CORONAL SOMATOSENSORY UNIT RESPONSES IN CATS WITH DORSAL COLUMN LESIONS

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INTRODUCTION

In the preceding study[8], we found that the effects of cervical dorsal column (DC) lesions were revealed by neurological and roughness-discrimination tests if the lesions destroyed at least 90% of the total DC cross-sectional area. Since the dorsal columns form the major spinal input to the coronal somatosensory cortex[12,13,18], unit recordings from this area should reflect the effects of DC lesions. In particular, we wished to determine the changes in cortical neuron response accompanying DC lesions which produce behavioral deficits. Accordingly, we recorded unit activity from the forepaw focus of coronal cortex[12], using most of the animals that had been behaviorally tested in the previous experiment. We found that only the DC lesions producing behavioral deficits (over 90% of the dorsal columns destroyed) significantly reduced the proportion of both responding and short latency (10 msec or less) units, reduced the proportion of units with receptive fields having sharp, well-defined borders, and increased the average area of receptive fields. Such lesions were estimated to involve approximately 90% of the DC fibers from the forepaw contralateral to the recording electrode; ineffective lesions spared a greater proportion of these fibers.

METHODS

Surgical procedure. Twenty-seven adult cats were anesthetized with sodium pentobarbital (35 mg/kg intraperitoneally); intravenous doses (0.1–0.2 ml of 65 mg/ml) were given so as to prevent spontaneous movements while maintaining brisk withdrawal reflexes. Following tracheotomy, the cat was placed in a stereotaxic apparatus. The coronal gyrus of the cerebral cortex was exposed unilaterally, the dura reflected, and a silver ball electrode (1 mm diameter) used to find the site of the largest potential evoked by needle stimulation of the central foot pad in the contralateral forepaw. This coronal site was remarkably constant in location with respect to the cortical landmarks. In two cats with chronic DC lesions, there was little or no evoked potential to contralateral forepaw stimulation, and the site had to be selected on the basis of cortical topography. Acute spinal lesions were made only after the microelectrode

was in the contralateral forepaw focus. During the experiment, sterile 5% dextrose–0.9% sodium chloride solution was administered frequently; rectal temperature (maintained by applications of heat when necessary), pulse, respiration, and expired carbon dioxide were monitored.

A closed chamber for unit recording was constructed directly over the cortical site. A plastic cylinder sealed at the bottom with Silastic rubber (Fig. 1) was held by a micromanipulator on the cortical surface, the dura pulled into place, and the rest of the cortical surface shielded with plastic film. Fast-setting Silastic A RTV mold-making rubber was poured over the protected cortex to fasten the cylinder to the exposed skull. Units were recorded with stainless steel microelectrodes inserted directly through the 20 sq. mm Silastic bottom of the cylinder.

Data acquisition. Extracellular microelectrodes were prepared from stainless steel wire and tested with a Tektronix Type 130 L-C meter by measuring the capacitance in 0.9% saline–0.5% agar at a frequency of 140 kc/sec[2]. Electrodes with a tip capacitance of 0.1–1.0 pF, total capacitance of less than 15 pF at 25 mm, and no sudden capacitance changes along the insulated shank were satisfactory. Progress of the electrode was controlled by a Kopf (Model 1207B) hydraulic micro-drive. The electrode was connected through a cathode follower to a Tektronix Type FM 122 low-level AC preamplifier. Electrical stimuli were delivered from a constant current stimulator (Electronics for Life Sciences CCS-1A) through needle electrodes to each forepaw and various cutaneous receptive fields. An on-line pulse-height discriminator and digital poststimulus histogram unit[15] was connected to a loudspeaker system to assist in identification of the receptive fields. The poststimulus histogram apparatus automatically displayed the responses of a unit for 10 time bins (0.8–40 msec each)

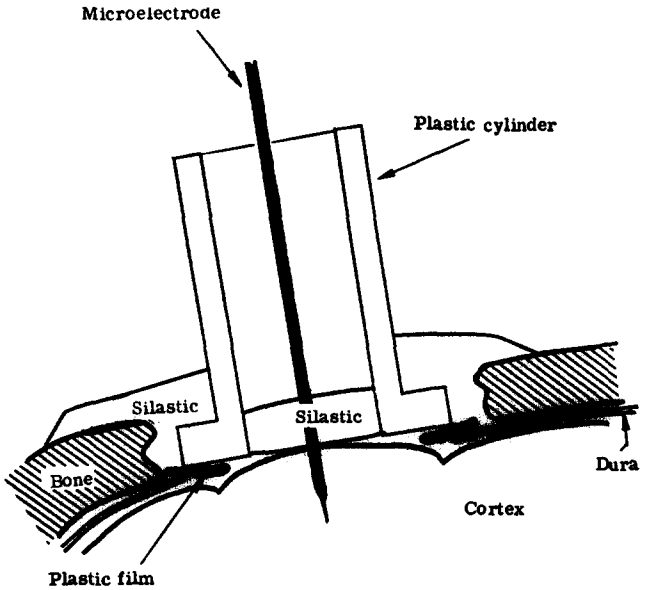

Fig. 1. Cortical chamber (cross-section).

after each stimulus for a total of 10–90 stimuli. Presumably, most unit recordings were from cell bodies since the spikes were usually negative, ranged from 0.1 to 25 mV in amplitude, and did not have the steep rising phase of spikes originating from fibers[10].

The electrode was advanced slowly during intermittent electrical stimulation and general manipulation of the cat's skin and limbs so that units which fired only in response to a stimulus could be identified. The following parameters were recorded for each unit: (1) cortical location, (2) rate and pattern of spontaneous activity, (3) response to natural stimulation, (4) location and size of receptive field, and (5) latency of response to electrical stimulation by needle electrodes in the receptive field. The natural stimuli included light or heavy tap, pressure, hair movement, joint position or movement, claw movement, and noxious pressure on skin or deep structures.

Peripheral receptive fields were initially located by examination of most of the cat's surface and were usually limited to the contralateral forelimb. Each receptive field was drawn on the cat's skin in ink; a light plastic sheet was then pressed against the skin and the field traced onto the plastic and the protocol notes. The area of each receptive field was measured with a planimeter.

Histology. The spinal lesions were reconstructed from alternate sections stained with Nissl cresyl violet or Weil stain. The size of chronic dorsal column lesions was determined by planimetry as previously described[8]. The size of the spinal lesions made during the acute experiments was determined by applying India ink to the in-

TABLE I

LESION SIZES AS PERCENTAGES OF THE AREAS INDICATED AT THE C_1 LEVEL

Cat	*Total bilateral DC*	*Contralateral DC*	*Contralateral CFP area*
Acute DC lesions			
35	39	40	39
40	28	42	38
41	64	62	61
43	69	77	75
44	75	79	72
45	70	78	87
46	71	71	68
Chronic DC lesions			
L38	38	26	28
L57	57	56	59
R69	69	83	96
L76	76	87	81
R82	82	77	76
L86	86	86	80
R86	86	84	78
L97	97	93	89
L100	100	100	100
R100	100	100	100
Lz100	100	100	100

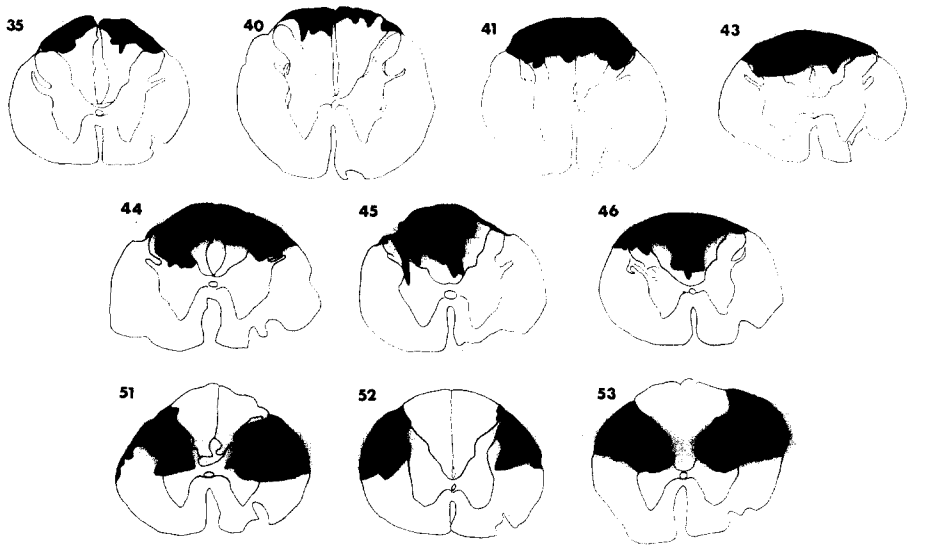

Fig. 2. Composite of histological reconstructions of all acute lesions in this study. Cats 35, 40, 41 and 43–46 received dorsal column lesions; cats 51–53 received dorsolateral lesions.

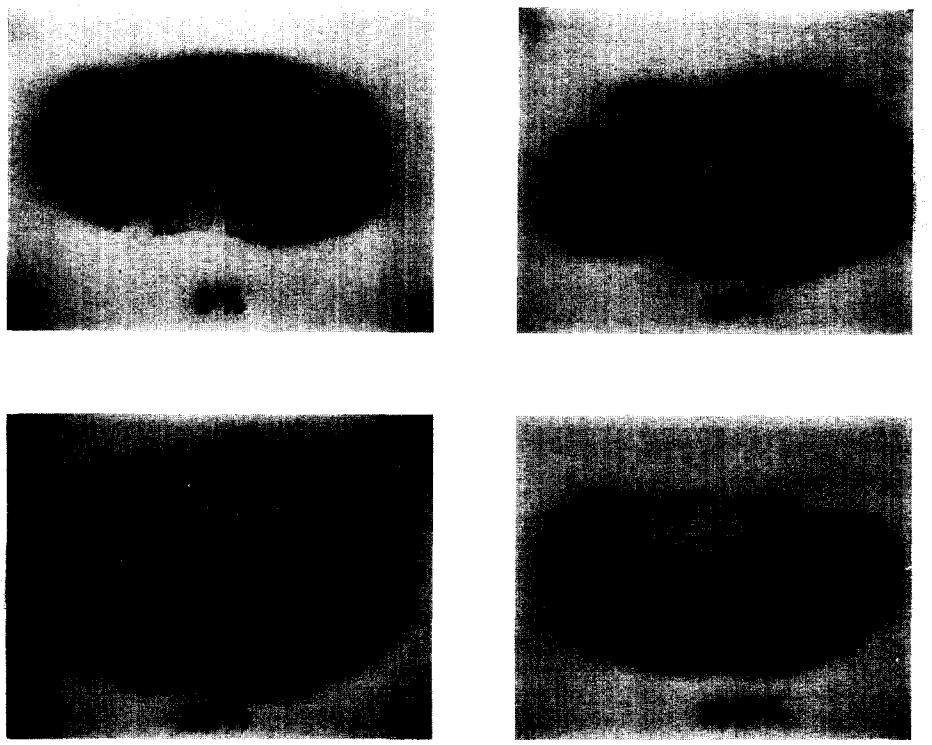

Fig. 3. Representative Weil-stained cervical sections from cats having chronic surgery, including one cat with a sham operation.

cision site for 10–30 sec, allowing the ink to distribute along all cut surfaces. The reliability of this technique was verified by the fact that in cats with chronic DC lesions, the ink never appeared below the surface of the area of degeneration.

In the text and data analysis, lesion size is expressed as the percentage of the total bilateral area which had degenerated at C_1. As discussed in the behavioral study[8], however, the critical DC fibers are those carrying information from the paws. At the C_1 level, these lie in the medial 65% of the DC as estimated by planimetric measurements, using the data obtained by Hekmatpanah[9], Liu[11], and Weaver and Walker[16]. Similar measurements were used to estimate that the lateral 37% of this medial area (or 24% of the total DC cross-section at C_1) is occupied by forepaw fibers. Lesion size is therefore also expressed in Table I as a percentage of both the contralateral dorsal column and the wedge-shaped area estimated to contain fibers from the forepaw contralateral to the recording electrode.

RESULTS

Units were recorded from 4 groups of cats: (1) 15 intact cats, (2) 7 with acute lesions of the dorsal columns, (3) 11 with chronic lesions of the dorsal columns, and

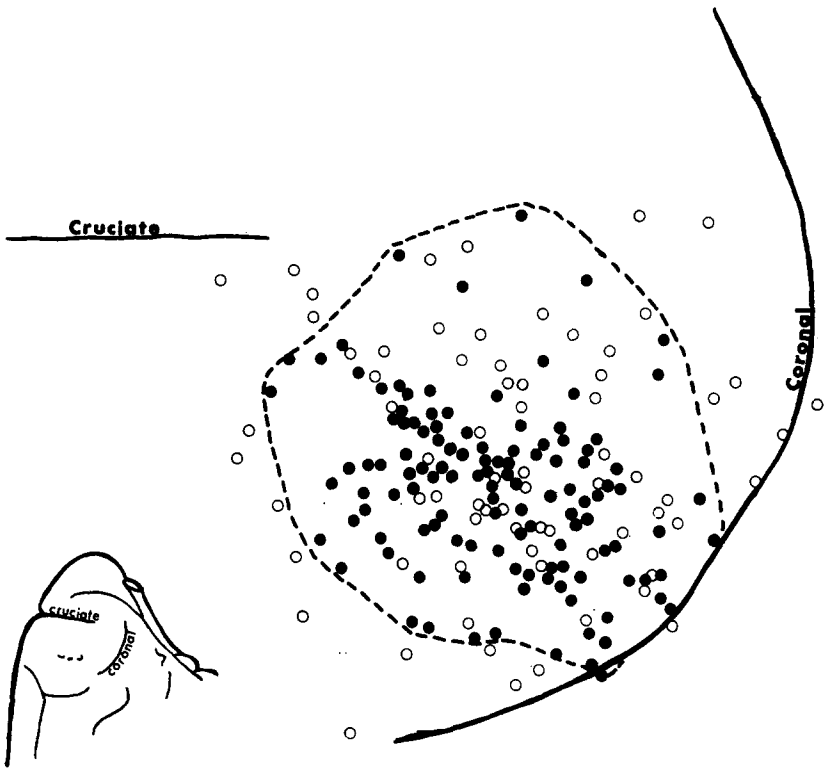

Fig. 4. Composite of all electrode track positions on the coronal gyrus. Dots represent tracks from which electrical activity was evoked by contralateral forepaw stimulation; circles, tracks without contralateral forepaw response.

(4) 4 with bilateral lesions of the dorsolateral (DL) columns. Acute lesions were made in some of the cats from the first group. All chronic lesions were made 45 days to 2 years before the acute experiments. Eight of the cats with chronic DC lesions had been tested in a roughness-discrimination task as previously described[8].

Spinal lesions made during acute experiments are shown in Fig. 2. Fig. 3 shows representative sections from cats with chronic DC lesions; the more detailed histology of these animals has been presented previously[8].

Identification of contralateral forepaw focus

Fig. 4 shows the cortical region of microelectrode exploration in all cats. The enclosed area includes all tracks in which stimulation of the contralateral forepaw elicited unit or slow potential responses. Units recorded from tracks outside this area are eliminated from this study; this includes 15 units from intact cats and 21 from animals with spinal lesions.

Evoked potentials

In 15 normal cats the positive coronal evoked potential had a latency of 8–9 msec and reached an average peak amplitude of 100 μV at 14–15 msec. The average evoked-potential amplitude of cats with chronic DC lesions was significantly decreased to about one-third that of intact animals ($P < 0.001$, Mann–Whitney U test). However, there was no relation between evoked-potential amplitude and the size of a chronic or acute DC lesion. In two cats the coronal evoked potentials were comparable in size before and after acute section of the dorsolateral pathway. In the only cat with a chronic bilateral dorsolateral lesion, contralateral forepaw stimulation evoked a large coronal potential.

Unit analysis

Responses to natural somatic stimuli. Of 506 units, 40 responded to brushing of hair, 206 to tapping, 37 to delicate claw movements, and 12 to more than one of these stimuli; 163 units did not respond to intentional natural or electrical stimulation. Six units responded to stimuli identified as joint flexion, deep pressure, or noxious stimulation of the skin. Of the 105 units studied in normal cats, 96 (91%) were active in the absence of stimulation and 70 (67%) responded to somatic stimuli: 10% to hair, 46% to tapping, and 1% to more than one type of stimulus (Table II). No claw units were found in intact cats.

One of the more striking findings among the animals with spinal lesions was that only 33% of the units in cats with chronic lesions involving over 90% of the total DC cross-sectional area responded to peripheral stimuli; this is a significant decrease from the 67% of responsive units in intact cats ($\chi^2 = 22.9$, $P < 0.001$). None of the other experimental groups differed significantly from the intact group in this respect except that 95% of the units in acute DC cats responded to somatic

TABLE II

SUMMARY OF UNIT DATA

Numbers in parentheses indicate percentages.

	Intact	*All chronic DC*	*Chronic DC 95%*	*Acute DC*	*Acute or chronic DL*
No. of cats	15	11	4	7	4
No. of units (total = 506)	105	301	97	42	58
No. spontaneously active (97% of all units)	96 (91)	223 (74)	90 (93)	34 (81)	45 (78)
No. which respond to peripheral stimuli (68% of all units)	70 (67)	190 (63)	32 (33)	40 (95)	43 (74)
Modality: No. of units examined, responding to:					
Hair only	11 (10)	17 (6)	7 (7)	7 (17)	5 (9)
Tap only	48 (46)	113 (38)	11 (11)	27 (64)	18 (31)
Claw* only	0 (0)	23 (8)	3 (3)	0 (0)	14 (24)
More than one stimulus	1 (1)	5 (2)	1 (1)	4 (10)	2 (3)
Other	10 (10)	32 (11)	10 (10)	2 (5)	4 (7)
No. with measured latency	37 (35)	98 (33)	15 (15)	25 (60)	37 (64)
No. with measured receptive field	44 (42)	92 (31)	8 (8)	29 (69)	19 (33)
No. with prolonged depression of post-response excitability**	2 (2)	29 (10)	10 (10)	0 (0)	0 (0)

* Claw units respond to minute, brisk movements of the claw but not to tapping or hair stimulation around the base of the claw. They adapt rapidly, are securely driven with a brief latency (median and mode = 13 msec).

** This term is defined in the text.

stimuli ($\chi^2 = 13.0$, $P < 0.001$); their DC lesions, however, were all less than 80%. In one cat (R69), in which 55% of 22 units responded, the chronic lesion was estimated to involve 96% of the contralateral forepaw DC fibers (Table I).

Spatial properties. Receptive fields were spots or irregular, sometimes stocking-like, areas confined to the contralateral forelimb. Fig. 5 illustrates the mean values for receptive field sizes for all units in each group of cats. The mean and median size of receptive fields in cats with chronic DC lesions greater than 90% is larger than in intact cats or in animals with less extensive spinal lesions (for the mean, $t = 5.37$, $P < 0.001$). Fig. 6 shows the frequency distribution of receptive field size for each group of cats. As a group, chronic DC lesions did not affect the relative incidence of sizes, but the cats with acute DC, acute dorsolateral and chronic dorsolateral lesions had smaller average receptive field areas and proportionately more receptive fields of the smallest size, 5 sq. cm or less, than intact cats. The differences in both mean and distribution are statistically significant (for the means, $P < 0.02$ by t-tests; for distribution, $P < 0.005$ by χ^2-tests).

Nine units (6 hair, 2 tap, and one not responding to stimuli) in intact cats were studied before and after cutting the dorsal columns bilaterally. The lesions destroyed 28–75% of the DC cross-sectional area (also, see Table I). Immediately

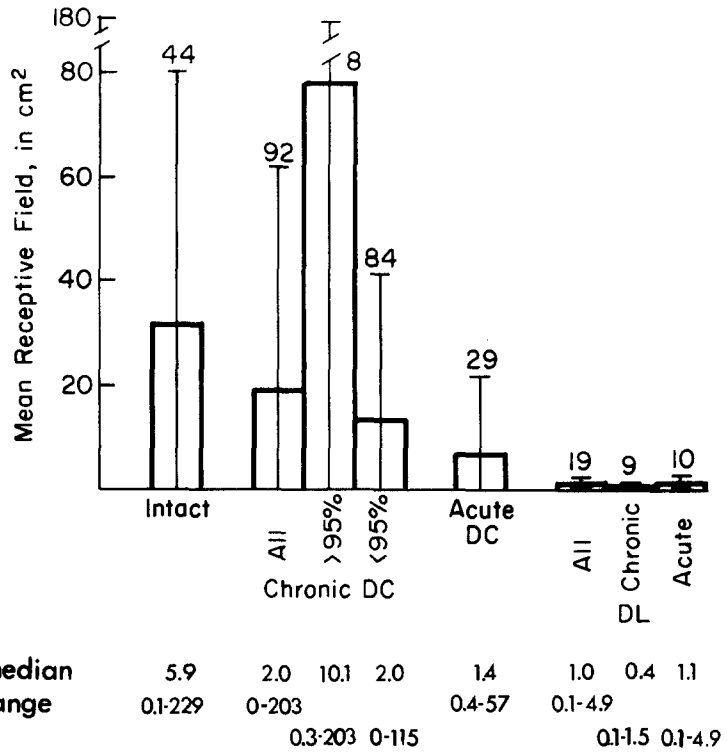

Fig. 5. The average of all measured receptive field sizes for intact cats and cats with dorsal column or dorsolateral lesions. The standard deviation and sample size (number of units) are shown on the graph; the range and median are shown at the bottom.

after the cutting, 3 units were less responsive to peripheral stimuli, but their excitability, as determined by poststimulus histograms, was restored to control levels within the hour. Six receptive fields remained exactly the same size and two increased in area 12–25%. On the average, their discharge in the absence of overt stimuli was unchanged.

In the intact animals, 42% of all units (63% of the units responding to somatic stimuli) had receptive fields with borders which could be sharply demarcated by moving the test probe a millimeter or two on either side of the border. The remaining receptive fields had borders which could be located only by test probe movements of at least 1 or 2 cm. In the animals with large chronic DC lesions (greater than 90%; see Table I), only 8% of all units (25% of those responding to somatic stimuli) had well-defined receptive fields. Thus, not only did the largest DC lesions increase the average receptive field size and decrease the proportion of cells which respond to somatic stimuli, but more of the cells which respond do so in a way which precludes drawing sharp boundaries around their receptive fields.

Eight units (7 tap, one unidentified) had 'labile' receptive fields. In this case the receptive field was clearly defined in at least two very different configurations, between which the field alternated over a period of minutes or hours. Four of the

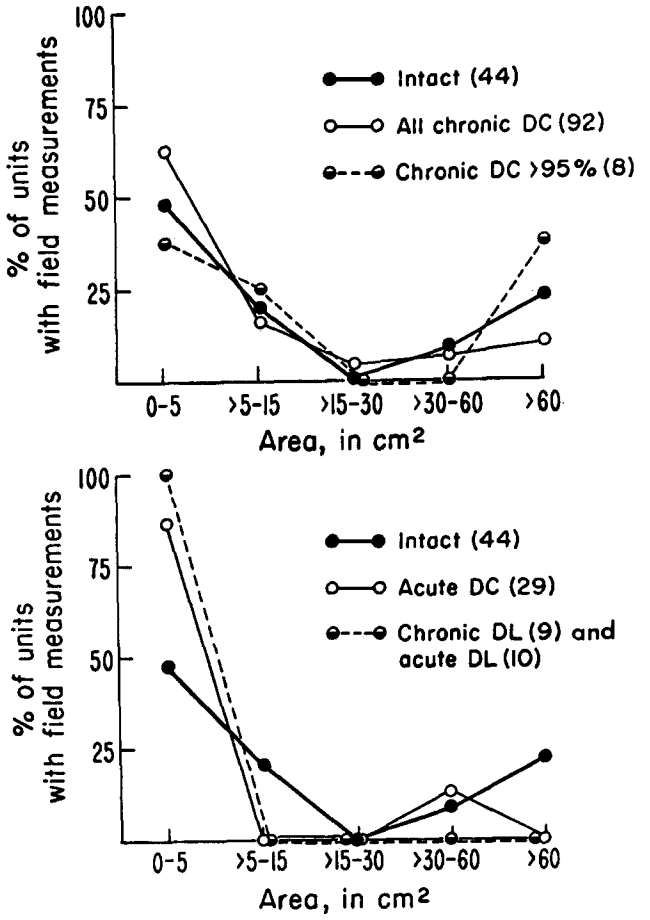

Fig. 6. The classification of receptive field sizes in intact cats and cats with dorsal column or dorsolateral lesions. The number in parentheses is the number of units in each group for which receptive field sizes were measured, and this number is represented as 100% on the ordinate.

receptive fields were small in both configurations, but 3 of them at some time covered the entire contralateral foreleg from the elbow although they were limited to one or two toes at another time (as much as a 300-fold change in size). Latencies for 6 such units ranged from 11 to 50 msec. These units were located throughout the contralateral forepaw focus, 0.4–2.6 mm deep (mean and median depth, 1.7 mm). Two of these units were recorded from intact cats, 4 from cats with chronic DC lesions less than 90%, one from a cat with chronic DC lesion greater than 90%, and one from a cat with an acute dorsolateral lesion.

Occasionally, it was possible to document the changes in the shape and position of receptive fields during prolonged study of a single unit. Fig. 7 shows the 4 tracings of the boundaries of one unit over a 3 h period.

Temporal properties. Fig. 8 illustrates the mean value of latencies for all units in each group of cats. There is no significant difference among the various groups

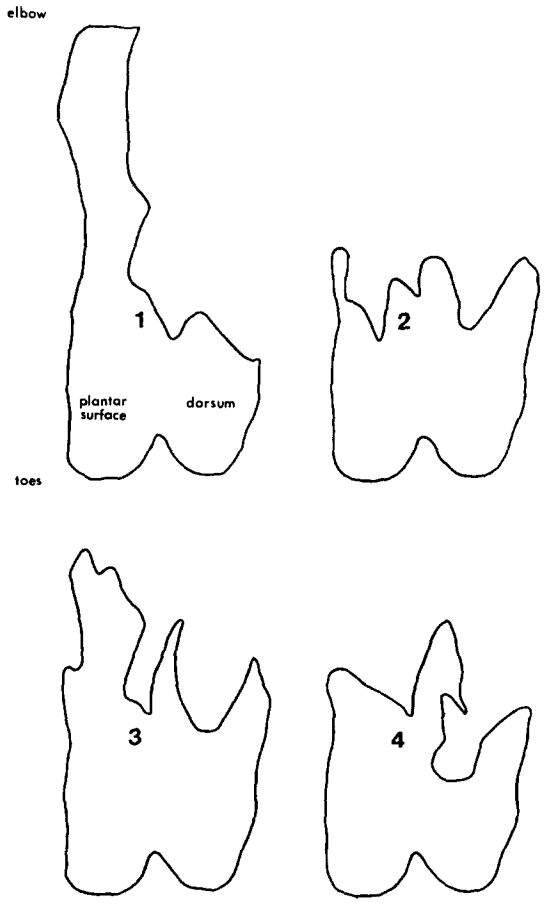

Fig. 7. Receptive field of a tap unit in an intact cat, drawn 4 times in a period of 3 h, to show the shifting boundaries. Boundaries were determined as the outermost points where there are at least 4 responses for each 5 taps. Field sizes are: area 1, 67.6 sq. cm; area 2, 52.2 sq. cm; area 3, 68.4 sq. cm; area 4, 52.0 sq. cm.

(*F*-test). However, as shown in Fig. 9, spinal lesions do affect the frequency distribution of latencies. For example, cats with chronic DC lesions have fewer short latency (10 msec or less) units than intact controls ($\chi^2 = 4.6$, $P < 0.05$). This difference, however, is attributable to the effect of the larger lesions (greater than 90%), for no short latency units were found among the 15 studied in these animals ($\chi^2 = 3.8$, $P = 0.05$). Cats with smaller chronic DC lesions do not have a significantly different proportion of short latency units than intact animals ($\chi^2 = 3.2$, $P > 0.05$). The proportion of units with latencies over 20 msec was not changed by chronic DC lesions regardless of size.

Acute DC and both acute and chronic dorsolateral lesions had a different effect on the frequency distribution of latencies. Compared to intact animals, there were more units with latencies of 20 msec or less in cats with acute DC lesions

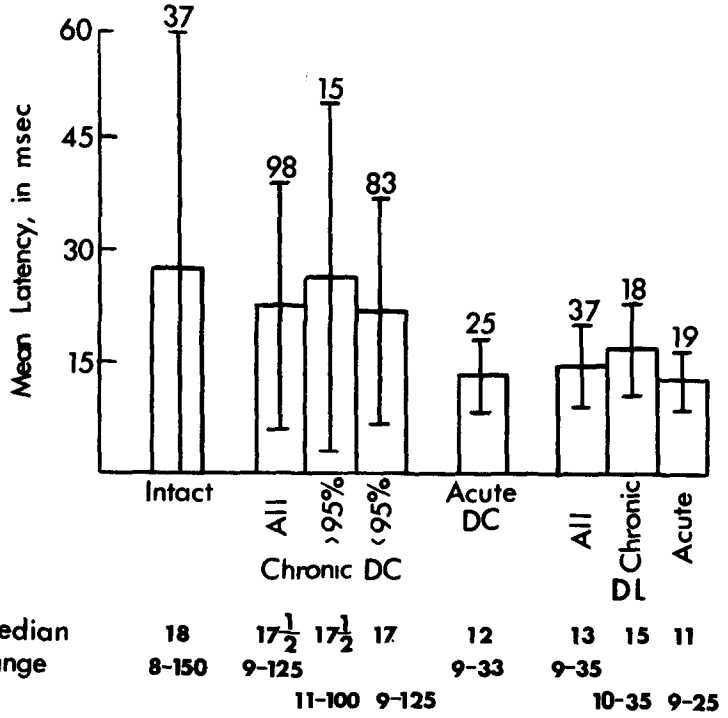

Fig. 8. The average of all measured latencies for intact cats and cats with dorsal column or dorsolateral lesions. The standard deviation and sample sizes (number of units) are shown in the graph; the range and median are shown at the bottom.

($\chi^2 = 10.1$, $P < 0.005$), cats with acute dorsolateral lesions ($\chi^2 = 9.5$, $P < 0.005$), and the cat with a chronic dorsolateral lesion ($\chi^2 = 4.5$, $P < 0.05$).

There is a highly significant relationship between latency and receptive field size ($\chi^2 = 15.1$, $P < 0.001$). Only 6 of the 79 units with latencies less than 20 msec had receptive fields larger than 30 sq. cm, while 15 of the 42 units with latencies 20 msec or longer had receptive fields larger than 30 sq. cm. This relationship is reflected in the finding of a relatively high proportion of both small receptive fields and latencies below 20 msec in cats with dorsolateral and acute DC lesions. However, the average receptive field size of the total group of chronic DC animals was not increased even though the cats with large lesions had a larger receptive field area and a smaller proportion of short-latency units. Latencies were recorded for 5 units before and after the dorsal columns were cut. Latencies of 3 units increased by 10–25% (original latency, 8–10 msec), while two remained the same.

Prolonged depression of post-response excitability. Most units respond with a burst or single spike to each electrical stimulus delivered at 1-sec intervals; response probability, as determined with the poststimulus histogram, does not increase as the stimulus frequency is decreased to 1/10 sec. However, 31 units consistently responded with increasing poststimulus probabilities as stimulus frequency was decreased below 1/sec. Fig. 10, for example, shows the poststimulus histogram of a unit responding

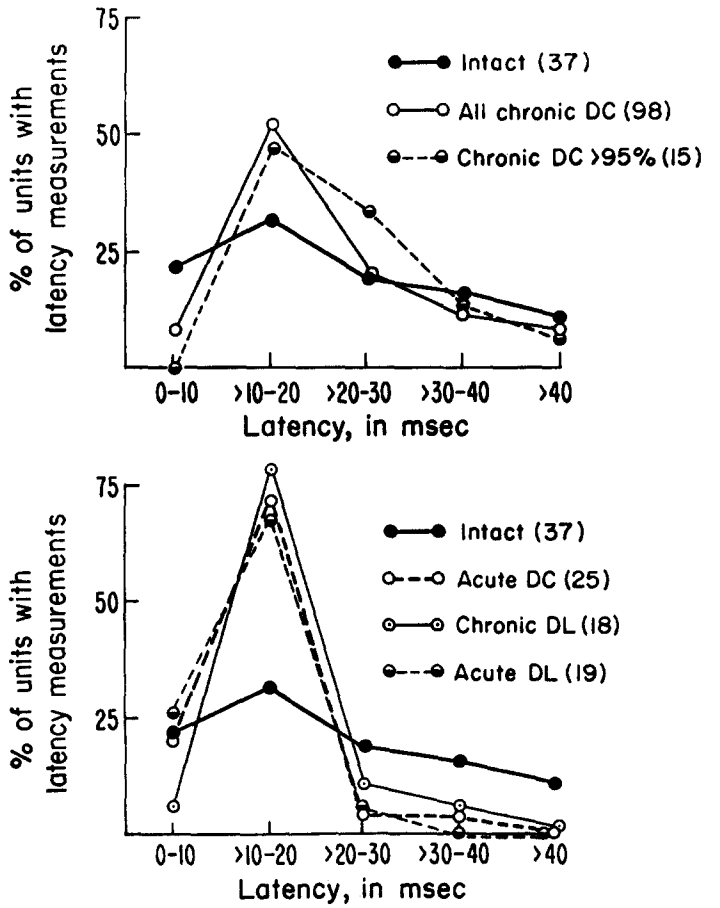

Fig. 9. The distribution of latencies in intact cats and cats with dorsal column or dorsolateral lesions. The number in parentheses is the number of units in each group for which latencies were measured, and this number is represented as 100% on the ordinate.

with only one-third as many spikes at a stimulation rate of 1/sec as it does for 1/4 sec. This prolonged depression of post-response excitability was found almost exclusively in cats with chronic DC lesions (29 in chronic DC, 2 in intact; $\chi^2 = 7.5$, $P < 0.01$).

Units showing this depressed post-response excitability are similar to the other units with respect to stimulus modality and cortical location (1.2–3.2 mm deep throughout the contralateral forepaw focus; median depth, 2 mm). However, these units do have large receptive fields (median, 10 sq. cm; range, 0.9–200 sq. cm) and long latencies (median, 35 msec; range, 14–150 msec). Three of the 31 units with post-response depression were among the 8 units with labile receptive fields, a significant correlation between the two properties ($\chi^2 = 5.0$, $P = 0.025$).

It is unlikely that post-response depression is due to anesthesia or other factors related to the length of the experiment. Of the 301 units recorded in chronic DC cats, 165 were recorded within 24 h after the initial dose of anesthesia and 20 (12%) of

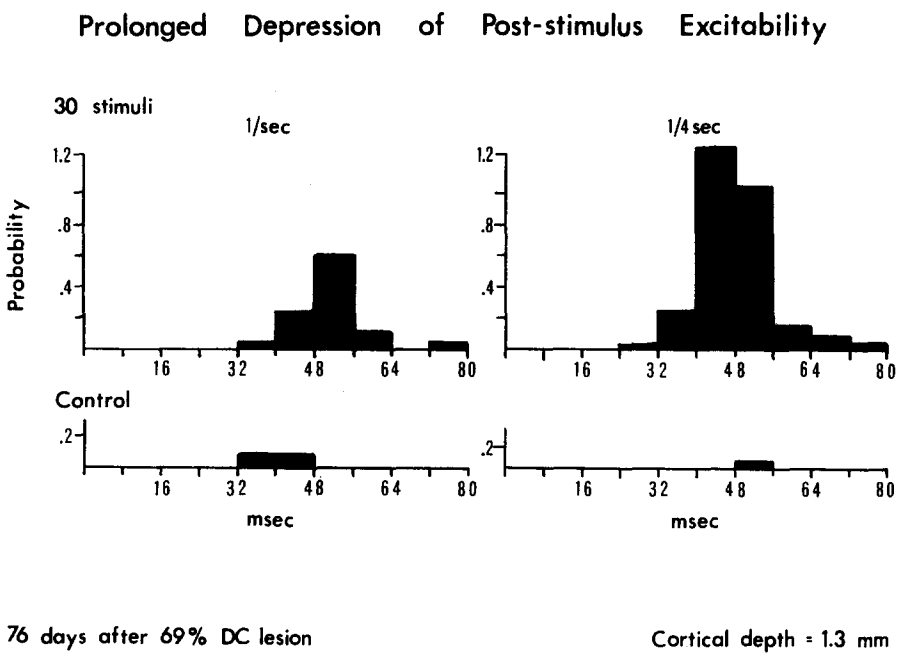

Fig. 10. Comparison of response properties of unit 24, cat R69, to two rates of electrical stimulation through skin needle electrodes on the contralateral forepaw (2.5 mA, 0.2 msec pulses). As stimulation frequency decreased to 1/4 sec, the unit responded to all of the stimuli and also increased the number of spikes per response. Control records show the occasional spontaneous activity in the absence of electrical stimulation.

these showed post-response depression whereas 9 (7%) of the 136 units recorded after the first 24 h showed this characteristic. This difference is not significant ($\chi^2 = 2.6$, $P < 0.10$). Proximity of the recording electrode to the cell, as indicated by unit amplitude and waveform, is known to affect unit excitability[14]. This factor is probably not related to post-response depression since only 4 of these 31 units were electrically positive, and none were larger than 1 mV.

The incidence of post-response depression is not related to the amount of chronic DC destruction. Four cats with 97–100% DC destruction had the same proportion of such cells (10%) as did 7 cats with 38%–86% destruction. Table III shows, however, that there is a distinct relationship between the incidence of cortical units with post-response depression and the length of time since previous dorsal column surgery. Note that the average postoperative period for all the DC cats having units with prolonged depression was 72 days, while DC cats without such units averaged 616 days since their most recent surgery. The times involved are clearly too long for the results to be attributed to acute surgical trauma. It is more likely that post-response depression reflects a slowly subsiding change in the functional integrity of input pathways.

Cortical organization. With the exception that claw units were not found in intact animals, spinal lesions did not affect the distribution of cortical units with

TABLE III

LIST OF ALL CATS WITH CHRONIC DC LESIONS, SHOWING RELATIONSHIP BETWEEN INCIDENCE OF UNITS WITH PROLONGED DEPRESSION OF POST-RESPONSE EXCITABILITY AND THE LENGTH OF TIME SINCE SURGERY.

Cat	*Total no. of units*	*% of these units having prolonged depression*	*Time between the most recent exposure of DC and the acute unit recording* (days)*	*Mean (days)*
L57	17	0	535	
L76	21	0	644	
L86	48	0	566	616
L97	35	0	723	
L100	31	0	613	
R69	22	4	78	
L38	31	6	42	
R82	18	11	46	72
Lz100	22	27	51	
R86	47	32	97	
R100	9	44	119	

* In each case except for L38 this indicates the time since the DC were cut. For L38 it is the time since a sham operation; the DC lesion had been made 699 days before the acute unit recording.

respect to their stimulus modality or cortical depth (Fig. 11). Hair and claw units are distributed evenly at depths of 0.5–3 mm, but are almost never found in the first 0.5 mm. Tap units, however, are found at all depths, and most frequently at 1.5 mm. Although claw units were mentioned by Brooks *et al.*[6], we did not search systemically for them until 151 units had been studied (including 69 of the 105 in intact cats); this fact may account in part for the distribution of this modality group.

DISCUSSION

Of the spinal lesions produced, only dorsal column lesions destroying over 90% of the total DC cross-sectional area were effective in (1) reducing the proportion of all responding units, short latency (10 msec or less) responses, and units with sharp, well-defined field borders; and (2) increasing the average area of receptive fields. Such lesions were estimated to involve over 89% of the DC fibers from the contralateral forearm (Table I). This finding is especially significant in view of the fact that only DC lesions of this size produced behavioral deficits revealed by neurological examination and roughness-discrimination testing[8]. Three of the 4 cats with extensive (greater than 90%) DC lesions in this study were the same animals showing deficits in roughness discrimination[8]; the 4th was not behaviorally tested.

Only 33% of the coronal units recorded from these 4 animals responded to somatic stimuli while 67% of the units were responsive in intact cats. In cats with chronic DC lesions of 38–86%, 77% of the units responded to peripheral stimuli

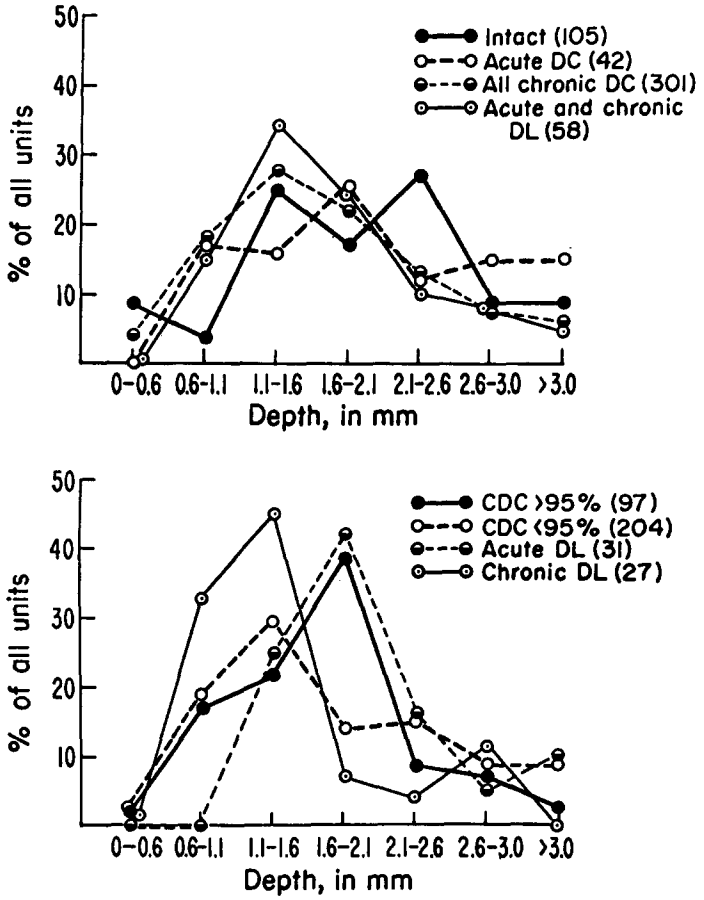

Fig. 11. The distribution of units by depth in intact cats and cats with dorsal column or dorsolateral lesions. The number in parentheses is the total number of units in each group, and this number is represented as 100% on the ordinate.

in spite of the presumed removal of much of their input. The one animal (R69) with a 69% total DC lesions had an estimated 96% destruction of contralateral forepaw DC fibers (Table I); the 22 units recorded were in all respects similar to those recorded from cats with smaller lesions except that only 12 (55%) responded to stimuli. This recalls Amassian's[1] finding that the primary evoked potential in somatosensory cortex is decreased very little by major decrements in afferent input. Whitehorn *et al.*[18] also found that it was necessary to produce deep lesions of the dorsal columns in order to substantially decrease cortical evoked potential amplitude. We found a significant reduction in evoked potential amplitude in cats with chronic DC lesions, but there was no relation between the size of the evoked potential and the DC lesion. In agreement with Whitehorn *et al.*[18], however, neither the less extensive acute DC lesions nor the acute or chronic dorsolateral lesions significantly affected evoked potential amplitude. The evoked potential, unit, and behavioral[8] studies, then, are

similar in showing that some measures of somatosensory function can be maintained by a fraction of the DC fibers.

The receptive fields of coronal neurons were usually areas of less than 5 sq. cm on the contralateral forepaw, but a wide range of field sizes was encountered (Fig. 5). Nonetheless, the receptive fields of units recorded from cats with extensive DC lesions were significantly larger than those encountered in other cats, a finding consistent with destruction of a spatially discrete, topographically organized system.

It was not possible to define sharply the receptive field borders of most coronal units recorded from the cats with extensive (greater than 90%) DC lesions. This was not so for intact animals or those with smaller lesions for, even though units with changing receptive field borders were very occasionally encountered in intact cats (Fig. 7), the border could be clearly outlined by small probe movements (approximately 2 mm) at each field configuration. The relatively poorly defined receptive field borders encountered in the cat with extensive DC lesions, however, could only be identified as approximately 2 cm bands surrounding the excitatory area. This may be due to small, rapid shifts in well-defined borders or a decrease in effective input as the stimulus is moved away from the excitatory center of the field.

Labile receptive field boundaries have been reported for neurons in the peri-cruciate cortex[7], but not in coronal cortex. However, this is a rare finding in coronal cortex since only 8 of 605 units had labile receptive fields; Brooks and Levitt[5] classified 5 of 160 sigmoid gyrus units as labile.

The cats with extensive chronic DC lesions also have a smaller proportion of units with short (10 msec or less) latencies as compared to intact cats or cats with smaller lesions. This finding is in keeping with the observation[13] that the short latency projection to the coronal somatosensory area is via the dorsal column–lemniscal system.

Morse *et al.*[12] reported that more than 90% of their coronal units, recorded from cats anesthetized with chloralose, followed peripheral stimulation at the rate of 2/sec and all units followed 1/sec. An appreciable number of units in this study (31 of 506) did not follow stimuli faithfully at 1/sec. Since only two of these were recorded from intact cats (2% of 105 units), it is possible that they could have been absent from the sample of 179 coronal units of Morse *et al.*, or classified as 'mute' since consistent response to natural stimulation would not have been observed. The proportion of such units cannot be related to the size of the DC lesion or to anesthesia, and the duration of post-response depression far exceeds that attributed to the activation of recurrent collaterals of cortical cells[3,4,17]. All chronic DC cats within 4 months of the most recent cervical cord surgery had units with prolonged post-response depression, but none of the units recorded from the chronic DC cats at least 1.4 years after surgery showed this property. Thus, prolonged post-excitatory depression appears to be a long-term but temporary state related to surgical exposure of the dorsal columns. The basis for this phenomenon cannot be determined from the information currently available.

An unexpected finding was that, compared to intact cats, units in cats with dorsolateral or acute DC lesions had smaller average receptive fields and a higher

proportion of small receptive fields and latencies below 20 msec. However, none of the 8 receptive fields studied before and after acute DC lesions were decreased in area. The curve for acute DC cats in Fig. 6 shows an absence of units with receptive fields greater than 60 sq. cm, suggesting that the lesions instead selectively decreased the proportion of units with large receptive fields. Similarly, it is possible that spinal lesions could increase the excitability of some cortical cells and thus shorten their latencies. However, none of the 5 units for which latencies were compared before and after an acute DC lesion acquired a shorter latency. Since units with long latencies are also likely to have large (greater than 30 sq. cm) receptive fields, these lesions may have selectively decreased the excitability of one class of cells; namely, those with large receptive fields and longer latency responses. The responses of such cells may be profoundly affected by any reduction in excitatory input such as might be produced by these less extensive lesions. The effect of the larger DC lesions might mask these more subtle changes.

The major findings, then, pertain to the cats with over 90% of the cervical DC cross-sectional area destroyed. The loss of behaviorally determined discriminative capacity in cats with DC lesions of this size[8] is presumably a reflection of the changes in cortical unit response observed in these animals.

SUMMARY

(1) Extensive chronic dorsal column (DC) lesions (greater than 90% of cross-sectional DC area) decrease the proportion of coronal somatosensory cells which respond to somatic stimuli, reduce the proportion of cells with sharp receptive field borders, and increase the average receptive field size. Such lesions were estimated to involve approximately 90% of the DC fibers from the forepaw contralateral to the recording electrode.

(2) Cats with extensive chronic DC lesions have proportionately fewer units with short latencies (10 msec or less) than intact cats.

(3) Cats with chronic DC lesions showed a higher proportion of units with prolonged depression of post-response excitability; this property was related, not to the extent of the lesion, but to the length of time since surgery.

(4) A higher proportion of units with latencies of 20 msec or less and small receptive fields (0–5 sq. cm) are found in cats with acute DC, acute dorsolateral, or chronic dorsolateral lesions.

(5) The results show that, as in behavioral studies, functional deficits can be detected following nearly complete DC lesions.

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