The Journal of Neuroscience, March 1988, 8(3): 833–859

Recovery of Tactile Function After Damage to Primary or Secondary Somatic Sensory Cortex in Infant *Macaca mulatta*

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These studies were designed to determine the basis for recovery of tactile function after the removal of primary (SI) or secondary (SII) cortex in infant *Macaca mulatta*. From previous studies we know that although removal of SI or SII in the adult macaque produces severe and irreversible impairment on a variety of tactile tasks, normal function can be obtained after partial or total SI lesions in the infant. From the present studies we have found that, as with SI, neither unilateral nor bilateral removals of SII in infants significantly affected the acquisition of or the performance on size tasks, but did cause a temporary delay in acquisition of texture tasks. Performance on texture threshold tasks was normal. The removal of the remaining SI in a juvenile animal that had received a unilateral SI lesion in infancy did not disrupt the recovered function, indicating that recovery is not mediated by the intact SI. However, when SI and SII were removed together from the same hemisphere in an infant, either sequentially or simultaneously, major impairment in the acquisition of texture tasks followed. These results suggest that although SI and SII are necessary for normal tactile function in the adult macaque, they show an equipotentiality for mediating normal tactile function after damage to either area in infants.

Localization of tactile function within the human neocortex resulted from studies of sensory deficits following damage to parietal cortex in patients (Critchley, 1969). Early behavioral studies of the effects of experimental brain lesions in nonhuman primates reported patterns of tactile deficits following removal of the primary somatic sensory projection area (SI) in the postcentral gyrus and posterior parietal areas similar to those described in clinic populations (Ruch and Fulton, 1935; Ruch et al., 1938). Subsequent studies in macaque monkeys with more restricted lesions to the separate cytoarchitectonic areas (3b, 1, and 2) of SI showed that distinguishable deficits were associated with each subdivision. Removal of area 3b severely affected all types of tactile discrimination, whereas lesions of area 1 affected only texture discrimination tasks, and of area 2, only size or shape tasks (Randolph and Semmes, 1974; Carlson, 1981). These behavioral findings were consistent with known differences between the cytoarchitecture, topography, and neuronal response characteristics of these areas (Powell and Mountcastle, 1959a, b; Paul et al., 1972).

Somatic sensory inputs are also represented in posterior parietal cortex, especially area 5 and parts of area 7b, and along the parietal operculum in the second somatic sensory area (SII). Recent studies of the functional extent of SII in *Macaca* (Friedman et al., 1980; Robinson and Burton, 1980a–c; Juliano et al., 1983) have provided better localization of this area for lesion-behavior studies (see Fig. 1). Contrary to earlier findings of no additional impairment after SI–SII removals (to that seen following SI lesions alone) in adult primates (Kruger and Porter, 1958; Orbach and Chow, 1959), lesions confined to SII (as defined physiologically) caused severe deficits on a variety of form and texture tasks (Ridley and Ettlinger, 1976, 1978; Murray and Mishkin, 1984). In contrast, lesions in the posterior parietal cortex, which do not interfere with the posterior segments of SI (i.e., area 2), have not caused tactile impairment on the types of tasks that are impaired after SI or SII lesions (Ruch and Fulton, 1935; Murray and Mishkin, 1984).

Though behavioral studies point to the critical significance of areas SI and SII for size and texture discriminations, the patterns of deficits do not suggest any specialization of function for these 2 major areas, as did selective subtotal lesions of SI cytoarchitectural areas. Yet physiological studies have shown that cells in these 2 major areas differ in the properties of receptive-field size, laterality of input, and submodality distribution, so that different behavioral functions might be expected (Whitsel et al., 1969; Robinson and Burton, 1980c; Ferrington and Rowe, 1981). The results of ablation studies mentioned above suggest that all 3 SI architectonic areas and SII must be intact in the adult macaques for normal tactile function. The multiple projections of tactile input within the postcentral gyrus and to SII might suggest, however, that any single area has the potential to mediate normal function. We expected that some selective differences between SI and SII might be revealed by studying the effects of SII and SI–SII lesions in infant macaques to complement our earlier studies of infant SI lesions (Carlson, 1984a–c). The studies in SI showed that following partial removals of area 3b or of 1 and 2 combined, infants recovered normal (or near-normal) function after about 3–4 months of training (Carlson, 1984b). With the thought that this recovery was mediated by the remaining areas in the damaged SI, the consequences of total unilateral SI lesions were explored (Carlson, 1984c). Surprisingly, infants with total SI lesions did not require months of training to achieve normal function; they learned at the same rate and efficiency as normal infants (Carlson, 1984a) and performed significantly better than did infants with partial SI le-

Received Mar. 30, 1987; revised July 31, 1987; accepted Aug. 10, 1987.

This work was supported by National Institutes of Health Grants NS15070, MH40157, and NS09809. We thank John Charlton, Ed Crader, Zeke Leventhal, and Gavin Perry for technical assistance, Joe Hayes for photography, and Jan Hoffmann and Sue Eads for secretarial assistance.

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sions and juveniles with total SI lesions. The results of the total SI lesions in infants led to the present experiment, in which the contributions of the remaining contralateral SI, and SII in the damaged and undamaged cortex, were explored.

Materials and Methods

Rearing procedures. Infant macaques were removed from their mothers at from 1 d to 1 month after birth and raised in heated incubators; they were fed a diet of Enfamil infant formula (Mead Johnson). Between 2 and 8 weeks of age they were trained to contact and pull a small metal handle that projected into the incubator in order to receive their daily feedings of formula. Records were made of daily weights, and supplemental feedings were given if adequate feeding was not obtained from the training sessions. On weekends, full feedings were given in the home incubator. After infants learned to pull the handles in the incubator, and then on the discrimination apparatus, they were subjected to cortical ablations.

Surgery. Animals were deprived of formula 12 hr prior to surgery. Aseptic surgical procedures were always used. After initial tranquilization with an intramuscular injection of ketamine hydrochloride (10 mg/kg), the animals were brought to a surgical depth of anesthesia by an intravenous injection of sodium pentobarbital (30 mg/kg). The heads were restrained in a stereotactic device. A large skull flap was removed and the dura incised and deflected medially, exposing the central (CS), intraparietal (IPS), and lateral (LS) sulci; the SI or SII hand areas were removed through suction with a 22-gauge probe. For total SI lesions, we removed all gray matter between the IPS and CS, medially to the postcentral sulcus and laterally to the tip of the IPS (Powell and Mountcastle, 1959a; Jones et al., 1978). The lesions extended down the posterior bank of the postcentral gyrus and into the lateral one-third of the anterior bank of the IPS. Lesions of the SII hand area were achieved by ablating the upper bank of the lateral sulcus. The lesion was started directly lateral to the tip of the IPS at a point where several branches of the middle cerebral artery emerge from the LS. These vessels were carefully preserved in those cases where SI was to be spared. The lesion was first extended to the fundus of the sulcus by following the course of the blood vessels to the insula. Once the insula was exposed, the lesion was extended from the superior limiting sulcus through the inner bank of the operculum rostrally, and caudally along the entire depth of the upper bank of the LS behind the insula for approximately 5–8 mm. Following surgical ablations, the areas were filled with Gelfoam and the dura and skull flap replaced with absorbable sutures. The connective tissue and skin were closed and animals were given prophylactic doses of antibiotic (Pro Cilin).

Behavioral training and testing. The apparatus, discrimination objects, and training and testing procedures were identical to those developed for earlier studies in infant *Macaca* (Carlson, 1984a–c). The apparatus was contained in a Wisconsin General Testing Apparatus (WGTA) within a darkened room. The animal was trained to reach through a tube to contact a pair of handles mounted on the apparatus. In this forced-choice situation, the animal had to contact the pair of handles and to pull forward on one, thereby indicating a choice. A signal from that handle was transmitted to the programming equipment, and "correct" responses triggered a solenoid to release a drop of Enfamil formula into a metal nipple in the cage front and to flash a yellow light. If the response was incorrect, a buzzer sounded, there was a flash of red light, and no formula was delivered. The apparatus contained 12 pairs of handles, one mounted vertically above the other, on a rotating carousel. After each response, the carousel moved backwards away from reach, rotated to the next pair of handles, and moved forward to present the next comparison. The 12 paired comparisons were presented repeatedly in rapid succession (see Carlson, 1984a).

The handles used for these tasks varied either in size (cross-sectional diameter) or in texture (grade of aluminum oxide abrasive cloth covering the handle). The smallest-sized handle or the smoothest-textured handle of any pair was designated as correct. The size handles were either 7, 9, 12, or 18 mm in diameter and 3.8 mm in length. The texture handles were all 3.8 mm in length and 1 cm in diameter; they were covered with 320, 120, 80, or 40 grains/linear in. (grains/in.) abrasive. Training began with the presentation of the easiest (i.e., most discrepant) pair of texture handles, 320–40 grains in animals tested on that single comparison until they achieved 80% correct in one session of between 50 and 120 trials. When the 80% criterion was reached, they were given a more difficult comparison, 320–80 grains/in., until criterion was reached. Then the most difficult comparison, 320–120 grains/in., was presented, followed by 120–40, 80–40, and 120–80 grains/in. as each comparison was mastered. By this method of *acquisition,* all combinations of handles were individually mastered. Errors to criterion served as the measure of efficiency of acquisition; curves of percentage correct across days illustrated the course of *learning.* Subsequently, a measure of *performance* was introduced, in which all 6 pairs of comparisons mastered during acquisition were presented together in the same session, the "alls" test. In this more complex task, the levels of difficulty changed on every trial, as easy, moderate, and difficult comparisons were presented in the same session, and what constituted the "correct" choice also changed. The average percentage correct over 5 sessions was used as a measure of performance. The same procedures were followed for the acquisition of size discrimination and for the presentation of the size "alls" task.

Histology. Following completion of all behavioral testing, the animals were given an overdose of sodium pentobarbital and immediately perfused transcardially with saline and neutral formalin. The brains were removed, photographed, and sectioned frozen at 50 μm; the mounted sections were stained with thionin. Lesion sites were reconstructed from sections using orthoplanometric procedures described previously (Jones and Burton, 1976). The cytoarchitectonic areas surviving in the vicinity of the lesions were noted, and identifiable borders plotted onto drawings of the sections. The thalamic nuclei showing profound retrograde cell degeneration were also identified.

Cell counts of surviving neurons were made in the lateral and inferior ventroposterior nuclei (VPL and VPI) to confirm the severity of the lesions. A cell was counted when its nucleus could be seen with a 40× objective. All cells found within a reticle field of 625 μm^2 were counted, at which point the stage was adjusted to an adjacent field. The boundaries of the areas to be surveyed were previously marked on the coverslips, using low-power objectives. As a limited number of fields could be counted within the boundaries selected per section, several adjacent sections, separated by at least 150 μm, were used until 20 fields had been analyzed. The selection process for the areas to be counted differed for VPL and VPI. In cases where SI had been lesioned, clear zones of gliosis were found in VPL. The cell counts in these cases were always made within the boundaries of maximal gliosis. When only SII was lesioned or where no lesions were present, there were no gliotic zones; counts in these cases were made over portions of VPL that were as nearly identical as possible to those regions counted in the SI lesion cases. These comparisons were based on such factors as matching sizes, shapes, and the locations of neighboring nuclei (i.e., the ventroposterior medial nucleus, centrum medianum, and geniculate nuclei). Counts in VPI were made only in cases that were processed from coronal sections because of the absence of cytoarchitectural distinctiveness of the posterior, dorsal, and ventral boundaries of this nucleus on horizontal sections. In addition, only the central, most obvious parvocellular portions of VPI were surveyed, to avoid potential erroneous counts of cells from neighboring nuclei. A consequence of these precautions was that the counts for VPI were obtained from approximately twice the number of sections needed to view 20 fields in VPL.

Results

The results from this study will be presented in 4 sections, with the first 3 sections arranged according to lesion group: SI, SII, and SI–SII. The fourth section will describe degenerative changes in the ventral thalamus associated with the different lesion groups. In each of the first 3 sections, a histological description of the cortical lesion will be presented, followed by a summary of the tactile discrimination capacity based on tests of size and texture discrimination abilities. The age of the animals and the location and size of all the lesions in this study are summarized in Table 1. The behavioral data following different lesions in the 9 animals will be referred to by animal number and a lower-case letter indicating which stage of lesion is being evaluated in the behavioral testing, e.g., Mm3a. An upper-case L or R indicates the left or right hemisphere.

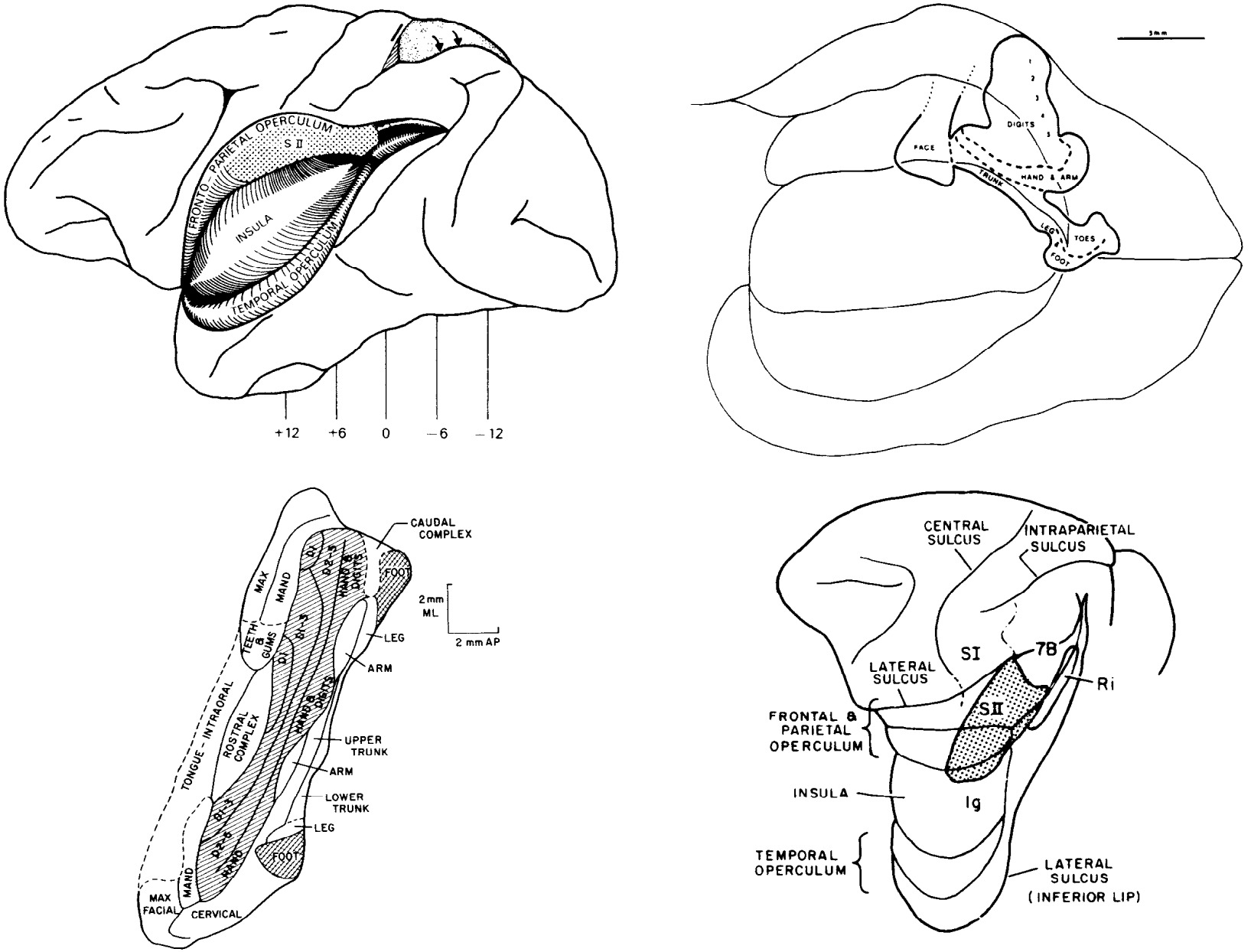
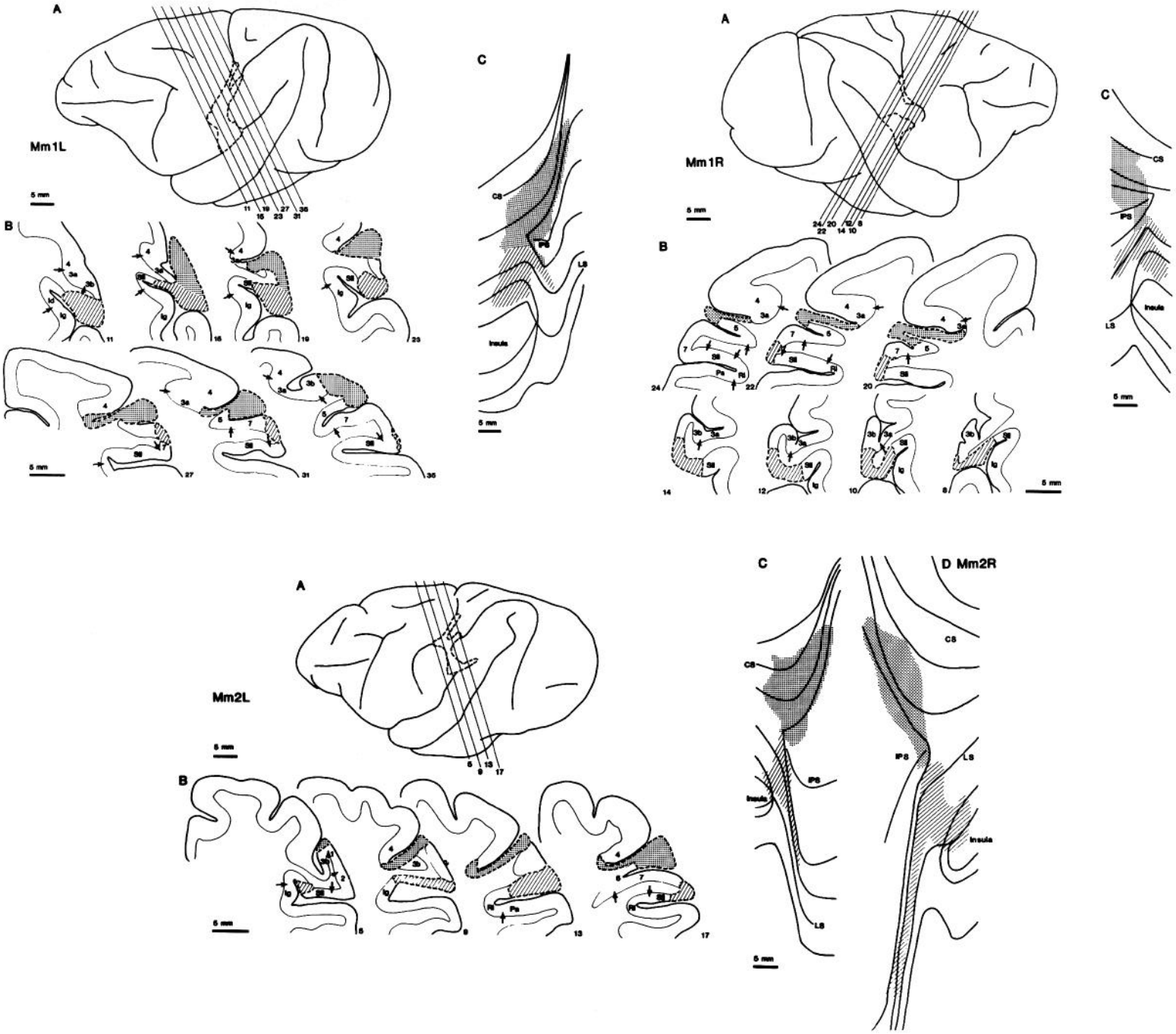


Figure 1. Location and/or organization of SII from different sources. *Upper left-hand corner,* schematic of the lateral view of the lateral sulcus used by Murray and Mishkin (1984) for SII lesion studies in adult *Macaca*; *upper right-hand corner,* summary diagram of body representation in SII in lateral sulcus demonstrated by connection pattern with SI (Friedman et al., 1980); *lower left-hand corner,* body map in SII obtained from electrophysiological recordings; and *lower right-hand corner,* location of that map on schematic of expanded lateral sulcus (Robinson and Burton, 1980a).


A
Mm1L
5 mm
11 19 27 35
15 23 31
B
4
3a
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C
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24 20 12 8
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5 mm

Table 1. Summary table for lesioned infants and adults

Animal	Lesion	Hemi-sphere	Surgery age (weeks)	SI Lesion size (mm^2)	SII Total (mm^2)	SII Inner	SII Outer	SII Pos-terior
Mm1a	SI	R	7.6	100	—	—	—	—
b	SI	L	130.9	200	—	—	—	—
c	SII	R	208.7	(100)	60	26	34	—
d	SII	L	228.0	(200)	61	33	22	6
Mm2a	SI	R	5.7	85	—	—	—	—
b	SI–SII	L	109.3	198	41	7	22	12
c	SII	R	144.6	(85)	54	12	14	28
Mm3a	SII	L	7.0	—	70	26	33	21
b	SI	R	25.3	108	—	—	—	—
c	SI	L	25.3	148	(70)	(26)	(33)	(21)
Mm4	SI–SII	L	7.6	132	91	6	48	37
	Sham	R	37.3	—	—	—	—	—
Mm5a	SII	R	5.6	—	56	17	31	8
		L	5.6	—	60	5	10	45
b	SI	L	28.4	175	(60)	(5)	(10)	(45)
Mm6	SI–SII	R	7.4	75	58	—	—	58
Mm7	SII	L	7.3	—	58	5	33	20
		R	7.3	—	54	9	12	33
Mm8	3b–SII	L	7.3	60	44	2	2	40
	SII	R	7.3	—	61	10	51	—
Mm9	1, 2–SII	R	7.4	95	13	3	10	—
	SII	L	7.4	—	41	—	41	—

R, right; L, left. —, No damage; ( ), previous lesion.

Lesions in SI in infant and adult animals

Two infant macaques were given SI lesions to determine the role of undamaged remaining somatic sensory cortical areas in the mediation of the normal tactile function found in an earlier study of total SI-lesioned infants (Carlson, 1984c). Following recovery of tactile function after the early SI lesion, the remaining SI or SI and SII were removed to see if the performance on the hand contralateral to the infant-lesioned hand (and ipsilateral to the recent lesion) was affected.

Mm1a refers to the first lesion in animal 1, which was in the right hemisphere. The reconstruction of the right hemisphere of this animal is illustrated in Figure 2 as Mm1R. The size of this lesion, as determined by measurement of the cortical surface area on the orthographic reconstruction in Figure 2*C* (of Mm1R) was estimated to be 100 mm^2 (see Table 1). This lesion was made at 7.6 weeks of age but was not measured until the animal was euthanized 5 years later. In this case all of areas 3b, 1, and 2 for the digit zone were damaged; some of area 3b was spared medial to IPS.

Mm1b (L) received a second SI lesion at 130.9 weeks. This second lesion was estimated to be twice the size of the first when measured in the adult brain. This large lesion extended laterally into the face area and caudally into the IPS, appearing to remove all of area 2 in that sulcus. All of areas 3b, 1, and 2 were removed medial to the IPS and lateral to the postcentral sulcus, the usual boundaries of the hand and arm area. Portions of areas 3a and 5 were also damaged (see Fig. 2).

Mm2a (R) received an SI lesion in the right hemisphere at 5.7 weeks of age and was euthanized more than 3 years later. At its maximum, this lesion destroyed nearly all of the posterior bank of the CS; damage extended into area 3a near the fundus

←

Figure 2. Localizations of lesions in *Mm1L*: *A*, Lateral view of left hemisphere showing the position of the lesions (*dashed lines*) along the central (*CS*), intraparietal (*IPS*), and lateral (*LS*) sulci. The position of selected parasagittal sections is shown by the *diagonal lines*, whose numbers correspond to the sections shown in *B*. *B*, The lesions have been reconstructed to approximate the tissue removed during surgery. The ablation intended for SI is *stippled* and that for SII is marked with *diagonal lines*. The cytoarchitectonic boundaries of surviving cortical areas in the vicinity of the lesions have been noted with *arrows*. These borders were not always clear where the tissue was excessively distorted by the lesions. *C*, Orthoplanimetric reconstruction of the cortical regions containing the lesions. These reconstructions were started from the most dorsal portion of the histological sections for each case. All of the reconstructions are not identical across the different cases because of differences in the planes of section, slight differences in the sulcal patterns, and especially the varying degrees with which the ablated regions could be estimated onto the sections. However, the areal extent of the lesions, the borders with the sulci, and the relative position of the lesions, where both were present, can be compared across cases. Localization of lesions in *Mm1R*: *A*, Lateral view of right hemisphere. *B*, Sections through the major extent of the lesions. *C*, Orthoplanimetric reconstruction of lesions. Localization of lesions in *Mm2L and Mm2R*: *A*, Lateral view of left hemisphere. *B*, Sections through the major extent of the lesions. *C*, Orthoplanimetric reconstruction of left lesion. *D*, Orthoplanimetric reconstruction of right lesion.

Table 2. Size comparison by SI-lesioned infants and juveniles

Animal	Age (weeks)	Hand	Size comparison: A. Errors to criterion (and age in weeks) during acquisition 7–18 mm	7–12 mm	7–9 mm	Total on 6 tasks	B. Percentage correct on individual comparisons on the "alls" task Age (weeks)	7–18 mm	7–12 mm	7–9 mm	Mean of all 6 comparisons
Mean normal		1st:	7.8 (15.2)	5.2 (15.6)	54 (15.9)	(115.1)	19.4:	98.4	95.4	76.7	(92.3)
infant		2nd:	25.0 (21.9)				21.4:	98.5	95.4	69.5	(90.2)
1–2 Month infant (ipsilateral lesion in adult)											
Mm1a	7.6	C-L	52 (68.0)	19 (68.3)	170 (70.3)	(285)	71.7	100.0	89.7	60.6	(88.2)
Mm1b	130.9	C-R	175 (183.7)	164 (189.9)	568 (190.9)[a]	(965)[a]	195.1	97.0	92.0	67.3	(85.5)
		I-L					201.7	94.8	88.5	57.7	(85.2)
Sham-operate											
Mm4b	37.3	C-L	1 (41.1)	1 (41.3)	3 (41.4)	(11)	42.3	100	99.0	77.0	(95.5)

[a] Never reached criterion.

of CS. Posteriorly, the lesion swept through area 1 and spanned both banks of the rostral third of IPS to include area 2 and the rostral edge of area 5 (Fig. 2). The size estimate of 85 mm² is similar to that for the previous infant-lesioned animal.

At 109.3 weeks of age, Mm2b received a complete SI and SII lesion of the digit zone in the left hemisphere (Mm2L) (Fig. 2). The SI removal included tissue in areas 3b, 1, and 2, and some unintentional damage in area 4's lateral border near the termination of the IPS. The SII lesion in this case will be discussed below (see Fig. 2).

In Mm3b, the SI lesions were made at 25.3 weeks in both the right and left hemispheres. The left SI, made in combination with an early SII lesion, was of average size but smaller than the right SI lesion (see Mm3R in Fig. 3, which is described later in the SII–SI section). The SI lesion in Mm3R may have spared some tissue in 3b medial to the tip of the IPS, but was complete in 3b, 1, and 2 in more lateral sections.

Size discrimination in SI-lesioned infants and adults

Only one of the animals receiving an SI lesion in one hemisphere as an infant, and in the remaining SI as an adult, was tested on size tasks on both hands following the second lesion. This animal showed some lasting impairment following the early SI lesion, but was not worse on size performance following the second SI lesion (see Mm1a L hand in Table 2). Mm1a received an SI lesion at 7.6 weeks, but was over a year of age (68.0 weeks) when first tested on size tasks. The errors to criterion for each of these size comparisons, and the total errors over the 6 size comparisons, were all slightly higher than the average for normal infants of 15–16 weeks of age, as shown in Table 2, A. When tested on the size "alls" task, performance on the easy and moderate-level tasks was near the normal average, but on the difficult task was 16% below normal in performance (see Table 2, B). The average performance over all 6 comparisons on the "alls" task was only slightly below average when Mm1b was tested on the hand contralateral to an SI lesion at 130.9 weeks. Training on the difficult 7–9 mm task was discontinued after failure to reach criterion over 10 sessions during 4 weeks of training. Performance on the size "alls" task on the hand contralateral to the latest lesion was depressed relative to that of normal infants and of the sham-operate. Performance on the ipsilateral hand was also depressed relative to that of normal infants and of the sham-operate (see Table 2, B) on the difficult comparison and on the average performance over all 6 size comparisons. Note that the performance on size "alls" was similar for the left hand following the early contralateral SI lesion and after the subsequent ipsilateral SI lesion. The role of the ipsilateral SI in mediating recovered size performance is equivocal. The results on this single animal showed that the late removal of the remaining SI did not change discrimination performance when compared to the change following the first lesion alone.

Texture discrimination by SI-lesioned infants and adults

Two animals demonstrating recovered texture discrimination capacity following early SI lesions were tested on both hands following the subsequent removal of the remaining SI. Neither animal showed a significant loss of the earlier-recovered function following the second lesion. Mm1a was tested first on texture (i.e., before size) and had considerable difficulty during initial shaping. This animal frequently adopted a position habit that resulted in many more errors and sessions needed to reach criterion on the first test series. When tested a second time on texture following size discrimination training, normal reacquisition scores and texture "alls" performance (see Mm1a contra-left in Table 3, A and B) were achieved. Texture performance was normal, or near normal, prior to the second SI lesion.

When Mm1b received an SI lesion in the left hemisphere at 130.9 weeks, opposite to the SI lesion received at 7.6 weeks of age, the hand contralateral to the late lesion was impaired compared to the hand contralateral to the early SI lesion. The more recent lesion also resulted in deficits on the texture "alls" performance to a greater degree in the hand contralateral than in the hand ipsilateral to that lesion (Table 3, A and B: Mm1b, C-R and I-L). When performance on the hand ipsilateral to the late lesion (contralateral to the early SI lesion) was compared to performance on that same hand about 2 years earlier, the recent overall performance was lower. Compared to the results from retesting of the sham-operate, reacquisition on the hand contralateral to the early SI lesion appeared normal, but performance on the texture "alls" task was low. When the other hand (contralateral to the late SI lesions) was compared to that

Table 3. Texture comparison by SI-lesioned infants and juveniles

Animal	Age (weeks)	Hand	Texture comparison: A. Errors to criterion (and age in weeks) during acquisition: 320–40 grains/in.	320–80 grains/in.	320–120 grains/in.	Total on 6 tasks	B. Percentage correct on individual comparisons on the "alls" task: Age (weeks)	320–40 grains/in.	320–80 grains/in.	320–120 grains/in.	Mean of all 6 comparisons
Mean normal		1st:	77.2 (20.3)	26.5 (20.9)	92.2 (16.3)	(249.5)	30.8:	97.8	94.8	63.7	(89.2)
infant		2nd:	23.4 (36.0)				36.3:	95.2	84.3	57.4	(83.6)
1–2 Month infant (ipsilateral lesion in adult)											
Mm1a	7.6	C-L1	907 (15.7)	533 (20.6)	357 (25.6)	(2060)	65.9	81.3	68.8	63.8	(69.0)[a]
		C-L2	444 (72.9)	29 (74.9)	68 (75.0)	(1191)	116.1	98.0	91.0	71.0	(85.3)
Mm1b	130.9	C-R	147 (132.1)	53 (133.1)	44 (134.6)	(873)	205.6	88.0	81.0	65.0	(70.2)
		I-L	12 (131.0)	31 (133.6)	30 (134.0)	(182)	207.0	88.4	89.1	80.7	(77.8)
Mm2a	5.7	C-L	70 (8.6)	131 (8.7)	537 (9.1)	(756)	12.3	100	89.4	68.5	(85.5)
		I-R					13.7	100	99.0	68.0	(92.0)
Mm2b	109.3	I-L	50 (133.3)	184 (133.6)	747 (134.6)	(1637)	143.3	96.0	79.0	72.0	(77.3)
Sham-operate											
Mm4b	37.3	C-L	17 (37.4)	40 (47.6)	185 (38.1)	(171)	39.6	100	98.1	76.0	(93.0)

[a] Percentage is based on 2 sessions.

of the sham-operate, it was relatively deficient on acquisition and "alls." Clearly, the second lesion did not impair the level of recovered texture discrimination capacity in the ipsilateral hand to the degree seen in the hand contralateral to the more recent lesion.

Mm2a was tested at 8.6 weeks on the left hand after an SI lesion at 5.7 weeks following size discrimination training. When initially tested on the hand contralateral to the infant SI lesion, the error scores were slightly inflated relative to the average for normal infants, but 2 sessions were given each day (in contrast to the procedure for normal infants). If days to criterion are compared, this acquisition performance was not different from that of normal infants. Texture "alls" performance was the same as in normal infants, as shown in Table 3, B (Mm2a, C-L).

As in the previous animal, some slight impairment was seen in the left hand (contralateral to an early SI lesion) when an SI–SII lesion was made in the left hemisphere at a later age (ipsilateral to the hand tested previously). Twenty sessions were required on the left hand after the later lesion to reacquire the difficult texture task. Performance on the texture "alls" on the left hand was lower following the late ipsilateral SI lesion (Mm2b) than following the early contralateral SI lesion. Neither size nor texture discrimination capacity could be measured in the right hand contralateral to the late SI lesion; tactile impairment was so severe that the animal never learned to discriminate between a single handle and no handle. Both texture acquisition and "alls" were depressed on the left hand as compared to performance on that same hand following the early SI lesion. Again, the degree of impairment following an early contralateral and late ipsilateral SI lesion did not approach that seen following a contralateral SI lesion in an older animal (Carlson, 1984c).

In summary, in the 2 infants in which recovery of texture capacity was seen following an infant SI lesion, removal of either SI or SI and SII in the undamaged hemisphere caused a slight reduction in the level of persisting recovery. The texture deficits following the late lesion were, in both cases, more severe for the hand contralateral to that lesion than for the hand contralateral to the early lesion, as was seen in the overall texture "alls" performance. The minor reduction in the level of recovery found following the late lesion demonstrates that the undamaged SI in the early-lesioned animal cannot alone account for recovery of tactile function after early SI damage.

SII lesions in infants

SII lesions were intended to extend up to the boundaries of SII (particularly the hand area), as suggested by electrophysiological mapping studies (Robinson and Burton, 1980a) and connectional patterns (Friedman et al., 1980) of adult *Macaca fascicularis*. SII lesions will be described in relation to the extent of the lesion on the inner and outer banks of LS, rostral to the posterior pole of the insula, and on the upper bank posterior to the insula. Posterior portions of SII will be defined as that part of the upper bank posterior to a line drawn from the tip of the insula to the tip of the IPS (see Fig. 1).

Five animals first received SII lesions between 5.6 and 7.4 weeks of age. One of these 5 received a unilateral lesion (Mm3L) and 4 received bilateral lesions, with only SII removed in each hemisphere (Mm5R and Mm5L, and Mm7R and Mm7L). In 2 animals, SII was removed in one hemisphere and some of SI was removed at the same time in the other hemisphere (Mm8R, Mm9L). Illustrations of the lesions in these animals are presented in Figure 3 (Mm3L, Mm5R and Mm5L, and Mm7L and Mm7R) and Figure 4 (Mm9). Measurements of the extent of SI and SII removals in each animal are presented in Table 1.

Animal Mm3L received a unilateral lesion of SII at 7 weeks, which spanned all of the parietal operculum except for a small region along the superior limiting sulcus, parallel to the posterior third of the insula. The lesion removed nearly all of the rostral SII except for a small part near the superior limiting sulcus and a small posterior segment bordering on the retroinsular area (Ri).

The animals with bilateral SII lesions had total removals in the same size range as that in Mm3L, but the distribution of the damage was not always equivalent. In the right hemisphere of Mm5a the lesion was primarily in the outer bank, with only minimal damage noted in the inner bank or posterior to the

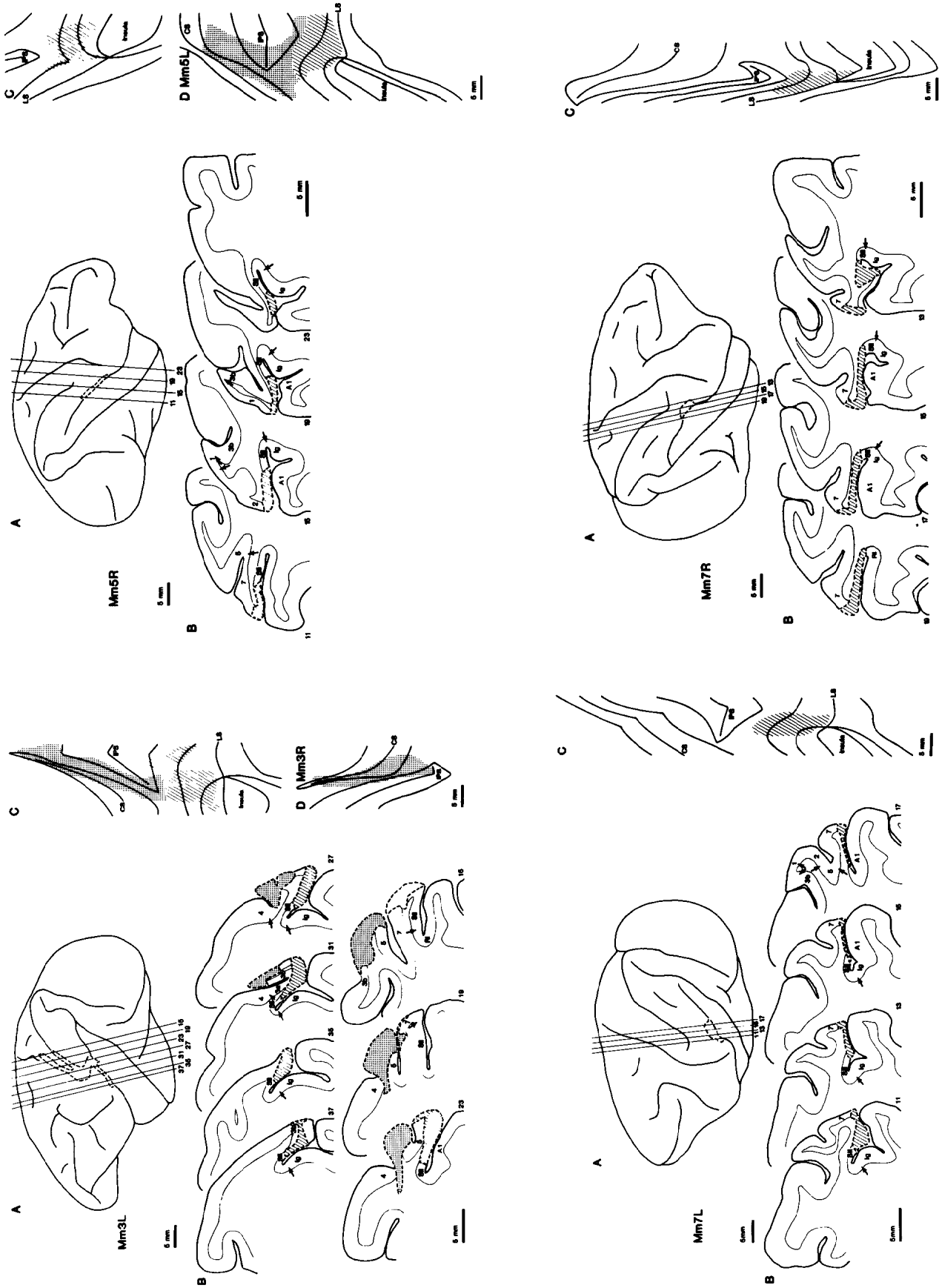


Figure 3. Localization of lesions in Mm3L, Mm3R, Mm5R, Mm5L, Mm7L, and Mm7R. *A,* Lateral view of left hemisphere. *B,* Sections through the major extent of the lesions. *C,* Orthoplanimetric reconstruction of lesions. *D,* Reconstruction of lesions on the right side of Mm3 and the left side of Mm5. See Figure 2 for further discussion.

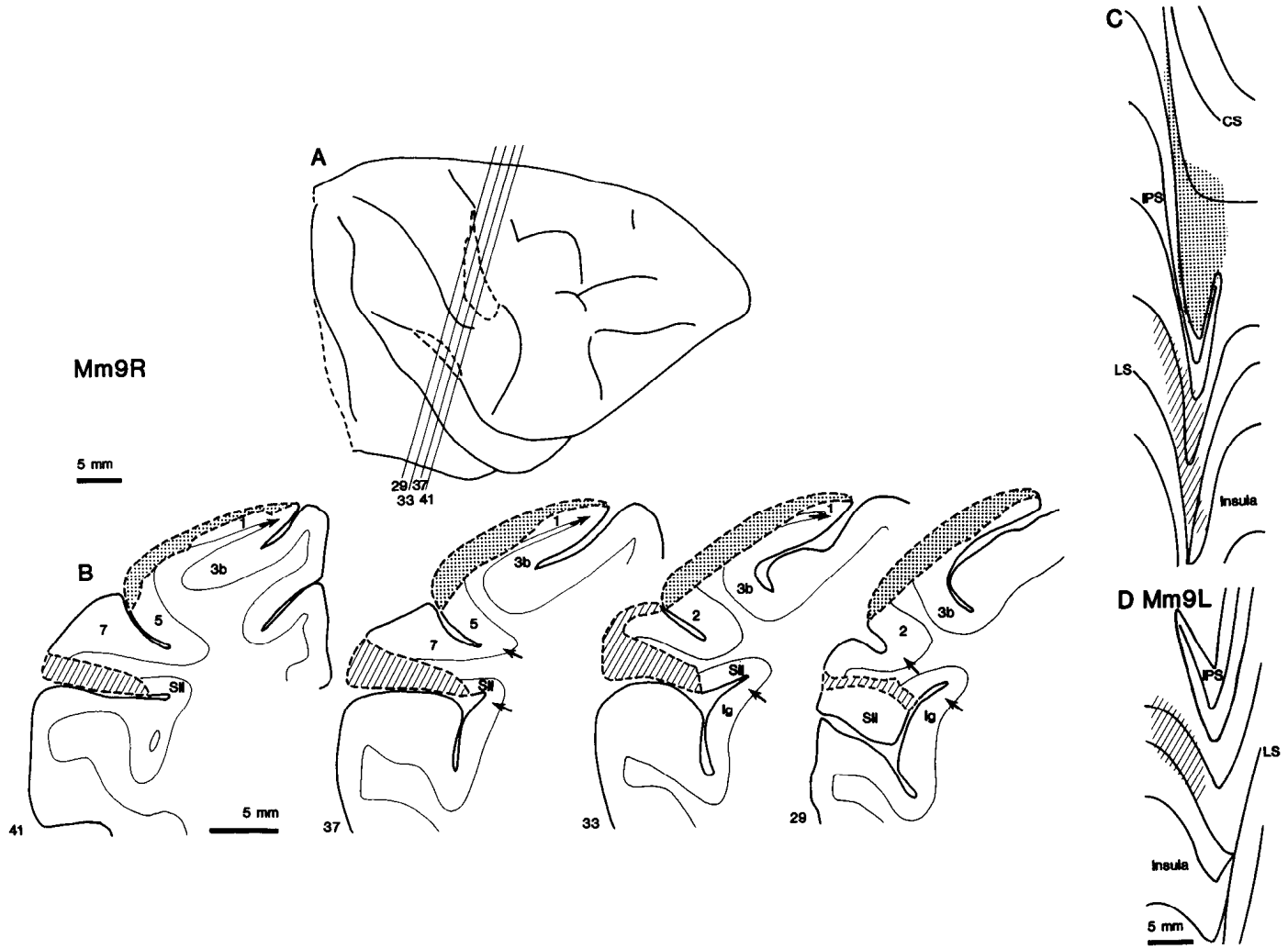


Figure 4. Localization of lesion in Mm9R and Mm9L. See Figure 2 for further discussion.

insula. In the left hemisphere, the same amount of tissue was removed, but primarily from the area posterior to the insula. The 2 SII lesions in Mm7 were also quite different from previous lesions and from each other. On the left side, the lesion was similar in size to those in Mm3 and Mm5, but spared the inner bank near the posterior tip of the insula; the lesion in the outer bank was complete and extended nearly to the fundus of LS in posterior regions of SII. The lesion on the right of Mm7 was of comparable size to other SII lesions (Table 1); the inner face of the parietal operculum near the posterior pole of the insula was mostly spared and the outer face was less fully damaged than in other cases. In all of these cases there was some damage to area 7 on the lateral convexity of the inferior parietal lobule. Ri was largely spared except for some damage in Mm7R (see section 20, Fig. 3), where the lesion reached the fundus of LS posterior to the insula.

In the 2 animals in which SII was ablated in one hemisphere and SI–SII in the opposite hemisphere, the SII lesions were more restricted, although generally of comparable size to other SII lesions. On the right side of Mm8 (not illustrated), the lesion was similar to that in Mm7L, especially in the vicinity of the posterior pole of the insula; nearly all of the outer bank of the parietal operculum was destroyed, while the inner bank was mostly spared. In Mm8R (not illustrated), the upper bank of LS behind the insula was less affected than in Mm7L. The remaining 3 SII lesions were smaller. The lesion on the right of Mm9, which resembled that in Mm7R, ablated most of the upper bank of the LS around the posterior pole of the insula, largely spared the inner bank next to the insula, and marginally affected the upper bank of LS within the first 2–3 mm posterior to the insula (Fig. 4). In Mm8L and Mm9L, the lesions were restricted to the outer bank of the parietal operculum; these lesions missed most of SII except for some of the face and the rostral half of the forelimb representations.

Size discrimination in SII-lesioned infants

None of the SII lesions, unilateral or bilateral, resulted in any retardation of size acquisition compared to normal infants, as is shown by the minimal number of errors in reaching the 80% criterion (Table 4, A). All animals reached that criterion in a single session for each comparison (Fig. 5, A, B). As a group, they made significantly fewer errors than did normal infants, averaged over the 6 tasks (Mann-Whitney test; $p \leq 0.05$), indicating more efficient than normal learning for the SII-lesioned animals. Some of this difference may relate to the older age of the SII-lesioned animals as compared to normal infants at the time of testing on size tasks. In contrast to previous studies (Carlson, 1984a–c), animals were tested on texture tasks prior

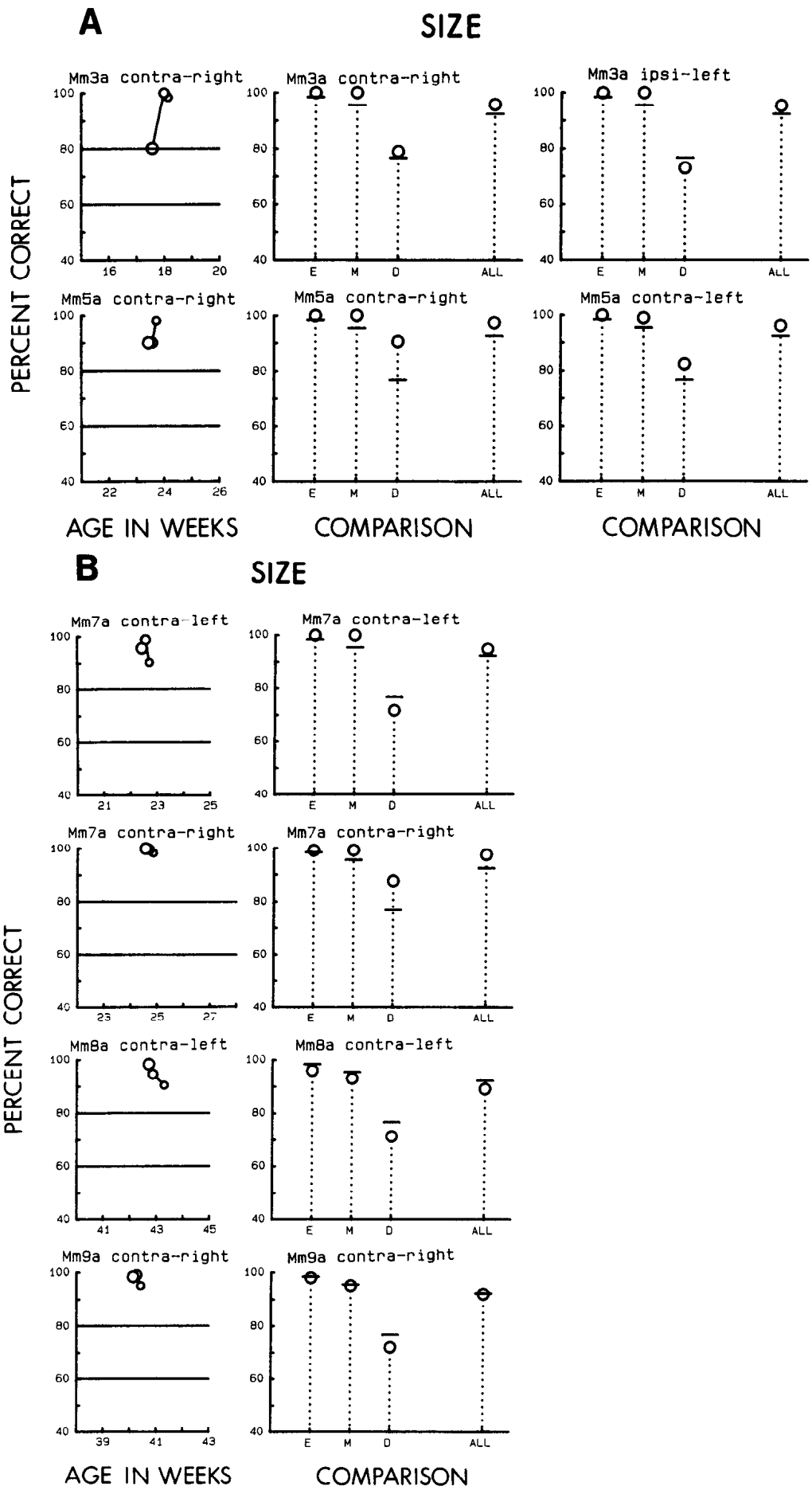


Figure 5. *A,* Acquisition curves and "alls" plots for Mm3a and Mm5a on size discriminations. Age in weeks at the time of testing is shown chronologically on the abscissa. The task difficulty is reflected in the size of *circles,* with the largest circle for the easiest size comparisons (7 vs 18 mm), the middle-diameter circle for the moderate-difficulty size (7 vs 12 mm) tasks, and the smallest circles for the percentage correct for the most difficult size comparisons (7 vs 9) (see Fig. 8*B* for further discussion). *B,* Acquisition curves and "alls" plots for size discriminations for Mm7a, Mm8a, and Mm9a.

Table 4. Size comparison by SII-lesioned infants

Animal	Age (weeks)	Hand	Size comparison: A. Errors to criterion (and age in weeks) during acquisition 7–18 mm	7–12 mm	7–9 mm	Total on 6 tasks	B. Percentage correct on individual comparisons on the "alls" task Age (weeks)	7–18 mm	7–12 mm	7–9 mm	Mean of all 6 comparisons
Mean normal infant		1st:	7.8 (15.2)	5.2 (15.6)	54 (15.9)	(115.1)	19.4	98.4	95.4	76.7	(92.3)
		2nd:	25.0 (21.9)				21.4	98.5	95.4	69.5	(90.2)
Mm3a	7.0	C-R	83 (17.6)	0 (18.0)	5 (18.1)	(96)	19.1	100.0	100.0	79.0	(95.7)
		I-L					20.3	100.0	100.0	73.0	(95.3)
Mm5a	5.6	C-R	12 (23.4)	7 (23.6)	2 (23.7)	(22)	24.6	100.0	100.0	90.3	(97.3)
		C-L	3 (27.4)	2 (27.6)	—	—	27.7	100.0	99.0	82.3	(96.2)
Mm7a	7.3	C-L	14 (22.4)	3 (22.6)	20 (22.7)	(46)	23.6	100.0	100.0	71.7	(94.8)
		C-R	0 (24.5)	1 (24.7)	3 (24.9)	(4)	25.7	99.2	99.2	87.6	(97.3)
Mm8a	7.3	C-L	2 (42.7)	7 (42.9)	12 (43.3)	(51)	43.9	96.1	93.1	71.3	(89.1)
Mm9a	7.6	C-R	2 (40.1)	1 (40.3)	6 (40.4)	(20)	41.4	98.0	95.1	72.0	(92.0)
Mean SII lesions			16.6 (28.3)	3.0 (28.5)	6.9 (24.7)	(34.1)[a]	29.2	99.0	98.1	79.2	(94.6)
Mean SI lesions			37.3 (22.8)	14.8 (22.9)[b]	72.0 (23.2)[b]	(146.7)[b]	24.7	100.0	93.8	65.8[b]	(92.5)

[a] Significant at $p \leq 0.05$ from normal infants.

[b] Significant at $p \leq 0.05$ from SI-lesioned infants.

to size tasks. In a previous study, normal infants were found to improve on size acquisition and size "alls" performance as a function of age (Carlson, 1984a).

On the size "alls" task, all SII-lesioned infants were equal to or better than normal infants in their level of performance (Fig. 5, *A, B*; Table 4, B). There were no significant differences in performance on size "alls" tasks between SII-lesioned and normal infants. There were also no detectable differences in size acquisition or size "alls" performance as a function of size or location of the SII lesions.

When compared to the SI-lesioned infants from a previous study (Carlson, 1984c), the SII-lesioned animals showed significantly fewer errors on the 7–12 and 7–9 mm acquisition tasks and over all 6 acquisition tasks, and showed significantly better performance on the 7–9 mm comparison in the size "alls" task (Mann-Whitney; $p \leq 0.05$) (see Table 8).

Texture discrimination in SII-lesioned infants

Acquisition of texture discrimination in SII-lesioned infants was protracted, especially for some cases and on the more difficult comparisons. Mm3a did not show inflated error scores on the 320–40 and 320–80 grains/in. comparisons, but did make more errors on the difficult 320–120 grains/in. comparison (see Table 5, A). As shown in Figure 6*A*, this animal was taken back to an easier comparison, 320–80 grains/in., on several occasions, because of its score of below 60% correct on that comparison. Criterion was reached on the difficult comparison after 14 sessions, whereas normal infants required 1–6 sessions to reach criterion (Carlson, 1984a). Performance for Mm3a on the "alls" task, with the hand contralateral to the 7 week SII lesion, was slightly lower than that of normal infants, and was even lower than Mm3a's performance with the hand opposite the 25 week SI lesion (Fig. 6*A*; Mm3a, ipsi-left) on the difficult comparison (320–180 grains/in.), but was normal on the average performance over all 6 comparisons.

Mm5a (Fig. 6*A*) was also normal in acquisition of the initial 2-texture comparisons, but made 10 times the normal number of errors on the difficult texture comparison on both the right and left hands. Difficulty with the 320–120 grains/in. comparisons was responsible for the large number of errors in the sum for all 6 comparisons for both hands during acquisition (Table 5, A). In spite of the obvious delay in texture acquisition for both hands of Mm5a, performance on the difficult 320–120 grains/in. comparison and on all 6 comparisons was slightly above normal (see Fig. 6*A*).

Mm7a (Fig. 6*B*) showed a significant delay in texture acquisition on the right hand but no delay when tested next on the left hand. The difficulty with the right hand was primarily on the 320–120 grains/in. comparison (see Table 5, A and Fig. 6*A*). When tested on the texture "alls" task, performance on the difficult 320–120 grains/in. task was slightly below normal for both hands, yet performance over all 6 tasks was like that of normal infants (see Table 5, B and Fig. 6*B*).

Mm8a required 30 sessions and made 10 times the normal number of errors to reach criterion on the left hand. The degree and pattern of impairment were equivalent to those seen on the left hand of Mm5a and the right hand of Mm7a after lesions of comparable size and location. Although performance on the 320–120 grains/in. comparison was slightly below normal, over all 6 comparisons performance on the "alls" task was similar to that of normal infants (see Fig. 6*B*).

Mm9a showed a moderate impairment on acquisition of the difficult texture task with the right hand, making twice as many errors and requiring many more sessions than normal infants to reach criterion. The errors with the right hand were less than half of those on the left hand of Mm8a (see Table 5, A and Fig. 6*A*); however, the pattern of performance on the texture "alls" task was nearly identical for the 2 animals.

In summary, statistical tests indicated that SII-lesioned infants made significantly more errors than normal infants did to

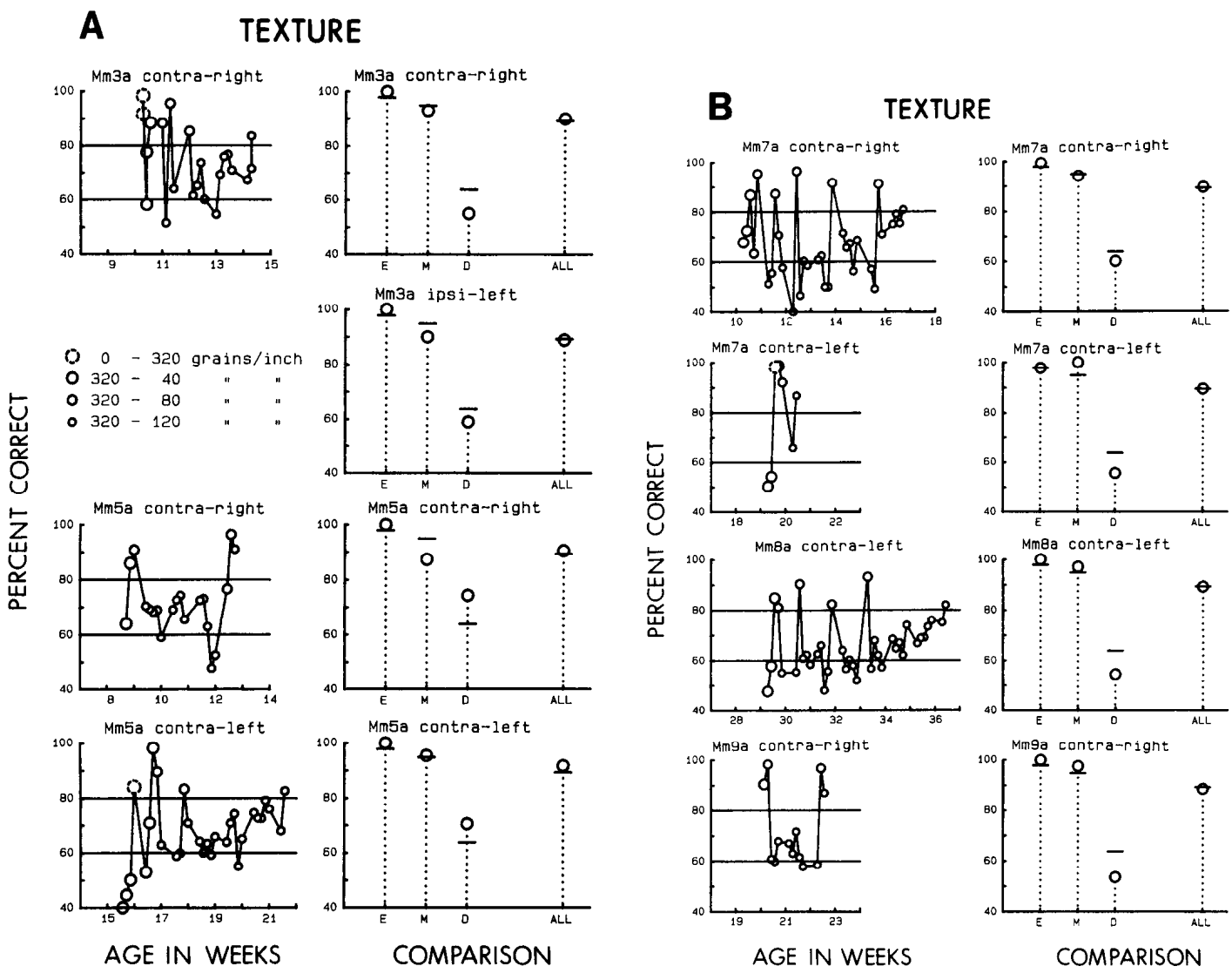


Figure 6. *A*, Acquisition curves and "alls" plots for Mm3a and Mm5a for texture discriminations (see Fig. 8*B* for further discussion). *B*, Acquisition curves and "alls" plots for Mm7a, Mm8a, and Mm9a for texture discriminations (see Fig. 3 for further discussion).

acquire the most difficult texture comparison of 320–120 grains/in., and made errors overall for the 6-texture comparisons (Mann-Whitney test; $p \leq 0.05$). No significant differences were found between performance scores for any of the "alls" comparisons. No significant differences were found between SII- and SI-lesioned animals on the texture acquisition tasks (see Table 8). On the texture "alls" tasks, SII-lesioned animals were significantly better on the 320–80 grains/in. comparison and significantly worse on the 320–120 grains/in. comparison than the SI-lesioned infants (Carlson, 1984c). SII lesions clearly retard the acquisition of texture discriminations in infant macaques, suggesting a role for SII in the mediation of texture in the infant. Texture discrimination capacities recover to normal levels after several weeks of training. The levels of acquisition impairment on difficult texture tasks, or of slight depression of "alls" performance on the difficult comparison, were trivial compared to the severe, irreversible impairment seen following SII lesions in adult macaques (Murray and Mishkin, 1984). By contrast, SII-lesioned infants are superior to normal and SI-lesioned infants on size acquisition, suggesting that SII does not mediate size capacity in the infant, although SII removals in the adult macaque result in severe deficits in size discrimination (Murray and Mishkin, 1984).

SI–SII lesions in infants and adults

For the combined SI–SII lesion group, there were 3 types of combination lesion, which will be discussed separately: simultaneous SI–SII, sequential SII–SI, and sequential SI–SII. Within each of these 3 groups, some animals were operated on as infants and others as adults. The different-aged animals will be discussed separately for purposes of analysis.

Simultaneous SI–SII lesions in infants

In animal Mm4a (L) (Fig. 7), the SI lesion extended deep into areas 3b and 3a for most of the mediolateral extent of the hand area, except for a small lateral portion that may have spared some of the digit 1 area. The SII lesion blanketed all of the outer face of the LS and spread over the adjoining inferior parietal lobule, as shown in Figure 7. This lesion destroyed all of SII but a small wedge of tissue located anteriorly near the superior limiting sulcus. This was the largest of the SII lesions seen in

Table 5. Texture comparison by SII-lesioned infants

Animal	Age (weeks)	Hand	Texture comparison: A. Errors to criterion (and age in weeks) during acquisition 320–40 grains/in.	320–80 grains/in.	320–120 grains/in.	Total on 6 tasks	B. Percentage correct on individual comparisons on the "alls" task Age (weeks)	320–40 grains/in.	320–80 grains/in.	320–120 grains/in.	Mean of all 6 comparisons
Mean normal infant		1st:	77.2 (20.3)	26.5 (20.9)	92.2 (16.3)	(249.5)	30.8	97.8	94.8	63.7	(89.2)
		2nd:	23.4 (36.0)				36.3	95.2	84.3	57.4	(83.6)
Mm3a	7.0	C-R	59 (10.4)	11 (11.0)	424 (11.1)	(524)	15.3	100.0	93.0	55.0	(90.0)
		I-L	37 (16.4)				16.6	100.0	90.0	59.0	(88.3)
Mm5a	5.6	C-R	96 (8.7)	14 (9.0)	954 (9.4)	(1126)	14.4	100.0	87.2	74.2	(90.4)
		C-L	322 (15.6)	11 (16.9)	810 (17.0)	(1155)	22.4	100.0	95.4	70.5	(91.6)
Mm7a	7.3	C-R	87 (10.3)	59 (10.7)	1578 (11.3)	(1737)	17.6	99.4	94.2	60.0	(89.6)
		C-L	159 (19.3)	15 (19.9)	87 (20.3)	(270)	21.3	97.8	100.0	55.6	(89.3)
Mm8a	7.3	C-L	76 (29.3)	14 (29.7)	1100 (29.9)	(1207)	43.9	100.0	97.0	54.5	(89.1)
Mm9a	7.4	C-R	14 (20.1)	2 (20.3)	485 (20.4)	(510)	23.4	100.0	97.5	53.6	(88.5)
Mean SII infants			116.1 (16.2)	18 (16.8)	776.9 (17.1)[a]	(932.7)[a]	22.6	99.6	94.9	60.5	(89.8)
Mean SI lesions			196.8 (16.8)	79.8 (18.0)	270 (185)	(781.0)	21.2	94.3	82.2[b]	74.8[b]	(84.5)

[a] Significant at $p \leq 0.05$ from normal infants.

[b] Significant at $p \leq 0.05$ from SI-lesioned infants.

either infants or adults and was equally distributed through the outer bank and the SII region posterior to the insula.

In animal Mm6a (R), the SI lesion spared some 3b medially, possibly in the arm area. In section 15 of Figure 7, some of area 3b in the hand area remains but areas 1 and 2 are gone. The 3b tissue remaining laterally in sections 19 and 21 most certainly were in the face area. The SII lesion was of average size, with damage largely restricted to that part of SII posterior to the insula. The lesion in the upper bank of LS extended to the fundus for more than 2 mm posterior to the insula.

The SI removal in Mm8a (L) was a partial lesion, restricted to the area 3b hand zone. The SII lesion was of average size and almost exclusively in the outer bank, with no involvement of SII posterior to the insula.

The SI lesion in Mm9a (R) was largely restricted to the hand zone in areas 1 and 2. Most of area 3b was spared as intended; a portion of area 2 in the IPS was ablated, as was the lateral third of the IPS. The SII lesion was very small and restricted to the outer bank (Fig. 4).

Sequential SII–SI lesions in infants and young animals

The SII lesion in Mm3b was described in an earlier section (see SI lesions, Fig. 3) as being large and including inner and outer banks and posterior SII. The SI lesion in Mm3b (in Table 1) went as far laterally as the face area in 3b and deep into the IPS in area 2. The SI lesion may have touched area 5 medially but not laterally. SI lesions were made bilaterally to determine the effects of an SI lesion alone at this age, for comparison with the SI–SII lesion.

The SII lesion in the left hemisphere of Mm5a (R) was described earlier (see above). This average-sized SII lesion was primarily in the area of SII posterior to the insula. The SI lesion was large throughout the hand area, extending deep into 3b and 3a and into area 2 in the IPS through the hand area.

Simultaneous SI–SII and sequential SI–SII lesions in older animals

In the right hemisphere of Mm1c at 208.7 weeks, the only tissue removed posterior to the insula was in area 7. Half the damage was within the inner bank (with some sparing near the posterior pole); the remaining damage was in the outer bank. No portion of SII lying near or just posterior to the insula was lesioned (see Fig. 2). The SI lesion was large but with some sparing laterally.

Mm1d received a left SII lesion of average size at 228.0 weeks; it was maximal along the outer and inner banks of the segment of LS that lies between the lateral tip of the CS and the anterior end of the IPS. The lesion was limited to the more superficial half of the inner face; this spared SII near the superior limiting sulcus. All of SII that lay along the upper bank of LS was spared, starting from the posterior pole of the insula. The SI lesion removed the entire hand area in 3b, 1, and 2.

At 109.3 weeks, Mm2b (L) received an extensive SI–SII lesion in the left hemisphere (Fig. 2). In SI, the lesion extended to area 3a and, in some regions, even into area 4. Posteriorly, the lesion was into the IPS but failed to reach the fundus. In the SII region, the lesion removed all of the parietal operculum bordering the posterior pole of the insula, and spread forward along the inner face of LS. This lesion destroyed most of the middle of SII, spared some of SII anteriorly on the outer bank of LS, and missed SII along the fundus of LS posteriorly.

At 144.6 weeks, Mm2c (R) received an additional SII lesion following an earlier SI lesion in the right hemisphere. The SII lesion removed nearly all of SII anteriorly on the inner and outer banks of LS and, except near the fundus, much of SII

Table 6. Size comparison by SI-SII-lesioned infants and juveniles

			Size comparison: A. Errors to criterion (and age in weeks) during acquisition				B. Percentage correct on individual comparisons on the "alls" task				
Animal	Age (weeks)	Hand	7–18 mm	7–12 mm	7–9 mm	Total on 6 tasks	Age (weeks)	7–18 mm	7–12 mm	7–9 mm	Mean of all 6 comparisons
Mean normal		1st:	7.8 (15.2)	5.2 (15.6)	54 (15.9)	(115.1)	19.4	98.4	95.4	76.7	(92.3)
infant		2nd:	25.0 (21.9)				21.4	98.5	95.4	69.5	(90.2)
SI–SII simultaneous											
Mm4a	7.6	C-R	9 (29.4)	36 (29.6)	358 (30.1)	(588)	33.6	100.0	93.0	70.0	(90.0)
Mm6a	7.4	C-L	33 (25.7)	3 (26.1)	18 (26.3)	(59)	27.4	100.0	95.6	59.6	(91.6)
Mm8a	8.3	C-R	48 (40.3)	8 (40.6)	46 (40.7)	(124)	37.3	96.2	95.2	71.8	(90.9)
Mm9a	7.4	C-L	80 (35.1)	6 (35.4)	8 (35.6)	(107)	36.4	100.0	95.1	77.0	(93.8)
Mm2b	109.3		N.T.				N.T.				
SII–SI sequential											
Mm3b	25.3	C-R	4 (56.4)	8 (56.6)	252 (57.1)	(294)	59.3	100.0	93.0	59.0	(88.7)
		I-L	37 (49.1)	19 (49.4)	803 (49.6)	(927)	55.3	100.0	92.0	72.0	(90.8)
Mm5b	28.4	C-R	6 (44.0)	16 (44.4)	378 (44.7)	(434)	47.7	100.0	87.0	59.0	(86.7)
		I-L					48.9	100.0	99.0	73.0	(94.7)
Mean SI–SII											
infants			30 (38.5)[a]	12.8 (38.8)[a,b]	176.7 (39.1)[b]	(266)[b]	40.3	99.4	93.2[b]	66.1[b]	(90.2)[b]
SI–SII sequential											
Mm2c	144.6	C-L	19 (153.7)	92 (153.9)	941 (154.6)[c]	(1052)	N.T.				
Mm1d	228.0	C-R	58 (237.6)	50 (237.9)	952 (238.1)[c]	(1060)[d]	243.6	90.8	69.7	66.3	(77.7)
		I-L	17 (234.6)	7 (234.7)	202 (234.9)[c]	(226)[d]	236.6	94.8	87.5	57.3	(79.2)
Mean SI–SII											
adults			31.3 (208.6)	49.7 (208.8)	698.3 (209.2)	(779.3)	240.1	92.8	78.6	61.8	(78.5)

[a] Significant at $p \leq 0.05$ from normal infants.
[b] Significant at $p \leq 0.05$ from SII-lesioned infants.
[c] Failed to reach criterion.
[d] Tested only on first 3 tasks.
N.T., not tested.

posterior to the insula on the upper bank of LS. The Ri area behind SII was partially damaged, as was area 7 medially.

Size discrimination

SI–SII-lesioned infants. In all cases of SI–SII-lesioned infants (Mm4a, Mm6a, Mm8a, Mm9a), acquisition of the size comparisons was equivalent to that of normal infants or slightly superior (Mm6a) (Fig. 8*A*). The latter possibility was due to the fact that Mm6a was older than normal infants at the time of testing. Performance on the size "alls" task overall comparisons for all cases was also equivalent to that of normal infants. In 3 out of 4 cases, however, performance was slightly below normal on the most difficult size comparison (7–9 mm).

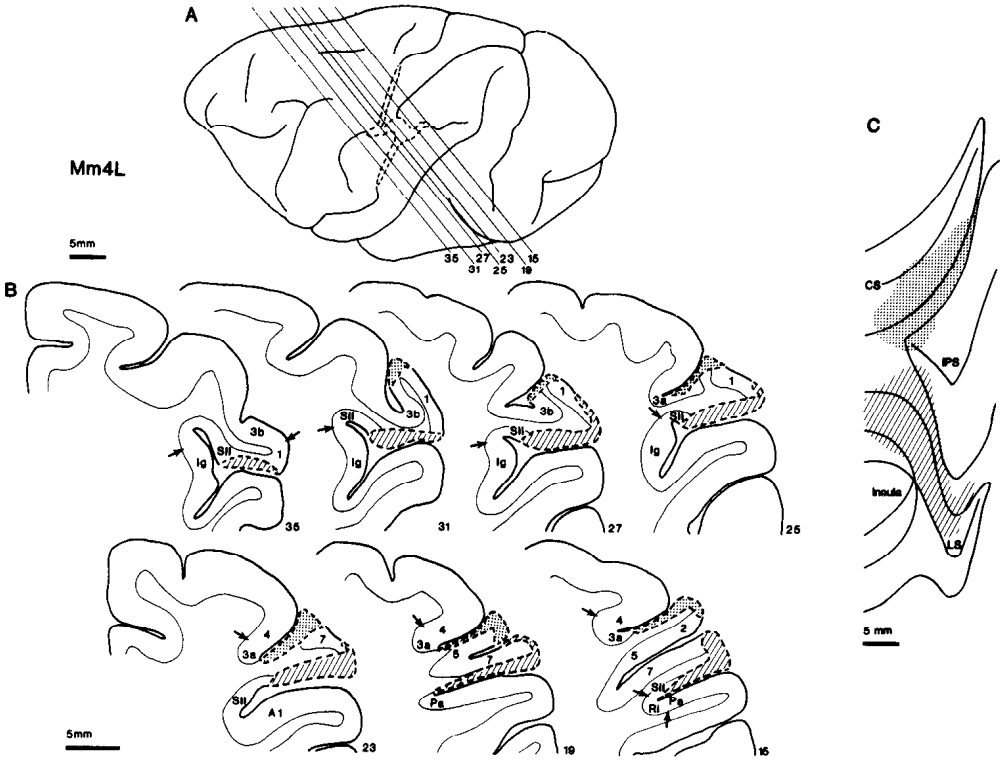
SII–SI-lesioned older infants. The 2 groups of infants [SI–SII simultaneous (Fig. 8*A*) and SII–SI sequential (Fig. 8*B*)] were combined for statistical comparisons (Table 6). As compared to the scores of normal infants, the error scores of infants with combined SI–SII lesions on the easy (7–18 mm) and moderate (7–12 mm) difficulty tasks were significantly higher, but no significant differences were seen on size "alls" tasks. When these animals were compared to SI-lesioned infants, no significant differences were seen during size acquisition or size "alls." However, compared to the scores of SII-lesioned infants, error scores on acquisition and performance on 7–9 mm, 7–12 mm, and overall tasks were significantly inflated (see Table 8).

SI–SII-lesioned adult animals. For both adult SI–SII-lesioned animals, impairment was seen on acquisition of difficult size comparisons, and this was related to poor overall performance on the size "alls" task (Fig. 8*C*).

Texture discrimination

SI–SII-lesioned infants. Although there was some evidence of discrimination impairment in the SI–SII-lesioned infants during acquisition of size comparisons, performance on the size "alls" tasks was not particularly depressed. The simultaneous removal of SI and SII had a devastating effect on both texture acquisition and texture "alls" (Fig. 9*A*). The most difficult tasks were not learned to criterion by any of the animals (Fig. 9*A*). Performance on the texture "alls" task was also impaired such that below-normal scores were obtained by nearly every animal, except on the easiest comparisons (Fig. 9*A*; Mm6a, Mm9a). In one case (Mm4a), performance was not even tested because criterion was never reached on the moderate-level task during acquisition (Table 7).

SII–SI-lesioned older infants. Mm3b received bilateral SI lesions at 25.3 weeks, following an earlier partial SII lesion in the left hemisphere. When tested on the right hand following the second-stage lesions, errors to criterion on all acquisition tasks were elevated compared to the performance of normal infants and of the sham-operate tested at the same age (see Table 7, A



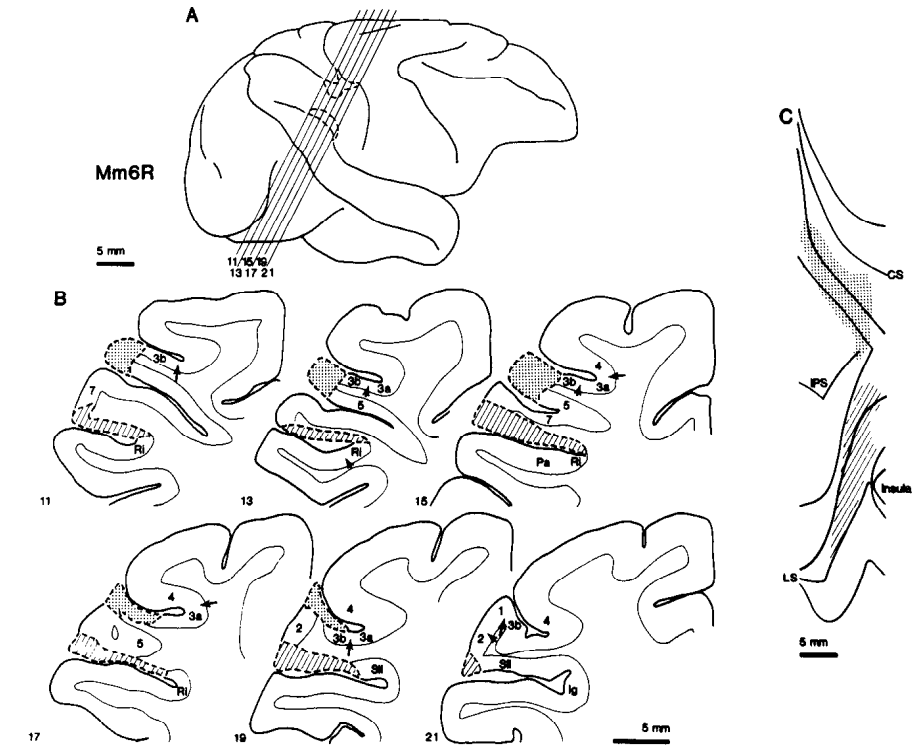
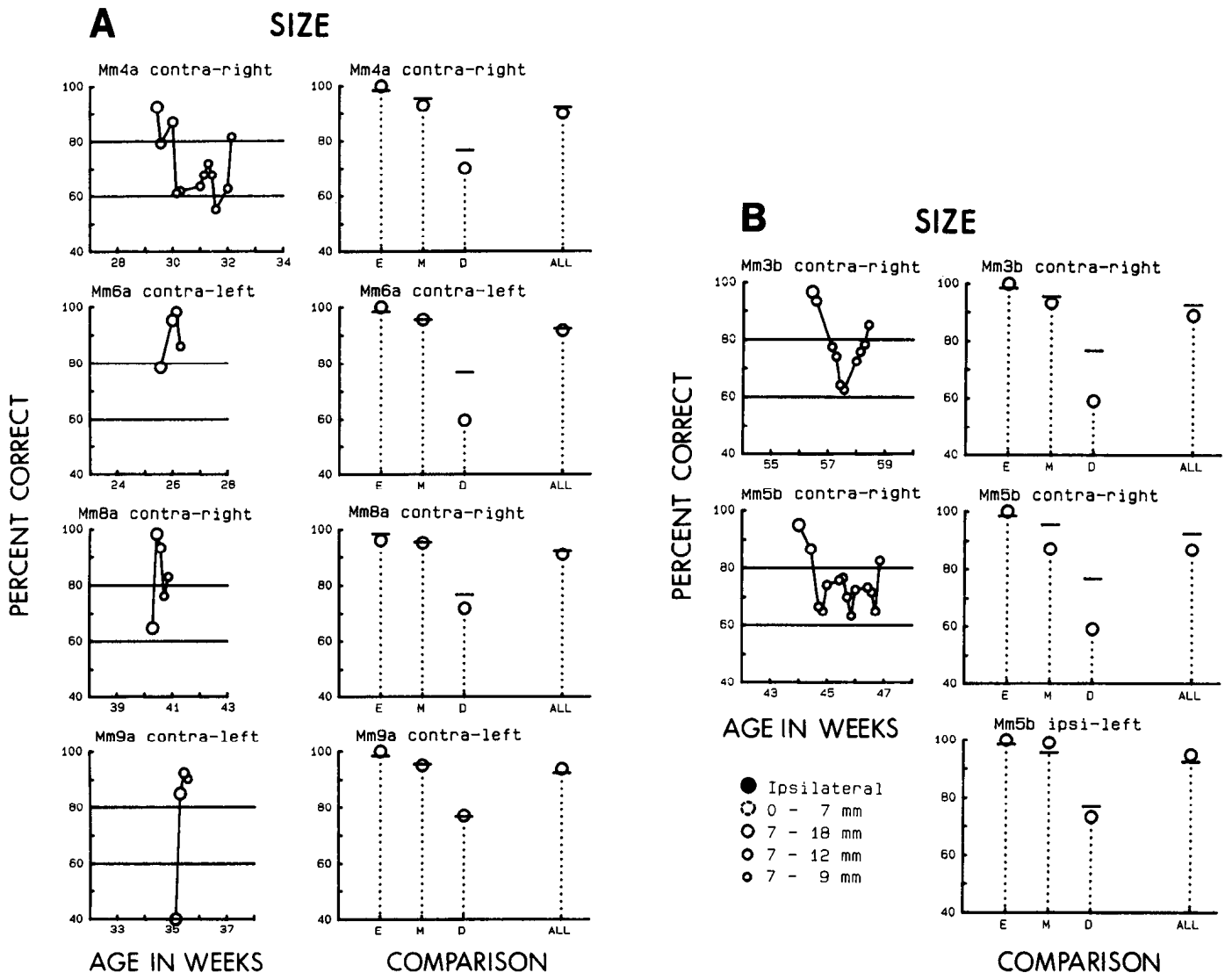


Figure 7. Localization of lesions in Mm4L and Mm6R. See Figure 2 for further discussion.

and Fig. 9*B*). However, if acquisition error scores for the right hand were compared to those for the left hand (contralateral to an SI-only lesion), the SI–SII lesion produced less of a deficit. Previous error scores on the right hand following the SII–only lesion were half of the total seen following the later bilateral SI lesions.

When the later "alls" performance on the right hand was compared to the performance following the SII-alone lesion, it



was lower overall (Table 7, B). Yet performance on the texture "alls" task was the same for the right and left hands, with each about 6% lower over all 6 comparisons, suggesting that the earlier, incomplete SII lesion did not augment the deficits caused by the later SI removals.

Mm5b also received an SI lesion at about 6 months of age, and showed a major deficit on even the easiest of texture tasks. Error scores were elevated for the 320–40 grains/in. tasks and the moderate-level task; 320–80 grains/in. was never mastered after 21 sessions. The texture "alls" task was not run because of the severity of disturbance found during acquisition (see Fig. 9*B*). The level of impairment after the later SI lesion exceeded that found after the earlier bilateral SII lesions in this animal or after the bilateral SI lesions made in Mm3b.

The data from SI–SII simultaneous and SII–SI sequential lesion groups were combined for statistical comparisons (Table 8). The errors to criterion during texture acquisition for the combined SI–SII lesion groups were significantly higher than those of normal infants on all levels of difficulty and over all 6 comparisons in the texture "alls" task. When the SI–SII-lesioned infants were compared to SI-lesioned infants, the only significant difference was in the difficult (320–120 grains/in.) comparison on the texture "alls" task. As compared to SII-lesioned infants, SI–SII-lesioned infants made significantly more errors on the easiest and moderate-level texture tasks and performance was significantly lower over all 6 comparisons in the texture "alls" task.

SI–SII-lesioned adults. When animal Mm2c was run on texture acquisition, criterion was not attained even after 20 sessions on the easiest comparison, interspersed with testing on 320 versus no handle as a correction procedure (Fig. 9*C*). The texture "alls" task was not given because of the severity of this deficit.

Beginning at 209.9 weeks, Mm1c was tested on texture with the left hand (contralateral to an SII lesion in the right hemisphere at 208.7 weeks, combined with an SI lesion at 7.6 weeks). Given what appears to be significant damage to both SI and SII in the right hemisphere, this animal did very well on all texture comparisons, reaching criterion on the difficult discrimination in one session. However, on the texture "alls" task, performance was below normal on the left hand contralateral to the recent SII lesion, and on the right hand contralateral to a late SI-alone lesion. When an SII lesion was made in the left hemisphere at 228 weeks and tested again with the right hand on texture tasks, texture acquisition was again remarkably normal. Yet performance on the texture "alls" tasks was, overall, lower than when tested prior to the later SII lesion except on the difficult com-

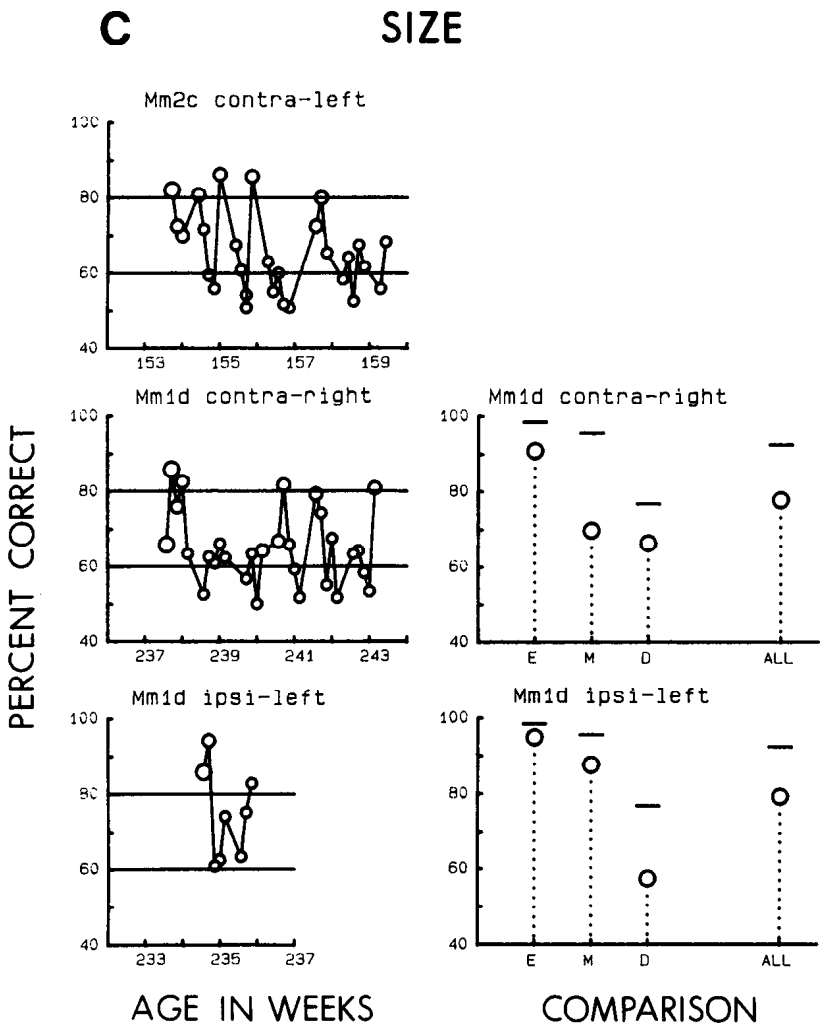


Figure 8. *A,* Acquisition curves and "alls" plots for Mm4a, Mm6a, Mm8a, and Mm9a on size discriminations (see Fig. 8*B* for further discussion). *B,* Acquisition curves and "alls" plots for Mm3b and Mm5b on size discriminations. Conventions used in acquisition curves in all figures are as follows. The filled *circles* represent performance on the ipsilateral hand on the easiest comparison and the *dashed circles* represent the comparison of the correct handle to *no* handle as a correction procedure. Percentage correct on the size "alls" task is plotted for the easy (*E*), moderate (*M*), and difficult (*D*) comparison and for the average of all 6 comparisons (*ALL*). The *horizontal lines* above E, M, D, and ALL represent the average performance level for normal infants (Carlson, 1984a). *C,* Acquisition curves and "alls" plots for Mm2c and Mm1d on size discriminations (see Fig. 5 for further discussion).

parison, on which performance was above the level of normal infants and at the level of the sham-operated animal. This animal had been tested on repeated occasions with each hand for 5 years, but the level of performance was still unexpected, given the major lesions in SI and SII received during this period, although both repeated testing and sequential—in contrast to simultaneous—lesions are commonly thought to ameliorate the effects of cortical removals.

When the remaining somatic sensory projection area (SII in the left hemisphere) was removed in animal Mm1d at 228 weeks, reacquisition with the contralateral hand was even better than in the sham-operated animal, and much better than in normal infants or after any other SI–SII combination lesion in other animals. The texture "alls" performance was superior to that of normal infants and similar to that of the sham-operate. The average performance over all 6 comparisons for the right hand was below that of normal infants and the sham-operate, and lower than for the left hand following this fourth lesion.

Cell degeneration in the thalamus

Profound retrograde cell degeneration was present in the medial half of the VPL in all cases involving combined lesions of SI and SII (Fig. 10, *A, B*; Mm4L). A similar pattern of degeneration was noted in those cases where only SI had been lesioned or where the sequentially placed juvenile lesion in the LS, after an infant SI lesion, missed SII (Fig. 10*E*; Mm1). Generally, few surviving cells were found in the central zone of maximum gliosis in the SI–SII lesion cases (Fig. 10*B*, Table 9). The average number of cells/625 μm^2 in VPL was greatest for the cases (Mm1, Mm8, Mm9) where the lesion in SII was not complete. Frequently, islands of surviving cells were seen within gliotic portions of VPL in these cases (Fig. 10*F*). No gliotic zones were detected in VPL in those cases involving only SII lesions (Fig. 11*A*; Mm7), even where the SII lesion was massive. For example, the largest lesion in the LS was made in Mm4, in which nearly all of SII was destroyed. As shown in Figure 11*A*, degeneration was obvious in VPL medial, i.e., the distal hand region, while the rest of the nucleus appeared normal. Cell counts, however, suggested that there was a decline of more than 20% in the forelimb portion of VPL when SII was adequately lesioned. Failure to see gliosis in VPL may have been due to the more scattered distribution of SII-projecting cells, as compared to those surviving cells in cases where only SI was ablated.

Additional changes were seen in the most inferior portions of the ventral nucleus in the region of VPI in cases with damage to SII. Massive gliosis was not seen, as some cells were always

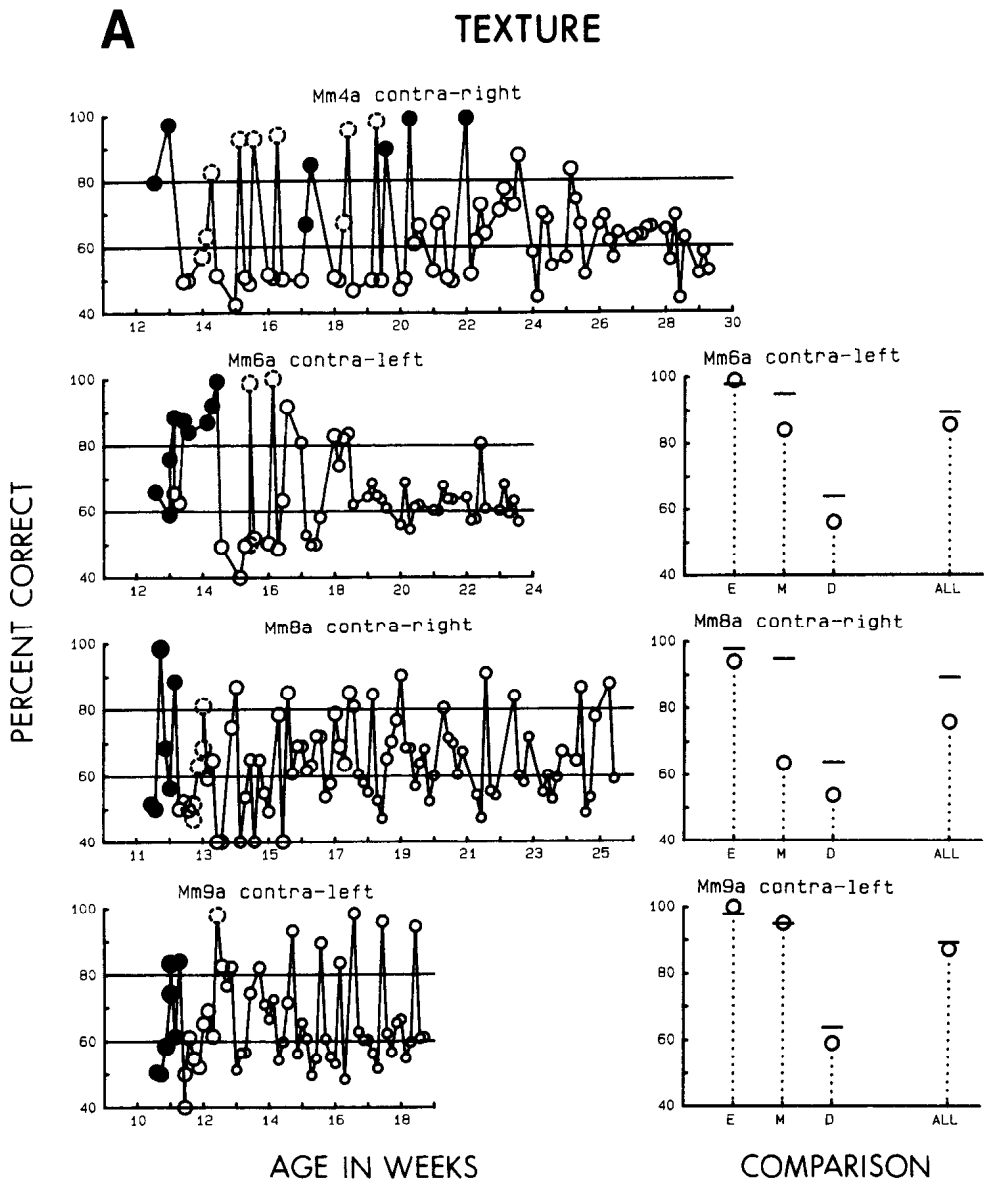


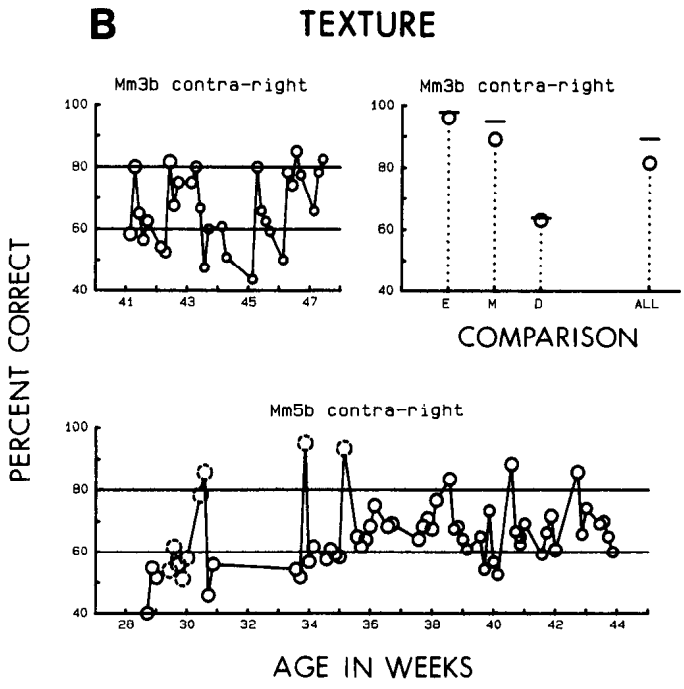
Figure 9. *A,* Acquisition curves and "alls" plots for Mm4a, Mm6a, Mm8a, and Mm9a on texture discriminations (see Fig. 8*B* for further discussion). *B,* Acquisition curves and "alls" plots for Mm3b and Mm5b on texture discriminations (see Fig. 8*B* for further discussion). *C,* Acquisition curves and "alls" plots for Mm2c, Mm1c, and Mm1d on texture discriminations (see Fig. 8*B* for further discussion).

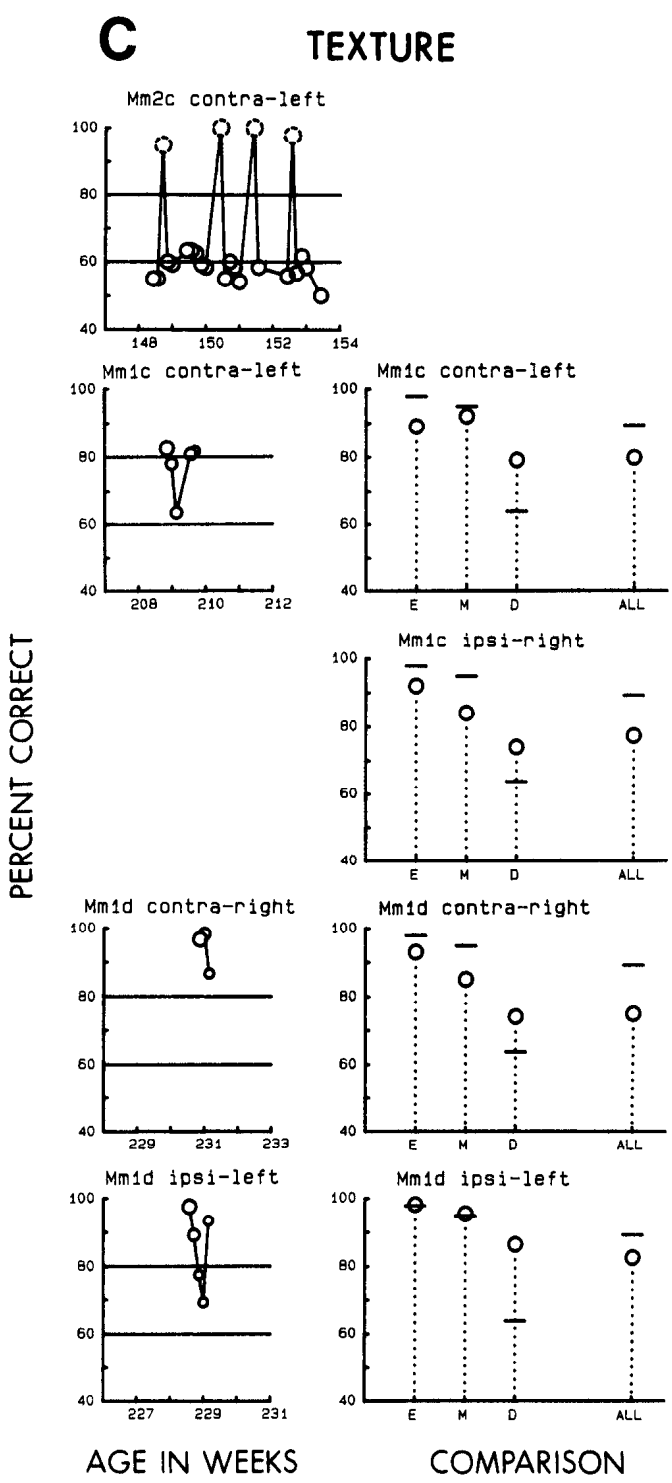
present in VPI (Figs. 10, *C, D*; 11, *A, B*) even when SII was extensively ablated. Cell counts were confidently made in VPI only in cases where the thalamus was sectioned coronally because of difficulties in defining the borders of this region. Comparisons of cell counts in VPI between normal and SII-lesioned cases showed a decline of 40–45% on average. In matched comparisons from the same animals, the differences were 63% in Mm6, in which no lesion was present in the left hemisphere and a large SII lesion was made on the right, and 46% in Mm9, in which a small lesion missed nearly all of the SII forelimb on the left and a moderate SII lesion was made on the right. In Mm7, where SII was bilaterally ablated, the counts in VPI were comparable (Table 9).

Cell degeneration was also seen in VPM in cases where the face area of SI was damaged when combined SI–SI lesions were too extensive. The anterior pulvinar nucleus (Pla) also showed striplike zones of cell degeneration in cases where the lesions invaded the lateral bank of IPS and the adjoining inferior parietal lobule.

Discussion

These studies of the effects on tactile discrimination tasks of ablating SI, SII, or SI and SII in different-aged macaques have shown that the recovery of tactile function observed in infants may be mediated within the remaining somatic sensory areas in the hemisphere with the initial lesion. Infant macaques readily learned to make size and texture discrimination with the hands contralateral to SI lesions made at 1–2 months of age (Carlson, 1984b); this recovery was minimally disrupted subsequent to a later lesion of SI in the opposite hemisphere. Infants with SII lesions also were not permanently deficient on tactile discrimination tasks. Surprisingly, removal of SII in infants was associated with improved acquisition on size comparisons; this finding may, however, have been confounded, as these animals were older than the normal infants at the time of testing. However, infants with SII lesions were retarded in learning texture discrimination, but performed normally on the texture "alls" task at the completion of acquisition training. The simultaneous





or sequential removal of SI and SII did not influence either size acquisition or size "alls" performance, but produced a severe, irreversible deficit in texture acquisition and performance. These results from combined SI–SII lesions indicate that in the infant either of these areas has the potential to mediate tactile learning and performance; this contrasts with the situation in the adult macaque, in which both SI and SII must be intact for normal tactile function (Ridley and Ettlinger, 1976, 1978; Murray and Mishkin, 1984).

The results raise questions concerning possible interactions between SI and SII when learning and performing tactile discriminations and the mechanisms underlying the adaptability of infant nervous systems to damage, as compared to the inflexibility seen in older animals with the same types of lesions. It is also relevant to consider the significance of the behavioral deficits for categories of tactile input to cortex, along with some of the behavior variables that may have influenced the results.

Task and experimental design variables

In these studies, acquisition tasks measured original learning of size and texture discriminations, and "alls" tasks evaluated performance thresholds. In earlier studies, acquisition was measured at only one level of difficulty, and ascending and descending series of threshold tasks were run after the entire acquisition series, for size and texture comparisons (Semmes and Mishkin, 1965; Randolph and Semmes, 1974; Murray and Mishkin, 1984). In this study, as in some previous studies (Carlson, 1981, 1984a–c), acquisition was begun with an easy task, followed by mod-

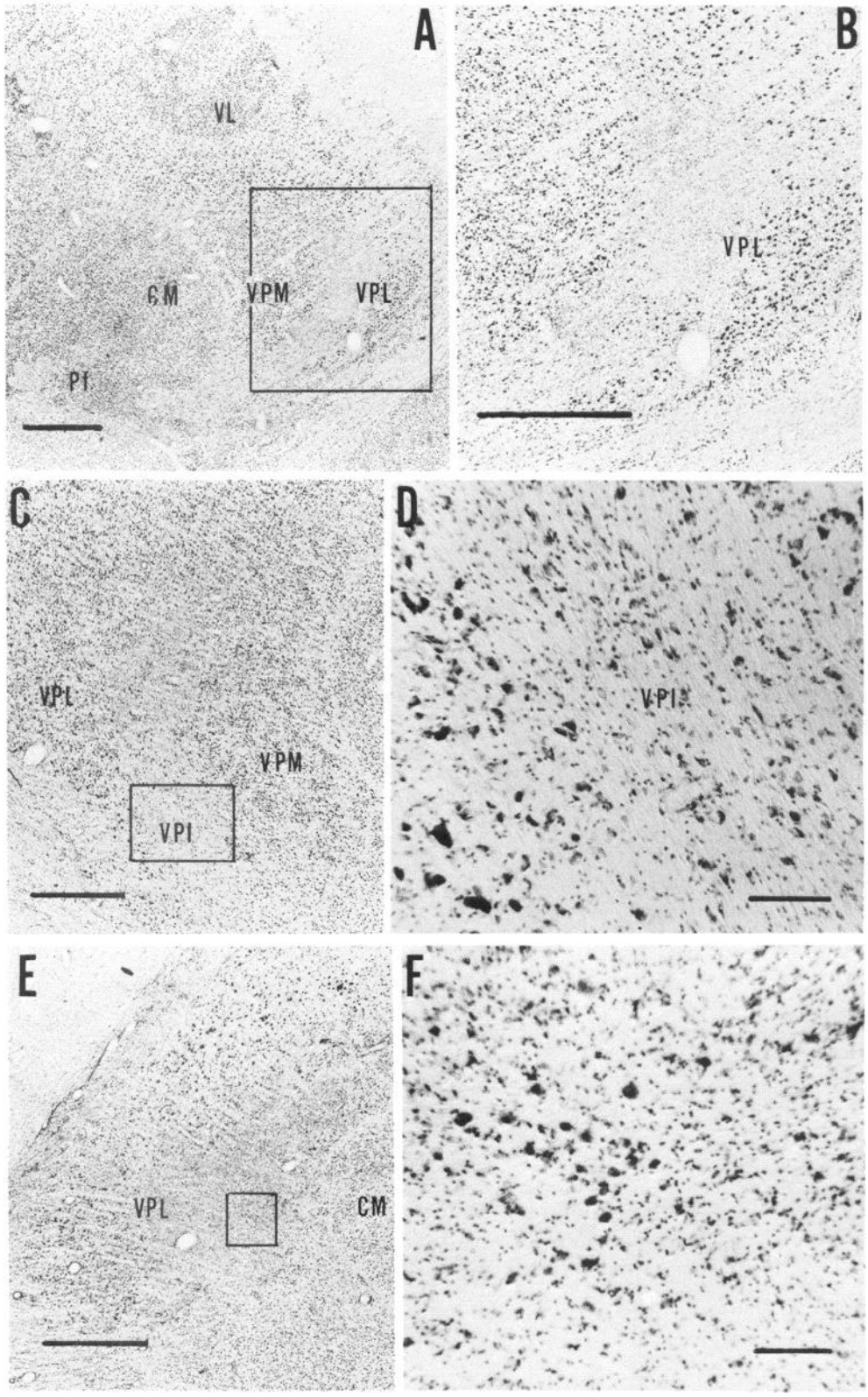


Figure 10. Photographs showing cell patterns in 50 μm frozen sections stained with thionin in selected ventroposterior nuclei following lesions to SI and/or SII. Horizontal section showing (*A*) low-power and (*B*) high-power appearance of gliotic zone in VPL of Mm4. Region enclosed by *box in A* shown at higher power in *B*. *C, D,* Coronal section through *VPL* and *VPI* in Mm6. Region of VPI enclosed by *box in C* enlarged in *D*. Note small, poorly stained cells of VPI in the center of photograph in *D*; darkly stained, larger cells in *lower left corner* are from VPL. *E, F,* Horizontal section through gliotic zone in *VPL* in Mm1. Note the small clusters of surviving cells in VPL. One of these is shown enlarged in *F*. Scale: 1 mm (*A, B, E*); 0.5 mm (*C*); 0.1 mm (*D, F*).

erate and difficult tasks as 80% criterion was reached on the previous tasks. This combined acquisition–threshold method gave an indication of general learning problems on the initial tasks and of the sensory-specific deficits in the subsequent, more difficult, tasks. A second, tactile performance measure, the "alls" test, was developed owing to the problems in interpreting errors to criterion on multiple acquisition tasks. In the "alls" test, all 6 acquisition comparisons were presented together. The animal was required to make a difficult relative judgment about each of the pairs in sequence. When all 6 pairs were presented

Table 7. Texture comparison by SI-SII-lesioned infants and juveniles

Animal	Age (weeks)	Hand	Texture comparison: A. Errors to criterion (and age in weeks) during acquisition: 320–40 grains/in.	320–80 grains/in.	320–120 grains/in.	Total on 6 tasks	B. Percentage correct on individual comparisons on the "alls" task: Age (weeks)	320–40 grains/in.	320–80 grains/in.	320–120 grains/in.	Mean of all 6 comparisons
Mean normal		1st:	77.2 (20.3)	26.5 (20.9)	92.2 (16.3)	(249.5)	30.8	97.8	94.8	63.7	(89.2)
infant		2nd:	23.4 (36.0)				36.3	95.2	84.3	57.4	(83.6)
SI–SII simultaneous											
Mm4a	7.6	C-R	2639 (13.4)	1190 (24.0)[a]		(3829)		N.T.			
Mm6a	7.4	C-L	306 (12.6)	23 (17.0)	2168 (17.2)[a]	(2630)	25.6	99.0	84.0	56.0	(85.3)
Mm8a	7.3	C-L	376 (11.3)	796 (14.1)	1757 (17.7)[a]	(3115)	27.3	94.1	63.5	53.7	(75.7)
Mm9a	7.4	C-L	407 (10.0)	66 (10.1)	2179 (13.0)[a]	(2682)	19.3	100.0	95.0	59.0	(87.2)
SII–SI sequential											
Mm3b	25.3	C-R	74 (41.1)	366 (41.4)	611 (43.4)	(1102)	210.9	96.0	89.0	63.0	(81.5)
Mm5b	28.4	C-R	1098 (30.7)	884 (40.4)[b]		(1982)		N.T.			
Mean SI–SII infants			837.5 (19.9)[b,c]	543.2 (24.5)[b,d]	1678.5 (22.82)[b]	(2374.5)[b]	—	97.3	82.9	57.9[c]	(82.4)[b,d]
SI–SII sequential											
Mm2c	144.7	C-L	949 (148.4)[c]					N.T.			
Mm1c	208.7	C-L	21 (208.9)	84 (209.0)	22 (209.7)	(227)	210.9	89.0	92.0	79.0	(79.7)
		I-R					212.0	92.0	84.0	74.0	(77.3)
Mm1d	228.1	I-L	3 (228.6)	13 (228.7)	72 (228.9)	(88)	229.6	98.2	95.5	86.4	(82.5)
		C-R	2 (230.9)	2 (231.0)	16 (231.2)	(22)	232.6	93.0	85.0	74.0	(74.8)

[a] Failed to reach criterion.
[b] Significant at $p \leq 0.05$ from normal infants.
[c] Significant at $p \leq 0.05$ from SI-lesioned infants.
[d] Significant at $p \leq 0.05$ from SII-lesioned infants.
N.T., not tested.

together in a single session, it was even more important to compare the handles in a pair, as a handle that was correct on the last trial could be incorrect on the next. Performance on the difficult-level comparisons actually drops from that measured on acquisition with that same pair, but performance is not influenced as much by repeated testing as is acquisition. The "alls" task makes a good repeated measure in the case of sequential lesions, in calculating the long-term effects of lesions, or in comparing the present level of discrimination capacity after different previous histories of acquisition. For this study, in which ani-

Table 8. Statistical comparison of lesion groups based on Mann-Whitney tests (percent level of significance)

	SII				SI–SII			
	Size		Texture		Size		Texture	
	Acq.	Perf.	Acq.	Perf.	Acq.	Perf.	Acq.	Perf.
Normal								
1–4	NS	NS	NS	NS	0.04↑	NS	0.004↑	NS
1–3	NS	NS	NS	NS	0.05↑	NS	0.01↑	NS
1–2	NS	NS	0.01↑	NS	NS	NS	0.02↑	NS
Sum	0.05↓	NS	0.03↑	NS	NS	NS	0.03↑	0.03↓
Total SI								
1–4	NS		NS	NS	NS		NS	NS
1–3	0.05↓	NS	NS	0.03↑	NS	NS	NS	NS
1–2	0.03↓	0.01↑	NS	0.05↓	NS	NS	NS	0.03↓
Sum	0.04↓	NS	NS	NS	NS	NS	NS	NS
SII								
1–4					NS	NS	0.04↑	NS
1–3					0.02↑	0.03↓	0.01↑	NS
1–2					0.02↑	0.03↓	NS	NS
Sum					0.01↑	0.03↓	NS	0.01↓

NS, not significant.

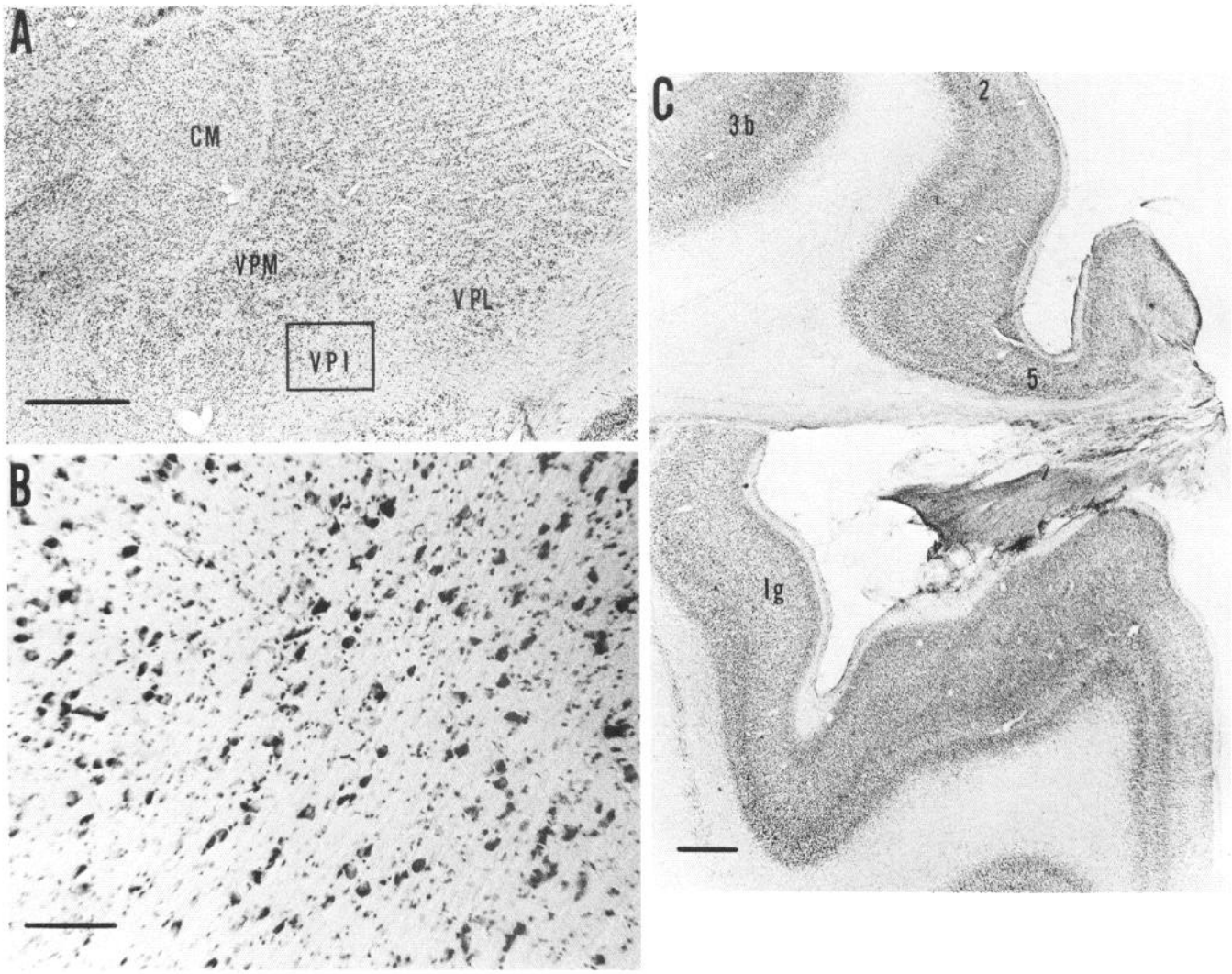


Figure 11. Photographs of thionin-stained, 50 μm frozen sections from Mm7R. *A,* Low-power view shows absence of gliosis in VPL or VPI following an SII lesion. *B,* Enlarged view of portion of VPI enclosed in *box in A. C,* View of lesion along the parietal operculum that selectively invaded SII without disrupting white matter underlying areas 3b, 1, and 2. Scale: 1 mm (*A, C*); 0.1 mm (*B*).

mals received lesions at different ages and were tested multiple times on the same tasks, it is particularly important to consider the limitations or advantages of the acquisition and performance tasks. It was necessary to consider the order of task presentation in this study as normal infants improve in their ability to discriminate size with age, whereas their ability to discriminate texture is actually better in the early weeks and months of life (Carlson, 1984a). The superior performance of SII-lesioned infants on size tasks may relate to being tested late on those tasks, but it was the texture tasks that were most sensitive to the SII and SI–SII lesions in infants.

Both acquisition and performance scores may reflect the period of time between the surgery and the beginning of behavioral testing. Some of these animals were pretrained to pull the discrimination handles before surgery and could be tested within days of surgery so that the immediate consequences of the lesions could be determined. The early testing of most infants on texture tasks in this study may have contributed to some elevation of acquisition on the first task, but would not appear to have affected the last (and most difficult) or the later "alls" task. Our results suggest that the selective deficits on texture, but not size, tasks are due to a modality-specific impairment, rather than to their being the first tasks presented after surgery.

Although there is considerable evidence in the literature on brain lesions and behavior that sequential lesions are less detrimental to behavior than are simultaneous lesions (Finger, 1978), the degree of impairment in these studies relates more to the number, size, and cortical area damaged than to the order of lesions. Simultaneous and sequential removal of SI and SII results in severe impairment on texture tasks. The major finding that removal of the remaining somatic sensory area in a damaged hemisphere either reverses or prevents recovery of function after SI or SII lesions in infants is robust enough to be seen on acquisition and performance tasks, regardless of the delay in testing order and with 1- or 2-stage surgical procedures.

The role of SII in tactile discrimination

SII was a region critical to the recovery of texture discrimination capacities following an SI lesion. Does this finding reflect a normal function of SII that was isolated by the lesions, or a consequence of adaptations by the immature nervous system to injury? A number of observations suggest that SII contributes

to the ability to compare surface textures. First, Murray and Mishkin (1984) showed that lesions to SII in adult monkeys destroy texture discrimination on tasks similar to those used here. In addition, lesions to SII alone in infants caused protracted learning of texture, but not size, comparisons. This indicated that the acquisition of a forced-choice comparison was not disrupted, but that only the ability to compare objects with different surface textures was impaired. Furthermore, impairment on texture tasks was extreme when lesions to SI were sequentially or simultaneously combined with lesions of SII in infants or juveniles.

The completeness of the SII lesions through the hand area was difficult to evaluate, as there are no sulcal landmarks to indicate the location of the hand area. The hand representation in SII is generally believed to extend from several millimeters along the inner and outer bank of LS at the anteroposterior level of the posterior tip of the insula to a few millimeters posterior to the insula. The SII lesions in the present study were in 3 locations: equally distributed in the anterior and posterior portions of SII, primarily anterior, and primarily posterior. Lesions of SII alone in all 3 locations caused significant impairment during texture acquisition, usually followed by recovery when tested on "alls." An SII lesion in any location that was combined with an SI lesion, simultaneously or sequentially, led to severe, irreversible impairment on texture acquisition and "alls." We have not been able to determine whether there is a critical area or areas in SII that must be removed to produce impairment. Rather, it appears that even a minimal amount of damage to any single region—inner bank, outer bank, or SII posterior to the insula—can cause a deficit in texture discrimination, particularly in combination with an SI lesion. These findings are consistent with previous reports, in which the hand area of SII was shown to extend from a portion of the parietal operculum located anterior to the posterior tip of the insula to the upper bank of the LS, just posterior to the end of the insula (Friedman et al., 1980; Robinson and Burton, 1980a; Juliano et al., 1983).

Table 9. Mean[a] cell counts/625 μm^2

	No lesion	SI	SII	SI–SII
VPL				
Horizontal				
Mm1R				3.65
Mm1L				3.45
Mm2R				1.4
Mm2L				1.9
Mm3R		7.4		
Mm3L				3.45
Mm4L				1.8
Mm5L				2.9
Mm5R				
Coronal				
Mm6R				2.85
Mm6L	44.3			
Mm7R			36.5	
Mm7L			35.8	
Mm8R			31.1	
Mm8L				4
Mm9R				10.4
Mm9L			26.7	
VPI				
Coronal				
Mm6R				13/15
Mm6L	35.8			
Mm7R			19.8	
Mm7L			17.5	
Mm8R			16.55	
Mm8L				13.2
Mm9R				11.85
Mm9L			21.85	

[a] Mean based on 20 fields per case.

Modality representation in SII

The physiology of SII in primates is generally consistent with a role for this cortical area in normal tactile behavior, such as texture comparisons. The glabrous surface of each of the distal digits is represented with some degree of topographical segregation; cells in this portion of SII have small receptive fields, and adjacent serial sequences of cells provide an accurate map of the skin surfaces that are stimulated (Robinson and Burton, 1980a; Burton and Carlson, 1986). Consequently, SII has the capacity to resolve and localize stimuli on the fingertips. In SII, the predominant, and possibly the only response to cutaneous stimulation is to rapidly adapt (Robinson and Burton, 1980c; Sinclair and Burton, 1984; Burton, 1986). SII also contains neurons that, at least in the cat, have been shown to respond to the Pacinian corpuscle range of high-frequency vibrations (Ferrington and Rowe, 1981; Fisher et al., 1983). Recent assessments of the peripheral receptors that contribute to texture perception have suggested that rapidly adapting mechanoreceptors (Meissner type low-frequency and Pacinian type high-frequency) contribute to the ability to distinguish between textured surfaces (Johnson, 1983; Lamb, 1983; LaMotte and Whitehouse, 1986). Thus, the relative restriction of cells with rapidly adapting properties in SII would be suited for texture comparisons.

The role of SII in size distributions is more difficult to appreciate. Infants with SII lesions learn size comparisons. In adult macaques, Murray and Mishkin (1984) and Ridley and Ettlinger (1976, 1978) have, in contrast, shown severe deficits in size-comparison behavior after SII lesions. However, the presence of an SII lesion in an infant with a total SI lesion did not produce greater impairment in learning or performance on size tasks than had been seen with SI-only lesions in infants (Carlson, 1984b, c). Previously, the relearning of size after a partial SI lesion in infants was hypothesized to be through the spared regions of SI on the side of the lesion or through SI in the opposite hemisphere (Carlson, 1984b). On the basis of persistent recovery even after total SI lesions, SII or the opposite SI was suggested as the source for sparing (Carlson, 1984c). Here we have shown that the recovered discrimination of size in infants may be due to regions beyond areas 3b, 1, and 2 of SI, SII, or the opposite SI. The difference between adults and infants in the recovery of size discrimination after SII lesions may be due to the greater recovery capacities of infants, as has been reported with regard to other cortical systems (Kennard, 1942; Goldman, 1974). Additionally, however, the recovery in infants may reflect the fact that the potential for making size comparisons is more widely distributed to cortical areas not ablated. Potential sites may be area 3a, which was never completely undermined, area 5, which receives proprioceptive inputs and was also spared, or motor cortex, which receives input from a variety of peripheral mechanoreceptors (Lemon and Porter, 1978).

The unresolved issue is whether SII is involved with size discrimination behavior in a normal adult monkey, in addition to other areas that appear potentially capable of providing for this behavior in infants following SI–SII lesions. Studies of SII neurons have reported inconsistently on the presence of distinct proprioceptive inputs to this cortex in primates (Burton, 1986). As shown previously (Randolph and Semmes, 1974; Carlson, 1981), selective damage to area 2, which responds to stimulation of joint and muscle receptors (Powell and Mountcastle, 1959b; Burchfiel and Duffy, 1972; Iwamura et al., 1980), selectively interferes with size discriminations. The significance of information about finger movement and position that can be obtained from proprioceptive input would appear to be critical for the discrimination of objects of different size and shape. The lesions made by Murray and Mishkin (1984) may have been more extensive than were needed here to demonstrate alterations in texture discrimination behavior in infants, and it is possible that their reported alterations in size tasks may reflect this difference in lesion size. Note, however, that in Mm4, where the SII lesion was clearly more extensive posteriorly, there was a normal performance on size tasks, and the acquisition curve in this animal was delayed only to the extent seen previously with infant SI lesions. Consequently, assuming a role for some of the areas surrounding SII (area 7b and Ri) in size comparisons is overly speculative.

Another possible difference between infant and adult cases may be the degree of inadvertent damage to SI. The middle cerebral arterial supply to the hand area of SI exits the LS immediately over SII. In all of these experiments, we first isolated these arteries in cases where SI was to be spared. Preserving these vessels was more difficult when larger SII lesions were made. Circulation in this region was of concern in an earlier recording study (Robinson and Burton, 1980a), since we found that disappearance of activity in SII often was subsequently correlated with evidence of vascular hematomas in the white matter adjacent to SII. The possibility of inadvertent damage due to circulatory insults was not discussed in previous lesion studies (Ridley and Ettlinger, 1976, 1978; Murray and Mishkin, 1984).

SII's hierarchical position in processing tactile inputs

The observed deficiencies in relearning size and texture comparisons following SII lesions touch on an hypothesized role for SII in a hierarchical model for tactile perception. According to Mishkin (1979), SII's projections to the ventromedial limbic area may provide a sequential input to tactual memories and sensory–limbic interactions in tactual learning. In the context of the theory, SII is assigned a role in somesthesis that is equivalent to the role played by the inferior temporal cortex in vision. This stepwise model is supported by recent evidence showing projections to layers III and IV of SII from SI, but return connections to only the supra- and infragranular layers of SI (Friedman, 1983; Friedman et al., 1986). These variations in the cortical layers reached by different classes of corticocortical connections have been hypothesized to reflect feedforward and feedback regulation of the flow of sensory information in the cortex (Tigges et al., 1973; Gilbert and Kelly, 1975; Rockland and Pandya, 1979; Maunsell and Van Essen, 1983). Consistent with this view, Pons et al. (1986) have reported that in adult macaques, SII cannot be activated by cutaneous stimuli after SI is lesioned; this is suggestive of the dependence of SII on input from SI. Additional support for a hierarchical processing of somatosensory information through SII comes from anatomical studies showing "feedforward" connections between SII and insular and retroinsular areas (Friedman et al., 1986).

Another issue that relates to our behavioral findings and theories of the flow of somatosensory processing in the cortex is the source of thalamic input to SII in the adult macaque. Previously, it was suggested that areas of VPL projected to SI and SII (Kruger and Porter, 1958; Roberts and Akert, 1963; Burton and Jones, 1976). It has recently been shown that SII also receives a substantial input from a ventral part of VPL (referred to as VPI), especially in primates (Friedman and Murray, 1986). In agreement with this view was the finding that cell counts in VPI were substantially reduced in cases where SII had been damaged. However, cell counts in VPL, in these cases, were also consistent with previous findings that VPL, in addition, projects to SII in a variety of species (Burton and Jones, 1976; Burton, 1984, 1986; Burton and Carlson, 1986). If VPI projects exclusively to SII, and VPL predominantly to SI and less extensively to SII, then it is of interest to know the nature of the submodality of these 2 thalamic projections. If the size and texture deficits in the adult after SII lesions are due to the interruption of the input to SII from SI, then the lack of cutaneous and noncutaneous input to SII could explain size and texture deficits. Yet in the infant, SII lesions and SI–SII lesions involve only deficits in texture discrimination capacity. Is this to be taken to mean that SII is dependent on SI only for cutaneous input in the infant? Actually, SII in infants can function alone to mediate texture discrimination capacity, so it must have an alternative source of cutaneous input when SI is gone. Such a source could be from VPL directly, rather than through SI to SII. Recent studies have suggested that VPL projects more widely to SI and surrounding areas in the infant than in the adult macaque (Cheema et al., 1986). VPL and VPI may also project more redundantly to SI and SII in the infant than in the adult. Such a phenomenon of exuberant thalamocortical connections might serve as a basis for the different behavioral results in infant and adult macaques (Clarkson et al., 1987).

A hierarchical model of somesthesis involving SII as the "pivotal link" to tactual memories and "limbic" motivations to perform does not explain why infant monkeys were able to learn size and texture without SII, learned size without SI and SII, and failed on texture only in the presence of combined lesions. If connections from SII are necessary for higher cortical processing of tactile information, how is normal tactile learning and performance obtained with the SII lesion in infants, even if exuberant VPL input relays necessary information to SII? If these SII–limbic connections seen in the adult are necessary for its tactile learning, some substitute for that pathway may exist in the infant. The present studies suggest that one should speculate about exuberant pathways to limbic areas from SI, as well as try to explain normal tactile function after early SII lesions.

Mechanisms for recovery

Potential mechanisms underlying the differences between the recovery of adult and infant animals from comparable lesions have been reviewed previously (Goldman, 1974; Finger, 1978). Thus, others have found that extensive lesions in sensorimotor cortex (Kennard, 1942; Passingham et al., 1983) prevented recovery of skilled motor behavior, or that lesions of both orbitofrontal and dorsolateral frontal cortex eliminated performance in a delayed-choice paradigm (Goldman, 1974). Our present finding that texture discriminations could not be learned

after combined SI–SII lesions is another example of the failure to find recovery when the lesioned area is extended to include adjacent areas with related, but not identical, functions. One possible conclusion from such observations is that the mechanisms supporting recovery in infants with more limited lesions must depend on activity in the surviving areas or on the interactions between the surviving tissue and other parts of the brain. In the context of texture discriminations, recovery was possible in infants with either SI or SII spared, but not when both were damaged. Consequently, it is possible that the mechanisms responsible for recovery are common to the 2 regions.

One mechanism for recovered function that has received considerable attention in studies of the visual system of rodents has been the sprouting of collateral connections (Schneider and Jhaveri, 1974). There is little anatomical evidence of such phenomena in postnatal primates. In addition, the behavioral changes seen here and after total SI lesions (Carlson, 1984c) were evident the day following surgery and would, therefore, be present too soon for sprouting of substantial distant connections. Such rapid recovery suggests physiological changes, albeit these could derive from altered connections (see below).

Another mechanism is that of alterations in behavior based on functional substitution by remaining portions of the nervous system. In this context, SI and SII might be defined as being more functionally equivalent in infants, while during maturation some of this pluripotentiality is lost. The extent of substitution would be limited, however, to those cortical regions that have access to similar cutaneous sensory inputs, as the texture discrimination tasks have been shown to depend on areas of cutaneous input (Randolph and Semmes, 1974; Carlson, 1981).

Substitution of modality or cue dependency may occur in the case of behavioral tasks that can be solved using several of a finite combination of cues or strategies. For example, substitution may be more likely when the required discrimination is more general, or where both proprioceptive and cutaneous cues are available to perform correctly. Thus, the cues needed to learn size comparisons may be easier to master than those for texture tasks; the correct choice may be accessible from a wider repertoire of inputs. This may explain the survival of the ability to make size comparisons after combined lesions. A critical implication is that substitution need not arise from surviving tissue responding to the behavioral situation in the same way as the lesioned tissue, or that the undamaged area(s) develop novel physiology, but that the animal learns to utilize the capacities of the surviving tissue, i.e., "learns to walk in a new way." The behavioral tasks must properly tap the unique capabilities of the different cortical areas in order to reveal whether a specific behavioral function has recovered, and whether the surviving tissue has substituted. A shift in cue-dependence does not seem likely in the present experiments, as the solution of the size and texture discrimination tasks has been shown repeatedly to depend on a highly specific stimulus dimension that is associated with selective categories of cutaneous or noncutaneous input (Randolph and Semmes, 1974; Carlson, 1981).

If some form of substitution of cortical areas occurs in infants, what might be responsible for its absence in adults? Might the cause be the role of corticocortical connections in providing input to a "higher" area, or the modulatory effects of such reciprocal connections between cortical and/or subcortical structures? Several studies have shown that the responses of visual (Schiller and Malpeli, 1977; Spear et al., 1980) and somatosensory (Pons et al., 1986; Burton and Robinson, 1987) cortical areas can be altered (possibly through the inputs or through modulation) to block activity in interconnected cortical areas. For example, in primates, area 18 does not respond to visual stimuli when area 17 is cooled (Schiller and Malpeli, 1977); in cats, the lateral suprasylvian visual area is similarly affected by ablating area 17 in adult but not infant animals (Spear et al., 1980). In the somatosensory system, Pons et al. (1986) have reported that ablating SI in adult primates makes it impossible to activate responses to stimulating the hand and digits in SII. These findings suggest that, through maturation, the interconnections between SI and SII may establish a potential for SI to gate or inhibit activity in SII. With SI lesioned, SII in an adult primate might then be unable to respond normally to cutaneous inputs. SII in infants might then be expected to be more responsive after an SI lesion if such gating functions are late to develop. The faster recovery seen with total SI lesions (Carlson, 1984c) could then be explained as being based on the degree of suppression of SII by any remaining SI tissue; with all of SI gone, SII would be able to respond maximally to the sensory inputs from the hand mediated through the thalamus. A similar competitive argument has been presented previously to explain more efficient language development in children whose dominant hemispheres have been more extensively damaged (cited in Carlson, 1984c). One problem with this explanation is the delayed learning of texture comparisons seen after infant SII lesions, since the idea of the control exerted by SI onto SII would not predict that SII lesions would cause changes in SI. However, evidence for reciprocal modulatory effects has been seen between SI and SII in cats following lidocaine injections into one cortical somatosensory area, while recording from the other ipsilateral region (Burton and Robinson, 1987). Hence, it is possible that alterations in SI activity might occur in primates after an SII lesion. Schiller and his colleagues have similarly seen some altered activity in area 17 following the cooling of area 18.

Findings on the elimination of exuberant synapses in the cortex of macaques may describe the anatomical basis for alterations in the physiology of surviving tissue in infant brains. The age range during which recovery was obtained in the present and earlier (Carlson, 1984b, c) experiments matched the 3–4 month postnatal period when maximum synaptic exuberance has been found throughout the cerebral cortex of primates (Rakic et al., 1986). The time course of synapse elimination, which is one of the regressive changes that are important in the development of the central nervous system (Purves and Lichtman, 1985), also corresponds to the period when other plastic changes have been observed, such as the formation of ocular dominance columns. Previously, we suggested that the presence of exuberant connections in the immature primate cortex might affect recovery of tactile capacities after total SI lesions (Carlson, 1984c). It now seems possible that the early lesions may modify synapse elimination in related cortical areas, such that the uninjured areas may continue to mediate normal functions. This hypothesis would suggest that in the uninjured region, connections persist that would have competed unsuccessfully with the cortical connections that were removed by the lesions (Caminiti and Innocenti, 1981). Three categories of connections may persist: commissural, from the same somatic sensory areas in the opposite hemisphere; association and numerous subcortical inputs, including thalamic (specific and intralaminar nuclei); and striatal, locus coeruleus, and basal nucleus of Meynert. One or more of these connections might, then, provide the basis for

altered activity in the surviving tissue and, thereby, some recovery of tactile discrimination abilities. One intriguing possibility might be that the connections normally eliminated provide the means for modulating the flow of sensory inputs through the interconnected somatic sensory areas of SI and SII.

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