ANALYSIS OF RESIDUAL WEIGHT DISCRIMINATORY ABILITY AND EVOKED CORTICAL POTENTIALS FOLLOWING SECTION OF DORSAL COLUMNS IN MONKEYS[1,2]

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(Received December 18, 1963)

Complete section of the dorsal columns at the level of C_2—C_3 caused a transient loss in weight discriminatory ability, which was recovered with training. Additional lesions of the restiform bodies in the caudal medulla were without effect. An alternate sensory pathway in the anterior columns was suggested when combined anterior and posterior lesions produced a greater initial deficit than that occurring after complete section of the dorsal columns. Parietal lobectomy secondary to dorsal column lesions produced a slight but persistent deficit in weight discrimination, indicating that the pathways responsible for recovery of function project in part to the parietal lobe. Electrical stimulation methods confirmed the existence of an afferent pathway from deep nerves to the parietal cortex, independent of the dorsal column relay system.

Clinical studies, notably those by Head and his co-workers (1906, 1911), indicate that lesions of the parietal lobe and, at a lower afferent level, of the dorsal columns severely affect weight discrimination. Ruch (1934) found that weight discriminatory ability is not focally localised in the parietal lobe; bilateral ablations of the postcentral gyri or of the posterior parietal lobes did not permanently impair weight discrimination by monkeys. Furthermore, complete ablation of the parietal lobe reduced rather than abolished the ability of chimpanzees to discriminate between weights (Ruch, Fulton, and German, 1938). Similarly, Sjöqvist and Weinstein (1952) found that weight discrimination was not permanently impaired by section of the medial lemniscus in the midbrain of the chimpanzee, nor by section of the superior cerebellar peduncle alone. However, combined lesions of the medial lemniscus and dentato-rubro-thalamic fibres produced a profound loss of discriminatory ability. Sjöqvist and Weinstein's findings, considered with those of Ruch, suggested that cerebellar projections to the precentral cortex may subserve weight discrimination.

The existence of a cerebellar component from the forelimbs in the cervical dorsal columns (Ferraro and Barrera, 1935) affords an opportunity to test this thesis. The unexpected finding that transection of these columns in monkeys causes only

1. Supported in part by a grant from the Washington State Research Fund for Biology and Medicine.

2. Submitted by Dr. Devito in partial fulfilment of the requirements for the Ph.D. degree, University of Washington.

a transient loss of the ability to discriminate weights led to an investigation of what other spinal pathways might be involved in this function. Lesions were made in the restiform bodies and the anterolateral columns. In one animal, parietal lobectomy was performed after section of the dorsal columns in the belief that any further deficit would give information about the termination of the tracts mediating the residual function. In addition, the spinal pathways for impulses from skin and muscle were traced by the electrical method in both monkeys and cats.

METHODS

Monkeys *(Macaca mulatta* and *Cercocebus torquatus atys)* were trained to discriminate weights on the horizontal frictional pull-in apparatus devised by Klüver (1933). The animal was presented with two strings attached to boxes identical in all respects but weight, and was trained to select the lighter box, which contained food. The weight of the standard box was 270 gm. To eliminate visual cues, the boxes were kept on the same sides of the table, and the weights were shifted in a predetermined random order. In one series, the standard weight of the box was varied in order to establish that the animal was responding to the lighter box and not to an absolute weight. A masking noise was used to cover possible sound differences. In some instances, the table was covered so that the monkey could not see the boxes until the one it had selected was pulled to the end of the table. Learning was indicated when a monkey began to test the resistance to tugs on the strings before pulling in a box.

The weight ratios were decreased when the general level of performance reached 80 per cent correct choices. To obtain a more complete curve, the attainment of a criterion was not required on small weight differences. The weight difference was continually reduced until the level of performance fell to 40 or 50 per cent error trials. On small weight differences, a cautious habit of working was encouraged by occasional "double-baiting," i. e., rewarding the incorrect choice. Two or three preoperative series were obtained in an effort to ascertain the animal's true capacity. After three weeks without trials, pre-operative retention tests were given to determine whether discriminatory ability would be lost during a period about equal to the interim necessary for post-operative recovery. Most animals recovered sufficient motor power within three weeks after operation to permit retesting.

In six monkeys, the dorsal columns were sectioned at the level of C_2-C_3 alone or in combination with other neural lesions. The dorsal columns were interrupted at the primary operations on three animals (DC1, DC2, DC3). In one of these (DC3) lesions were placed in the restiform bodies at a second operation. Parietal lobectomy with bilateral ablation of sensory area II was performed subsequent to dorsal column section in another (DC1). In monkey DC5, restiform body lesions preceded dorsal column section. The anterolateral columns were sectioned bilaterally at C_2-C_3 in monkey DC6, and the dorsal columns were divided at a second operation. The sixth animal (DC4) sustained combined section of the

anterior and posterior columns, but did not recover sufficient motor ability to permit postoperative testing.

In terminal experiments on the monkeys and acute experiments on cats, the animals were anesthetized with sodium pentobarbital or Dial, and the cortices were explored for evoked potentials. Peripheral nerves were stimulated with square waves, 0.5 msec. in duration. The potentials evoked were recorded with wick electrodes and led into a differential amplifier.

RESULTS

After complete bilateral division of the dorsal columns (fig. 1A-C), monkeys DC1, DC2 and DC3 performed weight discriminations as indicated in fig. 2, which

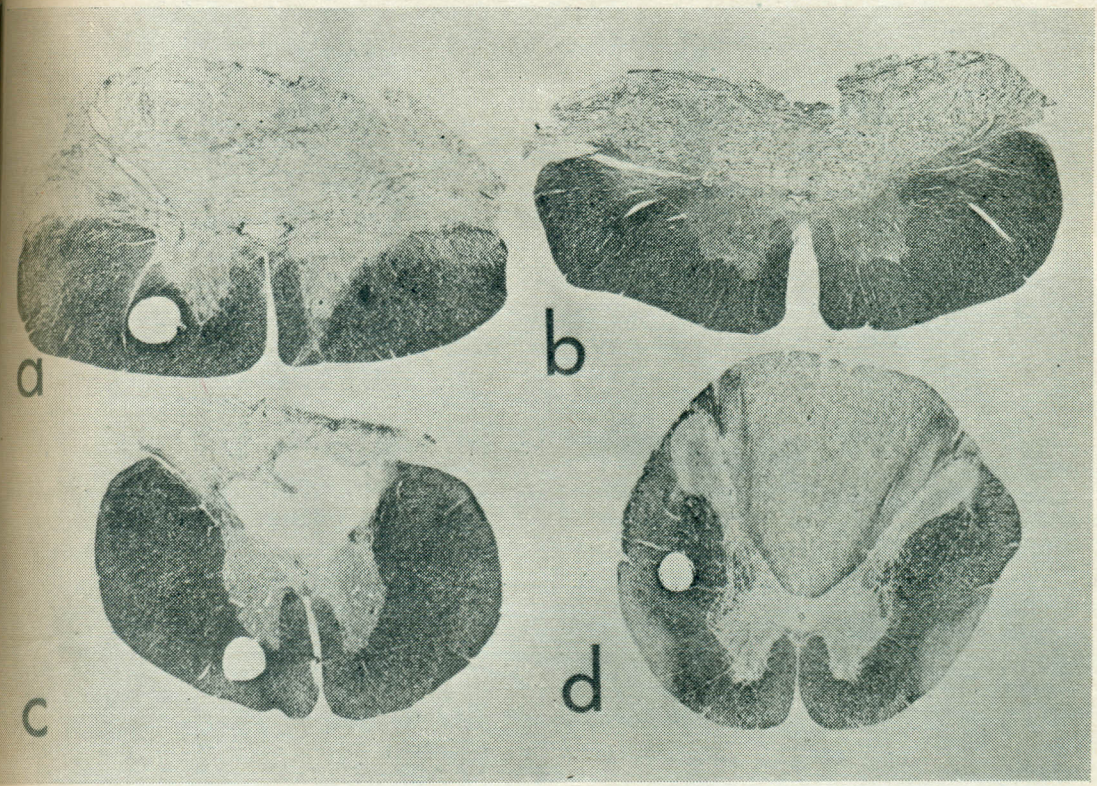

Fig. 1. Spinal cord lesions in monkeys, Weil stain, 20X. Circular puncture marks indicate right side. A, monkey DC1; dorsal and dorsolateral columns are completely replaced by scar tissue at level of C_{2-3}. B, monkey DC2; dorsal columns and upper borders of dorsolateral columns show complete degeneration at C_{2-3}. C, monkey DC3; dorsal columns are completely severed at C_{2-3} with no injury to other tracts. D, monkey DC6; degeneration of ascending fibers in dorsal and anterolateral columns at C_1. Intact fibers from C_3 show dark against fibers involved in lesions medially and lateral degenerated fibers from C_2 which were cut at time of anterolateral cordotomy.

shows the mean percentage error scores. The post-operative discrimination series illustrated do not show the initial deficits, which were greater in DC1 and DC2 than in DC3.

In monkeys DC3 and DC5, both the dorsal columns and the restiform bodies were sectioned, but not in the same order in both animals. The mean percentage errors by both monkeys before and after section of the restiform bodies is shown in fig. 3. The weight discriminatory ability of monkey DC5 was the same after dorsal column lesions as it had been after the lesions of the restiform bodies; however, the columns were found to be incompletely divided. The extent of the restiform body lesions in monkey DC3 is shown in fig. 4.

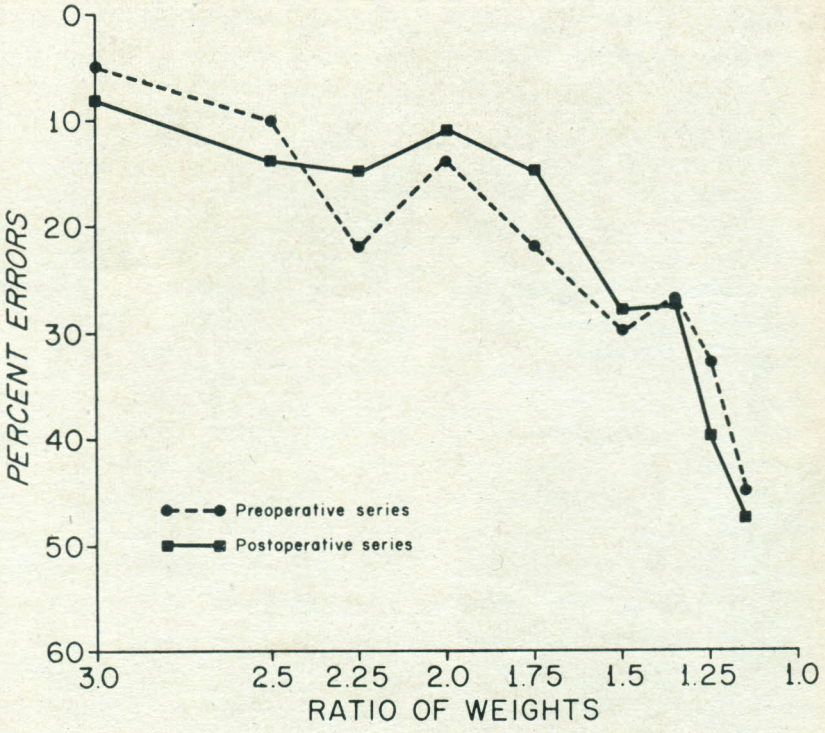

Fig. 2. Mean percentage errors of monkeys DC1, DC2, and DC3 in discriminating decreasing weight differences before and after dorsal column section.

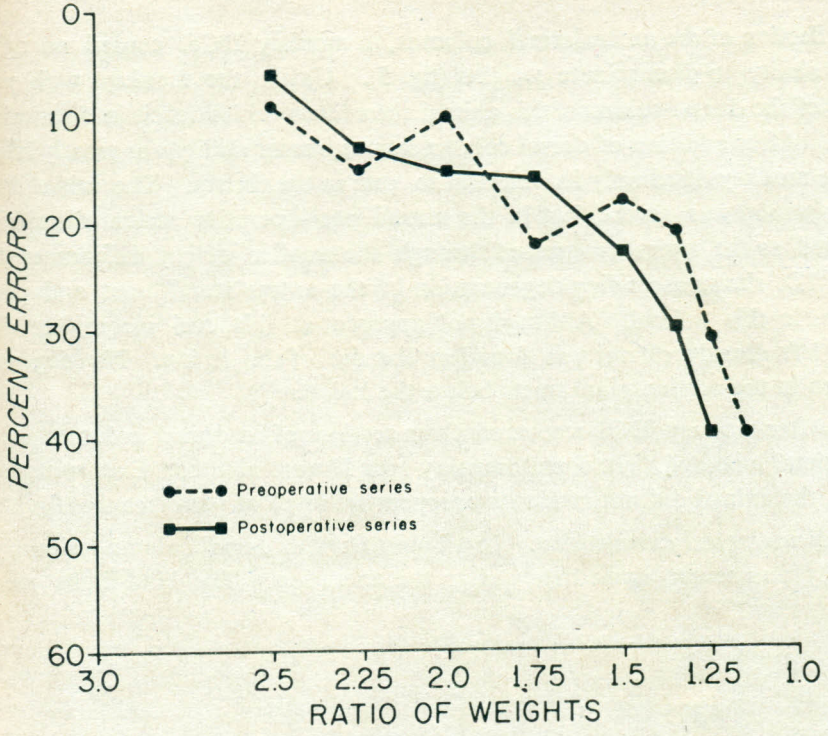

Fig. 3. Mean percentage errors of monkeys DC3 and DC5 in discriminating decreasing weight differences before and after section of restiform bodies.

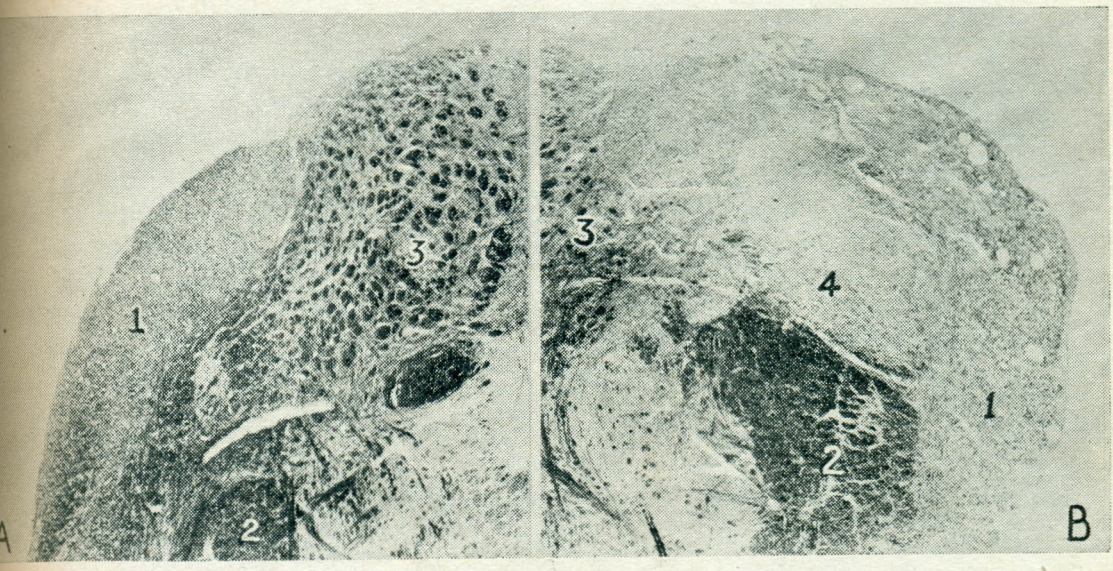

Fig. 4. Restiform body lesions in monkey DC3. On right side, majority or fibers in restiform body are demyelinated just rostral to lesion (A). On left side, restiform body is completely replaced by scar tissue at site of lesion (B). 1, restiform body; 2, tract of spinal V; 3, spinal vestibular nucleus and tract; 4, external cuneate nucleus. Weil stain, 37X.

Section of the anterolateral columns, in monkey DC6, caused no apparent loss of ability to discriminate weights (fig. 5). Unlike the monkeys with primary lesions of the dorsal columns, this animal showed no initial deficit in discriminatory ability. After addition of dorsal column lesions, motor ability was greatly impaired, and the monkey used only its left arm to pull in the boxes. The initial deficit in discrimination was severe, but in the second post-operative series, discrimination improved as the series progressed through the smaller weight differences (fig. 5). Figure 1D. shows ascending degeneration in the anterolateral tract and posterior columns in this animal. Although not apparent at this level, incomplete division of the left dorsal column was found at the site of the lesion. No degeneration was seen in the corticospinal tracts below the lesion.

After monkey DC1 recovered from section of its dorsal columns, its entire left parietal lobe and right somatosensory area II were ablated. Even with repeated testing, the animal did not attain its preoperative level of performance (fig. 6).

Neurological examination. In all animals with dorsal column lesions, motor disability was severe immediately after the operation. The hind-limbs recovered

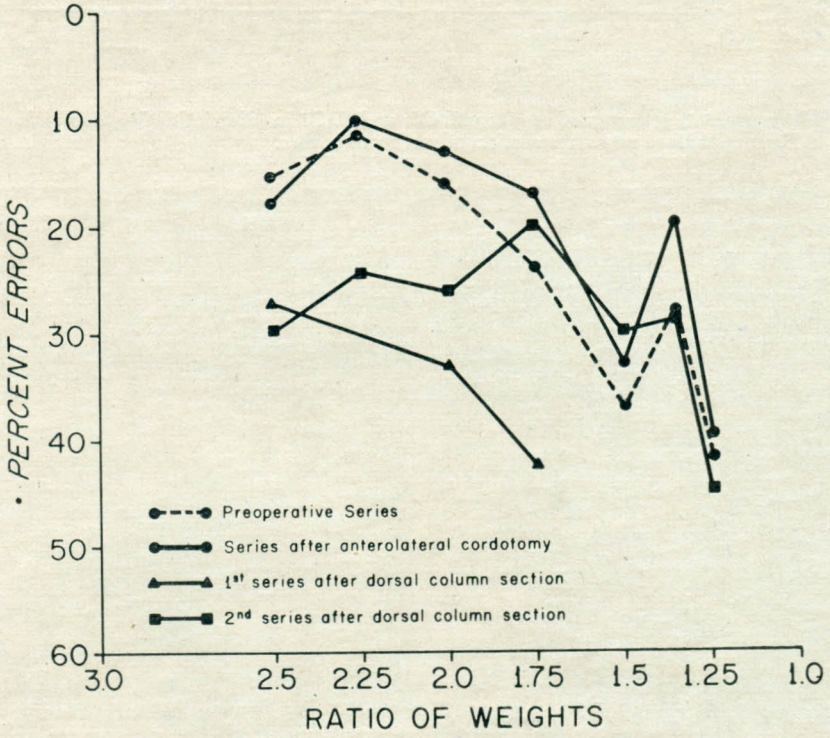

Fig. 5. Percentage errors in weight discrimination by monkey DC6 before and after anterolateral cordotomy and after additional dorsal column lesions.

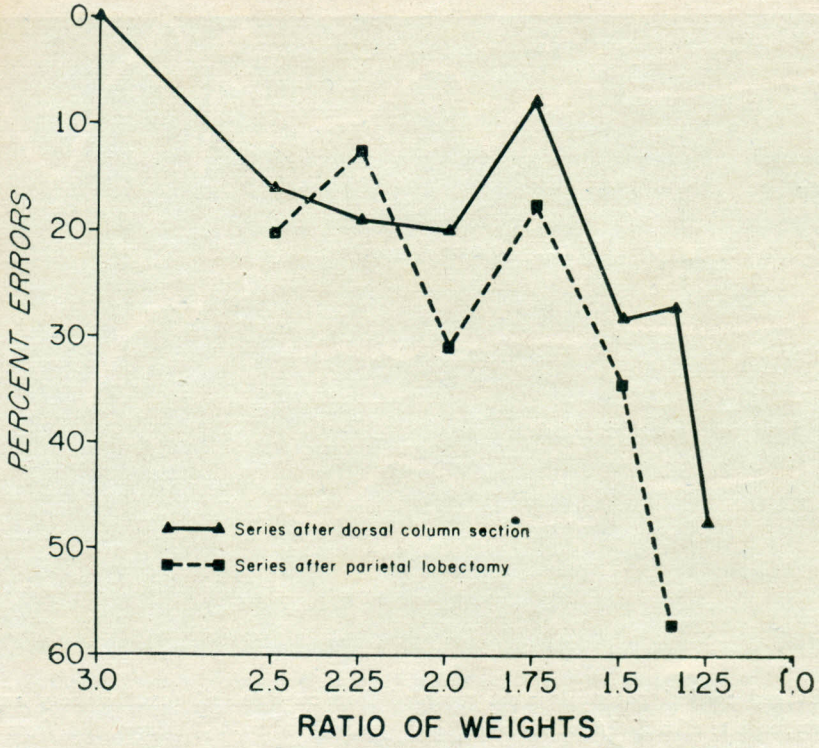

Fig. 6. Weight discrimination curves comparing performance of monkey DC1 after recovery of function from dorsal column lesions with that following parietal lobectomy.

more quickly than the forelimbs, and by the end of one week, the animals were able to move about on all fours with a hopping movement. Monkey DC3, with lesions confined to the dorsal columns, i.e., not extending into the lateral corticospinal tracts, was able to sit up nine hours after the operation and to stand on the third post-operative day. By the end of three weeks, all animals grasped sufficiently well to permit retesting. Obvious dysmetria of the forelimbs was present for about six weeks, and small errors in direction persisted in animals maintained for six months to two years. After two months, no impairment in equilibrium or gross movements was detectable during ordinary cage activity.

The hopping reaction returned during the first or second post-operative month. Contact placing returned later than hopping, and was elicited only occasionally during repeated testings. Proprioceptive placing was often elicited when there was no response to contact. All tests were performed with the animals bindfolded.

Effects of spinal lesions on evoked cortical potentials. After acute section of the dorsal column in cats, the cortical responses were extremely small or absent, but they increased in amplitude over a period of several minutes (fig. 7).

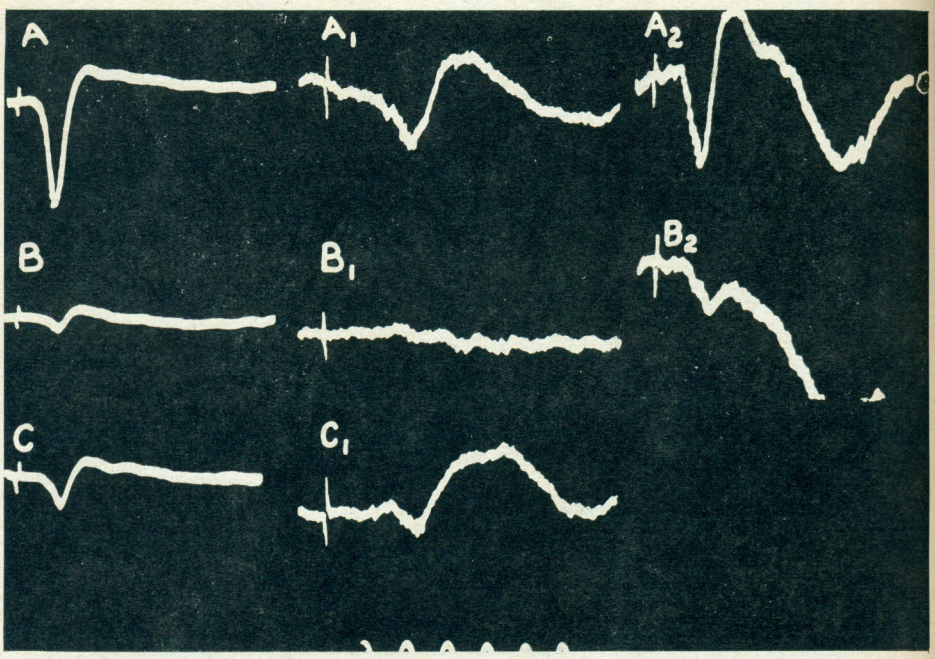

Fig. 7. Evoked cortical potentials in cat recorded from contralateral somatosensory area II in response to stimulation of radial (A) and gastrocnemius (A_1) nerves, and from contralateral somatosensory area I in response to stimulation of popliteal nerve (A_2). Cortical responses shown occurred before dorsal column section (A), immediately after section (B), and 30 minutes later (C). Responses to gastrocnemius and popliteal nerve stimulation were recorded at higher gain than were those to radial nerve stimulation. Time, 60 c.p.s.

Occasionally, periods as long as 30 minutes passed before responses were obtained. Primary potentials were recorded focally in the contralateral precentral and postcentral gyri following stimulation of forelimb nerves in monkeys DC2, DC3, DC5 and DC6, which had chronic dorsal column lesions.

DISCUSSION

Although perception of weight differences may depend upon touch-pressure sensibility as well as upon proprioception, discriminative functions in both sensory spheres are generally assigned to the dorsal columns. In this study, three monkeys with their dorsal columns completely interrupted at the level of C_2–C_3 lost their weight discriminatory ability only temporarily. This ability returned to preoperative levels with retraining. The initial deficit in weight discrimination was less in monkey DC3 than in monkey DC1 and DC2, a difference which correlated with the lesser motor involvement and the confinement of damage to the completely interrupted dorsal columns in this animal.

Ferraro and Barrera (1934) did not observe grasping with the forelimbs for three or four months after dorsal column section, but the earlier recovery in the persent series may have resulted from increased motivation in the testing situation. For example, monkey DC1 made no attempt to use his hand until he was placed in the test cage, whereupon he immediately grasped the string and pulled in the box. The effect of dorsal column section upon the hopping and placing reactions are similar to those following ablation of the postcentral gyri (Bard, 1937-38) in that contact placing is severely impaired while the hopping reaction is only temporarily diminished.

The recovery of discriminatory ability after section of the dorsal columns raises the question of whether such lesions essentially denervate the parietal lobe or whether the impulses necessary to weight discrimination reach this area by other pathways. The decrease in ability after the left parietal lobe and the right sensory area II were ablated indicates that some fibres subserving weight discrimination after dorsal column section terminate in the parietal lobe. However, the residual ability makes evident the participation of extra-parietal areas and/or the ipsilateral cortex. Existence of a fibre pathway to the parietal cortex independent of the dorsal columns was confirmed by electrical stimulation, since stimuli applied to cutaneous and muscle nerves after interruption of the dorsal columns evoked discretely localised potentials in contralateral somatosensory areas. Furthermore, Gardner and his co-workers (1952, 1953) found that hemisection of the cord and section of the opposite dorsal column failed to abolish cortical potentials evoked by stimulation of deep or cutaneous nerves, i. e., impulses reached both cerebral hemispheres if one anterolateral region of the cord remained intact.

Anterolateral cordotomy alone did not affect weight discrimination. After the dorsal columns in this monkey were damaged, the initial deficit in weight discrimination was greater than that in monkeys with dorsal lesions alone, and the animal's recovery of weight discriminatory ability could be explained on the basis of a sparing of fibres in the left cuneate fasciculus. On the right side, the interruption of the dorsal columns was complete, but the descending motor tracts were not damaged. However, the right digits never grasped, and right arm was used only for support. Thus, it appears difficult to determine the role of other spinal pathways in the retention of weight discrimination after dorsal column section since the animal with combined spinal lesions may not have sufficient motor co-ordination to be able to work in the testing situation.

In view of Sjöqvist and Weinstein's (1952) finding that weight discrimination depends upon a cerebellar relay as well as upon the medial lemniscus, the restiform bodies were sectioned in an attempt to interrupt a larger component of the spinocerebellar fibres than that represented by the dorsal columns. The restiform

body lesions alone or in combination with section of the dorsal columns did not affect weight discrimination. However, the lesions were placed too far caudally to interrupt all fibres contributing to the inferior cerebellar peduncle.

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