ACTA NEUROBIOL. EXP. 1971, 31: 263-289

A STUDY OF CEREBELLAR DYSKINESIA IN THE BILATERALLY DEAFFERENTED FORELIMBS OF THE MONKEY (MACACA MULATTA AND MACACA SPECIOSA)

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The central nervous system controls the contraction of skeletal muscle tone, posture, locomotion and skilled movements via two types of motor neurons, alpha and gamma (Eldred et al. 1953, Granit 1955, Jansen 1966). The gamma neurons innervate the intrafusal muscle fibers of the muscle spindle stretch receptors, while the alpha neurons innervate the extrafusal muscle fibers forming the great preponderance of the muscles (Leksell 1945, Hunt 1952, Barker and Ip 1961). The gamma motor neurons usually anticipate or precede the firing of the alpha motor neurons (Merton 1953, Granit 1955). This sequence of motor discharge emphasizes the ignition and modulation of alpha motor neurons via the muscle spindle reflex. It has been postulated that disruption of this alpha-gamma linkage would result in disturbances in tonic and phasic motor function (Merton 1953, Eldred et al. 1953, Denny-Brown 1966).

The dystonia, dysmetria, tremor and decomposition of movements following cerebellar ablation has recently been attributed to functional disruption of the alpha-gamma linkage (Granit et al. 1955, Higgins and Glaser 1964, Van Der Meulen and Gilman 1965, Glaser and Higgins 1966 and Denny-Brown 1966). The compensation of these motor deficits has also been correlated with the recovery of this linkage (Van Der Meulen and Gilman 1965, Higgins and Glaser 1965). These experiments have evaluated the motor deficits and their compensation in terms of central and peripheral reactivity of the gamma motor innervation of the muscle spindle and its sensitivity to tonic and phasic stretch (Van Der Meulen and Gilman 1965, Glaser and Higgins 1966). While it is clear from these experiments that the response of the myotatic reflexes of the

limbs in the decerebellate-decerebrate cat can be correlated with the motor impairment, the effect of cerebellectomy on the direct modulation of the alpha motor neurons can be evaluated only by selectively paralyzing the gamma motor neurons or by interrupting the afferent input from the muscle spindles.

Physiological studies (Eldred et al. 1953, Granit 1955, Granit et al. 1955, Dow and Moruzzi 1958, Denny-Brown 1966) of the exaggerated skeletal muscle tone in the decerebrate animal have established that this spasticity is due to the release of gamma motor neurons and increased myotatic reflexes. Deafferentation abolished this gamma rigidity. However, if a vermal cerebellar lesion is made in a decerebrate-deafferent preparation skeletal muscle tone returns (Batini et al. 1957 and Denny-Brown 1966). This tonic contraction is due to an increased firing of alpha motor neurons and is referred to as alpha rigidity. Both alpha and gamma rigidity are compensated in time (Denny-Brown 1966). Thus, it is impossible in the experiments with chronic decerebellate-decerebrate cats with intact stretch reflexes (Van Der Meulen and Gilman 1965, Glaser and Higgins 1966, Denny-Brown 1966) to evaluate the effect of cerebellectomy on the direct alpha motor neuron modulation and especially to determine the effect of cerebellar lesions on any but the simplest tonic and phasic reflexes.

The simplest procedure to determine the role of the alpha-gamma linkage in the modulation of motor acts is to deafferent a limb and study its voluntary and reflexly induced movements (Knapp et al. 1963, Taub et al. 1966, Bossom and Ommaya 1968). Denny-Brown (1966) has deafferented a single limb in one decerebellated monkey and noted the presence of a tonic flexor posture in the deafferented limb due to tonic alpha motor neuron discharge. He found that the coarse tremor of the intact limb during fending against a pin prick was absent from the deafferented limb. He concludes that "cerebellar tremor must therefore reflect some conflict of alpha with recovering spinal gamma effect".

A more parsimonious explanation for the disappearance of the coarse tremor in the deafferented limb can be offered. That is, the fending movements in the deafferented limb were primarily associated nongoal directed acts, in contrast to the goal directed movements of the intact limb. Growden et al. (1967) found that cerebellar ataxia and tremor never occurred during associated movements in animals whith pyramidal lesions, but occurred during the correction of an error in a goal directed act. Mott and Sherrington (1895) and Twitchell (1954) have emphasized that a unilaterally deafferented limb shows a grave paresis of goal directed acts and an abundance of associated (mittlebeweigung) movements.

In contrast with unilateral deafferentation, a monkey with bilateral

dorsal rhizotomy shows marked recovery of reflexes originating outside the denervated area and recovery of skilled use of the limbs (Knapp et al. 1963, Taub and Berman 1964, Bossom and Ommaya 1968). These observations have stimulated us to study: (i) the role of the limb afferents on the cerebellar modulation of motor acts of associated and goal directed movements; and (ii) the role of the limb afferents in the compensation for cerebellar dyskinesia. A preliminary report has been published in abstract form (Liu and Chambers 1967).

MATERIALS AND METHODS

Seven young adult male monkeys were used in this study. Two monkeys were *Macaca mulatta* and five were *Macaca speciosa.* All animals were handled until they were gentle and would take food from the observer and groom the hair on his hand. The gait, posture, tone and placing reactions were examined daily. After two weeks of pre-

TABLE I

Group I: Bilateral deafferentation and subsequent cerebellar lesions		
M8 speciosa	2/ 9/67 Bilateral deaff. C2-T3 (intradural)	
	3/15/67 Left cerebellar dentate-interposed	
	3/19/67 Right cerebellar fastigial, dentate-interposed	63 days survival
M9 mulatta	2/11/67 Bilateral deaff. C2-T3 (intradural)	4 days survival
M1 speciosa	6/ 4/66 Bilateral deaff. C3-T3[a] (extradural left VR C8, T1 were cut)	
	6/29/66 Bilateral cerebellar dentate-interposed	128 days survival
Group II: Cerebellar lesions followed by deafferentation		
M3 speciosa	8/ 4/66 Unilateral cerebellar right dentate-interposed	
	9/27/66 Bilateral deaff. C2-$T3_b$ (extradural)	
	10/10/66 Deaff. (intradural) right C4; Left C3 and T1	
	3/15/67 Left cerebellar dentate-interposed	254 days survival
M4 speciosa	12/27/66 Unilateral cerebellar right dentate-interposed	
	1/31/67 Bilateral deaff. C2-T4 (intradural)	40 days survival
M6 speciosa	2/ 8/67 Unilateral cerebellar right dentate-interposed	
	2/20/67 Bilateral deaff. C2-T3 (intradural)	
	5/ 8/67 Bilateral thalamic lesion (VL)	98 days survival
M7 mulatta	2/ 8/67 Unilateral cerebellar right dentate-interposed	
	3/21/67 Bilateral deaff. C2T-3	98 days survival

[a] Right C3-C4 dorsal roots — few fibres remained intact.
[b] Right C4; Left C3 and T1 dorsal roots — few fibres remained intact.
Abbreviations: M, macaca; C, cervical dorsal roots; T, thoracic dorsal roots; VL, nucleus ventralis lateralis of thalamus; VR, ventral root.

operative testing, movies were made of the animals to show the limb response during feeding, grooming, placing, climbing and walking. Electromyographic and accelerometer records of limb movements during fending off a pin prick or a bushy brush, feeding, grooming and in scratching to local application of itch powder (cowhage) were made at least two times before subjecting the animals to surgery.

The animals were divided into two experimental groups (Table I). Group I consisted of three animals with bilateral deafferentation of the forelimbs (C2-T3) and later unilateral or bilateral cerebellar dentate and interposed nuclear lesions. Group II consisted of four animals with initial unilateral cerebellar nuclear lesion and subsequent bilateral deafferentation of the forelimbs. All surgical procedures were performed with rigid aseptic technique and the animals were anesthetized with Nembutal. Dorsal rhizotomy was first attempted by an extradural approach in two animals but this was found to be unsatisfactory for achieving a complete deafferentation. The other six animals were deafferented by the intradural approach employed by Taub et al. (1966).

All roots were sectioned during visualization with the aid of a Zeiss operating microscope. The roots and rootlets were magnified 4 to 16 times and no attempt was made to preserve the blood vessels accompanying the dorsal roots. However, special care was taken to prevent injury to pathways in the lateral funiculus. The cerebellar lesions were made by aspiration. After surgery the animals were examined, as they were preoperatively, with frequent recordings of movements by movies, electromyographs and the accelerometer.

All deafferented animals mutilate their fingers by biting them. This self mutilation was prevented by fashioning a celluloid cone which was laced around the neck and inserted over the shoulder and upper arms. This cone was removed during all test periods and could be frequently left off for days.

When the cerebellar-deafferented animal had recoverd the ability to extend his limbs when suddenly dropped, to visually place, to scratch a local area treated with itch powder and to feed himself, he was tested with vision excluded by a rubber mask. All of the above tests were usually conducted with the animals seated in a primate chair. Three animals without vision blocked were trained to reach about 5 cm above or below and 10 cm away from their mouths and grasp food. Each animal was trained to reach above his mouth if the upper lip was touched with a piece of biscuit and below his mouth if his lower lip was touched. After learning this task the animal was blindfolded and his performance noted with vision excluded.

All animals with and without vision excluded were tested for the

completeness of the deafferentation by pin prick, touch, tendon reflexes and strong mechanical stimuli. This method of testing clearly revealed an incomplete deafferentation in the two animals with attempted rhizotomy via the extradural approach (M1, M3; Table I). One of these animals (M3) was reoperated and the intact rootlets cut intradurally.

The animals were sacrificed by an intracardiac perfusion technique (Koenig et al. 1945) while under Nembutal anesthesia. The cervical and thoracic spinal cord was carefully exposed and visualized at 40 × magnification for the identification of the severed dorsal roots. The spinal cord and brain were removed and prepared for study of the lesions. The cerebellum and brainstem were sectioned and stained by the Mahon and Nissl methods. The cervical and upper thoracic spinal cord was serially sectioned and stained by the Bodian silver technique for any remaining intact axons in the dorsal root spinal entry zone and for inadvertent damage of axons in the lateral and ventral funiculi.

RESULTS

I. Recovery of movements in the deafferented arms

Analysis of the time course and pattern of recovery of movements in the bilaterally deafferented forelimbs on the Group I animals reveals a rather stereotyped picture. On the first day after deafferentation of the forelimbs the speciosa monkeys righted themselves in hind quarters and then stiffly elevated the forequarters and head to assume a seated or a low crouched position with the ischial tuberosities 3 or 4 cm off the cage floor. The trunk and neck were held stiffly erect. The forelimbs were mildly protracted and adducted at the shoulders and flexed 10–20° at the elbows. The wrists and fingers were mildly flexed and the hands were fully pronated. These animals could walk in a low crouch with stiff trunk and neck and immobile forelimbs. In standing and walking they remind one of the posture and gait of the penguin. When they fell, they made no attempt to grasp the cage with the forelimbs or to extend them for support. Rather they would grasp the bars of the cage floor with their hindlegs or widen their base. They would attempt to climb with the forelegs maintaining a fixed position while using their teeth to grasp the bars. This motor performance persisted for three to four days.

However, during this time movement of the forelimbs could be elicited by several means. If the animal was threatened by a brush or pin pricked on the face, he lowered his head, flexed his truck and both forelimbs were jerkily elevated and flexed at all joints, the hand being carried above and behind the ears. The same movements were initiated by threat of sudden dropping or during rappid approach for visual placing

on a table top or to the home cage. Less marked flexor movements were initiated by offering food to the monkey's mouth. He would extend his head for the proffered food and both forelegs would become strongly flexed at elbow, wrist and fingers, with the limb further adducted and retracted at the shoulder during feeding, the clenched hand being brought to the level of the clavicles. Although the animals could not reach for or grasp the food, if it was placed in their flexed hand, they would attempt to seize it by lowering the head, as the associated flexed and adducted limbs moved toward the clavicle. They were so frustrated by this procedure that they would grasp the deafferented foreleg with one of their feet and try to guide it to their mouths.

That the forelimbs were capable of more precise movement at this time (1–4 days) was clearly illustrated by the monkeys moving their hands to their mouths and carefully biting the nails or the skin of the balls of the thumb and first finger (Table II). During biting the hand was supinated and pronated without ataxia or tremor. Also the pouch was emptied by the wrist during this period. On the second and third days a modification of the pattern of flexor movements to visual threat, pin prick and drop reaction was noted. This consisted of dorsiflexion of the wrist and fanning and extension of the fingers while the hands were still carried behind the ears.

By the sixth or seventh day the reflex responses to these stimuli consisted of two major movements — first flexion of the arms moving the fanned and extended fingers behind the ears with the palms facing upward and immediately following a second pattern of movements, protraction of the shoulder, extension of the elbow and supination of forearm. This pattern of movement of flexion followed by extension also occurred during visual placing and reaching for food. The flexor phase to drop, visual threat, and pin prick continued for several weeks (Table II).

Supporting and propping reactions with the forelimbs were first apparent in the latter part of the first week. The forelimbs were abducted and extended with the weight being borne on the dorsum and radial side of the wrist. Pushing the animal laterally or forward led to flexion of the limbs followed by weak extension. If these displacements were sudden, the animal fell. The response to push markedly improved during the second and third weeks. This improvement corresponded to the inhibition of flexor and initiation of extensor patterns during visual and drop placing reflexes.

At this time the animals could support themselves in the forequarters alone and could make some visual stepping responses. The response to pushing backward was adequate only in one animal (M1, incomplete

TABLE II

Rate of recovery of some forelimb movements after bilateral deafferentation and the effects of prior unilateral cerebellar lesions

Movements	Animal number				
	M8	M1	M3	M6	M7
Visual placing	LD 21 d	LD 21 d	LD 21 d	LD 21 d	LD 30
	RD 21 d	RD 21 d	RCD 57 d	RCD 30 d	RCD 47 d
Drop reflex	LD 21 d	LD 21 d	LD 21 d	LD 21 d	LD 46 d
	RD 21 d	RD 21 d	RCD 57 d	RCD 30 d	RCDN (57 d)
Fending	LD 21 d	LD 21 d	LD 7 d	LD 8 d	LD 42 d
	RD 21 d	RD 21 d	RCD 57 d	RCD 31 d	RCDN (57 d)
Reaching for food	LD 8 d	LD 10 d	LD 14 d	LD 8 d	LD 14 d
	RD 8 d	RD 8 d	RCD 46 d	RCD 20 d	RCD 41 d
Pick up raisins with fingers	LD 21 d	LD 21 d	LD 21 d	LD 37 d	LD 46 d
	RD 21 d	RD 21 d	RCDN (200 d)	RCDN (86 d)	RCDN (57 d)
Empty pouch	LD 4 d	LD 1 d	LD 3 d	LD 3 d	LD 8 d
	RD 4 d	RD 1 d	RCD 8 d	RCD 7 d	RCD 35 d
Grooming	LD 21 d	LD 21 d	LD 21 d	LD 16 d	LD 35 d
	RD 21 d	RD 21 d	RCD 130 d	RCD 37 d	RCDN (57 d)
Biting fingers	LD 4 d	LD 1 d	LD 4 d	LD 4 d	LD 2 d
	RD 4 d	RD 1 d	RCDN (200 d)	RCDN (86 d)	RCDN (57 d)
Choreo-athetoid movements	LD 35 d	LD 35 d	LD 39 d	LD 52 d	LD 35 d
	RD 35 d	RD 35 d	RCD 39 d	RCD 52 d	RCD 35 d

Abbreviations: D, deafferented forelimb; CD, deafferented limb with prior cerebellar involvement; R, right; L, left; d, days after deafferentation; N (— d), never returned in survival period after deafferentation.

dorsal rhizotomy) which could poorly retract, abduct and extend his limbs. At no time did any of these animals use a quadrupedal gait or attempt to climb. However, the animals seldom had ideal opportunities to run or climb as they were housed in regular cages. Nevertheless normal animals housed in similar cages frequently climbed and used a quadrupedal progression.

On reaching for food, the initial flexion phase was inhibited by 8–14 days (Table II). At this time the limb was jerkily and writhingly circumducted and extended with the fingers of the supinated hand in full extension and abduction. Grasping a piece of food was by flexion of all fingers. Even when the hand closed on the food, there was frequently repeated opening and closing of the fingers. If they missed the food, they made jerky and repeated movements at the wrist, or they might flex the arm slightly before again jerkily grasping at the food. If food was placed in the hand, by the fourth or fifth day the animals would adduct the arm against the chest and flex the elbow to carry the food

to the mouth. These movements were not so ataxic or jerky as when reaching for food and the hand seldom hit the face or missed the mouth. If it did, there was a jerky correction and rarely one or two oscillations. The animals usually fed from the ulnar side of the supinated hand. If the piece of food was larger than they could take in one bite, they frequently held it against their lips for repeated small bites. The food was rotated by wrist movements against the lips or in contact with the surrounding hair and tremor or ataxia was seldom seen.

By the end of the second week the finger and wrist movements during feeding had greatly improved. They could rotate the biscuit with their fingers or grasp the food in mid-supination pronation position or with the hand fully pronated.

By the third and fourth weeks, feeding movements had recovered to the point where they could feed from either side of their hand in supination or pronation and during bites they would move their hands several centimeters from the mouth, inspect the food, rotate and bring it back accurately for another bite. At other times the arm was fully extended or partially so with abduction or extension and the grasped food brought to the mouth accurately. That they did not always carefully look at the arm or food grasped in the hand was evident by the fact that they frequently relaxed their grasp of the food and brought the empty clenched hand to their mouth. They would then look at the hand open it as if they wondered what happened to the food.

During the third week the reaching for food near the mouth, with the upper arm flexed and adducted against the body was quite accurately performed, and with wrist and hand movements they could grasp small bits of food such as raisins. If this size food was placed in the center of their palms, they could manipulate it with their fingers and release it accurately to their lips. On reaching for food with the arm fully extended, the limb was still ataxic at the shoulder and they usually missed small bits of food and corrected with a jerky ataxia, sometimes showing a few oscillations, they became excited and started whole-handed grasping at the food (Fig. 1*DJ*). If the extended upper arm was stabilized by the observer, the finger and hand movements showed much greater accuracy.

To test the effect of vision on the recovery of movements at this stage the animals were placed in a seated position and blindfolded by a rubber mask. When food was touched to the lips, the animal extended the head and attempted to bite the food, both forelegs coming into associated flexor and adductor movements with the hand tightly flexed at the level of the clavicles. After the animals were prevented from taking food by grasping it with their lips and teeth, they would raise their hands

toward the mouth, frequently missing the mouth or hitting their face and correcting in a jerky fashion with occasional oscillations. They were obviously excited and irritated since they would grasp at the food touching their lips and seize it only accidentally.

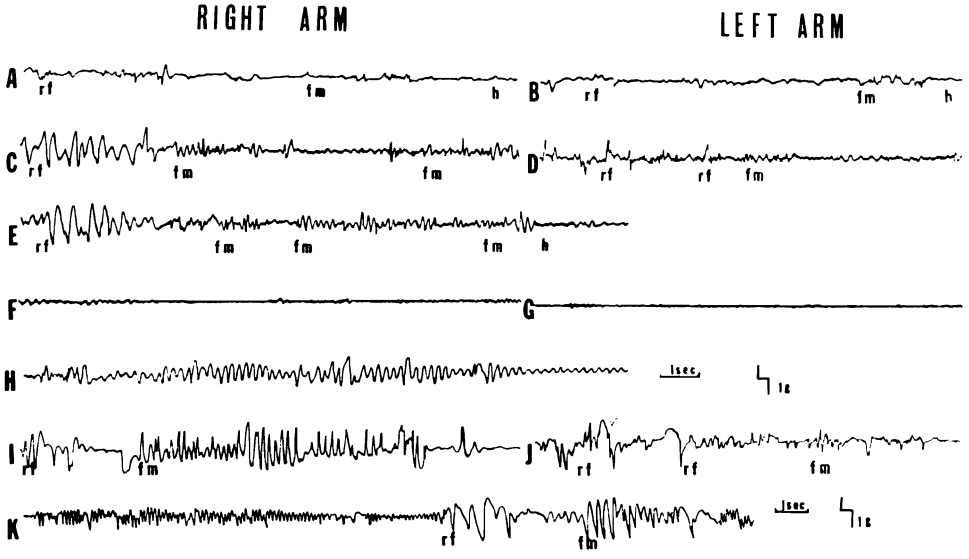

Fig. 1. Accelerometer records of arm movements during feeding of M6 showing ataxia and tremor following unilateral dentate and interposed nuclear lesions and the effect of subsequent bilateral deafferentation of the forelimbs. *A, C, E, F, H, I, K,* right arm; *B, D, G, J,* left arm. *A* and *B*: normal animal reaching for food (*rf*), feeding at the mouth (*fm*), and holding (*h*) food between bites. *C*: ataxia and tremor of the ipsilateral arm 2 days after a right cerebellar dentate and interposed nuclear lesion (Fig. 6*A*). *D*: normal movements of the contralateral limb recorded on the same date as *C*. *E*: ataxia and tremor ten days after cerebellar lesion. *F* and *G*: records of movements of the right arm (*F*) and the left arm (*G*) one day after deafferentation and 13 days after cerebellar lesion. Note oscillation (tremor) in *F* as the right arm attempts to place food in the mouth. *H*: supination-pronation tremor of the right arm induced by frightening the animal on the second day after deafferentation and 14 days after the cerebellar lesion. *I* and *J*: records of limb movements during feeding 22 days after deafferentation and 34 days after the cerebellar lesion. Note cerebellar ataxia and tremor of the right arm (*I*) and some jerky movements of the left arm (*J*) while reaching with it fully extended at the shoulder level. *K*: Nine-per-second supination-pronation tremor induced by anticipation of feeding and intensified by teasing or threatening the animal is replaced by slower and more irregular oscillations during reaching for food and taking bites of food. Records made 51 days after deafferentation and 63 days after the cerebellar lesion. Calibrations same for Fig. 1–5, Amplitude, 1x force of gravity; time, 1 sec. Calibrations for *A–J* above trace for *J*, for *K* to the right of this trace. Abbreviations same as in Fig. 2.

After two or three sessions they became accustomed to the mask and the movements were less wild and jerky. At this time a piece of food too large for a single bite could be placed against their lips or inbetween the teeth, and the animals somewhat wildly grasping at it, would push it against their lips and then grasp it. Now they could take

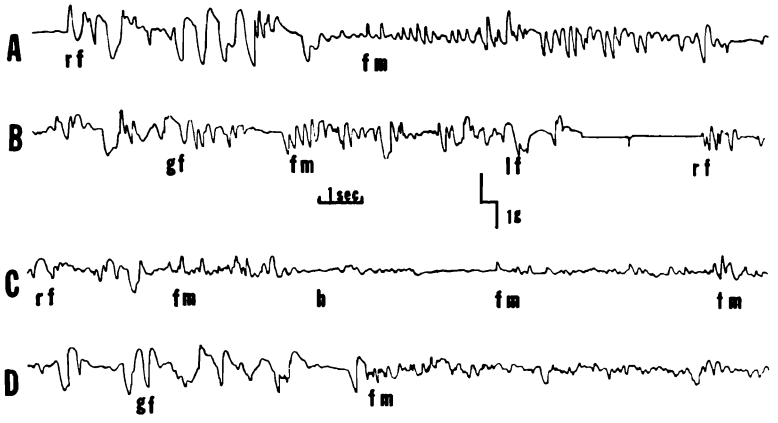

Fig. 2. Effects of blocking vision on the movements of the arms during feeding in mask adapted M6 with a right dentate and interposed cerebellar lesion and with both arms deafferented. The accelerometer records were taken 68 days after the cerebellar nuclear lesion and 56 days after deafferentation. *A* and *B*: records of the movements of the right arm during feeding: *A*, with vision and *B*, without vision; *C* and *D*: records of the movements of the left arm during feeding: *C*, with vision and *D*, without vision. Note that the movements of the limb, when food is held in the hand and the animal is taking repeated small bites (*fm*) is not noticeably changed by blocking vision. On the other hand the reaching (*rf*) and grasping (*gf*) with the hand in an effort to seize the food held between the monkey's teeth and lips is clearly more jerky and ataxic after excluding vision in the deafferented left arm (*C, D*) and not greatly altered in the right arm involved by the cerebellar lesion and deafferentation (*A, B*). Abbreviations: *rf*, reaching for food; *fm*, feeding at the mouth; *gf*, grasping for food held by the monkey's teeth and lips; *lf*, food dropped from hand; *h*, holding food in hand without feeding.

single bites of food, rotating the wrist and hand with the upper arm fixed against the chest. These movements were smooth and almost as accurate as when vision was not occluded (Fig. 2*CD*). The food, between bites, was kept in contact with the hair of the chin. Later the hand grasping the food was moved away from the mouth several centimeters and at times with the arms fully extended. The hand then was raised accurately back to the mouth. Frequently the food was dropped from the grasp and the empty clenched hand was brought to the mouth and the

animal would open the hand carefully and place it against his mouth as if the food might be in the center of the hand. It was clear that without vision, the movements were less accurate and more jerky, but once the animal had calmed to the mask and was feeding with upper arm fixed against the chest the hand and wrist movements were nearly as accurate as with vision.

The accuracy of the blindfolded animals in raising their hands to the mouth when food touched their lips or when replacing the grasped food in the mouth between bites, suggests that they knew the start position of the limb and that they were continuously aware of grasping the food and of the subsequent movements between bites. Passive movement of the limb from the start position (adduction and protraction of the shoulder and 10–20° flexion at the elbow) to a position of increased flexion or protracting or abduction led respectively to a consistent error of overflexion, undertraction or underadduction in reaching the mouth. If the error was not too great, the animals would hit their faces and make a series of corrective jerky movements. With a greater error the limb would return to the start position for a seated animal and then be raised accurately. The same disruption of movements occurs if the limb is passively moved between bites of food. Feeding movements are also upset by distraction and excitement of the animals. They apparently must focus their attention on the start position of the deafferented arm and on any of its subsequent movements during grasping and taking bites of food. This attention to movements must also apply to those of the head for it was noted that the animal could move his head between bites and the lowered hand holding food was accurately raised to the new position.

Grooming returned during the third to fifth week (Table II). At first the movements were clumsy and slow involving all fingers. There was no ataxia and tremor during these movements. The grooming although usually of the face or the hindlegs was at times of the other deafferented forelimb or the hair on the observer's hand. Cowhage placed on the dorsum of the head or side of the face was accurately localized and the itch area scratched without ataxia, although clumsily. The grooming and scratching movements during the fourth week (M1) and the fifth week (M8) showed considerable improvement in the use of the fingers. Hairs were spread and separated by abduction and adduction of the fingers and single hairs grasped between the thumb and the index fingers.

The recovery of movements in the deafferented forelimbs after one to two weeks is based on data from animals with prior or subsequent unilateral cerebellar lesions. Comparison of the movements of the deafferented forelimb immediately prior to and after a unilateral cerebellar

nuclear lesion in monkey (M8) revealed that a unilateral cerebellar lesion does not alter the recovered movements of the contralateral forelimb (Fig. 3*CDEF*).

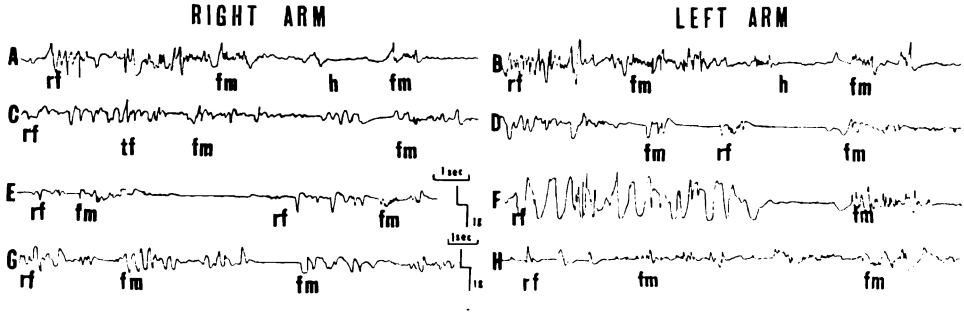

Fig. 3. Effects of unilateral and subsequent bilateral cerebellar nuclear lesions on the feeding movements of the deafferented arms of M8. *A* and *B*: records of movements of the arms before deafferentation. *C* and *D*: records taken 34 days after deafferentation. *E* and *F*: records made 38 days after deafferentation and 4 days after a left dentate and interposed nuclear lesion. *G* and *H*: records taken 63 days after deafferentation, 29 days after the left dentate and interposed and 25 days after a right cerebellar dentate, interposed and fastigial lesion. Note that the cerebellar ataxia and tremor occur ipsilateral to the dentate and interposed lesion (*F* and *G*). Abbreviations: *tf*, takes food in hand; other abbreviations same as in Fig. 2. Calibrations: *A–F* to the right of trace *E*; *G* and *H* to the right of trace *G*.

Also, a study of the deafferented limb in the Group II animals (M3, M4, M6 and M7) revealed that a prior unilateral cerebellar nuclear lesion did not alter the time course and pattern of recovery of the contralateral deafferented forelimb (Table II). Three of these animals of Group II had a simple deafferented limb in which the recovery of movements could be studied for considerable periods (M3, 156 days; M6, 86 days; and M7, 57 days).

Generally, the recovery was most marked in the second and third week but continued improvement of dexterity, smoothness of movement and reduction of ataxia on reaching was still taking place at 156 days. Two animals (M3 and M6) with prior unilateral cerebellar lesions improved to the point that their simple deafferented limbs could carefully groom a single hair. They could also reach out to grasp food with only slight jerky and ataxic movements of the shoulder girdle, even when the animals reached over their heads with the fully extended arm (Fig. 1*J* and Fig. 3*E*). When the animal fixated the shoulder and elbow joints, grasping at food with movements of the wrist and fingers was markedly improved.

Choreo-athetoid movements of the deafferented limbs appeared in all monkeys during the fifth to eighth weeks (Table II) and persisted largely unchanged in frequency or pattern throughout their survival periods. They occurred in the quiescent limbs while the animals were seated in their home cage or in the primate test chair. They disappeared during associated reflex and voluntary movements of the limbs. However, they would occur in one limb while the other limb was making voluntary movements. These motor acts consisted of isolated movements usually of single joints, which might be repeated several times or immediately involve another adjacent joint or a far distant one. These movements most frequently were asynchronous in the two limbs and usually involved movements at different joints. The movements in order of their frequency were: (i) flexion-adduction of the thumb followed immediately by extension and abduction; (ii) opposition and then spreading of the thumb and index finger; (iii) flexion then extension of the wrist; (iv) abduction then adduction of the wrist; and (v) supination then pronation of the arms.

There are at least three factors which may complicate the time course of the recovery of the deafferented forelimb: (i) difference in emotionality of the rhesus and the speciosa monkey; (ii) the use of the collar to prevent the animal from mutilating the deafferented forelimbs; and (iii) spinal cord in jury due to the wide exposure for rhizotomy and lack of proper dorsal ligamental support of the neck due to the extensive laminectomy (C2-T3).

The time course and recovery of movements in rhesus monkey, M7, was generally slower than in the speciosa animals (Table II). Reflex movements of fending, to visual threat, pin prick and to drop had essentially the same pattern as in the speciosa monkeys, but a slower recovery.

The slow recovery of feeding movements in the rhesus monkey was no doubt influenced by his refusal to take food in the first few days and his reluctance to take food from the observer's hand for 1 or 2 weeks. The lack of attempts of the rhesus monkeys to right themselves during the first 3 or 4 days, with the vicious biting of their deafferented arms or hands, in addition to the discrete biting of the nails and balls of the fingers, indicate that the differences of temperament might play a significant role in the recovery of the deafferented limb in the two species.

The necessity to apply a collar at frequent intervals for the prevention of self mutilation of the deafferented arms limited the use of the limbs at least for this purpose. If the restraining collar was left on continuously for two or three days, the animal's movements were poorer for one or two days after removal than prior to its application.

In all the speciosa monkeys a kyphosis at T2 and a depression of the

reflexes of the hind limbs occurred 3 or 4 weeks after deafferentation. Initially this involvement was hardly detectable clinically. Sometimes the symptoms were unilateral but soon became bilateral. The earliest symptoms were loss of tactile placing reactions. Later (5–10 weeks) hopping, supporting tone and walking became involved. The last deficits to appear were the use of limbs in visually guided behavior. Monkey M7 did not show these deficits of the hindlimbes even after 8 weeks. Involvement of the forelimbs during this period of motor impairment of the hind limbs was not as readily detectable due to its lack of autogenic reflexes, but the arms could not have been as seriously involved as the hindlegs.

II. Effects of cerebellar nuclear lesions on the movements of bilaterally deafferented forelimbs

In two animals with extensive but incomplete recovery of movements following deafferentation of both arms (M8 and M1, Table I, Group I), subsequent cerebellar lesions resulted in some decompensation and marked cerebellar dyskinesia (Fig. 3*F*). The decompensation and cerebellar ataxia was limited to the homolateral deafferented limb in M8 with a unilateral cerebellar lesion, and to both forelimbs in M1 with a bilateral cerebellar lesion. The resting posture of the cerebellar-deafferented (CD) forelimb showed an increased protraction and adduction of the shoulder and increased flexion of the elbow and fingers. The movements of the CD arm during feeding, grooming and postural adjustments were all slower, clumsier and the fingers could not pick up small bits of food. The grasp reflex of CD arms, although still strong, was noticeably weaker. There was also some exaggeration of the associated flexor movements of the CD arms as the animal extended his neck to take food offered to his mouth. However, there was no reversion to the flexor phase in visual placing and vestibular drop reactions.

In goal directed acts, such as grooming a site on which itch powder had been placed, or in grasping food, or placing food in the mouth, frequent errors of overshoot were committed by the CD arm and the subsequent attempts to correct for the errors resulted in ataxia and tremor (Fig. 3*F*). Adjustment of the CD forelimbs from a static posture to a new position also resulted in ataxia and tremor. If the CD forelimb was held abducted from the shoulder and suddenly released, it fell freely, rather than being lowered; and if the CD hand carrying food to the mouth was stopped by the observer and then suddenly released, the animal banged his face or body with the flexed arm. Braking movements preventing free limb drop and rebound slapping of the face can be accomplished in the deafferented forelimbs of animals without cerebellar

lesions, provided the animals are visually attending to the movements of the limbs.

Monkey 8 with both forelimbs deafferented, had subsequently a left interposed and dentate nuclear lesion and the cerebellar symptoms were confined to the ipsilateral forelimb (Fig. 3*F*). This CD limb was used for feeding only after the right limb was restrained. The cerebellar symptoms became bilateral after the placement of a right cerebellar lesion in the interposed dentate and fastigial nuclei (Fig. 3*G*). It was interesting to note after the right cerebellar lesion that oscillation of the head occurred during drinking from a fount or while trying to fixate a visual or auditory stimulus. These latter symptoms are attributed to a lesion of the right fastigial nucleus. After the second cerebellar lesion (right dentate, interposed and fastigial nuclei) a nine per second tremor was noted in the left forelimb when the animal was suddenly frightened or pushed to the left side. This tremor consisted of a rapid supination-pronation movement of the left arm and at times a tremulous movement of the thumb and index finger. This tremor disappeared when the limb was used for feeding and was replaced by cerebellar ataxia and ataxic tremor. Since the interval between the left and right cerebellar lesions was only four days, we do not know if the rapid tremor would have developed without adding the second cerebellar lesion.

With the passage of time the cerebellar deficits were somewhat compensated, but considerably less than would have been anticipated from cerebellar lesions in otherwise normal animals or, for that matter, the compensation which occurred in the afferented hind limbs of these animals.

III. The effects of bilateral deafferentation in animals with unilateral cerebellar neuclear lesions

In four animals (Table I, Group II) cerebellar nuclear lesions were made initially, and after varying periods of compensation both forelimbs were deafferented. All of these animals (M3, M4, M6, M7) had unilateral dentate and interposed nuclear lesions. They showed typical cerebellar symptoms limited to the ipsilateral limbs (*see* Growden et al. 1967). Immediately after the cerebellar operation, the involved forelimb was adducted at the shoulder and flexed about 50° at the elbow, with the forearm in mid-supination pronation and the fingers slightly flexed. The animals were reluctant to use the involved limbs for support or feeding. The hand, when forced to reach showed markedly extended and fanned fingers, which could not grasp small bits of food. With more attempts to use the limb wild ataxia and discoordination occurred in all goal

directed movements (i.e. grooming, feeding, placing reactions and attaining new support postures). The movements were slow in starting and stopping and abnormal fixation and irregular sequence of movements occurred in most acts. In attempting to pick up raisins, the arm extended with the fore arm held in midsupination, the wrist dorsiflexed and the fingers extended and fanned; the whole arm was then circumducted as an attempt was made to grasp the raisins in a flexed clawlike hand.

The inability to check movements following sudden release by the observer of the restrained arm during feeding led to the animal striking himself or letting the passively abducted arm fall freely without lowering it. After a few days the goal directed movements were better coordinated and the errors due to overshooting the target were less marked as were the subsequent attempts for correction (Fig 5*BD*). Thus the wild ataxia was gradually replaced by tremor. The slowness of movement and the impairment of checking gradually improved as did the dexterity of the fingers in grasping, grooming and manipulating small bits of food. The ataxia and tremor of the hind limb also showed similar compensatory changes. Fatigue, excitement, distraction and difficult tasks all partially decompensated the compensated movements. In addition an "anticipatory tremor" (Growden et al. 1967) of nine per second was observed in the cerebellar-involved limbs of M6, 5 days after a unilateral dentate and interposed lesion. This supination-pronation tremor occurred just prior to its use for placing or feeding and at other times, when the animal used the normal limb for feeding.

Bilateral deafferentiation of the forelimbs was then performed to compare the pattern, rate and extent of recovery of a simple deafferented limb with a limb involved by both cerebellar lesion and deafferentation. Data from such preparations also permitted one to determine if deafferentation led to decompensation of the "cerebellar-involved" limb. The time course and pattern of recovery of the deafferented forelimb of M3, M4, M6 contralateral to the cerebellar nuclear lesion did not differ from the time course and pattern of recovery of forelimbs that were subjected to deafferentation alone (*see* Section I, p. 267). The deafferented limb, ipsilateral to the cerebellar lesion, also showed the same general pattern of recovery but the recovery time was slower and less complete. The inappropriate flexor movements of the forelimbs to visual placing, vestibular drop reflex, visual threat and fending against pin prick was replaced by appropriate extension earlier in the simple deafferented limb than in the cerebellar deafferented limb (Table II). Other movements associated with feeding, grooming and postural adjustment of the limb also showed a similar slowness of recovery in the cerebellar-deafferented limb. The cerebellar deafferented limb even after considerable recovery could not be used for grooming and

feeding unless the animal was prevented from using the simple deafferented limb. A striking difference between the two limbs was seen in the self mutilation responses. The biting of the fingernails and skin of the ball of the fingers occurred only on the hand contralateral to the cerebellar lesion. We believe the failure to bite the CD limb is due to the initial slower recovery and persistent difficulty in maneuvering this hand for the delicate and deliberate biting. In contrast the early recovery of delicate hand movements during biting of the fingers of the simple deafferent limb may result in the formation of a habit, possibly within a critical period of time. That the habit may be the most important factor is supported by the continued biting of a deafferented limb after cerebellar lesions are subsequently added (M8, M1, M3), even though the movements are now ataxic and clumsy.

Cerebellar ataxia and tremor of the forelimb, during the first days after deafferentation were absent or difficult to demonstrate as there was little or no goal directed movement (*see* Fig. 1*FH*). The stereotyped, flexor, associated movements of the limbs occurring during the taking of food by the mouth, or the stereotyped movements of flexion during visual placing, visual threat and drop reflexes never showed any ataxia and tremor. Ataxia and tremor reappeared with the recovery of manual feeding, grooming, visual placing, fending against a pin prick or threatening brush or attaining a new support posture. At first, the ataxia was mild and primarily limited to the shoulder, as movements improved the ataxia and tremor became more marked and involved more joints.

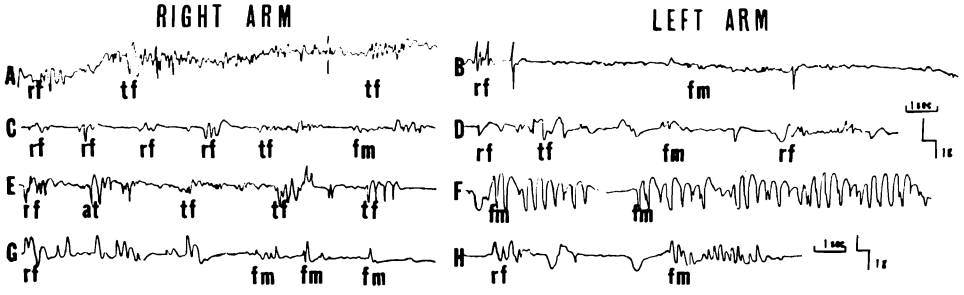

Fig. 4. Accelerometer records showing slow compensation of a unilateral cerebellar dyskinesia in the deafferented arms of M3 and the slight decompensation produced by a contralateral dentate and interposed nuclear lesion. *A* and *B*: records of arm movements 140 days after a right dentate and interposed cerebellar lesion and 73 days after deafferentation. *C* and *D*: records 223 days after right cerebellar lesion and 156 days after deafferentation. *E* and *F*: records 225 days after right cerebellar lesion, 2 days after a left dentate and interposed cerebellar lesion and 158 days after deafferentation. *G* and *H*: records 252 days after right and 29 days after left cerebellar nuclear lesions and 185 days after deafferentation. Abbreviations: same as in Fig. 2 and 3, except *at*: attempt to take food. Calibrations: *A–F* to the right of trace *D*; *G* and *H* to the right of trace *H*.

Clearly the cerebellar dyskinesia was decompensated by deafferentation (Fig. 1*EHIK* and 5*BDE*).

Monkey 6, on the first day after bilateral deafferentation showed a nine per second tremor of supination and pronation of the cerebellar involved forelimb (Fig. 1*F*). This tremor sometimes appeared in the pendant limb when the animal was offered food or threatened by another monkey or by the observer and disappeared with associated or goal directed movements (Fig. 1*HK*). It persisted unchanged throughout the survival period of 98 days. This dyskinesia had the same frequency of oscillation and pattern of movements as the "anticipatory tremor" seen before deafferentation.

Slow compensation of the cerebellar symptoms occurred in the ensuing months, but this was never as great in the deafferented as in the afferented limbs of other animals. Decompensation of the cerebellar symptoms by forelimb deafferentation occurred in both the fore and hind limbs. The hind limb decompensation was of shorter duration and less marked than for the forelimb.

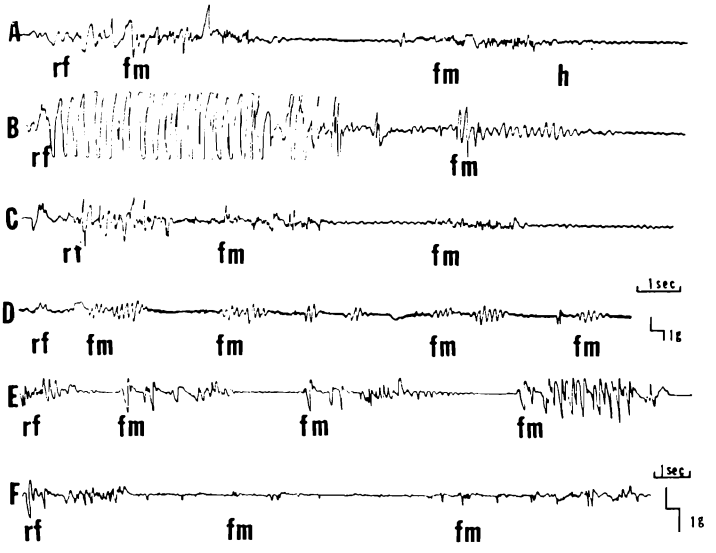

Fig. 5. Accelerometer records of M7 showing early stages of compensation of cerebellar ataxia and tremor in a forelimb, during feeding and their subsequent decompensation following bilateral deafferentation of the arms. *A*: record of movements of right arm prior to any operations. *B*: records of movements of right arm and *C*, of left arm 2 days after a right cerebellar dentate and interposed nuclear lesion. *D*: records of right arm 15 days after the cerebellar lesion. *E* and *F*: records 97 days after the cerebellar lesion and 56 days after deafferentation of the arms. *E*, movements of the right and *F*, of the left arm. Abbreviations: same as in Fig. 2 and 3. Calibrations: *A–D*, to right of trace *D*; for *E* and *F*, to the right of trace *F*.

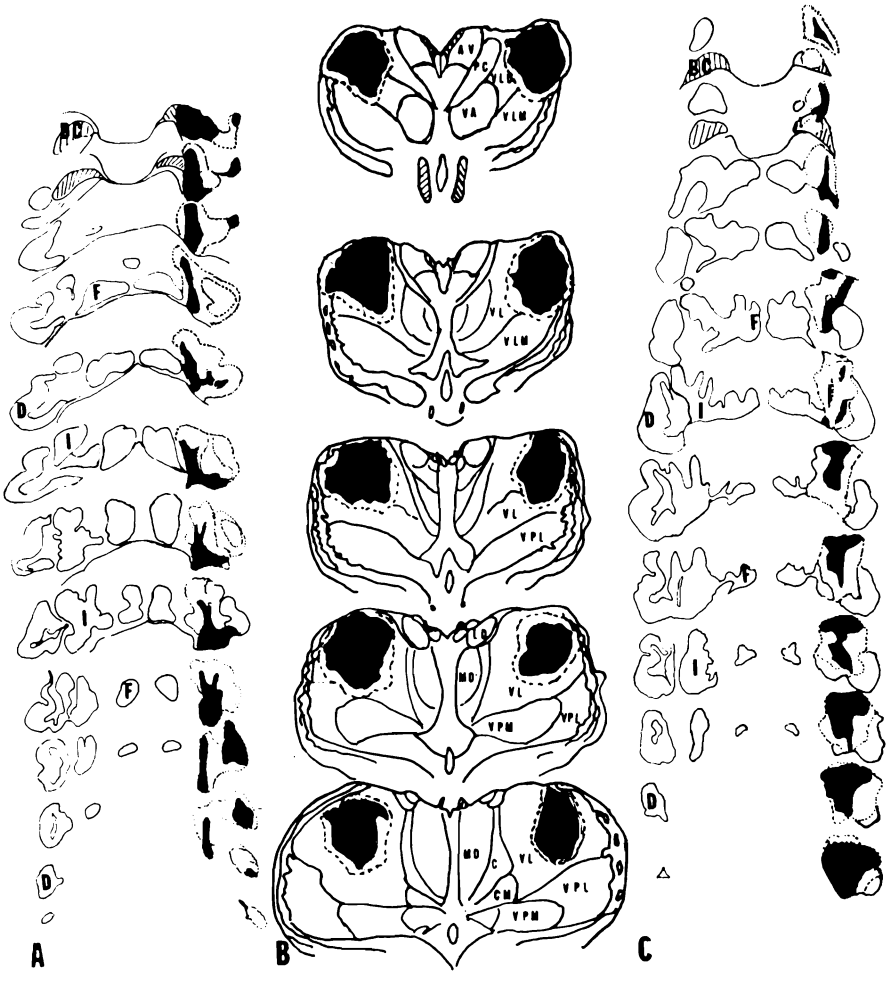

Fig. 6. Projections drawings of cerebellar and thalamic lesions in Monkey 6 and cerebellar lesion in Monkey 7. *A*: unilateral cerebellar lesion of the right brachium conjunctivum and interposed and dentate nuclei of M6. *B*: bilateral thalamic lesions of the cerebellar relay nuclei, ventralis lateralis of M6. *C*: unilateral cerebellar lesion of the right brachium conjunctivum and dentate and interposed nuclei of M7. Lesion cavity indicated by wavy parallel lines and gliosed region outlined by dashed lines. Abbreviations: D, dentate nucleus; F, fastigial nucleus; I, interposed nucleus; BC, brachium conjunctivum; AV, anterior ventralis; C, central lateralis; CM, centrum medianum; LD, lateralis dorsalis; PC, paracentralis; VA, ventralis anterior; VL, ventralis lateralis; VLM, ventralis lateralis, pars medialis; VLO, ventralis lateralis, pars oralis; VPL, ventralis posterior lateralis; VPM, ventralis posterior medialis.

Three attempts were made to further determine the source of this compensation: (i) a lesion was placed in the left cerebellar interposed and dentate nucleus of M3, 223 days after a right cerebellar nuclear lesion and 156 days after bilateral deafferentation; and (ii) bilateral stereotaxic lesions were placed in the nucleus ventralis lateralis of the dorsal thalamus of M6, 89 days after unilateral cerebellar nuclear lesions and 77 days after bilateral deafferentation. Monkey 3 showed new cerebellar symptoms of the limb ipsilateral to the most recent cerebellar lesion and decompensation of the contralateral limb (Fig. 4*FHEG*). Monkey 6 with the thalamic lesion (Fig. 6*B*) showed decompensation of movements in the cerebellar deafferented limb and the appearance of cerebellar symptoms in the other deafferent limb.

DISCUSSION

Although the effects of bilateral deafferentation of the forelimbs of the primate has been studied by several investigators (Knapp et al., 1963, Taub and Berman 1964, and Bossom and Ommaya 1968) with generally similar findings, we thought it necessary to further document the deficits and time course and pattern of recovery to serve as a basis for evaluating the effects of prior or subsequent cerebellar nuclear lesions. Previous studies (Taub and Berman 1968, and Bossom and Ommaya 1968) using the *Macaca mulatta* had been primarily concerned with the conditioned responses of the deafferented limbs and paid less attention to the time course and patterns of recovery of movements.

In the present study we have found the pattern of recovery to be similar for the *Macaca mulatta* and *Macaca speciosa.* The recovery was however slower in the mulatta than the speciosa. This we attributed to the fact that our two mulatta monkeys appeared more emotionally disturbed after deafferentation and were more hesitant to use the limb for several days.

The first movement to recover in the deafferented forelimbs was emptying of the pouch with the dorsum of the wrist and manipulating the fingers and thumbs so as to bite the nail and the skin of the tips of the digits (Table II). These movements were performed with precision and without ataxia and tremor. On the other hand when the animal tried to extend his forelimbs for visual placing to prepare for support on being suddenly dropped or to fend against a pin prick or reach for proffered food all joints flexed drawing the hand beside his head rather than extending the arm as in normal animals. The recovery from this flexor pattern was first noted in the dorsiflexion of the wrist and extension and fanning of the fingers. Only later (Table II) were the animals able to extend the elbow and protract the shoulder. It was

also noted that the monkeys could grasp food with the hand and manipulate it for small bites if the food was placed between their lips, while they were unable to reach for food at this time.

The recovery of reaching was characterized by the jerky sudden extension of the limb with a marked ataxia and missing the food. There was a clear lack of proper coordination of movements and proper joint fixation. However, if the shoulder and elbow were stabilized by the observer, the wrist and finger movements were adequate for seizing small bits of food. Although, there were deficits of movements both at the base and apex of the limb, the first recovery for all types of movements were distal before proximal and the animal soon learned to adduct and fix his upper arm and shoulder to use his limb better during feeding. That some distal movements were also severely involved initially, was seen by gradual recovery of the ability to pick up a raisin between the thumb and forefinger or to groom with the discrete digital movements of the normal animal. When the distal movements had recovered their delicate coordination and precision, the animals still showed poor fixation and coordination of the shoulder joint, especially when reaching for food with the arm fully extended above his head. These ataxic movements of the shoulder were never fully compensated. Thus it would appear that the deficits and pattern of recovery of movements are nearly the opposite to that found after bulbar pyramid lesions (Tower 1940, Bucy et al. 1966, Growden et al. 1967, Lawrence and Kuypers 1968). The failure of the unilateral deafferented forelimb of the monkey (Mott and Sherrington 1895, Lassek 1953 and Twitchell 1954) to perform any but the crudest voluntary movements and the failure to use this hand and fingers for feeding or grooming was interpreted as being primarily due to a loss of sensation from the lower arm and hand, which caused a disruption of the function of the somatomotor cortex and its efferent pathways (chiefly pyramid tracts). However, rather than disruption of the pyramidal function being the chief cause of the loss of voluntary movements, a loss of motivation to use this deafferented limb is indicated by the study of Taub and Berman (1964). They were able to get a recovery of voluntary movements of the hand by formal conditioning procedures.

On the other hand some responses for stance and locomotion (drop, tilt, visual placing) after bilateral deafferentation of the arms showed a more severe impairment and less compensation, than did the skilled voluntary movements of the hand. Denny-Brown (1966) suggests that these deficits for stance and locomotion may be due to loss of tonic neck reflexes and a release of body righting reflexes acting via the mesencephalic ventral reticulospinal tract.

The recovery of movements following unilateral (Twitchell 1954) or

bilateral (Denny-Brown 1966) deafferentation is said to be in part compensated by the animal employing the neck reflexes (with C2 and C3 intact, *see* Berman et al. 1960), the kinetic labryinthine reflexes, and visuomotor and possibly other afferent sources.

Taub and Berman (1968) and Bossom and Ommaya (1968) have made some attempts to determine the factors which aid in the recovery of goal directed movement following bilateral forelimb deafferentation. It is clear that formal conditioning, although requiring more trials to learn a particular response in a naive animal with deafferented arm than in a normal animal, is a potent means of aiding recovery of movements (Taub and Berman 1964). Such conditioned animals can learn motor tasks without the aid of vision (Taub and Berman 1964 and 1968). In our preparations the monkey after recovery of feeding with vision was able to reach and grasp food placed in between its teeth and to manipulate the food to take repeated small bites with vision occluded. They could protract, adduct and extend the forelimbs with food grasped in their hand and bring it accurately back to their mouth just as well without vision as with vision.

The ability of the deafferented arms to accurately locate an extra corporal position without vision was also obtained in this study. This was accomplished by first training the deafferented animal by touching the face and have him take food at a fixed distance from the mouth. If the upper lip was touched he reached 5 cm above and 10 cm away from the mouth; and if the lower lip was touched he reached 5 cm below and 10 cm away from the mouth, grasped the food and took it to his mouth. With vision excluded the animal could reach to the appropriate position in space when either lip was touched. But the grasping of food was by mere chance, the animal usually carrying the empty hand, to the mouth, then lowering the arm to the start position and trying again. At other times, he would move his empty hand from the mouth to the appropriate place in space and grasped for the food.

If the arm was passively displaced from the start position before the lips were touched, or from an assumed position during feeding, he missed the target. The error was related to the direction and extent of the passive movement. Passively increased flexion from the start position resulted in reaching above and too near the mouth even when the lower lip was touched. Other similar displacements such as abduction and protraction resulted in not enough adduction. Struggling or fending movements also interefered with these feeding movements when vision was excluded. If the animal's limb was passively moved so that he missed his mouth, he would usually lower the limb to the postural rest position and if possible adduct it against his chest and then repeat the feeding

movement accurately. At other times the animal might touch the side of the face and then make a corrective movement with the limb.

The above behavior with vision occluded suggests that the animal seated in the test chair knows the start position of his limb, keeps up with his motor discharges by central feed-back and has an intact body image. This behavior is similar to that described for a deafferented arm performing conditioned avoidance and visuomotor adaption to prisms (Taub and Berman 1968, and Bossom and Ommaya 1968) without vision of the arms. This type of conditioned behavior was postulated by Taub and Berman (1968) to be controlled by topological central feed-back or by an engram without any feed-back. Bossom and Ommaya (1968) found that lesions of the caudate nucleus abolished the reaching and grasping if vision was occluded, but had no effect on visually guided movements.

Unilateral cerebellar dentate and interposed nuclear lesions made prior to bilateral deafferentation of the arms does not alter the time course and pattern of recovery of the limb contralateral to the cerebellar lesion. The ipsilateral limb (cerebellar deafferented arm, CD) also showed the same general pattern of recovery as the simple deafferentated arm but it was much slower (Table II) and was wildly ataxic and poorly coordinated especially at the distal joints. Grasping, grooming and manipulating food for small bites with the CD arm was crudely performed with ataxia and tremor. The CD arm could not groom with delicate finger movements or pick up a raisin between his opposed finger and thumb. Also, he never mutilated the hand of the cerebellar involved limb, which we thought to be due to the lack of delicate hand movements during the early stages after deafferentation (*see* p. 279) of this CD arm. The ipsilateral influence of the dentate and interposed lesion was also found by first deafferenting both arms and subsequently making the cerebellar lesion. In this sequence the animals continued to perform the recovered movements but with marked ataxia and tremor and impairment of distal movements. These animals, however continued to mutilate the fingers even though the cerebellar hand lacked the delicate movements and was dyskinetic (*see* p. 279). The severe impairment of distal movements in the cerebellar deafferented arm is quite similar to the effects of combined pyramid and dentate and interposed lesions reported by Growden et al. (1967). It appears from these results that these cerebellar nuclei are important for the rate and extent of recovery especially of the distal movements of the deafferented limb and that this compensation may be in large part via the pyrimidal system.

Dentate and interposed lesions while producing profound distal impairment of movements and marked dyskinesia in a deafferented limb resulted in no detectable loss of recognition of goal position. For the

conditioned animal with or without vision would ataxically oscillate around the appropriate extracorporal space when the lower or upper lip was touched. However the animal's impaired grasp and ataxic tremor was so marked as to render the grasping of food nearly impossible when vision was excluded. It was also clear that vision could not greatly compensate for this deficit since he showed the same general reaction with vision.

The nature of the cerebellar deficit is suggested by two additional observations with the animal having full vision of his arms. If the deafferented cerebellar limb was passively extended and abducted to shoulder height and suddenly released the limb was not lowered to the rest position as was the simple deafferented limb, but fell as a dead member. Also, if the animal was feeding and the arm was restrained while he tried to carry it to the mouth the cerebellar deafferented limb hit the face while the simple deafferented limb was braked before striking the face. We believe that the primary deficit in the cerebellar deafferented arm is due to a failure to start and stop movements with the proper timing resulting in an overshoot. This overshoot with a preservation of a position endpoint and an attempt to correct for the error produces the ataxic tremor just as postulated by the experiment of Growden et al. (1967) from cerebellar lesions in the normal or a monkey with pyrimid section.

Denny-Brown (1966) observed that a unilateral deafferented limb in a cerebellectomized primate failed to show ataxia and tremor, while the afferented limb did. Thus he concluded that ataxia and tremor was a disturbance between alpha and gamma motor neuron regulation. We interpret his results as to the absence of ataxic tremor in the cerebellar deafferented limb to the fact, that the movements of this limb were associated movements without goal direction. For we never saw ataxic tremor in the cerebellar deafferented arm during associated arm flexor movements when the animal tried to take food with the mouth. We, however, saw ataxic tremor with emptying the pouch and when scratching or grooming a locally irritated area.

The proposed role of a disturbance of gamma motor neuron discharges as the primary causal factor for the motor deficits and their subsequent recovery for motor compensation in the cerebellectomized cat (Glaser and Higgins 1966 and Van Der Meullen et al. 1966) and monkey (Denny-Brown 1966) is questioned by the present observations. It appears likely that recovery of extracerebellar control of alpha and gamma motor neuron regulation occur concurrently and both are important for compensation following cerebellar lesions. For animals with major compensation of deficits from dentate and interposed nuclear lesions were decompensated and showed slow recompensation following bilateral deaf-

ferentation of the arms. Also cerebellar lesions in animals with previously deafferented arms showed slower compensation than animals with cerebellar lesions alone. The complexity of the neural organization responsible for cerebellar compensation was suggested by the decompensation of cerebellar dyskinesia by adding a pyramid lesion to a prior dentate and interposed nuclear lesion (Growden et al. 1967). This combination of lesions resulted in similar decompensation to that described in the present study and in both instances may be due to pyramidal disturbance, in the first instance by severing the tract and in this instance by reducing its functional activity.

SUMMARY AND CONCLUSIONS

Seven monkeys were studied after bilateral deafferentation of the forelimbs (C2-T3). Two of these had subsequent and four had prior stereotaxic lesions of the dentate and interposed cerebellar nuclei. Reflexes originating outside of the deafferented arms (vestibular drop, visual placing and postural responses to tilt) and volitional responses (feeding, fending, grooming and locomotion) were recorded clinically, cinematographically and with an accelerometer.

Bilateral deafferentation of the forelimbs resulted in a mild and enduring flexor posture of the arm. Initially an inappropriate flexion of all joints was elicited by drop, threat and reaching for food. These responses were replaced by appropriate extensor movements by three weeks. These movements were at first jerky and ataxic. There was a clear proximodistal gradient of impairment, with the fixation and movement of the proximal joints being most severely involved. Well coordinated wrist and finger movements without ataxia or tremor appeared as early as one day for biting the skin of fingers and by 21 days for grooming single hairs and picking up raisins. Postural response to tilt and movements of the limbs for walking and climbing showed the poorest recovery.

Subsequent unilateral dentate and interposed cerebellar lesions resulted in marked ataxia and ataxic tremor in goal directed acts (grooming, feeding) of the ipsilateral arm. Distal movemements of the ipsilateral limb after cerebellar lesion were so impaired that the hand could no longer be used for grooming single hairs or picking up raisins.

Unilateral cerebellar nuclear lesions followed by bilateral deafferentation resulted in similar cerebellar symptoms limited to the ipsilateral limb. The cerebellar deficits appeared only after the recovery of the goal directed movements and deficits were more severe than prior to the deafferentation. Recovered movements in the deafferent cerebellar animals were essentially unaltered by exclusion of vision.

It was concluded: (i) that deafferentation of the forelimbs results in

deficits of motor responses, which are the reciprocal of those following bulbar pyramid lesion; movements of the fingers and wrist are well executed, while fixation and movements of the proximal joints and postural locomotor use of the arm are severely impaired; (ii) a central programming of movements, with continuous monitoring by central feedback mechanisms must occur as the recovered movements do not depend upon vision; (iii) typical cerebellar dyskinesia is primarily of central origin for it develops in the deafferented limbs following dentate and interposed nuclear lesions. These deficits are not due to the disruption of the alpha-gamma linkage; an (iv) the grave impairment of the movement of the hands after cerebellar lesions and deafferentation suggest that the lateral cerebellar nuclei play an important role in the function of the pyramidal system in the monkey with deafferented arms.

This work has been supported by research grants from the U.S. Public Health Service number 5 RO1 NS08768 and UCPR-187-65 from the United Cerebral Palsy Foundation.

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Received 12 November 1970