colonies, and the genetic variance of the population is enhanced. Thus, these data best support a model of gradual evolution. The similarity between the social substructure of marmot populations and that of many other mammals suggests the generality of our conclusions (*14, 21*).

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Visual Effects of Auditory Deprivation: Common Intermodal and Intramodal Factors

Abstract. *Visual temporal acuity, as measured by the critical flicker frequency decreased and then increased during 24 hours of auditory deprivation. This intermodal effect is similar to intramodal changes in the critical flicker frequency of the nonoccluded eye during monocular deprivation; a single mechanism appears to underlie both phenomena.*

Until recently it has been assumed that different patterns of change in sensory sensitivity are generated for inter- and intramodal, nondeprived perceptual channels in response to sensory deprivation of a single modality. Unimodal deprivation results in a progressive, negatively accelerated improvement in the sensory sensitivity of nondeprived modalities for a variety of measures and modalities (*1*). These intermodality changes are predicted by and support ascending reticular activating system (ARAS) theories of sensory threshold regulation (*2*). Intramodal changes, on the other hand, are characterized by an initial depression in sensitivity followed by an enhancement phase. Thus, sensory deprivation of one eye results at first in a decrease in visual temporal acuity and then is followed by a progressive improvement of sensitivity in the nonoccluded eye (*3*). These intramodal changes have been explained on the basis of the law of denervation supersensitivity (*4*) as interpreted by Sharpless' (*5*) concept of disuse of neural pathways.

In a recent review of these areas (*6*), we have suggested that conceptual and empirical similarities between the two situations may make it fruitful to look for a common mechanism. In both cases, a compensatory change occurs in nondeprived sensory channels, the major difference being that the intramodal changes exhibit the depression-enhancement effect not observed in intermodal studies. As the short-term (less than 24 hours) intermodal effects of unimodal deprivation have not previously been investigated, an observation of the depression-enhancement component under these conditions would implicate a single rather than two separate mechanisms and open the way for a unified model to account for both intra- and intermodal effects. We now report for what we believe to be the first time that performance declines and then improves on an intermodal measure during unimodal sensory deprivation.

Twenty-two male university students were recruited and randomly divided into two groups of 11 subjects each. The experimental subjects were required to live, one at a time, for a 24-hour period at the University of Manitoba Sensory Isolation Laboratory. Auditory deprivation was achieved by confining these subjects to a sound attenuation chamber (mean reduction, 80 dB), by having them wear sound reducing (30-dB) earplugs, and by cautioning subjects against generating unnecessary noise (*7*). All living and experimental activities, including visual testing, took place in the chamber. Care was taken to ensure a patterned sensory environment for all modalities but the auditory. The deprivation setting and procedure has been discussed elsewhere (*6*). All members of both groups were instructed to have a normal night prior to the beginning of the experiment and to avoid all medications and alcohol. Since there are no outcome differences between a "confined-to-laboratory" and a nonconfined control group in auditory deprivation studies (*1, 6*), the 11 control subjects were required to report to the laboratory for testing at the same time intervals as the testing times for the experimental subjects. For comparison purposes the visual testing for critical flicker frequency (CFF) closely followed the procedure employed in previous interocular (*3*) and intersensory studies (*1*). The monocular CFF of the right eye was determined at 0, 3, 6, 9, 12, and 24 hours of deprivation for the experimental subjects and at the equivalent time for control subjects. Visual testing was preceded by a meal or a snack and by 15 minutes of dark adaptation. The CFF threshold for each subject consisted of the mean of eight trials, separated by 5-second intervals, obtained by the descending method of limits.

The stimulus consisted of a white light, at an initial frequency at a random value between 60 and 150 cycles per second. The light was presented by a cold cathode modulating lamp (Sylvania type R1131c) mounted at the rear of a standard viewing chamber (Lafayette model 1202C). The subject was required to centrally fixate the stimulus as it was presented through a Plexiglas diffuser 1.25 mm in diameter. The distance from the stimulus to the eye was 36.25 cm, and

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the visual angle subtended was 2° 10′, a value assuring full foveal stimulation. The flicker-generating apparatus (Lafayette model 12020) was set at a light-dark ratio of 0.5, and the lamp luminance during the "on" phase was approximately 25 cd/m^2.

The mean CFF of the experimental subjects was decreased at 6 hours, gradually returned to baseline, and by 24 hours of deprivation was dramatically increased over baseline values (Fig. 1). The control group, in contrast, showed relatively little variation.

A two-way analysis of variance for repeated measures revealed no group effect but reliable trials [F (1,10) = 14.90, $P < .005$] and group by trials interaction [F (5,100) = 4.69, $P < .001$] effects. Analysis of simple main effects revealed that, while the experimental group changed significantly over time [F (5,50) = 5.24, $P < .001$], the control group did not. One-tailed t-tests for related measures (with adjusted acceptance levels) showed that, at 6 hours, the experimental group showed CFF values significantly lower than baseline values [t (10) = 3.17, $P < .005$] while at 24 hours these values were significantly higher than baseline [t (10) = 2.53, $P < .025$]. Our hypothesis of depression followed by enhancement of visual temporal acuity in response to auditory deprivation was confirmed.

Our findings indicate that, when the times chosen for visual testing are the same between and within conditions of unimodal deprivation, the course of changes in sensory sensitivity are nearly identical. It seems reasonable to hypothesize a single mechanism of inter- and intrasensory deprivation effects.

The theories based on the ARAS, because they implicate a general arousal system, predict only the eventual enhancement of acuity found here (*2*). The denervation theory, on the other hand, predicts both depression and enhancement but cannot explain these cross-modal mediating effects, as changes in neural firing thresholds are confined to tissue between the brain and the partially destroyed (disused) nerves (*4, 5*). As an explanatory device for intramodal influences, denervation supersensitivity depends on the cortical receiving areas specific to the affected modality. At first look it appears much more difficult to find a site of interaction across, than within, modalities.

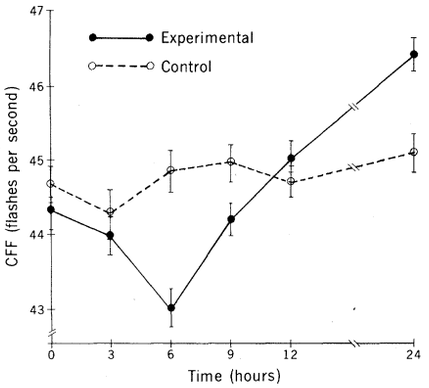

Fig. 1. Temporal changes in mean CFF values of subjects exposed to 24 hours of auditory deprivation and a nonconfined control group. The vertical bars are the standard errors for each group at each testing period.

We believe that this apparent theoretical impasse may be avoided by effectively combining the ARAS and functional denervation theories. It has long been known that the reticular formation receives afferent collaterals from all classical sensory pathways, that there is considerable convergence of these collaterals at the lower brainstem level, that activity at this level influences the ARAS at the mesencephalic level (*8*), and that stimulation at this higher level can lead to improved temporal acuity (*9*). In our model the reduced sensory input and the accompanying decrease in the firing rates at the lower reticular levels would lessen the activation operations of the ARAS. This, according to the reticular theories (*2*), will result in poorer sensitivity. When supersensitivity at the lower brainstem reticular formation sets in as a result of prolonged disuse of neural pathways (denervation), however, this supersensitivity will be passed on to the ARAS. The resultant increases in ARAS activity would (again in accordance with the ARAS theories) bring about a generalized increase in sensory sensitivity.

This unified theoretical model parsimoniously combines the ARAS and denervation theories and has the advantage of conceptual continuity and heuristic value. We recognize that generalization from our psychophysical data to the general case must await further investigations of various deprivation procedures and dependent measures, as well as that the validation of the model with reference to the underlying neurophysiological structures has to come from sensory physiologists studying the relevant systems in vivo.

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