Electromyographic biofeedback as a possible adjunct to the treatment of schizophrenic patients

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ELECTROMYOGRAPHIC BIOFEEDBACK

AS A POSSIBLE ADJUNCT TO

THE TREATMENT OF

SCHIZOPHRENIC PATIENTS

by

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Abstract

The symptomatology of schizophrenia can be understood partially as the result of autonomic nervous system hyperarousal. Electromyographic biofeedback was evaluated for its ability to reduce somatic arousal and thus schizophrenic symptoms. Twenty acute schizophrenic inpatients received relaxation instructions at the beginning of six, one-hour sessions (one baseline and five training sessions). During the training sessions, experimental patients received biofeedback signals from the forearm extensor and frontalis muscles for ten, one-minute intervals each. Control patients listened to calming music for a similar amount of time while the same muscles were monitored. Pre- and post-extensor muscle tension values demonstrated significant relaxation as a result of biofeedback induction. Psychopathology, as measured by MMPI scales 7,8, and 0, was shown to have been reduced at the same time. The relationships between muscle tension and various schizophrenic symptomatology measures were further examined; and several strong correlations, as well as a striking "inverted U" arousal-pathology curve, were found.

Introduction

Historical Review of Tension in Schizophrenia

From the earliest observations of the disorder called schizo-
phrenia, it has been noted that individuals suffering from the disorder
display an interruption of normal muscular function. For instance,
Freud spoke of the bound drives of the disorder, implying a neuromus-
cular dynamic, and Kraeplin noted that schizophrenics display what he
termed "writing imbalance" (Wulfeck, 1941). In early studies, simple
observation of posture, facial musculature, and affective flattening
produced the impression that the muscle activity of the schizophrenic
patient was reduced along with a generalized hyporeactivity (Angyal,
Freeman, and Hoskins, 1939). More exacting studies, such as the one
conducted by May (1948), measured pupillary response in patients and
also found reduced reactivity when compared to normals. Other early
studies, which relied upon mechanico-observational measures such as
eyeblink, knee-reflex, finger-tapping, and reaction time, reported sim-
ilar results, all indicating muscular hypotonicity in schizophrenia.
(For a complete review of early studies see Wulfeck, 1941 and Hoskins,
1944.)

The introduction of electromyographic techniques in the late
1930's allowed later researchers to measure muscle tension directly
through electrical monitoring of the action potentials produced by the
enervation of cells in muscle groups. Steger and Schaltenbrand (1940,
cited in Petursson, 1962) first used electromyography with catatonic
schizophrenics and discovered muscular hyperactivity (rather than hypo-
tonia) especially around the forehead, jaws, and neck. They were able

to relate this hypertonicity to the clinical picture of "waxy flexibility," which is the hallmark of the catatonic subtype of schizophrenia. Malmo and his associates performed a series of electromyographic studies from 1949 to 1952 (Malmo and Shagrass, 1949) and discovered that after experiencing a stressing event in the laboratory (e.g., a sudden loud noise), schizophrenics exhibit a short period of gross hyperactivity, whereas normal subjects do not exhibit the same response. They found the tension levels of the schizophrenic subjects to be similar to those of normals. Malmo, Shagrass, and Smith (1951) also found that related measures of autonomic nervous system arousal increased in an abnormal manner under stress. In a 1955 study, they found that frontalis muscle tension significantly differentiated between patients and normal controls, even at rest. Jurko, Jost, and Hill (1952), while not specifically investigating muscle tonicity, confirmed that several measures of arousal were abnormally elevated in schizophrenia. Gunderson (1953) found that muscular hypotonicity in schizophrenics accompanied arousal on 12 measures of sympathetic nervous system functioning. Ehrentheil (1962) attempted to evaluate the course of muscular tension in schizophrenic patients over a 30-year period by examining records of long-term patients, both those who had been hospitalized continuously and those who had been outpatients at least occasionally. While he admitted that the records he used were not ideally suited to conveying information about tonicity, Ehrentheil tentatively described the course of the muscular abnormality in these patients as a gradual reduction in tonicity over the 30 years after an initial marked elevation. In this description, one can discern the clinical picture of the "burned-out" chronic schizophrenic often seen in the long-term hospitalized patients (McDonald and Gynther, 1962). Whatmore and Ellis (1964) monitored the

tension of schizophrenic patients throughout their treatment including outpatient treatment and they observed an increased lability of muscle tension in the patients as well as a continuation of marked hypertonicity after their release from the hospital and the withdrawal of chlorpromazine. One intriguing finding was that a marked increase in muscular tension preceded a relapse among these outpatients. Goldstein (1964) performed a comprehensive examination of hypertonicity among emotionally disturbed patients using six muscle sites, including five measures of automatic arousal (i.e., galvanic skin response (GSR), both systolic and diastolic blood pressure, respiration, and heart rate). The frontalis and forearm extensor muscle sites of psychotic patients were found to be significantly more tense than normal controls under both resting and stress conditions. Using a standardized physical examination technique, Cheney (1969) palpitated muscles of psychiatric patients until they expressed pain. He considered the various levels at which pain was experienced to reflect degrees of muscle tension. Although this approach demonstrated that psychiatric patients in general maintain a higher level of muscle tension, his method was unable to differentiate specific diagnoses. Widmer (1976) found that, not only are schizophrenics characterized by increased muscular tonicity, but they are significantly less able to determine subjectively the level of their tension.

Before ending this section of the literature review, it is important to point out that only one of the studies since 1960 mentions the effects of major tranquilizers on muscle tonicity (Gellhorn and Loufbourrow, 1963), and this study reports that tonicity persisted after withdrawal of medication. Malmo (1970), in a review of his studies in the early 1950's, stated that the introduction of psychoactive drugs

would prevent further study of muscular tension in schizophrenics, pre-
sumably because such powerful tranquilizers would reduce tension to
normal levels or below. The work of Gellhorn and Loufbourrow, however,
provides evidence that Malmo's assumption was incorrect. Two other
studies, those of Lavallee (1977) and Nigl and Jackson (1979), also
demonstrated that, despite administration of narcoleptic drugs, schizo-
phrenic patients continued to exhibit abnormal hypertonicity.

Physiological Arousal in Schizophrenia

To make explicit what was sometimes only implicit in the foregoing
review of the research on muscular tonicity, hypertonicity can be con-
sidered to be indicative of a generalized arousal of the sympathetic
nervous system. Several of the investigations mentioned above (Goldstein,
1965; Gunderson, 1953; Jurko, Jost, & Hill, 1952; Malmo, Shagrass, &
Smith, 1951) reviewed several measures of physiologic arousal, including
tonicity, and concluded that schizophrenics display an abnormal degree of
physiologic arousal. A review by Tecce and Cole (1972) reported that, of
36 studies using schizophrenic subjects, 19 demonstrated that physiologic
measures were elevated, 9 showed hypoarousal, and 7 showed no difference
between groups. The studies in which the results did not conform to this
pattern used primarily G.S.R. and blood pressure.

Brill (1978) reviewed several articles which reported that physio-
logical arousal occurred in schizophrenics. His article focused on the
neurological mechanisms which could generate a condition such as schizo-
phrenic arousal and on the evidence to support these theoretical inter-
pretations. For example, it is commonly known that strong tranquilizers
are required to reduce florid schizophrenic symptomatology therapeutic-
ally. As Brill says,

> Catatonic schizophrenic patients may sit up and talk
> coherently after an intravenous injection of sodium
> amytal or pentothal in amounts that would promptly
> put a normal individual to sleep. As a group they
> are able to tolerate much larger amounts of pheno -
> thiazines and other tranquilizers than are normal
> people or other kinds of patients (p.666).

The same phenothiazine tranquilizers that control symptomatology also produce lower skin conductance (McDonald & Gynther, 1962; Spohn Thetford, & Cancro, 1971), which is another measure of somatic arousal. Thus tranquilizers, by reducing arousal (possibly a primary effect), control psychologic disorder (possibly the secondary effect). These studies show that the hyperarousal of schizophrenics is affected by the tranquilizing drugs in some manner at the neurophysiologic level.

In view of the evidence that hyperarousal is an integral part of the schizophrenic syndrome, it has been theorized that much of the symptomatology of this disorder can be explained as a disruption of the systemic arousal system (Storms & Broen 1969; Malmo, 1959; Tecce & Cole, 1972; Whatmore & Ellis, 1964). The following studies explore ways in which schizophrenic symptoms might be produced by physiologic hyper-arousal. Plutick, Wasserman, and Meyer (1975) have pointed out that a high degree of muscular tension would act to mask the facial depiction of emotion thus leading to flattening of affect. Auditory hallucinations have been found by Gould (1950) to be related directly to subvocal muscular contraction in speech areas. These contractions could be the result of nervous system arousal. Under normal conditions, the neuro-

logical impulses would be generated in the CNS, but inhibitory motor neurons or other neurological mechanisms would dampen or prevent any muscular actions. Hyperarousal would increase the normal impulses to a level that would overcome the normal inhibitions. Storms and Broen (1969) have provided an excellent and extensive review and demonstrated that associational and cognitive deficits found in schizophrenics could be associated with an arousal-attentional disturbance. Schacter (1962) performed a classic experiment in which normal subjects received a dosage of epinephrine, a sympathetic nervous system stimulant, without being told what effects to expect. After the time required for the drug to take effect, a confederate entered the room. In response to the task presented a written test, the confederate displayed either hostility (aggression) or playfulness (non-aggression). Subjects were found to model their behavior on the confederate's, but their behavior was extreme in both directions due to the stimulant they had unknowingly ingested. An observer, who was unaware of the experimental manipulation performed, described the subject's behavior as "inappropriate." The behavior displayed was very similar to the "inapropriate behavior" described in schizophrenia.

DSM III lists perceptual abnormalities such as sensation of bodily change and hypersensitivity to sound, sight, and smell as symptomatic of the schizophrenic disorder. Intuitively, it seems likely that these parasthesias could be closely related to the physiologic arousal of the individuals experiencing them.

Neurophysiological Basis of Arousal

If one accepts the hypothesis that a physiologic state of arousal accompanies the symptomatology of schizophrenia, one is not compelled to

accept the view that an underlying neurologic mechanism causes the biochemical disorders associated with the disease. This is only one possible theoretical formulation. The hypothesis that the disorder results from a physiologically pathological nervous system is an attractive one, however, because much evidence points to genetic predisposition and stress response factors in the disease. In response to almost any stimulus, the physiologically predisposed individual would display an abnormally high degree of somatic reaction which would appear in many somatic systems, including the musculo-skeletal system, and would produce even greater sympathetic arousal (Malmo, 1975). The acute schizophrenic episode would be seen as a "snowballing" of these mutually excitatory processes. Several theorists, however, have pointed out that the arousal found among schizophrenics might not be the result of neurologic anomalies, but might instead reflect some type of physiologic or psychologic "imbalance" or a combination of both (Stoyva & Budzinski, 1979). An imbalance of this kind would resemble that found in psychosomatic illness, a condition in which the function of an organ is disturbed because of emotional reactivity.

The earliest theorist to propose the "imbalance" formulation was Jacobsen. His early work "Progressive Relaxation" (1938) is now considered a classic in the biofeedback field even though it did not have much influence on the field for years after its publication. He wrote:

Wherever there is psychic disturbance trained observation
will reveal corresponding signs of neuromuscular hyper-
activity or hypoactivity ... which occur in the absence
of organ changes ... In these are found all degrees of
restless behavior or rigid posture of the individual. It
is generally agreed that prolonged worry and anxiety

often cause nervous disorder and likewise emotional

shocks, trauma, and maladjustment to environment (p.67).
Arieti (1956) tied worry and anxiety more closely to a neurologic
substrate when he postulated that a schizophrenic individual, in
attempting to escape from overwhelming anxiety,

avoids certain complicated psychological functions such

as interpersonal relations, certain types of symbolism

and planning which are predominantly mediated in the

cortical areas highest in the evolutionary scale, i.e.,

the most recently developed. These areas develop a

psychogenic hypofunction, and there is an attempt on

the part of the organism to return to a lower level of

integration and to a more primitive type of behavior.

This in turn further complicates the problems of ad -

justment and leads to further disequilibrium (p.326).

According to Brill (1978), Arieti located in the cerebral cortex
the neurologic mechanism that allows the rhinencephalon to become over-
active and to increase its inhibitory effect upon the hypothalamus. In
this way it would lead to generalized arousal. Whatmore and Ellis (1964)
and Whatmore and Kohli (1968) concentrated even more directly on neuro-
physiologic constructs and coined the terms "hyperponesis" and "hypopon-
esis" to describe the states of abnormal energy utilization. They stated
that this kind of imbalance is an etiological factor in many diseases.
In schizophrenia, they theorized, the posterior hypothalamus (responsible
for higher autonomic control) would be excited through afferent impulses
such as proprioceptive impulses from muscle groups. Excitation of the
thalamic region would produce increased arousal, resulting in a kind of
dysfunctional feedback loop by which the resulting increased muscle

tension would stimulate more proprioceptive impulses and thus produce even greater excitation. Brill has extensively reviewed research which would support this theoretical stance, including EEG and other medical research.

Biofeedback Treatment of Arousal

The description of schizophrenia as a psychophysiologic disorder which involves, in some manner, neuromuscular hypertension as well as the classical psychological symptomatology seems very appealing. For example, it provides a psychophysiologic rationale for the efficacy of the phenothiazine drugs which have been shown to act upon receptor sites in certain areas of the brain (Carlsson, 1974) and at the same time diminish the cognitive deficits so apparent in the disease. Also, it suggests that a holistic, mind-body approach could better address all facets of the disorder. Relaxation training, specifically electromyographic biofeedback-induced relaxation, could possibly be a fruitful approach to treatment of the disorder. One must be cautious, however, because this technique is new; and though promising, it lacks extensive experimental validation. In addition, biofeedback, like so many other new and dramatic methods that have come onto the scene, can be seen by professionals and patients alike as a "cure-all" for schizophrenia. However, if schizophrenia shares a physiologic substructure with other disorders, which have been successfully treated with biofeedback, e.g., hypertension, Renaud's syndrome, tension headache (Whatmore & Kohli, 1968), then it too may respond to biofeedback techniques.

If biofeedback were an epiphenomenal "cure-all" technique, no scientifically measurable results would be found. However, evidence reviewed above indicates that changes have been discovered following

biofeedback training. Still, it is advisable for an experimenter to be conservative in hypothesis formulation because biofeedback might induce measurable change at either the physiologic or psychologic level. The experimenter should consider whether the direct cause of the changes produced would be psychological (e.g., learning, desensitization, anxiety reduction), physiological (e.g., hypothalmic inhibition, pyramidal interactions) or both. At the neurological level of analysis, it is somewhat difficult to describe the rationale for the effectiveness of relaxation treatments. The field of neurophysiology has not advanced far enough to allow the identification of specific cortical and subcortical structures through which muscle relaxation mediates reduced autonomic arousal. According to Whatmore and Kohli (1968), experimentation has demonstrated that muscle effort, the opposite of muscle relaxation, influences autonomic activity in at least three neurological areas: 1) proprioceptive impulses act directly upon the hypothalamus to increase its excitability, 2) motor and premotor cortical neurons can affect the hypothalamus via basal ganglia and other direct pathways, and 3) pyramidal tract impulses can augment autonomic functions separately at the spinal level. Relaxation induced through biofeedback, then, could be assumed to have the opposite effect upon any or all of these three locations. Benson, Beary, and Carol (1974) coined the term "relaxation response" to describe the opposite of the "fight or flight" response, which is the sympathetic nervous system's reaction to danger. The relaxation response, they postulated, is mediated by the parasympathetic nervous system through integrated hypothalmic activity "in the area of the anterior hypothalamus" and extending "into the supra- and pre-optic areas, septum, and inferior lateral thalamus." The nervous system is so complex that it is, perhaps, impossible to attempt to describe

biofeedback's effects on specific brain structures. It is probably safe to conclude from the evidence reviewed that biofeedback does produce changes in muscle tension which in turn evidently produce some neuro-physiological changes.

It is also important to describe a psychological explanation for the efficacy of biofeedback mediated relaxation. This is an area which has been more extensively studied. In psychological theories of schizo-phrenia, the patient is often seen as having an inability to deal with overwhelming anxiety. There have been many studies in which biofeedback has been used as a tool to reduce anxiety. For object specific anx-ieties, (e.g., phobias) EMG biofeedback has been incorporated into Wolpe's desensitization procedures. In a number of recent studies non-specific anxiety has been treated with biofeedback with various levels of success (Canter, Condo, & Knot, 1975; Glueck, & Stroebel, 1975; Jessup, & Neufeld, 1977; Raskin, Johnson, & Rondesvedt, 1973; Townsend, House, & Addario, 1975). At a different level of analysis, biofeedback could be seen as a call to bodily awareness which would promote reality contact much as occupational therapy does. It might also act to lessen somatic delusions or unusual somatic concerns. Biofeedback can be thought to lead to internalization of the patient's locus of control and thus develop the patient's personal responsibility for his or her improvement.

The Present Study

Although there have been a considerable number of studies in which psychiatric patients have served as the subjects of biofeedback treat-ment (the above mentioned studies are just a partial listing), the specific disorder of schizophrenia and its treatment have been neglected largely by biofeedback researchers This may be due in part to Adler and

Adler's (1979) warning that relaxation training could precipitate an acute psychotic episode as a result of a decompression reaction. Only four studies were found in the literature that have mentioned biofeedback treatment for schizophrenics, and none found decompression among subjects. Lavalee et al. (1977) investigated the effects of the drug diazepam and EMG biofeedback and their combined use on the muscle tension of schizophrenics. They found additive results of the drug and EMG treatments initially, but over time, the EMG treatments were more lasting. Nigl and Jackson (1979) also investigated the interaction of drug treatment and biofeedback. They found that the phenothiazine drug they investigated had the capacity to potentiate much more rapid learning under the biofeedback condition. Acosta, Yamamoto, and Wilcox (1978) investigated EMG biofeedback relaxation learning in schizophrenic, neurotic, and tension headache patients. The results showed that schizophrenic patients were able to learn relaxation as well as the other groups. These studies demonstrate that schizophrenics, despite the symptomatology of their disorder, are able to learn to reduce muscle tension through biofeedback techniques. Only Staples (1978) has investigated the possibility of therapeutic benefits from relaxation training, and he obtained negative results. However, this study used a mixed diagnostic group as subjects with only 60 percent of the subjects having been diagnosed as schizophrenic. It is possible that the inclusion of several diagnostic categories masked any benefits that relaxation might have elicited specifically from the schizophrenics in the study.

No controlled research has been done on the possible therapeutic effects of EMG biofeedback treatment specifically for schizophrenic patients. The purpose of the present experiment, therefore, is to

investigate the effects of biofeedback mediated relaxation on schizo-
phrenic symptomatology, using the hypothesis that relaxation will
ameliorate the symptomatology through acting upon the underlying nervous
system arousal mechanisms. Because the resulting change could appear in
several areas of behavior, psychiatric ratings of patients and ward
behavior will be used as dependent variables as well as patients' self
reports of pathology.

Method

Subjects. Subjects were 20 psychiatric patients hospitalized with
the diagnosis of acute schizophrenia. The admissions of these patients
occurred between April and December 1978. Each patient's diagnosis was
confirmed by Basil Jackson, director of the Jackson Psychiatric Center
located at the hospital. There were three female patients and seven
males in the experimental group. There were five patients of each sex in
the control group. The mean ages of the patients in both groups were
quite similar, 30.3 in the experimental group and 30 in the control
group.

After their diagnoses were established, patients were asked to
participate in the study. Subsequently, each patient was assigned
randomly to the experimental or control group. The patient's psychi-
atrist and attending ward personnel were kept blind as to each patient's
treatment category. When patients began their participation in the
study, all were receiving what their psychiatrists considered to be
appropriate dosages of Promazine Hydrochloride (Sparine) as part of
their treatment regimen and were removed from all other psychoactive
medications. This was an attempt to control medication though not for

experimental analysis. Some departures from this medication control later occurred as a result of individual patient needs.

Apparatus. The bioelectric apparatus was a "Biofeedback Information System, Model 2 (BIF-2)." It was located in a small (12 foot X 14 foot), semisoundproofed room in the Jackson Psychiatric Center wing of the hospital. Subjects were allowed to recline in a position most comfortable to them in a black leather reclining chair. Skin electrode locations were cleansed with an alcohol preparation pad prior to each application to ensure the most efficient conductance of electric impulses.

The muscle sites chosen for monitoring were the frontalis and forearm extensor muscles. The frontalis is fairly easy to locate visually, and the electrodes were placed over the muscle that swelled when the eyebrows were raised. The extensor muscle was located by palpitation while the subjects flexed and extended their hands at the wrist. Three electrodes, mounted in a commercial band made of a rubber-like substance, were connected to the subjects' skin using Electro-gel for stronger conductance. The electrochemical impulses produced by the subjects were integrated by the BIF-2 system into microvolt/minute (μV /min) values which were visually displayed to the experimenter at the end of each minute interval. The display was out of the visual field of the patients. A 12-second period between each minute interval allowed the recording of the hundreths of microvolt/minute value display. For feedback the experimental subjects listened to electronic "clicks" or "pops" which reflected integrated muscle tension signals. The number of clicks heard by the patient indicated the immediate level of muscular activity that was occurring with increasing frequency of clicks signifying greater activity at the monitored muscle. The first three sub-

jects listened to feedback through headphones, but a small speaker was substituted subsequently for the remaining 17 patients to allow the experimenter to monitor the subjects' progress in relaxation more closely.

The Brief Psychiatric Rating Scale (Overall & Gorham, 1962) was used to measure the amount of change in pathology observed by the patient's psychiatrist or psychologist. This scale was designed for the rapid assessment of pathological behavior and measures behavior the rater actually observes as well as his/her evaluation of the patient's self-report. Sixteen well-defined symptom dimensions are measured with seven degrees of severity from "not present" (scored 1) to "extremely severe" (scored 7). The scores are added to produce a single numeric index of pathology. The scale has been demonstrated in several studies (Overall, Hollister, & Dalal, 1967; Gorham & Overall, 1964) to be a sensitive enough measure of pathology to detect change produced by pharmacologic intervention. The interrater reliability of this measure has been calculated to be .80 (Overall & Gorham, 1962).

The second instrument which was selected for assessment of be-havioral change was the Ward Behavior Inventory (WBI) (Burdock et al., 1968). It was designed to measure degrees of pathology through observa-tion by nonprofessional treatment personnel and has been demonstrated to be a sensitive enough measure to detect drug effects on pathological behavior. The WBI is made up of 138 items which describe observable units of behavior judged to be present or not present. The measure is made more precise by instructions given to the staff observers to base his/her response on only the 24 hour period before evaluation. The value of this instrument is its basic, operational description of be-havior (e.g., "Indicates that he does not know where he is." Y N). The

majority of the WBI items describe pathological behavior (e.g., "Laughs inappropriately." Y N). The greater the number of items marked Y, the more pathology the patient demonstrates. A number of items are worded in the opposite direction, to describe normal behavior (e.g., "Makes small talk with staff and other patients." Y N). The number of these items marked "N" is added to the number of "Y" responses to develop the pathology index. This construction corrects for observer response bias and guessing. The authors report that the interrater reliability of the instrument ranges between .57 and .87, with an average of .74. In one study, the authors thoroughly trained observers in the use of the inventory, and under these conditions the interrater reliability was .85.

The MMPI, which is widely used in psychiatric settings, was the instrument used to assess degree of pathology through patient self-reports. The 550 items are marked true or false by the patients, using the booklet form in this experiment. Results are reported in the form of scores which are standard scores with 50 as the mean and 10 as the standard deviation. The reliability of the individual scales (determined by test-retest procedures) range from .50 to approximately .92 (Gilliland & Colgin, 1951). The MMPI has been demonstrated to be a sensitive enough measure to detect the changes that result from pharmacologic intervention (Lewinsohn & Nichols, 1967; Klapper & McCollock, 1972; Mlott, 1973).

Procedure. All but the first of the six biofeedback training sessions were approximately one hour long. The first period lasted longer because, during that session, the subjects were asked for informed consent using the Marquette University Human Research Informed Consent form. Subjects were then given a standardized general explanation (Appendix A) of the purpose of this experiment and were shown how the

EMG apparatus works. Some patients were obviously fearful or suspicious. These individuals were shown the operation of the machine while it was attached to the experimenter. Through standardized instructions, the experimental group was then told that they would learn to control their muscular tension and to relax using the biofeedback signal provided by the machine. Patients in the control group were encouraged to relax by listening to music. Since there exists a discipline of music therapy, this was not considered a deception. After the introduction to the experiment, both groups were connected to the electromyograph. (The last 50 minutes of the first session established the three-phased pattern followed in each treatment session.) The first phase consisted of three one-minute measurements at one muscle site followed by three one-minute measurements at the other muscle site. The average of these three intervals at each site was considered to be reflective of the daily basal tension level for that muscle. The starting location was randomly varied. During the second phase of each session, both groups listened to a 20-minute tape of relaxation instructions modeled on Luthe's autogenic phrases and Jacobsen's instructions. It was standard procedure for the experimenter to leave the room during the presentation of the relaxation tape; but in exceptional cases, whenever a patient felt anxious if alone, the experimenter stayed with him or her. The experimenter returned to the room for the third phase, which consisted of twenty one-minute intervals, ten at each site. During this phase of the first session, the muscle tension of both groups was monitored at each site without feedback for either group. Both groups received the same treatment during this third phase of the session. At the end of the first session, the patient was asked to complete the MMPI. For the patient's convenience he/she was allowed to take the MMPI to the ward to

complete but was asked to do so before the next session. If a patient was unable to complete the MMPI alone, the experimenter or one of the ward personnel helped by reading the questions. The pretreatment WBI was distributed to attending ward personnel at this point, with instructions to evaluate each patient's behavior for the preceding 24-hour period. The Brief Psychiatric Rating Scale was distributed to attending psychiatrists. All but two of the scales were returned before the start of the second session.

The first two phases of session two were the same as those of session one. At the second session, however, the experimental manipulation was introduced during the third phase of the session. At this point the experimental subjects were given instructions emphasizing that they were to receive biofeedback from their own muscular activity. They were encouraged to learn to control the signal in some way which they were free to discover for themselves and thus control their muscles. They were then provided with feedback from the electromyograph in the same pattern of ten one-minute intervals at each muscle site as described earlier. The control patients, instead of receiving biofeedback signals, listened to five selections of tape recorded music which were judged to be calming. The tape was timed to correspond in length to the ten one-minute intervals of biofeedback produced at each site and included the interpolated 12-second intervals allowed for recording results. Both groups' tension levels were recorded by the experimenter on a prepared sheet at the end of each interval (during the 12-second interval) from the electromyograph's visual display.

Treatment sessions were held daily, Monday through Friday, unless other forms of treatment prevented them from being held. The third through the sixth sessions were replications of session two.

After the sixth session, the BPRS, and WBI were administered once again for the post treatment evaluation, and each patient was asked to complete the MMPI again. The control patients were fully informed about the purpose of the experiment at the end of the sixth session. After the patients had returned their completed MMPIs and the staff measures had been completed, the patients were given the opportunity, if they wished, to review the records of their muscle tension levels over the five training sessions.

<div align="center">Results</div>

Muscle Relaxation

Figure 1 shows the group means for extensor muscle tension
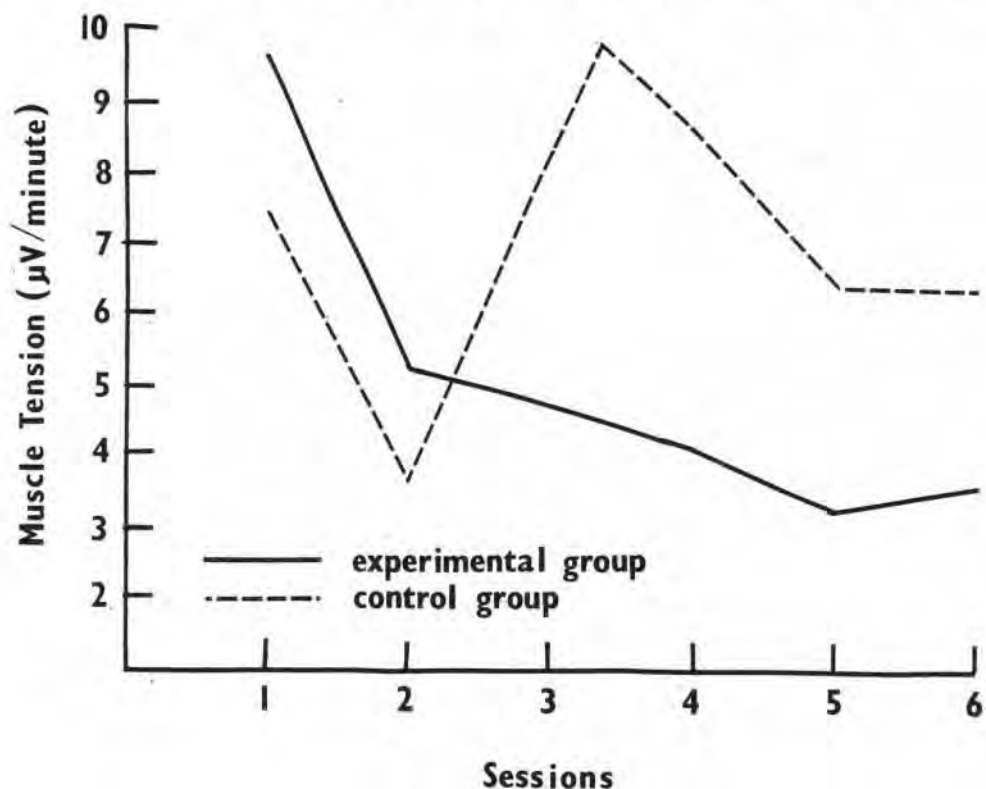

Figure 1. Mean extensor muscle tension at the beginning of each session

measured during the two sets of three one minute intervals obtained during the first phase of each session. The graphed results suggest that

steady improvement in muscle relaxation was achieved by the experimental group when compared with the control group. A similar graph (not shown) of frontalis muscle activity suggests no similar improvement at that site. A 2 X 6 analysis of variance (experimental vs control X 6 sessions) with number of sessions as a repeated measure was performed on both sets of muscle results. The biofeedback vs trials interaction was significant, $F(5,119) = 18.7$, $p < .05$, for the extensor muscle only.

A second statistical procedure, a one tailed t-test using regressed change scores (Posavac and Carey, 1980) to determine deviation, was performed to confirm the Anova results. Briefly, this procedure uses a regression of actual dependent scores to derive a formula that predicts dependent scores from independent values. The difference between these predicted scores and the actual scores observed is the regressed change score. The varience of these scores is a more sensitive measure of experimentally induced change in pre/post experimental designs. The results of this test indicate that both the frontalis and extensor muscles significantly relaxed as a result of biofeedback treatment. The experimental groups extensor mean decreased from 10.02 to 2.94 $/V$/min., while the control group's increased from 5.3 to 5.48 $/V$/min, $t(19) = -3.15$, $p < .05$. The experimental group's frontalis muscle decreased from 9.31 to 6.71, while the control group's muscle tension remained at 8.59, $t(19) = -2.25$, $p < .05$.

Reduction in Pathology

Group means for MMPI clinical scales were analyzed using a t-test with a standard deviation derived from the change scores to discover if the measured muscle relaxation was accompanied by changes in pathology. The WBI and BPRS results were also analyzed by t-tests to see if

behavioral changes were associated with muscle relaxation. The results

of these analyses are presented in Table 1.

Table 1

Change in Measures of Pathology
(t-tests performed on mean t scores)

MMPI	Mean Pre-Test Experimental	Control	Mean Post-Test Experimental	Control	Sd	t
1	63.0(18.6)	60.0(8.2)	62.4(15.6)	68.4(19.2)	8.8	1.0
2	69.8(24.6)	71.5(14.5)	65.4(24.1)	70.1(13.8)	9.2	.3
3	65.6(15.7)	64.0(10.6)	66.9(15.8)	67.8(14.8)	9.8	.3
4	73.8(13.7)	73.0(25.1)	76.2(9.9)	71.6(11.5)	6.4	.4
5	58.7(21.8)	53.7(14.8)	58.7(17.3)	50.0(16.2)	10.5	.3
6	79.9(18.7)	65.6(16.0)	76.4(17.4)	65.5(12.2)	23.5	.1
7	76.2(22.4)	79.1(16.4)	67.3(19.8)	72.9(11.9)	7.2	.4
8	90.2(26.2)	72.7(15.3)	79.4(26.4)	72.0(13.8)	9.7	1.0
9	75.0(17.6)	61.1(14.0)	64.0(17.0)	66.1(15.6)	12.2	1.3
0	57.9(15.3)	51.0(12.0)	60.1(13.7)	50.6(9.2)	5.3	.5
WBI	23.8(11.5)	20.3(8.9)	13.9(8.8)	14.6(7.5)	8.8	.3
BPRS	67.3(19.9)	46.5(4.0)	52.0(21.2)	40.0(8.0)	10.8	.9

* p < .05

The same measures of pathology were analyzed using the group mean

Table 2

Change in Measures of Pathology
(t-test performed on mean regressed
change scores)

Measures	Mean Regressed Score Experimental Group	Mean Regressed Score Control Group	Sd	t
MMPI				
1	-4.86	4.79	3.2	-3.0*
2	-1.63	1.86	2.2	-1.6
3	-1.21	1.27	2.1	-1.2
4	.89	-.84	1.5	.0
5	-.43	.44	2.5	.4
6	.47	-.44	.8	1.1
7	-5.00	4.97	2.0	-5.0*
8	-3.65	3.66	2.5	-2.9*
9	-2.90	2.98	4.4	-1.3
0	-6.00	5.90	3.0	-3.9*
WBI	-1.16	1.24	1.5	-1.6
BPRS	.92	-.90	4.2	.4

*p < .05

regressed change scores (Table 2) and using the standard deviation of the change scores in a t-test.

Relationship Between Muscle Tension and Behavior Pathology

In order to study more closely the relationship between tension and pathology a number of correlational analyses were conducted among the psychological instruments and muscle tension measures. Some of the relationships were found to be neither simple nor direct. Two factors had to be incorporated in the analyses to discover significant relationships. First, "impressionistic" observations by the writer had indicated that patients routinely seemed to display extreme tension at one muscle site and to have normal or lower tension at the other site. Secondly, as Tecce and Cole (1972) had pointed out, muscle tension is a somatic measurement that is especially prone to large individual variation. They suggested that individual differences should be removed by statistical procedures in order to produce the most accurate analysis. In the following analyses, individual variations were reduced by dividing the raw score values of muscle tension by each individual's lowest possible tension, or basal tension which, for purposes of this analysis, was considered to be the mean of the ten one-minute intervals at each muscle site during the first phase of the last of the six sessions. As was mentioned above, the two muscle sites were often widely disparate in the amount of tension they might display in an individual. It was therefore considered important to analyze them separately. Both muscle tension quantities were corrected, and high and low corrected muscle tension values were grouped for analysis. They will be referred to as the high pre-muscle corrected, the low pre-muscle corrected, high post-muscle corrected, and low post-muscle corrected measures.

The most striking finding to emerge from the correlations between tension and pathology measures was the relationship discovered between the Ward Behavior Inventory and corrected muscle tension. When lower
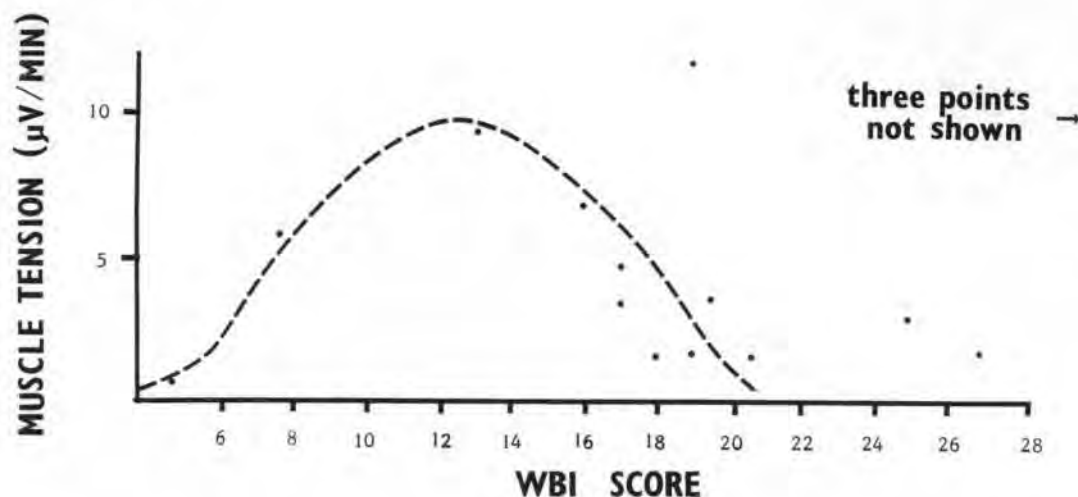

Figure 2. "Inverted U" function produced by the relationship of pathological behavior to muscle tension.

corrected pre-treatment tension values were graphed against the pre-WBI scores, it produced a curve (Figure 2) which appears to be an example of the the classic "inverted U" function (Tecce & Cole, 1972; Malmo, 1953; Hebb, 1955) found to be characteristic of the arousal performance relationship. The dotted line depicts the smoothing of the plotted values. The mathematical regularity of the function is quite apparent. After experimental treatment, a similar curve was not found because the corrected muscle tension values all fell below four microvolts/minute. A similar curve was produced by the relationship of the pre-MMPI 8 scale and lower corrected post-muscle tension elevation.

Less complex relationships between muscle tension and psychopathology were found in the analysis of MMPI results. All correlations use Pearson R procedures. The first step in the analysis simply correlates

pre- and post-muscle tension levels (corrected and uncorrected) with MMPI scales and the WBI. Correlation with the BPRS was not done because the staff psychiatrists returned too few ratings to allow valid analysis. Table 3 presents pre-treatment correlations and Table 4 post-treatment correlations.

Table 3

Correlations Between Pre-Test Psychopathology Scales and Muscle Tension Measures

MMPI	Frontalis	Extensor	High Corrected	Low Corrected
1	.17	.02	.13	.07
2	.47*	-.01	.35	.09
3	.18	.15	.18	.02
4	.00	.31	.06	.00
5	.08	-.32	.28	.25
6	.15	.00	.41*	.15
7	.36	.06	.39*	.05
8	.08	.17	.19	.21
9	.20	.29	.35*	.41*
0	.26	.04	-.01	.29
WBI	.00	-.23	-.11	-.18

*p< .05

Table 4

Correlations Between Post-test Psychopathology Scales and Muscle Tension Measures

MMPI	Frontalis	Extensor	High Corrected	Low Corrected
1	.15	.11	-.34	-.05
2	.21	.06	-.09	.24
3	.20	.10	-.27	-.05
4	.15	-.06	-.15	.11
5	.15	.13	-.05	-.39*
6	.03	.11	-.48	.01
7	-.04	.15	-.03	.23
8	.09	.31	-.20	.15
9	.37*	.06	.10	.27
0	.16	.02	-.49*	.03
WBI	.17	.20	.04	.16

*p< .05

If it were shown that changes in muscle tension are associated with specific changes in psychopathology, this would provide added indirect evidence that biofeedback was effective in the treatment of psychopathology. Data shown in the next two tables (Tables 5 and 6) correlate changes in muscle tension with changes in pathology. The latter is determined using the regression procedure used above, i.e., the predicted score subtracted from the actual score yields the change score for each individual.

Table 5

Correlations Between Regressed Psychopathology Change
Scores and the Regressed Change Scores of Corrected and
Uncorrected Muscle Tension

MMPI	Frontalis	Extensor	High Corrected	Low Corrected
1	-.28	.05	-.26	.19
2	.06	-.06	-.01	.16
3	-.11	.30	.06	-.01
4	-.14	-.18	-.14	.03
5	-.05	.18	.03	-.02
6	.04	-.22	.01	-.06
7	-.11	.20	-.15	.37
8	-.33	-.19	-.13	-.01
9	.17	.25	.12	.01
0	-.02	-.52*	-.43*	.17
WBI	.21	.06	.06	.15

$*p < .05$

An interesting problem for this experiment, dealing as it does with a psychophysiological treatment approach, is whether any physiological data can predict improvement in psychopathology. One might conjecture that, if such a relationship were found, it might lead to more refined treatment approaches. At the very least it would be another measurement the clinician could use which might prove resistant to faking, halo effect, etc., and thus add to the clinician's armamentarium of assessment

procedures. Table 6 presents Pearson's correlations between the pretreatment measures of muscle tension and the later change which resulted from treatment.

Table 6

Correlation Between Pre-Measures of Muscle
Tension and Change in Psychopathology

MMPI	Frontalis	Extensor	Pre-High Muscle	Pre-Low Muscle
1	.04	.00	-.28	-.02
2	.19	-.13	.11	.04
3	.27	.06	.07	-.26
4	.10	-.38	-.10	-.10
5	-.38	.09	.09	-.22
6	.30	-.17	.15	-.06
7	.23	-.45	-.53*	.09
8	-.41*	.24	.20	.28
9	.01	-.20	-.40	.09
0	-.29	-.02	.10	-.08
WBI	-.02	-.10	-.01	-.20

*p < .05

Discussion

Muscle Relaxation

Statistical analysis of the muscle relaxation results showed that muscle tension, especially the extensor muscle tension, was significantly reduced by biofeedback treatment over the six treatment sessions. The greater effectiveness of training for the extensor muscle may be because that muscle is strictly under voluntary control while the frontalis is influenced by both the sympathetic and voluntary nervous systems (Malmo, 1959).

The demonstration of a physiological change is an important finding because it is a partial confirmation of the experimental hypothesis, i.e., that biofeedback will alter nervous system arousal mechanisms. Physiological change is also important

because biofeedback is a manipulation which, without some independent physical measurement, is susceptible to the suspicion that the results are due to experimenter influence.

Although the change in measured relaxation was significant in this study, it did not reach levels obtained in earlier research (Lavalee, et al., 1977; Nigl & Jackson, 1979). The small size of the physiological response to biofeedback may have diminished the degree of psychological change as well. It is worthwhile to consider which uncontrolled variables might have produced the reduced strength of the biofeedback effect.

The most probable uncontrolled factor is the amount of variability in each patient's muscle tension. The standard deviation of muscle tension among the patients on one measurement, for instance, was between one half and one quarter of the range of all the tension values recorded (e.g., muscle tension ranged between four and thirty microvolts/minute and the standard deviation was approximately fifteen), a considerable degree of variability. It was obvious from tension values that muscle activity was fluctuating, sometimes moment-by-moment, in response to ward or treatment events and to such patient factors as mood and medication. Even momentary mental events (e.g., fantasies) would drastically alter patients' tension states. Part of this variability may be due to the disorder of schizophrenia itself. As Malmo, Shagrass, and Smith (1951), and Whatmore and Ellis (1964) have indicated, the psychopathology of schizophrenia includes a high degree of muscle tension lability.

The problem of muscle tension lability in this experiment was compounded by the very small N, which did not allow for the usual statistical reduction of the impact of variation in the analysis. This

occurred in many similar experiments as well. Overall, Hollister, and Dalal (1967) indicated that 40 patients in each group is a minimum number to establish certainty that treatment results actually induced by experimental manipulation could be shown statistically to be significant, i.e., that Type II errors would be avoided.

It is also likely that there were not enough training sessions to establish robust treatment effects. Staples (1975) has stated that five sessions appear to be the minimum number required to produce significant learning. However, his experiments dealt with several psychiatric diagnoses; and with schizophrenics, it is very possible that more sessions are needed to produce meaningful learning effects when such a severe disorder is present.

Another factor which may contribute to a weak biofeedback relaxation effect may have been the high degree of tension localization observed in the patients. Malmo (1959) reported that localization of tension to this degree was common and that EMG was much different from other physiological measures in this characteristic. He noted that the heart rate or blood pressure yield only one value no matter where recorded, but that "there are as many measures of muscle tension as there are muscles that can be recorded from." As suggested by Plutick, Wasserman, & Meyer (1975) and Goldstein (1965), this localization could be due to psychological factors as well as to physiological variations. Malmo concluded that, because of such site localization problems, cautious interpretation of negative results in the area of muscle tension is warranted.

In view of the localization problem, the combination of learning procedures recorded at two different muscle sites may have decreased the amount of actual learning that could have taken place at either site because each muscle could have learned relaxation independently rather

than generalizing to an overall state of relaxation as had been expected. Alternatively, one or the other muscle simply might not be amenable to conscious control. Localization such as that mentioned above would indicate that in this experiment, for example, learning which occurred at the extensor muscle may have inhibited possible learning at the frontalis site or vice versa.

The numerous factors reviewed above, which might mask biofeedback-induced change should be studied in future experiments. For example, future research designs might use only the extensor muscle, have a larger number of training sessions, and conduct two sessions or more per day.

Changes in Pathology

A regressed change t-test analysis (Tables 1 and 2) of MMPI scales 1,7,8, and 0 demonstrated significant change in the amount of pathology measured between the beginning and end of treatments. However, closer examination shows that the significant change in the 1 scale (Hypochondriasis) was produced by the increase in the control group's t score on this scale. The experimental group demonstrated no change on scale 1. The other scales do show significant changes in the expected direction by the experimental group. It is useful to consider the particular types of psychopathology reflected in these scales and their relationships to somatic arousal. A high scale 7 (Psychaesthesia) score is thought to reflect active, obsessively ruminative mentation. The reduction in this scale is especially salient to the experimental hypothesis because the avowed purpose of biofeedback is to calm inner excitation and reduce somatic arousal at the same time. The significant reduction in the 8 scale (Schizophrenia) is closely associated with reduction in somatic

hyperaousal as discussed in the introduction. This result is the single
most convincing confirmation of the experimental hypothesis.

The connection between the significant increase in the 0 scale
(increase in Social Introversion) to reduction in muscle tension is not
as clear as that shown on other scales. It could be postulated that, as
somatic arousal is reduced, the neurophysiological stimulation produced
by interpersonal contact becomes more overwhelming to the patient or that
the anxiety that is aroused by such relationships becomes more difficult
to deal with without some form of muscular reaction. Alternatively, it
is possible that those patients whose psychotherapeutic treatment enabled
them to relate to others in a more measured manner experienced a reduc-
tion in muscle tension.

There are several measures that share a conceptual relationship to
arousal but do not demonstrate similar significant improvement following
treatment. The 9 scale (Mania), by its very nature, should be closely
affected by levels of somatic arousal. The degree of change on this
scale closely approached, but did not reach, a significant reduction in
pathology level. Further reseach is necessary in this area to further
establish associations between change in somatic arousal and change in
pathology. It is important to establish whether the scales that do not
change are unchanged due to weak experimental effects, i.e., a low
degree of muscle relaxation, or whether the manipulation is simply
unrelated to such effects.

The multiple dependent measures of the present experimental design
allow a second way to analyze the psychological changes brought about by
biofeedback-induced relaxation. One can examine correlations between the
levels of change measured at each muscle and those changes brought about
in pathology as measured by the MMPI and WBI (Table 5). This analysis is

made more precise by using a regressed change score procedure, which yields change scores not subject to the difficulties found in difference scores. Such a procedure produces a more valid correlational analysis. The strongest relationship found using this procedure was a negative correlation between the amount of change in the 0 scale (Social Introversion) and the amount of tension change occurring in the extensor muscle. This negative correlation likely reflects the inability that patients had in reducing tension when other treatments encouraged them to increase their social interaction. Alternatively it may be that those patients who were least able to improve their interactions with others were best able to focus on somatic cues, master the biofeedback training, and thus reduce muscle tension. The 7 scale (Psychaesthesia) reduction is shown to be closely and positively related to the amount of tension reduction in the low-corrected muscle. The greater the reduction of muscle tension, the more the patient was able to moderate runaway mentation. The change in the 8 scale closely approaches, but does not reach, significant negative correlation to change in frontalis tension. This suggests that the more severe and/or stable the schizophrenic pathology was in a patient, the less likely he or she was to develop muscle relaxation skills. Overall, these results are difficult to interpret and strongly reflect the complexity of the relationship of muscle tension to pathology.

Pathology-Muscle Tension Relationship

The most striking results of this investigation were the correlational and especially the graphic analyses which show a close relationship between somatic arousal and certain dimensions of psychopathology measured by the MMPI and WBI.

The inverted U relationship discovered between physiological arousal and psychopathology, especially when seen in the graphic display showing the relationship between physiological arousal and MMPI and WBI measurements, is very much in accordance with the energy balance or hyperponesis theories of Jacobsen (1938), Whatmore and Ellis (1964), and Arieti (1965), which were reviewed in the introduction. This kind of relationship demonstrates the significant complexity of research in the psychophysiological area. Innumerable physiological factors and individual somatic variations must be taken into account in order to understand the processes involved (Tecce & Cole, 1972). For example in this experiment, a pattern of muscular tension which was signifcantly elevated at one site and yet normal at another had to be explained, while the complexity of this interaction was compounded further by the complexity of the schizophrenic disorder itself.

If further experimentation is able to confirm the existence of a curvilinear relationship between schizophrenic pathology and somatic arousal, such a relationship could have a considerable impact on the therapeutic and experimental approaches to schizophrenia. Attention to somatic arousal status might allow more discerning or exact treatment with neuroleptic drugs. As was mentioned in the introduction, excitatory factors in the patient's environment might themselves lead to hyper-arousal. Thus the treatment milieu might be "toned down," daily patient activities might be reduced, and a patient's family visits might be monitored to reduce stimulation.

Further analysis demonstrated significant correlations between muscle tension and measures of pathology before and after treatment. Table 3 shows that before treatment, the 2 scale (Depression) was strongly correlated in the positive direction to tension measured in the frontalis

muscle. This could be postulated to be quite directly associated with the dysphoria and distress of that symptom complex. The 6,7, and 9 scales (Paranoia, Psychaesthesia, and Mania) were positively related to the high-corrected muscle values. Thus the high tension found in that muscle seems to directly reflect in some way the active rumination, the interruption of normal interpersonal contact, and the overall hyper-arousal of the schizophrenic disorder. Scale 9 is positively correlated with the schizophrenic disorder and is also positively correlated to the low-corrected muscle values. That the 9 scale is related to both the ceiling and basal levels of tension, adjusted for individual differences, likely shows that this muscle scale does measure, to some degree, the qualities of psychic and somatic arousal. The 8 scale approached signifi-cant correlation with the low-corrected muscle values but, because of its curvilinear relationship to tension mentioned above, it would not be expected to have a significant linear relationship.

After treatment, the 6 scale was still correlated with the high corrected muscle value but this time in a negative direction. This suggests that the 6 scale might also have a curvilinear relationship with pathology and that measurement taken before and after treatment might capture either end of the curve. There was a negative correlation between the 5 scale and the low-corrected muscle value. The 5 scale and the low-corrected muscle values were also found to be related in the pathology change analyses, so this finding probably is not an anomaly. An explanation of this relationship probably involves the character-istics of the 5 scale that measures the intellectual bent of those who score high on it. These individuals may have approached the conceptual and learning aspects of biofeedback with more understanding. The 0 scale was negatively correlated to the high-corrected muscle value. The

explanation of this relationship is also difficult, but it may be due to the breakdown of normally strong inhibitions at the highest levels of arousal. Much further experimentation of the relationship of muscle tension and psychopathology is necessary.

Summary

This experiment demonstrated that biofeedback relaxation techniques could be successfully learned by schizophrenic patients. This success was achieved despite many factors, both physiological and psychological, which mitigated against it. This experiment further indicated that learning muscle relaxation techniques reduced severity of certain types of pathology. The specific areas of schizophrenic psychopathology that were reduced were bizarre mentation and highly active mentation, two symptoms which have a strong theoretical relationship to somatic hyper-arousal. Increased social introversion was also related to relaxation. The 9 scale (Mania) and the WBI closely approached but did not achieve significant reduction. High scores on the 8 scale (Schizophrenia) and 0 scale (Social Introversion) were demonstrated to limit learning to relax through biofeedback. Subjects who exhibited high arousal experienced the greatest reduction in pathology. However, physiological arousal was shown to be only one factor among those which are involved in determining degree of psychopathology.

Several relationships were shown to exist between muscle tension and pathology, some of which were curvilinear in nature. The 9 scale (Mania) was shown to be very strongly related and the 6 and 7 scales were more moderately related to somatic arousal. Further experimentation is necessary to examine the relationship of arousal and pathology, both in schizophrenia as well as other types of disorders.

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APPENDIX 1

Standardized Instructions Read to Patients

at Session 1

Experimental Patients -

As I said when I asked you to volunteer for this experiment, this is
a study of the effects of relaxation. Now I'd like to inform you more
specifically about its purpose. This study is intended to determine how
much relaxation is learned through biofeedback techniques and how this
might lead to a person feeling better. Biofeedback is a process by which
a machine tells a person how tense his or her muscles are by measuring
the electrical impulses the muscles produce. By listening to the bio-
feedback signal the machine gives out, a person can develop control over
his tension and learn to relax. Now, I'm going to read the Marquette
University Informed Consent Form to you. Then I will show you how this
biofeedback machine works.

Control Patients -

As I said when I asked you to volunteer for this experiment, this
is a study of the effects of relaxation. Now I'd like to inform you more
specifically about its purpose. This study is intended to determine how
much relaxation is learned through music relaxation and how relaxation
might lead to a person feeling better. This machine will tell me, by
measuring the electrical activity of your muscles, how much you are
relaxing. By listening to music a person can often quiet his or her mind
and "put out" thoughts and worries. This can help a person to relax.

Now I'm going to read you the Marquette University Informed Consent form and then I'll show you how this machine tells me how tense you are.

APPENDIX 2

Graphic Representation
of Experimental Procedures

Baseline Session

Experimental
and Control Groups:

Three 1-minute intervals at each muscle (measure-ment only) —— Twenty minute tape of relaxation instructions —— Ten 1-minute intervals at each muscle (measurement only)

Treatment Sessions

Experimental Group:

Ten 1-minute intervals at each muscle of biofeedback signals

Both Groups:

Three 1-minute intervals at each muscle (measure-ment only) —— Twenty minute tape of relaxation instructions

Control Group:

Ten 1-minute intervals listening to music (each muscle monitored)