H reflex in control subjects in the present work is similar to that obtained by Olsen and Diamantopoulos (1967) when they employed near-threshold stimuli. A valid comparison cannot be made with recovery curves obtained by investigators who have used stimuli of greater intensity (McComas and Payan, 1966; Takamori, 1967) or who have not specified the strength of stimulus (Magladery *et al.*, 1952; Ioku *et al.*, 1965). The early phase of facilitation after a conditioning volley is presumably due to recruitment of motoneurons which have been subliminally excited, as postulated by Magladery and co-workers (1952). The phase of unresponsiveness which follows the initial phase of facilitation is presumed to be caused by presynaptic inhibition or by inhibition arising from interneuronal activity at a spinal cord level, and its duration to be influenced by descending inhibition and excitation from supraspinal sources. It has been suggested that the second phase of facilitation in the recovery curve is caused by spindle afferent discharges during muscle relaxation (Granit *et al.*, 1966; McLeod and Van Der Meulen, 1967). The late phase of depression may be due in part to inhibition of the extensor motoneurons by afferent impulses derived from secondary endings in the muscle spindles (Granit *et al.*, 1966).

In the recovery curves of the H reflex in patients with cerebellar disorders, the early phase of facilitation appeared to be more pronounced than in those of control subjects; a similar observation was made by Olsen and Diamantopoulos (1967). Although the finding suggests that the motoneurone pool was more readily excited in the patients with cerebellar dis-

orders, it must be emphasized that, in three of the 10 patients, no early phase of facilitation was present. In the patients with cerebellar disorders there was also a significant shortening of the period of unresponsiveness, which indicates that the motoneurone pool recovers more rapidly after the conditioning volley in the patients with cerebellar disorders than in control subjects. This may be interpreted as an indication that there is increased alpha motoneurone excitability in these patients.

The apparent increase in excitability of the alpha motoneurons in patients with cerebellar disorders is not due to the employment of a higher intensity of stimulus in studying the recovery of the H reflex in these patients than that used in control subjects, since the stimulus strength was adjusted to the same level of intensity in both groups. The excitability of spinal motoneurons was not influenced by age, except possibly in patients over the age of 60. Although the average age of the patients with cerebellar disorders was slightly greater than that of the control subjects, this was clearly not a significant factor in causing the increased motoneurone excitability. It has been established by a number of groups of workers that there is increased motoneurone excitability in patients with upper motoneurone disorders (Magladery *et al.*, 1952; Olsen and Diamantopoulos, 1967; Takamori, 1967; Yap, 1967); the possibility must therefore be considered that the increased motoneurone excitability in the patients with cerebellar disorders was a consequence of damage or disease of the corticospinal tracts, rather than to the cerebellum. Patients were excluded from the study if they displayed clinical evidence of neurological disease in structures outside the cerebellum. None of the subjects studied showed clinical evidence of corticospinal tract involvement, such as increased muscle tone, clonus, or extensor plantar responses. Direct pathological confirmation was obtained in the case of the patient who died only nine days after electrophysiological studies had been performed. Increased motoneurone excitability was demonstrated by a pronounced initial phase of facilitation and an unresponsive period of 30 msec in the recovery curve. At pathological examination there was degeneration of the cerebellar cortex, but there was no evidence of corticospinal tract degeneration in sections of the brain-stem or cervical cord.

Olsen and Diamantopoulos (1967) studied the recovery of the H reflex in four patients with cerebellar hypotonia. They found an increased early facilitation, which has been confirmed in the present study, but did not observe any significant difference from normals in the duration of the unresponsive period. However, since they employed stimuli which were maximal for the H reflex rather than stimuli

near threshold intensity an alteration in this phase of recovery may have been obscured by the effects of muscle contraction. Castaigne, Cathala, Lacert, and Pierrot-Deseilligny (1966) have recently studied the effects of muscle stretch on the H reflex in patients with cerebellar disorders; they found evidence for increased alpha motoneurone excitability and a decrease of gamma motoneurone activity in the majority of their patients.

The H reflex is a monosynaptic reflex; the afferent pathway consists of fast conducting fibres, which presumably belong to Group Ia. It is known that Group Ia afferent fibres project to the cerebellar cortex and that the Purkinje cells have an inhibitory action on the intracerebellar and vestibular nuclei (Eccles, Ito, and Szentagothai, 1967). In pathological conditions in which there is a loss of Purkinje cells, this inhibitory action may become disturbed. The hyperexcitability of the alpha motoneurons which has been observed in the present work may be caused by interruption of an inhibitory reflex pathway those afferent limb ascends from the muscle spindle to the cerebellar cortex, and whose efferent limb descends to the spinal motoneurons. Alternatively, it may be caused by release of the reticulospinal and vestibulospinal pathways from tonic cerebellar inhibition (Terzuolo, 1959; Llinas, 1964; Batini, Moruzzi, and Pompeiano, 1957). There was no apparent difference in the amplitudes of the H waves following a conditioning stimulus in the patients with cerebellar disorders from those of control subjects over the range of stimulus intensities employed. If the physiological consequence of cerebellar disease were the removal of a direct tonic inhibition of alpha motoneurons, it might be expected that the amplitude of the H reflex response to a single shock would be enhanced. However, this would not necessarily be the case if in cerebellar disease there was an interruption of a reflex inhibitory pathway through the cerebellum on to spinal interneurons. Under such conditions, the monosynaptic reflex response of the motoneurone pool to a single stimulus would be unaltered, but because of interruption of the reflex inhibitory pathway, recovery of excitability would occur more rapidly than in normal individuals.

In cats, there is definite evidence of hyperactivity of the alpha motoneurons after acute cerebellectomy (Granit *et al.*, 1955; Van Der Meulen and Gilman, 1965) but there is no convincing evidence that the state persists in the chronically decerebellate animal (McLeod and Van Der Meulen, 1967). The reason for the difference between man and the cat is not clear. One important factor may be that in the experiments on cats, cerebellectomy was total with removal of the intracerebellar nuclei; in the human

subjects, the cerebellar lesions were only partial and in most cases probably involved chiefly the cerebellar cortex. Another factor may be that brain-stem structures compensate more rapidly in the cat than in man for the release of alpha motoneurone activity which follows damage to the cerebellum. Certainly there is a marked difference in the clinical states displayed by the cat and the monkey in recovering from total cerebellectomy. The cat develops a posture of extensor rigidity, whereas the monkey develops a flexion dystonia immediately after cerebellectomy; the cat can usually walk in two to three weeks after the operation, in contrast to the monkey which may not be able to walk for two to three months (Denny-Brown, 1966).

The alpha motoneurone hyperactivity in patients with cerebellar disorders may in part be responsible for their characteristically jerky incoordinate movements. The hypotonia of cerebellar disease is likely to be a result of depression of gamma motoneurone activity, for which Castaigne *et al.* (1966) have recently provided evidence.

SUMMARY

The H reflex was studied in 22 control subjects and 10 patients with cerebellar disorders. The strength of stimulus was adjusted to an intensity less than 1.2 times the threshold for the H reflex; pairs of identical stimuli were delivered, and the time-course of recovery of the amplitude of the H reflex was determined in every subject.

In the recovery curves of the control subjects there was an early brief phase of facilitation followed by a period of unresponsiveness of duration 45 to 109 msec (mean 72.6 ± 18.6 msec). In the recovery curves of the patients with cerebellar disorders there appeared to be a more pronounced phase of early facilitation, and a significantly shorter period of unresponsiveness of duration 15 to 43 msec (mean, 31.0 ± 8.8 msec). The more rapid recovery of motoneurone activity after a conditioning volley in patients with cerebellar disorders is considered to be the result of impairment of a reflex inhibitory action of the cerebellum on spinal neurones.

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