OPERANT ENHANCEMENT OF EEG THETA ACTIVITY

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SPECULATIONS ON THE BEHAVIORAL SIGNIFICANCE OF POSTERIOR THETA RHYTHM

During the last several years the interest in "operant" control of theta activity in the electroencephalogram (EEG) has rapidly increased. Apart from concern for actual scientific support, a wide range of clinical methods for "theta-control" are advertised by the biofeedback industry. This interest was partly stimulated by speculations on the involvement of theta activity in specific psychological and behavioral responses.

Pelletier (1974), for example, hypothesized that the presence of theta activity is a prerequisite for pain control. Studying an individual with abnormally high insensitivity to pain, he found an increase of posterior theta during painful stimulation.

A great deal of work is concerned with the significance of theta for meditative states. Most often cited in this respect is the paper by Banquet (1973). This investigation claims to have found in stage 1 of meditation a "dramatic increase in alpha abundance," high amplitude alpha waves in frontal areas, and no alpha blocking to external stimulation. Stage 2 was characterized by an increase of trains of occipital theta with a constant frequency that is different from the mixed frequencies found in drowsiness. Theta also persists when the eyes are opened and during the post meditation period. On the background of theta waves high frequencies in the beta range were observed. The work of Banquet (1973) failed to use an appropriate control condition, a flaw which characterizes most of the work on meditation. A group of beginners in transcendental meditation (TM) served as controls. It is therefore possible that the observed effects are due to the increased ability of trained TM practitioners to achieve a drowsy state, a criticism already confirmed by Fenwick (1974) who found no difference between TM patterns and drowsiness. Furthermore, in Banquet's study, no statistical analysis of the data was applied.

Williams and West (1975) confirmed the tendency of trained TM meditators to exhibit a significantly smaller alpha blocking effect during meditation; subjects instructed to relax served as the control group. The authors concluded that the meditation state is a state of prolonged drowsiness, but they also discussed the possibility of a state of sustained attention. Similar results are reported by Wallace et al. (1971) and Fenwick (1974). Generally, in spite of many interesting studies and speculations, there is little evidence for the existence of a special kind of theta state distinct from drowsiness in meditation.

Together with the increasing interest in meditation, theta was thought to be an indicator of hallucinatory and hypnagogic events as they occur during sleep stages 1 and 2 and other "twilight" states (Stoyva, 1973). Stoyva mentioned a special kind of "theta imagery." But as far as I know, no sufficiently controlled study exists which supports any of these speculations. The work of Green et al. (1971) claiming "vivid imagery" during theta stimulated a certain amount of practical applications of theta-training, but is based only on unsystematic observation and lacks an experimental design. From sleep research (Foulkes & Vogel, 1965) we know that with descending stages 1 and 2, reports of dreamlike experiences increase. Along with these speculations Budzynski and Peffer (1974) advanced the idea that "theta state" may be a prerequisite of very powerful memory consolidation as it occurs during posthypnotic suggestion; and in fact a machine for "twilight learning" has been built and is already being sold. However there is no solid evidence supporting this interesting hypothesis.

In the context of stress research the occurrence of theta activity is often considered as an indicator of a state of consciousness incompatible with anxiety (Stoyva, 1973). There are two controlled studies on the effect of alpha activity on anxiety relief during systematic desensitization (Birbaumer & Tunner, 1970; Benjamins, 1976). Both experiments support the notion that during and between imagination of anxiety provoking scenes, aversive arousal disappears with increasing alpha. Whether alpha presence or EEG synchronization is a necessary condition for anxiety reduction, as Birbaumer (1973) assumed, remains to be demonstrated empirically.

Better controlled studies exist on the relationship between theta activity and performance in vigilance tasks. Nevertheless the findings of the two main research groups (Lawrence & Johnson, 1976; Beatty, 1976) are contradictory. Since an excellent review by Lawrence and Johnson (1976) is available, only the main problems will be evaluated here. Beatty and his collaborators (Beatty et al., 1974; O'Hanlon & Beatty, 1975; O'Hanlon, Royal, & Beatty, see this volume) have reported an increased performance of student subjects in a radar monitoring task during posterior theta suppression and a decreased performance during theta augmentation in comparison to the same group of subjects without theta feedback. This effect was replicated using professional radar operators in a laboratory environment, but not in a natural radar monitoring situation, where no group decrement was observed under any condition (O'Hanlon, Royal, & Beatty, this volume).

As with biofeedback of other variables (e.g., heart rate), the effect of the training is either confined to the laboratory or there is no effect of training in those groups of subjects who need self-regulation. Lawrence and Johnson (1976) reviewed their own work in the light of Beatty's and O'Hanlon's research. Hord et al. (1975) used a yoked and one no-feedback group—a better controlled but less powerful design in comparison to the Beatty et al. (1974) within-subject bidirectional design (theta augmentation versus theta suppression). Hord et al. again used a 3-hour sonar task and applied theta suppression to their subjects, but used a different algorithm of EEG analysis. Beatty and his colleagues compute a theta ratio index for each second (number of theta waves divided by the total number of waves in the theta, alpha and beta frequencies) while Hord et al. worked with an analog filter system. It is not clear whether differences in the EEG analysis or in design are responsible for this discordance. Lawrence and Johnson (1976), after reviewing the relevant studies, state: "A performance decrement may be a necessary condition for the observance of any theta effect, and there are no data to suggest that theta suppression can lead to performance enhancement above initial levels."

Thus on the whole, the behavioral and psychological "meaning" of cortical theta activity is not clear. We believe one of the reasons for most of the problems in present research on theta is the fact that theta is not a unitary phenomenon. The characteristics of frontal theta and posterior or temporal theta may be entirely different. We observed unsystematically 10-20% high voltage theta trains during an alert awake state in frontal areas in comparison to a very weak posterior theta activity (Lutzenberger et al., in press). Low voltage theta with mixed frequencies in sleep stages 1 and 2 may be a very different physiological phenomenon than high amplitude regular theta during awake states and meditation. Different methods of analysis and feedback between the laboratories add a considerable amount of confusion; exact replications are virtually impossible because of different laboratory conditions. Most of the speculations on the psychological covariations of theta still await experimental support.

EFFECTS OF BIOFEEDBACK ON INCREASING THETA

It is generally assumed that high amplitude theta activity as an indicator of central synchronization is incompatible with the presence of central arousal patterns; low voltage theta is an integral part of the operational definition of sleep stages 1 and 2, and occurs also in REM sleep. Increased theta activity over large cortical areas should lead to a drowsy state or a state which is not characteristic of attentive wakefulness ("twilight state"). It may be a useful clinical goal to voluntarily increase the amount of theta in anxiety states, insomnia and other stress related disorders.

Unfortunately, the experimental setup of a biofeedback situation is incompatible with a drowsy state. It may be impossible to attend to the feedback during theta "state." On the other hand we know that operant learning is possible during a drowsy state and also in REM sleep (Salamy, 1970).

There are few controlled studies on the possibility of increasing theta activity through biofeedback. With the exception of our own work (Lutzenberger et al., in press) all investigators tried to control posterior theta. Brown (1971) and Klug and Brown (1974) reported a substantial increase in theta through biofeedback. Since they did not use a control group the effect may be due to increased drowsiness over the experimental session. The same effect could be responsible for the data reported by Green et al. (1971). Sittenfeld et al. (1976) similarly used a design which is inappropriate for the control of the drowsiness effect. Four groups of five subjects each with two different baseline frontal electromyogram (EMG) levels received theta feedback training (8 sessions), or combined EMG training (4 sessions) initially and theta training (4 sessions) afterwards.

They found that all groups with the exception of the group with high resting baseline EMG, which had only theta training for 8 sessions, increased theta activity over sessions. The second group with high EMG, which had the two stage training, increased theta activity as well. The authors interpret their results as supporting the "concept of shaping of low arousal" and as support of a "response patterning approach." In our opinion both explanations are inappropriate. The results could also mean that all groups adapt over sessions and became more and more drowsy, but the groups with a tonic higher tension level are unable to adapt as easily. This design again demonstrates the usefulness of EMG relaxation training for tensed subjects and it shows that muscular relaxed subjects have fewer difficulties in shifting to a drowsy state.

In summary, the possibility of increasing theta activity with feedback is still open to experimental testing. The clinical usefulness of theta biofeedback has not yet been demonstrated in controlled studies.

RESEARCH ON THETA BIOFEEDBACK FROM OUR LABORATORY

Two controlled studies were performed in our laboratory to test: 1) the effect of acoustic feedback on theta activity during increase task only; 2) the effect of the training session on a post training stress situation with and without feedback; and 3) the influence of simultaneous biofeedback of heart rate and frontalis EMG as pretraining on the biofeedback of EEG theta activity.

In the first study (Lutzenberger, Birbaumer, & Wildgruber, 1975) 10 subjects participated in eleven one-hour sessions (1 habituation session and 10 training sessions). A cross-over design was applied: Five subjects began with five sessions contingent feedback after a baseline session (CF, a soft pink noise on the right ear simultaneously with EEG activity of 3 to 7. 8 Hz with a minimal amplitude of 10;uM) and continued with 5 sessions of non-contingent feedback (NCF, pink noise on the left ear in the sequence of the tape recorded feedback signals produced by the same subject in the previous session); the remaining five subjects received the same treatment in reversed order. After the 6th and 11th sessions both groups were shown an aversive movie and were simultaneously presented with either the "non-contingent" feedback signal or the "contingent" signal, depending on group assignment. The entire experiment was run by an on-line program on a PDP lab 8/e computer. Posterior EEG, EOG, heart rate (HR) and subjective changes were recorded. (For a more detailed description see Lutzenberger et al., 1975).

The EEG was analyzed by a method similar in some respects to the method used by Beatty et al. (1974), but instead of zero crossing a maximum-minimum algorithm was used. Further, subjects received immediate feedback if more than one theta wave was detected in the EEG. A moving baseline was used in computation of the results; the mean amount of theta during the baseline of the same session was calculated and subtracted from the mean amount of theta during training. The statistical analysis was made within and across the 10 subjects. There was no significant difference between the CF and NCF condition or the test conditions (aversive movie) in any of the subjects. In contrast to Beatty's work, all subjects were informed of the nature of the feedback. Furthermore, falling asleep was carefully controlled: EEG and EOG data which indicated sleep stages 1 and 2 were eliminated.

Due to a high correlation between heart rate slowing and the amount of theta activity, and the positive effect of EMG training on theta biofeedback reported by Sittenfeld et al. (1976), a second study was designed to test the influence of the pretraining in simultaneous heart rate slowing and reduction of frontalis EMG on theta biofeedback (Lutzenberger, Birbaumer, & Steinmetz, 1977; Birbaumer, Lutzenberger, & Steinmetz, 1976).

Twenty normal subjects were exposed to four half-hour sessions with simultaneous feedback of heart rate and frontalis muscle (pretraining) following one baseline session. Ten subjects received contingent (CF), the other ten noncontingent feedback (NCF). Subjects were asked to lower heart rate and muscle tension (EMG). The feedback display of the heart rate was very similar to that developed by Lang (1974). The subject is seated before a large oscilloscope screen. Each heart cycle initiates a line starting on the left, which then extends across the screen. The length of the line is proportional to the length of each successive R-R interval. There is also a vertical target line for the heart rate (see Figure 1).

In the slowing task the horizontal line must extend past the vertical target. The target line is set initially at the median R-R interval of the subject. If the subject succeeds in extending the horizontal "heart rate line" for more than 10 heart beats, a new target line is set which is midway between the subject's previous and current medians ("rule of halves" after Lang's procedure). At the same time the horizontal line is moved up and down the screen proportionally to the integrated EMG amplitude of the subject. There is a horizontal target line for the EMG amplitude which is again initially set at the median of the subject's amplitude. The subject is asked to reduce his muscle tension as much as he can and to keep the horizontal line as long as possible below the horizontal target line. Subjects in the noncontingent feedback group (NCF) faced the same display as the contingent feedback group (CF) (see Figure 1) except that the horizontal line and the two target lines were not computed from the actual HR and EMG of the subject, but instead represented a random combination of HR and EMG of the past 2 minutes of the same subject.

Heart rate decreased up to 19 bpm with a mean of 4 bpm for the CF analysis was made within and across the 10 subjects. There was no significant difference between the CF and NCF condition or the test conditions (aversive movie) in any of the subjects. In contrast to Beatty's work, all subjects were informed of the nature of the feedback. Furthermore, falling asleep was carefully controlled: EEG and EOG data which indicated sleep stages 1 and 2 were eliminated.

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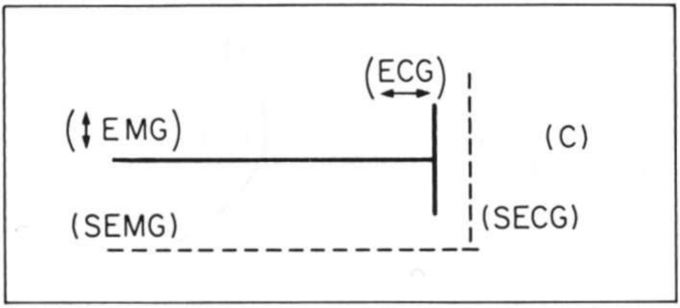

Figure 1. The solid line is moving up and down (EMG) and from right to left (HR). Dotted lines are target lines for EMG (horizontal line) and HR (vertical line). The counter (C) is placed in the right corner of the screen.

group within a session. There was only a weak decrease over sessions because of the strong adaptation effect.

EMG decreased substantially only in the first session and remained stable over the next three sessions. Differences between CF and NCF were significant for both measures. After pretraining subjects received eight sessions of acoustic feedback of their frontal EEG theta activity (Fz to mastoid), four sessions with CF and four sessions with NCF in balanced order. Again subjects were fully informed about the meaning of the feedback signal. In this study the feedback consisted of pink noise increasing in pitch against a steady soft background noise. Only theta periods longer than 1. 5 sec produced feedback.

To correct for artificial signals from eye movement, the feedback was immediately terminated if an EOG signal larger than 50 uV was detected. The noncontingent feedback (NCF) consisted of the tape recorded sequence of feedback signals produced by the same subject during the previous session.

After the fourth and eighth sessions a 4 min video film was presented showing aversive scenes from surgical operations and electroshock therapy. Five min before the film was shown subjects were told to produce the feedback as frequently as possible while waiting for the film. During film presentation and after the film they were again asked to regulate theta without any feedback for 4 min with eyes opened.

There was a weak significant increase of theta (15 to 20%) for the

CF condition over sessions but a decrease within the sessions. Pretraining had no influence on the performance during theta training.

A certain amount of control over theta in the presence of the stress inducing film was demonstrated: The CF group did not reduce theta in comparison to the NCF group which did. Whether this result could have any effect on subjective or behavioral responses remains to be investigated.

In contrast to our previous study (Lutzenberger et al., 1975) which indicated no effect of feedback on the EEG theta activity, there is a weak effect of contingent feedback in this experiment.

DISCUSSION

This discrepancy between the first (Lutzenberger et al., 1975) and the second study (Lutzenberger et al., 1977) could be due to a variety of effects which are not easy to evaluate, since the second study was not an exact replication of the first.

As Stoyva has suggested (see below), the negative results of the study may be the result of the fact that we excluded stage 1 sleep. In the study by Sittenfeld et al. (1976) stage **2** sleep was deliberately included. The reason for excluding stage 1 was a theoretical one: "Self-regulation" of central nervous functions in humans in our opinion should be the result of "cognitive processing" of an awake subject and should not be compared with a sleeping subject. Stoyva's argument is therefore correct for the first study. In the second study we included sleep stage 1, but contrary to Sittenfeld et al. (1976), who recorded theta from **C4 - O2,** we recorded theta from frontal areas. Since we found a small but significant increase over sessions, Stoyva's argument is strongly supported. The question remains, why there was no influence of the pretraining on the theta production as Sittenfeld et al. (1976) have reported.

In our opinion there still exists the possibility—which I mentioned above—that the high EMG group which didn't show theta increase was the only group that did not fall asleep within the sessions nor was there an effect across sessions because they were more tense than the other three relaxed groups. In this case they are not an appropriate control for a specific feedback effect. To put it ironically, insomniacs were compared with normals and a difference in sleep onset was found. But again, our second study is not an exact replication of Sittenfeld et al. (1976); all our critical arguments rest on the unproved—and the certainly wrong—assumption that both studies are comparable. Both results await an exact replication before even tentative conclusions are possible.

We conclude in agreement with Johnson (in these proceedings), that a positive effect of biofeedback posterior theta rhythm with the instruction to the subjects to increase theta still awaits experimental support.

Stoyva (personal communication) noted that the "discrepancy between the two sorts of results is puzzling," and that "the task remains of defining precisely the conditions which improve the ability to increase (sleep onset) theta rhythms. One important possibility may be the nature of the training employed in the Sittenfeld et al. (1976) experiment. This training was conceptualized as the 'shaping of low arousal' in which subjects would begin with an easy task and then shift to a more difficult one. The subject's first task was relaxation of the forearm extensor (1 session), next relaxation of frontal EMG (3 sessions) and finally, production of theta EEG (4 sessions). With such techniques, both forearm and forehead EMG values fall to very low levels (see Sittenfeld et al., 1976 for precise details on the training technique—since the latter are frequently critical in biofeedback learning). It seems quite possible that muscle relaxation may have to be very extensive before the CNS is affected in the direction of sleep. Consistent with such an interpretation is the experiment of Hodos (1962) who recorded EEG changes in 24 cats injected with Floxedil, a drug which blocks conduction at the motor end-plate and causes complete relaxation of the entire striate muscle system. As hypothesized, the cats regularly showed EEG changes in the direction of synchrony and sleep. Hodos' result suggests that muscle relaxation, as produced by biofeedback or other techniques, should be extensive before a shift to elevated theta activity is likely to occur."

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