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Compensatory plasticity and sensory substitution in the cerebral cortex

Josef P. Rauschecker

Cats deprived visually from birth show few overt impairments in their natural behavior. Therefore, they seem well suited as an animal model for the study of compensatory plasticity after early vision loss. It can be demonstrated that binocularly deprived cats show improved abilities of auditory localization, and at least equal tactile behavior compared to normal controls. Within the anterior ectosylvian cortex of binocularly deprived cats, where different sensory modalities come together, the anterior ectosylvian visual area is completely taken over by auditory and somatosensory inputs. Furthermore, the auditory spatial tuning of single units in this cortical region is sharpened significantly as a result of visual deprivation. Somatosensory compensation for early loss of vision can be demonstrated by a hypertrophy of the facial vibrissae, and a corresponding expansion of their central representation in the somatosensory cortex of binocularly deprived animals. The compensatory changes in the cortex can be explained by a reorganization of sensory representations under the guidance of sensorimotor feedback rather than by instruction through an extraneous 'supervisory' signal. These processes might form the neural basis of sensory substitution in blind humans.

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DO BLIND PEOPLE develop capacities of their remaining senses that exceed those of sighted individuals? This has been a question of debate for a long time[1]. Anecdotal evidence in favor of this hypothesis abounds. There are many examples of brilliant, blind musicians, including Louis Braille himself who, blinded at the age of three, later developed a system for reading and writing using tactile cues. Obviously, this system was based on the assumption that the blind have heightened sensitivity in their finger tips. A number of systematic studies have provided experimental evidence for compensatory plasticity in blind humans[2–7].

By contrast, empiricist scholars have argued often that blind individuals should have perceptual and learning disabilities in their other senses also, because vision is needed to 'instruct' them[8–10]. Without vision, the argument goes, neither a sense of space nor real knowledge of gestalt can be developed. Auditory space *per se*, it is asserted, does not exist, but has to be calibrated by vision, and visualization is needed for auditory- or tactile-form perception. This hypothesis receives support from an almost equal number of studies as the other hypothesis[11–13].

Thus, the question of whether intermodal plasticity exists has remained one of the most vexing problems in cognitive neuroscience[14–16]. One approach to solving the puzzle is to reduce it to the neural level, and develop an animal model. This would then enable the neural mechanisms underlying possible structural and functional changes in compensatory plasticity to be elucidated. An understanding of the neural mechanisms is also a necessary requirement for possible treatment, including the development of effective neural prostheses.

An animal model for human blindness, which has been used in neurobiological studies first by Wiesel and Hubel[17], is the binocularly lid-sutured cat. While some diffuse light can still reach the retina through the closed lids, all pattern vision is prevented, and the animals can, in effect, be regarded 'blind'. Lid suture can have physiological consequences that are

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different from dark-rearing or enucleation. However, it is preferable to enucleation as an experimental tool because, after the eyelids are reopened at the end of visual deprivation, any remaining visual functions in the brain can still be tested through the intact eyes. Lid suture is also preferable to dark-rearing in most cases, because it is difficult to provide a facility that can guarantee total darkness at all times.

Sound localization in blind cats

If binocularly deprived (BD) cats are to be used effectively as a model for human compensatory plasticity, the behavioral analogy has to be established first. In other words, the cats have to be trained to perform a task, such as auditory localization, that is considered critical for the question whether blind individuals improve or deteriorate in their ability to use their remaining senses. A number of studies have tried to tackle this problem in blind humans, and have obtained very different results (in favor of improvement: for example Rice *et al.*[3], Juurmaa and Suonio[4] and Muchnik *et al.*[7]; against: for example Spigelman[13] and Fisher[18]). The careful study by Muchnik and colleagues[7], for example, compared a total of 56 blind and 40 sighted subjects in various auditory tasks, among them their ability to localize sounds in eight different directions. It was confirmed that all subjects were without any history of neurological dysfunction, and had passed a hearing screening test. In all tasks, the blind subjects performed better than the sighted controls; in the auditory localization task specifically, blind subjects made fewer errors and had significantly better acuity ($P<0.01$). By contrast, the frequently cited study by Fisher[18], which measured sound localization in five blind and five sighted subjects, found no difference in their 'precision', that is, their resolution of relative sound location. Furthermore, the blind subjects consistently placed the presumed sound source at a wrong absolute location in space, that is, they appeared to have a reduced 'accuracy' of sound localization. However, this was attributed to the fact that the head position was not controlled.

In a design similar to that used by Muchnik and colleagues, BD cats, lid-sutured from birth, were trained to localize brief sounds, presented randomly at eight different locations, by walking towards the sounds' assumed azimuth position[19] (Fig. 1A). When localizing correctly, that is, within a certain criterion, the cats received a food reward. The comparison of localization error in BD and normal cats revealed that BD cats are more precise than sighted cats in localizing a sound source in space (Fig. 1B). The improvement was greatest in the rear-lateral positions, as has also been found in another human study[3]. In no case was a BD cat 'inferior' to a normal control.

Critics of the compensatory plasticity hypothesis might object that increased precision alone does not necessarily mean better localization performance, if (due to deficient calibration) the absolute position of the sound cannot be determined with the same accuracy as in normal controls[20,21]. In other words, similar to the subjects in Fisher's[18] study, blind cats might localize sounds with great precision but consistently at the wrong location in space. However, such a steady deviation from the true location is not apparent in BD cats: accuracy is normal or even

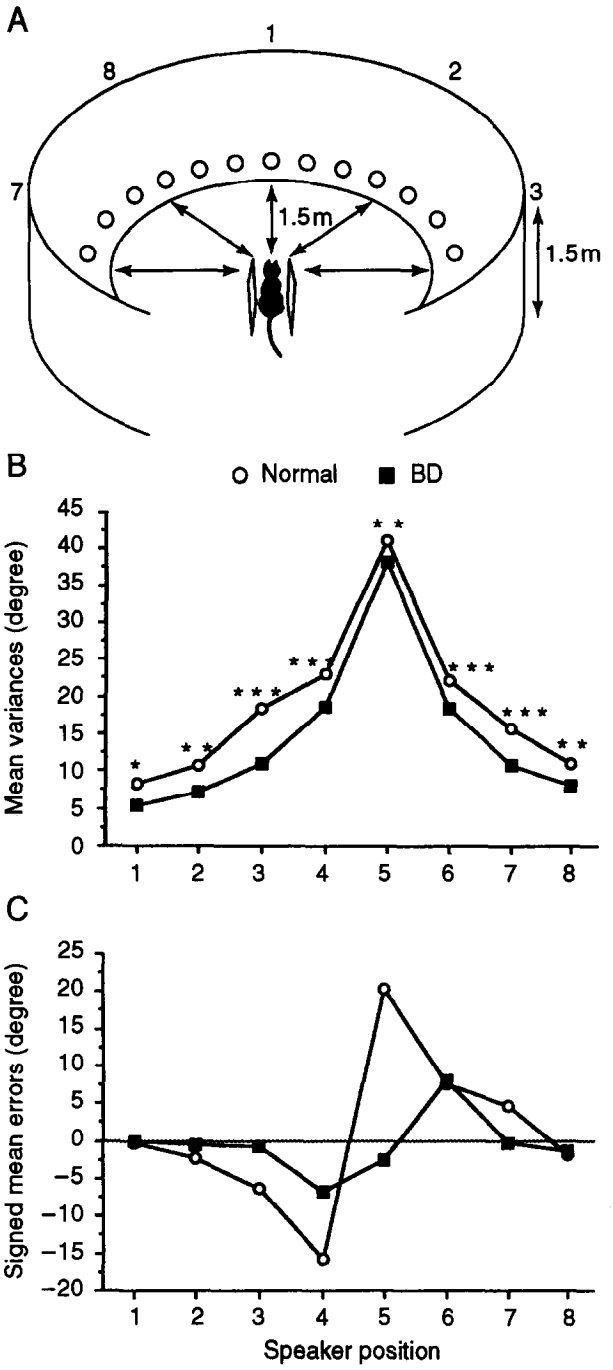

Fig. 1. ***Sound localization in binocularly deprived (BD) cats.*** *The animals were trained to walk toward a brief sound that was presented randomly at eight different azimuth positions. The distribution of sound localization error at each location was measured and compared with normal controls.* **(A)** *Behavioral testing apparatus, as installed in an anechoic room. Loudspeakers were mounted behind the arena walls 45° apart, from '1' (straight ahead) through '3' (90° to the right), '5' (straight behind) and '7' (90° to the left) with sham speakers in between. Positions 4, 5 and 6 are not shown. For more details, see Ref. 19.* **(B)** *'Precision' and* **(C)** *'accuracy' of sound localization compared with sighted controls. Precision is related inversely to the width of the distribution of sound localization error; accuracy is related inversely to the deviation of its mean from zero. Precision is improved significantly in blind cats ($P<0.002$; two-way ANOVA) with the greatest improvement at rear-lateral positions (3, 4 and 6, 7). Different significance levels of pairwise comparisons are indicated with a different number of asterisks. Accuracy is the same in both groups, when signed mean errors are assessed; it is also improved for blind cats ($P<0.02$; two-way ANOVA), when absolute mean errors are used* (modified from Ref. 19).

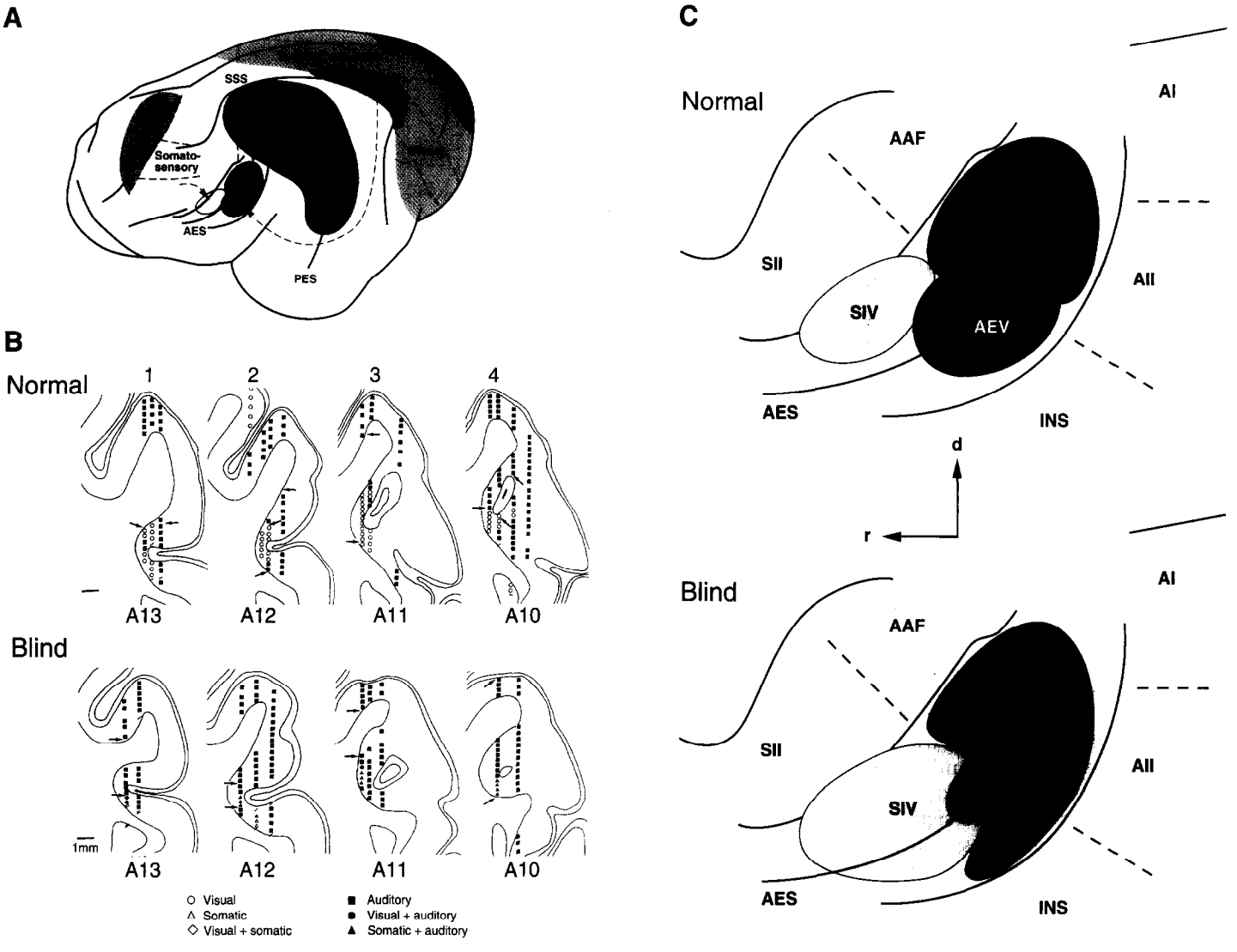

Fig. 2. ***Expansion of auditory and somatosensory regions into normally visual territory in anterior ectosylvian (AE) cortex after visual deprivation from birth.*** **(A)** *Lateral view of a cat's brain shows the site of AE sulcus (AES) as a region in which input from different sensory modalities comes together. Abbreviations: SSS, suprasylvian sulcus; and PES, and posterior ectosylvian sulcus.* **(B)** *Electrode tracks through the AE region from semichronic recordings in normal and BD cats. A purely visual area (AEV), which is found in normal cats, is absent in binocularly deprived animals (from Refs 26 and 28).* **(C)** *Schematic display of the crossmodal expansion in AE cortex. Abbreviations: AI and AII, primary and secondary auditory field; AAF, anterior auditory field; AEA, anterior ectosylvian auditory area; AEV, anterior ectosylvian visual area; SII, second somatosensory area; SIV, fourth somatosensory area*[29]*; and INS, insular cortex.*

improved (Fig. 1C). It is important to note that to reduce error, head position was always kept constant during presentation of the brief sounds.

Having demonstrated the improvement of sound localization in early blind cats, the next question that arises is whether sighted cats with sufficient training could eventually perform equally well. While the normal controls received the same amount of training as the BD cats in this specific task, this does not take into account the 'natural training' that BD cats receive by being forced to use auditory cues for orientation throughout life. One of the key questions in compensatory plasticity is, therefore, whether reducing visual activation, especially early in life, is necessary for an improvement of nonvisual modalities by providing a competitive advantage to them. Alternatively, or in addition, it could be that the increased attention devoted to these other senses helps to sharpen them. The answer to this question can come only from considering the neural basis of compensatory changes in the brain. A comparison between early and late blind cats would also be useful in this context, but at present this comparison remains equivocal[19].

Central correlates of auditory compensation

In mammals, including humans, the executive mechanisms responsible for auditory localization seem to be located mostly at the cortical level, because sound-localization ability is disturbed profoundly after cortical lesions[22–24]. Therefore, any neuronal changes underlying the improvement of sound localization in blind cats have to be sought first in the various areas of auditory cortex. The auditory portion of the anterior ectosylvian (AE) cortex has emerged recently as being possibly specialized for sound localization[25–27]. In the AE sulcus, representations of the three main modalities (visual, auditory and somatosensory) are located, with some overlap, in close vicinity to each other (Fig. 2A). The auditory part of the AE sulcus ['field AES' (Ref. 30) or AEA (Ref. 28)] adjoins another auditory area on the AE gyrus [classically referred to as the anterior auditory field[31,32] (AAF or A)]. Large numbers of spatially tuned neurons have been found in both of these

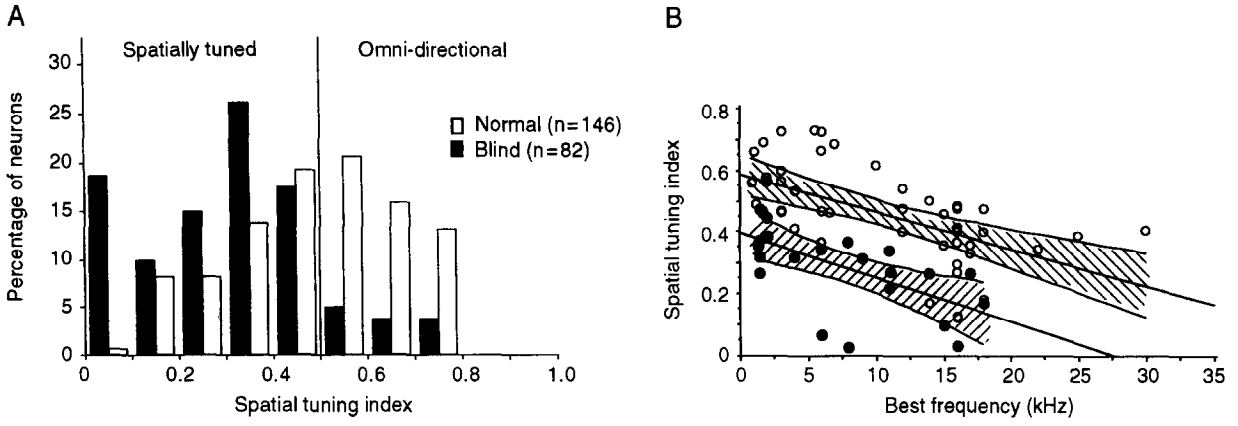

Fig. 3. ***Auditory spatial tuning of single neurons in anterior ectosylvian cortex after visual deprivation from birth and in normal controls.*** **(A)** *Spatial tuning index*[83] *is overall sharper in blind cats; fewer neurons are 'omnidirectional', as defined by a spatial tuning index of less than 0.5. The spatial tuning index is defined as the ratio between the smallest and largest response to stimulation from different speaker positions*[33]*. Other indicators of sharpness of spatial tuning, for example, the halfwidth of spatial tuning curves, give the same result.* **(B)** *The sharpening of spatial tuning holds throughout the whole frequency range. Filled circles represent blind cats; open circles represent normal controls. Regression lines and 95%-confidence intervals (hatched areas) for both groups are shown. Best frequencies are those frequencies of a pure-tone stimulus that lead to a single-unit response with the lowest threshold or elicit the highest number of spikes in a peri-stimulus time histogram* (from Ref. 26).

fields, which together have been termed the AE region[28].

The question related to compensatory plasticity is twofold: is the number of sharply tuned auditory spatial filters in the AE region increased, and is the overall sharpness of their spatial tuning enhanced as a result of visual deprivation? Studies in my laboratory, conducted in BD cats reared under the same conditions as those used in behavioral testing, revealed that this is indeed the case (Fig. 3). Furthermore, the portion of the AES in which neurons can be activated by auditory stimuli is expanded vastly, and the part of AES that is purely visual in normal cats {anterior ectosylvian visual area [EVA (Ref. 34) or AEV (Ref. 35)]} has almost disappeared. It is taken over by auditory and (in its rostral portion) somatosensory inputs, and only some bimodally activated visual neurons remain[28] (see Fig. 2).

It appears, therefore, that the behavioral improvement of sound localization ability in blind cats could be explained by these neural changes: the sharpening of auditory spatial filters and the increased number of such spatially tuned neurons in AES which, together, refine the grain of a spatially filtered auditory environment. Of course, the ultimate proof for this conclusion has to await further experimentation, for example, by selective lesioning of AE cortex.

Since spatially filtered auditory information has to be transformed into motor commands, most likely via the superior colliculus (SC), before it can lead to improved behavioral performance, the ties from the auditory cortex to the SC (Ref. 36) are of interest too. Indeed, the size of the projection from the AE to the SC is increased in BD cats[37]. In addition, it has been shown that the SC of BD animals contains a higher proportion of auditory neurons[38–40]. This can now be reinterpreted as reflecting the changes in the cortex and the corticotectal pathways. A potential problem is the matching of the reorganized auditory cortical input, which does not seem to be arranged as a spatial representation in its auditory domain, to the space map in the SC. One has to be cautious, therefore, in interpreting changes in the topography of the SC after early blindness[20,41,42]. Especially in animals that move their eyes, the relationship between their visual and auditory maps might not be fixed[43,44]. Instead, head-centered sensory maps are replaced in the SC by a map of motor error[45]. Recalibration of this map might require unexpected adjustments in response to the reorganization at the cortical level.

Somatosensory compensation for early blindness

That somatosensory information is used to compensate for the loss of vision in blind individuals has often been hypothesized also[4]. Again, the rival hypothesis has claimed that spatial orientation by the blind on the basis of tactile cues should deteriorate, because vision might be required to establish all knowledge of spatial relations[9,11]. When blind cats are tested in a spatial-maze task, they are not impaired in learning and solving the task, even if it is changed from trial to trial[46,47]. On closer inspection, it becomes obvious that the blind cats make extensive use of their facial vibrissae in forming a spatial image (or 'cognitive map') of their environment[47].

One curious observation is the hypertrophy of facial vibrissae in BD cats[48,49], which essentially leads to an increased range of these important tactile organs. The mechanism for this hypertrophy has yet to be determined, but the most plausible interpretation is that the increased usage of the whiskers in BD cats leads to stimulation of growth factors located in the whisker pads. In search for a possible central correlate of this hypertrophy, we turned to a different species, the pigmented mouse which, like all rodents, has a highly specialized region in its somatosensory cortex for the representation of its facial vibrissae. In this study, for technical reasons only, the eyes were removed at birth rather than lid-sutured, and deprivation continued for several months. In flatmounts of the cerebral hemispheres, which were stained either for cytochrome oxidase or for Nissl bodies, the barrel field representing the facial vibrissae was expanded significantly compared with normal littermates;

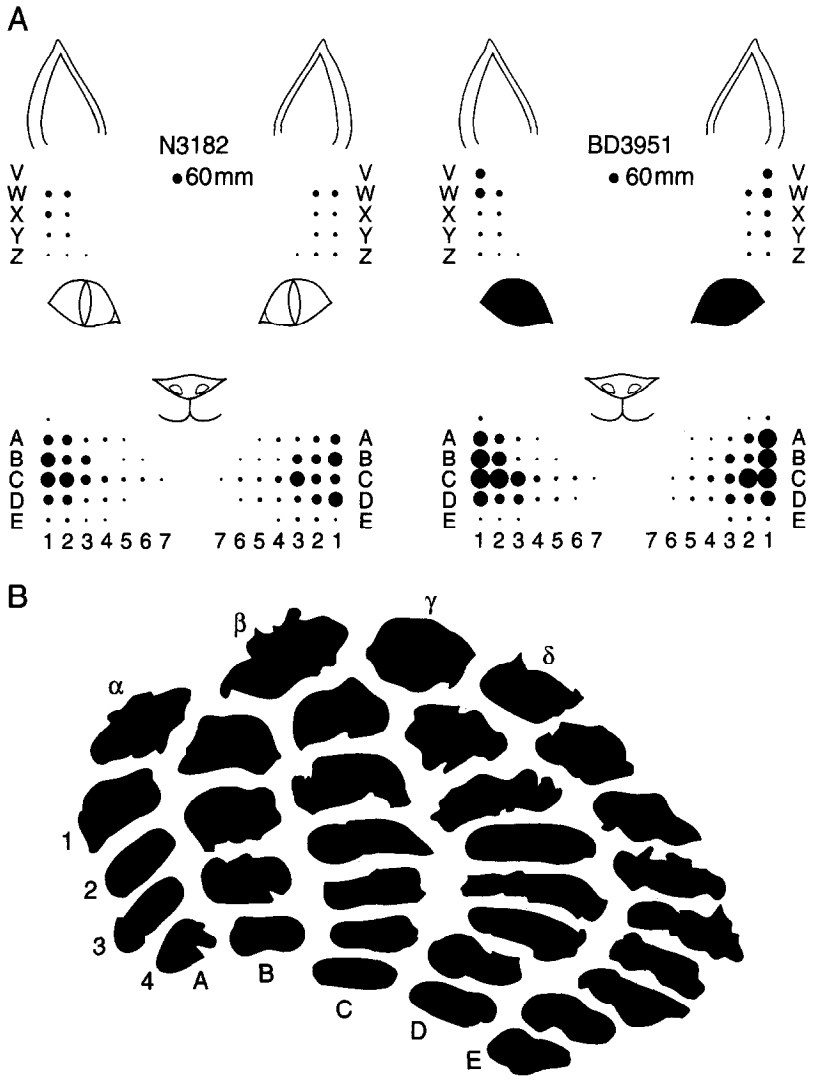

Fig. 4. ***Signs of somatosensory compensation in visually deprived animals.*** **(A)** *Hypertrophy of facial vibrissae in a blind cat* **(right)** *as compared to a normal control* **(left)**. *Each whisker position is identified by a row (A–E and V–Z) and a column (1–7). Diameter of circles in each position is proportional to the length of the corresponding whisker* (from Ref. 49). **(B)** *Expansion of the whisker barrel field in the somatosensory cortex of neonatally enucleated mice. Average barrel size in the blind group is compared with that in normal litter mates. The overall difference in size is highly significant ($P<0.0001$, t test). Most individual barrel positions also show a significant difference, as indicated by a different number of asterisks (significance levels: $P<0.05$ to $P<0.0005$; binomial test). Letters and numbers again refer to rows and columns of barrel position* (from Ref. 49).

individual barrels were enlarged by up to one-third[49] (Fig. 4). In addition, binocularly enucleated (BE) mice showed a similar hypertrophy of the vibrissae as BD cats showed.

When the total size of the flatmounted cerebral hemispheres is compared in normal and BE mice, they do not differ significantly. Therefore, the enlargement of the barrel field seems to occur at the expense of other cortical regions. In rhesus monkeys, it has been shown that visual cortex is reduced significantly in size after binocular enucleation[50]. Thus, it might be concluded that the barrel fields (and other somatosensory regions) in BE mice, like auditory regions in BD cats, also expand into formerly visual territory. The expansion of the barrel field in neonatally enucleated rodents was subsequently confirmed independently[51,52]. A takeover by auditory and somatosensory input of brain regions that are normally visually activated has also been reported in the naturally blind mole rat[53,54].

Sensory substitution in humans

With the advent of computerized measuring techniques, electric or magnetic surface potentials recorded from the human brain in response to sensory stimuli can now also be localized much more precisely. Measurements of regional cerebral blood flow or of differences in blood oxygenation levels using positron emission tomography (PET) and functional nuclear magnetic resonance imaging (MRI), respectively, in conjunction with modern imaging techniques, advance the understanding of localized events in the human brain even further. Studies that measure event-related potentials (ERP) have established that regions in the parietal cortex are activated more strongly by moving visual stimuli in deaf than in normal individuals[16]. In addition, deaf subjects responded more quickly and more accurately than hearing subjects to the visual stimuli. Functional MRI reveals a profound reorganization of language areas in the brain of deaf subjects using sign language[55]. Recent results of ERP and PET or single photon emission computed tomography (SPECT) studies in blind humans indicate activation of areas that are normally visual during auditory stimulation[56,57], haptic mental rotation[58] or Braille reading[59]. Transcranial magnetic stimulation shows an expansion in the representation of the reading finger in sensorimotor cortex of Braille readers[60].

These results correspond extremely well with the findings in visually deprived cats, and confirm the validity of that animal model for studies of early blindness and compensatory plasticity. The findings, in cats, humans and monkeys[61], of activation by auditory or somatosensory stimuli of brain regions designed for the processing of vision, show the extreme plasticity of the brain in adapting to changes in its environment. However, these findings also pose an interesting philosophical question: what is the kind of percept that a blind individual experiences when a 'visual' area becomes activated by an auditory or tactile stimulus? Do blind individuals 'see' their environment with their tactile senses, as has been suggested by the term 'facial vision'[62]? Do they see sounds in ways similar to a sonar system[2]? Or does the visual area simply get transformed into an auditory or somatosensory representation by the new type of input? In other words, is the percept determined by the type of sensory input or by the (functionally preordained) brain region that receives it? Undoubtedly, an auditory stimulus will still be perceived as a sound by blind individuals, because primary auditory regions are also activated. However, does the co-activation of 'visual' regions add anything to the quality of this sound that is not perceived normally, or does the expansion of auditory territory simply enhance the acuteness of perception for auditory stimuli?

A common code for sensorimotor integration

The answer to the above questions might be found more easily if we consider the projection targets rather than the inputs of a brain region. In order for a behavioral reaction to a particular stimulus to be appropriate, it is necessary that, at the interface, the same code is used regardless of sensory modality[45]. Thus, a cortical module at any one processing level applies the same type of operation

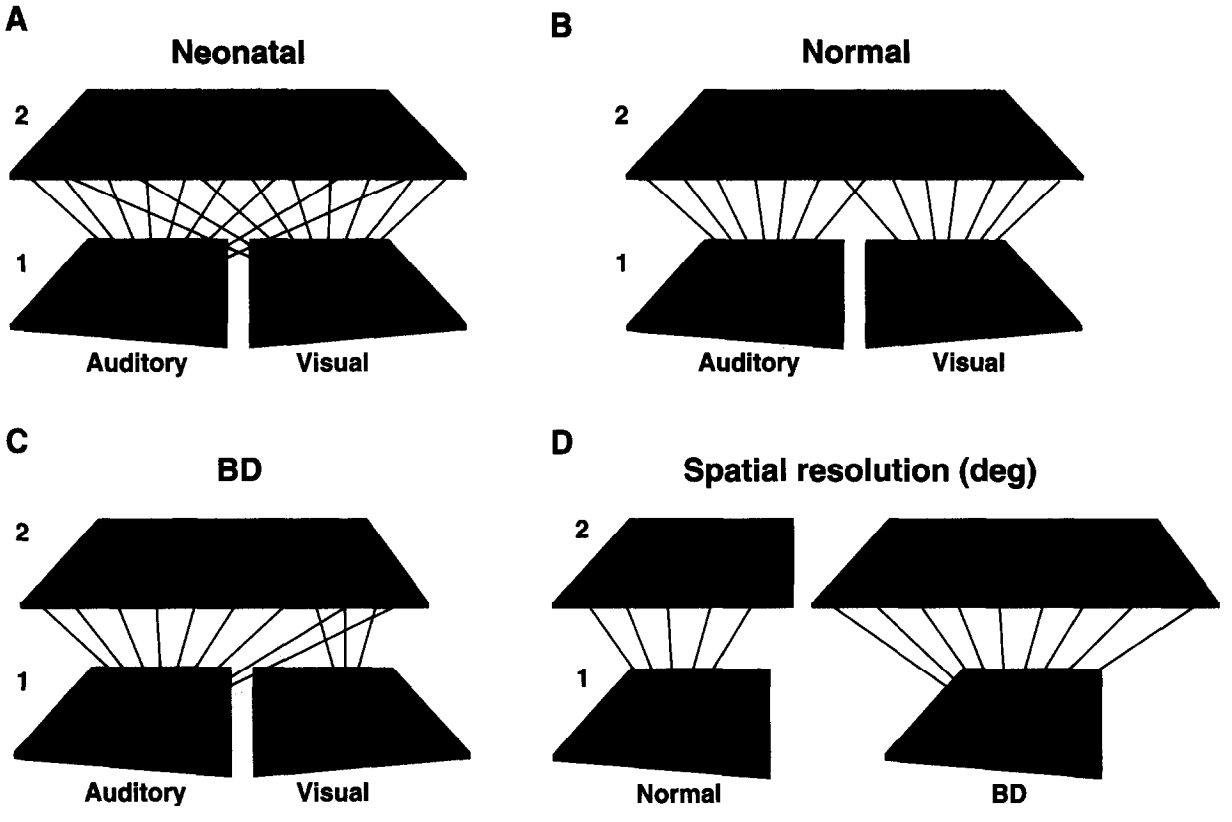

Fig. 5. ***Schematic representation of compensatory plasticity by changes in synaptic connectivity.*** *The example of auditory compensation for visual deprivation in anterior ectosylvian (AE) cortex*[28] *is used for illustration.* **(A)** *The initial condition in newborn cats is assumed to be highly 'exuberant'*[82] *with a large region of multimodal overlap.* **(B)** *In cats reared normally, the overlapping region is reduced by activity-dependent selection. Some connections, rather than being uncoupled completely, might become suppressed by inhibition and remain as 'masked' silent inputs.* **(C)** *Binocular deprivation (BD) provides a competitive advantage to auditory input, which takes over a larger part of AE region than normal, and a larger portion remains multimodal. Similar models could be developed with an initial condition of sparse overlap and ensuing sprouting of new connections.* **(D)** *The sharpened spatial tuning of auditory units (Fig. 3 and Ref. 26) can be explained within the same model. The expanded region of cortex, with a larger number of neurons, accommodates the same range of positions in auditory space. Therefore, a given number of neurons is devoted to the processing of smaller angles in binocularly deprived cats, which provides the system with better spatial resolution (higher spatial 'sampling rate'). It should be noted that no space map has yet been found in auditory cortex, as suggested here for the purpose of clarity, but the same argument holds for other forms of space coding.*

to different types of input and transforms them into a specific response. In the case of AES, input from different sensory modalities arrives in the same cortical region before being passed on to the SC, where the information converges onto single neurons[63,64].

There is good reason to believe that neighboring cortical areas share certain functional aspects, defined partly by their common projection targets. In cat AES, the function shared by all sensory modalities seems to be spatial processing[28]. Therefore, a common code for spatial information that can be interpreted by the SC has to be used. A compensatory expansion of AEA at the expense of AEV thus results in finer resolution of auditory behaviors mediated by the SC rather than in a reinterpretation of auditory signals as visual.

The functional consequences of activation of auditory cortex by 'abnormal' visual input have been discussed in another case of crossmodal plasticity, which is quite different from the present examples. In this experimental paradigm, all target regions for optic-nerve fibers in a newborn hamster or ferret are removed surgically and, at the same time, auditory (or somatosensory) afferents are destroyed. Under these conditions, optic-nerve fibers are found to innervate nonvisual thalamic regions[65,66]. Consequently, visual receptive fields are found in single units of, for example, auditory cortex. These animals, without visual cortex or optic tectum, seem to be capable of orienting towards visual stimuli[67]. Thus, it appears that visually activated auditory cortex can indeed be used for seeing.

Neural mechanisms of compensatory plasticity

No new mechanisms have to be postulated to explain the crossmodal changes involved in compensatory plasticity at the synaptic level. Various forms of changes in the afferent-activity pattern can have the same effects at the level of cortex. It is known that experience-dependent cortical plasticity involves changes of synaptic efficacy that follow Hebbian rules[68–71]. Similarly, peripheral lesions might unmask hidden inputs that do not normally lead to suprathreshold activation of a postsynaptic neuron[72–74]. Even simple behavioral training can have the same effects and lead to an apparent expansion of cortical tissue that is activated during a particular task[75]. In extreme cases, where expansions over several millimeters have been observed[76], axonal sprouting of intrinsic connections might be involved[77,78]. In all of these cases, expansion of the more active pathways or brain regions occurs at the expense of another pathway or region. This competition between neural representations with different activity levels seems to be one of the fundamental principles of cortical plasticity.

In the past, such mechanisms have been described only for changes within a single modality, where neighborhood relationships are defined on the basis of topography. Thus deprivation of one eye involves an expansion of the neighboring ocular dominance stripes from the other eye[70]. Deafferentation of the hand leads to an expansion of the adjacent face region[76]. Cochlear lesions of a certain frequency band lead to an expansion of neighboring frequencies in the auditory cortex of various species[79–81]. By contrast, in a region such as AES, where different sensory representations adjoin each other, and neighborhood relationships are defined by common function, the laws of cortical plasticity lead to changes across modality borders. Thus, visual deprivation leads to an expansion of the neighboring nonvisual areas into normally visual territory (Fig. 5).

Intermodal plasticity might involve any or all of the aforementioned neural mechanisms: unmasking of silent inputs; stabilization of normally transient connections[82]; axonal sprouting; or a combination of them. From a functional viewpoint, such intermodal plasticity enables an individual to optimize their capacities at a different level. For this conclusion to be completely valid, more careful behavioral studies need to be performed in concert with neurobiological investigations, including neuroimaging in humans.

Concluding remarks

In summary, the brain possesses the capacity to reorganize itself after peripheral injury or deprivation in such a way that it enables neighboring cortical regions to expand into territory normally occupied by input from the deprived sense organs. This plasticity might not be restricted to developmental periods, but may be available, at least to some extent, throughout life[83]. On the basis of the neural mechanisms, our question posed initially about the relative effects of deprivation versus training can now also receive at least a preliminary answer. In a competitive system, as described above, any factors that lead to increased contrast between differently active regions will affect the outcome. Thus, inactivation of one brain region by deafferentation or deprivation will accelerate the expansion of a competing pathway. At the same time, increased attention or training devoted to this other pathway will help also.

In the present scheme, representational plasticity occurs without the involvement of an extraneous, 'supervisory' signal that 'instructs' the cortical maps to change in a particular way. Spatial, cognitive maps are capable of self-organizing with the aid of sensorimotor feedback from their own target regions, which operate on the basis of a modality-independent code[84–86]. Similarly, no privileged role needs to be postulated for vision having to 'instruct' other senses[10,20] because it is the interaction of sensory with motor experiences that leads to the calibration of sensory maps[87,88]. As the role of sensorimotor feedback in compensatory plasticity becomes clearer, it might be, therefore, that the much discussed 'mobility training'[14,89] is one of the most important factors for rehabilitation in the blind.

Finally, a word of caution might be in order to calm overoptimistic interpretations. The enhanced nonvisual abilities of the blind are hardly capable of replacing fully the lost sense of vision because of the much higher information capacity of the visual channel. However, they can provide partial compensation for the lost function. With a more complete understanding of the events leading to this compensation, it might become possible to exploit this capacity for reorganization by instructing individuals with lost sensory functions to take advantage of this reorganization or by designing more sophisticated sensory prostheses[90].

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Books Received

Review copies of the following books have been received. Books which have been reviewed in Trends in Neurosciences *are not included. The appearance of a book in the list does not preclude the possibility of it being reviewed in the future.*

Louis W. Chang (ed.) *Principles of Neurotoxicology* Marcel Dekker, 1994. $195.00 (xviii + 800 pages) ISBN 0 8247 8836 2

P. Jeffrey Conn and Jitendra Patel (eds) *The Metabotropic Glutamate Receptors* Humana Press, 1994. $99.50 (x + 277 pages) ISBN 0 89603 291 4

V. Darley-Usmar and A.H.V. Schapira (eds) *Mitochondria: DNA, Proteins and Disease* Portland Press, 1994. £50.00/$80.00 (xi + 286 pages) ISBN 1 85578 042 9

Ravi Iyengar (ed.) *Heterotrimeric G-Protein Effectors (Methods in Enzymology, Vol. 238)* Academic Press, 1994. $80.00 (xxix + 456 pages) ISBN 0 12 182139 0

B. Jönsson and J. Rosenbaum (eds) *Health Economics of Depression (Perspectives in Psychiatry, Vol. 4)* John Wiley & Sons, 1993. £39.95 (viii + 153 pages) ISBN 0 471 93746 0

Michael A. Kaliner, Peter J. Barnes, Gert H.H. Kunkel and James N. Baranuik (eds) *Neuropeptides in Respiratory Medicine (Clinical Allergy and Immunology, Vol. 4)* Marcel Dekker, 1994. $195.00 (xv + 693 pages) ISBN 0 8247 9199 1

George Paxinos, Ken W.S. Ashwell and Istvan Törk *Atlas of the Developing Rat Nervous System* (2nd edn), Academic Press, 1994. $125.00 (xxvii + 552 pages) ISBN 0 12 547610 8

Nancy J. Rothwell and Frank Berkenbosch (eds) *Brain Control of Responses to Trauma* Cambridge University Press, 1994. £50.00 (ix + 342 pages) ISBN 0 521 419395

Wilhelmus J.A.J. Smeets and Anton Reiner (eds) *Phylogeny and Development of Catecholamine Systems in the CNS of Vertebrates* Cambridge University Press, 1994. £95.00 (xvi + 488 pages) ISBN 0 521 44251 6

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Lennart Stjärne, Paul Greengard, Sten E. Grillner, Tomas G.M. Hökfelt and David R. Ottoson (eds) *Molecular and Cellular Mechanisms of Neurotransmitter Release (Advances in Second Messenger and Phosphoprotein Research, Vol. 29)* Raven Press, 1994. $157.50 (xix + 569 pages) ISBN 0 7817 0220 8

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