Journal of Behavioral Medicine, Vol. 13, No. 5, 1990

Cephalic Blood Flow Correlates of Induced Headaches

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Accepted for publication: December 23, 1989

The purpose of this study was to examine the cephalic blood flow patterns of subjects with frequent headaches during a headache induction procedure. Thirty-six subjects with muscle-contraction, migraine, or mixed headache symptoms were exposed to a 1-hr stressor designed to induce a headache while multiple cephalic blood volume pulse amplitude (BVPA) was measured. Thirty subjects reported a headache during the procedure, and the procedure was associated with significant changes in several cephalic BVPA measures. Between-group differences were found for several cephalic BVPA sites and there were significant correlations between induced headache activity and cephalic BVPA measures for most subjects. The results support causal roles for psychosocial stressors and cephalic blood flow in muscle-contraction and migraine headache.

KEY WORDS: cephalic blood flow; headaches; migraine; muscle contraction; stress.

INTRODUCTION

Recently published research has examined the psychophysiological correlates of muscle-contraction and migraine headaches (see reviews by Andrasik *et al.*, 1982; Anthony, 1988; Bakal, 1982; Cohen, 1978; Haynes, 1981:

This research was supported by NIH Grant 5R01NS18759.
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0160-7715/90/1000-0467$06.00/0

Haynes *et al.*, 1982; Sturgis *et al.*, 1981; and studies by Blanchard *et al.*, 1983; Bolardi *et al.*, 1988; Gannon *et al.*, 1981, 1987; Haynes *et al.*, 1983; Juge, 1988). Such efforts have been helpful in determining the pathophysiology of these headache types and in suggesting potential intervention strategies and targets (Blanchard and Andrasik, 1982, 1985: Beaty and Haynes, 1979).

Change in extracranial cephalic blood flow[4] in response to stressors has long been suggested as a possible mechanism to account for migraine pain and associated symptoms (Appenzeller, 1976; Drummond and Lance, 1983; Edmeads, 1977; Juge, 1988; Lauritzen *et al.*, 1983; McCaffrey *et al.*, 1986; O'Brian, 1971; Wolff, 1963). Cephalic vascular factors have also been suggested as a possible causal factor for muscle-contraction headache (Gannon *et al.*, 1987; Haynes *et al.*, 1982; Onel *et al.*, 1961). In several studies (Gannon *et al.*, 1981, 1987) significant differences have been noted in cephalic blood flow patterns between persons with and persons without frequent muscle-contraction headache and, among persons reporting frequent headaches, within persons, between headache and nonheadache states.

Changes in cephalic blood flow may cause headache pain in two ways: (a) through anoxia and elevated lactic acid concentrations associated with restricted blood flow (ischemia) and sustained contraction of the striated muscles in the neck and head region and/or (b) through compensatory distention of the cephalic arteries and accompanying events (e.g., increases in circulatory neurokinins, catecholamines, vasopressin, and platelet serotonin) in response to localized ischemia at other sites (Bruyn, 1980; Dalessio, 1982; Saper, 1983). These physiological pain mechanisms may be triggered by a sustained psychosocial stressor and/or by the termination of a stressor (as in a "rebound" cephalic artery–arteriole distention) (Adams *et al.*, 1980; Anthony *et al.*, 1967; Bakal, 1975; *et al.*, 1988; Sturgis *et al.*, 1981).

The hypothesized causal role of cephalic blood flow is congruent with both the vascular theory of migraine pathophysiology, which hypothesizes that migraine is primarily a result of changes in cephalic blood flow, and the neurogenic theory of migraine, which hypothesizes that both migraine and changes in cephalic blood flow are secondary to a central neurogenic dysfunction (Bruyn, 1980; Johnson, 1978; Malgren and Hasselmark, 1988; Spierlings, 1988). The hypothesized causal role of cephalic blood flow is also congruent with the muscle tension-vascular theory of muscle-contraction headaches, which proposes that muscle-contraction headache is primarily a result of sustained contraction of the muscles in the head and neck region and associated modifications in cephalic blood flow patterns. Finally, previous studies have not consistently supported the widely held belief that muscle tension lev-

[4]The term "cephalic," in reference to blood flow, is used as a term of convenience to refer to "extracranial cephalic."

els of the head and neck are higher in those who suffer from headaches compared to those who do not and are higher during headache states compared to nonheadache sites in chronic headache sufferers (Haynes *et al.*, 1982). Consequently, it is possible that the mechanisms responsible for the pain of muscle-contraction headache include a vasomotor system which is abnormally reactive to the biochemical changes associated with normal muscle tension levels rather than abnormally elevated muscle tension levels.

Examination of the vascular pathophysiology of muscle-contraction and migraine headache is clinically useful in two ways: (a) to help determine the degree to which these headache types are qualitatively different entities or represent only quantitative differences on continua of interest, such as intensity (see discussions by Bakal, 1975; Takeshima and Takahashi, 1988), and (b) to suggest methods of treatment alternative to those currently in use.

Despite promising findings on the role of cephalic blood flow in headache, many studies have not been designed in a manner that facilitates confident inferences about cause–effect relationships. Most previously published research has involved (a) between-group or between-headache state comparisons, (b) nonvascular psychophysiological measures, (c) stressors with a low ecological validity (e.g., of relatively short duration), (d) cross-sectional rather than longitudinal, and correlational rather than manipulation, experimental designs, and (e) a focus on nomothetic rather than idiographic analyses (Haynes, 1981). These studies have been helpful in identifying correlational relationships but do not exclude the possibility that observed changes in psychophysiological responses followed, rather than preceded, the headache state (Gannon *et al.*, 1987) and do not address individual differences in the causes of headaches.

To examine the relationship between cephalic vasomotor responses and muscle-contraction and migraine headache, the current study used a longitudinal "induced headache" design similar to that used by Gannon *et al.*, (1987). Subjects with frequent headaches were exposed to a 1-hr stressor that had been previously shown to cause headaches in 80% of subjects exposed to it. Multiple cephalic blood flow measures were taken before, during, and following the stressor.

METHOD

Subjects and Recruitment

Subjects were 36 persons (mean age, 29.5 years; 23 women) with frequent headaches with nonorganic etiology (17 were diagnosed as suffering from muscle-contraction headache, 5 from migraine, and 14 had mixed

symptoms). Potential subjects responded to bulletins, newspaper announcements, and announcements in university classrooms soliciting participants for a "headache research project." Each subject was paid $10 for participating.

Diagnostic Procedures

Diagnostic procedures and criteria were identical to those used by Gannon *et al.* (1987) and outlined by Diamond and Dalessio (1978), Dalessio (1982), and Haynes (1981). Diagnostic criteria included the presence or absence of prodromes, laterality and location of head pain, associated symptoms, response to vasoactive medication, duration and intensity, precipitating factors, and the results of previous medical examinations. All subjects were required to have seen a physician about their headaches in the past year. Subjects in the muscle-contraction headache group were required to have at least four headaches per week (mean, 5.7/week), for least 2 years (mean, 7.8 years), and no history of migraine headaches. Subjects in the migraine headache group were required to have at least two migraine headaches per month (mean, 3.9/month), for at least 2 years (mean, 13.4 years) and no more than one muscle-contraction headache per week (mean, .25/week). Subjects in the "mixed headache" group reported frequent headaches (mean frequency, 5.5/week) for at least 2 years (mean, 6.6 years) with symptoms that were consistent with both migraine and muscle-contraction headaches (e.g., bilateral occipital tension and nausea).

To be included in the study, each subject had to provide consistent information about his/her headache parameters during a telephone diagnostic interview and a lengthy personal interview. Any subject who provided inconsistent information across the two screening interviews was excluded from the study. Approximately 65% of the volunteers did not meet the reliability and/or diagnostic criteria.

Procedures[5]

Subjects who volunteered to participate in the research program were initially contacted by phone and the program outlined to them. Those who still expressed an interest in participating (none refused) were administered a 15-min telephone screening interview focusing on their headache symptoms.

[5]The procedures reported in this section are similar to those used by Gannon *et al.* (1987).

Those who met the diagnostic criteria of their respective headache groups (about 50% failed to meet these criteria) were scheduled for a personal interview and the headache induction session. During the personal interview, the exact procedures were again explained. To help minimize intrinsic demand to report a headache during the procedure, subjects were informed that the purpose of the study was to investigate the physiological responses of individuals during various conditions and that they may or may not experience a headache during the session. Each subject was shown the psychophysiology laboratory and those wishing to participate (none refused) signed an informed consent and were given a detailed diagnostic interview.[6] Those subjects who reported symptoms consistent with those reported in the first interview and with the diagnostic criteria for each group (four subjects were excluded from the study at this point) were then taken to the psychophysiology laboratory. Subjects were required to be headache free at the beginning of their laboratory session; those reporting a headache were rescheduled. Subjects who had ingested vasoactive beverages (e.g., coffee, cokes) within 2 hr of the session were also rescheduled.

The laboratory was 3 × 3 M. sound-attenuated and temperature-controlled (76–79°F) isolation room with a comfortable reclining chair (reclining at approximately 45°); the recording equipment was housed in a separate room. Subjects received all instructions via audio tape. Preliminary instructions included a description of the procedures, recording methods, and electrode placement. Electrodes and sensors were attached by two experimenters who were blind to the diagnostic group of the subject. The subject was then left alone and the following experimental phases ensued.

Baseline. Subjects were instructed to sit quietly, relax, and await further instructions. This phase lasted 15 min.

Stress. Subjects were informed that "in this phase we are attempting to see how accurately and quickly you can think." They were exposed to a cognitive stressor consisting of arithmetic problems (e.g., 237–349) every 15 sec for 1 hr. In addition, they were informed that if their performance fell below the average for college sophomores, they would hear a buzzer. Buzzes (approximately 50 dB) were presented 22 times throughout the hour on a set variable-interval schedule. This stressor has been used in numerous studies and shown to result in significant increases in multiple psychophysiological indices, subjective reports of stress, and headache reports by about 80% of subjects (Gannon *et al.*, 1987).

Recovery. Following the last arithmetic problem, subjects were informed that no more problems would be presented and were instructed to relax and await further instructions. This phase lasted 10 min.

[6]This structured interview has been used in several previous studies (e.g., Gannon *et al.*, 1981, 1987; Haynes *et al.*, 1983) and been shown to have a reasonable degree of predictive validity.

Following the recovery phase, subjects were informed that the session was over, debriefed about the experimental procedures, and encouraged to use whatever methods they found effective in alleviating headaches that may have developed. Referral for medical attention was available but none of the headaches reached a severity where referral was appropriate; no subjects expressed the desire for medical attention, and all subjects indicated their ability to successfully deal with their headaches.

Apparatus and Dependent Measures

Headache Pain Ratings. These were taken nine times during each session: (a) immediately after sensors were attached and the experimenters left the room (prebaseline), (b) at the end of the baseline condition (postbaseline), (c) at the end of every 10-min period during the stressor, and (d) at the end of the recovery condition. At each sampling point, subjects responded to a tape-recorded query, "Please rate your current headache activity on a 10-point scale, with 0 representing no headache and 10 representing an extremely painful headache.

Six cephalic blood volume pulse amplitude (BVPA) measures were taken continuously during the experiment: two supratrochlear sites (above the eyebrows), two superficial temporal sites (at the bifurcation of the temporal artery), and two spinalis–semispinalis sites (by the second cervical vertebra). These sites were selected for measurement on the basis of previous hypotheses regarding the pathophysiology of headache and/or pain site involvement (Bakal, 1982; Dalessio, 1980; Gannon *et al.*, 1987; Haynes *et al.*, 1982).

Psychophysiological data were collected utilizing Grass photoelectric reflectance plethysmographs, a Grass polygraph (7-D), and low-level DC preamplifiers (Model 7PIB, TC = 0.1). All BVPA data were initially recorded in analogue form using a Hewlett–Packard six-channel FM tape recorder. The data were then fed through a computer-generated A–D conversion system employing an Apple IIe and an Issacs A-D converter which continuously sampled BVPA waveforms, computed peak-to-valley amplitudes, and excluded probable artifacts.

To ensure consistency across subjects in the placement of sensors, experimenters used illustrated protocols and carefully measured sites. All measures were tested prior to the beginning of the experimental phases to ensure scorable responses and initial BVPAs were set at 2-cm peak-to-valley amplitudes. To minimize movement artifact, sensors were attached at the measurement site with transparent double-sided tape; additionally, surgical tape was placed over and around the sensor to provide additional placement security.

Data Reduction and Analysis

BVPA data used in the statistical analyses were the means of the four 15-sec periods preceding a headache report. Data were analyzed as either absolute values or change scores. When using absolute values, larger values indicated greater vasodilation; when using change scores, larger values indicated greater vasoconstriction (e.g., baseline BVPA − stressor BVPA). Because the psychophysiology measures involved six conceptually related dependent variables, they were subjected to an overall MANOVA with Hoteling–Lawley Trace, followed by appropriate ANOVAs and post hoc tests. Preliminary analyses revealed no main or interaction effects for gender, and gender was not entered as a variable in subsequent analyses.

RESULTS

Subjective and Psychophysiological Responses to the Stressor

The mean headache reports across the three phases of the experimental session are illustrated in Fig 1. Eighty percent of the subjects developed headaches during the 1-hr period. The overall MANOVA on BVPA change scores indicated a significant effect for time [$F(54,2036) = 2.04, p < .0001$] and follow-up ANOVAs indicated significant effects for time for the right temporal [$F(8,263) = 4.88, p < .001$], left temporal [$F(8,255) = 2.29, p < .05$], and right spinalis [$F(8,263) = 2.16, p < .05$]. Post hoc Scheffe analyses indicated that these results were attributable to significant cephalic vasoconstriction during the second to seventh stressor periods, compared to baseline.

Between-Group Differences in Psychophysiological Responses to the Stressor

During the 60 min stressor 16 of 17 muscle-contraction headache subjects, 13 of 14 mixed headache subjects, and 2 of 5 migraine headache subjects reported a headache.

An overall MANOVA for all subjects and follow-up ANOVAs indicated significant between-group differences for three cephalic BVPA sites: left frontal [$F(2,32) = 3.68, p < .05$], right temporal [$F(2,33) = 4.22, p < .05$], and right spinalis [$F(2,33) = 7.03, p < .01$]. As illustrated in Figs. 2–4, the muscle-contraction and mixed headache groups were characterized

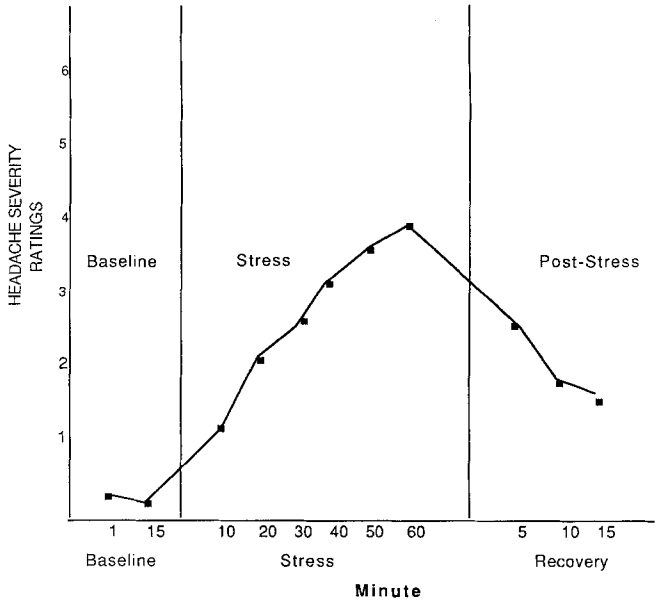

Fig. 1. Mean headache reports (10-point scale) across the three phases of the experimental session.

by cephalic vasodilation and the migraine headache group by vasoconstriction during the stressor.

Correlates of Induced Headaches

A correlation matrix was constructed to examine the linear association among headache activity during the stressor, cephalic BVPA responses during the stressor, and headache activity reported during the initial interview, for all subjects combined. This resulted in 66 correlation coefficients, of which 46 were significant at the .05 level (4 significant correlations would be expected by chance). Some of these results are summarized below.

The *frequency* of daily headaches reported during subjects' initial interview was significantly correlated with the mean [$r(34) = .45$, $p < .05$] and maximum [$r(34) = .42$, $p < .05$] severity of induced headaches. There were no significant correlations between duration or intensity of the headaches reported during subjects' initial interview and induced headache activity, nor were there significant correlations between frequency and severity of headaches reported during subjects' initial interview and any BVPA measure. However, the *duration* of headaches reported during subjects' initial in-

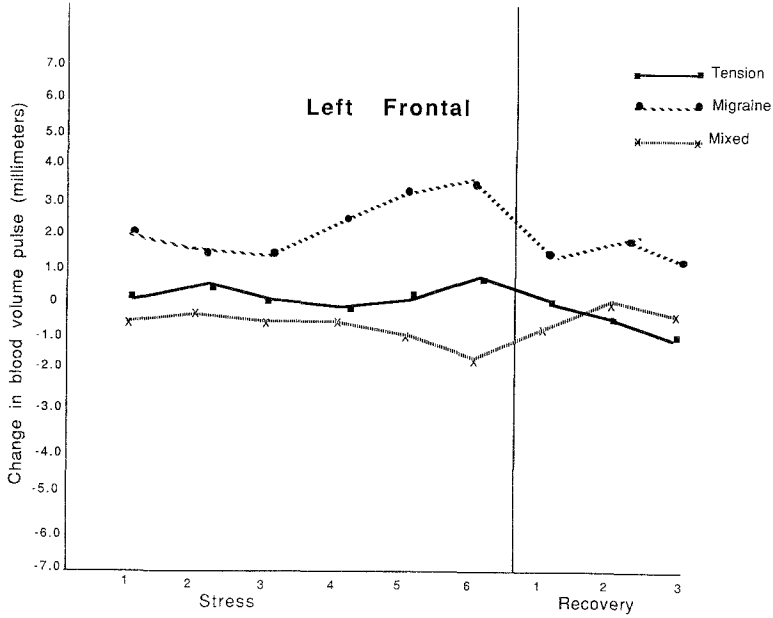

Fig. 2-4. Change scores in BVPA across measurement periods by group. Higher change scores = greater vasoconstriction.

terview was significantly correlated with greater constriction during the stressor for the left frontal site [$r(34) = .34, p < .05$] and right temporal site [$r(34) = .38, p < .05$] and less poststress recovery for the left frontal site [$r(35) = .37, p < .05$].

Interestingly, there was a significant inverse correlation between a family history of headaches and the probability [$r(34) = -.42, p < .05$], peak intensity [$r(34) = -.33, p < .05$], mean intensity [$r(34) = -.32, p < 05$], and latency (minutes of the stressor before a headache was reported) [$r(34) = -.52, p < .05$] of induced headaches.

Within-Subject Correlations

In light of the present authors' emphasis on individual differences in causal variables and causal paths, a correlation matrix including cephalic BVPA and induced headache activity variables was constructed for each subject who reported an induced headache. Because of possible serial dependency and the small number of data points (nine), the results of these analyses should be considered exploratory.

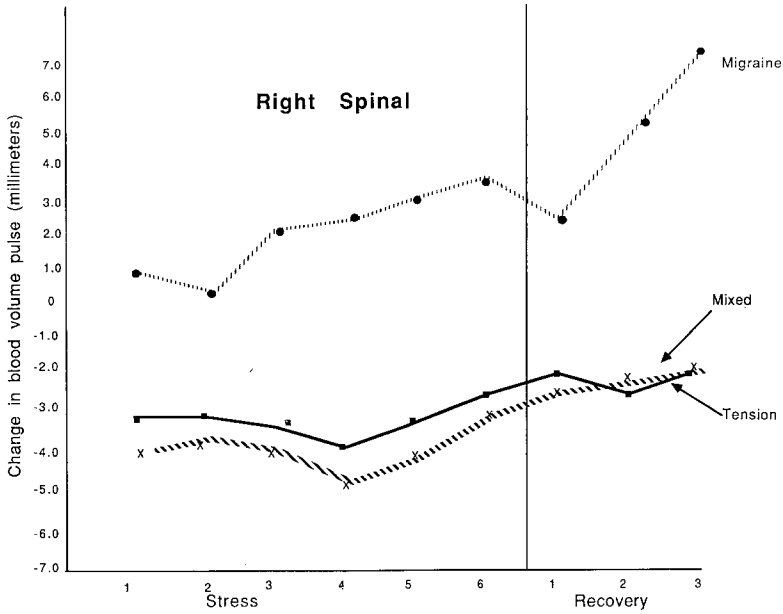

Fig. 2-4. Change scores in BVPA across measurement periods by group. Higher change scores = greater vasoconstriction.

There were significant correlations between induced headache reports and cephalic BVPA measures for most subjects and the number of significant correlations exceeded that expected by chance. However, no consistent pattern of relationships emerged. For example, a number of subjects demonstrated significant relationships (e.g., r = .60, .68, .69) between maximum headache ratings and right and left frontal BVPA responses during stress; other subjects demonstrated equally significant correlations between headache ratings and BVPA responses at other sites. Some subjects demonstrated significant correlations with induced headache reports for almost all cephalic BVPA, others for only a few BVPA measures, and nine subjects demonstrated no significant relationships.

DISCUSSION

This is the second study (see also Gannon *et al.*, 1987) to demonstrate that headaches can be *induced* by a psychological/environmental stressor. Although a causal role for environmental stressors and headache has long been hypothesized, previous studies have relied on correlational, rather than

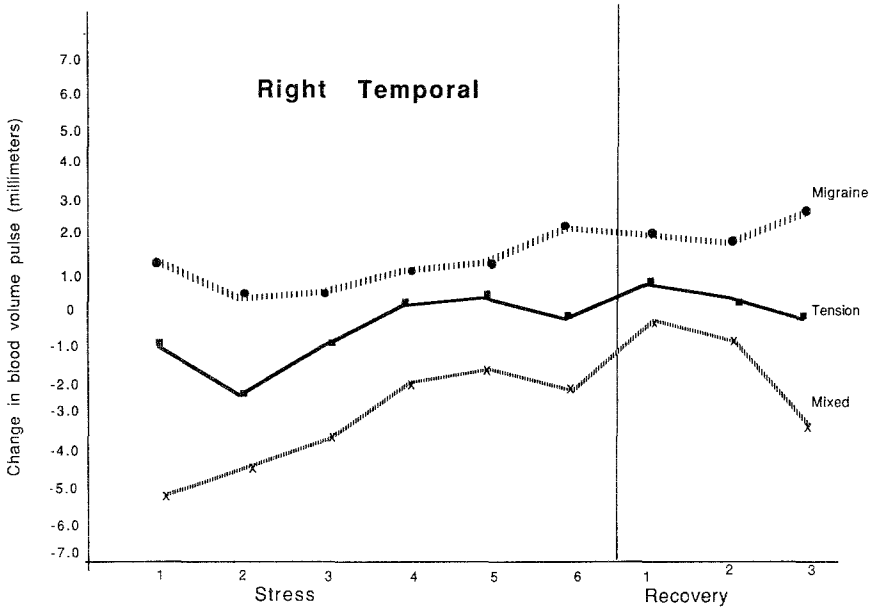

Fig. 2-4. Change scores in BVPA across measurement periods by group. Higher change scores = greater vasoconstriction.

manipulation, research designs for support. The hypothesis that headaches can be triggered by psychological/environmental stressors is also strengthened by recent time-series studies (Dalkvist *et al.*, 1983; Levor *et al.*, 1986) which have demonstrated that daily stressors often *precede* headache occurrence for some persons.

The present results also support the popular belief about the relative importance of stress as a cause of muscle-contraction and migraine headache. Migraine is believed to have a stronger genetic component than has muscle-contraction headache, and in the present study, a family history of headache was associated with less headache intensity during the stressor.

The results from this study are consistent with those from other studies (Gannon *et al.*, 1981, 1987; Haynes *et al.*, 1987; Onel *et al.*, 1961) that muscle-contraction headache may be associated with significant modifications of cephalic blood flow patterns. Whether these changes are a primary causal variable of head pain, and/or correlates of central autonomic or neurotransmitter processes, remains unresolved. The findings suggest, however, that clinical attention to cephalic blood flow may serve as an adjunct or alternative to traditional methods of treatment involving muscle-tension biofeedback and relaxation training.

Several previous studies (see review by Haynes *et al.*, 1982) have suggested that the temporal artery may be particularly reactive to psychosocial stressors and the results of this study are consistent with that hypothesis. Additionally, the results of this study are consistent with the hypothesis that temporal artery dilation may occur as a response to lower level vasoconstriction.

The results of this study also supported the importance of individual differences in the pathophysiology of headache. Significant correlations between induced headaches and cephalic blood flow patterns were found for most subjects. However, the specific cephalic BVPA patterns that significantly correlated with headache activity differed across subjects and were unrelated to self-reported pain site.

The between-group differences in cephalic blood flow patterns during the stressor (vasodilation by migraineurs, vasoconstriction by others) suggest that different pathophysiological mechanisms may be involved for subjects with muscle contraction vs. subjects with migraine symptoms. Between-group differences in the direction of BVPA response to the stressor are difficult to interpret without additional data on the characteristics of headaches reported during the induction procedure. The headaches experienced by the migraine subjects, for example, could have been either muscle-contraction or migraine.

Caution in the interpretation of the results of this study are warranted for several reasons: (a) although the subjects would be described as "severe" by most diagnostic criteria, they were not "clinical" cases because they were not seeking treatment at the time of the study, (b) the sample sizes were not optimally large, (c) cephalic BVPA patterns may vary across stressors, (d) unmeasured arteries may play an important role in the pathophysiology of chronic headache, (e) the factors controlling vasomotor responses are not established, (f) the degree to which cephalic BVPA patterns in this analogue situation and in the natural environment are correlated has not been established, and (g) differential responses to stressors of headache and non-headaches subjects were not addressed in this study.

ACKNOWLEDGMENT

The authors would like to express their appreciation to Dennis Molfese for valuable technical assistance.

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