

tion of van der Waals radii, 2.17 Å for Xe (from the element, 11), 1.35 to 1.50 for F.

It has been suggested (12) that the difference in Xe to F bond lengths of 2.00 Å in XeF₂ and 1.95 Å in XeF₄ may result from the Xe to F bonds having more *s*-orbital character in the latter compound (13).

JOHN H. BURNS

Reactor Chemistry Division

P. A. AGRON

HENRI A. LEVY

Chemistry Division, Oak Ridge

National Laboratory,*

Oak Ridge, Tennessee

References and Notes

1. R. E. Rundle, *J. Am. Chem. Soc.* **85**, 112 (1963); K. S. Pitzer, *Science* **139**, 414 (1963); L. L. Lohr and W. N. Lipscomb, *J. Am. Chem. Soc.* **85**, 240 (1963).
2. H. A. Levy and P. A. Agron, *J. Am. Chem. Soc.* **85**, 241 (1963).
3. J. A. Ibers and W. C. Hamilton, *Science* **139**, 106 (1963).
4. D. H. Templeton, A. Zalkin, J. D. Forrester, S. M. Williamson, *J. Am. Chem. Soc.* **85**, 242 (1963).
5. H. H. Claasen, H. Selig, J. G. Malm, *ibid.* **84**, 3593 (1962).
6. D. F. Smith, *J. Chem. Phys.* **38**, 270 (1963).
7. W. R. Busing and H. A. Levy, *Am. Cryst. Assoc. Boulder Meeting Abstr.*, July 31, 1961.
8. W. R. Busing, K. O. Martin, H. A. Levy, "ORFLS, A fortran crystallographic least squares program," Rept. No. TM-305, (Oak Ridge National Laboratory, 1962).
9. D. W. J. Cruickshank, *Acta Cryst.* **9**, 757 (1956).
10. W. R. Busing and H. A. Levy, unpublished data.
11. D. R. Sears and H. P. Klug, *J. Chem. Phys.* **37**, 3002 (1962).
12. P. A. Agron, G. M. Begun, H. A. Levy, A. A. Mason, C. G. Jones, D. F. Smith, *Science* **139**, 842 (1963).
13. We thank Dr. D. F. Smith of the Oak Ridge Gaseous Diffusion Plant for preparing the compound and checking its purity.

* Oak Ridge National Laboratory is operated by Union Carbide Corp. for the U.S. Atomic Energy Commission.

25 February 1963

Electroencephalographic Changes after Prolonged Sensory and Perceptual Deprivation

Abstract. Seven days' exposure to unpatterned light and white noise produced a significantly greater decrease in occipital lobe frequencies than did the same period of darkness and silence. This differential effect may be related to the greater behavioral impairments which seem to occur after prolonged exposure to diffuse light and noise.

It has been shown that the intellectual and sensorimotor impairments resulting from prolonged perceptual deprivation are greater than those occurring after prolonged sensory deprivation (1-3). These two terms are employed in the

sense advocated by Kubzansky (4), in which sensory deprivation refers to an attempt at "an absolute reduction in variety and intensity of sensory input," for example, the use of darkness and silence, whereas perceptual deprivation refers to "reduced patterning, imposed structuring, and homogeneous stimulation," for example, the use of translucent goggles, white noise, constant hum, and so forth. The purpose of this experiment is to determine whether the behavioral differences between the two conditions are accompanied by any differences in electrical activity of the brain. We already know that perceptual deprivation produces a decrease in occipital lobe frequencies (5). However, it is not known whether sensory deprivation produces a similar decrease and, if it does, whether it is of the same magnitude.

A group of 40 male university students were used, ten in each of four conditions. Each condition lasted a week. In the first, sensory deprivation, the subjects were placed individually in a dome-shaped isolation chamber. The details of this chamber are given in an earlier publication (1). They were required to lie quietly on an air mattress under constant darkness and silence (70 db attenuation). They could sit up or stand up only when eating or when using the toilet facilities located several feet away. Singing, humming, or any other vocal activity was not permitted. No gauntlet-type gloves or any other form of manual restrictions were imposed.

In the second condition, perceptual deprivation, the subjects were again isolated individually but under constant, unpatterned light and white noise. They wore a pair of translucent goggles which reduced the level of ambient illumination in the chamber from 90 to approximately 20 ft-ca (under the goggles). The goggles excluded all pattern vision. Each subject also wore a set of earmuffs through which white noise was constantly presented somewhat above the threshold of hearing. The other restrictions were similar to those in the first group.

The third condition was a control for the recumbent or prone position which the two experimental groups had to assume most of the time. In this condition the subjects were placed, in groups of three or four, in a large room near the isolation laboratory. They were required to lie quietly on air mattresses arranged parallel to one another on the floor. They were allowed to sit up only

when eating and to stand up only when going to the washroom, 15 feet away. Apart from these restrictions on gross body movements their environment was quite "normal." They were allowed to talk, read, listen to the radio, and watch television, and all lights were put out at night.

Finally, an ambulatory control condition was used. The subjects of this group merely came to the laboratory for electroencephalographic records, re-

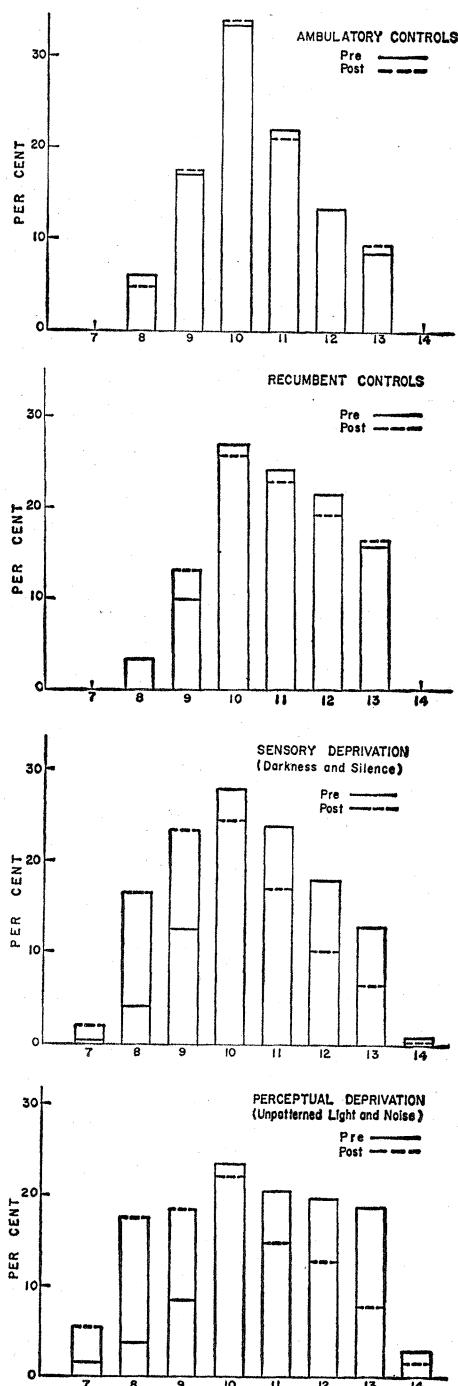


Fig. 1. Mean frequency spectrum of the two experimental and two control groups before and after the 1-week experiment. Abscissas are frequencies.

Table 1. Mean occipital lobe frequencies before and after two experimental and two control conditions (40 subjects, ten in each group).

Perceptual deprivation			Sensory deprivation			Recumbent controls			Ambulatory controls		
Pre	Post	Diff.	Pre	Post	Diff.	Pre	Post	Diff.	Pre	Post	Diff.
11.70	10.60	-1.10	10.05	9.00	-1.05	11.88	11.60	-0.28	9.99	10.09	+0.10
11.89	10.15	-1.74	10.33	10.18	-0.15	9.72	9.48	-0.24	11.84	11.97	+0.13
10.50	9.34	-1.16	10.67	9.73	-0.94	10.04	10.01	-0.03	11.50	11.53	+0.03
12.70	11.06	-1.64	10.32	8.85	-1.47	11.20	11.03	-0.17	10.50	10.47	-0.03
12.29	11.23	-1.06	11.80	11.05	-0.75	11.06	10.94	-0.12	10.22	10.23	+0.01
11.14	10.73	-0.41	11.42	10.60	-0.82	11.10	11.30	+0.20	11.04	10.79	-0.25
11.26	10.00	-1.26	11.00	10.09	-0.91	10.65	10.56	-0.09	9.65	9.59	-0.06
10.07	8.84	-1.23	10.70	9.87	-0.83	10.20	10.60	+0.40	10.37	10.49	+0.12
9.79	8.72	-1.07	10.13	9.40	-0.73	11.78	11.72	-0.06	10.02	10.30	+0.28
10.10	8.63	-1.47	11.38	10.49	-0.89	11.84	11.85	+0.01	9.79	9.53	-0.26
<i>Means</i>			<i>Means</i>			<i>Means</i>			<i>Means</i>		
11.14	9.93	-1.21	10.78	9.93	-0.85	10.95	10.91	-0.04	10.49	10.50	+0.01

turned home, and then reappeared a week later for the second record.

Electroencephalographic (EEG) tracings were taken by an Offner eight-channel machine, model D3, before and after each of the four conditions. Blood sugar level was controlled for by placing the subjects on a fixed feeding schedule and taking records in the morning, after breakfast. In order to exercise more control over possible drowsiness or decreased alertness in the experimental subjects, tracings were taken after they emerged from isolation. They were first given a battery of performance tests, breakfast, opportunity to wash up and then records were taken. These preliminary activities, which took approximately 2½ hours, eliminated any drowsiness at recording time which may have existed earlier. The controls were subjected to a similar routine. No differences in activity levels or in alertness could be detected between the experimentals and controls. In order to obtain a quantitative measure of EEG changes, two types of analyses were made. In the first, the mean occipital lobe frequency of each subject was determined. This involved counting the number of waves occurring in each of 200 1-second samples of artifact-free occipital lobe tracings. For this purpose, a cursor (a special EEG ruler) was employed. To avoid any bias the records were scored "blindly," that is, the technician was not told what groups the tracings came from or whether they were "pre" or "post" records. The second method involved a frequency spectrum analysis of the type suggested by Engel *et al.* (6). This consists of counting the number of waves in each 1-second period and expressing the number of 1-second

periods containing a particular frequency as percentages of the total 200-second period.

Table 1 shows the mean occipital frequencies of the 40 subjects before and after a week. It can be seen that all 20 experimental subjects show a post-isolation decrease in mean frequency. Furthermore, the decrease in frequency appears to be greater for the perceptual than for the sensory deprivation group. Of the former group, nine out of ten subjects show a decrease greater than 1 cy/sec, whereas only two of the latter group do so. On the other hand, the two control groups exhibit no consistent trend. Some subjects show an increase in frequency but others show a decrease. The mean "pre-post" difference for both control groups is almost zero. An analysis of variance of the "pre-post" difference scores of the four groups yielded a highly significant F ratio ($p < .001$). Subsequent *t* test analyses (2-tailed) revealed that the slight difference between the control groups was not significant ($p > .70$). However, both experimental groups differed significantly from the controls ($p < .001$). Furthermore, the mean decrease in frequency of the perceptual deprivation group was significantly greater than that of the sensory deprivation group ($p < .01$). Figure 1 shows the mean frequency spectrum of the four groups of subjects before and after a week. It can be seen that the "pre and post" spectrum of the control groups is almost identical. However, in both experimental groups the post-isolation spectrum shows a noticeable shift towards the lower end of the frequency scale.

In addition to the changes in occipital lobe frequencies, the records of the ex-

perimental subjects were also characterized by an excess of slow or theta activity, particularly in tracings from the temporal lobes. However, the incidence of these theta waves appeared to be the same for both experimental groups.

These results indicate that both sensory and perceptual deprivation can produce a disturbance of the electrical activity of the brain. Furthermore, this disturbance is greater under perceptual than sensory deprivation. This differential effect may be related to the greater behavioral impairments which seem to occur after prolonged perceptual deprivation (1-3). The fact that the recumbent condition did not affect EEG activity is also paralleled by a lack of behavioral deficits under this condition (2). Thus there appears to be a close correspondence between behavioral performance in deprivation conditions and the state of electrical activity of the brain. Since our EEG records were not taken during isolation but after its termination, one might interpret our results as indicating that the physiological effects of sensory deprivation wear off more quickly than those of perceptual deprivation. Although this is a possibility, we prefer to interpret the data as indicating the presence of EEG differences *during* the two types of isolation. This conclusion is supported by the fact that greater behavioral impairments occur under perceptual deprivation regardless of whether the performance measures are taken during or after the isolation period (1, 2, 7).

JOHN P. ZUBEK
G. WELCH

Department of Psychology and
Faculty of Medicine, University
of Manitoba, Winnipeg, Canada

References and Notes

1. J. P. Zubek *et al.*, *Can. J. Psychol.* **14**, 233 (1960); **15**, 83 (1961).
2. ———, *Perceptual and Motor Skills*, **15**, 171 (1962).
3. J. Vernon and J. Hoffman, *Science* **123**, 1074 (1956); T. H. Scott *et al.*, *Can. J. Psychol.* **13**, 200 (1959); E. Z. Levy *et al.*, *J. Am. Med. Assoc.* **169**, 236 (1959); T. I. Myers *et al.*, in *Progress Report on Studies of Sensory Deprivation* (U.S. Army Leadership Human Research Unit, Presidio of Monterey, Calif., March 1961).
4. P. E. Kubzansky, in *The Manipulation of Human Behavior*, A. D. Biderman and H. Zimmer, Eds. (Wiley, New York, 1961), p. 51.
5. W. Heron, *Sci. Am.* **196**, 52 (1957); J. P. Zubek *et al.*, *Science* **139**, 490 (1963).
6. G. L. Engel *et al.*, *A.M.A. Arch. Neurol. Psychiat.* **51**, 134 (1944).
7. This project was supported by the Defence Research Board, Canada, project 9425-08. We thank Miss S. Oliver, Miss G. Levins, and Dr. M. G. Saunders, EEG Department, Winnipeg General Hospital, for technical assistance.

21 January 1963