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PSYCHOPHYSIOLOGICAL REACTIVITY IN MIGRAINE HEADACHE

BY

© KENNETH STEPHEN MEEN

A THESIS

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled Psychophysiological Reactivity in Migraine Headache submitted by Kenneth Stephen Meen in partial fulfilment of the requirements for the degree of Master of Education in Counselling Psychology.

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Charles C. Anderson

Date 7 October 1981

Dedicated to my father, whose frequent admonishments to seek gainful employment served as impetus for the eventual completion of this thesis.

ABSTRACT

This study employed the concept of psychophysiological reactivity in an attempt to further delineate physiological responses to a psychological stimulus, and to relate these responses to the migraine headache response, within the broader context of the individual uniqueness paradigm. Migraine subjects were compared with nonmigraine subjects on measures of basal electromyographic activity, electrodermal activity, digital skin temperature, and the reactivity of these three physiological measures to a psychological stimulus.

The subjects for this study included 39 migraineurs who were recruited from the community, and screened on the basis of headache activity and symptom characteristics. A form of cohort matching, whereby the migraine subjects were required to bring same-sexed, similar aged friends who were screened for absence of headache activity, was implemented to secure a relatively well-matched control group. Each subject was seen individually, for a single one-hour session. Frontalis electromyopotential, palmar electrodermal activity, and digital skin temperature were all recorded through surface electrodes during the four experimental conditions. The first condition was a 15 minute adaptation condition, during which subjects were requested to sit quietly in a reclining chair, keep their eyes open, relax, and listen to soft background music. The second condition (baseline), which was of 3 minutes duration, was similar to the adaptation condition, except that subjects were requested to keep their eyes closed. Then came the stimulus condition, at the beginning of which subjects were instructed to mentally subtract 7 from 1000 in serial fashion, as fast as possible

until told to stop. After 3 minutes came the order to stop, and the subjects were then instructed to relax for 5 minutes at which point they were finished the procedure. This last condition was termed "recovery" condition.

A 2 Group x 4 Conditions ANOVA with repeated measures for conditions was carried out separately for each of the 3 physiological modalities. The only significant effect involving groups was a simple-main effect on conditions across groups, indicating that the migraine group had a significantly higher EMG level than the nonmigraine group in the adaptation condition, but that this difference did not persist in other conditions. It appears that migraineurs have higher levels of muscle tension than nonmigraineurs with eyes open, but not with eyes closed. The linear regression of EMG on time, which was calculated to deal with flux within conditions, showed that the nonmigraine group within the adaptation condition had EMG values which were initially lower than those of migraineurs, but which approached the latter group's value toward the end of the condition.

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Chapter I

INTRODUCTION

Rationale

Headache is one of the most pervasive complaints of mankind. An estimated 50% of the population has suffered severe or disabling headaches at some time in their lives (Ziegler, 1976). Migraine headache is also relatively common, afflicting about 15 to 20% of men, and 23 to 29% of women (Waters and O'Connor, 1975). The Ad Hoc Committee on Classification of Headache (1962) describes migraine as:

> recurrent attacks of headache widely varied in intensity, frequency, and duration. The attacks are commonly unilateral in onset; are usually associated with anorexia and sometimes with nausea and vomiting; in some are preceded by, or associated with conspicuous sensory, motor and mood disturbances; and are often familial.(p. 127)

While the pathophysiology of migraine is not well understood, recent investigations have contributed much to the elucidation of the cause and mechanism of migraine. Migraine may be broached from a number of perspectives including psychological, genetic, biochemical, neurological, vascular and electrophysiological; while advances have occurred in all of these areas, only some aspects of migraine are directly relevant to the present discussion.

It is known that the pain of migraine headache is associated with vascular phenomena, namely the edemous vasodilation of extracranial arteries. Dalessio (1978) indicated that the migraine syndrome is the result of a combination of vasodilation and a sterile inflammation which occurs as a result of the secretion of vasoactive substances at the site of the distended blood vessels. Vasodilation appears to be a

rebound phenomena resulting from the depletion of circulating serotonin, following an initial increase of this substance. Increased serotonin secretion, thought to be the indirect result of endocrine changes, dietary factors, or stress, causes vasoconstriction (Kudrow, 1978). Thus migraine follows a two-step process, the first characterized by vasoconstriction, followed by vasodilation.

There are potentially many factors involved in the etiology of migraine; any factor which initiates cerebrovascular constriction thereby setting of the two-stage mechanism, could be implicated. Bakal (1975) reports that a minority of headaches are caused by factors other than nonspecific psychological stress. A number of investigators are in agreement with this contention, and thus stress is commonly seen as the major trigger of the migraine mechanism. Support for this position is provided by the observation of a correspondence between stressful events and the onset of migraine. For example, Henryk-Gutt and Rees (1973) found that over half of their subjects suffered their first migraine attack during a period of emotional stress. Further, it was found that over half of the attacks of 43 subjects' were coincident with a stressful event. In an early study, Marcussen and Wolff (1949) were successful in provoking migraine attacks by introducing an arousing topic.

Yet stress alone does not appear to be responsible for migraine attacks. Henryk-Gutt and Rees (1973) found no evidence that migraineurs had been subjected to greater levels of stress than nonmigraineurs had. However, the results of psychological tests suggested that migraineurs experienced relatively low stress situations as threatening. As Bakal (1975) has pointed out, the term "stress" cannot be efficaciously used

in any absolute sense. Some situations are perceived as more stressful to some individuals than to others. It appears then, that there is something peculiar about the way in which migraineurs respond to events they find stressful that triggers the two-stage mechanism resulting in migraine headache. As has been suggested in various studies dealing with personality and migraine, there may well be constitutional tendencies that predispose migraineurs to respond more strongly to stimuli than nonmigrdineurs do. Biological theories emphasize various biochemical, genetic, neurological, and vascular morphological and functional aspects of migraine. While both of these types of variables are undoubtedly involved in the etiology of migraine, it appears that many researchers see the crux of the matter centering on the way in which biological mechanisms respond to psychological stimuli. This notion is implicit in stress theories of migraine, and is consistent with the Biopsychological approach promulgated by Bakal (1975) and later by Adams, Feuerstein, and Fowler(1980). Migraineurs may have predilections both biologically and psychologically to respond to stress in their own peculiar manner.

Under the rubric of the Biopsychological approach there are a number of ways in which stress can theoretically trigger a migraine attack. A number of writers have alluded in general terms to the role of stress in the etiology of migraine. Dalessio (1980) indicates simply that the presence of migraine is a sign that the individual is under stress, and that relief from anxiety is a legitimate form of treatment. Kudrow (1978) believes that stress results from anticipation or from an adaptation effort. Yates (1980) sums up this position perspicuously:

> migraine sufferers are characterized by a high degree of autonomic lability which results in objectively low stress situations being reacted to as threatening, triggering sympathetic activity in the autonomic nervous system. (p. 427)

The concept of heightened arousal in the sympathetic branch of the autonomic nervous system is consistent with psychological studies of migraine, and offers a potential explanation for migraine onset. Appenzeller (1976) has suggested a biochemical connection between sympathetic over-arousal and migraine attacks. An indirect empirical connection between migraine and sympathetic arousal has been implied as a result of the success of treatment programs directed at decreasing sympathetic activity through the promotion of various relaxation techniques (eg. Sargent et.a, 1980). In some cases relaxation was confounded with increases in skin temperature, which in themselves were thought to have a direct therapeutic physiological effect on the vasculature (Tarler-Benlolo, 1978).

Whether stress results in generalized sympathetic arousal which in turn sets off the migraine attack, or whether it is primarily the activation of specific vascular responses that results in headache is not entirely clear. The latter explanation is attractive by virtue of the known vascular involvement in the migraine mechanism. This situation is further confused by the fact that vascular responses are part of generalized sympathetic arousal as well as part of the orienting response (O.R.) and the defensive response (D.R.). For example, Boudewyns (1976) found that skin temperature decreased when normal subjects were exposed to stressful conditions. Decreases in skin temperature are directly related to decreased blood flow in tissue (Taub, 1977). Various vascular

phenomena observed in migraineurs tend to bolster the specific vascular response concept of migraine etiology. A number of studies indicate that the vascular responses of migraineurs to various physical stimuli are different from the responses of non migraineurs (eg. Appenzeller, Davison, & Marshall, 1963; Downey and Frewin, 1972). More important to the present discussion are findings of atypical responses to psychological stimuli; these studies are psychophysiological in the sense that they focus on physiological responses to environmental stimuli. They are directly related to the question of how stress causes migraine. One such study by Price and Tursky (1976) found that migraineurs were unable to comply with the demand that they produce digital vasodilation, while nonmigraineurs were able to comply. Migraineurs instead tended to constrict or show no change over time. This tendency was even more marked for temporal artery blood volume changes. The authors note that this is an example of how migraineurs respond to demanding situations. This tendency to vasoconstrict could be analogous to the initial vasoconstriction of the two-stage headache mechanism.

Price and Tursky's results are profound in that they demonstrate the effect of arousal on vascular responses. However, the question of whether this was a specific vascular response or just an aspect of general arousal is unanswered. Results of an experiment carried out by Bakal and Kaganov (1977) indicate that migraineurs respond with stronger EMG values than did either a group of tension headache subjects, or a group of control subjects. Pulse velocities were greater for the two headache groups than for the controls. These findings do not support the notion that the vascular response per se, apart from physiological

indices of arousal in other modalities is responsible for migraine attacks.

The orienting response (O.R.) and defensive response (D.R.) are both aspects of autonomic arousal and are convenient constructs for providing conceptual continuity between environmental stressors and physiological responses. In this respect the O.R. and D.R. provide ways in which individuals' responses to environmental stressors can be related to the etiology of migraine. As Cohen (1978) has indicated, claims that migraineurs have unstable autonomic nervous systems are not well documented. The relationship between stress and migraine is not clearly understood, but the few observations of physiological responses to various psychological stimuli that are documented have helped further define that relationship.

The theory of Response Specificity speaks directly to the issue of differential activation of physiological modalities in response to environmental stimuli. At one extreme, general activation theories hold that the sympathetic nervous system responds in a diffuse, undifferentiated manner, whereas at the other extreme lies the theory that various systems are highly dissociated (Duffy, 1972). Response Specificity is more consistent with the latter than the former, while the concepts of O.R. and D.R. tend to be more closely related to the former. "Individual uniqueness" is a special case of Response Specificity referring to the tendency of subjects with similar characteristics or symptoms to respond differently from subjects without those special characteristics (Engel, 1972). A synonymous term is "symptom specificity." This theory would suggest that migraineurs are more susceptible to stress - induced

activation of the physiological mechanism associated with that complaint (Cohen, Rickles, & MacArthur, 1978). Thus one would expect migraineurs to be more reactive to stress in the vascular system that nonmigraineurs would be. Support for this theory was provided by Moss and Engel (cited in Engel, 1972) who found that patients with hypertension reacted more strongly in blood pressure than did arthritics, who reacted more strongly in the muscles of symptomatic joints.

Cohen, Rickles, and MacArthur, (1978) investigated response stereotypy in migraineurs, and found that they responded more uniformly to different stimuli in six different psychophysiological measures than did nonmigraineurs. Their experiment did not look for individual uniqueness or symptom specificity; rather they compared the two groups' responses across a number of tasks. Stoyva (1979) advocates the use of "stress profile"as an initial step for patients about to receive general relaxation training. The profile consists of an initial baseline period, followed by the introduction of a mental stressor, followed by a period of relaxation. Stoyva indicates that migraineurs tend to show a stronger vasoconstriction response than other responses, and alternately that tension headache patients respond with stronger muscle tension of the head muscles. This observation seems to be tantamount to symptom specificity. He goes further to say that after the cessation of the mental stressor some individuals show slower recoveries in one or more of the physiological measures, and that such tendencies can be related to the particular stress-related disorder which an individual is suffering from. Unfortunately this author does not provide data to support his observations.

Purpose of the Study

The purpose of this study was to examine psychophysiological data and compare migraineurs with nonmigraineurs within the context of the "individual uniqueness" or "symptom specificity" paradigm. More specifically, this study was directed toward comparing migraineurs' and nonmigraineurs' physiological responses to a demand situation, and the subsequent recovery of these responses, with the intent of clarifying the mechanism by which stress leads to migraine headache. The following questions were asked:

1. Do migraineurs differ from nonmigraineurs on basal levels of electromyopotential (EMG), digital skin temperature (DST), or electrodermalpotential (EDR)?

2. Is there a difference between migraineurs and nonmigraineurs in the response of the three physiological measures (EMG, DST, & EDR) to a demand situation?

3. Is there a difference between migraineurs and nonmigraineurs in the recovery of the three physiological measures (EMG, DST, & EDR) after cessation of the demand situation?

4. Does the relationship between the three physiological responses (EMG, DST & EDR) appear to be different for migraineurs in comparison to nonmigraineurs?

Chapter II

REVIEW OF RELATED RESEARCH

Just as migraine headache is multifaceted, so is the research which seeks to apprehend this painful malady. Migraine is at once a psychological, genetic, biochemical, neurological, vascular and electrophysiological event; while all of these factors are pertinent to migraine etiology and treatment, and research exists in all of these areas, there remains much to learn. In fact recent thinking has cast serious doubts upon some contemporary conventionally monolithic conceptualizations of migraine headache, and consequently has led to a host of new unanswered questions. One such question entreats the clarification of the relation of stress to the etiology of migraine, and has biological psychological and biopsychological elements. In this chapter each of these three elements serves as a heading, under which relevant research is organized and discussed. Preceding this is a definition of migraine with a description of the various types.

Definition of Migraine

The Ad Hoc Committee on Classification of Headache (Friedman et.al., 1962) describes migraine as:

> recurrent attacks of headache widely varied in intensity, frequency, and duration. The attacks are commonly unilateral in onset; are usually associated with anorexia and sometimes with nausea and vomiting; in some are preceded by, or associated with conspicuous sensory, motor, and mood disturbances; and are often familial. (p. 127)

Migraine headache is then subdivided into five subtypes, each of which shares some, but not all of the features mentioned above. In "Classic" migraine, the headache is preceded by a prodrome which consists of

transient visual and other sensory or motor manifestations. "Common" migraine has no prodrome, is less often unilateral, and is often associated with various environmental, occupational, or menstrual variables. "Cluster" headache is typically unilateral; brief in duration; occuring frequently for a period of time, followed by a long period of remission. "Hemiplegic" and "Opthalmoplegic" headache are featured by persisting sensory and motor manifestations. "Lower-half" headache is centred primarily in the lower half of the face (Friedman et.al., 1962). Some authors have indicated that, while these subdivisions of migraine into various types can be convenient, often "pure" cases are infrequent, and often the more general term is used (eg. Dalessio, 1980).

Biological Aspects

The cranial vascular events that accompany migraine, and their part in the migraine mechanism, have been recognized for some time now. That migraine is referred to as "vascular" headache is testimony to the perceived importance of these vascular phenomena. Numerous studies have demonstrated increased blood flow in intra and extracranial arteries during the headache period. Skinhoj (1973) found significant hyper-fusion of blood in the internal carotid area during migraine, but a reduced perfusion during the prodromal phase. Skinhoj further posits that angiographic studies indicate that the basilar artery is also involved. O'Brian (1967) was among the first to document changes in cerebral flow during migraine, and while his early experiments found decreases in blood flow during migraine, later experiments found increases. O'Brian also measured reductions in blood flow during prodromata.

In a review of the haemodynamics of migraine, O'Brien (1973) concluded that "the headache stage is associated with vasodilation of both cerebral and noncerebral cranial vessels" (p. 162). He also indicated that the prodrome is associated with vasoconstriction, although the latter is not necessarily the cause of the former. These findings, along with similar findings by many other investigators, provide support for the classic two-stage theory of migraine, which holds that the vasodilation phase is a homeostatic rebound response to initial vasoconstriction, and that edemous vasodilation is responsible for the severe pain of migraine.

One of the earliest studies implicating vascular events in migraine etiology (Tunis and Wolff, 1953) found that the temporal artery was significantly larger in a headache-free migraine group than a non-headache group. Further, the artery was larger during headache than during a headache-free period. These authors also found that the migraineurs "exhibited greater variability of the contractile state of the observed vascular bed" (p. 557). Tunis and Wolff also reported that variability in the contractile state of parts of the cranial vasculature was modified in association with mood alteration, feelings of tension, sustained effort, and restlessness. The authors concluded that the vascular events were related to sustained adaptive reactions to stress. While the observations are not detailed, nor are the circumstances under which they were made, this presumably direct connection between life stress and cerebral vascular events is a rare and provocative observation.

Some recent studies suggest that the relationship between migraine and cerebral blood flow is more complex than previously thought. For example, Edmeads (1977) found discrepancies between "spatio-temporal

distribution of blood flow and the distribution of symptoms, and of the disordered cerebral vasoreactivity during attacks" (p. 151). He concluded that the vascular theory of migraine etiology is correct but incomplete. Blau (1978) goes further than this and challenges the classic two-stage theory of migraine. He suggests that migraine symptoms are not due to extracranial arterial vasodilation, but rather are due to calibre variation in the leptomeningeal circulation. Blau further posits that some migraineurs are dilators and some are constrictors. This line of thought appears to have received little follow-up, although Dalessio (1980) has recently pointed out the lack of correspondence between alterations in blood flow and migraine symptoms. It appears then that a general, but not perfect relationship exists between cerebral blood flow and migraine.

The theory of vascular etiology of migraine has been extended by some authors who have suggested that migraine is a generalized vasomotor dysfunction (eg. Kudrow, 1978). Downey and Frewin (1972) found that migraineurs' vascular response in the hands to a cold stimulus was less than in normals, but that migraineurs had higher basal blood flows. They concluded that their results are consistent with the notion of generalized vasomotor dysfunction. Appenzeller, Davison, and Marshall (1963) provided further evidence that migraineurs have an abnormal vascular temperature response in the hand, and concluded that an abnormality in the control of blood flow exists. However, this theory is not without critics. Morley (1977) reviewed the evidence and concluded that there was not one study in support of this theory that did not suffer from serious methodological flaws. Hence, while general vasomotor instability may exist in

migraineurs, it has not yet been unequivocally demonstrated. Some authors have postulated that a general instability of the autonomic nervous system is responsible for unstable central vasomotor control mechanisms which lead to migraine (eg. Dalessio, 1980). This hypothesis is in need of verification.

Biochemical factors appear to have a major role in the etiology of migraine. While a detailed account of particular biochemical events associated with migraine is beyond the scope of this discussion, a general account follows. As Blau (1978) indicated, in addition to vascular or neural mechanisms, a hormonal element must be involved; while the emphasis has been on vascular aspects of migraine, vascular and biochemical events are inextricably intertwined, and hence there is no good argument against conceptualizing the migraine mechanism as primarily biochemical in nature. Biochemical factors may well affect the extent of vasomotor reactivity, and have a major part in determining whether or not an attack occurs (Blau, 1978). For example, migraine is often associated with menses. Dalessio (1980) indicated that vascular dilation alone is not painful, and that it is the combination of vasodilation and the presence of certain biochemical substances in the blood vessel walls, resulting in a sterile inflammatory reaction which creates the pain. These substances also influence local vasomotor control, and thus are compatible with the vascular theory of migraine etiology. Such substances include amines (serotonin, catecholamines, histamine, tyramine), vasoactive polypeptides (bradykinen, angiotensin), lipids (various free fatty acids, prostoglandine), and hormones (oestrogens, progestogens) (Anthony and Lance, 1975).

Serotonin is one vasoactive substance which has received special attention because of its implication in migraine onset. An increase in free plasma serotonin causes vasoconstriction, and has been shown to correspond with various preheadache phenomena such as prodrome and mood changes. Some of this substance is thought to become sequestrated into the vessel wall causing sterile inflammation and pain. Sudden withdrawal of serotonin (due to depletion and metabolization) and lose of its vasotonic influence, leads to vasodilation of the cranial vessels (Anthony and Lance, 1975). Dalessio (1980) found a concurrent increase in neurokinin and protease, both of which are strong vasodilator substances, and which also affect a reduction in the pain threshold. Dalessio indicated that these substances are secreted as a result of neuronal excitation "in a variety of circumstances" (p. 108). Thus, while investigators are delineating biochemcial mechanisms associated with vasodilation and pain, it is still unclear precisely how this chain of events is initiated, even though it is known that these events are associated with increased sympathetic nervous system activity (Dalessio, 1980). Appenzeller (1976) has suggested a biochemical connection between sympathetic over-arousal and migraine attacks.

Psychological Aspects

Various theories have been spawned that impute various roles to psychological factors as they relate to migraine etiology. Under the rubric of psychosomatic medicine exists the theory that particular personality characteristics are associated with particular psychosomatic disorders. Friedman (1976) indicated that the "personality structure of the migraine patient has been described as compulsive, with outstanding

performance due to the emphasis on perfectionism, overconscientiousness, and ambition" (p. 71). Friedman further reported that observations indicate "that hostile impulses are basic to neurotic conflicts in patients, and that these individuals rechannel their hostilities into ambitious striving for success" (p. 72). This description of the migraine personality occurs with ubiquity in the literature. Bakal(1975) adds that primary trait is repressed hostility, which is often the consequency of situations which produce resentment and rage, and which neither be acknowledged nor expressed. While similar personality traits have been often reported, according to Harrison (1975) many of these findings are based on surveys which lacked basic experimental controls.

In his review of psychological testing and headache, Harrison (1975) concluded that the results of surveys with adequate controls reveal that, as a group, migraineurs do not differ from controls on I.Q. scores. Migraineurs were found to have increased scores on the Hypochondriasis and Hysteria scales of the MMPI, but as others have pointed out (eg. Adams, Feuerstein, and Fowler, 1980), these scores would normally be elevated for subjects who experience pain. Harrison also suggested that elevated scores on the Psychoasthenia scale were observed, thus indicating obsessive - compulsive tendencies. He further concluded that migraineurs scored higher than nonmigraineurs on scales measuring "well-defended neurotic anxiety" (p. 183). Studies of tests measuring patients' self-perceptions indicate that "headache patients think poorly of themselves" (p 183). While numerous clinical studies and anecdotal reports have identified unexpressed anger and hostility as prototypic migraine characteristics, Harrison indicated that the results of well-

controlled studies are not clear in this regard. He concluded that migraineurs perform differently from nonmigraineurs on psychological tests, but rightfully warned against accepting any particular etiological hypothesis on the basis of these observations.

A variation of the theory that particular personality traits give rise to particular psychosomatic complaints is the theory of emotional specificity which holds that specific physiological response patterns are elicited by specific emotional patterns (Adams, Feuerstein, and Fowler, 1980). A failure to find support for specificity theories such as this one, has led to their general rejection (Grinker, 1973). The mechanisms by which certain traits or emotions lead to migraine have seldom been adequately articulated.

Graham (1972) has written of an inherent difficulty in statements of psychological causation: such statements can mean that either an internal psychological state or an external psychological stimulus is responsible for the physical consequence. When the focus is on a psychological state, Graham maintains that questions of psychosomatic etiology are virtually impossible to answer, largely due to the fact that both the psychological state and the physical state are observed together, thereby precluding the designation of which is cause and which is effect. Further, if a psychological stimulus is shown to produce a physiological response, psychological states cannot be inferred from the stimulus, as they may well be the consequence of the physiological response. Recognition of these difficulties has led some investigators toward attempts at discovering those responses which are due to psychological stimuli, rather than attempting to deal with psycho-

logical states (Graham, 1972). Thus the more fruitful investigations more clearly delineate the stimulus.

Focussing on the association between situational variables and migraine avoids the problems of dealing with psychological states, and has helped to identify some common precipitators of migraine headache; in these cases the onset of headache was observed as a response to certain external stimuli. For example, Henryk-Gutt and Rees (1973) found that over half of their subjects suffered their first migraine attack during a period of emotional stress, and further, that over half of the attacks of 43 subjects [redacted] coincident with a stressful event. In an early study, Marcussen and Wolff (1949) were successful in provoking migraine attacks by introducing an arousing topic. Another situational psychological stimulus which appears to result in headache is relaxation following a sustained effort, or the "let-down" phenomena, for which there appears to be no adequate explanation. Thus migraine often occurs on weekends and the first day of a holiday (Anderson, 1980). BaRal (1975) lists noise, bright lights and lack of sleep as other precipitating stimuli, plus some situational factors which are ostensibly not psychological, such as specific dietary factors and alcohol ingestion.

While stress is a situational variable that innumerable investigators have implicated as the primary stimulus for migraine headache, this concept suffers serious shortcomings which tend to undermine attempts to investigate its etiological role. Apart from generating correlations between stressful events and migraine, studies dealing with stress typically do not further our understanding of the way by which

stress leads to migraine, because the term "stress" obfuscates the relationship between stimulus and response. The pervasive statement about stress is exemplified by Anderson's account, which states generally that migraine attacks tend to be associated "with responses to external or intrapsychic stresses" (1980, p. 416). Stress is thought to trigger sympathetic arousal which sets off the migraine mechanism (Yates, 1980). Unfortunately, there appears to be no evidence showing that migraineurs are sympathetically overaroused (Bakal, 1975), nor is there evidence that migraineurs are subject to more stress in their lives (Henryk-Gutt and Rees, 1973). That therapies aimed at reducing sympathetic arousal by inducing relaxation are successful, does not show that migraineurs were initally more aroused than nonmigraineurs. However, Friedman (1976) indicated that it is the significance of the stressful event rather than the stress itself that precipitates migraine. Bakal (1975) indicated that "stress" cannot be used as a specific term, as "any stimulus situation is capable of acquiring stressful properties" (p. 375). Thus, as Malmo (1972) points out, "overdependence on the concept of 'stress' makes one neglect the 'stimulus side' of the problem" (p. 968). While the relation of stress to migraine is an interesting observation, it is of limited utility in broaching the question of <u>how</u> stress leads to migraine, and thus contributes little to the elucidation of the migraine mechanism. The nature of the stress and the resulting physiological changes must be defined with greater precision (Malmo, 1972). In this way the important intermediate events can be studied. Bakal (1975) has advocated a "biopsychological" approach which would use established constructs of psychophysiology to explicate

relationships between stimuli and physiological responses, and thereby provide the precise data that is capable of enhancing our understanding of migraine.

Biopsychological Aspects

Both biological and psychological factors are undoubtedly involved in the etiology of migraine, and migraineurs may have predilections both biologically and psychologically to respond to stress in their own peculiar manner. However, neither of these two aspects of migraine has successfully explained the headache response in its entirety. Bakal (1975) has suggested that a synthesis of models is required in order to further specify the physiological properties of migraine. This synthesis would allow specific physiological responses to well-defined psychological stimuli to be quantified. There are a number of psychophysiological constructs which can potentially further our understanding of the headache response.

The notion of the "orienting response" (O.R.) is one psychophysiological concept which can tie specific physiological responses to simple stimuli. Bodily changes occurring as part of the O.R. include increased muscle tone, increased electrodermal activity, vasoconstriction in the limbs, and cephalic vasodilation (Beatty, 1975). It has been suggested that the two-stage vascular mechanism of migraine headache might be set off by an exaggerated O.R. (Adams, Feuerstein, and Fowler, 1980). The logic of how this would occur is not clear however, as the two-stage mechanism should begin with cephalic vasoconstriction. The cephalic vascular component of a "defensive" or "startle" response (D.R.) is vasoconstriction (Beatty, 1975), and hence would be more likely to

precipitate the two-stage vascular mechanism of migraine. Muscle tension, peripheral vasoconstriction, and electroder[redacted]tivity react the same in the D.R. as in the O.R. If this theory were applicable to migraine elicitation then, one might expect migraineurs' D.R.s to be more intense than those of nonmigraineurs. Or, in view of the data on psychological traits, perhaps migraineurs would show a D.R. in some situations which would elicit only an O.R. in nonmigraineurs.

The O.R. and D.R. paradigm has been extended to include patterns of physiological responses (Lacy & Lacy, 1958). While there is some confusion in the literature over what particular term applies to what particular pattern, the terminology of Sersen, Clausen, and Lidsky (1978) is adhered to here:

> Stimulus-response <u>specificity</u> refers to the tendency for different stimuli to produce unique patterns of autonomic responses. In contrast, individual response <u>stereotypy</u> reflects a tendency of the individual to display an idiosyncratic response pattern to all stimuli. (p. 60).

Cohen, Rickles and MacArthur (1978) investigated response stereotypy in migraineurs by having a group of migraineurs and a group of control subjects participate in seven different tasks while six different physiological responses were measured. Direct differences between migraineurs and nonmigraineurs were observed on EMG levels, which were lower across all tasks for migraineurs than for nonmigraineurs. Migraineurs also had warmer hand and head temperatures than nonmigraineurs. While these authors obtained many significant interaction effects for groups, tasks, and physiology, they did not perform the post-hoc tests which would be required to show differences between groups on physiological reactivity to stimuli. However, using nonparametric rank tests on

range corrected data, they found that the migraine group responded with more stability across the physiological measures than did the non-migraine group. That is, the difference in stressors had less effect on migraineurs' response patterns than on those of the control group subjects; migraineurs' physiological responses showed fewer shifts in rank order across stimuli than did those of nonmigraineurs. They concluded that the stability of migraineurs' patterns is evidence for response stereotypy. One explanation which was offered for this finding was that a rigid response pattern is an adaptation to keep the unstable physiological system under control.

A special case of response specificity is "individual uniqueness", which refers to the tendency of subjects with similar characteristics or symptoms to respond differently from subjects without those special characteristics (Engel, 1972). A synonymous term is "symptom specificity." While general activation theories indicate that the sympathetic nervous system responds in a diffuse, undifferentiated manner (as in the O.R.), the symptom specificity hypothesis prefers that individuals with a particular psychosomatic complaint are more susceptible to stress-induced activation of the physiological mechanism associated with that complaint (Cohen, Rickles, and MacArthur, 1978). Thus migraineurs would be expected to display greater reactivity to stress in the vascular system than nonmigraineurs would be. Support for this theory was provided by Moss and Engel (cited in Engel, 1972), who found that patients with hypertension reacted more strongly in blood pressure than did arthritics, who reacted more strongly in the muscles of symptomatic joints. The individual uniqueness theory tacitly underlies certain medical diag-

noetic tests (Engel, 1972), and in this respect could benefit practitioners faced with the difficult task of diagnosing migraine headache.

To date, it appears that increased vascular reactivity, within the context of the individual uniqueness hypothesis, has not been reported in a migraine population. Sturgis (1979) studied reactivity in migraineurs, tension headache patients, and normals and found no significant differences between groups. This one aspect of her study appears to be conceptually similar to the individual uniqueness approach, except that the stimuli consisted of 15 successive slide presentations, which were treated as one 15 level dimension of the design. This large number of levels introduces a complexity that could be a detriment to a study designed primarily to look at individual uniqueness. Sturgis' study is also not entirely clear with respect to which comparisons were made. However, she did find that migraine and muscle tension headache subjects failed to habituate in the digital blood volume pulse response over a two session period, whereas normals did habituate. Sturgis concluded that further research would be needed to determine if the failure to habituate was a measure of true reactivity.

Bakal and Kaganov (1977) compared migraineurs and muscle contraction headache patients on measures of superficial temporal artery pulse velocity responses, and E.M.G. responses to nonaversive auditory stimuli, during which the subjects were required to do nothing. They found no statistically significant differences between the two groups on reactivity. This aspect of this study addresses the issue of individual uniqueness, in that it sought to find differences in physiological

responses between two groups of subjects, each with a particular symptom. An interesting observation of these authors' was that migraineurs had higher baseline E.M.G. values than either controls or muscle contraction headache patients. This would not be predicted by theories of symptom specificity, as the musculature is ostensibly not the physiological modality which underlies migraine symptoms. In view of Bakal and Kaganov's conclusion regarding the apparent lack of features distinguishing migraineurs from muscle-contraction headache patients, it appears that individual uniqueness might be more readily observed in a comparison between migraineurs and nonheadache control subjects. Other investigators have questioned whether migraine and muscle-contraction headaches can really be differentiated (eg. Ziegler, 1979).

Price and Tursky (1976) compared migraineurs to nonmigraineurs in a study which required subjects to attempt to induce digital and cephalic vasodilation using biofeedback instruments. While nonmigraineurs were able to vasodilate, migraineurs instead showed marked vasoconstriction, particularly in temporal arteries. Thus it appears from this study that activation of vascular responses occurred for migraineurs only. The authors concluded that migraineurs reacted adversely to a demand situation. This study did not address other physiological modalities, and consequently it is unknown how the two groups might have compared on these measures.

Stoyva (1979) claims to have observed symptom specificity in his use of "stress profiles." The profile consists of an initial baseline period, followed by the introduction of a mental stressor, followed by a period of relaxation. Although no data is provided, Stoyva indicates

that migraineurs tend to show a stronger vasoconstriction response than other responses, while tension headache patients respond with higher levels of tension of the head muscles. Stoyva further indicates that some individuals show sustained activation of the physiological system that is related to the particular stress-related disorder from which the individual is suffering. These are provocative observations which beg to be verified. Thus, while a number of investigations have alluded to psychophysiological aspects of migraine that address etiological questions, it appears that further research is warranted.

Chapter III

METHOD

This chapter contains specific information regarding the selection of subjects, the experimental apparatus and setting, and the procedure which was followed. Following is the identification of experimental variables and an explanation of the data reduction. The final section relates the experimental design parameters and provides a general overview of the statistical analyses.

Subjects

The experimental subjects consisted of 39 migraine sufferers and a quasi-matched sample of 39 nonmigraine sufferers. The migraineurs were recruited from the community by means of newspaper and television announcements, and were required to fulfill various characteristics. They were between 18 and 55 years of age, and had a minimum 2 year history of headache with a frequency of at least one per month. With the exception of oral contraceptives, they could be on medication as long as their headaches remained uncontrolled. In order to eliminate subjects whose headaches were clearly of a nonvascular type, only subjects whose headaches were unilateral in onset were included. Further, subjects were required to have 3 of 4 symptoms: nausea or vomiting usually accompanying headache, positive family history of migraine, independent diagnosis of migraine by a physician, sensitivity to light usually accompanying headache.

The initial migraine subject pool consisted of over 70 individuals, each of whom was asked to bring a same-sexed friend of within 5 years of

the subject's age, who neither currently nor in the past suffered from migraine headaches. Further criteria for inclusion into the non-migraine control group included: a maximum of 1 headache per month, not unilateral in onset, able to continue with a relatively normal routine during headache, headache responsive to non-prescription analgesics or no medication required. Of the inital subject pool, 32 were able to find a matched individual who fulfilled the control group criteria. Eight more control subjects were solicited by the experimenter and were matched with migraine sufferers. From this group of 40 matched pairs, one was dropped because of spoiled data, leaving the final count at 39 matched pairs.

Table 1 reports more complete information on the 2 samples. The groups contain identical proportions of male to female, each with 9 of the former and 30 of the latter. The mean age for the migraine group is 37.1 years with a standard deviation 9.20, while the mean age for the nonmigraine control group is 36.44 years with a standard deviation of 9.57. The difference between the mean age of the 2 groups is not statistically significant ($t=.31$, $p > .05$). The mean headache frequency per year of the migraine group is 42 with a standard deviation of 20.56, while the mean headache frequency per year of the nonmigraine control group is 6.10 with a standard deviation of 5.87. The difference between the mean frequency of the two groups is statistically significant ($t=10.49$, $p < .01$), with the migraine group having significantly more headaches per year.

Apparatus

Experimental Setting. All experimental procedures were carried

Table 1

Age, Sex, and Headache Frequency per Year

for Each Experimental Group.

GROUP	SEX		AGE IN YEARS		HEADACHE FREQUENCY PER YEAR		N
	M	F	$\bar{X}$	S.D.	$\bar{X}$	S.D.	
Migraine Group	9	30	37.1	9.20	42.0	20.56	39
Matched Control Group	9	30	36.44	9.57	6.10	5.87	39
Both Groups	18	60	36.77	9.33	24.05	23.49	78

out in a quiet room, the decore of which was intended to promote relaxation. Lighting was soft and indirect, and homey pictures and posters adorned the walls. A tape-recording of soft, classical music was played throughout the experimental sessions. The subjects were seated in an upholstered reclining chair. The temperature of the room was maintained within the range 21.5 °C to 23.5 °C.

Physiological measures. Digital skin temperature was monitored with a Biofeedback Technology model 302 electronic thermometer. The thermometer was taped to the volar surface over the second phalanges of the non-dominant middle finger. Integrated electromyographic activity of the frontalis muscle was measured with an Autogen Systems Incorporated model 1700 electromyograph. The bandwidth was set at the 400 to 500 Hz position. The electrodes were of the Ag/AgCI, triple contact type. They were placed centrally on the forehead, 2 inches apart; 1 inch above the eyebrows. Digital electrodermal activity was monitored with an Autogen Systems Incorporated model 3400 Feedback Dermograph connected to triple contact type electrodes. The ground electrode was attached to the volar surface over the first phalanges of the dominant middle finger. The two active electrodes were similarly attached, one each to the index and ring finger of the same hand. The 3 instruments were connected through Autogen Systems Incorporated model I-5000 optical isolators to an Autogen Systems Incorporated model 5600 Data Acquisition Centre. The data were printed on an Autogen Systems Incorporated model P-5000 tape-printer.

Procedure

This experiment was preliminary to a parent project investigating

treatments for migraine headache. As the migraine group were to carry on with treatment and further study, they had previously attended a seminar in which information was gathered and they had received a general orientation to their impending treatment programme. The proceedings of the seminar were independent of this current experiment, and the two were carried out in different facilities. During the experiment the explanations (rationale) given to the two groups contained minor differences in order to make them applicable to each group. There is no obvious logical reason to suspect that the slight differences in treatment received by the two groups led to any spurious systematic variance between the two. These differences are given further consideration in the following paragraphs and may be examined in detail in the appendices.

The experimental procedure was conducted in one 30 minute session for each subject, and all subjects were run individually. Upon arriving at the room in which the experiment was to take place, the subject was directed to the reclining chair. Nonmigraine subjects were required to complete a brief questionnaire (Appendix A). Migraine subjects had filled out their questionnaire (Appendix B) at a previous meeting. Following this the subject was read the explanation of the study (Appendix C), and connected to the 3 devices monitoring activity in the 3 physiological systems. The subject was not allowed access to any feedback from the monitoring devices. The subject was then read the instructions (Appendix D) which directed him to sit quietly with eyes open and listen to the background music. Following this 15 minute adaptation period the subject was instructed to sit with his eyes closed

(Appendix E). This began the 3 minute baseline period. After this period the subject was instructed to mentally subtract serial 7's from 1000 as fast as possible until told to stop (Appendix F). After 3 minutes the subject was told to stop and report his answer. The recovery period then began, at the beginning of which the subject was instructed to relax and listen to the music without interruption for 5 minutes (Appendix G). After the initial 15 minute adaptation period subjects kept their eyes closed throughout the remaining conditions.

The investigator then explained to the subject the meaning of the data which were recorded on the tape. The output displays of the instruments were then shown to the nonmigraine group, who then received a brief 5 minute talk on biofeedback and were allowed to briefly work with the instruments.

Data Reduction

Digital electrodermal activity was measured in micromho units of conductance, digital skin temperature was measured in degree Fahrenheit, and frontalis electromyographic activity was measured in microvolts. Where appropriate, degrees Fahrenheit were converted to degrees Celsius. Under the adaptation condition these three physiological measures were sampled for 10 seconds once per minute. Under the remaining 3 conditions (baseline, mental arithmetic, and recovery) they were sampled for 10 seconds, three times over each minute. Each data point consisted of an average value of the 10 second input signal. The Data Acquisition Centre was programmed to calculate this value and print it out automatically as the data were being collected. The standard deviations of each data point were also automatically calculated. The mean and

standard deviation values were the raw data used in all analyses.

Research Design and Statistical Analyses

This study is of the mixed design type involving a comparison between 2 groups under 4 conditions. The independent measure is a 2 level classification variable on the basis of which the 2 groups were formed. The 4 conditions (adaptation, baseline, stimulus, recovery) comprise the 4 levels of the dependent variable. Each of the 3 physiological modalities represents a separate dependent variable. This study may be viewed as three separate 2 groups X 4 conditions experiments, as the data for each modality are analyzed independently of that of the other modalities. Thus the design consists of three separate 2 factor experiments with 2 levels on one factor and 4 on the other. Since data were acquired from the same subjects under all 4 conditions, the second factor has repeated measurements.

To assess groups and conditions effects a 2 X 4 analysis of variance with repeated measures on conditions was calculated for each physiological modality. Each data point consisted of a mean score of all measures taken within each condition for each subject. Because this analysis did not deal with trends across time within conditions, a series of linear regression equations were derived to describe the average change of each physiological variable over time within each condition. All data points within each condition were preserved in this latter analysis.

Chapter IV

RESULTS

Chapter IV contains the results of all statistical analyses. First the analyses which delineate between groups and conditions comparisons are presented, followed by appropriate post hoc tests. Following this a method for explicating general trends within conditions is discussed and the results presented.

Analyses of Variance

The major analyses were carried out with three separate 2 groups (migraine, nonmigraine) x 4 conditions (adaptation, baseline, stimulus, and recovery) analyses of variance with repeated measures (ANOVAR) on conditions. One ANOVAR was done for each of the 3 physiological modalities: digital skin temperature (DST), electromyopotential (EMG), and electrodermal response (EDR). The cell values consist of the mean score of all measures taken within a particular condition. For example, the 15 measures within condition A for each subject were used to calculate a single mean value for this condition. The results of these analyses appear in Table 2.

The analysis of the EMG data yielded a significant conditions effect, F (3,228)=27.19, $p< .01$. Scheffé post hoc comparisons (Appendix H) indicated that the value within the adaptation condition was significantly greater than the values within the remaining 3 conditions. A significant groups x conditions interaction was also observed, F (3,228)=4.31, $p< .01$. A Scheffé post hoc test of simple-main effects on conditions across groups indicated that migraineurs had

a significantly higher EMG level than nonmigraineurs on condition A (adaptation), but that this difference did not persist on other conditions (Figure 1, upper graph). There were no other significant effects for the EMG data.

The analysis of the DST data yielded no significant results. Although the mean values within conditions were consistently higher for nonmigraineurs in comparison to migraineurs, this difference failed to reach significance (Figure 1, middle graph). For groups, F (1,76)= .047, p= .828; for conditions, F (3,228) = 1.66, p= .176; for groups x conditions interaction, F (3,228) =.156, p= .926.

The analysis of the EDR data yielded a significant conditions effect, F (3,228) = 44.93, p <.01. Scheffé post hoc comparisons indicated that the mean values within conditions A and B were significantly smaller than the mean values within conditions C (stimulus) and D (recovery). While the values for the migraine group were consistently lower than the values for the nonmigraine group, this difference failed to reach significance (F (1,76)= .987, p= .324). There were no other significant effects for the EDR data.

Linear Regression Analyses

The rationale for using Linear Regression analyses was to compensate for information about trends within conditions that was not considered in the ANOVAR. An important aspect of the data is that changes occurred across time; while the ANOVAR dealt with static measures and gave rise to inferences about the net conditions effect, they did not delineate or describe the flux within conditions. Thus, linear regression analysis was implemented to help illustrate the trends

within conditions. Since the object was to provide an average statement about the change in particular physiological measures with change in time, in all cases the regression of each physiological variable on time was computed. The physiological values served as dependent measures. Separate regression equations were derived for each group within each condition. These calculations were intended to serve as a descriptive adjunct, and are treated as such; no tests of significance are forthcoming. Comparisons between slopes have meaning in the sense that a larger slope is indicative of a greater rate of change, and that different signs indicate different directions in the relationships. Differences which exist between groups in the distance of the regression lines from the abscissa are perfectly linear and constant within each condition, and as such, reflect data similar to that which was dealt with in the ANOVAR. Thus Y-intercept values are not given direct consideration.

EMG Responses. Figure 2, part A displays the linear regression equations of EMG responses on time. Examination of each condition shows that the slopes are very similar for both groups. Under condition A both groups show small positive slopes, indicating a slight average increase of EMG with time, with the nonmigraine group having a slightly larger value. For condition B, both groups exhibit a small average decrease in EMG with time. Under condition C, while migraineurs show a minimal decrease in EMG with time, and nonmigraineurs a minimal increase, the difference between the two slopes is negligable. Both groups show a small decrease in EMG with time under condition D.

DST Responses. Figure 2, part B displays the linear regression

equations of DST on time. Under condition A both groups show a small average increase in temperature over time, with the value for migraineurs being slightly larger. The slopes of condition B indicate that, while migraineurs' temperature responses increased minimally with time, the average responses of nonmigraineurs' decreased minimally with time. These two slope values differ marginally, with the slope of the migraine group having a slightly greater absolute value. Both groups show slight average increases with time under condition D.

EDR. Figure 2, part C displays the linear regression equations of EDR on time. Under conditions A, B, and D both groups show a small average decrease over time. Under condition C migraineurs show a small increase in EDR with time, while nonmigraineurs show a small decrease. The difference between the two is small. The rate of change of EDR with time is slightly higher for migraineurs under conditions A and D, while for nonmigraineurs the slope was greater in condition B.

General Trends. Examination of all linear regression analyses indicates that there are no notable differences in slope between the two groups. The regression lines do not cross under any of the conditions, and it appears that the general trends within each condition are similar for both groups.

Table 2

Analyses of Variance Assessing The Relationship Between Two Groups Across Four Conditions with Repeated Measures on Conditions

	EMG Responses				DST Responses				EDR Responses			
Source	df	MS	F	p	df	MS	F	p	df	MS	F	p
Groups:												
Between	1	9.655	2.138	.148	1	8.23	.047	.828	1	97.47	.987	.324
Within	76	4.517			76	174.01			76	98.75		
Conditions:												
Between	3	22.65	27.194	.001	3	1.625	1.66	.176	3	93.07	44.93	.001
Within	228	.833			228	.978			228	2.07		
Groups x Conditions:												
Between	3	3.59	4.311	.006	3	.152	.156	.926	3	1.07	.516	.671
Within	228	.833			228	.978			228	2.07		

Chapter V

DISCUSSION

Summary of Findings

The purpose of this experiment was to provide some clarification of the way in which stress leads to migraine by measuring well-defined physiological responses to a standard stimulus, and making comparisons between migraineurs and nonmigraineurs. The hypothesis of "individual uniqueness" or "symptom specificity" provided the theoretical context within which the comparisons were made, and gave rise to certain questions which seemed to address the major concern of the study. In the broadest form, the question asked if migraineurs display psychophysiological reactivity which differs from that of nonmigraineurs. While this study focussed on understanding the etiology of headache, clarification of the headache response could also prove beneficial to the development of more efficacious treatments for migraine.

The first specific question asked if migraineurs differ from nonmigraineurs on basal levels of EMG, DST or EDR. EMG levels were found to be significantly greater for migraineurs than for nonmigraineurs under the adaptation condition, but this difference disappeared under the remaining 3 conditions. Comparing the regression lines of migraineurs and nonmigraineurs under the adaptation condition, it appears that the difference starts out large and gradually decreases throughout the condition, largely due to an initial lower value for the nonmigraine group. There is no ready explanation for why the nonmigraine group's EMG level increased across the condition. One could speculate that this was caused by increased effort on the part of migraineurs to keep their eyes

open, and that the group difference in the slope of this increasing value was due to a ceiling effect for the migraine group who already had higher values. Putting aside the trend within conditions, the difference between migraineurs and nonmigraineurs is consistent with the findings of Bakal and Kaganov (1978), and Cohen, Rickles, and MacArthur (1978). The only systematic difference between adaptation and baseline conditions was that subjects were required to keep their eyes open in the former, but closed throughout the rest of the conditions. These results cannot be interpreted as reflecting higher levels of general activation for migraineurs with eyes open, as neither DST nor EDR showed a similar effect. Hence, this difference between groups appears to be specific to the EMG response. This finding does not appear elsewhere in the literature. The fact that one group had greater muscle tension than the other, but only with their eyes open, does not readily suggest an interpretation in terms of migraine activity. If, as some authors have suggested, migraine and muscle contraction headaches are not the discrete entities that they have traditionally been seen as, then a logical causative connection is possible: namely, that muscle contraction is associated with a cerebral vasoconstriction phase which represents the initial phase of the two-stage vascular migraine mechanism. Of course this is highly speculative, and would require verification , although Bakal and Kagonov (1978) also concluded that muscle tenesion appears to be a predisposing factor in migraine. This finding readily fits the concept of "dysponesis", which refers to faulty or misdirected efforts which lead to unnecessarily high levels of muscle tension, and negatively influence the organism (Whatmore & Kohli, 1979). This theory would hold

that, in keeping their eyes open, migraineurs covertly invoked bracing efforts which resulted in increased (and unnecessarily high) levels of muscle activity.

The basal DST level of the nonmigraine group was higher than that of the migraine group, and persisted across all conditions. However, this difference failed to meet significance. In contrast, Cohen, Rickles and MacArthur (1978) found that migraineurs had warmer hand temperatures than nonmigraineurs. There is no apparent reason for these two different findings. This particular result says little about group differences with respect to migraine etiology, although cooler hands are one measure of sympathetic arousal.

There was no significant difference between groups on basal electrodermal response (EDR), although nonmigraineurs had higher levels across all conditions. The EDR is directly proportional to the level of autonomic arousal (Shapiro & Surwin, 1979). A trend toward greater general arousal in nonmigraineurs is in conflict with the notion that migraineurs are more aroused, although it is compatible with theories of physiological specificity which would only preempt nonmigrainers from showing greater vascular responses. It is also possible that the non-significant EDR difference is due to the fact that nonmigraineurs had warmer hands than migraineurs; warmer hands might have more surface moisture than cooler hands. The nonsignificance of the EDR and DST measure is largely due to the fact that the variability of these measures within groups was large.

Psychophysiological reactivity was statistically defined as a significant conditions effect with post-hoc tests yielding differences between

conditions B (baseline) and C (stimulus). Findings of reactivity in the temperature modality for migraineurs, but not for nonmigraineurs (given by a post-hoc test for simple-main effects, following a significant groups x conditions interaction effect), would have been evidence for individual uniqueness or symptom specificity. Reactivity occurring for DST, but not EDR and EMG, would have been strong evidence for the specific activation of the modality associated with the mechanism of the complaint.

A significant conditions effects was found for EDR and EMG. For the EMG data the only significant specific comparisons were between condition A and each of conditions B, C, and D. The mean EMG value within condition C was higher than that in condition B. and D. Thus the effect was in the expected direction, despite failure to reach significance. For the EDR data the conditions effect was also significant, with post-hoc tests finding that the mean values within conditions A and B were significantly smaller than those within conditions C and D. The mean of condition D was smaller than C, but significantly so. There was no significant conditions effect for DST, although the mean for condition C was smaller than that of both conditions B and D. Thus, group differences aside, it appears that the introduction of the stimulus in condition C did not elicit a response in the EMG and DST modalities that was strong enough to reach statistical significance. In view of the relatively large sample, it appears that the stimulus was simply inadequate to evoke a strong enough reaction in these modalities. The exception was EDR which is a highly labile and sensitive measure (Shapiro & Surwit, 1979), no doubt one reason for its popularity as a psychophysiological measure.

The only groups x conditions interaction was discussed above. The lack of simple-main effects on conditions B and C across groups indicates that differential reactivity between groups was not observed for any of the three modalities. Thus, this study was unable to successfully demonstrate symptom specificity.

While the basic statistical reason for lack of significant findings in this study is that the variability within groups was too large in relation to the systematic variance between groups, there are also some practical experimental reasons which might explain the lack of significance of the results. As previously mentioned, it appears that the stimulus was not effective in eliciting sufficiently large EMG and DST responses. While this could be rectified by introducing a stimulus with a stronger psychological effect, part of the problem may also be due to the dependent measures. Since much of the error variance is attributable to the large number of data points within each condition, one way to decrease this intracondition variability would be to reduce the time within each condition. Unfortunately this would also attenuate the amount of information gathered.

The intracondition interval is also determined by the latency of the three response modalities. A shorter interval would cause the loss of data on the DST response toward the end of condition C. The longer interval provides more EMG data than required and thus contributes unnecessarily to the error variance. Each case thus places constraints upon the length of time over which data must be collected. One way around this would be to alter the dependent measure that required the longer interval, in this case, DST. This seems not only possible, but

in some respects desirable. From a response latency point of view, the time lag between vasomotor state and tissue temperature is well documented (Taub, 1977). If this lag were eliminated by taking direct blood flow measures, the intracondition intervals could be shortened.

Other aspects of the DST response also detract from its efficacy as an index of the vascular phenomena associated with migraine. The vascular response in migraine headache that is of foremost importance occurs in cephalic vessels. Other vascular phenomena are secondary, and in some cases poorly documented. Because of apparatus limitations, DST was used as a dependent variable in place of direct measures of cephalic vascular events. The various allusions in the literature to general vasomotor instability, and the known peripheral vascular responses to the O.R. and D.R., both suggest that DST was a reasonable substitute. However, it is not surprising that this measure did not behave according to theory, as the relationship between peripheral blood flow and cephalic blood flow is poorly defined. Although earlier investigators thought that there was a simple relationship between the two (Yates, 1980), recent investigations by Mathews et.al. (1980) found that peripheral and cephalic blood flow do not always simply covary. In order to test out specific biopsychological theories, the primary vascular response would be the ideal measure, particularly when this physiological modality is the one of most purported importance to the migraine response. In this sense the DST measure was not the most appropriate one for answering the theoretical questions posed in this study.

The structure of this study might not have been an appropriate

context within which to look for biopsychological factors which would distinguish migraineurs from nonmigraineurs, and clarify the headache response; stress might not be differentially manifested within this experimental and theoretical formulation of psychophysiological reactivity. The intensity of the response might be of lesser importance than the failure to habituate to repeated presentation of a stimulus. This alternate view of reactivity was suggested by Sturgis (1979), who also indicated that comparisons between physiological modalities are also plausible within this approach.

Implications for Further Research

The results of this study are modest, but suggest a few specific directions for further investigation. The finding of increased EMG values for migraineurs when their eyes were open, but not closed, might qualify other findings by other researchers, and is in need of further study to identify its relevance. This might have implications for those treatments which use EMG biofeedback; perhaps patients should learn to control and reduce their EMG levels while keeping their eyes open.

While this investigation was unsuccessful in its attempt to use the concept of psychophysiological reactivity to further delineate the migraine headache response, other studies have proved the utility of other specific applications of the general psychophysiological approach in furthering our understanding of various psychosomatic disorders, including headache. Indeed this approach has at its disposal many established constructs and techniques which can be implemented. It has previously been suggested that the concept of habituation might prove useful in further defining the headache response. Response lability is

another such concept which could be investigated. Such psycho-physiological studies may eventually prove valuable for purposes of classification, diagnosis, and treatment. The prevalence of headache in the population demands that it receive a large share of experimental attention, in hopes of ultimately ameliorating the pain and suffering.

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APPENDIX A

Control Group Data Form

Name:

Age: Sex:

Estimated headache frequency per year:

Are your headaches one-sided? ____ yes ____ no

Are your headaches severe enough to prevent you from maintaining your normal routine? ____ yes ____ no

What, if any, medication do you take for headache pain?

Does the medication lessen the headache pain? ____ yes ____ no

APPENDIX B

Name: _____ Address: _____

Phone: _____ Sex: e_____ Postal Code: _____

INCLUSION CRITERIA:	Criteria
1. What is your age?	_____(18 to 55)
2. How often have your headaches occurred in the last 2 months?	_____(less than 3X per day & than 1 per month)
3.(a) Do you take any medication for your headache?	_____
(b) What is the name of the medication?	_____
(c) How well does it control headaches?	_____(No)
(d) Currently using oral contraceptives?	_____(No); pregnant? _____(No)
(e) Are you on any other medications? specify:	_____
4. Have your headaches occurred one or more times pre month over the past 2 years?	_____(Yes)
5. Are you currently receiving any form of psychotherapy?	_____(No)
6. Do you ever experience sensory losses or paralysis of some muscles during a headache?	_____(No)
7. Do you suffer from a convulsive disorder (epileptic seizures)?	_____(No)
8. Do you have any form of heart disease or disorder?	_____(No)
9. Do you have any health problem such as diabetes, hypertension, etc.? specify:	_____

APPENDIX C

Explanation of the Study

Migraineurs:

"Today's session will last approximately 30 minutes. What I am going to do is attach you to 3 biofeedback instruments in order to see what levels of activity you produce in 3 different physiological systems: (a) skin temperature, (b) muscle tension, and (c) skin perspiration (indicate corresponding instruments). These instruments will not shock you or harm you in any way, they merely attach on to the surface of your skin with these wires. We are hooking you up today in order to find out how your body activity corresponds to your headache pattern, and, how the relaxation treatment program changes both your body activity and your headache pattern. Do you have any questions?"...

Non-Migraineurs:

"We are currently engaged in a study which seeks to develop better treatments for migraine headache. Part of the study is concerned with finding differences between people who have migraine headaches and those who do not. We wish to compare levels of body activity to determine how migraineurs differ from non-migraineurs. In order to make a good comparison we needed to find people without migraine who are similar to the people with migraine who are involved in the project. That is why we asked our clients to find friends of the same sex and of similar age.

Today's session will last about 30 minutes. I am going to attach 3 biofeedback instruments to measure levels of activity in 3 physical

systems: skin temperature, skin perspiration, and muscle tension (point out instruments). These will not shock or harm you in any way, they merely attach to your skin surface with these wires. Do you have any questions?...

APPENDIX D

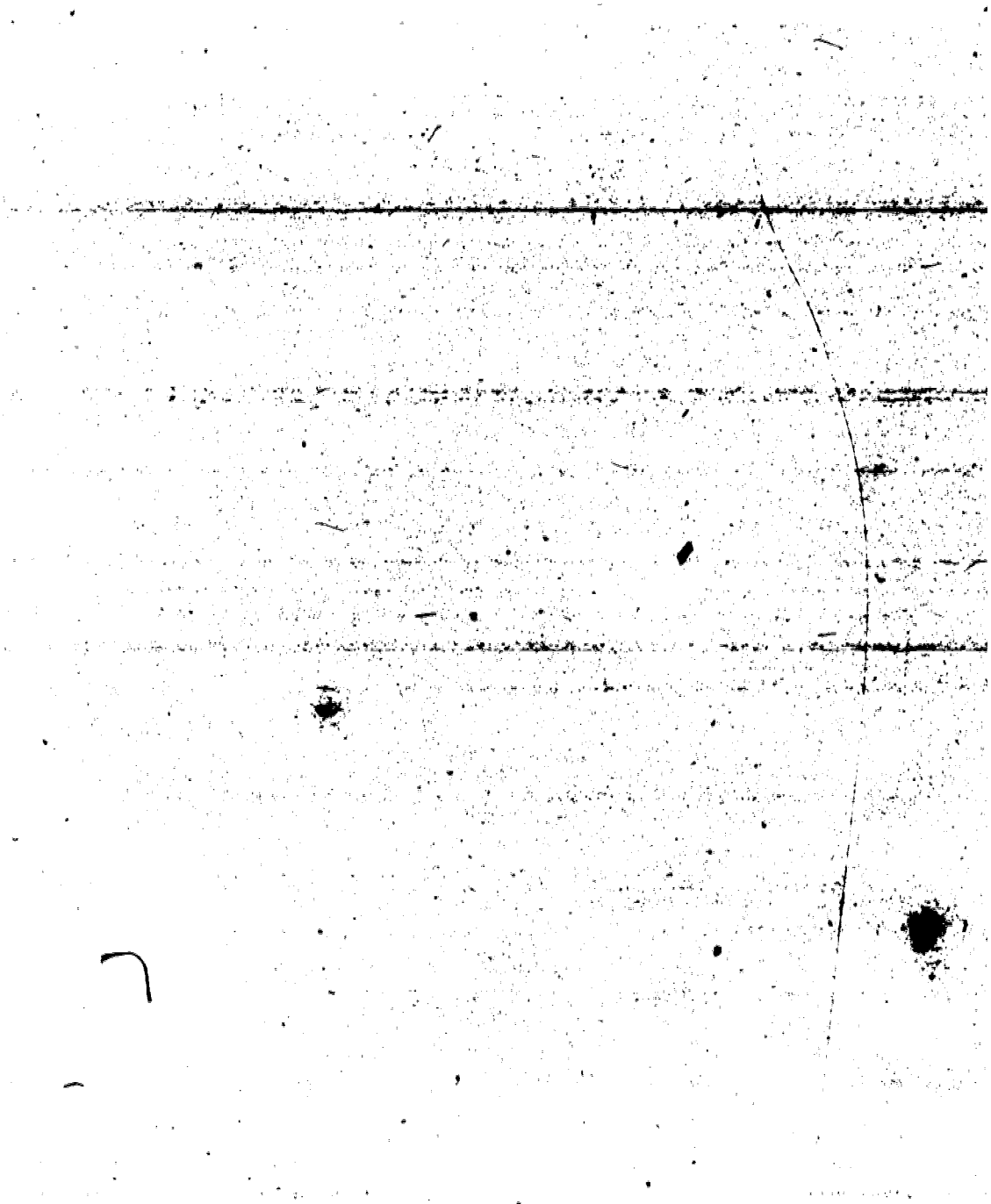
Instructions

For the next 15 minutes I would like you to relax comfortably with your eyes open and just listen to the music being played in the background. Try to avoid unpleasant thoughts and just enjoy this 15 minutes of rest. After 15 minutes I will ask you to sit for about 10 minutes with your eyes closed. Please try to sit quietly, without looking around or talking, and try to keep your hands still on the arm rest with your palms up. Do you have any questions? Okay then, starting with your eyes open, just relax and I will tell you when 15 minutes are up."

APPENDIX E

Instructions after Adaptation Period

"And now I would like you to sit for about 10 minutes with your eyes closed. Try not to fall asleep."



APPENDIX F

Instructions after Baseline Period

"Okay, while keeping your eyes closed now I want you to perform a mental task for me. I want you to subtract 7 from 1000 and then to continue subtracting 7 from your answers as fast as possible until I tell you to stop. Do this in your head, not out loud. Okay, so 1000 minus 7 is .... now keep going to yourself.

APPENDIX G

Instructions after Stimulus Condition

"Stop. What number did you get to? Now I just want you to relax with your eyes closed and listen to the music without interruption for 5 minutes, and then we are finished."

APPENDIX H

Table A

Scheffé Post Hoc Comparisons

EMG

main effect for conditions; Scheffé d.f.=1, 166.35.

comparison	F	p
A with B	30.46	< .05
A with C	18.42	< .05
A with D	26.24	< .05
B with C	1.51	> .05

simple main effects:

groups at condition A $F = 10.98$ (1, 166.35), $p < .05$

groups at condition B $F = .49$ (1, 166.35), $p > .05$

EDR

main effects for conditions, Scheffé d.f.=1, 71.8:

comparison	F	p
A with B	.034	> .05
A with C	5.61	< .05
A with D	4.11	< .05
B with C	6.51	< .05
B with D	4.88	< .05
C with D	.117	> .05

FIGURE 1

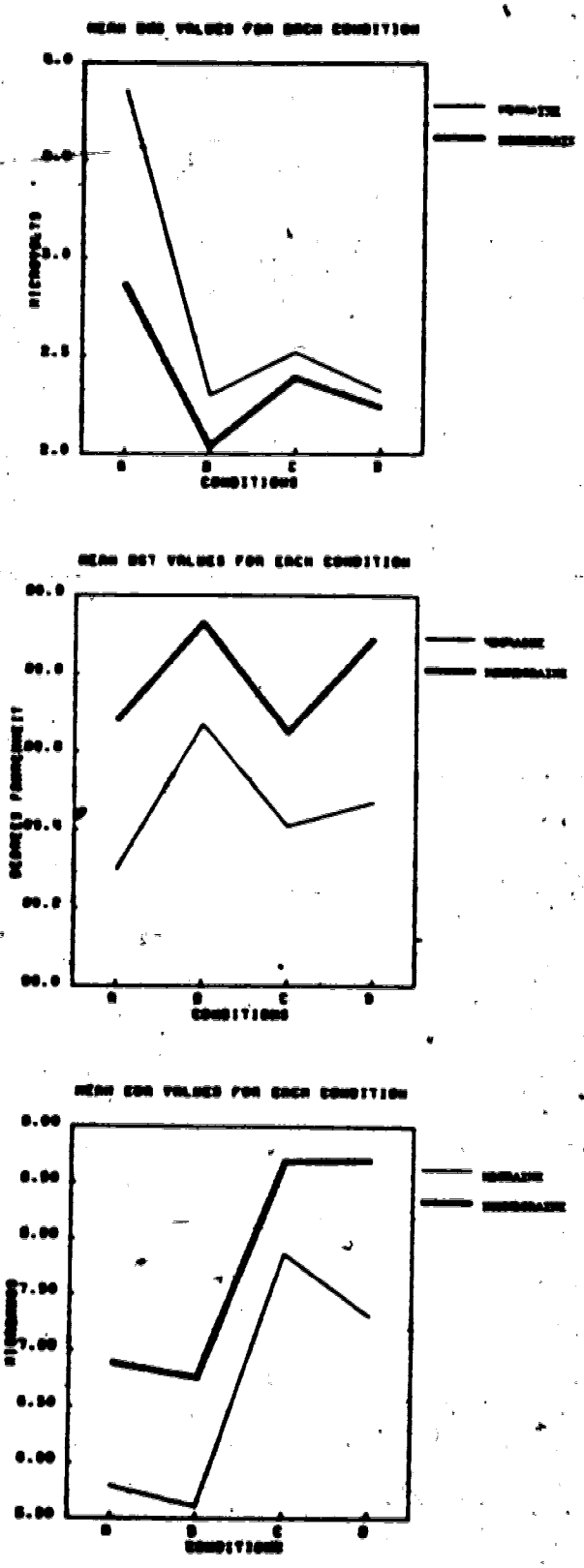


FIGURE 2, Part A

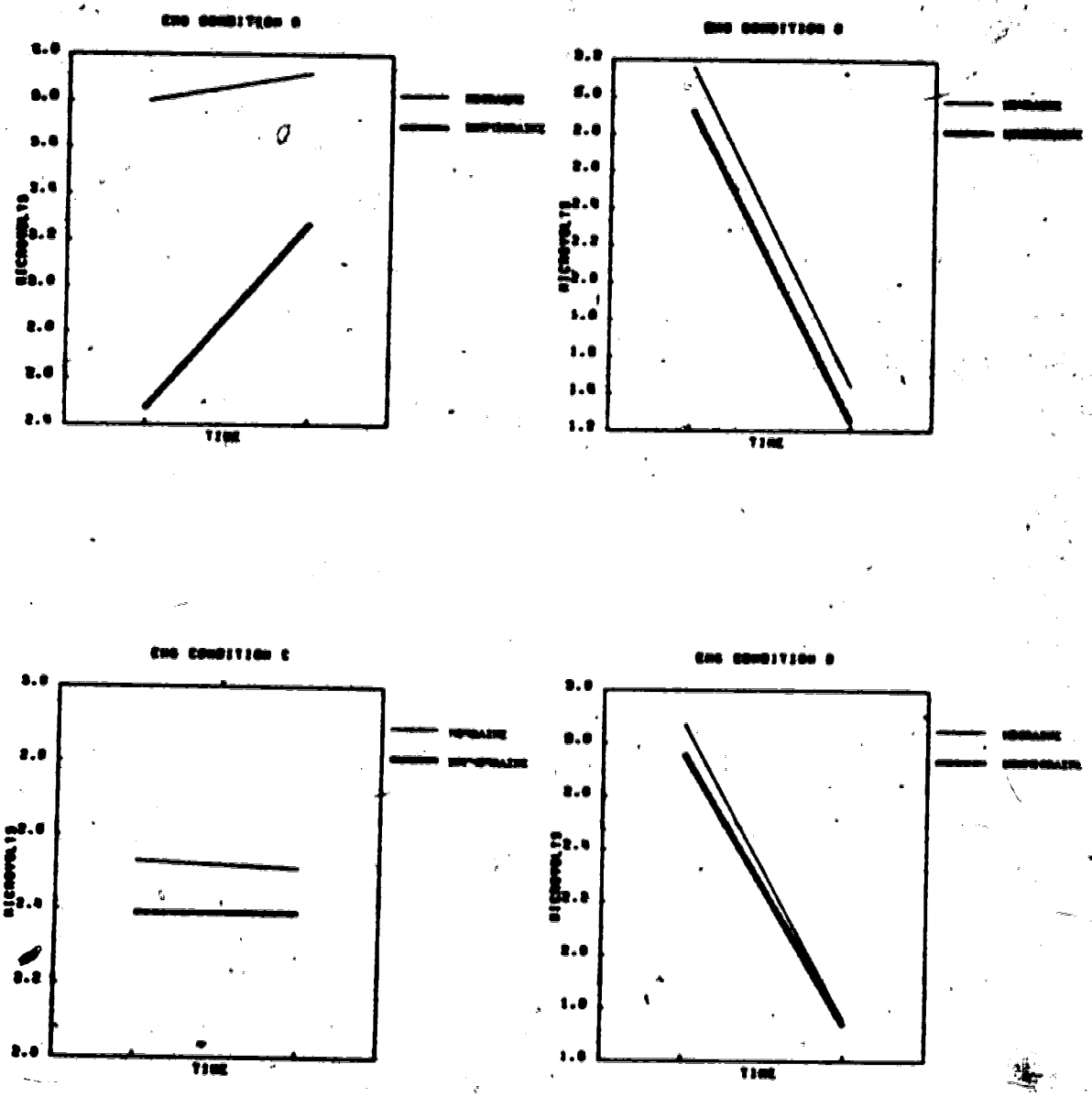


FIGURE 2, Part B

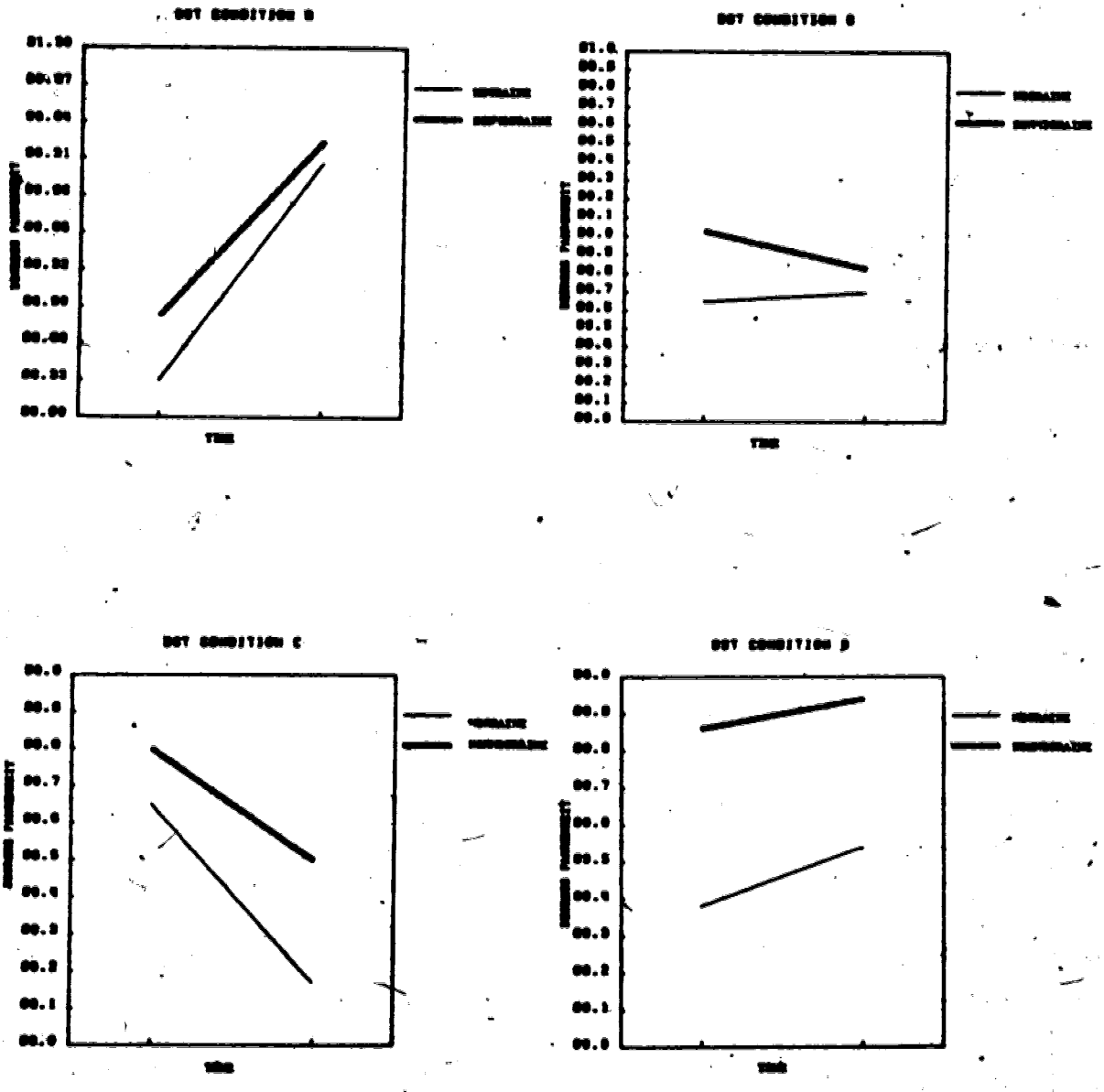


FIGURE 2, Part C