

type is fully determined by day 15, and any reversal takes 5 to 10 days more. The type of progeny produced during the test period (days 15 to 17), therefore, must be a consequence of the preceding thermoperiodic treatment.

The results of this experiment (Fig. 1 and Table 1) show that females of *N. vitripennis* produce nearly all of their progeny as diapause larvae when the warm period of the "day" is less than about 13 hours, but that diapause is absent when the warm period is more protracted. Females of *N. vitripennis*, therefore, are able to distinguish a "short-day" thermoperiod from a "long-day" thermoperiod and produce diapausing or nondiapausing progeny accordingly. The critical thermoperiod in these experiments was about 13 hours out of 24, whereas the critical photoperiod measured earlier (12) was 15¼ hours out of 24. However, little importance is attached to this difference; it is almost certainly associated with the slow rate of cooling and warming consequent upon the manner of transfer from one incubator to the other. More abrupt temperature transitions would undoubtedly produce a critical thermoperiod closer to that for light.

A resonance experiment, in which 12 hours at 23°C was coupled with 12, 24, 36, 48, and 60 hours at 13°C (Table 1), failed to show that diapause induction was a rhythmic function of the length of the temperature cycle (T), presumably because of the weakness of temperature as an entraining agent in cycles longer than 24 hours.

In a 24-hour cycle, the proportion of wasps producing diapause broods when the warm period lasted 12 hours was significantly greater than that for control females kept at 18°C throughout ( $\chi^2 = 26.72$ ;  $P < .001$ ). Similar results have been described by others for the moths *Ostrinia nubilalis* (13) and *Pectinophora gossypiella* (14), although in both cases the effects were weaker than those for 12 hours of light. Similarly, discrimination between a short and a long thermoperiod, described for *O. nubilalis* (15) and *Acronycta rumicis* (16), was, for both species, considerably weaker than the corresponding photoperiodic effect. My data for *N. vitripennis*, however, show that thermoperiod and photoperiod are equally effective in 24-hour cycles; indeed both are considered to be manifestations of the same phenomenon. Furthermore, since the switch in metabolism can be controlled by thermoperiod in the absence of light, any model for the

*Nasonia* clock in which light plays a part in induction ("external coincidence") must be ruled out. Since a specific temperature-sensitive phase seems unlikely, the most probable explanation for this phenomenon is a model of the "internal coincidence" type, involving the mutual interaction of the "dawn" and "dusk" oscillators.

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5. In "resonance" experiments a short light period (usually 8 to 12 hours) is coupled with different periods of dark to provide environmental light cycles (T) up to 72 hours or more in length. If the photoperiodic clock incorporates a circadian oscillation (that is, with an endogenous periodicity,  $\tau$ , close to

24 hours), the product of photoperiodic induction (for example, diapause) is observed to be high when T is close to  $\tau$  or modulo  $\tau$  (that is, the two oscillating systems resonate), or low when T is not close to modulo  $\tau$  (that is, the two oscillating systems do not resonate).

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## Visual Effects on Alpha Feedback Training

**Abstract.** *Presenting an audible indication of subjects' electroencephalographic alpha activity under conditions of dim ambient illumination led to systematic increases in alpha density, while in total darkness the same procedure did not. These results support the view that feedback training can be clearly demonstrated only when factors leading to a suppression of alpha activity are present in the environment.*

Much of the current interest in electroencephalographic (EEG) alpha activity centers around the apparent ability of individuals to learn volitional augmentation of alpha densities in their EEG. Kamiya (1) and Mulholland (2) have independently demonstrated that providing a subject with feedback concerning the presence or absence of his own EEG alpha activity makes it possible for him to alter the amount of such activity seen in the record. In addition, it has been suggested that volitional increases in alpha density lead to changes in subjective mood (3, 4), and it has even been proposed that feedback techniques may be useful as a means of "mapping the subjective space of consciousness" (5).

The effects of feedback training usually have been demonstrated in one of two ways, both of which contain inherent methodological shortcomings. The first procedure involves comparing periods during which subjects are instructed to maximize alpha density with

similar periods when they are instructed to minimize density. Although it is easy to demonstrate differences between periods when subjects are instructed to produce alpha activity and periods when they are instructed to block it, such differences rarely reflect symmetrical increases and decreases. Usually subjects learn to block alpha activity volitionally within one or two trials, and impressive differences with this procedure tend to be the result of unidirectional rather than bidirectional control.

The second procedure involves comparing initial baseline levels with the higher densities seen after training. Such a comparison more appropriately addresses the question whether individuals can learn to augment their level of alpha activity significantly. It then, however, becomes crucial to specify the nature of the baseline level from which increases can be measured. Novelty and apprehension are known to depress the subject's initial alpha density

during baseline determination, but as factors such as these dissipate, one tends to see a spontaneously rising baseline. To attempt to follow such a rising baseline, however, through the use of resting periods, interspersed throughout the training sessions as "floating" baselines, does not permit separation of the effects of feedback training from those increases which are due to changes in arousal or motivation; for the subject, definition of a period as a rest period produces changes in both motivation and arousal.

The present report is intended to help clarify the circumstances under which subjects are able to learn to augment alpha density and shed light on some of the possible mechanisms involved. Specifically, we were concerned with determining whether subjects could learn to significantly exceed the alpha density of their own baseline obtained under optimal circumstances. Because of the known overriding influence of visual stimulation on alpha activity (2), the first study was conducted in total darkness, although to lessen the likelihood of their falling asleep subjects were instructed to keep their eyes open throughout the experiment. Prior to the actual experiment, every effort was made to reassure the subjects and make certain that they were comfortable and understood the equipment and procedures. Nine paid volunteer college students served as subjects, and were recruited for an experiment in "the control of physiological responses." The subjects were given six training sessions, approximately 1 week apart. The monopolar occipital ( $O_2$ ) to right mastoid EEG signals were recorded on a Beckman polygraph and filtered with step-function cutoffs at 8 and 12 hz (6). Although the amplitude levels necessary to trigger the feedback stimulus were set in discrete 5- $\mu$ v steps from 10 to 25  $\mu$ v for a given subject, depending on initial levels of alpha density, the data used in this report were collected by using an invariant threshold of 15  $\mu$ v for all subjects. Each session consisted of (i) two 3-minute baseline periods, (ii) a 5-minute orientation period during which feedback was provided and subjects were encouraged to experiment, and (iii) ten trials of 2 minutes each, alternated with 1-minute rest periods. The presence of alpha activity raised the pitch of a feedback stimulus tone from 280 to 360 cycles per second, and activated a clock, the output of which recorded alpha density in seconds per minute. The subjects

were instructed that during the trials they should keep the higher (alpha) tone on as much of the time as possible. The absence of any tone served to signal a rest period. At the beginning of each rest period a digital display was illuminated briefly to give the subject a cumulative total of his alpha activity for each trial, and the subject was required to repeat the total out loud.

An examination of the results for the first session, as shown in Fig. 1, reveals that although subjects' initially high alpha activity was decreased markedly by opening their eyes in total darkness, they had recovered much of this drop by the middle of the initial 5-minute orientation period when the feedback tones were first available to the subjects. These increases occurred within 2 or 3 minutes, without volitional effort. Whether they represent true learning or adaptation is unclear, but the rapidity of these effects is different from what is usually described in alpha feedback training. It furthermore appears that subjects had approached their asymptotic density during the initial orientation period. The subjects exhibit a non-significant maximum further increase of 7.2 percent from their highest single minute during the orientation period to the highest density reached during any of the ten trials where subjects were instructed to maximize alpha density ( $t = 1.81$ ,  $P > .10$ ). Although in the group data the resting levels tended to be below trial levels, these differences were not significant.

Within the first session, subjects'

highest trial densities were significantly lower than their initial eyes-closed baseline densities ( $t = 2.46$ ,  $P < .05$ ). When both trial and resting averages are examined for all six sessions, with an analysis of variance repeated measures design, none of the differences are significant (trials:  $F = 0.19$ ,  $P > .20$ ; rest:  $F = 0.05$ ,  $P > .20$ ), and the largest difference between any two trial averages is only about 4 seconds of alpha activity in a minute. The trial average for the sixth session was not greater than the level of alpha density reached during the third minute of the orientation feedback period ( $t = 0.35$ ,  $P > .20$ ).

These findings contrast with data reported by others, which imply large increases in alpha density as a consequence of alpha feedback training. We had previously proposed that subjects in the alpha feedback situation are involved in overcoming factors which normally exert an inhibitory influence on alpha activity (7), and we reasoned, therefore, that the lack of consistent increases beyond 2 minutes in our subjects when run in total darkness may well have stemmed from the relative absence of inhibitory stimuli in the situation, especially visual stimuli which Mulholland and Peper have emphasized as primary influences on alpha activity (2, 8). Another group of seven subjects was therefore placed in the same training situation with auditory feedback and exposed to the identical procedure used with the previous group, adding only the presence of dim ambient light.

The results for these subjects are

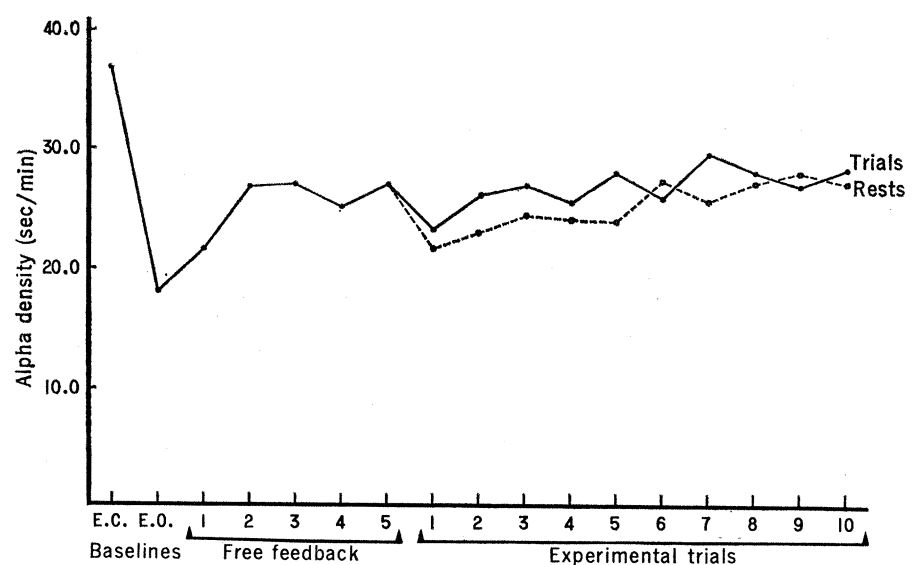


Fig. 1. Average alpha densities for several segments of the first day with total darkness. All points are averaged over nine subjects, with eyes-closed (E.C.) and eyes-open (E.O.) baselines averaged over 3 minutes and trials averaged over 2 minutes. The alpha density is expressed as the number of seconds that alpha activity is present in the EEG per minute.

shown in Fig. 2A. With this new group, the same drop from eyes-closed to eyes-open baseline is evident. The recovery during the free orientation period preceding the trials is not, however, as great as that seen in darkness. Unlike the earlier group, this second group tested in ambient light exhibited alpha activity during trial periods which was significantly higher than that seen during resting periods ( $t = 4.33$ ,  $P < .01$ ) and, furthermore, densities increased beyond the orientation period, as evidenced in a significant 96.8 percent increase in densities from the highest minute during the free orientation period to a subject's highest trial density ( $t = 4.06$ ,  $P < .01$ ).

Figure 2A resembles some of the reported findings (1, 3, 9), showing clear-cut increases in alpha density with training. These data would, of course, be

even more impressive if the initial eyes-closed baseline were not included in the graph. Most previous reports on the effects of alpha feedback have shown little in the way of such a baseline, and have not considered the possible effects of ambient light.

The significance of the presence of light is seen most clearly by comparing the absence of continuing augmentation in the original group of subjects, run for six sessions in total darkness, with the same group's response during a single session in the presence of dim ambient light. Eight of the original nine subjects were presented with the identical auditory feedback procedure they had previously experienced, with only dim ambient light added. The results for this original group, during their seventh session, the first session with light in the room, are shown in Fig. 2B. Recovery

from the initial drop takes place slowly, and a significant increase of 55.7 percent was evident from subjects' highest minute during the orientation period to their highest trial density ( $t = 3.04$ ,  $P < .02$ ). The difference between trial and resting averages was significant ( $t = 2.47$ ,  $P < .05$ ).

Tests between the results of the first session in total darkness and the subsequent session with dim ambient light indicate that the session with light for those same subjects was significantly different, both in trial averages ( $t = 9.11$ ,  $P < .001$ ) and resting averages ( $t = 5.57$ ,  $P < .001$ ), from their performance in darkness. The session with light for the independent group of subjects also differed significantly from the session in total darkness, both in trial averages ( $t = 1.96$ ,  $P < .10$ ) and resting averages ( $t = 2.64$ ,  $P < .05$ ). No significant differences exist between the two sessions with light.

Though there may have been some effects of alpha feedback training for six sessions in the dark, the data appear to indicate that the response of the two groups of subjects to alpha feedback training is more alike in the presence of light than the responses of one of those groups with and without the presence of dim light.

The significant differences in the relative level of resting alpha densities between light and dark conditions are probably a function of the fact that light allows the subject to engage in visual scanning during the resting periods, in contrast to the limited scanning possible in total darkness. These results help to explain the discrepancy between Kamiya's findings (1), which showed resting periods at about the same level as during the trial periods, and Brown's results (3), where subjects exhibited resting levels significantly lower than those seen during feedback. Kamiya used an auditory feedback stimulus, and usually had subjects close their eyes. Brown, in contrast, used a visual stimulus, a procedure which necessitated the subjects' keeping their eyes open in a lighted room.

The popular view that alpha feedback training somehow permits individuals to increase alpha densities significantly above those obtained during a comfortable baseline with eyes closed in a non-drowsy, relaxed state cannot be supported by our data. Indeed, despite many training sessions, we have failed to note any individuals whose capacities for producing alpha activity were altered by the feedback procedure. What

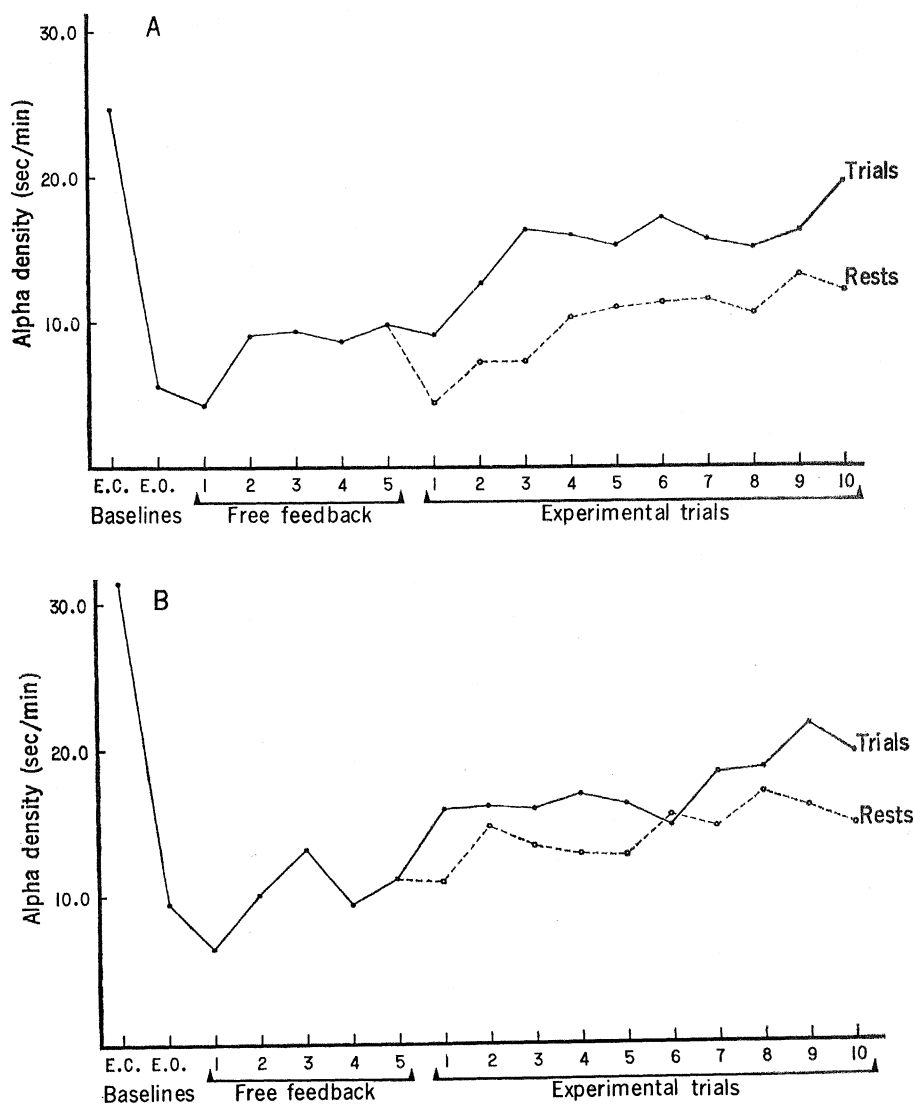


Fig. 2. (A) Average alpha densities for the first day with the seven subjects who began with dim light. (B) Average alpha densities for the seventh day with eight subjects from the original group, their first day with dim light. In both cases, eyes-closed (E.C.) and eyes-open (E.O.) baselines are averaged over 3 minutes, the free feedback period over 5 minutes, and individual trials over 2 minutes.

does seem clear, however, is that alpha feedback training can lead to large and significant changes in alpha densities when conditions have lowered alpha density below the levels seen spontaneously under optimal conditions. Subjects can acquire volitional control over alpha activity only under conditions which normally lead to decreased densities. Thus, during the initial orientation in the dark, when subjects experiment, but are not especially set to increase alpha density, they achieve a level approximating the level reached with additional training. In the light condition, however, there are marked volitional increases. In spite of these increases, however, in the three procedures reported here, as well as during a number of other procedures run in our laboratory, we have not seen alpha densities beyond an individual's initially demonstrated normal physiological range.

While ambient light is sufficient in the present studies to act as a suppressing stimulus, it is likely that other stimuli such as anxiety or physical stress may, in some circumstances, also lead to suppression which persists. Although no increases in alpha density are seen when the situation presents few suppressing stimuli, alpha feedback training may enable a subject to overcome suppressing effects when they are present. The subjective experiences reported to be associated with alpha feedback training (3, 4) may be understood as a consequence of acquiring skill

in disregarding stimuli in the external and, perhaps, internal environment which would ordinarily inhibit alpha activity. Increased alpha densities, then, may best be viewed not as an end in themselves, but as one convenient index of a subject's ability to acquire this skill.

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## Strychnine and Memory Process

Alpern and Crabbe (1) reported that strychnine facilitates the long-term store of memory and that their data could not be explained by the consolidation hypothesis. I believe their data can also be explained by the fact that strychnine facilitates the consolidation of short-term memory even if injection was administered 24 hours after the last training.

On the basis of the total error scores, during daily retention tests of mice prior to criterion (at least 4 consecutive days), Alpern and Crabbe found that mice exposed to a maze for 2 days and given low doses of strychnine for 10 consecutive days [the experimental group or LD (1-10)] showed significantly improved learning in the retention test when trained again, as compared to the control group, which received the vehicle alone for 10 days between train-

ing and retraining. Alpern and Crabbe say that the consolidation argument is vitiated because two additional control groups, given the strychnine injection once either at day 1 [group LD (1)] or at day 5 [group LD (5)] after the last day of initial training showed no statistical differences from the control group. The daily scores on the "number of initial errors" of the above four groups (as estimated from figure 1 of Alpern and Crabbe) could lead to a different conclusion. The performance on retention test day 1 was facilitated as compared to the last day of initial training in two groups; that is, LD (1-10) with 0.7 less error (from 2.5 to 1.8) and LD (1) with 0.5 less errors (from 2.75 to 2.25). Mice in these two groups had received strychnine 24 hours after their last day of initial training. In

contrast, a performance decrement was found in two other groups in which mice received no strychnine for at least 5 days after the last day of initial training: the control group with 0.5 more errors (from 2.5 to 3.0) and LD (5) with 0.6 more errors (from 2.1 to 2.7). According to the savings scores above, the administration of strychnine 24 hours after the last training day facilitated the consolidation process even though retrograde facilitation for only a few hours after final training has been reported (2).

Alpern and Crabbe have excluded the possibility that strychnine enhanced short-term memory consolidation, because group LD (1), which also received strychnine 24 hours after the last training, was not statistically different from the control group. This inference was based on total error scores before criterion was reached (at least 4 retention days) rather than savings scores between days. In addition, I have been told by Alpern that there were no significant differences between the scores of day 1 of retention tests even in the case of group LD (1-10) and the control group. Even with the pooled scores, the facilitation effect in LD (1-10) is very small. Any facilitation in group LD (1) may be lost because of its overall small magnitude. In any case, the reasons for strychnine facilitation of performance only in group LD (1-10) remains to be examined. I suggest that the consecutive injection schedule may, through an unknown mechanism, prolong the labile phase of (short-term) memory into a period longer than 24 hours, so that a cumulative effect on memory store can be seen.

Additional control groups—such as LD (2-10), with the vehicle injection at day 1 after the initial training and strychnine injection at low dose daily for 9 days, LD (1-5) and LD (6-10)—would provide information on whether consolidation could be manipulated with strychnine for a period of up to 10 days. Without these controls, the consolidation hypothesis cannot be rejected.

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