Mashayekhi, Nancy

LATERALIZED CLASSICAL MIGRAINE: A CONTROLLED INVESTIGATION OF COGNITIVE AND EMOTIONAL DIFFERENCES

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LATERALIZED CLASSICAL MIGRAINE: A CONTROLLED INVESTIGATION OF COGNITIVE AND EMOTIONAL DIFFERENCES

by

NANCY MASHAYEKHI

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4/25/87 ______________ Louis J. Gerstman
date Chair of Examining Committee

April 27, 1987 ______________ Herbert D. Saltzstein
date Executive Officer

Jane Healey Ph.D.

Alfred Frontera M.D.
Supervisory Committee

Paul Fedio Ph.D.

Crawford Clark Ph.D.
Outside Readers

The City University of New York

ABSTRACT

LATERALIZED CLASSICAL MIGRAINE: A CONTROLLED INVESTIGATION OF COGNITIVE AND EMOTIONAL DIFFERENCES

by

Nancy Mashayekhi

Advisor: Dr. Louis Gerstman

Although the primary cause of migraine is still elusive, it is generally agreed that the autonomic, sensory and motor disturbances frequently accompanying an attack, originate from central nervous system dysfunction.

A notable feature of some syndromes, is unilateral aura and pain, factors not previously considered. This study of lateralized classical migraineurs [sharply defined prodromes and contralateral neurological disturbances] examines affective and cognitive correlates of locus of pain in migraine.

Migraineurs are often characterized as obsessive, compulsive, success oriented, prone to depression and humorless. Personality tests as the MMPI and Rorschach have failed however, to distinguish a migraine personality. One of the problems may be that the diagnostic

categories of these measures are based on characteristics of psychiatric patients, not differences from a normal population. Patterns that might be characteristic, albeit not pathologic, may be overlooked.

These traits may result from the neurological process which precipitates the migraine attack and may closely approximate those distinctive personality alterations seen in patients with lateralized hemispheric injury. Therefore, the Bear and Fedio Personal Inventory was chosen for this study because it was designed to determine differences in psychosocial behavior between patients with lateralized neurological dysfunction, in particular, temporal lobe epileptics. Since specific clusters of behaviors differentiated the right focus from the left focus patients, this measure seemed more appropriate than standard personality tests for discerning the possibility of a migraine "personality".

Evidence of neurological dysfunction in lateralized classical migraine suggested the possibility of distinct cognitive sequelae, therefore migraineurs were compared on neuropsychological measures selected for their sensitivity to lateralized neurological disorders.

Subjects with predominately unilateral migraine and normal subjects with uncomplicated tension headaches were compared on the Personal Inventory. Classical migraineurs scored higher than the tension headache group on six traits. Almost all differences were attributable to right migraineurs who scored higher than left migraineurs and the control group on 4 traits [emotionality, anger, obsessionalism and viscosity], and higher than control subjects on guilt, sadness and paranoia.

Virtually no differences were found between groups on neuropsychological measures.

It was concluded that the results of this study support a subcortical model of migraine.

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Important knowledge about the functional asymmetry of the cerebral hemispheres has been gained by the behavioral assessment of patients with central nervous system insult. Early observations of patients with discrete lesions of the left hemisphere revealed impairment of speech and related language disorders, whereas language disturbances were rarely seen in patients with right hemisphere lesions. Expressive language deficits result from lesions in the frontal lobe [Broca, 1865], while lesions in the temporal and parietal language areas produce receptive language deficits [Wernicke, 1874].

Unique functions of the right hemisphere were noted as early as 1867 [Quaglino & Borelli]. In the absence of visual impairment, right hemisphere lesions were found to impair patients' ability to recognize objects or persons, and produced deficits of visual spatial organization, visual memory and visual construction [Badal, 1888; Jackson, 1874; Kimura, 1961; Kleist, 1916; Milner, 1958; Weisenberg & McBride, 1935].

Development of commissurotomy techniques, in cases of intractable seizures, permitted the study of the behavior of patients with relatively intact but separate hemispheres. The results of these studies confirmed earlier observations that the two hemispheres contribute quite differently to cognitive function [Bogen & Vogel, 1962; Gazziniga, 1970; Hecaen, 1962; Milner & Taylor, 1972; Nebes, 1974; Sperry, 1961].

The evidence of predominately left hemisphere involvement in language functions and right hemisphere mediation of nonverbal, visual perceptual functions has led to countless studies of brain-behavior relationships. The most important impression however, was a caution not to attribute an all or nothing approach to laterality of function [Gazzaniga, 1977]. Individual differences are well documented, particularly in left handers [Goodglass & Quadfasel, 1954] and females [Bryden, 1979; McGlone, 1980]. Further, right hemisphere lesions do not completely destroy nonverbal functions and left hemisphere damage leaves some residual language. Bogen & Vogel [1962] demonstrated that the right hemisphere has some capacity to comprehend language though it can not speak. Therefore, the two hemispheres work in reciprocal fashion, in concert to subserve not only cognitive but emotional behavior.

Functional asymmetry may even extend to subcortical structures. Thalamic stimulation and post thalamotomy studies suggest that the left thalamus is more involved in language functions, whereas the right thalamus seems to contribute to the recognition of complex visual patterns [Darley, Brown & Swenson, 1975; Fedio & Van Buren, 1975; Gorelick, Heir, Benevento, Levitt & Tan, 1984; Henderson, Alexander & Nasser, 1982; Ojemann, 1976; Riklan & Cooper, 1975; Riklan & Levita, 1965]. Milner [1973] demonstrated a differential contribution of the mesial temporal structures, especially the hippocampus to memory. Removal of this structure on the left led to verbal memory difficulties while removal on the right interfered with nonverbal memory.

Anatomical, structural, chemical [Glick, Ross & Hough, 1982] and developmental asymmetries of the central nervous system abound and their influence on cognition is as yet, not fully understood. Geshwind & Galaburda [1985] present a comprehensive review of investigations in this area as well as some intriguing hypotheses for further research.

Interest in a possible relationship between affective states and hemispheric laterality was initiated by the observation of catastrophic reactions in patients with left hemisphere damage [Goldstein, 1939] and indifference reactions, often associated with unilateral neglect, in patients with right hemisphere damage [Denny-Brown, Meyer, & Horenstein, 1952; Hecaen, Ajuriaguerra & Massonnet, 1951].

Today there is a body of evidence which suggests a differential representation of emotional processes within the two hemispheres. The relevant data is derived from a broad spectrum of neuroscience research, ranging from lateralized biochemical processes to clinical descriptions of affective states in patient with lateralized pathology.

In fact, cognitive and affective disturbances that correlate with unilateral cerebral insult have been well documented in the more severe neurological disorders including temporal lobe epilepsy [Bear & Fedio, 1977; Flor-Henry, 1969a, 1969b; Geshwind, 1973; Serafetinides, 1965]. However, the sequelae of classical migraine, a common syndrome of lateralized headache with contralateral disturbances of cerebral function, have not been evaluated. Preliminary evidence suggests that although classical migraine is considered a minor neurological problem [rarely causes permanent physiological damage], there may be permanent affective and neurocognitive consequences which correlate with the side of the headache [Mashayekhi & Berger-Gross, 1982, 1983].

Pathogenesis and Etiology of Migraine

Syndromes involving paroxysmal unilateral cephalgia (head pain) have been described in medical literature for many centuries. In the 2nd century A.D. the term hemicrania was introduced to designate unilateral vascular headaches and their constellation of symptoms which occur in the absence of known pathology. This term has been modified, particularly from the French pronunciation "emicrania" and today, this familial disorder is more simply known as migraine, a group of syndromes which affects 25-29% of women and 15-19% of men in the general population [Waters, 1975].

In spite of a long history, the etiology and pathogenesis of migraine is still not well understood. Many syndromes ranging from mild, almost imperceptible symptoms to permanent neurological disturbances are associated with migraine, and no one theory, be it physiopathologic or biochemical, satisfactorily explains the manifestations of this disorder [Bruyn, 1976].

It is generally believed however, that in all the variants, abnormal cranial vascular changes occur prior to and during the attack, independent of systemic blood pressure and in the absence of known intracranial pathology. Although paroxysmal throbbing pain, frequently unilateral, is the usual presenting complaint, the neurological symptoms which may precede or accompany this event implicate a complex vasomotor disturbance.

Most commonly,the pain occurs in frontal, superorbital, temporal or parietal areas of the head. The headache is usually unilateral at least at onset [Bille, 1962; Heyck, 1981; Lance & Anthony, 1982] and may be associated with nausea, vomiting and hypersensitivity to noise, light or odors.

In classical migraine, there is a well defined prodrome or "aura" during which the migraineur experiences contralateral sensory or motor disturbances such as scintillating scotoma [luminous flashes, streaks or spots of light], paresthesia [abnormal sensations], hemianopsia [defective vision in one half of the visual field], tremors, stupor [Lee & Lance, 1977] or speech difficulties. If pain is experienced more posteriorly in the occipital regions, the prodroma may also include ataxia [loss of muscular coordination], vertigo, and visual disturbances such as nystagmus [involuntary rapid movement of the eyeball] and occasionally transient blindness [Bickerstaff, 1961; Scharf & Buntale, 1975].

In contrast, the characteristic throbbing headache of common migraine occurs without the "aura" experienced in classical migraine. The prodroma of this syndrome are vague, and edema, dysphoria or abdominal pain may precede the headache by hours or days.

Other variants include opthalmoplegic migraine, in which the unilateral pain is accompanied by oculomotor palsy arising from the third cranial nerve. Hemiplegic migraine involves the loss of one side of the visual field and is some cases, unilateral motor or sensory loss [Andersson, 1974; Charcot, 1890; Gilbert, Rappaport & Trump, 1974]. In some cases these signs may become permanent [Connor, 1962; Silverberg & Latkes, 1974]. Leon-Sotomayor [1974] has described a syndrome designated as "cardiac migraine", that is a combination of angina pectoris, in the absence of organic disease, and headache. He suggests that the chest pain is due to the same type of arterial spasm which produces migraine. Isolated visual migraine is characterized by perception of scintillating zig-zag lines spreading from central to peripheral areas or a bilateral central or paracentral scotoma and headache [Rydzewski & Pozniak-Patewica, 1975].

In children, migraine may be manifest as cyclic vomiting, abdominal pain, motion sickness and not uncommonly, as acute confusional states [Ehyai & Fenickel, 1978; Gascon & Barlow, 1970; Rabe, 1974; Riggs, 1973; Rothner 1979]. Some children also display abnormal 14 and 6 cycle per second EEG patterns [Edmeads, 1979; Essman, 1978; Whitehouse, Pappas, Escabe & Livingston, 1967]. This combination of symptoms is often difficult to distinguish from the epilepsies.

The many variants of cluster headache or [Horner's syndrome] may also be related to migraine. These unilateral headaches occur once or more daily and are accompanied by miosis [pupil contraction], ptosis [drooping of the eyelid] and tearing of the ipsilateral eye [Ekbom, 1974; Kudrow, 1974]. These so called clusters may persist for several days or weeks followed by remissions of weeks, months or years. There are usually no prodromal or premonitory symptoms associated with cluster headache. It should be noted that classical and common migraine may follow this cyclic pattern as well.

Periodically, the prodromal symptoms may present alone as migraine equivalents. Scintillating scotoma, abdominal pain, nausea, vomiting, diarrhea, vertigo, edema and psychic disturbances of sleep, mood and behavior have been reported to occur in the absence of headache [Friedman, 1972a; LaWall & Ooman, 1978; Reiman, 1973; Rydzewski, 1975].

A differential diagnosis between migraine and other recurrent or unilateral head pain can be made by a careful evaluation of the patients history, with special note made of the quality, frequency, duration, location and intensity of pain and any associated symptoms [Kudrow, 1976; 1979]. Other types of functional headaches i.e. tension, muscle contraction and those associated with depression and anxiety can be differentiated by their lack of complex symptomatology [aura], difference in quality and distribution of pain, are invariably bilateral and do not respond to antimigraine drugs [e.g.

ergotamine]. Temporal arteritis [inflammation of the temporal artery] presents as a unilateral headache centered around the affected artery but unlike migraine, there are general systemic reactions to a low grade infection [i.e. fever, weight loss and always a change in the sedimentation rate of white and red blood cells in peripheral blood]. The artery is prominent, nodular and extremely tender to pressure. Periodic headache accompanied by nasal stuffiness and painful sinuses suggests a diagnosis of acute sinusitis. Intracranial lesions such as vascular malformations, aneurysms [sacs formed by the dilatation of a vascular wall] angiomas [vascular tumors] and other expanding lesions often present as headaches which are similar to those of migraine; paroxysmal and always unilateral. These cases can be clarified by skull x-rays, cerebral angiograms and CAT-scan.

Classically, the prodroma of migraine is believed to be the result of hypoxia caused by vasoconstriction of intracranial and retinal arteries. The progression of neurological symptoms over several minutes excludes neuronal transmission and the rate of depression corresponds to that which spreads over the cortex after chemical or electrical stimulation [Leao, 1944] and resembles the Jacksonian March of epilepsy [Bucking & Baumgarten, 1974; Hare, 1966; Lashley, 1941; Milner, 1958]. Wolff [1962] demonstrated that inhalation of small amounts of amytal nitrate or 10% carbondioxide-oxygen mixture, both powerful cerebral vasodilators, will relieve the prodromal phenomena and in some cases prevents the progression of the attack. Larger amounts of amytal nitrate induce hypotension as well as vasodilatation. This reduces cerebral blood flow and as a result, the aura is intensified. However it has been demonstrated that there is an increase in cortical blood flow during spreading depression and a reduction, below normal, of 20-25% afterwards [Lauritzen, Jorgenses, Diemer, Gjedde & Hansen, 1982]. Further, positron emission tomography [PET], of patients with transient ischemic attacks, revealed that cerebral oxygen metabolism is maintained by local compensatory mechanisms in spite of the reduction in cerebral blood flow [Powers, Grubb & Raichle, 1984].

Regional blood flow [rCBF] studies indicate changes in intracranial circulation during a migraine attack [Edmeads, 1979; O'Brien, 1971, 1873]. During the prodroma, there is reduced rCBF bilaterally, but more marked in the area which relates physiologically to the manifest symptoms. Carotid angiograms obtained during the aura of migraine reveal no evidence of vasoconstriction although there is reduced blood flow [Edmeads, 1979]. The angiogram also remains normal when blood flow is increased in the headache phases, that is, no vasodilatation is apparent. The vascular changes must therefore take place in the arterioles which regulate cerebral blood flow, but are too small to be visualized. Xenon clearance studies of migraine patients also indicate that CBF is significantly reduced during the prodrome and increased in the headache phase. The entire hemisphere was affected although the angiograms were normal [Mathew, Hrastnik & Meyer, 1976; Sakai & Meyer, 1979; Skinhoj, 1973]. This evidence is contrary to that which is found in spreading cortical depression.

Differences between classical and common migraineurs are now being demonstrated. In classical migraine, there is a short focal increase in cerebral blood flow prior to the reduced flow; no changes were apparent at any stage of the common migraine attack [Olesen, Lauritzen, Tfelt-Hansen, Henriksen & Larsen, 1982]. These authors conclude that the ischemia recorded was not enough to cause the symptomatology of the migraine aura. These results suggest that migraine is a neurogenic event as spreading depression originates from cortical neurons, not vascular changes [Edmeads, 1982].

The painful phase of migraine is more difficult to interpret as the location and intensity of head pain is not helpful in identifying its source. Pain from both intra and extra cranial tissues may be felt in the same place and extend well beyond the initial source [Friedman, 1979b]. Dilatation of intra and extra cranial arteries has been reported by some investigators and an increase in blood flow is present during headache. In 1938, Graham and Wolff reported that the intensity of pain was parallel to the pulsation amplitude of large temporal arteries, branches of the external carotid artery. An injection of a vasoconstrictor reduced pulse amplitude and pain. This relationship can also be demonstrated in some patients who can obtain headache relief by learning to reduce temporal pulse amplitude through biofeedback training [Diamond, 1979; Jessup, 1978; Sargent, Green & Walters, 1972; Steiner & Arkin, 1978] or by compression of the artery itself [Drummond & Lance, 1982]. This phenomena, however, does not verify that the locus of pain is extracranial. Blood vessels do not operate independently and the action of vasoconstrictors or biofeedback on the rest of the vascular system cannot be ignored The reduction of pain may be only incidentally related to temporal pulsation change and both may be the result of intracranial vascular responses. Recently, plethysmography tests have demonstrated that the temporal artery on the headache free side is equally dilated [Rose & Amery, 1982]. Furthermore, dilatation of the temporal artery following bypass surgery, for vascular disease, does not induce headache [Kletter. 1979; Myers, Boone & Gregg, 1982].

In fact, vasodilatation is a normal sequelae to vasoconstriction and does not in itself produce pain. Dilatation following cessation of long term infusion of norepinephrine does not produce headache [Wolff, 1972]. Normal activities such as prolonged exercise, soaking in a hot tub or overeating can also induce vasodilatation without pain. These observations led Wolff to conclude that in addition to vasodilatation, local aseptic inflammation and substances which lower pain threshold must also be present to produce pain. A bradykinin type polypeptide which lowers pain threshold was subsequently identified in the fluid aspirated near the painful temporal area. It was also found, though in lesser quantities, in fluid taken from the opposite temporal area where there was no pain.

The rCBF studies indicate that increased blood flow in cerebral vessels may out last the headache by several days [Edmeads, 1979]. How ever, since the arterioles can not be visualized, there may be a reduction of actual vessel size without a measurable decrease in CBF. The angiogram studies have shown that blood flow may vary without a change in large vessel caliber. Unfortunately, most of the rCBF studies are of questionable value in determining blood flow changes during aura or headache. At present, these measures lack sufficient sensitivity to accurately record changes and may give evidence of an increase in flow when there is actually a decrease [Sadove, 1979]. Clearly the scanning of migraineurs during aura and headache with positron emission tomography would be more helpful in clarifying blood flow changes.

Blau [1978] argues that the vasomotor instability of the meningeal circulation is responsible for both the aura and headache. His examination of Wolff's pulse wave tracings, reveals that there was no difference between pulsation amplitude 36 to 72 hours prior to the migraine attack and at the time of the most intense pain. Further, these tracing have never been replicated [Hecyk, 1956]. Blau [1981] points out that in temporal arteritis, where inflammation is present, headache is prominent but none of the other symptoms which occur during the migraine headache are produced. Headache, vomiting and photophobia are however, the hallmarks of meningitis. Migraine pain is exacerbated by a rise in intracranial venous pressure as demonstrated by the Valsalva maneuver, coughing, sneezing or sudden head movement. This phenomenon is the hallmark of a lumbar puncture headache which can only arise intracranially. This suggests that migraine pain arises from pressure changes in the dural venal sinuses which have nocioceptive nerve endings along their whole intracranial course. Blau's evidence has cast considerable doubt on the extracranial locus of pain theory and, although the question is still being debated, most neurologists now agree, that the pain of migraine is of intracranial origin.

Attacks of migraine may be precipitated by various factors including stress, fatigue, alcohol, bright or flickering lights, changes in barometric pressure, or sensitivity to foods containing tyramine. This has led to such divergent theories of etiology as instability of the blood brain barrier [Harper, MacKenzie, McCulloch & Pickard, 1977]; blood platelet abnormality [Dalsgaard-Nielson & Genefke, 1974; Damassio & Beck, 1978]; epileptiform discharges [Basser, 1969; Lennox, 1960; Sommerville, 1976; Whitehouse et al, 1967]; disorder of central inhibition [Dalessio, 1979; Elliott, Frewin & Downey, 1974; Selby & Lance, 1960]; hormonal imbalance [Grant, Carroll, Goodwin & Pryse-

Davis, 1974; Horrobin, 1977; Sommerville, 1972a, 1972b]; faulty metabolism [Hannington, 1978] and food allergies [Monod, 1975; Ryan, 1974].

Despite all these theories of pathogenesis and etiology of migraine, no satisfactory explanation has been proposed which can account for the predominately lateralized neurological symptoms and locus of pain, or for the premonitory and prodromal symptomatology and precipitating factors. Bruyn [1982], speculates that the vascular response in cortex may be conciliatory to changes in branches of the basilar artery which supplies upper brain stem structures. He suggests that since parsimony is the main guideline in neurology, the smallest area which could account for all of the sensory, visual, motor, speech, autonomic and mental symptoms in migraine is that area of the upper brain stem which includes mesencephalic gray, basal thalamus, lateral geniculate body, posterior limb of the internal capsule, hypothalamus and hippocampus. Nothing is known about the balance between carotid and basilar perfusion, vasoactivity and its neurogenic control [Bruyn, 1982], but the notion of brain stem involvement should stimulate new investigations and observations in migraine. Clearly, migraine is not a simple vascular headache but rather the end result of a complex pattern of dysfunction within the central nervous system.

Psychopathology of Migraine

One of the most persistent and controversial notions in the study of migraine is psychogenic origin, that neurotic mechanisms are pathogenic to migraineurs. The putative interaction of psycho-social traits with physiologic factors can be summarized as follows. Individuals who are genetically endowed with abnormal microvascular reactivity to internal or external stress are prone to have conflicts between the desire to be dependent and repudiation of these wishes by their adult sense of pride [Graham; Rogado, Rahman & Cramer, 1970] and /or manifest obsessive compulsive traits as reaction formations to repressed anger [Friedman, VonStorch & Houston, 1954; Linford, 1975; Packard, 1979; Wolff, 1937]. Friedman [1979; 1982] characterizes the migraine patient as unable to handle his/her own aggressive energy, or that of others; prone to transitory depression; generally intense [humorless], overly conscientious, striving, meticulous in performance and often obsessed with details. These combinations of physiologic and psychic factors predispose an individual to develop migraine.

This notion implies two hypotheses. First, individuals with certain traits are prone to developing migraine and secondly, situations which produce psychic tension elicit migraine attacks. This hypothesis lacks support as the relationship between abnormal autonomic reactivity and psychic conflict has not been determined. A psychodynamic approach implicates conversion and somatization of psychic conflicts and emotion into the experience of pain. "That the choice of the head as a focus of pain is perhaps the natural outcome" [Friedman, 1982]. Symbolic function may underlie all behavior and perception but when the significance of a disease is sought through symbolic function, all illnesses are rendered equal and there is no way to distinguish them from hysteria [Savoldi, 1982].

The search for psychodynamic pathogenesis of migraine remains inconclusive. Partial support is documented in psychiatric interviews suggesting the existence of an obsessive compulsive migraine "personality" [Friedman et al, 1954; Graham et al, 1970; Wolff, 1937], but the lack of standardized procedures in this type of study makes objectivity difficult.

Moreover, most studies utilized highly selected subjects [migraineurs in psychotherapy] and did not include control groups which are essential for conclusive research [Schnarch & Hunter, 1980].

Studies incorporating psychological tests which have fixed procedures for examination and scoring [for example, MMPI, Rorscharch] have yielded little positive findings. More importantly, there are few published studies with control groups, matched on the basis of age, sex and socio-economic backgrounds.

Elevated scores on neurotic scales of the Edwards inventory were reported by Andersen and Frank [1981]; by Henryk-Gut Rees [1973] and Maxwell [1966], both on the Eysenck Maudsley Inventory; and Rogado, Harrison and Graham [1973] on the MMPI. Henryk-Gut and Rees [1973] and Kudrow and Sutkus [1979], found no differences on the MMPI; and the Frieburg Inventory failed to isolate any particular traits of migraineurs [Cuypers, Allenkirch & Bunge, 1980]. Zeitlin & Oddy [1984] report elevated scores on the anxiety, obsessional and somatic complaint scales of the Middlesex Hospital Questionnaire. No consistent findings were found in studies using the Rorschach [Cooper & Friedman, 1954; Kaldegg, 1971; Ross & McNaughton, 1954]. The lack of consistent results does not imply that there are no psychosocial traits which distinguish the migraineur, clearly some subjects perform differently, if not consistently as a group, on some psychological measures.

There are several factors which may be relevant to these discrepant findings. The lack of strict diagnostic criteria for migraine, and the pooling of subjects with different or mixed syndromes resulted in the evaluation of very different samples. Moreover, specific personality traits, may not be characteristic of all the syndromes of migraine. Secondly, associations between migraine and neurotic behaviors based on highly selected samples [clinical patients] may not be representative of migraineurs in general. Thirdly, trait categories in most tests are based on characteristic answers of psychiatric groups rather than on differences within a normal population. Patterns that might be characteristic, albeit not psychopathologic, may be overlooked.

In fact, the patterns of cognitive and emotional traits observed in migraineurs may be the result of neurological dysfunction. There is little evidence for this view because of the paucity of studies which correlate emotional and behavioral traits with specific neurological signs in migraine. Evidence from Cat-scans suggests that cerebral lesions are rarely associated with migraine [Carrera, Gerson, Schnur & McNeil, 1977; Mathew, Meyer, Welch & Neblet, 1976], but of those migraineurs scanned, 33-59% showed some abnormality [Cala & Mastaglia, 1980; Hungerford, du Boulay & Silkha, 1976; Masland, Friedman & Buckshaum, 1978; Mathew et al, 1976; Sargent, Lawson Solbach & Coyne, 1979]. Most commonly, there were indications of scattered cortical atrophy. Less frequently, low density areas, enlarged ventricles and some localized atrophy in temporal and parietal areas was reported. The incidence of abnormal brain scans in the general population has not been determined, therefore the significance of these reports cannot be ascertained.

Non specific abnormal EEG patterns in adults and 14 and 16 cycle per second patterns in children, have been reported [Edmeads, 1979; Essman, 1978; Hockaday & Whitty, 1969; Jay, 1982; Whitehouse et al, 1967]. Appenzeller [1975] reports that abnormal hypothalamic discharges precede the migraine attack. These factors suggest that functional cerebral changes may occur, but there is insufficient data to postulate the nature, or the specific anatomical loci of these changes. Techniques such as the EEG, may not be specific enough or sensitive enough to detect subtle functional changes.

This notion as well as Bruyn's [1976] hypotheses of brain stem involvement during the aura suggests another approach and the basis for the present study of psycho-social behavior in migraine. One of the most notable features of migraine and a factor which has not been previously considered, is the unilateral nature of the aura and pain. The emotional and behavioral characteristics of lateralized migraine may in fact, be more closely associated with those distinctive alterations seen in patients with unilateral cerebral dysfunction.

EMOTION: HEMISPHERIC ASYMMETRY

The suggestion from early clinical observations that lesions of the right and left hemispheres produced different emotional states was reinforced by reports of the emotional responses of patients who were subjected to unilateral carotid injections of sodium amytal to determine speech lateralization prior to brain surgery [Rossi & Rosadini, 1967; Terzian & Cecotto, 1964]. A depressive catastrophic response was reported to follow left hemisphere sedation [crying, anxiety] and an inappropriate euphoria [joking, spontaneous laughter] often followed right hemisphere inactivation. Ambidexterous patients who had bilateral speech representation displayed a euphoric response to both right and left sedation. The emotional responses to the injection occurred only after the sedative effects had cleared and the EEG over the sedated hemisphere returned to an activated state. These emotional responses however, could not always be replicated by other investigators [Milner, 1967].

These reports prompted Gainotti [1972a; 1972b] to investigate the effects of unilateral lesions on behavior during neuropsychological examinations. He found that emotional reactions to failure were significantly different in patients who had suffered lateralized cerebral vascular accidents. Left brain damaged patients tended to display catastrophic or anxious-depressive reactions [bursts of tears, refusal to continue testing] while patients with right hemisphere damage showed indifference to their disabilities [joking, minimization of their illness] and often anosognosia [hemi-attention] of the left side of their body and/or ambient space. Subsequent research has produced results which are generally congruent [Dobrokhtova & Braghina, 1974; Folstein, Maiberger & McHugh, 1977; Roth, 194949; Sackiem, Weiman, Gur, Greenberg & Hungerbuhler, 1980].

The depressive-catastrophic reactions vary according to the locus of the lesion in the left hemisphere [Gainotti, 1972b]. Transient emotional outbursts of short duration [tears, refusals] were most characteristic of the patients with Broca's aphasia. Patients with Wernicke's aphasia were more apt to curse and express discouragement. Anxiety reactions were more prominent in amnestic aphasia and tears came only after repeated failures to communicate. The statistical significance of these differences was not reported. The non-aphasic patients showed significantly less depressive-catastrophic reactions than the aphasic groups. Both aphasics and non-aphasics were significantly more anxious than patients with right hemisphere damage.

The phenomena of hemi-inattention is a striking example of right hemisphere contribution to emotional states. These patients ignore, disown or turn away from the affected left side of their body and/or ambient space. Mood shifts and denial are an integral part of the syndrome. [Heilman & Watson, 1977; Weinstein & Friedland, 1977].

Indifference and apathy are manifest when patients fail to recognize their own limbs or attend only to events and people on their right side. When questioned about the affected side however, an abrupt change of mood can occur. The patient may refer to his or her disabilities in a euphoric or facetious manner, i.e. a paralyzed arm belongs to a relative or is a Communist because it doesn't work; or with anger, striking or verbally abusing the limb

[Weinstein, 1955; Weinstein & Friedland, 1977].

In a review of patients with a more radical brain insult, adult hemispherectomy, Sackeim et al [1980] found euphoric moods frequently followed right cortex removal, but no particular emotional response to left hemispherectomy. Taylor [1972] however, reports that neurotic symptoms were most often associated with right temporal lobectomy and psychopathic disorders more frequent with left temporal lobectomy. These findings suggest that the temporal lobes may be differentially involved in the regulation of emotion [Bear, 1983].

Individuals with lesions of the right hemisphere also demonstrate a deficit in comprehending and communicating emotional material. Recall of stories with emotional content is impaired [Wechsler, 1973] as well as the ability to judge the emotional mood of the speaker through tone of voice, particularly in those patients with lesions of the parietal areas [Heilman, Scholes & Watson, 1975]. Ross and Mesulam [1979] report that right hemisphere damage can also cause the loss of prosody, the melodic line produced by variation of pitch, rhythm and stress of pronunciation that bestows emotional meaning to speech. These patients also have difficulty in comprehending the facial expression of emotion over and above their general impairment of facial recognition [Cicone, Wapner & Gardner, 1979].

Deglin and Nikolenko [1975] have also reported hemispheric differences in their investigation of the emotional effects of induced lateralized seizures. 40 patients with diverse types of depression and with schizophrenic were subjected to right or left ECT on alternate days. An analysis of the patients emotional state after the seizures, revealed significant association between non-dominant ECT and smiling, joking, laughter and elevation of mood . Dominant ECT led to anxiety, dysphoria or an intensification of an existing melancholia or delusional anxiety. This study would have been more informative had the researchers indicated whether or not the emotional shift was the same for both the schizophrenics and depressives, since these disorders are also thought to be somewhat lateralized.

These results are consistent with over 20 years of research in the use of ECT as a treatment for endogenous depression. Most investigators agree, that dominant ECT is significantly less effective than bilateral ECT [Cronin, Bodley & Potts, 1970; Halliday, Davison. Browne & Kreeger, 1968] and that the depression relieving effect of bilateral and unilateral non-dominant ECT are equivalent [d'Elia, 1970; d'Elia & Raotma, 1975; Sand-Stromgren, 1973]. Moreover, some researchers have found non-dominant ECT to be superior to bilateral ECT in alleviating depression [Cohen, Penick & Tarter, 1974; Halliday et al, 1968; Martin, Ford & McDonald, 1965; Zindin & Birchnell, 1968]. Most investigators report that reactive depression is less amenable to ECT therapy.

If different emotions are dependent on lateralized brain mechanisms, one would expect to find this organization reflected in the patterns of dysfunction in psychiatric disorders. The phenomena closest to positive and negative emotion seems to be the variance of mood in the affective disorders.

An evaluation of the performance of manic depressive patients on 26 neuropsychological variables sensitive to lateralized frontal, temporal and parietal function, suggests that both mania and depression are characterized by predominately right hemisphere dysfunction [Flor-Henry, 1976; Flor-Henry, Yeudall, Stefanyk & Howarth, 1975].

The performance of patients with affective disorders on dichotic listening tasks showed left ear decrements across conditions [verbal, non-verbal] that paralleled the performance of patients with right hemisphere lesions [Yozawitz & Bruder, 1978]. A reversal of this abnormal pattern to a normal one was observed to follow ECT [Strauss, Moscovitch & Olds, 1979] Similarly, performance by depressive patients suggested poor right hemisphere functioning on the Halsted-Reitan and other neuropsychological tests, and improved after ECT [Goldstein, Filskov, Weaver & Ives, 1977; Kronfol, Hamsher, Digre & Waziri, 1978].

The results of electrophysiological measures of depressive patients are more complex. d'Elia and Perris [1973] report greater desynchrony of the EEG over the left hemisphere and in a later study, that an increase of left hemisphere activation is relevant to the depth of the depressed mood

[Perris, 1975]. In a more sophisticated study with discrete electrode placement, the left and right frontal regions were found to contribute differentially to specific symptoms of depression. An increase in anterior right hemisphere activity covaried with the severity of the depressive affect [apathetic, withdrawn]. Symptoms of anxiety and ruminative ideation [demanding, provocative, angry] however, were associated with activation of the left frontal region [Perris & Monakhov, 1979].

This differential pattern was also demonstrated in Flor-Henry's [1976] study of the power spectral EEG over the temporal and parietal areas of unipolar and bipolar depressives and of manics. The power spectra were compiled for each subject during a neutral condition [eyes open] and during verbal and spatial tasks designed to activate dominant and non-dominant hemispheres respectively. A correlation of the results of these 3 conditions suggests that there are abnormal energy distributions in both hemispheres of depressed patients but they are significantly more extensive in the right. Mania on the other hand, was characterized by bilateral disorganization and specifically, failure of left hemisphere activation during verbal tasks.

The design of this study had been criticized because the myogenic potentials which are recorded when the patient blinks his eyes or speaks, may obscure the results. Flor-Henry [1979] defends his methodology by pointing out that very low [0-3Hz] and very high 50+Hz] bands were excluded from his analysis, eliminating some muscle artifacts. Further, he argues that it would be highly unlikely that his psychiatric patients would demonstrate lateralized muscle potentials that would coincide with lateralization found by other techniques.

Overall, evidence drawn from many lines of inquiry reflects the importance of the right hemisphere in the organization of mood. The depression syndrome is characterized by predominately right hemisphere dysfunction and patients with right hemisphere pathology exhibit characteristics of the depression syndrome. Unlike organic patients, clinical improvement in depression is accompanied by a return of normal EEG function and performance patterns.

The ECT literature has described the

efficacy of induced seizures as a form of therapy but today, patients suffering from endogenous depression are more frequently treated with anti-depressant drugs. The pharmacological action of anti-depressants are complex and the exact mechanism by which they relieve the symptoms of this disorder are unknown, but the primary action appears to be interference with the re-uptake and metabolism of norepinephrine and/or serotonin. This proposed action has given rise to the biogenic hypothesis of affective disorders, which actually incorporates two major theories. The first implicates functional changes in central noradrenergic systems and the second suggests that the primary abnormality is in the central serotonergic pathways. Studies of the biochemical correlates and pharmacological treatment of depression has led to speculation that there may be neurochemical lateralization in the central nervous system.

Mandell and Knapp [1979] found higher concentrations and a more active turnover rate of serotonin in right limbic and striatal areas of rat brain. In humans, higher concentrations of norepinephrine have been found in right thalamic nuclei [Oke, Keller, Medford & Adams 1978]. An increase in CSF 5 hydroxyindole acetic acid, a metabolite of serotonin was found to correlate with evoked potentials from the right hemisphere, in a mixed sample of psychiatric patients [Gottfries, Perris & Roos, 1974]. In a rare biochemical experiment with normal subjects, Sai-Halasz, Brunecker and Szara [1958] injected 30 physicians with dimethyltryptamine [a chemical closely related to serotonin] and found significant transient left sided neurological signs; hyper-reflexia and positive Babinski. Mental symptoms included spatial illusions, disturbances of body schemata, contraction of time and euphoria.

There is one study which suggests that the effects of anti-depressants may be lateralized. Depressed children improved their performance of right cognitive functions but not those of the left, during treatment with tricyclic anti-depressants [Brumback, Staton & Wilson, 1980]. These results are congruent with those which demonstrated a reversal of poor right hemisphere function following ECT treatment for depression. More likely, these results are due to a motor artifact. Right cognitive tests generally require more active manipulation, scanning and constructions. These skills are more "fluid", that is less overlearned or automatized, thus require more creativity and energy. Therefore, an increase in hemispheric activation, via "stimulants" will improve functions much more than verbal functions.

Not all emotional behavior however, can be attributed to the right hemisphere. Lesions, sedation and lobectomies of the left hemisphere are associated with distinct affective changes. In their neuropsychological evaluation of psychiatric patients, Flor-Henry et al [1975] found that schizophrenics showed a pattern of deficits that suggests dysfunction of the frontal and temporal areas of the left hemisphere.

Electrophysiological studies have indicated that in schizophrenia, the left hemisphere may be dysfunctional because of over activation. A greater desynchrony of the EEG over the left hemisphere of schizophrenics which can be normalized by phenothiazine [anti-psychotic] administration has been widely reported [Flor-Henry, 1974; Gur, 1978; Serefetinedes, 1973]. Gruzelier and Hammond [1976] observed that schizophrenic patients demonstrate a right ear deficiency in temporal discrimination on auditory tasks, although right ear acuity was abnormally good. These results are congruent with the notion of left hemisphere dysfunction. As with the EEG studies, the right ear auditory task errors were normalized following phenothiazine administration. Examination of lateral differences in event-related potentials [ERPs] from schizophrenics left and right hemispheres has shown greater variability in the ERP over the left hemisphere, suggesting greater fluctuations in the arousal level of the left side of the brain [Roemer, Shagass, Straumanis & Amadeo, 1978].

Other studies suggest that intrahemisphere transfer may also be faulty in schizophrenia. Beaumont and Dimond [1973] showed that schizophrenics had particular difficulty in inter versus intra hemispheric cognitive transfer, compared with normals. In further research on transcallosal communication, a sample of schizophrenics demonstrated a left hand anomia [difficulty in naming objects held in their left hand, out of view], suggesting difficulty in gaining effective access to right hemisphere information. Alpert and Martz [1977] found that schizophrenics who demonstrated a flattened affect, also showed difficulty in communicating emotion through tone of voice and difficulty in using the imagery of a word to improve performance on a learning task. These deficits suggest a problem in the use of right hemisphere contributions to verbal processes. This evidence may be relevant to the language processing deficits associated with schizophrenia [Mefferd, Lester, Wieland, Falconer & Pokarny, 1969]. The decreased and/or inappropriate emotional responses of schizophrenics may also reflect the loss of emotional contribution from the right hemisphere.

The primary action of anti-psychotic drugs such as chlorpromazine, is thought to be the blocking of dopamine in the CNS. This has given rise to the dopaminergic theory of schizophrenia which suggests that overactivity of some parts of the dopaminergic pathways may result in schizophrenic symptoms. This theory is compatible with the reports of psychotic episodes following the abuse of amphetamines, which cause dopamine release.

Evidence suggesting that dopaminergic systems are lateralized is scant. In rats, lateralization has been reported in right striatal areas [Dennenberg, 1983] but it may in fact vary with the individual animal [Glick, Weaver & Meibach, 1980]. In humans, a correlation was found between auditory evoked potentials from the left hemisphere of psychiatric patients and an increase of a dopamine metabolite, homovanillic acid, in CSF [Gottfries et al, 1974]. CNS levels of dopamine, then, appear to influence or be influenced by the activation level of the left hemisphere and this level of activation may influence emotional status.

Although most of the research on affective organization has relied on an abnormal model, there is some indication that there is lateralization of emotion in normal human beings as well. The evidence is based upon assessment procedures such as eye movements [Ahern & Schwartz, 1979; Tucker, Roth, Arneson & Buckingham, 1977], dichotic listening [Bryden, Ley & Sugerman, 1982; Ley & Bryden, 1982], and visual recognition of emotional expression [Ley, 1979; Ley & Bryden, 1978]. activation contributes to the expression and comprehension of affect. More specific conclusions cannot be drawn due to contradictory studies especially in the area of lateral eye movements [Ehrlichman & Weinberger, 1978]. and a number of methodological problems which confound this area of research [Bryden & Ley, 1984].

The study of psychosis in lateralized temporal lobe epilepsy [TLE] has also provided evidence that emotional tendencies may be differentially lateralized. In general, epilepsy can be defined as a paroxysm of abnormal neuronal discharge or seizure in brain involving the synchronized firing of millions of neurons. This results in an alteration of consciousness, often accompanied by convulsive movements depending upon the route over which the discharge is dispersed within the central nervous system. The seizure may be focal or generalized and can result from the presence of lesions or in the absence of known organic factors [idiopathic]. Like migraine, the epilepsies are a group of syndromes classified primarily by their paroxysmal clinical manifestations. They are associated with many etiological factors, and have similar pathophysiological mechanisms, but can develop in different areas of the brain Unlike migraine, they are characterized by differential electrographic changes. Transient seizures and convulsions may occur as a result of extra-cranial origins such as pregnancy [eclampsia]; metabolic disturbance [water or alcohol intoxication] or drug withdrawal, but by current definition, the diagnosis of epilepsy requires the chronicity of spontaneous seizures resulting from a cerebral defect [Glaser, 1973].

Temporal lobe epilepsy [TLE] comprises about 1/3 of all the epilepsies [Sutherland & Eadie, 1980]. The high incidence of these seizures is probably due to the particularly vulnerable nature of the temporal lobes to trauma, and formation of an epileptogenic foci. Parts of the temporal lobe are also particularly susceptible to anoxia and hypoglycemia, and their location makes them very vulnerable to head trauma. Mastoid and middle ear infections may spread to the temporal lobe and some viruses have a particular affinity for this area. Further, lesions and malformations which can occur in any part of the brain seem to have a greater epileptogenic potentiality in the mesial aspects of the temporal lobe. This may be due to the low seizure threshold of the hippocampus [Sano & Malamud, 1953]. There is also evidence that focal discharge originating in other areas may quickly spread to the temporal lobe and clinically mimic TLE [Livingston & Escobar, 1971].

The clinical manifestations of TLE are varied and include epigastric sensations, often described as fear; hallucinations of taste, smell, hearing or vision; memory disorders [deja vu, jamais vu]; dreamy states; primary automatisms and affective disorders. Because of diffuse limbic system connections with the temporal lobe, disturbances of emotion are not uncommon in TLE [Bear, 1985].

Initially, functional psychoses were thought to be associated with all the epilepsies [Clark, 1931]. Subsequent research demonstrated that they were more manifest in temporal lobe epilepsy and specifically; a schizophrenic like evolution from a dominant focus and a manic-depressive evolution from a non-dominant focus [Flor-Henry, 1969a, 1969b, 1979; Serafetinedes, 1965]. However, only a small percentage of temporal epileptics are actually psychotic and more frequently they are described as exhibiting personality changes [Currie, Heathfield, Hensen, Scott & Flynn, 1971; Migone, Donnelly & Sadowsky, 1970; Small, Milstein & Stevens, 1962].

Clinical descriptions of the personality traits of TLEs include suspiciousness, hypermorality, altered sexuality, humorlessness, hypergraphia, emotional lability, meticulousness, anxiety, viscosity, aggression and religiosity [Blumer, 1970; Dewhurst & Beard, 1970; Mitchell, Falconer & Hill, 1954; Mulder & Daly, 1952; Waxman & Geshwind, 1974; 1975]. Most studies of the epileptic personality have utilized the Rorschach test [Kelly & Marquilies, 1940; Lisansky, 1948; Piotrowski, 1947; Shaw & Cruikshank, 1957], an instrument which is not considered to be reliable for differential diagnosis [Tizard, 1962]. The findings have not only been contradictory, but the majority of studies are unacceptable because of gross methodological errors [e.g. biased samples; lack of, or poorly matched controls; unspecified groups of epileptics]. The MMPI has also failed to identify a TLE personality [Guerrant, Anderson & Fisher, 1962; Mathews & Klove, 1968;

Stevens, Milstein & Goldstein, 1972]. Tizard [1962] concluded from her review of the literature, that clarification of personality dimensions and the development of instruments to measure these parameters were necessary in order to elucidate this controversy.

Since psychiatric diagnostic categories did not typify the TLE patient, Bear and Fedio [1977] designed a behavioral inventory to determine specific psychosocial aspects of behavior. 18 psychosocial traits [each sampled by 5 questions] which in isolation are not indicative of psychosis but were reported to be associated with interictal behaviors, were selected. Although the inventory itself was drawn from a model of the interictal personality of the TLE, the application of this instrument was drawn from the reports of hemispheric asymmetries in emotionality, reviewed earlier in this paper. The most significant aspect of Bear and Fedio's research is their attempt to determine the differential effects of lateralized temporal dysfunction on psychosocial behavior by studying non-psychotic, idiopathic right and left focus TLEs. This approach then, incorporates a model of the temporal-limbic system and its role in emotionality. The Personal Inventory revealed a distinctive profile of the epileptic group [significantly higher scores on all 18 traits] and specific clusters differentiated the right focus from the left focus patients. Left TLEs were more concerned with religiosity and personal destiny, and showed more anger, paranoia, hypergraphia and catastrophic reactions to dissocial behavior. The right temporal patients on the other hand reported more elation, obsessionalism, viscosity, emotionality and sadness. The effects of lateralized seizure were similar to those found in patients with lateralized temporal lesions and the emotional effects of unilateral ECT.

Subsequent studies, of epileptics, with the Personal Inventory have produced mixed results [Brandt, 1985; Herman & Riel, 1981; Master, Toone & Scott, 1984; Mungas, 1982; Nielsen & Kristensen, 1981; Rodin & Schmaltz, 1984]. Some investigators argue that the Inventory does not produce a specific profile for the TLE. In return Bear [1985] claims that it is the relative specificity that is important, that is, are these clusters of behaviors the same or different than those caused by other neurological disorders. A true comparison of the studies is difficult because of differing groups and statistical analyses. Although there is the possibility that Bear and Fedio's sample was a highly selected group [Dodrill & Batzel, 1986] it is also probable that other samples were not select enough. Many investigators do not use special electrode placements [sphenoidal or nasopharyngeal] or EEG recordings during sleep to maximize the probability of detecting the presence of a temporal lobe focus in patients with generalized seizures [Hermann & Whitman, 1984]. Other variables which may be contributing to the conflicting reports are seizure frequency, psychiatric history and the fact that the diagnoses of "psychomotor" and "temporal lobe" seizure are often used equivocally, though many patients with TLE never have a psychomotor seizure and many with psychomotor seizures do not manifest a temporal lobe EEG focus [Stevens, 1966].

McIntyre et al [1976] found differences in cognitive style and comprehension of emotionally evocative written material. Subjects with a left TLE focus demonstrated a reflective tempo and a deficit in assigning affect labels to the emotionally evocative passages they read. The right focus subjects displayed an impulsive tempo and no emotional comprehension deficits.

Preliminary results of the performance of lateralized classical migraineurs on the Personal Inventory were similar to those of temporal lobe epileptics, at least for those with right hemisphere locus of pain [Mashayekhi & Berger-Gross, 1982; 1983]. The right focus patients scored significantly higher on emotionality, anger, sadness, elation, viscosity, obsessionalism, humorlessness than left focus migraineurs and tension headache controls.

In reviewing the overall effects of unilateral trauma to the cerebral hemispheres, a pattern of emotional reactions has emerged, that is some what consistent. Anxiety, catastrophic reactions and thought disorder may follow left hemisphere insult while disorders of mood are associated with right hemisphere dysfunction. Moreover, the existing evidence suggests that the right hemisphere may be more influential in the maintenance of emotional tone.

Just as lateralized temporal disorders have been associated with patterns of affective and personality changes, changes in affect may be accompanied by focal neuropsychological deficits. For example, depression and right focus TLE have been associated with nondominant hemisphere differences in dichotic listening and visual tasks [Berent, 1977; McIntyre, Pritchard & Lombroso, 1976; Yozawitz & Bruder, 1978]. while schizophrenics and left focus TLEs demonstrate some impairment in verbal/linguistic systems [Flor-Henry et al, 1975; Hammond, 1976; Yozawitz & Bruder, 1978]. Therefore, changes in affect and personality may be associated with cognitive asymmetries as well.

COGNITION: HEMISPHERIC ASYMMETRY

Performance of cognitive functions depends upon the combined workings of many cortical and subcortical processes. Disturbance of a particular cognitive function by a circumscribed lesion does not imply that the function is localized in that area. It is sufficient, that a link in that functional system is disrupted causing either a loss or a deficit in the performance of the function. Higher cortical functions may be disturbed by a lesion occurring in one of the many different areas of the functional system but they will be disrupted differently depending upon the link affected [Luria, 1977].

The analysis of how cognitive functions are impaired by lesions in different parts of the brain has lead to some understanding of general patterns of function and conversely, allows inferences to be made about hemispheric integrity from deficits in cognitive function. Dominance of either hemisphere is not an all or nothing phenomena and different patterns of lateralization appear to be correlated with early brain damage [Selnes, 1976], handedness

[Goodglass & Quadfassel, 1954; Satz, Achenbach & Fennell, 1967] and sex [Healey, 1980]. However, most right handers and a majority of left handers possess similar patterns of dominance.

Although many of the present notions about the laterality of cognitive functions are currently being revised, it is generally agreed that there is some functional differentiation between the hemispheres. The left hemisphere is seen as the predominate site of mediation for speech and language and is often described as analytic or concerned with sequential processing. Studies of patients with unilateral lesions or commissurotomized patients indicate that this hemisphere codes data into verbal symbols and organizes data by conceptual similarity [Nebes, 1974]. The right hemisphere is more dominant for nonverbal skills and considered to be more of a holistic or gestalt processor involved with the integration of information over space and time. This hemisphere handles its data by comparing or apposing perceptual constructs that defy verbal description and organizes its data on the basis of structural similarities [Bogen, 1969; Kimura, 1966; Vignolo, 1969].

Left hemisphere lesions commonly give rise to speech and language related disorders or more broadly, disruption of symbolic formulations [Dax, 1865; Dimond & Beaumont, 1974; Broca, 1861; Hecaen & Marcie, 1974; Russell, 1961; Sperry, 1874]. Damage to frontal lobe speech zones produces deficits in verbal fluency and expressive language functions, while lesions in language zones in the temporal and parietal areas result in deficits in receptive language functions [Geshwind, 1967; Goodglass & Geschwind, 1976]. Reading and writing may be disrupted from posterior lesions extending from the posterior temporoparietal area into the occipital lobe [Hecaen & Angelerques, 1964; Russell & Espir, 1961]. Right hemisphere lesions more often produce deficits of spatial orientation [Hernad & Doty, 1977], visual-spatial integration [Benton, 1979], and visuoconstructive performance [Parsons, Vega & Burn, 1969]. Patients with right hemisphere lesions perform poorly on tests involving the manipulation and appreciation of forms and spatial relationships [Weisenberg & McBride, 1935]. Visual memory and visual perception may also be impaired [Kimura, 1961; Milner, 1958].

There is some evidence to suggest that facial recognition may be more lateralized to the right hemisphere [Brown, 1972; Milner, 1967; Meadows, 1974]. These results however, appear to be dependent upon the functional demands of the task, that is, whether verbal or nonverbal figural components were the important factors. Berent [1981] demonstrated that when similarity of facial expression, was the comparative factor, a dimension that can be verbally encoded, there was evidence of left hemisphere processing.

Visual field differences and dichotic listening studies have provided data about hemispheric specialization in the intact brain [Kimura, 1966; Kimura, 1967]. The main premise in these paradigms is that stimulation arising from the right ear or right visual field arrives at the contralateral or left hemisphere first for processing and conversely, information from the left ear or left visual field arrives first to the right hemisphere. This is because ear and visual field hemispheric connections are crossed, giving the contralateral hemisphere a time advantage in receiving the stimulus. If the hemisphere that receives the information first is also the most efficient, because of its cognitive specialization at processing that stimulus, recognition or reaction time should be better or faster. This leads to the assumption that a faster reaction time or a recognition advantage is related to the cognitive specialization of the contralateral hemisphere [Bryden, 1982].

Normal subjects show a significant right-ear advantage in recall for dichotic verbal material and a left ear superiority for nonverbal material [Berlin, 1978; Bryden, 1969; Kimura, 1964; 1967]. Experimental designs employing tachistoscopic presentation of language related stimuli suggest a right visual field superiority and by inference, a left hemisphere superiority for language.

The performance of patients with a lateralized seizure focus has been examined on several neuropsychological dimensions. Analysis of a consonant-vowel [CV] dichotic listening task revealed that right focus TLEs and control subjects evidenced the expected right ear superiority [Kimura, 1967] while the left focus TLEs demonstrated a right ear deficiency. McIntyre et al [1976] report that patients with left TLE display a more reflective cognitive style and those with right TLE are more impulsive, than a normal control group, when performing the Kagan Matching Familiar Figures test. A significant difference was also found between the 3 groups on the WAIS vocabulary subtest. The control group had higher scores than the epileptic groups.

No significant right-left differences on the Wechsler Intelligence Scale for Children [WAIS] or The Wide Range Achievement Test [WRAT] were found in a population of well adjusted TLE children [Camfield, Gates, Ronen, Camfield, Ferguson & MacDonald, 1984]. Fedio and Mirsky [1969] however, report differences in the performance of TLE children, on more sensitive measures. Those with a right focus showed performance deficits, especially on visual memory tasks. Children with a left focus had language related difficulties, particularly in verbal memory.

The phenomena of cognitive asymmetries has been touched on only briefly here but there are some parting cautions which must be applied not only to the evaluation of the studies cited but to all such research. While it is clear that many behavioral and physiological measures are related to hemisphere specialization, many of the effects may be by-products of the experimental procedure itself. Further individual differences as well as sex and handedness may confound the results. Descriptions of functional lateralization are helpful in knowing what kind of laterality effects to expect from brain damage and are invaluable for inferential diagnosis of pathology from behavioral changes but they are not theories of hemispheric function [Bryden, 1983].

THE PRESENT STUDY

The reported psychosocial behaviors of some migraineurs are strikingly similar to those of the TLEs. Obsessionalism, hypermoralism, circumstantiality, humorlessness, aggression, anger depression, dependence and altered sexuality are all traits reported to be manifest in the migraine personality. If these behaviors are the sequelae of TLE involvement, it is possible that they may also result from the lateralized dysfunction in migraine. The hypothesis of a fundamental relationship between migraine and epilepsy is based on similarities of physiological changes [EEG abnormalities]; neurological symptoms; biochemical changes [an increase in serotonin levels during the attack [Essman, 1978]; and from cases where the identical aura leads to a classical migraine attack in some instances and a seizure in others [Basser, 1969]. Acceptance of such a hypothesis however, is not necessary to recognize that classical migraine, like epilepsy is a paroxysmal disorder with unilateral CNS focus, contralateral disturbances, has psychosocial behavioral concomitants and is the result of an ongoing [though self-limiting] complex pattern of lateralized dysfunction within the CNS.

It is appropriate then, to approach the investigation of the migraine "personality" in a way that has not been done before, that is, with the hypothesis that the psychosocial concomitants of migraine are the result of CNS dysfunction, and the specific effects of lateralization must be considered. Evidence of hemispheric asymmetry in emotionality drawn from unilateral lesion, ECT and TLE studies suggest that there may be significant differences in the affective behavior of classical migraineurs with a predominately right or left locus of pain.

The evidence of cerebral dysfunction suggests that there may be cognitive sequelae to classical migraine as well. There are few investigations of neuropsychological deficits in migraine and none which consider the lateralized nature of the disorder. Zeitlin & Oddy [1984] report significant differences in the cognitive performance of a mixed group of migraineurs [common and classical] who had severe headaches for at least 10 years, and a group of subjects who had never had a migraine headache [no medical history is reported]. The migraine group gave poorer performances on Trail Making A [a test of number sequencing], Paced Auditory Serial Addition Test, a choice reaction time test, and on a forced choice word recognition test. No relationship between test results and severity was found. Hooker [1985] found that common, classical and non-migraineur groups performed significantly different on several measures. For the classical migraine group, dominant hand motor speed was slower than the performance of a control group. Their dominant hand was also less dexterous than that of the common or control group. Both migraine groups performed in the normal range with their dominant hand on the Tactual Performance Test, but their score was significantly poorer than the control group. All groups performed within normal limits [WNL] on Digit Symbol and aphasia screening tests, but the classical migraineurs scored significantly lower. No significant differences were found between groups on the Trail Making test in this study. Hooker notes that on many measures, whether significantly different or not, the performance of the common migraine group was intermediate to that of the classic and control groups. He proposes this to be evidence of a continuum of impairment in approximate proportion to clinical manifestations [common migraine, no overt neurological disturbances; classical migraine, transient neurological dysfunction], and supports a single pathophysiological theory of migraine. All of the migraineurs, in the studies above, were clinic patients.

No significant differences between migraine groups and other subjects on intelligence measures have been reported [Hooker, 1985; Waters, 1971; Zeitlin & Oddy, 1984] except for the results of Schuchman & Thetford [1970] whose migraine sample was superior to normals on the Picture completion subtest of the WAIS.

A comparison of the performance of these sample populations, comprised of patients with the same disorder [paroxysmal headaches], but with different clinical manifestations [unilateral and bilateral focus of pain; presence or absence of contralateral neurological concomitants] leads to ambiguous results. The groups perform differently, but there was no clarification of what kind of processes may be contributing to the results. It is the same as drawing inferences from a group of patients with mixed lesions. As in the migraine personality studies, the pooling of patients with different syndromes results in the evaluation of very different samples.

Focusing on a distinct subgroup, with approximately equivalent symptomatology, allows the investigator to assume with some reasonable certainty, that he/she is looking at a sample with similar neurological processes even though the etiology and pathogenesis is unclear.

For that reason, the present study will concentrate on lateralized classical migraine, a syndrome with a distinct contralateral aura of motor, sensory or visual manifestations. Disorders other than migraine which manifest unilateral head pain are unsuitable as control groups because of their extracranial locus of pain [temporal arteritis] or the probability of more profound hemispheric insult [intracranial lesions]. Therefore, subjects with unilateral migraine will be contrasted with subjects presenting periodic tension headaches.

In summary, the aims of this study are:

1. To examine the migraine personality by contrasting the performance of subgroups of lateralized classical migraineurs and tension

headache groups.

2. To investigate the possibility of cognitive sequelae in classical migraine.

3. To ascertain whether there are distinct affective and cognitive patterns in individuals exhibiting classical migraine.

HYPOTHESES

1a] If specific, emotional and behavioral traits are associated with classical migraine, personality tests sensitive to differences in a normal population should differentiate migraine subjects from tension headache controls.

1b] More specific tests such as the Personal Inventory, will differentiate classical migraineurs from subjects with tension headaches.

2a] If classical migraine is the result of lateralized cerebral dysfunction, tests sensitive to behavioral and affective change in patients with lateralized neurological disorders will differentiate left and right focus migraineurs.

2b] More particularly, right focus migraineurs will perform differently than left migraineurs.

3a] If there are functional changes in classical migraine, neuropsychological measures which have been shown to be sensitive to lateralized neurological disorders will differentiate left and right focus migraineurs.

3b] Specifically, measures which place greater demands on the right hemisphere will be preformed better by left focus migraineurs, and measures which make more left hemisphere demands will be performed better by right focus migraineurs.

METHODS

SUBJECTS

Subjects with migraine were selected by the criteria of unilateral pain, distinct aura with visual, motor or sensory manifestations [classical migraine], a negative history of other neurological disease and no evidence of other headache syndromes [i.e. sinusitis, cluster, hypertension]. Two classical migraine groups were studied; predominately unilateral left focus of pain and predominately right locus of pain.

Subjects with tension headaches described as bilateral, with no prodromata and no history of other headache syndromes or neurological disease comprised the control group.

The three groups included university

students, staff and personnel from several major hospitals and any others who were willing to participate in a headache research project. Age, sex and education were comparable in the 3 groups.

Sample populations in prior migraine personality research, with the exception of the study by Henryk-Gut and Rees [1973] and that of Waters and O'Connor [1970], have been drawn from headache clinic patients or persons with migraine undergoing psychotherapy. This is a highly selective sample and not representative of all migraine sufferers in the general population. It did not include sufferers whose attacks are sufficiently mild or infrequent that they do not seek clinic treatment. Migraine sufferers who do seek clinical treatment differ in social class and intelligence from those migraineurs who do not [Waters, 1971]. Further, the personality of migrainous psychotherapy patients may be atypical and unrepresentative of all migraineurs. Psychotherapy patients are more likely to report having migraine, complain of severity and are more likely to seek clinical treatment [Schnarch & Hunter, 1980]. Generalizations based on these highly select populations may not be applicable to all migraine suffers particularly with regard to theories of causation or interaction of personality factors. Therefore none of the subjects will have reported a psychiatric history or attended a headache clinic.

A description of the participants, in the present study, can be found in Table 1.

TABLE 1. DESCRIPTION OF SUBJECTS

	MIGRAINE		
	RIGHT	LEFT	TENSION
NUMBER OF SUBJECTS	16	15	17
SEX M/F	5/11	6/9	7/10
AGE MEAN [RANGE]	33.1 [19-55]	35.3 [18-61]	37.2 [18-66]
HANDEDNESS R/L	13/3	15/0	12/5
EDUCATION/YEARS MEAN [RANGE]	14 [12-22]	15 [12-22]	15 [12-18]
AGE AT ONSET MEAN [RANGE]	16.4 [4-35]	19.6 [7-38]	18 [5-50]
DURATION/YEARS MEAN [RANGE]	16.6 [1-40]	14.4 [2-43]	19.2 [1-39]
FREQUENCY/MONTH MEAN [RANGE]	3.8 [.25-15]	2.4 [.16-10]	4.9 [.16-25]

MATERIALS

Personal Information Form

This two page form records general information; name, age, education and specific information about headache history. Questions about frequency, age of onset, location of pain [check right, left or bilateral as well as indicating by numbers on a schematic drawing of the head], intensity and quality of pain are included. A check list of prodromal symptoms, space for additional symptoms not included and a comment section, is provided. In addition, there are questions about medical history and drug usage. [See appendix A]

Bear & Fedio Personal Inventory

The Personal Inventory has demonstrated some ability to differentiate the same psychosocial traits attributed to the migraineur, in non-psychotic TLE groups and identify differences between patients with lateralized neurological dysfunction [Bear & Fedio, 1977]. Therefore, it is the most appropriate instrument to investigate the psychosocial profile of the migraine patient.

18 traits [summarized in Table 2] are

sampled by 5 items each and an additional 10

TABLE 2. DEFINITION OF TRAITS (adapted from BEAR and FEDIO 1977)

EMOTIONALITY	Deepening of all emotions
ANGER	Increased temper, irritability
OBSESSIONALISM	Ritualism, orderliness, compulsive attention to detail
VISCOSITY	Stickiness, tendency for repetition
GUILT	Tendency to self scrutiny and self recrimination
PARANOIA	Suspicious, over interpretive of motives and events
AGGRESSION	Overt hostility
SADNESS	Discouragement, self depreciation
PHIL. INTEREST	Incipient metaphysical or moral speculations
CIRCUMSTANTIALITY	Overly detailed, loquacious, pedantic
HUMORLESSNESS	Humor lacking or idiosyncratic, sobriety, ponderous concern
DEPENDENCE, PASSIVITY	"at hands of fate", cosmic helplessness
HYPERMORALISM	Attention to rules with inability to distinguish significant from minor infractions, desire to punish offenders
RELIGIOSITY	Holds deep religious beliefs
HYPERGRAPHIA	Keeps detailed notes or extensive diaries
ELATION, EUPHORIA	Exhilarated mood, grandiosity
PERSONAL DESTINY	Events given personalized significance, divine guidance ascribed to patients life
ALTERED SEXUALITY	Loss of libido, hypersexual

items modified from the MMPI Lie Scale, are included. Items are presented in random order. Scores on each trait represent the number of affirmative answers in that category [0-5]. Sample items are presented in Table 3.

TABLE 3. SAMPLE TRAIT ITEMS from BEAR & FEDIO PERSONAL INVENTORY

EMOTIONALITY	For me, feelings often take the place of thinking.
OBSESSIONALISM	I have a habit of counting things or memorizing numbers.
GUILT	I can never forgive myself for some of the things I've done.
AGGRESSION	I have a tendency to break things or hurt people when I get angry.
VISCOSITY	I cannot get off the point sometimes.
MMPI [lie scale]	I never feel like swearing.

Subjects fill out the questionnaire themselves. The cover page states that the purpose of the questionnaire is to investigate the relationship between certain medical conditions and personal habits, feelings and beliefs, and gives instructions for completing the Inventory by checking true or false to each item. The instructions emphasize that there are no right or wrong answers and a request for honesty is encouraged by assurances that their form will be processed without their name. In cases where the subject is reluctant to give his or her name, it may be omitted. [See appendix B]

Klein & Armitage Verbal & Spatial Test of Cognitive Osscilation [VSCO]

Hemispheric specialization for verbal and spatial performance has been well established in current literature. Klein & Armitage [1979] demonstrated the independence of these modalities by the use of two paper and pencil matching tasks. The verbal task requires the subject to indicate whether 2 letters [one uppercase, one lowercase] are the same or different [after Posner, Boies. Eichelman & Taylor, 1969]. In the spatial task, the subject must identify whether or not 2 random dot patterns are identical. Choices are made by crossing out S or D between each pair, during 3 minute trials.

In repeated measures over time, there was an inverse relationship between speed of performance on verbal matching and speed on the spatial matching. Because this test is sensitive to intra individual differences in lateralized activation, it may also be sensitive to subtle differences in lateralized classical migraine. The scores are the mean number of correct identifications in two 3 min. trials, in each condition. [See appendices C and D]

Purdue Pegboard

This test of executive motor function was designed to measure individual differences in a normal population. It has been found to be an effective discriminator of lateralized cerebral dysfunction [Costa, Vaughan, Levita & Farber, 1963]. The subject places pegs in a board with the preferred hand, nonpreferred hand and both hands together, as quickly as possible. The score is the mean number of pegs correctly placed in two 30 second trials in each condition.

Finger Tapping Test

This test consists of a telegraph tapping key with an electronic counter attached, to record the number of taps. It is a measure of pure motor speed. Marked slowing of the tapping rate may indicate contralateral cerebral dysfunction [Halstead, 1974]. Score is mean of 5, 10 sec trials.

Kagan Matching Familiar Figures [MFF]

Impulsivity is a psychological concept which may be basic to the overt expression of aggressive behavior [McIntyre et al, 1976], a trait often attributed to the migraine patient. Differences in conceptual tempo may reflect the tendency to internalize or externalize aggressive feelings. The MFF provides a measure of cognitive style; impulsivity versus reflection [Kagan, 1966]. Zern [1979] has reported that aggressive patients [assaultive or delinquent behavior] score more impulsively and those with internalized aggressive behavior [attempted suicide or depressives] demonstrated a more reflective style on the MFF. McIntyre et al [1976] found distinctly different response styles exhibited by left and right temporal lobe epileptics. Subjects with a left focus demonstrated a reflective tempo and the right focus subjects displayed a more impulsive tempo.

The MFF consists of 12 pictures of common objects. Subjects are asked to choose an identical picture from 8 others [7 of which are slightly different] presented at the bottom of the same page as the standard. Latency to first response and total number of errors for each item are recorded. Scores reflect mean latency time and number of errors.

Benton Form Perception Test

This test was designed to measure nonverbal tactile information processing [Benton, 1983]. It consists of two equivalent sets of 10 cards. On each card, there is a sandpaper geometric figure. Each subject is instructed to feel each card, which is concealed under a box, and visually identify it from 12 drawings on a card placed before him. One set is used for each hand. The scores represent the number correct for each hand.

LSU-Kresege Dichotic Listening CV Test

Pairs of nonsense consonant vowel combinations are presented simultaneously to each ear, via Koss HV/1A professional stereophonic headphones. The stop consonants /p/b/t/d/g/ and /k/are recorded such that each appear in all possible combinations. The subject records his answers on a form which has each possible choice; ba, da, ga, pa, or ta; randomly arranged in a line, for each presentation [See appendix E]. Each subject completes 60 trials in a forced choice paradigm.

That is, the subject must give two answers to each trial, even if he must guess. This reduces attentional bias [Clark, 1974; McNicol, 1971]. Most subjects have a right ear preference for speech sounds and correctly identify more CV's from that ear [Kimura, 1961; Milner, 1974]. Total correct for each ear and total number of correct pairs constitutes the 3 scores for each subject.

WAIS Subtests

Similarities

The subject must explain the commonality of word pairs in this test of verbal concept formation. There is a wide range of difficulty, ranging from the simplest which only the most impaired fail, to the most difficult which less than 10% of the population can answer correctly [Lezak, 1976]. This test is extremely sensitive to the effects of brain injury regardless of localization. Depressed scores are associated with frontal and left temporal lobe dysfunction.

Digit Span

In this task, subjects are asked to repeat number sequences both forwards and backwards. It involves auditory attention and measures immediate auditory memory span. The reversing sequence appears to rely upon internal visual scanning [Weinberg, Diller, Gerstman & Schulman, 1972]. Digit span is most sensitive to left hemisphere involvement [McFie, 1960].

Block Design

This subtest measures visual-spatial and visual- construction ability. Red and white blocks are manipulated to copy designs. It is the best WAIS measure of visual-spatial organization [Lezak, 1976].

PROCEDURE

Persons randomly drawn from the general population, who agree to participate in a headache research project, will complete the Personal Information form and the Personal Inventory. Information drawn from the medical and headache history, will determine group placement or elimination from the study.

Subjects will be tested individually [at a time when they are headache free] on all of the remaining measures. The tasks will be presented in the following order.

1. Klein & Armitage Letters
2. Klein & Armitage Patterns
3. Kagan Matching Familiar Figures
4. Purdue Pegboard

5. Similarities
6. Finger Tapping
7. Digit Span
8. Benton Tactile Form Perception
9. Dichotic Listening
10. Block Design
11. Repeat, Klein & Armitage Letters
12. Repeat, Klein & Armitage Patterns

The sequence of these tests is arbitrary and is intended only to alternate long and short measures and provide variety in types of tasks.

RESULTS

SECTION 1. Comparison of All Classical Migraine Subjects [N = 31] and Tension Headache Controls [N = 17].

Subject Characteristics

A summarization, of the sampled characteristics of the classical migraineurs as a whole [right and left] and the tension headache controls, can be seen in Table 4. There were no significant differences [T-Test] between the classical migraineurs and the tension headache controls in age, education, age at onset, duration of ill-

TABLE 4. STATISTICAL DESCRIPTION OF SUBJECTS

	MIGRAINE	TENSION
NUMBER OF SUBJECTS	31	17
SEX M/F	11/20	7/10
AGE MEAN [SD]	34.2 [11.5]	37.2 [12.7]
HANDEDNESS R/L	28/3	12/5
EDUCATION/YEARS MEAN [SD]	14.9 [2.9]	15.4 [1.9]
AGE AT ONSET MEAN [SD]	18 [8.9]	18 [9.5]
DURATION/YEARS MEAN [SD]	15.5 [10.9]	19.2 [12.]
FREQUENCY/MONTH MEAN [SD]	3.1 [3.5]	5 [7.5]
PAIN SEVERITY MEAN [SD]	3.8 [1.]	2.5 [1.2]
PAIN DURATION/HOURS MEAN [SD]	16.8 [18.4]	6.7 [11.2]
FAMILY HISTORY OF MIGRAINE	22/32	3/17

ness and frequency of headache.

The migraine group however, reported more severe pain, on a scale of 1-5 [P <.001] and their average headache was of longer duration [P<.04].

Sex and handedness [Chi-square] were not significantly different between the two groups. The presence of a family history of migraine was significantly higher in the migraine group [P<.001].

Bear and Fedio Personal Inventory

Figure 1 summarizes the performance of the combined migraine and tension headache groups on the Bear and Fedio Personal Inventory. Each trait is assessed by five questions therefore, possible mean scores range from 0 [no questions answered positively in that category] to 5 [all questions answered positively]. Scores on the ten MMPI items have been halved to fall within the same range. The grand means, over all traits, are shown on the far right.

Scores were evaluated by a T-test. The traits are ordered by their decreasing ability to differentiate the 2 groups [2-tailed probability].

The significance of the difference between the migraine group [right + left focus] and the tension headache controls, for each trait, are summarized above Figure 1.

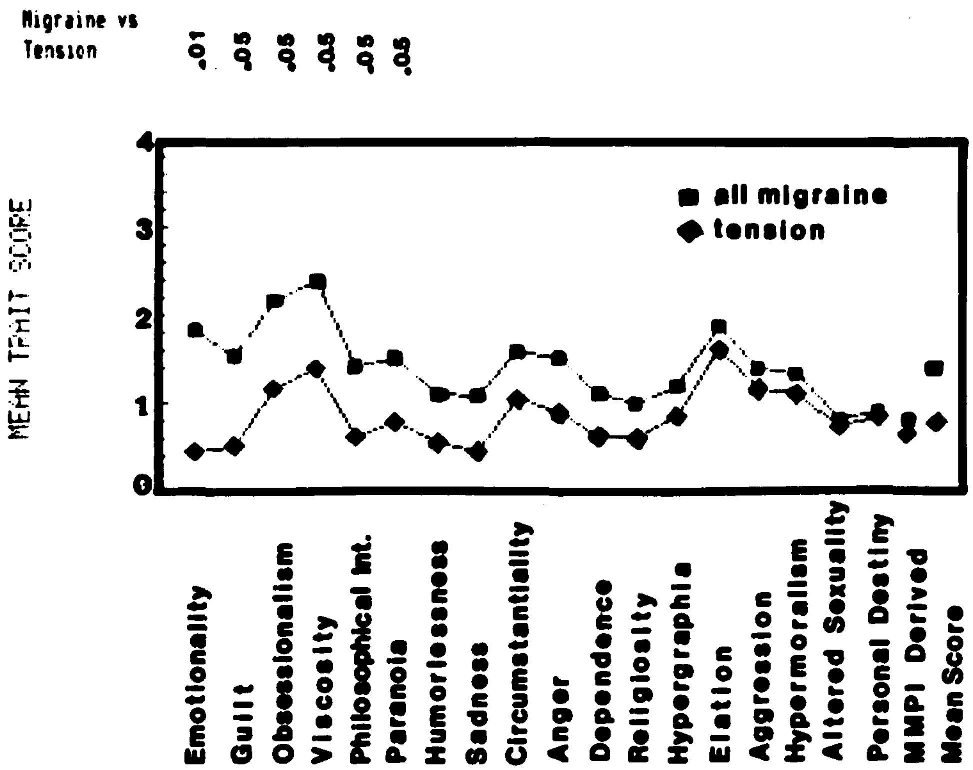


Fig. 1. Performance of all classical migraine subjects [left and right focus] and tension headache controls on the Personal Inventory [self-report]. Traits are ordered by decreasing ability to differentiate migraine from tension subjects [T-Test]. Mean scores over all traits are shown at right.

Overall, the combined classical migraine group endorsed more items on each trait, than the control group. The classical migraineurs scored significantly higher than the tension headache subjects on six of the traits. These traits were, emotionality [P< .01], guilt, obsessionalism, viscosity, philosophical interest, and paranoia [P<.05]. A summary of these results may be seen in Table 5.

TABLE 5. LEVEL OF SIGNIFICANCE: PERSONAL INVENTORY TRAITS

	ALL MIGRAINE vs TENSION
EMOTIONALITY	* *
GUILT	*
OBSESSIONALISM	*
VISCOSITY	*
PHIL. INTEREST	*
PARANOIA	*
HUMORLESSNESS	
SADNESS	
CIRCUMSTANTIALITY	
ANGER	
DEPENDENCE	
RELIGIOSITY	
HYPERGRAPHIA	
ELATION	
AGGRESSION	
HYPERMORALITY	
ALTERED SEXUALITY	
PERSONALITY	

T-Test * * = p <.01
* = p <.05

Items derived from the MMPI did not significantly differentiate the two groups. There was no significant difference between the migraineurs and the tension headache group, in the mean number of positively endorsed items, over all the traits [grand mean].

The means and standard deviations of the two groups, on each trait, may be seen in Table 6.

TABLE 6.	MEANS and STANDARD DEVIATIONS - TRAITS ALL MIGRAINE vs TENSION HEADACHE GROUP	
	MIGRAINE	TENSION
NUMBER OF SUBJECTS	31	17
	MEAN [SD]	MEAN [SD]
EMOTIONALITY	1.8 [1.6]	.47 [.72]
ANGER	1.5 [1.4]	.94 [1.1]
OBSESSIONALISM	2.2 [1.4]	1.2 [1.2]
VISCOSITY	2.4 [1.5]	1.4 [1.2]
GUILT	1.5 [1.6]	.53 [.72]
PARANOIA	1.5 [1.2]	.82 [.81]
AGGRESSION	1.4 [1.6]	1.2 [1.4]
SADNESS	1.1 [1.4]	.47 [.80]
PHIL. INTEREST	1.5 [1.4]	.65 [.79]
CIRCUMSTANTIALITY	1.6 [1.4]	1.1 [1.1]
HUMORLESSNESS	1.1 [1.1]	.59 [.71]
DEPENDENCE	1.2 [1.3]	.65 [.79]
HYPERMORALITY	1.3 [1.5]	1.1 [1.5]
RELIGIOSITY	1.0 [1.1]	.65 [.93]
HYPERGRAPHIA	1.2 [1.0]	.88 [1.0]
ELATION	1.9 [1.4]	1.5 [1.8]
PERSONAL DESTINY	.93 [1.1]	.88 [.86]
ALTERED SEXUALITY	.84 [.86]	.76 [1.1]
MMPI [lie scale]	.79 [.62]	.94 [.77]

Neuropsychological Measures

The performance of the classical migraineurs and the tension controls on the neuropsychological measures were analyzed by a T- test. The tension headache group performed significantly better [P<.05] than the migraine group, on the Purdue Peg-board, placing pegs with both hands simultaneously. The performance of the migraine group was within normal limits. No other significant differences were found on any of the other

TABLE 7. MEANS and STANDARD DEVIATIONS-NEUROPSYCHOLOGICAL MEASURES [ALL MIGRAINE vs TENSION GROUP]

	MIGRAINE	TENSION
NUMBER OF SUBJECTS	20	10
	MEAN [SD]	MEAN [SD]
PURDUE PREF HAND	15.4 [1.4]	15.9 [1.3]
PURDUE NON-PREF HAND	14.9 [1.5]	15.1 [.91]
PURDUE BOTH	12.3 [1.8]	13.6 [1.2]
K & A DOT	93 [23]	96 [21]
K & A LETTER	150 [25]	147 [27]
DICHOTIC CV LEFT EAR	28.5 [5.6]	27.7 [4.8]
DICHOTIC CV RIGHT EAR	42.0 [4.9]	39.7 [5.5]
DICHOTIC CV [R+L]	15.4 [5.9]	13.6 [6.]
WAIS DIGIT SPAN	11.9 [3.5]	11.0 [1.6]
WAIS SIMILARITIES	13.2 [2.5]	12.6 [1.1]
WAIS BLOCK DESIGN	11.5 [2.7]	12.1 [2.2]
BENTON TACTILE R HAND	9.9 [.36]	10. [0]
BENTON TACTILE L HAND	9.9 [.22]	9.8 [.42]
KAGAN MFFT ERRORS	10.0 [5.4]	9.8 [5.1]
KAGAN MFFT LATENCY	48.0 [28.7]	58.7 [28.2]
TAP PREF HAND	51.4 [6.8]	52.9 [4.8]
TAP NON-PREF HAND	47.7 [5.3]]	49.5 [3.5]

neuropsychological measures. The means and standard deviations of the two groups on each measure may be seen in Table 7.

SECTION 2. Comparison of The Left Focus Migraine Group, The Right Focus Migraine Group and Tension Controls.

Subject Characteristics

The characteristics of the two migraine groups [right focus and left focus] and the tension headache controls are summarized in Table 8.

TABLE 8. STATISTICAL DESCRIPTION OF SUBJECTS

	MIGRAINE		
	RIGHT	LEFT	TENSION
NUMBER OF SUBJECTS	16	15	17
SEX M/F	5/11	6/9	7/10
AGE MEAN [SD]	33.1 [11.1]	35.3 [11.8]	37.2 [12.7]
HANDEDNESS R/L	13/3	15/0	12/5
EDUCATION/YEARS MEAN [SD]	14.8 [2.7]	15 [3.2]	15 [1.9]
AGE AT ONSET MEAN [SD]	16.4 [8.9]	19.6 [8.3]	18 [9.5]
DURATION/YEARS MEAN [SD]	16.6 [10.9]	14.4 [11.3]	19.2 [12.]
FREQUENCY/MONTH MEAN [SD]	3.8 [4.1]	2.4 [2.6]	4.9 [7.5]
PAIN SEVERITY MEAN [SD]	4.1 [.87]	3.4 [1.1]	2.5 [1.2]
PAIN DURATION/HOURS MEAN [SD]	17.5 [20.1]	16.2 [17.1]	6.6 [11.2]
FAMILY HISTORY OF MIGRAINE	11/16	11/15	3/17

There were no significant differences [ANOVA] between the three groups in age, education, age at onset, duration of illness, frequency of headache or duration of pain. Both the right focus and left focus migraineurs reported significantly more pain [P<.001] than the tension headache controls.

Sex and handedness [Chi-Square] were not significantly different between the three groups. The presence of a family history of migraine was significantly higher in both the right and left focus migraine groups [P<.001] than in the tension headache controls.

Bear and Fedio Personal Inventory

Oneway analysis of variance was employed to evaluate the scores of the three groups [left migraine, right migraine and tension headache subjects] on the Bear and Fedio Personal Inventory with specific differences tested by the Student-Newman-Kuels procedure. This analysis revealed that almost all of the differences found between the combined [left + right focus] migraine group and the tension headache controls, reported in Section 1, were attributable to the performance of the right focus migraineurs.

The right focus migraineurs endorsed more items than both left focus migraineurs and tension headache controls on 15 of the traits. The exceptions were philosophical interest, religiosity and personal destiny.

The performance of the three groups is summarized in Figure 2. The traits are ordered

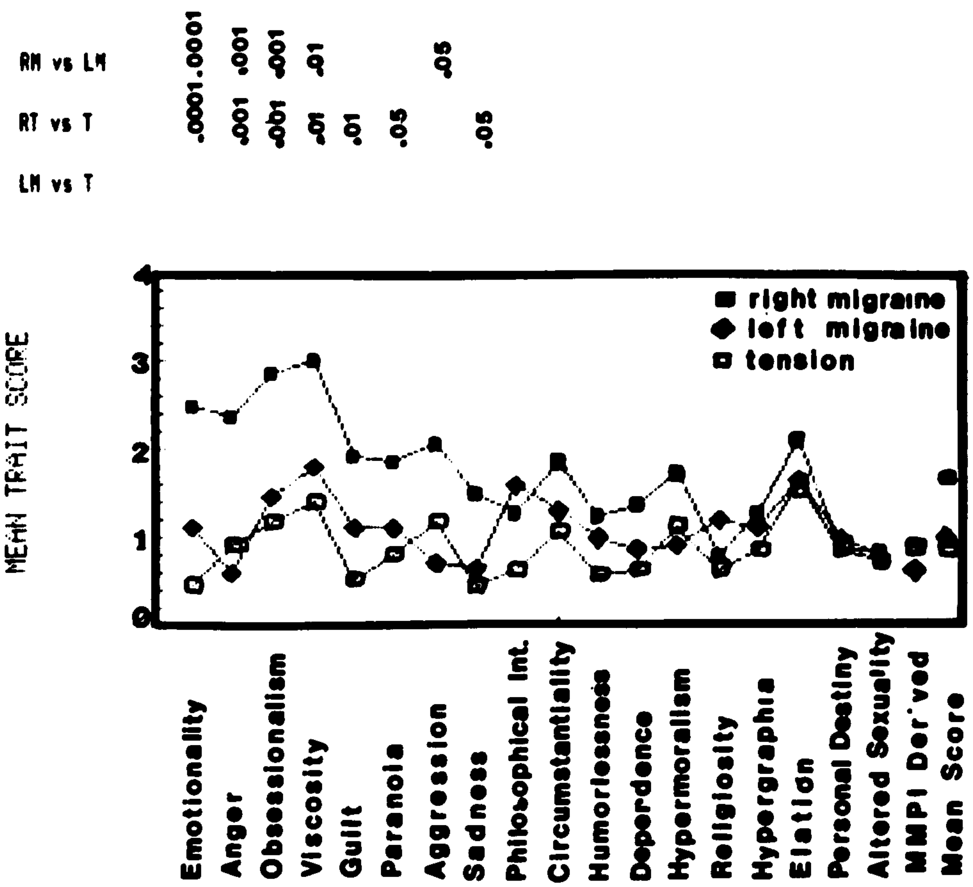


Fig 2. Performance of right focus migraine, left focus migraine and tension headache controls on the Bear and Fedio Personal Inventory [self-report]. Traits are ordered by decreasing ability to separate the groups [ANOVA]. Mean scores over all traits are shown on the right.

on the horizontal axis by their decreasing ability to differentiate the 3 groups [univariate F value]. [note: the resulting order of the traits, in this analysis, is not the same as the one which differentiated the combined migraine group and the tension headache controls].

The significance of right focus vs left focus migraineurs; right focus migraineurs vs tension controls; and left focus migraineurs vs tension controls; are summarized for each trait above Figure 2.

The right migraineurs scored sigificantly higher than both the left migraineurs and the tension controls on 4 of the traits. They reported more emotionality, anger, obsessionalism [P<.001] and viscosity [P<.01].

Higher scores on 3 more traits further distinguished the right migraine group from the tension controls; guilt, sadness and paranoia. In addition, the right migraine group reported significantly more aggression than the left migraine group. [See Table 9].

The left migraine group tended to endorse less items derived from the MMPI lie scale than the right migraine or tension headache groups [P<.08]. There were no significant differences between the grand means of the 3 groups.

TABLE 9. LEVEL OF SIGNIFICANCE: PERSONAL INVENTORY TRAITS

	ALL MIGRAINE VS TENSION	RIGHT MIGRAINE VS LEFT MIGRAINE	RIGHT MIGRAINE VS TENSION
EMOTIONALITY	* *	* * *	* * *
ANGER		* * *	* * *
OBSESSIONALISM	*	* * *	* * *
VISCOSITY	*	* *	* *
GUILT	*		* *
PARANOIA	*		*
AGGRESSION		*	
SADNESS			*
PHIL INTEREST	*		
CIRCUMSTANTIALITY			
HUMORLESSNESS			
DEPENDENCE			
HYPERMORALITY			
RELIGIOSITY			
HYPERGRAPHIA			
ELATION			
PERSONAL DESTINY			
ALTERED SEXUALITY			

ANOVA * * * = p <.001, * * = p <.01, * = p <.05

The means and standard deviations of the three groups, on each trait, may be see in Table 10.

TABLE 10. GROUP MEANS and STANDARD DEVIATIONS - TRAITS

	R. MIGRAINE	L. MIGRAINE	TENSION
NUMBER OF SUBJECTS	16	15	17
	MEAN [SD]	MEAN [SD]	MEAN [SD]
EMOTIONALITY	2.5 [1.5]	1.1 [1.4]	.47 [.72]
ANGER	2.4 [1.3]	.6 [1.1]	.94 [1.1]
OBSESSIONALISM	2.9 [1.4]	1.5 [1.1]	1.2 [1.2]
VISCOSITY	3. [1.1]	1.8 [1.6]	1.4 [1.2]
GUILT	1.9 [1.6]	1.1 [1.5]	.53 [.7]
PARANOIA	1.9 [1.1]	1.1 [1.3]	.82 [.8]
AGGRESSION	2.1 [1.7]	.73 [1.1]	1.2 [1.4]
SADNESS	1.5 [1.7]	.67 [.72]	.47 [.8]
PHIL. INTEREST	1.3 [1.4]	1.6 [1.4]	.65 [.79]
CIRCUMSTANTIALITY	1.9 [1.4]	1.3 [1.2]	1.1 [1.1]
HUMORLESSNESS	1.3 [1.2]	1.0 [1.1]	.59 [.71]
DEPENDENCE	1.4 [1.4]	.87 [1.1]	.65 [.79]
HYPERMORALITY	1.7 [1.6]	.93 [1.4]	1.1 [1.5]
RELIGIOSITY	.81 [.83]	1.2 [1.3]	.65 [.93]
HYPERGRAPHIA	1.3 [1.0]	1.1 [1.1]	.88 [1.0]
ELATION	2.1 [1.4]	1.7 [1.5]	1.6 [1.8]
PERSONAL DESTINY	.87 [1.2]	1.0 [.93]	.88 [.86]
ALTERED SEXUALITY	.87 [.96]	.80 [.77]	.76 [1.1]
MMPI [lie scale]	1.0 [.69]	.53 [.40]	.94 [.77]

Neuropsychological Measures

The performance of the right migraineurs, left migraineurs and tension headache controls on the neuropsychological measures was analyzed by Oneway analysis of variance. None of these measures significantly differentiated the three groups. The means and standard deviations of the 3 groups can be seen in Table 11.

TABLE 11. GROUP MEANS and STANDARD DEVIATIONS: NEUROPSYCHOLOGICAL MEASURES

	R. MIGRAINE	L. MIGRAINE	TENSION
# OF SUBJECTS	10	10	10
	MEAN [SD]	MEAN [SD]	MEAN [SD]
PURDUE PR. HAND	16.0 [1.5]	14.7 [.92]	15.9 [1.3]
PURDUE NON-PR. HAND	14.9 [1.9]	14.8 [1.1]	15.1 [.91]
PURDUE BOTH	12.3 [2.1]	12.3 [1.6]	13.6 [1.2]
K & A DOT	85 [23]	102 [21]	96 [21]
K & A LETTER	151 [29]	149 [22]	147 [27]
DICHOTIC CV L.EAR	30.5 [6.]	26.5 [4.8]	27.7 [4.8]
DICHOTIC CV R.EAR	40.8 [4.5]	43.4 [5.4]	39.7 [5.5]
DICHOTIC CV [R+L]	16.4 [6.5]	14.4 [5.4]	13.6 [6.]
WAIS DIGIT SPAN	11.7 [3.9]	12.1 [3.2]	11.0 [1.6]
WAIS SIMILARITIES	13.3 [2.9]	13.1 [2.1]	12.6 [1.1]
WAIS BLOCK DESIGN	10.8 [2.5]	11.3 [2.9]	12.1 [2.2]
BENTON TACTILE R.HAND	9.9 [.32]	9.8 [.42]	10. [0]
BENTON TACTILE L.HAND	9.9 [.32]	10. [0]	9.8 [.42]
KAGAN MFFT ERRORS	10.2 [5.2]	9.9 [5.8]	9.8 [5.1]
KAGAN MFFT LATENCY	41.9 [19.3]	54.2 [36.8]	58.7 [28.2]
TAP PREF HAND	51.8 [6.6]	51. [7.3]	52.9 [4.8]
TAP NON-PREF HAND	48.4 [5.4]]	47.1 [5.5]	49.5 [3.5]

A computed variable, Purdue ratio [preferred hand score - non-preferred hand score] / [preferred hand score + nonpreferred hand score] x 100 significantly differentiated the right migraineurs from the left migraineurs [P<.05]. The right migraine group demonstrated more asymmetry of performance, predominately due to the fact that they performed better with their preferred hand, than the left migraine group [P<.07].

SECTION 3. Nonparametric Analysis.

The small sample size raises the question of the propriety of conducting tests such as ANOVA and T whose stringent underlying assumptions about the parameters [homogeneity of variance and normal distribution] of the population under study, may not be met. Therefore the Mann-Whitney U test was used as a powerful nonparametric alternative to the T-test and Anova.

Generally, the results of this analysis were the same as the results of all of the parametric tests [all migraine vs tension; right migraine vs tension, left migraine vs tension and left migraine vs right migraine]. The Mann-Whitney U analysis also found additional differences between the right focus and left focus migraineurs and between the left focus migraineurs and tension headache controls.

The right migraineurs reported more severe pain [$P<.06$], endorsed more items on the MMPI lie scale and performed better with their preferred hand on the Purdue Pegboard [$P<.05$], and scored higher on trait Paranoia [$P<.07$]. The left migraineurs performed better than the right migraineurs on the Klein and Armitage pattern matching task [$P<.05$].

The left focus migraine group reported a longer duration of pain and endorsed more items on the trait philosophical interest [P<.05] than the tension headache controls.

A comparison of these two types of analysis, of the Bear and Fedio Personal Inventory traits, can be seen in Table 12.

TABLE 12. LEVEL OF SIGNIFICANCE: PERSONAL INVENTORY TRAITS

COMPARISON OF PARAMETRIC AND NON-PARAMETRIC ANALYSIS

	ALL MIGRAINE VS TENSION	R.MIGRAINE VS L.MIGRAINE	R.MIGRAINE VS TENSION	L.MIGRAINE VS TENSION
EMOTIONALITY	* * 0 0	* * * 0 0	* * * 0 0 0	
ANGER		* * * 0 0 0	* * * 0 0	
OBSESSIONALISM	* 0 0	* * * 0 0	* * * 0 0 0	
VISCOSITY	* 0	* * 0	* * 0 0 0	
GUILT	* 0		* * 0 0	
PARANOIA	* +	+	* 0 0	
AGGRESSION		* 0 0		
SADNESS			* 0	
PHIL INTEREST	* 0			0
CIRCUMSTANTIALITY				
HUMORLESSNESS				
DEPENDENCE				
HYPERMORALITY				
RELIGIOSITY				
HYPERGRAPHIA				
ELATION				
PERSONAL DESTINY				
ALTERED SEXUALITY				

ANOVA * * * = p <.001, * * = p <.01, * = p <.05, o = .05 < p <.07

M-W U 0 0 0 = p <.001, 0 0 = p <.01, 0 = p <.05, + = .05 < p <.07

SECTION 4. Analysis of Trait Items.

A chi square analysis was performed on each of the 100 inventory items [when the degrees of freedom are greater than one, fewer than 20% of the cells should have an estimated frequency of less than 5 (Cochran, 1954)]. Therefore, only those analyses which were significant and met this requirement are reported.

The percentage of each of the three groups, who endorsed the significant items, are shown in Figure 3.

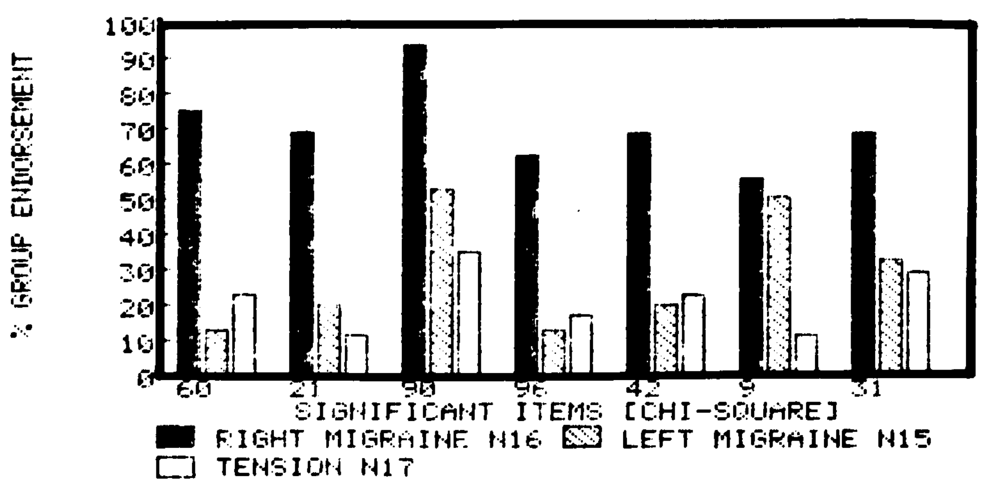


Fig. 3. Percentage of right focus migraineurs, left focus migraineurs and tension headache controls who positively endorsed items found to be significantly different [chi square]. Items are ordered by decreasing significance.

Visual analysis of Figure 3 suggests that the differences are largely due to the number of positive endorsements by the right migraine group. Items 60 and 90 are from trait viscosity; 21 and 42 represent trait obsessionalism; 9 is from trait emotionality and 96 and 31 represent anger and elation respectively. The specific items may be found in appendix B.

DISCUSSION

This investigation of cognitive and emotional anomalies associated with classical migraine, is characterized by four features that distinguish it from prior studies.

1. A substantial effort was made to focus on a distinct subgroup of migraineurs [lateralized classical] with approximately equivalent symptomatology, to maximize the assumption that the subjects in this sample have similar neurological processes.

2. Consideration of the lateralized nature of classical migraine and an attempt to determine the effects of a unilateral locus.

3. All subjects were free of prophylactic medications.

4. All subjects were fully functional and drawn from the general population rather than from headache clinics or persons undergoing psychotherapy.

The present study was designed with several questions in mind. First, are there emotional, cognitive and behavioral concomitants associated with the classical migraine syndrome? Secondly, could the performance of subjects with predominately right focus classical migraine, left focus classical migraine or tension headaches be dissociated by behavioral and affective changes reflecting lateralized neurological disorders? The answer to these first two questions is yes, and these findings will now be discussed. It is important to note that the characteristics identified in this study are not indicative [singly or in combination] of psychopathy, emotional or adjustment problems.

There were significant differences between the classical migraineurs and the tension headache group on the Bear & Fedio Personal Inventory. However, unlike patients with unilateral left and right TLE who displayed differential patterns on this measure, most of the positive findings were attributable only to the right migraineurs [the implications of this singular phenomena will be discussed later].

The results of this study appear to substantiate some of the impressions in the literature about the migraine personality. The most controversial and commonly cited personality trait of the migraine patient, is obsessionalism [Friedman et al,1954; Linford, 1975; Packard, 1979; Wolff, 1937]. The significant difference between the right and left migraineurs on this trait suggests that patients with right migraine are more apt to display this characteristic. This finding illuminates the lack of consistent results in studies which have not differentiated between right and left focus migraineurs. Studies which may have had a preponderance of left focus or bilateral migraineurs might yield negative results, while those with more right focus migraineurs, would suggest an obsessive personality.

The notion of migraine as a result of repressed anger [Friedman et al, 1954; Linford, 1975; Packard, 1979; Wolff, 1937] or somatization of emotion into the experience of pain [Friedman, 1982] might have been substantiated had both right and left migraineurs demonstrated a significant difference on these traits from the tension headache controls. However, only the right focus migraineurs reported significantly more anger and emotionality.

There was a significant difference between right and left focus migraineurs, in aggressive tendencies but, no differences were found between them and the non-migraine controls as suggested by Friedman [1979; 1982].

Although paranoia and viscosity have not been specifically reported in the migraine literature, the significant elevation of these traits in the right focus migraine subjects, in combination with the others, has probably led to the inconsistent inferences, about the migraine personality.

Reports of humorlessness and dependence were not substantiated in this study.

There was no evidence of the emotional, cognitive and behavioral sequelae often reported as a result of left hemisphere insult such as increased religiosity, preoccupation with personal destiny, hypergraphia and catastrophic reaction to dissocial behavior in the left focus migraine group, as measured by the Personal Inventory.

A summary of these data might suggest that the interaction of "migraine personality traits" and physiologic abnormalities [genetically predisposed] is pathogenic only, to right classical migraine. Although the mechanism of such an interaction is unclear, it is unlikely that the etiopathology of left and right migraine, would be different. Clearly the results of this study support the idea that the affective features associated with migraine are the result of CNS dysfunction.

The psychosocial traits reported by the right focus migraineurs are similar to those found in patients with other right CNS dysfunction such as TLE. Bear and Fedio's [1977] sample of right TLE patients also reported significantly more emotionality, obsessionalism, viscosity and sadness even though there was evidence of denial [by comparison to rater reports]. Unlike Bear and Fedio's left TLE subjects, there was no evidence that left focus migraineurs rated themselves harshly. Although the rater questionnaire was not used in this study, the significantly higher scores produced by the right focus migraineurs, on some of the traits, suggest that denial may not be a trait in this group.

The heightened emotionality, reported by the right focus migraineurs may be likened [though to a lesser degree], to the variation in mood seen in bi-polar disorder, which may be characterized by predominately right hemisphere dysfunction, [Flor-Henry,1969a, 1969b, 1976, 1979; Flor-Henry et al, 1975; Serafetinedes, 1965] and patients with right hemisphere damage [Weinstein, 1955; Weinstein & Friedland, 1977].

In summary, the characteristics reported by the right focus migraineurs are primarily overt expressions, such as emotionality, anger [although in the case of this sample, not repressed], obsessionalism and viscosity, which are congruent with those often ascribed to all migraine patients, and in particular, to those found in patients with right TLE or lesions in the right hemisphere. These findings do not suggest a migraine trait profile. The use of the Personal Inventory was to clarify reports of differing personality changes in classical migraineurs and relate them to what is already known about behaviors which result from lateralized hemispheric insult.

Given that the most popular concepts of the pathogenesis of migraine involve the notion of paroxysmal constriction and dilatation of the craniocerebral arterial tree raised a third question. What might be the cognitive sequelae of this transient, but repetitive, dysfunctioning by cerebral structures? The neuropsychological instruments utilized in this study for their sensitivity to lateralized dysfunction, failed to distinguish the three groups.

The lack of positive results does not rule out the possibility that the neurological process of lateralized classical migraine may compromise some cognitive functions. It simply suggests that the distinct neuropsychological impairments of TLE's [McIntyre et al, 1976; Kimura, 1963] and migraine groups [Hooker, 1985] were not evident in the present sample. It is possible that there are other cognitive dysfunctions, but the measures employed in the present study were insensitive to them. The cognitive impairments associated with migraine may not be of the classical type [sensory, motor, linguistic or perceptual] but more of a learning, processing or memory, deficit.

The significance of a better bimanual performance on the Purdue Pegboard by the tension controls, than that of the combined migraine group; and the faster performance of the right focus migraineurs vs the left focus group, with their preferred hand cannot be explained. All performances were WNL.

The lack of correlation [Appendix F] between migraine frequency, duration of illness and severity of illness, and the behavioral parameters, and the absence of cognitive correlates, argues against the possibility that the traits reported on the Personal Inventory reflect structural changes [cortical or subcortical] such as those proposed for the psychological changes, in temporal lobe epilepsy [Bear & Fedio, 1977; Bear, 1983]. It does suggest however, that the same neurological dysfunction that predisposes an individual to migraine, may alter the emotional system as well.

The duration and severity of TLE is associated with increased symptomatology, whereas the relatively mild insult of migraine may not produce a measurable change. The anticonvulsant drugs used to treat epilepsy have also been reported to produce interictal behavioral and personality changes [Hirtz & Nelson, 1985; Mattson, Cramer & Collins, 1985; Reynolds, 1983; Rivinus, 1982; Trimble & Reynolds, 1976; Trimble & Thompson, 1983] and may contribute to the relation between duration of illness and progressive behavioral, cognitive and affective changes [Engel Jr., Caldecott-Hazard, & Bandler, 1986].

The present investigation raises a fourth question, that is, why only classical migraineurs with a right locus of pain self-report significant psychosocial traits on the Personal Inventory? Many of the affective correlates found in this study are of a more fundamental nature, of an overt emotive behavior. These more primitive behaviors are known to occur in animals [Issacson, 1976; McLean, 1972; Papez, 1927] and may be more easily altered by minor insult or neurological dysfunction than the more ruminative, intellectualized [implicating higher cortical processing] psychosocial traits reported by the left TLE's. The migraine "disturbance" is clearly not as traumatic as a seizure and may not be sufficient to disturb cortical structures, irreversibly. In the absence of clear cortical anomalies [as measured by instruments in this study] in classical migraineurs, it is reasonable to suggest that the lack of involvement may be the reason why the left locus migraineurs do not exhibit the characteristic behaviors associated with either lesions or TLE lateralized to left cortical structures. This may also account for the fact that the right locus migraineurs displayed no evidence of the "denial and/or neglect syndrome" or that of emotional unconcern, often reported in right hemisphere lesions.

Clearly, this question cannot be fully answered by this investigation. However, the results do suggest the possibility that cerebral dysfuntioning may not be involved in the pathogenesis of the classical migraine syndrome [the considerations discussed herein apply only to classical migraine].

Classically, migraine is believed to be the result of paroxysmal vascular changes in cortex which are thought to be triggered by a wide variety of stimuli. However this explanation becomes cumbersome for explaining the multiple neurological phenomena involved in classical migraine. The symptoms are often quite uniform, in the same case, suggesting that the vascular change must always occur in the same place and repeatedly produce the same peculiar neurological disturbance. The hypothesis that spreading cortical depression, similar to that reported by Leao [1944], is responsible for the classical migraine attack would also need a fixed location to propagate.

The prodromata or aura of classical migraine is difficult to reconcile, when mapped on the cortex as a whole. The arterial organization and/or cytoarchitecture of the cerebral hemispheres do not support an explanation of neurological symptoms which are not contiguous or appear to skip over discrete areas [sparing of central vision though it occupies the major portion of striate cortex].

The proposal that subcortical structures are involved in the process of migraine has been strongly endorsed [Appenzeller, 1975; Basser, 1969; Bickerstaff, 1961; Bruyn, 1980, 1982; Herberg, 1975; Johnson, 1978; Lennox, 1960; Pearce, 1985; Sommerville, 1976; Van der Does de Willebois, 1932]; Whitehouse et al, 1967]. In his critical analysis of the presumably cortical origin of migraine, Bruyn [1982] presents a cogent argument for the displacement of the pathogenesis of the classical migraine syndrome from cortical to subcortical structures. He proposes that a more parsimonious region than cortex, which might accommodate all the visual, motor, sensory, speech, autonomic and mental symptoms of the classical migraine syndrome, may be located in the upper brainstem. This area includes the mesencephalic central gray, the basal thalamus, lateral geniculate body, posterior limb of the internal capsule, hypothalamus and hippocampus. The blood supply to this area has a common origin; three branches of the vetebro basilar artery [anterior and posterior choroidal arteries and the thalamogeniculate artery].

Many of Bruyn's deductions are pertinent to the scope of this study. Vegetative signs, such as nausea, vomiting, mood change, diarrhea, polyuria and loss of appetite, and dysphoria, common symptoms of migraine, are known to be influenced by hypophysis, hypothalamic and hippocampal structures. The expressive speech disturbances which occasionally accompany migraine may be explained by the evidence that electrical stimulation [Ojemann, 1976] and lesions of the left thalamus have been reported to produce expressive aphasia [Demeurisse, Derouck, & Crehaerts, 1982; Gorelick, Hier, Benevento, Levitt & Tan, 1984]. The cortical explanation of scintillating scotoma suffers from many cytoarchitectural challenges and this phenomena may be more parsimoniously localized to the lateral geniculate body, via its many projections to striate cortex. The cerebellar symptomatology, ataxia, nystagmus and vertigo has been explained by Bickerstaff [1961] to be a result of basilar artery involvement.

Current theories of the pathogenesis of migraine incorporate a vascular model and do not necessarily implicate a change of locus. Arterial dysregulation, of the basilar artery, occurring post bifurcation may indeed produce paroxysmal vascular spasms, thereby initiating the migraine syndrome.

The phenomena of spreading depression is not limited in animals to cortex, but can involve subcortical structures as well. Therefore it is theoretically possible that an occurrence in these structures might initiate the attack [Pearce, 1985].

The profusion of afferent and efferent connections, neuropeptides, amines and neurotransmitters known to be present in these subcortical structures may account for the widely diverse precipitants of a migraine attack such as stress, fatigue, alcohol, lights, foods and hormonal changes which have led to many theories of migraine pathogenesis. They include faulty metabolism [Hannington, 1978;] allergies [Monad, 1975; Ryan, 1974] and serotonin abnormalities [Dalsgaard-Nielson & Genefke, 1974; Damassio & Beck, 1978].

Although the locus of pain in the migraine syndrome is still controversial, most investigators posit intracranial origin. Blau [1978] suggests that the nocioceptive nerves of the dura of the venal sinuses may be involved. The effect may be due to vascular or CSF changes. A shift of the pathogenesis of classical migraine to subcortical structures does not facilitate an explanation at this time.

While there is no new compelling evidence to support any of the existing etiologies, most are enhanced by a shift from large areas to a more circumscribed one, which has neuroanatomic structures that are capable of producing the classical migraine syndrome. Many of these same brain structures, such as the hypothalamus, have long been implicated in emotional and behavioral function. The proximity of these structures allows one to postulate a common etiology, be it vascular, spreading depression, neurochemical or epileptiform discharge, of both the migraine syndrome and the emotional and behavioral concomitants.

The absence of neuropsychological anomalies by both the left and right migraineurs [as measured by the instruments in this study], in conjunction with the compelling argument not only of parsimony, but of experimental and empirical evidence, gives rise to serious consideration of a subcortical pathogenesis of classical migraine.

It would seem that the collective investigation of different migraine syndromes has led to a great deal of confusion, not only in identifying personality characteristics but perhaps of the pathogenesis of migraine itself. Focusing on a discrete subgroup of migraineurs with a lateralized locus of pain and contralateral neurological disturbances has clearly shed some light on the question of a "migraine personality" and adds some support to the notion of a subcortical etiology of the migraine syndrome. The similarity of these findings to those in other lateralized disorders provides some direction for future research.

Although the general measures utilized in this study failed to identify specific impairments, they provided basic information about the subjects' intelligence and motor and sensory abilities. These data established that there were no significant differences between the groups on these classical functions. Therefore, future studies should include instruments which assess other cognitive functions, for example, memory an attention.

Aphasic disorders have been reported in patients with left thalamic lesions as well as in those with left cortical dysfunction. This suggests that language functions such as fluency and naming should be assessed. Verbal and nonverbal memory dysfunctions have been noted in epileptic subjects and may be manifest in the classical migraine population as well. Recognition of emotional stimuli is reported to be impaired in some patients with right hemisphere disorders. Thus the classical migraineurs ability to comprehend and perceive emotional stimuli should also be investigated. Above all, the lateralization of the classical migraine syndrome should not be a neglected variable in any study of affect, cognition or etiology of this neurological disorder.

If additional anomalies can be

demonstrated, the lateralized classical migraineur may provide yet another population from which to gather information to aid in our understanding of functional hemispheric asymmetry.

Patient Information

Name:____sex:_____age:_____

Occupation:____

Hand used for writing:____

At what age did your headaches begin?___________________________

Number of headaches on the average per month?__________per week?____

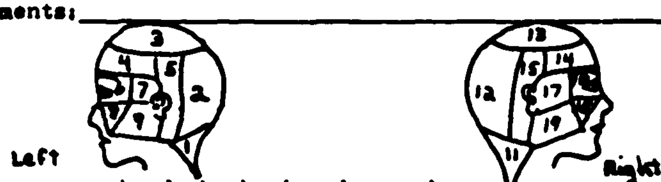
How long do they last?____

On which side do you usually have the most pain? Rt.____Left____Both_

Where do you have the pain? Indicate by numbers from drawing below.

areas:____

comments:____



Before your headache begins do you have

nausea?_______

stomache cramps?___

vomiting?__

diarrhea?__

blurred vision?___

dizziness?___

confusion?___

weakness?___

spots before your eyes?___

inability to speak?___

inability to read?___

chills?___

Nasal stuffiness?___

trouble walking?___

trouble breathing?___

partial blindness?___

Other?(please explain fully)____________________________________

____

What brings on a headache?

physical activity___ stress___ overtired___ flickering lights__

high altitudes___ a stuffy room___ after eating certain foods___

if so, which foods____________________ Other____________________

Alchohol ?________ ____

What makes your headache better?____________________

What makes your headache worse?____________________

On a scale of 1-5 (1=little discomfort - 5=severe pain), how would you rate your usual headache?____________________

How would you describe the pain? Pulsating or throbbing____Deep, boring____ Dull, nagging______

What time of day do your headaches usually occur?____________________

Have your headaches ever been diagnosed? Migraine_____Tension_____Cluster____ Other (please explain)____________________

Have you had any recent head injuries?____________________

If yes, please explain____________________

What medications do you take for your headaches?____________________

Have you ever had a seizure or convulsion?____________________

Other illnesses____________________

Other medications taken regularly____________________

Does anyone in your family have migraine headaches?____________________

If yes, who?____________________

Are you an only child?_____If not, what was your order of birth?__________

Have you ever had psychiatric or psychological treatment?____________________

If so, what type?____________________

Highest grade completed in school____________________

Comments:

Introduction

We are studying the relationship between certain medical conditions and personal habits, preferences, feelings and beliefs. We are now asking for your help in this study.

On the following pages there are statements of personal attitudes and opinions. For each statement, please indicate whether the statement seems true or false from your point of view. Simply place a check (✓) under the appropriate column for each item.

There are no right or wrong answers to this inventory; what is most important is the honesty of your answers.

Because some of the items deal with highly personal areas, we can assure you of the confidentiality of your responses. Each form will be given a computer code number and will be processed statistically without your name.

We plan to share with your physician any findings that would be helpful in future treatment. We hope that in this way your participation in the study will prove rewarding for you and other patients with similar illnesses.

PERSONAL INVENTORY

		True	False	II
1.	I think people would learn a lot from the story of my life.	( )	( )	31
2.	I have stronger feelings of happiness than most people.	( )	( )	32
3.	I feel like a pawn in the hands of others.	( )	( )	33
4.	I can never forgive myself for some of the things I have done.	( )	( )	34
5.	I have a habit of counting things or memorizing numbers.	( )	( )	35
6.	It makes good sense to keep a detailed diary.	( )	( )	36
7.	Recently more of my thoughts have something to do with sex.	( )	( )	37
8.	I never get angry.	( )	( )	38
9.	For me, feelings often take the place of thinking.	( )	( )	39
10.	Things which never attracted me before have become sexually appealing.	( )	( )	40
11.	I think that I have a special mission in life.	( )	( )	41
12.	I interpret things more deeply than most people.	( )	( )	42
13.	My religious beliefs have undergone magor changes.	( )	( )	43
14.	I am more sensitive to distractions than most people.	( )	( )	44
15.	I have gotten people angry by asking them to do so much for me.	( )	( )	45
16.	I never gossip.	( )	( )	46
17.	Powerful forces outside my control are working with my life.	( )	( )	47
18.	I keep a diary.	( )	( )	48
19.	It makes me personally furious to see people disobeying the law.	( )	( )	49
20.	Little things make me angrier than they used to.	( )	( )	50

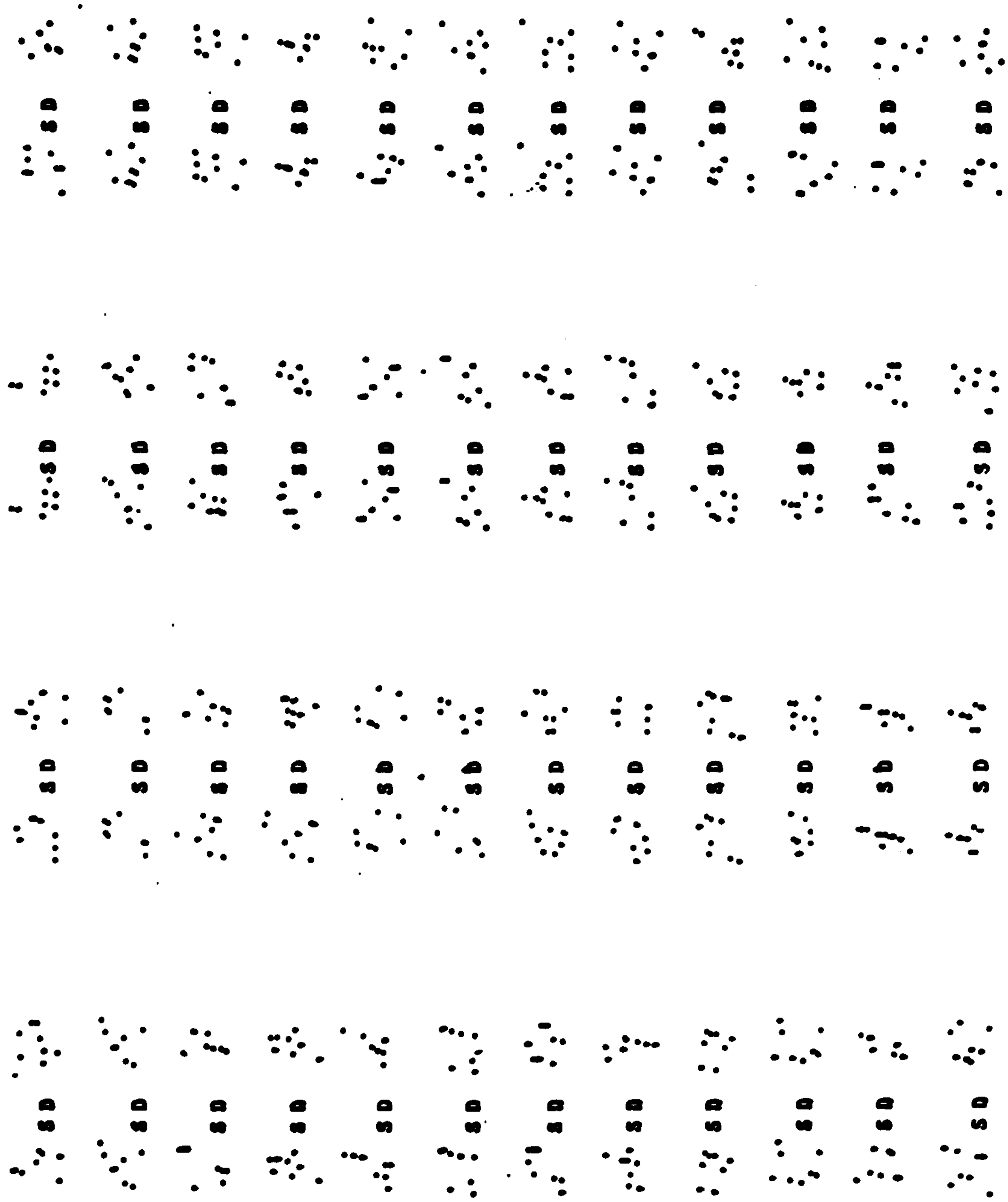
	True 1	False 0	II
21. If things are not just right, it upsets me.	( )	( )	51
22. Fate appears to be working against me.	( )	( )	52
23. Almost everything triggers some emotional reaction in me.	( )	( )	53
24. The Bible has special meaning which I am beginning to understand.	( )	( )	54
25. My temper has gotten me into trouble.	( )	( )	55
26. Sometimes I get terribly confused by little details.	( )	( )	56
27. Powerful forces are acting through me.	( )	( )	57
28. I seem to depend on other people for many things	( )	( )	58
29. Few things are really funny.	( )	( )	59
30. My table manners are just as good at home as when I am out in company.	( )	( )	60
31. Often I get into such a good mood that I do foolish things.	( )	( )	61
32. I am sure there is a significant meaning behind my suffering.	( )	( )	6
33. I have periods of weeks or months when I could not get going.	( )	( )	63
34. I am open to attack from many sides.	( )	( )	64
35. I cannot get off the point sometimes.	( )	( )	65
36. I am losing control of my temper more frequently.	( )	( )	66
37. Nothing is more important than trying to understand the forces that govern this world.	( )	( )	67
38. Life is a strain for me much of the time.	( )	( )	68
39. Sometimes I feel so helpless that I want people to do everything for me.	( )	( )	69
40. I never put off until tomorrow what I ought to do today.	( )	( )	70

		True 1	False 0	II
41.	Often I am the only one to stand up for what is right.	( )	( )	71
42.	Sometimes my mind gets stuck on so many different ideas that I cannot make a decision or do anything.	( )	( )	72
43.	When I get angry I often explode.	( )	( )	73
44.	Once I start to talk to someone, I have trouble breaking off.	( )	( )	74
45.	People do not seem to appreciate my jokes.	( )	( )	75
46.	I spend a lot of time thinking about the origins of the world and life.	( )	( )	76
47.	At elections I never vote for men about whom I know very little.	( )	( )	77
48.	I have had some very unusual religious experiences.	( )	( )	78
49.	Almost every day I am infuriated by cases where justice has not been done.	( )	( )	79
50.	It is useless to tell people something without giving them all the details.	( )	( )	80
51.	I have come to place faith in astrology, meditation or other spiritual ways of relating myself to the universe.	( )	( )	81
52.	My sexual activity has decreased.	( )	( )	82
53.	I write down or copy many things	( )	( )	83
54.	Emotions control my life.	( )	( )	84
55.	Much of the time I feel as if I have done something wrong or harmful.	( )	( )	85
56.	My feelings of hatred can be very intense.	( )	( )	86
57.	I like everyone I know.	( )	( )	87
58.	Before I make a decision, I need to know every detail.	( )	( )	88
59.	Sometimes I feel so good that ideas come into my mind faster than I can handle them.	( )	( )	89
60.	Sometimes my mind gets stuck on one idea so that I cannot make a decision or do anything.	( )	( )	90

		True 1	False 0	II
61.	I have not lived the right kind of life.	( )	( )	16
62.	I try to keep track of special details about my life and thinking.	( )	( )	17
63.	People tend to take advantage of me.	( )	( )	18
64.	I always tell the truth.	( )	( )	19
65.	I have had periods when I felt so full of pep that sleep did not seem necessary for several days.	( )	( )	20
66.	People should think about the point of many jokes more carefully instead of just laughing at them.	( )	( )	21
67.	I need more details than most people before I understand something.	( )	( )	22
68.	I have a tendency to break things or hurt people when I get angry.	( )	( )	23
69.	I am subject to big shifts in mood--from very happy to very sad.	( )	( )	24
70.	When I accidently hurt someone's feelings, I cannot forgive myself for a long time.	( )	( )	25
71.	I tend to get bogged down with little details.	( )	( )	26
72.	Finally I am beginning to understand the real meaning or nature of this world.	( )	( )	27
73.	I really am down in the dumps most of the time.	( )	( )	28
74.	I never laugh at a dirty joke.	( )	( )	29
75.	I would go out of my way to make sure the law is followed.	( )	( )	30
76.	I have more of a feeling than most people for the order and purpose of life.	( )	( )	31
77.	I am strongly attracted to members of my own sex.	( )	( )	32
79.	Sometimes without any reason or even when things are going wrong I feel excitedly happy, on top of the world.	( )	( )	34
78.	Sometimes I keep at a thing so long that others lose their patience with me.	( )	( )	33
80.	I really make myself suffer after even a small mistake.	( )	( )	35

		True 1	False 0	II
81.	People sometimes tell me that I have trouble getting to the point because of all the details.	( )	( )	36
82.	I would like to rip some people to shreds.	( )	( )	37
83.	I despise people who try to break the rules.	( )	( )	38
84.	I have trouble becoming sexually aroused.	( )	( )	39
85.	I have often felt so bad that I was close to ending my life.	( )	( )	40
86.	I read every editorial in the newspaper every day.	( )	( )	41
87.	The thought of revenge burns inside me.	( )	( )	42
88.	Most jokes do not seem funny to me.	( )	( )	43
89.	My emotions have been so powerful that they have caused trouble.	( )	( )	44.
90.	Sometimes a particular thought will run through my mind and bother me for days.	( )	( )	45
91.	I am often said to be hotheaded	( )	( )	46
92.	The furture seems hopeless to me.	( )	( )	47
93.	I am fortunate to receive so much help from people around me.	( )	( )	4
94.	I am very religious(more than most people) in my own way.	( )	( )	49
95.	I never feel like swearing.	(.)	( )	50
96.	When I think of some of the things people have done to me, it makes me absolutely furious.	( )	( )	51
97.	Sometimes I think an illness has been given to me so that I would meet certain people at the right time.	( )	( )	52
98.	I would like to write a book about my life.	( )	( )	53
99.	Religion and God are more personal experiences for me than for most people.	( )	( )	54
100.	There is too much foolishness in the world these days.	( )	( )	55

T SD t	A SD a	T SD l	N SD t
N SD e	I SD n	B SD b	B SD b
B SD a	N SD n	T SD b	H SD a
N SD e	H SD h	B SD l	I SD r
B SD n	E SD h	I SD i	T SD t
R SD n	T SD t	I SD i	A SD a
E SD t	H SD r	N SD n	B SD b
N SD n	I SD l	H SD i	E SD e
A SD e	R SD b	H SD h	A SD r
A SD a	I SD h	R SD r	R SD r
T SD a	E SD t	R SD r	R SD h
A SD b	E SD e	E SD e	H SD h



LSU-KRESGE DL CV TEST

CODE_______NAME_________________________________DATE_____________

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pa ga ta ba ka da
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ga pa da ka ba ta

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da ta pa ga ba ka
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da ta ba ga ka pa
ka da ba pa ta ga

ga da ta ka ba pa
pa ka ta da ba ga
ta ga da ba ka pa
ka ga ta da ba pa
pa ga ba ta da ka

CLASSICAL MIGRAINE SUBJECTS [N=31]

CORRELATIONS: LEVELS OF SIGNIFICANCE

	FREQ. OF HEADACHES	DUR. OF ILLNESS	SEVERITY OF PAIN
EMOTIONALITY	.233	.757	.313
ANGER	.299	.678	.176
OBSESSIONALISM	.332	.907	.645
VISCOSTIY	.162	.681	.336
GUILT	.576	.946	.944
PARANOIA	.230	.914	.958
AGGRESSION	.875	.169	.085
SADNESS	.567	.223	.287
DEPENDENCE	.646	.203	.938
CIRCUMSTANTIALITY	.754	.654	.598
HUMORLESSNESS	.640	.597	.700
ELATION	.768	.967	.647
PHIL.INTEREST	.389	.908	.214
HYPERMORALITY	.850	.263	.265
HYPERGRAPHIA	.117	.822	.365
PERSONAL DESTINY	.335	.678	.426
RELIGIOSITY	.080	.416	.195
ALTERED SEXUALITY	.118	.305	.152

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