The Role of Emg in Tension Headache (Muscle Contraction Headache, Psychophysiology).

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THE ROLE OF EMG IN TENSION HEADACHE

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THE ROLE OF EMG IN TENSION HEADACHE

A dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
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in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy

in

The Department of Psychology

by

David P. McAnulty
B.A., Harding University, 1981
M.A., Louisiana State University, 1984
May, 1986
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"The writing of many books is endless and excessive
devotion to books is wearying to the body"
Ecclesiastes 12:12

I am compelled to thank those important individuals who made feasible the accomplishment of this seemingly endless and oft wearying project.

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ABSTRACT

Since the formal proposal by the Ad Hoc Committee on the Classification of Headache (1962) which identified muscle contraction (or tension) headache as a distinct headache diagnosis, numerous attempts have been made to validate the purported etiological mechanism. Results of these investigations, while failing to consistently distinguish tension headache sufferers from either normals or other headache type sufferers on various psychological and psychophysiological measures, have pointed to a lack of homogeneity within this diagnostic category.

The present study was intended to identify two distinct subgroups of tension headache sufferers. Tension headache sufferers exhibiting significant EMG increments from a headache-free state to a headache state were compared to sufferers failing to show such increments and to normal controls.

The results indicated that headache subjects with EMG increments during headache were more similar to headache free subjects on psychological measures of illness behavior and health locus of control than those individuals without the EMG increments. In addition, all three groups could be classified with a high degree of accuracy based on psychophysiological response patterns to stress. Results are interpreted as confirmatory of the presence of two tension headache subgroups, one with the expected muscular involvement and another with, perhaps, more psychogenic involvement.
The Role of EMG in Tension Headache

In 1962, the National Institute of Neurological Diseases and Blindness appointed an Ad Hoc Committee on the Classification of Headache composed of headache researchers. This body proposed fifteen different categories of headache syndromes. These included Migraine Headache, Muscle Contraction Headache and Combined Muscle Contraction and Migraine Headache. In addition, a number of headaches involving obvious structural damage were identified. It has been suggested that the muscle contraction headache syndrome accounts for the vast majority of presenting headaches (Ostfeld, 1962; Waters & O'Connor, 1971).

Muscle contraction headache was described by the Ad Hoc Committee (1962) as "an ache or sensation of tightness, pressure or constriction; widely varied in intensity, frequency and duration; sometimes long lasting; commonly occipital; and associated with muscle contraction in the absence of permanent structural changes; usually as part of the individual's reaction during life stress."

More recently, Olton and Noonberg (1980), in a review of the adjectives used to describe muscle contraction (or tension) headache, found that such statements as "t tightness", "pressure", "soreness" and "tight band" were most commonly reported. It has further been suggested that the pain in this type of headache is dull, occasionally changing to a throbbing ache, and is usually
bilateral (Appenzeller, Feldman & Friedman, 1979). The location of the pain varies greatly, and, though generally occipital, it may radiate to the temporal or frontal regions (Friedman, 1979).

Studies of headache diaries provide further information relative to the characteristics of muscle contraction headache (Cohen & McArthur, 1981; Friedman, 1979; Raskin & Appenzeller, 1980). Specifically, the duration of these headaches may range from a few hours to weeks or months of constant pain (Diamond & Dalessio, 1978; Friedman, 1979). The time of onset is most often between the hours of four and eight A.M. or P.M. (Diamond & Dalessio, 1978). A significant proportion, however, report headaches increasing in severity as the day progresses (Appenzeller et al., 1979; Granberry, 1980).

The symptoms listed above which describe tension headache are subject to considerable individual variation (Diamond & Dalessio, 1978; Friedman, 1979), to such a degree that a clear understanding of what the classification actually represents has evaded researchers. In fact, a survey of recent literature quickly reveals that the classification scheme offered by the Ad Hoc Committee (1962) suggests a clearer picture than really exists. As the present review will demonstrate, recent experimental studies, whether involving symptoms reports, psychological assessments or physiological measures, have failed to identify a strong homogenous muscle contraction headache entity. **Differentiation of Tension Headache from Other Headaches**

A major emphasis in tension headache research has been the
reliable and consistent differentiation of this type of headache from migraine and other headache syndromes. To accomplish this goal, tension headache has been defined not only in terms of the presence of muscle contraction symptoms (listed above), but also in terms of the absence of migraine-like symptomatology (Hunter & Philips, 1981). These symptoms have included unilateral onset, visual prodromata, relief of pain using vasoconstrictive drugs, nausea and/or vomiting, throbbing (pulsating) pain and photophobia.

Even when both inclusion and exclusion decision criteria are used, differential diagnosis has been problematic. In fact, a significant proportion of disagreement in headache diagnosis has been documented, with reliability estimates ranging from 59% to 86.4% (Blanchard, O'Keefe, Neff, Jurish & Andrasik, 1981; Granberry, 1980; Turkat, Brantley, Orton & Adams, 1981). Furthermore, clinical observations have revealed that a number of muscle contraction headache sufferers show typical vascular symptoms (Appenzeller et al., 1979). Finally, studies comparing symptom reports from migraine and tension headache subjects suggest that these two categories may not be identifiable on the basis of traditional diagnostic criteria (Bakal & Kaganov, 1979; Granberry, 1980; Philips, 1977c; Ziegler, Hassanein & Hassanein, 1954).

At least three factor analytic studies based on headache inventories have attempted to substantiate the traditional diagnostic categories. Ziegler and his colleagues (1954) identified seven factors, three of which were presented as migraine dimensions, and one of which (a headache duration factor) was posited as a
muscle contraction factor. However, important characteristics usually associated with muscle contraction headache (as well as migraine headache) were notably absent from the factors. A second investigation identified 12 factors from which 13 clusters were extracted (Granberry, 1980). The results of this study suggest that more than four categories (i.e., migraine, tension, mixed and cluster) are needed to classify headaches. Furthermore, the factors identified failed to provide fine discrimination between headache groups. Finally, Arena, Blanchard, Andrasik and Dudek (1982) identified three factors on the basis of a 14 item headache questionnaire. Although the authors felt that the factors (one migraine, one tension and one duration) supported the existence of two major types of headache, a significant proportion of muscle contraction subjects loaded positively on the migraine factor and negatively on the tension headache factor. Similarly, a number of migraine subjects loaded positively on the tension headache factor.

These factor analytic studies suggest that the diagnostic criteria presently used are inadequate. In general, the factors obtained failed to correspond closely to diagnostic categories. This was especially true of the muscle contraction headache group. However, it should be noted that results in these studies depended to large extent on the discriminative ability of the headache questionnaire used.

Further evidence concerning the inadequacy of current headache nosology may be drawn from studies of pain and/or symptom reports across headache groups. Bakal and Kaganov (1979) investigated the
symptom characteristics of headache sufferers and found that frequency of headaches rather than specificity of symptoms discriminated between diagnostic categories. Traditional diagnostic items such as muscle tension and nausea were found to differentiate between chronic and non-chronic headache sufferers, not between migraine and muscle contraction headache subjects. Philips (1977c) likewise found that headache diagnosis was not associated with characteristics such as headache laterality, prodromata, nausea and pill taking. Rather, these variables which are usually thought to discriminate between migraine and tension headache were found to relate to gender and/or severity of headache.

Certain researchers have found contrary evidence. Allen and Weinmann (1982) reported that tension and migraine headache sufferers report pain in different ways. While no differences were noted in the total number of evaluative and sensory descriptors of pain, the subjects in each category generally endorsed different adjectives to describe their pain. Tension headache subjects also tended to use significantly fewer affective words. Moreover, the majority of pain descriptors were endorsed by less than 17% of tension headache subjects and only one descriptor was used by more than 30% of the individuals in this group. Again, this indicates a great deal of variability among tension headache sufferers.

Hunter and Philips (1981) also found tension headache sufferers to differ from migraineurs in their usage of pain descriptors. However, a number of problems with this study should be noted.
While the migraine sample was comprised of psychiatric patients, the tension headache group included both psychiatric and normal subjects. The results indicated that differences in pain reports between the two tension headache groups were often as marked as those between the migraine sample and the tension headache group taken as a whole. Again, as in the Allen and Weinmann (1982) study, a great deal of variability was noted within each tension headache group. Differentiation between the headache categories thus relied heavily on the migraineurs' consistent endorsement of certain items (e.g., sickening, nauseating, blinding).

In conclusion, the contradictory nature of the findings from investigations of headache characteristics suggests that the current nosology may be inadequate. It seems the diagnostic criteria presently used are too global to identify meaningful headache groups. This is especially true for the tension or muscle contraction headache category where a great deal of variability has been noted. The failure to identify a strong homogenous tension headache entity based on symptom reports may indicate that this category, as it is presently used, includes distinct subgroups differing from one another in critical respects. This would seem likely in light of the fact that muscle contraction headache is often diagnosed by the simple exclusion of symptoms typical of other headache types (Haynes, 1981; Philips, 1977b). Other possible interpretations for the lack of substantiation of a distinctive tension headache category in the studies reviewed may include the idea of a headache continuum (Bakal & Kaganov, 1977) or inconsistent
symptom reporting on the part of headache subjects (Blanchard et al., 1981). However, as mentioned previously much of the ambiguity in the nosology can be attributed to significant variance within the muscle contraction headache group alone.

Pathogenesis of Tension Headache

The possibility of tension headache subgroups is further evidenced by the lack of any definitive explanation of the etiology of the disorder. Various theories have been offered to explain the origin of pain in tension headache (Dalessio, 1978; Diamond & Dalessio, 1978; Kudrow, 1976; Wolff, 1963). All of these views underline to some degree the importance of abnormal contraction of head muscles. Wolff and his colleagues (Wolff, 1963; Tunis & Wolff, 1954) first advanced the notion that the sustained contractions of head muscles was responsible for the pain in tension headache. Since that time, most of the research and conceptualization has been based on this model.

The results of the early laboratory studies by Wolff (1963) are however, affected by a number of serious methodological flaws. First, the samples were extremely small, ranging from one to three subjects. Second, the subjects used were not tension headache sufferers but normal individuals. Similarly, head pain was experimentally induced and not the result of spontaneous headache onset. Finally, the physical and pharmacological interventions used were extreme and in no way corresponded to the usual onset of headaches.

A major weakness in the traditional view of tension headache
Pathogenesis concerns the fact that many of the findings in recent psychophysiological investigations have failed to support Wolff's model (Philips, 1978; 1980; Pickoff, 1985; Williamson, 1980). These investigations have addressed a number of tenets, delineated by Philips (1978), which are inherent to the muscle contraction model of tension headache. For instance, tension headache sufferers would be expected to show differences from normals with respect to resting EMG levels (tonic changes) and/or greater EMG reactivity to various stimuli, especially stressors (phasic changes). Also, differences in muscle tension should be obvious between headache and headache free periods. Finally, EMG levels should be directly related to pain reports. Findings in these areas are, however, inconsistent as the data in the following section indicates.

**Psychophysiological Investigations of Tension Headache**

**Resting EMG and tension headache.** Based on the current view of tension headache pathophysiology, a certain amount of individual response stereotypy involving the muscles of the head and neck would be expected. Response stereotypy refers to an individual's tendency to respond (usually to stressors) with a consistent physiological pattern. Researchers, in their study of response stereotypy have noted that pathways modulating stress related pathology may react by showing shifts in physiological reactivity or tonic adjustments (Lacey, Bateman & VanLehn, 1953; Steptoe, 1980). Thus, in tension headache, one would expect to find either abnormal resting levels of muscle activity and/or excessive muscle reactivity to various stimuli.
Several investigators have found that tension headache sufferers show higher levels of resting muscle activity when compared to normal controls (Andrasik & Holroyd, 1980a; Cohen, Williamson, Monguillot, Hutchinson, Gottlieb & Waters, 1983; Phillips, 1977a; Pozniak-Patewiecz, 1976). Phillips (1977a) compared tension headache sufferers to controls and other headache types on EMG measures taken at four locations (neck, frontalis, temporalis and trapezius). She found the tension headache group to show significantly higher resting EMG levels than controls. Pozniak-Patewiecz (1976) similarly observed significant differences between tension headache and normal subjects in resting EMG. More recently, Andrasik and Holroyd (1980a) supported these findings, noting that muscle contraction headache sufferers showed higher resting frontalis EMG when compared to controls. Finally, Cohen et al. (1983) found that a group of 11 tension headache subjects exhibited higher frontal EMG than controls during a relaxation condition.

Even though studies such as these strongly support the notion of abnormal resting EMG levels as a characteristic of tension headache, the findings are attenuated by two factors. First, some researcher have failed to differentiate headache free controls from tension headache subjects on the basis of muscle activity at rest (Acosta, Jamamoto & Wilcox, 1978; Anderson & Franks, 1981; Bakal & Kaganov, 1977; Martin & Mathews, 1978; Sutton & Belar, 1982). In several of these studies, however, a great deal of variability was noted in EMG levels among tension headache subjects. Such wide
individual differences might be explained in terms of subgroups, with a group showing abnormal resting EMG levels and another failing to show such elevations. Such variance within the tension headache category would have a tendency to cancel out groups differences between migraine and muscle contraction headache groups. Second, even when tension headache sufferers show higher resting EMG levels than normal controls, migraine sufferers show similar and often more pronounced elevations (Bakal & Kaganov, 1977; Cohen et al., 1983; Philips, 1977a; Philips & Hunter, 1982; Pozniak-Patewicz, 1976). Such findings are not necessarily detrimental to the view that EMG has etiological significance in tension headache. Even though present in several types of headache, abnormal resting levels of muscle activity may have a different significance in each. This idea is supported by a recent finding indicating that when multiple rather than single physiological measures are used, differences in physiological patterns between headache groups emerge (Cohen et al., 1983).

In summary, the inconsistent results regarding an abnormal resting EMG level in tension headache suggest that subgroups may exist. The critical differentiating variable in this respect might be the etiological mechanism involved in headache. On the other hand, abnormal resting EMG levels may simply be a consequence of prolonged headache. Yet another hypothesis is that elevated EMG is reflective of the frequency of headaches. Two studies by Haynes and his colleagues (Haynes, Griffin, Mooney & Parise, 1975; Vaughn, Pall & Haynes, 1977) did indicate that subject who
reported more frequent headaches exhibit overall higher EMG levels. Clearly, more research is needed in order to rule out a possible causal role of high levels of resting EMG in at least a certain subgroup of tension headache sufferers. However, it may be that other factors such as physiological reactivity (especially EMG) are more important in explaining the etiological mechanisms of tension headache.

**EMG reactivity in tension headache.** Numerous researchers have investigated psychophysiological response patterns in tension headache sufferers. In these studies, investigators have measured reactivity to neutral auditory or visual stimuli (Bakal & Kaganov, 1977; Brantley, 1980; Cohen et al., 1983), to painful stimuli (Feuerstein, Bush & Corbisiero, 1982) or to experimental stressors (Anderson & Franks, 1981; Andrasik, Blanchard, Arena, Saunders & Barron, 1982; Andrasik & Holroyd, 1980a; Brantley, 1980; Cohen et al., 1983; Feuerstein et al., 1982; Gannon, Haynes, Safranek & Hamilton, 1981; Philips, 1977a; Sturgis, 1981; Vaughn et al., 1977). The stressors used have included: (a) mental arithmetic, (b) difficult quizzes, (c) the cold pressor, (d) an occlusion cuff, (e) the Harvard step-up procedure, (f) aversive slides and (g) subject defined imaginal stressors. Since tension headache has been defined as a stress related disorder (Ad Hoc, 1962), studies of EMG reactivity are important in the investigation of etiological mechanisms for this type of headache.

Results from investigations of physiological reactivity in tension headache subjects remain equivocal. Studies considering the
orienting response to non-aversive stimuli have failed to
discriminate between headache and non-headache groups or among
headache diagnostic groups (Bakal & Kaganov, 1977; Cohen et al.,
1983; Philips & Hunter, 1982; Sturgis, 1981). Likewise a number of
experiments measuring phasic changes during stressors have found no
differences between tension headache sufferers and normals. Martin
and Mathews (1978) reported no differences in EMG reactivity to a
mental task between tension headache sufferers and control subjects.
Others, using mental arithmetic and imagined stressors, obtained
similar results (Andrasik et al., 1982a; Feuerstein et al., 1982;
Sturgis, 1981). Physical stressors have also failed to produce
greater EMG changes in tension headache sufferers (Anderson &
Franks, 1981; Andrasik et al., 1982a; Gannon et al., 1981).

In other studies, tension headache sufferers have been
reliably differentiated from non-headache controls on measures
of EMG reactivity to stress. Philips (1977a) found that tension
headache subjects respond to an imagined stressor with
significant increases in EMG. However, no control groups were
used. Results from an investigation by Anderson and Franks
(1981) though not statistically significant confirmed the
expected pattern. These researchers found that tension headache
subjects tended to have the highest elevations in frontal EMG
during stress when compared to normals and migraineurs. Brantley
(1980) observed that muscle contraction headache sufferers
responded to an imagined stressor with significant frontalis EMG
increments while normals did not. These findings were confirmed
by Philips and Hunter (1982) who observed tension headache sufferers to be significantly more reactive to imagined stress than a non-headache comparison group. More recently, Cohen et al. (1983) reported a pattern of EMG increase to a stressful quiz among tension headache sufferers. Similar changes were absent in headache-free controls.

In brief, while a number of studies do suggest abnormal phasic EMG changes in tension headache sufferers compared to normals, others do not. The discrepancy among these results may be explained by several factors. First, stimulus variables such as duration (Boxtel & VanderVen, 1978; Philips & Hunter, 1982) and stimulus specificity of physiological responses patterns elicited (Andrasik et al., 1982a) have been demonstrated to affect the results obtained. Second, and perhaps more importantly, subgroups within the muscle contraction headache group have been noted which may cancel out differences. Bakal and Kaganov (1977), though not obtaining significant differences between headache and non-headache subjects, did observe that a proportion of the tension headache subjects were responding with decreases or no changes in EMG and others with increases. Philips (1977a) similarly noted a great deal of variability across tension headache cases with respect to EMG reactivity to stress. There is clearly a need to determine whether such differences in reactivity relate to pathogenetic mechanisms.

EMG levels during headache and no-headache states. Regardless of whether tonic and/or phasic physiological abnormalities are noted
in tension headache sufferers, a significant correlation between levels of muscle activity and pain would be expected. Specifically, increases in EMG should correlate with headache onset and headache intensity. Two approaches have been taken in examining the relationship between muscle activity and head pain. First, EMG levels in tension headache sufferers have been measured both during headache and headache free periods. If head pain in tension headache is indeed caused by sustained contractions of the muscles of the head, a significant increase in muscle activity during headaches would be expected. Second, biofeedback outcome studies have examined correlations between EMG changes and changes in headache activity or pain reports in order to verify whether concomitant changes occur as would be anticipated.

When tension headache sufferers are considered as a group, differences on EMG measures between headache and no-headache periods generally fail to reach statistically significant levels. Pozniak-Patewiecz (1976) investigated EMG levels in 30 tension headache cases, along with various other headache cases, both during and in the absence of headache, and found no differences. However, a major weakness of the study was that the groups used for headache and headache-free recordings were composed of different subjects. Philips and Hunter (1982) likewise failed to find differences in mean temporalis and frontalis EMG levels during pain and pain-free states. These results are modulated by the fact that statistical analyses were not performed, and again, different subjects constituted the pain and pain-free groups. Philips (1977a) also
examined EMG levels in the frontal, temporal, neck and trapezius muscles in a small group of tension headache sufferers. No significant increases were noted in any muscle group. In another similar study (Martin & Mathews, 1978), not only did the investigators fail to find an increment in neck or frontal EMG during headache, but an actual decrement was noted for the frontalis muscle.

The results of these three studies indicate that muscle tension may not be closely associated with pain in tension headache. These findings concur with results from attempts to correlate pain reports and headache activity across a number of headache-free and headache-present sessions (Epstein, Abel, Collins, Parker & Cinciripini, 1978; Harper & Steger, 1978). In these studies, EMG measures were recorded over several sessions, and each time pain reports (on a six or seven point scale) were obtained from the patients. Correlations between the two measures were not significant.

Contradictory results have, however, been reported. Two studies have in fact documented some relation between EMG and head pain. Philips and Hunter (1982) found both frontalis and temporalis EMG levels to be significantly correlated with headache intensity reports across 23 tension headache cases. Haynes et al. (1983) compared frontal, neck and forearm EMG readings during headache and non-headache states. For the group as a whole, significantly higher neck and arm EMG activity were documented during headache. Moreover, in individual subject analyses, 7 of 9
tension headache sufferers exhibited greater neck EMG during headache, 2 of 6 greater frontal EMG and 4 of 6 greater forearm EMG. A major problem with this investigation, though, was the extremely small sample size \((n = 9)\), as well as the absence of any control group. In addition, the individual analyses reported (paired t-tests) were performed on autocorrelated data and therefore represent a violation of the underlying assumptions of the statistics used. Replication is clearly needed.

Although results for tension headache sufferers as a whole generally indicate a lack of concurrence between EMG levels and head pain, there is evidence that subgroups exist for which a positive correlation between pain and muscle activity does exist. Bakal and Kaganov (1977) found that muscle contraction headache subjects did show significant increases in frontalis EMG from no-headache to headache. However, they noted a greater range in EMG activity in this group than in controls. In fact, they remarked that a subgroup of muscle contraction subjects who did show very high values of EMG during headache was responsible for the significant differences in resting frontalis EMG between normals and tension headache patients experiencing a headache. Another group of patients failed to show such elevations. Similarly, Epstein et al. (1978) found that when six tension headache sufferers were considered as a group, correlations between EMG and headache were not significant. However, two individuals did show the expected pattern. In these subjects, EMG levels were positively related to headache reports.
In conclusion, results from studies investigating EMG levels in tension headache sufferers as a whole during both headache and headache-free periods fail to show any significant correlations between the two variables. However, when individual results are examined, a great deal of variability is observed. Such variations may be representative of subgroups with differing pathophysiological mechanisms. These subgroups could clearly cancel out differences which may occur between the group as a whole and controls. However, to date, no study has systematically identified and differentiated between tension headache sufferers who show significant increases in EMG levels during headache and those who show minimal or no changes in EMG.

Changes in EMG and pain resulting from biofeedback. A second method of investigating the relationship between EMG levels and headache pain has involved observing changes on these two variables as a function of EMG biofeedback. If headache pain is associated with increases in EMG levels, decreases in EMG would be expected to be accompanied by a reduction in headache activity. In general, the superiority of EMG biofeedback over no treatment or placebo treatment is well attested (Beaty & Haynes, 1979; Blanchard, Andrasik, Ahles, Teders & O’Keefe, 1980; Nuechterlein & Holroyd, 1980; Olton & Noonberg, 1980; Williamson, 1981). However, when changes in EMG and headache activity are closely investigated, results are much less clear.

A number of investigators have found concomitant reductions in EMG levels and headache frequency, duration and/or intensity as a
result of biofeedback. Budzinski and his colleagues (Budzinski, Stoyva & Adler, 1970; Budzinski, Stoyva, Adler & Mullaney, 1973) found that EMG biofeedback produced significant decreases in resting frontalis EMG. They also observed a significant reduction in headache activity which correlated highly ($r = .90$) with the EMG decreases. Hutchins and Reiking (1976) likewise found biofeedback-aided relaxation to result in significant decreases in headache duration, headache intensity and EMG levels.

Others, even though substantiating the effectiveness of EMG biofeedback in the treatment of tension headache, have failed to identify a strong correlation between EMG decreases and changes in headache activity. Cox, Freundlich and Meyer (1975) found that reductions in EMG accounted for only 18 percent of the variance in EMG biofeedback treatment effects. Another group of investigators (Holroyd, Andrasik & Noble, 1980) also noted significant decrements in muscle activity and headache symptoms as a result of EMG biofeedback. However, these were not significantly correlated. Philips (1977b) similarly reported that while biofeedback was found to be successful in decreasing headache activity, reductions in EMG levels, although significant, were not synchronous with the changes in headaches. She suggested that pain complaints may change more slowly than muscle activity, and the results could be explained in terms of such a time lag.

In light of these nonsignificant correlations between EMG decreases and reductions in headache symptoms, it has recently been argued that the effects of biofeedback are largely non-specific.
Hart and Cichanski (1981) found that while headache activity decreased as a result of EMG biofeedback, no changes in EMG levels across sessions were observed. More dramatic evidence, though, is gathered from studies involving manipulations of feedback contingencies in biofeedback. Holroyd and his colleagues (Andrasik & Holroyd, 1980b; Holroyd et al., 1984) discovered that regardless of whether subjects learned to decrease, increase or maintain constant levels of frontal EMG, similar reductions in headache symptoms occurred. These improvements were not accompanied by decreased EMG levels. Rather, (sham) feedback on success seemed more important as a determinant of headache activity change. Philips and Hunter (1981) found that training certain subjects to increase muscle tension levels and others to decrease them using analogue feedback resulted in significant differences in resting EMG levels between the groups. However, neither group showed a significant improvement in headache activity.

Studies such as these demonstrate dramatically that for tension headache subjects as a whole, EMG decreases and reductions in pain reports are not necessarily correlated. Still, a number of problems should be noted. The type of outcome measure used may be critical to the results obtained. It may be that changes in headache intensity, for instance, are showing a significant treatment effect while frequency and duration of headache do not improve. In such an instance, EMG reductions may not be expected. In addition, changes in cognitive processes (self-efficacy, depression, etc...) may actually account for more of the variance in treatment effects, yet
have no causal connection, while EMG might. Finally, the active ingredients in biofeedback have yet to be identified.

Several explanations regarding the active component of biofeedback have been offered. First, it has been suggested that pain reports and EMG levels may be modified at different rates (Philips, 1977b). Second, some researchers propose that during biofeedback subjects are actually learning cognitive coping strategies to deal with subjective tension (Andrasik & Holroyd, 1980b; Holroyd et al., 1985). Finally, certain investigators interpret changes in headache symptoms as resulting from the subjects learning to monitor the onset of headaches and discriminate tension levels (Holroyd et al., 1980; Philips & Hunter, 1981).

No study has attempted to correlate changes in EMG to changes in headache activity with tension headache subjects separated into two subgroups: (a) those showing significantly higher EMG levels during headaches and (b) those without such increments. Yet, there is considerable evidence that the degree of association between EMG decreases and reductions in headache activity is subject to a considerable amount of individual variation (Philips, 1977b). It may be that these two variables are correlated in the one group but not the other.

Conclusions. The present review of psychophysiological investigations of tension headache reveals a great deal of inconsistency in the results obtained by various researchers. Indeed, neither tonic nor phasic EMG abnormalities have been reliably documented in tension headache sufferers as a whole.
Likewise, results concerning the relationship between EMG and pain are merely suggestive. However, when results from these investigations are examined closely, the evidence concerning individual variability is quite strong. As a result, there is a high likelihood that real differences between subgroups of tension headache sufferers and no-headache controls are being concealed by the inclusion of these subgroups into one general tension headache category. Yet, to date, no study has examined the differences between tension headache subjects with abnormal EMG levels during headache and those without changes.

**Psychological Factors in Tension Headache**

Concurrently with psychophysiological investigations, a sizeable literature has addressed the role of personality factors in the development and maintenance of tension headache. Before considering results from these investigations, a number of qualifications are in order. First, in light of the correlational nature of the results, cause and effect conclusions are tentative at best. Treatment outcome studies of tension headache would in fact indicate that psychological symptoms may be consequences rather than causes of tension headache (Cox & Thomas, 1981; Holroyd et al., 1980). Investigations including other patient groups are needed to elucidate this issue. Second, as Philips (1976) pointed out, tension headache sufferers who seek medical attention may represent a subgroup more likely to exhibit psychological symptoms. Third, results are often difficult to integrate in view of the wide variety in the type
and quality of assessment tools used.

The psychodynamic view of tension headache suggests that this disorder represents a psychoneurotic reaction in which anxiety is somatized in the form of a physical symptom (Martin, 1978). Based on psychiatric interviews with 100 muscle contraction headache sufferers, Martin (1972) concluded that this type of headache was associated with anxiety, anger and frustration. He further noted that conflicts relative to dependence, sexuality and anger control were common. Most patients were seen as rigid, compulsive, perfectionistic and worry prone; they were also said to display a great deal of denial. Secondary depression was further noted. Weatherhead (1980) likewise viewed tension headache as an anxiety reaction secondary to external stress and internal conflicts. He regarded this type of headache as equivalent to a psychogenic or hypochondriachal headache, common in inadequate, passive individuals and obsessive-compulsive types.

To a large extent, these traditional views of the tension headache personality have not been supported by results of studies using objective measures. However, certain characteristics have been identified among tension headache sufferers. Kudrow and Sutkus (1979) found that individuals with these headaches were similar to mixed headache subjects in showing significantly more depression, psychic distress and somatization, as measured by the Minnesota Multiphasic Personality Inventory (MMPI), when compared to normals and migraineurs. Other investigators (Andrasik, Blanchard, Arena, Teders & Rodichok, 1982b) similarly found muscle contraction
headache patients to exhibit the highest level of pathology on five MMPI scales (1,2,3,7,8) relative to other headache groups. Finally, Sternbach and his colleagues (Sternbach, Dalessio, Kunzel & Bowman, 1980) reported that their sample of tension headache sufferers was more depressed and anxious (MMPI scales 2, 7) than migraineurs, while headache groups as a whole exceeded general medical patients on all but two scales (4 and 9).

Taken altogether, these studies suggest more anxiety, depression, somatization and general distress in tension headache sufferers. However, two factors should be considered. First, though statistically significant, differences in test profiles were not clinically significant (Sternbach et al., 1980). Secondly, a great deal of variability was noted among tension headache sufferers with some exhibiting more pronounced pathology (T scores $>70$ for scales 1,2,3) and others showing essentially no differences from normals and migraineurs (Andrasik et al., 1982b). This finding points to the possibility of subgroups differing in psychological characteristics.

Investigators have also used a number of other psychometric instruments in their attempts to distinguish tension headache sufferers from controls. Results from these studies have been inconsistent. Tension headache sufferers have been found by some to reveal significantly more depression than controls (Cox & Thomas, 1981; Sternbach et al., 1980). Others have failed to support such an association (Andrasik, Blanchard, Arena, Teders, Teevan & Rodichok, 1982c; Pratt et al., 1982). In addition, it has been
found that even when depression is present in tension headache sufferers (a) it appears to be reactive rather than causal (Cox & Thomas, 1981) and (b) a great deal of individual variation occurs both within the tension headache category and across headache diagnostic categories (Davis, Wetzel, Rushwagi & McClure, 1976).

Tension headache sufferers have additionally been viewed as highly neurotic (Martin, 1978; 1983). Although contradictory evidence has been obtained (Philips, 1976), the bulk of research indicates the presence of anxiety in tension headache sufferers. Andrasik et al. (1982c) supported this notion in finding muscle contraction headache patients to be more anxious and revealing significantly more psychosomatic symptoms than normals. Others have also found these patients to report higher levels of trait anxiety than non-tension headache subjects (Andrasik & Bolroyd, 1980a; Pratt et al., 1982). Anderson and Franks (1981) similarly noted tension headache sufferers were not only anxious but also insecure as measured by the Edwards Personal Preference Survey.

In summary, tension headache sufferers have not consistently been differentiated from controls with respect to psychological characteristics, excluding measures of anxiety. In addition, they cannot be reliably distinguished from other headache patients on psychological measures. However, a consistent trend has been observed for tension headache sufferers to reveal the most pronounced and most varied psychological symptoms of all headache groups (Andrasik et al., 1981; 1982c). This variability may be indicative of subgroups differing in psychological characteristics.
and perhaps in pathogenetic mechanisms. More specifically, it has
been pointed out that many studies of tension headache subjects may
actually be including genuine muscle contraction headache sufferers
and psychogenic headaches in one category (Andrasik et al., 1982b;
Sternbach et al., 1980; Turkat, cited in Martin, 1983).

Psychogenic headache refers to a category of headaches in which
the symptomatology is vague and varied, often resembling that of
tension headache, and for which no pathophysiological mechanism can
be found; these headaches are further believed to be caused by
environmental factors (e.g., secondary gain) and/or mental or
emotional factors (Adams, Brantley & Thompson, 1982; APA, 1980;
Sternbach et al., 1980) Although in the Diagnostic and Statistical
Manual of Mental Disorders (APA, 1980) muscle contraction headache
is not viewed as a psychogenic pain disorder, many researchers in
the area have failed to differentiate between these two groups
(Adams et al., 1982; Blanchard, 1982; Sternbach et al., 1980). It
may be that the psychogenic headache subgroup included in the
tension headache classification is accounting for some of the
inconsistent findings in psychological and psychophysiological
investigations. However, the nature and role of psychological
correlates remain to be identified for tension headache subgroups
with different psychophysiological characteristics.

The Present Study

From the above review of literature, it can be concluded
that the tension headache classification as it is currently
defined evidences a great deal of heterogeneity. Tension
headache sufferers do not uniformly reveal elevated scores on psychologic indices, pronounced EMG changes to stress or even EMG increases during headache. To date, these measures have been used in attempts to discriminate tension headache sufferers from other headache categories. However, only one effort has been reported which attempted to identify subgroups of muscle contraction headache sufferers using some of these same measures (Haber, Kuczmiarczyk & Adams, 1985).

These investigators conducted two exploratory studies to identify a muscle contraction headache subgroup and a psychogenic headache subgroup. An arbitrary criterion (median split) of 15 μv frontalis EMG during headache was used to assign subjects to groups. The muscle contraction headache group was found to exceed the psychogenic and control groups in EMG levels both during headache and in the absence of headache. In addition, only the muscle contraction group showed significant increases from non-headache to headache states. With regard to reactivity analyses, all groups were found to respond to imagined stressors with increased frontal EMG. While these studies offered promising results concerning the existence of subgroups within the muscle contraction category, certain problems should be noted. First, the headache groups were not differentiated from one another, yet were both distinct from controls, on the MMPI (scales 1 and 3). This raises some question as to the appropriateness of the label of 'psychogenic' headache for the one tension headache subgroup. In addition, the groups were defined in terms of headache EMG alone. Subjects showing very real
EMG increases from non-headache state to headache may have been included in the psychogenic group, while subjects with constantly elevated EMG may have been included in the muscle contraction group. Clearly, these methodological problems must be addressed.

The present study was intended to compare headache sufferers to one another using EMG as an independent variable. As noted earlier, when headache EMG levels are used as a dependent variable, the vast amount of intersubject variability may mask any significant differences when the group is considered as a whole. In the present study, subjects manifesting symptoms of muscle contraction headache who show EMG increments during headache (group 1) were compared to those who fail to show such significant differences (group 2). In this attempt to isolate homogenous subgroups, the following variables were used to compare these groups: self-reported headache pain reports, psychopathology, stress, and illness behavior. It was hypothesized that these variables would distinguish between the groups.

The following questions were addressed in the present study:

Specifically, do the groups differ with regard to their reports of headache activity and symptoms? It was hypothesized that no significant differences would be found between the groups on measures of headache intensity, duration, and frequency. This would explain why these two groups, though differing in headache EMG levels, have been included in the same group in previous studies. It was, however, expected that subjects in group 2 would show a tendency to report headaches as more incapacitating than group 1.
Do these groups differ from each other or controls with regard to psychological characteristics? It was hypothesized that group 2 would reveal significantly more anxiety and depression than either group 1 or a control group, and, that group 1 and the controls would not differ significantly on these measures. These hypotheses were based on the findings reviewed earlier which suggested that a proportion (but not all) of tension headache sufferers show pronounced psychopathology.

Do these groups differ with respect to illness behavior (i.e., overconcern with physical symptoms, minor symptoms interfering with major aspects of social and occupational functioning, expectancies regarding the locus of control for health)? Group 2 was expected to differ significantly from group 1, which would in turn differ from controls on measures of illness behavior and health locus of control.

Do these groups differ in their psychophysiological reactivity to stress? It was hypothesized that group 1 would show more physiological reactivity (especially EMG) to stressors than either group 2 or controls.

In summary, it was hypothesized that when tension headache sufferers are divided according to the degree of muscle activity increase they experience during headaches, different subgroups would emerge. It was further expected that the groups showing significant increases in EMG levels during headache would differ from the group without such elevations with regard to physiological and psychological characteristics. In general, it was expected that
group one would be more similar to the control group with respect to psychological characteristics, while group two would be more like the controls in terms of physiological reactivity.
METHOD

Subjects

Research subjects were recruited through newspaper advertisements and referrals from the medical community resulting in a sample of 28 tension headache sufferers. Of these, 9 failed to complete the project requirements and were therefore excluded from the final sample. An additional 10 headache free controls were obtained. These were volunteers recruited through the headache subjects (non-blood relatives and friends) or from the community. Controls with more than six reported headaches a year and/or psychiatric history were excluded from the study. One control subject initially accepted in the study was subsequently excluded when daily headache monitoring (described below) revealed several headaches. All headache subjects were offered treatment for their headaches in return for their participation in the study, to begin immediately following the assessment phase. All groups were equated on relevant demographic dimensions. Chi-square analyses for gender and race across the three groups ($\chi^2 = .0798$ and $.0054$) were non-significant. Likewise, no age differences were noted ($F = 1.366$, $p < .05$). Sex and race distributions, along with mean ages, for each group are presented in Table 1.

The following criteria were used in reaching diagnostic decisions (Ad Hoc, 1962; Philips & Hunter, 1981): tension headache sufferers were characterized by the presence of typical muscle contraction pain complaints such as aching, dull pain, tightness, tension and caplike or bandlike pressure. Furthermore, the presence
<table>
<thead>
<tr>
<th>Group</th>
<th>Sex*</th>
<th>Race*</th>
<th>Mean age** range</th>
</tr>
</thead>
<tbody>
<tr>
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<td>9 White</td>
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</tr>
<tr>
<td></td>
<td>5 Females</td>
<td>1 Black</td>
<td>22-66</td>
</tr>
<tr>
<td>2</td>
<td>4 Males</td>
<td>8 White</td>
<td>43.4</td>
</tr>
<tr>
<td></td>
<td>5 Females</td>
<td>1 Black</td>
<td>27-61</td>
</tr>
<tr>
<td>3</td>
<td>4 Males</td>
<td>8 White</td>
<td>35.5</td>
</tr>
<tr>
<td></td>
<td>5 Females</td>
<td>1 Black</td>
<td>26-54</td>
</tr>
</tbody>
</table>

* Chi-Square non-significant
** F non-significant
of no more than one of the following vascular symptoms was established: throbbing pain, nausea and/or vomiting. Subjects typically had experienced at least two headaches per week.

In addition to the inclusion and exclusion criteria established above, a number of conditions automatically excluded individuals from the study. These included: severe sinus headaches (with a confirmed diagnosis), temporomandibular joint syndrome (based on a confirmed dentist's diagnosis), structural damage or physical trauma (such as concussions or pinched nerves) and past or present major psychiatric illness (e.g., psychosis). Likewise, subjects who had not had a neurological screening including at least a CT scan, a brain scan and/or an electroencephalogram (EEG) were excluded from the present study.

Assignment to headache category was based on two independent diagnoses. A board certified neurologist diagnosed all subjects based on an interview and a neurological examination. Also, a doctoral student in medical psychology diagnosed the subjects using the criteria outlined above. Discrepancies between the two diagnoses were discussed and resolved before inclusion into the study. This procedure resulted in 100 percent diagnostic agreement.

Tension headache sufferers were subdivided into two groups: those exhibiting substantial increases in frontal EMG during headaches, and those not displaying such changes in EMG. Subjects were assigned to either group based on the results from two psychophysiological recordings, one conducted during a headache and one in the absence of head pain (see description of these sessions
under Procedure). For each subject a change score was computed (the ratio of resting headache EMG to resting no-headache EMG). Inspection of the distribution of change scores revealed that nine (47%) of the headache subjects exhibited less than 10 percent of an EMG increase from no-headache to headache. The remaining ten subjects (52%) all experienced EMG increases from no-headache to headache representing a 60 percent or more change. The distribution of EMG change scores is represented graphically in Figure 1. Based on this distribution, subjects with a change score (ratio) of more than 1.50 were assigned to group 1, those with a score of 1.10 or less were assigned to group 2.

Paper and Pencil Measures (see Appendix A).

One time measures. Subjects were given the Illness Behavior Inventory (Turkat & Pettegrew, 1983) and the Health Locus of Control (Wallston, Wallston, Kaplan & Maides, 1976) to reflect different aspects of illness behavior and beliefs.

The Illness Behavior Inventory (IBI) is a recently developed 20 item self-report scale designed to assess overt behavior performed by an individual which indicates he or she is physically ill or in physical discomfort. While normative data are yet limited, the authors report mean scores of 72.5 for diabetic patients labeled as high illness behavior patients and 53.7 for low illness behavior patients. The Health Locus of Control (HLC) is an 11 item scale designed to assess the extent to which individuals perceive their physical health as being under the control of chance or significant others. The higher
Figure 1. Frequency distribution of no-headache to headache EMG ratios.
the score, the more external one's health locus of control. In normal controls, mean scores have been reported between 33 and 36, while an average score around 40 has been obtained with medical outpatients.

**Bi-weekly measures.** All subjects were required to complete the Zung Self-Rating Scale (Zung, 1972) and the State-Trait Anxiety Inventory, Form X-2 (Spielberger, Gorsuch & Lushene, 1970) every two weeks in order to assess levels of depression and anxiety. The Zung is a self-report depression inventory for which considerable validity has been documented (Schaefer et al., 1985). Scores of 50 or less are considered normal, while mild depression is indicated by scores of 50 or less, moderate depression by 60 to 69, and severe depression by scores over 70 (Gabrys & Peters, 1985). The State Trait Anxiety Inventory (STAI), form X-2, is a measure of trait anxiety in widespread use. For working adults, mean scores of 34 (SD = 9) are reported. In general medical and surgical patients, those without psychiatric complications obtain a mean score of 41 (SD = 12.5), while the mean score for patients identified as having psychiatric complications is about 44 (SD = 14.1).

**Daily measures.** Subjects completed a daily headache record designed to monitor headache frequency, intensity and duration, as well as degree of incapacitation.

**Physiological Measures and Apparatus**

All psychophysiological measures were recorded from a Grass Model 7 Polygraph. Measures of heart rate, galvanic skin
conductance, skin temperature, cephalic vasomotor response, and frontalis and temporal EMG were obtained during a laboratory session. Both basal and phasic activity were assessed for all subjects in the absence of headache. In addition, for headache subjects, basal frontalis EMG activity was recorded in a second laboratory session, during a headache.

**EMG.** To record forehead EMG, a 16 millimeter active electrode was placed 2.5 centimeters above each eyebrow and centered over the pupil (Lippold, 1967; Williamson, Epstein & Lombardo, 1980). In recording temporal EMG, two four millimeter electrodes were vertically aligned on the right temporalis muscle, which was located by palpating the muscle tensed by the subject. All skin surfaces were first cleaned with isopropyl alcohol and lightly abraded. Beckman silver/silver chloride (Ag/AgCl) electrodes filled with Beckman electrolyte paste were used to detect EMG. Electrode resistance was checked to ensure it was below 10 Kohms; when readings exceeded 10 kohms, electrodes were reattached. Activity was measured at both sites by Grass model 7P3 pre-amplifiers, set on integrator mode. An integrated EMG reading expressed in microvolts was obtained by measuring the size of pen deflections from baseline, every ten seconds.

**Blood Volume Pulse (BVP).** Cephalic pulse amplitude was measured with a Grass PTT1-S photoelectric plethysmograph sensor placed over the superficial artery of the left temple. The sensor was attached with a Beckman electrode collar. The photocell was directed to a Grass 7P1 pre-amplifier at a setting of TC 0.8. Pulse
amplitude was measured in millimeters by the deflections of the pen from peak to trough. Three pulses were sampled every 10 seconds.

**Galvanic skin resistance (GSR).** Skin resistance was measured at the thenar and hypothenar eminences of the palm on the non-dominant hand. Skin surfaces were cleaned with isopropyl alcohol. Beckman Ag/AgCl 16 millimeters were then placed. Skin resistance was recorded from a Grass 7P1 pre-amplifier with input set at EGR and sensitivity at 0.5 Mv/cm. Skin resistance was expressed as the mean in microhms of pen tracings, sampled every 10 seconds. It was then converted to skin conductance, by the formula (Hassett, 1978):

\[
\text{Conductance (mhos)} = \frac{1}{\text{Resistance (ohms)}}
\]

**Skin temperature (ST).** Finger skin temperature was measured via a Cyborg YSI series thermistor attached to the distal phalanx of the index finger on the non-dominant hand. The thermistor was connected to a Grass 7P1 pre-amplifier set at EGR. Temperature readings, in degrees Celsius, were obtained from the pen tracings, at 10 second intervals.

**Heart Rate (HR).** Heart rate was measured with Beckman Ag/AgCl 16 millimeter electrodes applied in a standard lead I placement (Hassett, 1978). One electrode was placed on the dominant forearm while the other was positioned on the non-dominant forearm. Both electrodes were attached on the ventral surface of the forearm, 5 centimeters below the joint. Skin surfaces were cleaned and lightly abraded. Electrodes led to a Grass model 7P4 tachograph, from which heart rate in beats per minute was obtained by counting the number of pen deflections (R waves). Every other
ten second interval was sampled in this manner.

Procedure

The present study involved four weeks of self-monitoring by all subjects. During this period of time, subjects reported to the headache clinic on a bi-weekly basis.

First visit. During the first session, subjects signed an informed consent form. Subjects were also administered the State-Trait Inventory (STAI), the Zung and the Illness Behavior Inventory (IBI). They were then supplied with the necessary forms for and instructed in the daily monitoring procedures. In addition, headache subjects received instructions for the psychophysiological recording sessions (see below), to be conducted both during a headache and in the absence of headache. The order of these two sessions was counterbalanced for all subjects. Controls were only scheduled for the stress session.

Second visit. During the second visit, research participants returned with their home records for the first weeks. Any errors or misunderstandings in the self-monitoring procedures were clarified at this time. Subjects were also administered the Zung, STAI, and the Health Locus of Control (HLC) during this session.

Third visit. At the end of the fourth week of self-monitoring, subjects came in for their third visit. At this time, they completed the Zung and STAI.

Laboratory sessions. In addition to the bi-weekly sessions, subjects also presented for psychophysiological recordings. For the recording only (headache) session, headache subjects were notified
to contact the clinic during their next headache of at least moderate intensity (a 5 or more on a scale from 0 to 10). Upon calling, they were instructed to report to the lab within two hours of the onset of the headache without taking any medication for the pain. During this visit, subjects were seated in a comfortable chair in a room kept at constant temperature and insulated from extraneous noises. Electrodes were then placed. Following a ten minute adaptation period, five minutes of resting EMG were recorded.

For the stress (non headache) session, headache subjects were instructed to reschedule if they were experiencing a headache (or more than a mild headache, i.e., a 1 or 2 on a ten point scale, for subjects with constant head pain) at the appointed time. Upon arrival, subjects were seated and electrodes were placed by two examiners. Room conditions remained the same as during the other lab session. An adaptation period of 10 minutes was allowed during which time the subjects were instructed to sit quietly with their eyes open and to relax. The next five minutes constituted the baseline. Two stressor conditions were then administered consecutively, the first of which was followed by a five minute return to baseline. Stressors were counterbalanced to control for order effects.

One of the stress tests was the 12 minute tape-recorded Quiz Electrocardiogram (Schiffer, Hartley, Schulman & Abelman, 1976). This test consists of a series of 35 questions of increasing difficulty. The Quiz was presented to the subjects as a test of intellectual capacity. Subjects were asked to write their
responses on an answer sheet, in order to eliminate movement artifacts in the temporal EMG and BVP measures that might result from answering orally.

The second stressor consisted of a two minute imagined stressful scene. This scene was defined as one having recently occurred for the subject and was determined before the beginning of the session. At the end of the session, subjects were then asked to rate the clarity with which they were able to visualize the scene. They were also asked to rate the stressfulness of both the Quiz and the imagined stressor, on a 0 to 10 point scale. The mean clarity rating for the imagined stressor was 6.57; mean stressfulness ratings for the imagined scene and the Quiz were 4.64 and 4.81 respectively.
RESULTS

Manipulation check. An attempt was made to validate the meaningfulness of assigning headache subjects to group 1 versus group 2 based on the EMG ratio cutting score described earlier. A 2 x 2 repeated measures ANOVA was computed on frontal EMG, with Group (1 vs 2) as between factor and State (no-headache vs headache) as repeated measures factor. This analysis (see Table 2) resulted in a significant main effect for State (F=8.666, p<.01), indicating headache EMG was higher than non-headache levels. However, a significant Group X State interaction (Figure 2) showed that only group 1 exhibited EMG increases, while group 2 revealed a non significant trend toward EMG reduction during headache. Post hoc comparisons (Tukey HSD) indicated the groups were not significantly different from each other during either the headache or no-headache state alone (p<.05).

Headache data. Measures of headache intensity (0 to 4) and duration (0 to 24 hours) were obtained for all subjects in the headache groups. An average daily Headache Severity Index was computed from these values based on the formula:

\[
\text{Sum (Intensity X Duration)} / \text{Total Days}.
\]

In addition, a headache disability rating (1 to 5; see Appendix B for a description of this rating scale) was obtained for each subject. These two headache activity measures were then subjected to a one-way MANOVA. Results from this analysis (Table 3) confirmed
Table 2

Summary table for repeated measures ANCOVA of headache and non-headache frontalis EMG for groups 1 and 2.

<table>
<thead>
<tr>
<th>Source of Variance</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>Sig</th>
</tr>
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<tr>
<td>Group</td>
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<td>0.00</td>
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</tr>
<tr>
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<tr>
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<td>.001</td>
</tr>
<tr>
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<td>17</td>
<td>27.273</td>
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</tbody>
</table>
Figure 2. Mean EMG for headache and non-headache session.
Table 3
Multivariate tests of significance on headache indices across groups.

<table>
<thead>
<tr>
<th>Test name</th>
<th>Value</th>
<th>F</th>
<th>Hypoth. df</th>
<th>Error df</th>
<th>Sig</th>
</tr>
</thead>
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<td>2</td>
<td>16</td>
<td>.249</td>
</tr>
<tr>
<td>Hotellings</td>
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<td>1.51841</td>
<td>2</td>
<td>16</td>
<td>.249</td>
</tr>
<tr>
<td>Wilk's</td>
<td>.84048</td>
<td>1.51841</td>
<td>2</td>
<td>16</td>
<td>.249</td>
</tr>
</tbody>
</table>
the hypothesis that no significant differences between groups 1 and 2 exist on measures of headache activity ($F = 1.518, p < .05$).

**Psychological data.** Averages for each the Zung and the STAI over time were computed. A one-way MANOVA (with three levels: controls, group 1 and group 2) was then performed on these and the IBI and HLC scores. The results of this analysis (presented in Table 4) revealed a significant multivariate effect for psychological measures across groups. In addition, significant univariate effects for STAI, HLC and IBI scores were obtained. Differences between group means were further analyzed utilizing Tukey's Honestly Significant Difference test (Tukey 1977) for a posteriori comparisons. Results from these analyses appear in Table 5. No two groups differed from one another on the Zung. However, the control subjects had significantly lower STAI scores than either groups 1 or 2, who in turn did not differ from one another. On the IBI, controls differed significantly from group 2, while the mean score for group 1 did not differ from either group 2 or controls. Finally, both controls and group 1 had significantly higher HLC scores than group 2, but did not differ from one another.

**Stress data.** Physiological reactivity was assessed across groups for all physiological measures. The last two minutes of each baseline period were scored, according to the sampling rates described above, to determine the pre-stress scores. For the Quiz, the first six minutes were scored to represent the stress score. For the imagined stressor, the first two minutes constituted the stress score.
Table 4

Multivariate tests of significance on psychological measures across groups.

<table>
<thead>
<tr>
<th>Test name</th>
<th>Value</th>
<th>F</th>
<th>Hypoth. df</th>
<th>Error df</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pillais</td>
<td>0.85744</td>
<td>4.12753</td>
<td>8</td>
<td>44</td>
<td>.001</td>
</tr>
<tr>
<td>Hotellings</td>
<td>2.10526</td>
<td>5.26315</td>
<td>8</td>
<td>44</td>
<td>.001</td>
</tr>
<tr>
<td>Wilk's</td>
<td>0.27832</td>
<td>4.76155</td>
<td>8</td>
<td>44</td>
<td>.001</td>
</tr>
</tbody>
</table>
Table 5

Means for psychological variables across groups.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>1</th>
<th>2</th>
<th>F</th>
<th>Sig. of F</th>
</tr>
</thead>
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<tr>
<td>Zung</td>
<td>32.4</td>
<td>42.8</td>
<td>41.7</td>
<td>2.950</td>
<td>.07</td>
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<tr>
<td>STAI</td>
<td>26.7</td>
<td>41.9</td>
<td>44.5</td>
<td>11.357</td>
<td>.001</td>
</tr>
<tr>
<td>IBI</td>
<td>38.5</td>
<td>52.4</td>
<td>64.2</td>
<td>5.825</td>
<td>.01</td>
</tr>
<tr>
<td>HLC</td>
<td>26.9</td>
<td>31.1</td>
<td>40.8</td>
<td>7.076</td>
<td>.001</td>
</tr>
</tbody>
</table>

Note: Means not joined by a rule are significantly different (p<.05)
For each of five physiological variables (frontal EMG, temporal EMG, HR, ST and SCL) one way ANOVAs were computed to detect any differences between groups at baseline. The groups were found to differ (.05 level) on baseline frontalis EMG. No other difference reached significance. For four variables (temporal EMG, HR, ST, SCL), a 2 X 2 X 3 repeated measures, mixed effects ANOVA was then computed. Each ANOVA had two within factors, Condition (i.e., type of stressor: imagined vs quiz) and Time (baseline vs stress), and one between factor, Groups (control vs group 1 vs group 2). In the case of frontalis EMG, a 2 X 2 X 3 ANCOVA was performed to control for baseline differences. For BVP alone, a 2 X 3 (Condition by Group) repeated measures ANOVA was computed. The Time factor was eliminated, since change ratios (stress / pre-stress) were entered into the analysis rather than absolute scores.

The ANCOVA on frontalis EMG revealed a significant main effect for Time ($F=15.36, p<.001$) and significant Group X Time ($F=3.194, p .05$) and Condition X Time ($F=9.74, p<.005$) interactions (see Table 6). The interactions are presented in graphic form in Figures 3 and 4. Significant frontalis EMG elevations were thus elicited, though these were moderated by group membership and type of stressor independently. Simple main effects were tested with Tukey's HSD for the between factor and Bonferroni's t statistic (Miller, 1966) for within factors. Experimentwise error rate was held constant at .05 for these and all other post hoc comparisons. Analyses of means for the Group X Time interaction showed that only the control group experienced significant EMG increases during stress. Likewise,
Table 6

Summary table for repeated measures ANOVA on frontal ENG.

<table>
<thead>
<tr>
<th>Source of Variation</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>Sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group</td>
<td>41.437</td>
<td>2</td>
<td>20.719</td>
<td>.069</td>
<td>ns</td>
</tr>
<tr>
<td>Within cells</td>
<td>7179.467</td>
<td>24</td>
<td>299.144</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Within Subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Condition</td>
<td>773.571</td>
<td>1</td>
<td>773.571</td>
<td>2.728</td>
<td>ns</td>
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<tr>
<td>Group X Condition</td>
<td>529.641</td>
<td>2</td>
<td>264.821</td>
<td>.934</td>
<td>ns</td>
</tr>
<tr>
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<td>7090.387</td>
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<td>283.615</td>
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<td></td>
</tr>
<tr>
<td>Time</td>
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<td>384.752</td>
<td>15.361</td>
<td>.001</td>
</tr>
<tr>
<td>Group X Time</td>
<td>160.000</td>
<td>2</td>
<td>80.000</td>
<td>3.194</td>
<td>.058</td>
</tr>
<tr>
<td>Within Cells</td>
<td>626.182</td>
<td>24</td>
<td>25.047</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time X Condition</td>
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<td>203.868</td>
<td>9.474</td>
<td>.005</td>
</tr>
<tr>
<td>Group X Time X Condition</td>
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<td>10.442</td>
<td>.485</td>
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</tr>
<tr>
<td>Within Cells</td>
<td>537.969</td>
<td>24</td>
<td>21.519</td>
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<td></td>
</tr>
</tbody>
</table>
Figure 3. Frontalis EMG: Group X Time interaction

Figure 4. Frontalis EMG: Condition X Time interaction
investigation of the Condition X Time interaction indicated that while the Quiz evoked significant EMG elevations from baseline, changes during the Imagined scene were non-significant. Furthermore, pre-stress (baseline) frontalis EMG levels were equivalent across stressors; however, stress scores for the Quiz differed statistically from Image stress scores.

Temporal EMG increased significantly during stress as evidenced by a significant Time effect (F=7.08, p < .01). Reactivity varied, however, as a function of group. This was indicated by a significant Group X Time interaction (F=3.72, p < .04). Results appear in Table 7. Tests of simple main effects revealed no significant differences between means. As Figure 5 illustrates, members of the control group and group 1 tended to show temporal EMG increases during stress, while subjects in group 2 exhibited non-significant decreases.

Analysis of heart rate revealed significant effects for Time (F=47.03, p < .001) and Condition (F=6.32, p < .02), which were moderated by a significant Time X Condition interaction (F=28.46, p < .001). Differences between Groups on heart rate approached significance (F=3.15, p < .06). Results from these analyses appear in Table 8. The Time X Condition interaction, depicted in Figure 6, clarifies the Time and Condition main effects. Stress scores were always higher than baseline scores. The significant baseline to stress increases observed during the Quiz condition were responsible for this effect. Though pre-Image scores tended to exceed pre-Quiz scores, Quiz stress scores were more elevated than Image stress scores.
Table 7

Summary table for repeated measures ANOVA on temporal EMG.

<table>
<thead>
<tr>
<th>Source of Variation</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>Sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group</td>
<td>1718.281</td>
<td>2</td>
<td>859.140</td>
<td>1.832</td>
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<tr>
<td>Within cells</td>
<td>11723.513</td>
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<td>468.941</td>
<td>1.832</td>
<td>ns</td>
</tr>
<tr>
<td>Within Subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Condition</td>
<td>649.848</td>
<td>1</td>
<td>649.848</td>
<td>2.213</td>
<td>ns</td>
</tr>
<tr>
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<td>2</td>
<td>306.502</td>
<td>1.844</td>
<td>ns</td>
</tr>
<tr>
<td>Within Cells</td>
<td>7342.000</td>
<td>25</td>
<td>293.705</td>
<td>1.844</td>
<td>ns</td>
</tr>
<tr>
<td>Time</td>
<td>51.034</td>
<td>1</td>
<td>51.034</td>
<td>7.084</td>
<td>.013</td>
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<tr>
<td>Group X Time</td>
<td>53.524</td>
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<td>26.762</td>
<td>3.715</td>
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<td>Within Cells</td>
<td>180.101</td>
<td>25</td>
<td>7.204</td>
<td>3.715</td>
<td>.039</td>
</tr>
<tr>
<td>Time X Condition</td>
<td>9.133</td>
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<td>9.133</td>
<td>1.436</td>
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</tr>
<tr>
<td>Group X Time X Condition</td>
<td>22.887</td>
<td>2</td>
<td>11.443</td>
<td>1.799</td>
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</tr>
<tr>
<td>Within Cells</td>
<td>159.019</td>
<td>25</td>
<td>6.361</td>
<td>1.799</td>
<td>ns</td>
</tr>
</tbody>
</table>
Figure 5. Temporal EMG: Group X Time interaction.
<table>
<thead>
<tr>
<th>Source of Variation</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>Sig</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Between Subjects</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Group</td>
<td>2635.428</td>
<td>2</td>
<td>1317.714</td>
<td>3.149</td>
<td>.061</td>
</tr>
<tr>
<td>Within cells</td>
<td>10042.739</td>
<td>25</td>
<td>418.447</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Within Subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Condition</td>
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<td>1</td>
<td>124.592</td>
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<td>.019</td>
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<tr>
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<td>16.269</td>
<td>.825</td>
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<td>Within Cells</td>
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<td>1121.332</td>
<td>47.026</td>
<td>.001</td>
</tr>
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<td>Group X Time</td>
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<td>22.114</td>
<td>.927</td>
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</tr>
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<td>Within Cells</td>
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</tr>
<tr>
<td>Time X Condition</td>
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<td>542.258</td>
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<td>Group X Time X Condition</td>
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<td>37.121</td>
<td>1.948</td>
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</tr>
<tr>
<td>Within Cells</td>
<td>457.333</td>
<td>24</td>
<td>19.056</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Figure 6. Heart rate: Condition X Time interaction.

Figure 7. Digital skin temperature: Condition X Time interaction.
Digital skin temperature tended to decrease during stress, as a
significant main effect was obtained for Time (F=13.01, p<.001).
This decrease was more pronounced for the Quiz as indicated by a
significant Time X Condition interaction (F=6.56, p<.02). A
posteriori comparisons indicated Quiz scores exceeded Image
scores, regardless of Time. However, significant decrements in
skin temperature, during stress, were obtained only for the Quiz.
These results are represented in Table 9 and Figure 7.

Analysis of BVP revealed no reliable changes across Conditions
or Groups (Table 10). Likewise, SCL did not vary from baseline to
stress for either stressor. Differences between groups did,
however, approach significance (F=2.89, p<.074). Results of
analyses on SCL appear in Table 11.

Finally, a Discriminant analysis with backward stepwise
solution was conducted. In this type of analysis, all variables
are entered provided they meet the minimum tolerance level
(.001). Variables in the equation are then tested for removal on
the basis of their partial F values. Values of 1.0 were used for
F-to-enter and F-to-remove. The final equation adopted is that
which minimizes the overall Wilk's lambda.

Change scores from baseline (averaged over both baselines)
to stress (averaged over both stressors) for frontal and temporal
EMG, HR, SCL and ST were entered as independent variables. The
final solution retained temporal EMG, SCL and HR. Due to the
restricted sample size, the final Wilks' lambda was adjusted for
shrinkage by the formula (Cohen & Cohen, 1983):
Table 9

Summary table for repeated measures ANOVA on digital skin temperature.

<table>
<thead>
<tr>
<th>Source of Variation</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>Sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group</td>
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<td>214.889</td>
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</tr>
<tr>
<td>Within cells</td>
<td>5054.051</td>
<td>25</td>
<td>202.162</td>
<td></td>
<td></td>
</tr>
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<td>Within Subjects</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
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<td>10.988</td>
<td>13.008</td>
<td>.001</td>
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<tr>
<td>Within Cells</td>
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<td>.845</td>
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<tr>
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<td>4.998</td>
<td>6.558</td>
<td>.017</td>
</tr>
<tr>
<td>Group X Time X Condition</td>
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<td>.255</td>
<td>.335</td>
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<td>25</td>
<td>.762</td>
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</table>
Table 10

Summary table for repeated measures ANOVA on blood volume pulse.

<table>
<thead>
<tr>
<th>Source of Variation</th>
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<th>df</th>
<th>MS</th>
<th>F</th>
<th>Sig</th>
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<td><strong>Between Subjects</strong></td>
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</tr>
<tr>
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<td>.196</td>
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<td>.162</td>
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</table>
Table 11

**Summary table for repeated measures ANOVA on skin conductance.**

<table>
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<th>MS</th>
<th>F</th>
<th>Sig</th>
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<td></td>
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<td>.003</td>
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<td>.001</td>
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</tr>
<tr>
<td><strong>Within Subjects</strong></td>
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<td></td>
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<td></td>
<td></td>
</tr>
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<td>.000</td>
<td>.205</td>
<td>ns</td>
</tr>
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<td>.000</td>
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<td>.000</td>
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<td>.000</td>
<td>.167</td>
<td>ns</td>
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<td>.000</td>
<td>1.409</td>
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<td>.000</td>
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</tr>
<tr>
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<td>.000</td>
<td>.544</td>
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<td>.000</td>
<td>1.539</td>
<td>ns</td>
</tr>
<tr>
<td>Within Cells</td>
<td>.005</td>
<td>25</td>
<td>.000</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
\[ R = 1 - ((1 - R)n - 1 / n - k - 1) \]

where \( R = 1 - \lambda \). The resulting Wilks' lambda was .5699 (\( p < .02 \)), indicating that 43.01 percent of the variance in group membership could be accounted for by three physiological reactivity variables: temporal EMG, HR and SCL. Discriminant function coefficients and mean discriminant scores on the functions for each group are presented in Tables 12 and 13. Moreover, the two canonical discriminant functions derived in this analysis resulted in 70.37 percent accurate prediction of group membership, as indicated in the classification table (Table 14).
Table 12

**Standardized canonical discriminant function coefficients for variables retained in the final equation.**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Function 1</th>
<th>Function 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMG (temporal)</td>
<td>.91244</td>
<td>.16361</td>
</tr>
<tr>
<td>Heart Rate</td>
<td>-.59388</td>
<td>.77239</td>
</tr>
<tr>
<td>Skin Conductance</td>
<td>-.07911</td>
<td>.96293</td>
</tr>
</tbody>
</table>
Table 13

Canonical discriminant functions evaluated at group means.

<table>
<thead>
<tr>
<th>Group</th>
<th>Function 1</th>
<th>Function 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-.15923</td>
<td>.61473</td>
</tr>
<tr>
<td>2</td>
<td>-.78748</td>
<td>-.47836</td>
</tr>
<tr>
<td>3</td>
<td>.87690</td>
<td>-.25782</td>
</tr>
</tbody>
</table>
Table 14

Classification results from discriminant analysis.

<table>
<thead>
<tr>
<th>Actual group</th>
<th>number of cases</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>10</td>
<td>8</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>80%</td>
<td>0%</td>
<td>20%</td>
<td></td>
</tr>
<tr>
<td>Group 2</td>
<td>8</td>
<td>2</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>25%</td>
<td>50%</td>
<td>25%</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>9</td>
<td>2</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>22%</td>
<td>0%</td>
<td>78%</td>
<td></td>
</tr>
</tbody>
</table>

Percent of cases correctly classified: 70.37%
DISCUSSION

Results from the analyses of the EMG data across headache and non-headache states would support the underlying assumption of the present study, namely that at least two distinct subgroups are engulfed in the general muscle contraction or tension headache category. Indeed, subjects were sorted into those showing at least 60 percent increase in EMG from no-headache baseline to headache (group 1) and those showing no such changes (i.e., less than 10% increments) based on visual inspection of the data. Statistical analyses supported this distinction, since only group 1 showed significant increases. As expected, the groups could not be distinguished from one another on the basis of either no-headache or headache EMG alone. Results also indicated that headache EMG increments for group 1 were sufficiently pronounced to cause a significant difference between headache and non-headache EMG levels when both groups were collapsed, even though group 2 actually demonstrated a trend toward lessened EMG during headache.

Considered in light of other research reports, these observations help to clarify contradictory findings. While certain investigators have documented higher EMG levels during headache for tension headache subjects (Haynes et al., 1983), others have found either no differences (Philips, 1977a) or even decreases (Martin & Mathews, 1978). In the present study, one group clearly demonstrating increases during headache was distinguished from
another which showed decreases. The existence of these two subgroups would explain the great intragroup variability in EMG activity reported by others (Bakal & Kaganov, 1977; Epstein et al., 1978). It could be that, due to the restricted sample sizes reported, the percentage of subjects with the expected EMG elevations during headache varies from study to study and thereby results in conflicting findings.

In sum, these findings point to the need to consider EMG levels during headache and non-headache states in combination, rather than relying on a single assessment. This study differs from previous attempts to identify a subgroup of 'true' muscle contraction subjects, with EMG pathology, in that those have depended on a headache EMG reading alone, with no regard for initial headache free levels (Haber et al., 1985; Philips & Hunter, 1981).

Turning to the headache diary data, results supported the hypothesis that groups 1 and 2 would not differ in terms of headache activity. In fact, no differences were obtained on a headache index combining frequency, intensity and duration, thereby demonstrating that differences observed between the groups on other measures, whether physiological or psychological, could not be attributed to differing headache severity. The hypothesis that group 2 would report headaches as more incapacitating, on the other hand, was not supported by the present data. This failure to document such a difference might be accounted for by (a) the restricted range of possible scores on the disability rating scale (0 to 5) and/or (b) the inclusion of operationalized descriptors for the rating scale.
(cf. Appendix B), which allowed little room for a subjective evaluation of one's headache.

Psychological test scores were predicted to discriminate among the groups, with group 1 presenting as the more similar to controls. This hypothesis was largely supported, with the exception of measures of anxiety and depression. None of the groups differed in levels of self-reported depression. This finding has been reported by others, who compared tension headache subjects to controls (Andrasik et al., 1982c; Pratt et al., 1982). In this absence of clinically elevated depression scores for either headache group, the traditional view of tension headache as highly associated with depression was not confirmed.

This investigation likewise concurred with others in obtaining significantly higher levels of anxiety in tension headache sufferers as a whole, regardless of subgroup membership (Andrasik et al., 1982c; Andrasik & Holroyd, 1980a; Blaszczynski, 1984; Pratt et al., 1982). The absence of differences between groups 1 and 2 is analogous to the results of a study conducted by Naliboff and colleagues (Naliboff, Cohen & Yellen, 1982) with a chronic pain population. These investigators were unable to differentiate patients with an organic basis to their pain from those with functional etiology on indices of psychopathology. This would suggest that pain, regardless of its origin, is associated with elevated psychological test scores, including anxiety.

Differences between the groups on measures of illness behavior and health beliefs followed the hypothesized pattern. On the IBI,
group 2 differed significantly from controls, with group 1 falling in between. The IBI was developed to identify patients with excessive or inappropriate illness behavior; scores on the scale correlate positively with medical expenditures, frequency of medical utilization, number of days in the hospital, and other similar indices (Turkat & Pettengrew, 1983). Therefore, when compared to no headache controls, it appears that subjects who fail to exhibit the expected ENG elevations during headache constitute a group of individuals prone toward exaggeration of and overreaction to physical symptoms. As such, these patients would be expected to be more susceptible to secondary gain for pain behavior. Tension headache subjects with the expected 'muscle contraction' pathophysiology, on the other hand, do not respond to illness any differently than healthy controls, in spite of suffering with chronic, recurrent head pain.

Results with the HLC are even more striking. Controls and group 1 had equivalent means, which were significantly lower than mean scores for group 2. In other words, members of the latter group tended to attribute their illness and physical symptoms to external factors, such as chance or powerful others. This finding is noteworthy in that previous studies have not found tension headache sufferers as a group to differ from controls in terms of health locus of control (Andrasik et al., 1982c; Pratt et al., 1982). Changes on this dimension, though, have been correlated with improvement during biofeedback treatment (Holroyd et al., 1984).

Thus, differentiation of two subgroups of tension headache
sufferers with significantly different HLC scores may have direct implications for treatment outcome and/or recommendations regarding optimal treatment modality. Indeed, a number of studies have emerged which would suggest that internally oriented subjects respond better to progressive relaxation than externals (Lewis, Biglan & Steinbock, 1978), while the latter learn EMG biofeedback more quickly (Prager-Decker, 1978). Factors accounting for such differences may include the fact that biofeedback provides external cues and, therefore, would be more appealing to externally oriented subjects. On the other hand, it may be that patients with a 'psychogenic' profile are more responsive to the placebo effect associated with the 'high-tech' aspect of biofeedback treatment. Further research is needed to answer these questions.

The delineation of psychological differences among controls, tension headache sufferers with raised headache EMG during headache and patients without these increments is remarkable in that the criterion used for group assignment (EMG ratio) is not itself a psychological dimension. Moreover, the only other reported study which attempted to identify tension headache subgroups in a similar manner (Haber et al., 1985) failed to note any psychological differences (on the MMPI) between groups, even though one group was labeled a 'psychogenic pain' group. Thus, in an area where discrepant results abound, the identification of physiologically distinct subsets of tension headache sufferers with meaningful psychological differences is particularly encouraging. It would seem that a subgroup does exist with no evidence of abnormal muscle
activity during headache, showing a psychological profile indicative of excessive illness behavior and external locus of control for health, along with anxiety. This clinical picture would be concordant with a DSM III diagnosis of 'psychogenic pain disorder.'

With respect to psychophysiological reactivity to stress, contrary to the hypotheses, group 1 was not found to exhibit the highest levels of reactivity. Neither was evidence of muscular response stereoptypy obtained for either headache group. In fact, main effects were not obtained on any single psychophysiological measure. A close analysis of the results reveals a number of similarities to, as well as discrepancies from, previous findings.

With particular respect to frontalis and temporal EMG, no significant group differences were obtained. Considered together, the groups revealed increased EMG during stress. However, an interaction effect showed that, surprisingly, only controls experienced significant increments in (frontalis) EMG. Group 1 showed a similar, though non significant, pattern while group 2 revealed a slight decrease. This absence of group differences, while in contrast to a number of reports (Brantley, 1980; Cohen et al., 1983; Philips, 1977a, Philips & Hunter, 1982), replicates results of previous researchers (Andrasik et al, 1982a; Feuerstein et al., 1982; Gannon et al., 1981; Martin & Mathews, 1978; Sutton & Belar, 1982) who failed to differentiate controls and tension headache subjects with respect to EMG reactivity to stressors. In fact, Martin and Mathews (1978) also noted a tendency for controls to exceed headache subjects in phasic EMG changes.
In light of this absence of significant group differences, it is precarious to discuss distinct response patterns. Nevertheless, a clear trend for controls and group 1 to show parallel responses was noted. Group 2 tended to have higher baseline levels with slight decrements during stress. The absence of pronounced EMG reactivity for group 1 might be interpreted as negating pathogenic stress responsiveness as an etiological mechanism. However, a recent study by Borgeat, Hade, Elie and Larouche (1984) suggests that tension headache subjects and headache free individuals do not differ in EMG levels during rest or voluntary muscle contraction. Rather, tension headache subjects simply show more pain sensitivity in response to sustained (voluntary) contractions of scalp muscles. Interestingly, the authors observed the presence of a subgroup of headache subjects who were maximally sensitive to the contractions, while others felt no discomfort. These data would fit with the different response patterns obtained for groups 1 and 2 in the present study.

Other psychophysiological variables failed to distinguish between groups at any point. For heart rate, skin temperature, frontal and temporal EMG, the stress conditions resulted in significant changes. Therefore, the manipulation was effective for all measures, omitting skin conductance and pulse volume. Yet, significant interactions for HR, frontal EMG, and ST indicated the Quiz produced more marked responses across all groups than the imagined stressor.

While groups were not differentiated at rest or during stress
on individual psychophysiological measures, when these were combined, a certain degree of psychophysiological patterning emerged. This was evidenced by the high degree of accuracy in classification of subjects (70.37%) based on three combined variables: temporal EMG, HR and SCL change scores. Unfortunately, these obtained patterns do not fit into any theoretical model for headache psychophysiology and are, therefore, difficult to interpret.

In conclusion, the present study identified two subgroups of tension headache patients, one with EMG increments from no-headache baseline to headache, the other without. These groups were undistinguishable based on headache EMG alone, non-headache EMG alone, headache index and headache disability ratings. The groups tended to differ in terms of their EMG stress reactivity patterns, though neither differed from controls. Significant psychological differences were documented between the groups, indicating the group without headache EMG pathology resembled a psychogenic or conversion headache type.

The results of this study are noteworthy in that they afford some clarification of discrepancies in the tension headache psychological and psychophysiological literature simultaneously. In addition, they confirm the idea that psychogenic/conversion type headaches are difficult to distinguish from muscle contraction headache and therefore commonly fused into one category. Though suggestive of differing etiologies for these two groups, the present investigation does not allow direct investigation of pathogenic
mechanisms. Based on the present findings, a plausible model would emphasize secondary gain, conditioning and modeling factors as causative factors for group 2. For group 1, pathophysiologic elevations in EMG may be involved in headache onset. These elevations may represent responses to stress, which though not excessive are accompanied with an increased sensitivity (as per Borgeat et al., 1984).

The present study did not directly address the etiological role EMG may play in headache, even for the muscle contraction group (group 1). It has recently been suggested that EMG may simply be a corollary of headache or a consequent factor (Pickoff, 1985; Ramirez, 1985). Nevertheless, the present findings are provocative and would indicate a need for further exploration of these two subgroups. Pickoff (1985) may be right in suggesting that muscle contraction is a cause of headache for some, a consequence for other, and absent for still others.

A number of limitations should be identified. First, controls in the present investigation were not yoked (i.e., did not receive two psychophysiological recordings). It would, however, be useful to determine base rates of cross-session EMG variability for headache free individuals. No headache to headache changes for tension headache subjects should be examined in light of these base rates. Second, only temporal and frontal EMG were recorded; perhaps, occipital and trapezius muscle should be sampled as well. To date, data on different site recordings are mixed. Yet, Pritchard and Wood (1983) have highlighted the danger of relying on
frontalis EMG alone.

A third problem concerns the fact that group 2 tended to have more elevated baseline EMG levels, although this difference was not significant. An ideal design would partial out subjects with constantly elevated EMG into a third group. Future research is needed to determine how individuals in this category might differ from the groups identified in the present study. Finally, future studies should consider reactivity both in the absence of and during headache. The combination of headache and stress has been shown to be important by several investigators (Hussey et al., 1985; Thompson et al., 1984). Etiological implications have yet to be delineated.

In spite of these limitations, the findings obtained in this effort are encouraging. Future research is clearly indicated and the present results are considered exploratory. A relatively new area of emphasis in tension headache research has certainly been identified which may lead to a refinement of both diagnostic procedures as well as subject-to-treatment matching.
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sufferers treated with cognitive therapy or biofeedback?

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Turcat, I.D., & Pettegrew, L.S. Development and validation of the


APPENDIX A

Paper and Pencil Measures
Zung Self-Rating Scale

Rate what proportion of the time you feel the following statements depict you, using a scale from 1 to 4 where:

1. A little of the time
2. Some of the time
3. A good part of the time
4. Most of the time

1. I feel down-hearted and blue
2. Morning is when I feel best
3. I have crying spells or I feel like it
4. I have trouble sleeping at night
5. I eat as much as I used to
6. I still enjoy sex
7. I notice that I am losing weight
8. I have trouble with constipation
9. My heart beats faster than usual
10. I get tired for no reason
11. My mind is as clear as it used to be
12. I find it easy to do the things I used to do
13. I am restless and can't keep still
14. I feel hopeful about the future
15. I am more irritable than usual
16. I find it easy to make decisions
17. I feel that I am useful and needed
18. My life is pretty full
19. I feel that others would be better off if I were dead
20. I still enjoy the things I used to do
Self-Evaluation Questionnaire

Directions: A number of statements which people have used to describe themselves are given below. Read each statement and then rate how you generally feel on the 1 to 4 rating scale below. There are no right or wrong answers.

1. I feel pleasant
2. I tire quickly
3. I feel like crying
4. I wish I could be as happy as others
5. I am losing out on things because I can't make up my mind soon enough
6. I feel rested
7. I am "calm, cool, and collected"
8. I feel that difficulties are piling up so that I cannot overcome them
9. I worry too much over something that really doesn't matter
10. I am happy
11. I am inclined to take things hard
12. I lack self-confidence
13. I feel secure
14. I try to avoid facing a crisis or difficulty
15. I feel blue
16. I am content
17. Some unimportant thought runs through my mind and bothers me
18. I take disappointments so keenly that I cannot put them out of my mind
19. I am a steady person
20. I get in a state of tension or turmoil as I think over my recent concerns and interests

<table>
<thead>
<tr>
<th></th>
<th>almost never</th>
<th>almost always</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
<td>20</td>
<td>1</td>
<td>2</td>
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</tbody>
</table>
For each statement below circle the number which indicates how much you either agree or disagree with it. Use the following scale:

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<thead>
<tr>
<th>Statement</th>
<th>Strongly Disagree</th>
<th>Strongly Agree</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. If I take care of myself, I can avoid illness</td>
<td>1 2 3 4 5 6</td>
<td>1 2 3 4 5 6</td>
</tr>
<tr>
<td>2. Whenever I get sick, it is because of something I've done or not done</td>
<td>1 2 3 4 5 6</td>
<td>1 2 3 4 5 6</td>
</tr>
<tr>
<td>3. Good health is largely a matter of good fortune</td>
<td>1 2 3 4 5 6</td>
<td>1 2 3 4 5 6</td>
</tr>
<tr>
<td>4. No matter what I do, If I am going to get sick I will get sick</td>
<td>1 2 3 4 5 6</td>
<td>1 2 3 4 5 6</td>
</tr>
<tr>
<td>5. Most people do not realize the extent to which their illnesses are controlled by accidental happenings</td>
<td>1 2 3 4 5 6</td>
<td>1 2 3 4 5 6</td>
</tr>
<tr>
<td>6. I can only do what my doctor tells me to do</td>
<td>1 2 3 4 5 6</td>
<td>1 2 3 4 5 6</td>
</tr>
<tr>
<td>7. There are so many strange diseases around that you can never know how or when you might pick one up</td>
<td>1 2 3 4 5 6</td>
<td>1 2 3 4 5 6</td>
</tr>
<tr>
<td>8. When I feel ill, I know it because I have not been getting the proper exercise or eating right</td>
<td>1 2 3 4 5 6</td>
<td>1 2 3 4 5 6</td>
</tr>
<tr>
<td>9. People who never get sick are just plain lucky</td>
<td>1 2 3 4 5 6</td>
<td>1 2 3 4 5 6</td>
</tr>
<tr>
<td>10. People's ill health results from their own carelessness</td>
<td>1 2 3 4 5 6</td>
<td>1 2 3 4 5 6</td>
</tr>
<tr>
<td>11. I am directly responsible for my health</td>
<td>1 2 3 4 5 6</td>
<td>1 2 3 4 5 6</td>
</tr>
</tbody>
</table>
Circle the response which best reflects your feelings with the statement. The following scale is to be used for your response to each item:

1. Strong agreement with statement
2. Moderate agreement with statement
3. Slight agreement with statement
4. Slight disagreement with statement
5. Moderate disagreement with statement
6. Strong disagreement with statement

1. I see doctors often 1 2 3 4 5 6
2. When ill, I have to stop work 1 2 3 4 5 6
3. I complain about being ill when I feel ill 1 2 3 4 5 6
4. Most people who know me are that I take medications 1 2 3 4 5 6
5. I stay in bed when I'm ill 1 2 3 4 5 6
6. I work fewer hours when I'm ill 1 2 3 4 5 6
7. Even if I don't feel ill at certain times, I find that I talk about my illness anyway 1 2 3 4 5 6
8. I do fewer chores around the house when I'm ill 1 2 3 4 5 6
9. Others often behave towards me as if I'm ill 1 2 3 4 5 6
10. I seek help from others when I'm ill 1 2 3 4 5 6
11. Although I very seldom bring up the topic of my illness, I frequently find myself involved in conversation about my illness with others 1 2 3 4 5 6
12. Others seem to act as if I am more ill than I really am 1 2 3 4 5 6
13. When I'm ill I work slower 1 2 3 4 5 6
14. My illness or aspects of it is frequently a topic of conversation 1 2 3 4 5 6
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</thead>
<tbody>
<tr>
<td>15. I leave work early when I'm ill</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>16. When I'm ill people can tell by the way I act</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>17. I avoid certain aspects of my job when I'm ill</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>18. Often I act more ill than I really am</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>19. I have large medical bills</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>20. I take rest periods when I'm ill</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>
APPENDIX B

Headache Disability Rating Scale
How much did this headache interfere with your daily routine?

(Place number in space provided)

1. did not interfere with my daily routine
2. some interference with my daily routine
3. interrupted my daily routine
4. had to go to bed as a result
5. had to go to doctor or emergency room as a result
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Supervisor: Phillip J. Brantley, Ph.D.
March, 1982 - Louisiana Correctional System. Conducted intelligence and educational testing, interviews of prison inmates. Funds provided from grant monies through LeHigh University. Supervisors: Frank M. Gresham, Ph.D. William F. Waters, Ph.D.


TEACHING EXPERIENCE:


CLINICAL SUPERVISION EXPERIENCE:

June, 1984 - Chief Extern and Coordinator of Psychology Consultation/Liaison Service, Earl K. Long Memorial Hospital. Responsible for direct case supervision for four clinical psychology graduate students, and coordination of patient referrals and case dispositions. The C/L Service receives over 200 cases/year exhibiting the full range of adult psychopathology. Supervisor: Phillip J. Brantley, Ph.D.

ADMINISTRATIVE EXPERIENCE:

1984-1985 Admissions Committee student representative (Behavioral Medicine track)

1985-1986 Intern Training Committee intern representative

PROFESSIONAL ORGANIZATIONS:

American Psychological Association
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Behavioral Medicine Special Interest Group of AABT
Southeastern Psychological Association
GRANTS:

1983  (Co-author) Behavioral Science, Research and Patient Education. A three year training grant for expansion of Louisiana State University Medical School Family Practice Residency program at Earl K. Long Memorial Hospital ($497,690 plus indirect costs). From National Institute of Health. Approved but not funded.

1983  (Co-investigator) Training Family Physicians to Prevent Patient Overutilization of Community Services. A one year research grant from the Department of Health and Human Services ($49,371). Approved but not funded.

RESEARCH EXPERIENCE:

1985  Research associate. Miriam Hospital Risk Factors and Chronic Pain Clinics. Member of interdisciplinary teams investigating assessment issues and behavioral treatment outcome for smoking cessation, weight loss and chronic pain. Responsibilities included data analyses and report writing.

1983-1985  Research assistant to Phillip J. Brantley, Ph.D. Investigations involving the psychophysiological, psychological and biochemical assessment and behavioral treatment of chronic headache, medical patients expectations from family doctors. Responsibilities included project design, data collection, data analyses and report writing.

1983-1985  Research associate. Louisiana State University. Member of a team investigating social perceptions and attitudes toward intelligence testing and special education. Responsibilities included project design, data collection, data analyses and report writing.

1983-1984  Research assistant to William F. Waters, Ph.D. Investigations of the psychophysiology of headache, validation of the Autonomic Nervous System Inventory. Responsibilities included project design, data collection, data analyses and report writing.

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PAPER PRESENTATIONS:


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Approved:

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