The effects of altering respiratory parameters upon certain autonomic and somatic variables

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Thesis submitted to the Department of Psychology, University of Cape Town, in fulfilment of the requirements for the degree of Master of Arts in Psychology.

Cape Town
South Africa
November, 1978

Acknowledgements

I wish to express my gratitude to the following people :

Dr. G.S. Saayman, my supervisor, for his continuous help, advice and sympathy.

Mr. J. de Wet for his help in revision of the thesis upon re-submission.

M. and S. Hardy, my parents, for support and financial assistance with equipment. Without them this thesis would not have been possible.

H.V. Gibson for much assistance in almost every practical aspect of this thesis.

The Human Science Research Council and the University of Cape Town for bursaries which assisted in financing the research.

The students and friends who volunteered for the experiment, in spite of the great inconvenience it often caused them.

A.R. Timm (Electrical Engineering Department, U.C.T.) who designed and built the EEG Analyser, devoting to it much of his own time.

G. Pearson (Mechanical Engineering research student, U.C.T.) who built the respiratory strain gauge belt.

M.R. White (Physiotherapy Department, U.C.T.) for advice regarding the teaching of the deep breathing technique.

Dr. T.J. van der Werff (Bio-engineering Department, U.C.T.) for the loan of the strain gauge coupler, without which respiratory measurements could not have been taken.

Professor C.E. Lewer Allen (Professor of Orthopaedic Surgery Department, U.C.T.) for the advice and references in the field of electromyography.

Dr. I.A.P. Tunley (Physical Medicine Department, Groote Schuur) for advice concerning muscular tension recordings.

Professor A.W. Sloan (Professor of Physiology, U.C.T.) for the references and advice concerning the respiratory system, and for permitting me to attend Course 1 lectures.

L. Gilbert (Psychology Department, U.C.T.) for advice on statistical issues.

A. Reynolds (Psychology Department Technician, U.C.T.) for help with management of the equipment.

L. Elley who typed the first draft of this thesis in her own time.

P. Gray-Taylor (Psychology Department) for help with the computer and computer programme, and for volunteering as a subject in this research.

A.J. Gibson who recruited all the volunteers whom I needed for my pilot studies.

J. Hutchings who typed this thesis under much pressure.

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SUMMARY

AIMS OF THE RESEARCH

Psychophysiological research has shown that high physiological arousal in the autonomic and somatic nervous system is associated with emotionality, e.g. anxiety. It was the aim of the present research to investigate the use of a deep breathing technique which would be easily amenable to voluntary control, in order to train subjects to lower their level of physiological arousal.

INTRODUCTION

High physiological arousal in the somatic and autonomic nervous systems has long been associated with emotional states such as joy, pain and anger (Cannon, 1915). Psychophysiological research at present has indicated a positive correlation between high arousal and psychopathological states such as anxiety and schizophrenia (Alexander, 1972, see review by). Similar differences in arousal level have been reported in normal subjects differentiated into high and low emotionality scores using psychometric tests e.g. the Taylor Manifest Anxiety Scale (TMAS) (Kelly et al, 1970). A psychophysiological approach to the treatment of maladjusted emotional states would appropriately attempt to deal with the physiological over-arousal symptoms observed in such states. In the present research, deep breathing was used as a means of lowering physiological arousal in normal subjects. This approach in treatment of maladjusted emotional states is based upon two main assumptions : i.e. firstly, it is postulated that the effects of deep breathing upon arousal level would be comparable in normal and emotionall

maladjusted groups, provided the technique be effectively practiced. Secondly, it is argued that conteracting the physiological symptoms associated with emotional states would be therapeutic in cases of emotional maladjustment such as anxiety. The validity of these two basic theoretical assumptions are discussed below.

The present findings have been concerned with the effects of deep breathing upon arousal level in normal subjects. It should be noted however, that the differences in physiological arousal existing between pathological and non-pathological emotional states, has been found to be of a quantitative, and not of a qualitative nature. Studies of normal subjects undergoing transient periods of heightened emotionality (e.g. Wenger, 1948, 1960; Wenger and Ellington, 1963; Wenger et al, 1957) have shown the same pattern of increased sympathetic activity and muscular tension to be present as that reported in studies of various psychodiagnostic groups such as schizophrenics and neurotics (Alexander, 1972, see review by). Other studies, using normal subjects differentiated into, for example, high and low anxiety or depression groups, using psychometric tests, have reported the same direct relationship between physiological arousal and emotionality (e.g. Balshar, 1962; Katkin and McCubbin, 1969; McFarland and Coombs, 1974; Smith, 1973). Furthermore, psychotic, neurotic and normal control groups have been reported to differ as to degree of arousal, the psychotics showing the highest levels of physiological arousal, and normal control groups ranking lowest on the arousal continuum (e.g. Eysenck, 1956; Goldstein, 1965). There remains to consider the significance of quantitative differences in initial level of physiological arousal upon the outcome of the deep breathing technique, i.e. upon lowering physiological arousal. In this study, the normal subjects used in all probability had

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lower initial levels of physiological arousal than would be found in either transitory or pathological emotional states. In certain instances, initial arousal level is a crucial consideration in assessing induced physiological changes in arousal : e.g. if an organism has reached its maximum or minimum arousal level, any attempts to initiate further changes beyond these limits would prove fatal : thus such changes will most often prove unsuccessful due to homeostatic mechanisms responsible for maintaining level of arousal within the limits required for survival (Lacey, 1956, 1967). In the present study, the effects of deep breathing would only differ in direction of change from more highly aroused groups, if all the subjects were to begin the experiment at their minimum level of arousal. This represented an unlikely possibility. The presence of unidirectional, lowering of arousal level in this study, would indicate that deep breathing may be used to lower physiological arousal. However, further research would be required to indicate whether the concentration required in practicing the deep breathing technique may be successfully applied to psychopathological groups. The present research findings concerning the success of deep breathing in lowering physiological arousal would be most applicable to normal subjects suffering from transient anxiety of phobia.

The second theoretical issue which is considered, regards the effectiveness of the physiological approach to the improvement of maladjusted states : this depends upon the importance of the role of physiological symptoms in subjective evaluation of the emotionally-disturbed state. Emotions such as anxiety (pathological and non-pathological) are objectively assessed by such techniques as interview of the subject and self-descriptive psychometric tests. Both

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of these techniques rely upon subjective state for diagnosis. The importance of physiological cues in evaluation of subjective emotional state has been investigated in certain drug studies, in which high physiological arousal was artificially induced. From these experiments the following conclusions may be drawn :

1. Physiological cues are consciously used in evaluating subjective emotional state: however, the presence of physiological symptoms of an emotion (such as anxiety) does not necessarily result in the subjective feeling usually associated with these symptoms; e.g. Ackerman and Sachar (1974) reviewed studies where anxiety neurotics were given serum lactate injections (the effect of this injection is to simulate the physiological symptoms of high arousal associated with anxiety). A significant proportion of the patients were reported to say that the only reason why they did not experience "mental fear" or "panic" was because of the reassuring presence of the physician, who had told them about the effects of the injection; some of the patients were reported to interpret their state of high physiological arousal as subjectively felt anxiety, in spite of their knowledge that the physiological symptoms had been artificially induced.

2. The physiological symptoms associated with a subjective emotional state are essential for the experience of that state - e.g. Schacter and Singer (1962) successfully evoked feelings of anger or euphoria, in a predictable fashion, according to which of the two states was suggested to normal subjects who had received standardized injections of epinephrine. However, none of the subjects given a placebo injection reported a subjective

emotional state, even when it was strongly suggested to them.

These studies indicate that physiological symptoms are important in subjective evaluation of emotional state. Thus, dealing efficiently with the symptoms of unpleasant emotional states could prove effective in relieving such states.

Turning now to research findings in abnormal psychophysiology, high levels of physiological arousal have commonly been reported in states of anxiety (Lader, 1975, see review by); e.g. when compared to normal control groups, anxious subjects have been reported to have higher skeletal muscular tension (Balshan, 1962; Goldstein, 1964 ; Smith, 1973); higher heart rates (HR) and blood pressure (Goldstein, 1964 ; Kelly et al, 1970; Friedman and Bennett, 1974); higher overall sympathetic activity (Smith and Wenger, 1965); higher pulse rate (Bloom et al, 1976). Similarly, high physiological arousal in the autonomic and somatic nervous systems has been found to be a characteristic of most other psychopathological states, e.g. depression (McCannon, 1973; Reite et al, 1974); schizophrenia (De Wolfe and Youkilis, 1975; Fenz and Velner, 1970; Zahn et al, 1976).

There have been inconsistencies in research findings concerning the precise nature of the physiological arousal symptoms associated with emotionally-disturbed states (Alexander, 1972, see review by). e.g. Golstein (1964 a) found no difference between anxious and normal subjects in testing frontalis EMG tension, while Smith (1973) reported higher frontalis EMG tension in questionnaire rates anxiety rated subjects. These inconsistencies have been largely the result of differences in degree of pathology, type of phenomena observed, scoring procedure and similar technical difficulties. However, there has been widespread experimental support indicating that high physiological arousal in the autonomic and somatic nervous systems is associated

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with states of emotionality, both pathological or transient (Duffy, 1972, see review by).

Various techniques have been investigated with a view of counteracting the physiological symptoms accompanying emotional states involving nervous tension, by reducing arousal level, e.g. biofeedback techniques have mostly dealt with single physiological parameters, such as increasing hand temperature so as to relieve migraine headaches (Sargent et al, 1973; Turin and Johnson, 1976). Some techniques aiming at a more general state of relaxation have required the use of highly specialised equipment, e.g. electrosleep, where a small current is circulated through electrodes placed over the forehead and ear; this procedure was reported to be successful for treatment of sleep disturbances (Borkovec and Weerts, 1976).

Luthe's (1963) use of autogenic training is a similar technique to deep breathing, both aiming at a self-induced state of general relaxation without the use of any laboratory equipment. Autogenic training requires the subject to concentrate in turn on each part of the body, suggesting that feelings of warmth and heaviness are being experienced. Deep breathing consists of concentrating passively on a single physiological process, i.e. breathing deeply in a systematic way. These differences in procedure may cause one or the other technique to be applicable to different subject populations; i.e. subjects who have concentration problems would probably find it easier to practice a repetitive passive task such as deep breathing. Others who find a monotonous task difficult to follow would respond more readily to autogenic training.

RESPIRATORY ACTIVITY AND PHYSIOLOGICAL AROUSAL

Psychophysiological research has often reported a direct correlation between physiological arousal, as indicated by electro-encephalographic (EEG) wave patterns and respiratory activity e.g. Goldie and Green (1961), Aserinsky (1965), Timmons et al (1972). Decreasing respiratory activity has also been reported to be a useful means of inducing relaxation (Grim, 1971; Harris et al, 1976).

From the physiological standpoint, the experimenter was unable to find conclusive evidence dealing with the effects of various modes of breathing upon specific physiological systems such as the ANS; it should be noted however that links between the respiratory system, and the ANS and the skeletal muscles have been known to exist, even if their precise function remains unclear (Comroe, 1974), It would prove difficult to objectively predict the effects of deep breathing upon physiological arousal. The present prediction that decreased physiological arousal in the ANS and skeletal muscles would accompany the practice of deep breathing was based upon results observed in pilot studies of the effects of deep breathing upon arousal. Furthermore, in a personal communication with physiotherapist Miss White (1975), it became apparent that the relaxing effects of deep breathing were recognised and exploited, although the physiological mechanisms involved in this procedure remained obscure.

The present research was an exploratory study into the effects of the deep breathing technique upon various physiological parameters i.e. Skin Resistance (SR), Heart Rate (HR), masseter and corrugator muscle tension. It was predicted that during the practice of deep breathing, arousal level would decrease, as indicated by increased

SR level, decreased HR and muscular tension.

METHODOLOGY

Two groups each consisting of 6 subjects were used in a repeated measures design: the experimental group was instructed in the technique of deep breathing by the use of certain exercises which resulted in the subject breathing slowly and deeply, using his whole lung at each breath. The control group was instructed in the technique of progressive relaxation: they were taught how to locate a tensed-up muscle and to relax it. Both groups each attended a total of eight 25 minute sessions per subject; these 8 sessions consisted of one habituation session; two pre-treatment baselines which were meaned so as to obtain a reliable recording of baseline activity; the training sessions during which no recordings were taken followed. Then, three treatment sessions were run consecutively followed by one post-treatment baseline. The measurements taken were palmar SR, HR, masseter and corrugator muscle tension (EMG). The recordings were made on a Beckman Dynograph Type RP; for HR and EMG measurements, Beckman A-C D-C Couplers Type 9806A, functioning the A-C mode was used. A Beckman GSR Coupler Type 9842 with a belt containing four strain gauges was used for SR recordings, the latter being made using the Time Mark System.

A rank order system was used for analysis of the EMG data. The SR and HR data were analysed using the 3-way ANOVA with repeated measures on B and C and where appropriate this was followed by a simple main effects and pairwise comparisons for 3-way ANOVA. The session was divided into three main phases, and the second

phase/...

phase subdivided into two parts to yield four 5-minute periods which were compared to each other. The following diagram illustrates the means by which each sessions was divided in the statistical analysis :

PHASES WITHIN EACH 30 MINUTE SESSION			
	FIRST PHASE	SECOND PHASE	THIRD PHASE
Time recorded	5 minutes	12½ minutes	7½ minutes
Time used in statistical analysis	5 minutes	last 10+ minutes	last 5 minutes

+ the second phase was subdivided into two 5 minute periods phase 2(i) and 2(ii).

RESULTS

For SR, the ANOVA indicated significant interaction effects between factors ABC, at the .05 level ($F = 1.83$, $df = 12,120$). These results were followed by an Analysis of Simple Interaction Effects (ASIE); where appropriate, more detailed information was obtained from Analysis of Simple Simple Main Effects (SSME). Significant SSME F-ratios were followed up by a comparison of cell means using Tukey's HSD test. In the final analysis the SR results indicated significantly higher SR during the second half of the deep breathing period (factor C3) compared to SR in the initial period of the session (factor C1) both in the second and the third treatment sessions (factors B3 and B4) at the .05 level ($t' = 3.68$, $df = 4,75$).

The only significant HR results indicated differences between

treatment/...

treatment means as a function of phases (factor C) significant at the .01 level, $F = 4.51$, $df = 3,30$). This finding permitted no differentiation to be made as a function of groups or sessions. The gradual decrease in HR as a function of phases (factor C) was to be expected under baseline rest conditions. No relevant conclusions could be drawn from this finding.

According to the rank order procedure used in this research, the only EMG results found reliable were the greater masseter muscle tension decreases during phase 2 in treatment sessions 2 and 3 as compared to the first baseline and first treatment sessions. Considering the magnitude of these differences in masseter muscular tension, there was 37% more EMG tension decrease during phase 2(i) of treatment sessions 2 and 3 (i.e. during the first part of deep breathing) than in the corresponding phase of the first baseline and first treatment sessions. The same trend was observed during phase 2(ii), although there was 57% more masseter EMG decreases in treatment sessions 2 and 3 as compared to the first baseline and first treatment session.

DISCUSSION

In the present research, it was found that deep breathing was accompanied by decreases in arousal in at least some physiological systems, i.e. there were significant SR increases in treatment sessions two and three, during the second half of deep breathing (phase 2(ii)); similarly, masseter muscle tension was found to be lower by an average of 47% during the deep breathing period of treatment sessions two and three, than in the corresponding phase of the first baseline and first treatment session.

It should be noted that the lack of significant drop in physiological arousal in the first treatment session is not altogether surprising, as prior to this session, the subjects had only practiced the newly-learnt technique once, for a period of ten minutes, (after receiving the instructions during the training session). The significantly increased SR and drop in masseter muscle tension observed in treatment sessions two and three followed the same trends : namely, SR rose during the first half of deep breathing during these treatment sessions, but this increase only reached significance during the second half of deep breathing (phase 2(ii)). The masseter muscle tension decrease was 20% greater during phase 2(ii) as compared to phase 2(i) (representing the second and first halves of the deep breathing period). The effects of deep breathing in lowering arousal in two of the four physiological measurements recorded were found to be specific to the deep breathing period, as no significant findings were obtained for phase 3 (i.e. after cessation of deep breathing). It may be concluded that the deep breathing technique consistently

lowered/...

lowered physiological arousal in two of the four physiological measurements taken.

The muscular relaxation technique seemed not to have been as effective in lowering physiological arousal; no significant effects were found for treatment sessions, although SR increases were significant in phase 2(ii) of the post-treatment baseline i.e. during the phase equivalent to that in which muscular relaxation was practiced during treatment sessions. This finding could be interpreted as indicative of the control group exhibiting gradual lowering of physiological arousal as a function of habituation to the experimental situation. Alternatively, it could be postulated that the significant SR increases observed in phase 2(ii) of the post-treatment baseline is due to a generalization of treatment effects to that session, without the subject's conscious inducement of relaxation. This could be compared to a classical conditioning effect with the experimental situation acting as a conditioned stimulus. The view of muscular relaxation occurring as a learnt response to environmental stimulus, instead of being a state induced through certain exercises, has recently received support (Connor, 1974, see review by). It may be postulated that muscular relaxation actively induced on demand is difficult to put into practice, in spite of the subject's knowledge of how to locate and release muscular tension. However, it is possible that relaxation as a response to a specific stimulus (here the experimental situation) could have occurred during the post-treatment baseline.

In attempting to account for the physiological mechanisms or processes involved in the correlation observed in this research

between/...

between deep breathing and significant SR increases and masseter tension decreases, only indirect evidence is available. It should be noted that no assessment of the respiratory changes involved in deep breathing (e.g. minute ventilation, oxygen consumption) was possible here, due to lack of suitable equipment. However, subjective reports consistently indicated that deep breathing involved "breathing less than usual".

Considering the means by which respiratory activity may influence the other major systems, only indirect experimental evidence may be brought up as the relationships between the major physiological systems remain to be fully explored; however, it has been recognised that such connections do exist e.g. Ganong (1971) suggested connections between the inspiratory-expiratory respiratory centres (in the pons and medulla) and the neocortex as although inspiration and expiration were not usually conscious events, they were both under voluntary control. Ganong (1971) noted that pain and emotional stimuli affected respiration, thus suggesting afferents from the limbic system and hypothalamus. Further, movements of the joints stimulated the respiratory centres, presumably through impulses in afferent pathways from proprioceptors in muscles, tendons and joints (Ganong, 1971).

In a more recent and comprehensive textbook concerning the respiratory system, Comroe (1974) referred to the location of the respiratory centres in the medulla and pons as strategic for interactions with other systems, because of the Reticular Formation (RF) consisting of many groups of richly interconnecting neurons, is also situated there Besides, the medullary respiratory centre, responsible for rhythmic breathing, has connections with the respiratory centres

in/...

in the pons, with areas of the hypothalamus and cortex, and with the Reticular Activating Cortex (Comroe, 1974).

With consideration for the above evidence, it may be concluded that the respiratory system has had effects upon the other major physiological systems via one or all of the following structures :

1. The sino-aortic chemoreceptors.
2. The central chemoreceptors in the mid-brain which lie close to the Reticular Formation.
3. Via the respiratory muscles and gamma motoneurons.

In conclusion, the present research findings indicated decreased arousal in at least some physiological systems during the practice of deep breathing by normal subjects. The learning of the technique does not require the use of equipment, and is easy to master : the present results suggest that two separate practice sessions of approximately ten minutes each would be sufficient for the consistent lowering of physiological arousal associated with deep breathing to be observed.

Research findings are presented in the introduction (see page 3) which indicate that high physiological arousal symptoms are an important source of information in subjective assessment of emotional states (such as anxiety). The present research has indicated that the practice of deep breathing is accompanied by decreased physiological arousal in certain systems and it may therefore prove useful in alleviating discomforting physiological symptoms : it is noteworthy that high physiological arousal has in some cases proved sufficient in inducing subjectively felt anxiety, even though the symptoms had been artificially induced by a drug

(Ackerman/...

(Ackerman and Sachar, 1974, see review by). It cannot be expected however, that such a physiological approach would be sufficient in dealing with unpleasant emotional states, which also have cognitive associations.

The usefulness of deep breathing with psychodiagnostic groups therefore is suggested to be worthy of further investigation.

Abstract

Psychophysiological research into psychopathological states has found more sympathetic nervous system activity, higher muscular tension and respiratory rates in psychodiagnostic groups compared to normal control groups. The present research has used the technique of Deep Breathing to lower physiological arousal in normal subjects. Deep Breathing was compared to a muscular relaxation technique in a repeated measures design. Skin resistance and muscle tension results indicated that physiological arousal was significantly lowered during the latter part of Deep Breathing, in the last two (out of three) treatment sessions. It was concluded that Deep Breathing consisted of a reliable means of lowering physiological arousal, and could prove useful in states of over-arousal such as anxiety and phobia. Further investigation as to its applicability to psychodiagnostic states was suggested. The muscular relaxation technique showed no significant treatment effects. In the post-treatment baseline, skin resistance was significantly increased during the phase corresponding to that in which relaxation had been practiced during the preceding treatment sessions. It was suggested that this finding could be attributable to poor voluntary control over the muscular relaxation technique; this explanation was indicated by the Control Group's failure to successfully induce, maintain and terminate the physiological effects widely associated with lowering of arousal (relaxation) during their practice of the technique.

1. Aims of the Research

In reviewing psychophysiological research, it was found that high physiological arousal in the autonomic and somatic nervous systems was associated with emotionality, e.g. anxiety. It was the aims of the present research to investigate the use of a deep breathing technique which would be easily amenable to voluntary control, in order to train subjects to lower their level of physiological arousal. Normal subjects were used, as it was pointed out (see Section 2.1) that the difference in physiological arousal existing between emotionally disturbed and normal subjects were of a quantitative, and not of a qualitative nature. Thus, if the deep breathing technique were to be consistently associated with lowered physiological arousal in normal subjects, the technique could be used with normal subjects in preventing the onset of transient anxiety states such as phobia. In the long term, the present investigation would indicate the relevance of further research into the use of deep breathing in pathological high arousal states, such as chronic anxiety.

In the present research, several physiological measurements were taken so as to gain a representative view of physiological arousal i.e. in the autonomic nervous system, heart rate and skin resistance were recorded; in the somatic nervous system, electromyographic recordings of tension in the masseter and corrugator muscles were taken. Electroencephalographic recordings of occipital activity were also included; this data could not be used in the results due to certain technical difficulties involved in the analysis of the data. A new method of EEG analysis is described fully in the appendix, which may prove valuable to future researchers.

2. Introduction/..

2. Introduction

High somatic and autonomic arousal have been reliably associated with various psychopathological states (discussed in more detail in Section 2.1). Furthermore, high physiological arousal has been associated with normal subjects with high ratings on psychometric tests, e.g. the Taylor Manifest Anxiety Scale (TMAS) (Kelly et al., 1970); and with normal subjects exposed to stressful situations (e.g. threat of electric shock, Bloom et al., 1976). High physiological arousal has thus been generally associated with both pathological and non-pathological emotional states, such as anxiety. As early as 1915, Cannon (1915) noted that high physiological arousal was associated with various behavioural states, not necessarily subjectively unpleasant ones, e.g. hunger, joy or pain. The particular "psychological" label attached to such an aroused state was dependent upon cognitive and situational variables. It is noteworthy that psychophysiologists have been unable to differentiate between various psychodiagnostic groups physiologically, e.g. between anxiety and schizophrenia, (Alexander, 1972, see review by).

In summary, physiological arousal in the Autonomic Nervous System (ANS) and somatic nervous system is found to increase in the presence of emotional behaviour, such as anxiety or joy. Emotion may be said to consist of undifferentiated physiological arousal, varying only in degree, and presumably duration, according to the intensity of the emotional state. Most psychopathologies consist of emotional disturbance; e.g. depression, anxiety. Thus, a psychophysiological approach to the treatment of maladjusted emotional states would appropriately attempt to deal with the physiological overarousal observed in such states.

The present/...

The present research has undertaken to investigate whether physiological arousal could be lowered in a predictable manner, by the use of a deep-breathing technique. Normal subjects were used for this purpose. It is necessary therefore to discuss the extent to which the present findings can be considered relevant to a psycho-physiological approach to emotionally disturbed states. If the difference in physiological arousal, existing between emotionally disturbed and normal subjects were of a qualitative nature, it would follow that the present investigation would be of no relevance to pathological emotional states. Taking the example of medical pathology, it would be meaningless to investigate the effects of a certain medicine upon bronchitis, using healthy subjects. Physiological arousal however, cannot be considered to be a "sympton" such as bronchitis; it refers to the level of activity observed at a given moment, in the ANS and somatic nervous system. In any given subject, regardless of his psychodiagnostic status, this level of physiological activity fluctuates according to emotional state. The difference between psychodiagnostic and normal groups may be expected to lie in degree and duration of the increased physiological arousal e.g. non-pathological emotional states such as transient anxiety, are commonly experienced by normal subjects in response to life-stresses, such as a dentist appointment or current economic difficulties. High physiological arousal in transient states of emotional disturbance was reported by Wenger and his colleagues (Wenger, 1948, 1960; Wenger and Ellington, 1943; Wenger et al, 1957). These authors have undertaken a thorough study of autonomic function, using a comprehensive number of measurements, as well as large groups of subjects of various ages, from which they derived normative tables of autonomic balance, which they call the $\bar{A}$ factor. This overall

score of/...

score of autonomic balance was derived from a number of measurements of various autonomic variables (e.g. blood samples for hormone content, salivary output, HR, palmar and volar skin conductance, pulse pressure, consisted of some of the measures taken). The raw scores obtained from a large number of normal subjects, were transformed into Z and T-scores to make up the overall $\bar{A}$ factor normative table. These studies were well controlled (e.g. for humidity, temperature, season, sex, age) and thus consist of some of the most important studies made of autonomic function even today.

Wenger (1941) found that a high $\bar{A}$ score (indicating predominantly parasympathetic activity) was correlated with less emotional, more behaviourally controlled, as well as shyer children. Further, Wenger and Ellington (1943) found a correlation between high $\bar{A}$ in children and lower basal metabolic rate, less fatigue symptoms, amongst other personality traits of no great interest to the present paper (e.g. less friendly and gregarious, etc.). Wenger (1948) conducted a study based on the normative tables derived from a vast number of subjects, upon 225 patients with diagnosis of operational fatigue, 98 hospitalized psychoneurotics and 488 unselected preflight students (normal controls); all subjects were recruited from the Army Air Force. He found significantly higher sympathetic arousal (low $\bar{A}$ score) in the operational fatigue group using several measurements (salivary output, palmar conductance, diastolic and systolic blood pressure, sinus arrhythmia, HR, finger temperature), as well as higher respiratory rate and tidal air mean. Sublingual temperature did not discriminate between groups. The hospitalized neurotics also had a tendency to be sympathetic dominant, but the trend was not as pronounced as in the operational fatigue group.

Wenger et al/...

Wenger et al (1957) reviewing their past research, made the observation that the $\bar{A}$ factor was related to personality patterns and diagnostic categories; e.g. anxiety psychoneurotics, battle fatigue subjects, asthmatics were found to have an overall low $\bar{A}$ score (indicating sympathetic dominance).

Other studies using normal subjects differentiated into, for example, high and low anxiety or depression groups, using psychometric tests, have reported similar findings concerning the direct relationship between physiological arousal and emotional maladjustment (e.g. Balshan, 1962; McCubbin, 1969; McFarland and Coombs, 1974; Smith, 1973).

Thus, high physiological arousal is a characteristic of increased emotionality, irrespective of the presence of pathology. Further support for the view that no qualitative physiological difference exists between groups differing in degree of psychopathology, can be found in studies involving comparisons between psychotic, neurotic and normal groups; e.g. Eysenck (1956) conducted a study on neurotic, psychotic and normal subjects, whom she compared on basal skin resistance and the skin resistance response during a rest period, then while the subjects answered by "yes" or "no" to the Maudsley Medical Questionnaire, and finally in response to a loud noise. The psychotic group had the highest levels of skin resistance at rest. The neurotics had higher skin resistance values than normal controls, but not as high as the psychotic group. Eysenck's (1956) results indicated that there was a quantitative rather than qualitative difference between neurotic and psychotic groups. This quantitative difference was also observed in scores of restlessness (number of times a subjects moved, coughed, etc.). The psychotic being the most restless group, and the neurotics "also inclined to be fidgety, p.268". Similar

conclusions/...

conclusions were reached by Goldstein (1965) in a study conducted on mixed psychotic, neurotic, psychopathic and normal groups.

Jurko et al (1952) suggested a qualitative difference between psychodiagnostic groups. Jurko et al (1952) compared a psychoneurotic, an early paranoid schizophrenic and normal control group in a frustration test. They found that the various groups released their energy during frustration via different systems, e.g. normals showed disturbance predominantly through a rise in skin conductance and cardiovascular activity, the neurotic group responded through all the physiological systems (electrodermal, cardiovascular, and respiratory activity, as well as through skeletal muscle tension). The early schizophrenics responded mainly through the cardiovascular and muscular systems. Only superficially do these findings suggest qualitative differences between psychodiagnostic groups, i.e. all the groups responded to stress with increased physiological arousal. It is of little consequence to the present research whether physiological arousal is expressed through different systems according to the nature of the emotional disturbance. It is hypothesized that deep-breathing will result in lowered physiological arousal. Several physiological measurements were taken so as to assess whether the effects of deep breathing were general rather than specific.

It may be concluded that high physiological arousal is a phenomenon common to normal and psychodiagnostic groups. A more basic issue regards whether the effects of deep breathing upon physiological arousal would be comparable regardless of the initial arousal level; in the present study, the normal subjects in all probability had lower levels of physiological arousal than emotionally-disturbed groups (transitory or pathological). If deep breathing were to have varying effects upon physiological arousal, according to initial level of arousal, the

present/...

present study would be of limited value to lowering arousal in states of emotional disturbance. Physiological arousal varies in degree on a continuum between high and low levels of arousal. There is a maximum and a minimum level of arousal beyond which the organism could not function. Homeostatic mechanisms in the various systems have the function of maintaining a certain level of arousal which is most suitable to the efficient performance of metabolic activities (Ganong, 1971). These homeostatic mechanisms are present in the ANS (e.g. the baroreceptors of the carotid sinus and aortic arch); in the skeletal muscles (e.g. the spindle organs and gamma motoneurons) and in the respiratory system (e.g. the chemoreceptors of the carotid sinus) (Darrow, 1943; Gellhorn, 1953, 1954, 1967). If an organism has reached its maximum or minimum arousal level any attempts to initiate changes in the same direction will most often prove unsuccessful due to the homeostatic mechanisms responsible for maintaining level of arousal within certain limits. Such homeostatic restraint of response was investigated by Lacey (1956, 1967) who noted that the recorded autonomic response to a stimulus is a function both of the magnitude of the autonomic response, as it would be seen in the absence of contrary changes, and of the promptness and vigour of secondarily induced autonomic changes that serve to restrain and limit the effects of the initial disturbance. Applying these observations to the present study, the effects of deep breathing upon physiological arousal would only differ in direction of change if the initial arousal level were to be at its minimum or maximum value, and changes were to be attempted beyond these limits. In this research, this represents an unlikely possibility, i.e. only if all the subjects started off at their minimum arousal level on all of the physiological measurements taken, would such restraint of response be apparent. The consistent

presence of/...

presence of two-directional changes wohld suggest that such a phenomenon was occurring. However, the finding of a unidirectional lowering of physiological arousal in the present sample would indicate that deep breathing may be used to lower physiological arousal. However, further research would be needed so as to establish whether deeply disturbed patients are capable of following the instructions for the use of the technique. Deep breathing is a fairly simple technique for normal subjects to apply; but it could prove difficult to psychodiagnostic subjects were they incapable of maintaining their attention upon the breathing for any length of time. It should be emphasised however that the purpose of the present research was to firstly investigate whether deep breathing, when properly exercised, was reliably associated with lowering physiological arousal. If this were to be the case, it could be argued that deep breathing would be valuable in lowering physiological arousal in any subject who would successfully apply the technique, i.e.with minimal ability of concentration. It would be especially applicable to normal subjects suffering from transient anxiety.

Another theoretical issue to consider at this stage, regards the effectiveness of the physiological approach to improvement of maladjusted states: this depends upon the importance of the role of physiological symptoms in the emotionally-disturbed state. It is to be noted that emotions such as anxiety (pathological and non-pathological) are objectively assessed in a large part, by such techniques as interview of the subject and self-descriptive psychometric tests. Both of these techniques rely upon subjective state for diagnosis. It is thus important to determine the importance of the physiological state in determining the subjective emotional state.

Various views/...

Various views have been put forward as to the importance of physiological symptoms in maladjusted states; e.g. Wolpe (1971) defined anxiety as "an individual organism's characteristic constellation of autonomic responses to noxious stimulation, p.341". This view suggests that physiological criteria represent alternative means of describing anxiety from such a standpoint, the state of anxiety could no longer be experienced were its physiological correlates to be effectively removed. However, there has been evidence indicating that the physiological symptoms of emotional states are only components of such states. Other important aspects of emotion consist of cognitive and situational variables. Certain drug studies have been reported, which shed some light upon the role of physiological arousal in subjective emotional states. e.g. Schacter and Singer (1962) elicited radically different affective responses to a standardized infusion of epinephrine in normal subjects : subjects reported feelings of anger or euphoria, in a predictable fashion, according to experimental manipulation (i.e. amount of prior explanatory information about the effects of the injection; and the type of affective responses exhibited by stooges who the subjects thought had received the same injection as themselves). However, the placebo group, who had received a saline injection, failed to report any affective state in spite of having been subjected to the same experimental manipulation as the epinephrine group. It is apparent from this study, that the presence of physiological arousal (induced by epinephrine) was essential for the subject's experience of an emotional state. However, given the appropriate physiological state of arousal, negative or positive emotions could be experienced depending upon situational variables; it is noteworthy that those subjects who had been told in advance of the physiological effects of

epinephrine/...

epinephrine, did not report any emotional state despite the suggestion of such a state by the stooges behaviour. Thus, cognitive variables were also important.

Similar results were obtained on studies conducted on anxiety neurotics (Ackerman and Sachar, 1974, see review by). In these experiments, anxiety neurotics were given serum lactate injections (the effect of this injection is to simulate the physiological symptoms of anxiety, i.e. high physiological arousal). A significant proportion of the patients were reported to say that they did not experience "mental fear" or panic, "a fact that the patients attributed to the reassuring presence of the physician" p.76.

It is apparent from these reports, that the patients perceived and generally used their physiological state when interpreting their subjective feelings (i.e. anxiety). A certain proportion of the patients did in fact report feelings of anxiety in response to the serum lactate effects. However, given two contradictory sources of information regarding their subjective states, (e.g. physiological anxiety symptoms, and a reassuring environmental situation coupled with the knowledge of the effects of injection), some subjects would choose one of the sources of information, while fully aware, that given the appropriate set of environmental circumstances, their interpretations of their subjective state would have been based upon their physiological state.

Concerning the importance of physiological symptoms in determining a subjective emotional state, the following points arise out of these studies :

1. The presence of physiological symptoms of an emotion (such as anxiety) does not necessarily result in the subjective feelings usually associated with them.

2. The physiological/...

2. The physiological symptoms associated with a subjective emotional state are essential for the experience of that state - i.e. in the above reported studies, subjects given the placebo injection never reported a subjective emotional state, even when it was strongly suggested to them (Schacter and Singer, 1962). Thus, dealing efficiently with the physiological symptoms of unpleasant emotional states could prove effective in relieving such states.

In the next section (2.1), the psychophysiological research conducted on the physiological correlates of emotionally maladjusted states is reviewed, so as to support the consistent link between high physiological arousal and these states.

2.1. Research findings in abnormal psychophysiology.

In states of anxiety, high levels of physiological arousal have commonly been reported (Lader, 1975, see review by). Skeletal muscular tension has been reported to be higher in anxious subjects than in non-anxious, normal subjects (Balshan, 1962; Goldstein, 1964, and Smith, 1973). Certain autonomic measurements have also been found to distinguish anxious from non-anxious subjects in baseline rest conditions. e.g. Goldstein(1964 a) found anxious women patients to have a higher Heart Rate (HR) and systotic blood pressure than normal controls.

Taking several measures of autonomic function from which an overall score of autonomic balance, referred to as the $\bar{A}$ factor, was derived, Smith and Wenger (1965) reported predominant sympathetic activity in Ph.D. students about to take their oral examination, than in the same subjects one month after the examination.

Kelly et al (1970), using several psychometric tests, compared

subjects/...

subjects with high anxiety and depression ratings to matched controls on four autonomic measurements (forearm blood flow, HR, Skin Resistance (SR) and cutaneous vasomotor status). Only forearm blood flow and HR were found to be higher in the anxiety-depression group.

Friedman and Bennett (1977) reported a significant correlation between hypertension and anxiety in 834 outpatients.

Respiratory rate was reported to be faster at rest in anxious non-patient subjects diagnosed psychometrically (Balshan, 1962) and in anxious women-patients (Goldstein, 1964).

Thus, most studies reported above have found high sympathetic arousal, muscular tension and fast respiratory rate to be associated with anxiety in resting baseline conditions. The exception to this generalization is in measurements of electrodermal activity, which in some instances was found to be indicative of low sympathetic arousal e.g. (Goldstein, 1964).

A number of studies have dealt with physiological responses of anxious subjects to stimulation.

Significant differences between anxious and normal control subjects have been reported with greater consistency in experiments where subjects were exposed to some form of stimulation, expecially when stress was induced (Balshan, 1962; Alexander, 1972).

Balshan (1962) found that the differences in physiological arousal level in anxious subjects (as compared to non-anxious subjects) was accelerated in response to a one-minute noise than at rest : greater overall EMG tension and higher palmar skin conductance were observed during the noise.

Fenz (1964) found the skin resistance response to neutral and anxiety words to be much greater in parachutists on the day of their first jump than on previous days, and than other normal controls

on the/...

on the same day.

Goldstein (1964) noted that six out of the seven muscles from which EMG recordings were obtained showed more tension in anxious women patients in response to a white noise, than in normal controls. This contrasted with the finding of only two of the seven muscles being more tense in resting conditions. The anxious patients exhibited the same pattern of autonomic activity in response to noise as they did in baseline rest conditions i.e. higher HR, systolic blood pressure and lower skin conductance than normal controls.

Kelly et al (1970) came up with a puzzling finding when comparing anxious subjects with matched controls, first at rest and then during $2\frac{1}{2}$ minutes mental arithmetic under pressure : while the anxious subjects showed the expected trend of higher HR and forearm blood flow during rest, this trend was reversed during the stress task. This finding may have been due to the already high levels attained in these two physiological variables at rest, by the anxious patients. This finding could be interpreted in the light of Lacey's (1956, 1967) concept of the law of initial value i.e. that the higher the initial value of an autonomic function, the smaller will be its capacity for further increase before homeostatic mechanisms reverse this trend so as to restore a state of balance in the ANS. Thus, in a group of subjects exhibiting high resting levels of sympathetic activity, it may be postulated that when exposed to stress, a compensaotry, parasympathetic-like response could well be observed, thus seemingly yielding contradictory findings.

Bloom et al (1976) induced anxiety in female student subjects by threat of an electric shock. The group exposed to threat (96 subjects) had higher pulse rate, finger pulse volume and reported more anxiety on the Affect-Adjective Check List (AACL) than did the

non-threat/...

non-threat groups.

Research findings thus indicate that high physiological arousal is most frequently found in states of high anxiety as compared to low anxiety states. This was found to apply both to pathologically anxious patients relative to normal control groups. and to subjects with high scores on anxiety scales relative to low scorers.

High physiological arousal is a characteristic of most other psychopathological states, e.g. depression (McCarron, 1973; Oken et al, 1960; Reite et al, 1974; Whatmore and Ellis, 1962), schizophrenia (Berger, 1964; De Wolfe and Youkilis, 1975; Fenz and Velner, 1970; Malmo et al, 1951 b; Zahn et al, 1976).

Although there has been strong support indicating that high arousal was associated with emotionally disturbed states, the field of abnormal psychophysiology has been noted for its contradictory research findings (Alexander, 1972, see review by). For example, Kelly et al (1970) found no difference in testing forearm extensor EMG tension in subjects scoring high on the Taylor Manifest Anxiety Scale, as compared to subjects having low anxiety scores on this test. Goldstein (1964 a) found higher EMG tension in the same muscle group in anxious patients compared to normal controls. On the other hand, Goldstein (1964 a) found no difference between anxious and normal subjects in testing frontalis EMG tension, while Smith (1973) reported higher frontalis EMG tension in questionnaire rated anxiety subjects. Katkin and McCubbin (1969) found no relationship between Taylor Manifest Anxiety Scale (TMAS) scores and autonomic activity. Using the same test (TMAS) McFarland and Coombs (1974) found no difference in basal HR between subjects differing in anxiety scores.

Two main sources for the inconsistencies observed in abnormal psychophysiological correlations have been identified by researchers

in the field/...

in the field : technical difficulties e.g. type of physiological phenomenon under consideration (e.g. with electrodermal activity, various authors have referred to basal resistance, conductance, potential changes, number of unelicited electrodermal responses, and magnitude of elicited responses as indices of level of activity - Edelberg, 1972, see review by). Other technical difficulties refer to the influence of varying environmental variables (e.g. humidity and temperature) on certain physiological measurements e.g. skin resistance response (Eysenck, 1956). Furthermore, researchers have used groups differing as to degree of chronicity of psychopathology, length of hospitalization and diagnostic procedure e.g. Greenfield et al (1963) measured the electrodermal response (EDR) in depressed patients and normal control subjects. No relationship was found between psychiatric diagnosis and EDR. However, when subjects were separated into depressed and non-depressed groups on the basis of the MMPI depression scale, the depressed subjects were found to be low EDR responders.

A second major source of inconsistencies in abnormal psychophysiology, is no doubt genuine differences in physiological functioning existing between individuals. e.g. Lacey (1950. 1956, 1967) and Lacey et al (1953) have investigated individual physiological response patterns, and a brief summary of their conclusions was put forward below :

1. In any given situation, some individuals react more in one autonomic function than in another.

2. In a specific situation, individuals differ as to which function they react most in.

3. Some individuals always react with the same response pattern regardless of the type of stress; others vary from stress to stress.

The most/...

The most comprehensive effort to study psychopathological groups in terms of certain physiological response patterns was undertaken by Shipman and Colleagues with a certain amount of success. (Heath et al, 1965; Shipman et al, 1964; Shipman et al, 1970 a and 1970 b). However, some of their findings were negative (Heath et al, 1965). Response patterns were not found to be consistent in relation to personality variables; instead, individuals were found to show a consistent response pattern to various situations (Shipman et al, 1970 a and 1970 b). The response pattern approach could only prove valuable in limited cases, e.g. to psychotherapists who are able to spend the time required in finding the appropriate physiological response pattern applicable to each individual patient.

In spite of the inconsistencies which have been found in abnormal psychophysiological correlations, there are certain generalizations which may be made in the field, which have received widespread experimental support. These generalizations refer to principles of physiological function differentiating normal and emotionally disturbed states. Two such criteria were found to consistently differentiate between emotionally disturbed and normal groups.

1. Emotionally disturbed subjects were found to have higher levels of autonomic and somatic arousal (See Section 2.2).

2. Psychodiagnostic groups have been shown to have impaired homeostatic functioning as compared to normal control groups (See Section 2.3).

2.2. High physiological arousal as a defining criterion of maladjusted states.

High physiological arousal at rest has frequently been associated

with emotionally/...

with emotionally disturbed states (Alexander, 1972, see review by). When several physiological measurements are taken, most researchers have reported high arousal in one or more of these functions; e.g. Goldstein (1964 a) found greater EMG tension in 6 out of 7 skeletal muscles in anxious patients compared to normal controls. Kelly et al (1970) found anxiety-depressive patients to be more autonomically aroused on two out of four functions, than were normal control groups. For the aims of the present research, it was of little importance that high arousal could be manifested in only certain autonomic or somatic functions. The deep breathing technique had the aim of having the general effect of lowering arousal in whichever functions that were relatively overaroused. For this purpose, several physiological measurements were taken.

The main body of research findings indicating that high physiological arousal was characteristic of the various psychodiagnostic groups has already been presented in Section 2.1.

2.3. Deficient homeostatic functioning as a defining criterion of maladjusted states.

It should be noted that the present criterion has mainly been derived from studies of psychodiagnostic groups. It is thus not to be investigated in this research which was conducted on normal subjects. It is however noteworthy that poor homeostatic functioning has been a characteristic of maladjustment and should be investigated in future research on the effects of deep breathing.

In summary, in a physiological system where there is adequate homeostatic functioning, it may be expected that :

1. There will be a fairly stable state of arousal in the various physiological/...

various physiological systems, as homeostatic reflex mechanisms will be maintaining arousal as near as possible to the normal set-point (Ganong, 1971). Psychodiagnostic groups have been reported to show more fluctuation or unelicited physiological responses than normal control groups : e.g. Psychodiagnostic groups have shown more irregularity in respiratory pattern (Malmo and Shagass, 1949; Jurko et al, 1952); more irregularity in electromyographic (EMG) responses (Malmo et al, 1951 a; Malmo et al, 1951 b) and more abrupt body movements (De Wolfe and Youkilis, 1975); more heart rate instability (Altschule, 1953, see review by; Alexander, 1972, see review by; Malmo and Shagass, 1949); more spontaneous (unelicited) electrodermal responses (Bernstein, 1965; Dengerink, 1971; Gatchel and Proctor, 1976 (in experimentally stressed group), McCarron, 1973; Zahn et al, 1976); more variability in second-to-second analysis of the EEG (McCarron, 1973); Alexander et al (1963) reported more high frequency oscillation in skin resistance, finger blood volume, HR and EMG in a mixed psychiatric patient group when compared to normal controls. Van de Velde (1976) in a ten year longitudinal study on schizophrenics observed abnormally frequent shifts in type of symptom reported, response to drugs, body weight and daily rectal temperature.

2. In the presence of a startling stimulus, a temporary state of high physiological arousal may be expected to result. If there is efficient homeostatic functioning, there will be quick restraint of this arousal response. Psychodiagnostic groups have generally been observed to be over-responsive to environmental stimulation in autonomic/somatic nervous systems than normal controls, e.g. In response to stress, psychopathological groups exhibited higher HR responses (Fenz, 1971; Hare, 1974; Valins, 1967); low electrodermal

responsiveness/...

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responsiveness/...

responsiveness (Fenz and Velner, 1970; Hare and Craigen, 1974; Stern et al, 1965); higher EMG responsiveness (Malmo et al, 1951 b; Balshan, 1962; Goldstein, 1964, 1965; Fenz and Velner, 1965); higher blood pressure responses (Malmo et al, 1951 b; Goldstein 1964, 1965); higher respiratory rate in response to stress (Fenz and Velner, 1970). Balshan (1962) reported more palmar skin conductance increases in anxious patients than in normal controls.

3. Pre-stimulus levels of physiological arousal (referred to by Ganong (1971) as the "normal setpoint") will be restored at a rapid rate after a disturbance, by efficient homeostatic functioning. Slower physiological recovery in autonomic and somatic nervous systems after a disturbance have been a characteristic of psychodiagnostic groups compared to normal controls, e.g. Malmo et al (1951 a) reported that psychoneurotics showed persistence of arterial blood pressure at high levels throughout a stress experiment. Similar findings were reported concerning slow recovery of the EDR in psychodiagnostic subjects (Depue, 1976; Eysenck, 1956; Hare, 1968).

These observations concerning deficient homeostatic functioning in psychodiagnostic groups is of theoretical interest to the present research, i.e. in emergency situations, such as emotional disturbances, physiological arousal increases over the normal setpoint. In this context, psychodiagnostic groups have been said to react to ordinary life situations as if they were "emergencies" (Cannon, 1915). Constant interference could realistically lead to disbalance of homeostatic functions, as the latter unsuccessfully strive to maintain and restore its normal setpoint. Thus, subjects could be taught to practice deep breathing so as to prevent overarousal from occurring, or to rapidly restore the normal setpoint when overarousal could not be avoided.

In/...

In the long-term, restoration and maintenance of normal arousal levels, through the deep breathing technique could be postulated to simultaneously restore normal homeostatic functioning. The investigation of such a phenonemon would require long-term observations of psychodiagnostic groups with clear homeostatic deficiencies coupled with physiological overarousal. However, the first step in such an investigation is to establish that deep breathing is associated with lowering of physiological arousal; this therefore was the aim of the present research.

2.4. Practical approaches in abnormal psychophysiology

There have been several techniques which have been used in abnormal psychophysiology in dealing with emotional maladjustment. These techniques generally have had one aim in common : that of lowering physiological arousal, e.g. muscular relaxation was reported to be incompatible with anxiety by Jacobson (1938), and the progressive muscular relaxation technique had been used in behaviour-therapy to eliminate states of anxiety and such phobic behaviour (e.g. Wolpe, 1958).

This view that lowering of physiological arousal be therapeutic to anxiety states received support from animal studies, e.g. Anderson and Brady (1976) and Corwin et al (1926) reported experiments in which conditioned fear responses in animals were prevented by beta-adrenergic (sympathetic) blockade or vagal (parasympathetic) stimulation; these two experiments indicated that conditioned fear responses (which are physiologically comparable to anxiety) are to a large extent mediated by the sympathetic nervous system. It is thus of some significance that induced states of low physiological a arousal, e.g. Transcendental Meditation (Kanellakos and Lukas, 1973;

have been reported/...

have been reported to have long-term psychotherapeutic effects, e.g. Boudreau (1972); Tjoa (1972); Hough (1975, see review by).

Mostly, techniques developed with the aim of eliminating high arousal states have dealt with specific physiological functions showing overarousal, e.g. decreasing of frontalis muscle tension using biofeedback was reported to relieve tension headaches (Budsynski et al, 1973). Welgan (1974) successfully decreased gastric acid secretion in ulcer patients using biofeedback. Biofeedback of relevant muscles was reported to relieve chronic asthma and Parkinsonian disease (Stenn et al, 1976). The use of biofeedback to increase hand temperature was reported by Sargent et al (1973) and Turin and Johnson (1976) to successfully relieve migraine and tension headaches; this method was derived from Luthe's (1963) method of Autogenic Training. Other specific uses of biofeedback have been to decrease total respiratory resistance in airways in treatment of asthma (Levenson, 1974; Feldman, 1976; Tal and Miklich, 1976; Vachon and Rich, 1976); voluntary production of high alpha abundance was reported to relieve phobias (Keefe, 1971) and prevent recurrent headaches (Gannon and Sternbach, 1971). The main difference between biofeedback techniques and the presently used deep breathing technique, is the emphasis laid by the latter technique upon altering physiological function in a generalized rather than specific manner. This preference for a non-specific approach to lowering high arousal regards not only to more widespread applicability of such a technique, but also in certain dangers inherent in biofeedback techniques in which specific systems are tampered with, e.g. Obrist (1975) after extensive research into the cardiovascular system has pointed out that there were possible dangers in interfering with individual physiological systems, i.e.

"Any interference/...

"Any interference with such function such as by decreasing kidney blood flow, immediately initiates a series of compensatory adjustments by the cardiovascular system so profound that a permanent hypertension can ensue" (p.104).

There have been a number of techniques however, which have had the aim of inducing a general state of physical relaxation with therapeutical aims. For instance, sleep disturbances have been treated by electrosleep : a small current is circulated through electrodes placed above the eye and behind the ear; the immediate effects are relaxed drowsiness (Borkovec and Weerts, 1976; Rosenthal and Wulfsohn, 1970; Sergeev, 1963). This method was reported to have been successful in desensitizing a phobia (Feinberg et al., 1974). There are two main drawbacks with using electrosleep : firstly the need for highly specialized equipment; and the fact that the application of external stimulation such as an electric current may have unknown undesirable side-effects.

Luthe's (1963) use of autogenic training is the closest parallel technique to deep breathing, both aiming at a general state of relaxation. In autogenic training, the subject repeats certain phrases suggesting feelings of heaviness and warmth (generally associated with relaxation) in each part of the body. A combination of the autogenic training procedure and biofeedback (the latter informing the subject as to the success of his efforts at hand warming) was reported as effectively relieving headaches (Sargent et al., 1963; Turin and Johnson, 1976). This technique of autogenic training, as used by Luthe (1963) has the effect of inducing a lowered state of physiological arousal in a generalized manner. The difference between autogenic training and deep breathing consists of procedural differences in achieving relaxation. Autogenic training requires the subject to

concentrate/...

concentrate in turn on the various parts of his body, suggesting to himself that feelings of warmth and heaviness are experienced. Deep breathing consists of concentrating more passively on a single physiological process, i.e. that of breathing deeply in a systematic way. The only difference which is immediately apparent between the two techniques, is that autogenic training requires a great deal more concentration upon the procedure involved, while minimal effort is required to practice deep breathing, due to the repetitive nature of the technique. In a subject who finds it difficult to remain relaxed, autogenic training may prove difficult in that feelings of warmth and heaviness induced in various parts of the body may not be maintained as the subject proceeds to other areas. In summary, the two techniques, if properly practiced, may be expected to have very similar effects i.e. lowering physiological arousal in a generalized manner. However, the techniques involve different procedures which may cause one or the other to be more easily applicable in different subject populations. For example, emotionally disturbed subjects who have concentration problems would probably find it easier to practice a repetitive task such as deep breathing. Other subjects may find a monotonous task difficult to follow, and would more readily benefit from autogenic training.

A few studies have reported the use of respiratory techniques as a psychophysiological approach to psychotherapy. Grim (1971) found that respiratory feedback facilitated relaxation after self-induced muscle tension resulting in anxiety. Harris et al (1976) used a method of paced respiration where a light was switched on and off in order to halve breathing rate - the variable of attention was controlled for. The paced respiration group showed fewer and smaller electrodermal responses than the control group in response to painful

electric shock. Heart rate, however, showed no significant differences between groups. These respiratory techniques studies suggest that breathing may be a means of altering overall physiological function; in addition they require little specialized equipment in their application. In the present study the possibility of using a respiratory technique having the general effect of lowering autonomic and somatic arousal level was investigated; the technique used required no equipment: subjects simply received instructions as to how to alter their breathing. It was considered important to let the subjects perform their own respiratory adjustments according to whether they felt comfortable or not : this prevented long-term undesirable side-effects such as over or under breathing; such effects could have more easily occurred if equipment over which the subjects had no control had been used in regulation of breathing pattern. The rationale for using the technique of deep breathing, with a view of inducing widespread changes in the autonomic and somatic nervous systems is presented in the next section.

3. The Technique of Deep Breathing as a Means of Improving Adjustment in Terms of the Criterion of Physiological Arousal

3.1. Description of the technique of deep breathing

The technique of deep breathing consisted of the following elements :

1. A passive concentration on the air flowing in and out of the lungs as the subject breathed.
2. The use of the full lung, from abdominal to thoracic regions, at each breath.
3. The filling of the lungs from the abdominal (bottom) to the thoracic (top) regions.
4. The emptying of the lungs from thoracic to abdominal regions (top to bottom).
5. Each complete breath was to be taken slowly (so as to prevent hyperventilation).

3.2. Psychophysiological studies indicating a link between respiration and physiological arousal.

Psychophysiological research has reported a correlation between physiological arousal and respiratory activity, e.g. Goldie and Green (1961) have found respiration to be a good index of arousal. They reported thoracic respiration in drowsy subjects, shifting to abdominal breathing as the subject became aroused. Bülow (1963) found both respiration and EEG frequency to be good indicators of arousal. He also points to a correlation between respiratory, circulatory, muscular autonomic functions and general changes in the EEG along

the continuum of arousal. In his studies he found, for example, that a switch from alpha to theta rhythm in the EEG was always accompanied by a decrease in ventilation. With increases in level of wakefulness there was increase in ventilation. This finding was supported by Aserinsky (1965) who reported that during sleep, when the eyes are in motion there was an increase in alpha activity together with a depression of ventilation, and a possible slowing of the heart.

On a similar line, Timmons et al (1972) reported that during sleep a reduction of the amplitude of the abdominal respiratory movement was concomitant with transitions from alpha to theta in the EEG. With larger amplitudes of abdominal respiratory movements (which may be seen as increases in ventilation), 81% of the transitions from theta to alpha occurred. The authors suggest manipulating respiratory patterns to alter 'state of consciousness'.

These studies, therefore, reinforce the notion of respiration not being independent of physiological function as a whole, but changing in behavioural states such as arousal and relaxation.

Other indications arising out of psychophysiological research, as to the possible link between respiratory activity and arousal, are with regard to its use as a means of relaxation. Grim (1971) found that decreasing respiratory activity through the use of feedback facilitated the process of muscular relaxation.

Harris et al (1976) reported that electrodermal responses to tones and shock were smaller in a group instructed in slowing down their respiratory rate than in a Control group. In that study, the factor of 'paying attention' to respiratory activity was controlled for by having the control group performing an attention task (counting the light flashes to which the experimental group was synchronizing their respiratory rate).

3.3. The predicted effect of deep breathing upon physiological function.

The experimenter was unable to find research either from physiological or psychophysiological literature, dealing with the effects of various modes of breathing upon physiological function. Thus, it would have been difficult to predict the physiological effects of deep breathing from an objective standpoint. However, the experimenter had had the opportunity to observe, during the course of certain pilot studies (one of them performed on an experienced Yoga student) that decreased physiological arousal (specifically in the form of large increases in skin resistance) accompanied the practice of deep breathing; subjective reports indicated that a state of relaxation was experienced during the exercise of the deep breathing technique. Further, it was found that deep breathing was used concurrently with other relaxation techniques by physiotherapists (White, 1975). In a personal communication with Miss White (1975) it became apparent that the relaxing effects of deep breathing were recognized, although the reasons for the effectiveness of the technique were not known. It was thus thought useful to conduct an exploratory study into the effects of the deep breathing technique upon various physiological parameters, i.e. upon skin resistance (SR), heart rate (HR), masseter and corrugator muscle tension and finally the occipital electroencephalogram (EEG). The latter measurement had to be discarded due to faulty analysis of a great portion of the data (see Section 4.2.7.). It was predicted on the basis of the pilot studies conducted, and the evidence from subjective reports, that deep breathing would lower arousal level, as indicated by increased SR level, decreased HR and muscular tension. Deep breathing was compared

to the/...

to the technique of muscular relaxation, so as to assess whether the two techniques differed from one another in their effect upon physiological arousal. Three baseline and three treatment sessions were run: it was expected that the deep breathing and muscular relaxation groups would show significantly greater decreases in physiological arousal in treatment as compared to baseline sessions.

Deep breathing was an easy technique to learn and practice. It was thought possible that deep breathing could prove useful in restoring low levels of physiological arousal in psychopathological groups such as anxiety neurotics. It was pointed out in Section 2 that perception of physiological state played a crucial role in a subject's judgement of his subjective state. If it were established in the present reaearch that deep breathing successfully and reliably lowered physiological arousal in normal subjects, further research could then be carried out using psychopathological groups.

In summary, it was postulated that the deep breathing technique would have the effect of lowering physiological arousal, in a similar way as is observed during muscular relaxation. The present research had primarily exploratory aims.

4. Methodology

4.1. Subjects.

The subjects in the present study comprised of 12 subject volunteers, 9 of whom were recruited from the Psychology I course at U.C.T. The other 3 subjects were also university students at U.C.T; two of them were recruited by one of the Psychology I students. The other one was a friend of the experimenter's. They ranged in age between 17 - 26 years with a mean age of 18,75 years.

Subjects were required to fill in a questionnaire in which they were asked if they suffered from any form of chronic illness at the time of the experiment (e.g. asthma). All the subjects were found to be in good health at the time of the experiment, and none reported being on any form of medication.

Those subjects who continued participating in the experiment to the end proved extremely co-operative. Good rapport was established with all subjects by the end of the habituation session (which was the second encounter between the experimenter and the subjects).

4.1.1. The Experimental Group

The experimental group consisted of 6 subjects, with a mean age of 18,33 years. There were three males and three females in this group.

The experimental group were instructed in the technique of deep breathing by the use of certain exercises (described in Section 4.3.6.) during a single training session of approximately 30 minutes.

4.1.2./...

4.1.2. The Control Group

The control group consisted of 6 subjects, with a mean age of 19,17 years. The group comprised itself of 3 males and 3 females.

The control group were instructed in relaxation technique making use of muscular contraction-relaxation exercises which they learnt in a single training session of approximately 30 minutes (described in Section 4.3.7.).

4.2. Apparatus

4.2.1. The Experimental Setting

The subjects were seated in a medium sized room which had the appearance of a bedroom. The apparatus was in an adjoining room, the wires passing through the wall separating the two rooms, to a console mounted next to the subject; the electrodes were plugged into the console. The subject could thus neither see nor hear the equipment which was in the laboratory.

The subject was seated on a bean bag. A switch signalling system was placed at the approximate level of the subject's right hand. The room also contained a buzzer, which the experimenter used to signal certain instructions to the subject. The purpose of this signalling device (switches and buzzer) is fully explained in a subsequent section (4.3.3.).

4.2.2. The Dynograph

A six channel Beckman Dynograph Type RP was used for all the physiological measurements, with its accompanying pre-amplifiers (Type 482 and 481b), and 3 Power amplifiers Type 482 M8. The

settings/...

settings and coupler used for each function monitored is described separately below.

The data was printed on Beckman chart paper by 6 pen galvanometers. The operation paper speed was run at 1 cm per second.

4.2.3. The Tape Recorder

All the data was recorded on an Ampex Magnetic Tape Recorder Model PR - 500 run at 1 $^{7}/_{8}$ IPS (inches per second).

Unfortunately, only one magnetic tape reel was available to the experimenter, so that all the data could not be stored on tape. When the reel was filled, the EEG data was analysed, and the reel erased so as to make space for further data.

4.2.4. The Electrodes

Beckman miniature biopotential skin electrodes (11 mm) was used for all measurements, excluding the EEG and respiration (electrodes used for EEG were described under the EEG equipment section).

The centre part of the Beckman electrode is the sensing element, consisting of a silver-silver chloride pellet.

Beckman Bulletin 5047 give the following specifications concerning the bipolar electrodes :

Offset voltage	:	less than 250 microvolts after pre-soaking in saline.
Electrode-to-skin offset voltage	:	300 microvolts (varying according to type of electrolyte paste used and condition of skin

Polarization/...

Polarization voltage	:	5 microvolts per 24 hours with electrodes placed face to face in saline at constant temperature.
Electrode-to-skin impedance	:	500 ohms using standard ECG lead with normal skin preparation. 100 ohms in other applications.

A low impedance lead was used for skin resistance recordings, thus it may be assumed that electrode-to-skin impedance for SR was 100 ohms.

4.2.5. Skin Resistance Equipment

A Beckman GSR coupler Type 9842 was used on the Time Mark System. This yielded data in a digital form.

Two Beckman miniature biopotential electrodes (described in Section 4.2.4.) were used for measurement of palmar skin resistance to a 10 ua constant current.

4.2.6. Heart Rate Equipment

A Beckman A-C D-C coupler Type 9806 A, functioning in the A-C mode was used for recordings of heart rate.

Two Beckman miniature biopotential electrodes (described in Section 4.2.4.) were placed over the chest area.

4.2.7. EEG Equipment

The EEG was recorded using an A-C D-C coupler Type 9806 A functioning in the A-C mode. Round silver electrodes (a.5 cms diameter) were used for the EEG. These were disposable electrodes,

but could/...

but could be used repeatedly by adhering a new strip of stick-on foam over the electrode at each application. It was ensured that the foam was not too sticky but simply loosely adhered to the hair. Two hair clips were clamped on either side of each electrode, over the protruding foam. A hair net was then tied over the entire head. This arrangement was chosen after having tried many other types of electrodes, because of its proving to be the most stable means of placing electrodes on the scalp. The relatively large, flat surface of the electrodes ensured good contact, and provided a quick way of application. This means of securing the electrodes had the great advantage of not requiring the usual discomforts associated with for example, rubber bonnets which have to be fastened below the chin. The hair net was tied at the back of the neck and the subject was in no way inconvenienced by its presence during the experiment.

The EEG data was analysed by an EEG Power Spectrum Analyser, built for the purposes of the present experiment by Mr. A. Timm (M.Sc Electric Engineering, U.C.T.).

Unfortunately, when the EEG Analyser was first designed, it was found to accept too restricted an amplitude range. Thus, the very high theta amplitudes and the very low beta amplitudes could not be coped with. The Analyser was sent to Johannesburg to Mr. Timm for modification. Mr. Timm was not familiar with the EEG signal and had restricted the amplitude range of the Analyser so as to keep the costs of the apparatus to a minimum, as Mr. Timm was aware of the experimenter's restricted funds. After the appropriate modifications had been made to the analyser, it proved to be faultless and provided a most useful research tool which could be used in future research. Very little of the EEG data was available in this research,

however/...

however, due mainly to the shortage of magnetic tape reels (which had to be ordered from the U.S.A.) It proved impossible to keep, for example, one common baseline and treatment session for all subjects, thus allowing some meaningful statistical analysis and comparison, as some subjects started before the Easter vacation, some who went away for the vacation only started later. Due to the initial large drop-out rate, certain subjects were recruited much later after the first pre-vacation group; thus, the subjects did not attend the same sessions during the same period. However, a full description of the modified Analyser, its validation data, design diagrams, scoring procedure as well as a few samples demonstrating the relationship between the Analyser's results and visual inspection of the EEG tracing were included in the Appendix for the benefit of future researchers.

One great advantage in the EEG analyser, was that it was designed so as to analyse the EEG data 32 times faster than the recording speed, i.e. the EEG signal was recorded on the FM tape at the speed of 1 $^{7}/_{8}$ IPS (inches per second) and played back through the EEG Power Spectrum Analyser at 32 times the recording speed, i.e. at 30 IPS. The advantage in reproducing the data at 32 times faster during analysis, was that the data was analysed at great speed, thus saving a lot of time, e.g. 30 minutes recording was analysed in less than a minute.

4.2.8. EMG (Electromyography)Equipment

An A-C D-C coupler Type 9806 A was used in the A-C mode for recording of surface EMG.

Beckman miniature biopotential electrodes (described in Section

4.2.4./...

4.2.4.) were used for recording of EMGs.

4.2.9. Respiratory Rate Equipment

A Beckman strain gauge coupler Type 9803 was used together with a respiratory transducer consisting of an expandable belt containing 4 strain gauges.

The respiratory transducer was built for the purposes of the present experiment and was designed for maximum sensitivity. The 4 strain gauges were built into a flexible metal triangle which was strapped around the chest by an adjustable belt. As the subject inspired and expired the changes in pressure caused by expansion of the chest muscles at each breath were transduced into electrical recordings and traced on paper by the pen galvanometers.

It was observed that during breath holding after a normal inspiration the strain gauge system was able to detect changes in pressure arising from heart beat. Thus, it could be assumed that the respiratory transducer was sensitive enough to record the least respiratory movement, permitting approximations of respiratory amplitude, as well as precise recordings of breathing rate.

4.3. Procedure

4.3.1. Electrophysiological Recording Procedure.

As soon as the subject reported to the laboratory, he was instructed to sit upright on the bean bag and make himself comfortable.

The skin electrodes were attached first (EMG, skin resistance and earth electrodes). Each of the appropriate electrode sites were cleaned with cotton wool which had previously been immersed in

alcohol/..

alcohol. DB electrode paste was rubbed into the skin until most of it had been absorbed: a dry tissue was then used to wipe off excess electrode paste so as to leave the skin surface dry. The centre part of each electrode was filled with electrode paste, and the electrode (mounted on a circular sticky collar) was then stuck on firmly over the appropriate site. A ground electrode which was common to all transducers was placed on the back of the right hand.

The EEG electrodes were applied in a manner similar to that described for the skin electrodes. However, the electrode paste was not wiped off by a dry tissue after rubbing it in. Instead, the electrode, covered with a thin layer of electrode paste, was applied over the appropriate areas, and the adhesive piece of foam was gently pushed down around the electrode site: two hair clips were then used over the foam so as to clamp down both sides of the electrode. A hair net was placed over the whole head and tied firmly at the back of the head.

The respiratory transducer, mounted on an adjustable belt was tied around the chest.

All the leads plugged into a console, and from there went through the wall via electrically shielded leads to the couplers of the dynograph situated in the adjacent monitoring room. The experimenter then checked each channel for artifacts and, if necessary, made adjustments to the electrodes or monitoring apparatus. The subject was then told that the experiment had started, was briefly reminded of the instructions, the door was closed and the subject was left alone in the room with a small reading lamp on. At the end of the session the experimenter opened the door, put the main lights on

and/...

and proceeded to unwire the subject and clean the skin with cotton wool immersed in water. The electrode paste was found to come loose quite easily with water, and the subjects preferred the use of water to alcohol for the cleaning up of the skin.

The procedure for monitoring each of the different physiological functions is described separately below :

a. Skin Resistance -

The GSR coupler Type 9862 was used on the Time Mark System. This yielded digital data, amenable to manual scoring procedures. The electrodes were placed on the subject's left hand, one on the palmar area, and the other on the bone of the lower wrist. The electrode placement is illustrated in the photograph below :

The GSR coupler setting was on operate, a 10 ua constant current was circulated, and the toggle switch, selecting the mode

of operation/...

of operation of the coupler was set on TM (Time Mark System)

The pre-amplifier was switched off, as the GSR coupler used on the Time Mark System by-passed the pre-amplifier. The power amplifier was set at X.1. The frequency selector on the power amp was in the Hi-out position, and the high frequency filter set on 3.

The Time Mark System operated by tracing marker pips of fixed amplitude: the number of markers per second were directly proportional to the value of basal resistance. Resistance values in kohms could thus be derived from the marker data, using the linear equation $y = bx + c$ (the transformation of number of marker pips per second into kohms is described in Section 4.4.1.)

b. Heart Rate -

The A-C coupler type 9806 A was set up as follows :

Time constant	: .03
Hi-frequency filter	: 3
Pre-amplifier	: 1 mv/cm
Power amplifier	: X.1
Power amplifier amplitude filter switch	: Hi-out

Heart rate was measured by electrodes placed over the upper chest area. The exact placement of the electrodes differed slightly according to the type of clothing worn by the subject. However, the two electrodes were placed roughly under the collar bone (left or right) and 10 cms below the neck in the middle of the chest. This electrode placement is illustrated in the

photograph/...

photograph below :

This electrode placement was chosen so as to avoid the chest muscles which would have caused interference on the recording.

c. The Electroencephalogram (EEG) -

The settings used on A-C coupler Type 9806 A were :

Time constant	:	0.3
High frequency filter	:	3
Pre-amplifier	:	0.5 mv/cm
Power amplifier	:	X.1.
Power amplifier amplitude filter switch	:	Hi-out

Bipolar EEG recordings were obtained from the occipital

areas/...

areas: the O1 and O2 positions were used to record the EEG (Jasper 10-20 system, 1958).

d. Muscular potential recordings (electromyography) -

The EMG recordings were taken from the corrugator and left masseter muscles.

The corrugator muscle is situated above the outer third of the superciliary arch; its function is to wrinkle the forehead in vertical folds (Walthard and Tchicaloff, 1961). This muscle was chosen because of its being the 'frowning' muscle, i.e. when contracted the individual is said to be frowning. It was the opinion of the experimenter that this group of muscles would give a good indication of muscular tension in the face.

The electrodes were placed by asking the subject to frown. The position of the muscle of both sides of the eyebrows was noted by the experimenter, by placing two fingers over the required spots, and the electrodes were immediately stuck on.

The masseter muscle is situated in front of the tragus above the angle of the jaw; its function is to click the lower against the upper teeth, with the mouth in the usual position of closure (Walthard and Tchicaloff, 1961).

The masseter muscles are the ones which are tensed up when the jaw is clenched; this was also postulated to be a frequently tensed muscle, and thus appropriate for measuring facial muscle tension.

The electrodes were placed by asking the subject to clench his jaw with mouth shut. The masseter muscle of the left jaw moved as this task was done, sometimes remaining slightly protruding. In such cases, the electrodes could be placed by

visual/...

visual inspection of the muscle profile. If the experimenter was unsure of whether the muscle was adequately detectable visually, the muscle was found by feeling the upper jaw area: in the jaws clenched position, the masseter muscle is a hard lump easily detected by touch.

The A-C couple Type 9806 A was set as follows :

Time constant	:	0.03 (cut-off frequency 5.3 Hz)
High frequency filter	:	2
Pre-amplifier	:	1 mv/cm
Power amplifier	:	X.1
Power amplifier amplitude filter switch	:	Hi-out

4.3.2. Experimental Design

The experiment consisted of a total of 8 sessions per subject. It had originally been planned to have 10 sessions instead of 8. However, the difficulty involved in subject recruitment for such a lengthy experiment forced the experimenter to reduce the number of sessions.

The 8 sessions were divided as follows :

FIGURE 1

	30 MINUTE SESSIONS			
1 Habituation Session (no measurement taken	2+3 Pre-treatment baselines	4 Training	5+6+7 Treatment	8 Post-treatment baseline

Each/...

Each session (except for the training session, No. 5) was subdivided into three phases, which are illustrated in Figure 2.

FIGURE 2

PHASES WITHIN EACH 30 MINUTE SESSION					
	First Phase	BUZZER	Second Phase	BUZZER	Third Phase
Baseline Instruct-ions	5 minutes Sit still Eyes closed		12½ minutes Sit still Eyes closed		7½ minutes Sit still Eyes closed
Treatment Instruction	Sit still Eyes closed		Relax/deep breathing		Sit still Eyes closed

Description of the three phases of the sessions -

Phase 1 :

This consisted of 5 minutes during which physiological functioning was allowed to settle down to a steady baseline. It should be noted that the subject had been seated for at least 15-20 minutes prior to this first phases of the session, during the wiring-up process. Five minutes was thus sufficient, most functions having settled down after the second minute (e.g. HR, EMG, EEG) and SR taking slightly longer.

At the end of Phase 1, a buzzer was sounded by the experimenter so as to signal the subject that he had reached the end of the first phase. The subject was required to respond to the buzzer by opening their eyes, and operating a toggle switch which made a light come on in the experimenter's room. Once the light had been switched off by the subject another buzzer was sounded to indicate to the subject

to close/...

to close his eyes and continue with the session.

Phase 2 :

12½ minutes. Only the last 10 minutes of this phase were statistically analysed, as it was considered that the signalling procedure between the two phases could have caused a certain amount of physiological disturbance; the first 2½ minutes would allow normalisation of physiological function.

After the second phase, exactly the same signalling procedure was used as was described after the first phase.

Phase 3 :

7½ minutes, of which only the last 5 minutes were used in the statistical analysis, for the same reasons as given for the omission of the first 2½ minutes of Phase 2 of the session.

At the end of Phase 3, the experimenter opened the door, put the light on, and proceeded to unwire the subject.

4.3.3. Experimental Procedure

Prior to the first habituation session, the subject was taken round the laboratory so as to view the experimental setting. The experimenter explained where the electrodes would be placed. The subjects were told during this first encounter that the experiment would involve 10 sessions run over a period of 3 to 4 weeks. Due to the high drop-out rate following the disclosure of the length of the experiment, the experimenter reduced the number of sessions to 8, run over 2½ to 3 weeks.

The experimenter was deliberately vague concerning the purpose of the experiment: the subjects were told that it would mainly

involve/...

involve the investigation of normal physiological function 'as it would be if they had to be sitting still for a while'. The experimenter hinted at the practice of certain 'exercises' later on in the experiment, but the subject was not told the nature of the exercises, as 'it had not been decided yet which ones he'd be doing'.

When finally the training session came up, the subject would be told that the exercises consisted of either breathing or muscular relaxation. The experimenter was careful to put across a certain amount of doubt concerning the possible usefulness of the training and of the exercises. This was done so as to confuse the subject concerning the aims of the experiment, thus preventing him from knowing what was expected of him, on how to please the experimenter: the latter attitudes in subjects were found in a pilot study to be variables interfering with the objectivity of the subject, causing tension and thus defeating the aims of the research. Emphasis was laid on 'doing the exercises' and the concept of relaxation was avoided as much as possible in either group.

The subject received relatively lengthy instruction before the first session run, where he was given the following information. These instructions were never read out to the subject. The experimenter memorized the contents and repeated them to the subject; it can be expected that in some instances a few words were replaced by others. However, the content was roughly as follows, and was given to the subject in the order presented below :

Instructions given before the first experimental session :

a. Each session which you will attend will be divided into three parts. I shall signal you at the end of the first, and

again/...

again after the second part of the session, by sounding a buzzer. (The experimenter then walked out of the room into the adjoining laboratory and pressed the buzzer, then returned and made sure the subject had heard it - if it was reported to have startled them, a pillow was put over the buzzer so as to muffle the sound).

b. When you hear the buzzer for the first time you will then know that the first part of the session is over. You must then open your eyes, and signal me with one of these switches near your right hand. (The subject was shown the three switches. There was a green, a yellow and a red one). Which switch you will select will depend on how you felt during the time you were sitting before I sounded the buzzer. If you felt drowsy, press the green switch. If you were particularly alert or restless, press the red switch. If you cannot make up your mind, or felt you were neither one more than the other, press the yellow switch. (This switch system was introduced for two reasons, outlined at the end of the present section).

c. Once you have pulled down the switch which you have chosen, put it up again, as otherwise I cannot use the buzzer. After you have closed up the switch, I shall buzz you again immediately. You will then close your eyes again and sit still for the second part of the session. This part is the longest one. Try not to get restless, nor to fall asleep. At the end of this second phase, I shall again buzz you. We shall then repeat the same procedure as last time: you select the appropriate switch pull it down then switch it off. After the second buzz, close your eyes for the last part of the session.

d. At the end of the last part of the session, I shall just walk into the room and unwire you.

e./...

e. Please remember not to fall asleep during the session.

The subject was given as much explanation as he asked for concerning the signalling system. During the habituation (first) session, red, yellow, and green transparent papers were stuck on to a piece of cardboard containing three square holes. On the green paper was written, with a black cokey pen : low alertness. On the red paper : high alertness. Nothing was written on the yellow paper. This piece of cardboard was placed in front of the dim light which was left on during the session. The subject could thus check up which switch stood for what, by glancing at the papers. After the first session, the subjects could remember quite easily which switch they wanted to use.

These comprehensive instructions were only given at the beginning of the first session. For the other sessions, shorter instructions were given.

The signalling procedure had two functions in the experiment :

1. It had been noted in a pilot study that subjects tended to fall asleep during the session. The cutting up of each session into three, with the signalling system occuring twice during the session relieved the monotony and helped to prevent the subject from sleeping.

2. The drowsy-alert signal could be recorded so as to compare subjective reports to physiological measurements. The subject frequently reported having pressed the green light, not because he had been drowsy, but because he had felt very relaxed. The experimenter explained that the green light could be used whenever the subject felt in a low state of alertness, the nature of such a state was said to be unimportant. However, the subject

was/...

was always asked whether he had fallen asleep, so as to ensure whether the green light had stood for drowsiness or relaxation.

4.3.4. Baseline Procedure

(Session 2, 3 and 8).

1. Today I'm just wanting to see what your physiological measures would be like when you are just sitting still, but do not fall asleep.
2. Remember that the session is split into three parts, and I shall signal you with the buzzer after the first and second phases are over - do not forget to signal me back with one of the switches.
3. Keep your eyes closed throughout the session, except when you are busy signalling me after the buzzer.
4. Try not to get restless and make yourself comfortable, especially in the middle portion of the experiment which is a little longer than the others.

4.3.5. Treatment Procedure

(Sessions 5, 6 and 7)

1. Today I want you to practice the breathing/muscular control exercises I showed you during the training, but only during the middle part of the experiment.
2. During the first part of the session, just sit still like you have done in the first sessions. Only start the breathing/muscular control exercises after I have signalled that the first phase of the session is over.
3. Do the breathing/muscular control exercises during the

whole/...

whole of the second part of the session, right up until you hear me buzzing you for the second time. Then, stop the exercises immediately, open your eyes and simply press any of the switches - this time do not bother to select one over the other, as I'm only interested in the physiological effects of the exercises. (This served to detract the subject's attention from suggestions of relaxation, which were made to seem irrelevant to the exercises).

4. The last part of the session is the same as the first part. you just sit still with eyes closed until I walk in.

Frequently subjects asked what effect the exercises were supposed to have on them. The experimenter would then say that she had not the slightest idea herself, that they were exercises practiced by some eastern people, and that this study was actually the first one to investigate them at all, and that therefore she could not, as yet, foresee the results of the experiment. The subjects were told to 'just do the exercises without thinking of anything else about them'.

The subjects were asked after the session very casually 'whether they had been uncomfortable in doing the exercises'. Subjective reports were noted on the Beckman paper recording once the subject had left. The experimenter was most careful never to show signs of increased interest according to the quality of the reports. All reports were responded to in the same casual manner and leading questions were never asked.

4.3.6. Deep breathing training procedure

When the subject arrived at the laboratory for the deep breathing training, he was immediately told that there would be no wiring up

involved/...

involved in this session, but that he would be required to do certain exercises in breathing.

The subject was told that it was essential that he sit very upright during the practice of the breathing exercises, as otherwise part of the lung would be blocked. He was made to sit on the floor with his back and shoulders touching the wall. He was told that he could leave his legs straight or if it were more comfortable, he could sit with his knees crossed. Most subjects preferred the latter position. The experimenter then checked that the subject was sitting upright, and corrected his posture if necessary.

The experimenter sat opposite the subject on the floor, and proceeded to explain the nature of the exercises to be practiced, and at the same time did them herself, so as to demonstrate them to the subject.

The following instructions were not read out, so that a few words may have varied from subject to subject. However, the content and order of presentation was kept constant :

Exercise 1 :

This exercise had the purpose of making you use your whole lung at each breath. Remember it is a training exercise, you will not actually be doing this during the session itself. Take a deep breath then expire slowly, emptying your lung from the top to the bottom; when you finish expiring normally, do a forced expiration until you can no longer push out any air: then hold this position for as long as you can, and notice where all your respiratory muscles are, from the bottom to the top.

Then, when you can no longer hold your breath, simply let go your stomach muscles, and air will automatically be drawn in from

the/...

the bottom of your lung, and then carry on the breath until you feel your whole lung is full. Then repeat the exercise three times. (The experimenter then did the whole exercise in front of the subject, and then told the subject to do it, with eyes closed, so as to concentrate on which muscles are being exercised, and on filling the lung from bottom to top, and emptying it from top to bottom).

Exercise 2 :

Now you have learnt to fill up your lung in the abdominal area. The next exercise is one where you must be able to follow your breathing and stop it at the other levels of the lung. First at the mid-chest. Fill your lung from the bottom area and proceed upwards: stop when you feel that you have reached the level of the mid-chest. Then empty the lung from the top to the bottom. (Experimenter demonstrated the exercises and urged the subject to do the same three times with eyes closed).

Exercise 3 :

Now repeat what you have just done, only this time do not stop at the mid-chest level: instead, once you feel that you have reached that level, carry on and fill up the upper lung. When you reach that part of the lung, you will feel your shoulders moving slightly backwards: exaggerate this movement slightly while practicing this exercise. (The experimenter demonstrated the exercise, and asked the subject to repeat it three times with eyes closed).

Exercise 4

Now I want you to take a full breath, doing all the exercises

at once/...

at once. Start off by filling your lung fron the abdominal area (the experimenter pointed to the lower lung) to the mid-chest and without stopping right up to the upper chest area until a slight backward movement of the shoulders is felt. Maintain this position a little while, then start expiring, emptying first the upper chest area with a slight drop of the shoulders, then the mid-chest, right down to the abdominal area. Do a forced expiration, hold your breath for as long as you can, then let go all the muscles and let the air flow in, first in the abdominal area, then right up to the upper chest in a smooth motion, without stopping. (The experimenter demonstrated the exercise and asked the subject to do the same three times with eyes closed; the subject was told to keep his mind on his breathing and make sure he was following the instructions of inspiring from abdominal to upper chest area, and expiring in the opposite direction at each breath).

Exercise 5

Now you have learnt how to take a complete breath; the exercises exaggerated each movement slightly for you to get a clear idea of how to take the full breath. However, if you were to take many breaths like that consecutively, you would feel dizzy, as you would be overbreathing. Now, take the breath, following the same principles, but without forcing the lung in any way: inspire from bottom to top, but gently and smoothly as is comfortable for you. No movement should actually be observed from outside. It is the quality of the deep breathing that is important, the quantity will depend on how much air you are needing. If you are taking in too much air, you are overbreathing; you will feel

dizzy and automatically you will readjust your breathing so as to take in less air, or take it in more slowly. Because you are breathing so deeply, you will find that you have to breathe more slowly to keep the amount of air constant. If you are underbreathing, you will feel that you are short of breath, choking or gasping for air. That means that you are breathing too slowly or that you are pausing for too long between breaths. Remember it is not necessary to pause at all, in fact, try to keep the breathing as smooth as possible. You will find that after a few breaths you are readjusting your breathing automatically so that it is comfortable. Just keep your mind on your breath, and follow it through all its motions as if you were watching something passively.

Exercise 6 :

Now I leave you for ten minutes; just sit upright on the bean bag as you will be doing during the next session, then breathe as I have just shown you. Remember the important points: inhale from the bottom to the top of the lung, exhale from the top to the bottom, keep your mind on your breath so that you can monitor it. If you feel dizzy, you are overbreathing, make the necessary adjustments. If you feel short of breath, you are underbreathing, try to analyse why this is so and adjust your breathing accordingly, keep your eyes closed.

The subject was left in the room for ten minutes with only the reading light on. The experimenter then walked in and asked the subject whether he had felt discomfort. If the subject reported any, the experimenter explained to him again what was probably wrong (over or under breathing most frequently). Sometimes the subject

reported discomfort in maintaining the upright position. The experimenter would then seek to seat him upright so that he would be supported by the bean bag right up to the shoulders - the bean bag was chosen because of its being easily moulded so as to fit a subject's posture.

4.3.7. Muscular Relaxation Procedure

The subjects were informed upon arriving at the laboratory that there would be no wiring up involved in this session as they were to be trained at certain muscular exercises.

The subject was told to seat himself as upright as possible in such a way as to be well supported by the bean bag and comfortable. Some time was allowed for the subject to achieve the required position.

He was then given the following instructions, which were not read out so that a few words may have varied from subject to subject. However, the content and order of presentation were the same for all subjects.

1. I am going to be showing you certain exercises which have the purpose of showing you how to keep a muscle contracted, and more important, how this can show you how to keep the muscle loose (the word relaxed was not used so as not to suggest the state of relaxation to the subject). We are now going to contract then loosen each muscle in the body in turn. You must keep your eyes closed and every time you work on a muscle, keep your mind on it as it contracts then gets loosened.

2. The experimenter first started with the subject's left

foot/...

foot, until he felt tired and could no longer keep it up; then he was to loosen it, let the foot fall in whichever position it chose and keep their minds on the foot, so that they could feel the difference between a contracted and a loose muscle. The experimenter demonstrated to the subject how to do the muscular contraction and relaxation, and then asked the subject to do the same.

3. After the left foot had been done, the experimenter went over the following muscles, in that order, repeating the instructions to contract the muscles until the subject felt tired, and then to loosen up the muscle and let it become limp so that it could get into any position it found comfortable.

1. The left foot
2. The left leg up to the knee
3. The left thigh
4. The right foot
5. The right leg up to the knee
6. The right thigh
7. The hips
8. The stomach
9. The left hand (in a fist)
10. The left arm below the elbow
11. THe left shoulder up to the elbow.
12. The right hand (in a fist)
13. The right arm below the elbow.
14. The right shoulder up to the elbow.
15. The neck (twisting the head from side to side with neck muscles tensed up).
16. The face (screw up the face, with lips tight and eyes

closed tightly).

4. After all the exercises had been completed, the subject was told : Now I am going to call out all the different places where the muscles we have just worked on are situated. Keep your eyes closed. When I call out a certain location like right foot, or stomach, do not contract the muscle like you did last time, instead think of the muscle and try to see to what extent it is contracted. If it is so, loosen it as much as you can. If you find this difficult then contract the muscle again then loosen it. If it is already limp, then do nothing, just be sure it is as limp as it can be. I will follow the same order as during the previous exercises, from the feet to the head, the left limb always first.

(The experimenter proceeded to call out the 16 different locations and if any tension was noticeable in a muscle and the subject did not relax it when it was called out, the experimenter drew the subject's attention to the muscle and asked him to contract it then let it limp.

5. Now I am going to leave you alone, for you to go through the muscles again on your own. Remember to start with the left foot and proceed upwards to the head. Try and tell me quickly the parts of the body you will be trying to keep limp during these exercises.

(The subject was then required to repeat or point to the 16 parts of the body he was to concentrate on - if the subject persistently picked the right limb first, he was not corrected as this was thought trivial to the practice of relaxation).

Remember it is not necessary to contract the muscles at all, as by now you will be able to judge whether a muscle is contracted

or not/...

or not, from having done the exercises. Only contract a muscle if you cannot get it uncontracted any other way. Then, remember which muscle it was, as you will probably find that it will often be contracted; next time spend some time on the muscles you found most often contracted in today's session, and be sure you loosen them up.

(The subject was left alone for ten minutes to practice the exercises with only the reading light on).

6. After 10 minutes the experimenter walked in and asked the subject whether any muscles had been particularly contracted. The subject was told to remember this/these muscles during the next session where he would be going through each muscle as he had been doing in the last 10 minutes, without contracting any of them. He was reminded that it was important to keep the eyes closed and to passively observe each muscle, judge its degree of contraction, then loosen it completely. After he had gone over all the muscles in the actual sessions, he would then be required to remain passively aware of his body so that he would be able to detect any muscular contraction without actually concentrating too hard on the task.

4.4. Scoring Procedure

4.4.1. Skin Resistance Scoring Procedure

Using the time mark system, the number of marker pips (or intervals between marker pips) per second could be converted into kohms resistance values using a linear equation $y = bx + 3$ (see Section 4.3.1. a). The skin resistance raw data was scored manually, by

measuring/...

measuring the number of centimeters between the first 13 marker pips (i.e. 12 intervals) of each minute of the session which was analysed; this data was then transformed into number of intervals between marker pips per second (this procedure was adopted so as to get a representative reading of the number of intervals per second for each minute, even when resistance values were changing at a fast rate).

The number of intervals per second obtained for each minute was then converted into resistance values (kohms) using the equation

$$y = 960x$$

which was derived in the following manner.

As the number of marker pips per second was directly proportional to the value of basal resistance, the linear equation $y = bx + c$ could be used to derive resistance value for any given number of intervals per second. In the equation $y = bx + c$

- y = resistance value to be derived.
- b = slope of the line (i.e. the rate at which the time marks per second changed with changes in resistance).
- x = number of intervals per second (or number of time mark per second).
- c = the point where the graph intercepts the y-axis (in this instance, $c=0$: see Figure 3 on page 61).

The calibration system built into the coupler allowed for the value of b to be derived (i.e. the slope of line). Appropriate x and y values necessary for the calculation of b could be obtained for the calibration setting used in the recording sessions.

For calibration, the coupler settings were kept identical to those used during the recording sessions i.e. a 10 ua constant current

was used/...

was used with a 1 second time constant.

In the formula

$$y = bx$$

(where b is a constant), b may be calculated by the following equation :

$$y = \frac{y_2 - y_1}{x_2 - x_1}$$

where y_2 and y_1 are any two known resistance values, and x_2 and x_1 their corresponding number of time marks per second. The values obtained from the calibration settings used in the present experiment, were as follows :

20k : 0.13 intervals per second.

100k : 0.21 intervals per second.

500k : 0.63 intervals per second.

The value of b was derived from each of the three x and y values.

1. When y_1 = 500k and y_2 = 100k

   then x_1 = 0.63 and x_2 = 0.21

   $$b = \frac{500 - 100}{0.63 - 0.21} = 952.38 \qquad \text{(i)}$$

2. When y_1 = 500k and y_2 = 20k

   then x_1 = 0.63 and x_2 = 0.13

   $$b = \frac{500 - 20}{0.63 - 0.13} = 960 \qquad \text{(ii)}$$

3. When y_1 = 100k and y_2 = 20k

   then x_1 = 0.21 and x_2 = 0.13

   $$b = \frac{100 - 20}{0.21 - 0.13} = 1\ 000 \qquad \text{(iii)}$$

Thus, three b values were obtained, for 20k, 100k and 500k and these b values ranged between 952.38 and 1 000.

This/...

This error was checked so as to ensure that it fell within the 5% error margin specified in the Beckman chart, i.e.

If error (magnitude or discrepancy between the 3 b value) = e and full range values from which b was derived = y (resistance values)

$$e = 1\ 000 - 952.38 = 47.62$$

$$y = 1\ 000k - 20k = 980k$$

$$\text{Percentage error} = \frac{e}{y} \times 100$$

$$\frac{47.62}{980} \times 100 = 4.86\%$$

This was within the 5% error margin.

Obviously, one b value only had to be used in the score transformation. The b value obtained from

$$b = \frac{500k - 20k}{0.63 - 0.13}$$

i.e. 960, was chosen, as this was the score obtained from the whole calibration range (20k to 500k)

This yielded the equation

$$y = 960x$$

i.e. Resistance value to be derived = 960x where x is number of intervals per second.

The following calculations show that y = 960x result in score transformations in which the number of time marks per second was directly proportional to resistance changes. i.e.

(i) $100k = 960x$

$$x = \frac{100}{960} = 0.1 \text{ int. per sec.} \qquad \text{(i)}$$

(ii)/...

CALIBRATION OF GSR COUPLER.

Resistance value = bx + c

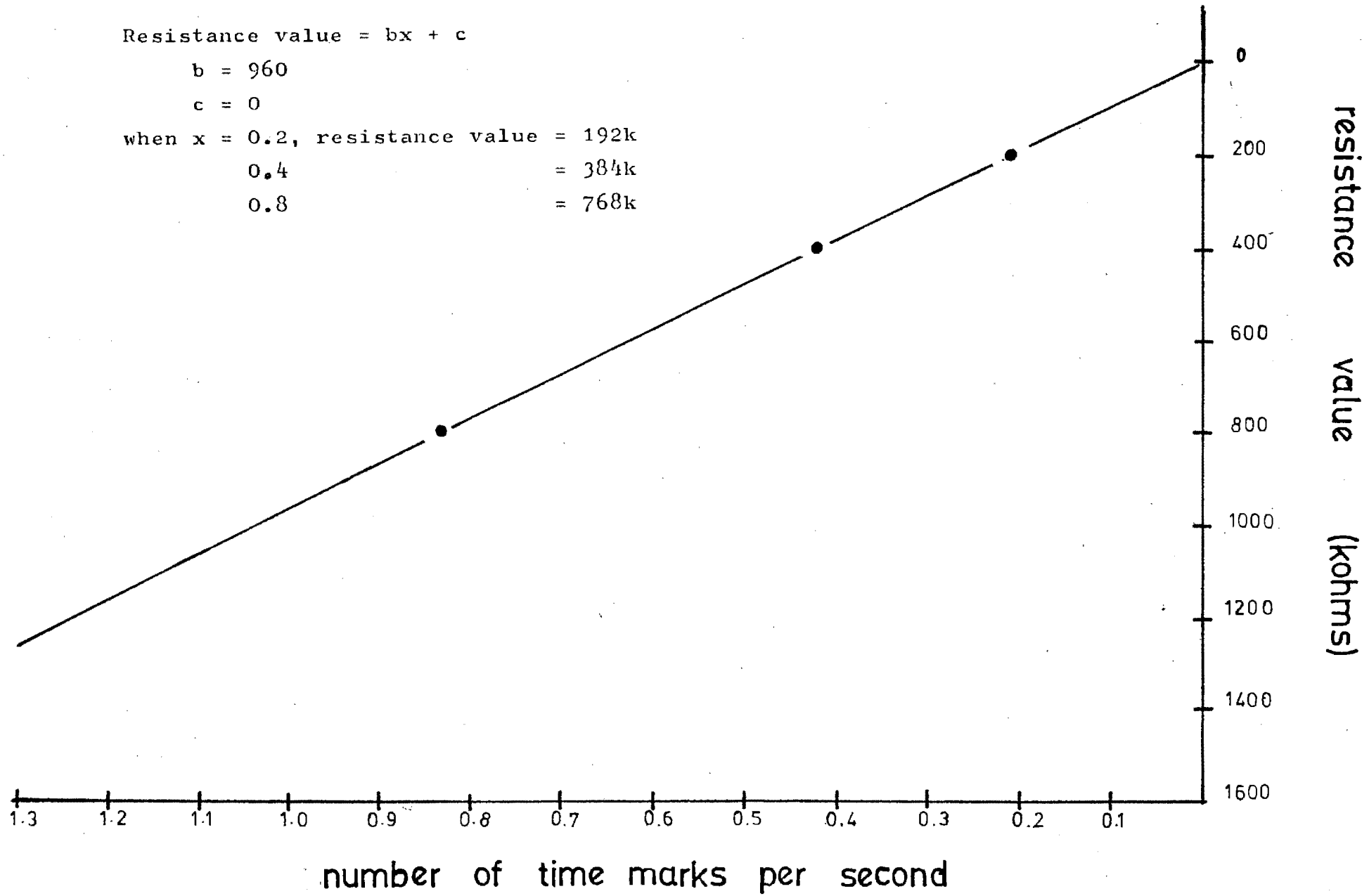
b = 960

c = 0

when x = 0.2, resistance value = 192k

0.4 = 384k

0.8 = 768k



(ii) $20k = 960x$

$$x = \frac{20}{960} = 0.02 \text{ int. per sec.} \qquad \text{(ii)}$$

(iii) $500k = 960x$

$$x = \frac{500}{960} = 0.52 \text{ int. per sec.} \qquad \text{(iii)}$$

Now,

$$\frac{100k}{20k} : \frac{500k}{100k}$$

$$5 : 5$$

Substituting

0.1 for 100k (i)

0.02 for 20k (ii)

0.52 for 500k (iii)

$$\frac{0.1}{0.02} : \frac{0.52}{0.1}$$

$5 : 5.2$ (taking the 5% error into account.

4.4.2. Heart Rate Scoring Procedure.

For Heart Rate the number of beats for each minute was counted, and the mean was worked out from the readings obtained for each phase of the session. A heart beat was not always present at the very beginning of the first, nor at the end of the fifth minute. In such a case, the missing beat would be counted as either thirds or fourths of a beat, as was appropriate.

4.4.3. EMG (electromyography) Scoring Procedure

The/...

4.4.3. EMG (electromyography) Scoring Procedure

The analysis of the EMG dats had to be done by visual inspection using a rank order system. This was necessitated by shortage of appropriate equipment while recording : no EMG coupler was available and an A-C coupler had to be used with its inadequate frequency range for EMG recording. The signal could not be amplified to the maximum capacity (of the dynograph), due to the high signal-to-noise ratios involved in recording surface EMG. The low amplitude of most EMG signals recorded would have made any scale of values, such as millimeters so small that comparisons would be unreliable.

Therefore, a sample of EMG data from both muscle groups (masseter and corrugator) were cut out from the end of each phase (phase 1, 2(i), 2(ii) and 3) and put alongside each other. The subject's samples for each session were included in the Appendix. The data for each session was ranked from most tense (which was given the rank 1) to least tense (which was given the rank 4). When two or more phases had an equal amount of tension, they were awarded the same rank. In cases in which tie scores were found, it was considered appropriate to give the next phase the rank immediately following the last one e.g. if phase 1 were given the rank 1, and phase 2(i) and 2(ii) both ranked second, the third phase would be ranked 3 and not 4. This procedure was contrary to that habitually used when tie scores are encountered, but was adopted in consideration of the nature of the data and the information which was required from it, i.e. tie scores indicated no change in muscular tension; thus, it would be inappropriate to award a score of 4 to the third phase, for example, in a case where no muscular tension was noticeable in the first two phases, while the same score would be obtained in a case where muscular

tension/...

tension decreased throughout the session. The use of continuous ranks regardless of tie scores thus gave some indication of the degree of muscular tension decrease as well as of the presence of such drops in muscle tension. When sessions were compared, it was thought appropriate that those sessions in which 4 noticeable decreases in EMG amplitude should be distinguished from those in which 1 or 2 such changes were found. It should be noted that before adopting this procedure, consideration was given to the possibility of tie scores being consistently followed by larger EMG amplitude decreases than were the progressively decreasing EMGs. This was not found to be the case however, as inspection of the EMG samples included in the Appendix will indicate.

4.5. Statistical Procedure

A different statistical procedure was used for skin resistance and heart rate on the one hand, and the electromyogram on the other hand. These are reported separately in Sections 4.5.2. to 4.5.4. However, the procedure used in data collection for statistical analysis is first outlined below.

4.5.1. The division of the experimental session into four phases in the statistical analysis

It was stated in Section 4.3.2. that each experimental session was divided into 3 phases (see Section 4.3.2.) The following table (Figure 4) illustrates those parts of the three phases which were used in data analysis for the different physiological parameters. The rationale for this choice of data is subsequently presented.

Figure/...

FIGURE 4 : Division of the Experimental Sessions into phases for statistical analysis

	First Phase	Second Phase		Third Phase
Total	5 min	$12\frac{1}{2}$ minutes		$7\frac{1}{2}$ min
Analysed +	5 min	Last 10 min.		Last 5 min.
	Phase 1	5 mins Phase 2 (i)	5 mins Phase 2 (ii)	Phase 3

+ It should be noted that Skin Resistance data was derived using the last minute of each of the four 5 minute periods labelled C1, C2, C3 and C4.

The EMG samples presented in the Appendix were also taken from the last minute of each phase.

The first $2\frac{1}{2}$ minutes of the second and third phases were not analysed as the subject was frequently disturbed by the buzzer. Further, he was required to select one of three switches indicating his level of arousal in the preceeding phase. These $2\frac{1}{2}$ minutes allowed for the subject to settle down to his previous physiological functioning.

The SR data was collected in a slightly different way to HR. This method was chosen because it was found that SR was extremely sensitive to environmental conditions and changed rapidly (rose or

went/...

went down), at a relatively fast rate but was slow to recover. Thus, the last minute of each four 5 minute period was considered most representative of the level of SR attained by the subject in each of these four periods. This decision was taken only after having analysed the whole 20 minute period for each subject. The buzzers for example frequently caused a sharp drop in SR which would slowly regain its previous level. The effect of the buzzer was often still noticeable in certain subjects after 2½ minutes. The subsequent rises in SR were thus masked by the effect of the buzzer.

The last 10 minutes of the second phase were split up in the analysis into two 5 minute periods, so as to compare the various treatments over time. e.g. When the relaxation and deep breathing techniques were compared, it was found that in the first five minutes the relaxation group frequently attained a higher level of SR than the deep breathing group. However, this effect was not maintained over the second five minute period by the relaxation group. On the other hand, the deep breathing group continued to show a rise in SR and at the end of the second phase they had attained a higher level of SR than the relaxation group. This pointed to qualitative differences between the two techniques: the relaxation group achieved physiological relaxation more rapidly than the deep breathing group, but could not maintain this state over the entire time. The experimental group, however, in the last two treatment sessions, showed a continuous rise in SR during the practice of deep breathing.

4.5.2. Statistical Procedure used for Skin Resistance and Heart Rate Data

This experimental design indicated the use of the 3-way Analysis of Variance (ANOVA) with repeated measures on factor B (sessions) and

C/...

C (experimental phases). Where appropriate, the ANOVA was followed by a Simple and Main Effects test, and Multiple and Pairwise comparisons for the 3-way ANOVA with repeated measures on Factor B and C.

4.5.3. Definition of the levels of the factors in the 3-way ANOVA

The 3-way ANOVA with repeated measures on factors B and C was used. Factor A consisted of the 2 groups of 6 subjects (experimental and control). Factor B represented the experimental sessions. Factor C was the phases within each experiment. So as to facilitate the interpretation of the ANOVA summary tables, Figure 5 provides a list of the factors and each level of the factors is defined.

FIGURE 5 : Definition of the 3 factors in the ANOVA

Factor A	:	A1	:	Experimental group
		A2	:	Control group (relaxation)
Factor B	:	B1	:	Mean of pre-treatment baselines 1 and 2
		B2	:	First treatment sessions.
		B3	:	Second treatment session.
		B4	:	Third treatment session.
		B5	:	Post-treatment baseline.
Factor C		C1	:	First Phase.
		(C2	:	Phase 2 (i) (5 mins. treatment)
	+	(		
		(C3	:	Phase 2 (ii) (5 mins. treatment)
		C4	:	Third Phase

Factor B had 5 levels (see Figure 5). The experiment consisted of two pre-treatment baseline sessions, followed by three treatment

sessions/...

sessions and finally one post-treatment baseline session (6 sessions).

This yielded an unbalanced experimental design. However, it was considered most desirable to obtain two pre-treatment baseline recordings, so as to enhance the likelihood that a representative measurement of normal physiological function would be obtained. These two pre-treatment baselines were meaned, and treated as a single session in the statistical analysis. This gave a balanced experimental design, and is illustrated in Figure 6.

Thus, there were two levels of factor A (experimental and control group); 5 levels of factor B (pre-treatment baseline, 3 treatment sessions, post treatment baseline); and 4 levels of factor C (phase 1, phase 2(i), phase 2(ii) and phase 3).

4.5.4. Statistical Procedure used for EMG Data

The EMG data could not be subjected to non-parametric techniques usually used with ranked data (e.g. Mann-Whitney test) as only four ranks were used and quite a number of tie scores were encountered. It was not possible to pool the different subject's EMG samples and then rank them, due to differences in initial EMG tension levels; these initial value differences were of no relevance to the present study which concerned itself with changes in EMG tension. Thus, for the six subjects in each group, each phase was awarded ranks 1 to 4; each rank could be expected to occur six times (once for each subject); if tie scores occurred, some of the ranks would be encountered more than once per subject. It was the purpose of the experimenter to find a means of obtaining meaningful data analysis from the ranked EMG samples, which would allow comparisons to be made between the various sessions. For this purpose the

following/...

FIGURE 6 : SUMMARY OF THE EXPERIMENTAL DESIGN

		FACTOR B : EXPERIMENTAL SESSIONS					FACTOR C PHASES WITHIN SESSIONS		
FACTOR A	A1	B1	B2	B3	B4	B5	C1	C2	C3
	Exp. Group	Mean Baselines 1 + 2	Deep Breathing	Deep Breathing	Deep Breathing	Post-treatment baseline	Baseline eyes closed	Deep Breath treat-ment or eyes closed	Baseline eyes closed
	A2 Control Group	"	Relaxa-tion	Relaxa-tion	Relaxa-tion	"	"	treat-ment (relax) or eyes closed	"

following procedure was adopted: for every session, the ranks obtained by each subject as per phase were summed to get each phase's total rank; e.g. if all six subjects obtained a rank of 1 in the first phase, the group's total rank for that phase would be 6. Where there were scores missing for a session, the group's mean rank was used in its place so as to keep the number of scores constant from session to session for purposes of comparison. When only part of a subject's EMG samples were available, the whole session for that subject was excluded. Corrugator and messeter muscles were analysed separately. The total ranks obtained for each group were represented in tabular form in the Appendix. The lower the score representing total rank, the greater the EMG tension for that phase; and the higher the score representing total rank, the lower the EMG tension could be expected to be. From these total ranks, change scores were computed for phase 2(i), 2(ii) and 3 by subtracting total rank in the first phase from each of the following phases. This did away with the problem of differing initial values and permitted direct inter-session and inter-group comparisons on the basis of change scores; e.g. if the first phase of session A and B both had a total rank of 9, and phase 2(i) of session A had a total rank of 12 while the same phase of Session B had a total rank of 15, session A's change score would be 3 and session B's, 6. It could then be concluded that there had been greater EMG tension decrease in phase 2(i) of session B than in the corresponding phase of session A : i.e. the greater the difference between a given phase's total rank and that of the first phase, the larger was the decrease in muscular tension represented.

Taking an extreme case, if phase one was always given the rank 1 and phase four the rank of 4, then their respective total ranks would be 6 and 24, and the change score between phase 1 and 4 would

be/...

between change scores, which here is 36. Thus, if two sets of change scores differed by a score of 12, it would probably be more meaningful to the reader to know that the difference between the sessions involved is of $33^{1}/_{3}\%$ ($\frac{12}{36}\%$) of the maximum difference obtainable.

5. Results

5.1. Skin Resistance Results

A three-way ANOVA (Analysis of Variance) with repeated measures on factors B and C was used to analyse the skin resistance (hereforth referred to as SR) data. These ANOVA results are presented in table 1. Significant F-ratios were found for factor C at the .05 level of significance (F 2.92, df = 3, 30). Significant interaction effects between factors ABC were also present, at the .05 level of significance (F 1.83, df = 12, 120).

The significant F-ratio obtained for factor C may not be interpreted in view of the significant interaction effects found for factors ABC.

The significant ABC interaction found in the ANOVA was further investigated using an Analysis of Simple Interaction Effects (hereforth referred to as ASIE). These results are presented in table 2.

None of the AB interactions were found to be significant. These results indicate that the experimental and control groups displayed similar SR trends during the various sessions.

The ASIE revealed one significant AC interaction at the 0.25 level. (AC at B5, F 3.34, df = 3, 75). More detailed information concerning the significant AC interaction at B5 was obtained by an analysis of Simple Simple Main Effects (hereforth referred to as SSME). These SSME results are presented in table 3. The control group's cell means profiles were represented graphically in Figure 8. A significant SSME F-ratio was obtained for factor C at A2B5 at the .05 level (F 2.74, df = 3,75). Referring to Figure 8, both the upward SR trend at level C3, and the sharp drop in SR at C4 appears

to be/...

to be atypical of the SR trends observed for factors C3 and C4 at the other levels of B. The cell means of the post-treatment baseline (factor B5) were compared for the control group using Tukey's HSD test (Pairwise comparisons). These results are presented in table 4, and indicated significantly higher SR at level C3 (phase 2(ii)) compared to C1 (phase 1) at the .05 level (t' 3.68, df = 4,75).

In the SSME results in table 3, it should be noted that the F-ratio for C at A1B5 fell 0.15 short of the .05 level of significance (F 2.72, df = 3,75); furthermore, a SSME test had to be performed for C at A1B5 as a follow-up of an independently significant SIE (BC at A1). With due caution for the consideration that C at A1B5 did fall just short of significance, it was considered possible that genuine differences in SR were present for the experimental group in the post-treatment baseline's various phases. Referring to figure 7, it may be noted that the experimental group's SR dropped sharply during phase 2(ii) (factor C3); this trend continued to the end of the session in phase 3 (factor C4). In view of the non-significant F-ratio obtained for C at A1B5 in the SSME, it was expected that cell means comparisons using Tukey HSD test would yield no significant results, the latter being a conservative statistical test; however, the cell means of the post-treatment baseline for the experimental group were compared, using Tukey's HSD test, so as to assess whether any pair of means differed to a near-significant level: these results are set out in table 4. Only one pair of means approached a near significant difference, i.e. SR was lower in phase 3 (factor C4) than in phase 2(i) (factor C2), but fell short of significance by 0.44 (t' 3.73, df = 4,75).

Analysis of SIE of factors BC revealed one significant F-ratio

(BC at A1, at the .05 level; F 1.85, df = 12,120). This indicated significant differences in SR trends for the experimental group, as a function of sessions and phases. The SSME results indicated that the experimental group's SR trends in the various phases differed significantly during the second treatment session (C at A1B3, at .05 level; F 2.72, df = 3,75) and during the third treatment session (C at A1B4 at .025 level; F 3.34, df = 3,75). As was pointed out in discussing the SSME results for significant AC interaction, the Fr ratio obtained for C at A1B5 was considered worthy of further investigation. The latter Tukey HSD ratios were reported previously in this section. In figure 7, the SR cell means profiles for the experimental group (A1) are graphically represented. These graphs make it difficult to gain a visual understanding of the SR trends for factor B3, B4 and B5 at the various levels of C, due to the differences in initial SR level. The SR trends at levels of C for factors B3 and B4 tend to give the impression that similar trends were present, for example for factor B1. These differences in initial SR value are of no relevance to the present experiment (a glance at table 5 will indicate that SR changes were unrelated in magnitude and direction to initial value in phase 1). Referring back to figure 7, it is apparent that were all SR values at C1 (initial SR level) to be identical, the upward SR trend observed at levels C2 and C3 of factor B3 and B4 would in no way be parallel to the SR trends observed for C2 and C3 at the other levels of B. On the other hand, the apparently great interaction effect observed for factor B5 at levels of C would only consist of a sharp SR drop at level C3, were differences in initial SR level to be eliminated. In figure 7, the SR trends for factors B2, B3 and B4 appear to differ significantly from those observed

at/...

at B5, as the latter's line cuts across all three of these sessions (B2, B3 and B4). This effect, however, is due to differences in initial SR value, and is therefore an artificial observation. It is thus not altogether surprising that no significant SSME ratios were obtained for C at A1B5, while those for C at A1B3 and C at A1B4 were found to be significant. It is undeniable, however, that the sharp SR drop for factor B5 at level C3 may represent a genuine difference in SR trend. This will be elaborated upon in the discussion of the SR results.

The cell means of the second treatment session (factor B3) and those of the third treatment session (factor B4) were compared, for the experimental group, using Tukey's HSD test. These results are set out in table 4. The HSD test indicated significantly higher SR at level C3 (phase 2(ii)) as compared to level C1 (phase 1), for both factors B3 and B4 (the second and third treatment sessions) at the .05 level (t' 3.68, df = 4,75).

No Simple Main Effects (hereforth referred to as SME) test was run on the non-significant two-factor interactions, as significant interactions were found in the ASIE (see table 2).

TABLE 1/...

TABLE 1 : Skin Resistance ANOVA Summary.

SOURCE	SS	DF	MS	F RATIO	SIG. LEVEL
Between Subj.					
A	333558	1	333558	.289062	
SUBJ.W.G.	1.15393E+07	10	1.15393E+06		
Within Subj.					
B	810048	4	202512	1.15139	
AB	224886	4	56221.5	0.319649	
BxSWG	7.03540E+06	40	175885		
C	67326	3	22442	3.05994	.05
AC	3240	3	1080	0.147257	
CxSWG	220024	30	7334.13		
BC	90426	12	7535.5	1.52984	
ABC	112890	12	9407.5	1.90989	.05
BCxSWG	591080	120	4925.67		

Check on homogeneity of error terms

F.Max. (Subj.W.G.) = 1.42353 DF = 2,5

F.Max. (B x SWG) = 2.2155 DF = 2,20

F.Max. (C x SWG) = 3.26312 DF = 2,15

F.Max. (BC x SWG) = 1.04022 DF = 2,60

TABLE 2 : SKIN RESISTANCE RESULTS

Analysis of Simple Interaction Effects (ASIE)

SOURCE	DF	F-RATIO	SIGNIFICANCE LEVEL
AB at C1	4	0.82721	
AB at C2	4	0.33337	
AB at C3	4	0.421751	
AB at C4	4	0.189359	
ERROR	42		
AC at B1	3	0.216557	
AC at B2	3	0.668163	
AC at B3	3	2.55892	
AC at B4	3	0.288773	.025
AC at B5	3	3.42625	
ERROR	75		
BC at A1	12	1.99282	.05
BC at A2	12	1.44742	
ERROR	120		

TABLE 3 : SKIN RESISTANCE RESULTS

Analysis of Simple Simple Main Effects (SSME) (for significant ASIEs AC at B5 and BC at A1

SOURCE	DF	F-RATIO	
A at B5C1	1	0.821658	
A at B5C2	1	0.221698	
A at B5C3	1	1.27609E-02	
A at B5C4	1	6.35802E-02	
ERROR	13		
C at A1B5	3	2.66391	
C at A2B5	3	3.29491	.05
ERROR	75		
B at A1C1	4	0.789203	
B at A1C2	4	1.10786	
B at A1C3	4	1.12162	
B at A1C4	4	1.0918	
ERROR	42		
C at A1B1	3	0.410482	
C at A1B2	3	0.297465	
C at A1B3	3	2.72758	.05
C at A1B4	3	3.40563	.025
C at A1B5	3	2.66391	
ERROR	75		

TABLE 4 : SKIN RESISTANCE RESULTS

Comparisons of Cell Means by Tukey's HSD Test

C AT A1B3				
	C1	C2	C3	C4
C1	-	2.69263	3.71414 +	3.18117
C2	-	-	1.0215	0.53297
C3	-	-	-	0.488534

C AT A1B4				
	C1	C2	C3	C4
C1	-	3.24222	4.25264 +	3.1756
C2	-	-	1.01041	6.66213E-02
C3	-	-	-	1.07703

C AT A1B5				
	C1	C2	C3	C4
C1	-	0.516315	2.276215	2.770345
C2	-	-	2.79253	3.28666
C3	-	-	-	0.49413

C AT A2B5				
	C1	C2	C3	C4
C1	-	3.19229	3.98618 +	1.24915
C2	-	-	0.793893	1.94314
C3	-	-	-	2.73703

SIGNIFICANT RATIOS :

C AT A1B3 : C1 C3 at the .05 level
(t' 3.68, df = 4,75)

C AT A1B4 : C1 C3 at the .05 level.
(t' 3.68, df = 4,75)

C AT A2B5 : C1 C3 at the .05 level
(t' 3.68, df = 4,75)

TABLE 5 : SKIN RESISTANCE MEANS AND STANDARD DEVIATION

Mean Phase One : Phase 1

Mean First Half Second Phase : Phase 2(i)

Mean Second Half Second Phase : Phase 2(ii)

Mean Third Phase : Phase 3

	Pre-Treatment Baseline	First Treatment	Second Treatment	Third Treatment	Post-Treatment Baseline
EXPERIMENTAL GROUP					
Phase 1	333.833	497.5	469.333	492.167	543
SD	193.975	350.555	384.008	328.495	404.079
Phase 2 (i)	352.833	495.167	550.167	589.5	558.5
SD	234.344	306.538	427.355	384.941	442.049
Phase 2 (ii)	380.667	485.5	580.833	619.833	474.667
SD	287.405	290.987	449.587	356.931	329.515
Phase 3	356.	461.667	564.833	587.5	459.833
SD	234.572	274.916	434.257	376.641	313.59
CONTROL GROUP					
Phase 1	362.167	332.	534.167	341.667	375.833
SD	169.304	332.829	348.072	177.411	159.2
Phase 2 (i)	374.	341.5	509.833	447.	471.667
SD	174.583	340.455	340.886	332.732	223.459
Phase 2 (ii)	365.167	359.167	493.833	438.333	495.5
SD	161.621	333.049	327.679	304.499	245.374
Phase 3	376.667	374.333	495.833	460.167	413.333
SD	143.062	322.941	284.879	265.143	209.579

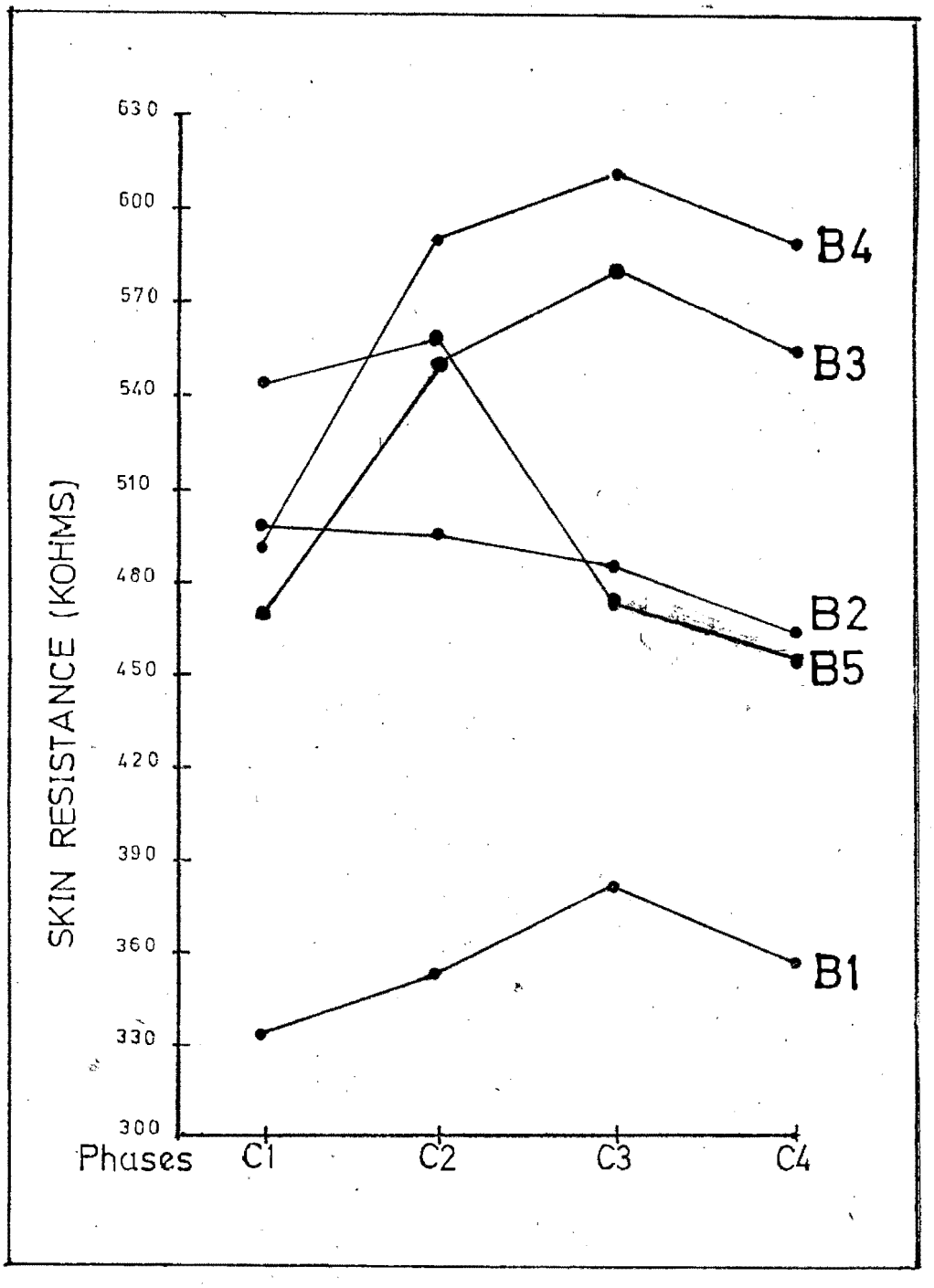


Figure 7 : Skin Resistance (kohm) cell mean profiles : Experimental Group (Factor A1)

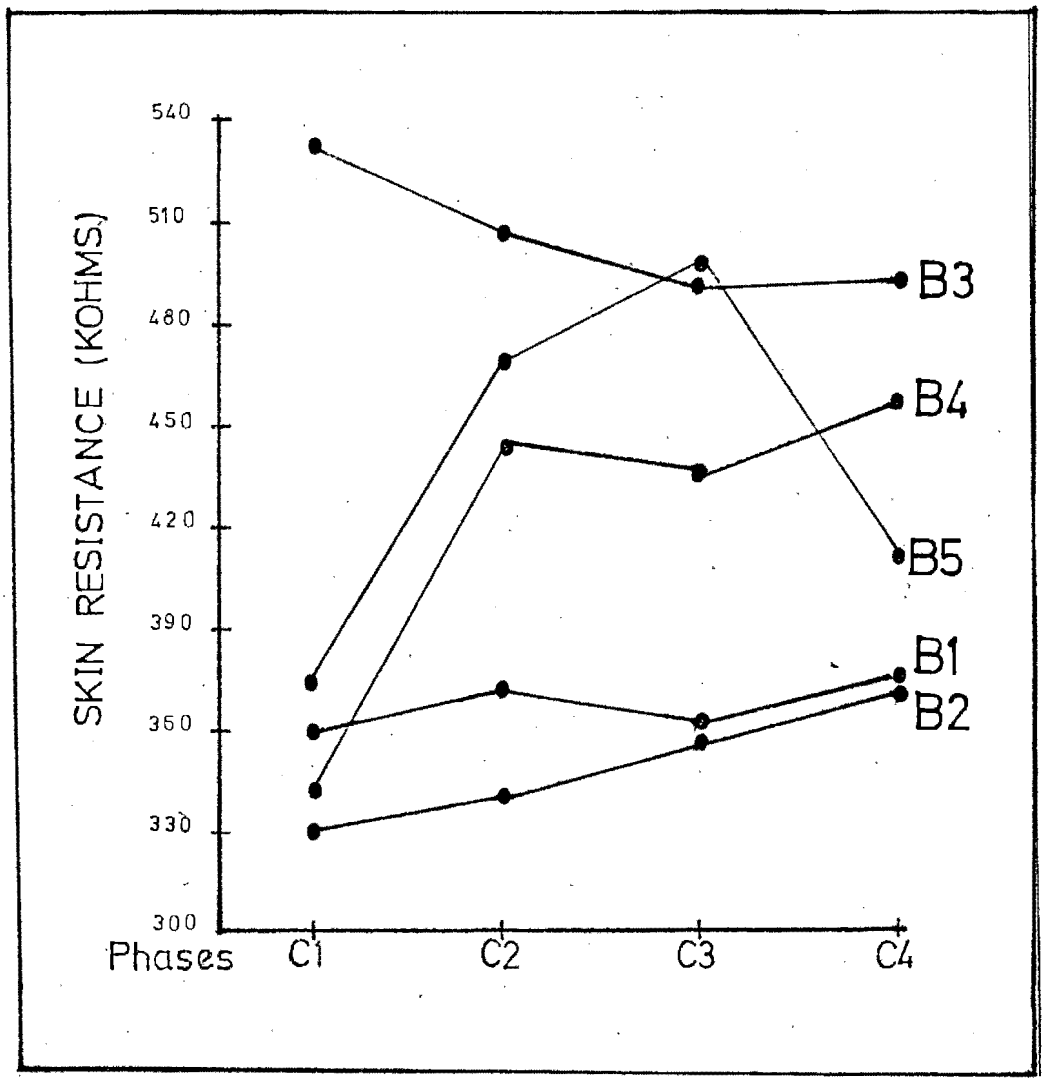


Figure 8 : Skin Resistance (kohms) cell means profiles : Control Group (Factor A2)

5.2. Heart Rate Results

The ANOVA results for HR have been summarized in table 6. THe HR means and SD are set out in table 7.

In the ANOVA, the only significant F-ratio was for factor C at the .01 level (F 4.51, df = 3,30). As no significant interaction effects were found, pairwise comparisons of treatment means for each level of Factor C by Tukey's HSD test was indicated. The treatment means for factor C are presented in table 8. Figure 9 graphically illustrates the HR treatment means profiles for factor C: Pairwise comparisons of these treatment means was not considered meaningful to the present investigation, i.e. such a test would give no indication of differences between either the various treatments, or between the two groups. Referring to figure 9, it is apparent that there was an overall, gradual drop in HR from phase 1 (factor C1) to phase 3 (factor C4). This trend is to be expected from subjects sitting still over a period of 25 minutes, and here is not statistically related to treatment or group effects.

TABLE 6/...

TABLE 6 : Heart Rate ANOVA Summary

SOURCE	SS	DF	MS	F. RATIO	SIG. LEVEL
Between Subj.					
A	579.375	1	579.375	0.168972	
Subj. W.G.	34288.2	10	3428.83		
Within Subj.					
B	270.562	4	67.6406	0.681732	
AB	831.375	4	207.844	2.0948	
BxSWG	3968.75	40	99.2187		
C	188.25	3	62.75	4.86749	.01
AC	9.1875	3	3.0625	0.237557	
CxSWG	386.75	30	12.8917		
BC	53.625	12	4.66875	0.919023	
ABC	44.8125	12	3.73437	0.767995	
BCxSWG	583.5	120	4.8625		

Check on homogeneity of error terms

F.Max. (Subj.W.G.) = 3.2354 DF = 2,5

F.Max. (B x SWG) = 1.14759 DF = 2.20

F.Max. (C x SWG) = 1.04359 DF = 2,15

F.Max. (BC x SWG) = 1.10785 DF = 2,60

TABLE 7 : Heart Rate (Beats per Minute) Means and Standard Deviations.

Mean Phase One : Phase 1

Mean First Half Second Phase : Phase 2(i)

Mean Second Half Second Phase : Phase 2(ii)

Mean Third Phase : Phase 3

EXPERIMENTAL GROUP					
	Pre-Treatment Baseline	First Treatment	Second Treatment	Third Treatment	Post-Treatment Baseline
Phase 1	75.8	69.1	75.7	74.7	73.2
S.D.	17.1	15.9	20.3	19.7	17.1
Phase 2 (i)	72.5	70.9	74.7	74.1	70.5
S.D.	17.2	12.7	17	16.8	16.7
Phase 2 (ii)	72	69.7	75.5	73.6	70.7
S.D.	17.9	13.2	17.2	17.1	17
Phase 3	72.6	68	73.3	70.2	70
S.D.	17.6	15.6	19.1	16.6	15.9
CONTROL GROUP I					
Phase 1	71.1	74.6	69.6	70.1	66.6
S.D.	8.6	15.4	11.2	6.7	9.4
Phase 2 (i)	69.5	74.3	68.3	68.3	66.6
S.D.	8.5	16.2	111.1	8.1	9.2
Phase 2 (ii)	68.9	72.7	67.7	68.3	66.4
S.D.	8.4	13.9	10.4	7.8	9.4
Phase 3	68.6	72.4	67.6	67.5	65.7
S.D.	8.7	12	9.1	6.5	6.5

TABLE 8 : Heart Rate Treatment Means for Factor C (Beats per Minute).

C1	C2	C3	C4
72.05	70.97	63.422	55.928

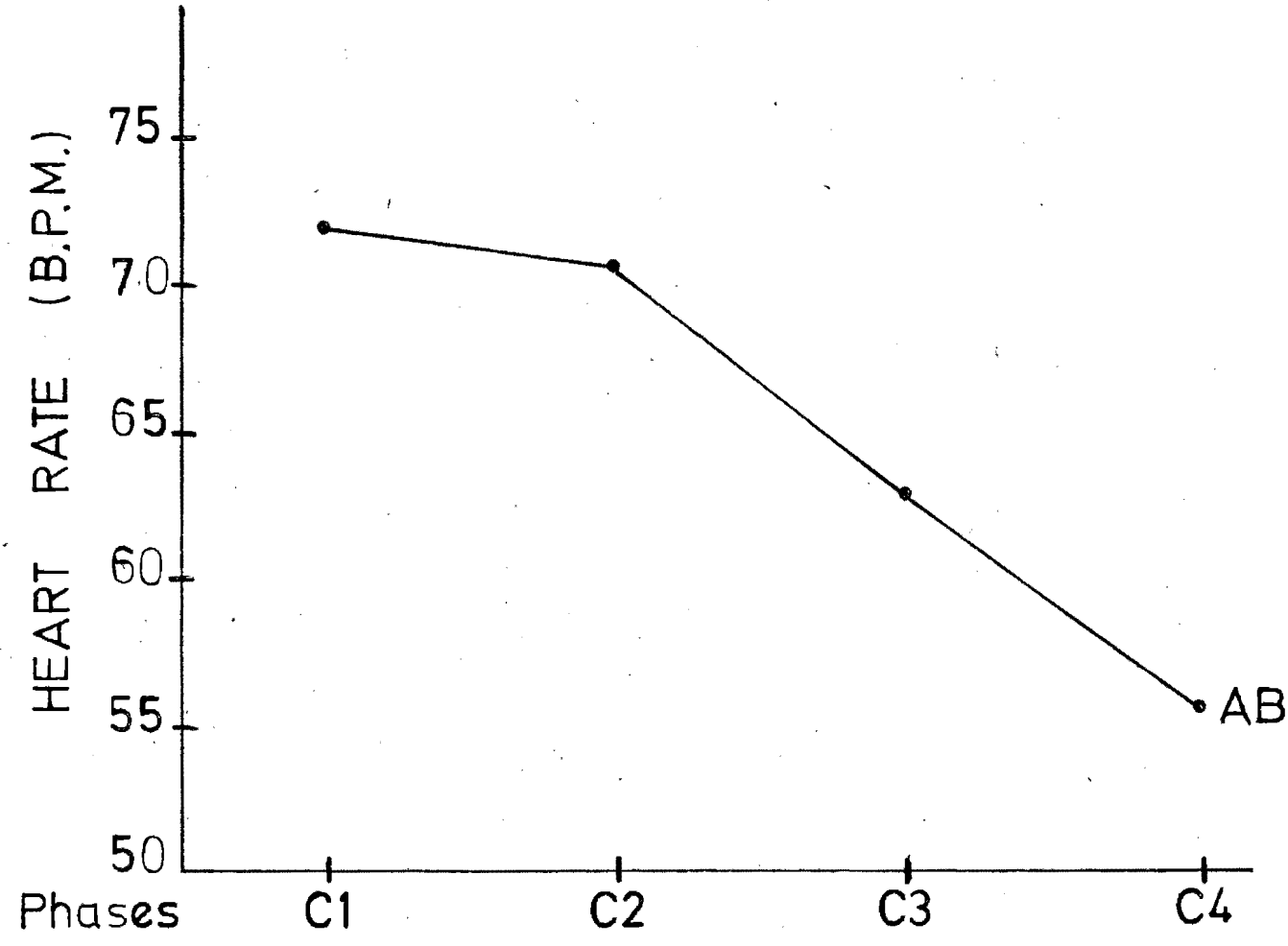


Figure 9 : Heart Rate treatment means profiles for Factor C.

5.3. Respiratory Rate Results

It should be noted that respiration was the dependent variable in this experiment, and thus not necessarily amenable to statistical analysis. However, the respiratory rate data was analysed by the 3-way ANOVA with repeated measures on factor B and C. The significant F-ratio for error term C x SWG confirmed the invalidity of subjecting the dependent variable to statistical analysis, as homogeneity of variance could not be assumed.

The chief interest in respiratory rate analysis, was to obtain the changes in breathing rate in the various treatment situations. Table 9 represents the changes in respiratory rate for each session, taking mean breaths per minute (hereforth referred to as bths/min) in the first phase as the initial value. Positive scores indicate a rise in breathing rate, negative scores a drop in respiratory rate.

It was found that during the last 10 minutes of the deep breathing sessions, breathing rate dropped by 11.08 breaths a minute (bths/min) in the first treatment session; by 11.23 bth/min in the second treatment session; and by 9.26 bth/min in the third treatment session. During the relaxation treatment sessions, bth/min dropped by 1.16 and 1.01 for the first and second treatment sessions, and rose by 1.01 bth/min in the third treatment session.

During baseline conditions, the average drop in breathing rate for the experimental group, during the second phase of the session was 0.72 bths/min. In the control group, breathing rate went up by 0.63 bth/min in the pre-treatment baselines, and dropped by 0.47 bths/min in the post-treatment baseline.

TABLE 9/...

TABLE 9 : Respiratory Rate Change Scores.

Mean deviations from the first phase, for the second and third phases of the 5 sessions. Positive values represent a drop in respiratory rate.

EXPERIMENTAL GROUP					
	Pre-Treatment Baseline	First Treatment	Second Treatment	Third Treatment	Post-Treatment Baseline
Phase 2(i)	1.02	11.47	11.04	9.88	0.32
Phase 2(ii)	0.87	10.68	11.42	8.63	0.66
Phase 3	0.66	3.09	0.74	0.95	1.3
CONTROL GROUP (RELAXATION)					
Phase 2(i)	-0.47	1.08	0.81	-0.63	-0.06
Phase 2(ii)	-0.79	1.25	1.21	-1.39	0.53
Phase 3	-0.92	0.11	-0.04	-1.53	0.39

+ Standard deviations not included, as deviation scores were not taken from an arbitrary initial value and begative scores were included. Mean and SD of above change scores were not computed.

5.4./...

5.4. Muscular Tension Results

The procedure used for scoring muscular tension was described in Section 4.4.3. which should be referred to if there is inadequate understanding of the data presented in Tables 10 to 13. The samples used for EMG data analysis were included in the appendix.

Briefly, part A of each Table gives the total rank obtained by a group for each phase of each session; the larger the rank the more the EMG tension decrease represented, and the smaller the rank the least the EMG tension decrease. A table of change scores derived from the total ranks (part B of the table) represents the amount by which phase one's total rank differed from that of each of the subsequent phases. A large change score indicates greater EMG tension decreases than small or negative score; the maximum EMG tension decrease would have the score of 18 and the least EMG decrease a score of -18. 0 would represent no tension changes. Part C of the Table ranks sessions according to their change scores. As there was a total of 6 sessions, the ranks ranged between 1 and 6.

Rank 1 was given to the smallest change score (least EMG tension decrease) and Rank 6 to the largest change score (greatest muscular tension decrease). The hypothesis was that according to probability theory treatment and baseline sessions should have each 50% chance of ranking first and second, as of ranking fifth and sixth.

5.4.1. Masseter Muscle Results

Table 10 gives the ranking order of the experimental group for the masseter muscle, for phase 2(i), 2(ii), and 3. It should be remembered that phases 2(i) and 2(ii) of treatment sessions represent the deep breathing period. For both phases 2(i) and 2(ii), the

first/...

first baseline session and the first treatment sessions respectively got the ranks 1 and 2, thus representing those sessions in which the least muscular tension decrease occurred. This consists of a 50% distribution of the lower ranks between treatment and baseline sessions. However, treatment sessions 2 and 3 ranked fifth and sixth during both phases 2(i) and 2(ii), thus suggesting greater EMG decreases for the whole period of deep breathing in these treatment sessions relative to other sessions. As to the magnitude of the difference between change scores for phase 2(i), the first two ranks obtained a joint change score of 0 (no EMG decrease) while treatments 2 and 3 had a change score of 13.2 making a difference of 13.2 between the two sets of change scores. This is above the set amount of 12 as minimum difference to be found reliable. Considering that 36 would be the maximum difference obtainable between two sets of change scores, a difference of 13.2 represents 37% ($\frac{13.2}{36}$ %) more EMG decrease in the high ranking sessions compared with the low ranking ones. For phase 2(ii) the lower ranking sessions had a change score of 2.4 while for the fifth and sixth ranks the change score was 20.4, a difference of 18 points between the lower and higher ranking sessions. This represents 57% more EMG decrease in phase 2(ii) of treatment sessions 2 and 3 than in the corresponding phase of pre-treatment baseline 1 and treatment 1. It is not altogether surprising to find little EMG tension decrease in treatment 1 which was reported to have been extremely uncomfortable by the experimental group: the skin resistance data showed consistent trends, suggesting increased levels of arousal. It may be considered that the first treatment session was unrepresentative of the other treatment sessions as to EMG tension patterns, as was found for skin resistance (see Section 6.2.)

It/...

It should be noted that were the first treatment's EMG data excluded, pre-treatment baseline 2 would rank second, and the change score for the first two baselines in phase 2(ii) would increase from 2.4 to 3, as compared to 20.4 for treatment sessions 2 and 3, yielding a difference of 17.4 (than 12) between the two; this represents 48% more EMG decrease in treatment sessions 2 and 3 compared to pre-treatment baselines 1 and 2. It may be concluded that greater EMG tension decreases occurred during treatment sessions 2 and 3 than in baseline sessions 1 and 2; it may also be concluded that treatment session 1 had reliably less masseter EMG decreases than did the other two treatment sessions. For phase 3, baselines 1 and 2 ranked first and second; baseline 3 and treatment 3 ranked fifth and sixth. The first two ranks had the joint score of 3 and the last two of 21.3. This amounts to a difference of 18.3 between them ($>$ than 12). However, baseline 3 ranked fifth with treatment 3 sixth, thus consisting of 50% probability for treatment on baseline session to be found in the higher ranks in the third phase.

Considering the masseter muscle EMG data for the Control Group (Table 11), the following ranks were obtained: for phase 2(i), baseline 2 and treatment 3 both ranked first. Treatment 2 and 1 ranked fifth and sixth respectively. For phase 2(ii), baseline 2 and 1 ranked first and second; baseline 3 and treatment 1 ranked fifth and sixth. This gives a distribution of 3 baselines and 1 treatment in the first two ranks, and of 3 treatments and 1 baseline in the last two ranks, when the whole of phase 2 is considered. This amounts to 66.67% probability of finding a baseline session in the first two ranks and equally of finding a treatment session in the last two ranks. Considering the differences between change scores, baseline 2 and treatment 3 obtain a total of 4 for phase 2(i) and treatment 2 and

1 a/...

1 a score of 8.8, a difference of 4.8 in change scores which cannot be considered reliable, especially in view of only 66.67% probability which was found in the ranking distribution for both phases 2(i) and 2(ii). For phase 2(ii) a total of 2 is obtained from change scores of baselines 1 and 2; baseline 3 and treatment session 1's change scores amounted to 10.8, the difference between the sets of change score being 8.8 (< than 12) representing 24% more masseter EMG decrease in the high ranking sessions compared with the low ranking ones. During phase 3, baseline session 2 still occupied the first rank, with baseline session 1, treatment sessions 2 and 3 all occupying the second rank with change scores of 5. Baseline 3 and treatment 1 remained at the fifth and sixth ranks respectively with change scores of 15.6, consisting of 10.6 (< than 12) difference between the sets of change scores i.e. 29% more EMG decrease in high ranking sessions compared with low ranking ones. The masseter EMG change scores (part B of the tables) were used for graphically representing the EMG tension changes in Figure 10. The six sessions were represented alongside each other for the experimental group and the control group so as to allow inter-session comparisons. The dotted line with the value of 0 on the Y-axis indicated no EMG tension decrease.

5.4.2. Corrugator Muscle EMG Results

The ranks obtained for corrugator EMG by the experimental group (see Table 12) were: for phase 2(i), baseline 3 and treatment 1 both came first; baseline session 2, treatment sessions 2 and 3 all ranked third, and baseline 1 sixth. This corresponds to equal probability of finding a baseline or treatment session of the lower or higher ranks. The total change score for the first two ranks (baseline 3

and/...

and treatment 1 both came first; baseline session 2, treatment sessions 2 and 3 all ranked third, and baseline 1 sixth. This corresponds to equal probability of finding a baseline or treatment session of the lower or higher ranks. The total change score for the first two ranks (baseline 3 and treatment 1) was 0 and for the last two ranks (treatment 3 and baseline 1), 6. This gives a difference of 6 (< than 12) in change scores, and thus was not considered a reliable difference. For phase 2(ii) baseline 3 ranked first, and baseline 2 together with treatment 3 ranked second with a total change score of 1. Treatment 2 and baseline 1 ranked fifth and sixth with a score of 8, thus amounting to a difference of 7 (< than 12) between change scores. The ranking distribution for this phase cannot be used to distinguish between baseline and treatment sessions, with two baselines and one treatment session in the lower ranks, and one treatment with one baseline session in the higher ranks. For phase 3, treatments 2 and 3 ranked first and second with a total change score of 2; baselines 2 and 3 ranked fifth and treatment 3 sixth, with a total change score of 13.2. This yields a difference of 11.2 (< than 12) between change scores which is just under the level considered reliable: this represents 31% more EMG decrease in the treatment sessions than in the baselines during phase 3.

For the Control group (see Table 13) during phase 2(i), baseline sessions 1 and 3 ranked first and second with a total change score of 3; treatment sessions 3 and 1 ranked fifth and sixth with a total change score of 11, the difference between these scores being 8 (< than 12), representing 22% more corrugator EMG decrease in treatment sessions 3 and 1 compared with baseline sessions 1 and 3. There was a 100% consistency in treatment v/s baseline ranking, with

two/...

two baselines ranking first and second, and the upper ranks being o occupied by two treatment sessions, It may be concluded that larger EMG tension drop did occur in treatment sessions 1 and 3 than in baseline 1 and 3 (the first pre-treatment baseline and the post-treatment baseline) although the difference between change scores was not great enough to be considered reliable. A similar ranking distribution occurred in phase 2(ii), with baselines 2 and 1 ranking first and second with a total change score of 3; fifth and sixth were treatment sessions 3 and 2 with a total change score of 13. The difference between change scores amounts to 10 (< than 12), indicating 28% more EMG tension decrease in the high ranking sessions compared with the low ranking ones. It should be noted that different baselines and treatment sessions were found in lower and higher ranks for both phases 2(i) and 2(ii). However, the ranking distribution does reflect a higher than chance probability of finding baselines or treatments in the first and last ranks. During phase 3, a 50% probability ranking distribution was found, with baseline 2 and treatment session 3 occupying the first and second ranks respectively with a total change score of 5; treatment session 2 together with baseline 1 ranked fourth and baseline 3, sixth with a total change score of 15. The difference between change scores is 10 (< than 12). The corrugator EMG tension decreases are graphically represented in Figure 11: the graphs were constructed in the same manner as was Figure 10 (see page 98).

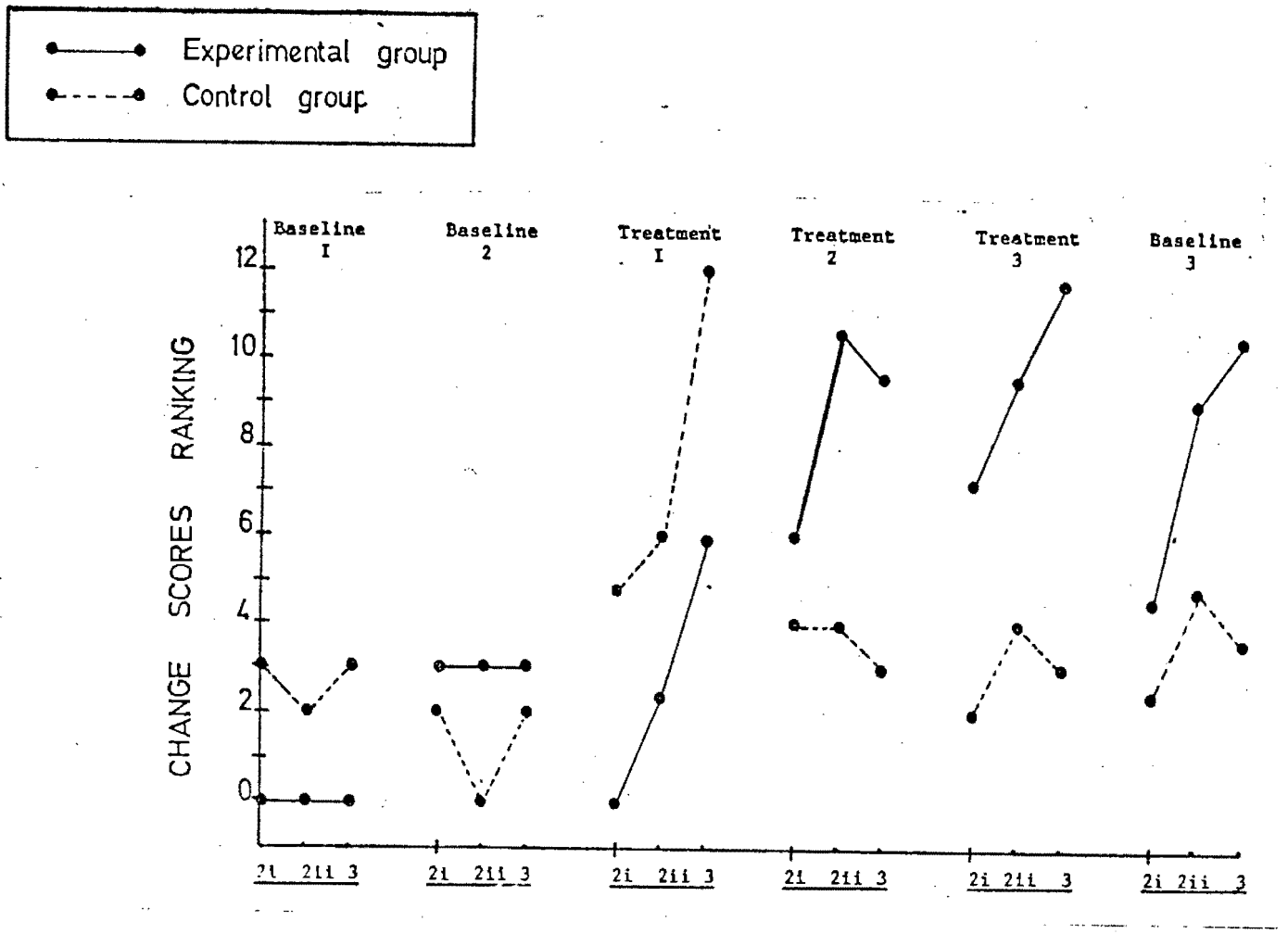


FIGURE 10: Masseter EMG Tension.

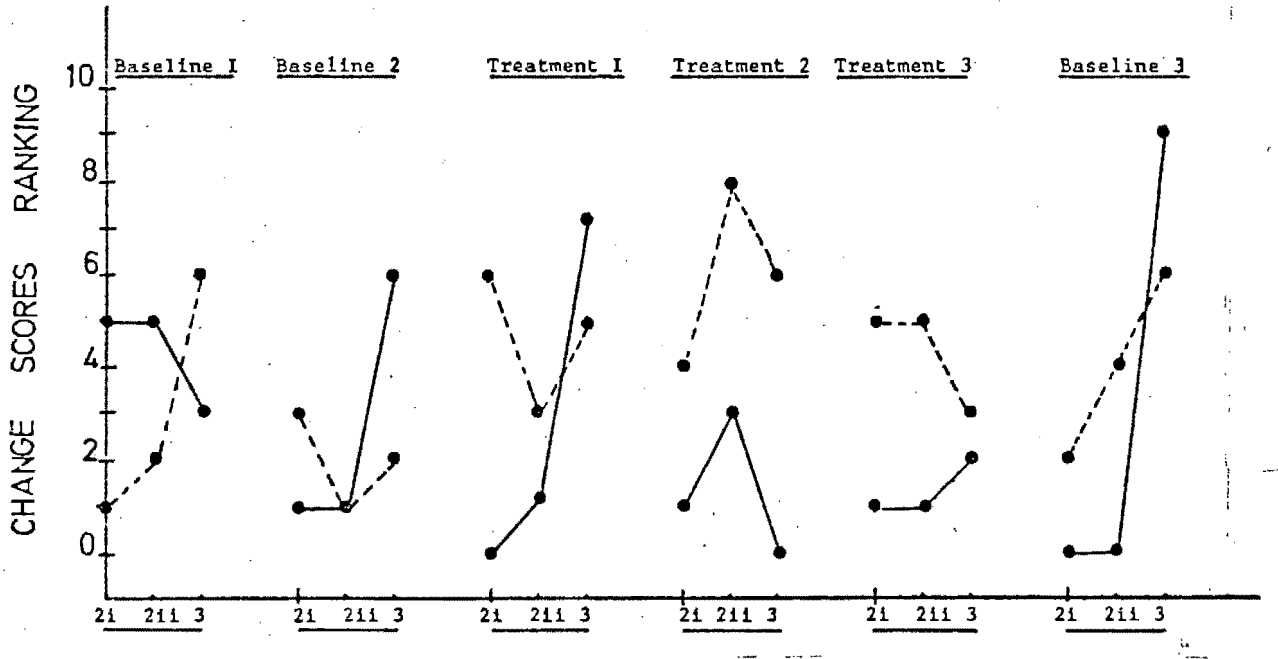


FIGURE 11 : Corrugator EMG Tension.

The value of 0 on the y-axis represents no EMG tension changes.

TABLE 10 : Masseter EMG Results : Experimental Group

PART A

	Pre-Treatment Baseline 1	Pre-Treatment Baseline 2	Post-Treatment Baseline 3	Treatment 1	Treatment 2	Treatment 3
Phase 1	9.6	7.5	7.5	9.6	6	7.2
Phase 2(i)	9.6	10.5	12	9.6	12	14.4
Phase 2(ii)	9.6	10.5	16.5	12	16.8	16.8
Phase 3	9.6	10.5	18	16.6	15.6	18

PART B

	Pre-Treatment Baseline 1	Pre-Treatment Baseline 2	Post-Treatment Baseline 3	Treatment 1	Treatment 2	Treatment 3
Phase 2(i)	0	3	4.5	0	6	7.2
Phase 2(ii)	0	3	9	2.4	10.8	9.6
Phase 3	0	3	10.5	6	9.6	10.8

P.T.O. for PART C

TABLE 10 (continued)

PART C : Masseter EMG Results : Experimental Group

Ranking of change scores

Phase 2(i)	Total change score
1. Pre-treatment baseline 1	0
. Treatment session 1	
3. Pre-treatment baseline 2	
4. Post-treatment baseline 3	
5. Treatment session 2	13.2
6. Treatment session 3	
Phase 2(ii)	
1. Pre-treatment baseline 1	2.4
2. Treatment session 2	
3. Pre-treatment baseline 2	
4. Post-treatment baseline 3	
5. Treatment session 2	20.4
6. Treatment session 3	
Phase 3	
1. Pre-treatment baseline 1	3
2. Pre-treatment baseline 2	
3. Treatment session 1	
4. Treatment session 2	
5. Post-treatment baseline 3	21.3
6. Treatment session 3	

TABLE 11 : Masseter EMG Results : Control Group

PART A

	Pre-Treatment Baseline 1	Pre-Treatment Baseline 2	Post-Treatment Baseline 3	Treatment 1	Treatment 2	Treatment 3
Phase 1	9	9	9.6	6	6	9
Phase 2(i)	12	11	12	10.8	10	11
Phase 2(ii)	11	9	14.4	12	10	13
Phase 3	12	11	13.2	18	9	12

	Pre-Treatment Baseline 1	Pre-Treatment Baseline 2	Post-Treatment Baseline 3	Treatment 1	Treatment 2	Treatment 3
Phase 2(i)	3	2	2.4	4.8	4	2
Phase 2(ii)	2	0	4.8	6	4	4
Phase 3	3	2	3.6	12	3	3

P.T.O. for PART C

TABLE 11 (continued)

PART C : Masseter EMG Results : Control Group

Ranking of change scores

Phase 2(i)		Total change scores
1.	Pre-treatment baseline 2	4
1.	Treatment session 3	
3.	Post-treatment baseline 3	
4.	Pre-treatment baseline 1	
5.	Treatment session 2	8.8
6.	Treatment session 1	
Phase 2(ii)		
1.	Pre-treatment baseline 2	2
2.	Pre-treatment baseline 1	
3.	Treatment session 2	
3.	Treatment session 3	
5.	Post-treatment baseline 3	10.8
6.	Treatment session 1	
Phase 3		
1.	Pre-treatment baseline 2	
2.	Pre-treatment baseline 1	5
2.	Treatment session 2	
2.	Treatment session 3	
5.	Post-treatment baseline 3	15.6
6.	Treatment session 1	

TABLE 12 : Corrugator EMG Results : Experimental Group

PART A

	Pre-Treatment Baseline 1	Pre-Treatment Baseline 2	Post-Treatment Baseline 3	Treatment 1	Treatment 2	Treatment 3
Phase 1	7	10	9	9.6	12	13
Phase 2(i)	12	11	9	9.6	13	12
Phase 2(ii)	12	11	9	10.8	15	14
Phase 3	11	15	15	16.8	12	15

PART B

CHANGE SCORES

	Pre-Treatment Baseline 1	Pre-Treatment Baseline 2	Post-Treatment Baseline 3	Treatment 1	Treatment 2	Treatment 3
Phase 2(i)	5	1	0	0	1	1
Phase 2(ii)	5	1	0	1.2	3	1
Phase 3	3	6	6	7.2	0	2

P.T.O. for PART C

TABLE 12 (continued)

PART C : Corrugator EMG Results : Experimental Group

Ranking of change scores

Phase 2(i)		Total change score
1.	Post-treatment baseline 3	0
1.	Treatment session 1	
3.	Pre-treatment baseline 2	6
3.	Treatment session 2	
3.	Treatment session 3	
6.	Pre-treatment baseline 1	
Phase 2(ii)		
1.	Post-treatment baseline 3	1
2.	Pre-treatment baseline 2	
2.	Treatment session 3	
4.	Treatment session 1	
5.	Treatment session 2	8
6.	Pre-treatment baseline 1	
Phase 3		
1.	Treatment session 2	2
2.	Treatment session 3	
3.	Pre-treatment baseline 1	
4.	Pre-treatment baseline 2	13.2
4.	Post-treatment baseline 3	
6.	Treatment session 1	

TABLE 13 : Corrugator EMG Results : Control Group

PART A

	Pre-Treatment Baseline 1	Pre-Treatment Baseline 2	Post-Treatment Baseline 3	Treatment 1	Treatment 2	Treatment 3
Phase 1	9	9	11	9	8	8
Phase 2(i)	10	12	13	15	12	13
Phase 2(ii)	11	10	15	12	16	13
Phase 3	15	11	20	14	14	11

PART B

CHANGE SCORES

	Pre-Treatment Baseline 1	Pre-Treatment Baseline 2	Post-Treatment Baseline 3	Treatment 1	Treatment 2	Treatment 3
Phase 2(i)	1	3	2	6	4	5
Phase 2(ii)	2	1	4	3	8	5
Phase 3	6	2	9	5	6	3

P.T.O. for PART C

TABLE 13 (continued)

PART C : Corrugator EMG Results : Control Group

Ranking of change scores

		Total change scores
Phase 2(i)		
1.	Pre-treatment baseline 1	3
2.	Post-treatment baseline 3	
3.	Pre-treatment baseline 2	
4.	Treatment session 2	
5.	Treatment session 3	11
6.	Treatment session 1	
Phase 2(ii)		
1.	Pre-treatment baseline 2	3
2.	Pre-treatment baseline 1	
3.	Treatment session 1	
4.	Post-treatment baseline 3	
5.	Treatment session 3	13
6.	Treatment session 2	
Phase 3		
1.	Pre-treatment baseline 2	5
2.	Treatment session 3	
3.	Treatment session 1	
4.	Pre-treatment baseline 1	15
4.	Treatment session 2	
6.	Post-treatment baseline 3	

6. Discussion

6.1. Research Strategy

The running of eight sessions in a repeated measurements design was used so as to determine the effects of two main relaxation techniques (deep breathing and muscular relaxation) upon level of physiological arousal. The following strategies were used to control for some of the more frequent methodological shortcomings which were discussed in Section 2.1. (pages 12-17.)

1. A repeated measures design was used so as to control for the large inter-individual differences in physiological function. Thus, each technique could be assessed from the differences found between baseline rest and treatment conditions in the same subjects.

2. An habituation session, prior to the first baseline session allowed for the subject to become familiar with the experimental procedure thus controlling for the effect a novel and strange situation could have had upon physiological arousal. A few subjects at the beginning of the experiment were required to attend two habituation sessions. However, the subject had visited the laboratory prior to the first habituation session, and it was found that he reported complete familiarity with the experimental setting and procedure after the first habituation session; the second habituation session unnecessarily added to what subjects considered an already lengthy experimental program, and it was thus eliminated.

3. Readings from two pre-treatment baselines were meaned to ensure that representative samples of baseline physiological

function/..

function were obtained for each subject for comparison with the three treatment sessions.

4. Three treatment sessions permitted the evaluation of practice effects upon the technique, as well as the consistency with which treatment effects were recorded.

5. A post-treatment baseline was introduced so as to ensure that any physiological changes occurring during the course of the experiment were due to treatment effects, and not, for example, to habituation to the experimental situation.

6. Five physiological parameters were measured (excluding respiratory rate which was the dependent variable) so as to get a representative view of physiological arousal. Goldstein (1965) suggested that conclusions arising out of psychophysiological research could well depend upon the particular measurements used. Lacey (1950) noted that individuals reacted more in one function e.g. HR, while others reacted more in another function, e.g. blood pressure. Monitoring several physiological parameters was thus believed to provide a reliable means of assessing the effects of the treatments upon physiological function.

7. Each of the two groups (experimental and control group) contained an equal number of males and females (3 males and 3 females in each group) thus controlling for the effects of sex differences upon physiological function.

8. Subjects were of approximately the same age (17-26 years) thus controlling for age variables. The main shortcomings of the present research was that only 6 subjects were used in each group. This was chiefly due to difficulty in recruiting volunteers in the present experiment. It was partly compensated for by the running of several sessions per subject, thus ensuring that reliable

physiological/...

physiological measurements were obtained.

The scope of this research was largely restricted by equipment shortage. Some equipment without which the research would have been difficult, was obtained by special arrangements (e.g. the respiratory transducer, the EEG Power Spectrum Analyser). As such complex equipment as the EEG Analyser was being used for the first time in the present experiment, it was inevitable that certain difficulties be encountered primarily in its use. It was unfortunate that the shortage of magnetic tape reels made it impossible for the experimenter to store all the data until the analyser was modified so as to function optimally. Various attempts were made to order more reels; however, these had to be ordered from the U.S.A. and when the order finally arrived, the EEG Analyser was already modified, and a large part of the data had been lost. The EEG Power Spectrum Analyser had been built so as to accept too limited an amplitude range. The restriction of amplitude range had kept the cost of the machine to a minimum, as Mr. Timm was aware of the experimenter's restricted funds, (see Section 4.2.7.)

A second major shortcoming due to equipment shortage in this research, regarded the unavailability of means of recording ventilation changes, thus making it impossible to objectively define the changes involved in deep breathing. It was fortunately possible to gain some idea of the breathing changes involved mainly from the Heart Rate data (HR most often shows parallel changes with respiratory activity) and subjective reports. However, the present research must be considered as having had primarily exploratory aims, mainly that of demonstrating that the technique of deep breathing can be used as a consistent reliable means of lowering physiological arousal.

Future/...

Future research will be needed to gain objective knowledge of ventilation changes involved, so as to arrive at an adequate explanation of the success of the technique in lowering physiological arousal.

6.2. Discussion of Skin Resistance Results

Before interpreting SR results, the relationship between SR level and arousal is to be noted. High palmar skin resistance has been associated with such behavioural states as sleep, relaxation, boredom, while low levels of palmar SR were found to accompany states of emotional arousal, alertness, and psychopathology (Edelberg, 1972, see review by; McDonald et al, 1976; Mundy-Castle and McKiever, 1953).

McCurdy (1950) reported from various studies correlations ranging between .53 and 1.00, most of them above .70, linking subjective evaluation of stimuli intensity and the magnitude of skin resistance drop in response to these stimuli (psychogalvanic reflex). The present researcher has had the opportunity of observing a close connection between subjective reports of alertness or relaxation and level of SR. In one pilot study conducted on an experienced yoga student who was to practice certain breathing exercises, the experimenter noticed an initial rise in palmar SR upon initiation of one type of breathing exercise, followed by a sharp drop in SR; subsequently SR again rose markedly until the end of the session. After the session the yoga student reported having felt relaxed during the first part of the session during the one type of breathing exercise; she then stated that she could no longer keep on the breathing and had felt very restless as she felt uncomfortable and cold. Finally, she had resumed a different type of breathing exercise which had permitted

her/...

her to relax again. It is noteworthy that the verbal report parallels the trends found in SR very closely, in a pattern of relaxation-alertness-relaxation.

From this instance and several other times when the close link between subjective relaxation-alertness and SR level was observed over the course of the experiment, it was considered that the SR data was the single most important physiological parameter in the present research.

Briefly summarising the significant SR results presented in section 5.1., the experimental group were found to have significantly higher SR levels in phase 2(ii) (factor C3) than in phase 1 (factor C1) of treatment sessions 2 and 3; the experimental group's SR in the post-treatment baseline showed a near-significant drop in phase 3 (factor C4) as compared to phase 2(i) (factor C2) (falling 0.4 of the .05 level of significance in Tukey's HSD test).

For the control group, only two pairs of means were found to differ significantly by Tukey's HSD test: that was in the post-treatment baseline, where SR was significantly higher in phase 2(ii) (factor C3) than in phase 1 (factor C1).

The present discussion was conducted with reference to the graphs in Figure 12 illustrating changes in SR from the arbitrary initial value of 500k, so as to allow meaningful visual comparisons between sessions and groups, without regard to differences in initial SR level. It should be noted that there were initial differences in SR level from one individual to the other, and for the same individual at various sessions which although not significant, made it difficult to visually compare changes in SR between groups and sessions; the present discussion has concerned itself with comparisons of SR changes quite regardless of the initial value which was not found to have a

significant/...

significant effect on SR changes; a look at table 5 (page 82), where means and SD of SR for each group, session and phase were tabulated will indicate that increases or decreases in SR levels were not related to low or high initial values (SR level in the first phase).

The graphs in Figure 12 which represent the changes in SR in the 5 sessions, were thus designed in the following manner: the dotted line parallel to the X-axis represents SR level at the end of the first phase of the session. This line may thus be referred to as the "Initial Value" of SR. The three points above or below this dotted line in each graph, indicate the changes in SR (in khoms) which occurred in the subsequent phases relative to the initial value (i.e. relative to SR value at the end of the first phase). Two resistance values were derived from the second phase, the last 10 minutes of which were subdivided into two 5-minute periods, the first of these was labelled 2(i) and the last as 2(ii) (refer to Figure 4, page 65). SR changes in the two parts of the second phase and the third phase were calculated by subtracting the resistance values of these phases from that of the first phase. These change scores were expressed as deviations from an arbitrarily set initial level of 500k. This initial value was chosen so as to avoid the use of negatives: the means and standard deviations of these change scores may be found in Table 14.

Thus a score of 520k indicated a rise in SR of 20k, and 480k represents a drop in SR by 20k after the first phase. In the graphs in Figure 12, the initial value of SR at the end of the first phase for each session was represented by a dotted line having the arbitrary value of 500k on the Y-axis. The points falling below the 500k line indicate a decrease in SR: those lying above the dotted line represent an increase in SR; the five sessions were graphically represented

Table 14/...

TABLE 14 : Skin Resistance (KOHMS) : Change scores in the second and third phases from initial value in phase 1 (arbitrarily set at 500k)

First half of second phase : Phase 2(i)

Second half of second phase : Phase 2(ii)

Third phase : Phase Three

	Pre-Treatment Baseline	First Treatment	Second Treatment	Third Treatment	Post-Treatment Baseline
EXPERIMENTAL GROUP					
Phase Two I	519	497.67	580.83	597.33	515.5
S.D.	46.39	55.12	183.61	120.47	78.40
Phase Two II	546.83	488.	611.5	627.67	431.67
S.D.	101.84	71.03	236.72	90.22	187.24
Phase Three	522.17	464.33	595.5	595.33	386.17
S.D.	50.64	83.36	192.3	129.75	165.23
CONTROL GROUP (RELAXATION)					
Phase Two I	511.83	509.5	475.67	605.33	647.0
S.D.	53.02	16.3	38.72	160.24	139.6
Phase Two II	503.0	527.17	459.67	596.67	619.67
S.D.	79.4	60.81	46.48	131.98	107.69
Phase Three	514.5	542.33	461.67	618.5	537.5
S.D.	82.11	116.90	127.95	96.31	143.07

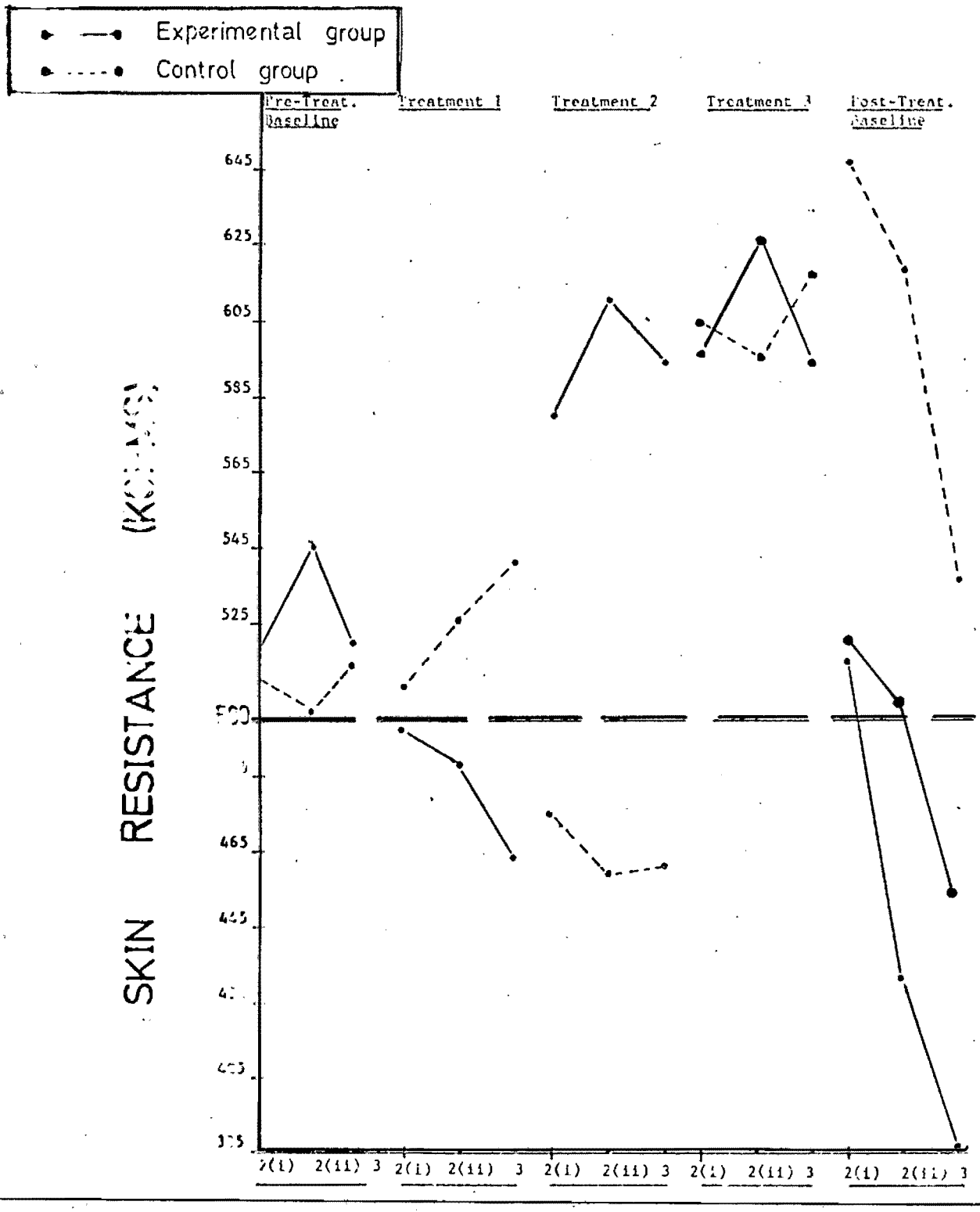


Figure 12 b : SR changes in the Experimental Group in the Post-Treatment Baseline excluding S_E

on the same set of axes alongside each other. On the extreme left is the pre-treatment baseline; and the other sessions follow in the order in which they were run, with the post-treatment baseline lying on the extreme right hand side of the graph. Figure 12 permits direct comparisons of SR changes in the various sessions by visual inspection.

6.2.1. The Experimental Group : SR changes

The experimental group showed significantly higher SR means in phase 2(ii) compared to phase 1 of the second and third treatment sessions. Referring to Figure 12, it is apparent that SR steadily increased during both parts of phase 2 (consisting of the whole deep breathing period). During the second half of the deep breathing period (phase 2(ii)) SR level was significantly higher than in phase 1 at .05 level (t' 3.68, df = 4,75). In the second treatment session, the mean SR in phase 1 was 469.333k, and had increased to 580.8331 at the end of phase 2(ii). The pattern of SR change during this treatment session is noteworthy, i.e. there was an increase of 80.8k in SR during the first half of the deep breathing period (i.e. phase 2(i)); by the end of that period (phase 2(ii)) SR had increased by 111.5k. During the third phase, when deep breathing was discontinued, SR remained at high levels, but no further increases occurred; in fact, there was a 16k drop in SR after the end of deep breathing (i.e. after phase 2(ii)). These trends tend to suggest that there were significant increases in SR levels during the second half of deep breathing which were discontinued after cessation of the breathing, i.e. that the SR increases were specific to the deep breathing period. During the third treatment session, the experimental

group/...

group exhibited exactly the same pattern of SR changes as in the second treatment session (see Figure 12), i.e. SR increased steadily during the whole deep breathing period (phase 2(i) and 2(ii)). Only in phase 2(ii) were these increases significantly higher than in phase 1, at the .05 level (t' 3.68, df = 4,75). Mean SR in phase 1 of the third treatment session was 492.167k; in phase 2(ii) SR level reached 619.833k, a rise of 127.7k from initial SR value in phase 1. This represents a slightly greater net increase in SR during the third as compared to the second treatment session (see figure 12). It may be noted that SR showed a steeper rise throughout the deep breathing period of treatment session 3, as compared to treatment session 2. Another similarity in SR trends in treatment sessions 2 and 3 in the experimental group, is that after cessation of deep breathing (during phase 3), SR dropped slightly in treatment session 3 by 32.3k from the level reached at the end of deep breathing in phase 2(ii).

These trends, which are common to both treatment sessions 2 and 3, for the experimental group, are extremely relevant in assessing the importance of treatment effects, i.e. increases in SR are normally associated with low physiological arousal. In the present experimental situation, the subjects sat for 25 minutes in a darkened silent room: it may therefore be expected that SR would increase gradually over the whole experimental session. Such a trend is apparent in the pre-treatment baseline. Furthermore, it may also be considered possible that these SR increases would be accentuated over the sessions, as the subject grew more familiar with the laboratory situation. From figure 12, it as apparent that the experimental group's SR trends differed from the above-mentioned relaxation - laboratory habituation pattern in two ways:

Firstly/...

Firstly, SR was found to be significantly higher during phase 2(ii) than during phase 1, in treatment sessions 2 and 3; no significant findings for phase 3 were observed. If the subjects were getting more relaxed as a function of time, it would be expected that SR would rise even higher during phase 3 which is that last part of the session. It could be argued that the buzzer which was sounded after the end of phase 2 disturbed the experimental group, thus preventing further SR increases during the 7½ minutes that followed; it should be noted, however, that the same buzzer was used after phase 1 of each session; figure 12 indicates that in the 7½ minutes following the first buzzer (i.e. phase 2(ii)), the experimental group did show significant SR increases in treatment sessions 2 and 3, thus contradicting the proposition that the buzzer could account for the non-significant differences in SR in phase 3 as compared to phase 1, of treatment sessions 2 and 3.

In accounting for the significant increases in SR from phase 1 during the second half of the deep breathing period (phase 2(ii)), in treatment sessions 2 and 3, the factor of habituation to the experimental situation must be considered. The subjects could have shown significant SR increases from initial level at the end of phase 2, and not during phase 3, for various reasons unrelated to treatment effects, e.g. knowledge that the experimental session was nearing its end, and thus preparing themselves for activity once more. It should be noted that no statistical backing may be relied upon in accepting or rejecting this explanation of SR trends in the experimental group: referring to figure 12, two observations may be made, however, which put into question the validity of this argument. Firstly, were the subjects to respond to the buzzer after phase 2(ii) with alert, highly aroused anticipatory behaviour, SR would not have remained at such high levels during phase 3. It should be noted that

SR did not continue increasing during phase 3, but nor was there any significant drop in SR; it should be remembered that SR readings were taken at the last minute of each phase; thus, during phase 3, $7\frac{1}{2}$ minutes after the second buzzer, SR remained at high levels; in treatment session 2, SR dropped by 16k from the level reached in phase 2(ii); in treatment session 3, SR dropped by 32.3k from the level reached in phase 2(ii), These drops must be considered in relation to the significant SR increases in phase 2(ii) of these two treatment sessions, which were 111.5k and 127.67k respectively.

A further argument against the significant SR increases in phase 2(ii) of treatment session 2 and 3 being due to habituation to the laboratory situation, lies in the near significant SR decreases found for the experimental group in the post-treatment baseline. Figure 12 clearly illustrates the difference in SR trends in the experimental group during the post-treatment baseline. After a slight increase in SR during phase 2(i), which was comparable in magnitude to pre-treatment baseline changes, SR dropped steadily from initial value in phase (by 68.33k in phase 2(ii) and 113.83k in phase 3). The near-significant F-ratio obtained from the experimental group during that session (falling short of the .05 level of significance by 0.15) indicates that a SR pattern opposite to that observed during the treatment sessions was present. This observation does indicate that the significant SR increases during treatment sessions 2 and 3 were most probably not due to habituation to the laboratory situation. However, such a near-significant drop in SR during the latter part of the post-treatment baseline sets it off from the trend observed in the pre-treatment baseline (see figure 12). The data was scanned for extreme scores; one subject (henceforth referred to as S_E) was found to show unusually extreme scores in

phase/...

phase 2(ii). Figure 12B shows the SR changes of the experimental group in the post-treatment baseline when S_E was excluded from the group: this graph (figure 12b) was represented on tracing paper so that it could be superimposed upon figure 12 for purposes of comparison: it may be noted that in figure 12b the drop in SR during the second half of phase two (2(ii)) was considerably reduced when S_E was excluded (consisting of a 16k drop from phase 2(i) instead of 83.8k). However, during the third phase of the post-treatment baseline, the drop in SR was accentuated when S_E was excluded. This may be due to S_E having reached a plateau after the large SR drop which had already occurred in phase 2(ii), thus no longer showing SR decreases in the third phase while the other subjects did. This drop in SR in the final phase of the last experimental session was common to the control group. Most subjects reported feeling restless towards the end of the post-treatment baseline, as the long-term experiment had proved to be long and they had felt glad it was over. The post-treatment baselines had been specifically resented by the experimental group who had enjoyed the practice of deep breathing but found the last baseline to be a "drag". Both parts of the second phase of the post-treatment baseline showed comparable SR changes to the corresponding phases of the pre-treatment baseline for the experimental group when S_E was excluded.

6.2.2. The Control Group : SR changes

The only significant pair of means found to differ for the Control group, was in the post-treatment baseline where SR was significantly higher at the end of phase 2(ii) than in phase 1 (a rise of 119.667k

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from initial level). After consultation of figure 12, it is immediately apparent that the post-treatment baseline SR trends for the control group was more comparable to the third treatment session (i.e. the preceding session) than to the pre-treatment baseline. The significantly higher SR in phase 2(ii) compared to phase 1, in the post-treatment baseline for the control group could be interpreted in two ways. Either it could be postulated that this trend reflected habituation effects to the experimental situation, thus suggesting that the control group showed no treatment effects in SR trends during the experiment. Or the increased SR levels in phase two of the post-treatment baseline could be due to a generalization of treatment effects to that session, without the subject's conscious inducement of relaxation. This could be compared to a classical conditioning effect with the experimental situation acting as the conditioned stimulus. In favour of this latter argument it may be noted that throughout the treatment sessions, subjects in the control group exhibited poor voluntary control over the technique i.e. assuming the validity of the widely established inverted correlation between SR level and relaxation (or physiological arousal, see Section 6.2), skin resistance trends may be regarded as indicative of behavioural relaxation and physiological arousal. The following trends all tend to suggest poor voluntary control of the muscular relaxation technique by the control group : (it should be noted that these trends were not statistically significant).

1. In the first treatment session, SR continued to rise at approximately the same rate (as indicated by the slope of the line) after the cessation of the relaxation phase (phase 2(i) and 2(ii)).

2./...

2. In the second treatment session SR decreased during relaxation. This effect may not be attributed to poor mastery of the technique in view of the slight SR increases apparent in the first treatment session where SR rose at a constant rate throughout the session. It is noteworthy that subjects in the control group reported having felt bored during this session which was a repetition of the previous one and the fourth session attended. The technique of muscular relaxation thus seemed to prove ineffective in removing restlessness associated with feelings of boredom during this session.

3. In the third treatment session evidence of inadequate voluntary control of the muscular relaxation technique can be inferred from the drop in SR which occurred during phase 2(ii), after the initial SR increase; further, it may be observed that after the relaxation phase, in the third phase of that session, SR started rising sharply once more.

Voluntary control of a certain technique is here considered to involve successful inducement, maintenance and termination of the physiological effects associated with it whenever it be required. In terms of SR, the subjects showed deficiencies in all three of these criteria during the treatment sessions. In terms of termination of the technique, subjects reported that they found it difficult to re-instate muscular contraction in their bodies after they had achieved a relaxed state. (The term relaxation was frequently used by subjects as synonomous with successful application of the technique). It should be noted that no physical exercise was performed by the control group during treatment sessions, i.e. they were required

to/...

to think of each muscle group alternatively and to loosen up any contraction they detected in themselves (they had learnt to detect muscular contraction during the training session). It is thus possible to postulate that in the post-treatment baseline, muscular relaxation may have occurred as a classically conditioned response to the experimental situation (buzzer, or other environmental variables), without conscious inducement of the state. It is noteworthy that Connor (1974) suggested from a review of several other studies and his own experiment, that the actual muscular exercises involved in progressive relaxation techniques were all but superfluous to the inducement of relaxation: he presented evidence of the relaxation instructions playing a more important role than the exercises; the viewing of relaxation as a response to stimuli (e.g. instructions) was suggested in place of it being seen as a "state". This view would favour the suggestion that muscular relaxation be more useful as a temporary response to arousing stimulus than as a long sustained state of low physiological arousal.

6.2.3. Comparison of deep breathing and muscular relaxation effects upon skin resistance changes

Skin resistance data was regarded as the most important single physiological parameter in evaluating treatment effects in the present study (Section 6.2). Figure 12 represents the three treatment sessions and the two baselines alongside one another. From the SR data it was concluded that treatment effects could be separated in terms of net SR increase during the second phase of treatment sessions 2 and 3, and in terms of consistency with which SR increases were

observed/..

observed during the second phase of the treatment sessions (including to what degree these SR increases were specific to treatment effects). In terms of net SR increase during the second phase of the treatment sessions, the technique of deep breathing seemed to have been more effective (significantly higher levels of SR were found at the end of deep breathing in treatment sessions 2 and 3; no significant differences in SR were noted in the control group during treatment sessions). In terms of the consistency with which SR increases were induced and the degree to which these SR changes may be regarded as specific to treatment effects, deep breathing again proved to be a superior technique. In figure 12, it may be noted that SR changes during deep breathing showed a trend which could possibly have been due to practice effects, with the first treatment session being ineffective in increasing SR, the second treatment session having significantly increased SR and in the third treatment session, the SR changes were induced more rapidly (as indicated by level of SR reached in phase 2(i)) and to a greater extent. Further SR increases occurred only during the second phase of the two last treatment sessions and not during the third phase when deep breathing was discontinued; thus the effect of this technique upon SR were specific to the treatment. A third point which indicates the high degree of effectiveness of the deep breathing technique upon SR increases (reflecting lowered physiological arousal) is that SR continued to rise throughout the practice of deep breathing in treatment sessions two and three. It was pointed out previously that the SR data indicated that the muscular relaxation technique seemed relatively unreliable in its voluntary application. The variable which could be most important in differentiating the two techniques may have been the successful focussing of attention upon a monotonous

stimulus. Respiratory activity consisted of a single, mechanical, systematic physiological process, and subjects reported no difficulty in remaining aware of it. It became clear from the verbal reports of subjects that being aware of their respiratory activity during deep breathing was found to be very restful and relaxing (due probably to the absence of distracting thoughts which could have caused restlessness). The relaxation group seemed to find it difficult to remain attentive to the relaxation procedure. In conclusion it may be said that deep breathing was both more effective in inducing low arousal levels; and more amenable to voluntary control than was muscular relaxation (in terms of SR increases).

6.3. Discussion of Heart Rate Results

No significant treatment or group differences were found for HR. It was noted by Harris et al (1976) that Heart Rate was often a poor physiological index of relaxation; in a variety of studies, plus their own on the effects of a paced respiration technique upon SR and HR responses to shock, they reported significant results for SR data but not for HR. The reason for the lack of significant results for HR as compared for example to SR, or muscular tension may be postulated to be a result of the sensitive homeostatic mechanisms situated in the carotid sinus and aortic arch (baroreceptors) which swiftly counteract any changes in autonomic balance in the cardiovascular system: changes in skin resistance (due to activity of the sweat glands) are brought about solely by the sympathetic nervous system which innervates the sweat glands (in spite of acetylcholine being the neurochemical transmitter, a characteristic usually associated with parasympathetic nerves - Carmichael et al, 1941;

Darrow, 1943; Goadby and Goadby, 1949). Thus little restraint of SR changes is to be expected from such single innervation; on the other hand, the cardiovascular system is dually innervated, and it may be expected that such a system will show restraint of response whenever a change is induced.

The graphs in Figure 13 which represent the changes in HR in the five sessions, for each group, were constructed so as to make the HR changes in the two groups directly comparable by visual inspection. The same method was used as in the SR graphs, i.e. the dotted line parallel to the X-axis represents HR level during the first phase of the session. This line may thus be referred to as the "initial value" of HR. The three points above and below this dotted line in each graph, indicate the changes in HR (in beats per minute) which occurred in the subsequent phases relative to the initial value (i.e. relative to HR value during the first phase). HR changes in the two parts of the second phase and in the third phase were calculated by subtracting the HR values of these phases from that of the first phase. The change scores thus obtained were represented in Table 15. In the graphs, the points falling above the dotted line (which has a value of 0 bpm) represent a rise in HR, while those below the dotted line indicate a drop in HR; the five sessions were graphically represented on the same set of axes alongside each other. On the extreme left is the pre-treatment baseline; and the other sessions follow in the order in which they were run, with the post-treatment baseline lying on the extreme right hand side of the graph. Figure 13 permits direct comparisons of HR changes in the various sessions by visual inspection.

TABLE 15/...

TABLE 15 : Heart Rate (Beats per Minute) Change Scores during the second and third phases, from initial value in phase 1. (Positive values represent a drop in HR, and negative values a rise in HR).

EXPERIMENTAL GROUP					
	Pre-Treatment Baseline	First Treatment	Second Treatment	Third Treatment	Post-Treatment Baseline
Phase 2 (i)	3.2	-1.8	1	0.6	2.7
Phase 2 (ii)	3.7	-0.6	0.2	1.1	2.5
Phase 3	3.1	1.1	2.4	4.5	3.2
CONTROL GROUP (RELAXATION)					
Phase 2 (i)	1.6	0.3	1.2	1.7	0
Phase 2 (ii)	2.2	1.9	1.9	1.8	0.2
Phase 3	2.6	2.2	2	2.6	0.9

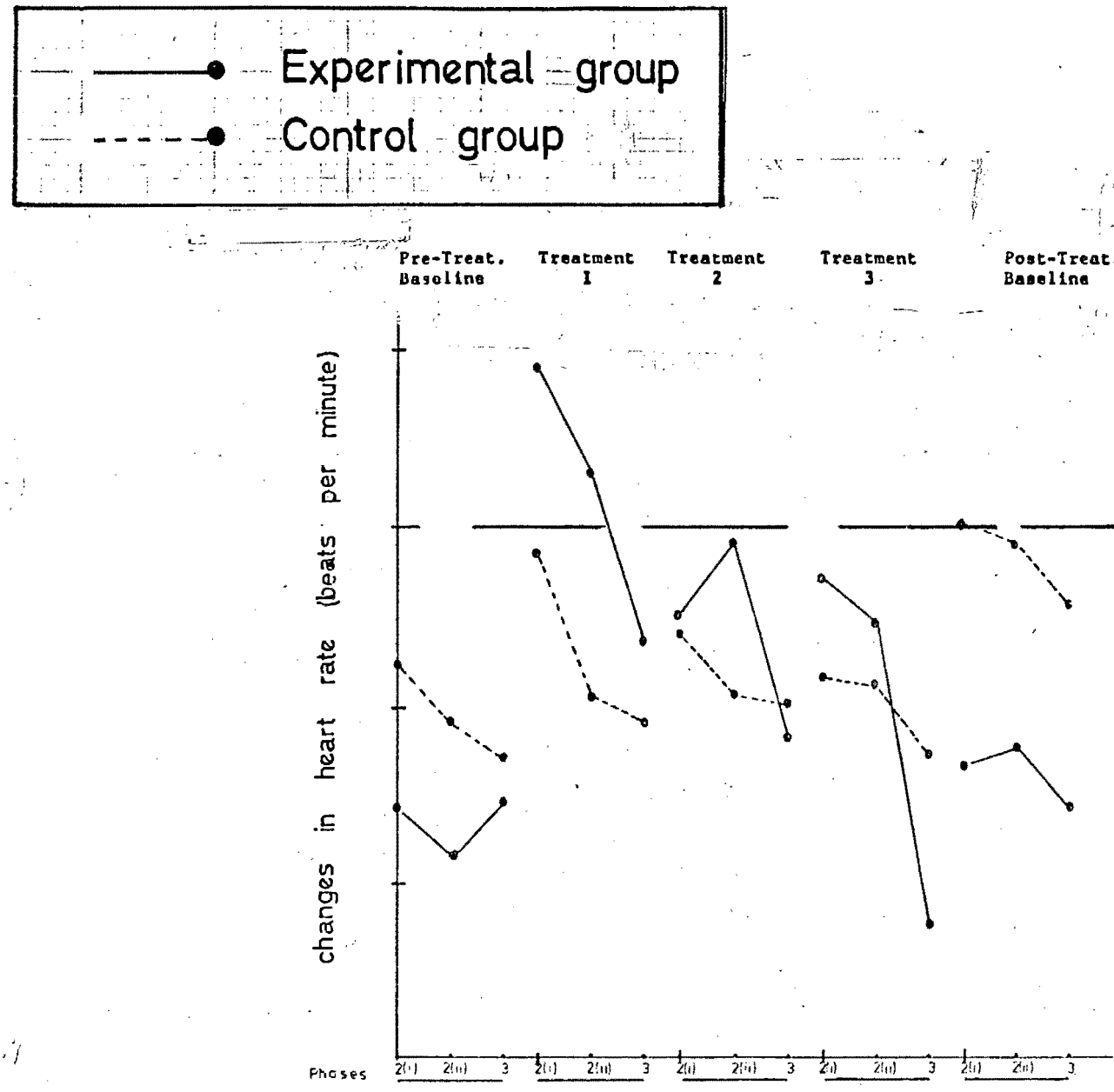

FIGURE 13 : Heart Rate (beats per minute) changes in Experimental Group and Control Group during the five sessions.

NEGATIVE VALUES REPRESENT A RISE IN HR, POSITIVE VALUES A DROP IN HR.

6.4 Discussion of EMG Results

The only EMG data which were found reliable according to the criteria set in Section 4.4.3 was for treatment sessions 2 and 3 of the experimental group which were found to have 37% more masseter EMG decreases than baseline session 1 and treatment session 1 in phase 2(i); 57% more masseter EMG decrease than baseline session 1 and treatment session 1 in phase 2(i). The finding of high physiological arousal in masseter EMG data during treatment session 1 was in accordance with skin resistance and heart rate data for that session which was reported to be uncomfortable by the subjects who probably had not yet mastered the deep breathing technique. Two of the subjects had seated themselves too low on the bean bag with their legs crossed underneath them, and got pins and needles in their legs which caused them additional discomfort. It was found that if treatment session 1 were to be excluded, baseline session 2 would rank second (with baseline session 1 first). For phase 2(ii) there would still be a 48% more masseter EMG decrease in treatment sessions 2 and 3 as compared to baseline sessions 1 and 2.

A few comments regarding certain trends in EMG tension changes are here relevant. Figures 10 and 11 (in the results section, page 98) represent masseter EMG tension change scores for all the sessions alongside one another. From figure 10, it is apparent that the experimental group show the greatest EMG tension decrease in the post-treatment baseline, the second, and the third treatment sessions. Figure 11 indicates that very little corrugator EMG decrease occurred in these three sessions, most especially in the second and third treatment sessions. Deep breathing thus seems to have had different

effects/...

effects upon masseter and corrugator EMG tension. Considering the functions of these two muscle groups, the corrugator muscle would tense up when subjects frown - which may be expected during thought concentration. The masseter group would tense up when subjects clenched their jaws, which is popularly associated with feelings such as anger, frustration and other related emotions. It could be expected that during the practice of deep breathing there would be a certain degree of thought concentration, as subjects were asked to passively concentrate on their breathing. However, the great amount of masseter EMG tension decrease during the second phase of the last two treatment sessions indicated that a certain amount of muscular relaxation could have occurred during deep breathing. The finding of the lack of EMG tension decrease in one group of muscles (here the corrugator) compared to another (masseter) may be related to studies of response specificity which have demonstrated that persons can react to various situations with tension in different muscles, e.g. Goldstein (1972, see review by) reported studies in which discussion of hostility were associated with increased forearm EMG; during a session concerned with sex, leg tension rose. Thus, it may be postulated that the lack of correlation found between masseter and corrugator muscles is due to such symptom specificity. It can be concluded that while the practice of deep breathing in treatment sessions 2 and 3 was accompanied by relatively large decreases in masseter EMG tension, that corrugator EMG tension only decreased slightly.

Another muscular tension trend which is noteworthy is the sudden decrease in corrugator muscle tension in the third (final) phase of baseline 2, baseline 3 and treatment 1 in the experimental

group/...

group (see Figure 11, page 98). It is noteworthy that baseline 3 and treatment 1 were the sessions during which the subjects reported frustration and discomfort respectively. The sudden corrugator EMG decrease in the final phase of a session may have been related to subjects knowing that the end of the session was near and thus feeling some relief.

In the control group, corrugator EMG tension decreased most in the post-treatment baseline (baseline 3) and the second treatment session. These results are partly in accordance with skin resistance data which indicated that low levels of physiological arousal were maintained during baseline 3; however, the skin resistance data indicated increased physiological arousal in treatment session 2; furthermore, subjective reports suggested that irritation had been felt during the session. It could be postulated that the subjects in the control group managed to keep their corrugator muscle tension low during the second treatment session by consciously reducing muscular tension during the session, according to the technique which they were practising, i.e. they were taught to detect muscle tension in each part of the body and to relax the tensed up muscles. Further, they were told to pay special attention to muscles which were most contracted. Looking at the corrugator EMG samples for that session in the control group (see Appendix) it should be noted that 5 out of 6 subjects started off with rank 1 (most tense EMG) in the first phase and proceeded to decrease EMG tension in the other phases. Only 1 subject showed the reverse trend of increased EMG tension during that session.

Although none

of the control group's differences between sets of change scores exceeded 12, there was 28% more corrugator muscle tension decrease in treatment sessions 3 and 2 compared to baseline sessions 1 and 2 during phase 2 (ii).

6.5 The effects of deep breathing upon physiological arousal

In the present research, it was found that deep breathing was accompanied by decreases in physiological arousal, as indicated by increased levels of skin resistance and decreased masseter muscular tension; however, no significant changes were found for HR. Corrugator muscle tension did not increase, but decreased only slightly. Subjective reports unanimously indicated that deep breathing involved "breathing less than usual". In view of these findings, it is suggested that during deep breathing there was a gradual decrease in minute ventilation (amount of air inspired or expired each minute) due perhaps to the link between metabolic rate and ventilation, i.e. metabolic rate is described by Ganong (1971) as the amount of energy consumed per unit of time. In an individual who is not moving and who has not eaten recently, energy output mainly appears as heat (Ganong, 1971), and a low metabolic rate results. During states of low arousal (referring to parasympathetic dominance in the ANS, low muscular tension, respiratory activity and EEG frequency) metabolic rate is low, as little energy is being consumed. On the other hand, in high metabolic states, there will be high levels of physiological arousal (in the form of muscular tension, sympathetic dominant activity in the ANS and fast voltage on the EEG). Consequently, the term low metabolic rate would always imply a state of low physiological arousal, and high metabolic rate, a

state/...

state of high arousal.

In the present experiment, the subject sat still with eyes closed in a quiet, dark, warm environment. He was instructed not to eat for at least one hour prior to the experiment. It may thus be postulated that the experiment was conducive to a low metabolic rate, as little energy was being consumed. However, the presence of anxiety and tension would have caused tensing of the muscles and increased secretion of epinephrine, both of which elevate metabolic rate.

The technique of deep breathing seemed to have effectively prevented the presence of anxiety and tension, or of any arousing thought, during the course of its practice. This aim could have been partly achieved by passive concentration upon respiratory activity which would prevent disturbing thoughts from arising.

The subject's attention during deep breathing was involved in monitoring his respiratory activity; this was a simple task demanding little effort to be exerted, while at the same time requiring continuous attention. This aspect of the deep breathing could be comparable to repeating a 'mantra' (a sound) such as practiced in most forms of meditation (e.g. in Transcendental Meditation). The repetition of a monotonous sound (in the case of deep breathing, the continuous monitoring of breathing activity) has been postulated to have in itself a relaxing effect upon the organism (Beary et al, 1974). This could be attributed to the monitoring task obstructing anxiety-provoking or other distracting thoughts resulting in tension and arousal.

It should be noted that the control group (Section 4.3.7) controlled for the effect of deep breathing being limited solely to the passive concentration involved in its practice, as they were also

required to concentrate on their task of maintaining low muscular tension.

If decreased ventilation is assumed from the finding of lower physiological arousal associated with deep breathing, it is noteworthy to consider what changes have been reported to accompany such low ventilation. Taking the extreme case of hypoxia, there would be general sympathetic effects and increased muscular tonus (Gellhorn, 1967). However, if the ventilation decrease occurred gradually it may be expected that no hypoxia occurred (except during treatment session 1, where there was increased respiratory rate in the third phase: this may have been due to lack of oxygen, or excess carbon dioxide during deep breathing, which had not been properly mastered). It should be noted that deep breathing involved an increase in tidal volume (amount of air moving in with each inspiration, or out with each expiration). Comroe (1974) stated that increased tidal volume resulting in greater lung inflation, increased the number of inhibitory nerve messages from the lung to the respiratory centres: this reflex reaction has the function of preventing overbreathing by overflation of the lung. During deep breathing increased tidal volume was made necessary in that the whole lung had to be used; this increased tidal volume however, could not result in too much lung inflation because of reflex adjustments arising from the inhibitory nerve messages from lung to the respiratory centres. This would explain why deep breathing was a relatively easy technique to master, as hyperventilation was reflexly prevented without conscious interference by the subject: during deep breathing the increased tidal volume involved in the use of the whole lung was taken very slowly, thus preventing sudden lung inflation and over-

from occurring.

A further reason for supposing that deep breathing would result in reduced ventilation, was that accumulation of carbon dioxide (CO_2) was prevented by regulating a full expiration, whereby the whole lung was emptied. CO_2 receptors (e.g. in the carotid and aortic bodies and in the mid-brain) are extremely sensitive to CO_2 concentration; a slight increase in CO_2 is enough to induce stimulation of respiratory activity. On the other hand, O_2 receptors are relatively insensitive, e.g. they only respond when O_2 concentration in inspired air is decreased to 8% instead of 20,05% (Comroe, 1974). During deep breathing, quite a significant drop in O_2 inhalation (e.g. by slow intake of air) would not result in stimulation of respiration, as there was no CO_2 accumulation. Besides, such a drop in O_2 inhalation would occur naturally as a consequence of the reduced metabolic needs of the body during deep breathing, such as pointed out earlier in this Section.

Considering the means by which respiratory activity may influence the other major systems, only indirect experimental evidence may be brought up, as the relationships between the major physiological systems remain to be fully explored; however, it has been recognised that such connections do exist, e.g. Ganong (1971) suggested connections between the inspiratory-expiratory respiratory centres (in the pons and medulla) and the neocortex, as although inspiration and expiration were not usually conscious events, they were both under voluntary control. Ganong (1971) noted that pain and emotional stimuli affected respiration, thus suggesting afferents from the limbic system and hypothalamus. Further, movements of the joints stimulated the respiratory centres, presumably through impulses in afferent pathways from proprioceptors

in/...

in muscles, tendons and joints (Ganong, 1971).

In a more recent and comprehensive textbook concerning the respiratory system, Comroe (1974) referred to the location of the respiratory centres in the medulla and pons as strategic for interactions with other systems, because of the Reticular Formation (RF) consisting of many groups of richly interconnecting neurons, is also situated there. Besides, the medullary respiratory centre, responsible for rhythmic breathing, has connections with the respiratory centres in the pons, with areas of the hypothalamus and cortex, and with the Reticular Activating cortex (Comroe, 1974).

With consideration for the above evidence, it may be expected that the respiratory system has had effects upon the other major physiological systems via one or all of the following structures :

1. The sino-aortic chemoreceptors.
2. The central chemoreceptors in the mid-brain which lie close to the Reticular Formation.
3. Via the respiratory muscles and gamma motoneurons.

Each of these possibilities were discussed separately.

6.5.1 The sino-aortic chemoreceptors as mediators of respiratory effects upon the other major physiological systems

The chemoreceptors situated in the carotid and aortic bodies have both afferent and efferent connections with the ANS (Comroe, 1974); when the chemoreceptors are stimulated by inadequate ventilation, the following physiological functions are activated: breathing rate, blood pressure, heart rate, secretion of the adrenal medulla and cortex, and that of the posterior pituitary gland; further there is

increased/...

increased activity of the cerebral cortex (the chemoreceptors have connections with the cerebral cortex via the 9th and 10th cranial nerves, Comroe, 1974). Gellhorn (1967) reported experimental evidence (translated from German) indicating that the chemoreceptors of the sino-aortic area had a direct effect upon the gamma motoneurons, thus indirectly provoking changes in muscular tone with changes in respiratory activity. e.g. Hypercapnia (induced by inhalation of gas mixtures rich in CO_2) and asphyxia were reported to produce generalised sympathetic discharges and increased tone of skeletal and abdominal muscles. The above evidence indicates that respiratory activity could have effects upon both ANS and skeletal muscles via the peripheral chemoreceptors. Under good conditions of oxygenation, as was found in deep breathing, it may be expected that the opposite effects to hypercapnia and asphyxia would be observed, with parasympathetic effects and loss of muscular tone being mediated by the sino-aortic chemoreceptors.

6.5.2 The respiratory centres in the medulla : effects upon Reticular Activity.

It was pointed out earlier that the location of the respiratory centres in the midbrain (medulla and pons) was considered a strategic one for interaction with other systems, as the Reticular Formation, consisting of many groups of richly interconnecting neurons is also there. Under drastically altered respiratory conditions (e.g. hypercapnia, asphyxia) widespread arousal response are induced which were reported to involve the brain stem as a whole (Gellhorn, 1967). It may be postulated that overarousal of the respiratory centres generalize to the whole Reticular Formation beside which they are

situated;/...

situated; high intensity stimulation of Reticular Formation has been reported to activate the posterior hypothalamus which in turn elicits widespread sympathetic discharges, increased muscular tone and shivering, and also involves cortical desynchronization (Gellhorn, 1964, 1967). On the other hand, it may be considered that during deep breathing involving slow rhythmic discharges in the respiratory centres, there would be low intensity stimulation of the Reticular Formation; Gellhorn (1964, 1967) has given evidence of such low intensity stimulation of Reticular Formation leading to activation of the anterior hypothalamus associated with parasympathetic discharges and loss of muscle tonus. It is, therefore, not unlikely that deep breathing could have partly influenced the other major systems via the Reticular Formation.

6.5.3 The respiratory muscles and the gamma motoneurons : effects upon physiological arousal

The connections between the skeletal muscle system and the autonomic nervous system was by far best documented by Gellhorn (1958 a and b, 1964, 1967). These two major systems were found to be connected via the gamma motoneurons, e.g. if during and following a rise in the pressure of the isolated carotid sinuses (normally leading to increased parasympathetic activity) the activity of a single gamma fibre is recorded in the anterior root of the lumbar cord, there is a brief increase in gamma activity, followed by a complete inhibition outlasting stimulation period by several minutes. A similar effect was reported when the afferent muscle spindle discharges were recorded, Further, it was noted that the inhibitory

action/...

action but not the temporary phase of excitation was absent after denervation of the carotid sinus, indicating that the diminuition in muscle tone associated with increased pressure in the carotid sinus is due to a lessened activity of the gamma system. Further, Gellhorn developed a detailed thesis concerning the means by which cues from the muscles may alter brain excitability, e.g. Gellhorn (1964) described his animal experiments, where proprioceptive impulses were eliminated through drugs such as Intocostrin or Anectine; the administration of these drugs were followed by EEG synchrony, as in sleep, hypothalamic potentials undergoing similar changes. Flaxedil, when administered intra-arterially did not alter the EEG, but did only when injected intramuscularly. Gellhorn (1964) therefore, points out that the effect only arises when the neuro-muscular junctions are blocked, thus eliminating the possibility of a direct effect on the brain. By eliminating proprioceptive impulses, he demonstrated a shift in cerebral balance towards relaxation, a tendency to sleep and lessened responsiveness to emotional excitation and paid, the latter two indicating lessened sympathetic activity.

Thus drugs (curare) which reduce proprioceptive cues from the muscles, alter hypothalamic activity: when there is decreased pro-prioceptive feedback, Gellhorn (1958 a and b) shows that the excita-bility of the posterior hypothalamus, responsible for sympathetic activity, is lowered. He suggests this to account for the diminuition in emotional reactivity associated with skeletal muscular relaxation. Gellhorn (1958 c) suggests a model for viewing emotion: Tonic and phasic proprioceptive discharges act on the hypothalamus and reticular formation, changing the balance and reactivity of the ergotropic and

trophotropic /...

trophotropic systems. This he points out affects awareness, mood and emotional state.

It was considered that deep breathing would prevent muscular contraction (or tension) in all the respiratory muscles (ranging from the lower abdominal area to the upper thoracic region). Deep breathing consisted of slow, long inspirations during which there was slight contraction of the respiratory muscles, this was followed by complete relaxation of these muscles during expiration (which is a passive process effected by relaxing the respiratory muscles). This continuous process of contraction-relaxation could be said to be comparable to the technique of progressive relaxation. It is uncertain, however, whether such relaxation would generalize to other than respiratory muscles : Shedivy and Kleinman (1977) found no concomitant relaxation in other somatic muscles with decreases in frontalis EMG tension achieved by biofeedback. In the present study, masseter EMG tension was found to have decreased by about 50% more during deep breathing in treatment sessions 2 and 3 compared with baseline sessions 1 and 2 and treatment session 1. However, corrugator muscle tension decreased less during deep breathing than in the other sessions. It may be concluded that during deep breathing at least some muscle groups could have had greater decreases in EMG tension than under baseline conditions. It is noteworthy that even if the greater decreases in EMG tension were specific to some muscles only, that may have lowered metabolic rate to quite a significant degree, as in the present experimental setting muscular contraction may have been the chief source of energy consumption considering the effect that concentration upon respiratory activity would have had on anxiety-provoking or otherwise arousing thoughts.

Although the lack of established data in physiology makes it

impossible/...

impossible to state how changes in respiratory activity may have effected widespread changes in the other major physiological systems, there was some evidence indicating that functional connections between the major physiological systems occurred via the chemoreceptors of the sino-aortic bodies, the midbrain Reticular Formation where the respiratory centres are located, and the gamma mononeurons stimulated by proprioceptive cues from the respiratory muscles. All of these mechanisms or only one or two of them may possibly have been responsible for the significant changes in skin resistance and masseter muscle tension indicative of lowered arousal in at least some important physiological systems, which was observed during the present experiment.

6.6. Conclusion

The deep breathing technique used in the present research consisted of an easily learnt technique, requiring no special equipment for learning or practice. It proved superior to muscular relaxation in its effects of lowering physiological arousal as indicated by SR rises and masseter EMG decreases. This was so especially with reference to the degree of voluntary control which the groups were found to have over the respective techniques: e.g. deep breathing was associated with immediate and continuous rise in SR throughout its practice; these trends were slightly reversed upon cessation of deep breathing. It should be noted that were the actual muscular exercises of contraction-relaxation performed by the

control/...

control group during the training session, voluntary control of the technique may well have improved. These exercises were not performed as they would have caused interference over the physiological recordings, and may well have required a longer treatment session. No conclusion may thus be reached as to the differences between the two techniques if it were possible for subjects to go through the whole progressive muscular relaxation exercises. However, the subject could discriminate between a tensed-up and relaxed muscle after the training session and judging from the high rises in skin resistance in the first part of relaxation during treatment session 3, the subjects were perfectly able to induce relaxation. The difficulty seemed to lie in maintaining the relaxation; subjects reported that their minds tended to wander after a while and that they could not passively concentrate on their muscles throughout the required period. It may be assumed that one advantage deep breathing had over muscular relaxation was the ease with which subjects could keep their minds upon their breathing: this task was reported to be pleasant and relaxing.

6.7 Pointers to future research

The present research was a pioneering study of the physiological effects of deep breathing. It would be most valuable for future research to establish the exact nature of the respiratory changes induced during deep breathing, as this would allow more certain speculation concerning the means by which the physiological changes associated with the technique occurred.

Further the present research has only recorded EMG tension changes in two muscle groups; masseter muscle tension was found to

decrease/...

decrease to relatively great degree, while corrugator muscle tension was not reliably affected. It would be of great interest to obtain EMG recordings from several other muscles, so as to assess the importance of muscular relaxation effects during deep breathing.

The present research could not use psychopathological groups as subjects, as the effects of deep breathing were as yet unexplored. However, the results have indicated that the technique is reliably associated with lowering of physiological arousal. It would be of great interest to investigate the effects of deep breathing in psychopathological groups where underarousal is found. It could be discovered that deep breathing would increase physiological arousal level in such a group. This effect would be most likely to be found in the case where very low HR is found due to respiratory effects upon HR (e.g. during exercise, breathing rate and HR increase; during sleep, both functions generally are found to decrease).

In this research, elaborate exercises were used in teaching the deep breathing technique: it would be of interest to compare this present method with one where simple instructions are given (e.g. "reduce your respiratory rate", or "breathe slowly and deeply"). The exercises used in this research were found useful in a pilot study, as a means of effectively preventing over and under-breathing in a short period of time. It is not impossible though that simple instructions would induce the same changes as were found using the present technique, even if more practise time is required by subjects. Such an extra control group could not be run in the present experiment, due mainly to difficulties encountered in subject recruitment.

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APPENDICES

7. The EEG Power Spectrum Analyser

It was pointed out in Section 4.2.7 that initially, the EEG Power Spectrum Analyser accepted too restricted an amplitude range for the full EEG signal, resulting in the 1 - 7 Hz and 13 - 40 Hz bands not being successfully analysed. Due to this fault, to the delay necessitated by the modifications to the EEG Power Spectrum Analyser and to the shortage of magnetic tape reel on which to store the data, over half of the EEG data was either lost completely or inadequately analysed. Thus, it became impossible to use the EEG data. (See Section 4.2.7).

However, after appropriate modifications to the EEG Power Spectrum Analyser had been completed, the experimenter became impressed by the close correspondence between the analyser's percentage results for the various frequency bands (1 - 7 Hz, 7 - 10 Hz, 10 - 13 Hz, 13 - 40 Hz) and visual inspection of the EEG tracing; the EEG Power Spectrum Analyser had the additional advantages of being relatively inexpensive and time-saving. Thus, it was considered useful to include a full description of the Analyser's design and mode of operation, as well as a few samples of the EEG tracings accompanied by the results obtained by the EEG Power Spectrum Analyser.

7.1 Design of EEG Power Spectrum Analyser

The device monitors the Power Spectrum of an Electroencephalograph Signal, and delivers four dc output signals proportional to the energy in the following EEG frequency bands :

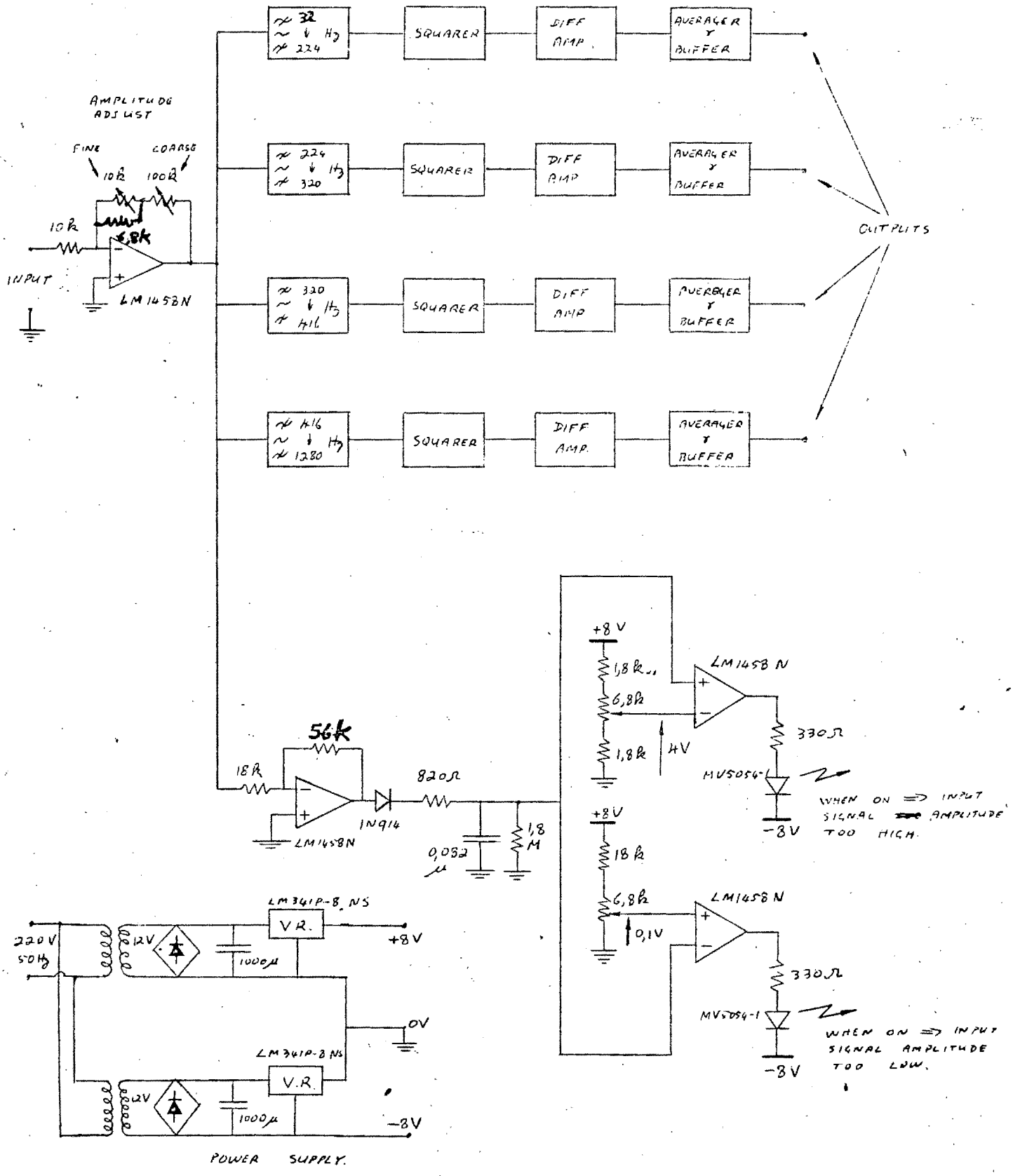
(i) 1 Hz - 7 Hz

(ii) 7 Hz - 10 Hz

(iii)/...

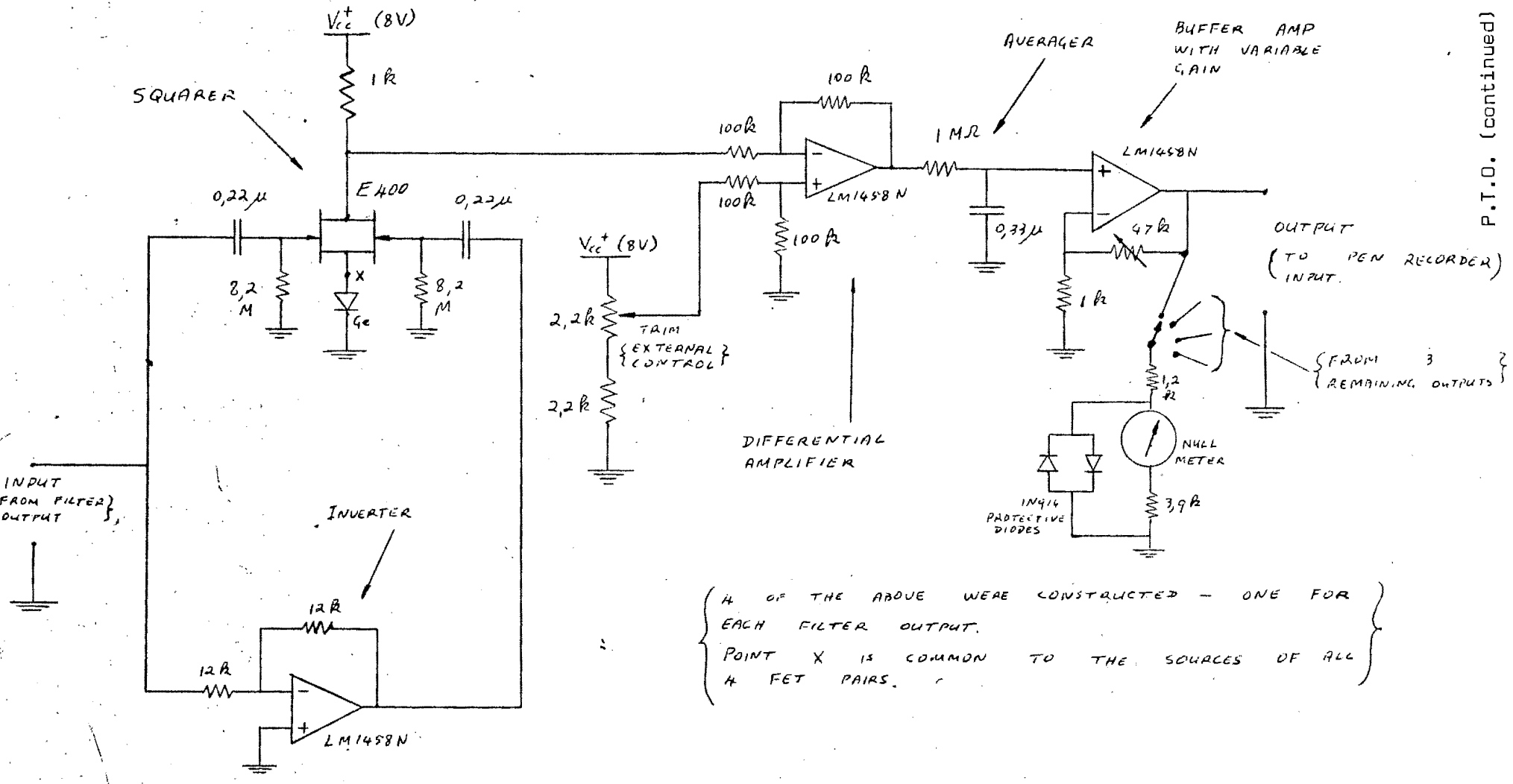
FIGURE 14

COMPLETE CIRCUIT OF EEG POWER SPECTRUM ANALYSER.



P.T.O. (continued)

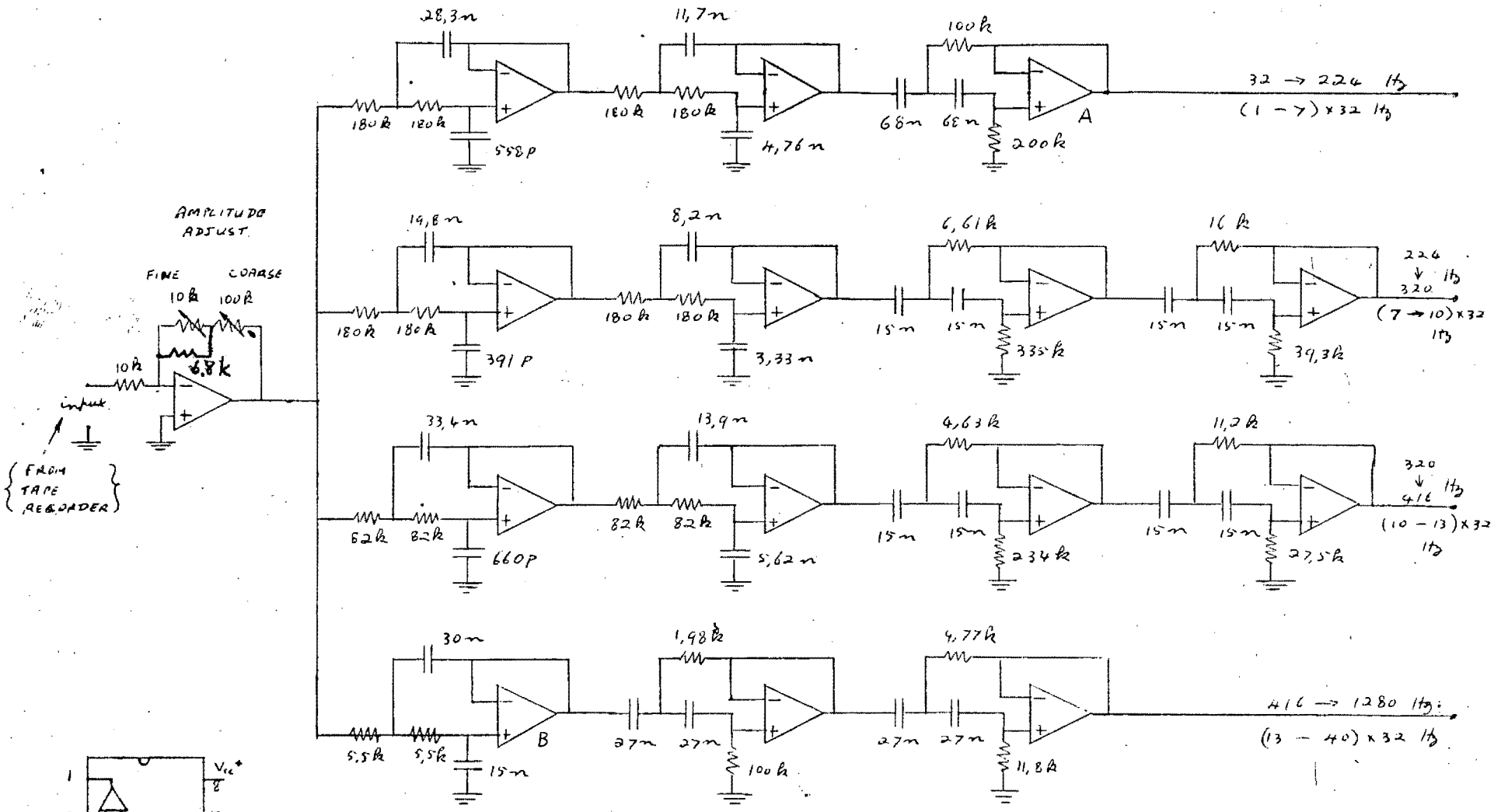
CIRCUITRY FOR POWER SPECTRUM ANALYSIS OF SIGNAL



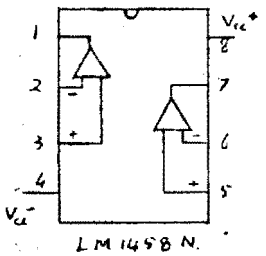
{4 OF THE ABOVE WERE CONSTRUCTED — ONE FOR EACH FILTER OUTPUT.
POINT X IS COMMON TO THE SOURCES OF ALL 4 FET PAIRS.}

P.T.O. (continued)

FILTERS



EACH OUTPUT CONNECTED TO ITS RESPECTIVE SQUARER.



ALL OP-AMPS TYPE LM1458.

{ EXCEPT FOR A & B, ALL FILTERS DESIGNED FOR 80 dB/DECADE ROLL OFF PER SLOPE, WITH 1 dB PASS BAND RIPPLE (CHEBYSHEV). A & B EACH HAVE $\alpha = \sqrt{2}$ — IE FOR SMOOTHEST PASS BAND RESPONSE. }

(iii) 10 Hz - 13 Hz

(iv) 13 Hz - 40 Hz

Note : The EEG signal must first be recorded on any FM tape recorder and then played back through the EEG Power Spectrum Analyser at 32 times the recording speed. The filter cut-off frequencies are therefore 32 times higher than those mentioned above.

Specifications :

(i) Input Resistance ..... 10 k

(ii) Output Resistance 1

(iii) Output Short Circuit Duration ..... Indefinite

(iv) Meter Scale ..... 10 mV/ graduation

(v) Filter Frequency Bands :

32 Hz - 224 Hz

224 Hz - 320 Hz

320 Hz - 416 Hz

416 Hz - 1280 Hz

7.2 Operating Instructions :

(i) Allow a couple of minutes warm-up time after switch-on, to allow certain circuit elements to stabilise thermally.

(ii) In the absence of input signal, interrogate each output with the "Output Select" Switch, and adjust the "Trim" potentiometer of the corresponding output to register a nul on the meter.

(iii) The amplitude of the input signal should neither be too high nor too low. Set the Amplitude Control Potentiometers so that

neither/...

neither of the two indicator lights are on. Preferably somewhere near the mid-point of the two dial extremeties.

Note : It in unimportant if either of the two lights flashes on occasionally, as long as the bulk of the signal falls in the stipulated range. In the event of both lights flashing regularly however, adjust the Amplitude Controls so that the "low" indicator flashes more often than the "high".

If the signal goes much beyond the "high" limit, the device saturates. However, the occasional spike can be tolerated, since there is not much energy in it compared to the bulk of the signal.

If the signal drops below the "low" limit, it starts becoming comparable with the voltage offsets. This is unimportant provided the outputs are accurately nulled. However, it is better to have the signal large enough to swamp out these small offset voltages.

Using an Ampex FM Tape Recorder Model RP.500, the EEG Signal was recorded at $1^{7}/_{8}$ I.P.S. (Inches per Second) and reproduced at 30 I.P.S. through the Analyser, i.e. 32 times faster. The leads from the Analyser's four frequency band outputs were plugged into the input socket on the Dynograph (on the Power Amplifier) and the toggle switch placed in the Hi-In position. A photograph of the EEG Analyser connected up to the Dynograph and Ampex Tape Recorder was included so as to illustrate the above instructions.

One great advantage in the EEG analyser, was that it was designed so as to analyse the EEG data 32 times faster than the recording speed. Thus 30 minutes recording was analysed in less than one minute.

Another advantage of the EEG Power Spectrum Analyser is that it was able to read the EEG signal from the FM recorder, in analog form: this eliminated the need for digital transformers in data quantification.

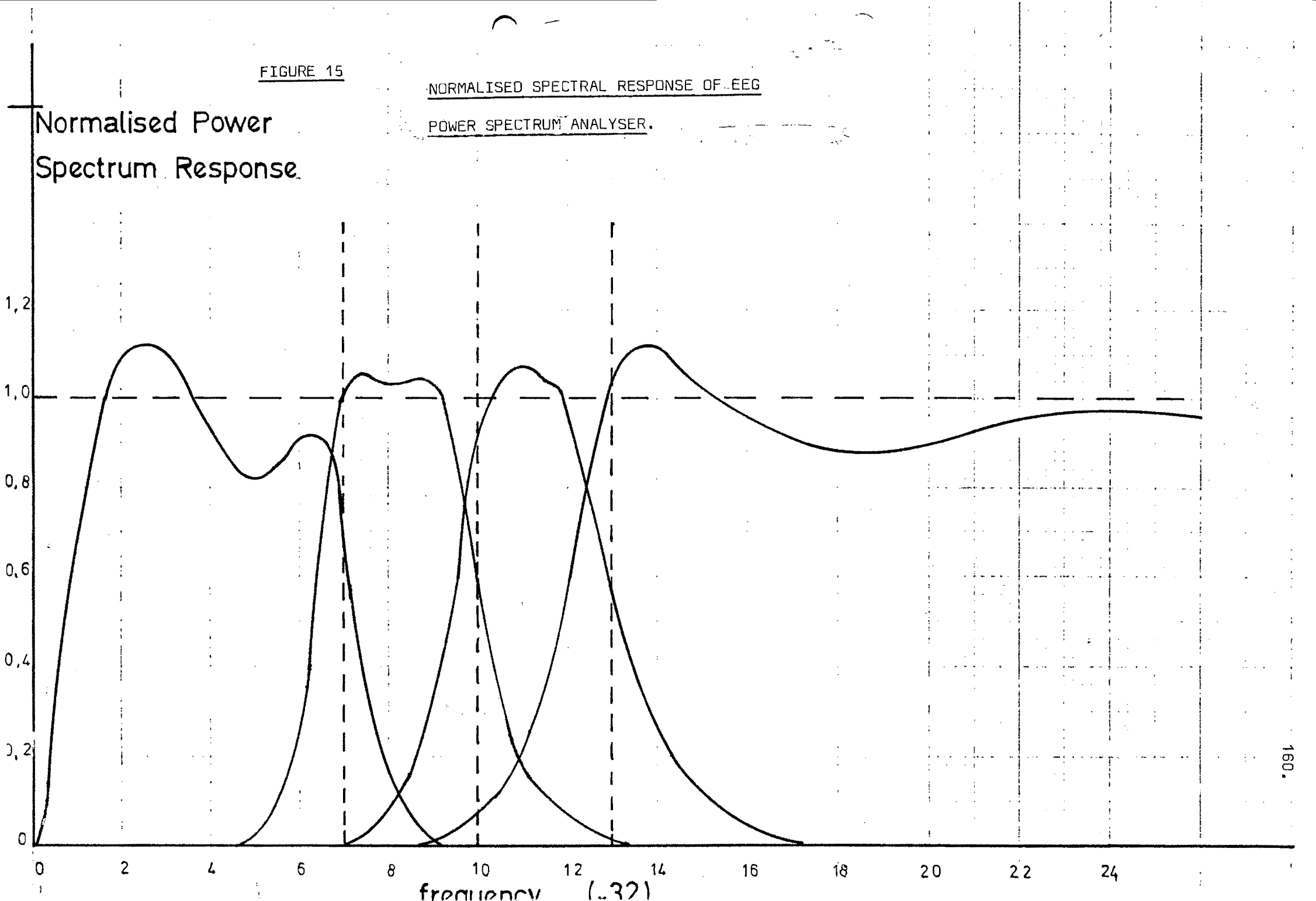
Initially/..

Initially, the EEG data was to be analysed by a system of Band-Pass filters, fed into a set of frequency sensitive switches. The output was given in digital form by electro-mechanical counters. This apparatus took a long time to be perfected and eventually was discarded as a means of analysis. The main problem lay with the relatively long latency inherent in a mechanical system such as in digital counters, compared with the speed at which the EEG signal varies, e.g. if two waves within the same frequency bands (e.g. 7 - 10 Hz) were to follow each other at extremely short intervals, or even, as is frequently the case in the 13 - 40 Hz range, be slightly superimposed

upon/...

FIGURE 15

NORMALISED SPECTRAL RESPONSE OF EEG POWER SPECTRUM ANALYSER.



upon each other, the digital counter could only count one of the waves.

7.3 Validation of the EEG Spectrum Analyser

The Analyser was validated and the error margin quantified by calculating the shape of the curves of the normalized spectral responses of the EEG Analyser were using a sine-wave frequency generator and a dc voltmeter (See Figure 15). The procedure for validating the output signal given by the EEG Power Spectrum Analyser is as follows :

1. With the sine-wave frequency generator, feed a signal of about 1 volt (rms) into the analyser.
2. Amplitude controls potentiometers are used to adjust the input signal so that it is neither too high nor too low - this ensures homogeneity in input signal amplitude.
3. Feed the outputs of the 4 channels in turn to a dc voltmeter set on about 3 volt scale (any scale between 2 - 10 would do as long as it is ensured that the meter does not go over-range).
4. The first channel (1 - 7 Hz) x 32 will peak at about 2.8 (x 32) Hz, i.e. 90 Hz.
5. The second channel will peak between (7 x 32) Hz and (9 x 32) Hz (224 Hz and 288 Hz).
6. The third channel will peak between (10 x 32) Hz and (12 x 32 Hz) (320 Hz and 380 Hz)
7. The fourth channel will peak at about (13.5 x 32) i.e. 430 Hz.

Put in non-technical terms, the above method of validation consists of feeding into the Analyser a signal of known frequency, and checking

that/...

that the appropriate frequency channel registeres the signal. The normalised spectral response curves obtained show that when the above validation test is performed, each frequency band will register the appropriate frequency signal : if the frequency band 7 - 10 Hz is taken as an example, all 8 Hz signals will be registered in that band; thus the 'peak' response will be between 8 and 9. At 7 Hz, some of the signal on the lower side of 7 Hz will be registered in the 1 - 7 Hz band; some of the 7 Hz signal on the higher side of 7 Hz in the 7 - 10 band.

Thus the normalised spectral response to a 7 Hz signal will be lower in the 1 - 7 Hz and 7 - 10 Hz bands, than the 8 Hz signal in the 7 - 10 Hz band.

It was necessary to express the cutoff frequency bands as 'overlapping' as whole numbers were used. It may be considered, that were it possible to express the 'overlap' frequencies in fractional form, 1 - 7 Hz would end on the first half of the 7 Hz frequency and 7 - 10 Hz would register the second half of the 7 Hz frequency. It should be noted that the design of the Analyser reduced the 'overlap' error inherent in such fractionation of the intermediate frequencies, in the following ways :

1. The frequency bands were multiplied by 32.
2. Further these frequency bands were squared by the analyser.

Taking 1 Hz as an example, the Analyser read 1 Hz as 32^2, i.e. 1 024 Hz. It becomes evident that the resulting overlap frequencies were extremely reduced by the squarer and the multiplication of the frequency bands by 32.

7.4/...

7.4 EEG Data Scoring Procedure

An example was used to demonstrate the means by which the output of the EEG Spectrum Analyser was quantified : see Figure 16 for an EEG analog dc output tracing, in the form yielded by the Power Spectrum Analyser.

In Figure 16, point A represents one moment in time in the 30 minute recording period. Point A1 represents the 1 - 7 Hz energy band; Point A2, the 7 - 10 Hz energy band; Point A3, the 10 - 13 Hz energy band and Point A4 the 13 - 40 Hz energy band. The dot over each of the 4 points (A1, A2, A3, A4) has been put over the baseline of each of the dc tracings.

Measuring the number of centimeters between baseline and the dc output tracing, the following numbers were obtained :

A1	= 4	mm	:	(1 - 7 Hz)
A2	= 1.75	mm	:	(7 - 10 Hz)
A3	= 1.5	mm	:	(10 - 13 Hz)
A4	= 1	mm	:	(13 - 40 Hz)
TOTAL	8.25	mm		

The total (8.25 mm) represents the energy output between 1 Hz to 40 Hz.

So to find out the percentage of energy in each of the frequency bands, each point is divided by the total energy and multiplied by 100.

e.g. In the 1 - 7 Hz band :

$$\frac{4}{8.25} \times 100 = 48.5\%$$

In/...

In the 7 - 10 Hz - 21.2% energy (low alpha)

In the 10 - 13 Hz - 18.2% energy (high alpha)

In the 13 - 40 Hz - 12.1% energy

Taking point B in figure 16 as a second point during the session, two kinds of information can be derived, concerning EEG changes which occurred at point B, relative to point A.

i.e. 1. The energy distribution in the four bands at point B may be compared to that of Point A.

e.g. Point B had the following percentage energy distribution :

1 - 7 Hz : 57.1%

7 - 10 Hz : 17.9%

10 - 13 Hz : 14.3%

13 - 40 Hz : 10.7%

Thus there was a larger proportion of energy in the 1 - 7 HZ at Point B, and a smaller proportion of the remaining frequencies at point B, relative to point A.

2. The changes in energy in each frequency band, at point B relative to point A may be calculated.

e.g. The following measurements were taken at the two points :

	1 - 7 Hz	7 - 10 Hz	10 - 13 Hz	13 - 40 Hz
Pt A	4 mm	1.75 mm	1.5 mm	1 mm
Pt B	4 mm	1.25 mm	1 mm	0.75 mm
A + B	8 mm	3.0 mm	2.5 mm	1.75 mm

1 - 7 Hz/...

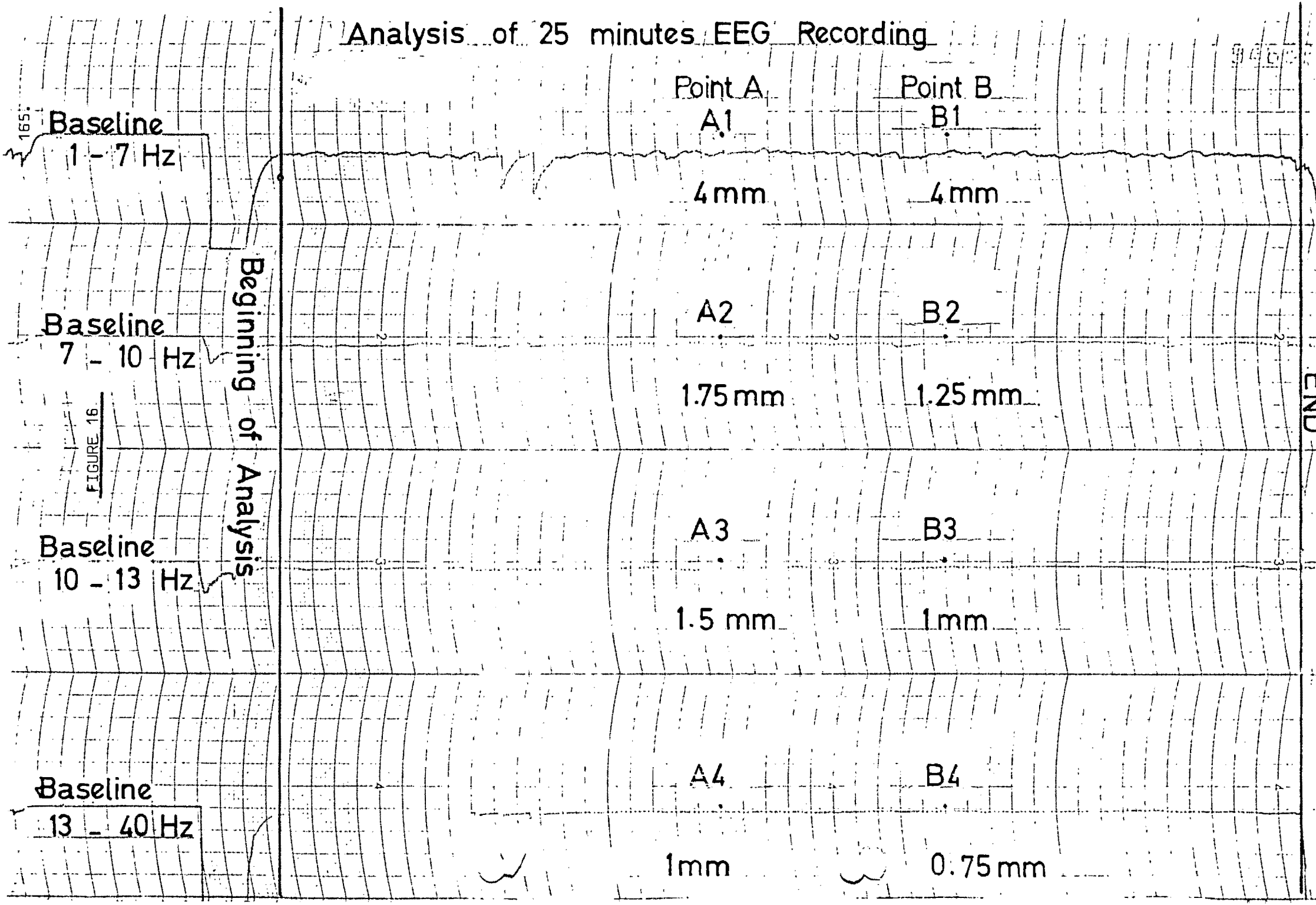

Analysis of 25 minutes EEG Recording
Point A
Point B
A1
B1
4 mm
4 mm
Baseline
1 - 7 Hz
Beginning of Analysis
A2
B2
1.75 mm
1.25 mm
Baseline
7 - 10 Hz
A3
B3
1.5 mm
1 mm
Baseline
10 - 13 Hz
A4
B4
1 mm
0.75 mm
Baseline
13 - 40 Hz

FIGURE 16

1 - 7 Hz : There was no change in energy output in that band at point B relative to point A

7 - 10 Hz : There was 58.3% energy in that band at point A.

$$\frac{A}{A+B} \times 100 = \frac{1.75}{3} \times 100 = 58.3\%$$

At point B there was

$$\frac{1.25}{3} \times 100 = 41.7\% \text{ energy}$$

in the 7 - 10 Hz band

Thus the energy output in the 7 - 10 Hz band decreases at point B relative to Point A :

Point A_2 - B_2 = percentage decrease

58.3% - 41.7% = 16.6%

There was a 16.6% decrease in energy in the 7 - 10 Hz band at point B relative to Point A.

In summary, the EEG Power Spectrum Analyser may be used in two main ways so as to quantitatively express EEG energy changes in the course of an experimental session :

1. Proportion of energy distribution in the frequency bands may be compared over time during the session.
2. Energy output changes in a single band may be analysed over time during a session.

7.5 EEG Power Spectrum Analyser results compared with visual inspection of the EEG tracing

Each of the tables in this section, represent readings obtained

from/...

from the EEG power Spectrum Analyser's tracings (which were included in this section). The Power Spectrum Analyser's readings for each phase of a particular session is given for each frequency band in millimeters (mm) and in percentage form derived according to the method delineated in the previous section. The EEG tracing corresponding to each of the three tables is represented so as to allow visual inspection of the data; this provided a means of assessing the relationship between the results obtained from the EEG Power SPectrum Analyser and the EEG tracing itself. Typical EEG tracings from each frequency band were presented in tables 16 - 18 facilitate interpretation by visual inspection of the EEG signal.

Table 16 represents the EEG Power Spectrum Analyser's results for the third treatment session of a deep breathing subject (experimental group). Most noteworthy in this table, is the sudden rise in energy of the 1 - 7 Hz band during phase 2(ii): simultaneously there is a drop in percentage energy in the other three bands. Thus, there should be found a greater amount of 1 - 7 waves (i.e. high amplitude slow waves) and further, less waves in the alpha and beta bands in phase 2(ii) compared with phase 2(i). The EEG tracing of the second and third minutes of phase 2(i) and the two last minutes of phase 2(ii) are represented in figure 17. It should be noted that there was indeed more alpha spindles and beta activity in phase 2(i) while in phase 2(ii) almost no beta activity is seen, the alpha spindles are rarer, while there is an increase in slow and large theta wave activity.

TABLE 16 : Sample 1 of EEG Power Spectrum Analyser Readings

(See Power Spectrum tracing overleaf)

		ENERGY OUTPUT				
		1-7 Hz	7-10 Hz	10-13 Hz	13-40 Hz	Total in mm
Phase 1	mm	9.25 mm	6.25 mm	7.75 mm	3 mm	26.25 mm
	%	32.24 %	23.81 %	29.52 %	11.43 %	
Phase 2(i)	mm	10.5 mm	8.75 mm	12 mm	2.25 mm	33.5 mm
	%	31.34 %	26.12 %	35.82 %	6.72 %	
Phase 2(ii)	mm	20.5 mm	8.75 mm	9.75 mm	1.8 mm	40.8 mm
	%	50.25 %	21.45 %	23.9 %	4.41 %	
Phase 3	mm	17.75 mm	6.5 mm	11.25 mm	3.25 mm	38.75 mm
	%	45.81 %	16.77 %	29.03 %	8.39 %	

TABLE 17 : Sample 2 of EEG Power Spectrum Analyser Reading

(See Power Spectrum tracing overleaf)

		ENERGY OUTPUT				
		1-7 Hz	7-10 Hz	10-13 Hz	13-40 Hz	Total in mm
Phase 1	mm	1.5 mm	8 mm	12.75 mm	6 mm	41.75 mm
	%	35.93 %	19.16 %	30.54 %	14.37 %	
Phase 2(i)	mm	16.5 mm	13	16.5 mm	9 mm	55 mm
	%	30 %	23.64 %	30 %	16.36 mm	
Phase 2(ii)	mm	8.25 mm	8.25 mm	9 mm	8 mm	33.5 mm
	%	24.63 %	24.63 %	26.87 %	23.88 %	
Phase 3	mm	14 mm	11 mm	14.75 mm	8 mm	47.75 mm
	%	29.32 %	23.04 %	30.89 %	16.75 %	

The above table represents the second treatment session of a subject in the relaxation group (Control Group). Comparing the EEG Power Spectrum results at beginning and the end of the relaxation period (phases 2(i) and 2(ii) respectively) two observations may be made : Firstly, there was a decrease in the energy output of the 1 - 7 Hz band, and a simultaneous increase in the energy output of the 13 - 40 Hz band. This should be represented visually by increased beta activity and decreased slow wave activity, particularly in the 1 - 7 Hz band in phase 2(ii) compared to phase 2(i). Secondly, comparing the total energy output (in millimeters) for the 4 frequency band, phase 2(i) had a total of 55 mm while phase 2(ii)'s total only came up to 33.5 mm.

This/...

This indicates that less energy was present in phase 2(ii) compared with phase 2(i), and should be represented on the EEG tracing as a flattening out of the EEG signal: minutes 2 and 3 of phase 2(i) and the last two minutes of phase 2(ii) are represented in figure 18. It may be noted that there was less slow wave activity, more 13 - 40 Hz activity and a flattening out of the EEG signal in phase 2(ii) compared with phase 2(i), as was indicated in the EEG Power Spectrum Analyser's results.

FIGURE 17

EEG Power Spectrum Analyser Output tracing for readings given in Table 16 (EEG Sample one)

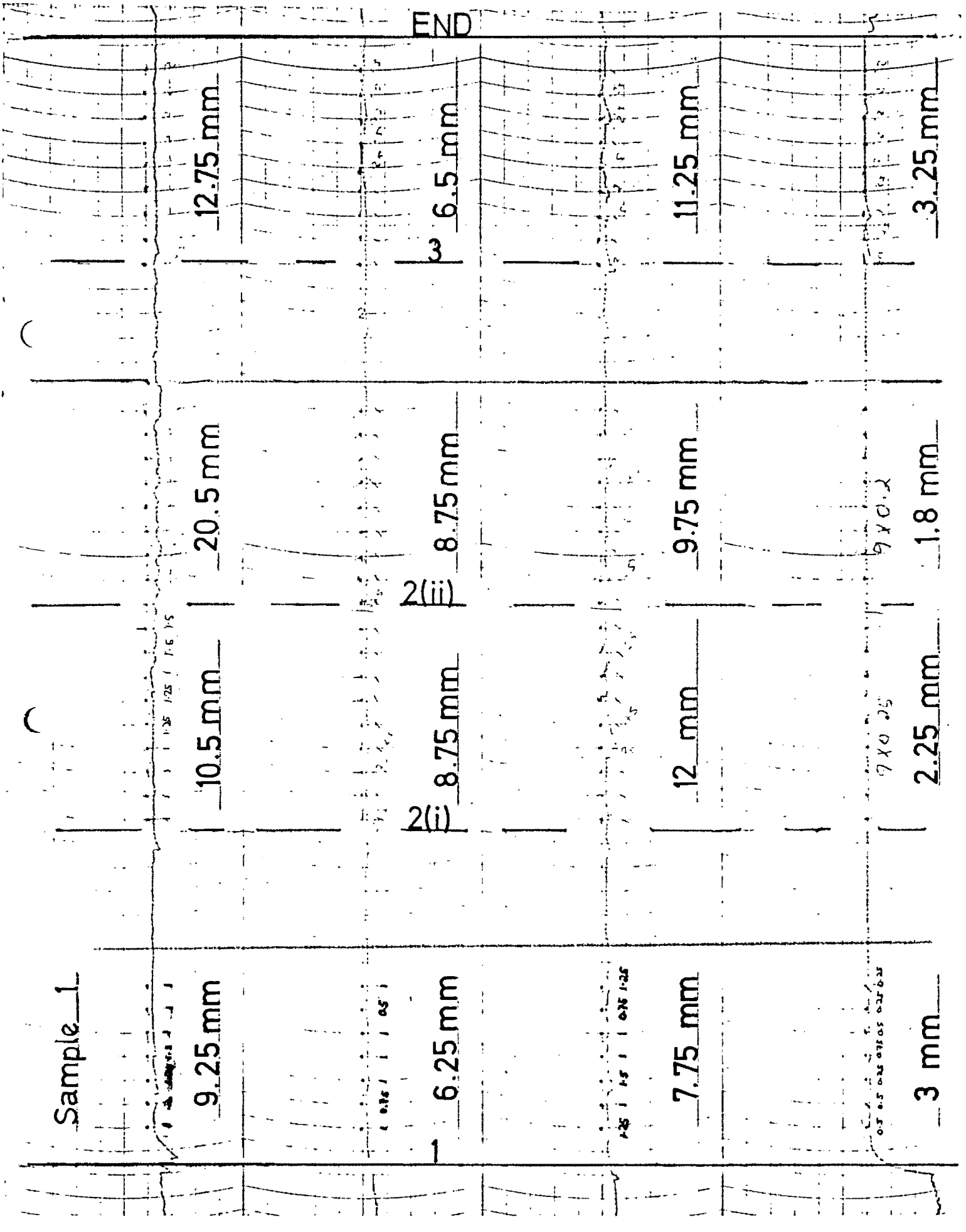


FIGURE 18

EEG Power Spectrum Analyser Output tracing for readings given in Table 17 (EEG Sample two)

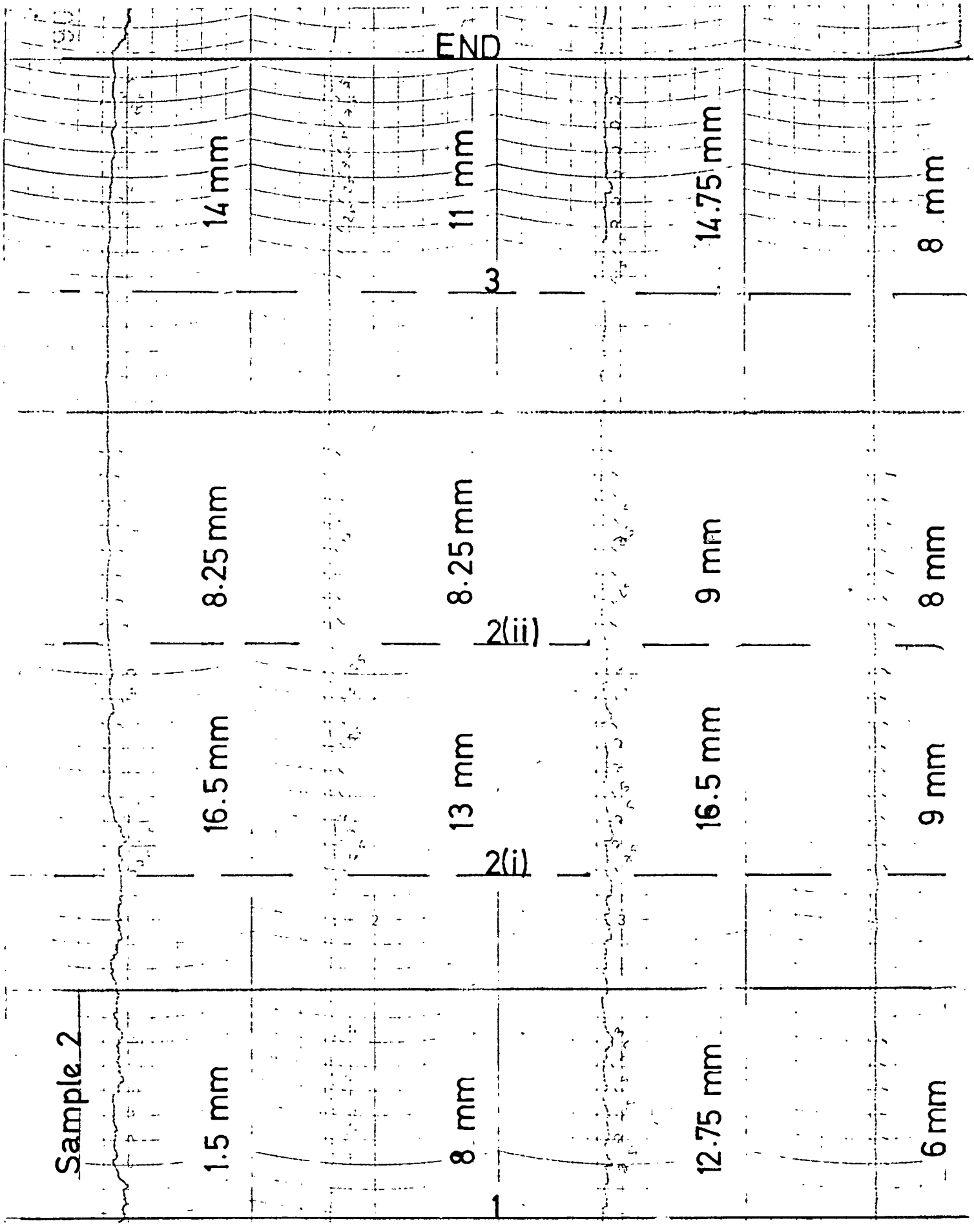


FIGURE 19

EEG Power Spectrum Analyser Output tracing for readings given in Table 18 (EEG Sample three)

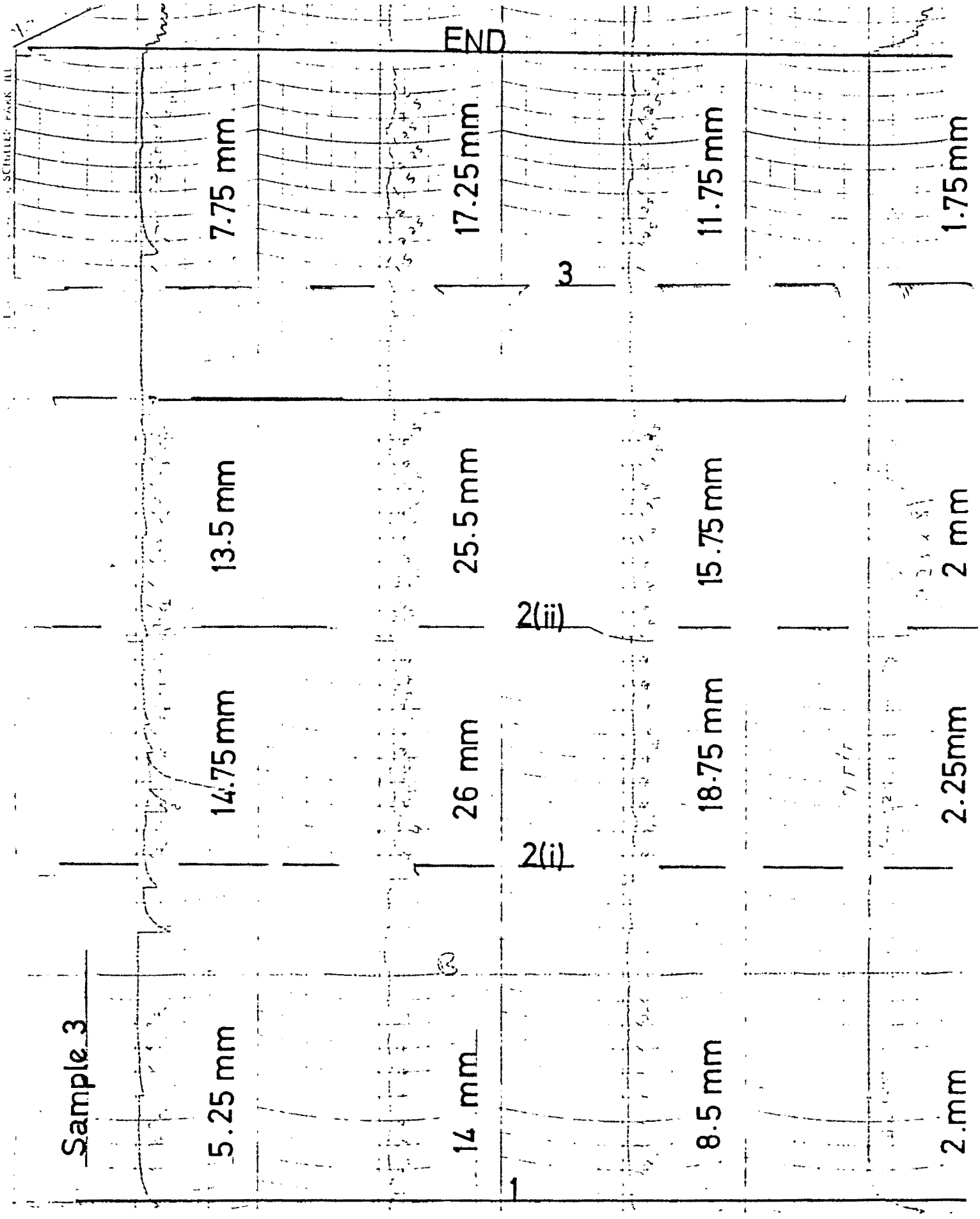


TABLE 18 : Sample 3 of EEG Power Spectrum Analyser Readings

(See Power Spectrum tracing overleaf)

	ENERGY OUTPUT				
	1-7 Hz	7-10 Hz	10-13 Hz	13-40 Hz	Total in mm
Phase 1	mm 5.25 mm	14 mm	8.5 mm	2 mm	29.75 mm
	% 17.65 %	47.06 %	28.57 %	6.72 %	
Phase 2(i)	mm 14.75 mm	26 mm	18.75 mm	2.25 mm	61.75 mm
	% 23.89 %	42.11 %	30.36 %	3.64 %	
Phase 2(ii)	mm 13.5 mm	25.5 mm	15.75 mm	2 mm	56.75 mm
	% 23.79 %	44.93 %	27.75 %	3.52 %	
Phase 3	mm 7.75 mm	17.25 mm	11.75 mm	1.75 mm	38.5 mm
	% 20.13 %	44.81 %	30.52 %	4.55 %	

The above table represents the EEG results from the Power Spectrum Analyser, for the second treatment session of one of the two subjects in the control group. In spite of small fluctuations in the percentage energy distribution, it should be noted that no great changes occurred throughout the session; however, there was a slight shift towards increased energy in the slow wave bands in phase 2 compared to phase 1. Further there was a slight increase in the 13 - 40 Hz band and decrease in the 1 - 7 Hz band in phase 3 compared to phase 2. These differences were not substantial. On the other hand, comparing the total energy output as measured in millimeters, for all four frequency

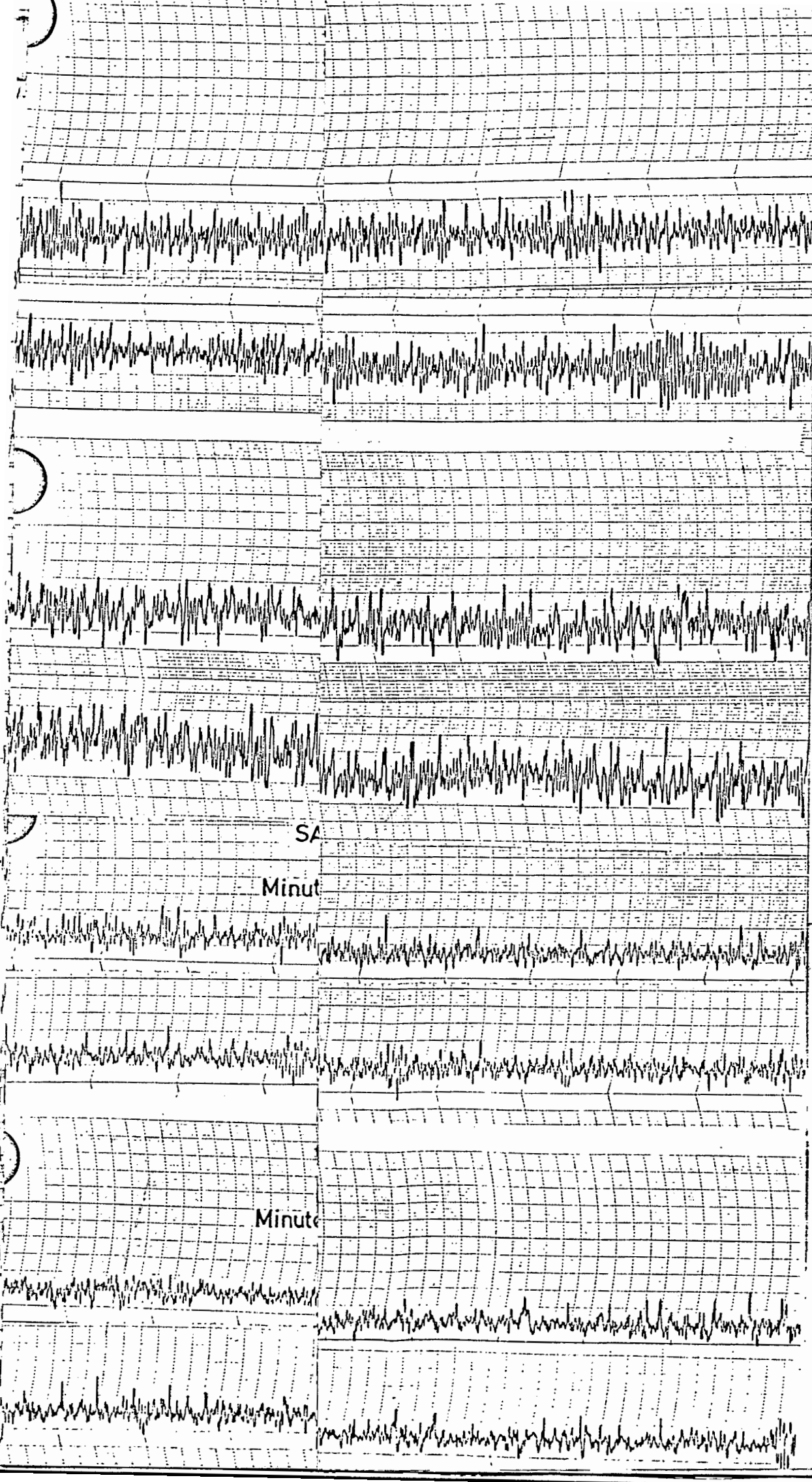
bands/...

bands in the various phases, there was approximately double the amount of energy in phase 2(i) and 2(ii) as compared to phase 1. During phase 3, the energy output was lowered, although not quite to the level registered in phase 1. On the EEG tracing, these changes in total energy output should be seen as changes in amplitudes (with the largest amplitude being found in phases 2(i) and 2(ii)). Little change could be expected in the frequency band; however, relating the shifts in percentage of energy in each frequency band; however, relating the shifts in percentage of energy in each frequency band band with the changes in total energy output, it may be expected that slower larger waves in the 1 - 7 Hz band would be more frequent during phase 2 than phase 1; further in phase 3, the drop in total energy output together with the shift towards the faster frequency bands (10 - 13 Hz, 13 - 40 Hz) should be seen as a greater amount of low amplitude fast waves in phase 3 compared with phase 2. Figure 19 represents minutes 1 and 2 of phase 1; minutes 4, 5, 6 and 7 of phases 2(i) and 2(ii); and minutes 4 and 5 of phase 3 alongside each other. It should be noted that the same observations which were made concerning the energy and frequency distributions in these various phases, with regards to the EEG POwer Spectrum Analyser results, apply to visual inspection of the EEG tracing. i.e. There was an increase in total energy output during phase 2 as compared to phase 1. This was accompanied by a slight increase in slow wave frequencies most especially in the 1 - 7 Hz band. During phase 3 there was a drop in total energy output due mainly to a decline in large amplitude slow wave frequency and an increase in 10 - 13 Hz and 13 - 40 Hz wave frequency.

It may be concluded that the results obtained from the EEG Power SPectrum Analyser closely parallel the conclusions drawn from visual

inspection/...

inspection of the EEG tracing. These observations demonstrate the utility of the Power Spectrum Analyser in EEG research where distinctions are to be made between theta activity, low and high alpha activity and beta activity as well as in EEG research concerned with amplitude changes. In samples 2 and 3 it became apparent from simultaneous observations of amplitude and frequency changes that the Analyser's results were in agreement with the common observations of a reverse relationship between EEG frequency and amplitude : i.e. the smaller the wave's frequency, the greater is its amplitude. The larger the EEG frequency, the greater its amplitude.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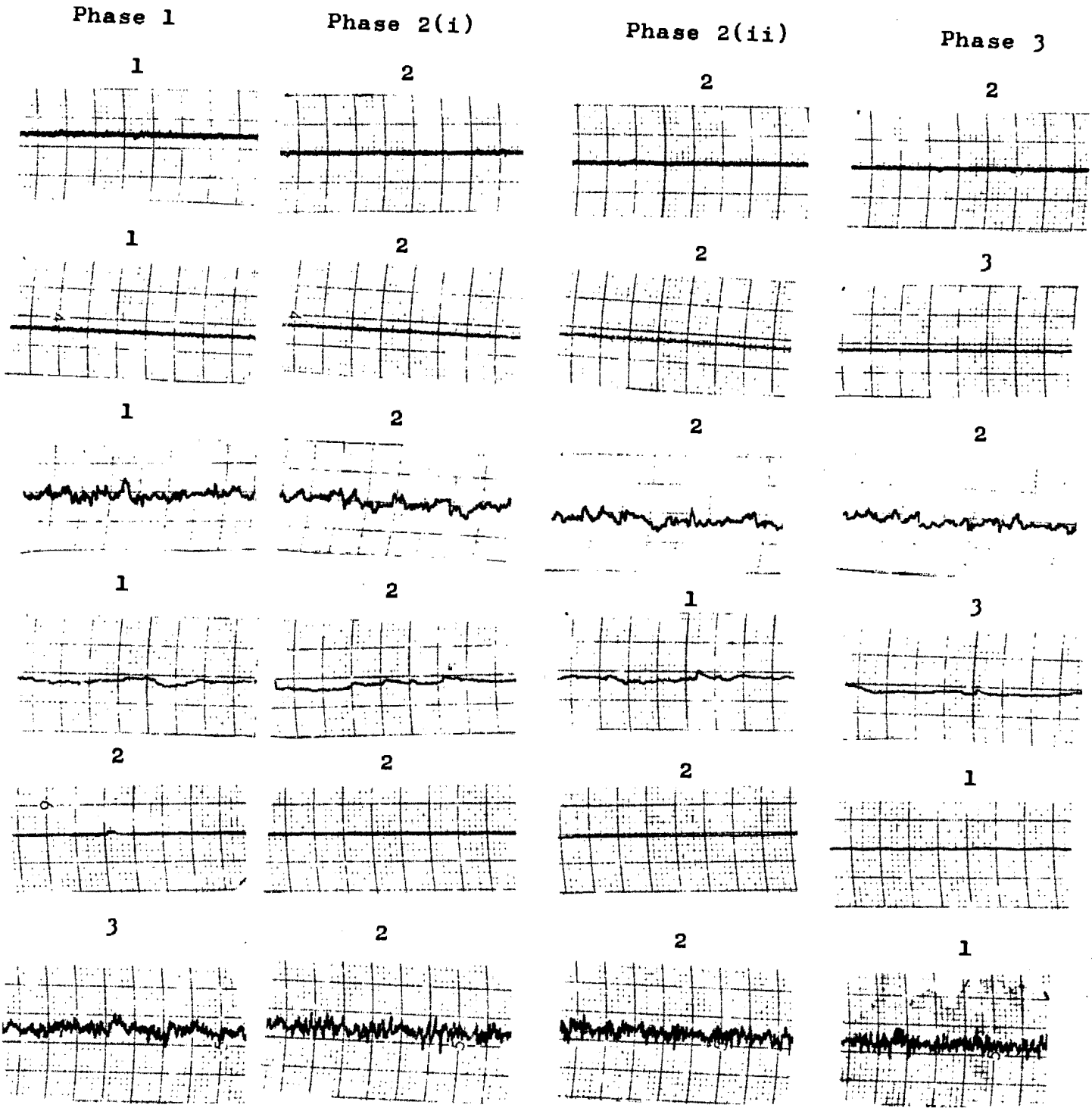
MASSETER MUSCLE - BASELINE 1

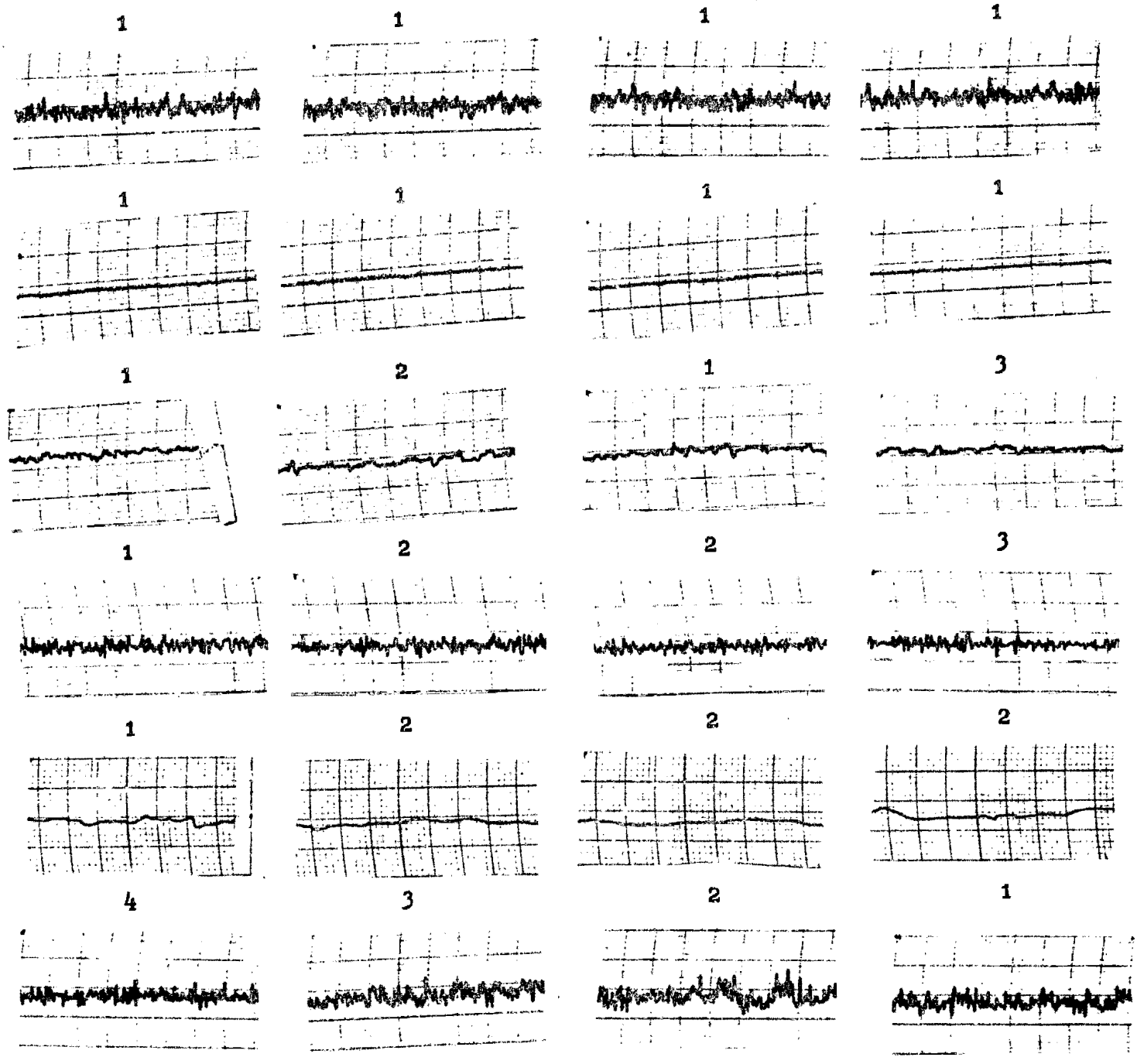
CONTROL GROUP I



MASSETER MUSCLE - BASELINE 2

CONTROL GROUP I

Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
1	1	1	1
1	1	1	1
1	2	1	3
1	2	2	3
1	2	2	2
4	3	2	1

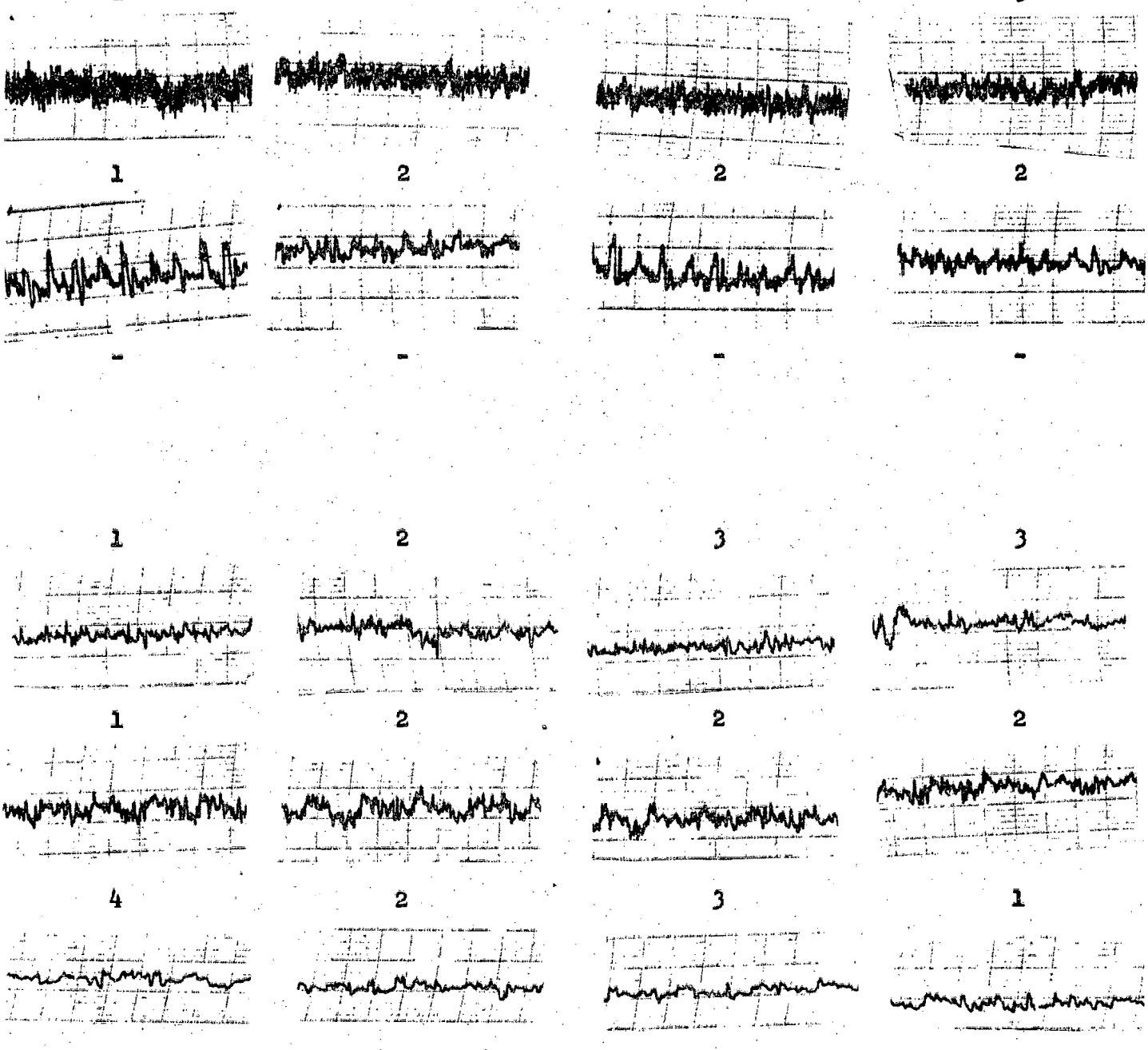


Masseter/...

MASSETER MUSCLE - BASELINE 3

CONTROL GROUP I

Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
1	2	2	3
1	2	2	2
-	-	-	-
1	2	3	3
1	2	2	2
4	2	3	1

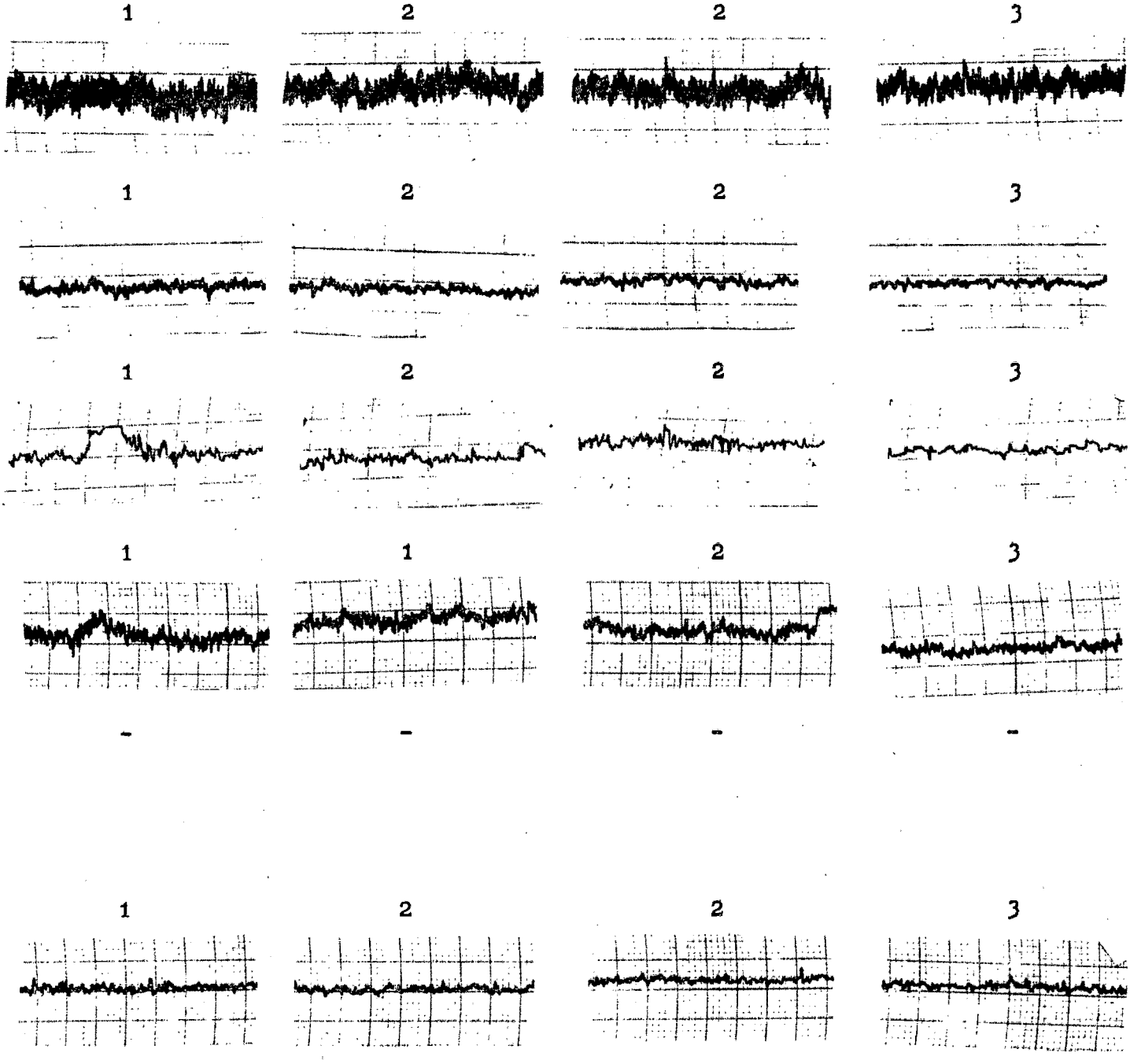


Masseter/...

MASSETER MUSCLE - TREATMENT SESSION 1

CONTROL GROUP I

Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
1	2	2	3
1	2	2	3
1	2	2	3
1	1	2	3
-	-	-	-
1	2	2	3

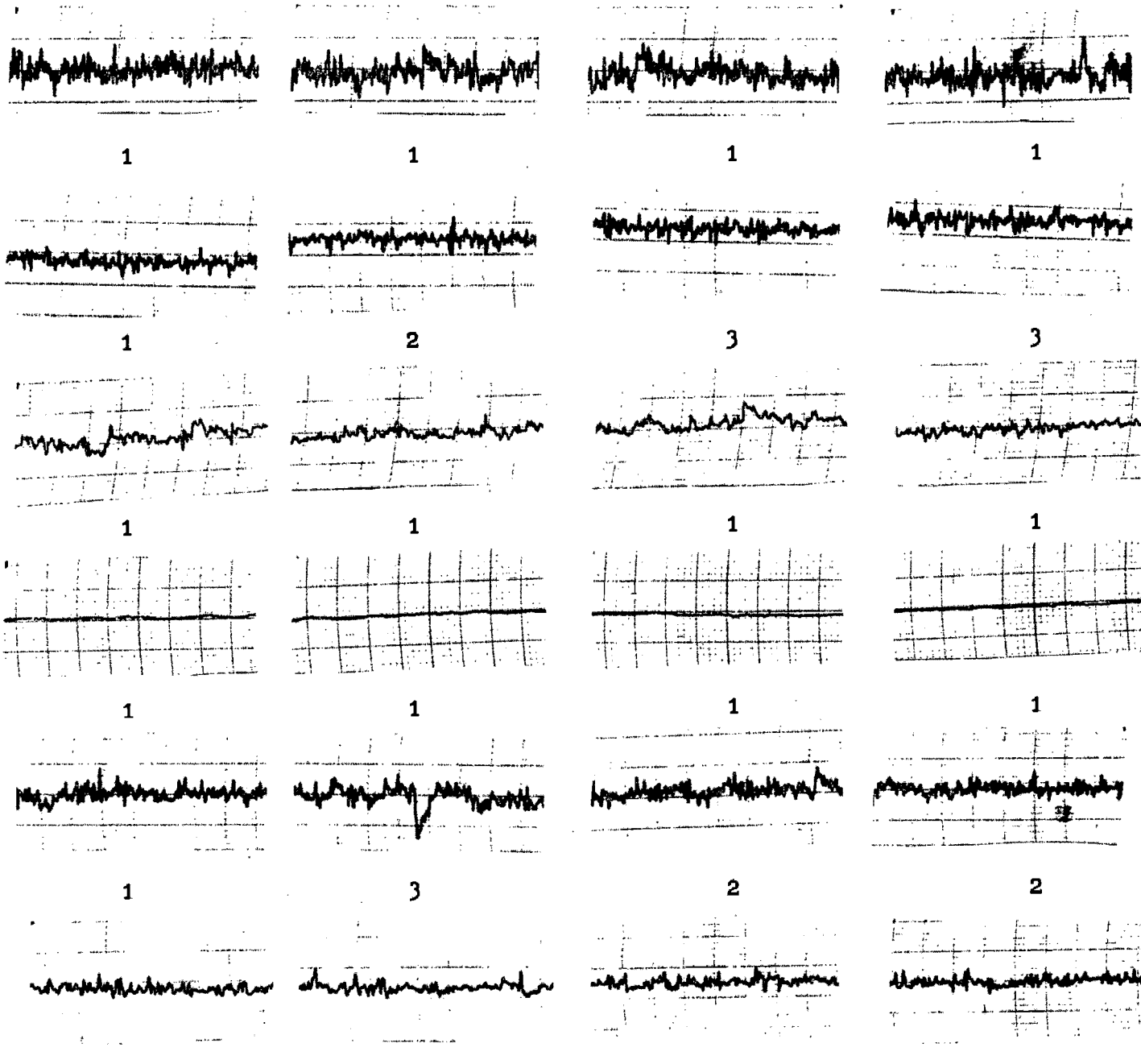


Masseter/...

MASSETER MUSCLE - TREATMENT SESSION 2

CONTROL GROUP I

Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
1	2	2	1
1	1	1	1
1	2	3	3
1	1	1	1
1	1	1	1
1	3	2	2

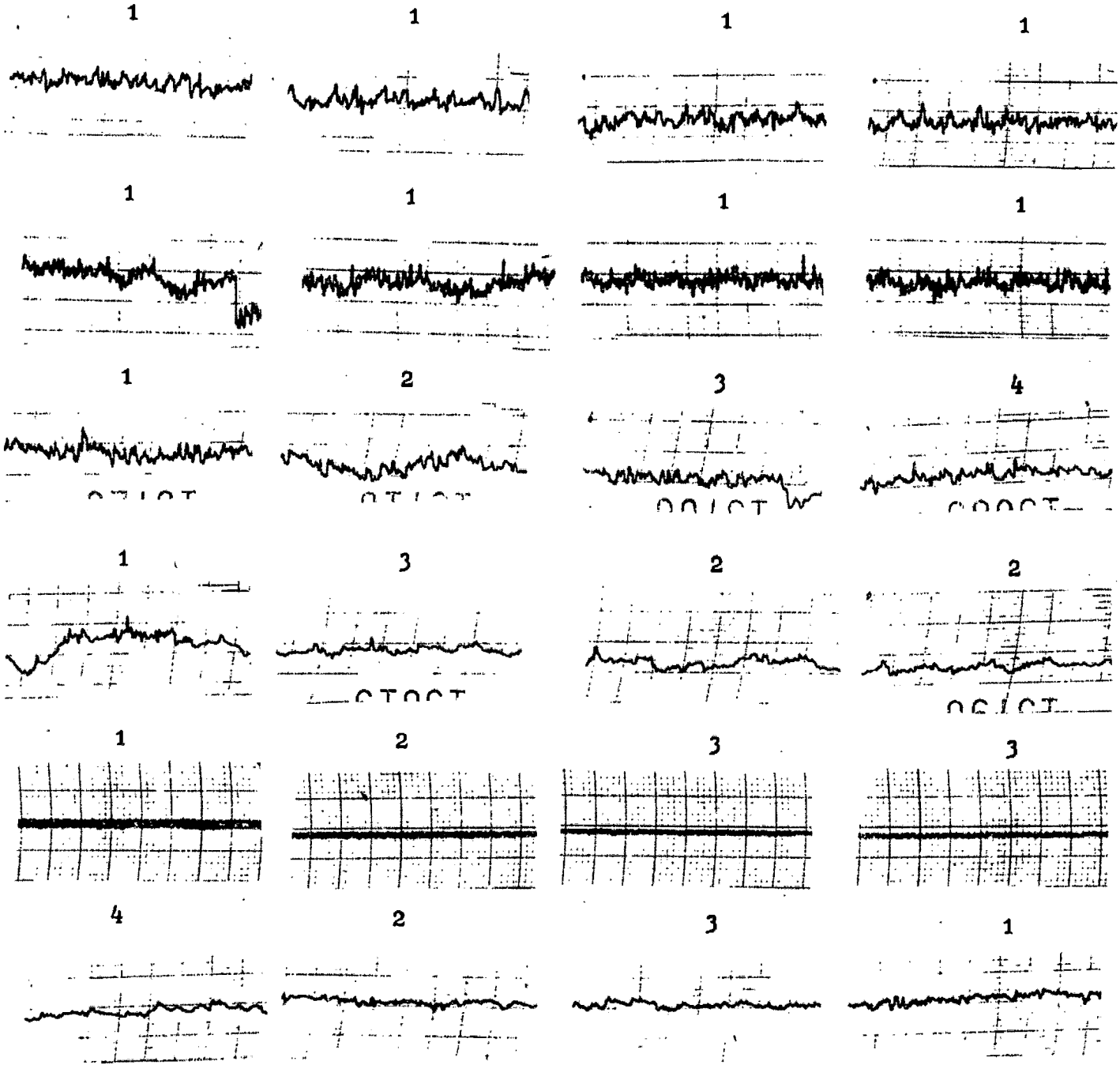


Masseter/...

MASSETER MUSCLE - TREATMENT SESSION 3

CONTROL GROUP I

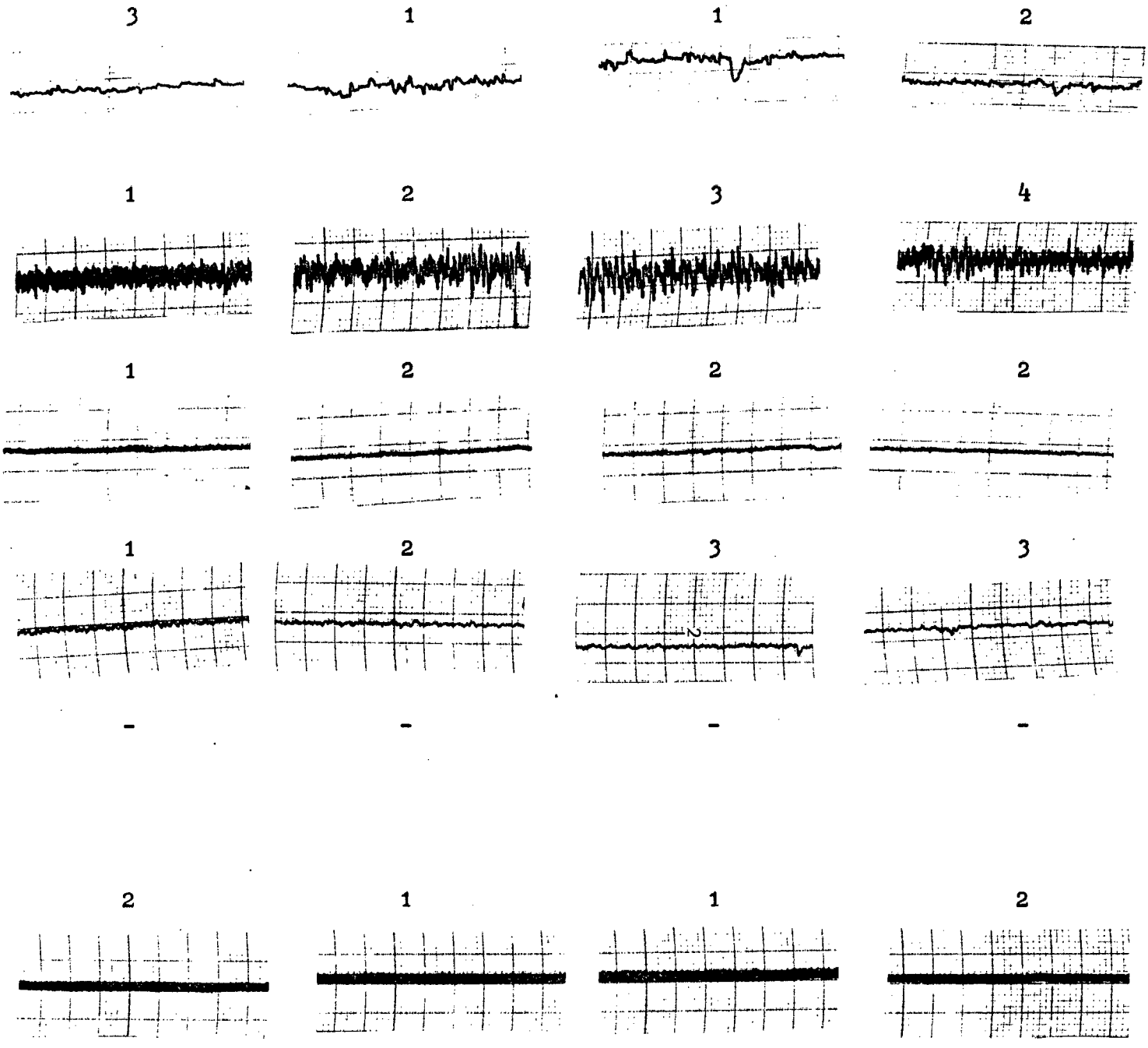
Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
1	1	1	1
1	1	1	1
1	2	3	4
1	3	2	2
1	2	3	3
4	2	3	1



MASSETER MUSCLE - TREATMENT SESSION 1

EXPERIMENTAL GROUP

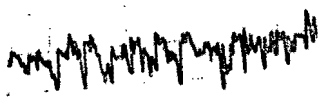
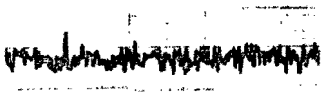
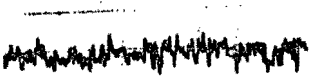
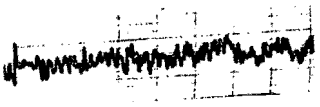
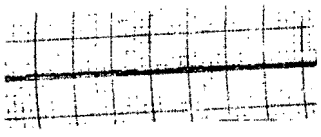
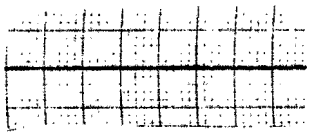
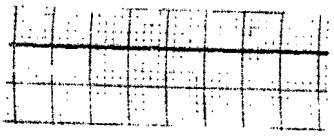
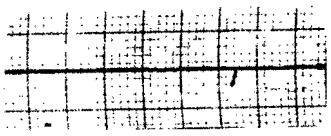
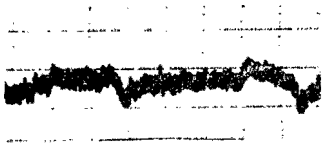
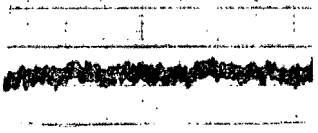
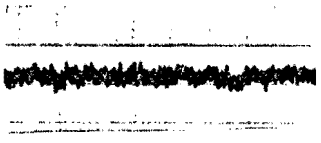
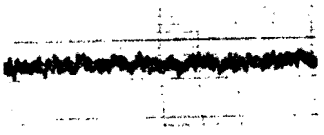
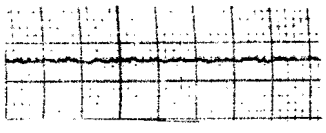
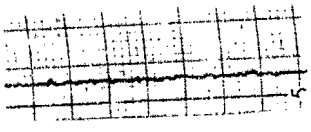
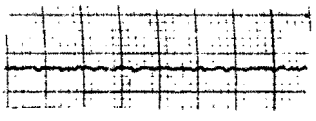
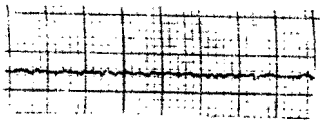
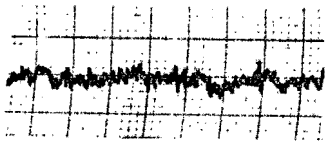
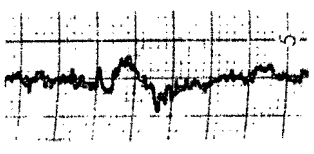
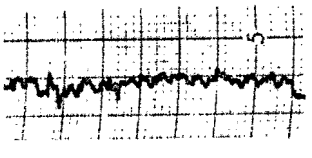
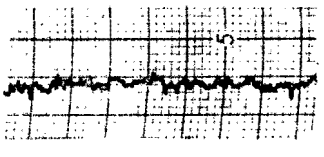
Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
3	1	1	2
1	2	3	4
1	2	2	2
1	2	3	3
-	-	-	-
2	1	1	2



Masseter/...

MASSETER MUSCLE - TREATMENT SESSION 2

EXPERIMENTAL GROUP

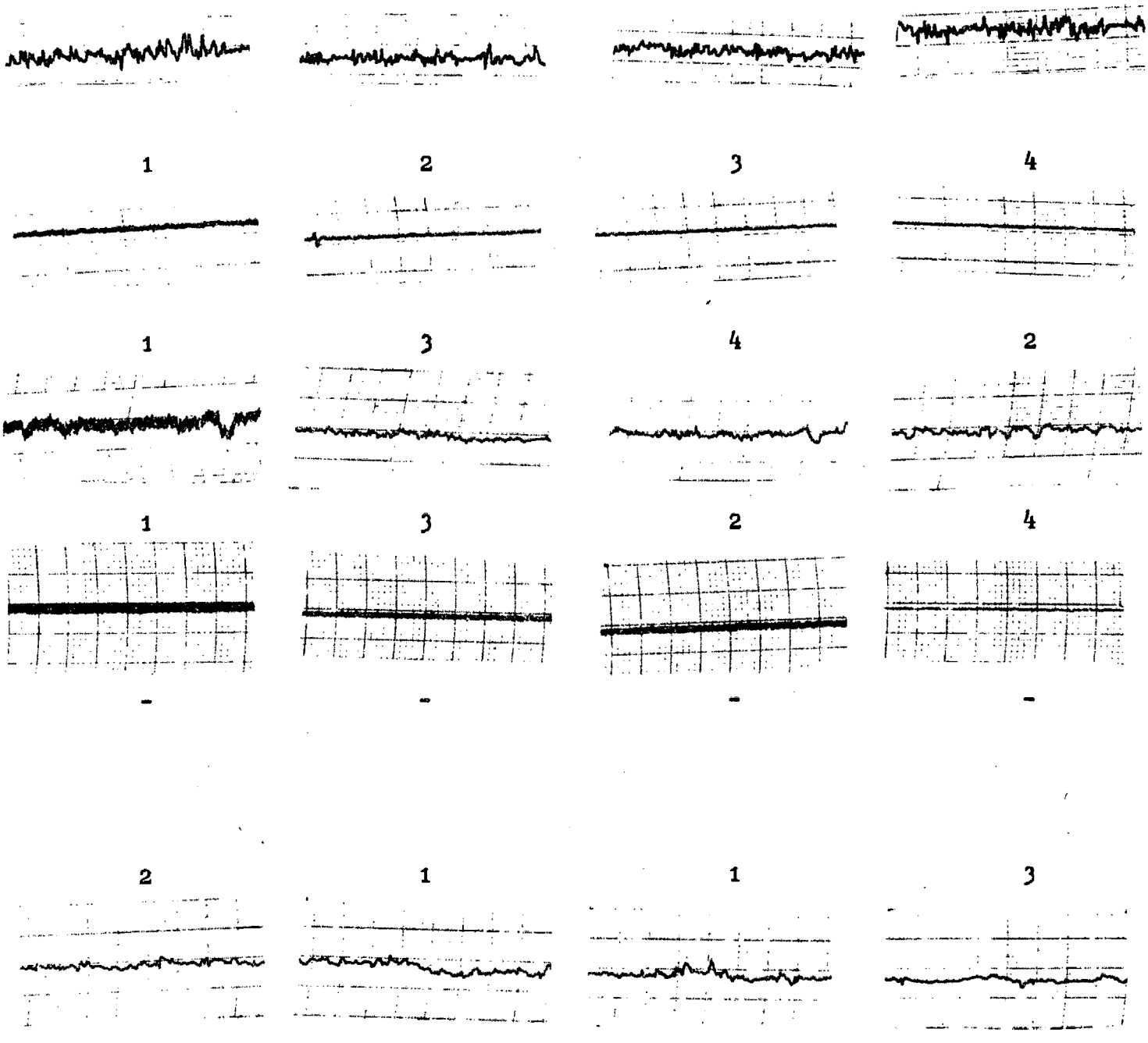
Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
1	2	4	3
			
1	2	2	2
			
1	2	2	3
			
1	2	3	1
			
-	-	-	-
1	2	3	4
			

Masseter/...

MASSETER MUSCLE - TREATMENT SESSION 3

EXPERIMENTAL GROUP

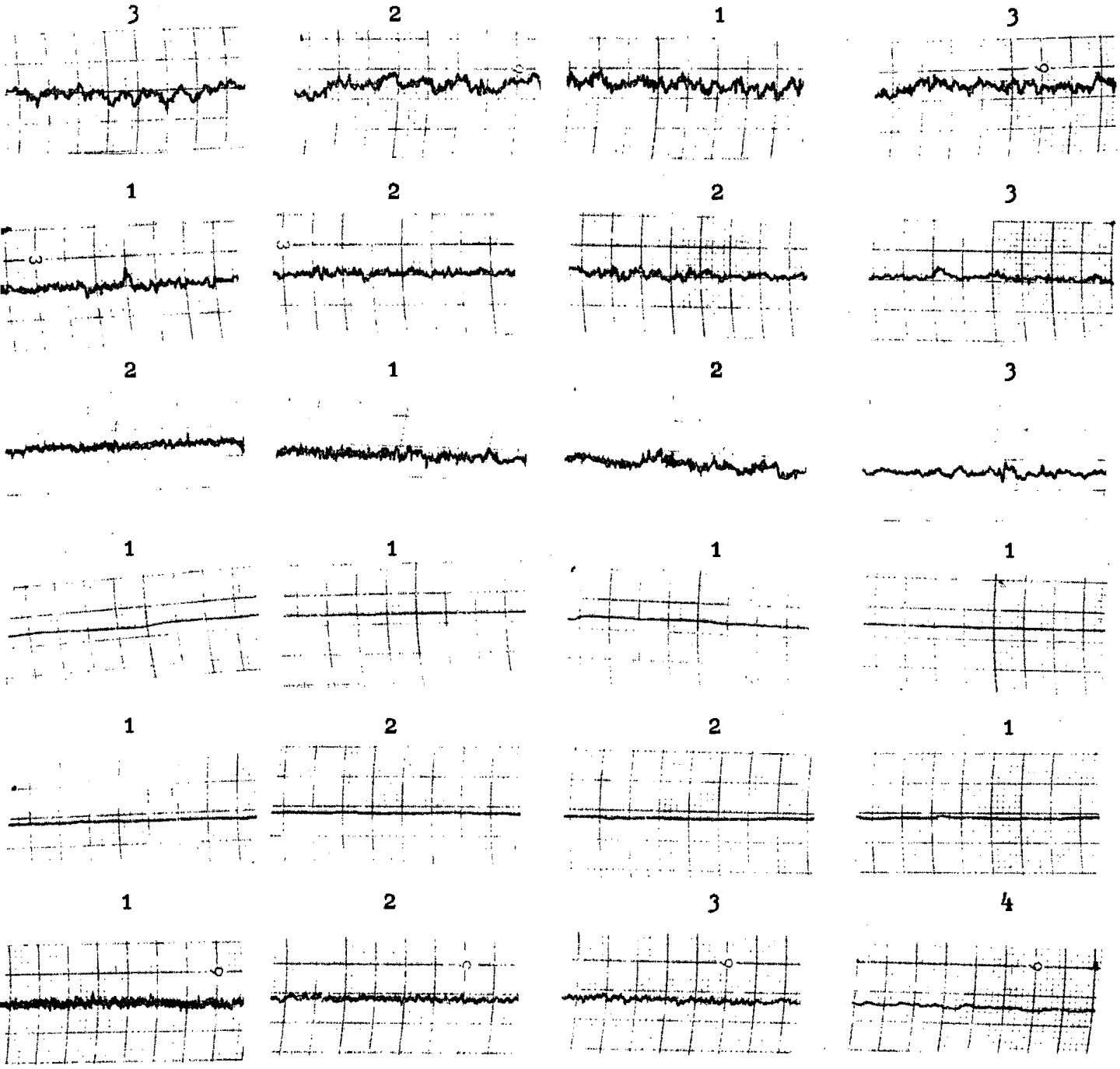
Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
1	3	4	2
1	2	3	4
1	3	4	2
1	3	2	4
-	-	-	-
2	1	1	3



CORRUGATOR MUSCLE - BASELINE 1

CONTROL GROUP I

Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
3	2	1	3
1	2	2	3
2	1	2	3
1	1	1	1
1	2	2	1
1	2	3	4

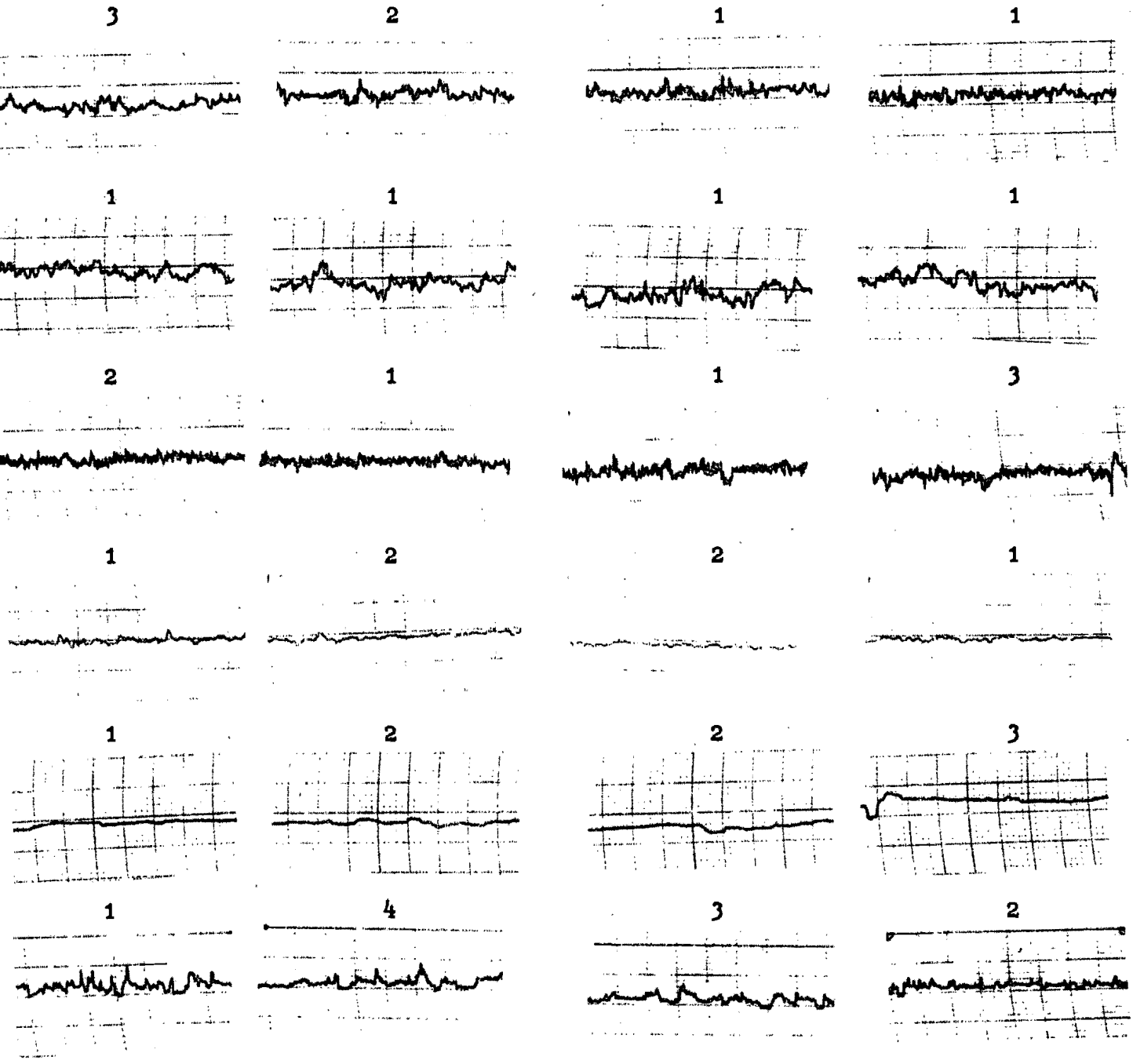


Corrugator/...

CORRUGATOR MUSCLE - BASELINE 2

CONTROL GROUP I

Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
3	2	1	1
1	1	1	1
2	1	1	3
1	2	2	1
1	2	2	3
1	4	3	2

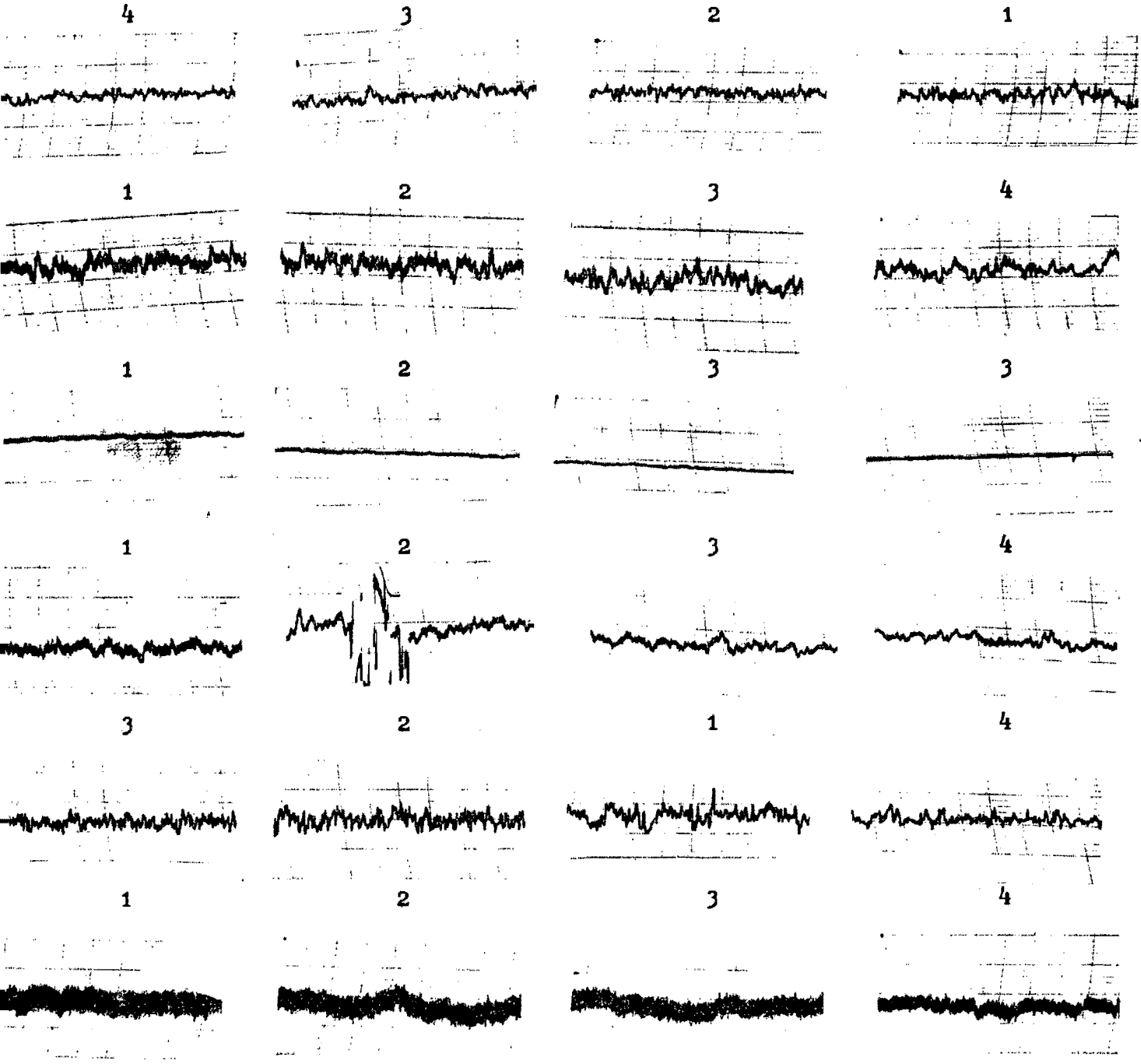


Corrugator/...

CORRUGATOR MUSCLE - BASELINE 3

CONTROL GROUP I

Phase 1 | Phase 2(i) | Phase 2(ii) | Phase 3

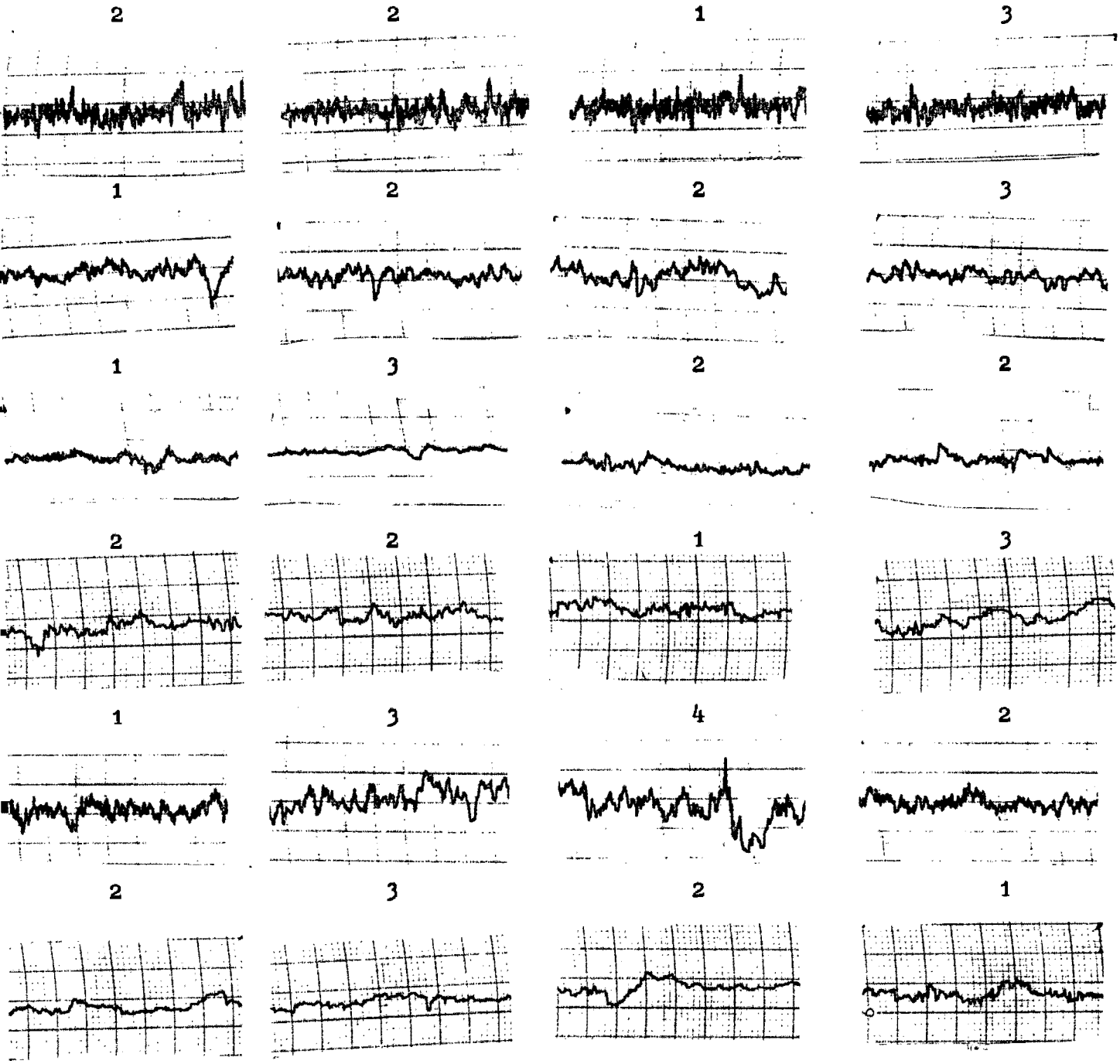


Corrugator/...

CORRUGATOR MUSCLE - TREATMENT SESSION 1

CONTROL GROUP I

Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
2	2	1	3
1	2	2	3
1	3	2	2
2	2	1	3
1	3	4	2
2	3	2	1

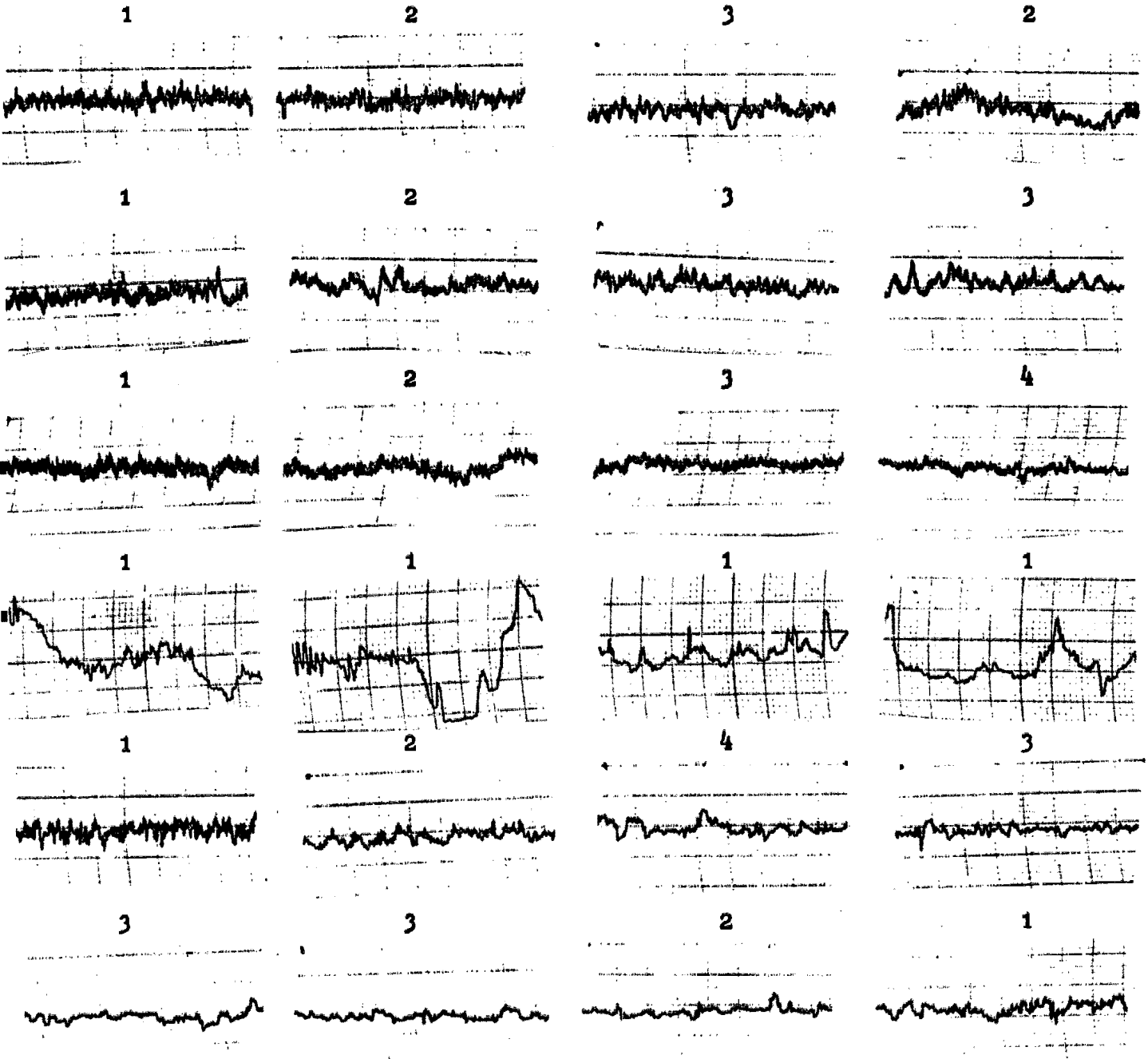


Corrugator/...

CORRUGATOR MUSCLE - TREATMENT SESSION 2

CONTROL GROUP I

Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
1	2	3	2
1	2	3	3
1	2	3	4
1	1	1	1
1	2	4	3
3	3	2	1

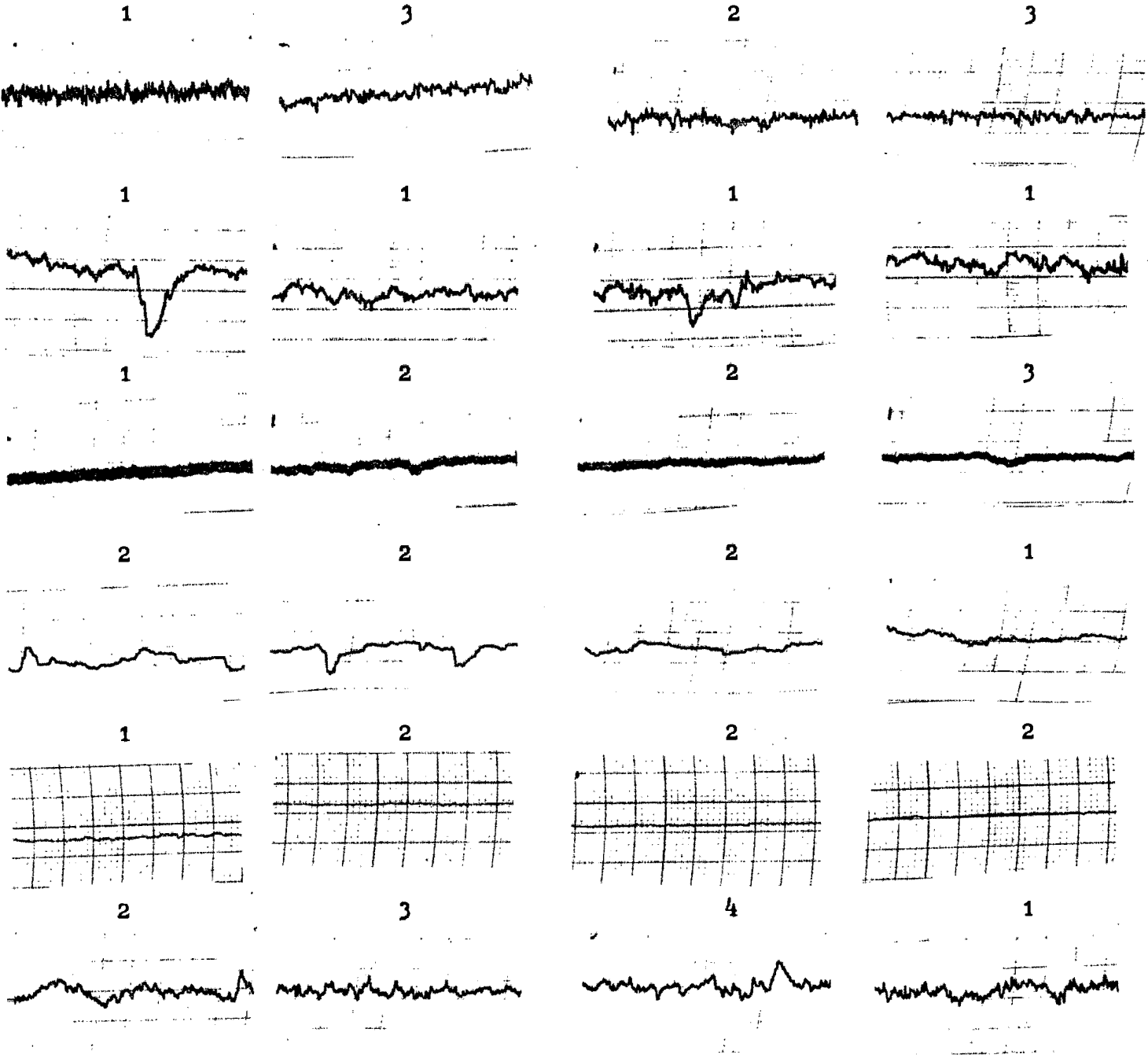


Corrugator/...

CORRUGATOR MUSCLE - TREATMENT SESSION 3

CONTROL GROUP I

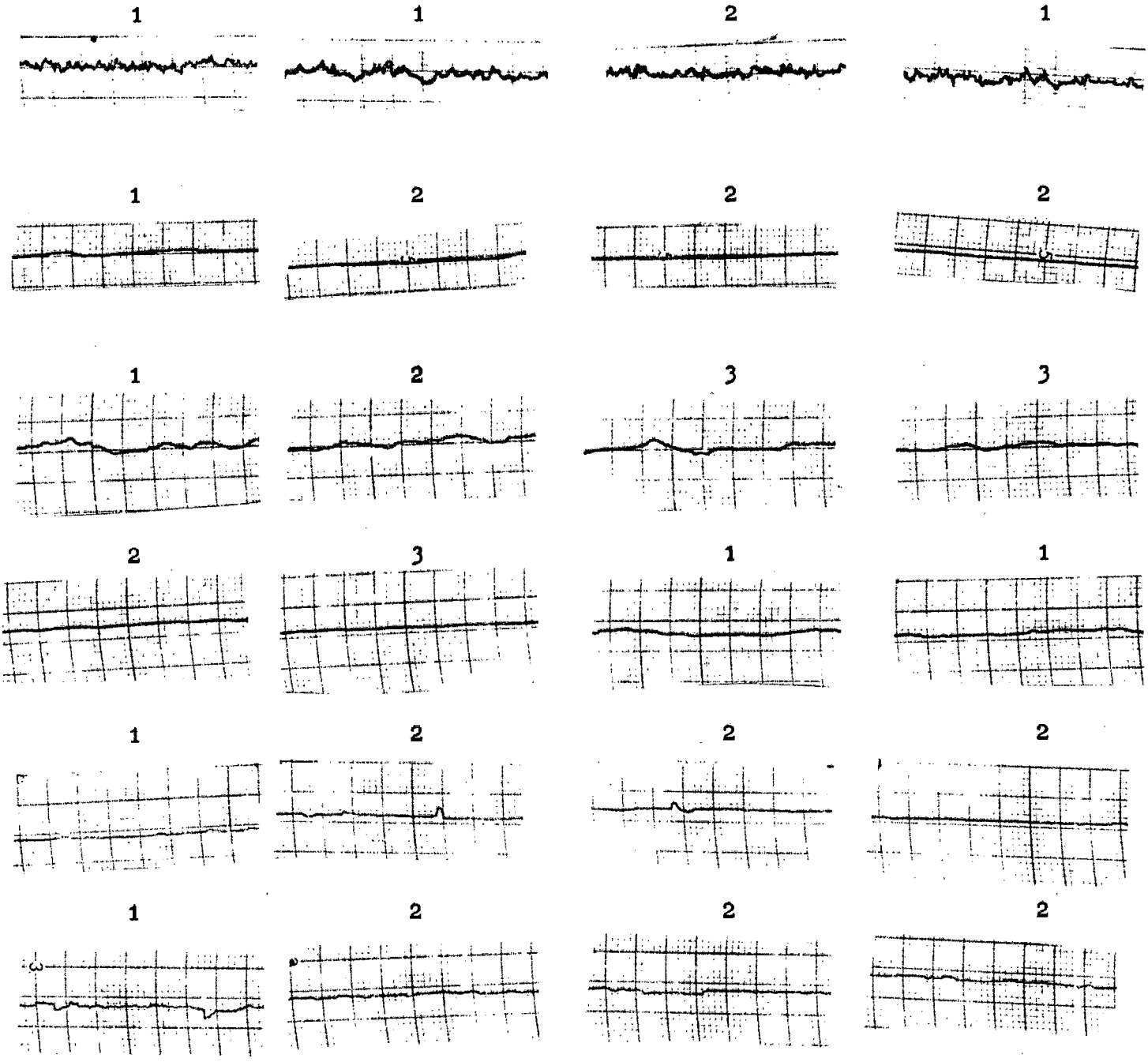
Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
1	3	2	3
1	1	1	1
1	2	2	3
2	2	2	1
1	2	2	2
2	3	4	1



CORRUGATOR MUSCLE - BASELINE 1

EXPERIMENTAL GROUP

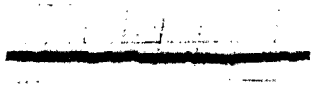
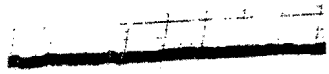
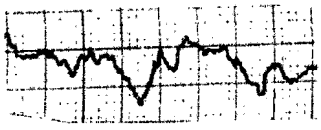
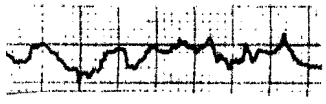
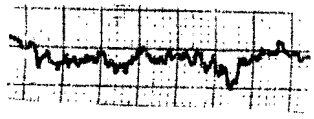
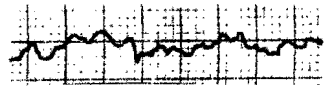
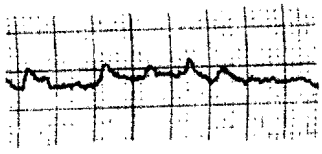
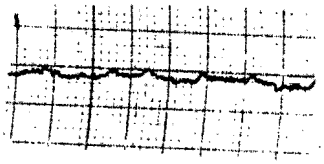
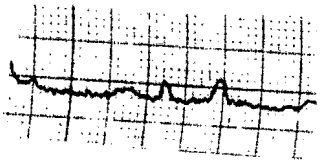
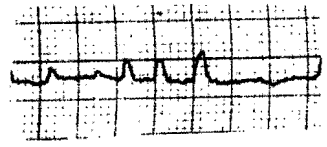
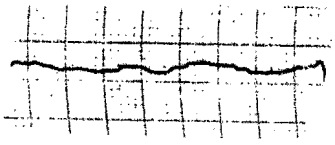
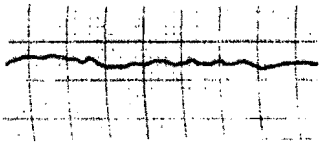
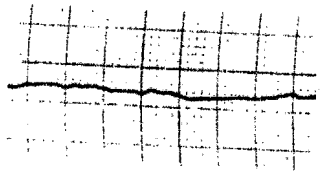
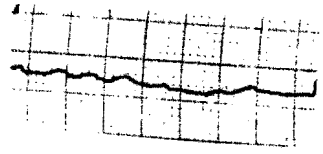
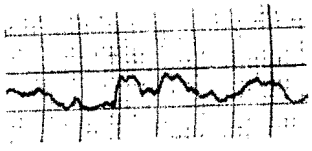
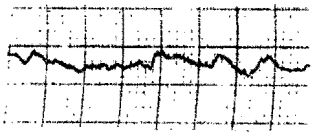
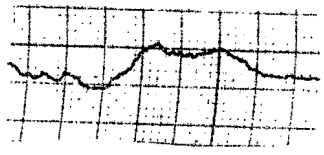
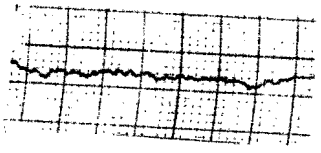
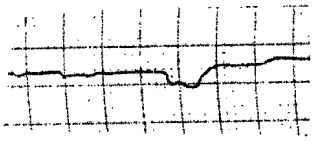
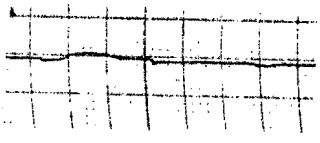
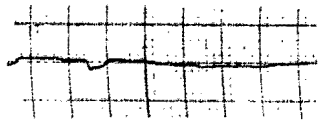
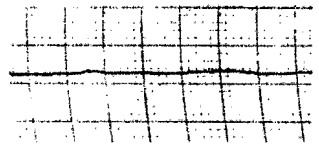
Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
1	1	2	1
1	2	2	2
1	2	3	3
2	3	1	1
1	2	2	2
1	2	2	2



Corrugator/...

CORRUGATOR MUSCLE - BASELINE 2

EXPERIMENTAL GROUP

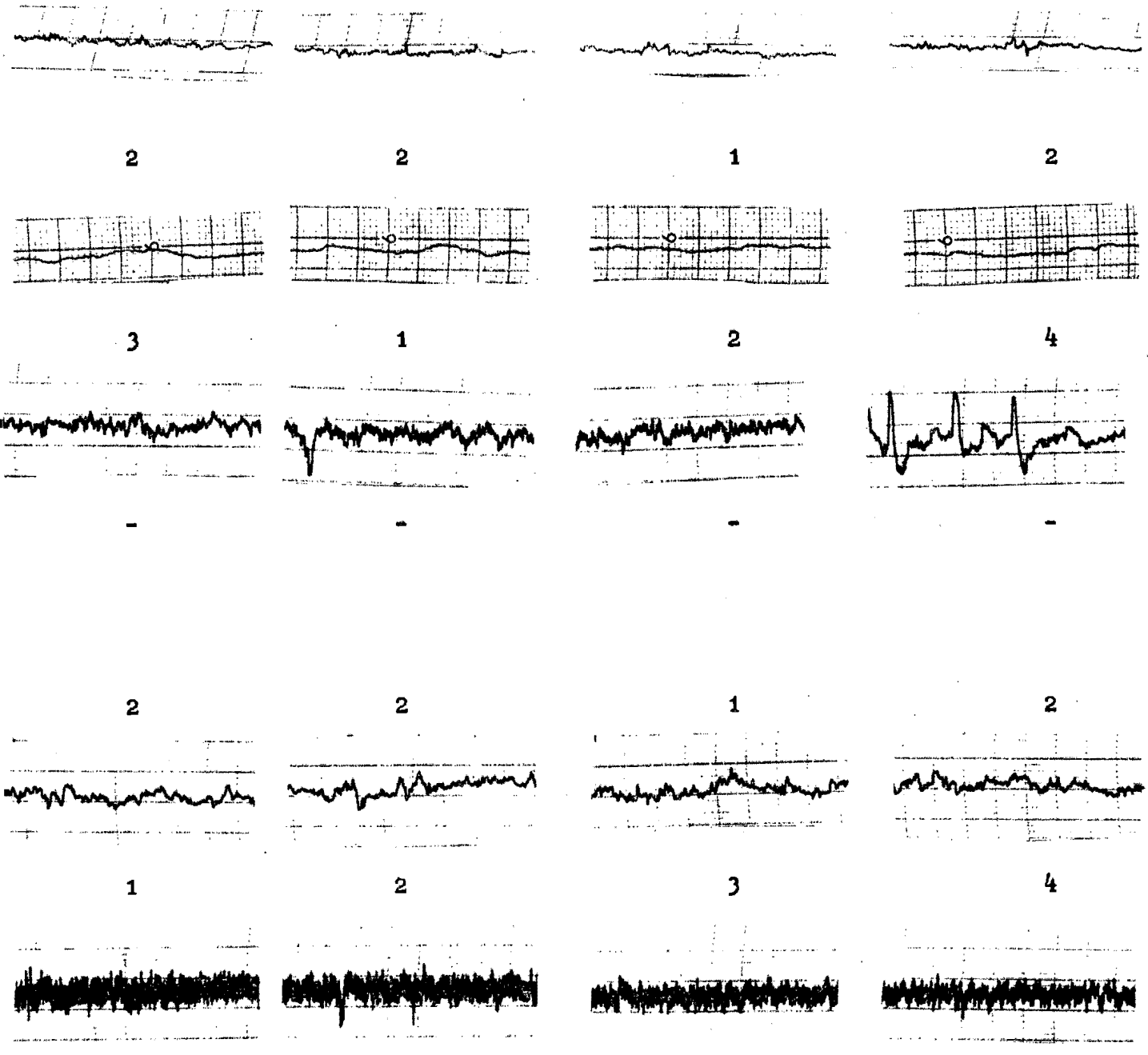
Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
1	2	2	1
			
3	2	1	4
			
1	1	1	2
			
3	2	2	1
			
1	2	3	4
			
1	2	2	3
			

Corrugator/...

CORRUGATOR MUSCLE - BASELINE 3

EXPERIMENTAL GROUP

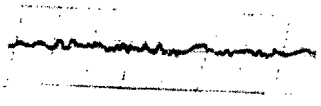
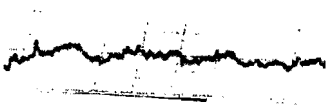
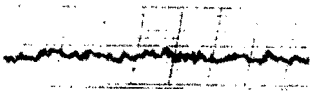
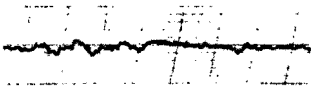
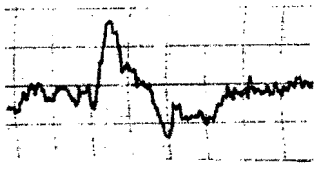
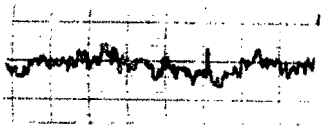
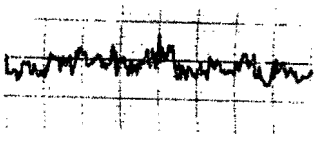
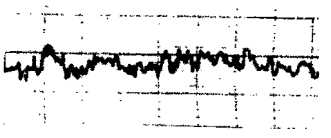
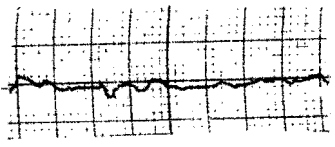
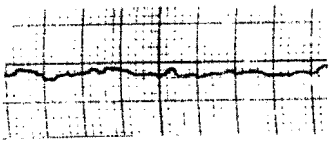
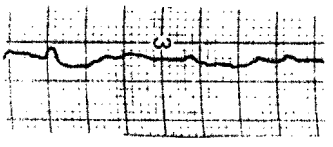
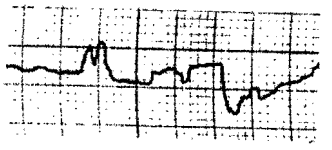
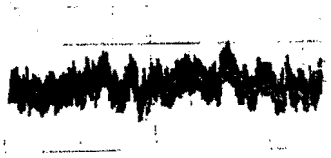
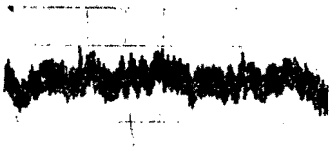
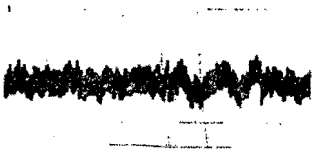
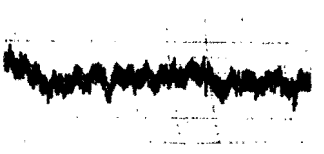
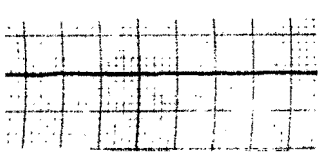
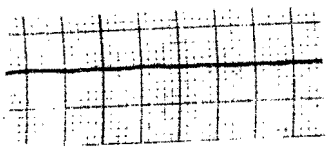
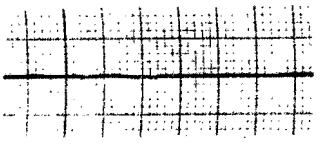
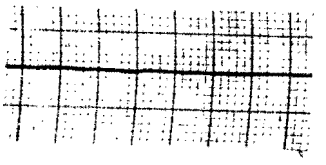
Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
1	2	2	3
2	2	1	2
3	1	2	4
-	-	-	-
2	2	1	2
1	2	3	4



Corrugator/...

CORRUGATOR MUSCLE - TREATMENT SESSION 1

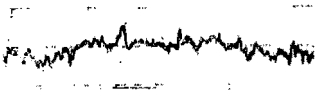
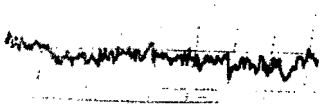
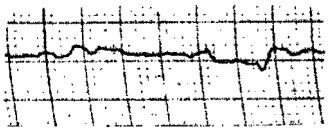
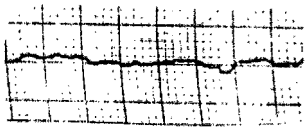
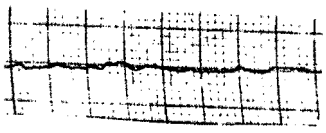
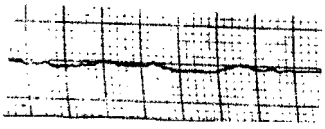
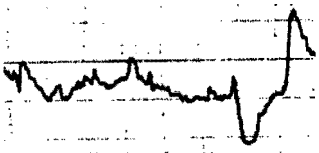
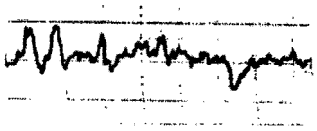
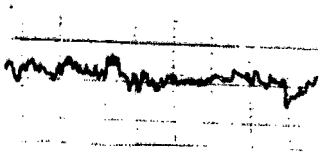
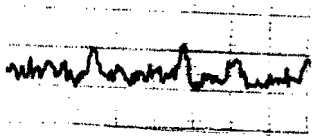
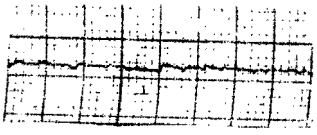
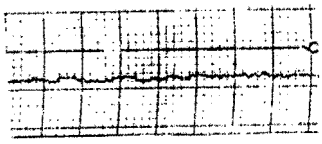
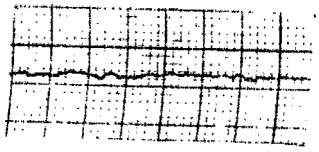
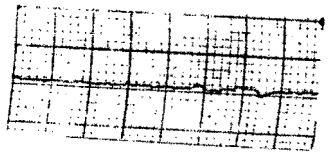
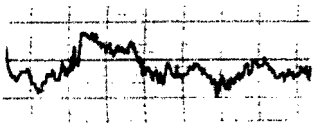
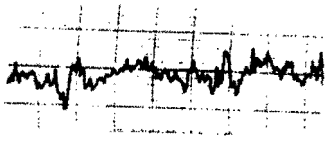
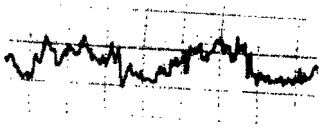
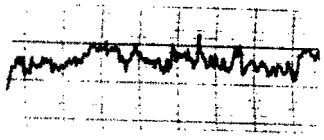
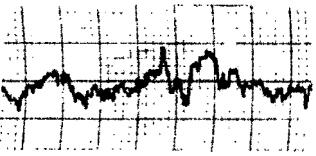
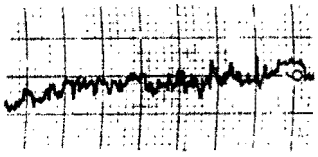
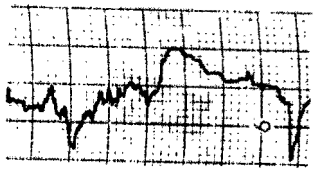
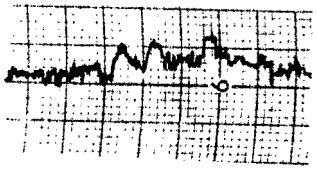
EXPERIMENTAL GROUP

Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
3	2	1	4
			
-	-	-	-
2	2	1	3
			
1	1	3	2
			
1	2	3	4
			
1	1	1	1
			

Corrugator/...

CORRUGATOR MUSCLE - TREATMENT SESSION 2

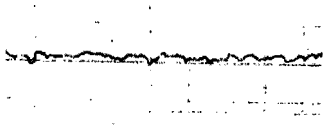
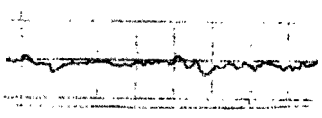
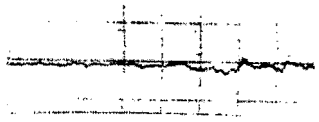
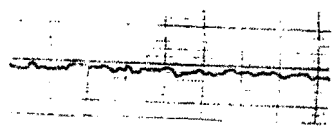
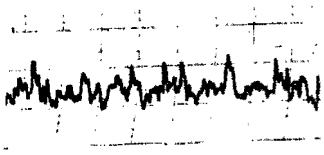
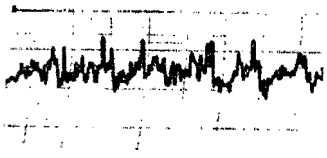
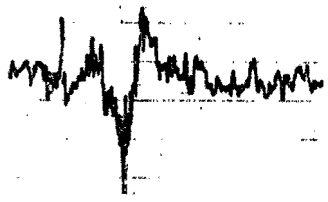
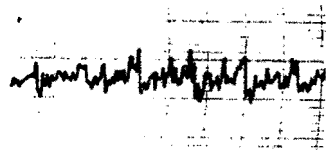
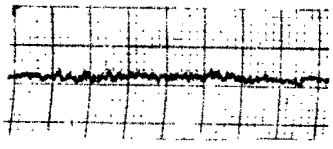
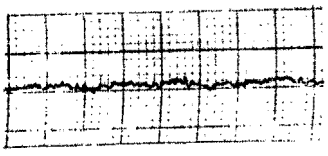
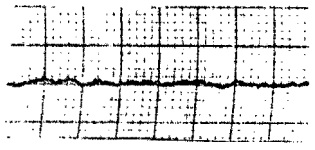
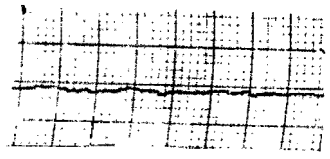
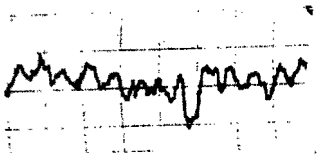
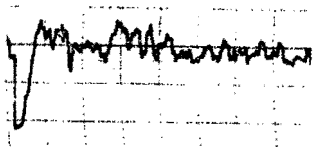
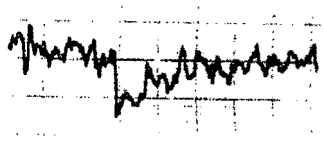
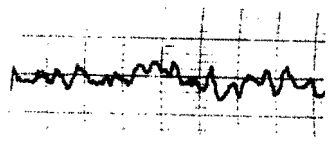
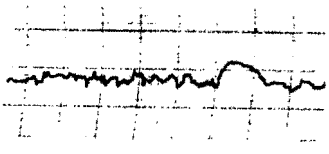
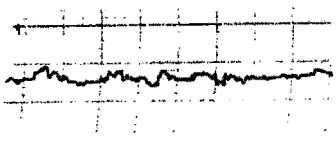
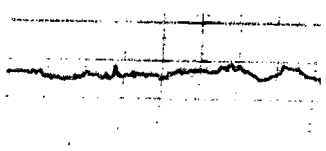
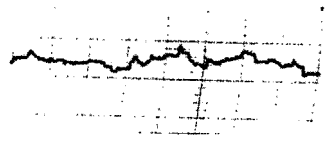
EXPERIMENTAL GROUP

Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
4	3	2	1
			
1	2	4	3
			
3	2	1	1
			
1	1	2	2
			
1	2	2	2
			
2	1	4	3
			

Corrugator/...

CORRUGATOR MUSCLE - TREATMENT SESSION 3

EXPERIMENTAL GROUP

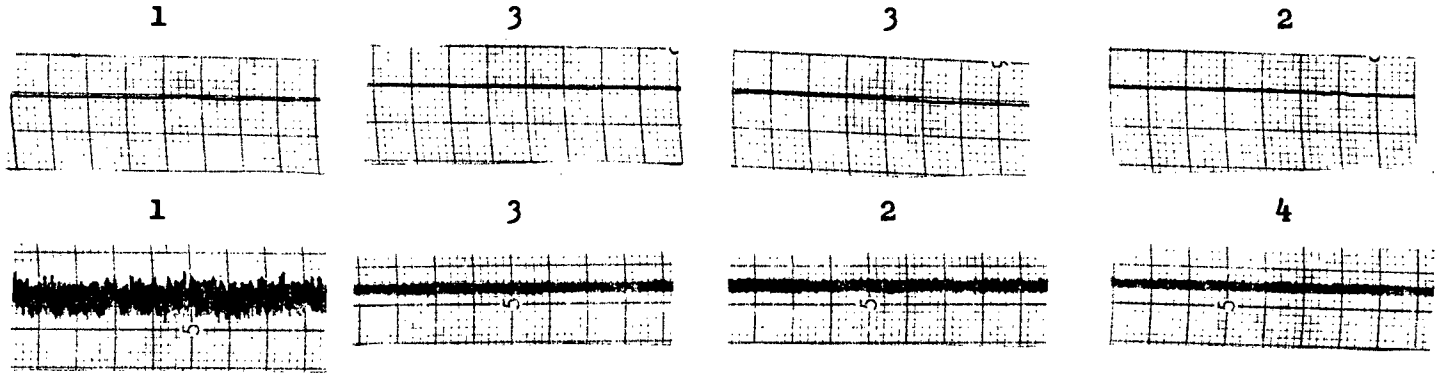
Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3
3	1	1	2
			
1	3	4	2
			
3	1	1	2
			
1	2	3	4
			
3	2	1	4
			
2	3	4	1
			

MASSETER MUSCLE - BASELINES 1, 2, 3

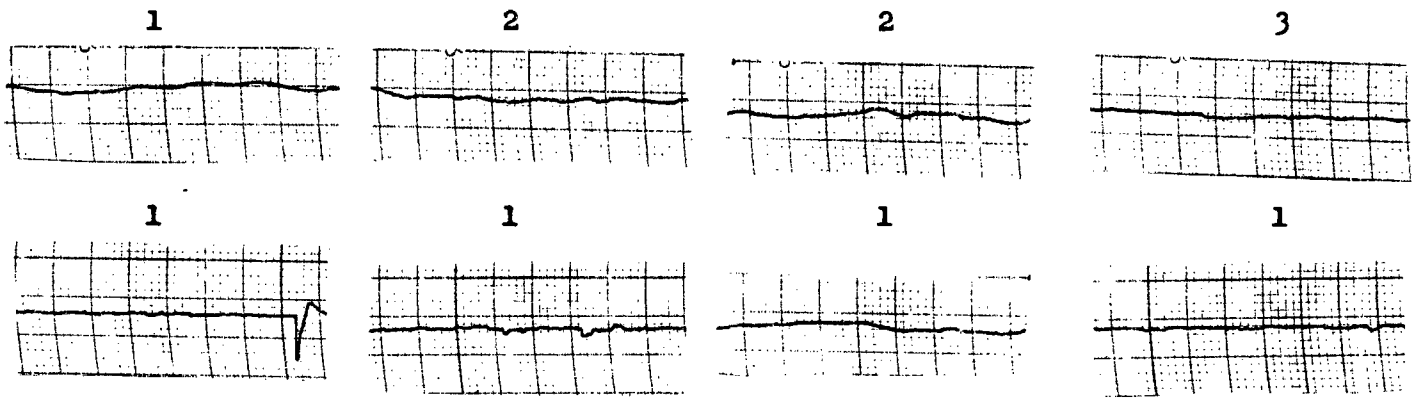
CONTROL GROUP II

Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3

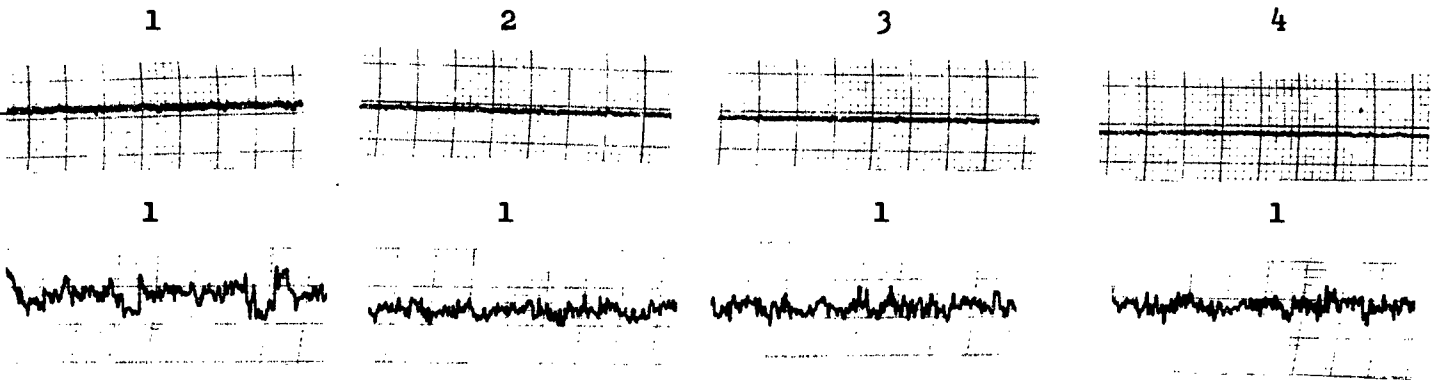
BASELINE 1



BASELINE 2



BASELINE 3



Masseter/...

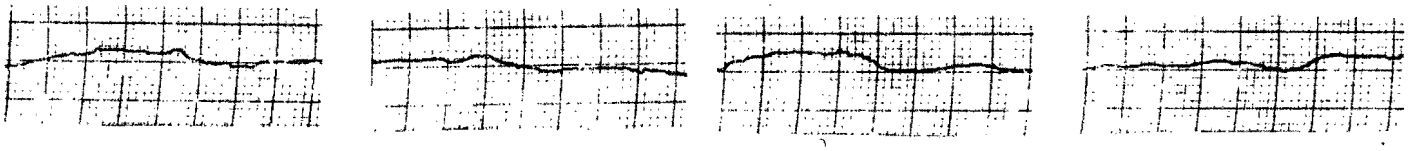
MASSETER MUSCLE - TREATMENT SESSIONS 1, 2, 3

CONTROL GROUP II

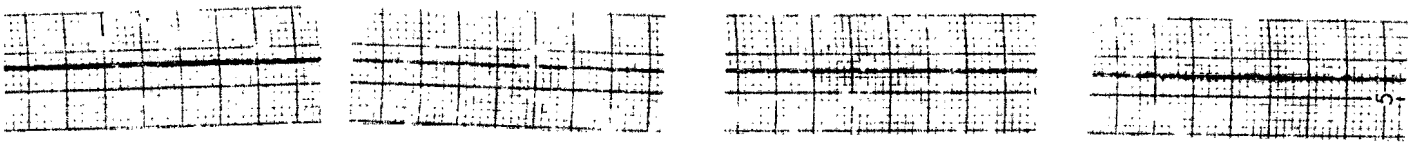
Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3

TREATMENT SESSION 1

2 2 1 3



1 3 2 1



TREATMENT SESSION 2

\- - - -

2 3 4 1

TREATMENT SESSION 3

\- - - -

2 2 2 1

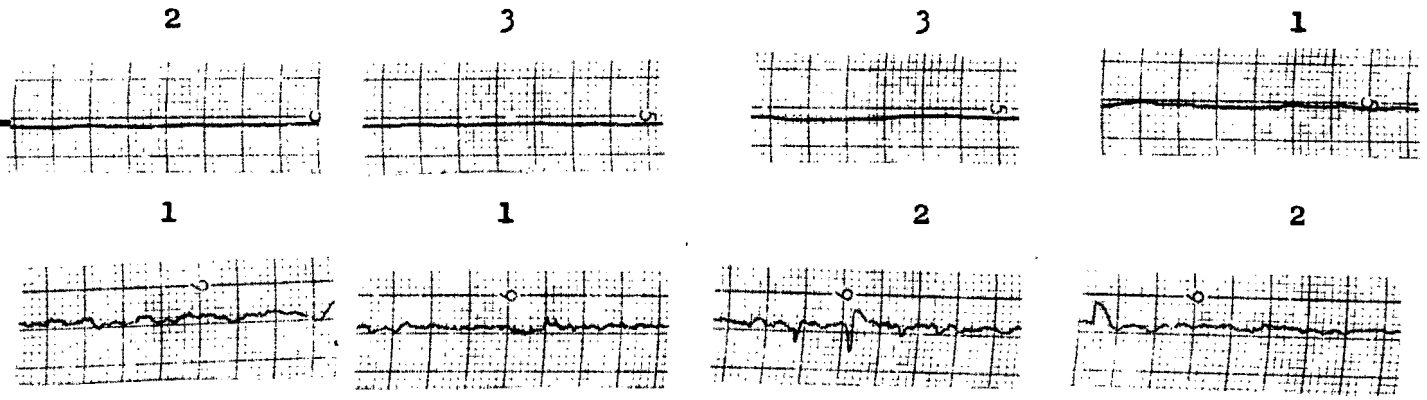
Corrugator/...

CORRUGATOR MUSCLE - BASELINES 1, 2, 3

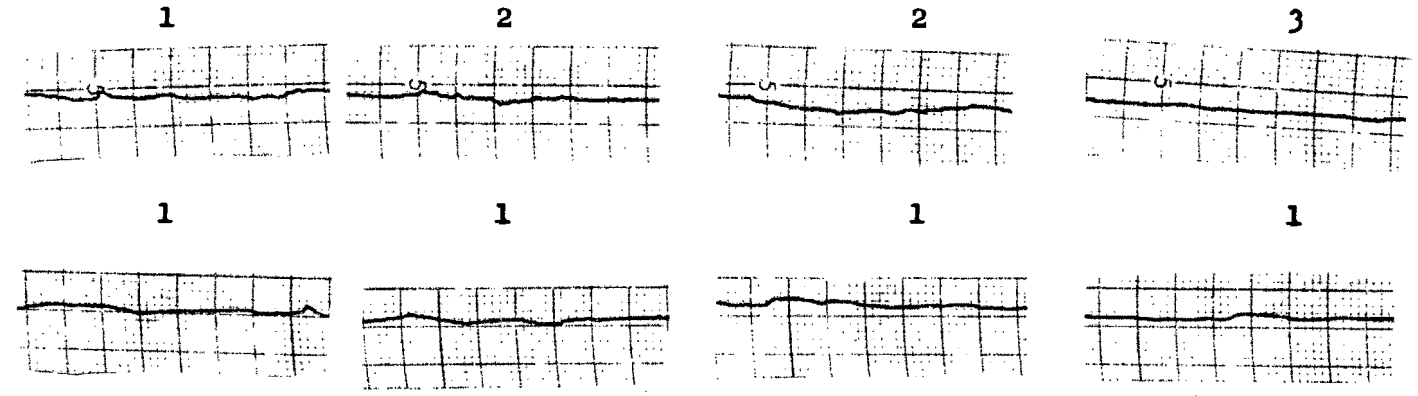
CONTROL GROUP II

Phase 1	Phase 2(i)	Phase 2(ii)	Phase 3

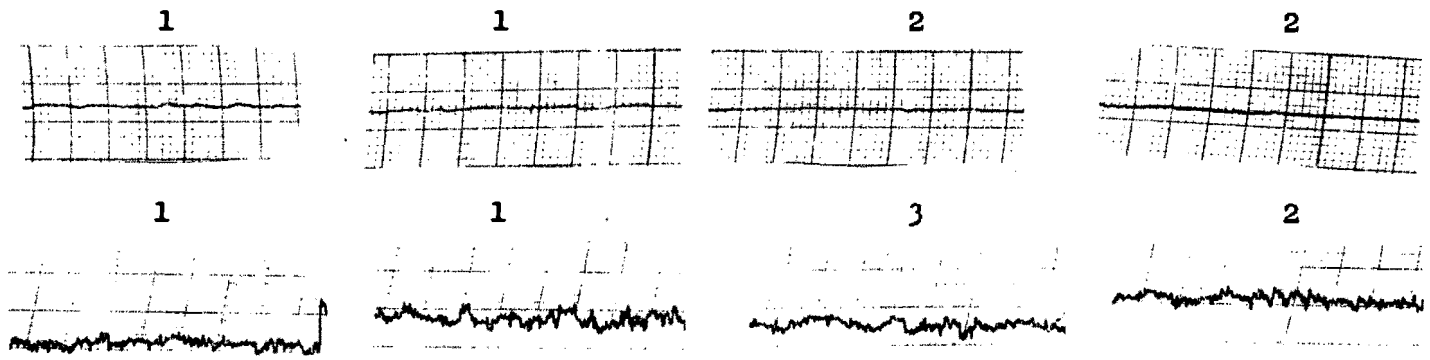
BASELINE 1



BASELINE 2



BASELINE 3



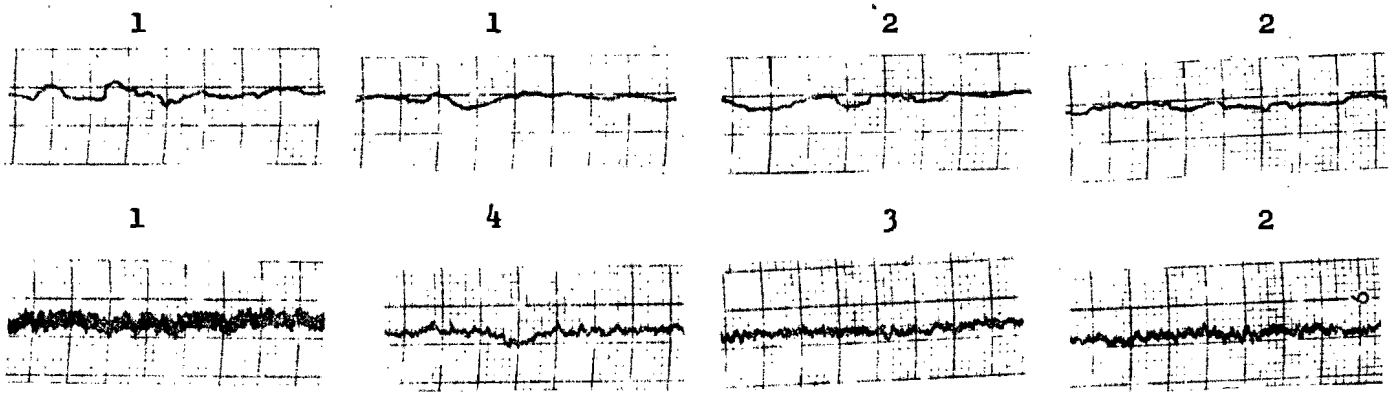
Corrugator/...

CORRUGATOR MUSCLE - TREATMENT SESSIONS 1, 2, 3

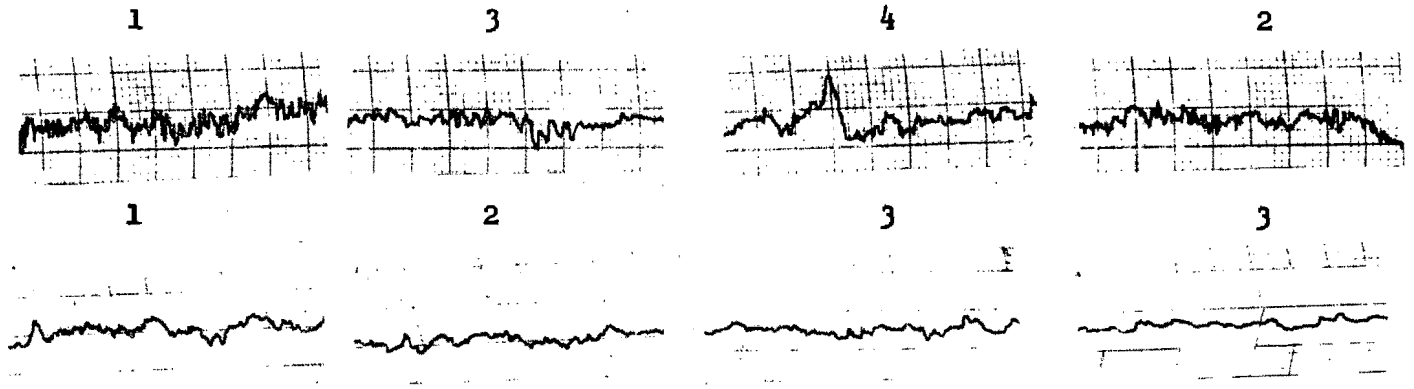
CONTROL GROUP II

Phase 1 Phase 2(i) Phase 2(ii) Phase 3

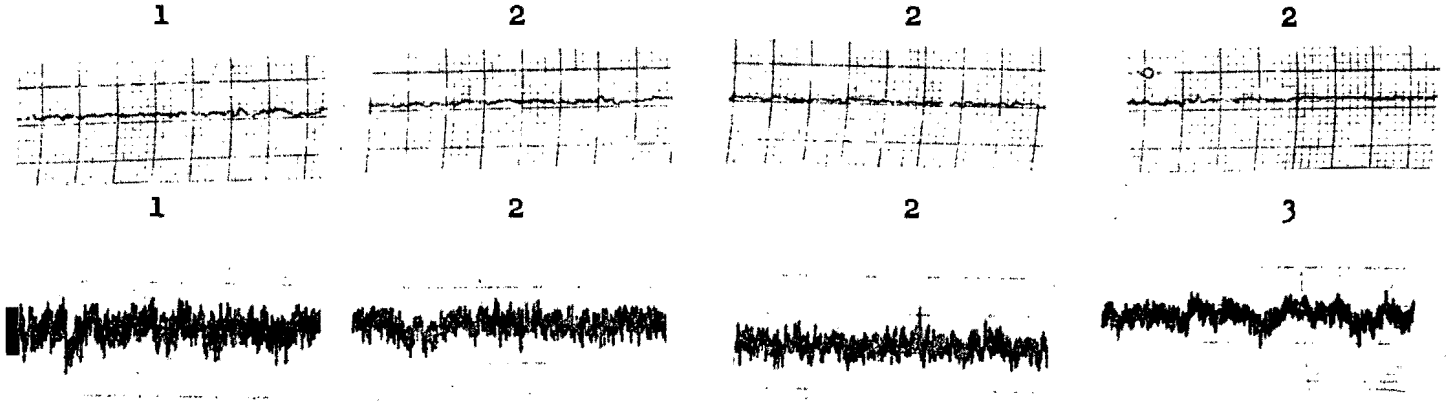
TREATMENT SESSION 1



TREATMENT SESSION 2



TREATMENT SESSION 3



Masseter/...