Electrophysiological Changes in Humans During Sensory Isolation

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EEG, EMG, EOG, FOG, and Electrophysiological Measures were obtained from 18 normal volunteer female subjects during a 4-hour period of sensory isolation of the water-tunnel variety. The results showed significant decrease of EEG voltage and frequency, of ECG rate and an increase in some of the electrophysiological measures. The findings are discussed in terms of progressive reduction of cortical activities as distinct from sleep and diverse autonomic reactivity resulting from an overall reduction of sensory input.

The relatively short-term electrophysiological changes in human subjects associated with various forms of experimental sensory isolation are as yet little known. Although studies have been published in the past regarding the effects of sensory deprivation on EEG variables, the emphasis was either on the long-term or chronic aspects of such changes or in comparing baseline with isolated samples during the experiment. The aim of this study is to focus on an extensive number of electrophysiological variables (EEG, EOG, EYE movement and Electrophysiological phenomena) as recorded continuously in healthy female volunteers during a 4-hour period of sensory isolation. The latter was achieved by creating an artificial underwater environment into which the subjects were safely immersed. The same subjects were also studied at a different time regarding the effects of the same electrophysiological variables of perceptual isolation, to be reported separately.

MATERIAL AND METHOD

The Ss of this study were 18 paid volunteer female nursing students between the ages of 18 and 19. The criteria for selection were freedom from major physical or emotional disability, no previous sensory isolation experience and regular menstrual pattern for the previous year.

The sensory isolation laboratory consists of a 12 x 27 x 7' light and soundproof room containing a circular tank (8 ft diameter x 8 ft deep) of slowly circulating water at 94°F, a bed and an adjoining 12 x 8 recording room.

In addition to pre- and post-vital indices, the following measures were taken: Holtzman Inkleblot Test, Conners' anxiety and hostility measures and continuous recordings of the following: five channels of EEG, one channel of electromyogram (EMG) (lateral to midline at level of hyoid bone), two channels of electrooculogram, for lateral and vertical eye movements (LEOG and VEOG), two channels of basal skin resistance (in Ohms), one channel of encephalogram and two channels of esophageal GSR calibrated in units of conductance (dorsal and palmar) and finally one channel of EEG monitoring R to R intervals for variation in pulse rate only.

The EEG electrode sites selected from the left hemisphere of the head were in accordance with the International Federation of Electrode placements. Electrodes were placed over the frontal (F3), parietal (P3), occipital (O1), anterior temporal (T1), posterior temporal (T2) and the central vertex (Cz). Five channels of EEG data were recorded using the following montage: F3-F4, Fz-T4; C3-T3; Cz-Fz; F7-T8.

Skin surfaces over the scalp region were thoroughly cleansed with acetone followed by an application of Beckman electrode paste that was gently worked into the electrode sites to lower skin resistance. A mound of laboratory paste (approximate 1 cm) was spread over the site to form a base for placement of the Grass model E51G electrode. A piece of gauze soaked in flexible collodion was dried in place over the electrode to form an insulation against moisture collection at the electrode site. After all electrodes were firmly placed on the scalp, a "mutfly dry" diaper liner and a surgical cap were used to cover the head. A large self-adhering surgical drape was carefully secured to the skin surfaces at the hairline to exclude all water. All electrodes exited from the drape at a single point and...
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were waterproofed and shielded by a latex surgical cigarette drain which extended above the water line in which the subject floated. The subject floated at neutral buoyancy in a supine position rendering the face of the neck area vulnerable to leakage of water. A layer of petroleum jelly at the area of irregularity in the skin in the area retarded water seepage into the scalp area of electrode application. Potentials were recorded at standard sensitivity of 60 μV/7.5 mm., with a time constant of 0.3 seconds. A Beckman Type II Dynograph was used.

The psychological measures, including selection interview and MMPI data as well as elicited spontaneous talk tape-recorded during the experimental run will be reported separately. The electrophysiological data, which are the object of this report, were sampled as follows regarding statistical analysis. As baseline we used a five-minute recording immediately preceding the experimental situation. Thereafter and every 4 minutes a 10-second interval was analyzed throughout the 4 hours of the experiment. These 10-second epochs were evaluated visually manually regarding EEG (only one channel assessed, i.e., P3-O1) frequency and voltage (the latter assessed planimetrically according to a modification of the method described by Bruck). ECG, eye movements (EOG), gross body movements (CMM) and Electrodermal events. As criteria for change were accepted. EEG frequency = 1 to 2 cps, EEG amplitude ± 36%, ECG = ± 2 beats, electrodermal events ± 2 responses, lateral eye movements ± 2 and vertical ones ± 1. Means and standard deviations were calculated for these data as well as EEG voltage/frequency ratios, and lateral/vertical eye movement ratios and t tests were done comparing the values of the first 2 versus the last 2 hours of the 4-hour experiment.

RESULTS

Of the 10 Ss, 3 dropped out at various stages of the experiment and their data are not included here. For the remainder, a comparison of the values (Table 1) obtained during the first half-time period of the experiment (first 2 hours) versus the second half-time period of the same experiment (last 2 hours) revealed the following significant differences.

1. EEG voltage amplitude: There was a significant drop in the second half-time period.

2. EEG frequency: There was also a significant slowing in the second half-time period, but not as great as the voltage decline.

3. The ratio of voltage to frequency (v/f) showed a smaller value for the second half-time period of the experiment.

4. Corresponding with the above, there was also a significant slowing of the cardiac rate in the second half-time period.

5. Regarding electrophysiological activity, and with the exception of basal dorsal and palmar readings, they all showed a significant increase in the second half-time period.

6. There were no significant changes between first and second half-time period for the rest of the variables (i.e., LEQG, VEQG, L/V EOG ratios, GMM, EMM and Basal Dorsal and Basal Palmar CSB).

DISCUSSION

The main finding—in terms of EEG effects of sensory isolation—are a progressive decline of voltage accompanied by a decrease of frequency over the four hour period of the experiment. The cardiac rate did also decrease whereas electrodermal measures were the only ones that showed an increase. Previous electromyological studies of sensory deprivation differ in a number of methodological characteristics. Firstly, none of them, to the best of our knowledge, employed the present water-tank method. In this respect our method effectuated a profound sensory isolation, whereas in the other studies as it will be shown shortly, the condition was one of moderate restriction of sensory input.

TABLE 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>1st 2 Hours</th>
<th>2nd 2 Hours</th>
<th>DF</th>
<th>t test</th>
</tr>
</thead>
<tbody>
<tr>
<td>EEG voltage</td>
<td>Mean 0.2786 SD 0.0044</td>
<td>Mean 0.6705 SD 0.2933</td>
<td>33</td>
<td>0.21772*</td>
</tr>
<tr>
<td>EEG frequency</td>
<td>77.2305 SD 4.3510</td>
<td>74.8914 SD 3.5233</td>
<td>27</td>
<td>2.96077*</td>
</tr>
<tr>
<td>EEG Vd/Fr ratio</td>
<td>0.039 SD 0.0067</td>
<td>0.0401 SD 0.0082</td>
<td>48</td>
<td>2.84325*</td>
</tr>
<tr>
<td>LEQG</td>
<td>7.4854 SD 1.7805</td>
<td>7.6681 SD 1.1599</td>
<td>99</td>
<td>1.09731</td>
</tr>
<tr>
<td>VEQG</td>
<td>4.2445 SD 1.0903</td>
<td>4.3968 SD 1.1093</td>
<td>30</td>
<td>0.36694</td>
</tr>
<tr>
<td>L/V EOG ratio</td>
<td>1.6403 SD 0.3381</td>
<td>1.1944 SD 0.5023</td>
<td>65</td>
<td>0.0065</td>
</tr>
<tr>
<td>EOG</td>
<td>12.6845 SD 0.4961</td>
<td>14.9406 SD 1.0064</td>
<td>65</td>
<td>0.00097</td>
</tr>
<tr>
<td>GMM</td>
<td>38.73 SD 3.1388</td>
<td>39.73 SD 3.4722</td>
<td>13</td>
<td>0.28183</td>
</tr>
<tr>
<td>LMM</td>
<td>1.5507 SD 0.3294</td>
<td>2.9777 SD 0.5077</td>
<td>65</td>
<td>0.0074</td>
</tr>
<tr>
<td>Electrodermal, USR</td>
<td>5.5645 SD 0.3714</td>
<td>5.8802 SD 0.4752</td>
<td>65</td>
<td>0.58208</td>
</tr>
<tr>
<td>Electrodermal, dorsal</td>
<td>3.2248 SD 0.3992</td>
<td>3.8876 SD 0.4513</td>
<td>65</td>
<td>0.26764</td>
</tr>
<tr>
<td>Electrodermal, palmar</td>
<td>3.970 SD 0.3681</td>
<td>6.9876 SD 0.4627</td>
<td>65</td>
<td>0.00736</td>
</tr>
<tr>
<td>Basal dorsal</td>
<td>9.4121 SD 1.3161</td>
<td>5.5002 SD 1.4116</td>
<td>65</td>
<td>0.58208</td>
</tr>
<tr>
<td>Basal palmar</td>
<td>11.3211 SD 1.8170</td>
<td>11.8061 SD 1.2061</td>
<td>65</td>
<td>0.35039</td>
</tr>
</tbody>
</table>

*Statistically significant at the .05 level.
† The figures are planimetric means and refer to area measurements expressed in sq. cm.
E, a.

For voltage conversion the formula —

v = d . 1

E, the millivolt signal (50 μV), a, the area figure given in the table, d, the calibration deflection (25 μV), and i, the corresponding length (15 cm.), for a, above.

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Secondly, no attempt had been made so far to examine the effects of sensory isolation on other EEG variables in addition to frequency, or on the various other physiological functions as studied here. Finally, as mentioned in the introduction, most previous studies lasted for days or weeks whereas the emphasis here was on the acute or short-term effects of such isolation. Only one study to our knowledge focused on such short-term effects but because of dissimilarities in terms of methodology (perceptual rather than sensory isolation) and subjects involved (male schizophrenic patients instead of normal subjects) comparisons are difficult. In the study just mentioned, frequency of occipital alpha was found to decline following one hour's sensory isolation, consisting of bright diffuse red light seen through translucent eye caps and muffling noise to a low audible level while the patients lay supine with instructions to lie still. No voltage or other physiological measurement were made. Prior to this study, the earliest EEG effects of sensory isolation were documented by Heron. Again methodological differences make comparisons difficult despite the similarity of findings. Heron reported on the EEGs of eight subjects who experienced isolation for 96 hours. The subjects were situated on a bed in a lighted cubicle. Their auditory and tactual perception was limited. Translucent goggles prevented patterned vision but did admit diffuse light. EEG potentials were sampled at the beginning of the experiment and twice daily for the duration and also during hallucinations. After a battery of perceptual tests were given at the termination of the isolation, a final recording was made. All records were made with subjects awake. Slower frequencies at the bipolar parieto-occipital leads were seen at the termination of isolation. The alpha rhythm deteriorated and as the interval of isolation increased, a quantitative analysis of wave frequencies revealed progressively slower activity. After 96 hours there was more slow activity than after just 48 hours. Even analysis of the records at 24 hours showed a similar trend. Three and one-half hours after isolation termination, these changes persisted. During hallucinations the EEG was found to be similar to that of an alert person. Thus our findings of reduced EEG frequency are in agreement with published reports. Furthermore, our study shows that such a reduction can be effected by a different method of sensory interference than the ones mentioned in these reports and also by acting for a shorter period of time. However, interestingly enough, such frequency reduction as effected here is not of the same magnitude as another EEG change in our study, namely the voltage reduction. Such a finding has implications that go beyond the specific field of sensory isolation. It had been assumed in the past, for instance, that there is a reciprocal relationship between frequency of the EEG and the corresponding voltage, and techniques were evolved on the basis of such assumptions in order to measure drug effects, levels of vigilance and intracranial disease. The notion was accepted as "apparent" that "during low level cerebral activity (such as lack of sensory stimulation in sleep), relatively large amplitude sleep waves predominate on the EEG." Our findings suggest that there are certain states, indeed, when the relationship between frequency of the EEG and corresponding voltage is not reciprocal. Furthermore, this differentiates the EEG of profound sensory isolation from the slow-wave sleep where voltage is found increased. Thus, there is additional evidence here that, for the time-span of this study at least, we are not dealing with sleep but with awake subjects adapting to profound sensory isolation. A general characteristic then of such an adaptation seems to be a progressive reduction of the level of the cerebral activities as monitored by the EEG. The corresponding reduction of cardiac rate would point to an associated autonomic reduction of activity as well were it not for the concurrent increase in electrodermal measures. This dichotomy raises intriguing questions regarding response specificity of the cardiac versus the dermal autonomic sub-systems vis-à-vis sensory isolation and its consequences.

REFERENCES