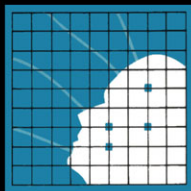


TASK
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REPORT

19



BIOFEEDBACK

Task Force Reports

This is the nineteenth report in a monograph series authorized by the Board of Trustees of the American Psychiatric Association to give wider dissemination to the findings of the Association's many commissions, committees, and task forces that are called upon from time to time to evaluate the state of the art in a problem area of current concern to the profession, to related disciplines, and to the public.

Manifestly, the findings, opinions, and conclusions of the Task Force Reports do not necessarily represent the views of the officers, trustees, or all members of the Association. Each report, however, does represent the thoughtful judgment and consensus of the task force of experts who formulated it. These reports are considered a substantive contribution to the ongoing analysis and evaluation of problems, programs, issues, and practices in a given area of concern.

Donald Langsley, M.D.
President, APA, 1980-1981

BIOFEEDBACK

Report of the Task Force on Biofeedback of the
American Psychiatric Association

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FOREWORD

We wish to acknowledge the cooperation and assistance of the Biofeedback Society of America in making available to us a series of their own Task Force Reports. While the present report is focused primarily on issues relevant to psychiatry, their reviews have provided valuable background. We are indebted to Dr. Johann Stoyva for supplying important information regarding the biofeedback therapy of headaches. Drs. Ernest R. Hilgard and Milton Perloff provided detailed comments on all aspects of the report. We appreciate the patience of the Council on Research and Development and the continuing support of Dr. Edward Sachar who was responsible for the liaison between the Council and this Task Force.

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CHAPTER I

INTRODUCTION

What is Biofeedback?

The effective functioning of any system—electrical, physiological, or social—is contingent upon the return to the system of information concerning its performance. The loop which provides the system with information about its output is called feedback. All of the homeostatic mechanisms of the body depend upon complicated interrelated feedback loops which serve to stabilize the internal environment. Similarly, all learning depends upon knowledge of results. Practice makes perfect only if the individual is aware of how well he performs as he practices.

An illustration of the significance of feedback involves the acquisition of speech. The child learns to speak by imitating the sounds he hears; the adequacy of learning depends upon his ability to compare the sound of the words he generates with that of the words generated by those around him. For this reason, individuals who become deaf later in life continue to speak without much difficulty, but children born deaf were, until relatively recently, unable to speak. Only when special techniques were developed by which the deaf child could learn the consequences of his attempts to speak, using visual and tactile cues to make up for his hearing deficit, did it become possible for totally deaf individuals to learn intelligible speech. In a sense, teaching a deaf person to speak is an example of biofeedback. Alternative feedback channels not ordinarily available to the individual must be provided so that he may acquire volitional control over speech. It is not surprising that the earliest applications of biofeedback were in the context of rehabilitation. These included not only teaching the deaf to speak or the blind to see with their hands and ears, but also in the retraining of muscles after injury or a paralytic disease, particularly when it was necessary for the individual to utilize the remaining muscle groups in a novel manner. Though the accomplishments of rehabilitation were impressive, the full implications of providing new channels of feedback to the individual were not generally appreciated because they were used to teach “ordinary” skills, within the behavioral repertoire of most individuals.

The Confluence of Findings that Led to the New Field of Biofeedback

A number of diverse studies during the 1960s appeared to demonstrate that an evolving technology provided the means to teach volitional control over physiological functions which were generally believed to be involuntary. Since a major strand running through this work was couched in conditioning terminology, it seems relevant to review the major theoretical controversies which inspired much of the research.

For many years it had been accepted that physiological changes such as increases in heart rate, the electrodermal response, blood pressure and the like could only be brought about in a reflex way. Classical Pavlovian conditioning was seen as the mechanism whereby visceral learning took place. In this process the organism is conceived as essentially passive, automatically responding to what is being done to it. For example, a painful shock may elicit an increase in heart rate. If the shock is consistently preceded by a tone, the tone will begin to elicit the same response in a lawful, predictable fashion. The issue is not one of motivation. It does not matter what the organism wants; rather the response is evoked involuntarily in a reflex-like manner, initially by the shock and subsequently by the tone as well. Using such procedures, any physiological change that could be elicited involuntarily by one stimulus as an unconditional reflex could be conditioned so that it would be evoked by a different stimulus as a conditional reflex. (Those theories which emphasized the role of conditioning in the acquisition of the physiological responses underlying phobias, anxiety states, and other conditions of inappropriate physiological arousal leaned heavily on the Pavlovian model to explain how some innocuous stimuli acquired the ability to elicit physiological responses that would have been an appropriate reflex response only to fight or flight stimuli.)

An entirely different mechanism, however, is relevant to the learning of volitional behavior. This model, known as instrumental or operant conditioning, involves reinforcing the organism when it is producing a desired response. In animal research, for example, the rate and consistency of lever pressing can readily be controlled by the frequency and consistency with which the hungry animal is rewarded with food (contingent upon the desired response). Whereas in Pavlovian conditioning the organism's "response" involves any physiological change normally only involuntarily elicited by a reflex, in operant conditioning the response is any item of behav-

ior normally under the organism's volitional control. One may think of the reinforcer in operant conditioning as merely serving to make it worthwhile for the organism to choose to do something that in any case was already well within its capabilities. Human operant conditioning, for example, involves systematically providing some desired reward—money, tokens, or most often, social reinforcers such as encouragement—as, for example, an agoraphobic patient walks farther and farther away from the safety of his room. The patient knows how to walk; the therapist using the operant procedure makes it important enough for the patient to practice that skill despite his fears.

While the application of operant conditioning procedures to modify the behavior an individual chooses to carry out makes intuitive sense, it seems somewhat odd to request that an individual raise his heart rate since this seems outside of his capabilities regardless of the inducements we choose to offer. Indeed, a two-factor theory of learning—classical conditioning for visceral learning and instrumental conditioning for the acquisition of behaviors—originally outlined by Skinner (1935; see also Mowrer, 1960) seems to best fit the available data.

During the 1960s, a number of investigators sought to apply the operant model as a means of modifying visceral activity. For example, Engel (1972) initiated studies to control some cardiac arrhythmias. Noting that slight changes in heart rate would virtually eliminate the arrhythmias, he set about training patients to control their heart rates. Using electronic equipment to compare the frequency on a beat-by-beat basis, he arranged matters so that when subjects continued to raise their heart rates even by a slight amount, a signal light would be turned on. He then instructed subjects to keep the signal light on. Conceptualizing the light as a reward, he observed that subjects could indeed learn to increase their heart rates. Though the extent of control was limited, there was no doubt that subjects did indeed learn to alter their heart rates and in some cases gained control over their arrhythmias. Heart rate control was also documented by Shapiro, Tursky, and Schwartz (1970) who went on to show that the change in heart rate was independent of changes in blood pressure. Kimmel (1967), and subsequently Shapiro, Crider, and Tursky (1964), documented operant control of the electrodermal response. Basmajian (1963) showed that the firing of single motor neurons could similarly be brought under volitional control, a control sufficiently delicate to allow some subjects to send Morse code in this fashion.

These studies, most of which were carried out with human subjects, seemed to show that, contrary to previous beliefs, appropriate

technology could help individuals acquire volitional control over a number of different autonomic functions. Further, this control seemed to involve considerable specificity and could not be explained simply as due to "thinking arousing thoughts." Nonetheless, there was considerable controversy concerning these studies, and a number of scientists were unwilling to accept the observations as compelling evidence that cortical control over autonomic activity had been acquired. Rather, they argued that subjects merely learned to change their physiological response either by playing mental games, taking advantage of classically acquired conditional reflexes in their imagery or perhaps utilizing covert muscular activity. It remained for Miller and his associates to document the radically new nature of the emerging technology.

In a now classic paper, Miller (1969) reported a number of studies carried out on curarized animals—thereby eliminating the possibility of muscular mediation—which documented that a variety of vascular responses could be brought under operant control. For example, heart rate and blood pressure could be varied independently of each other, intestinal motility could be altered, and the specificity of the responses was sufficiently great that it was possible to train rats to increase blood flow in one ear while simultaneously decreasing it in the other ear (DiCara and Miller, 1968). The importance of showing that operant procedures could directly cause dramatic changes in autonomic bodily functions was illustrated in a study by DiCara that showed that rats could be operantly trained to slow their heart rate until the heart stopped. This experiment graphically revealed the power of this new procedure and at the same time highlighted why some physiologically-oriented scientists had difficulty in conceiving that nature would entrust such an important function as heart rate to the volitional control of any organism. Nonetheless, Miller's work effectively served to provide the scientific legitimacy needed for the emerging field. It appeared to resolve the lingering doubts about the possibility of achieving operant control over physiological processes and seemed to justify a major effort to develop this new technology into a useful therapeutic modality.

The third major interest developed independently and involved the work of Kamiya (1969) who appeared to demonstrate that brain waves, most notably alpha wave activity, could be brought under operant control. Though brain wave activity had not really been considered analogous to visceral activity, it had nonetheless been assumed to be beyond volitional control. Kamiya not only documented that subjects increased alpha density in a dimly lit environment but also found that these increases appeared to persist beyond the training. His subjects

reported that they preferred the experience of increasing alpha density to that of blocking alpha. Finally he found that many subjects reported feelings that had traditionally been associated with meditative disciplines, such as calm, passive, relaxed, and pleasant, while producing large amounts of high density alpha in their EEG.*

The findings of Kamiya were widely reported in the media and supported by the work of Brown (1971), Nowlis and Kamiya (1970), Hart (1968) and others. Equally important perhaps, these findings were congruent with the observations of investigators who noted large high-density alpha in the EEG of Zen masters and yogas respectively seeking to establish a link between the consequences of meditative training and alpha feedback training. Though these studies tended to lack systematic controls, they caught the imagination of many serious scientists, the media, a considerable number of technologically oriented individuals, and a sizable segment of the counter-culture. The means appeared at hand to achieve within a few, relatively simple sessions results that had previously required years of difficult discipline and training. The presumed alpha state appeared to link meditative states, the creative process, the hypnotic phenomenon, and much else that many individuals deemed desirable. The possible significance of these phenomena for psychiatry seemed self-evident and will therefore be discussed in some detail in this report.

Other work in EEG control showed that individuals could be trained to control their evoked cortical response (Fox and Rudell, 1968), to vary the amount of theta in their EEG (Beatty, Greenberg, Deibler, et al, 1974), and even to gain control over the sensory motor rhythms which increased their threshold for convulsions, making this type of training a potentially useful treatment for epilepsy (Serman and Friar, 1972).

Biofeedback and Relaxation

As has already been noted, the use of muscle tension feedback has a considerable history in rehabilitation. However, the application of this technique to lower the overall level of muscle tension did not develop until the late 1960s. Stoyva and Budzynski (1974) showed that frontal muscle feedback could be employed to train people in general relaxation, and that while EMG feedback was initially specific in its

*Independently of Kamiya, Mulholland (1968) reported that subjects could learn to increase or decrease alpha density. However, he did not generally observe the association between alpha density and subjective states reported by Kamiya.

effects, it soon generalized so that as the level of frontalis muscle tension was reduced, individuals became progressively more relaxed. This was indicated by the level of muscle tension in other muscle groups as well as by their verbal reports. This type of training has been widely applied in the treatment of tension headaches and other high arousal states.

Another parameter which has been used for biofeedback training is finger temperature. It has long been recognized that anxiety leads to peripheral vasoconstriction—the cold hand of the frightened person. Objective psychophysiological studies have shown that finger temperature and forehead temperature vary inversely so that increasing finger temperature and decreasing forehead temperature are associated with increased relaxation and decreased anxiety. In the context of an early feedback study, Sargent, Green, and Walters (1973) noted one of their patients who had frequent migraine attacks became symptom free in the context of an experiment involving training to raise finger temperature. This encouraging finding has led to the use of finger temperature training as a treatment of migraine, based in part on the logic that the same process which causes vasoconstriction of the forehead is likely to decrease the vasodilation of the cerebral vessels which is the generally accepted mechanism underlying acute migraine attacks. A later section will deal with an evaluation of the studies treating headache with biofeedback and the present scientific status of that procedure. We are concerned here, however, with one of the byproducts of the interest in biofeedback—the rediscovery of relaxation training.

The history of relaxation training as part of various meditative disciplines goes back to early antiquity. Its more systematic and scientific study began with the work of Schultz (1932) in Germany and of Jacobson (1938) in the United States. Schultz developed a procedure he referred to as autogenic training (Schultz and Luthe, 1959) which he saw as embodying the major healing aspects of hypnosis stripped of some of its overtones. Autogenic training involved a series of learned exercises to induce calmness and relaxation. They included heaviness of the hand and, most interestingly, warmth of the hand, followed by training involving coolness of the forehead, evenness of respiration, and so on. This procedure, requiring a considerable period of training and extensive practice on the part of the student, was seen as producing predictable neurophysiological changes with profound salutary effects on a wide range of psychosomatic and psychiatric disorders. Though almost unknown in the United States until the 1960s, this procedure has long been widely used in the German-

speaking countries and in Japan. There is an extensive clinical literature on the application of this technique.

The work of Jacobson on the use of progressive relaxation stems from a somewhat different theoretical framework and focuses almost exclusively on learned muscle relaxation. While the procedures are somewhat different, the end result is intended to train individuals to be able to relax profoundly. Some of the salutary consequences ascribed to this technique show considerable overlap with those claimed for autogenic training.

Other procedures which have recently aroused modest scientific and considerable popular interest such as Transcendental Meditation (TM) seem to produce similar kinds of physical and mental relaxation. Indeed, in their popular book, Benson and Klipper (1975) discuss the relaxation response as a generic concept which has profound therapeutic effects for the individual.

The Two Major Categories of Phenomena Which Are Currently Called Biofeedback

The initial impetus toward the concept of biofeedback derived from an apparently novel procedure which would permit the acquisition of volitional control over involuntary processes. These procedures, which seemed to combine the most advanced electronic technology with the most advanced psychological technology, appeared to have the capacity of producing both very profound and very specific changes (e.g., with feedback an individual could learn to selectively fire one particular muscle bundle without causing any action potential in those surrounding it). It is easy to see why the promise of exquisitely specific behavioral interventions would suggest a myriad of new therapeutic possibilities. Some procedures, such as Engel's work on the treatment of incontinence, are dramatic examples of specific, clinically-effective applications (Engel, Nikoomanesh, and Schuster, 1974). On the other hand, in many instances the individual's ability to gain control over some particular physiological parameter may not be related to any therapeutic effects.

In contrast to highly specific effects, the term biofeedback has also been applied to an effort to train individuals to alter their overall state of arousal. In this context, it has been used quite loosely, especially since it has become a fashionable term. Thus, it is common for individuals to consider themselves as practicing biofeedback when teaching one of the many forms of relaxation training. It was this kind of

general effect which was initially claimed for alpha feedback training. The only form of feedback training which directly relates to muscle relaxation involves EMG feedback. Though this procedure tends to be effective, the extent to which feedback itself is important rather than instructions, the individual's expectancies, and the cognitive changes associated with them, remains to be evaluated. In the next section, we will discuss some of the conceptual and methodological problems in evaluating the effects of biofeedback. While there seems little doubt that relaxation training can produce profound and important changes, it remains to be established how much the specific feedback procedures add other than to make simple meditative or self-hypnosis procedures more palatable.

No commentary on biofeedback would be complete without some reference to its link with current metaphysical preoccupations. The "new morality" manifested, for example, by the flower children of the 1960s represents a developing *zeitgeist*. That development is reflected in the current growth of fundamental Christianity, Zen Buddhism, Transcendental Meditation, *est*, etc. To the individual who has adopted a metaphysical position focusing on personal integrity, biofeedback appears to be an easy way of taking charge of one's body. Like the Tantric yogi, he would accept total responsibility for his bodily functions (and to everything else in the universe) and biofeedback seems like Yankee know-how's answer to the austerity of apprenticeship to a Tibetan Rinpoche. The virtues of integrity and responsibility are obvious, but the dangerous paradoxes they represent have often been overlooked by occidental enthusiasts. For example, one sees manic depressives purchasing biofeedback gadgets and refusing to take lithium.

It is not intended here to do more than acknowledge that biofeedback impinges on moral and metaphysical areas that provide profound paradoxes. Nonetheless, it is essential that these issues be approached with an open mind lest one overlook important and relevant truths because of the manner in which they may be presented.

Summary

Biofeedback brings together innovative procedures which enable an individual to learn control over some processes over which he previously had little or no control. Though initially this was conceptualized primarily as a conditioning process, it seems likely that the availability of information about the consequences of one's efforts is

for human subjects the important novel feature in this learning process. This belief is the reason these techniques are referred to as biofeedback rather than as conditioning. We have reviewed some of the early observations which helped create wide interest among the public and the scientific community. (The journal of biofeedback, *Biofeedback and Self-Regulation*, has gained well over 3000 subscribers in the first four years of its existence.) The initial period of enthusiasm has finally given way to serious attempts on the part of reputable scientists to evaluate the early claims. While the clinical interest and the push toward application have continued with little restraint, it has become evident that many of the early claims for alpha feedback training have not been supported. Similarly, the early enthusiasm concerning the specific treatment of hypertension with blood pressure feedback has turned out to be premature, since it was no more effective than simple relaxation training. This latter observation, however, has considerable clinical significance and is important in its own right. On the other hand, some specific uses of biofeedback have received empirical support, and it seems highly likely that additional, specific therapeutic applications of biofeedback will be identified and find their application in general medicine. These issues will be discussed in the third section of this report.

From a scientific point of view, the single most troublesome problem that has faced biofeedback research is the unexplained inability of Miller and his associates to replicate their dramatic findings with curarized animals (Miller, 1969). These observations were originally made by a number of investigators within Miller's laboratory and were replicated by others, but for reasons which have remained obscure, can no longer be reproduced (see Dworkin and Miller, 1977). This is important because the work with curarized animals had shown magnitudes of effects far greater than those obtained with man or animals without curare. Not only did these studies provide a hard scientific basis for biofeedback, but they also provided the hope that we might find techniques analogous to curare which would yield massive effects.

The remainder of this report will seek to present a balanced view of the present state of knowledge concerning biofeedback and its clinical application. The first section will seek to assess biofeedback from the point of view of specific versus nonspecific effects. This will be followed by a section summarizing the work using biofeedback with psychiatric patients. A separate section will deal with the biofeedback of brain electrical activity other than alpha. It is followed by a discussion of the medical uses of biofeedback. The last section is a general discussion of the more salient aspects of the work reported and a summary of the overall conclusions.

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CHAPTER II

ASSESSMENT OF BIOFEEDBACK THERAPY: SPECIFIC VERSUS NONSPECIFIC EFFECTS*

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The question which must ultimately concern every clinician is¹ the effectiveness of the particular treatment he contemplates using. Such a question would seem easily answered by keeping proper records and assessing how well patients do with different therapeutic approaches. However, as much as many of us would like to assume that the physician's only concern is whether a treatment "works," therapeutic effectiveness alone has never been accepted as the sole criterion for assessing a treatment's potency. Thus, Christian Science and other forms of faith healing have undoubtedly brought relief to a great many individuals. Nonetheless, few physicians would be comfortable in suggesting such a course of "treatments" even if they knew that the patient was suffering from a functional ailment and might well obtain relief by way of faith healing. Indeed, most physicians and probably most patients, given the choice between a rational therapy and an equally effective faith healing procedure, would choose the former.

Medicine is not simply a pragmatic art concerned only with relieving the patient's discomfort. Rather, it is an enterprise that has always been characterized by the search for specific therapies, a search dating back to antiquity, which has begun to live up to its promise only within the last century. Previously, physicians had few effective specific treatments at their disposal. They learned, however, to recognize different kinds of diseases and their natural history. They developed an understanding of supportive measures and identified ways of relieving some of the patient's discomfort. For the physicians of earlier

*The substantive research upon which the theoretical outlook presented in this paper is based was supported in part by grant MH 19156 from the National Institute of Mental Health and by a grant from the Institute for Experimental Psychiatry. Appreciation for their helpful comments is expressed to David F. Dinges, Emily Carota Orne, William M. Waid, Mae C. Weglarski, and Barbara R. Brandt.

times, medicine was truly both an art and a science. The importance of such nonspecific factors as food, rest, and general hygiene was recognized along with such factors as the will to live. Nonetheless, there was a continuing search for specific remedies. Even while the physician could not necessarily provide a powerful therapy, he was committed to search for a rational therapy and to expand his knowledge even while he recognized its current limitations.

Though faith healing was already differentiated from medical therapy by the early Egyptians, a true understanding of nonspecific factors in treatment required a clarification of the pathological mechanisms involved in illness. As long as fever was seen as a pathology, cold packs and quinine were, in a sense, specific therapies. It required an entirely different level of understanding of bacteriology before quinine was recognized as a specific therapy for malaria, but only as an antipyretic in the treatment of virtually all other febrile conditions. Thus the concept of specific treatment has always reflected the underlying level of understanding of the conditions being treated.

Due to the discovery of a large number of specific therapies, modern medicine has become remarkably effective. There is relatively little attention paid to traditional supportive measures and the kind of nonspecific procedures which constituted much of medical practice early in the century. We are so used to seeing dramatic changes in the patient's condition following specific therapies that we tend to underrate the potential effectiveness of nonspecific factors; yet to distinguish between these two remains the central issue of assessing all treatment.

Unfortunately, the process of establishing a specific effect is by no means simple and often cannot be accomplished solely on the basis of clinical results. Many of the problems which face biofeedback research are closely analogous to those that were encountered earlier in psychopharmacology. While the pragmatic, empirical basis of much psychopharmacological research is acknowledged, no one would consider undertaking a clinical study of a new drug without extensive documentation of its effects *in vitro* and through the use of animal models. The more that is known about the various pharmacological effects of a drug, the more one is likely to be able to justify a clinical trial. Thus, while our discussion will focus mainly on the assessment of clinical studies, it should be emphasized that perhaps the greatest lack has been systematic research demonstrating clinically relevant effects with biofeedback in animals or normal human subjects.

Following a brief discussion of the problems inherent in assessing biofeedback, these issues will be illustrated by reviewing the available data in the treatment of different kinds of headaches. An analysis

will be carried out to clarify the kind of empirical questions which need to be addressed.

The Placebo Control

Psychopharmacology has developed the placebo control because any pill given with conviction by a physician is likely to produce some therapeutic effect. It also soon became apparent that if the physician knew which pill was the active drug and which was the placebo, the patients given the real drug almost invariably did better. However, here too subtle differences in the way patients were treated soon became apparent. Most physicians find it extremely difficult and uncomfortable to knowingly administer an inert agent and can rarely do so with conviction. Further, the physician who knows that the patient is receiving an experimental drug tends to be considerably more careful in evaluating the patient and expresses far more interest and genuine concern about side effects. In contrast, the patient receiving a placebo who reports a peculiar ringing in his right ear will be treated with benign neglect. For reasons such as these, double- and triple-blind procedures are now considered necessary in psychopharmacology. However, even with these, care is still essential. For instance, I recall a double-blind study of phenothiazines where the medical staff was indeed kept blind but where the patients had no difficulty determining whether they were on the active drug because of the ubiquitous dry mouth which was associated with it. This underlines the need to use active placebos which produce analogous side effects in order to make sure that the patient, as well as the medical staff, is kept blind.

As useful as placebo controls are, they are more complex procedures than they appear to be. Two examples seem particularly relevant. A number of studies comparing morphine with placebo did not find a significant difference in pain threshold during carefully controlled studies in the laboratory. It is worth noting that were it not for the dramatic clinical effectiveness of morphine, a "hard nosed" colleague might well have assumed that there is no specific therapeutic action of morphine. Fortunately, further work eventually identified the problem. The pain threshold for a stimulus such as electric shock, with a rapid onset and offset, is relatively unaffected by morphine. On the other hand, the threshold of ischemic muscle pain or other pain stimuli which lead to a gradually increasing pain intensity, particularly involving C fibers, is clearly increased by morphine as compared to placebo (Beecher, 1959). This illustrates the need to make certain

that the dependent variable chosen reflects that mechanism which is affected by the drug under investigation.

Another difficulty with placebo controls is relevant to the present discussion. The widely used mild tranquilizer, meprobamate (Equanil or Miltown) did not yield better results than placebo in several carefully controlled clinical trials. Uhlenhuth, Rickels, Fisher, et al (1966) carried out a study with profound implications. The physicians in the study were instructed to administer the drug either with great conviction, assuring the patients that they were receiving a potent drug which was specific to their difficulty and would help them, or to give the drug in a "scientific" and "neutral" manner, indicating that it might be helpful to them. Drug and placebo were administered in a double-blind fashion with both styles of interaction. Interestingly, when the medication was given in a "scientific" manner there was no difference between drug and placebo. However, when the medication was given in a way that served to convey the expectation of improvement, meprobamate was significantly more effective than placebo, even though they were both administered in a double-blind fashion. It would appear that some specific therapeutic effects can be seen only if the drug is administered to patients with appropriate expectancies.

These last two points are emphasized because there is a risk of overlooking a potent, specific therapeutic effect even in what appears to be a well-designed double-blind study and underline the importance of not dismissing potentially promising techniques on the basis of a single study.* Of course, this risk is minimized if one does not expect complete answers to come from clinical studies but other information, *in vitro* studies, and laboratory research with normals relating to the pathophysiology of the condition to be treated are also available.

Problems of Assessing Biofeedback Research

The first stage of research is to discover a phenomenon worthy of systematic assessment. It is hardly sensible to design a complex study to investigate an effect that is either trivial or has not been documented by simple clinical observation. Biofeedback therapy initially appeared to be based on sound animal studies which documented that

*It should be noted that there are negative as well as positive placebo effects (Lasagna, Mosteller, Von Felsinger, et al, 1954). Thus, patients may show a wide variety of side effects to placebo, including a profound worsening of the presenting complaint. Since patients may have very different attitudes toward biofeedback than toward medication or psychotherapy (see page 35 in Dr. Stroebel's chapter), the possibility of negative placebo effects due to such differential expectancies also needs to be recognized.

profound changes in visceral responses could be produced by the systematic application of instrumental conditioning. These studies were extended to show that in normal human subjects significant changes could also be brought about. However, it soon became evident that with human subjects the information component of the conditioning procedures was more important than the specific reinforcing component—hence the emphasis on feedback rather than on conditioning. Against this background, biofeedback technology evolved to treat an exceedingly broad range of medical and psychological problems.

A burgeoning amount of literature reported results with individual cases and small series of cases. Studies of larger numbers of cases including control patients have begun to appear only recently. Thus, now one can begin to form an assessment of biofeedback as a specific treatment. We will review the difficulties facing such a task because, just as there is a tendency to initially accept early findings uncritically and with great enthusiasm, there is an equally troublesome tendency to dismiss promising findings as “simply due to non-specific effects” as critical studies are published which may themselves suffer from serious methodological limitations.

Unfortunately, any dramatic and novel treatment procedure will, of course, embody placebo components every bit as profound as those associated with medication (Shapiro, 1964; Stroebel and Glueck, 1973). The problems of control for such nonspecific placebo components are even more complex than those in psychopharmacology noted earlier.

The most rudimentary control is to compare treated and untreated groups. If there are no differences found with such a comparison, it is very unlikely that an important therapeutic effect is overlooked. Indeed, there is neither a specific therapeutic effect nor a placebo effect worth considering. If we find the treated group does significantly better than the untreated controls, we have, however, established only that there is a therapeutic procedure which deserves further evaluation. Even this conclusion should be reached with caution if it turns out that self-selected patients seeking biofeedback are used for the treated group and others are used for the untreated group. Unfortunately, what to the statistician is an obvious and rudimentary *sine qua non* of evaluation—random assignment to groups—is difficult to achieve in clinical practice and therefore lacking in many of the reports. Patients who view themselves as being treated do better than those who view themselves as not being treated, the important dimension being that treated patients expect to improve. This expectation, combined with what is interpreted to be a powerful treatment, will tend to produce a difference.

We ought not, however, to conclude that simple observational data comparing treated and untreated patients is necessarily trivial, if the condition being treated is clearly delineated and if the natural history of the disease is known. Such data may be quite powerful. For example, the first reports of successfully curing subacute endocarditis with penicillin were quite sufficient to electrify the medical community because the natural history of the disease was progressive deterioration and death. Under such circumstances, a control group is hardly necessary. The smaller the difference in results between the novel treatment and the natural history of the disease with a variety of other treatments, the more important controlled evaluation research becomes. Few biofeedback studies have concerned themselves with the natural history of the conditions that are being treated or have considered the results obtained with alternative therapies which, depending on their nature, would give some estimate of the extent that expectational effects can affect the condition. Many conditions treated by biofeedback, such as headache, insomnia, and simple anxiety states, are notoriously responsive to placebo, and results can be assessed only by long-term follow-up. Differences in therapeutic outcome measured over a few weeks are unlikely to reflect the kind of improvement which therapy should aim for. Thus, only if patients with these conditions continue to do well a year or two after treatment commences is it likely that one is dealing with specific therapeutic effects.

Though the discussion below will focus on various methodological refinements which should improve the adequacy of assessing possible specific effects of biofeedback, it is all too easy to become preoccupied with the methodological elegance of a design and overlook the far more important issue of considering the natural history of the illness for which a specific treatment is being evaluated.

What Constitutes an Appropriate Control Group for Biofeedback Therapy?

As in all therapy research, an appropriate control group cannot be defined without a very clear understanding of the hypothesis being tested. For example, to determine whether a particular treatment is better than no treatment is relatively easy. One needs only to compare an untreated group with the treated group.* However, as has been discussed above, without controlling for placebo effects it is virtually impossible to assess the meaning of such findings—especially if the

* It turns out that an untreated group which is evaluated twice as part of the study is still more likely to show improvement than an untreated group without such evaluation (see Paul, 1966).

condition being treated is known to respond to many types of therapeutic attention and various kinds of specific interventions. Therefore, the question of greater relevance is whether the treatment under investigation is a specific therapy. In other words, is the treatment in question more effective than can be accounted for by nonspecific factors? This demands control procedures which eliminate the specific therapeutic component but are careful to include the other factors which might contribute to improvement, such as maximizing the patient's expectation of help, encouraging him, impressing him, providing a vehicle for the therapist's interest to be expressed, and the like.

While it seems a simple matter conceptually to define what is meant by specific and nonspecific treatments respectively, this distinction is often by no means intuitively clear. Indeed, what constitutes a specific treatment depends upon the investigator's theoretical orientation. For example, the psychopharmacologist studying the action of an antidepressant drug views the nature of the interaction between the physician and his patient as a nonspecific factor. Indeed, within the limitations of the placebo model discussed above, the placebo group will have the benefits of the same helpful doctor-patient interaction as those receiving the drug. From the point of view of a therapist studying the psychological treatment of depression (e.g., Beck, 1973), the patient's progressive feeling of mastery in terms of success defined by the therapist is the specific therapeutic factor, whereas miscellaneous noncontingent encouragement would be a nonspecific aspect of the total therapeutic situation. If there is an antidepressant effect of both drug and a specific psychotherapeutic treatment, it is possible, depending upon the circumstances, for either of these effects to obscure the therapeutic effect of the other.

In the case of biofeedback therapy, a specific effect is generally conceptualized as the consequence of learning to control some physiological parameter through biofeedback training. Biofeedback therapy, like many behavior modification techniques, has a compelling rationale and provides graduated steps indicative of improvement (as defined by the therapist and accepted by the patient), with ample opportunity for the patient to see the improvement. These aspects have profound effects on patients' expectancies and may well have much to do with a positive therapeutic outcome. Conceptually, however, they are clearly distinct from the specific biofeedback effects which are presumed to be the mechanism by which improvement is mediated. For this reason, research needs to distinguish between the specific effects involving the learned control of some physiological parameter as opposed to improvement due to nonspecific effects me-

diated by factors such as strong positive expectancies which the patient brings that are further enhanced by many aspects of the procedure.

Yoked controls. One procedure of apparent methodological elegance involves the use of yoked controls. In an alpha feedback study, for example, the first subject may be given alpha feedback training that uses a tone to signify changes in alpha density. The tone is recorded, and the next subject, who is the yoked control, is fed back not the tone reflecting changes in his own alpha density (which are, of course, analyzed subsequently) but rather the tone that had been produced by the first subject to whom the control is "yoked." Among the virtues of this procedure is that both the experimental group that receives feedback and the yoked control group that receives false feedback have precisely the same number of "success experiences." Since success experiences are considered important reinforcers (which, according to some theories at least, might well lead to differences in outcome), it was felt that the yoked control was particularly well suited to eliminate this potentially powerful but, from the point of view of the biofeedback researcher, nonspecific effect.

One of the difficulties, which is usually overlooked in yoked control studies, is that all too frequently the investigator is fully aware which subjects receive contingent feedback and which subjects are the yoked controls. This raises the possibility of inadvertent differential treatment, much as in single-blind studies where the physician knows which patient receives drug and which receives placebo. It is, however, possible to control this problem by using an arrangement where the therapist does not know whether the feedback signal is contingent upon the subject's physiological response or yoked, and a technician sets the machine according to a predetermined schedule prior to each treatment session.

While such a procedure would resemble a double-blind study in psychopharmacology, it still suffers from the limitations of inert placebos. With most biofeedback modalities, subjects are readily able to control their response in one or the other direction from the beginning of training. For example, with alpha feedback training, in the presence of ambient light it is an easy matter to block alpha simply by focusing one's gaze, though it may be difficult or indeed impossible to increase alpha above a certain point. With muscle tension, it is an easy matter to volitionally tense the muscles and cause dramatic changes in the feedback signal, though it is difficult to relax beyond a certain point. Heart rate can usually be increased volitionally (from a low base rate), and so on. Most individuals who undergo biofeedback training test out the system's response early in training; while they soon learn that they

have imperfect control, they also learn that they have some control during feedback training. In our own studies using yoked controls in alpha feedback training, we noted that subjects became aware that they had no control during the first session and reported experiences quite different therefore from those individuals who received contingent feedback and observed that they could affect the feedback tone (Lynch, Paskewitz, and Orne, 1974). The awareness of some control versus a lack of control creates a very different situation for the experimental subject as opposed to his yoked control. It is hardly surprising, therefore, that this superficially elegant procedure has had only limited utility in actual practice.

Nevertheless, it is possible to design a reasonably effective yoked control procedure. All subjects begin with a contingent biofeedback signal, giving them some experience with the signal in the beginning and gradually phasing out the contingent feedback, replacing it with noncontingent feedback. All of this must be done in a way that is neither apparent to the investigator nor to the patient. Unfortunately, the technical difficulties are formidable, and if a patient again tests out his control after the feedback signal has been totally yoked, he will still learn that he has no actual control. Thus, while properly executed yoked controls may be effective, there inevitably remains some uncertainty and therefore they have not been used a great deal in clinical studies.

Alternative control procedures. A different approach to the placebo control is to use feedback from a physiological system for which control could be learned but which is not expected to affect the symptom being treated. Care must be taken, however, to avoid a feedback modality that could potentially have a specific effect in its own right. For example, in the treatment of migraine where temperature feedback is often used, one might decide to use feedback of the heart rate. Simple sensors attached to the fingers would include a thermister for temperature information and a photoelectric plethysmograph from which the heart rate signals can be taken. The slowing of the heart rate might be used as a control procedure, but some would argue that this might be associated with relaxation and have a specific effect. Thus, it might be better to use variability of heart rate as a control procedure to avoid this possibility. While feedback of an irrelevant physiological system is one of the best kinds of active placebo controls, it has rarely been used because the quality of the feedback may be very different from a different physiological system. Further, the question can always be raised whether the control procedure might not in and of itself have a specific action.

An elegant control procedure which avoids the problems of false

feedback in the sense of providing experience of partial learned control and the difficulties associated with finding an appropriate inert physiological system to use instead of false feedback takes advantage of the fact that in most modalities subjects have considerable control from the beginning over one direction of the physiological response but not over the other. It is technically possible to accurately feed back decreases in alpha density which, as has been pointed out, subjects can generally produce at will without training, but to provide random false feedback for increasing alpha density. As a consequence, the subject who tests the system becomes aware that he has some control over the feedback tone when he blocks alpha. However, as he tries to increase alpha density, the tone is not contingent upon his response. This kind of procedure is probably the best way of providing an active placebo control for biofeedback research but, to my knowledge, it has only been used in one study (Otis, McCormick, and Lukas, 1974).

There are, of course, entirely different approaches to determine whether biofeedback is a specific treatment that capitalizes on individual differences. For example, if increasing finger temperature is in fact the mechanism by which a patient learns to control his Raynaud's disease, one ought not only to find differences in the incidence and severity of attacks between a group of patients treated with temperature feedback and a control group, but also to find a correlation between the ability to control finger temperature and the remission of clinical symptoms within the treatment group. Though this type of data was provided by Budzynski and his colleagues (Budzynski, Stoyva, and Adler, 1970; Budzynski, Stoyva, Adler, et al, 1973) in their studies of tension headache, it is unfortunately not generally reported.

Clinical data about biofeedback therapy that should become available. There is little excuse for not systematically recording and reporting the relationship between the acquisition of control over the particular physiological function being trained and clinical outcome. Such data involve no cost to the patient and relatively little effort to the clinician. As we have emphasized earlier, follow-up over a long period is critical to assess the effectiveness of biofeedback. One needs to know if there are any effects before being able to assess specific effects.

Finally, it is striking how few biofeedback studies have compared their results to the effectiveness of those medical procedures which are the standard, accepted treatment for the disorder in question. For example, the yardstick for assessing biofeedback treatment of migraine would seem to be the results of ergotamine therapy rather than a no-treatment control group. Such a comparison would, of course, not address the issue of whether biofeedback is a specific treatment but

rather how effective a treatment it is when compared to what is now considered standard therapy—information that is also essential for informed clinical decisions.

Is Biofeedback Treatment of Headache a Specific Therapy?

Thus far, the problems of control in biofeedback research have been discussed in the abstract. It is not possible in this report to deal with the burgeoning amount of literature in detail. However, it is useful to examine the evidence for and against the view that biofeedback of headaches is a specific treatment as an example of the kind of unresolved questions which characterize the field. The review will compare the treatment of tension headache with the treatment of classic migraine and does not attempt to be exhaustive but rather to discuss those studies which are relevant to the assessment of biofeedback as a specific therapy. We shall examine the studies, not necessarily from the point of view of the authors but rather how they relate to the issue of assessing the magnitude of potential placebo effects. Finally, we shall contrast the treatment of tension headache and migraine because this, in and of itself, may be the most meaningful evidence indicative of a specific biofeedback effect in the therapy of headaches.

An overview of findings in the treatment of tension headaches. Tension headaches are one of the most ubiquitous and uncomfortable psychosomatic complaints. Constant and nagging pain is generally experienced on both sides of the forehead or neck, though it is frequently described as pressure or “as if a band were being tightened around my head.” Headaches of this kind may occur frequently, even daily, and while the pain may be very troublesome, it does not usually prevent the patient from carrying out his daily routine. Dalessio (1972) summarizes the etiology as due to sustained skeletal muscle contraction associated with some vascular constriction in the relevant nutrient arteries.

Several studies indicate that increased frontalis muscle tension is associated with tension headaches. Thus, the frontalis EMG scores of patients tested while experiencing a tension headache were significantly higher than comparable scores of patients not experiencing such headaches while being tested (Sainsbury and Gibson, 1954). As a group, patients with tension headaches had significantly higher levels of frontalis EMG than individuals who did not suffer from tension headaches (Budzynski, Stoyva, Adler, et al, 1973; Phillips, 1977a,

1977b). Dramatically higher baseline frontalis EMG levels were reported for 18 patients with severe tension headaches than analogous EMG levels from non-headache controls (Hutchings and Reinking, 1976).

In the first controlled studies using EMG feedback (Budzynski, et al, 1973), three groups of six patients were compared. The first group had frontalis EMG feedback, the second group had false feedback, and the third group was a no-treatment control. Patients were given relaxation instructions to practice at home and had 16 thirty-minute treatment sessions over a period of eight weeks. Prior to treatment, all patients had a two week pretraining baseline. It was noted that approximately 25 percent of the subjects showed transitory reduction in headaches during these two weeks; however, these patients were excluded from the study. The main findings were that frontalis EMG feedback training resulted in a significant decrease in frontalis EMG in the group receiving feedback but not in the control group with false feedback. Four of the six patients in the treatment group showed a large decrease in headaches, while subjects in the other groups showed far less improvement. A high correlation was observed between the amount of EMG decrease in the treated group and clinical improvement. After 18 months, four of the six patients in the biofeedback treatment group maintained their gains. An ancillary criterion measure was a significant reduction in the use of headache medication or tranquilizers by patients who received biofeedback therapy but not in either the pseudo-feedback group or the no-treatment control group.

Subsequently most studies reported a 50 to 70 percent reduction in the frequency and severity of the headaches (e.g., Cox, Freundlich, and Meyer, 1975; Hutchings and Reinking, 1976; Phillips, 1977a). Most studies reported three- to six-months follow-up. However, one investigation, combining psychotherapy with EMG feedback, reported 80 percent improvement which persisted with follow-up extended over several years (Adler and Adler, 1976).

These selective findings would suggest that EMG feedback is indeed a specific therapy for muscle tension headache: a clear rationale is available, supported by studies of the pathophysiology of muscle tension headaches; one study showed that false feedback— analogous to the yoked control—was ineffective but actual feedback was effective; a highly significant correlation was seen in another between acquiring control over frontalis muscle tension and the diminution of headaches.

The important finding that contingent biofeedback was necessary to achieve the results was replicated by Wickramasekara (1972) who

used a false feedback period prior to the true feedback. He reported that there was no effect from random feedback but that patients improved dramatically with true feedback. There is only one aspect of these findings that should give one pause: namely, there is no placebo effect whatsoever reported in this study. Similarly, a more recent study comparing a group receiving false feedback with a group receiving true feedback (Kondo and Canter, 1977) noted 80 percent headache reduction with actual feedback but only a trivial decrease with false feedback. Though these findings are certainly encouraging, the inference is limited by the fact that it is quite easy for patients to become aware that feedback is noncontingent with EMG. They have only to wrinkle their brow to notice that there is no contingent change in the feedback tone. Not only is the false feedback an inactive placebo, but also the therapist is aware which group is being treated—a factor which in and of itself may play a major role in outcome.

Only one study employed false feedback in a way that was analogous to an active placebo, providing an estimate of the possible magnitude of placebo effect. Otis, McCormick, and Lukas (1974) arranged for feedback from the trapezius muscle to be contingent for increasing muscle tension but false feedback for decreasing muscle tension. Such a procedure would allow the patient to test the feedback procedure, and if he contracted his trapezius muscle, he would immediately alter the feedback tone. However, the presumed therapeutic procedure was to relax the trapezius muscle and regarding this aspect, the patient received only false feedback. The physiological data supports the view that subjects learned to tense the muscle but did not learn to relax it. Nonetheless, two-thirds of the group were without headache by the end of the study. A no-treatment control group showed no improvement. This ingenious study, which unfortunately did not include a group receiving true feedback for relaxing as well as tensing the muscles, nonetheless raises serious questions about any conclusions based on false feedback. As was noted earlier, this is the kind of control procedure worthy of more widespread usage.

A recent study comparing frontalis EMG feedback with a cognitive therapy focusing on stress coping (Holroyd, Andrasik, and Westbrook, 1977) was instructive about possible investigator bias. Here the stress coping procedure, which was designed to teach patients to think about and manage their present situation better, resulted in a 75 percent improvement versus only a 25 percent improvement in the biofeedback group. This was observed despite the fact that the EMG feedback group showed a significantly greater decrease in frontalis muscle tension than did the cognitive group. It will hardly be surprising that these authors were particularly interested in cognitive ther-

apy. The results they reported with cognitive therapy were of the order of magnitude others reported with EMG feedback, but their results with EMG were significantly below that of other studies. From the point of view of this discussion, the most interesting aspect of their report is that it is possible to see patients significantly decrease their frontalis tension, objectively verified by EMG measurements, without a concurrent improvement in clinical status.

Another recent study (Epstein and Abel, 1977) reported that while EMG biofeedback was very effective in reducing tension headache frequency, they failed to note the correlation between headache and clinical improvement earlier reported by Budzynski, et al. Further, none of their patients were able to reduce the level of EMG without feedback, suggesting that more evidence is needed about the necessary relationship between actual EMG changes and clinical improvement.

Perhaps the most relevant results in assessing the clinical effectiveness of EMG biofeedback is the comparison between the effectiveness of relaxation training and biofeedback. Four studies specifically address this issue. Haynes, Griffin, Mooney, et al, (1975) compared EMG feedback with relaxation training specifically designed for this group and a no-treatment control. Both of these treatment groups showed a significant reduction in headache compared with the controls but did not differ from each other. Cox, Freundlich, and Meyer (1975) compared biofeedback, relaxation training, and a placebo group. Again, both biofeedback and relaxation training groups were significantly improved compared to placebo in terms of frequency, intensity, hours of headache per week, and the amount of medication ingested per week. One study compared EMG training with relaxation training. Both groups who had EMG feedback showed greater improvement than the group which had only relaxation training. There was no difference between the group with EMG training alone and that with the combination of EMG training and relaxation (Hutchings and Reinking, 1976). Another study, however, reports exactly opposite findings in a study comparing four groups: EMG feedback, EMG feedback with relaxation training, relaxation training, and a no-treatment control. Chesney and Shelton (1976) report that both groups with relaxation training do equally better clinically than the biofeedback and the no-treatment control groups.

Summarizing these data, it seems clear that EMG biofeedback in the treatment of tension headache is significantly better than no treatment. While some studies find a strong relationship between the degree of relaxation which is achieved and clinical improvement, others fail to find such a relationship. While the degree of improvement is

generally over 50 percent with EMG feedback, it is as little as 25 percent in one study oriented toward cognitive therapy and 40 percent in another oriented toward relaxation training. Finally, two studies fail to show any difference in outcome between relaxation training and EMG biofeedback. One study shows EMG biofeedback to be dramatically better than relaxation training, while another reports relaxation training showing a greater benefit of the same order of magnitude with relaxation training than others report with biofeedback. Strikingly, no study shows a combination of relaxation training and biofeedback to be more effective than one or the other alone.

These findings reflect the limitations of the research design: the investigators were not blind, only one study used an active placebo and the likelihood of the strong investigator bias effects exists. Some conclusions may nonetheless be drawn with reasonable certainty. Tension headache appears to respond to either biofeedback therapy or relaxation training. The likelihood of improvement with either of these procedures is probably related to the investigator's preference for, or perhaps effectiveness with, the particular therapy. Though either of these approaches may exert a specific effect, the studies taken as a whole show that the responsivity of patients may vary considerably with the same treatment. The adequacy of the placebo controls in all but one of these studies is such that these effects could potentially be accounted for in terms of nonspecific factors. Perhaps the most serious deficits in these studies are the lack of comparability of patient populations, the lack of information about the natural history of the disorder, and the failure to compare biofeedback with alternative medical management.

An overview of findings in the treatment of migraine. The classic migraine is typically preceded by a warning aura, characterized by intense, throbbing headache, predominantly on one side of the forehead, and commonly associated with nausea. The etiology of migraine is generally related to dilation and spasms of the cerebral vessels. Attacks can usually be aborted successfully by the early administration of ergotamine. There is a strong familial incidence, and the frequency of attacks is, like tension headaches, related to general stress factors. Migraine and tension headaches are accompanied by high frontalis EMG levels. Bakal and Kaganov (1977) reported higher EMG frontalis levels in patients with migraine as compared with tension headaches, both during headache and in headache-free intervals.) Nonetheless, initial efforts to apply EMG feedback to migraine have generally been unsuccessful (e.g., Feuerstein and Adams, 1977; Phillips, 1977a; Wickramasekera, 1973). However, since the first reports by Sargent, Green, and Walters (1972) concerning the successful use

of temperature feedback of the fingers in the treatment of migraine, this procedure has been used by a large number of clinicians with highly encouraging results. The procedure merely involves attaching a thermister to the patient's finger. The output is fed into a device that modulates a tone so that it reflects changes in finger temperature. Typically the patient is asked to decrease the loudness of the tone which reflects an increase in finger temperature.

In the initial studies and in most clinical applications, the patient is instructed to repeat to himself phrases borrowed from autogenic training, in particular "I feel relaxed," "My hands are heavy and warm." The original Menninger Clinic study (Sargent, et al, 1973) reported significant improvement in 80 percent of 43 migraine patients who were followed for more than 150 days. However, autogenic phrases are not essential. Thus, for example, Reading and Mohr (1976) treated six patients unresponsive to drug therapy and reported an average symptom reduction of 70 percent without relaxation instructions. Turin and Johnson (1976), in a similar study of seven patients, reported improvement in all of their patients, but the average symptom reduction was between 40 percent to 50 percent. In a very large series treated at the Institute of Living, Stroebel (personal communication) reported approximately 80 percent improvement.

Few of these studies have, however, used independent controls, and none have used false feedback. The data which seems most directly relevant to the specificity of handwarming is a clinical report of Wickramasekera (1973) who, having tried unsuccessfully to treat two migraine patients with EMG training, offered them another type of feedback, an offer which was only reluctantly accepted after some time. Both of these patients improved dramatically when they received temperature feedback. The only other partial attempt to control for placebo effects is in the Turin and Johnson (1976) study where three of the seven patients initially received feedback for hand cooling and failed to improve despite the cognitive set that the feedback training should help. However, these patients, as well as the four other patients, improved with handwarming.

One way the issue of specificity could be addressed is to examine the relationship between the extent to which patients learn to increase finger temperature and their clinical response. However, such data are not available except in the Turin and Johnson (1976) study where there is no significant relationship.

There are, however, studies which have compared finger temperature biofeedback in the treatment of migraine with relaxation. Blanchard, Theobald, Brown, et al, (1977) compared temperature feedback combined with relaxation training with a group which received only

relaxation training. They observed significant clinical improvement in both groups with no difference between them. Andreychuk and Skriver (1975) compared temperature feedback, self-hypnosis, and alpha feedback training and found all three equally successful in the treatment of migraine. Finally, Price and Tursky (1976) compared vasodilation feedback, relaxation training, and an untreated control group and observed that patients receiving either treatment improved significantly more than the control group. Thus, despite a high degree of consensus among workers in biofeedback that handwarming is effective in the treatment of migraine, there is little hard evidence.

Certainly the available data would not permit the conclusion that temperature feedback training is necessarily a specific therapy in the treatment of migraine. On the contrary, there are no active placebo studies, there is no demonstration that clinical improvement is contingent upon learning the skill of handwarming, and relaxation training seems as effective as biofeedback.

Additional considerations concerning the biofeedback treatment of migraine and tension headache. It is certainly possible that all of the available findings are due to powerful placebo effects, and one is tempted to dismiss biofeedback as a specific therapy for headache. The best available evidence indicates that relaxation training is as effective as biofeedback for classic migraine and tension headaches. However, while relaxation training is a nonspecific treatment from the point of view of biofeedback therapy, it is far from a true nonspecific effect. Though appropriate rapport between doctor and patient is important, relaxation training involves learning a specific skill which a patient is required to practice. Individuals differ in their ability to acquire this skill and in their willingness to exercise it. While clinical reports tabulate only whether patients are offered the training, techniques for evaluating the ability of patients to relax successfully need to be utilized, and the frequency of practice carefully assessed. If future research documents a relationship between the acquisition and practice of relaxation skills and therapeutic outcome, the procedure can and should be separated from the general category of nonspecific responses.

One of the important questions which will need to be resolved is whether relaxation training involves the identical underlying process as that which follows from biofeedback training. In some studies, the groups receiving biofeedback and biofeedback plus relaxation training are significantly better than those receiving relaxation training alone. In others, those receiving relaxation and biofeedback plus relaxation training are both better than biofeedback alone. But in none of the studies does the combination of biofeedback and relaxation

training do better than either of the two modalities alone. This suggests the possibility that the underlying processes are indeed similar. Unfortunately, all of the available studies using this type of factorial design are carried out by individuals who tend to focus preferentially on either biofeedback therapy or relaxation training, and little concerted effort is made to provide a meaningful rationale for the combination of these two approaches. Relaxation training requires the patient to be passive and allow the process to take place; biofeedback tends to demand more active participation, especially in the early stages of training. Unless some concerted effort is made to integrate these two approaches, it is not surprising that the patient would tend to focus his response on one or the other of these modalities if he is presented with both. This might be an alternative explanation for why no additive effect of these two approaches has thus far been documented.

The noteworthy observation, however, is that EMG feedback is consistently reported not to be particularly effective in the treatment of migraine, while handwarming does not seem to relieve tension headaches. It is difficult to reconcile the notion that biofeedback leads to a process identical to that of relaxation training with the apparent specificity of the modality of biofeedback for the nature of the headache. Unfortunately, with the exception of a clinical descriptive report by Wickramasekera (1973) where two migraine patients failed to improve with EMG feedback but showed dramatic improvement with handwarming, no substantive study addresses this question. Nonetheless, it is the one point about which there is virtual unanimity among biofeedback therapists, and its rigorous test would go far toward documenting a specific effect associated with biofeedback training of hyparoused states. Technically it would be relatively straightforward to compare handwarming and EMG feedback in the treatment of carefully diagnosed patients with classic migraine and tension headaches. Half of each patient group would be assigned to EMG feedback and the other half to temperature feedback. The therapists treating the patients could avoid discussion about the patients' headaches and thus be kept blind as to the kind of problem involved. Assessment would be carried out by an independent clinician, and after a period of 12 weeks, a cross-over design would make it possible to determine definitively whether there is a specific differential effect due to the biofeedback modality. If there are no differences between these two types of biofeedback, there would be little remaining doubt that we have been dealing with a complex, impressive, and up-to-date placebo. On the other hand, if, as I believe is likely, it turns out that the clinical wisdom of the field is based on actual differences in effec-

tiveness, then a specific effect of biofeedback training will have been documented.

If specific effects of biofeedback in the treatment of headache can be established, it would become all the more important to begin a serious examination of relaxation therapy as an important specific therapeutic intervention. Teaching individuals to relax involves a special kind of skill learning, different from simple attention or encouragement, and also likely to be more than some form of complex placebo. Serious attention needs to be paid to the question of whether individuals differ in the extent to which they can benefit from relaxation training and biofeedback, and how these modalities can be combined to yield a synergistic effect. Finally, we need to explore the effect of psychological factors as they interact with the kind of therapeutic outcomes that may be expected from relaxation training, biofeedback, and various specific psychotherapeutic interventions.

It is easy to become unduly critical of biofeedback as a field since one would have expected, given the widespread interest in this modality and the claim of biofeedback to involve the acquisition of highly specific skills, that its specificity of therapeutic action would have long since been documented. It is well to remember, however, that most of the issues which bedevil biofeedback are those with which psychiatry has long had to struggle. While it is far easier to specify the nature of the intervention, making a controlled study of biofeedback considerably less complex than similar studies of psychotherapy outcome, it still remains a formidable task. An effort has been made to illustrate the complexity of the issues and why definitive answers are not yet available. Some possible ways of beginning to address the question of whether biofeedback therapy for headache is a specific treatment have been suggested. Until such answers are available, one can hardly justify the claim that biofeedback is a specific treatment of headache, but one ought to be equally cautious in prematurely rejecting it.

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CHAPTER III

APPLICATION OF BIOFEEDBACK TECHNIQUES IN PSYCHIATRY

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Numerous anecdotal, single case, and uncontrolled small sample studies claim efficacy for the treatment of a variety of psychiatric conditions with biofeedback (Blanchard, et al, 1974; Blanchard, et al, 1978; Legalos, 1973; Rickles, et al, 1978; Wentworth-Rohr, 1977) However, to the extent that the domain of psychiatry encompasses disorders enumerated in DSM-III, no *convincing* evidence supports the application of biofeedback as a specific treatment for any listed condition, including the psychophysiological disorders listed under category 316.00. Positive outcomes have been reported only when the biofeedback techniques are combined with a passive, non-threatening therapeutic alliance, including components of psychotherapy, behavior therapy, Progressive Relaxation, Autogenic Therapy, physical therapy, Relaxation Response, Quieting Response, imagery, hypnotic variants, and coaching procedures (Fuller, 1977; Gaarder and Montgomery, 1977).

This makes it difficult to evaluate the efficacy of biofeedback techniques in psychiatry. Namely, are aspects of this mixture of biofeedback technology and other techniques essential for therapeutic results? Based on present evidence, this question is difficult to answer. Stroebel and Glueck (1973) have speculated:

... if it ever will be possible to separate out the real effects in human subjects. Probably not, much to the anguish of our scientific side, which demands objectivity, experimental precision, and epistemological surety in our quest for a scientific understanding of man and his problems. (p. 379)

They have even suggested that in encouraging self-responsibility on the part of patients, biofeedback techniques may serve as nonspecific

“ultimate placebos” by fostering the persistence of these effects over time, independent of the therapist.

As noted elsewhere in this report, the concept of specificity of biofeedback modalities is increasingly unclear (Silver and Blanchard, 1978). Even in relatively non-controversial applications of biofeedback technique, namely physical medicine and neuromuscular re-education, the issue of specificity versus nonspecificity of effects is coming under increasing scrutiny (Wolff, 1978).

Whenever biofeedback therapy is used—particularly in a psychiatric setting—it involves far more than the use of sophisticated electronic techniques to inform the patient about a particular set of physiological responses. Certainly psychotherapy *per se* seems too narrow a term to describe the process in which a therapist deals not only with cognitive material but with associated physiological manifestations as well. The integration of biofeedback technology adds a new dimension to treatment, and the term “psychophysiological therapy” more appropriately describes the actual process. Such an approach is highly congruent with recent trends re-emphasizing the medical model in psychiatry.

While the therapist is acutely tuned to sensing latent covert structures and dynamics in linguistic-cognitive communications from a patient, both he and the patient are generally less sensitive to associated covert physiological variations. Biofeedback has often been described as a “real-time physiological mirror” and may well serve to extend both therapist’s and patient’s sensitivity to subtle emotional-physiological components of the basic psychotherapeutic process. From this point of view, biofeedback may be seen as an adjunctive tool for the refinement of more traditional therapeutic approaches rather than as an independent entity. For this reason it is felt that biofeedback therapy—particularly involving psychiatric problems—should not be undertaken without a background of traditional psychodynamics, psychological defense mechanisms, and an understanding of presumptive psychopathology and physiopathology lest there be undesirable treatment consequences.

The Use of Psychophysiological Therapy with Psychiatric Patients

Except for occasional anecdotal reports, little has been published about the treatment of psychiatric inpatients with biofeedback. In a study of over 60 inpatients with a variety of psychiatric diagnoses

(neuroses, affective disorders, schizophrenia), who were referred for biofeedback therapy because of a secondary diagnosis of a psychophysiological somatic condition, the improvement of the somatic complaints did not exceed that expected by chance. Further, while many patients showed transient improvement of the primary psychiatric complaint in response to the demand characteristics of the clinic, to the biofeedback instrumentation and the extra staff attention, no significant improvement that could be ascribed to biofeedback therapy was noted in a single instance. Psychotic patients, particularly those with paranoid and delusional features, tended to show increased confusion and disorganization when exposed to biofeedback instrumentation (Glueck and Stroebel, 1975; Weber, 1977).

Although no other series of cases have been reported, Marcus and Levin (1977) reported that biofeedback has helped one patient to break through a characterological denial of paranoid hyperalertness accompanied by somatic activation. They speculated that obsessive patients might be able to accept ego dystonic observations from an objective, impartial machine. Rickles (1976) noted that the biofeedback machine may serve as an auxiliary inanimate therapist and may provide the patient with an opportunity to learn more about transitional phenomena and the vicissitudes of transitional or facilitating objects in adults.

A number of investigators (Rickles, et al, 1978; Stroebel and Glueck, 1978) have observed that many psychiatric patients are able to acquire voluntary self-regulation skills using biofeedback while immersed in the demand characteristics (Orne, 1962) of the treatment context. However, the transfer of such skills to a situation outside the clinic setting is limited and insufficient to have significant impact on the major psychiatric problem. Stroebel and Glueck (1979) and Schneider and Weiss (1978), in treating over 600 and 1000 patients respectively, have observed that depression, whether overt or masked, is a major factor in the unsuccessful outcome when psychosomatic conditions are treated with biofeedback as an adjunct to other, traditional therapies. Depressed patients tend not to assume responsibility for practicing self-regulation techniques. Therefore, failure to detect hyperarousal in the patient's EMG, thermal, or galvanic skin responses strongly suggests that the somatic complaints of the patient have a hysterical or masked depression basis and will therefore not respond (even in the short-term) to psychophysiological therapy with biofeedback as an adjunct. A treatment of the underlying depression or other somatic equivalent with more traditional therapeutic modalities is indicated.

The Use of Tranquilizers to Facilitate Biofeedback Therapy

A number of investigators (Fuller, 1977; Gaarder and Montgomery, 1977; Stroebel and Glueck, 1976; Wickram, 1976) have emphasized the relatively passive conditions necessary for the acquisition of voluntary self-regulation of physiology by means of biofeedback. Patients experiencing pain, whether functional or somatic, are generally incapable of achieving the passive state necessary for the acquisition of these new low arousal skills. One strategy to overcome this problem is to use minor tranquilizers to decrease the patient's mental anxiety or to give an analgesic for somatic pain during the course of initial acquisition of biofeedback skills. Such procedures have not proven to be effective in treating over 600 patients with psychosomatic disorders (Stroebel and Glueck, 1979). On the contrary, it was observed that patients simultaneously receiving minor tranquilizers required considerably more therapeutic time to acquire voluntary self-regulation skills. It was hypothesized that the medication chemically interrupted the very pathways used in acquiring such skills.

Potential Uses of Biofeedback Therapy in the Psychiatric Context

There is increasing concern among physicians and laymen alike about the widespread use and abuse of minor tranquilizers prescribed to chemically interrupt stress mediated psychiatric symptoms. While there are no hard data, there is a growing consensus that biofeedback techniques may facilitate the treatment of perceived stress induced states of physiological hyperarousal (Rickles, et al, 1978).

Though biofeedback therapy has little application with the major psychiatric disorders, it is likely to be useful with psychosomatic illnesses. Sifneos (1973), Nemiah (1978), and others report that a surprisingly large percentage of patients with psychosomatic symptoms may be characterized as being alexithymic. This condition has been described as an impoverished fantasy life with a resulting utilitarian way of thinking, a tendency to use action to avoid conflicting stressful situations, a marked constriction in experiencing emotions, and particularly a difficulty in finding appropriate words to describe their feelings (Sifneos, 1973).

Taking medication to relieve a symptom or pain is apparently within the belief structure of an alexithymic, psychosomatic patient whereas referral for insight therapy from a psychiatrist or psychologist

is not. The use of impressive and complex electronic technology, which is an aspect of biofeedback, seems congruent with the passive structure of such patients and may therefore provide an acceptable route to insight-oriented psychophysiological therapy. A significant new population of patients who would normally be resistant to psychiatric referral may now accept treatment in the context of biofeedback therapy. Such a procedure would be sharply in contrast to encouraging the use of medication which allows the patient to expose himself to ever greater stress without dealing with the root causes of the underlying psychic conflicts (Schwartz, 1977; Selye, 1974; Stroebel, 1981).

Two case reports (Gaarder, 1976; Korein, et al, 1976) have noted that persistent dystonias and tardive dyskinesias become less pronounced in states of low arousal. They report on patients who benefited from biofeedback therapy for tardive dyskinesia.

Biofeedback techniques have been used to help produce low arousal, generally in the context of other forms of relaxation training. Such uses include creating states of relaxation for patients in traditional psychotherapy or in behavior therapy, particularly in the context of systematic desensitization. Similarly, biofeedback techniques have been used to create low arousal states to facilitate uncritical free association as practiced in a variety of traditional psychotherapeutic contexts. However, no systematic studies documenting the importance of biofeedback techniques, over and above the contribution of relaxation instructions, are currently available.

Biofeedback procedures may prove to be an important new noninvasive research tool which will permit stabilization of a physiological state wherein associated psychological content may be examined (Stroebel and Glueck, 1978). While there is no convincing evidence that biofeedback is helpful in the treatment of disorders of volitional control such as obesity, smoking, or drug abuse, it is possible that instrumentation signaling activation could prove helpful to patients in confronting the denial which makes treatment of these conditions so difficult.

Possible Complications of the Adjunctive Use of Biofeedback Therapy

Many popular presentations of biofeedback imply that these techniques are benign, with few, if any, undesirable side effects. This view is alarming to experienced clinicians knowledgeable about psychopathology and psychodynamic mechanisms. Schultz and Luthe (1969)

have already documented an extensive set of criteria for contraindications and necessary precautions in the application of autogenic therapy which are likely to be equally relevant to biofeedback procedures. Psychiatric complications of biofeedback therapy in individuals who are presumed to be somatic "non-psychiatric" patients have been reported by Rickles (1976). He noted three variations of symptom substitution in patients with somatic problems being treated with biofeedback. The first is classical symptom substitution where a new psychophysiological symptom is substituted for another as the original one is resolved. The second is transformation of a somatic symptom into either a neurotic symptom, an affect such as depression, or into behavior such as some form of acting out. The third is transmission of the somatic symptom as it resolves into a significant person by provocative, projective identification, usually within the family unit.

More recently, the potentials and pitfalls of biofeedback as an inanimate therapist in the intense transference problems of patients with borderline features have been described by Rickles, et al, (1978). The importance of carefully assessing the patient's medical and psychiatric status prior to considering biofeedback therapy cannot be overemphasized. With widespread and enthusiastic media descriptions of biofeedback, ready access to biofeedback instrumentation by the public, and an increasing number of lay biofeedback therapists, the likelihood of patients receiving ineffective therapy for their conditions (e.g., biofeedback treatment of a depression related headache) is greatly enhanced. Complications may involve not only a delay in reaching appropriate help but can also be quite serious when borderline or paranoid patients are given biofeedback therapy.

Behavioral Medicine

The emergence of biofeedback technology and increasing investigation of its specific versus nonspecific aspects in facilitating the treatment of stress related disorders has been concurrent with the development of a new, broader field of interest identified as "behavioral medicine." This approach is likely to have far reaching implications for the treatment of psychosomatic conditions where patients have traditionally resisted referral to psychiatrists or psychologists. This new specialty area recognizes that there is a large domain of physiological functioning that is responsive to behavioral stimuli, is potentially adaptive, and is vulnerable to dysregulation as well as responsive to voluntary self-regulation (Schwartz, 1977; Stroebel and Glueck, 1978). By emphasizing self-responsibility and providing a belief struc-

ture acceptable to patients suffering from psychosomatic disorders, behavioral medicine may ultimately expand the scope of psychiatry in facilitating the treatment of a large number of conditions currently being treated less appropriately with traditional physical and psychiatric approaches.

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CHAPTER IV

**BIOFEEDBACK OF BRAIN
ELECTRICAL ACTIVITY**

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Attempts have been made to control quite a number of brain electrical phenomena. Although alpha biofeedback has often held the center of the stage, attempts at conditioning other forms of brain electrical potentials are of interest, both for the same reasons that alpha biofeedback is of interest and also for reasons inherent in the particular brain electrical phenomena under consideration. Table I offers a very short overview. The various phenomena are listed, and sections in which they are discussed are indicated.

At the most practical level, all attempts at operant control of brain electrical potentials raise the same issues: (1) Can one learn to control brain electrical activity? (2) Can this learned control of brain electrical activity be put to some practical use? Considering various forms of brain electrical activity allows one to take a less restricted approach to these questions and evaluate them from a variety of different points of view. In the sections to follow, we will see illustrated, repeatedly, how the superficial simplicity of these two questions is misleading.

Another set of issues has to do with questions of facilitating self-awareness, exploring the nature of consciousness, and in general creating a context that allows a more complete acknowledgment of responsibility for "mind." These issues are interesting and perhaps ultimately more important than the issues that are the focus of this paper. However, for the practicing psychiatrist, this medically oriented review seems appropriate.

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TABLE I. Some "Other-Than-Alpha" Brain Electrical Potentials That Can Be Controlled Voluntarily or Can Be Operantly Conditioned

<i>Section</i>	<i>Possible Physiological Significance</i>	<i>Possible Clinical Application</i>
A. ONGOING RHYTHMS		
1.	(a) 4-8 Hz (theta) Occipital (b) Frontal	Drowsiness Calmness
2.	12-18 Hz (beta)	Motor Inhibition
3.	40 Hz	Learning
B. EVENT-RELATED POTENTIALS		
1.	Lambda Waves	Saccades (rapid corrections of eye position)
2.	Cortical Sensory Evoked Potentials	Processing of Sensory Input
3.	Contingent Negative Variation (CNV)	Readiness to Respond
4.	Brainstem Evoked Potentials	Primary Sensory Input

A. Ongoing Rhythms

1. *Theta*

Theta activity refers to EEG activity in the sub-alpha range (4-7 Hz). Although theta tends to be associated with drowsiness and the first stages of sleep, there is evidence (reviewed by Schacter, 1977) that the issue of theta is much more complex. In particular, it has been suggested that high voltage, regular theta from the front of the head may be associated with calm, awake, meditative states, in contrast to the low voltage mixed theta from the back of the head that is the more conventional sign of drowsiness. The issue is complicated by the fact that light sleep and hypnagogic imagery may be both psychologically and phenomenologically quite indistinguishable from some meditative states in which persons think that they are calm and awake. Thus, theta offers three interesting problems. There are two standard questions: (1) Can theta be controlled?, and (2) Can one do something useful with that control? Then there is the somewhat more specific question: (3) Do frontal and occipital theta have different significances? Unfortunately, we have no good answers to the last question, and so far a relatively negative answer to the question of utility. However, two experiments will serve to illustrate the status of our three questions.

a. *Occipital Theta*

Beatty and his co-workers (1974) assumed that if theta represents drowsiness and if theta control can be taught, then control of drowsiness can be taught. The practical application of such a technique is obvious. For example, a major problem for people doing real-life vigilance tasks, such as standing radar watch, is the so-called "vigilance decrement" which develops over time. After a period, rare targets are missed or responded to slowly. These lapses appear to be associated with drowsiness and with theta activity.

O'Hanlon, Royal, and Beatty (1977) reported that occipital theta is relatively easy to condition. They measured theta as the ratio of waves in the 3-7 Hz band relative to total waves in the 3-30 Hz band. They used a quiet, 400 Hz tone which increased in intensity for one second if the theta during the preceding second equalled or differed from a criterion value in the desired direction. The criterion value was reset every thirty seconds to be the mean theta ratio for the preceding 30 seconds. Training sessions were one hour long on the first day and one-half hour long on the second day.

Although subjects could learn either to augment or to suppress theta, the prime interest was in learned theta suppression. Performance on a radar monitoring task was measured, first in the laboratory and then in an operational environment at the U.S. Naval Pacific Missile Base. In the latter case, subjects were volunteer, enlisted personnel and were expert radar operators. Results were statistically significant throughout. With prior training in theta suppression, theta feedback improved vigilance performance. However, the vigilance performance improvement was less as one moved from naive operators to experienced operators, and from experienced operators in the laboratory to experienced operators in the operational situation. Performance was negligibly improved in the best operators at their usual task. Thus, the general use of theta suppression feedback for radar operators is probably not indicated although perhaps it would be of value if inferior or poorly trained operators had to be pressed into service.

b. *Frontal Theta*

Frontal theta may be a reflection of an alert, tranquil state rather than of drowsiness, although this remains to be demonstrated unequivocally. In any case, frontal theta is much harder to condition than is occipital theta. An illustrative study was conducted by Birbaumer (1977) and his group. To obtain good frontal theta conditioning, a prior training in EMG frontalis relaxation and heartrate slowing was required. To this training was added theta feedback. A "pink" noise was fed to the right ear simultaneously with EEG activity in the 3.0-7.8 Hz band with a minimum amplitude of more than 10 microvolts.

In the test procedure, the subjects were shown aversive scenes from surgical operations and from electroshock therapy in a four-minute video display. A statistical control over theta in the presence of stress inducing film was demonstrated. The results were, apparently, rather weak and extremely difficult to achieve. In conclusion, Birbaumer states, "Whether the results could have any effect on subjective or behavioral responses remains to be investigated."

In the case of theta, then, the evidence seems to be that theta control can be learned. One can imagine situations where learned theta control could have utility, as for example, if poorly trained radar operators had to be pressed into service during a war but an immediate, practical utility for theta control training has not been demonstrated. There remains also the suggestion that frontal theta and occipital theta may be different phenomena. If so, this has interesting theoretical, as well as practical implications, but again, the case is not proven. For the clinician, there is thus far nothing here of demon-

strated clinical utility, and while continued research seems indicated, it is carefully controlled studies that are needed.

2. *Beta*

Clinically, this is the most interesting of the "other-than-alpha" brain electrical potentials. There is evidence that training in the production of beta rhythms can reduce epileptic seizure frequency in humans. A considerable controversy has arisen concerning this clinical application. I will try to provide a view of some of the controversy and then consider in some detail the most recent experimental evidence supporting this application.

Sensorimotor rhythm, or SMR, is a rhythmic 12-16 Hz activity arising from the sensorimotor cortex of the cat. Gastaut, et al, (1964) described an analogous rhythm in humans which they referred to as mu rhythm. Sterman and Wyrwicka (1967) studied SMR in cats who were trained to remain motionless. Subsequent studies by Sterman's group showed that cats could learn to voluntarily increase SMR and that cats over-trained in this procedure had elevated seizure thresholds. This, naturally, suggested that training humans in SMR might convey a similar increase in seizure threshold. Such a finding would have possible clinical significance in the treatment of epilepsy.

SMR is an interesting subject in itself. Kuhlman (1978) argues that mu and SMR are indeed analogous, but human mu has a center frequency of 10.1 Hz—right in the alpha range. Perhaps one should not then refer to 12-14 Hz premotor activity in man as SMR. However, with the above caveat in mind, we will use Sterman's convention and refer to 12-14 Hz as SMR whether in cat or man. SMR in cats appears to arise in the same area of the brain from which sleep spindles arise, and training in SMR results in the appearance of a more normal sleep spindle picture in epileptics who previously showed a reduction or absence in such activity. Hauri (1978) also reports that SMR training can benefit insomniacs. The work on cats and on sleep in humans is of intrinsic interest but serves here only to illustrate that learned control of SMR can, indeed, be demonstrated and that it has physiological consequences.

The crucial issue of clinical application in human epilepsy actually involves two related sub-issues. First, is biofeedback training capable of altering seizure frequency in epileptic humans? Second, does SMR have some special seizure-reducing effect? For example, Wyler and his group have studied the effect of operant conditioning on epileptic unit activity and of operant conditioning on epilepsy both in man and in animals. In considering their studies, Johnson (1977) argues, "The most obvious factor correlative with the diminution in ictal

activity concurrent with operant conditioning was not the production of any specific EEG frequency but simply the production of desynchronized single unit activity within the epileptogenic focus." Thus, there are some critics of Sterman who feel that even if the operant control of brain electrical activity can reduce seizure threshold, one can still argue against the specificity of the SMR.

There are also critics who have questioned the existence of a relationship between operant control of brain electrical activity and clinical response in epilepsy. For example, Kaplan (1975) studied 6-12 Hz training of epileptics. She failed to get EEG evidence of training and so could not attribute the clinical changes she observed to learned EEG control. However, it is worth noting that she used no shaping, no artifact control, and a simple on-off signal. She also did not negatively reinforce low frequencies. Nevertheless, her failure was applauded by Gastaut (1974) who then generously counseled . . . "not to condemn (SMR conditioning) out of hand . . . but to wait until its value, even its existence, has been demonstrated before proposing that it be used." It is worth noting that reference to Gastaut's early work on mu rhythm is absent from Sterman's more recent publications.

Thus, we have two questions: (1) Does SMR conditioning affect clinical epilepsy in man?, and if so, (2) Is this specific to the conditioning of SMR or due to some other less specific effect?

As Sterman has pointed out, there are formidable problems in evaluating clinical outcome studies of SMR training in epilepsy. With drugs, for example, one can define the therapeutic blood level. With biofeedback, idiosyncratic EEG parameters in epilepsy make it difficult to define a standard "blood level" of voluntary EEG control. In addition, quantitative evaluation of seizures is also difficult. With double-blind drug studies, large numbers of subjects can be used to wash out the noise in the measurement of seizure frequency and so achieve results that inspire some confidence. Biofeedback training is much more time consuming than drug administration studies and, hence, tends to involve smaller numbers for the statistics.

Then, given a good clinical study, the theory can still be questioned. Johnson (1977), for example, points out how one could attribute any positive result either to reduced arousal indicated by the observed state of immobility or to increased arousal indicated by the observed suppression of high voltage/low frequency EEG. Finally, in the field of biofeedback, the box-score approach (how many studies work; how many do not) is always misleading because there are too many variables to combine studies from different laboratories.

A good review of the other workers in this area can be found in the paper by Johnson (1977) and in the paper by Sterman and McDonald

(1978) which will be considered at some length, since this latter paper is an index of the state of the field.

The particular study in question was a double cross-over, single-blind, A-B-A design using eight selected epileptic subjects who had histories of poor seizure control with anticonvulsive medication for a period of three years and systematic logging of seizure incidents for at least three months. The biofeedback consisted of a system which aborted the reward whenever high voltage transients occurred and provided both a continuously varying indication on the positively rewarded frequency and a digital indication of reward for simultaneous suppression of the negative frequency and production of the positive frequency. Each patient was trained with two frequencies alternately, one being positively reinforced and the other negatively reinforced during one trial, and then with the contingencies reversed in another. In each case, one of the frequency bands was 6-9 Hz. For one-half of the subjects, the second frequency band was 10-15 Hz; for the other half of the subjects, the second frequency band was 18-23 Hz.

Reinforcement was provided for trains of the appropriate frequency band lasting more than .5 seconds and concomitant suppression of the other frequency band. A minimum of 10 laboratory training sessions was given each patient, and during this time the criteria for positive and negative frequencies were set individually for the patient. After this, the patients were sent home with technicians who established a procedure and schedule for continued home practice. During the home practice period, the patient returned to the laboratory at two-week intervals for recording and training, and the home training itself was recorded on a polygraph at alternate two-week intervals. At the end of a three-month period of home training, the reinforcement contingencies were reversed without the patient's knowledge.

There were other pertinent details in this rather careful and complex experimental design, but the above will serve our purposes here. The important issues are careful and prolonged laboratory training; continuous indication of the positive frequency; and reward contingent on producing the positive frequency, diminishing the negative frequency and avoiding high voltage artifacts. Seizure incidence records were compared statistically before, during, and after the experimental periods.

Six of the eight patients reported significant and sustained seizure reductions which averaged 74 percent following reinforcement of either the 12-15 Hz or the 18-23 Hz with suppression of the 6-9 Hz. The response to reinforcement for the 12-15 Hz was specific, with seizure rates returning to baseline when reinforcement contingencies

were reversed. Reduced seizure rates following reinforcement for 18-23 Hz were not altered when contingency was reversed.

The authors suggest that the 18-23 Hz reward may normalize the EEG in patients who have frequent, abnormal, slow EEG patterns. "The failure of these patients to effectively reverse this change when rewarding contingencies were reversed could reflect a homeostatic effect, making it more difficult to decrease high frequencies and/or increase abnormal frequency activity." By contrast, they suggest that 12-15 Hz is effective in those epileptic patients who have shown general disturbance in sleep patterns and conclude that different training strategies may be required for different pathological manifestations.

Kuhlman and Kaplan (in press) discuss the issue of EEG biofeedback in the treatment of epilepsy at length. They contrast attempts to teach epileptics an awareness of their abnormal EEG patterns so that seizures can be aborted (unsuccessful) with the teaching of faster rhythms (successful). This latter they refer to as "neural exercise," and they argue that the normalization of slow hypersynchronous neural activity (rather than SMR learning) is the therapeutic force in successful biofeedback treatment of epileptics.

In summary, then, it would seem that the question about voluntary control of beta activity can be answered affirmatively. Furthermore, it would appear that with careful selection of patients and with very prolonged and meticulous training, certain patients will experience a reduction in seizure frequency with reinforcement of fast activity. The specificity of SMR in this reduction in seizure activity remains in question, and the practical utility of this procedure would still appear to be severely limited by the time, the cost, and the degree of patient cooperation that is required.

3. *Forty Hertz*

Almost all of the work on 40 Hz has been carried out by Sheer and his colleagues. Much of this is reviewed by Sheer (1975) and two recent abstracts have been published by Newton (1976). Basically, 40 Hz is considered to be an extension of beta activity. Sheer argues that the closer one looks at low amplitude/fast frequency desynchronized records, the more one can notice a buried, rather synchronous 40 Hz activity. This rhythm is found in subjects doing arithmetic problems or engaged in other problem-solving activities.

Operant control of 40 Hz, however, poses very special problems. To begin with, muscles produce electrical activity (EMG) of very high amplitude, with the power centered quite close to 40 Hz. Second, brain 40 Hz activity from the scalp is rarely more than about 5 microvolts, and so it is easily lost in amplifier noise, cable noise, etc., even

if muscle activity is adequately controlled. These technical difficulties may account for the failure of other laboratories to follow up on Sheer's ideas, and the conditioning of 40 Hz remains to be widely replicated.

Since 40 Hz seems related to problem-solving, Sheer has considered the possibility that 40 Hz training could improve problem-solving ability and, indeed, has presented some evidence that might be construed as supporting this position. Because of the audacity of this suggestion and because of the formidable technical problems involved, expert replication will be absolutely essential.

4. *Asymmetry*

Although as early as 1971 Peper (1971) was working with biofeedback training for alpha asymmetry, very little progress has been made in this area.

The idea of learning EEG asymmetry has a certain appeal. It seems that use of left hemisphere propositional cognition produces a right-greater-than-left EEG asymmetry, while right hemisphere appositional cognition gives the opposite left-greater-than-right EEG amplitude. For an example of this research showing such task induced asymmetry, see Doyle, Ornstein, and Galin (1974), and for a critical evaluation, see Donchin, Kutas, and McCarthy (1977). Suppose one could learn to suppress the left hemisphere (turn on asymmetrical left alpha). Then one could "turn on" an alternate mode of thinking. Or, perhaps a dyslexic who fails to turn on the left hemisphere to read could be taught to appropriately engage the left hemisphere. Indeed, there is one report by O'Malley and Connors (1972) of a dyslexic with a suppressed evoked potential on the left who was trained to increase alpha on the left. This training resulted in an increase in the amplitude of his visual AEP on the left. The effect of this on his reading could not, of course, be separated out from other things such as the enormous amount of attention he received over the training period.

Some of the most recent work in this area has been done by Dumas and Merts (in prep.). Feedback was in the form of two tones. One tone indicated that right alpha was above baseline and left alpha was below baseline, while the other indicated the reverse, R. alpha below and L. alpha above. Bilateral suppression or enhancement, even if asymmetrical, produced no feedback signal.

After three days of training, only five of ten subjects gave evidence of learning, a percentage roughly equivalent to that reported by others using different feedback techniques. A questionnaire was used to explore the strategies subjects employed in obtaining the desired asymmetry. The expected reports of visual-holistic thoughts during right alpha suppression and verbal-sequential thoughts during left suppres-

sion were not obtained. However, open inquiry suggested that a sharp body image was associated with left activation and a diffuse body image with right activation.

We can be reasonably certain that different asymmetries can be produced by different tasks and that voluntary control of asymmetry can be learned by talented subjects. For practical utility, the prospects are not bright but, as a tool for the phenomenological investigation of consciousness, the technique seems worth pursuing.

B. Event-Related Potentials

The term "event-related potential" generally refers to a scalp-recorded event that is more or less time-locked to some observable event such as a sensory stimulus or a motor act. Since, for the clinician, these are of academic interest only, we might as well include intracranial recorded activity in the same section and, thus, having descended to the level of animal experimentation, we might as well go the whole way and include single- and multiple-unit activity along with gross electrode slow wave activity. There is further justification for combining such a heterogeneous collection of phenomena. Not only do operant conditioning studies of such potentials have no apparent clinical applications, but they also address similar theoretical and philosophical issues.

Conditioning of unit activity also requires some mention, if only for reasons of priority. Even before Kamiya began his pioneering work, Olds and Olds (1961) were conditioning single units. They observed that some units conditioned more easily than others and that conditioning a unit in one area would result in controlling units in quite remote locations. Following the Oldses, others demonstrated that single unit activity, multiple unit activity and slow wave evoked potential activity from a variety of areas could, indeed, be brought under voluntary or operant control.

Fortunately, a volume edited by Chase (1974) brings together reviews of much of this work. To simplify, the studies to date have shown that the more some electrical pattern is part of a voluntary repertory, the easier it is to bring under voluntary control. If one wishes to control some electrical event and disassociate it from other events, whether they be sensory input or motor output, then the more variable and unstable the event is, the more easily it can be controlled. Hard-wired and invariable events are the most difficult to control. In a classical study, Fetz and Finocchio (1971) were able to condition either cortical units or electromyographic response. They found that

there were certain, almost invariant, pairings such that conditioning of either the unit or the EMG resulted in activity in the other member of the pair. They then attempted to disassociate such paired unit activity and EMG responses. They discovered that animals could learn to emit bursts of cortical cell activity and totally suppress the related EMG activity. Although less effort was spent in the other direction, it appeared more difficult to reduce cell activity during conditioned EMG activity, and they failed to suppress cell activity entirely when the paired EMG activity was being elicited.

1. *Lambda Waves*

Some of the work done in animals has utilized brain stimulation as a reward. This allows one to deal with a completely paralyzed preparation. In such cases, somatic motor activity can be ruled out as a mediator of the operant control phenomenon, although autonomic responses are still left uncontrolled. In a review such as this, it would be inappropriate to attempt doing justice to the physiological and neuroanatomical details of all these studies. Instead, I have selected one animal experiment for illustrative purposes.

A psychiatrist reading this literature cannot help being struck by the apparent disbelief of physiologists when confronted by evidence that the brain can, on the basis of its past history and without benefit of apparent sensory input or motor output, undertake to initiate something on its own. On the other hand, demonstration of self-evident truths is not a waste of time, for sometimes nature hides rather nasty surprises. Happily, this has not been the case in studies of cerebral conditioning.

The only work on lambda waves appears to be that done by School and Rowland (1976) using cats as subjects. Lambda waves are brief, high amplitude, biphasic waves arising in the visual cortex about 30 msec after the start of a saccade. A saccade is that rapid movement of the eye used in correcting eye position. When the eye is tracking a moving object and loses its target, it makes an abrupt movement to get on target again. In looking about a scene, as the eye moves from one object to another, the same, very rapid sort of movement occurs. There is evidence that this rapid movement is associated with a kind of blanking of visual perception. For example, place a large mirror in front of you, put two dots on it, and then look from one dot to another. You will make saccades, and you will not be able to see your eye moving. Now, if you move the mirror while you look at one dot, you easily see the movement that the eye makes in this tracking operation. Thus, tracking eye movements do not produce blanking, nor do tracking movements produce lambda waves.

The lambda wave seems to be the neurophysiological correlate of the saccade. Its exact function is unknown, but since it follows the saccade, it is not unreasonable to believe that it may have something to do with this phenomenon of blanking out of visual perception while the relationship of the visual field to personal space is being reorganized.

Now, when a cat is totally paralyzed with Flaxadil, lambda waves can still be observed. Since there is no eye movement in such animals, the only explanation is that the brain is generating the command for a saccade even though it has no saccade to show for it.

For the experiment in question, the cats were implanted with electrodes in a brain reward center, and each cat had indicated by bar-pressing that stimulation in that brain area was, in fact, rewarding. Using operant procedures, the cats readily learned to produce or to inhibit lambda waves in order to be rewarded. When cats first learned to make or inhibit saccades, they could produce lambda waves under paralysis. They were also capable of learning initially under paralysis.

In summary, a cat can be trained to produce lambda waves in order to obtain rewarding brain stimulation, and the same cat can also suppress lambda waves if that is required for the rewarding brain stimulation. In other words, given feedback based on a brain state ordinarily associated with eye movement, the cat could learn to produce or avoid this brain state even though eye movement was prevented by drug induced paralysis.

The inference from this is that cats can certainly learn to control their brain electrical activity independent of any motor behavior or sensory feedback normally associated with that particular brain state. Thus, it is not necessary to have a motor act or sensory feedback from a motor act in order to learn to produce or inhibit at least one brain state. For many of us, it seems an inescapable fact that one can learn to intend something and that a change in intention is associated with some change in brain function. The fact that one can discover a particular intention related to a particular electrical phenomenon is then less than surprising.

2. Auditory Evoked Responses in Humans

Although one can voluntarily alter evoked responses by attention, either with or without changing receptor orientation, there is only a single report in English of operantly conditioned auditory cortical evoked responses in human beings. This is one reported by Rosenfeld, et al, (1969). In this study, they presented tone pips every four seconds over a headset. For conditioning they selected a

negative-going peak at 200 msec. They measured the averaged electrical activity for 200 msec prior to the stimulus and took this as a baseline. Then they centered a 20 msec window at 200 msec and rewarded the subject each time his electrical activity following a tone pip was increased one standard deviation above that baseline level. Money was used as a reward, and enhancement of this negative evoked potential peak apparently was learned.

There was no attempt to teach the subjects to reduce their negative EP activity; they were only taught to increase it. Furthermore, no consistent response to the question, "What did you do to get rewarded?" was found. The effects were small—30 percent success when chance was 16 percent—but nevertheless the phenomenon appears to be demonstrated.

Roger and Galand (1977) reported on conditioning of visual and somatosensory evoked potentials, as well as auditory evoked potentials. Their interesting and provocative studies are so far available only in French.

3. *Contingent Negative Variation (CNV)*

The CNV is a slow, negative wave which develops following a warning stimulus when the subject expects an imperative stimulus (i.e., a stimulus that requires some sort of response). In a sense, this can be looked upon as a classically conditioned phenomenon rather than an operant phenomenon and indeed it was originally viewed this way by Grey Walter, et al, (1964). This tradition of classical conditioning slow wave activity is still being pursued in Lelord's laboratory (Lelord, et al, 1976).

With respect to the classical CNV, the more certain the subject is of the arrival of the imperative stimulus and the more effort required for the response, the larger the negative wave. More recent studies have fractionated this negative wave into an early frontal component, which seems to be related to alerting, and a later central component, which seems to be associated with readiness.

One can, however, leave the classical procedure with a paired conditioned and unconditioned stimulus and adopt a more operant viewpoint. Thus one can ask whether the subject could internally initiate events that produce the contingent negative variation. The answer is affirmative. In some experiments carried out by McAdams, et al, (1966), experienced laboratory workers attempted simply to "think CNV." This effort was successful in producing the slow negative potential and qualified the CNV for a place in the list of brain electrical potentials brought under voluntary control.

4. *Brainstem Evoked Response*

The brainstem or far-field evoked response consists of a series of small ($<.5 \mu v$) wavelets in the first 10 msec for a stimuli. Their resistance to such things as severe cortical damage and anesthesia make them an unlikely candidate for biofeedback control. Nevertheless, Finley, et al, (1978) present very convincing evidence of conditioning for Waves IV-V (lemniscal-collicular responses with a 5-6 msec latency). Successful conditioning was associated with lowered auditory threshold. The questions raised by this brief report are extensive, and to deal with only a few could be misleading. In any case, such counter-intuitive results deserve careful consideration.

Conclusion

It can be said with some confidence that a wide variety of brain electrical events can be brought under operant control. Although there are many details to be filled in, generally it seems that some more or less "hard-wired" electrical events are associated almost invariably with other events such as sensory input or motor output. Nevertheless, it is the plasticity of the brain that is most impressive. Philosophically, if we accept Bateson's (1972) view of the mind as involving extended closed communication loops, and if we postulate an optimum ecology for the mind, then the limits of the mind's ability to control the brain seem still untested.

The philosophical relevance of other-than-alpha conditioning is not matched by clinical relevance. SMR conditioning is the sole possible exception. In the other case where conditioning of humans is unequivocal (i.e., posterior theta), the applications are unimpressive. In the other cases, to paraphrase Gastaut, one needs to be convinced that conditioning actually exists before clinical relevance can be evaluated.

It does appear that training epileptics to produce SMR (or perhaps to simply suppress slow and increase fast activity) has clinical promise. Skilled professionals, highly motivated subjects, and a well-equipped laboratory are essential for adequate evaluation of this promise, and although SMR feedback is expensive and time consuming, support for continuing clinical research on this subject is thoroughly justified.

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CHAPTER V

ON THE NATURE OF ALPHA FEEDBACK TRAINING*

Martin T. Orne and Stuart K. Wilson

I. INTRODUCTION

A new kind of interaction between man and his body, *biofeedback*, elicited enthusiastic interest in many sectors of the scientific community in the late 1960s. A number of investigators had shown that automatic electronic sensing and feedback of a wide variety of usually unconscious physiological functions allowed individuals to directly influence internal processes that had previously been considered beyond volitional control. These included galvanic skin response (Crider, Shapiro, and Tursky, 1966), heart rate (Engel and Chism, 1967; Engel and Hansen, 1966), blood pressure (Shapiro, Tursky, Gershon, and Stern, 1969), evoked cortical potentials (Fox and Rudell, 1968; Rosenfeld, Rudell, and Fox, 1969), and EEG (Hart, 1968; Kamiya, 1969; Mulholland, 1968). Perhaps most impressive was the elegant demonstration by Miller and DiCara (1967) that curarized animals could acquire instrumental control over visceral and glandular responses.

EEG brain alpha wave feedback had particularly struck the imagination of researchers and public alike. Alpha waves—the large sinusoidal 8–13 cycle per second EEG activity—had been linked by earlier studies (Lindsley, 1952; Stennett, 1957) to intermediate levels of arousal. The alpha rhythm was felt to be most prominent when the individual was neither drowsy nor hyperalert. Within this theoretical context, Kamiya (1969) demonstrated that individuals could control

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alpha density through feedback and consequently maintain higher alpha levels. Further, this enhanced alpha density was associated with pleasant, relaxed feelings (Brown, 1970, 1971; Hart, 1968; Kamiya, 1969). These results thus suggested that alpha feedback was a method by which modern man might achieve direct control over the level of his neurophysiological arousal and, therefore, over his anxiety and dysphoria. The potential, not only for the troubled individual but for everyone, appeared unlimited and held out the promise of our advancing beyond the age of drugs into an age of direct, conscious control of many psychobiological processes.

In the discussion to follow, we seek to evaluate the disparate scientific observations that made this dream plausible. We also focus on the line of research carried out at the Unit for Experimental Psychiatry specifically intended to clarify those aspects of alpha feedback training, and of the alpha mechanism itself, that are crucial to the potential therapeutic application of alpha feedback training. Finally, we try to spell out to what extent these hopes now seem justified and the possible directions of future research.

II. STUDIES SUGGESTING ALPHA FEEDBACK TRAINING MAY INFLUENCE SUBJECTIVE EXPERIENCE

Berger (1929) demonstrated in his initial studies that the predominant EEG rhythm in relaxed individuals sitting with their eyes closed in a darkened room is alpha. He found that when the individual becomes drowsy, alpha activity rapidly disappears, while a stimulus that causes the individual to be startled, surprised, anxious, or frightened blocks the presence of alpha, at least temporarily. Later, Jasper (1936) suggested, and Lindsley (1952) and Stennett (1957) tried to document, that the relationship between alpha density and activation or arousal (both physiological and subjective) may be described by an inverted U-shaped function. They felt that during high arousal, as in anxiety-tension, alpha density seemed reduced and that it approached minimal levels with extreme excitement or panic. Alpha density was at maximal levels during alert, but relaxed, nonfocused mind-wandering. It disappeared from the EEG record with the onset of sleep. Thus, maximal alpha density appeared to reflect an intermediate level of arousal, that level at which an individual is neither drowsy nor hyperalert but rather comfortably relaxed. If alpha feedback training could teach an anxious individual to produce high alpha density he might concomitantly reduce his level of arousal to relaxed alertness, with its associated subjective state of pleasant relaxation.

The issue to be resolved seemed to be whether it was possible to learn to control such neurophysiological functions directly.

The initial enthusiasm for alpha feedback training appeared particularly warranted because brain functioning, in contrast to heart rate or blood pressure, logically seems to be more closely connected with subjective experience. Further, while not dealing directly with alpha feedback, the studies of feedback control over other visceral states, such as blood pressure or galvanic skin response, provided substantial scientific support for the view that feedback might be used to gain control over otherwise automatic physiological processes. Some investigators, from purely teleological deduction, felt even then that nature could never afford to leave life-supporting homeostatic systems to the capriciousness of conscious intent. However, the original study of heart rate feedback with curarized rats had dramatically shown that an animal could be induced to slow its heart, even to the point of death (Miller and DiCara, 1967).

A. Subjective Identification of Alpha Production

In light of the hypothesized relationship between alpha and arousal, Kamiya's (1969) anecdotal report of early work showing that subjects could learn to recognize the presence of alpha in their EEG was of great conceptual importance in providing a logical link to suggest that direct biofeedback of alpha wave production might produce desired subjective experiences. While observing the clinical EEG of a number of subjects, Kamiya instructed them to indicate whether they were producing brain wave state A (alpha) or brain wave state B (non-alpha) each time a bell rang. He provided feedback by telling them whether their statements were correct. Over a period of several hours some subjects apparently learned how to correctly identify alpha 100% of the time. Further, in Kamiya's later experiments on training the subject to enhance or suppress alpha, spontaneous alpha density during rests between training trials was higher than before, apparently because these subjects preferred the high alpha state.

Kamiya (1969) felt that it was not possible to conclude from the available data that the presence or absence of alpha was associated with perceptible alterations in subjective experience, but he reported that the subjects appeared to have gained some control over their brain wave states. It was not clear to Kamiya how and to what extent alpha production itself was represented in conscious experience; nor was it clear whether in order to identify it, the person was associating

certain levels of arousal or other behaviors with the concomitant changes in alpha production. He proposed that the data did suggest that it was possible to learn both to control alpha and to produce specific subjective states by attending to simple biofeedback signals based on EEG activity.

Given the above observations, it seemed reasonable to interpret Kamiya's (1969) finding as indicating that feeding back the presence or absence of alpha would allow an individual to learn to produce maximal levels of alpha density. This, in turn, would produce a level of arousal between drowsiness and hyperalertness—a state of mind (and body) that, furthermore, might produce the salutary effects reported by meditators, who also seemed to have high-amplitude and high-density alpha in their EEG (Anand, Chhina, and Singh, 1961; Wenger and Bagchi, 1961).

B. Meditation and Alpha Waves

Another important theoretical support for the use of alpha feedback training emerged from an increasingly widespread interest in Eastern religions in general and meditation in particular. Previous studies of the physiological status of Far Eastern meditators during normal waking and meditation produced apparently striking confirmation of the notion that alpha waves were directly related to relaxed states of mind. Anand *et al.* (1961) and Wenger and Bagchi (1961) studied the EEG of yogis and reported that their brain waves showed a predominance of very-high-amplitude alpha waves. Further, the kind of stimuli that normally caused subjects to block alpha failed to block alpha production in meditating yogis, whose discipline trains them to turn inward and ignore the outside world. Kasamatsu and Hirai (1966) studied Zen masters, who, in their meditation, are trained to remain open and seek to experience even mundane stimuli as continually new and fresh. They also noted very-high-amplitude alpha in these subjects. In contrast to yogis, however, these individuals not only showed the usual alpha blocking response to novel stimuli but continued to block alpha indefinitely, even to the same trivial stimulus. In other words, the meditating Zen masters failed to habituate.

These studies of Zen masters and yogis, considered together, were of special interest, not only because they suggested that meditators in general tended to have large amounts of high-amplitude alpha, but also because their EEG demonstrated alpha characteristics commensurate with their mental discipline. The meditating yogis failed to show alpha blocking in response to a stimulus, while the Zen masters

failed to show habituation. Thus, particular states of mind seemed reliably associated with easily measured neurophysiological processes.

III. THE BASIC ALPHA FEEDBACK EXPERIMENTS

Taken together, the several lines of preliminary inquiry described above were felt to be potential evidence for the idea that alpha feedback might be developed into a major tool for the self-control of subjective experience. All that seemed necessary was the proper electronic equipment, adequate training methods, and properly motivated individuals. A number of studies that supported this general hypothesis soon appeared.

A. *Some Encouraging Alpha Feedback Results*

Kamiya (1969), following up his early experiments on the identification of alpha, used electronic circuitry to identify the presence or absence of alpha waves in the EEG. He arranged the equipment so that either a light or a tone would go on whenever alpha waves were present. The subject sat in a dimly lit room and attended to either a visual or an auditory feedback signal. He then was trained to produce or block alpha by instructions to keep the signal on or off, respectively. Kamiya, as well as Hart (1968) and Mulholland (1969) independently, showed that in such conditions subjects could exert volitional control over the presence or absence of alpha. Kamiya pointed out that this control was manifested most dramatically in the ability to reduce alpha but added that subjects seemed to prefer the alpha state. Further, they tended to describe the state in characteristic terms, such as *relaxed*, *calm*, and *pleasant*. Brown (1970) found similar reports of relaxation, total concentration on the feedback light with a loss of awareness of the surroundings, etc. Interestingly, although Mulholland's subjects also were able to increase alpha, they did not report many of the striking subjective changes found by Kamiya and others.

The subjective experiences apparently associated with alpha wave production were explored more carefully by several investigators but substantiated perhaps most intricately by Brown (1971). Using appropriate electronic circuitry, she illuminated different colored lights, depending on the type of EEG wave in the subject's record. For example, blue or red lights were used for alpha, red or green for beta, and green or blue for theta. The subjects were encouraged to play with the lights for an hour and try to associate specific feelings with each of them. Forty-five subjects received this kind of feedback.

For each subject, one of the two possible light colors was associated with one of the three EEG frequency bands identified above. The subjects were then asked to sort more than 100 mood-descriptor terms into the appropriate red, blue, green, or white bin, symbolizing the three colored lights and no particular color association, respectively. Brown (1971) compared their sorting with the sorting performed by 45 control subjects who had not undergone the three-light feedback and had not associated any colors with the experimental situation. She was able to show that the experience of linking an EEG state with a colored light significantly changed the mood terms sorted with that color. Descriptors significantly associated more frequently with alpha colors were *calm, peaceful, pleasant, at ease, neutral, illusion, dreamlike, mysterious, and uncertainty*. Beta wave production (low voltage or small waves of greater than 13 Hz) was associated with feelings of being angry, aggravated, irritated, impatient, unhappy, troubled, frustrated, touchy, shaky, and investigative, as well as with feeling a void inside.

Thus, a much more specific assessment of the associated subjective experiences again seemed to confirm Kamiya's (1969) original reports. It appeared, then, eminently reasonable to try to utilize alpha feedback training as a means of helping the individual learn to gain control over the extremes of arousal. The only further requirements seemed to be an appropriate learning context for the subject and the necessary learning schedules.

IV. ESSENTIAL REFINEMENTS OF ALPHA FEEDBACK METHODS

If the therapeutic applications of the above findings were to be justified, several issues of both practical and theoretical importance required attention. Perhaps the most readily apparent problem was the wide individual differences in alpha density found among subjects—an observation Berger (1930) made early in his research. Some subjects in a darkened room show almost continuous alpha, which may persist even in the presence of light, while others show none. In a dimly lit room, under novel circumstances, Kamiya (1969) observed that most subjects had relatively low levels of alpha, which gradually increased over the session. Individual differences in baseline alpha density and the rising levels of alpha density that occurred during sessions presented serious methodological problems for efforts to document the effectiveness of feedback enhancement of alpha density.

A. Control of Subject, Methodological, and Situational Factors

The solution to the problem of individual differences originally attempted by Kamiya (1969) was to equate individuals with widely differing levels of baseline alpha production by setting the electronic filter gains arbitrarily for each subject so that the alpha-on signal would be presented 50% of the time regardless of the actual amount of alpha shown on the EEG record. Working in an operant conditioning context, Kamiya could equate, between subjects, the amount of positive reinforcement—the subject's feeling of success—in the task. Unfortunately, this procedure tended to focus attention away from the individual's actual changes in alpha density and artificially created a situation in which changes in apparent alpha density were emphasized. Only in later work (Nowlis and Kamiya, 1970) was any attention paid to the interaction between the initial level of alpha density and the effects of training procedures.

Other means of equating extreme differences between subjects were also employed in the later Kamiya studies. For example, Nowlis and Kamiya (1970) provided feedback to subjects with their eyes closed but asked some subjects to keep their eyes open if their initial alpha density was high. The latter condition would depress the high resting alpha levels and thus bring the starting alpha density of these subjects to a level more similar to that of individuals with moderate alpha density.

In these early alpha feedback studies the assumption was made that alpha density somehow reflected a basic psychobiological process, and little attention was paid to whether the individual's eyes were open or closed or to whether the circumstances were novel or the subject was well habituated; nor was there much concern with whether the feedback modality was auditory or visual. The possible interactions among initial baseline alpha levels, the circumstances of recording, and subsequent changes in alpha density were not considered. However, these issues must be taken into account, and extensive baseline measures of alpha density levels must be obtained before the results of feedback training can be compared between laboratories.

B. Replication of Alpha Feedback Results with Refined Methodology

Our first study sought to replicate the findings reported by Kamiya (1969) and others mentioned above (Brown, 1971; Nowlis and

Kamiya, 1970) but hoped, by attention to methodological detail, to gain a clearer understanding of the process. To facilitate analysis, eyes-closed and eyes-open baselines were obtained at both the beginning and the end of the experiment. The learning trials consisted of 2-min periods interspersed with 1-min rest periods. In order to demarcate clearly the beginning of rest, the feedback signal was arranged to provide a green light for the presence of alpha and a red light for the absence of alpha. The light was turned off to signal the onset of the rest periods. Instead of arbitrarily setting the electronic equipment to register 50% alpha, we set the equipment to reflect the presence of alpha as defined by standard definitions for the hand scoring of EEG wave forms. To accomplish this goal, a special filter with extremely sharp cutoffs, providing almost immediate discrimination of alpha, was developed (Paskewitz, 1971).

In addition to recording EEG from monopolar frontal (F4) and occipital (O2) electrode placements referenced to the ipsilateral mastoid, the procedure, followed in virtually all the early studies, also involved the recording of eye movements, heart rate, and the electrodermal response. Continuous paper recordings were made on a Beckman dynograph, and the data were also recorded on magnetic tape. The feedback system used occipital EEG signals, with the specially developed hybrid filters having step-function cutoffs at 8 and 12 Hz. There was a further amplitude criterion of 15 or 20 μV , depending on the particular experiment. At the completion of each session, a postexperimental interview was carried out during which the subject was asked about both the strategies employed to increase alpha density and the nature of his experiences during the experiment.

The first study included an initial session devoted to classical conditioning, followed by two feedback sessions on successive days (Lynch, Paskewitz, and Orne, 1974; Paskewitz, Lynch, Orne, and Costello, 1970). The results demonstrated that individuals did indeed learn to increase alpha density across trials, as had been reported by others. Figure 1 shows the effect of alpha feedback on seconds per minute of alpha produced by 16 males. Using visual feedback, subjects quadrupled the amount of alpha emitted during their first feedback session. However, we noted that this apparently dramatic increase took place from a very low initial level of alpha density. Thus, they went from an average of 2 sec/min of alpha density to an average of 8 sec/min of alpha density during the ten 2-min trials interspersed with 1-min rest periods.

Previous experimenters (Kamiya, 1969; Mulholland, 1969) had shown that subjects could volitionally block alpha as well as increase it when given appropriate instructions. We were also able to confirm

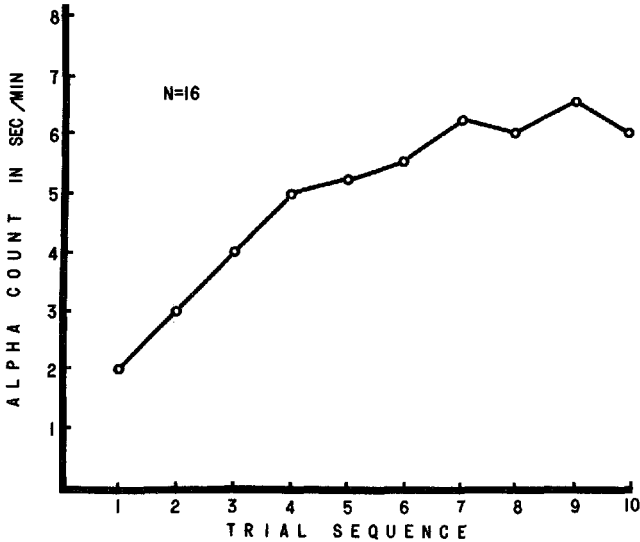


FIGURE 1. Seconds per minute of criterion EEG alpha produced during the first day of binary alpha wave feedback by visual display. Ten 2-min feedback trials are presented.

this finding in the same study. Thus, on the second day of feedback training, subjects had five feedback trials with instructions to augment alpha, followed by several trials during which they were alternately told to increase and decrease alpha density. Figure 2 certainly seems to document the claim that subjects can be taught to reduce, as well as to increase, alpha; however, careful examination indicates that something other than learning could explain this observation. On the very first trial during which subjects were told to "keep the red light on," alpha density dropped to a level nonsignificantly below the initial trial on Day 1, when feedback training with the visual display was started. Since subjects were producing almost no alpha under these circumstances, performance during subsequent "alpha-off" trials could not manifest any significant increase in alpha blocking from that seen during the first trial. It would, therefore, appear inappropriate to speak of subjects' learning to block alpha, since this is a skill that they seem to possess from the very beginning.

C. The Effects of Alpha Feedback on Subjective Experience

Care was taken in this study to solicit subjects for an experiment in conditioning rather than running self-selected individuals who wanted to be trained to increase alpha density. Only rarely did we encounter any subjective reports reminiscent of those described by

Kamiya (1969). In those occasional instances when subjects did report a kind of calmness or relaxation, it was invariably associated with the feedback trials, when the actual alpha density was, of course, far lower than during the rest periods in total darkness. While we were not prepared to dismiss the possibility that alpha feedback training might lead to systematic subjective effects, such effects were clearly not a simple function of alpha density. If this were the case, subjects would have reported being in an "alpha state" during the baseline periods of rest, when the actual alpha density was significantly higher than during feedback trials in the presence of light. We never encountered a subject giving such reports, and we therefore concluded, even at this early stage, that the subjective changes could not be simply a matter of the level of alpha density. However, it was felt that they might conceivably involve an increase in alpha density under circumstances that normally depress it.

D. Alpha Density during Light Feedback versus Resting in Darkness

The nature of the results of feedback training during the first study may be understood more clearly when placed in the context of

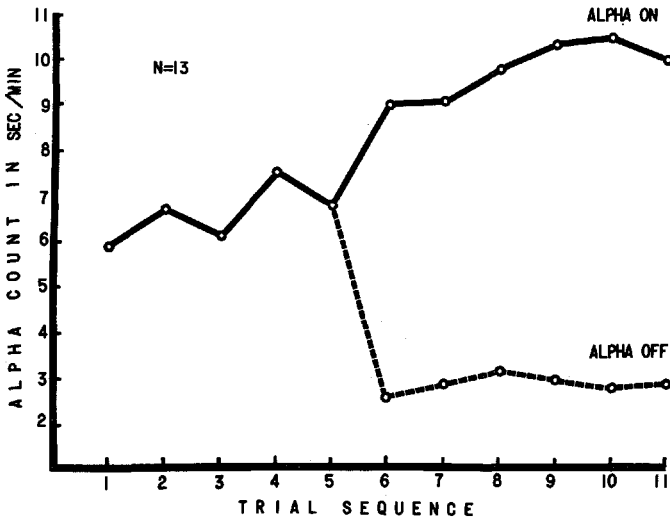


FIGURE 2. Seconds per minute of criterion EEG alpha produced during the first day of binary feedback by light display. Five 2-min enhancement feedback trials were followed by 12 discrimination trials with alternating instructions to enhance and inhibit alpha production.

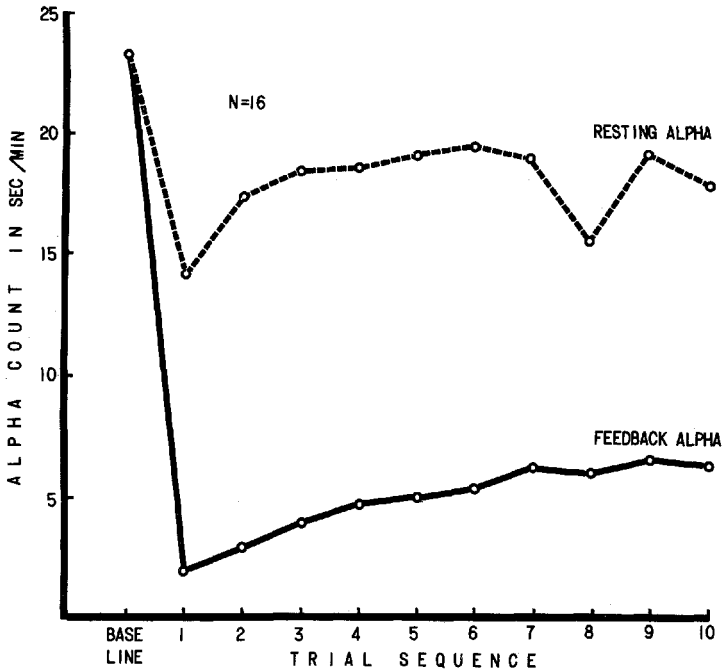


FIGURE 3. Seconds per minute of criterion EEG alpha produced during binary feedback with light display compared with baseline and rest period levels. The alpha feedback data are the same as those presented in Figure 1. Baselines and 1-min rest period alpha levels interspersed between 2-min feedback trials were obtained while the subject was in total darkness.

the alpha density during the initial eyes-open and eyes-closed baselines in total darkness as well as the alpha density during the rest periods. In these intervals, the feedback light was turned off and the feedback room again became totally dark. It is evident in Figure 3 that subjects in total darkness began with a spontaneously high baseline level of alpha density, which was promptly depressed by the visual feedback stimulus. However, during the rest period, when the room again was in total darkness, the alpha density returned to the much higher baseline levels.

V. THE SIGNIFICANCE OF THE VISUOMOTOR SYSTEM FOR ALPHA FEEDBACK TRAINING

As Berger (1929) had already recognized, the presence of light is typically associated with a precipitous drop in alpha density. It

seemed that the increase in alpha density associated with visual feedback, a circumstance that normally suppresses alpha, involved learning to avoid attending directly to the visual stimuli. Therefore, since the alpha density with visual feedback was of a far lower order of magnitude than that produced spontaneously in total darkness, it seemed more appropriate to speak of individuals' learning to disinhibit—in the Pavlovian sense—the alpha blocking effects associated with the presence of light, rather than to consider these data as a demonstration of learning to increase alpha.¹ Mulholland (1969) had independently shown that the process of habituation to the feedback stimulus is reflected by a gradual increase in the length of alpha bursts associated with it. Thus, the increase is also a product of adaptation to the feedback signal rather than of learning alone. This phenomenon explains in part why one typically sees a gradual increase in alpha density during feedback, regardless of the subject's success in producing alpha density greater than baseline levels.

In view of the dramatic effects associated with the visual feedback system, it seemed evident that if one hoped to find a true enhancement of alpha density, it would be necessary to carry out feedback training in the absence of light. Thus, we sought to determine whether individuals starting feedback training with alpha density already at a high baseline level could learn to increase alpha density to significantly higher levels. Accordingly, feedback signals were changed to tones, and all light was eliminated from the experimental room. The presence of alpha was signaled by a 75-dB tone presented at 360 Hz, and its absence was signaled by a 75-dB tone presented at 280

¹ We are seeking to make a distinction—which is a topic not commonly addressed in the learning literature—between the learning of a skill as opposed to the exercising of that skill under circumstances which normally inhibit it. Consider, for example, a student who is capable in mathematics but suffers from a test phobia which inhibits his test performance. If one were to operationalize learning to do mathematics simply by how well a student does on a test, one would confound the individual's true mathematical skill under optimal circumstances with the inhibition of that skill induced by the circumstance of taking a test. The most effective way to increase such an individual's performance would be through various procedures that would help disinhibit the anxiety effects associated with taking a test; in contrast, the student who cannot do mathematics will benefit most from encouragement, a good tutor, and lots of homework. Though in both instances one might observe improved test performance, it would be brought about by conceptually distinct processes: disinhibition in one case and learning in the other. There is little evidence to show that alpha feedback training leads to learning analogous to that of learning mathematics—despite feedback training, subjects rarely exceed their optimal alpha baseline level. Conversely, much apparent learning to increase alpha density seems to involve a process analogous to that of the student with the test phobia learning to effectively exercise a known skill during a test by disinhibiting his anxiety response to the situation.

Hz. The frequency difference was easily discriminated by the subject, and the tones were not experienced as noxious. In pilot studies, we determined that it made no intrinsic difference which tone was used to signal alpha and which was used to signal nonalpha.

A. Alpha Feedback in Total Darkness versus Dim Ambient Light

A study was conducted with nine subjects run in total darkness for six sessions, each separated by approximately one week (Paskewitz and Orne, 1973). Monopolar EEG recordings of the right occipital and the right frontal brain areas, each referenced to the right mastoid, were made. After an initial 3-min eyes-closed and a 3-min eyes-open baseline, an orientation period of 5 min was provided during which feedback was available, and the subject was encouraged to experiment with the tones to learn how his thoughts and behavior could affect them. The subject was then instructed to try to keep the high-pitched tone on and was given ten 2-min feedback trials interspersed with 1-min no-feedback rest periods. All feedback training was carried out in total darkness.²

Although during the first session subjects' initial high alpha activity was reduced markedly when they first opened their eyes in total darkness, they recovered much of this drop by the middle of the initial 5-min orientation period (Figure 4). These increases occurred within 2 or 3 min without instructions to augment alpha density. Whether they represent true learning or adaptation is unclear, but the rapidity of the increase was different from what was usually described as occurring with feedback training. Further, during the later sessions, this initial drop in alpha density during eyes-open baseline became

² At Dr. Kamiya's suggestion, two procedural changes were incorporated: (1) Subjects also received digital feedback indicating the amount of alpha they had produced during each of the 2-min periods by means of a digital display that indicated the number of seconds of alpha during the preceding 2 min and that was lit for 5 sec immediately at the conclusion of each 2-min trial before the 1-min rest period started. This feedback was deemed important to maintain motivation, since subjects could not really judge how well they were doing by listening to the tones. Further, the digital display provided information concerning even relatively small changes. Subjects were required to read the display out loud, thus providing feedback to the experimenter about their continuing alertness. (2) The frontal output was used as the basis of feedback. However, as in our previous studies, occipital alpha was also recorded, and the changes in occipital alpha, which were essentially parallel to those of the frontal alpha, were used as the basis for analysis.

Alpha Densities - Day One - Total Darkness (N = 9)

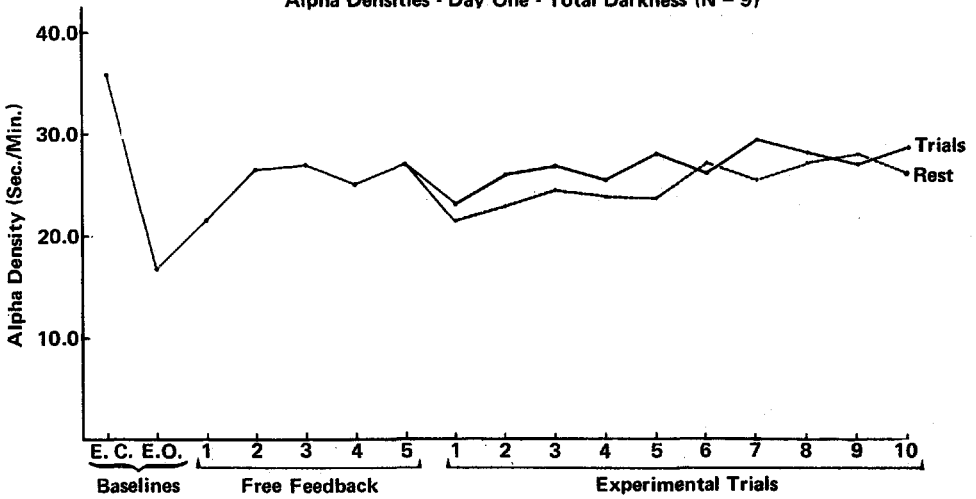


FIGURE 4. Mean seconds per minute of criterion EEG alpha produced on the first day of binary feedback by tones. The subjects were in total darkness during the auditory feedback as well as during baseline and rest periods. E. C.: eyes closed; E. O.: eyes open.

progressively less, presumably as subjects ceased to orient in a situation that was no longer novel.

The data suggested that subjects approached their maximal alpha density during the initial orientation period. The highest alpha density reached during any of the 10 alpha augmentation trials was only 7.2% more than that during this orientation period ($t = 1.81, p > 0.10$). Although in the group data the resting levels tended to be below trial levels, these differences were not significant.

When both trial and resting averages for all six sessions were examined with an analysis of variance, repeated-measures design, not one of the differences was significant (trials: $F = 0.19, p > 0.20$; rest: $F = 0.05, p > 0.20$). The largest difference between any two trial averages was only about 4 sec of alpha activity per 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 in the first session ($t = 0.35, p > 0.20$). Thus, within sessions or across sessions, no evidence indicative of learning to augment alpha density beyond the highest half-minute of alpha during the initial eyes-closed baseline period was noted in any of the subjects. Most important, subjects' initial eyes-closed baseline was not significantly exceeded at any time during the six days of training (Figure 5).

It appeared that by eliminating light from the feedback setting,

one also eliminated any evidence of alpha augmentation during feedback training. These data, in conjunction with extensive pilot studies, led us to conclude that subjects do not appear to exceed their initial optimal baseline levels of alpha density with feedback training. Evidence of learning was present only if alpha density levels had somehow been depressed. To document this last point, it was necessary to clarify the relationship of these data to the effect of light on alpha density.

So that we could confirm the essential effect of the presence of light, the subjects who previously had failed to show any evidence of learning after six sessions spread over six days were asked to return for one additional day of feedback. Eight of the nine subjects were able to participate. Their EEG response in the identical experiment except for the presence of ambient light was far more similar to that of earlier subjects given light-signal feedback than it was to their own past performance during six sessions in total darkness (Figure 6). Recovery from the initial drop took place slowly, but their highest trial alpha density was 55.7% higher than their highest minute during the orientation period ($t = 3.04, p < 0.02$). The difference between trial and resting averages was significant ($t = 2.47, p < 0.05$). Tests between the results of the first session in total darkness and the subsequent session with dim ambient light indicated that the session with light for those same subjects was significantly different—both in reduced trial averages ($t = 9.11, p < 0.001$) and reduced resting averages ($t = 5.57, p < 0.001$)—from their performance in darkness.

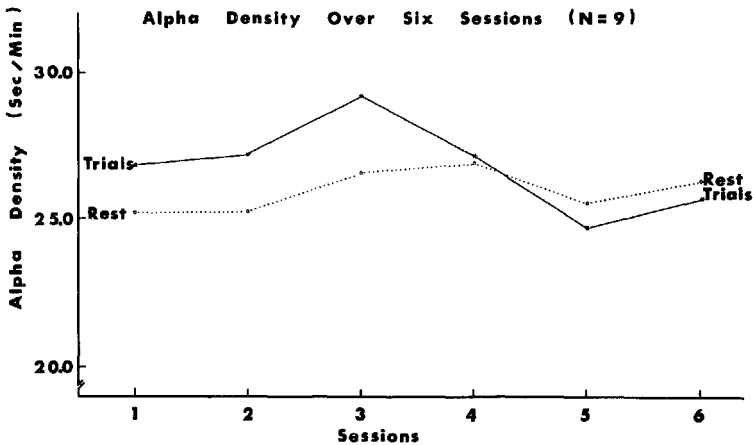


FIGURE 5. Mean seconds per minute of criterion EEG alpha produced during each of six separate sessions of binary alpha wave feedback by tones. The subjects were in total darkness during the auditory feedback as well as during baseline and rest periods.

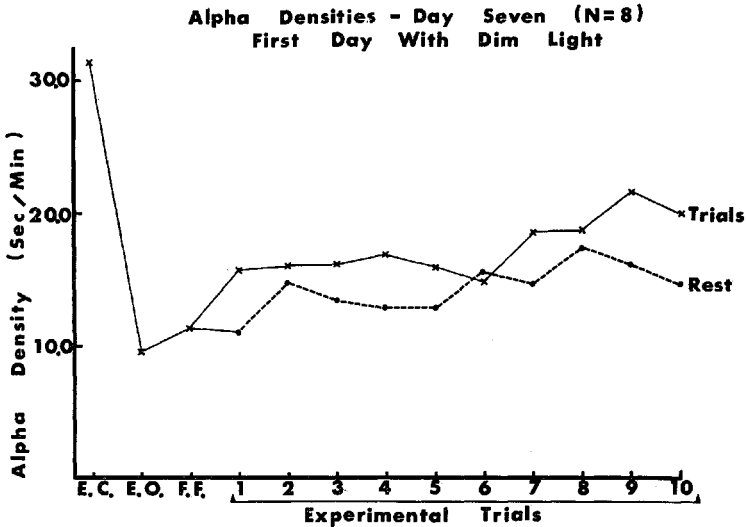


FIGURE 6. Mean seconds per minute of criterion EEG alpha produced during the seventh session of binary alpha wave feedback by tones. In this session, auditory feedback was presented while the subjects sat in a dimly lighted room. E. C.: eyes closed; E. O.: eyes open; F. F.: free feedback.

The importance of light, which had long been noted and again underlined in the earlier studies, was now clearly identified as being of major significance to any understanding of the alpha feedback experience. Further, the data supported the hypothesis that the apparent augmentation of alpha density during feedback occurred only when alpha density previously had been depressed by light. The increment in density shown during feedback seemed to involve the individual's gradually learning to ignore the stimuli that had been responsible for alpha suppression in the first place; that is, to cease orienting to visual stimulation.

B. "Looking" and Alpha Density

Mulholland (1969) previously suggested that alpha production was intimately related to visuomotor activity, specifically that of the triad of visual accommodation (convergence, pupillary constriction, and lens accommodation) rather than to visual stimulation, visual attention, or attention itself. He argued that only to the degree that

attention is coupled with oculomotor control is it likely to be linked with alpha. This hypothesis regarding the connection between alpha production and the triad of accommodation was tested by Pollen and Trachtenberg (1972), who demonstrated that alpha blocking still occurred when the visual task was arranged so that feedback for accommodative effort was neither available nor required (accommodation was blocked with a cycloplegic agent, and lenses were provided to allow focused vision). Thus, although there are obvious limitations to the use of peripheral nerve blocks to examine central nervous system performance, this study suggested that the specific nature of the link between alpha and vision was still obscure. However, the general conclusions that could be reached included that, in some way, visual activity had a powerful influence on alpha density. Our data were also in agreement with this idea. Therefore, we sought to tease apart the relationship between visual attention and that of attention in general with regard to alpha density.

An unpublished study³ compared attempting to see a barely perceptible visual stimulus with attempting to hear a barely perceptible auditory stimulus. Nine subjects participated in the experiment, which was conducted in a totally dark room. Baselines for eyes-closed and eyes-open alpha density were obtained. Counterbalanced sequences of an auditory and a visual attention task were then conducted as follows: The subject was told that sometime after a tone sounded a very dim light would be turned on. As soon as he perceived that the dim light was actually on, he was to press a button to let the experimenter know. The contingencies were arranged so that the very faint and difficult-to-identify light was turned on some 45 sec after the signal. A closely analogous task involved the identification of the presence of an auditory stimulus that was barely above threshold. This task, although equally difficult for the subject, had a significantly different impact on alpha density.

As Figure 7 shows, alpha density dropped precipitously—approaching zero in several subjects—as soon as the signal was given to search for the light and well before the stimulus was actually present. Once the light was identified, alpha density tended to increase again. In some cases, the light was not actually identified by the subjects, but the effort of trying to locate it was nonetheless sufficient to depress alpha density. Thus, even in total darkness, the attempt to see an object served to depress alpha density. Visual search produced alpha levels that were significantly below those during the auditory task (*ts*

³ Paskewitz and Orne, 1973.

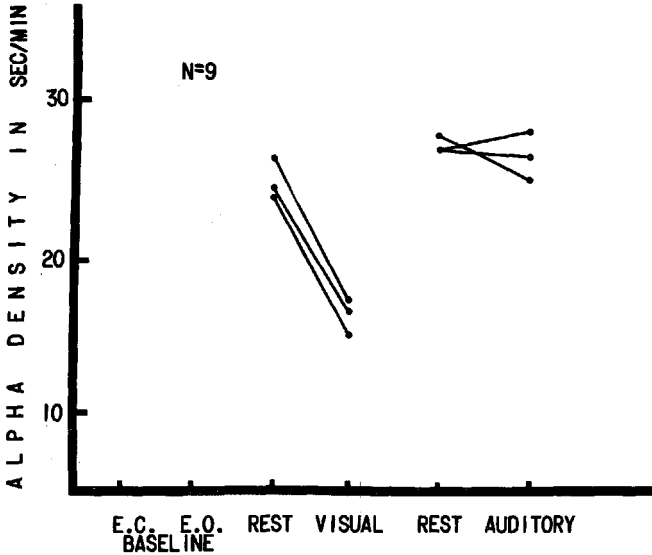


FIGURE 7. Mean seconds per minute of criterion EEG alpha produced during visual and auditory searches. The subjects sat in total darkness during both tasks. E. C.: eyes closed; E. O.: eyes open; Visual: visual search period; Auditory: auditory search period.

for the three trials, 3.08, 2.40, and 3.45; $p < 0.01$) and rests (t s for the three trials, 4.43, 2.60, and 3.08; $p < 0.01$). Alpha density during auditory search was not significantly below resting levels of alpha density (t s for the three trials, 1.23, 0.18, and 1.16; $p > 0.10$). Clearly, in contrast to visual search, the auditory search task caused very little drop in alpha density.

It is apparent that the attempt to see, even in the total absence of visual stimuli, is sufficient to produce alpha blocking. Thus, these findings replicated the visual-attention effects on alpha density reported by Adrian and Matthews (1934), supported by Durup and Fessard (1935), and suggested as part of the definition of alpha by Storm van Leeuwen and committee (1966). However, it would appear that the actual relationship of alpha rhythm to visual activity, brain activity, and subjective state is considerably less clear than one might expect 40 years after those simple and elegant studies that first demonstrated the connection between alpha density and the visuomotor system. Certainly, our work within the feedback setting did confirm and expand upon some of the original observations of the alpha rhythm's basic characteristics.

The primary finding was that the visuomotor system is of overriding importance in the suppression of, and in subsequent learning to

enhance, alpha production in the typical feedback session. This connection between visual processes and alpha density is particularly direct for parieto-occipital alpha but, although still present to a considerable degree, is less so for temporal, central, and frontal areas of the brain. The importance of these regional differences in alpha density and alpha dynamics for alpha enhancement effects has not been fully clarified. However, early published reports (Brown, 1970; Kamiya, 1969; Nowlis and Kamiya, 1970) of the subjective effects of alpha increases did not direct attention to lateralized or regional differences in brain function. The enhancement of alpha and the subjective effects were demonstrated with occipital alpha recording but were assumed, or implied, to be whole-brain phenomena based on a change in overall psychophysiological state.

With the above data available, it seemed apparent that the effects of alpha feedback training should be reconceptualized as an experience that teaches the subject to augment alpha under circumstances that ordinarily reduce the amount of alpha in the EEG (Paskewitz *et al.*, 1970). The visuomotor system seems to be the overriding factor determining alpha levels in those circumstances in which the person can see visual patterns or, in a totally dark room, attempt to see them. Further, alpha enhancement under conditions that include the subject's eyes being open in a dimly lighted room seems to require different learning strategies, such as avoiding looking at anything directly. In addition, it may have different subjective effects when compared with alpha increases that might occur in a subject sitting with closed eyes in a totally dark room (Plotkin, 1976b; Travis, Kondo, and Knott, 1975).

Of considerable importance to the utilization of alpha feedback in a totally dark room is whether, under such conditions, a subject can increase his alpha density over an optimum eyes-closed resting baseline level. This question remains the center of current controversy. The recent papers by Hardt and Kamiya (1976) on the affirmative side and Plotkin (1976a) on the negative side adequately review the conflicting evidence. It would seem that it may be possible, as Hardt and Kamiya have pointed out, that different kinds of alpha recording and different feedback techniques, as well as longer periods of training than those described here, would allow subjects to demonstrate significant increases in alpha. However, we would agree with Plotkin that the burden of proof remains with those who make the claim. A convincing demonstration of such alpha changes has not yet been forthcoming.

Given that previous feedback enhancement of alpha wave density appeared to result from the lifting of alpha suppression mechanisms,

such as those connected with light, the exploration of other potential sources of alpha inhibition seemed the most important area for further research. As we noted above, in the absence of light the suppression of alpha density that seemed to occur in the routine feedback setting could be seen only during the first session, when the subject opened his eyes in the total darkness. Once this relatively brief blocking effect is overcome—a process that occurs spontaneously during the first 2-3 min of the free-play period—the subject's alpha level again approximates that of the initial baseline, and no further augmentation can be seen during training. It seemed appropriate, therefore, to recognize that it might be necessary to reconceptualize the nature of the mechanisms underlying alpha density changes in the alert subject.

VI. ALPHA AND AROUSAL/ACTIVATION

The findings on levels of alpha density during visuomotor activity had begun to clarify some of the issues surrounding alpha blocking in the feedback context. At the same time, the basic link between alpha density and arousal began to appear to be much more complex than earlier workers had assumed. The initial experimentation, as reported above, had implicated the visuomotor system much more than arousal in alpha density changes, but no controlled manipulation of activation/arousal had been carried out. Therefore, the most appropriate next step seemed to be to explore directly the key hypothesis justifying the use of alpha feedback in the clinical setting: that alpha density is linked to subjective and physiological arousal by an inverted U-shaped function.

In brief, both Lindsley (1952) and Stennett (1957) hypothesized that alpha density is related to activation or arousal by an inverted U-shaped function. That is, alpha density was felt to be at a maximum during alert, but relaxed, nonfocused mind-wandering, while dropping to zero with the onset of sleep. During physiological and subjective arousal, as in anxiety-tension, alpha density seemed reduced and approached minimal levels during periods of extreme excitement or panic. These assertions, based on laboratory-manipulated changes in arousal, appeared to receive further support from clinical research (Cohn, 1946; Costa, Cox, and Katzman, 1965; Jasper, 1936; Lemere, 1936; Ulett and Gleser, 1952; Ulett, Gleser, Winokur, and Lawler, 1953) on neurotic and schizophrenic patients with constant high arousal. Therefore, we began our exploration of the relationship of alpha density to levels of arousal with the assumption that the hypothesized relationships were essentially confirmed. However,

it was apparent that visuomotor activity had to be controlled if we were to obtain uncontaminated observations on alpha-arousal interactions.

Since the previous data suggested that visuomotor activity in an alert subject under lighted conditions overwhelmed the effects on alpha density of any other behavior, the additional alpha blocking from activation/arousal might be difficult to discern under those circumstances. It was expected that a highly anxious person in a lighted room would block alpha more from his visuomotor activity than from the effects of fear itself. However, it was assumed that only learning to inhibit the fear effect would produce the dramatic subjective change relevant to controlling emotional turmoil in response to stress. For this reason, the presence of ambient light was eliminated from further experiments, and the hypothesized arousal mechanisms that might be responsible for reducing alpha density below optimal levels became the focus of attention.

A. High Levels of Arousal—Fear

We were left to confirm the assumption that since activation/arousal leads to decrements in alpha density, as did visuomotor activity, feedback training might permit the subject to disregard the alpha blocking effects of anxiety, just as it did those of light. It seemed entirely plausible that an anxious or aroused individual with reduced alpha in a totally dark room might learn to increase alpha density with feedback training and thereby learn to inhibit the mechanisms responsible for the physiological and psychological concomitants of anxiety.

Therefore, a study was specifically designed to: (1) establish during Day 1 the relaxed individual's optimal initial baseline; (2) create anxiety or fear in the subject over returning to the laboratory for a second session so that baseline alpha density for his second session presumably would be depressed; (3) show, then, how alpha feedback training can serve to increase alpha density even in total darkness, if it had initially been depressed by this situational anxiety; and (4) create a situation in which the subject would periodically be placed in jeopardy of being shocked (which would, presumably, again depress the level of alpha density) and in which an increase in alpha density would reduce or eliminate the likelihood of being shocked. In other words, the paradigm would approximate the all-too-common life situation in which the anxiety response is counterproductive and must somehow be controlled.

In an experiment by Orne and Paskewitz (1974), subjects first came to the laboratory to participate in a simple alpha feedback training experience. Every effort was exerted to make the subject comfortable and relaxed. A number of baselines were obtained, and feedback was given in the presence and the absence of ambient light. At the conclusion of this initial session, those subjects who had greater than 25% alpha were given the option of returning for a second session. It was explained that although it was very important for them to return, they were under no pressure to do so since the subsequent sessions involved receiving mildly uncomfortable to quite painful electric shocks to the calf of the leg. Thus, the experimenter refrained from actually reassuring the potential volunteers, although he made it clear that no injury would result. Of the 22 eligible subjects, 10 agreed to continue.

During the second session, two large silver electrodes and a ground were attached over the right gastrocnemius muscle, after the routine sensory electrodes had been positioned for recording EEG, EOG, GSR, and heart rate. The subject was informed about the nature of the silver shock electrodes but was given no instructions regarding when shocks might occur, since it was felt that any ambiguity about the shock would maximize anxiety. The experimenter left the room, the lights were turned out, and the entire session was conducted in total darkness.

Eyes-closed and eyes-open baselines as well as four routine 5-min feedback trials were given to the subject before shock instructions occurred. It was then explained that during the next part of the experiment he would, from time to time, receive electric shocks. "Jeopardy" periods (those times when he was in danger of being shocked) would be signaled by a third tone, clearly distinct from the alpha and no-alpha tones. This third tone would be on only when he was not producing alpha. Simply by turning on alpha, he could turn off the jeopardy tone and prevent his being shocked. It was emphasized that the only time he could be shocked would be while the jeopardy tone was on. Therefore, the more alpha he could produce, the less the likelihood of his being shocked.

Following these shock instructions, the subjects were given five 5-min feedback trials. Each of these trials was divided into 10 contiguous half-minute segments, 5 of which were jeopardy segments during which the third (or shock warning) tone was always present simultaneously with the no-alpha tone. During the other 5 segments, only the usual alpha or no-alpha tones were presented.

The shock contingencies were, in fact, arranged so that subjects received one to two shocks during each 5-min feedback segment.

Shock intensity was varied during the experiment, with only one or two being sufficiently intense to feel painful (since the purpose of the shock was to create apprehension rather than to inflict discomfort). These same procedures were repeated during a third visit to the laboratory.

The findings did not confirm the predictions of the theory. The initial alpha baselines during the second session were just as high as those in the first session, when no shock threat was present. During the first four feedback trials, alpha density was sustained at baseline levels (see Figure 8). The lack of alpha blocking following the shock instructions was most striking, in view of previous reports that fear causes drops in alpha density (Stennett, 1957). Alpha density did drop slightly, but transiently, during the first two jeopardy periods themselves. However, by the third jeopardy feedback period, alpha density levels were no different than those during nonshock feedback trials. The data from the third session showed alpha density differences between jeopardy and nonjeopardy periods only during the first jeopardy feedback trial. The group mean alpha density was equivalent to baseline levels during the rest of the trials. Thus, neither the anticipation of receiving electric shock nor the signal of the imminent

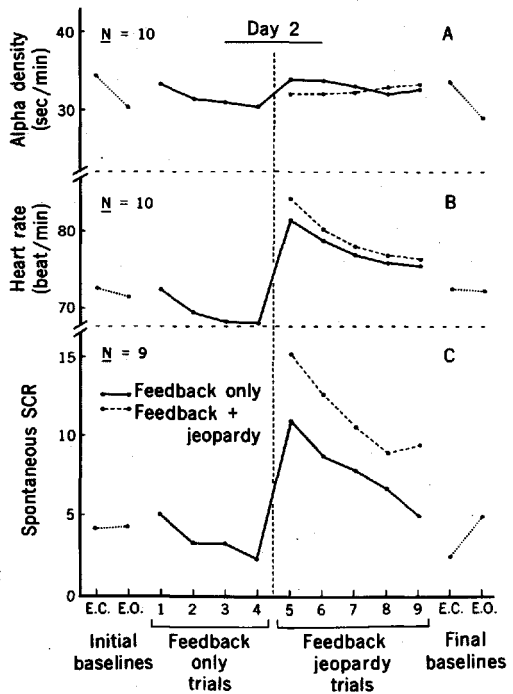


FIGURE 8. Mean seconds per minute of criterion EEG alpha, heart rate, and spontaneous skin conductance responses (SSCR) for the first day of shock (Day 2). E. C.: eyes closed; E. O.: eyes open.

onset of shock served to reduce the subjects' production of alpha density levels comparable to those found during resting baselines in the dark.

In an interpretation of these data, the first possibility to be considered was that the shock manipulation was not successful in making the subjects anxious. However, postexperimental inquiries clearly substantiated predictions that subjects would be anxious. Furthermore, during the experiment itself, visual observation (an infrared video system, included as a safety precaution, had permitted unobtrusive observation in the total darkness) revealed that the facial expression and demeanor of the subjects clearly suggested that they were anxious. Finally, other physiological data, notably heart rate and electrodermal responses, substantiated the subjects' reports and our behavioral observations. For example, as can be seen in Figure 8, when shock instructions were given, an instantaneous and dramatic increase in heart rate of well over 10 beats per minute took place ($t = 2.98, p < 0.01$). Pertinently, heart rate was significantly higher during jeopardy periods than during nonjeopardy periods ($t = 2.05, p < 0.05$), and when shock trials were over, heart rate returned to baseline levels. A second measure of activation, the number of spontaneous skin conductance responses (SSCRs), showed a closely analogous sequence of arousal. Data from the third session showed alpha, heart rate, and SSCR patterns very similar to those of the second and, therefore, replicated the findings.

Thus, neither the apprehension about the shock session in general, which might have been reflected in a drop in the second session's initial baseline densities, nor even the acute fear of being shocked resulted in the anticipated sharp drop in alpha density. The expected relationship between high levels of activation and reduced alpha density did not materialize. The data clearly indicated the lack of a necessary relationship between alpha density and the apprehension, anxiety, fear, or arousal levels of the subjects in this experiment. The discrepancy between these observations and previous reports (Lindsay, 1952; Stennett, 1957) of a link between alpha density, on the one hand, and subjective state and physiological arousal, on the other, clearly suggested that the old hypothesis required further exploration.

Insofar as the above results might reflect on the possible effects of alpha feedback training, they must be considered tentative because of the lack of yoked noncontingent feedback controls and the use of selected volunteer subjects. However, these findings call into question the assumed relationship between subjective anxiety-tension and alpha density, the basic notion upon which the rationale for the use of alpha biofeedback to reduce the effects of stress was founded. The

study suggests that the simplistic assumption that alpha density always reflects a specific level of physiological activation/arousal does not hold, at least following and/or during alpha feedback training. Although these data, since they were collected during alpha enhancement feedback, cannot directly demonstrate the inadequacy of the inverted U-shape hypothesis describing the relationship between alpha density and arousal, they call into question the continued, unconsidered use of this conceptualization of the relationship of subjective and objective arousal with EEG alpha wave generation.

It is possible that the older literature (Lindsley, 1952; Stennett, 1957) suggesting a connection between high levels of arousal and decreased alpha density reflects a fortuitous combination of situation-specific factors and mediating influences that are not yet understood. Several phenomena were not adequately considered or controlled in previous studies. For example, the effects of novelty on the interaction between alpha density and arousal appear to be of considerable importance, particularly during the first visit to the laboratory. Johnson and Ulett (1959) found an inverse relationship between alpha density and Taylor Manifest Anxiety in 44 males during the first baseline recording session. However, they noted that this relationship was not present during the second and third visits to the laboratory. Johnson and Ulett recognized that they were not dealing with a simple relationship between optimal tonic level of alpha activity and anxiety but, rather, with a correlation that followed from differential response to a new and subjectively important experimental context. This point has been independently documented by Evans (1972) with regard to attempts to relate hypnotic responsivity to a subject's baseline alpha density. Most previous EEG studies of patients or of laboratory manipulation of fear have been carried out with subjects during their first experience with EEG recording. Thus, the interactions among the effects of novelty and fear with cortical activation cannot be separated without further controlled experimentation that takes these underlying factors into account.

The failure in earlier work to distinguish between studies performed with subjects having their eyes open in the presence of some ambient light versus those with subjects with their eyes closed or in the total absence of light produced even more confusion. The presence or absence of light not only interacts with habituation to the environmental situation but also plays a major role itself, with or without alpha feedback. Thus, attempts to relate current data to previous studies are frequently frustrated by the absence of standardized recording conditions in work performed before the effects of these phenomena were clearly recognized.

Early studies (Adrian and Matthews, 1934; Berger, 1929; Thiesen, 1943), which reported the alpha blocking effects of anxiety and arousal, typically used stimuli that were both novel and anxiety arousing. Further, little concern was given to concurrent visual activity. However, it now seems plausible to consider that any drop in alpha density that previous studies ascribed to arousal might actually have been the result of orienting to novelty or the visual activity provoked by the same stimulus responsible for emotional arousal.

Finally, Surwillo (1965) criticized Stennett's (1957) frequently cited study of the inverted U-shaped function as the result of an erroneous analysis of the data. Surwillo used the relationship between alpha amplitude and heart rate in his subjects to show that a single individual rarely demonstrates the inverted-U function. He found that his data, as well as Stennett's, produced such a curve only if subjects who increased alpha with increasing arousal were juxtaposed with those who decreased alpha with increasing arousal. The combination of the two limbs thus formed then created the inverted-U shape. However, this juxtaposition was possible only if the relative level of activation among the subjects was ignored. Thus, some of the key data ostensibly supporting the hypothesis have themselves been questioned.

It appeared possible, then, that our failure to find significant changes in alpha density with high arousal might be in agreement with Stennett's (1957) data as interpreted by Surwillo (1965), while still serving to discredit the inverted U-shaped function hypothesis. It was clear that it was necessary to reexamine carefully the nature of the high arousal end of the curve. However, the data for the low end seemed much less likely to be confounded by the above problems. For example, subjects falling asleep would have their eyes closed and would thus be exposed to the same low visual stimulation rates. Indeed, as will be demonstrated, our data seemed to confirm the older literature (Lindsley, 1960; Stennett, 1957) on the relationship between drowsiness and low alpha density.

B. Low Levels of Arousal—Drowsiness

Initially, practical concerns over obtaining valid baseline alpha densities against which to compare feedback results led us to examine some of the circumstances under which measures of alpha density were or were not characteristic of the individual. The initial 3-min eyes-closed and final 2-min eyes-closed baselines of subjects coming to the laboratory for a variety of feedback sessions were evaluated

(Paskewitz and Orne, 1972). The 24 subjects were primarily males who had participated in at least three laboratory feedback sessions.

The average intercorrelation (Pearson) between the mean alpha density for the six periods (two baselines during each of three visits) was 0.76, with individual coefficients ranging from 0.67 to 0.95. In spite of the generally high correlations, some baselines were highly atypical and failed to reflect the subject's usual alpha density. Baselines with reductions in alpha density of greater than 50% during 30-sec intervals were examined more closely in a subset of 9 subjects for whom eye movement data were available. Of 22 atypical baselines, 15 were accompanied by slow eye movements, a characteristic precursor of the onset of sleep (see Table 1).

Thus, a study of the reliability of baseline EEG alpha measures also clearly documented the now well-established relationship between the onset of drowsiness, which merges into Stage 1 sleep, and a corresponding decrease in alpha density. It is tempting to accept these data as documenting the relationship between low arousal and the absence of alpha. Here too, however, caution is needed. The drop in alpha density may not be a function of low arousal at all; rather it may be an incidental manifestation of the active processes associated with sleep onset.

For example, if one examines nighttime sleep records, there are periods when individuals show a great deal of arousal. Notably, REM is associated not only with the rapid eye movements that give the sleep stage its name but also with other manifestations suggesting heightened arousal, such as penile erection and marked variation in heart rate. Nonetheless, during these periods there is a disproportion-

TABLE 1
Number of Episodes of Slow Eye Movements Compared with Number of Periods of Atypically Low Alpha Density in 1-Min Samples from the Baseline Recordings of Nine Subjects

		Does Baseline Contain Atypically Low Minute?		
		Yes	No	
Are slow eye movements present?	Yes	15	5	20
	No	7	27	34
		22	32	54

$$\chi^2 = 13.27, p < 0.001^a$$

^a The χ^2 values are based on data from 9 of 24 subjects. The authors recognize the compromise of the assumptions of the χ^2 statistic in that different subjects may be disproportionately represented. It seems reasonable to consider each depressed baseline as an independent event, however, even though it may occur more frequently in one subject than another.

ately small increase in alpha, especially when one considers the amount of mentation associated with dreaming as well as the autonomic arousal.

A similar paradoxical relationship between alpha density and arousal indices is suggested by the periods of GSR storms during Stage 4 sleep (Burch, 1965). This fascinating phenomenon does not seem to be accompanied by large changes in other physiological parameters, such as heart rate and respiration, but again, we are unaware of any evidence suggesting that alpha density normally increases during such periods.

Thus, activation within sleep appears to demonstrate a major separation of what seems to be a relatively unified physiological arousal in the waking individual. No considerable emergence of increased alpha density during normal sleep has been documented, yet arousal from these EEG sleep stages may be followed by reports of vivid dreaming experiences. We see, then, an apparent clear separation of processes signaling increased subjective and physiological activation from cortical alpha production. The absence of evidence demonstrating a continued link between brain-stem activation, the cortex, and subjective experience strongly suggests that the decrease in alpha density seen as an individual approaches sleep may reflect an active disengagement of alpha-wave-producing mechanisms from the cortex, rather than a low level of general brain arousal. The specific concomitants of the connection between general activation/arousal and cortical arousal indices such as alpha wave production has yet to be determined.

At this juncture, it may be concluded that a number of subjectively and objectively different mechanisms might have one final common effect: a reduction in alpha production. As demonstrated by the preceding experiments, both attempting to see an object and drowsiness have alpha blocking effects. While high arousal appeared, in previous experiments, to result in reduced alpha density, it is not clear at this point whether this was an independent effect, secondary to increased eye movements, or the effect of unspecified mechanisms. However, given the several apparently fundamentally different types of alpha blocking, it would follow that different skills might be necessary to learn to augment alpha density, depending on the nature of the primary stimulus that is depressing alpha activity. With this new perspective, it becomes relatively meaningless to speak of alpha feedback training in a generic sense. We have to understand what influences have served to depress alpha density below the person's optimum levels in each specific feedback circumstance.

The problem of specificity of response in biofeedback has also been explored by Schwartz (1972, 1974, 1975), particularly with regard to cardiovascular parameters. His group's further work on brain processes has led them to postulate that patterns of brain and peripheral physiological processes, rather than isolated parameters, may be more meaningfully linked to cognitive-affective experiences. Schwartz (1976a,b) suggested that emotions and conscious states must be seen as emergent properties of neural patterning—perhaps, for example, in interactions between the two hemispheres—rather than merely as functions of general neurophysiological activation. Although such a perspective adds to our ability to plan meaningful experiments, an understanding of the nature of phenomena such as lateralization of hemispheric activation depends on the central issue of the significance of alpha density for brain arousal or activation, as a whole or in regions. This significance is by no means clear at this point, and therefore, we shall not seek to comment further on this line of inquiry.

VII. IS DIRECT AWARENESS OF ALPHA WAVE PRODUCTION POSSIBLE?

Since a number of underlying relationships between alpha and subjective experience were now at least vaguely apparent, the conceptual importance of Kamiya's (1969) early study—reported anecdotally, to demonstrate that subjects could rapidly learn to discriminate between alpha and no-alpha periods in their own EEG—became even greater. The most direct approach to the potential link between subjective experience and alpha production seemed to lie in attempting to replicate, with more rigorous controls, the original Kamiya finding that subjects could learn to identify periods of alpha wave production. Very early in our pilot work, we had run one subject in 2 of his total of over 30 sessions while providing him with a manipulandum so that he could signal the presence or absence of alpha as he thought it occurred. Visual inspection seemed to support Kamiya's observations that a subject could learn to identify alpha periods, but a number of problems made it very difficult to quantify such data. Therefore, we did not at that time pursue the matter further. However, the findings summarized above had convinced us of the need to address systematically the basic question of whether alpha bursts were reliably accompanied by an identifiable alteration in subjective experience.

An appropriate procedure was devised (Orne, Evans, Wilson, and Paskewitz, 1975) to allow for a more rigorous test of Kamiya's (1969) hypothesis. Subjects were automatically signaled periodically with a tone and required to indicate, by pressing the appropriate one of two buttons, whether they believed that they had or had not just been generating alpha. If the subject answered correctly, the signal tone was replaced by another, somewhat higher tone. If the choice was incorrect, the tone merely terminated. Thus, this situation provided "feedback" regarding only the presence or absence of alpha each time the subject responded to the tone. It is evident that this approach fits the classical signal detection model. Such a paradigm makes it possible to separate the accuracy of correctly identifying the presence of alpha independently from the accuracy of correctly identifying the absence of alpha. Further, it permits the identification of guessing strategies.

Though conceptually the experiment seemed straightforward and potentially elegant in its approach to the problem, the execution proved to present a series of unexpected problems. For example, even though care was taken to choose subjects with moderate amounts of alpha in order that alpha and no-alpha events would be equally frequent, these subjects, although well acclimated to the laboratory, showed a considerable increase in alpha, without feedback, during the second session and an even greater rise in the third session. Because of this dramatic increase in alpha density, finding periods of non-alpha with a duration of even 1-3 sec was very difficult. Thus, inequalities in the time intervals between alpha and no-alpha events developed.

The results were examined from several different perspectives. First, a day-by-day chi-square analysis for each subject suggested that correct discrimination was being acquired over time, but a more careful analysis showed that a significant chi-square reflected, in large part, an increase in correct guesses during alpha events with a corresponding increase in incorrect guesses during no-alpha events. Thus, the results were apparently a function of response bias on the part of the subjects, who seemed to believe that their alpha density gradually increased across days.

Further assessment through a one-sample runs test and a signal detection analysis confirmed that response bias was the central factor in producing the results. Although the relatively small number of trials and the possible violation of some of the underlying assumptions of signal detection make the results of such an analysis less than ideally clear, it did show that there was a very low d' index of discriminability. The response bias criterion showed signs of a strong "alpha" response bias effect that was relatively consistent throughout the

series, except for the seventh session, when two of the subjects reported feeling extremely drowsy.

Legewie (1975) and Pavloski, Cott, and Black (1975) also used this alpha/no-alpha discrimination procedure in experiments attempting to replicate Kamiya's (1969) original findings. Neither group was able to demonstrate that their subjects could actually discriminate between these two EEG states. When trial probabilities and confounding cues were controlled, the subjects could not determine at any one moment whether alpha or no-alpha was occurring in their EEG recording. In summary, these alpha state discrimination studies suggested that the apparent ability to discriminate between alpha and no-alpha events during the pilot studies was probably an artifact of the individual's strategy within the experiment. For example, our subjects tried to increase their incidence of alpha without instructions to do so and followed this attempt with the strong tendency to choose "alpha" more often than "no-alpha" for their decision.

While it would be all too easy to dismiss Kamiya's (1969) anecdotal findings in light of the above studies, we are not yet prepared to do so. The number of subjects examined for the ability to discriminate alpha and no-alpha conditions is small, and our automated procedures may be obscuring the issue as much as helping to clarify it. Thus, our failure to replicate the earlier Kamiya results may be as much a function of our approach as of the nature of alpha. However, while it is, of course, possible that it is necessary to train individuals with longer windows than those that were used in these studies, it would seem essential that more carefully controlled positive observations be obtained before we are justified in assuming that the simple presence of alpha has cortical representation.

The line of inquiry into alpha and its connections with subjective experience had thus demonstrated that: (1) subjects do not appear to learn to increase their alpha density above their resting baseline through feedback; (2) visuomotor activity is of prime importance in depressing optimal alpha density and in subsequently learning to enhance alpha; (3) high levels of alpha density can be present even during very high arousal and subjective fear during alpha feedback; (4) the absence of alpha during activation/arousal changes during sleep suggests that whatever relationship exists in the waking state between alpha density and arousal levels is not readily seen during sleep itself; and (5) subjects may not be able to discriminate directly between alpha and no-alpha events during waking states. In sum, the view that alpha production is closely related to subjective experiences, has specific cortical representation, and alone reflects level of activation/arousal cannot be justified with currently available data.

Given these observations, it seems that the entire basis justifying the potential benefits of alpha feedback training is lacking, and accordingly, one might well choose to dismiss this entire line of inquiry. However, throughout our efforts to understand alpha feedback, we have become increasingly aware of the need to understand the underlying processes, and we have been forced to reevaluate issues that were assumed to be resolved by previous work in order to reconcile the conflicting reports in the literature. Of several issues that arose, the single most important factor, which has been essentially ignored in the reported work to date, related to systematic individual differences in the dynamics of the alpha response. Such differences may provide further clarification of the nature of the conflicting findings reported above.

VIII. THE EFFECTS OF COGNITIVE ACTIVITY ON ALPHA DYNAMICS

The view that alpha blocking is always associated with concentrated mental activity was first hypothesized by Berger (1929) and was supported by Adrian and Matthews (1934). Several subsequent studies seemed to demonstrate a clear relationship between mental tasks themselves and the blocking of alpha activity in the EEG (Chapman, Armington, and Bragdon, 1962; Darrow, Vieth, and Wilson, 1957; Glanzer, Chapman, Clark, and Bragdon, 1964; Glass, 1964, 1967; Lorens and Darrow, 1962). We also found, in early studies, that combining the task of incrementing alpha through feedback with a cognitive task such as subtracting by sevens produced more alpha blocking.

Individual differences in the degree of blocking, depending on the person's proficiency at the task and his self-paced rate of performance, seemed to substantiate such an interpretation. For example, one subject, choosing to do an arithmetic task more quickly than he could readily manage, showed large amounts of blocking, while another, going more slowly than justified by his skill in arithmetic, showed little blocking. It appeared obvious that the task difficulty at any given time was determined not only by the task itself and the individual's proficiency in the task but also by the individual's rate of task performance (Paskewitz and Orne, 1972). However, several other studies (discussed below) also seemed to indicate that there are other individual differences that might mediate the different alpha blocking reactions between persons.

A. *Previously Reported General Effects of Tasks on Alpha Density*

Mundy-Castle (1957) found that both mental arithmetic and imagery could be carried on without necessarily leading to alpha blocking. He concluded from his studies that there was no one-to-one relationship between alpha blocking and visual activity or attention. Further, Chapman *et al.* (1962) noted that mental arithmetic reduced alpha in an eyes-closed but increased it in an eyes-open condition. Kreitman and Shaw (1965) observed, in a study of eight subjects, that alpha density increased in some individuals during most tasks. Legewie, Simonova, and Creutzfeldt (1969) replicated a previous finding (Creutzfeldt, Grünewald, Simonova, and Schmitz, 1969) that a number of experimental tasks performed during an eyes-open condition increased temporo-occipital alpha in seven of eight subjects, while decreasing it when their eyes were closed. Thus, this group of studies tended to concentrate on the interaction between direction of alpha change during a task and visuomotor effects.

In contrast, Pollen and Trachtenberg (1972) focused on the impact of task difficulty. By varying the demand on mental effort, they found that in an eyes-closed condition, no alpha blocking occurred during the easier parts of their progressively more difficult range of tasks. In those sections that demanded greater mental effort, alpha blocking was present and continued until the problem was solved. Their results thus suggested that alpha augmentation might be expected only during lower-level mental effort. Any differences in alpha attenuation between subjects over different tasks could then be attributed to individual differences in task-related skills or effort.

In sum, the literature has concentrated on the effects of light on alpha changes during a task or on experienced task difficulty. However, the effects of the novelty of the experimental setting and the tasks were not well controlled. Further, the meaning of the fact that some individuals, when performing a mental task with their eyes open, augmented alpha was not clarified.

B. *Individual Differences in Alpha Response to a Task*

In view of the possible individual differences inherent in previous data and their potential practical and theoretical import, the effect of cognitive tasks on alpha density was reexamined (Orne *et al.*, 1975), with particular attention given to the control of novelty effects and to

the elimination of light from the experimental setting. Subjects were run through the same baseline recordings and essentially similar tasks on three different days, both as a preliminary familiarization with procedures in order to control novelty and to permit selection of those who were to participate in a feedback study to extend over several days.

The three sessions were designed to record alpha density while the subject sat in a totally dark room. Conditions included were eyes-open and eyes-closed resting baselines, as well as carrying out a number of tasks requiring different levels of cognitive effort. Following the initial eyes-closed and eyes-open baselines, a number of 90-sec serial subtraction tasks using several different numbers, as well as descending subtraction, were interspersed with 1- and 2-min baselines. The subtraction tasks varied in difficulty from simply counting backward by ones to the most difficult descending subtraction task. For the latter, the subject began by subtracting 9 from a three-digit number, then 8 from the remainder, then 7 from that remainder, and so on until reaching 2, when he began again with 9, 8, 7, etc., until told to stop.

The tasks were followed by ones designed to elicit left- or right-hemisphere activation specifically, such as verbal and mathematical problems for the left and visualization of scenes and visuospatial problems for the right. Five problems of each of the two types were performed in a counterbalanced order, with intervening 20-sec rests separating them. Essentially similar, although slightly modified, tasks and baselines were carried out during all three sessions. Thus, it was possible to compare alpha changes between tasks after novelty had been eliminated. All EEG data were obtained from bilateral recordings of monopolar occipital EEG, with the right mastoid used as reference, and recorded on paper. Criterion alpha was measured by use of a 15- μ V amplitude standard for the presence of alpha.

During the first session, there was a general tendency to block alpha while performing the tasks, although some subjects blocked alpha much more than others. However, when the data from the second session were examined, strikingly specific individual differences in alpha dynamics emerged. Among these subjects, all of whom were used to the experimental procedures and were dark-adapted, seven responded to subtraction by ones by incrementing their alpha density above their own baselines and four responded by blocking alpha. However, given the Pollen and Trachtenberg (1972) findings on task-difficulty effects, one would anticipate that all subjects would block alpha during the difficult descending subtraction.

Subjects were therefore divided on the basis of whether they increased or decreased alpha density while counting by ones, so that we could see if this dichotomy would differentiate them when they performed descending subtraction. Figure 9 shows the mean percentage of left-hemisphere alpha density of two groups: four alpha blockers (dotted lines) and seven alpha augmenters (solid lines). The individual was assigned to the augmenting or blocking group on the basis of his alpha density change from baseline during subtraction by ones in the second session. Subjects who blocked alpha while counting backward by ones also did so during descending subtraction. However, contrary to expectations, those who increased alpha density while performing the simple task increased it during the difficult one as well!

As Figure 9 demonstrates, the two kinds of alpha response to a task are not related to differences in resting alpha density either during the initial baseline or in the rests preceding the tasks. Since the two groups were defined by the direction of their alpha response during subtraction by ones, it is hardly surprising that their alpha density is significantly different during that task. However, the continued differences (Trial 1, $t = 1.82$, $p = 0.05$; Trial 2, $t = 3.09$, $p < 0.01$) in their alpha response to the much more demanding descending subtraction task were remarkable, particularly since these differences were not related to the individual's success or speed in counting backward during the descending subtraction task.

The consistency of an individual's alpha response to a task is further demonstrated by the continued significant differences between these two groups, separated by direction of alpha change with subtraction by ones on Day 2, during the descending subtraction task

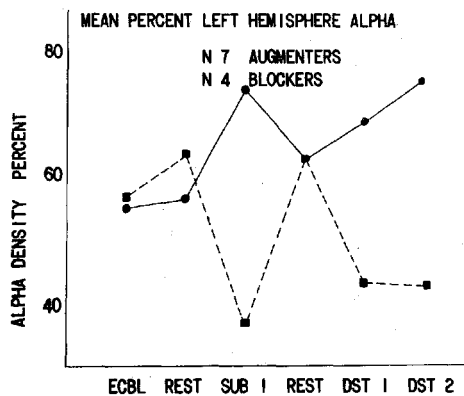


FIGURE 9. ECBL: eyes-closed baseline; SUB 1: subtraction by ones (1's); DST 1 and 2: Descending Subtraction Tasks 1 and 2.

on Day 3. Again, the two groups showed their characteristic directions of response during the task, and their alpha densities were significantly different ($t = 3.02$, Trial 1; and 3.58 , Trial 2; $p < 0.01$ for both). The Pearson correlations between Day 2 and Day 3 alpha density change scores during the descending subtraction tasks were 0.56 , Trial 1, and 0.66 , Trial 2 ($p < 0.05$). Pearson correlations of alpha density between tasks on the same day were uniformly above 0.66 , regardless of the differences in the difficulty of the task. Thus, the individual differences in alpha dynamics appeared to be more important modifiers of alpha density response than task difficulty on the second and third days of the experiment.

The bimodality of these response characteristics, evident during the second and third days, was not present in the first day. On the contrary, a fairly uniform tendency to block alpha while performing a cognitive task was apparent. So that we could determine whether there were any individual differences reflected in Day 1 data, the number of subtraction tasks (total possible, five) during which an individual showed alpha augmentation was tabulated. Seven subjects identified as augmenters on Day 2 augmented alpha during a mean of 1.86 of the 5 subtraction tasks on Day 1, while six identified as blockers on Day 2 augmented during a mean of 0.33 tasks on Day 1 ($t = 2.69$, $p < 0.25$). (Two subjects who did not complete the third day are included in the Day 1 data.) Thus, an individual characteristic that was easily identified in Day 2 data was also present on Day 1 but not readily discernible because of the relatively uniform response to novelty.

These striking, reliable, and significant differences in the direction of alpha density changes during cognitive tasks, although observed by others in the past, have tended to be ignored because they were masked either by the presence of light or by novelty on the first day of testing. Therefore, they have been taken to represent random variation in alpha blocking. However, the persistent direction and amount of alpha change that occurred in our subjects across tasks and across days suggests that what may be manifest in these phenomena is a powerful and pervasive characteristic of the person's neurophysiological dynamics, rather than merely phasic changes whose nature is closely tied to his immediate mental effort or content. Thus, the same subjective experience and objective performance in some subjects may elicit considerable alpha blocking, in others alpha augmentation, and in still others little or no change in alpha density.

Clearly, such individual differences in response to cognitive tasks are not taken into account by current theories regarding alpha,

activation, behavior, and subjective experience outside of the feedback context. Still, one might consider dismissing them as irrelevant to the general activation/arousal theory justifying the use of alpha feedback in a clinical setting. However, conceptually similar spontaneous changes in alpha density occurred during high activation/arousal in an alpha feedback experiment (Wilson, Orne, and Paskewitz, 1976). Some individuals blocked alpha during fear of electric shock and some showed no change, while others increased alpha; all these different responses occurred during periods of large increases in autonomic indices, such as heart rate and spontaneous skin conductance activity. Thus, these individual differences may be quite pertinent to an understanding of the conflicting reports in alpha feedback research.

Travis *et al.* (1975), for example, reported that only about 60% of their subjects felt the attempt to enhance their alpha density as a neutral or pleasant experience. This kind of variability in reports of positive subjective experience has been explained in a number of ways. For example, Walsh (1974) showed that subject expectations and demand characteristics of the experiment have a significant impact on whether the individual reports positive experiences. However, individual differences in alpha dynamics such as those reported here may help explicate the findings in a more basic and ultimately more useful manner. They may also clarify the controversy surrounding the potential of individuals to augment alpha density over baseline levels. Our data demonstrate that such increases are possible, at least in some persons. However, they have occurred in response to difficult cognitive tasks, or high activation/arousal, rather than during relaxation.

In sum, although there is little question that the nature of a cognitive task or an emotional experience has an impact on alpha dynamics, directing data analysis toward individual differences permits the identification of another important dimension in alpha phenomena. This dimension has previously been obscured by the effects of light or by orientation to novelty on the first day of participation in an experiment. Once these effects are controlled by the subject's being adapted both to darkness and to the circumstances of the experiment, individual differences in alpha dynamics become evident. It seems apparent that one cannot expect to apply alpha feedback to obtain predictable results unless these powerful systematic individual differences are better understood and taken into account. Otherwise, the results of alpha feedback can, at best, be no more than confusing and, at worst, detrimental to some of those whom we would hope to aid.

IX. OVERVIEW AND PROSPECTS

What may we then consider to be established conclusions regarding the relationship between alpha and subjective experience? *First*, contrary to our initial naive hopes, we cannot assume that high alpha density is uniformly accompanied by a moderate physiological arousal or subjective calm. It is now clear that a number of different mechanisms influence alpha density and interact to determine an individual's tonic levels and phasic changes in alpha. *Second*, visuomotor activity is of primary importance in determining alpha density and in the subject's learning to augment alpha in the presence of light. It would appear that feedback training carried out in light requires the development of different skills and may have very different subjective and objective results than that carried out in total darkness. *Third*, the widely accepted inverted U-shaped function hypothesized to relate alpha density to activation needs to be reevaluated. *Fourth*, while in early work we could not get people to exceed baseline alpha levels, it is now clear that some subjects do—in response to activation. *Fifth*, very important systematic individual differences in alpha dynamics must be taken into account in any further studies of the relationship between cortical electrical activity, subjective experience, and behavior, as well as in alpha feedback training research.

The disappointing overall results of alpha biofeedback training, compared with the initial hopes, have forced a reconceptualization of the necessary conditions for the clinical application of alpha feedback. Our results suggest that once novelty and visuomotor effects are eliminated, alpha augmentation may be the product of relaxation in one individual and of hyperarousal in another, while a third may show little relationship between subjective state and alpha density. Thus, regardless of the area of the brain from which recordings are taken, or the pattern of other autonomic parameters, uniform subjective experiences over a population of subjects are unlikely to emerge from a single direction of alpha change. Unless the individual differences are taken into account, it would seem foolhardy to expect that alpha feedback would lead to uniform effects once novelty and visuomotor factors are excluded.

In sum, the research that has followed the original reports of a reliable connection between alpha production and subjective experience has tended to negate and/or qualify the early results. However, three more recently defined areas of inquiry must be understood before the final chapter on the potential subjective effects of alpha enhancement through feedback training can be written. More careful examination of the relationship of specialized areas of the brain to

behavior, as well as of the specific pattern of physiological reactions associated with particular emotional states, must be carried out. Perhaps most important to any future applications of EEG alpha feedback will be an in-depth exploration of individual differences in alpha dynamics. The potential new integration of basic neurophysiological and neuropsychological perspectives that may follow would then permit a more scientifically mature second approach to the use of this elusive method of interacting with man's neurophysiological self.

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Chapter VI

MEDICAL USES OF BIOFEEDBACK

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Many of the most interesting applications of biofeedback therapy relate to problems generally considered medical in nature. It is noteworthy that a number of these problems—hypertension, peptic ulcer, cardiac arrhythmias, asthma, and Raynaud's disease—have long been considered psychosomatic illnesses. The findings that biofeedback and relaxation therapies enable some patients to exert control over relevant physiological processes support that categorization. While ultimately it will be important to identify the specific factors responsible for a positive therapeutic outcome, this section will focus on the potential effects of the totality of procedures involved in biofeedback therapy in order to assess which areas of bodily dysfunction may be subject to treatment by means of mental mechanisms. It seems important at least to indicate the areas in which the total biofeedback package may be effective and where it should be explored systematically in future research. We will also, where possible, report comparative studies which indicate the relative roles of informational feedback, relaxation effects, and enhanced medication compliance in ameliorating the medical problem.

This chapter will provide an overview with suitable references for colleagues interested in pursuing this field in more depth, rather than try to cover the area exhaustively. Except as otherwise noted, biofeedback therapy refers not only to the use of electronic techniques to provide information for the patient and therapist, but rather to the total treatment context (which may share much with other more traditional psychotherapeutic approaches, including the nonspecific factors associated with them).

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Blood Pressure Control

Hypertension. One of the medical applications of biofeedback which has aroused the most interest is the reduction of high blood pressure. Following their development of a technique for monitoring blood pressure (BP) continuously for short periods of time without using invasive procedures—the technique relies on automated detection of Korotkov sounds—Benson, Shapiro, Tursky, et al, (1971) trained a group of seven hypertensive patients to lower their systolic BP with beat-by-beat feedback that indicates whether a patient has lowered his pressure below a criterion level. In this manner five of the seven patients decreased their systolic BP by 16 mm Hg or more. After this promising beginning, a number of subsequent studies were done, several of which clarified the mechanisms by which biofeedback might be operating. Relaxation with (Patel and North, 1975) or without (Benson, Rosner, Marzetta, et al, 1974; Taylor, Farquhar, Nelson, et al, 1977) adjunctive biofeedback was shown to lower BP, as was meditative practice (Stone and De Leo, 1976). However, BP biofeedback appeared to be operating in a somewhat different fashion for patients trained over a protracted period. Kristt and Engel (1975) studied five hypertensive patients who were hospitalized for three weeks and given intensive training in BP lowering and increasing using beat-by-beat systolic BP biofeedback. (No control group for relaxation was run.) By the study's end, the patients could raise or lower systolic BP without significantly changing heart rate, triceps brachii muscle tone, or occipital alpha-wave EEG prevalence. This finding suggested that biofeedback was capable of producing a quite specific change in the response under training, with minor effects on other variables, in contrast to relaxation techniques which produce generalized effects. It is of note that the BP changes obtained in these studies are of clinically significant levels, averaging 28.1/15.0 mm Hg in one study of 17 patients (e.g., Patel and North, 1975).

Another finding related to biofeedback training in hypertensive patients concerns the question of whether complex training equipment and procedures are necessary for clinical benefit. Two studies argue that they are not. (Carnahan and Nugent, 1975) 100 hypertensive patients were randomly assigned to two groups. One group was treated with only the clinic's standard medication protocol; the second was treated in the same manner, except that each patient was also given a sphygmomanometer and asked to record his BP twice daily at home. The second group thus had a simple biofeedback system for home monitoring of BP fluctuations. At the end of the six-month study, the systolic BPs of the second group were significantly lower (by 11.4 mm

Hg). Diastolic pressures were similar in the two groups, probably because the treatment protocol dictated that medications be added only until the patient's diastolic BP was 90 or less. In a related study (Haynes, Sackett, Gibson, et al, 1976) patients with hypertension who took their medications erratically were given BP cuffs for home use (and special instructions about pill taking). Their medication-use compliance increased, and their BPs were significantly reduced. Thus in hypertension, biofeedback may contribute to BP lowering by specific, physiological learning effects leading to reduced BP (Kristt and Engel, 1975), by relaxation effects (Benson, Rosner, Marzetta, et al, 1974; Taylor, Farquhar, Nelson, et al, 1977), or by producing life-style changes, including increased compliance with medication regimen (Haynes, Sackett, Gibson, et al, 1976).

Hypotension. This much less common medical problem was treated successfully in a single patient with orthostatic hypotension as a result of a spinal cord injury (Bucker and Ince, 1977). The patient was given extensive biofeedback training in increasing his BP. He gradually was able to produce large changes in this variable (48 mm Hg), enabling him to assume the erect posture for protracted periods of time—something he had not previously been able to do. With time his ability to maintain an adequate BP when erect became such that he did not have to exert much effort to do so. Physiological studies indicated that the patient had learned specifically to increase peripheral resistance. As this patient had had this problem for two years, was incapacitated by it, and was quite motivated to ameliorate his situation, his ability to maintain a satisfactory BP when erect does appear to have been a specific effect of biofeedback training. The relative importance of the informational feedback to the patient in his acquiring the skill, and to his therapist—thereby enabling the latter to encourage and request appropriate actions by the patient for increasing BP—is unclear.

This study emphasizes that biofeedback is not necessarily hypoaousal training. Feedback can also be used to train patients to increase arousal, as in hypotension.

Cardiac Arrhythmias

Another area in which biofeedback has been studied is the amelioration of cardiac arrhythmias. Small groups of patients with different types of arrhythmias have been studied, at times with clinical benefits. In most cases, these exploratory studies were conducted without control groups.

Premature ventricular contractions. Particular interest has centered on this arrhythmia because of its relationship to sudden death in certain patients (Gradman, Bell, and DeBusk, 1977) and its known susceptibility to higher nervous system influences (Lown, Verrier, and Rabinowitz, 1977). Three studies have documented effects of heart rate (HR) biofeedback training on premature ventricular contractions (PVCs). Typically the training procedure involves giving the patient informational feedback related to HR increases and decreases, and then training him intensively in the direction of HR change which minimizes PVC frequency. In one study (Weiss and Engel, 1971) of eight such patients, five showed a reduction in PVC frequency during training. Definite clinical benefit persisted for over 21 months in one patient, and possible lasting benefit occurred in three others. In a second study (Pickering and Miller, 1977) two such patients both showed an ability to reduce PVC frequency significantly; in one this ability was beneficial outside the training situation. One patient with PVCs in a parasystolic rhythm was trained in a third study (Pickering and Gorham, 1975). While HR increases were associated with less frequent PVCs later rather than earlier in training, no mention is made of clinical benefit. These studies indicate that biofeedback can modify the frequency of PVCs in a substantial percentage of patients who have been studied. However, to date, no delineation has been made of what types of PVC patients are most likely to benefit, nor has the role of relaxation—which has been shown to affect some such patients beneficially (Benson, Alexander, and Feldman, 1975)—been clearly separated from the specific learning effects of the biofeedback training procedure.

Wolff-Parkinson-White Syndrome. In this cardiac condition the conduction of the excitatory electrical activity from the heart's atria to its ventricles follows an aberrant and accelerated pathway. It is of particular clinical interest because patients with this syndrome tend to have the arrhythmia, supraventricular tachycardia, which in some instances can be so rapid and sustained as to be fatal. The use of biofeedback with two such patients has been studied. One of these studies produced a very striking instance of the power and specificity of the biofeedback paradigm, and will be discussed at some length. Here a 29-year-old woman with this syndrome was given two types of biofeedback training, one being HR control training which she did successfully, and the second being modification of the atrio-ventricular conduction pathway (Bleeker and Engel, 1973). In this second type of training she was required to increase or decrease the percentage of her heart beats conducted by the abnormal pathway. She was able to

do this to a significant extent, thus showing that she developed separate abilities to modify two different processes within the heart. This provides a cogent instance of the potential specificity of the biofeedback procedure. No information about the impact of this training on the frequency of tachycardia episodes in this patient was given.

A second patient with Wolff-Parkinson-White syndrome was studied in a similar fashion (Weiss, Brady, and Mac Farlane, in prep). She also showed the ability to modify her HR and conduction pathway in the laboratory, although effects in the latter variable were less dramatic because her aberrantly conducted beats were less frequent than the first patient's. However, this patient's biofeedback training had no demonstrable effect on the frequency of her tachycardia episodes, perhaps partly because she tended to deny her problem and so was unwilling to practice her biofeedback technique regularly at home as a preventive measure against tachycardia.

Supraventricular tachycardia. Three patients with ectopic atrial arrhythmias have been treated with HR biofeedback. Two had supraventricular tachycardias occurring intermittently and of several years duration (Engel and Bleecker, 1974). One patient was trained with both HR slowing and HR speeding to maximize voluntary control, while the second was trained in HR slowing. In both patients the ability to control HR was demonstrated in the laboratory and a striking clinical improvement occurred and persisted for several months of follow-up. One other atrial arrhythmia patient was trained with less success (Weiss, Brady, and Mac Farlane, in prep.). He had a five-year history of paroxysmal atrial flutter and fibrillation, requiring several medications to control it. A series of relaxation training sessions was unsuccessful in ameliorating his arrhythmia as was a trial of HR biofeedback.

Sinus tachycardia. Four patients with sinus tachycardia have been successfully treated with HR biofeedback aimed at decreasing HR (Weiss, Brady, and Mac Farlane, in prep.; Engel and Bleecker, 1974; Scott, Blanchard, Edmundson, et al, 1973). Amelioration of the arrhythmia both inside and outside the laboratory for an extended period of time was reported for some of these patients.

Atrial fibrillation. Bleecker and Engel (1973) trained one group of six patients with chronic atrial fibrillation in slowing and speeding their ventricular HRs. All patients successfully learned to produce sequential increases and decreases in this variable, and pharmacological studies indicated that alterations in vagal nerve tone to the atrio-ventricular node mediated these effects. No clinical changes in the patients were described. One patient with paroxysmal atrial fibrilla-

tion had an intensive period of several hours of HR slowing biofeedback in an effort to interrupt an episode of fibrillation. This was not successful (Gottlieb and Engel, in prep.).

Complete heart block. Three patients with this arrhythmia of complete atrio-ventricular heart block (also called third degree heart block) were given HR biofeedback training to increase their ventricular HRs (Weiss and Engel, 1975). None of the patients learned this successfully. Since ventricular HR is little influenced by neural effects, the authors concluded that this was the likely reason for the learning failure.

Raynaud's Disease

Three studies have described improvements in patients with Raynaud's disease who received either temperature biofeedback or blood-volume biofeedback from the affected area (Jacobsen, Hackett, Surman, et al, 1973; Surwit, 1973; Schwartz, 1973). In the first study the patient also received suggestions for relaxation (Jacobsen, et al, 1973). The patients generally were able to demonstrate several-degree Centigrade increases in digit temperature in the laboratory. The relative contributions of feedback and relaxation were evaluated in two studies (Surwit, Pilon, and Tenton, 1978; Jacobson, Manschreck, and Silverberg, 1979). Both found that finger temperature biofeedback added nothing to the efficacy of autogenic (Surwit, et al, 1978) or relaxation (Jacobson, et al, 1979) therapy in producing increased finger temperature and an amelioration of clinical state. Clinical benefits persisted in seven of 11 patients contacted two years later (Jacobson, et al, 1979) and appeared to depend on continued regular relaxation practice. This finding of continuing benefit depending on continuing practice of relaxation has been commonly described in behavioral treatments of hypertensives (Luborsky and Greer, in prep.).

Finally, the promise of such behavioral treatment for Raynaud's disease patients is underscored by Stroebel and Glueck's (in press) report that approximately 80 percent of the patients that they treated with a combination of biofeedback, psychotherapy, and relaxation showed significant improvement at two-year follow-up.

It must be noted that here, as in most other areas described in this section, no control groups are included in the studies. Furthermore, there has been no assessment of the effect of these procedures on patients' clinical status by observers blind to the experimental inter-

vention. Studies with such control groups and with blind observers are necessary for more definitive assessment of the value of these procedures.

Gastrointestinal Dysfunctions

Fecal incontinence. One of the areas in which biofeedback has become well accepted as a treatment modality is in the amelioration of fecal incontinence. This advance followed the demonstration by Engel, Nikoomanesh, and Schuster (1974) of the applicability of feedback training to such patients. Using a recording device which permits rectal distension by a balloon—simulating a fecal bolus—and the monitoring of both internal and external anal sphincter tone in response to this stimulus, they were able to train six of seven patients with several-year histories of severe fecal incontinence to improve their continence significantly. The method involved initially having the patients watch the monitoring described above on a polygraph after they had been instructed in the appearance of the normal sequence of events in the continent individual, i.e., rectal distention followed by internal sphincter relaxation, but with external sphincter contraction. Thereafter, the patients practiced producing external sphincter contractions in response to rectal distentions by the balloon. After they successfully mastered this maneuver, they were gradually weaned from reliance on the biofeedback by covering the polygraphic tracing. After treatment four of the six patients were completely continent during follow-up periods of six months or more. Two of the patients were significantly improved, although not completely continent. In a second report, Cervulli, Nikoomanesh, and Schuster (1976) studied 40 such patients. Follow-ups of four months or longer showed that 70 percent of patients treated in this manner had at least a 90 percent reduction in episodes of incontinence. Thus, biofeedback of this type—in this case the training of an elicited, rather than a spontaneously occurring, response of the skeletal muscle anal sphincter—is the best therapy for the problem.

Diarrhea. Furman (1973) studied five patients with so-called functional diarrhea. These patients had between four and 15 bowel movements per day, usually preceded by abdominal cramps. Emotional stress reportedly worsened the problem. In this open exploratory study, patients were trained with an electronic stethoscope (connected to a speaker) on their abdomen. They were trained both to

increase and decrease bowel sounds activity. Within five sessions, all patients reportedly showed some ability to control their bowel sounds using the auditory biofeedback. Furthermore, all patients also showed clinical improvement in their symptom, with considerable normalization of bowel function. The dependence of the clinical improvement on the bowel sounds control appears equivocal, however, as improvement preceded control of bowel sounds in some patients; nor was improvement dependent on the degree of bowel sounds control the patient achieved. This interesting study deserves replication, controlling for nonspecific factors which may have been operating, and attempting to define the importance of biofeedback in the patients' improvement.

Vomiting. An impressive case study in the biofeedback treatment of chronic, ruminative vomiting in an infant was carried out as a last-resort therapeutic measure (Land and Melamed, 1969). The nine-month-old baby was quite malnourished as a result of the vomiting, despite a variety of therapeutic efforts. In this extreme situation it was decided to use aversive conditioning in an effort to stop the vomiting. The infant's vomiting was monitored by electromyographic (EMG) recordings and by watching the child. At the earliest sign of the infant's initiating vomiting, electric shocks were administered to his leg until the vomiting stopped. After a few repetitions of this sequence, the child stopped vomiting, and in a short time was discharged in considerably improved nutritional state. He continued to improve, thereafter achieving normal nutritional status. This is one of the rare human biofeedback studies which has used shock as a negatively reinforcing stimulus. Its dramatic circumstances and successful outcome are noteworthy. The intervention in a pediatric setting, to ameliorate a developmental problem, and the child's subsequent normative development, also are instructive.

Peptic Ulcer. While no successful attempt to ameliorate the clinical state of duodenal ulcer patients has been reported, two studies have shown that individuals can exert some voluntary control over gastric acid secretion when given feedback relevant to this variable (Welgan, 1974; Whitehead, Renault, and Goldiamond, 1975). In one study (Welgan, 1974), for example, ten patients with (active or inactive) duodenal ulcers were trained over two sessions to reduce gastric acid secretion by means of continuous feedback of the pH of their aspirated gastric fluid. Significant reductions in gastric acid secretion were obtained, and control procedures suggested that these were a result of the biofeedback. No follow-up effects of this training on the patients were reported.

Miscellaneous Applications

Stuttering. Biofeedback has been applied to the amelioration of speech dysfluency. Lanyon, Barrington, and Newman (1976) taught eight stutterers to relax their masseter muscles by means of EMG biofeedback. Subsequent practice in reading with and without this biofeedback showed that the patients read significantly better in each case with the biofeedback. While the study did not control for adaptation effects to the procedure, the authors argue that the levels of stuttering achieved by all of their patients during biofeedback are much lower than those occurring by adaptational effects in other studies. The positive influence of expectancy of benefit from the new procedure was also not controlled for, but this study merits attempts at replication with appropriate control conditions. In a second study, one patient was given EMG biofeedback from the neck areas lateral to the larynx (Hanna, Wilfling, and McNeill, 1975). The patient reduced his stuttering during biofeedback as compared to the no-feedback or false-feedback conditions. In a third study, an elderly patient with speech dysfluency appeared to benefit similarly from masseter muscle EMG biofeedback (Weiss, Carson, and Brady, 1979). However, her improvement was shown to be related to the opportunity to speak regularly with an interested listener rather than specifically to result from the biofeedback practice.

Asthma. The use of biofeedback techniques in asthma has been evaluated in a preliminary way in two studies. Each fed back to the patients an index of airway resistance and used this to train the patients to reduce this parameter of pulmonary function. Auditory (Feldman, 1976) or visual (Vachon and Rich, 1976) feedback was used. In each study the patients were able to produce significant decreases in total respiratory resistance. Vachon and Rich (1976) employed a control procedure in which another group of patients were given reinforcement signals which were unrelated to their airway resistance. No decrease was seen, suggesting that the reduced respiratory resistance was indeed secondary to the biofeedback. No information regarding changes in the patients' clinical states was presented, and it remains to be seen if biofeedback is of clinical value in the treatment of asthma.

Urological applications. Wear, Wear, and Cleeland (1979) recently described an application of biofeedback in improving urinary sphincter control. A group of eight patients, most with urinary incontinence or retention, was given EMG feedback from the periurethral voluntary muscles by means of urethral catheter. Four of the patients showed a moderate or marked clinical benefit from the training.

Rehabilitation medicine. Another medical area in which biofeedback is in wide use is rehabilitation medicine. This therapeutic application relies on EMG feedback. First employed by Marinacci and Horande (1960) and greatly developed by Basmajian (1974), biofeedback has been applied to the rehabilitation of a large variety of congenital and acquired central and peripheral nervous system disorders. This general area has been well and critically reviewed elsewhere (Inglis, Campbell, and Donald, 1976); only a brief overview will be presented here. The two main rehabilitative uses of biofeedback are the training to decrease skeletal muscle activity in overactive muscles (e.g., spasticity or dystonias like torticollis) and the training to increase muscle activity in underactive or atrophied muscles (e.g., following stroke or peripheral nerve injury). While large numbers of patients with a wide variety of such disorders have been treated, few controlled studies have been done. Two well-controlled studies confirm the impressions that: (1) patients with increased muscle tension following injury to an area—here the neck—can learn to reduce the increased muscle tension with EMG feedback from that site (Jacobs and Felton, 1969); and (2) that patients with muscle weakness following stroke can learn to increase strength in the affected muscles better with EMG biofeedback from the weak muscle as an adjunct to their standard physical therapy regimen than patients who had only physical therapy (Basmajian, Kukulka, Narayan, et al, 1975). Of particular interest to psychiatrists is the fact that a large number of patients with spasmodic torticollis have been treated with EMG biofeedback from the sternocleidomastoid muscles. Follow-up of three months or more indicated that 40 percent of the patients treated in this fashion maintained a significant benefit (Brudny, Korein, Grynbaum, et al, 1976). While further controlled studies are necessary in this area, biofeedback does seem an extremely promising therapeutic modality in rehabilitation medicine.

What Processes are Included in Medical Biofeedback?

(1) *Biofeedback as a skill learning procedure.* Medical biofeedback initially was conceived as a training procedure for imparting to patients skills which might ameliorate their illnesses. This is the perspective from which most of the preceding discussion has been given. In studies where patients learn both to increase and to decrease the level of an illness variable, this viewpoint seems appropriate. However, experience with biofeedback also has suggested that addi-

tional processes go on during this training (Weiss, 1977), and these will be discussed below.

(2) *Biofeedback as information for the patient.* While some patients with medical problems are aware of fluctuations in the level or intensity of their illnesses, e.g., patients with tension headaches, many others are not. Biofeedback changes this situation, making the patient aware of fluctuations in the level of his illness, e.g., BP, in a detailed way. The patient thus can learn, for example, what higher or lower BP feels like, and what effects on this variable occur with changes in mental and physical activity, locale, time of day or week, other individuals, and the like. This cataloguing of the effects of a variety of factors on the illness variable offers the patient an additional dimension which he will wish to consider in planning his priorities and activities.

(3) *Biofeedback as information for the physician.* Physicians are accustomed to using the monitoring of bodily processes in their patients as informational feedback in order to optimize their treatment programs. Electrocardiograms, x-rays, and the evaluation of blood chemistries are examples of this. Recently, there has been an increasing trend to home-monitoring of such variables as well, including cardiac arrhythmias (Pickering, 1976) and hypertension (Julius, Ellis, Pascual, et al, 1974). Biofeedback also provides the therapist with a great deal of information about relationships between the patient's illness and his activities, etc. as discussed above. This is particularly useful in psychosomatic medicine, encouraging therapists to look for physiological indices of illness processes—as with frontalis EMG activity in tension headache patients—and to focus on such indices as objective criteria of therapeutic gain. In this sense especially, biofeedback is part of the growing movement toward behavioral medicine (Birk, 1973; Williams and Gentry, 1977).

Also, biofeedback often provides the therapist more precise information about change in a patient's functioning than would otherwise be available (Woodridge and Russell, 1976). Recording the amount of time a patient meets a given standard for HR control or the level of EMG activity from a muscle under training are examples of such exact information available from biofeedback devices. This information can be useful to the physician in evaluating the patient's progress.

(4) *Biofeedback as an opportunity for relaxation.* Many biofeedback studies have involved protracted training over many sessions, each lasting a half hour or more, with the patient sitting alone in a room attending to repetitive stimuli. This situation includes several features which have been described as appropriate for eliciting relaxation (Benson, Rosner, Marzetta, et al, 1974). While relaxation is discussed

at length elsewhere in this report, its participation in some of the effects attributed to biofeedback also is noted here.

(5) *Biofeedback as a placebo.* As a relatively new treatment, biofeedback is likely to involve important placebo effects. New therapies typically evoke positive expectations from patients and therapists which can be beneficial in themselves. However, they are not at all integral to the new therapy. Biofeedback's emphasis on complicated and impressive equipment, protracted treatment, and a medical setting all may positively influence the outcome, independent of the biofeedback itself. As studies with appropriate control groups are done (e.g., Patel and North, 1975; Haynes, Griffin, Mooney, et al, 1975) and as the limitations of biofeedback become more clearly defined and more widely disseminated, the placebo effects should wane.

Biofeedback as a Research Tool

Another aspect of the medical uses of biofeedback is its application to the increased understanding of the pathophysiology of illness. If one can train a patient voluntarily to increase and decrease the frequency of an arrhythmia or the level of his BP, an excellent opportunity is provided to study the mechanisms by which the symptom is brought under control. Interventions with autonomic nervous system blocking agents have been one such approach. The results of these studies with arrhythmia patients, for example, led in one case (Weiss and Engel, 1971) to the inference that increased vagal nerve activity to the heart was capable of suppressing PVCs in some patients, an hypothesis that was subsequently supported by several other studies (Waxman, Downer, Berman, et al, 1974; Weiss, Lattin, and Engelman, 1975; Waxman and Wald, 1977).

Conclusion

It seems clear that the biofeedback treatment package can produce beneficial effects in several illness states—hypertension, hypotension, some cardiac arrhythmias, Raynaud's disease, stuttering, urinary and fecal incontinence, and in rehabilitation medicine. It also seems clear that relaxation effects, placebo factors, and increased compliance with the medication regimen may participate in these effects. Most convincing is the evidence for biofeedback's unique therapeutic efficacy in the training of skeletal muscle responses—for example, in sphincter control for fecal and urinary incontinence, and in rehabilitation medicine. Biofeedback is not limited to training for hypoarousal.

This is illustrated by successful treatment of hypotension and of disorders involving underactivity of skeletal musculature—e.g., in rehabilitation medicine.

Two major questions remain unanswered. First, long-term adherence to the practice of biofeedback or relaxation/meditative techniques is necessary to produce sustained improvement in chronic clinical problems such as hypertension or headaches. It remains to be established whether biofeedback, relaxation or meditative practice will lead to better adherence long-term. Second is a corollary question, namely whether certain personality types will have better results with one technique or another. One might assume that more quantitatively-oriented or more skeptical individuals would do better with biofeedback, while more imagery-oriented or religious individuals would do better with meditation. The answers to these questions are likely to be forthcoming from research conducted in the next few years.

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Chapter VII

SOME GENERAL ISSUES CONCERNING BIOFEEDBACK THERAPY

Biofeedback is an interesting and novel concept which involves enabling individuals to acquire the ability to control some of their own basic physiological processes. This requires making available the necessary information, the prerequisite motivation, and the existence of some neural mechanism by which cortical control can be exercised. Work in biofeedback has helped to clarify the extent to which it is possible to control physiological parameters previously assumed to be outside of an individual's volitional control. Further, biofeedback has served as an effective research tool to assess not only what is humanly possible but also to help establish how control is mediated.

As far as psychiatric treatment is concerned, biofeedback therapy has not been found useful with the more severe psychiatric disorders. It has been effective as an adjunct to psychiatric treatment to help control tension and concurrent anxiety as well as to treat specific psychosomatic complaints. Though clinical reports vary in their enthusiasm, they do not provide compelling evidence of specific effects in this area. The few studies that are available indicate that hypnotic therapy, meditation, or relaxation training are equally effective. It is clear that biofeedback therapy, with rare exceptions, cannot be considered a powerful specific therapy analogous to those treatments of biological psychiatry which, while affected by psychological factors, have direct effects on some aspect of the nervous system. Nonetheless, because biofeedback involved advanced technology, complex equipment, and facilitates precise measurement of physiological responses, both the public and the medical profession have tended to characterize it as different from psychological therapies. This assumption is not justified by the available evidence.

It would be foolhardy, however, to dismiss the dramatic effectiveness of biofeedback therapy for some psychobiological disorders. Though far and away the most extensive use of biofeedback has been in the treatment of stress related disorders where the technology is used to train the patient to induce a state of low arousal, there are some circumstances where biofeedback involves training the patient to maintain an increased muscle tone in specific ways. A good example

is the treatment of orthostatic hypotension following spinal cord injury (Bucker and Ince, 1977), referred to in the section by Weiss. Systematic training allows the patient to maintain his blood pressure. Again, treatment of fecal and urinary incontinence with biofeedback involves teaching the patient to maintain a high tonic level of activity in the relevant sphincters. In these applications as well as in neuromuscular rehabilitation, biofeedback appears to be a specific therapy but, with the possible exception of the biofeedback treatment of epilepsy, we know of no such specific biofeedback therapy directly relevant to the treatment of psychiatric disorders.

The most promising use of biofeedback relevant to psychiatry and psychosomatic medicine is in the control of stress related difficulties. There is considerable evidence, discussed earlier, that biofeedback therapy is effective in the treatment of migraine, muscle tension headache, some psychophysiological disorders, particularly stress related problems. As we have emphasized, in actual treatment biofeedback itself forms only one component of a treatment package designed to produce profound physical and mental relaxation. Biofeedback training in the office is generally not effective unless patients can be induced to practice the skill on a regular basis outside of the office in their natural environment. When patients systematically practice maintaining a state of hypoarousal, results tend to be far more satisfactory. However, the treatment procedure then shares many important attributes with other forms of relaxation training, self-hypnosis, meditation exercises, and the like. All these therapies inevitably include a certain amount of attention by the therapist, the inculcation of positive expectancies, some skill training, encouragement, facilitated by perceptible changes defined as related to improvement, and so on. Perhaps most important, all of these procedures require the patient to interrupt a busy schedule to carry out the exercises at various points during the day and thereby serve to interrupt the progressive stressing of the individual due to the mounting pressures of the day.

It is worth recalling that the therapeutic effects of relaxation therapy were already recognized by the Greeks, and some version of relaxation training can be found in virtually all cultures, suggesting that these tap a very basic and important health facilitating function. Though in most instances various procedures to induce hypoarousal—biofeedback, self-hypnosis, meditation, relaxation training—yield similar results, it would be unfortunate to ignore the dramatic effect some of these simple and safe procedures are able to exert on the patient's well being. Perhaps it is less important whether biofeedback provides a unique form of treatment than that some effective technology of trained hypoarousal be made available on a broad scale. It

would seem surprising indeed if the judicious use of biofeedback could not help some patients to better learn the skill of gaining control over their own level of arousal. Thus, while therapeutic results are usually documented on the basis of subjective reports, biofeedback can and should be useful as a means of helping to assess therapeutic outcome in the treatment of stress related conditions for both patient and therapist. Recognizing that biofeedback does not provide a uniquely different result in the treatment of stress, research should be addressed to assessing which combination of techniques will be most effective and most efficient for most patients. Further, it would be important to consider some of the possible moderator variables—for example, individual differences in hypnotizability are likely to affect which treatment approach will be most appropriate for a particular patient. Only after every effort has been made to develop the best available treatment package would it again become worthwhile to explore the relative importance of the various components that are involved in that package.

Similarly, even when competent psychotherapists have begun to work with hypoarousal training, they have rarely considered these procedures in the context of a psychotherapeutic process. With some notable exceptions (e.g., Rickles, 1976), the psychotherapist changes roles, so to speak, to become either biofeedback expert, hypnotist, or guru, clearly distinguishing between his activities in these areas and his role as a psychotherapist. There has also been little effort to integrate biofeedback with other forms of relaxation training to induce hypoarousal (the work of Stoyva and Budzynski, 1974, is a notable exception), and to integrate these into an overall psychotherapeutic approach.

In a careful review of the field, it has become increasingly clear that biofeedback therapy is not simply a matter of using complex electronic technology to provide information to the patient and therapist. It is by no means sufficient to employ sophisticated equipment capable of identifying the appropriate physiological responses. Rather, it makes a great deal of difference how the procedure is presented to the patient; the way in which the patient is helped to understand his role in the treatment procedure; how he is motivated to apply himself to the task of learning volitional control; the degree to which he can be persuaded, cajoled, and motivated to practice the acquired skills in real life situations; and so on. Certainly there is no independent science or profession of biofeedback therapist. Rather, it is a technique to be mastered by trained health professionals to be utilized in the professional's specific area of competence.

The Task Force recognizes that a physician interested in using

biofeedback therapy will need to acquire the technical skills of operating the equipment and an understanding of its capabilities. As important as these skills are in actual practice, it is no less important to have an appreciation of the pathology to be treated and an understanding of the psychobiological problems involved in the therapy of physiological disorders. While lay individuals may readily master the technical skill involved in using biofeedback equipment, the assumption that they are therefore competent to treat medical and psychological disorders is incorrect. Treatment by biofeedback therapy should be conducted in an overall therapeutic context by a highly trained health professional who is qualified to treat the problem in question equally well by means other than biofeedback.

For these reasons, the Task Force is opposed to the credentialing of biofeedback therapists as independent professionals. While a technician may readily be taught the use of the equipment, there is ample evidence that merely exposing an individual to biofeedback does not constitute effective treatment,* and therefore biofeedback should be employed by psychiatrists, psychologists, or medical practitioners familiar with the technology in their own special area of competence.

We would hope that psychiatric understanding of a large number of stress related disorders will, on the long run, benefit greatly from the development of biofeedback technology and its integration into the treatment process. Thus, some sophisticated clinical reports describing the effect of biofeedback therapy emphasize the vital role psychological factors play in determining whether a patient will continue with the treatment program, whether he is able to transfer his learning from the laboratory to life situations, and, finally, whether he will continue to practice the skill of controlling his physiology after he has learned to do so successfully. It would stand to reason that appropriate psychotherapeutic treatment would help many patients to utilize the skills that they might have learned through biofeedback or relaxation training. A patient may find it easy to reject a therapist's interpretation of his tension headache as expressing masked rage because he experiences the headache as happening to him and beyond any control. It becomes quite a different matter, however, when a

*Just as a roentgenologist employs x-ray technicians, it may be appropriate for psychiatrists or psychologists to employ a technician to carry out some aspects of biofeedback therapy. However, the technician is not an independent therapist; just as it would be inappropriate for a physician who is not an expert in roentgenology to hire a technician and ask him to take and interpret films, it would similarly be inappropriate for a psychiatrist unfamiliar with this technology to employ or refer patients to a technician untrained and unlicensed in medicine or psychology. The key point always remains that biofeedback therapy is administered in a larger therapeutic context by an individual trained to understand the pathology and to assess the results of this treatment as opposed to other treatments which might be employed for the same condition.

patient has learned that it is possible for him to control his headache by carrying out relaxation exercises (whether the skill is taught by way of EMG feedback or relaxation training) yet he simply finds it "too difficult" to devote the few minutes a day necessary to maintain his skill. The latter instance involves not carrying out an action which is clearly within the patient's abilities. Consequently, it becomes far easier for the patient to accept responsibility for what has now become his actions and to begin the process of understanding and thereby modify the role headache plays in his psychic economy.

Similarly, the judicious combination of EMG and temperature feedback training with other techniques of relaxation therapy in a systematic fashion should be encouraged. While one is tempted to interpret the studies showing no difference in the effect of biofeedback therapy versus practice with simple self-hypnosis formulas to indicate that there is little justification for expensive equipment, such a view ignores the fact that therapists vary greatly in their ability to teach relaxation. Even if the mechanisms were identical—an assumption which has not been fully documented—some patients find it far more comfortable to use biofeedback as the method for learning to relax, while others find it easier to relax while being taught self-hypnosis. In any case, the biofeedback instrumentation can provide useful information for both patient and therapist concerning the patient's gradual mastery of the relevant relaxation skills. By the same token, some form of relaxation exercise is probably the easiest way to teach individuals to practice the self-control of arousal without biofeedback equipment. Thus, an integrated approach should ultimately allow a larger percentage of patients to benefit from self-induced low arousal, it should also facilitate the objective assessment of the patient's progress, and it should effectively serve to bridge the gap between psychotherapy and techniques more directly designed to modify the stress response.

Conclusions

A careful review of existing clinical studies, experimental research, and clinical reports led the Task Force to the conclusion that biofeedback therapy should not be contemplated as a separate psychiatric treatment. There is also no psychiatric condition for which biofeedback as such is the treatment of choice. However, clinical data indicates that a number of psychophysiological disorders respond well to biofeedback therapy. Unfortunately, little is known about long-term

consequences of biofeedback treatment, and the excellent clinical response to biofeedback is paralleled by equally good therapeutic effects following several different kinds of relaxation training. Until more evidence is available, particularly involving long-term follow-up, no single form of biofeedback therapy can be endorsed as a specific treatment for any of the psychophysiological disorders at the present time.

The technology of biofeedback is reasonably well developed and safe. Nonetheless, it should be applied by individuals intimately familiar with both the psychological and physiological aspects of the procedure. It is particularly important that psychiatrists or other physicians contemplating the use of this technology have a clear appreciation of how and why it works, since only then does it become possible to integrate biofeedback with other therapeutic modalities.

It may help to consider the analogy to psychopharmacology. Some 25 years ago there were psychiatrists who used psychotropic agents and others who used psychotherapy, whereas today no clinical psychiatrist would fail to recognize the importance of both. As a profession we have learned to integrate the therapeutic modalities and use them synergistically. For biofeedback to play a significant role in psychosomatic medicine, it will be necessary for a similar kind of synthesis to occur. The technique of biofeedback will need to be integrated into the overall therapeutic approach. Only then are we likely to see the kind of longterm results reported by those few individuals who appear to have achieved a creative synthesis in the manner in which the techniques are combined.

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