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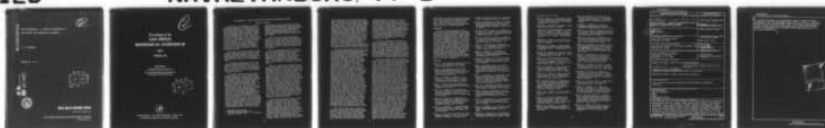
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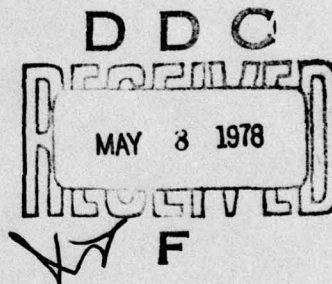
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EEG BIOFEEDBACK: A CRITICAL EVALUATION OF  
THE RESULTS AND UNDERLYING RATIONALE

R. E. TOWNSEND

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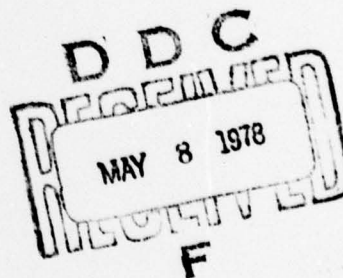
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## EEG BIOFEEDBACK: A CRITICAL EVALUATION OF THE RESULTS AND UNDERLYING RATIONALE

Richard E. Townsend\*

The application of biofeedback to the electroencephalogram (EEG) involves basically the same procedures as biofeedback - mediated control of any other physiological activity. The subject is provided with a visual or auditory signal that makes available information about the presence or absence of the desired EEG activity. There is now little question that within certain limits, individuals can learn voluntary control of the frequency content of the EEG. If one examines the current literature, it also is apparent that interest has now shifted from the question of if, to the questions of how voluntary control of the EEG is accomplished, whether or not the self-regulated EEG parameters can exceed normal baseline limits, and finally, what benefits, if any, can be expected from self-regulation of the EEG.

The question of how subjects achieve voluntary control of the EEG is one of direct operant conditioning vs. mediating variables. That is, can an individual learn direct control of his EEG or is he simply learning to produce a certain behavioral state which in turn will result in the desired EEG change?

It was perhaps prophetic when, in the first stages of alpha feedback popularity, Green, Green, and Walters (1970) stated that "there is no such thing as training in elicitation of certain brain waves, we only train to produce certain subjective states that have been shown to be associated with the desired brain waves."

In the case of alpha activity the evidence to date does suggest that control is achieved predominantly through the use of oculomotor control or attentional factors. Mulholland and co-workers have published a series of studies evaluating the role of the visuomotor system in the control of alpha activity (Mulholland & Runnals, 1962; Mulholland & Evans, 1966; Mulholland & Peper, 1971; Mulholland, 1972, 1974). They found that to increase alpha activity, subjects learn to inhibit "looking behavior" and to decrease alpha activity subjects learn to increase looking behavior. Plotkin (1976) reached similar conclusions and stated that "in the feedback augmentation of occipital alpha, enhancement and suppression of this rhythm is always mediated by learned control of oculomotor processes" (p. 92). The role of attentional factors in alpha control has been well-known since the earliest biofeedback studies when Kamiya (1974) first taught subjects to use close-up visual imagery to suppress alpha, and relaxation and calm to enhance alpha activity. More recently Paskewitz and Orne (1973) and Lynch, Paskewitz, and Orne (1974) concluded that there is no direct learning

of alpha control, rather subjects learn to dis-inhibit various attentional and arousal factors that normally block alpha activity. Consistent with this interpretation, they also found that biofeedback could not increase alpha activity above that observed during baseline sessions with eyes closed or in darkened rooms with eyes open (Paskewitz & Orne, 1973).

As with other state-related physiological variables, it has also been shown that subjects can learn to control alpha activity in the absence of any feedback. Beatty (1972) verbally gave subjects appropriate strategies to produce behavioral states associated with alpha or absence of alpha and they were able to produce the desired EEG changes without feedback.

Thus, it seems apparent that direct operant control of alpha activity has yet to be demonstrated. As Lynch and Paskewitz (1971) stated, "... alpha activity is mediated by a whole host of factors and the likelihood of ever controlling or eliminating all these factors to observe only simple alpha conditioning is extremely small. Thus ... any discussion of the 'operant control of alpha activity' seems to us, operationally indefensible."

Since it is apparent that individuals can, by one strategy or another, learn to produce voluntary changes in EEG alpha activity, the next question is, why would one wish to produce these changes? In the early 1970s, there was an abundance of publications describing the benefits of the "alpha state." Alpha enhancement was associated with feelings of serenity, relaxation, and improved task performance (Nowlis & Kamiya, 1970), improved delayed recall (Gannon & Sternbach, 1971), extrasensory perception (Honorton, Davidson, & Bindler, 1971), and decreased need for sleep (Regestein, Buckland, & Pegram, 1973). The broad ranging expectations for alpha enhancement were perhaps epitomized by Nideffer (1973) who suggested alpha would be useful in the treatment of hypersensitive conditions, increase the ability to withstand stress, increase self-acceptance, tolerance, understanding, and empathy for others, and would enable individuals to recognize and experience a non-critical, ego-free state. In the face of such positive benefits, it is no wonder that laymen and some researchers embraced alpha as the panacea of the 1970s.

The uncritical acceptance of alpha was short-lived however, and evidence quickly began to accumulate, suggesting that a more cautious approach was required. The subjective states of calmness, tranquility, inner peace, and altered states of consciousness often associated with alpha enhancement were found to be a function of depth of relaxation (Marshall & Bentler, 1976) and expectation (Walsh, 1974; Plotkin, 1976) rather than the abundance of alpha per se. Other studies demonstrated that alpha enhancement does not prevent the effects of sleep loss (Hord, Tracy, Lubin, & Johnson, 1975; Hord, Lubin, Tracy, Jenama, & Johnson, 1976), does not provide a recuperative

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break during sustained performance (Kamiya, 1972), does not aid memory or choice reaction performance (Beatty, 1973), and is not compatible with cognitive tasks requiring mental effort (Orne, Evans, Wilson, & Paskevitz, 1975). Alpha alone was found to have no significant effect on pain reduction (Melzack & Perry, 1975), and did not reduce tension headaches (Lehmann, Lang, & Debruyne, 1976). The positive therapeutic and subjective effects first attributed to alpha enhancement have now been shown to be largely due to the general relaxation that is often associated with abundant alpha activity. Thus we are led full circle to the statements made by some investigators at the beginning of the alpha era - alpha is a correlate of certain behavioral states, especially relaxation and lack of oculomotor activity. If the behavioral state is "good" for you and you use that state to produce alpha enhancement, then you will experience positive effects from the behavioral state, but not from the alpha per se.

Theta and the sensorimotor rhythm are the other two EEG frequency bands that have been subjected to voluntary manipulation by biofeedback methods. Since theta has been closely associated with drowsiness and light sleep it is natural that investigators would attempt to increase drowsiness by training for enhancement of theta, and prevent drowsiness by training for suppression of theta. As in the case of alpha enhancement, enhancement of theta activity appears to be dependent on establishment of a behavioral state which is accompanied by maximal theta, e.g., very deep relaxation and drowsiness. This was best demonstrated by Sittenfeld, Budzynski, and Stoyva (1976) in a study of electromyographic (EMG) feedback followed by theta feedback. Subjects with high and low EMG levels were chosen. A comparable number of high and low EMG subjects were given either theta only training or EMG relaxation training followed by theta feedback training. The high EMG subjects were only able to produce increases in theta if they were initially given EMG training to produce a relaxed, drowsy state. Low EMG subjects did no better with the two phase training than they did with the theta training alone. Thus it appears that theta enhancement is mediated by the behavioral state of deep relaxation or drowsiness. Producing a drowsy state as a way to enhance theta would seem to be useful in cases of sleep-onset insomnia and the converse, prevention of drowsiness as a way to suppress theta, would seem to be useful in situations where drowsiness is not desirable. Attempts to use theta suppression as an aid in maintaining performance by preventing lapses due to drowsiness, have met with mixed success. Subjects trained to enhance theta, performed less well on a vigilance task than subjects trained to suppress theta (Beatty, Greenberg, Deibler, & O'Hanlon, 1974) but in a subsequent study, theta suppression had no effect on vigilance performance (O'Hanlon & Beatty, 1977).

There is also evidence that sleep deprived subjects, those who would be most in need of theta suppression training as a way to maintain performance, are unable to maintain theta suppression during task performance (Wilson, Mord, Townsend, & Johnson, 1976). One of the problems in using theta suppression as a method to maintain performance, is the fact that there can be fairly sub-

stantial changes in the amount of theta with no accompanying changes in performance. In 1962, Williams, Granda, Jones, Lubin, and Armington pointed out that the amount of theta is not related to performance in rested subjects. A recent replication using more precise spectral estimates of theta intensity also found no relationship between reaction time or errors of omission and the amount of theta activity in either the 8 seconds or the 1 second just before stimulus presentation in rested subjects (Townsend, Mord, & Johnson, 1976). In the sleep deprived subject, however, theta intensities do become predictive of performance (Williams et al., 1962).

In answer to the question of whether direct, operant conditioning of theta has occurred, the answer appears similar to that for alpha activity. There is no convincing evidence that theta enhancement or suppression represents anything more than an EEG correlate of voluntary changes in behavioral state, e.g., deep relaxation and drowsiness produce theta enhancement and alert wakefulness produces theta suppression. As in the case of alpha control, the best supported and most parsimonious explanation for theta control is that the observed EEG changes are secondary to voluntary changes in arousal level.

The third major application of biofeedback to the EEG has been to enhance the sensorimotor rhythm (SMR). Interest in SMR as a means for suppressing seizure activity developed from the findings of Sterman, LoPresti, and Fairchild (1969) that in cats, training to enhance a 12-16 Hz rhythm over centro-frontal cortex caused delayed onset of convulsions following injection of a convulsant drug, compared to seizure onset latencies in cats not trained for SMR. Subsequently, Sterman, Howe, and MacDonald (1970) noted that SMR trained cats had greater spindling when asleep and, thus, proposed that SMR and sleep spindles have a common neural basis concerned with the suppression of movement. Despite what appeared to be a well-founded relation of SMR enhancement and sleep spindles to movement suppression in cats (Chase & Harper, 1971; Roth, Sterman, & Clemente, 1967; Sterman, Wyrwicka, & Roth, 1969), the evidence for a scalp-recorded SMR in man has been less consistent, and no significant relationship has been found between sleep spindles and movement suppression in man (Johnson, Hansen, & Bickford, 1976). A major problem in the interpretation of the effect of SMR on seizure rates has been the tendency to report results based on very small numbers of patients (one to four), a lack of appropriate control groups, and a lack of quantitative evidence for enhancement of SMR accompanying seizure suppression. Reductions in seizure rates have been obtained with biofeedback training for 12-14 Hz activity (Sterman & Friar, 1972), 12 Hz activity (Finley, Smith, & Etherton, 1975), 6-12 Hz activity (Kaplan, 1975), combined enhancement of 18-23 Hz with suppression of 6-9 Hz activity (Sterman, MacDonald, Lucia, & Walsh, 1976), and combined EMG relaxation and alpha enhancement (Johnson & Meyer, 1974). Johnson (1977) has suggested that EEG biofeedback may serve the function of focusing attention and thus may inhibit seizure activity just as mental arithmetic or digit counting can suppress seizure activity. Studies of epileptogenic and non-epileptogenic single units (Wyler, Lockard, Ward, & Finch, in



press) suggest that any controlled EEG activity may help to prevent recruitment of surrounding neurons into a large enough focus to initiate an ictal event. Thus, it is not clear that SMR, if it exists in man, has any unique seizure suppressing function compared to other EEG frequencies.

### Conclusions

The fact that the gross frequency content of the EEG can be voluntarily manipulated is not new. Berger (1930) observed that subjects could suppress and enhance the new found alpha or "Berger" rhythm by opening and closing their eyes. While this may seem trivial compared to the electronic sophistication of EEG biofeedback, there is little evidence to suggest a fundamental difference. After seven years of fairly intensive study, there is no convincing evidence for direct operant control of EEG activity in man, or that enhancement or suppression of any given EEG frequency band per se will result in significant changes in an individual's mental or physical health. In its broadest sense, EEG biofeedback might best be viewed as a teaching machine which can help individuals learn to maintain certain behavioral or mental states consistent with production of a desired EEG pattern. Whether such procedures have a uniquely useful application has yet to be clearly demonstrated.

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