PLASTICITY OF SENSORY AND MOTOR MAPS IN ADULT MAMMALS

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KEY WORDS: spinal cord, thalamus, auditory system, ventroposterior nucleus, nerve regeneration

INTRODUCTION

This review addresses questions about the capacity of sensory and motor maps in the brains of adult mammals to change as a result of alterations in the effectiveness of inputs, the availability of effectors, and direct damage. The issue of the mutability of maps in adults is important because sensory and motor representations occupy much of the brains of mammals, regardless of the complexity and extent of neocortex (e.g. Kaas 1988, Wall 1988, Maunsell & Newsome 1987); behavioral recovery occurs after damage to central representations (e.g. Bornschlegl & Asanuma 1987, Dürsteler et al 1987, Eidelberg & Stein 1974); and such changes may relate to improvements in sensory and motor skills with experience (e.g. Gibson 1953). In addition, features of reorganization that are apparent in sensory and motor maps may characterize less easily studied areas of the brain.

Specific questions addressed in this review are as follows:

1. An obvious question is, “Does map reorganization occur in adult as well as developing mammals?” The mutability of developing sensory maps as a result of environmental manipulations, especially sensory deprivation, is well documented and accepted (see Sherman & Spear 1982, Kaas et al 1983, McKinley et al 1987, Wall 1988), but many changes appear to occur only within a “critical period” of development. The often less dramatic evidence for alterations in adults may seem
questionable, open to alternative explanations, or so limited in extent to be unimportant (see Killackey 1989 for review). Thus, it is useful to consider the now extensive evidence for map reorganization in adult mammals.

2. Since sensory systems consist of a number of separate representations that are interconnected in a semihierarchical fashion, another obvious and long-standing question is, “What stages are capable of reorganization?” Early evidence for plasticity was obtained at the level of sensory inputs to the spinal cord (e.g. Liu & Chambers 1958, Devor & Wall 1978), and at least some of the changes described in primary somatosensory cortex could simply result from the relay of subcortical alterations. There now is evidence, however, for the restructuring of maps at three levels of cortical processing in the somatosensory cortex, and for changes that cannot be easily attributed to the relay of subcortical reorganizations.

3. To date, the bulk of the evidence for plasticity in maps stems from experiments in the somatosensory system. Thus, “Are other sensory and motor systems capable of reorganization?” Recent experiments on the motor, visual and auditory systems suggest that the capacity to reorganize characterizes all central representations and may be a general feature of brain tissue.

4. “How rapidly does cortex reorganize?” This is an important issue because different mechanisms have been proposed for rapid and slow alterations.

5. “What are the magnitudes of the observed changes?” Small alterations are compatible with synaptic and other modifications within previously existing structural frameworks, whereas larger modifications may imply the sprouting and growth of new connections.

6. Finally, “What are the relative extents of changes at different stages of sensory hierarchies?” These are critical to evaluate because higher stations have been hypothesized to be more plastic (e.g. Merzenich et al 1988, Pons et al 1988).

Much of the research on the plasticity of neural maps in adult mammals has been on primary somatosensory cortex. Primary somatosensory cortex is a convenient place to look for plasticity in the somatosensory system because S-I contains a relatively large, two-dimensional map that in many species is largely accessible on the dorsolateral surface of the brain. Changes in the smaller, three-dimensional representations in the brainstem and spinal cord are more difficult to demonstrate, and the higher order maps in cortex can be less responsive to sensory stimuli under typical recording conditions and can have more complex maps in which changes
in the representation may be less obvious. Thus, it has been logical and productive to look for plasticity in S-I.

REORGANIZATION OF S-I (AREA 3B) OF MONKEYS

A number of experimental approaches have demonstrated somatotopic changes in the representation of skin receptors in area 3b or S-I proper of monkeys. Although the term S-I has been used to include up to four separate representations in monkeys and higher primates, only area 3b appears to be the homolog of primary somatosensory cortex or S-I of the nonprimates (Kaas 1983). A popular site of investigation has been area 3b of somatosensory cortex in the relatively unfissured brains of such New World monkeys as owl monkeys, squirrel monkeys, and marmosets. These and other monkeys have a large, central portion of area 3b devoted to the glabrous or ventral surface of the hand (Figure 1A; also see Merzenich et al 1978, Sur et al 1982, Carlson et al 1986). The arrangement of skin parts on the ventral hand (Figure 1B), the palmar pads, and glabrous phalanges is preserved but distorted in the cortical map (Figure 1C). Each digit is represented in order, with proportionally more cortex devoted to the sensitive digit tips. The spatial arrangement of the palmar pads, allowing for a partial split of the palm between the thenar and hypothenar pads, is also maintained. The representation of the dorsal or hairy skin of the hand occupies little tissue, and the location of dorsal skin in the map is variable across and within species. The representation of the back of the digits and hand, however, is often split into separate locations lateral and medial to the representation of glabrous skin (Figure 1C) and into islands of cortex within the representation of glabrous skin.

The Effects of Removing an Input

Cutting or crushing the median nerve, which subserves the thenar half of the glabrous hand from D1 through the middle of D3, produces, over time, a dramatic change in map organization (Merzenich et al 1983a, Huerta et al 1986). The nerve section, by deactivating inputs from half of the hand, deprives a large lateral sector of the hand representation in area 3b of its normal source of activation (Figure 1D). Before regeneration occurs, the deprived cortex becomes responsive to new inputs largely from the dorsal surface of the hand but also from parts of the palmar pads with preserved cutaneous innervation. There is no recovery of responsiveness to the denervated skin, apparently because intact nerves have a very limited capacity to sprout and claim vacant territory (Jackson & Diamond 1981,
Kinnman & Aldskogius 1986). A new somatotopic organization is created in area 3b, the representations of the dorsal surfaces of digits 1–3 become many times larger than in normal animals, and receptive fields on the dorsal hand for neurons in this altered cortex are much smaller than normally seen for cortical neurons representing the dorsal hand. The time course of the change is not well-known, but some of the reactivation occurs within hours of the nerve section, and further alterations in responsiveness and representational detail appear to occur over days to possibly months (Merzenich et al 1983b).

The effects of deactivating a peripheral nerve can be partly or totally reversed by nerve regeneration. Regeneration after nerve crush in humans is typically followed by complete sensory recovery and no notable impairments (see Wall & Kaas 1985 for review). This behavioral recovery apparently reflects complete or nearly complete and errorless regeneration (Lisney 1983). Recordings from the hand representation in area 3b of monkeys after nerve crush and regeneration reveal maps that not only are totally normal (Figure 1F), but when the same monkey is recorded both before and after nerve crush and regeneration, individual features of the maps return (Wall et al 1983). These results demonstrate that nerve regeneration after nerve crush can be extremely accurate, hence the recovery of normal function, and that changes produced during reorganization can be reversed, even to the extent of returning individually unique features. Although the cortical map is plastic and it does change after nerve damage, the recovery of a specific, previous organization indicates that original connection patterns persist, have some advantage, and previous activation patterns are likely to reappear.

Another outcome occurs if a nerve is cut, repaired by suturing the cut ends together, and allowed to regenerate (Paul et al 1972, Wall et al 1986, 1988, 1989b; Figure 1G). After such repair, regeneration is incomplete and disorderly (Florence et al 1988). The resulting complex of changes in cortex incorporates (a) sections of reorganized cortex that are not recovered by inputs from the regenerated nerve and (b) sections of somatotopically disorganized cortex that are activated by the regenerated nerve (Wall et al 1986). Because nerve regeneration is incomplete, original sources of activation do not return for some parts of the deprived zone of cortex. The cortex remains reorganized and responsive to intact inputs from other nerves. However, disorderly nerve regeneration produces other patches of cortex that are responsive to median nerve inputs, but these patches do not form a continuous, orderly, somatotopic map. In addition, neurons in many reactivated cortical locations have several, separate receptive fields. Thus, peripheral axons previously subserving a restricted skin location regenerate to several different skin locations. Furthermore, some cortical
neurons become abnormally responsive to deep or pacinian receptors, thereby suggesting that regenerated terminals are established in the median nerve skin field with inappropriate receptors. The results demonstrate that there may be no central correction for disorder in peripheral nerves; however, a normal control map may be reestablished after section, repair, and regeneration of sensory nerves of a single finger (Allard et al 1989). Thus, central corrections may occur for the more local disorder in axons regenerating in a finger.

Evidence for a limit on the extent of cortical reorganization is seen after cutting both the ulnar and radial nerves leaving only the median nerve to the hand (Huerta & Wall 1987). This procedure produces a more extensive zone of deprived cortex that is not completely reactivated after recovery. The representation of the median nerve expands over much of the hand representation, but small zones of silent cortex remain. Thus, parts of large zones of deactivation may remain unresponsive even after long recovery periods.

Further evidence that the reactivation of cortex is limited by the size of the deprived zone is seen after cross repair and regeneration of nerves to the hand (Wall et al 1986, Figure 1H). In this procedure, both the ulnar and median nerves are cut, the proximal end of the ulnar nerve is crossed and sutured to the distal end of the median nerve, and the proximal end of the median nerve tied to prevent regeneration. As a result, the ulnar nerve regenerates into the skin territory of the median nerve, and the skin territory of the ulnar nerve remains denervated. The cross repair results in islands of cortex in the ulnar nerve zone of area 3b that are activated by afferents terminating in the skin field of the median nerve. Thus, the overall cortical map does not reorganize to reflect the altered targets of the regenerated ulnar nerve (also see Allard et al 1989). Furthermore, a behavioral compensation does not occur, and such monkeys persistently mislocalize stimuli on inner to the outer side of the hand. As after median nerve cut and regeneration, a somatotopically disordered array of reactivated patches of cortex is seen after nerve cross and regeneration. Thus, local disorder in the regenerated ulnar nerve is not corrected centrally. Also, even though the regeneration of the ulnar nerve is in the skin territory of the median nerve, the reactivation is in the cortical territory of the ulnar nerve. Thus, there is no central correction for the new skin location for the ulnar nerve afferents. Furthermore, the dorsal hand, which is innervated by the intact radial nerve, has an expanded representation, indicating the reorganization of some of the deprived cortex and the formation of a new activation pattern that is not completely displaced by inputs from the regenerated nerve. Finally, large zones of silent cortex persist, thus suggesting that the incomplete regeneration of the ulnar nerve together with
the total lack of regeneration of the median nerve produced a zone of
deprived cortex that was, as for the two nerve cut preparation, too large
for complete reactivation. However, such zones of silent cortex do not
always occur. In similar experiments, where both the median and ulnar
nerves were cut, no significant zones of silent cortex were observed in
recordings from area 3b after a recovery period (Garraghty et al 1990c).

Reorganization of area 3b has also been demonstrated after removal of
a single digit in owl monkeys (Merzenich et al 1984, Figure 11). Detailed
microelectrode maps before and weeks after removal of digit 3 at the base,
revealed that the cortical territory normally activated by stimulating digit

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Figure 1 The reorganization of primary somatosensory cortex (area 3b or S-I proper) of
monkeys after different experimental manipulations. A. The location of area 3b on a dor-
solateral view of an owl monkey brain. Because of the lack of a central fissure, most of the
representation of the body is in the cortex exposed on the surface of the anterior parietal
cortex. The foot and body are represented medially, the hand in the middle, and the face
laterally. B. The topographic order of the hand is largely preserved in the cortex, but the
palm is partially split and the dorsal surfaces of the hand and digits are largely split to medial
and lateral cortical locations (shading). Digits and pads are traditionally numbered, and
insular, hypothenar, and thenar pads are lettered. C. The representation of the hand is
distorted in shape to fit a rectangle of cortex, digit tips occupy the anterior half of the digit
regions. D. Section or crush deactivates the median nerve and deprives most of the lateral
half (hatched) of the hand representation of normal activation. E. During long-term de-
activation of the median nerve by crush or section, the deprived cortex is topographically
activated by stimulating the dorsal surfaces of digits 1–3, and by insular pads and pad 3.
F. After nerve crush and regeneration, a normal map and even the map specific to an individ-
ual is recovered. G. After regeneration of a cut and reconnecting median nerve, parts of the
deprived territory remain responsive to other inputs, thus indicating incomplete regeneration
and recovery, and parts become responsive to median nerve inputs, but in a somatotopically
disorganized pattern that indicates incomplete compensation for disorder in the regenerated
nerve. Some recording sites (dots) have multiple receptive fields. H. After section and suture
of the median nerve to prevent regeneration, and cross suture of the cut ulnar nerve to the
peripheral stump of the median nerve and subsequent regeneration into the median nerve
territory, regions of unresponsive cortex occur as a result of removing inputs from the median
nerve and part of the ulnar nerve. The cortical territory of the ulnar nerve is partly activated
in a somatotopically disorganized manner by ulnar nerve axons regenerated into the median
nerve skin. Again, some recording sites have multiple receptive fields. I. After removal of
digit 3, cortex formerly devoted to that digit becomes activated by the glabrous skin of D2
and adjoining palm. J. Weeks of tactile stimulation of D2 and D3 resulted in an expansion
of the tactile representation of D2 and D3, especially by increasing the tactile activation of
the adjoining part of area 3a. K. After ablation of the cortex representing D3 (left), the lesion
site shrinks over weeks, and some of the sites along the margin of the lesion that were
formerly responsive to D2 and D4 become responsive to D3. L. After a period of hand
use after two digits have been fused, the normally sharp boundaries between the digit
representations are blurred by many neurons having receptive fields on both fused digits. M.
Electrical stimulation of an electrode site in cortex representing D3 produces an expanded
zone of cortex with matching receptive fields on D3. See text for references.
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A. Brain Map

B. Map Somatopic Order

C. Normal Map

D. Zone Deprived by Nerve Section

E. Reorganization after Median N. Section or crush

F. Recovery after Regeneration of crushed nerve

G. Altered Recovery-Regeneration of Cut N.

H. Altered Recovery after Regeneration of Ulnar N. into Median N. Skin

I. Reorganization After D3 Ablation

J. Expansion after D2 & D3 Stimulation

K. Recovery of Ablated Representation of D3

L. Reorganization After Digit Fusion

M. Expansion After Electrical Stimulation

E.S.
3 becomes responsive to light touch on the glabrous surfaces of digits 2 and 4, and to some extent palmar pads.

These results indicate that the same cortical territory can reorganize in different ways depending on what inputs are removed. After section of the median nerve subserving glabrous digits 1–3, most of the reactivation is from inputs from the dorsal surface of the hand, and the cortical territory of the glabrous surface of D3 is largely reactivated by inputs from dorsal skin of D3. After removal of inputs from both the dorsal and glabrous skin of D3, however, the reactivation of the same cortical zone is from inputs from glabrous surfaces of D2 and D4. Both inputs from dorsal and glabrous skin apparently can activate the cortical territory of glabrous D3. The dorsal D3 inputs may dominate when inputs from the glabrous surfaces of adjacent digits are missing, whereas inputs from glabrous D2 and D4 may dominate when inputs from dorsal D3 in addition to glabrous D3 are missing.

**Effects of Cortical Lesions and Cortical Stimulation**

Another way of evaluating the potential of cortical representations to reorganize has been to ablate all cortex representing some part of the hand (see Jenkins & Merzenich 1987). Weeks to months after removing all cortex responsive to light touch on the glabrous surface of D3 in owl monkeys, some recording sites in cortex next to the lesion that were formerly responsive to other parts of the hand become responsive to D3 (Figure 1K). Thus, there was recovery of some of the cortical inputs previously removed by the lesion. Yet, the amount of cortex activated by inputs from D3 remained small, and larger lesions including the cortex devoted to digits 2–5 were not followed by the recovery of representations of these digits. The somatotopic map in area 3b of monkeys can also be changed, at least for short periods, by repeated intracortical microstimulation of a given cortical site (Recanzone & Merzenich 1988). Neurons close to the stimulated site (~ 500 μm) come to have receptive fields closely matching those of the stimulated neurons (Figure 1M).

**Effects of Changing Skin Stimulation Patterns**

Another way of changing cortical organization has been to fuse the skin of adjacent digits (Figure 1L; Clark et al 1988). Normally, neurons in 3b of monkeys have receptive fields restricted to a single digit. This condition is changed, however, when the skin of two adjacent digits is fused by cutting and suturing skin to create a continuous skin surface across the two digits. Recordings made weeks later from area 3b revealed a number of receptive fields that bridged the skin of both of the fused digits. This did not seem to be the result of a mechanical spread of the stimulus or the
sprouting of peripheral nerves across digits, since receptive fields remained on two digits when recordings were made immediately after the digits were separated. In related experiments, transplantation of skin and its neurovascular supply from one finger to an adjacent finger in monkeys appeared to result in a cortical representation of the digits that integrated the representation of transplanted skin with that of surrounding skin (Clark et al. 1986, also see Merzenich et al. 1988). This result is in contrast to the lack of integration into the larger map that is observed after redirection of the ulnar into the median nerve skin (Wall & Kaas 1986).

Changes in the somatotopic organization of cortical maps of the hand in monkeys may also occur as a result of restricted, excessive tactile stimulation (Figure 1J). When monkeys were trained to maintain contact with a rotating disk with one or two digits, there was evidence for an expansion of the cortical representation of these digits (Jenkins et al. 1990). Much of this expansion appears to be in the adjoining part of area 3a, however, which normally is activated largely by deep (muscle) receptors, but also to some extent, especially for the hand representation, by cutaneous receptors. Thus, the major effect of the stimulation may have been to potentiate cutaneous over deep receptor activation.

S-I OF CATS, RACCOONS, RODENTS, AND BATS

Many of the studies of cortical map reorganization have been in S-I of nonprimates. Some of the earliest studies were on S-I of cats. Raccoons provide the advantage of studying a large hand area in S-I in which individual digits are demarcated by shallow sulci. Rats have been useful in studies in which it is important to study larger numbers of animals or to have a clear morphological substrate such as the “barrel field.” Bats provide an opportunity to study a cortex in which the representation of the forelimb is quite different.

The Effects of Removing Peripheral Inputs

The results of several studies on nonprimates are in agreement with those on monkeys that show that cutting a peripheral nerve or removing a skin field alters the cortical map; the deprived cortex in S-I is not permanently deactivated, but it becomes responsive to remaining inputs. In an early landmark study, Kalaska & Pomeranz (1979) reported that several weeks after section of the nerves to the forepaw in adult cats, cortex formerly responsive to the forepaw became responsive to the normally innervated forearm (however, the change was more impressive in kittens). Similarly, when the hindlimbs and tails of cats were deafferented by selectively sectioning dorsal roots entering the spinal cord, portions of S-I normally
representing these body parts were activated by stimulating the abdomen, thorax and forelimb, and no silent cortex was observed (Franck 1980). Earlier, Metzler & Marks (1979) had shown that such changes in S-I can be quite rapid by recording from neurons formerly responsive to the upper hindlimb after reversal blocks of inputs from the upper hindlimb produced by injections of local anesthetic. While most recorded neurons in the cortex for the upper hindlimb could not be driven after the block, over a quarter of the sampled neurons immediately acquired new receptive fields on the hindpaw or abdomen. More recently, Brandenberg & Mann (1989) have recorded from the forepaw cortex in S-I of cats weeks after crush and at least partial regeneration of the sensory nerves to the skin of the forepaw. This procedure revealed a small proportion of abnormal neurons that responded to stimulation of the hindpaw as well as the forepaw, thus suggesting that some effects of reorganization as a result of denervation can persist for some time after reinnervation. Finally, the removal of a forepaw digit in adult cats is followed by a dramatic expansion of the representations of adjacent digits in contralateral S-I when measured by the 2-deoxyglucose technique for revealing metabolically active cortex (Juliano et al 1990).

Removing inputs at a higher level by damaging the spinal cord may produce somatotopic reorganizations in S-I of cats as well. In an early study, Levitt & Levitt (1968) reported that the responsiveness of the hindlimb cortex of adult cats to low-threshold cutaneous stimuli was abolished by lesions of both the contralateral dorsal and lateral columns, although the topography of cortex subserved by inputs above the lesion was unchanged. McKinley et al (1987) recently demonstrated, however, that after deactivation of the hindlimb cortex of S-I by lower spinal cord transections in kittens, this cortex ultimately becomes responsive to the trunk. Reactivation of deprived cortex was more extensive in kittens sectioned at 2 weeks than 6 weeks of age, but the magnitude of the changes that occurred after the sections at 6 weeks of age suggests that some plasticity could follow such sections in adult cats.

Alterations produced by digit removal have been extensively investigated in raccoons. These carnivores have the advantage of a large representation of the glabrous forepaw that is separated from other parts of S-I by the fusion of the coronal and postcruciate fissures (Welker & Seidenstein 1959). Furthermore, spurs on the triradiate sulcus separate palm and digit representations, and digit 2 and 3 representations, and cortical dimples may indicate further divisions. Thus, the locations of the representations of individual digits and pads of the forepaw can be closely approximated from surface landmarks.

The first experiments were on the long-term effects of digit removal in
young, 2–8-week-old raccoons, and the reactivation of the deprived cortex by inputs from adjoining digits and parts of the palms was observed (Kelahan et al 1981). Subsequent experiments demonstrated a similar reorganization of S-I after removal of digits in adult raccoons. The immediate effect of removing digits in adult raccoons appears to be a profound deactivation of the cortex for that digit. On the same day of amputation, Rasmusson & Turnbull (1983) found that responsiveness in the deprived cortex to adjacent intact digits was largely restricted to responsiveness to the offset of skin indentation, presumably as a result of the offset of inhibition generated by the stimuli. In similar experiments, however, Kelahan & Doetsch (1984) found that some neurons in the deprived zone of cortex were immediately activated by low-intensity stimulation of adjacent digits. A consistent finding was that over several weeks, the deprived cortex became highly responsive to light cutaneous stimuli on the digits next to the removed digit (Rasmusson 1982, Rasmusson & Turnbull 1983, Kelahan & Doetsch 1984). An even more dramatic reorganization of S-I cortex was reported for a raccoon that had been caught in the wild at some unknown length of time after the loss of a forepaw (Rasmusson et al 1985). In this raccoon, recording sites throughout the forepaw region of S-I were responsive to stimuli on the forelimb stump, and some sites were responsive to tactile stimulation of the glabrous hindpaw. These observations raise the possibility that the entire, quite large forepaw area of S-I in adult raccoons can reorganize and become responsive to other inputs. Another possibility is that some or much of the reactivation was from persisting forepaw nerve terminations in the stump.

Cortical reorganization has been studied in S-I of adult rats after section of the sciatic nerve (Wall & Cusick 1984), after a cytotoxin has been used to kill primary neurons in the sciatic nerve (Wall et al 1988), and after denervation of a row of mystacial vibrissae follicles (Welker et al. 1988). In normal rats, about 85% of the hindpaw representation depends on the sciatic nerve and 15% on the saphenous nerve. After section of the sciatic nerve, the representation of the saphenous nerve expanded about three times in surface area into, but not completely filling, the territory of the sciatic nerve. The expansion occurred rapidly, within 1 or 2 days, and remained stable for at least several months; however, about half of the total hindpaw representation remained unresponsive to cutaneous stimuli. The removal of sciatic nerve neurons with the toxin, ricin, produced a nearly identical result. Since most dorsal root ganglion cells in adults survive peripheral nerve section, maintain central connections in the spinal cord and dorsal column nuclei, and have some level of spontaneous activity, these experiments indicate that the presence or absence of sciatic nerve terminals is not a major factor in determining the extent of plasticity.
Microelectrode recordings of the face representation in S-I of adult mice (Welker et al 1988) and 2-deoxyglucose measures of metabolic activity in adult rats show that representations of normally innervated vibrissae expand into the cortical territories of denervated vibrissae within months after deactivation (Kossut et al 1988, Kossut 1988).

The effects of amputating a body part on S-I organization has also been studied in an Australian fruit bat, the flying fox (Pteropus). Though most of the forelimb digits have been modified to support the wing membrane, the first digit protrudes from the wing and is used for climbing. In normal bats, D1 has a large representation in caudal S-I (Calford et al 1985). Weeks after amputation of D1, recordings in the deprived cortex, where neurons previously had receptive fields restricted to D1, reveal new receptive fields that include the skin of the intact adjacent wrist and metacarpals, and the adjacent wing membrane (Calford & Tweedale 1988, 1990). Immediately after the amputation, the deprived cortex was silent, but within 20 h, responsiveness returned and the deprived neurons had large, new receptive fields. Over a period of days, however, the new receptive fields became more restricted in size.

Peripheral denervations in adult mammals not only produce reorganizations in somatotopy in S-I, but also alter the expression of inhibitory neurotransmitters and levels of metabolic activity. Lesions of the follicles of mystacial vibrissae in rats and mice are followed in days by a decrease in the deprived cortex in the expression of the inhibitory neurotransmitter, GABA, that lasts for weeks (Welker et al 1989). Reductions in the expression of the metabolic enzyme, cytochrome oxidase, also occur in the deprived cortex (Wong-Riley & Welt 1980). Such lesions also produce increases of about 40% in the sizes of metabolically active columns of tissue in S-I for adjacent vibrissae as revealed by 2DG uptake (Kossut et al 1988; also see Melzer et al 1989).

**Reorganization of S-I Following Peripheral Stimulation**

There is some evidence that increasing the activity in peripheral nerves increases the extent of the cortex activated by these inputs. In an early report, Spinelli & Jensen (1979) recorded from the cortex in kittens that had received avoidance training by shocking one forearm in a daily session. Spinelli & Jensen noted that the representation of the forearm was many times larger in S-I contralateral than ipsilateral to the shocked forearm. While the effects were attributed to changes in brain development, similar effects may occur in adults. More recently, Recanzone et al (1990) reported that a single session of 6–8 hours of electrical stimulation of a cutaneous nerve in adult cats is rapidly followed by at least a short-term expansion of the cortical representation of that nerve. After periods of stimulation
of pairs of mystacial vibrissae in rats, neurons in S-I enlarge their receptive fields to include both whiskers (Yun et al 1987, Delacour et al 1987).

REORGANIZATION OF HIGHER CORTICAL AREAS IN PRIMATES

After sources of input have been removed, somatotopic reorganizations have been observed in area 1 of anterior parietal cortex and S-II of lateral parietal cortex of monkeys. Area 1 was once included with area 3b and other areas in a large "S-I" (see Kaas 1983 for review). It is now clear, however, that both areas 3b and 1 contain separate, congruent, and parallel representations of cutaneous receptors in a manner analogous to V-I and V-II of visual cortex. Though both areas 3b and 1 receive direct input from the ventroposterior nucleus, the input to area 1 is sparser and from thinner axons and axon branches. Area 1 receives its major feed forward inputs from area 3b. Lesions of area 3b deactivate area 1, but lesions of area 1 do not deactivate area 3b (Garraghty et al 1990b). Thus, area 1 can be considered as a higher somatosensory area than area 3b.

Although area 1 has not been as extensively studied as area 3b after peripheral nerve manipulations, the normal dependency of area 1 activity on area 3b suggests that all changes observed in area 3b should be reflected by similar changes in area 1. This assumption appears to be largely supported. Thus, nerve section produced somatotopic reorganizations in both areas 3b and 1 of monkeys (Merzenich et al 1983a), and nerve section, repair, and regeneration resulted in similar mixtures of reorganization and disorderly reactivation in both fields (Paul et al 1972, Wall et al 1986). The details of the topographical transformations that were produced were different in areas 3b and 1 in both types of experiments, however, and these differences were interpreted as evidence that some reorganization occurs within area 1. Thus, area 1 reorganization may reflect both relayed and intrinsic plasticity.

More recently, major somatotopic changes have been reported for S-II of macaque monkeys after partial lesions of area 3b and adjoining somatosensory fields of the anterior parietal cortex. S-II receives dense inputs from all anterior parietal fields, and sparse inputs from the ventroposterior nucleus of the thalamus (see Kaas & Pons 1988 for review). S-II is completely dependent on anterior parietal cortex, probably from both direct and indirect inputs from area 3b, for activation by cutaneous stimuli (Pons et al 1987, Garraghty et al 1990c, Burton et al 1990). Thus, lesions of the anterior parietal cortex that remove cortex representing the hand in areas 3a, 3b, and 1 and 2 abolish all activation of neurons in S-II.
by stimulation of the hand. Indeed, the cortex normally responsive to stimulating the hand in S-II is initially unresponsive to the stimulation of any body part after the lesion. Recordings made weeks after the lesion, however, reveal a complete recovery of responsiveness in the deprived cortex to stimuli to other parts of the body, largely the foot (Pons et al 1988; Figure 2).

These results provide compelling evidence that somatotopic changes do occur in the cortex, and are not solely relayed from subcortical structures. It seems unlikely that the cortical lesions produce subcortical plasticity that is somehow relayed to S-II. Rather, changes in the effectiveness of connections in S-II from anterior parietal cortex would be the likely source of the reactivation. The results also suggest that higher cortical stations have greater potential for plastic changes than lower stations. When two nerves are cut or damaged to denervate most of the hand, parts of the representation of the hand in areas 3b and 1 may not recover responsiveness to cutaneous stimuli (e.g. Wall & Kaas 1986, Huerta & Wall 1987). Removing all of the inputs from the hand by cortical lesion, however, is followed by complete reactivation of S-II. Possibly, higher stations such as the parietal ventral area (Krubitzer & Kaas 1990) have even more potential for reorganization.

SOMATOTOPIC REORGANIZATION AT SUBCORTICAL LEVELS

Two extreme positions are (a) that all reorganization in the somatosensory system occurs at the cortical level, and (b) that all changes in cortex reflect alterations relayed from subcortical levels. This review is largely concerned with cortical maps, because changes can be more clearly demonstrated in these larger, more accessible, two-dimensional representations, and any failure to find evidence of mutability at cortical levels would suggest that looking at subcortical levels would be unrewarding. The reviewed evidence, however, strongly suggests that plasticity occurs at both cortical and subcortical levels.

There have been some questions about the existence of somatosensory reorganization in the spinal cord of adult mammals. Peripheral afferents enter the spinal cord and terminate in the dorsal horn or in the lower brain stem, where they form somatotopic representations. The possibility of extensive reorganization of the map in the spinal cord of adults was suggested by early evidence for collateral sprouting of intact inputs into regions of the spinal cord of cats deprived by partial denervation (e.g. Liu & Chambers 1958). Researchers using current techniques have failed to
Figure 2  The second somatosensory area, S-II, depends on inputs from representations in the anterior parietal cortex, largely area 3b, for activation. Thus, a lesion of the hand representations of the anterior parietal cortex (areas 3a, 3b, 1 and 2) deactivates the hand region of S-II, which becomes responsive to the foot. A. The locations of S-II in the opened central and lateral sulci of a macaque monkey. B. A strip lesion of hand cortex deactivates the hand region of S-II. C. The normal map of the contralateral body surface in S-II. D. The altered maps months after lesion of anterior parietal cortex. Based on Pons et al (1988). Figure reproduced from Kaas & Garraghty (1989).
demonstrate such sprouting (e.g. Rodin & Kruger 1984), however, and alternative explanations of previous results have been offered (see Pubols & Bowen 1988 for review). Yet, the lack of convincing evidence for sprouting does not mean that reorganization does not occur by other means, and the electrophysiological evidence for plasticity seems more compelling.

The immediate effect of denervation by nerve cut or section of dorsal roots seems to be to completely deactivate neurons with receptive fields in the denervated skin (Wilson & Snow 1987, Pubols & Goldberger 1980, Brown et al 1984, Devor & Wall 1978, Lisney 1983, Pubols 1984). Evidence has been presented for rather extensive reorganization in the dorsal horn after weeks of recovery (Basbaum & Wall 1976, Devor & Wall 1978, 1981), but such changes have not always been reported (Brown et al 1984). Recent reports describe a more limited spatial reorganization in the spinal cord that is restricted to the formation of new receptive fields very close to the original receptive fields (Wilson & Snow 1987). These authors conclude that the magnitude of the change is so small that it would be easily missed in most experiments.

There have been only a few experimental investigations of plasticity in the dorsal column–trigeminal complex after peripheral nerve injuries in adult mammals. Miller et al (1976) reported that cutting all but one of the dorsal roots related to the hindlimb in cats immediately deactivated many neurons in the gracile nucleus. They observed an increase in the size of the representation of the intact root after months of recovery, however, and fewer nonresponsive neurons. When all the dorsal roots for the hindlimb were cut (Dostrovsky et al 1976), the immediate effect was an increase in the number of neurons with receptive fields on the abdomen. Furthermore, some of the neurons with receptive fields on the leg acquired new receptive fields on the abdomen during a reversible cold block of the afferents from the hindlimb. More recently, Kalaska & Pomeranz (1982) studied kittens weeks after forepaw denervation and found that in the dorsal part of the cuneate nucleus, where neurons normally have receptive fields on the forepaw, many neurons had receptive fields on the wrist, forearm, or trunk. These experiments indicate that some degree of somatotopic reorganization occurs in the dorsal column nuclei after peripheral injury or deactivation, and that changes can be quite rapid. However, reorganization may be quite limited or absent in some circumstances. Waite (1984) reported that 2 months after section of the infraorbital nerve to whiskers in adult rats, there was no evidence for plasticity in the trigeminal nuclei.

Somatotopic plasticity has also been reported for the ventroposterior (VP) nucleus of the thalamus of adult rats and cats after the partial removal of inputs. In an early landmark study in adult rats, Wall & Egger (1971) ablated much or all of the gracilis nucleus, which represents the hindlimb,
and found no immediate somatotopic change in the VP. Yet recordings one week or more after the lesion provided evidence for an expansion of the forepaw into the hindpaw representation, and the authors concluded that thalamic cells projecting to hindlimb cortex had acquired new receptive fields on the forepaw. The reported expansion was small, however, on the order of a 200 μm or less. Thus, it would be useful to look for the relay of such proposed changes by recording from primary somatosensory cortex, where the forepaw and hindpaw are represented in quite separate locations. In monkeys, section of the dorsal column input to the gracilis nucleus was followed by an expansion of the forelimb representation into the most lateral part of the VP, where the hindlimb is normally represented (Pollin & Albe-Fessard 1979). Even earlier evidence for somatotopic change stems from studies showing that the receptive fields of some neurons in the VP can immediately change from one skin location to a nearby location where the skin region of the original receptive field is anesthetized by subcutaneous injections of procaine (Nakama et al 1966). More recently, extensive reactivation of the hand subnucleus of VP by inputs from the dorsal hand was found in squirrel monkeys with chronic section of the median and ulnar nerves to the glabrous hand (Garraghty et al 1990a).

REORGANIZATION OF VISUAL REPRESENTATIONS

Although the organization of the visual cortex has been considered to be highly stable in adult mammals, recent evidence indicates that considerable retinotopic reorganization can occur as a result of removing some of the retinal inputs. In mammals with frontally directed eyes, most visual areas can be activated by retinotopically matched inputs from either eye. In adult cats, parts of primary (V-I or area 17) and secondary (V-II or area 18) visual cortex were completely deprived of their normal sources of activation by removing one eye, and placing a 5–10° lesion near the central vision in the retina of the remaining eye (Kaas et al 1990). By itself, a lesion of the retina had little impact on the retinotopic organization of visual cortex. Recordings in rows of electrode penetrations across cortex produced normal sequences of receptive fields for each eye until the region of the cortex deprived by the lesion was encountered. Then, neurons only responded to the intact eye, since retinotopically matching receptive fields would be in the location of the retinal lesion. When recordings were made weeks after the removal of one eye and the placing of a small lesion in the other eye, the receptive field progressions indicated that the retinotopic
organizations of both V-I and V-II were altered. Recordings outside the deprived zone of cortex produced normal progressions of receptive fields for the remaining eye. In contrast, recordings in the binocularly deprived zone of cortex revealed that neurons had acquired new receptive fields that were displaced from expected locations in the lesioned part of the retina to intact parts of the retina around the lesion (Figure 3). The reorganization was not retinotopic, in that the new receptive fields did not form retinotopic progressions. Rather, neurons across 2–3 mm of cortex typically had similar, highly overlapping receptive fields in a part of the retina next to...
the lesion. In addition, neurons in adjacent recording sites in the deprived zone of cortex sometimes had quite separate receptive fields on opposite sides of the lesion. A few recording sites were activated from locations on both sides of the lesion. Although the extent of the reorganized cortex was large, as much as 4-6 mm in width, larger retinal lesions of 10-15°, depriving an even larger zone of cortex, produced a fringe of deprived cortex with reactivated neurons and new receptive fields, and a central core of silent cortex where neurons were not driven by visual stimuli. Comparable results were obtained in a monkey where matching lesions were placed in the fovea of each eye (Heinen & Skavenski 1988). Neurons in V-I initially unresponsive to visual stimuli later became responsive. These observations, though quite limited in extent, are important in that they indicate that the visual cortex, like the somatosensory cortex, is capable of reorganizing as a result of restricted deprivations to create new representations of receptor surfaces.

Other evidence for the reorganization of the visual cortex after partial deprivation stems from experiments on cats in which the optic chiasma was sectioned (Milleret & Buser 1984). This procedure produced a decrease in average receptive field sizes in area 18 (V-II) of cats by removing activation from the ipsilateral temporal retina. Over a period of weeks, however, neurons in area 18 progressively recovered large receptive fields, but only if visual experience was allowed. In another type of experiment (Krubitzer & Kaas 1989), partial lesions of area 17 in owl monkeys were found to deactivate a part of the middle temporal visual area, MT, which receives direct inputs from area 17. Recording immediately before and

Figure 3  The reorganization of the visual cortex after retinal lesions in cats. A region of primary visual cortex (area 17) was deprived of its normal sources of visual activation by removing the ipsilateral retina and placing a 5° lesion in the retina of the contralateral eye. Rows of recording sites (such as row A 1–5 on the lower right) outside the deprived zone of cortex produced normal progressions of receptive fields in the contralateral visual hemifield (A 1–5, upper). Rows of recording sites across the deprived zone (such as row B 1–5) would normally produce similar progressions of receptive fields corresponding to the lesioned portion of the retina. Instead, receptive fields cluster around the margins of the lesion (or the projection of the lesion into the visual field), and some recording sites produce split receptive fields on opposite sides of the lesion (B1). A dorsal view of the cat brain on the lower left shows the location of area 17 in the right hemisphere. An enlargement of a portion of cortex indicates recording sites in area 17, including a portion schematically unfolded from the medial wall of the hemisphere. The long dashed line marks the lateral border of area 17 corresponding to the zero vertical meridian (line of decussation), and the short dashed line marks the zero horizontal meridian separating the rostral representation separating the rostral representation of the lower visual quadrant from the lower representation of the upper visual quadrant. Recordings in area 18, the second visual area, revealed a similar reorganization. Based on Kaas et al (1990).
after the lesion revealed a narrow fringe of cortex around the deactivated zone in MT where neurons could be activated, but had slightly displaced receptive fields that corresponded to parts of the retina still represented in area 17. In related experiments in macaque monkeys, Dwayne et al (1987) recorded from neurons in MT before and over days after a partial lesion of MT, and found a scattering of neurons with expanded receptive fields.

After lesions of the retina, retinotopic changes similar to those observed in the cortex, but of lesser magnitude, have been reported for the lateral geniculate nucleus (Eysel et al 1980, 1981, Eysel 1982). Small, 3–4° lesions placed in the paracentral retina of adult cats completely deactivated a narrow column of neurons in a layer of contralateral LGN. After 1 month or more of recovery, however, such neurons regained excitability to visual stimuli within receptive fields displaced from the region of the lesion to the immediate surround of the lesion. The spread of excitation into the deactivated projection column in the LGN was estimated to be of the order of 100–200 μm. Lesions producing larger zones of deactivation resulted in a persistence of unresponsive neurons, except at the border of the deprived zone.

REORGANIZATION OF AUDITORY CORTEX

The possibility of reorganization of auditory cortex has been investigated recently by placing small unilateral lesions in the cochlea of adult guinea pigs (Robertson & Irvine 1989). Guinea pigs have two tonotopically organized areas in auditory cortex that have been termed the caudal and rostral fields (Robertson & Irvine 1989) because homologies between these fields and the primary field A-I and the primary-like field R (rostral) of primates and squirrels and A (anterior) of cats remain uncertain (see Luethke et al 1988 for review). Immediately after the cochlear lesions, neurons within the deprived isofrequency bands in the rostral field were depressed, but could be activated by more intense tones of frequencies spared by the lesion (increases of 30+ dB). After one month or more, neurons in the deprived cortex had response thresholds that were close to normal for sound frequencies adjacent to the frequency range damaged by the lesion. More limited results suggested that a similar change occurred in the caudal auditory field. Thus, the auditory cortex of adult mammals has the capacity to expand the representations of sound frequencies related to intact portions of the cochlea into zones of cortex deprived of normal sources of activation by cochlear damage (Figure 4). Such potential for reorganization apparently exists in at least two auditory fields (also see Weinberger & Diamond 1987).
Figure 4  The reorganization of auditory cortex after partial lesions of the cochlea. Lesions of a part of cochlea devoted to a restricted range of auditory frequencies deprived a zone of the contralateral auditory cortex of its normal source of activation. Initially, neurons in the deprived zone had greatly depressed responsiveness to auditory stimuli, but over one month or more, these neurons became highly responsive to frequencies represented in adjoining isofrequency bands. The location of the auditory cortex is shown on a dorsolateral view of the guinea pig brain above. Recordings were from a rostral auditory field that is primary-like, and may be A-I (see text). Reorganization was also noted in an adjoining posterior auditory field. Normal “A-I” is depicted on the lower left, which shows a rostrocaudal progression of the representation of low-to-high frequencies and the dorsoventral course of isofrequency lines. Frequency change is gradual rather than stepwise as shown. A cochlear lesion produces a profoundly deprived zone of cortex, which is later activated by inputs from adjoining intact portions of the cochlea, as shown in the lower right. Based on Robertson & Irvine (1989).
REORGANIZATION OF MOTOR CORTEX

The motor cortex of mammals includes a primary field, M-I, and typically several other motor fields. Systematic maps or representations of motor movements have been demonstrated in M-I of rats and other mammals by electrical stimulation; the most detailed maps are produced by low levels of current passed through the tip of microelectrodes that penetrate to the deeper layers of cortex. Although strong or repetitive electrical stimulation of the cortex can alter the types of movements produced at cortical sites (e.g. Nudo & Merzenich 1987, Brown & Sherrington 1912), thereby demonstrating a form of plasticity in the motor cortex of adults, the map can be quite stable over time when near-threshold levels of stimulation are used (Craigs & Rushton 1976). Nevertheless, recent experiments have shown that the portions of M-I of adult rats can change if parts of final motor pathways are eliminated (Donoghue et al 1990, Sanes et al 1988, 1990). In these experiments, sectioning of a motor nerve to muscles of part of the body or the removal of a part of the body have been used to eliminate the possibility of movement for these body parts. The immediate effect in cortex was that the stimulation of sites that formerly moved the inactivated or removed body part no longer produced movements.

Within hours of the lesions, however, stimulation of these sites resulted in the activation of new sets of muscles (Figure 5). For forelimb amputations, representation of the shoulder in M-I enlarged. After section of the branches of the facial nerve that innervate the musculature for the facial vibrissae, the representations of both the eyelid and eye and the forelimb regions enlarged.

MECHANISMS OF REORGANIZATION

The two main mechanisms proposed for the observed changes in sensory and motor maps have been the growth (collateral sprouting) of new connections, and alterations in the effectiveness of previously existing connections by forming new synaptic contacts or potentiating existing synapses (see Wall 1977, Devor & Wall 1981, Kaas et al 1983, Pearson et al 1987, Wall 1988). Since many of the changes are quite rapid, over hours to days, most investigators favor the interpretation that at least these early changes are the result of potentiating previously existing connections. This interpretation seems possible in that the magnitudes of the cortical changes are often, but not always (e.g. see Rasmusson & Nance 1986), within the extents of individual thalamocortical axons, which can be 1 mm or more in width (e.g. Landry et al 1982, Florence & Casagrande 1987, Garraghty...
Facial nerve section

Figure 5  Reorganization of the primary motor cortex (M-I) after section of the facial nerve to the musculature of the facial vibrissa. A dorsolateral view of a rat brain above shows the locations of the primary somatosensory (S-I) and motor (M-I) areas. The normal organization of the motor map is approximated on the lower left. Weeks after section of branches of the facial nerve that innervate the vibrissa, electrical stimulation of cortex formerly devoted to producing movements of vibrissa, instead produced eye and eyelid as well as forelimb movements. Thus, the effective eye and forelimb regions of M-I that were enlarged into vibrissa cortex deprived by inactivation of the effectors. Other experiments involving forelimb amputation enlarged the representation of shoulder movements. Based on Sanes et al (1990). N, neck; Tr, trunk.

et al 1989). Furthermore, somatotopically “inappropriate” projections of thalamic neurons with receptive fields on one toe to cortex representing another toe have been demonstrated electrophysiologically in cats (Snow et al 1988). Thus, expansions of representations could occur simply by extending the activating effectiveness of synapses from the dense core to
the sparse fringe within the terminal arbors of axons when the fringe extends into the deprived zone of cortex. Of course, lateral intrinsic connections within representations, which can be quite extensive (e.g. Rockland & Lund 1982, DeFelipe et al 1986), could also increase the zone of effective spread, and changes relayed from one to the next level in a cascading fashion could ultimately become very extensive. In addition, the dendritic branches of individual neurons can extend over hundreds of microns. Finally, at least some structures appear to have maps of inputs that are normally subthreshold or unexpressed, but they may gain potency when the inputs of the dominant map are removed (see Rhoades et al 1987). Thus, existing connection patterns are widespread enough to be compatible with the extents of most or all observed reorganizations. The growth of axons (see Goldberger 1981) and dendrites (see Purves & Hadley 1985) may indeed contribute, but such growth may not be of enough magnitude to be the major factor in the reorganization of brain maps (see Rodin & Kruger 1984, Rodin et al 1983, Stelzner & Keating 1977, Baisden et al 1980, Rasmusson & Nance 1986, Rasmusson 1988).

A number of ways of increasing synaptic effectiveness have been postulated (e.g. Wall 1977, Wall et al 1989a). An immediate enhancement of previously existing excitatory pathways may result by the deactivation of tangentially spreading inhibitory connections, while other, more slowly developing potentiations may follow the reduction of inhibition as a result of activity-dependent regulation of neurotransmitters and metabolic enzymes. For example, nerve section (Garraghty et al 1990a) and eye removal (Hendry & Jones 1988) reduce the expression of GABA in cortex, and presumably increase the excitability of neurons (see also Dykes et al 1984). The renewal of synaptic boutons is a feature of reorganizing cortex after lesions (see Ganchrow & Bernstein 1981), and alterations in synapse shape, number, size and type may be important in map plasticity (Greenough & Chang 1985; Markus & Petit 1989). The blocking of retinal impulses in adult monkeys appears to induce changes in neuronal structure and synapses in visual cortex (Wong-Riley et al 1989), and removing thalamic projections to motor cortex is followed by an increase in synapses related to previously existing inputs from parietal cortex (Ichikawa et al 1987).

Another feature of reorganization is that it sometimes, but not always, appears to be topographic. The changes described in the visual cortex appear to be simply of reactivation without retinotopic remodeling, and the reactivation of digit cortex in raccoons does not create a somatotopic order within the reactivated zone. However, months after digit removal (Merzenich et al 1984) or nerve section (Merzenich et al 1983a) in monkeys, there is evidence for the presence of topographically ordered represen-
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tations of preserved inputs in the deprived cortex. The emergence of such order could require factors in addition to the increases of effectiveness of previous connections as a result of the deactivation of alternative inputs. Thus, theories of synapse selection based on coactivation and competition have been proposed as mechanisms for topographic "self-organization" (see Willishaw & von der Malsburg 1976, Pearson et al 1987, Merzenich 1987).

Recent research suggests a role for the excitatory amino acid receptor, N-methyl-D-aspartate (NMDA) in cortical reorganization (see Cotman et al 1988). The NMDA receptor appears to regulate the flow of calcium ions into neurons, and the presence of calcium may be a factor in strengthening synapses. The NMDA receptor has been implicated in use-dependent development plasticity in the visual cortex (Gruel et al 1988, Kleinschmidt et al 1987, Bear et al 1987), and the receptor may have a similar role in adult plasticity.

CONCLUSIONS

The reviewed research supports a number of conclusions on the nature and extent of reorganization of sensory and motor maps in adult mammals.

1. Reorganization of sensory maps follows changes in neural activity patterns induced by the relative inactivation of feedforward pathways produced by lesions or removals of sensory surfaces, peripheral nerves, or more central structures. Motor maps reorganize by changing the effectiveness of outputs to remaining effectors when the access to the preferred effectors is blocked by removal or lesions.
2. Increases in the relative activity of parts of pathways in a sensory system can increase the sizes of the representations of those parts in central maps. Alterations are produced by both sensory and electrical stimulation.
3. Ablation of parts of representations can be followed by partial or complete recovery of those parts in reorganized representations.
4. Reactivation sometimes, but not always, creates a topographic pattern.
5. Sensory maps both early and late in processing hierarchies are mutable, but changes are more dramatic in higher stations, possibly because of the accumulation of serial changes and greater potential for plasticity.
6. Sensory maps in the somatosensory, visual, and auditory systems are capable of change.
7. Sensory maps in a range of mammalian species have been shown to change. The clear implication is that adult plasticity is a feature of all mammalian brains.
8. Some changes are so rapid, within hours, that the potentiation and modification of existing synapses rather than growth of new connections is the likely explanation. Since other modifications appear to take place over longer periods of weeks and perhaps months, sprouting of central axons may be an additional factor, but clear experimental evidence is needed. Sprouting may be much more significant in developing brains. The emergence of topographic features in some reorganizations may depend on synapse selection based on competitive interactions and temporal correlations and discorrelations in neural activity.

9. There is presently little evidence related to the behavioral consequences of map reorganization, if any, but partial recoveries of lost abilities following central damage and improvements and alterations in sensory and motor skills are possibilities (see Merzenich et al 1988, Wall & Kaas 1985).

ACKNOWLEDGMENTS

I thank P. E. Garraghty and S. L. Florence for comments on the manuscript.

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