HEADACHE AND DEPRESSED AFFECT IN A NON-CLINIC COLLEGE STUDENT POPULATION: A SEVERITY-CONTINUUM ANALYSIS

by

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ABSTRACT

Data are reported on two related studies of headache-mood covariation. In Study 1, 305 (136 M; 169 F) introductory psychology students (M age 19.50) completed the Beck Depression Inventory (BDI) and a Headache History Questionnaire (HHQ; Thompson & Collins, 1979) in groups of approximately 40. Severe/recurrent headache was reported by 35.4% of the sample, with typical episodes comprising mixed (migraine-tension) features. Rated severity, but not chronicity or frequency, predicted BDI score, F (1,303) = 13.865, p <.0002, as did four HHQ dimensions derived by principal axis factoring.

In Study 2, 36 subjects (18 M; 18 F) who reported frequent headache on the HHQ self-monitored headache features and depressed affect three times daily (10:00 a.m., 4:00 p.m., 10:00 p.m.) over 28 consecutive days, using a modified form of the Headache Frequency Record (HFR; Bakal & Kaganov, 1976) and the Depression Adjectives Check Lists (DACL; Lubin, 1967). Two models of headache-mood association were evaluated: (a) Diamond's (1983) conception of headache as a somatic manifestation of masked depression; and (b) Sternbach's (1974, 1976) view of depression in headache as a secondary consequence of chronic pain. The

severity-continuum model of headache (Bakal, 1975; Featherstone, 1985) was adopted, with no attempt to select or differentiate on the basis of headache typology.

Severe/recurrent headache was reported by 90.3% of the subsample, who endorsed a common symptom profile of mixed headache. Verbal and visual analogue severity measures intercorrelated highly, (r = .908, p <.000), and taken together were predictive of DACL scores, F (1,781) = 73.827, p <.000. Twelve subjects (6 M; 6 F) evidenced a weak severity-mood association, with no uniform temporal pattern. Neither explanatory model of the association was clearly supported. Reported family prevalence of headache on the HHQ differentiated among subjects who did and did not evidence the association, F (3,23) = 3.104, p <.046. It was concluded that headache-mood covariation did exist in some subjects, but that no uniform temporal pattern was evident. Future research should attempt to isolate differentiating characteristics among clinic and non-clinic headache populations selected with reference to both reported headache frequency and measured depression.

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PART I

A REVIEW OF THE HEADACHE LITERATURE

INTRODUCTION

The extant literature concerned with headache in its various clinical forms suggests a relatively high prevalence of the disorder within the general population (Bakal, 1975; Friedman, 1979); actual estimates have ranged from approximately 15% of adult men to 20% of adult women (Newland, Illis, Robinson, Batchelor, & Waters, 1978; Waters 1970; Waters & O'Connor, 1971). The vast majority of these headache complaints are not directly attributable to known physical causes. Although severe recurrent headache constitutes one of the most frequent complaints of adults seeking outpatient medical services (Cypress, 1981), prevalence estimates vary widely from source to source. As such, current figures must be regarded as preliminary. The picture is further complicated by the suggestion that only a small percentage of complaints leads to medical consultation (Andrasik, Holroyd, & Abel, 1979; Banks, Beresford, Morrell, Waller, & Watkins, 1975; Waters & O'Connor, 1975), an observation that invites concern about the representativeness of clinical samples generally (Merskey, 1982). There does appear to be broad consensus, however, that recurrent or chronic headache constitutes the most common health complaint facing modern community medicine.

Over the years, several authoritative texts and papers on the descriptive and biological aspects of headache have appeared in the medical literature (e.g., Appenzeller, 1976; Dalessio, 1972; Lance, 1978; Ryan & Ryan, 1978). For the most part, these have confined themselves to matters of classification and diagnosis, biological etiology, and medical intervention. In contrast, the present review is concerned with contributions to the headache literature derived from psychological investigations -- specifically, research concerned with a purported relationship between depressed affect and headache activity.

The first chapter of the review examines issues of classification and diagnosis. Major idiopathic headache syndromes are reviewed briefly, as are problems of definition and differentiation, and known or suspected etiology. The second chapter focuses on psychological investigations of headache. Specifically, the chapter examines issues of headache research from the perspective of applied psychology, particularly the process of headache assessment. The third and final chapter of the review examines the literature concerned with personality and affective variables in headache, and the findings of psychometric and related investigations.

1. REVIEW OF CLINICAL HEADACHE SYNDROMES

Pain has been defined briefly as, "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (IASP Subcommittee on Taxonomy, 1979, p. 250). This definition both conveys the multidimensional and subjective nature of pain, and permits a distinction between pain as a sensory experience and the psychosocial phenomenon of "pain complaint" -- the act of presenting with the symptom of pain (Sternbach, 1976). This distinction underlies a number of difficulties inherent in classifying and measuring headache as a variety of pain (e.g., Bradley, Prokop, Gentry, van der Heide, & Prieto, 1981).

Philips (1977) proposed a tri-component perspective on head pain, encompassing: (a) physiological change (muscle contraction, peripheral vasoconstriction or dilatation); (b) subjective concomitants (distress, fatigue); and (c) behavioural and motivational correlates (attention-seeking, self-medication). Not all components need appear in, or be associated with, the clinical presentation of headache. Inter-component associations are assumed to be mediated by psychological factors -- personality, attitudes, expectations, and the like.

Headache complaints can be differentiated along several descriptive dimensions, such as site, rapidity of onset, duration, severity, frequency, and association with neurological signs (Lance, 1978). Ideally, classification and diagnosis are firmly grounded in contemporary knowledge of pain mechanisms. Such has not always been the case, however, as considerations of taxonomy have taken precedence over those of etiology (Saper, 1983). There are several difficulties evident in headache classification schemes generally. While rigid inclusion and exclusion criteria are desirable, they serve to exclude the larger number of atypical cases observed in clinical practice (Pearce, 1975), particularly those characterized by mixtures or combinations of syndrome-specific symptoms (Saper, 1983). Clinical and experimental investigations of headache have been hindered by definitional imprecision with regard, for example, to the number of symptoms required to establish diagnosis (Olesen, 1978).

Currently, the most frequently employed diagnostic scheme in both research and clinical practice is that first proposed by the Ad Hoc Committee on the Classification of Headache (1962) of the National Institute (U.S.) of Neurological Diseases and Blindness. Through incorporating then current clinical and experimental research findings with both inference and consensual validation among its members, the Committee defined and delineated (Table 1)

Table 1

Classification of Headache

1.	Vascular Headache of Migraine Type
2.	Muscle-Contraction (Tension) Headache
3.	Mixed (Combined) Headache
4.	Psychogenic Headache (Headache of Delusional, Conversion, or Hypochondriacal States)
5.	Headache of Nasal Vasomotor Reactions
6.	Nonmigrainous Vascular Headache
7.	Traction Headache
8.	Headache Due to Overt Cranial Inflammation
9-13.	Headache Due to Disease of Ocular, Aural Nasal and Sinusal, Dental or other Cranial Neck Structures
14.	Cranial Neuritides
15.	Cranial Neuralgias

15 major varieties of headache disorder. For the purposes of this review, a distinction is drawn between those forms secondary to known physiological causes (Categories 5-15), and those not readily attributable to specific causes (Categories 1-4). Respectively, these forms correspond to the taxonomic classes of "organic" (secondary) and "idiopathic" (primary, essential, functional). The present review is confined to discussion of the latter class of headache -- migraine, muscle-contraction or tension headache, mixed or combined headache, and psychogenic headache -- as it is among these forms that psychological factors have been most often implicated.

Migraine Headache

Migraine is commonly defined as a headache disorder characterized by recurrent episodes of intense head pain of sudden onset -- migraine is often referred to as "paroxysmal" in nature -- varying widely in frequency, intensity, and duration (Lance, 1978). In its various clinical forms, migraine is estimated to affect between 15% and 30% of the adult population at one time or another (Markush, Karp, Heyman, & O'Fallon, 1975; Waters & O'Connor, 1975). Prevalence estimates of pediatric migraine range from 2.5% of all children under age seven or 1.4% of all pre-adolescents (Bille, 1962, 1981), to 5.5% of all children under age eleven (Oster, 1972). The disorder is more often seen clinically in women than in men, though approximately

60% of all pediatric cases are male (Prensky & Sommer, 1979; Raskin & Appenzeller, 1980), a developmental trend that reverses in the direction of higher prevalence among females after age eleven (Bille, 1981).

In most cases, migraine episodes appear unilaterally at onset, and may be associated with loss of appetite, nausea, and vomiting. Often, these are preceded by neurological or mood disturbances -- what are referred to as "prodromata". The disorder is polysymptomatic in nature, typically comprising central, peripheral, and autonomic features (Friedman, 1976). In fact, head pain is not invariably the key clinical feature. In the absence of actual headache complaint, there may appear sensory, motor, ophthalmologic, or behavioural manifestations. Many "migraineurs" report the belief that their episodes are precipitated by any number of specific or non-specific stimuli or events, such as fatigue, intense or flickering light, ingestion of certain foods, exposure to high altitudes, or meteorologic changes.

Where head pain is the central feature of the episode, it is most often experienced unilaterally in the frontal and temporal regions, but may radiate through other regions of the head, or project downward into the neck and shoulders (Lance, 1978). Typically, pain quality is described as "dull" at onset, increasing rapidly in severity, and assuming a pulsating or "throbbing" quality that wanes as

the episode progresses. Headache duration is typically under 24 hours, though exhaustion and lethargy may persist thereafter. Episodes are relatively infrequent (1 to 10 per month), but may occur at any time of the day.

The Ad Hoc Committee (1962) identified five variants of the migraine syndrome: (a) classic migraine, characterized by a sharply-defined prodromal phase, unilateral throbbing head pain, loss of appetite and nausea or vomiting; (b) common migraine, which is not accompanied by clear prodromata, and may be unilaterally distributed and of longer duration than the classic form; (c) hemiplegic (ophthalmologic) migraine, in which head pain is associated with ipsilateral visual phenomena, extraocular muscle palsy, and temporary hemiparesis or hemiplegia; (d) basilar artery migraine, which is associated with severe and throbbing occipital head pain and vomiting preceded by clear prodromata; and (e) cluster headache, characterized by unilateral head pain, flushing of the skin, perspiration, lacrimation, and rhinorrhea. Of these, the common, classic, and cluster variants have been the most studied.

In its classic form, migraine occurs bi-phasically. The initial or prodromal phase may appear transiently and inconsistently, or regularly, developing slowly over 10 to 60 minutes preceding head pain (Friedman, 1975). The most commonly reported pre-headache "warning" symptoms include visual disturbances -- fortification spectra, hemianopia,

hemianopic scotomata, illusions of moving lights, zig-zag patterns -- which appear anywhere from 30 seconds to 60 minutes prior to head pain onset. These may affect both visual fields sequentially (Wakefield, 1975). Also common are sensory distortions (e.g., numbness or tingling in the face or hands), alterations of consciousness or awareness (e.g., an undue sense of well-being despite prior experience with phasic headache symptomatology), or motor phenomena (e.g., temporary hemiparesis). While the precise pathology underlying these alterations is not well understood, there appears to be consensus in favour of cerebral vasoconstriction as either a cause or concomitant. The headache phase of the migraine episode most often involves unilateral head pain as described above, and appears to be associated with a "rebound" vasodilatation, perhaps mediated by autoregulatory mechanisms that serve to restore full blood flow to regions partially deprived during the vasoconstrictive prodromal phase (Wakefield, 1975).

Current evidence suggests that headache associated with age of onset during childhood to early adulthood is usually migrainous; onset in midlife is more suggestive of the partial influence of psychological factors and tension headache symptomatology, while onset during later years may indicate organic pathology (Kunkel, 1979). The various subtypes may affect the same individual over the lifespan, but the common variant is far more prevalent than is the

classic (Wakefield, 1975). The symptom profile in pediatric migraine closely approximates that observed in adults (Ryan & Ryan, 1978), but is more likely to be characterized by a preponderance of abdominal and gastrointestinal features over actual head pain. With age, a general increase in headache symptomatology is accompanied by a corresponding diminution of abdominal complaints.

The prognosis in pediatric migraine is generally favourable. Approximately 33% of confirmed cases show complete remission on follow-up in adolescence; the remainder show at least some improvement (Prensky & Sommer, 1979; Saper, 1983). There is also evidence that males show substantially greater improvement with age than do females; prevalence estimates are slightly higher for male children, while the majority of adult migraineurs are women (Bille, 1981; Hockaday, 1978; Rothner, 1979). Sex differences in prognosis may be attributable in part to the provoking impact of estrogen in females on migraine predisposition (Saper, 1983).

Muscle-Contraction (Tension) Headache

Muscle-contraction or tension headache is generally thought to be the most common form of head pain complaint in adults (Friedman, 1979; Kudrow, 1976; Lance, 1978; Martin, 1972). Estimates of its prevalence in the adult general population range from 20% to 25% or higher (Ostfeld, 1962; Waters &

O'Connor, 1975). While the disorder is also thought common in childhood, most forms of pediatric headache appear to be vascular (Rothner, 1979; Ryan & Ryan, 1978).

Martin (1972) provided a graphic description of the syndrome:

> Usually, the muscle contraction headache is described as being a steady nonthrobbing ache. Patients may describe the sensation of "tightness", "pressure", "drawing", and "soreness". Usually these head pains and their related sensations are bilateral and most frequently center about the occiput and posterior cervical regions. They may extend to the temples, jaws, portions of the face, or top of the head. Frequently, a "band-like" constriction about the head is noted. This may be compared to the sensation of pain by prolonged wearing of a hat that is too tight. Commonly the scalp is tender, and the patient may complain that combing or brushing the hair intensifies the distress. Such headaches may be fleeting or they may persist for days. Tension headache most frequently occurs in the afternoon or evening, but may be present after what has seemed to be a sound sleep. (p. 16)

In this conceptualization, severity of head pain is assumed to be a direct consequence of, and in direct proportion to, the degree of contraction present in implicated muscle groups (Ostfeld, 1962). Thus, a causal link is drawn between a putative physiological state and a resultant subjective experience of pain (Philips, 1980).

Unlike migraine, tension headache is generally experienced as bilateral and occipital-suboccipital rather than frontal and unilateral-frontal-suborbital in location, dull and aching rather than throbbing, frequent rather than infrequent in occurrence, and lacking in identifiable

prodromata. Typically, headache onset is gradual, and perceived to coincide with or follow emotional or situational stresses (Friedman, 1979; Martin, 1972).

Combined (Migraine-Tension) Headache

Combined headache comprises a syndrome marked by concomitance of both migraine and tension headache symptoms (Ad Hoc Committee, 1962; Kunkel, 1979). Clinical presentation may comprise any combination of vascular and muscle-contraction features, but the most common complaint is of a tension-like headache. Episodes may occur daily or almost daily, and are periodically accompanied by transient migrainous symptoms or other vascular phenomena. In certain cases, acute migrainous features may appear against a tonic background of tension headache symptoms.

The syndrome is relatively rare in children, but becomes more prevalent during the adolescent years (Rothner, 1979). Actual estimates of prevalence in both pediatric and older populations have been difficult to obtain, primarily because the syndrome presents a complex diagnostic picture.

Psychogenic Headache

Also referred to as headache of delusional, conversion, or hypochondriacal states, psychogenic headache has been defined as a syndrome in which, "the prevailing clinical disorder is a delusional or a conversion reaction and a

peripheral pain mechanism is nonexistent" (Ad Hoc Committee, 1962, p.128). Some disagreement exists as to the prevalence of the disorder; while some writers have noted the relative paucity of pre-adolescent cases (e.g., Barlow, 1979), others have described psychogenic headache as the most common variant among pediatric cases, suggesting a prevalence as high as 24% of all extant headache complaints (Rothner, 1979; Ryan & Ryan, 1978).

To the extent that the experience of pain is generally associated with some alteration in affective state, all forms of headache can be said to involve a "psychogenic" component (Weatherhead, 1980). For this reason, confusion has persisted with regard to use of the category; it has been applied to virtually every variant of idiopathic headache in which psychological factors have been observed. Packard (1976) reported that most physicians surveyed applied the diagnosis to tension-like headaches having no ostensible organic basis, and to headaches perceived as secondary to stress. None regarded migraine as a psychogenic variant.

Weatherhead (1980) proposed that the mere perception of psychogenic factors could not be taken as the defining feature of the disorder, and that the diagnosis should not be applied to tension-like headache per se. Rather, the term should be used only in reference to a predominately psychological disorder in which headache is the chief

presenting complaint. Variants of the syndrome include tension-like headache associated with generalized anxiety, hysterical forms of headache complaint characterized by conversion symptomatology, and headache secondary to "reactive" depression. Evidence of psychogenic precipitation generally suggests the need for evaluation of current and long standing personality functioning.

While its status as a diagnostic entity is currently in question, considerable confusion and error might be averted by restricting application to clinical presentations of headache that closely approximate the syndrome as defined by the Ad Hoc Committee (1962).

Problems of Headache Classification

The preceding reviews of the idiopathic forms of headache are drawn from several authoritative sources (Ad Hoc Committee, 1962; Friedman, 1976; Kudrow, 1976; Kunkel, 1979; Weatherhead, 1980). Actual clinical presentations seldom conform to "textbook" descriptions, however, and several authors have commented on the problems inherent in attempting to differentiate clearly among individual headache complaints (Friedman, 1979; Lance, 1978; Martin, 1972; Waldbott, 1962; Waters, 1970).

According to Ziegler (1979), "The first unsolved problem in the field of headache relates to terminology. Is any specific headache migrainous or tension? Clinicians have

long dealt with this problem by defining many episodes as 'combined', but this begs the fundamental question" (p. 444). Headache specialists have tended to accept the validity of the Ad Hoc Committee's (1962) classification scheme, which Ziegler (1979) cautioned serves at best to differentiate only among headache complaints for which there are determinable organic bases, such as head pain secondary to trauma or ocular disease. Among the idiopathic variants, etiology is largely undetermined or multifaceted. Clinical presentation often comprises a mixture of symptoms suggestive of several diagnostic forms (Lance, 1978; Waldbott, 1962). As such, differential diagnosis is often complicated by symptom heterogeneity.

Classification of migraine headache. The current conceptualization of migraine, which in the main resembles that first proposed by the Ad Hoc Committee (1962), comprises little more than a clinical description of the typical vascular episode (Waters & O'Connor, 1971). At present, there is no definitive "test" for migraine, and diagnostic accuracy is dependent on the depth and clarity of the patient's symptom report. While there is some evidence for specific vascular changes either during or between episodes, the disorder is widely regarded as functional to the extent of not being linked to any known pathophysiology (Appenzeller, Davison, & Marshall, 1963; Walshe, 1969; Wolff, 1963). Electroencephalographic (EEG) studies have

reported some mild abnormalities in migraineurs relative to non-migraineurs (Hockaday & Whitty, 1969; Hoefer, 1967; Lees & Watkins, 1963; Selby & Lance, 1960). These findings may not be generalizable, as they were derived from self-selected clinical populations (Parsonage, 1975). In conclusion, the validity of the migraine variant has not been enhanced by attempts to identify any unique underlying pathophysiology.

Bakal and Kaganov (1979) examined symptom profiles among "chronic" and "non-chronic" migraine and tension headache sufferers. Those who reported frequent and severe episodes were found to be equally familiar with symptoms and head pain locations thought characteristic of, and specific to, one or the other syndrome. This finding suggests that, in several respects, migraine and tension headache patients may be more similar than dissimilar. To the extent that this is so, differential diagnosis may be complicated by symptom heterogeneity where relative specificity is expected. The clinician or researcher may be faced with the task of determining, by whatever criteria, which and how many uniquely migrainous features are required to establish a diagnosis. To date, attempts to identify isolated defining characteristics have been problematic (e.g., Ziegler, Hassanein, & Hassanein, 1972).

Classification of tension headache. The use of the term "tension" is intended to highlight the distinct features of

the syndrome (Olton & Noonberg, 1980): the subjective experience of head pain; a sustained contraction of musculature in the scalp, neck, and shoulder regions; and certain personality characteristics or "lifestyle" features of the patient. To a degree, evaluating the tension headache literature involves first determining an author's intended usage of the term. The orientation or focus of a given publication is largely dependent on the author's particular field of expertise; specialists in psychosomatic medicine might be expected to hold somewhat different views on the dynamics of tension headache than those common to neurology or psychophysiology. Evaluation of the literature relies in part on the standardization of key terms. In this respect, the criteria first proposed by the Ad Hoc Committee (1962) are not sufficiently specific to facilitate the degree of consistency required for generalization across different studies and populations.

It is widely held that sustained muscle contraction constitutes the principal defining feature of tension headache (Ziegler, 1979). Recent psychophysiological studies, however, have demonstrated that elevated muscle contraction is no more characteristic of tension headache than of other idiopathic variants (Acosta, Yamamoto, & Wilcox, 1978; Magora, Magora, & Abramsky, 1974; Philips, 1977; Pozniak-Patewicz, 1976). In fact, Bakal and Kaganov (1977) reported higher resting levels of muscle tension in a

group of migraineurs relative to tension headache subjects. Although both groups showed higher resting tension levels than did non-headache controls, they did not differ significantly from one another.

Philips (1978) derived and evaluated seven key assumptions implied in the Ad Hoc Committee's (1962) conceptualization of tension headache:

1. Sustained contraction of key musculature in the scalp, neck, and shoulder regions is a necessary precondition for the development and maintenance of the disorder. The Ad Hoc Committee's (1962) description implied a direct correspondence between muscle tension and head pain: "It is associated with sustained contraction of skeletal muscles in the absence of permanent structural change, usually as a part of the individual's reaction during life stress" (p. 128). This statement has often been interpreted as implying a causal link. Overall, the muscle contraction hypothesis has received only partial confirmation (e.g., Bakal & Kaganov, 1977) and, as such, elevated tension cannot be regarded as the key defining feature of the disorder.
2. A strong association exists among headache severity and frequency, resting levels of muscle tension, and behaviours motivated by head pain. Utilizing self-medication rate per unit of time as a measure of

pain-motivated behaviour, Philips (1977) reported a weak association between medication frequency and headache activity; medication frequency and resting muscle tension were inversely related. These findings have not since been replicated, however, and have been discussed only briefly in the author's more extensive reviews of the psychophysiology of tension headache (Philips, 1977, 1980). To the extent that medication frequency provides a valid and sensitive measure of pain-motivated behaviour, Philips' preliminary observations seem somewhat puzzling. Whether, as Philips (1978) has maintained, the second assumption of the tension headache model is inherent in many contemporary treatments (e.g., relaxation training), an emphasis on medication habits alone offers little to further our understanding of the association between muscle contraction and head pain. Given the widespread acceptance of this assumption, however, it should be of greater interest to researchers.

3. During headache, a close correspondence exists between pain intensity and tension or contraction in the implicated musculature. Philips (1977) failed to demonstrate significant elevations from normal resting levels in any of four muscle groups monitored -- neck, trapezium, frontalis, temporalis -- during either headache or non-headache measurement periods.

Several investigators have failed to link head pain and muscle tension (e.g., Epstein & Abel, 1977; Epstein, Abel, Collins, Parker, & Cinciripini, 1978; Martin & Mathews, 1978; Thompson, Haber, & Tearnan, 1981). Martin (1983) reasoned that these findings reflected partial "contamination" of tension headache samples by psychogenic cases, suggesting the need for more rigorous pretreatment evaluation. In any event, there is little conclusive evidence for the third assumption.

4. Frontalis muscle tension alone can be taken as indicative of the individual's muscle-contraction profile. Although frontalis (forehead) tension is widely regarded as the principal determinant of pain in tension headache, it cannot be assumed to be predictive of tension in other sites in the manner implied (Philips, 1977, 1978). Generalization of muscle tension and relaxation is an issue central to the evaluation of relaxation-based interventions (Stoyva, 1979), but remains largely unresolved at this time. Only further research will establish the validity of the fourth assumption.
5. Elevated tension is a tonic (sustained) condition, and should be evident in resting musculature, even during headache-free periods. Tonic abnormalities are observed in few cases (Philips, 1978), and some investigators have reported failure to demonstrate

even phasic (transient) muscle tension responses to subject-defined stressors (Bakal & Kaganov, 1977; Philips, 1977). As such, the fifth assumption is not supported.

6. Increments from resting tension level are associated with head pain. While there is some evidence that experimental stimulation of sustained muscle tension is associated with pain complaint, the actual intensity of pain elicited by these techniques is substantially less than that typically reported by headache sufferers, even though absolute tension levels during simulation procedures are actually higher (Philips, 1977). These observations contradict the sixth assumption.

7. Induced decrements in tension are associated with reductions in headache severity and duration; long-term reductions in tension lead to lower headache frequency. This assumption is widely held despite, for example, evidence that muscle relaxants are generally ineffective in the treatment of tension headache (Ostfeld, 1962). Several studies have reported virtually no relationship between muscle tension levels and treatment outcome (Andrasik & Holroyd, 1980; Epstein & Abel, 1977; Haynes, Griffin, Mooney, & Parise, 1975; Holroyd & Andrasik, 1978; Holroyd, Andrasik, & Westbrook, 1977). Although reductions in muscle tension may be associated with

reductions in pain intensity, the two variables evidence different rates of response, suggesting their dissociation in the clinical presentation of headache.

Philips (1978) concluded that tension headache and migraine subjects could not be clearly differentiated on the basis of muscle tension. In general, the psychophysiological approach to tension headache is somewhat problematic, in that the individual may not evidence either an abnormal muscle tension, or an association with headache complaint (Philips, 1980). These studies have assumed a certain homogeneity of pain behaviour among patients assigned the same diagnoses, largely ignoring the subjective dimensions of headache. Most often, there has been failure, either to support the muscle-contraction model, or to identify alternative pathogenic mechanisms (Martin, 1983).

Alternatives to Typological Classification

Although the classification scheme proposed by the Ad Hoc Committee (1962) has clearly demonstrated its utility in differentiating between primary and secondary forms of headache, it has proven more problematic in the delineation of idiopathic subtypes (Ziegler, 1979). Nevertheless, it has met with widespread acceptance in both the clinical and research communities, and few competing systems have emerged since its introduction. There have, however, been promising developments.

Ziegler, Hassanein, and Hassanein (1972) examined interrelationships among migraine symptoms through factor analysis. A group of 300 patients presenting with recurrent headache was administered a 27-item symptom questionnaire. Principal components analysis yielded a small number of factors comprising distinct symptom clusters, characterized by: nearly constant head pain, with some tension, but no migrainous, features; unilateral head pain focused above the eye; nausea before and during head pain; and focal neurological symptoms before, during, or after head pain. No single factor comprised all of the essential features of migraine -- unilateral head pain, nausea, visual prodromata -- and the symptom clusters obtained did not correspond closely to clinical descriptions of the disorder.

Using a similar approach, Kroner (1983) administered a comprehensive health questionnaire containing 82 headache-related items to a group of 302 chronic headache patients. Factor analysis yielded two stable symptom clusters, corresponding closely to the Ad Hoc Committee's (1962) descriptions of migraine and tension headache.

Although innovative, these approaches were nevertheless problematic. Rather than develop alternatives to typological classification, the authors chose not to challenge the authority of the existing scheme. For example, Ziegler, Hassanein, and Hassanein (1972) interpreted certain of their factors as more or less descriptive of migraine or tension

headache, and simply excluded from further analyses what they assumed to be "irrelevant" factors. As such, their interpretations, and those of Kroner (1983), were bound by extant conceptualizations of headache.

The Diagnostic Process and Headache Classification

The medical diagnosis of headache has been described in detail by Kunkel (1979) and Saper (1983) as comprising a clinical interview, a comprehensive headache history, physical examination, and specific diagnostic studies as indicated. Throughout, the process is subject to errors of self-report and retrospection.

Diagnosis usually begins with a detailed clinical interview, addressing the patient's health background, family history of headache, dietary habits, substance use or abuse, and details of current daily activities including employment (e.g., are there specific stressors associated with the patient's work or work environment?). A comprehensive headache history should follow, focusing on the patient's pain complaint, including age and circumstances of initial onset (e.g., early onset in childhood or adolescence is more suggestive of migraine); head pain location; frequency (e.g., migraine is supposedly episodic and relatively infrequent, while tension or psychogenic headache may occur almost daily); duration; pain quality; diurnal qualities; prodromal features and

associated symptoms; precipitating, exacerbating, or relieving factors; and other pertinent information.

Physical examination is required to rule out organic pathology. Where secondary headache is suspected, specific diagnostic studies (EEG, Computerized Axial Tomography, angiography) are usually ordered. There are, however, no specific tests for idiopathic headache.

Headache diagnosis is subject to several errors (Friedman, 1979). The patient's headache history is most often varied, and not clearly suggestive of any specific syndrome. He or she may display different headache features at different times, and it is rare for a migraineur not to experience tension-like or combined headache on some occasion. More problematic is the observation that milder headache complaints are seldom the focus of clinical presentation (Andrasik, Holroyd, & Abel, 1979; Banks, Beresford, Morrell, Waller, & Watkins, 1975; Olton & Noonberg, 1980; Waters & O'Connor, 1975). The more numerous and intense the symptoms experienced, the more likely is the patient to seek medical attention. As such, clinical samples are by definition non-representative.

There is little hard evidence bearing on the validity or reliability of headache diagnosis (Hoelscher & Lichstein, 1984). Blanchard, O'Keefe, Neff, Jurish, and Andrasik (1981) compared the diagnoses of a certified neurologist

with those of a psychologist for a group of adult headache patients seeking treatment. They reported moderate to high concordance on diagnoses of migraine (84.2% agreement) and combined headache (61.5% agreement), suggesting good interdisciplinary reliability in headache diagnosis where strict inclusion-exclusion criteria are applied.

Differential diagnosis. In practice, headache complaints are differentiated on the basis of symptom profile. Migraine and tension headache, for example, are usually differentiated on the basis of such factors as pain location (unilateral versus bilateral distribution), quality (throbbing or pulsing versus dull and aching), onset characteristics (presence versus absence of prodromata), and associated symptomatology (presence versus absence of visual or gastrointestinal complaints). Clear differentiation may not always be possible, however, in that the patient often presents a varied symptom profile (Olton & Noonberg, 1980; Ziegler, Hassanein, & Hassanein, 1972). Among migraineurs, for example, vascular episodes are often associated with, or preceded by, tension-like head pain (Pearce, 1977). In some cases, tension headache may be a precursor to migraine.

Because symptom heterogeneity is so common, clear guidelines for differential diagnosis are needed (Friedman, 1979). These should be based on established knowledge of both the mechanisms and expression of head pain, and on reliable and verifiable headache history data derived from

defined populations. Because primary diagnosis of idiopathic headache is based on verbal reports of pain rather than specific diagnostic tests, differentiation is often a process of "diagnosis by exclusion". Thus, tension headache is often diagnosed by ruling out migraine through absence of prodromal features or associated symptoms. Similarly, adequate criteria are lacking for the differentiation of tension and psychogenic headache. In a sense, the emphasis placed on the migraine-tension dichotomy has served to obscure both the combined and psychogenic variants (Thompson & Figueroa, 1983). There is mounting evidence that a substantially greater number of chronic headache patients meet the diagnostic criteria for combined headache than for either migraine or tension headache (Anderson & Franks, 1981; Cohen, 1978), and that tension headache samples employed in clinical research are often at least partially contaminated by combined or psychogenic cases (Haber, Kuczmierczyk, & Adams, 1985; Martin, 1983).

Wolff's early investigations of headache during the 1930's and 1940's (cited in Dalessio, 1972) reported the presence of tension headache symptoms in many migraineurs. These symptoms were thought to be secondary to migraine, and no causal significance was ascribed to their occurrence. From Wolff's time forward, the belief in a clear migraine-tension dichotomy persisted, with few challenges. In recent years, and particularly since the publication of a

critical paper by Bakal (1975), several studies and reviews have challenged this conceptualization (Anderson & Franks, 1981; Andrasik, Blanchard, Arena, Saunders, & Barron, 1982; Bakal & Kaganov, 1977, 1979; Gannon, Haynes, Safranek, & Hamilton, 1981; Haynes, Cuevas, & Gannon, 1982; Kaganov, Bakal, & Dunn, 1981; Philips, 1977, 1978; Philips & Hunter, 1982; Sutton & Belar, 1982; Thompson & Figueroa, 1983; Thompson, Haber, Figueroa, & Adams, 1980). Bakal's work is discussed at greater length in a later section of this review; it need only be noted here that the usual approach to differential diagnosis has met with recent criticism.

Bakal and Kaganov (1977) had tension and migraine headache subjects monitor headache activity and pain locations daily over a 2-week period. All were found to be equally familiar with symptoms and locations believed characteristic of one, but not the other, headache type. Symptom heterogeneity was evident in both groups, as were relatively unique symptom profiles, regardless of prior diagnosis. Similarly, Bakal and Kaganov (1979) compared symptom profiles of occasional versus chronic headache sufferers. Those initially diagnosed with migraine were familiar with all symptoms and pain locations, and reported experiencing these more often than did subjects initially assigned the diagnosis of tension headache. Further, migraineurs reported a greater frequency of tension headache than vascular symptoms. These findings were replicated by Thompson, Haber, Figueroa, and Adams (1980).

Kaganov, Bakal, and Dunn (1981) reported similar observations of symptomatology in a non-clinic population. The data suggested that, the more headache was perceived as bothersome, the more likely it was to comprise both tension and vascular features, the latter being associated with more severe episodes. Overall, symptom profiles were similar among problem and non-problem headache subjects. There was no evidence of symptom clustering consistent with typological groupings.

In recent years, psychophysiological assessment techniques have been increasingly employed in efforts to clarify the distinction between migraine and tension headache. Weatherhead (1980) proposed that, because the pain mechanisms underlying psychogenic headache are either undetectable or non-existent, differential diagnosis with tension headache must rely on the evaluation of key physiological variables, such as muscle tension. He further noted that single-site response measures -- frontalis electromyographic (EMG) activity, for example -- were inadequate to the task of differentiation.

Overall, psychophysiological investigations of headache have yielded conflicting findings with regard to differentiation (Anderson & Franks, 1981; Bakal & Kaganov, 1977, 1979; Cohen, 1978; Morley, 1977; Vaughn, Pall, & Haynes, 1977), reflecting in part the paucity of studies comparing headache disorders along physical dimensions. To

date, the majority of these has reported mainly frontalis EMG in tension headache subjects (e.g., Bakal & Kaganov, 1977; Vaughn, Pall, & Haynes, 1977), or indirect measures of vascular activity in migraineurs (e.g., Cohen, 1978; Morley, 1977). Pozniak-Patewicz (1976) reported higher resting levels of tension in the scalp and neck muscles of migraineurs relative to tension headache subjects, during both headache and headache-free periods. He concluded, however, that muscle-contraction was of only minor significance in both types.

Anderson and Franks (1981) compared migraine, tension, and headache-free individuals on a variety of psychophysiological measures during simulated conditions of stress and relaxation. Under resting conditions, tension headache subjects did not evidence higher frontalis or forearm EMG activity, or differ in blood pressure, heart rate, peripheral temperature, or electrodermal activity (EDA). Migraineurs exhibited a non-significant trend toward lower peripheral temperature and higher heart rate during rest, in a manner suggestive of a generalized vasomotor abnormality. No group differences were reported for any of the measures, in either displacement from resting to stress levels, or average levels during stress. Overall, there was no clear evidence of any relationship between headache activity and frontalis tension in tension headache subjects; the findings for migraineurs were less conclusive.

Evidence to date suggests that psychophysiological measures, particularly frontalis EMG, have only limited utility in the differential diagnosis of idiopathic headache. In fact, there have been reports that some migraineurs display higher resting levels of muscle tension during headache-free intervals than do tension headache patients (Philips, 1977), suggesting that muscle tension may not be the appropriate criterion for differentiation. Further, the episodic nature of migraine renders it difficult to investigate through psychophysiological means (Botney, 1981). Still, the approach is common to much of the current assessment and intervention literature.

The Psychobiological Model of Headache

Thompson and Figueroa (1983) noted two significant developments in the conceptualization of headache during the preceding two decades. The first of these comprised the system of classification proposed by the Ad Hoc Committee (1962), prior to which idiopathic headache was believed to indicate an underlying neurological disorder. Theirs was the first attempt to develop a systematic headache typology, one which would meet with and maintain widespread acceptance in the medical community.

The second important conceptualization of headache appeared in the early writings of Bakal and his associates (Bakal, 1975, 1980, 1982; Bakal & Kaganov, 1977, 1979;

Bakal, Kaganov, & Demjen, 1983; Kaganov, Bakal, & Dunn, 1981). In the "psychobiological" model (e.g., Bakal, 1975), headache disorders are conceived as existing along a continuum of severity, rather than in terms of a symptom-determined typology. Differences among major idiopathic variants are viewed as quantitative rather than qualitative.

As is noted above, headache patients seldom display symptom profiles consistent with textbook descriptions. Similarities in reported features among headache patients suggest common mechanisms in the development and maintenance of the disorder (Bakal, Kaganov, & Demjen, 1983). In Bakal's model, headache results from a complex interplay of environmental, physiological, genetic-biochemical, and psychological factors. This perspective has facilitated a shift in emphasis away from hypothesized psychological antecedents toward the processes that mediate and maintain headache.

Central to this conceptualization is the notion of a psychobiological "predisposition" for headache, which emerges in the presence of stressful stimuli or events, and plays a critical role in headache episodes that seem to be precipitated by identifiable antecedents (e.g., psychosocial stress, illness, ingestion of certain foodstuffs), or that seem to appear spontaneously. The transition from episodic to chronic headache results from an increasing autonomy of

underlying physiological mechanisms. Over time and repeated episodes, the individual's predisposition for headache may come to function more or less independently of the psychosocial stresses that, in earlier stages of the disorder, play a triggering or precipitating role.

In so-called tension headache, persistent muscle contraction may arise as a physiological response to perceived stress. Over time, this response may come to function independently of stress. On examination, the individual may report a persistent sensation of tightness or pressure in the head and neck regions, and the perception that his or her headaches are not precipitated by recognizable causes. In this manner, a pattern of repeated episodes may establish itself, reflecting the interaction of an apparent inability to effectively cope with head pain of mild to moderate severity during early stages of the disorder, and progressive involvement and precedence of physiological mechanisms that mediate headache and headache predisposition.

Over time, vascular features may also appear in the headache profile, accompanied by a variety of autonomically-mediated symptoms, such as nausea or vomiting. Chronic headache thus represents one extreme of a severity continuum, rather than a unique disorder distinct from less frequent or severe forms (Bakal & Kaganov, 1979). Similar psychobiological processes are assumed to underlie all

chronic headache complaints, though actual mechanisms of head pain may differ from case to case.

Evidence for the psychobiological model. According to Featherstone (1985), "There is increasing, undismissable evidence that idiopathic headaches are a single continuum of a multiple-symptom process, of varying severity and of varying symptom combinations, which is satisfactorily described by the headache severity model" (p. 197). The author proposed a variation on Bakal's (1975) model, in which headaches of mild severity are characterized by few associated symptoms; as severity increases, so too do the number and variety of symptoms.

Several lines of evidence converge in support of the severity-continuum perspective. Many studies have reported that both muscle contraction and vascular symptoms are associated more with head pain severity than with specific headache syndromes or types (Bakal & Kaganov, 1979; Deubner, 1977; Drummond & Lance, 1984; Featherstone, 1985; Kaganov, Bakal, & Dunn, 1981; Newland, Illis, Robinson, Batchelor, & Waters, 1978; Waters, 1973). Symptoms believed specific to migraine -- nausea, vomiting, unilateral head pain having a throbbing quality -- have also been reported in supposedly confirmed cases of tension headache (Allen & Weinmann, 1982; Thompson, Haber, Figueroa, & Adams, 1980). Both variants may be associated with muscle tenderness during headache (Oleson, 1978; Tfelt-Hansen, Lous, & Oleson, 1981).

Biological investigations of headache have established certain similarities between migraine and tension headache, on measures of EMG (Bakal & Kaganov, 1977; Pozniak-Patewicz, 1976), blood platelet (Muck-Seler, Deanovic, & Dupel, 1979; Rolf, Wiele, & Brune, 1981), and vasomotor (Cohen, 1978) activities. The only consistently reported physiological difference has been the observation of a dilatation of the scalp vasculature during the headache phase of migraine (Featherstone, 1985).

Studies of personality and other psychological variables are examined in Chapter 3 of the present review. It need only be noted here that personality differences between tension headache and migraine patients have not been reliably demonstrated (Andrasik, Blanchard, Arena, Teders, & Rodichok, 1982; Kudrow & Sutkus, 1979; Sternbach, Dalessio, Kunzel, & Bowman, 1980; Werder, Sargent, & Coyne, 1981). Similarly, treatment outcome studies have documented few differences in effectiveness among different headache treatments; clinicians have reported equal success in treating both migraine and tension headache with analgesics, tricyclic antidepressants, or non-steroidal anti-inflammatants (Carasso, Yehuda, & Streifler, 1979; Diamond & Medina, 1981; Mathew, 1981; Saper, 1978). Only the ergot compounds (e.g., ergotamine) have proven specificity of action in the treatment of migraine (Cohen, 1978).

Taken together, the above observations seem most supportive of the severity-continuum perspective on headache, as a viable and heuristic alternative to the typological approach. Generally speaking, however, the medical community has been slow to accept a unitary model of headache mechanisms (Bakal, 1980). While the occurrence of tension headache symptomatology in migraineurs is widely acknowledged, it is most often interpreted as evidence of a susceptibility to other headache disorders; that many tension headache patients also experience vascular symptoms is usually overlooked or ignored.

Etiological Factors in Headache

Current interventions for headache tend to be symptom-focused rather than prophylactic, and fail to adequately account for either antecedents or consequences of headache activity (Martin, 1983). If, for example, depressed affect were found to be a consistent antecedent of headache, then it might be desirable to devise a treatment that would facilitate greater sensitivity to, and control over, mood status. Ideally, treatment would be directed at underlying causes rather than presenting symptoms alone, the ultimate goal being prevention. The process would need to begin, however, with the identification of etiological factors.

In examining the etiology of headache, it should be noted that idiopathic head pain is a relatively non-specific

complaint, suggestive of a potentially broad range of medical conditions (Friedman, 1979). Neither location nor intensity of pain provide reliable clues to etiology, and none of the idiopathic variants is consistent with regard to these and other features. A variety of factors can interact in the precipitation, exacerbation, maintenance, and alleviation of headache (Korczyn, Carel, & Pereg, 1980).

One approach to examining etiology involves the study of associations among head pain and other physical complaints or disorders. Featherstone (1985) reviewed the medical records of 1,414 life insurance applicants, and obtained 200 cases of confirmed or suspected idiopathic headache. The complaint of recurrent head pain was associated with higher prevalences of hypertension, vertigo, gastroesophageal reflux, peptic ulcer disease, depression, anxiety, and (in men) irritable bowel syndrome. There have been further reports of associations among headache and disorders of vestibular function (Kuritzky, Toglia, & Thomas, 1981; Moretti, Manzoni, Caffaira, & Parma, 1980), essential hypertension (Graham, Bana, & Yap, 1978), and depression and anxiety (Cox & Thomas, 1981; Ziegler, 1979).

Etiology of migraine. Over the years, several etiological models of migraine have been proposed. Wolff (cited in Dalessio, 1972) posited a mechanical or chemical stimulation of peripheral pain receptors. Sicuteri, Anselim, and Fanciullaci (1973) postulated the role of a

hypersensitivity to serotonin in the central nervous system, while Dalessio (1978) implicated inflammatory responses in extracranial tissue. None of these models has generated supportive research.

To date, a study reported by Bille (1981) constitutes the only systematic longitudinal investigation of the inheritance of migraine in the headache literature. A 23-year follow-up was conducted on a group of former pediatric migraine cases, greater than 50% of whom currently had at least one child aged four or younger. Of these, approximately 30% had one or more child with migraine. The examination of hereditary factors in migraine is complicated by the fact that a positive family history is itself employed as a diagnostic criterion; the majority of patients examined will therefore report a positive family history (McGrath, 1983). Further, determination of inheritance is usually accomplished through patient interviews rather than by examination of relatives or their medical records. The role of heredity in migraine remains largely unresolved, and progress relies in part on developments in other areas of research (Smith, 1980).

Although it is widely believed that migraine is linked to psychosocial stress, there is little supportive evidence for this association (Anderson, 1980). Henryk-Gutt and Rees (1973) reported that 54% of headache episodes in an adult sample were perceived as secondary to stress. Several

authors have suggested that both migraine and tension headache patients report greater life stress because of personality styles that consistently draw them into conflict with their environments. Among these individuals in particular, stress is often perceived as a headache precipitant (Anderson, 1980; Andrasik, Blanchard, Arena, Teders, Teevan, & Rodichok, 1982; Friedman, 1979; Henryk-Gutt & Rees, 1973). Bakal (1975, 1982) might argue that migraine initially perceived as secondary to stress could be expected to develop functional autonomy over time, such that both vascular features and the anticipation of head pain might themselves act as stressors, provoking further pain. To date, a link between migraine and stress has not been conclusively demonstrated.

There is some evidence, based mainly on clinical observations, of family environments characterized by rigidly enforced norms of behaviour, emphasis on individual achievement, and the use of subtle forms of punishment for nonconformity or noncompliance (Friedman, 1975). In this view, the migraineur perceives no outlet for the expression of negative emotion, particularly anger, and this conflict emerges as a major precipitant of head pain. In the absence of controlled prospective studies on the family environments of migraineurs, however, the hypothesis is difficult to evaluate.

The etiology of migraine appears complex; any purported "cause" is probably too simplistic. The wide variety of hypothesized causes suggests a diversity of pathological mechanisms in the disorder (Pearce, 1975).

Etiology of tension headache. Historically, the etiology of tension headache has been presumed largely psychophysiological; head pain is viewed as secondary to sustained contraction of key muscles in the head, neck, and shoulder regions. Pikoff (1984) and Haynes, Cuevas, and Gannon (1982) summarized the evidence for this model as follows: Experimental investigations of muscle contraction in tension headache subjects have yielded inconsistent findings. Although elevated muscle tension is significant in some cases, it does not account for a majority of the variance in headache activity. Similarly, studies of vascular factors have been equivocal; it is not clear whether tension headache is associated more typically with localized vasoconstriction and ischemia, or with localized vasodilation. Overall, research to date has lacked sufficient specification of primary and differential diagnostic criteria, making comparisons among different studies and populations difficult. Further, the stress analogues typically employed in simulation studies have usually comprised discrete physical or cognitive stimuli, whereas patient-defined precipitants of headache are more often tonic, psychosocial, and environmental in nature (Diamond & Dalessio, 1978).

The clinical presentation of tension headache comprises a striking degree of individual variation in extent of association between psychophysiological variables and head pain complaint, and it is not possible to infer causal links among these factors (Haynes, Cuevas, & Gannon, 1982). To date, no study has adequately accounted for the possibility that tension headache itself produces changes in both vasomotor and musculoskeletal functioning.

Precipitants of headache. Many headache patients, particularly migraineurs, identify specific stimuli or situations as precipitants or aggravants of headache. The list of factors implicated includes substance use or abuse (e.g., alcohol or drugs), interpersonal conflict, emotional upset, fatigue, modelling of headache-associated behaviours, secondary gain for head pain complaint, anxiety, tension, depression, ingestion of certain foodstuffs, menstrual period, exposure to bright light or loud sound, and meteorologic changes (Blanchard, Andrasik, & Arena, 1984; Kunkel, 1979).

In migraine, dietary factors are often implicated. Many migraineurs claim that specific foods can trigger vascular headaches. There is some evidence that vascular head pain may follow the missing of a meal (Dexter, Roberts, & Byer, 1978), or the ingestion of foods containing tyramine (e.g., chocolate, cheese, red wine). Tyramine is believed to provoke a generalized vasoconstriction, probably through

stimulation of norepinephrine release (Dalton, 1975; Ghose, Coppen, & Carroll, 1978; Medina & Diamond, 1978; Ryan, 1974). Knowledge of these mechanisms is incomplete, however, and the role of tyramine is itself in question. The few controlled studies reported have not been generally supportive of the tyramine hypothesis (Moffett, Swash, & Scott, 1974; Shaw, Johnson, & Keogh, 1978).

Korczyn, Carel, and Pereg (1980) examined the relationship between head pain and a variety of potential physical precipitants -- anemia, elevated blood pressure, disturbances of visual acuity and intraocular pressure -- among headache-free and migraine, tension, combined, and undiagnosed headache subjects. No associations were found between head pain and any of these stimuli. Similarly, Drummond (1985) studied patient perceptions of headache "triggers" in migraine, tension, and combined headache. Migraineurs reported a greater number and variety of triggering stimuli (e.g., light glare, alcohol ingestion, menstrual cycle) than did other patients, and a greater range of variables that aggravated ongoing headache (e.g., head movements).

Summary Comments on Chapter 1

In assessing the literature on headache description and classification, several issues emerge. From the perspectives of both research and clinical practice, the current system

of classification (Ad Hoc Committee, 1962) seems unsatisfactory. Derived originally from clinical observations and impressions, it has garnered little empirical support since its introduction. Most problematic is the widespread observation of symptom heterogeneity among what were formerly thought of as distinct headache syndromes, particularly those of idiopathic etiology.

There are at present no specific or clinically feasible diagnostic tests by which to classify idiopathic headache, and the task of differential diagnosis is subject to errors of self-report and retrospection. Researchers and clinicians have disagreed on interpretation and operational definition of diagnostic criteria such as "tension". These problems, in turn, suggest a number of critical areas for further study: the reliability of primary and differential diagnostic criteria; the comparative psychophysiologies of the major idiopathic variants; and the development and evaluation of specific diagnostic tests.

Despite the difficulties inherent in the use of retrospective symptom reports, and the relative infrequency of "pure" headache syndromes in clinical practice, preliminary evidence does suggest a fairly reasonable degree of interdisciplinary concordance on diagnosis when clear inclusion-exclusion criteria are employed. Nevertheless, the perspective of migraine and tension headache as discrete diagnostic entities now seems untenable. In response, some

investigators have proposed the severity-continuum perspective as an alternative to typological classification (Bakal, 1975, 1980, 1982; Featherstone, 1985).

According to Lechin and van der Dijs (1980), "The great quantity of physical and biochemical disorders reported in headache patients correspond to phenomena which are fragments of a whole process. Consequently, whatever hypothesis is formulated regarding the physiopathology of (migraine), it should explain satisfactorily the greatest number of these phenomena in order to win minimum acceptability" (p. 81-82). Review of the literature to date suggests that satisfactory etiological models of idiopathic headache have yet to be advanced.

2. ISSUES IN PSYCHOLOGICAL RESEARCH ON HEADACHE

Conceptually and operationally, the psychological literature concerned with headache can be classified as follows: (a) early investigations of psychosomatic factors (e.g., Wolff, cited in Dalessio, 1972); (b) studies of personality and affective variables in headache (e.g., Arena, Andrasik, & Blanchard, 1985; Kudrow & Sutkus, 1979; Martin, 1972); (c) psychophysiological investigations (e.g., Bakal & Kaganov, 1977; Cohen, 1978; Epstein & Abel, 1977; Martin & Mathews, 1978); and (d) clinical trials of behaviourally-based interventions (e.g., Budzynski, Stoyva, Adler, & Mullaney, 1973; Chesney & Shelton, 1976; Cox, Freundlich, & Meyer, 1975; Haynes, Griffin, Mooney, & Parise, 1975; Holroyd, Andrasik, & Westbrook, 1977). Martin (1983) reviewed 136 psychological studies of headache published between 1972 and 1982. The majority (75%) concerned treatment; fewer focused on etiology (15%) or classification (10%).

Generally speaking, psychological studies of idiopathic headache have adopted and maintained the prevailing medical view that migraine and tension headache are distinct disorders with different etiologies. This chapter examines basic issues in the first two categories of headache investigation identified above, with regard specifically to

inclusion-exclusion criteria, subject selection and assignment, and headache assessment. The reader is referred to the sources cited for more detailed reviews of the psychophysiological and intervention literatures.

General Issues in Headache Research

Defining inclusion-exclusion criteria. Chapter 1 examined issues of headache classification and diagnosis, and identified a need for greater clarification. Not surprisingly, the application of diagnostic schema in headache research has proven problematic, in particular the derivation and specification of inclusion-exclusion criteria (Olton & Noonberg, 1980). Two principal trends have been evident: (a) reliance on the independent diagnoses of qualified medical personnel; and (b) derivation of criteria from specified clusters of headache symptoms (e.g., Ekbom, Ahlboorg, & Schele, 1978; Kroner, 1983; Price & Tursky, 1976). Neither strategy has facilitated the establishment of homogeneous subject groupings, in that both are based on criteria that have not been empirically validated (Olton & Noonberg, 1980).

The diagnostic scheme proposed by the Ad Hoc Committee (1962) was designed in part to stimulate treatment outcome research, by permitting comparisons among different headache populations. Its criteria, however, lack the degree of specificity required for adequate evaluation of validity and

reliability . The problem is compounded by the fact that so few studies have correctly or adequately applied the criteria as specified. Thompson (1982) reviewed a total of 50 headache treatment studies published between 1971 and 1979. Of these 56% failed to specify inclusion criteria, 24% provided only vague criteria, and only 20% reported specific criteria. In the last of these groups, there was considerable variation in the criteria employed. Within the total group, 20% employed specific medical tests. Of these, 22% reported using neurological examinations (the actual tests performed were reported in fewer than half of the studies); 6% reported other specific medical procedures.

Failure to correctly apply diagnostic criteria has greatly hindered comparisons among different studies and populations. It is possible that the samples employed were relatively heterogeneous. Virtually all of the studies reviewed by Thompson (1982) classified headache cases as either migraine or muscle-contraction; few were classified as combined or psychogenic.

To the extent that migraine represents a discrete disorder characterized by a distinct symptom profile, it should not often be confused with other idiopathic variants. Few studies (e.g., Feuerstein & Adams, 1977; Sturgis, Tollison, & Adams, 1978) have actually differentiated migraine subtypes, or addressed the possibility that treatment outcome might differ among these.

Tension headache is unquestionably the most frequently studied idiopathic variant. Despite the common assumption of an underlying pathology involving sustained contraction, only one study (Epstein & Abel, 1977) out of 50 reviewed by Thompson (1982) employed psychophysiological measures in defining headache groups. Overall, little attention has been devoted to the physiological basis of tension headache complaint, and few studies (e.g., Philips, 1977) have examined combined headache subjects.

Subject selection and control procedures. The combined headache category has proven particularly problematic in treatment outcome research, as it has not been possible to ascertain whether observed change has been due to alterations in either or both the migrainous and tension components. As of Thompson's (1982) review, only a small number of single case studies had reported monitoring changes in headache activity by headache type (Feuerstein & Adams, 1977; Sturgis, Tollison, & Adams, 1978). Headache intervention has become a particularly active area of research in recent years, with several reviews appearing in the behavioural literature in particular (Adams, Feuerstein, & Fowler, 1980; Beatty & Haynes, 1979; Blanchard, Ahles, & Shaw, 1979; Martin, 1983; Philips, 1978). Generally, these have reported somewhat conflicting findings in outcome by technique, and have attributed the confusion in part to methodological errors -- problems of subject selection,

classification, and assignment; procedural errors; inadequate or inappropriate pre- and posttreatment evaluation; and questionable data interpretation.

According to Friedman (1979), individuals who present clinically are not representative of the general headache population. Usually, they report more marked frequency, intensity, and duration of headache activity, and less relief of pain through self-medication. It is likely that subject selection in pain intervention research generally is confounded by the bias inherent in self-selected clinical samples (Crook, Rideout, & Browne, 1984; Merskey, 1980). There is clearly a need for more rigorous psychological characterization of the pain population examined; the further the subject base varies from a random sample, the more "abnormal" is any subject likely to appear (Merskey, 1982). In recent years, considerable evidence has amassed of abnormal illness behaviour among chronic patients presenting in pain clinics (Pilowsky & Spence, 1976).

Selection problems have confounded virtually all studies of personality characteristics of pain patients (Merskey, 1982). Representativeness has been a particularly critical concern. Several headache treatment studies have recruited subjects through media advertisements (e.g., Budzynski, Stoyva, Adler, & Mullaney, 1973; Chesney & Shelton, 1976; Holroyd, Andrasik, & Westbrook, 1977). In the absence of normative data, there are hazards in attempting to

generalize from recruited samples to the larger population of headache sufferers (Belar, 1979).

Blaszcynski (1984) argued the need to control for chronic pain per se as a determinant of personality, particularly in research aimed at demonstrating links between these variables. Sternbach and Timmermans (1975) reported the existence of "neurotic" traits secondary to chronic pain in a medical population. These were noted to subside with pain following successful surgery and rehabilitation. Woodeforde and Merskey (1972) similarly reported evidence of changes in personality secondary to chronic pain.

A second distinct issue in subject assignment involves the drawing of comparisons among headache types. Treatment studies have typically compared migraine and tension headache subjects to "headache-free" controls; seldom have simultaneous comparisons been made among individuals reporting different symptomatologies (Bakal, 1980).

Overall then, much of the psychological literature on headache to date has been confounded by critical methodological flaws, including problems of classification and assignment, particularly with regard to the application of diagnostic criteria; bias in subject selection; and failure to employ appropriate control groups or conditions. In light of these observations, the material examined in Chapter 3 of the present review should be interpreted with

caution. The investigation of personality and emotional variables in headache represents a tentative approach to the study of headache, one subject to potential pitfalls.

Methods of Headache Assessment

Measuring pain and its correlates. Problems of defining and assessing pain phenomena were alluded to briefly in Chapter 1. The first major issue in assessment concerns the lack of a generally accepted operational definition of the variable in question -- pain can be conceived as either or both a sensory and an affective experience. The former conceptualization effectively precludes any direct measure of pain, and although psychophysical scaling provides a quantitative method of evaluating sensory phenomena (e.g., Blanchard, Andrasik, Arena, & Teders, 1982; Gescheider, 1976), the investigator is more or less reliant on self-report, or indirect physiological or behavioural measures. The latter conceptualization offers potentially more promise for researchers concerned with the behavioural dimensions of the pain experience. In any event, pain cannot be reduced to a unidimensional event, and quantification is therefore often problematic.

It is precisely because of its inherently subjective nature that pain is difficult to evaluate consistently. The individual's perception of the pain experience is subject to several influences: early learning experience; personality

factors; associated fear or anxiety; and degree of attention focused on the experience. The evaluation of pain measures can be problematic because, in the final analysis, only the individual can accurately assess the quality and intensity of his or her pain experience (Huskisson, 1974). Some writers (e.g., Hendler, 1981) have gone so far as to conclude that pain is essentially unmeasurable. Though this position is somewhat extreme, many authors have voiced serious reservations about existing pain measures (e.g., Sternbach, 1976; Tursky & Jamner, 1982; Wolff, 1980).

Beecher (1959) defined pain as a multidimensional experience comprised of the interaction between: (a) a sensory component, defined operationally through measurement parameters (quality, intensity, location); and (b) a reactive component, involving affective responses to nociceptive stimulation, fears about its significance, and concerns about coping potential. Historically, pain researchers have emphasized the former component, particularly the parameter of intensity (Melzack, 1980). Consistent with the multidimensional perspective, Richardson, McGrath, Cunningham, and Humphreys (1983) proposed that pain be conceptualized in terms of distinct cognitive, physiological, and behavioural response modes. While the first of these is necessarily subjective and difficult to access, behavioural responses, though reflective only indirectly of the pain experience (Melzack,

1980), can be assessed in relatively objective ways (e.g., medication rate, frequency and duration of pain complaint).

From a different perspective, pain can be conceived as a predominately psychological event, subject to personality and emotional influences (Tursky & Jamner, 1982). Assessment may involve deriving individual pain profiles. While this approach does little to clarify pain severity, it may suggest individual differences in tolerance and coping capacity. Fordyce (1978) noted that, because the experience of pain is usually accompanied by distinct observable behaviours -- assuming intact sensory and processing apparatus -- it can be defined operationally in terms of these behaviours. This perspective implies objective description and measurement, but the behaviours in question must be regarded as correlates or concomitants of pain, and do not define the individual's pain experience per se.

The assessment process in headache research. Assessment plays a critical role in headache research generally (Thompson & Figueroa, 1983). For example, psychophysiological assessment can, in some cases, aid in determining the mechanisms underlying head pain (Andrasik, Blanchard, Arena, Saunders, & Barron, 1982; Raczynski, Thompson, & Sturgis, 1982), the effectiveness of ongoing treatment (Haber, Thompson, Raczynski, & Sikora, 1983; Thompson, Raczynski, Haber, & Sturgis, 1983), or the process of appropriate subject selection (Raczynski, Thompson, & Sturgis, 1982; Thompson, 1982).

Despite recognition that classification is problematic for headache research generally, only recently have issues of diagnosis been seriously considered (Raczynski, Thompson, & Sturgis, 1982). As is noted above, several studies have failed to specify or correctly apply inclusion-exclusion criteria. In reviewing the assessment literature, Thompson and Figueroa (1983) noted only one treatment study (Epstein & Abel, 1977) in which EMG measures were employed as inclusion-exclusion criteria for determination of tension headache. Few studies have specified either migraine subtypes or the criteria by which migraineurs were differentiated from tension headache cases. These findings suggest the need for more rigorous pretreatment evaluation of headache activity. To the extent that the success of any treatment is ultimately dependent on the correct identification of the target disorder and its parameters, accurate diagnosis must precede intervention (Friedman, 1979; Lake, 1981; Thompson, 1982).

According to Thompson and Figueroa (1983), a comprehensive evaluation of headache activity should incorporate features of the patient's head pain complaint, pain-related behaviours, and psychophysiological correlates of head pain. To this end, a variety of headache assessment instruments has been devised or adapted, including structured interviews, headache history questionnaires, symptom checklists, self-monitoring methods, functional analyses of headache activity, and psychological tests.

Lake (1981) conceptualized headache assessment as a bi-level process. The most elementary (topographic) level involves description of headache through the response modes of observable behaviour, physiological activity, and cognitive activity. Affect comprises an interaction of physiological and cognitive activities. The second level of assessment involves a functional analysis of antecedents and consequences of headache related behaviours. Antecedents may be generated internally (affects, cognitions) or externally (life stresses). Consequences comprise reinforcements, either positive (e.g., secondary gain for symptom expression in the form of attention or sympathy), or negative (e.g., continued self-medication following previous successful treatments). The implication is that head pain complaints often appear similar at the topographic level, but involve different antecedents or consequences. In most psychological studies of headache, antecedents have been examined under the rubric of personality or affect; consequences have been largely ignored or overlooked (Norton & Nielson, 1977).

The medical diagnostic process in headache was examined in Chapter 1 of the present review. Thompson (1982) outlined an analogous process of assessment that extends beyond the medical, topographic approach. Assessment begins with a general physical or neurological examination, to rule out head pain secondary to organic pathology. The second step comprises a clinical interview, through which headache

history and other pertinent data are obtained, and verbal symptom profiles are evaluated against diagnostic criteria. Psychological factors may also be assessed briefly during the interview, and preliminary diagnostic hypotheses drawn. Typically, medical diagnosis concludes at this point. The third step in the process, one critical to individualized treatment, involves patient self-monitoring of headache activity -- symptomatology, intensity, frequency, duration, pain quality, situational triggers or aggravants -- over an extended period of time. The data obtained can then be used to confirm or rule out initial diagnoses. These procedures have been described in detail by Collins and Thompson (1979).

In addition to self-monitoring, the assessment may incorporate the observations of the clinician or of individuals close to the patient. These can provide valuable information about antecedents of headache and pain-related behaviour. In addition, psychophysiological measures may aid in differentiating headache types, and in evaluating treatment response at a later time. Again, the utility of these procedures is limited, and appropriate only in certain cases.

Assessment in headache research has most often involved reliance on indirect measures of headache activity, such as self-medication rate (Philips, 1978). In some cases, headache frequency, intensity, and duration have been

combined in a single index, without concern that these parameters might be differentially responsive to treatment. In the absence of specific tests for different headache types, some researchers have derived idiosyncratic diagnostic criteria (e.g., Newland, Illis, Robinson, Batchelor, & Waters, 1978), which in turn has complicated the task of comparing findings across different studies.

The headache history. Some form of headache history has become a more or less routine component of the initial patient interview. Because the method relies on retrospection, it is subject to errors of memory (Cohen & McArthur, 1981). Chronic headache patients usually seek treatment only when their episodes are most severe, increasing the likelihood of somewhat exaggerated pain reports. Typically, the history comprises a series of questions, administered verbally or by questionnaire, concerned with various aspects of the headache experience (Ryan & Ryan, 1978; Thompson & Collins, 1979).

Headache questionnaires and checklists. By far, the most frequently employed non-medical assessment tool in headache research is some form of headache symptom questionnaire or checklist (e.g., Bakal & Kaganov, 1977; Kaganov, Bakal, & Dunn, 1981; Thompson & Collins, 1979; Thompson & Figueroa, 1983; Thompson, Haber, Figueroa, & Adams, 1980). Somewhat surprising, then, is the paucity of validity and reliability data bearing on these instruments generally. Most are

devised for use in single investigations, and are not subjected to standardization procedures.

Typical questionnaire items (e.g., Ziegler, Kodanaz, & Hassanein, 1985) focus on key aspects of the headache experience, including frequency (On average, how often have you had these headaches in the past year?); duration (On average, how long do these headaches usually last?); location (Do you have pain on only one side of your head during the headache?); associated symptoms and prodromata (Do you have vomiting during the headache? Do you have spots, lines, "heat waves" before your eyes before headache?); and perceived precipitants (Does emotional stress bring on your headache?). A variety of response formats is used, most often some form of verbal or numerical rating scale (e.g., Thompson & Collins, 1979).

The Headache Assessment Questionnaire (HAQ; Bakal, 1982), devised to aid in identifying psychological antecedents and correlates of headache activity, consists of 48 statements about thoughts and feelings experienced at headache onset. Most of these express distressing cognitions or negative affects that serve to exacerbate or maintain headache. In a recent psychometric study, Penzien, Holroyd, Holm, and Hursey (1985) found the HAQ useful for discriminating among head pain complaints of varying frequency (high versus moderate). Factor analysis suggested six dimensions: nonproductive rumination; self-denigration; irritation over

recent events; tension and worry; and two factors that were not readily interpretable. There were strong associations among these dimensions and other measures of headache activity, suggesting that more severe and frequent headache is generally accompanied by negative thoughts and feelings at onset. There was also evidence that headache sufferers experience these more when headache is attributed to exogenous factors, and when they perceive themselves as unsuccessful in preventing headache. Overall, the findings were supportive of Bakal's (1975) reports of increments in negative cognition and affect with increasing headache severity.

The Melzack Pain Questionnaire (MPQ; Melzack, 1975) has been shown to be useful, both in assigning patients to diagnostic groups without access to pain location data (Dubuisson & Melzack, 1976), and in discriminating between responses to clinical versus experimental (induced) pain (Crockett, Prkachin, & Craig, 1977). The scale comprises three classes of qualitative terms used to rate the subjective experience of pain: sensory (temporal, spatial); affective (tension, fear, autonomic symptoms); and evaluative (overall perceived intensity). Using the MPQ in conjunction with a headache questionnaire and symptom data, Allen and Weinmann (1982) reported that migraineurs described head pain in more intense terms than did tension headache subjects, though the differences were not

significant. Pain descriptions alone yielded accurate diagnoses: migraineurs tended to describe head pain in vascular terms, while tension headache subjects described pain in muscle-contraction terms.

Headache questionnaires have been employed in several studies of head pain complaint, in both clinical (Ziegler, 1979; Ziegler, Kodanaz, & Hassanein, 1985) and non-clinical (Friedman, 1979; Ziegler, Hassanein, & Couch, 1977) populations. Overall, the findings of these studies have suggested the following: Consistent with epidemiological studies (e.g., Bille, 1962; Waters, 1974), most populations examined have evidenced a high prevalence of at least occasional headache. Headache-prone individuals tend to report episodes of both mild and intense severity at different times. Severe headache is more often reported by women than men, while reports of mild headache only are more common among men. Onset of headache activity typically occurs within the first two or three decades of life. Psychological factors are often implicated in severe tension headache, particularly when emotional stress is perceived as a precipitant.

Epidemiological and normative headache data are usually obtained through large scale surveys. It has been suggested (Waters, 1978) that discrepancies among prevalence estimates reflect variations in the questionnaires used. Despite a clear need, there have been few validity and reliability

studies of these measures. It is therefore not clear whether they are capable, for example, of yielding comparable severity estimates over repeated administrations. There appears to be little correspondence between questionnaire estimates of severity and those obtained through daily headache ratings (Holroyd, Andrasik, & Westbrook, 1977).

In a study of headache in a college student sample, Thompson and Collins (1979) reported that those assigned to a "problem headache" group were less consistent over time in their reports of head pain severity, associated symptomatology, and attribution of headache to tension or stress. Test-retest reliability was not influenced by self-monitoring of headache activity during a 3-month period between administrations.

Andrasik and Holroyd (1980) examined the test-retest reliability and concurrent validity of a brief headache questionnaire administered to both a clinic and a nonclinic sample. Consistent with earlier findings (Thompson & Collins, 1979), questionnaire reports of headache symptomatology displayed reasonable test-retest reliability over a 2-week period. Nonclinic subjects, however, reported greater headache intensity, frequency, and duration at re-administration. Little correspondence was noted between questionnaire reports of headache and daily headache recordings; the latter provided the more valid and reliable measure. There is evidence that daily recordings are less

subject to errors of memory (Ciminero, Nelson, & Lipinsky, 1977), suggesting that retrospective items may not yield the most reliable estimates of headache activity.

Headache questionnaires are subject to other sources of error. In some cases, forced-choice response formats are employed. Generally, correspondence between forced and multiple-choice formats is low (Thompson & Figueroa, 1980, 1983); permitting a wider response range may increase the sensitivity and utility of a measure. Many headache questionnaires have failed to incorporate the assessment of temporal features (e.g., pre-headache, concurrent, and post-headache phenomena) without resorting to memory. Retrospection may be problematic for those episodes in which pain location shifts during the course of the headache.

The extant view of migraine and tension headache as discrete disorders has greatly influenced questionnaire development (Bakal & Kaganov, 1979). Individual items or descriptors are most often restricted to symptoms diagnostic of headache as defined medically (Ad Hoc Committee, 1962). Some questionnaires incorporate only a limited number of non-vascular descriptors, with principal emphasis on migraine (e.g., Deubner, 1977; Ekbom, Ahlboorg, & Schele, 1978; Ziegler, Hassanein, & Hassanein, 1972). Measures so designed largely overlook the occurrence of tension-like symptoms in individual headache profiles.

Self-monitoring of headache activity. Assessment of pain parameters -- intensity, frequency, duration -- usually comprises some form of self-rating measure administered in the form of a "headache diary". Most often, this consists of a verbal-numerical scale on which the descriptive phases of headache are assigned numerical values. The diary technique was first proposed by Budzynski, Stoyva, Adler, and Mullaney (1973), who had subjects record bi-hourly ratings of head pain intensity on a 6-point numerical scale (0: "no headache activity"; 6: "extremely intense and incapacitating headache"). Subsequent investigations (e.g., Epstein & Abel, 1977) employed fewer daily ratings, in part to minimize retrospective responding.

Cohen and McArthur (1981) had migraine and tension headache patients self-monitor headache activity daily over 16 consecutive months, comprising pre- and post-treatment phases of biofeedback training. Activities monitored included perceived headache type, intensity, duration, degree of incapacitation, associated symptomatology, self-medication, and perceived effectiveness of treatment. Regardless of initial diagnosis, all patients reported occurrences of both vascular and tension headache; the former were given higher ratings on intensity, duration, incapacitation, and medication frequency. The findings were taken as evidence of discrete headache types. Similar results were obtained by Blanchard, Andrasik, Arena et al.

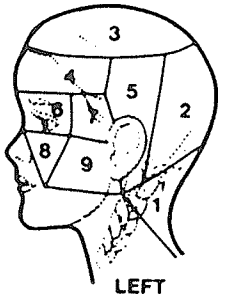
(1984). Vascular episodes were associated with a higher frequency of headache-free days per week, while tension neadache episodes were rated higher on daily average intensity.

Bakal and Kaganov (1976) devised a headache diary instrument with explicit emphasis on pain location. The Headache Frequency Record (HFR; see Figure 1) incorporates a 6-point intensity scale, diagrammatic specification of 19 possible head pain locations, and a symptom list comprising vascular (throbbing or pulsing pain, nausea, vomiting, visual disturbances, light sensitivity) and tension headache (dull and aching pain, sensations of tightness and pressure) features. Subjects also record self-medication. Using HFR data obtained from migraine and tension headache subjects, Bakal and Kaganov (1977) found that all were equally familiar with symptoms and head pain locations usually regarded as specific to one or the other disorder.

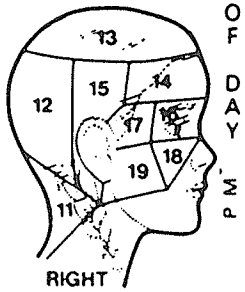
Headache diaries possess the advantages of relative ease of use and of yielding reasonably accurate profiles of daily fluctuations in headache activity. Overall, they appear to display acceptable sensitivity and reliability (Collins & Martin, 1980; Collins & Thompson, 1979). To their detriment, headache diaries tend to highlight the sensory qualities of head pain at the expense of the reactive-affective dimension (Andrasik, Blanchard, Ahles, Pallmeyer, & Barron, 1981).

Figure 1. Headache Frequency Record (HFR; Bakal & Kaganov, 1976).

Code _____ Date _____



LEFT



RIGHT

TIME OF DAY

INTENSITY

AM/PM	0	1	2	3	4	5	MEDICATION	NOTES	SYMPTOM LIST
12									I am experiencing:
6									throbbing or pulsating pain ( )
8									dull and aching pain ( )
10									feelings of tightness and pressure ( )
12									nausea ( )
2									vomiting ( )
4									visual disturbances ( )
6									light sensitivity ( )
8									
10									
12									

HEADACHE FREQUENCY RECORD

Few studies have examined the issue of compliance with headache self-monitoring procedures generally (Thompson & Figueroa, 1983), nor have any focused on compliance as it pertains to the dimensions of symptomatology, intensity, duration, and medication use. There is some evidence that the sensitivity of these procedures is enhanced when subjects are knowingly monitored by an external observer (Lipinsky, Black, Nelson, & Ciminero, 1975; Nelson, Lipinsky, & Black, 1975).

It is often noted that headache patients, particularly migraineurs, describe past episodes with little precision; the experience of pain is not well consolidated in memory (Oleson, 1978). Self-report of pain is also complicated by the limitations inherent in our pain vocabulary. Despite variations in symptomatology across different head pain complaints, headache sufferers tend to describe their episodes in similar terms. It may be that certain pain descriptors, such as "severe" or "unbearable", hold different meanings for different individuals and across different symptom profiles or severities (Blanchard, Andrasik, Arena, & Teders, 1982). It is therefore important to supplement the headache diary with some measure of the reactive-affective dimension of head pain.

Verbal rating and scaling methods. Tursky (1976) devised a technique for concurrent assessment of both the sensory and reactive components of pain, comprised of descriptive

adjective lists. Subjects reported the relative meanings of pain-related terms as they corresponded to their own experiences. Enmore and Tursky (1981) reported population scale values for the descriptors, and demonstrated their utility in assessing biofeedback treatment effects among migraineurs.

Andrasik, Blanchard, Ahles, Pallmeyer, and Barron (1981) correlated descriptive adjective data with numerical headache diary ratings derived from migraine, tension, and combined headache patients. All recorded four intensity ratings per day throughout 1-month baseline, 6-week treatment, and 2-week follow-up phases. At the end of each day, adjectives were chosen from "intensity" and "reactive" lists that best described head pain experienced during the day. Higher correlations were obtained among indexes of severity from the diary and adjectives lists than were obtained between severity and the reactive adjectives, suggesting dissociation between pain intensity and affective reaction to pain. The findings were consistent with the clinical observation that patients who present with severe head pain complaint don't always show visible signs of distress.

Blanchard, Andrasik, Arena, and Teders (1982) utilized Tursky's (1976) adjective lists to examine response to pain among migraine, tension, combined, and cluster headache patients, and headache-free subjects, and found that

different symptom profiles did not affect the perception of pain intensity. Those who reported the most intense vascular headaches consistently scaled the reactive adjectives in the direction of severe distress.

The visual analogue scale (VAS) is a direct scaling technique which, when applied to the measurement of head pain, requires the subject to rate intensity by marking a point along a straight vertical or horizontal line representative of a pain continuum (Harvey & Hay, 1984; Ohnhaus & Adler, 1975; Scott & Huskisson, 1976). Although clear and simple to administer, the VAS is often associated with a tendency to interpret responses as though the scale had true interval or ratio properties, with each scale unit corresponding to an equal psychological unit of pain. Further, the VAS has most often been employed as a measure of pain conceived as a unidimensional sensory phenomenon, varying only in perceived intensity (Ohnhaus & Adler, 1975).

Melzack and Torgerson (1971) were among the first to devise multidimensional pain descriptor scales. They subjectively grouped clinical pain descriptors along different dimensions -- sensory (spatial, temporal, qualitative), affective (tension, fear), and perceived intensity (mild, moderate, severe). To date, the only notable application of this approach has been in the development of the MPQ (Melzack, 1975). Within the context of a multimodal assessment, however, it should be possible

to evaluate the sensory, affective, and perceived severity dimensions of head pain.

Assessment of antecedents and consequences of headache. While little attention has been focused on the assessment of headache-related behaviours (Thompson & Figueroa, 1983), some authors have advocated the evaluation of headache from a functional perspective (Lake, 1981; Norton & Nielson, 1977). To date, there has been no systematic investigation of the antecedents and consequences of headache-related behaviours. While certain events or stimuli may be seen to precipitate or aggravate headache, the actual sequence of events appears exceedingly complex and interactive. It is not known whether headache is more susceptible to immediate or remote influences (e.g., ingestion of certain foodstuffs versus the cumulative effects of psychosocial stress). Patient reports suggest a wide array of stimuli and behaviours that impact on headache complaint.

In many cases, recurrent head pain appears to be maintained by the consequences of its expression, when headache behaviours -- complaint of pain, bodily expression of suffering, self-medication -- are reinforced by environmental circumstances. Intervention should incorporate some attempt to manipulate these consequences (Yen & McIntyre, 1971). Budzynski, Stoyva, Adler, and Mullaney (1973) found that patients who reported some form of secondary gain for pain complaint were less responsive to biofeedback treatment.

Psychophysiological assessment of headache activity. The overall findings of psychophysiological studies of headache were examined in Chapter 1. Generally, psychophysiological measures have proven only moderately useful in either diagnosis or determination of etiology. In the context of individualized headache assessment, however, they may be useful in evaluating patient suitability for, or response to, behaviourally-based treatments, such as progressive relaxation or biofeedback.

According to Thompson and Figueroa (1983) research on the psychophysiological assessment of headache has been subject to several methodological problems. Raczynski and Thompson (1982) reviewed general assessment practices in several studies and found that the majority had failed to account for relevant subject variables, such as age, sex, inclusion-exclusion criteria, neurological or other medical findings, and self-medication practices. Similarly, most had failed to consider important procedural issues, such as the rationale for selection of measures, measurement sites, or adequate baseline periods. Finally, certain quantification problems were evident, including errors in artifact detection, and the measurement of physiological activities over time and across conditions.

Standardized Psychological Tests in Headache Research

In addition to headache questionnaires and other specialized assessment instruments, a variety of standardized psychological tests, mainly objective personality inventories, has been employed in headache research. Generally, these studies have focused on the exploration of etiological or diagnostic hypotheses, rather than on headache assessment per se. To date, the most frequently employed tests include the Minnesota Multiphasic Personality Inventory (MMPI; Hathaway & McKinley, 1943), the Beck Depression Inventory (BDI; Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961), and the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970; Spielberger, Gorsuch, Lushene, & Vagg, 1977). The findings of recent psychometric studies are discussed in Chapter 3 of the present review. This section briefly examines the descriptive aspects of various tests, and issues pertinent to their use in headache research.

The MMPI (Hathaway & McKinley, 1943) is the most frequently reported test in psychometric headache research. Because of its status as the most studied objective test of personality -- the annual rate of publication internationally currently exceeds 200 reports (Butcher & Finn, 1983) -- data on its development, standardization, composition, interpretation, and application are available in several sources (e.g., Dahlstrom, Welsh, & Dahlstrom,

1972; Golden, 1979; Graham, 1987), and are discussed here only briefly. Empirically-derived and consisting of 566 true-false statements, the MMPI yields scores on four validity and 10 basic clinical scales, which are in turn converted to standard or "T-scores" (M 50; SD 10). T-scores higher than 70 are regarded as clinically significant.

The rationale underlying the use of the MMPI in headache research has derived from theoretical formulations examined in Chapter 3 of this review. These have postulated the existence of a unique "headache profile", most often involving traits of obsessive-compulsiveness, rigidity, hostility, and depressed affect. As such, research to date has focused on clinical scales measuring Hypochondriasis (1), Depression (2), Hysteria (3), and Psychasthenia (7). The depression (MMPI-D) scale, for example, contains 60 items that assess symptomatic depression -- poor morale, pessimism about the future, general dissatisfaction with life, psychomotor retardation, physical complaints, worry, tension, and denial of impulses. While highly elevated scores (> 80) are suggestive of clinical depression, more moderate elevations suggest a general attitude or lifestyle characterized by pessimism and poor morale (Graham, 1987).

The MMPI has demonstrated its advantages as an assessment device in the study of headache and other physical complaints. In addition to its extensive psychiatric applications, normative data are available on large

outpatient medical populations (Swenson, Pearson, & Osborne, 1973).

The BDI (Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961) is a clinically derived 21-item multiple-choice questionnaire assessing complaints, symptoms, and attitudes related to ongoing depression. Each item consists of four self-evaluative statements numbered from 0 to 3, with higher scores indicating greater severity. The BDI yields total scores ranging from 0 to 63, associated with four "levels" of depression: normal, non-depressed (0-9); mild (10-15); moderate (16-23); and severe (24-63). Specific scales include sadness, pessimism, sense of failure, dissatisfaction, feelings of guilt, suicidal wishes, irritability, withdrawal, and indecisiveness. The BDI has been shown to fully sample six of nine depression criteria specified in DSM-III-R (American Psychiatric Association, 1986), and partially assess an additional two (Moran & Lambert, 1983).

Recent meta-analyses (Edwards, Lambert, Moran, McCully, Smith, & Ellingson, 1984; Lambert, Hatch, Kingston, & Edwards, 1986; Moran & Lambert, 1983) suggest that the BDI compares well with alternative depression scales, and provides a particularly sensitive measure of treatment response. The BDI has been used to examine a purported association between headache activity and depressed affect. Both the MMPI-D and BDI have been criticized, however, for

inclusion of somatic and vegetative signs equally characteristic of the non-depressed medically ill and the elderly (Turk, Rudy, & Stieg, 1987). Classification into depressed and non-depressed groups is often problematic, particularly among young and college-aged subjects (Deardorff & Funabiki, 1985; Hatzenbeuhler, Parpat, & Matthew, 1983; Zimmerman, 1986). Further, BDI data are unstable in the mild to moderate ranges of depression, and should be supplemented by other criteria . A comprehensive critique of these measures is beyond the scope of this review, however; the reader is referred to the sources cited above for further information.

The Depression Adjective Check List (DACL; Lubin, 1965, 1967; Lubin & Levitt, 1979) consists of two sets of descriptive word lists. The first set comprises four equivalent lists of 32 non-overlapping adjectives; the second, three similar lists of 34 adjectives. Unlike the MMPI-D and BDI, the DACL is designed to measure mainly transient (state) depressed mood rather than chronic enduring (trait) depression. The availability of brief equivalent forms has made the measure useful in repeated measurement designs, particularly those involving day-to-day fluctuations in mood level (Lewinsohn & Lee, 1981).

The State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970; Spielberger, Gorsuch, Lushene, & Vagg, 1977) is the most carefully developed and rigorously

examined measure of anxiety, viewed as both an exogenous state and an endogenous trait (Levitt, 1980). Comprised of state (Y1) and trait (Y2) forms, the inventory consists of 20 statements of thoughts and feelings, to which the subject responds on a 4-point scale. The STAI has been used to explore an hypothesized relationship between headache activity and anxiety (e.g., Andrasik & Holroyd, 1980; Henryk-Gutt & Rees, 1973).

Issues in psychometric headache research. Andrasik, Blanchard, Arena et al. (1982) proposed a set of criteria by which to evaluate psychometric studies of headache, with reference to: (a) specification of inclusion-exclusion criteria to ensure subject assignment to relatively homogeneous groups; (b) simultaneous investigation of 2 or more diagnostic types or levels of severity to determine, for example, the specificity of a proposed psychological construct; (c) use of appropriate control subjects or groups (e.g., non-headache or other chronic pain patients); and (d) use of multidimensional tests (e.g., MMPI) or test batteries. To date, few studies have met more than one of these criteria (Henryk-Gutt & Rees, 1973; Kudrow & Sutkus, 1979; Philips & Hunter, 1982; Sternbach, Dalessio, Kunzel, & Bowman, 1980), and none has satisfied all criteria. Several conceptual and methodological problems are evident in the literature generally. For the most part, studies to date have relied on subject retrospection, and have therefore

been subject to errors of memory (Gatchel, Deckel, Weinberg, & Smith, 1985). Also evident has been a failure to control for relevant subject characteristics, such as age, sex, and duration of headache complaint (chronicity).

There is a growing consensus that subject samples employed in psychometric studies may be biased in the direction of psychopathology, if only because of the experience of more frequent and severe head pain (Arena, Andrasik, & Blanchard, 1985). For example, headache patients seen clinically often show significant elevations on a number of MMPI scales as their symptom profiles more closely approximate that associated with tension headache as opposed to migraine (Sternbach, Dalessio, Kunzel, & Bowman, 1980). This finding may be more suggestive of the significance of "pain density" (Sternbach, 1974) than of differences among headache groups as typically derived. Further, those MMPI scales of particular interest -- Hypochondriasis (1), Depression (2), Hysteria (3), and Psychasthenia (7) -- are often elevated in patients with <u>any</u> chronic physical illness (Harrison, 1975; Merskey, Brown, Brown, Malhotra, Morrison, & Ripley, 1985; Naliboff, Cohen, & Yellen, 1982; Sternbach, 1974; Watson, 1982). As such, elevations on these scales among headache patients (e.g., Ajwani & Ajwani, 1984; Formisano, 1985; Roccatagliata, 1984) must be interpreted with caution.

For the most part, psychometric studies of headache are correlational in nature; firm etiological conclusions cannot be drawn from associations between test scale elevations and headache activity (Arena, Andrasik, & Blanchard, 1985; Blanchard, Andrasik, & Arena, 1984). The "direction-of-causality" issue is problematic; it cannot be ascertained whether headache is caused, maintained, or exacerbated by the kinds of variables accessed through psychological tests, or indeed whether these variables are altered by chronic head pain (e.g., Sternbach, 1974). Sternbach and Timmermans (1975), for example, noted that psychological test responses were often modified by pain interventions.

Arena, Andrasik, and Blanchard (1985) examined MMPI, BDI, and STAI profiles of chronic and non-chronic headache sufferers, assuming that higher scale elevations among chronic patients would suggest alterations in psychological functioning secondary to pain. Alternatively, failure to obtain group differences among subjects showing significant elevations would suggest the precedence of dysfunctional traits or predispositions. Chronicity had no discernable influence on any of the clinical scales. Intercorrelations among scales, age, and chronicity, however, suggested that certain personality traits, independent of head pain, had become more ingrained over time. Overall, the findings indicated that characterological traits, as opposed to

day-to-day fluctuations in affect, for example, play important etiological roles in chronic headache.

Summary Comments on Chapter 2

It would appear that, regardless of the orientation of headache research -- diagnostic, etiologic, psychometric -- there is a clear need for either closer adherence to existing classification guidelines (e.g., Ad Hoc Committee, 1962), or the development of valid and reliable alternatives to typological diagnosis. Reliance on external diagnosis without adequate validity checks, and the practice of deriving cut-off points (number of symptoms required to confirm diagnosis) are questionable strategies. This issue is of principal concern to those who maintain a typological perspective on headache.

Clearly, researchers cannot regard either self-selected clinic or media-recruited samples as representative of the general headache population. Equally critical, particularly where group comparisons are made among different headache "types", is the selection of appropriate control groups or conditions. Ideally, these should include both headache and headache-free subjects.

Review of the intervention literature suggests a need for more rigorous, preferably multimodal, pretreatment assessment. This should comprise a comprehensive headache history, headache questionnaires or symptom checklists, and

critically, daily and systematic self-monitoring of headache activity over an extended period, with provision for validity and compliance checks.

Research to date suggests poor correspondence between questionnaire and self-monitoring data, owing in part to limitations in the questionnaires typically employed (e.g., reliance on retrospection, forced-choice response formats, insensitivity to temporal qualities of head pain). The assessment of antecedents and consequences of headache would seem to hold particular promise for enhancing behavioural interventions directed at contingency-maintained features of headache, and for determining important maintaining, exacerbating, and relieving factors.

Standardized psychological tests have proven useful in clarifying associations among psychological variables and parameters of headache. The psychometric literature, however, evidences a number of methodological flaws, such as the failure to control relevant subject variables or incorporate appropriate control groups. Essentially, these are the same types of problems that have limited other areas of headache research. More critical perhaps is the observation that subject samples may, for reasons that are not fully understood, be biased in the direction of psychopathology. Problems of subject selection and assignment are greatly simplified in the severity-continuum perspective, which effectively precludes reliance on

clinical headache samples. Research can then focus, for example, on associations among test scale elevations and parameters of head pain in non-clinic headache populations.

Psychometric studies of headache have been predominately retrospective and correlational in approach. The problem of retrospection could be addressed, however, either through prospective designs or greater reliance on self-monitoring of headache. To date, no true prospective psychological studies of headache have been reported. The correlational nature of psychometric research is not necessarily a liability; thus far, the research has been largely exploratory and prototypical. From this perspective, our knowledge of psychological factors in headache may be further enhanced by well-designed correlational studies, even if causality cannot be ascertained. While procedures have been developed by which to manipulate psychological variables such as mood, these generally result in phasic alterations. Presumably, psychological factors that impact on headache complaint in the real world are long standing in nature. Certainly, patient-identified stressors are generally tonic rather than phasic. Finally, designs such as that reported by Arena, Andrasik, and Blanchard (1985), while precluding clear determination of causality, do suggest potential approaches that might allow for relatively naturalistic observation of headache.

3. PERSONALITY AND AFFECT IN HEADACHE

Over the years, a wide variety of psychological constructs has been implicated in the etiology and expression of headache -- anger, anxiety, conflict, dependency, depression, ego defenses, fear, guilt, hysteria, inability to expression emotion, obsessive-compulsiveness, and sexual dysfunction. One area of particularly intensive study is that concerned with specific personality variables (Blanchard, Andrasik, & Arena, 1984). The earliest such reports took the form of extended case descriptions and uncontrolled studies, predominately from the psychoanalytic perspective (e.g., Wolff, cited in Dalessio, 1972). More recent investigations have involved the use of standardized psychological tests.

Personality Variables in Headache

A recent review by Adler, Morrissey-Adler, and Friedman (1987) outlined historical developments in the conceptualization of headache generally, with particular reference to migraine. The term "migraine" is derived from the French "migrein" or "megrim", in reference to severe headache (Onions, Friedrichsen, & Burchfield, 1966), and was in turn derived from the late Latin "hemicrania" or "half-headed", reflecting the characteristic unilateral

distribution of head pain associated with the disorder. Migraine has been known and written about for at least 2000 years, and purely phenomenological accounts in early Greek and Roman writings, with their explicit emphasis on "sick" and "blinding" headache, are similar to contemporary clinical descriptions (Friedman, 1975). The informal beginnings of headache diagnosis have been accredited to Aretaeus of Cappadocia, who differentiated among "heterocrania" (probably migraine), "cephalgia" (other acute forms of headache), and "cephalea" (chronic headache).

Historically, the conceptualization of migraine has closely paralleled current thinking in medicine generally (Adler, Morrissey-Adler, & Friedman, 1987). Early Middle Eastern writings stressed that headache was caused by forces -- often spiritual -- outside of the individual. It is interesting to note that the word "pain" in fact derives from the Latin "poena", meaning penalty or punishment. The practice of medicine remained undifferentiated from religion for many centuries. Toward the latter 19th Century, a view of migraine arose in which headache was attributed to "faulty habits of life". This in turn gave rise over time to the concept of a constitution or personality unique to migraine. A stereotype of migraine as a "disease of the intelligent, conscientious professional classes" (Pearce, 1977, p. 126) gained popularity, but epidemiological studies (e.g., Waters, 1971) have long since discredited this view.

During the same era, the pioneering works of Gowers and others in the field of neurology stressed the purely biological basis of headache and other physical complaints.

Early psychosomatic investigations. The so-called "personality theory" of headache emerged as a variant of Alexander's (e.g., 1950) theory of emotional specificity, which postulated that specific emotional traits, in conjunction with a genetically-linked "organ weakness" -- a predisposing vulnerability in a specific organ system -- led to the development of specific psychosomatic disorders. Early psychosomatic investigations of headache were directed at determining psychological antecedents, rather than psychophysiological components, of chronic headache disorders, and sought to establish the significance of dysfunctional emotions and personality traits among migraineurs in particular.

Touraine and Draper (1934) described a constitutional personality type in migraine, comprised of: "some physical acromegaloid traits accompanied by retarded emotional development and superior intelligence ... migraine appears when these individuals lose the protection of home, particularly maternal dependency, which has helped them avoid facing the responsibilities of living alone" (Alexander & Flagg, 1965, p. 894). Wolff (cited in Dalessio, 1972), whose work was inspired by the pioneering

investigations of Alexander (1950), Dunbar (1935), Fromm-Reichmann (1937), and Knopff (1935), described the "migraine personality" as ambitious, perfectionistic, orderly, inflexible, and excessively reserved. Vascular episodes were said to be triggered by affective states marked by feelings of frustration, self-doubt, fear of criticism or failure, and resentment. Furmanski (1952) reviewed these and similar clinical observations, and described the typical migraineur as anxious, obsessive, and lacking in the ability to openly confront and express feelings of hostility and resentment.

In recent years, several investigators (Anderson, 1980; Bille, 1962, 1981; Menkes, 1974; Ryan & Ryan, 1978; Saper, 1983) have described the childhood migraineur as alert, ambitious, anxious, conscientious, courteous, delicate, dependent, depressed, docile, easily frustrated, fearful, inflexible, ingratiating, intelligent, nervous, obstinate, polite, reserved, sensitive, and shy. For the most part, these characterizations were derived from clinical impressions rather than systematic studies.

Specific personality variables. A variety of personality variables has been implicated in the chronic headache profile. While early psychosomatic investigations focused on putative intrapsychic conflicts involving the expression of anger or other negative affect, later studies examined specific personality variables.

There have been numerous clinical reports of a higher prevalence of "neurotic" traits among migraineurs (Henryk-Gutt & Rees, 1973) and tension headache patients (Andrasik, Blanchard, Arena et al., 1982; Andrasik & Holroyd, 1980; Philips, 1976). Psychometric studies have reported, for example, MMPI profiles marked by elevations on Scales 1 and 3, relative to 2 -- the so-called "neurotic triad" (Lanyon, 1968) -- in tension (Andrasik, Blanchard, Arena et al., 1972; Harper & Steger, 1978; Martin, 1972) and combined headache (Weeks, Baskin, Rapoport et al., 1983), or among chronic headache sufferers generally (Kudrow & Sutkus, 1979). Overall, however, headache patients have been found to measure only moderately higher on neuroticism than headache-free individuals; differences between normals and neurotics are typically more striking (Cuypers, Altenkirch, & Bunge, 1981). There is some evidence of greater somatic preoccupation on the MMPI among tension, combined, and psychogenic headache patients, relative to headache-free subjects (Haber, Kuczmierczyk, & Adams, 1985; Mathew, Stubits, & Nigam, 1982; Weeks, Baskin, Rapoport et al., 1983; Werder, Sargent, & Coyne, 1981).

Over time, the migraine personality came to be thought of as comprising traits of perfectionism, rigidity, and obsessive-compulsiveness (Wolff, cited in Dalessio, 1972). Recent findings do not support this formulation, however. Kudrow and Sutkus (1979) reported essentially normal MMPI

profiles among migraineurs. Blaszczynski (1984), employing the Hysteroid-Obsessoid Questionnaire (Caine & Hope, 1971), found no evidence of greater obsessionality among classic migraine and tension headache patients, relative to headache-free and other chronic pain groups.

The findings on anger and hostility have been less conclusive. Martin (1972) reported evidence of poorly-controlled anger and hostility in the MMPI protocols of tension headache patients. Henryk-Gutt and Rees (1973) compared classic and common migraine and undifferentiated non-vascular headache patients on responses to the Buss-Durkee Hostility-Guilt Inventory (Buss & Durkee, 1957). Classic migraine patients, and in fact all female patients undergoing treatment, displayed higher levels of hostility. Blaszczynski (1984) reported greater hostility among classic migraine and tension headache subjects relative to headache-free controls, on both the Buss-Durkee and the Direction of Hostility Questionnaire (Caine, Foulds, & Hope, 1967). Headache subjects, however, did not score significantly higher on either measure relative to non-headache chronic pain patients. Finally, Andrasik, Blanchard, Arena et al. (1982) found no differences between migraine and tension headache subjects in expressed dislike and distrust of others.

Despite numerous clinical descriptions of migraineurs as perfectionistic and success-oriented, there appear to be no

significant differences among headache types on measures of achievement motivation (Andrasik, Blanchard, Arena et al., 1982).

Problems in research on personality variables. Recent medical research has concentrated on the search for specific biological precursors of headache, with personality and emotional factors viewed as less significant (Pearce, 1977). Nevertheless, personality variables are still commonly regarded as central to the chronic headache profile, and their potential etiological significance remains a viable area of enquiry (Friedman, 1975). Several conceptual and methodological problems have been evident in the research conducted to date, however.

Harrison (1975) reviewed several psychometric studies of headache published prior to 1975; virtually none of these was controlled. All headache patients, regardless of diagnosis, were characterized as more anxious, hysterical, and vulnerable than headache-free individuals. As was noted in Chapter 2, it is difficult to ascertain, in the absence of controlled longitudinal research, whether such variables constitute precursors of headache, or the consequences of living with chronic pain (Arena, Andrasik, & Blanchard, 1985; Bakal, 1982). Apart from the issue of causality, specific procedural errors in these studies have been noted by several authors (e.g., Andrasik, Blanchard, Arena et al., 1982; Blanchard, Andrasik, & Arena, 1984; Martin, 1983;

Passchier, van der Helm-Hylkema, & Orlebeke, 1984; Weeks, Baskin, Sheftell et al., 1983; Williamson, 1981), including reliance on anecdotal evidence, failure to specify diagnostic groups, prohibitively small sample sizes, lack of control for either unbalanced age or sex distributions, and the use of idiosyncratic or non-standardized assessment instruments.

Affective Variables in Headache

Research on mood states. Contrary to Alexander's (1950) theory of psychosomatic specificity, there has been little agreement as to whether certain emotional states are more or less implicated than others in the development and maintenance of somatic complaints, or whether there are identifiable links between emotions and physical symptoms (Epstein & Kaplan, 1984). There is also little agreement on the pathogenic role of the suppression of affect in symptom formation.

Martin (1983) proposed two strategies for evaluating associations among mood states and headache activity: (a) subject self-monitoring and reporting of mood (e.g., Lubin, 1967; McNair, Lorr, & Droppleman, 1971) during headache and headache-free intervals; and (b) comparison of headache and non-headache controls on responses to laboratory manipulations of mood status (e.g., Velten, 1968). While there are numerous anecdotal reports of mood and headache

activity, little systematic research has been conducted consistent with either strategy.

Arena, Blanchard, and Andrasik (1984) reported an isomorphic relationship between mood and head pain among migraine, tension, and combined headache patients, such that increments in headache activity were associated with same-day mood shifts. The correlations were not significant, however. In an effort to determine whether mood states were predictive of headache, Harrigan, Kues, Ricks, and Smith (1984) had migraineurs monitor headache and mood over an extended period. Feelings of constraint and fatigue were consistent predictors of subsequent headache, with mood generally lowest during headache and throughout the preceding day. In a similar study, Harvey and Hay (1984) reported more depressed affect on the DACL (Lubin, 1967) on headache days, but more positive mood status -- reports of feeling more alert, attentive, energetic, and clear-headed -- during the previous day.

To date, little attention has been focused on situational affect and headache activity, or on the prospective investigation of mood change and headache (Arena, Blanchard, & Andrasik, 1984; Harvey & Hay, 1984). As such, it is not yet clear whether mood influences subsequent headache or vice-versa, or whether the two should properly be regarded as concomitants of a common process.

Sternbach (1974, 1976) reviewed the literature on personality variables in chronic pain patients, and concluded that, as pain status proceeds from acute to chronic, autonomic signs and symptoms of anxiety (elevated heart rate, blood pressure, and muscle tension) tend to habituate out, while vegetative signs of depression and hypochondriasis (disturbances of sleep, appetite, and sexual drive, irritability, somatic preoccupation) take precedence. It may be the case, then, that changes in mood status are caused by headache.

Headache and anxiety states. Many headache-prone individuals seen clinically present as anxious (Crisp, Kalucy, McGuiness, Ralph, & Harris, 1977; Gianotti, Cianchetti, & Taramelli, 1972; Martin, 1966; Martin, Rome, & Swenson, 1967; Packard, 1976, 1979; Ziegler, 1979; Ziegler, Rhodes, & Hassanein, 1978). To date, however, there have been no systematic studies of anxiety in chronic headache patients; anxiety has simply been assumed to be characteristic of the head pain profile, particularly among tension headache patients.

A few psychometric investigations have provided some clarification on the role of anxiety. There is evidence of higher levels of anxiety among tension headache patients relative to migraine and combined headache sufferers, on both the MMPI (Andrasik, Blanchard, Arena et al., 1982; Blanchard, Andrasik, Arena et al., 1984; Mathew, Stubits, &

Nigam, 1982) and the STAI (Blanchard, Andrasik, Arena et al., 1984). Ziegler, Kodanaz, and Hassanein (1985), however, found no association between tension headache activity and anxiety as assessed by the Bendig-Taylor Anxiety Scale (Bendig, 1956).

Extending Sternbach's (1974, 1976) observations to chronic headache, it may be that depressive features are more likely to dominate the picture in most headache cases seen clinically, and therefore, headache treatment samples.

Headache and depression. In the psychobiological model of headache examined in Chapter 1, depression, anxiety, and feelings of helplessness are seen as significant cognitive components of the chronic headache profile (Bakal, Demjen, & Kaganov, 1984). A recent review of psychological factors in headache (Blanchard, Andrasik, & Arena, 1984) listed a total of 37 studies and reports concerned with headache activity and depression, most of which comprised unsystematic clinical observations. The majority of these concluded that depression is far more prevalent among tension headache sufferers than among migraineurs. A comprehensive discussion of the phenomenon of depression is beyond the scope of this review; the reader is referred to several current sources (e.g., American Psychiatric Association, 1986; Beckham & Leber, 1985; Cancro, 1985; Taska & Sullivan, 1983). For the purposes of this discussion, however, some clarification of basic terms and issues might be useful.

Depression has been defined generally as, "A clinical syndrome consisting of lowering of mood-tone (feelings of painful dejection), difficulty in thinking, and psychomotor retardation ... (it) ordinarily refers only to the mood element, which in psychiatry would more appropriately be labeled dejection, sadness, gloominess, despair, despondency, etc." (Hinsie & Campbell, 1970, p. 200). The term is used in reference to a mood, symptom, or disease entity (Cancro, 1985), and several authors have specified a distinction between the subjective mood and somatic-vegetative aspects of depression (Pilowsky, Chapman, & Bonica, 1977; Garron & Leavitt, 1983; Maruta, Swanson, & Swenson, 1976). Not unlike pain, depression is a multidimensional construct (Shaw, Vallis, & McCabe, 1985), comprised of biological, psychological, and psychosocial components. It also represents a continuum of severity ranging from mild dysphoria associated with daily life stresses to a major clinical syndrome involving alterations in mood, vegetative functions, cognitive and psychomotor activities, and psychosocial functioning (Turk, Rudy, & Stieg, 1987). In this conceptualization, depressed mood or affect represents only one aspect of a multicomponent syndrome (Cancro, 1985; Shaw, Vallis, & McCabe, 1985).

Independent of severity, the prevalence of depression in the general population is estimated at between 8% and 20% (Taska & Sullivan, 1983). Estimates of its co-existence with

chronic pain have ranged from 10% (Blumer & Heilbronn, 1982; Chapman, Sola, & Bonica, 1979; Ward, Bloom, & Friedel, 1979) to as high as 50% (Large, 1980). Conversely, a high proportion of clinically depressed patients also report pain symptoms (Delaplaine, Iffabumuyi, Merskey, & Zarfas, 1978; von Knorring, Perris, & Eisemann, 1983).

The association between chronic pain and depression is highly complex, and the research evidence to date largely inconclusive (Turk, Rudy, & Stieg, 1987). Diagnostic signs and symptoms of depression -- psychomotor retardation, fatigue, anorexia and weight loss, insomnia, somatic preoccupation -- are observed in approximately 50% to 80% of general medical inpatients (Cavanaugh, Clark, & Gibbons, 1983). Psychiatric inpatients, particularly the depressed, frequently report vague or specific pain complaints (Baker & Merskey, 1967; Lindsay & Wyckoff, 1981; von Knorring, Perris, & Eisemann, 1983; Ward, Bloom, & Friedel, 1979). Similarly, chronic pain patients may develop symptoms of depression in response to pain itself (Sternbach, 1974).

Using a structured psychiatric interview, Kashiwagi, McClure, and Wetzel (1972) reported some degree and form of mood disturbance among a high percentage of chronic headache patients, but no differences between migraine and tension headache sufferers. Couch, Ziegler, and Hassanein (1975) reported a small but significant association between depression and migraine severity, particularly striking

among a subgroup who evidenced neurological symptoms. Barolin (1976) reported a higher prevalence of depression among headache-prone, relative to headache-free, subjects. Similarly, Ziegler, Rhodes, and Hassanein (1978) found evidence of more severe depression in a non-clinic sample of migraineurs relative to headache-free individuals. Higher levels of depression were associated with more severe and disabling episodes.

Blumer and Heilbronn (1982) reported a high incidence of self-reported depressed affect among tension headache and other pain patients; the former were more likely, however, to deny emotional problems or suicidal ideation. Both groups reported family histories of depression, chronic pain, alcohol abuse, and physical disability. Drummond (1985) compared common and classic migraine, tension, combined, and cluster headache patients on responses to a variety of measures. Tension headache subjects reported a higher incidence of depressive episodes and family or work-related problems than did the remaining headache groups. Depression was negatively correlated with migraine symptomatology, and was more likely when head pain was constant or nearly constant, and among tension headache subjects who also experienced neurological symptoms.

Diamond (1983) and Diamond and Dalessio (1978) proposed a model of the association between depression and headache in which the former is given precedence. Depression is

associated with a broad spectrum of cognitive, affective, and somatic manifestations, only one of which may be recurrent head pain. In this formulation, headache associated with depression has a number of key features: (a) episodes tend to occur at regular intervals in close correspondence with daily life events, and often follow periods of work or stress; (b) head pain displays diurnal variability, tending toward greater severity during the morning hours; and (c) response to standard analgesic medications is poor. The patient most often presents with a constellation of somatic complaints -- chronic general head or other pain, sleep disturbance, severe insomnia and early awakening, anorexia and weight loss, decreased sexual drive -- suggestive of depression. There is also often a history of severe intermittent headache over a period of years, with more recent onset of mild-to-moderate daily episodes.

Diagnosis follows a careful examination of headache history, with special reference to long standing habits, traits, coping methods, personal and family histories of mood disturbance, and perceived precipitants of both headache and depression. Differential diagnosis focuses on ruling out possible organic etiology (Friedman, 1979). Where tension-like headache predominates, one might expect manifestations of a reactive mood disturbance (e.g., anxiety). Head pain may comprise the principal or sole presenting complaint, with depressive symptomatology denied or masked.

Often, the premorbid picture is one of an inadequate or compulsive personality, though this is not always the case (Weatherhead, 1980). There is some evidence that children who experience both headache and depression also display greater social withdrawal, poorer school performance, sleep disturbance, and fatigue, relative to healthy children (Ryan & Ryan, 1978).

Saper (1983) described "somatized depression headache" as a psychogenic variant comprising a somatic manifestation of a mood disturbance of neurotic or even psychotic character. Head pain may be a depressive equivalent. This is in contrast to conversion headache, in which visible signs of distress are rarely observed. The disorder is usually associated with vegetative signs of depression.

Weatherhead (1980) concurred generally with Diamond's (1983) formulation, but argued that recurrent tension headache is associated only with depressive episodes marked by anxiety, such that muscle-contraction pain comprises a secondary somatization of anxiety. Because not all depressive episodes are marked by anxiety, tension headache is not always part of the clinical picture. In short, tension-like headache more commonly occurs in reactive mood disturbance resulting from either endogenous (e.g., feelings of guilt) or exogenous (e.g., personal loss) stresses.

The mechanisms of depressive headache have not yet been determined. Dalessio (1968) hypothesized that chronic tension headache is associated with a functional disorder of the nervous system, whereby the brain is unable to inhibit, and is prone to misinterpret, sensory information originating in normal or minimally-damaged peripheral tissues. Misinterpretation of input in turn hastens the development of a chronic functional pain syndrome. Whatever the merits of this hypothesis in accounting for the sensory aspects of tension headache or the transition from occasional to chronic headache, it does not directly address the role of depressed affect in the clinical picture.

Psychometric studies have provided only mixed support for the putative association between headache and depression. Werder, Sargent, and Coyne (1981) reported a fairly high prevalence of depressive symptomatology among migraine, tension, and combined headache patients administered the MMPI. A few studies (Andrasik, Blanchard, Arena et al., 1982; Blanchard, Andrasik, Arena et al., 1984; Weeks, Baskin, Rapoport et al., 1983) reported higher depression scores on the MMPI and BDI among combined headache patients relative to migraine and tension headache cases. Blanchard, Andrasik, Neff et al. (1982) found depression most evident among tension headache subjects. Further, depression correlated negatively with treatment response, a finding also reported by Levine (1984). In contrast, Ziegler,

Kodonaz, and Hassanein (1985) found no association between tension headache frequency and scores on the Zung Depression Scale (Zung, Richards, & Short, 1975).

The mediating influence of headache chronicity is not clear. Ajwani and Ajwani (1982) reported higher MMPI-D scores among more chronic headache sufferers. A longer and more extensive headache history was associated with greater life disruption, and a closer link between head pain and behavioural patterns suggestive of avoidance. Philips and Jahanshahi (1985), however, found no evidence of greater depression among more chronic patients. There is some evidence that ongoing depression is positively related to headache frequency (Garvey, Schaffer, & Tuason, 1983).

Several studies have yielded indirect evidence of a headache-depression association. It has been established, for example, that certain tricyclic antidepressants (e.g., amitriptyline) can effectively alleviate some cases of migraine and tension headache in the absence of depressive symptomatology (Blumer & Heilbronn, 1982; Couch, Ziegler, & Hassanein, 1975, 1976; Diamond, 1975; Diamond & Baltes, 1971; Gomersall & Stuart, 1973; Sjaastad, 1980; Ward, Bloom, & Friedel, 1979). The most responsive cases are said to be those characterized by severe headache without depressive features. Sjaastad (1980) identified a subgroup of chronic headache patients who evidenced a mixture of both vascular and muscle-contraction features as most responsive to

antidepressant therapy. These findings must be interpreted cautiously, however. It may be that amitriptyline, for example, possesses both antidepressant and analgesic-like properties (Fields, 1981; Merskey, Brown, Brown, Malhotra, Morrison, & Ripley, 1985; Monks & Merskey, 1984). Although the precise mechanisms of action are unclear, there is evidence that the tricyclics produce more rapid improvements in pain than in affective status (Sherwin, 1979), suggesting multiple sites of action.

In a study of EMG biofeedback, relaxation, and stress management approaches to treatment, Cox and Thomas (1981) reported concurrent, though not correspondent, reductions in headache activity and self-reported depression following treatment. Determination of depression was not clarified, however, and head pain and mood did not display identical response times.

Problems in research on affective variables. The significance of depressed affect in certain headache profiles has been widely accepted since the association was first studied by Diamond (1964), Lance and Curran (1964), and Dalessio (1968). It is not possible, however, to infer a causal connection between the two from observations of their temporal association. Both Diamond (1983) and Weatherhead (1980) hypothesized that mood disturbances may be accompanied by headache as a secondary somatization. It is important to note that some degree of mood disturbance

might be expected secondary to chronic headache (Martin, 1978). From this perspective, Sternbach's (1974, 1976) observations of a link between chronic pain and depression are particularly important. Specifically, there is evidence that chronic pain per se can lead to anxiety and depression (Merskey, 1978; Woodeforde & Merskey, 1972); the view of depression as secondary to pain is one widely held by both physicians and patients (Blumer & Heilbronn, 1982). Standardized psychological tests such as the MMPI and BDI may be particularly sensitive to affective involvement where depression is denied or not reported.

In addition to the issue of causality, there have been a number of problems evident in studies attempting to link headache and affect. There are at present no reliable estimates of the prevalence of depression among headache patients, or vice-versa. There are also problems in attempting to generalize findings obtained from clinical samples to the general population. Specifically, evidence of multiple referrals among headache and other chronic pain groups suggests the possibility that conversion or hypochondriacal features figure prominently in symptom profiles of those patients who seek treatment in more specialized pain clinics (Merskey, Brown, Brown, Malhotra, Morrison, & Ripley, 1985; Woodeforde & Merskey, 1972).

It has been noted that severe depression is relatively rare among chronic pain patients (Pilowsky, Chapman, &

Bonica, 1977), and that less extreme expressions of dissatisfaction with general functioning are far more common (Pelz & Merskey, 1982). In any event, tne typical clinical presentation of depression (American Psychiatric Association, 1986) is seldom observed in pain patients. Further, symptoms of pain and depression may alternate, rather than appear concurrently, in the clinical picture (Blumer, 1975; Blumer & Heilbronn, 1982). Alternatively, chronic pain may mask depressed affect (Sternbach, 1974, 1976). The role of denial in the overall psychological make-up of the chronic pain patient is not well understood at this time.

It does appear, then, that headache and depression co-exist in some fashion in certain individuals, though the precise nature of this relationship is not known. There are as yet little reliable data bearing on the competing hypotheses of headache as a secondary somatic manifestation of depression (Diamond, 1983; Diamond & Dalessio, 1978; Weatherhead, 1980) and mood disturbance as a secondary consequence of chronic pain (Martin, 1978; Sternbach, 1974, 1976).

Summary Comments on Chapter 3

The research examined in this chapter has provided support generally for the role of personality and affective variables in headache, but little conclusive evidence. Early

characterizations of the "headache personality" were derived from clinical observations on select headache populations. Later psychometric studies focused on evaluating specific personality variables, such as neuroticism, anger, hostility, obsessive-compulsiveness, and rigidity. The findings of these studies were somewhat inconsistent, however.

There is some evidence of fluctuations in mood coincident with headache activity. The significance of depressed affect in certain chronic headache sufferers is suggested by clinical observations, psychometric data, and findings on both antidepressant and relaxation-based treatments. It is not clear, however, whether headache in these individuals is secondary to underlying depression, or represents a precursor to mood disturbance. These competing views have yet to be tested empirically in a single study.

Several conceptual and methodological problems have limited the progress of research on psychological variables in headache. Because of the correlational nature of virtually every study reported to date, and in the absence of controlled prospective research, neither causality nor direction of causality can be ascertained. Specific procedural problems evident in these studies have included the use of self-referred clinical samples, unspecified or inadequate inclusion-exclusion criteria, lack of appropriate control groups or conditions, and the use of idiosyncratic

or non-standardized assessment instruments. These problems notwithstanding, the bulk of the evidence to date is at the very least suggestive of an association between headache activity and certain personality and affective variables.

4. SYNTHESIS OF CHAPTERS 1-3

Chapters 1 through 3 provide an overview of the current headache literature, with special reference to issues of classification, assessment, and the significance of psychological factors in headache. The bulk of the evidence on classification suggests that the typological perspective, as exemplified in the Ad Hoc Committee's (1962) classification system, is inadequate, and has met with increasing resistance in recent years (Anderson & Franks, 1981; Bakal, 1975, 1980, 1982; Lance, 1978; Olton & Noonberg, 1980; Philips, 1977, 1978; Waters & O'Connor, 1971; Ziegler, 1979).

To date, the principal challenge to typological classification has come from proponents of the severity-continuum perspective (Bakal, 1975, 1980, 1982; Bakal & Kaganov, 1977, 1979; Bakal, Demjen, & Kaganov, 1984; Bakal, Kaganov, & Demjen, 1983; Featherstone, 1985; Kaganov, Bakal, & Dunn, 1981), which holds that the essential differences among headache complaints are quantitative rather than qualitative. Several lines of evidence appear to support this perspective, though not in any particular formulation.

The severity-continuum perspective appears to offer a variety of research alternatives. To the extent that head pain is viewed as a continuous variable, with more extensive and multisystemic symptomatology associated with greater severity, the need for discrete headache types is precluded. Consequently, problems of subject selection and assignment -- nonrepresentativeness of clinical samples, adherence to rigid diagnostic criteria, selection of appropriate control subjects -- are largely mitigated. More specifically, the severity-continuum perspective invites research on nonclinic populations. In recent years, several nonclinic studies have been reported (Andrasik & Holroyd, 1980; Andrasik, Holroyd, & Abel, 1979; Crisp, Kalucy, McGuiness, Ralph, & Harris, 1977; Featherstone, 1985; Kaganov, Bakal, & Dunn, 1981; Korczyn, Carel, & Pereg, 1980; Passchier, van der Helm-Hylkema, & Orlebeke, 1984; Penzein, Holroyd, Holm, & Hursey, 1985; Thompson & Collins, 1979; Thompson, Haber, Figueroa, & Adams, 1980; Ziegler, Hassanein, & Couch, 1977; Ziegler, Rhodes, & Hassanein, 1978), many of which have employed college student samples.

Andrasik, Holroyd, and Abel (1979) reported a relatively high prevalence of headache complaint in a large college student sample; approximately 20% of a sample of 1,161 reported experiencing at least three headache episodes per week, and virtually all reported at least occasional headache. The majority of those with headache attributed

their episodes to muscle tension rather than vascular causes. Overall, the findings suggested a sufficiently high prevalence to warrant further investigation of headache activity among college students. From the severity-continuum perspective, it is perhaps less critical that the findings would not be immediately generalizable to headache clinic patients (i.e., who would, on average, fall higher on the severity continuum) than it is to note that clinic populations, from which the bulk of our understanding of headache is derived, are not believed to be representative of the headache population generally.

Review of the headache assessment literature suggests a need for concurrent self-recording of both the sensory (quality, intensity, location) and reactive aspects (affective responses, pain-motivated behaviour) of headache, over extended periods of time under natural conditions. This end might best be met through a strategy combining both headache history data and daily self-monitoring of headache using some form of diary. The HFR (Bakal & Kaganov, 1976) described in Chapter 2 focuses on the sensory (intensity) aspect and associated headache symptomatology. Daily HFR data might be supplemented by some variation on Tursky's (1976) descriptive adjectives procedure, such as that reported by Blanchard, Andrasik, Arena, and Teders (1982), providing some measure of the reactive dimension.

There are few clear guidelines as to appropriate length of the self-monitoring process, though this might be estimated through reference to expected frequency of headache. In a college student population, for example, prevalence data (e.g., Andrasik, Holroyd & Abel, 1979) would suggest a self-monitoring phase of 2 to 6 weeks to adequately sample headache activity. Intervention studies (Chesney & Shelton, 1976; Cox, Freundlich, & Meyer, 1975; Haynes, Griffin, Mooney, & Parise, 1975; Holroyd, Andrasik, & Westbrook, 1977) often report shorter baseline periods.

Although the findings on personality factors in headache have so far been inconsistent, there does appear to be an association between headache and certain affective variables. There is some evidence, for example, of mood shifts prior to or during headache (Arena, Blanchard, & Andrasik, 1984; Harrigan, Kues, Ricks, & Smith, 1984; Harvey & Hay, 1984), suggesting the value of incorporating a mood measure (e.g., DACL) into the self-monitoring process (Martin, 1983).

Anxiety has been implicated in headache, in both clinical reports (Crisp, Kalucy, McGuiness, Ralph, & Harris, 1977; Gianotti, Cianchetti, & Taramelli, 1972; Martin, 1966; Martin, Rome, & Swenson, 1967; Packard, 1979; Ziegler, 1979; Ziegler, Rhodes, & Hassanein, 1978) and psychometric studies (Andrasik, Blanchard, Arena et al., 1984; Mathew, Stubits, & Nigam, 1982). Extrapolating from Sternbach's (1974, 1976)

observations on chronic pain, one might expect symptoms of anxiety to be more pronounced during the early stages of an individual's headache disorder, later giving way to vegetative and other signs of depression as the disorder progresses or is maintained.

Several authors have examined the association between headache and depressed affect (Barolin, 1976; Blumer & Heilbronn, 1982; Couch, Ziegler, & Hassanein, 1976; Diamond, 1983; Diamond & Dalessio, 1978; Drummond, 1985; Kashiwagi, McClure, & Wetzel, 1972; Saper, 1983; Weatherhead, 1980; Ziegler, Rhodes, & Hassanein, 1978). In Diamond's (1983) formulation, headache is seen as a possible manifestation of depression, occurring in close association with a variety of predominately somatic complaints suggestive of mood disturbance. Most often, these episodes are described in tension-like terms. Weatherhead (1980) noted that the occurrence of tension headache symptomatology is most often associated with a reactive depression, marked by anxiety.

If Diamond's (1983) view is correct, then one would expect close association among headache activity, mood status, and perhaps perceived daily life stress over the course of an extended period of self-monitoring. Further, these associations might be most evident among individuals who score higher on psychological test scales measuring depression (BDI, MMPI-D). Alternatively, and in view of a documented interaction between affect and pain chronicity

(Sternbach, 1974, 1976), one might instead examine scores on some measure of anxiety (Weatherhead, 1980). Within a nonclinic sample, one would expect a proportionately smaller number of "chronic" cases.

From the severity-continuum perspective, one would also expect depressed affect to be more prevalent among those who report a mixture of headache symptoms -- a profile of periodic vascular episodes over a tonic background of tension headache, for example -- and somatic complaints consistent with depression. Mood status and headache activity would covary, particularly during more severe episodes. To the extent that headache complaint is more often associated with reactive depression, anxiety might dominate the presentation. With reference to Sternbach's (1974) notion of pain density, however, and despite a conceptual link between anxiety and muscle tension, one might alternatively expect greater evidence of depressed affect among those who report frequent tension-like or combined headache; vascular episodes are reportedly less frequent, and therefore associated with lower pain density. To date, no study has attempted to examine these associations in either clinic or nonclinic populations.

In conclusion, there seems to be ample justification for further application of the severity-continuum model in headache research, as a viable alternative to the typological perspective. In this view, research need not be

limited to clinical studies. A variety of assessment instruments is available by which one may study headache and its correlates under relatively naturalistic conditions, precluding reliance on laboratory analogue or simulation procedures. Among the largely unexplored and potentially fruitful areas of investigation is that linking headache and mood status. This area seems particularly suited to a severity-continuum analysis, in which critical variables are conceived as continuous in nature.

PART II

A STUDY OF HEADACHE AND DEPRESSED AFFECT

5. THE PRESENT INVESTIGATION

The present investigation examined the association between headache activity and depressed affect in an undergraduate college student population. The first of two studies reported surveyed headache history and current depression in a large sample of students, and provided the basis of selection for a second, intensive study of ongoing headache activity and depressed affect.

Study 1

Method

Subjects. The subjects consisted of 305 university students (136 males; 169 females) between the ages of 18 and 35 (*M* 19.50; SD 2.80) enrolled in introductory level psychology courses at the University of Manitoba during the fall semester, 1987. These were recruited by means of standard experimental sign-up booklets distributed during class meetings. In order to minimize reliance on memory for headache pain, and to ensure a purely non-clinic sample, only those volunteers who met the requirements of no fewer than two headache episodes per month and no current treatment for headache or depression were employed. Subjects signed up for one of seven available appointment hours

scheduled during a 2-week period, with a maximum of 40 subjects permitted to sign up for each appointment time.

Materials. All materials employed are contained in Appendixes A, B, and E.[1] Individual headache history was assessed through a 26-item headache history questionnaire (HHQ) adapted from Thompson and Collins (1979). The HHQ contains six items concerned with duration of headache complaint (chronicity), frequency, perceived severity, associated symptoms, and family prevalence of headache, and 20 rating scale (5-point) items by which to record the frequencies of specific headache features -- pain quality, location, laterality, accompanying mood and symptoms, precipitants and aggravants, degree of associated incapacitation, and relieving factors. These are scored by recording raw numerical ratings; no composite HHQ score is computed. Individual items were derived with reference to clinical studies of headache. The original normative sample consisted of 101 undergraduate college students between the ages of 18 and 23. Item by item test-retest reliability at 3 months ranged from .14 to .90; coefficients for the 20 symptomatology items in isolation ranged from .14 to .81. Self-monitoring of headache activity between administrations of the HHQ was shown to have no appreciable effect on reliability (Thompson & Collins, 1979).

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[1] To conform with thesis binding requirements, materials contained in Appendixes A through F are reduced by 25%.

Consistent with a purported relationship between headache and depressed affect, and in order to identify individuals who experienced depressive episodes of clinical severity, all subjects were administered the BDI (Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961). The rationale and basic structure of the BDI are discussed in Chapter 2 of the preceding review. Concurrent validity coefficients range from .62 to .77 against clinical ratings (Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961; Bumberry, Oliver, & McClure, 1978; Strober, Green, & Carlson,1981); correlations with the MMPI-D and other depression measures range from .41 to .80 (Burkhart, Gynther, & Fromuth, 1980; Schwab, Bialow, & Holzer, 1967; Seitz, 1970; Shaw, Vallis, & McCabe, 1985). Test-retest reliability ranges from .69 to .90 (Gallagher, Nies, & Thompson, 1982; Strober, Green, & Carlson, 1981), with coefficients of internal consistency in the range of .58 to .93 (Reynolds & Gould, 1981).

Procedure. At the time of recruitment, the procedures of Studies 1 and 2 were made explicit in a brief spoken presentation, and in written form on the sign-up booklets. Subjects met with the investigator on a single occasion in groups of approximately 30 to 40 in a controlled classroom setting (seating capacity 60). After presentation of questionnaire instructions, all were administered the HHQ and BDI in a single packet, in counterbalanced order. Instructions were given to complete all items in serial

order, without returning to any item or measure once it had been completed. The questionnaire packet was completed while seated at individual desks, and subjects were requested not to sit in close proximity to one another or speak during test completion. It was thought that group administration in an "examination-like" atmosphere might facilitate compliance, while the relative privacy of the seating arrangement might help minimize interpersonal influences on subject response set.

All materials were coded numerically to facilitate both subject confidentiality and contact of volunteers for the second study. Subjects were assigned a number between 1 and 312 which they maintained throughout the entire investigation. Upon completion of the HHQ and BDI, subjects were requested to indicate whether they were interested in participating further for experimental credit by printing their name, telephone number, and hours available for contact on a consent form attached to the questionnaire packet. Those interested were asked to complete the form, detach it from the packet, and submit it separately. Subjects were not informed of the basis of selection for Study 2, but were told that a total of five experimental credits would be awarded for full participation. A single experimental credit was awarded for participation in Study 1, which took between 30 and 40 minutes to complete.

Subjects who scored in the moderate to severe range (25 to 60) on the BDI were contacted by telephone to meet with the investigator during scheduled office hours. Two subjects who openly acknowledged current depressed affect when asked about BDI responses were advised discreetly of the availability of counseling services on campus. Neither presented as clinically depressed, nor did they report past or current histories of treatment for depression or headache complaint. The decision whether or not to seek counseling was therefore left to individual discretion. Both expressed strong interest in continuing their participation in the study, and were permitted to do so.

Hypotheses and Data Analysis

The following hypothesis was evaluated:

1. Current depression (total BDI score) would be positively associated with reported duration of headache complaint or chronicity (HHQ item 2), frequency (item 3), and severity (item 4) of headache. This prediction was derived from: (a) research linking chronic pain and vegetative signs of depression (Martin, 1978; Saper, 1983; Sternbach, 1974, 1976); (b) Diamond's (1983) observation that chronic patients often evidence histories of both depression and severe intermittent headache, with more recent onset of daily mild headache episodes;

and (c) Sternbach's (1974, 1976) notion of pain density (frequency) as it might relate to depression and the perception of pain severity. This association was expected to hold regardless of whether headache was secondary to (Diamond, 1983; Saper, 1983; Weatherhead, 1980), or predictive of (Martin, 1978; Sternbach 1974, 1976), depression. In Diamond's (1983) analysis, co-existence of severe headache and depression is more characteristic of periodic or "non-chronic" headache sufferers; presumably, the sample employed in the present study conformed generally to this profile.

The above hypothesis was evaluated through separate multiple regression analyses, employing chronicity, frequency, and severity as predictors of depression scores. Because the HHQ yields data on other critical headache attributes -- pain locations, associated symptoms, exacerbating and relieving factors -- group data were subjected to principal axis factoring (PAF) to determine the minimum number of dimensions that would account for variance in HHQ item scores. These factors were then employed as predictors of depression (BDI score) in a stepwise multiple regression.

Results

Characteristics of the sample. Group means and standard deviations for the 26 items of the HHQ are reported in Table 2, as are data derived from the normative sample of the HHQ (Thompson & Collins, 1979); frequencies of the six symptoms noted in HHQ item 5 are listed in Table 3. Severe or recurrent headache was reported by 35.4% of the sample. Average duration of headache complaint (chronicity) ranged from 6 months to 5 years, with an average frequency of between one and four episodes per month. These were most often rated as sufficiently intense to be both noticeable and moderately disruptive of concentration. On average, at least one immediate family member or relative was reported to experience recurrent headache. The most commonly noted symptom associated with headache was need to avoid bright light (62.3%); the least frequently noted, nausea or vomiting (17.4%). The most frequent features of headache included exacerbation by noise or bright light (HHQ item 9), throbbing or pulsing pain (item 10), bilateral pain (item 17), improvement upon lying down (item 22), and perceived cause in tension or stress (item 25). Based on relative frequencies of these features, the sample endorsed a common headache profile comprised of bilateral throbbing head pain -- perceived as triggered by tension or stress, exacerbated by noise or bright light, and relieved by rest. Scores on the BDI ranged from 0 to 49 ($\underline{M}$ 10.528; $\underline{SD}$ 1.093).

Table 2

Mean Ratings on the Headache History Questionnaire (HHQ) from Study 1

Variable		Thompson & Collins (1979) M	Study 1 (n=305) M	SD
HHQ	1	2.6	1.65	1.12
	2	2.1	1.66	1.14
	3	3.5	2.72	1.04
	4	3.5	2.69	1.04
	5	1.8	0.90	0.77
	6	2.7	1.69	1.06
	7	1.6	0.74	0.91
	8	2.1	1.20	1.10
	9	1.9	0.89	0.89
	10	2.3	1.32	1.11
	11	3.1	2.09	1.12
	12	2.5	1.79	1.12
	13	3.1	2.35	1.17
	14	2.0	1.35	0.90
	15	1.3	0.53	0.75
	16	3.6	2.29	0.99
	17	2.5	1.65	1.09
	18	2.1	0.82	0.87
	19	3.3	2.69	0.99
	20	2.2	1.55	0.95

Table 3

Reported Frequencies of Headache Symptoms (HHQ) from Study 1

Symptom	Frequency (%) Thompson & Collins (1979)	Study 1 (n=305)
Dizziness	2.7	43.3
Nausea/Vomiting	1.0	17.4
Visual Problems	16.8	31.5
Loss of Appetite	12.9	26.9
Avoid Light	20.8	62.3
Mood Changes	81.7	37.7

Main findings. Contrary to Hypotheses 1, total BDI score was not predicted by either chronicity, F (1,199) = .138, p >.05, or frequency of headache, F (1,303) = 3.668, p >.05. Severity was, however, predictive of BDI score, F (1,303) = 13.865, p <.0002. Chronicity correlated negatively, but not significantly, with frequency (r = -.105, p >.05); low positive correlations were obtained among severity and both chronicity (r = .190, p <.007) and frequency (r = .017, p >.05).

Principal axis factoring of the HHQ yielded four stable dimensions, comprising: (a) a general factor stressing long standing incapacitating head pain consistent with vascular headache (HHQ items 1, 2, 4, 7, 8, 15, 16, 19, 20, 21, 23, 26); (b) a general factor suggesting long standing unilateral or bilateral head pain (items 1, 2, 7, 8, 17); (c) unilateral or bilateral head pain associated with loss of appetite (items 5-4, 7, 8, 16, 17); and (d) head pain associated with sadness or depression (item 12). Criteria for setting the number of factors extracted were derived with reference to Comrey's (1978) cautionary notes on the eigen value-one procedure, and Loehlin's (1987) discussion of the scree test. Following varimax rotation, new variables were created from the factors obtained by adding the scores of their respective items. When entered in a stepwise regression (Table 8), Factor (a) was predictive of BDI score, F (1,196) = 23.063, p <.0000, as were Factors

(d), F (2,195) = 15.097, p <.0000, and Factors (b) and (c) taken together, F (4,193) = 8.347, p <.0000.

Study 2

In order to examine a purported relationship between headache and depression, volunteers from Study 1 who reported frequent headache episodes were employed in an extended self-monitoring study of headache activity -- occurrence, severity, associated symptomatology, perceived cause -- and depressed affect.

Method

Subjects. Subjects consisted of 36 volunteers (18 males; 18 females) from Study 1 who met the basic criterion of at least one headache episode per week (HHQ item 3). Data from five subjects who reported fewer than 10 episodes during Study 2 were excluded from analysis. The mean age of the final sample of 31 (14 males; 17 females) was 19.60 (SD 2.20).

Materials. All materials employed are contained in Appendixes C, D, and F. Headache activity was assessed using a modified version of the HFR (Bakal & Kaganov, 1976), the rationale and basic structure of which are discussed in Chapter 2 of the preceding review. For the purposes of the present study, only the intensity scale and symptoms list of the original HFR were retained. To facilitate

multidimensional assessment of head pain, the modified HFR incorporated items from the Numerical and Adjectival Pain Descriptors lists (Blanchard, Andrasik, Arena, & Teders, 1982), a non-standardized measure derived from Tursky's (1976) descriptive pain lists. The intensity measures were arranged in three lists, one of which provided verbal equivalents for a 6-point numerical peak intensity scale comparable to that of the HFR. The remaining lists were comprised of verbal pain descriptors: one assessing the sensory (intensity) dimension of pain; the other, the reactive (affective) dimension. The procedure also incorporated a 12-item perceived cause checklist as a measure of "daily life events" (Diamond, 1983) influencing headache activity. It was assumed that the inclusion of verbal intensity measures would clarify and standardize intended meanings of scale values for each subject, and mitigate problems associated with the use of visual analogue measures discussed in Chapter 2 of the preceding review.

Also administered were two measures assessing depression and depressed affect, respectively: the MMPI-D scale (Hathaway & McKinley, 1943); and three forms (A, B, C) of the DACL (Lubin, 1967). The former was administered as a supplementary depression measure to the BDI (Study 1), and was not employed in the evaluation of hypotheses. The purpose and structure of each of the measures is examined in Chapter 2 of the preceding review. The MMPI-D has been

shown to correlate with other clinical measures (e.g., interview data) in the range of .29 to .38. Test-retest reliability varies according to population, ranging from .60 to .80 for short intervals (< 2 weeks) to between .35 and .60 for longer periods (> 1 year). Internal consistency ranges from .60 to .90 over most populations (Dahlstrom, Welsh, & Dahlstrom, 1972; Golden, 1979; Graham, 1987). The DACL has demonstrated low to moderate concurrent validity with the MMPI-D (.25 to .53) and BDI (.38 to .66) (Christenfeld, Lubin, & Satin, 1978; Lubin, 1965; Lubin & Himelstein, 1976; Lubin & Levitt, 1979). Test-retest reliability is predictably low (.19 to .24), as the measure was designed to assess changes or fluctuations in mood. Alternate forms reliability ranges from .80 to .93; internal consistency ranges from .82 to .93. Mean scores on the DACL range from between 7 and 10 for normal individuals (males and females) to between 14 and 20 for depressed patients (Lubin, 1967).

The DACL was embedded within the modified HFR, such that each HFR form comprised seven items, assessing: associated symptoms (throbbing or pulsating pain, tightness and pressure in the head, visual disturbances, dull and aching pain, nausea or stomach pain, vomiting, light sensitivity); responses to verbal sensory and reactive pain descriptors; perceived cause or causes (allergies, hunger, fatigue, alcohol or drugs, food or drink, stress, poor sleep, anger,

depression or sadness, anxiety or tension, physical illness); verbal peak intensity; head pain intensity (VAS); self-medication; and depressed affect.

Procedure. The study was conducted in two phases. In the first of these, subjects met with the investigator as a group on a single occasion in a controlled classroom setting. They were then given a brief spoken introduction to the materials and general procedures of the study, following which they were administered the MMPI-D while seated as in Study 1. All forms were coded numerically, with subjects retaining numbers assigned previously.

Completion of the MMPI-D took between 10 and 20 minutes, following which subjects were introduced to the self-monitoring procedure, requiring completion of three daily recordings of headache activity and mood over 28 consecutive days via the modified HFR. Forms A, B, and C of the DACL were presented randomly within each subject's packet of 84 HFR forms. Ratings were completed at or near three specified times -- 10:00 a.m., 4:00 p.m., and 10:00 p.m. -- during each of the measurement days. On those occasions in which headache was absent, subjects completed only items 4 and 7 of the HFR. Thus, mood ratings were to be completed for all measurement times, independent of headache occurrence. Subjects were required to complete one HFR form for each measurement period of the study, and to maintain the sequence assigned; all forms were pre-dated with times

specified. The procedure was estimated to take no longer than 20 to 30 minutes per day, or approximately 10 to 15 hours over the course of the study. Each subject's materials were presented in manila folders, on the front of which were attached instructions for self-monitoring.

Compliance with the procedure was monitored on a weekly basis through periodic telephone and in-person contacts with each subject. Experimental credits were awarded at the rate of one per each completed week of the study, with an additional credit awarded for completion of the final week. Subjects were required to return completed HFR forms once weekly during office hours, at which time they received credits due. All 36 subjects completed the entire study, and general compliance with procedures was not problematic.

Upon completion of the final week of self-monitoring, and at the time of awarding final credits for participation, subjects were given verbal and written descriptions of the basic rationale and aims of Studies 1 and 2, and were encouraged to contact the investigator at the end of the academic year about individual findings on headache and mood. All were provided information on an independently-run headache treatment study that was currently recruiting subjects. No subject was requested to make enquiries about the treatment study; contact was at the discretion of the individual.

Hypotheses and Data Analysis

The following hypotheses were examined:

1. Association was predicted among: (a) ratings on the sensory (HHQ item 2-1) and reactive (item 2-2) pain descriptors lists; (b) peak intensity or severity (item 4); and (c) the visual analogue intensity scale (item 5). These were conceived as alternative intensity or severity scales that would yield comparable ratings in practice. It was reasoned that the provision of explicit verbal labels for numerical scale values would result in basic equivalency of measures.
2. Assuming the validity of Hypothesis 1, a computed severity index comprised of the mean of these four measures was predicted to associate positively with scores on the DACL. This hypothesis was derived from previous reports of a headache-mood association (Arena, Andrasik, & Blanchard, 1984; Harrigan, Kues, Ricks, & Smith, 1984; Harvey & Hay, 1984).
3. Positive associations were predicted among: (a) the severity index; (b) DACL scores; and (c) a symptom profile suggestive of tension-like or combined headache (item 1). Association between depressed affect and tension-like or combined headache was predicted by Diamond (1983) and Weatherhead (1980). That these episodes would be perceived as more severe

and incapacitating was derived from the severity-continuum perspective (Bakal, 1975, 1982), and from Sternbach's (1974, 1976) concept of pain density.

In addition, temporal associations between headache intensity (severity index) and mood status (DACL) were examined. If headache were secondary to depression (Diamond, 1983; Saper, 1983; Weatherhead, 1980), then one might expect evidence of depressed affect prior to headache onset (Harrigan, Kues, Ricks, & Smith, 1984; Harvey & Hay, 1984). If depression were secondary to headache, however, then one might expect either a near-constant depression of affect among the most chronic cases (i.e., high frequency headaches), or evidence of depressed affect at or following headache onset (Martin, 1978; Sternbach, 1974, 1976).

Hypotheses 1 and 3 were evaluated through Pearson correlations computed among variables specified. In the latter case, the procedure involved first computing indexes to comprise headache profiles based on ratings of symptoms suggestive of migraine (throbbing or pulsating pain, visual disturbances, nausea or stomach pain, vomiting, light sensitivity), tension (tightness and pressure in the head, dull and aching pain), and combined headache (any or all symptoms). The obtained indexes were then correlated with raw DACL scores. Hypothesis 2 was evaluated through multiple regression of the severity index defined above on

raw DACL scores. Temporal associations between headache severity and mood status were evaluated through cross-lagged panel correlations (e.g., Cook & Campbell, 1979) computed among the severity index and DACL scores obtained at lags of one and two measurement times, within and across days.

Results

Characteristics of the sample. Mean HHQ data for the 31 subjects retained in Study 2 are reported in Table 4. Frequencies of the six symptoms noted in HHQ item 5 are listed in Table 5. Severe or recurrent headache was reported by 90.3% of the subsample. Chronicity ranged from 6 months to 5 years, with an average frequency of between one and seven episodes per week. These were rated as sufficiently intense to be both noticeable and moderately disruptive of concentration. On average, at least one immediate family member or relative was reported to experience recurrent headache. The most commonly noted symptoms, endorsed by 74.2% of the group, were mood changes and need to avoid bright light; the least frequent was nausea or vomiting (12.9%). The most frequent features of headache episodes included exacerbation by noise or bright light (HHQ item 9), throbbing or pulsing pain (item 10), bilateral head pain (item 17), exacerbation by head movements (item 19), improvement upon lying down (item 22), head pain beginning in the morning and worsening through the day (item 23), and

Table 4

Mean Ratings on the Headache History Questionnaire (HHQ) from Study 2

Variable		Thompson & Collins (1979) M	Study 2 (n=31) M	SD
HHQ	1	2.6	1.97	0.95
	2	2.1	1.81	1.17
	3	3.5	2.71	1.10
	4	3.5	2.81	1.01
	5	1.8	1.07	0.89
	6	2.7	1.90	1.08
	7	1.6	0.87	1.05
	8	2.1	1.65	1.28
	9	1.9	1.07	0.77
	10	2.3	1.45	1.18
	11	3.1	2.36	1.14
	12	2.5	1.97	1.25
	13	3.1	2.52	1.06
	14	2.0	1.84	1.04
	15	1.3	1.00	0.97
	16	3.6	2.10	1.11
	17	2.5	2.10	1.01
	18	2.1	1.03	0.75
	19	3.3	2.90	1.04
	20	2.2	1.74	1.06

Table 5

Reported Frequencies of Headache Symptoms (HHQ) from Study 2

Symptom	Frequency (%) Thompson & Collins (1979)	Study 2 (n=31)
Dizziness	27.7	54.8
Nausea/Vomiting	1.0	12.9
Visual Problems	16.8	48.4
Loss of Appetite	12.9	38.7
Avoid Light	20.8	74.2
Mood Changes	81.7	74.2

perceived cause in tension or stress (item 25). Based on the relative frequencies of these features, subjects endorsed a common headache profile comprised of bilateral throbbing head pain, beginning in the morning and increasing in severity through the day, perceived as triggered by tension or stress, exacerbated by noise, bright light, or head movements, and relieved by rest. Scores on the BDI ranged from 0 to 49 (M 11.806; SD 10.117); scores on the MMPI-D[2] ranged from 14 to 40 (M 24.258; SD 5.508).

Group means and standard deviations for the seven HFR items (i.e., across all measurement periods) are reported in Table 6. Frequencies of the seven symptoms and 11 causes specified in HFR items 1 and 3 are listed in Tables 7 and 8. Self-medication through non-prescription analgesics (ASA, acetaminophen, ibuprofen) was reported for 79.6% of all headaches noted. The mean DACL score for the group was 9.494 (SD 4.192).

Main findings. For the purposes of comparison, the main analyses performed in Study 1 were repeated on the Study 2 subsample. Total BDI score was predicted by both headache frequency, F (1,29) = 7.991, p <.008, and severity, F (1,29) = 5.675, p <.024; as in Study 1, chronicity was not predictive of BDI score, F (1,26) = .870, p >.05. Table 9

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[2] Raw values were transformed to T-scores. Butcher and Tellegren (1978) have recommended the use of standardized test scores in research only when such scores have clear interpretive value, as is the case with scores on the MMPI.

Table 6

Mean Ratings on the Modified Headache Frequency Record (HFR) from Study 2 (n=31)

Variable	M	SD
Symptom Total	1.51	0.79
Severity 1	1.46	1.32
Severity 2	1.36	1.39
Cause Total	1.71	1.58
Peak Severity	2.22	1.20
Severity 3	1.81	1.39
DACL	9.49	4.19

Table 7

Reported Frequencies of Headache Symptoms (Modified HFR) from Study 2 (n=31)

Symptom	Frequency (%)
Throbbing/Pulsing	30.1
Tightness/Pressure	47.6
Visual Disturbances	7.4
Dull/Aching Pain	44.0
Nausea/Stomach Pain	5.0
Vomiting	0.2
Light Sensitivity	16.8

Table 8

Reported Frequencies of Perceived Causes of Headache (Modified HFR) from Study 2 (n=31)

Cause	Frequency (%)
(Not Sure)	32.5
Allergy	1.2
Hunger	9.5
Fatigue	34.9
Alcohol or Drugs	8.9
Food or Drink	2.5
Stress	30.2
Poor Sleep	23.2
Anger	6.8
Depression/Sadness	9.6
Anxiety/Tension	24.9
Illness	13.9

Table 9

Intercorrelations Among Severity Measures and the DACL (Modified HFR) from Study 2 (n=31)

Measure	1	2	3	4	5	6
Severity 1	--	.917	.899	.887	.969	.294
Severity 2		--	.880	.890	.966	.316
Peak Severity			--	.923	.957	.271
Severity 3				--	.949	.266
Severity Index					--	.294
DACL						--

$p<.000$, two-tailed test

reports Pearson correlations among: (a) the four severity measures of the modified HFR, which ranged from .880 to .923 (p <.000) with a mean of .899; (b) the severity index; and (c) DACL scores. The high intercorrelations among severity measures confirmed Hypothesis 1. Multiple regression yielded an association between the severity index and DACL scores, F (1,781) = 73.827, p <.0000, confirming Hypothesis 2. Correlations among the severity index, headache profile indexes, and DACL scores are listed in Table 10. Contrary to Hypothesis 3, no association was found among headache severity, mood status, and tension-like symptomatology. The highest correlations were obtained between the combined profile index and those of migraine (r = .663, p <.000) and tension headache (r = .655, p <.000). The combined index was negatively related to severity (r = -.556, p <.000).

Figure 2 depicts correlations among the severity index and DACL. These were computed concurrently and at lags of one or two measurement times. All associations were positive but weak; correlations at lag 2 were non-significant. Associations were strongest among successive recordings of the same measure (test-retest or lagged autocorrelations): The DACL correlated positively with DACL-1 (r = .299, p <.000); the severity index similarly correlated with prior ratings (r = .339, p <.000). Correlations for simultaneous recordings of the severity index and DACL were in the range of .293 to .294 (p <.000). Group findings did not suggest

Table 10

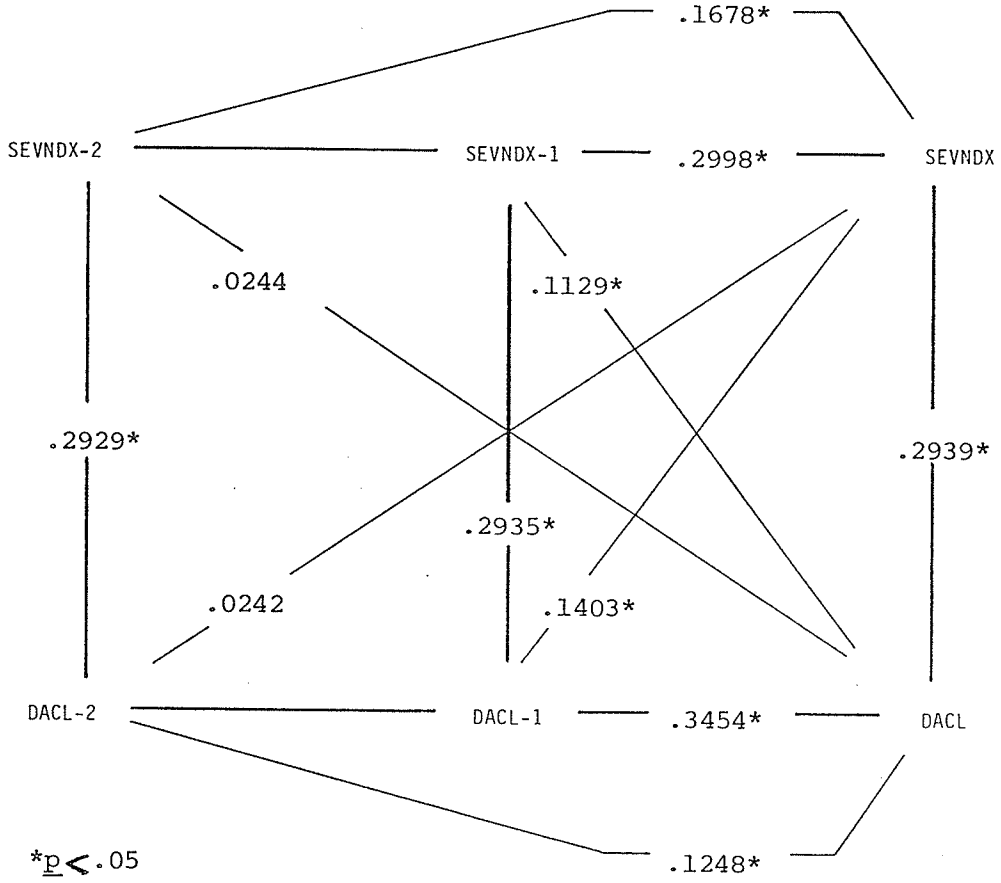
Intercorrelations Among Headache Profile Indexes, Severity Index, and DACL from Study 2 (n=31)

Measure	1	2	3	4	5
Migraine	--	-.132	.663	-.257	-.100
Tension		--	.655	-.478	-.117
Combined			--	-.556	-.165
Severity Index				--	.294
DACL					--

$p<.001$, two-tailed test

Figure 2.
Cross-Lagged Panel Correlation of DACL

SEVNDX: Study 2 (n=31)



*p < .05

	M	SD
DACL	9.494	4.193
SEVNDX	1.717	1.326

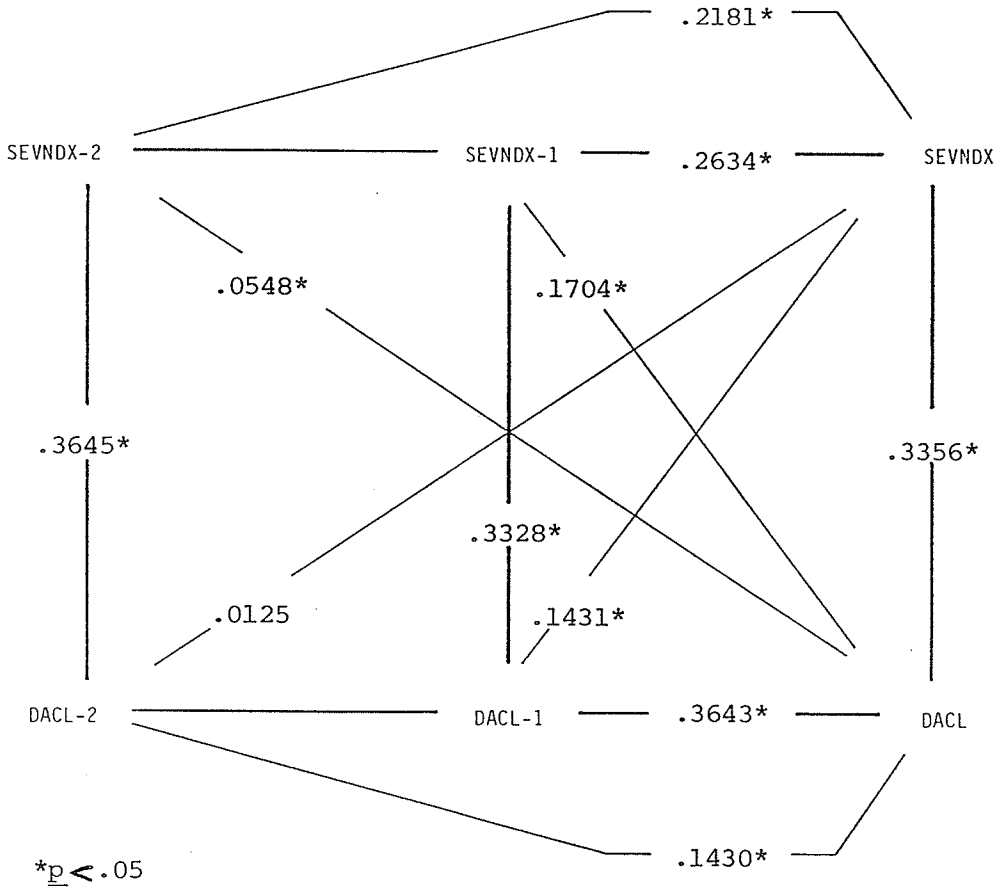
headache-mood association. To test for possible sex differences in association, lag correlations were performed separately on data obtained from males and females (see Figures 3 and 4). Females evidenced a more consistent pattern of association between headache severity and mood status than did males, for whom low negative correlations were obtained for the severity index and DACL at lags 1 (r = -.035, p >.05) and 2 (r = -.134, p <.031). Again, group findings by sex did not suggest headache-mood association.

Neither the DACL nor the severity index showed any systematic variation secondary to either time of day, F (2,2520) = 1.717, p >.05; F (2,781) = .885, p >.05, or to repeated measurement over successive days of self-monitoring, F (27,2495) = 2.292, p <.0002[3] F (1,26) = 1.342, p >.05.

Individual findings on headache-mood association. Because group data were not suggestive of a headache-mood association, cross-lagged panel correlations were computed by subject, to examine individual differences in degree of association between headache severity and depressed affect. Setting a minimum criterion of at least one significant correlation of .45 or greater between either or both

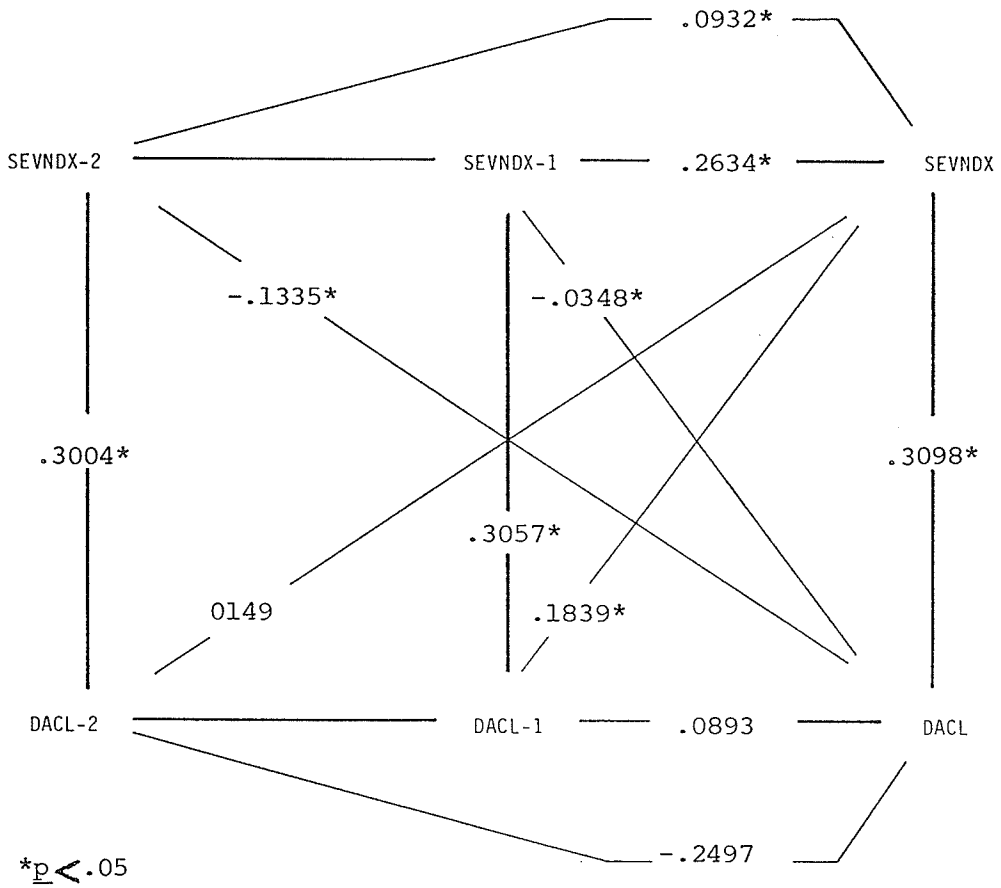
[3] While the reported F value was significant, it did not actually reflect the effects of day of study with days entered in sequential order.

Figure 3. Cross-Lagged Panel Correlation of DACL and SEVNDX: Females (n=17).



	M	SD
DACL	9.420	4.375
SEVNDX	1.470	1.075

Figure 4. Cross-Lagged Panel Correlation of DACL and SEVNDX: Males (n=14).



*p < .05

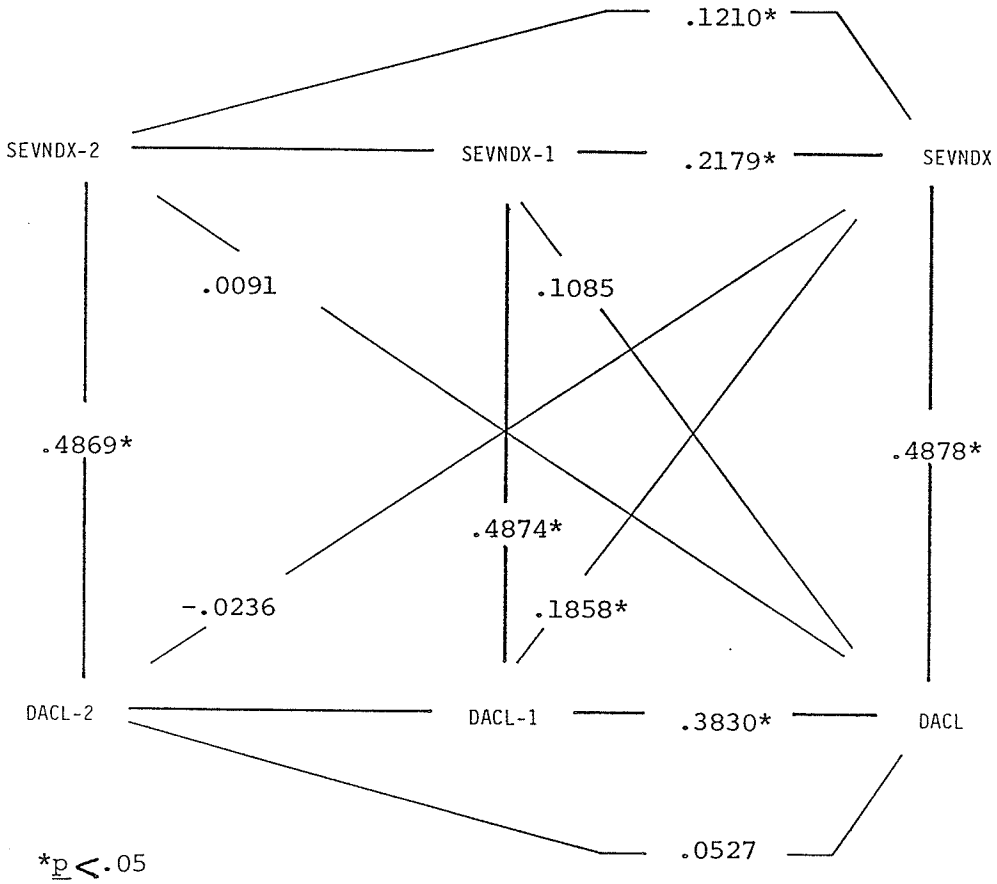
	M	SD
DACL	9.586	3.964
SEVNDX	1.107	0.832

concurrent (synchronous cross correlations) and "lagged" scores on the severity index and DACL, 12 subjects (6 males; 6 females) with a mean age of 20.58 (SD 1.171) were selected for closer examination. Individual findings (see Figures 5 to 17) were as follows:

1. Nine subjects (53, 79, 134, 141, 148, 161, 168, 266, 305) showed positive associations among concurrent severity index ratings and DACL scores -- i.e., significant synchronous cross-correlations in the range of .467 (p <.000) to .819 (p <.001). In most cases, stationarity[4] was evident. These findings suggest association between concurrent headache severity and mood status in some, but not all, subjects.
2. Three subjects (134, 141, 168) showed a positive association between current headache severity and mood status at the previous (lag 1) measurement time, with correlations in the range of .446 (p <.002)

[4] An index of the stability of the measures correlated over time, as suggested by stable synchronous correlations over successive measurement periods (Cook & Campbell, 1979).

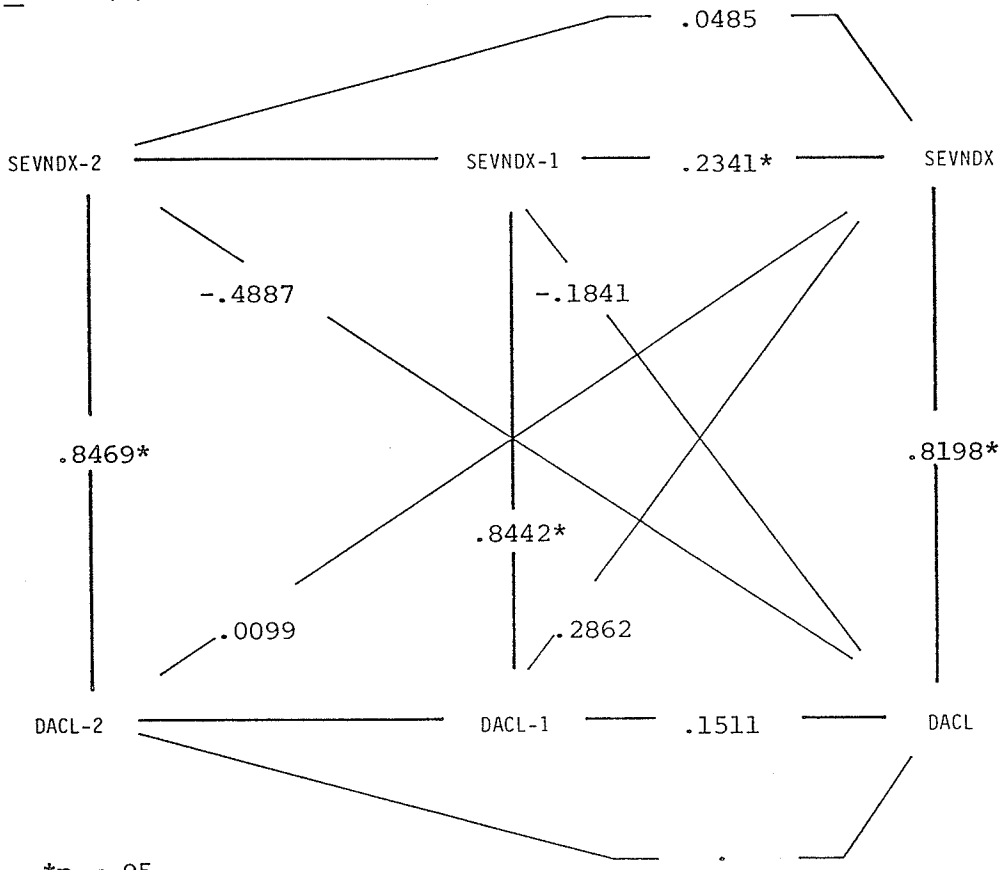
Figure 5. Cross-Lagged Panel Correlation of DACL and SEVNDX: Subjects Showing Headache-Mood Association (n=12)



	M	SD
DACL	9.774	3.734
SEVNDX	2.073	1.419

Figures 6-17. Individual Data on Cross-Lagged Panel Correlation of DACL and SEVNDX.

S# 79 (M)

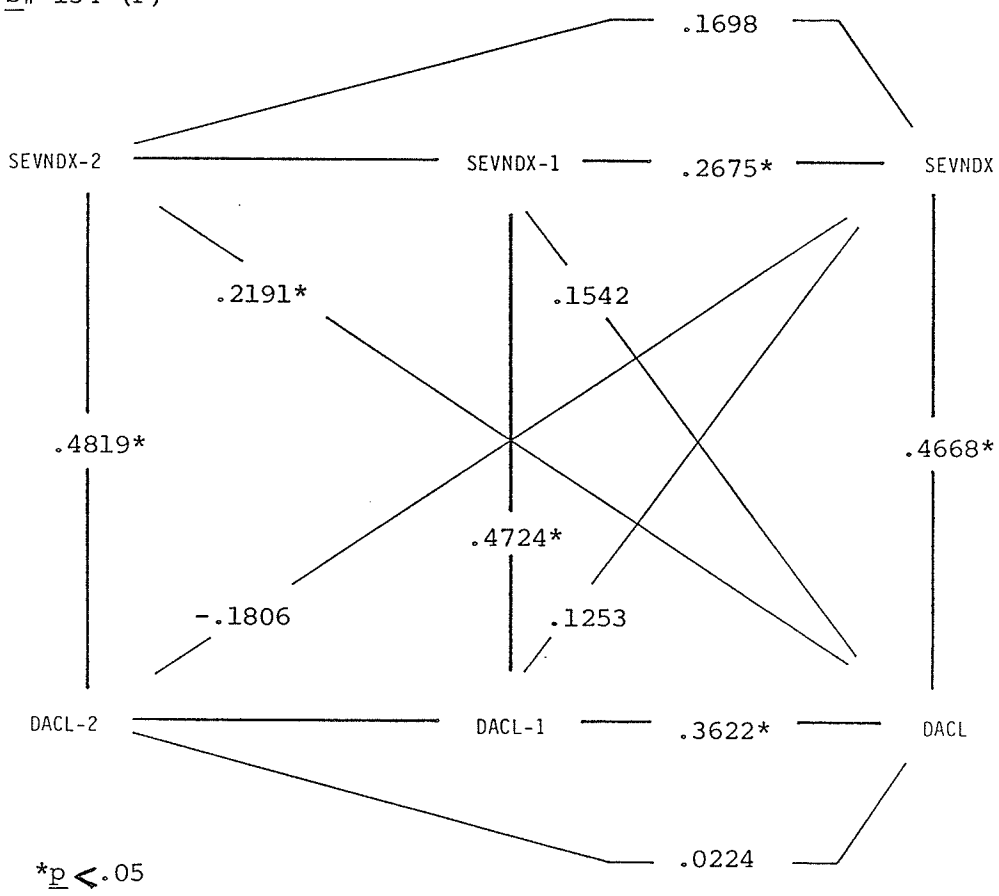


*p < .05

. correlation could not be computed

	M	SD
DACL	8.893	3.611
SEVNDX	2.115	1.299

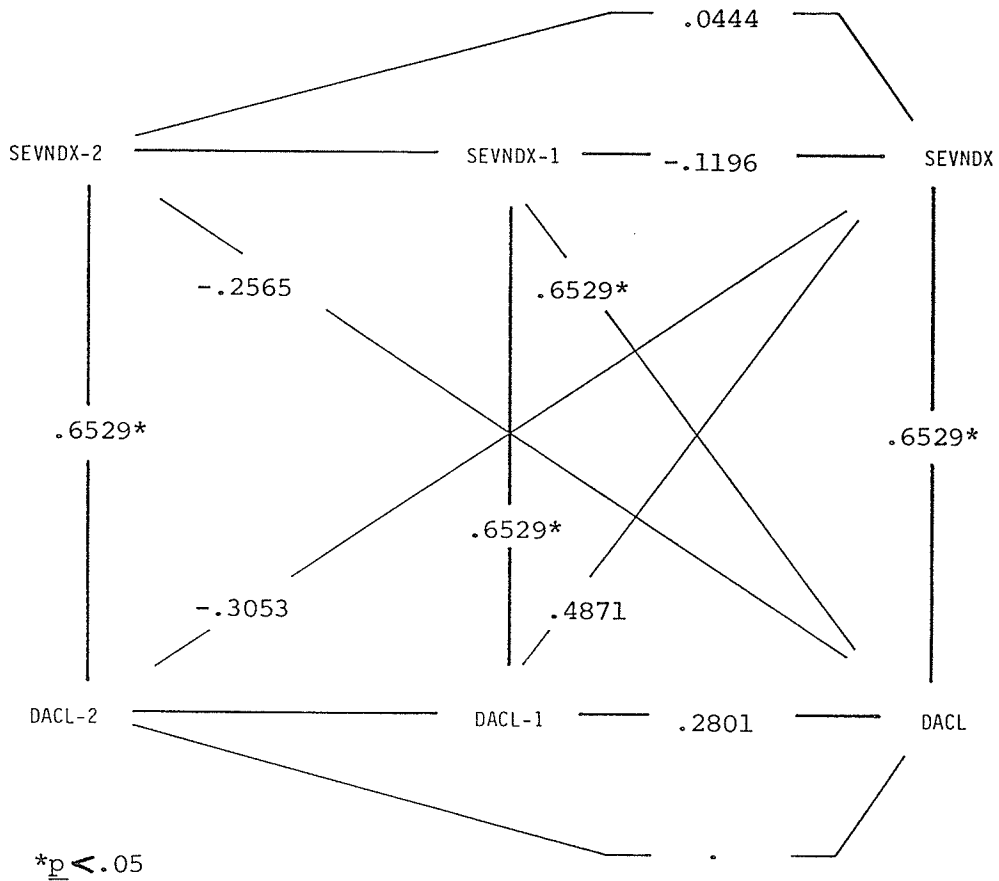
S# 134 (F)



*p < .05

	M	SD
DACL	11.121	3.921
SEVNDX	1.701	1.328

S# 141 (M)

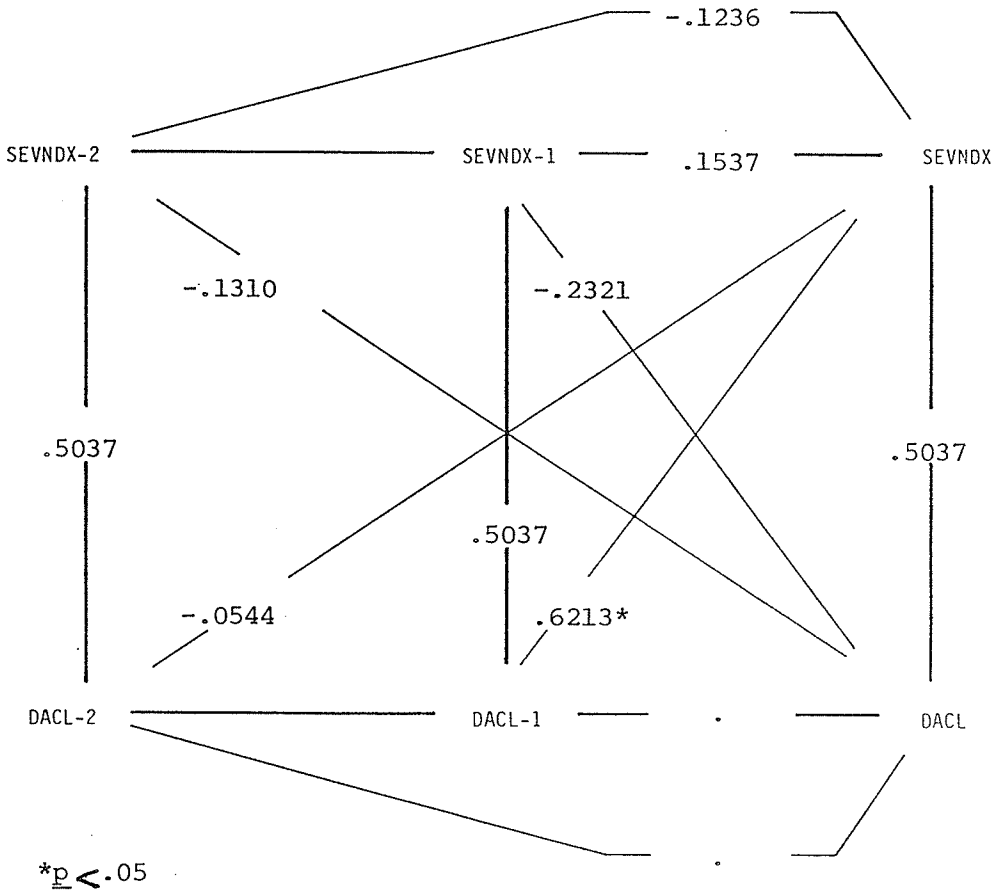


*p < .05

. correlation could not be computed

	M	SD
DACL	9.800	3.297
SEVNDX	1.443	0.858

S# 145 (M)

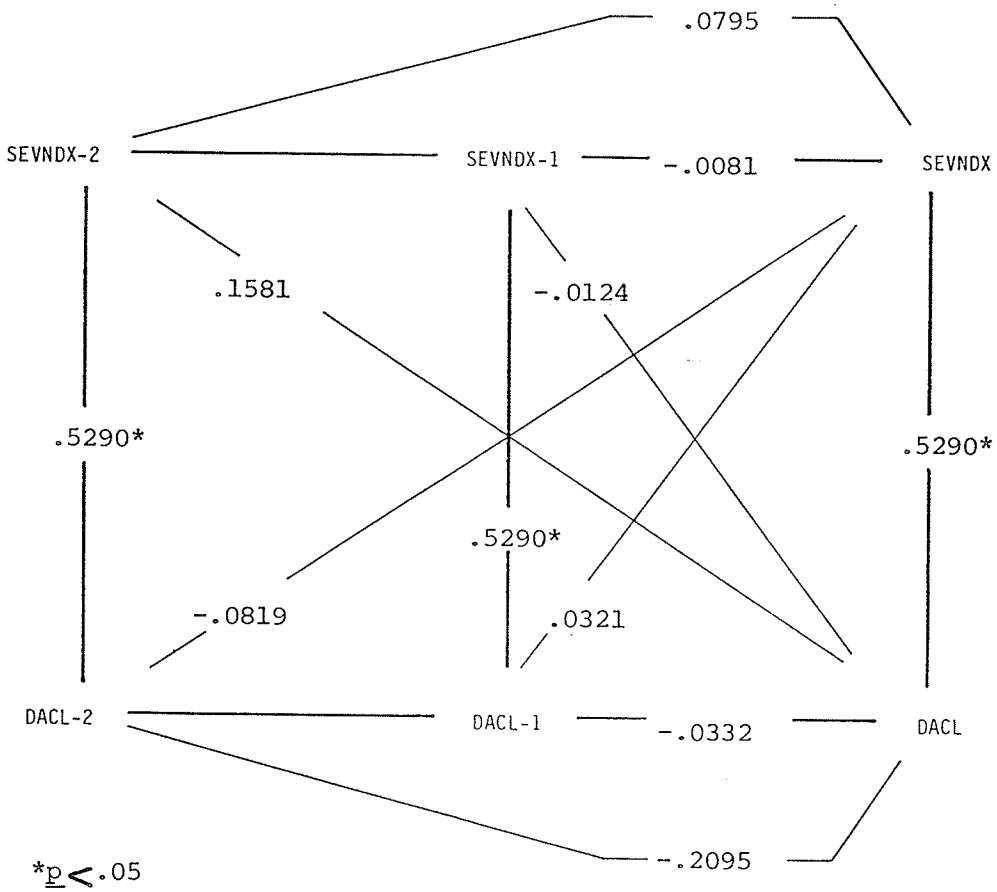


*p < .05

. correlation could not be computed

	M	SD
DACL	9.952	2.145
SEVNDX	1.813	0.931

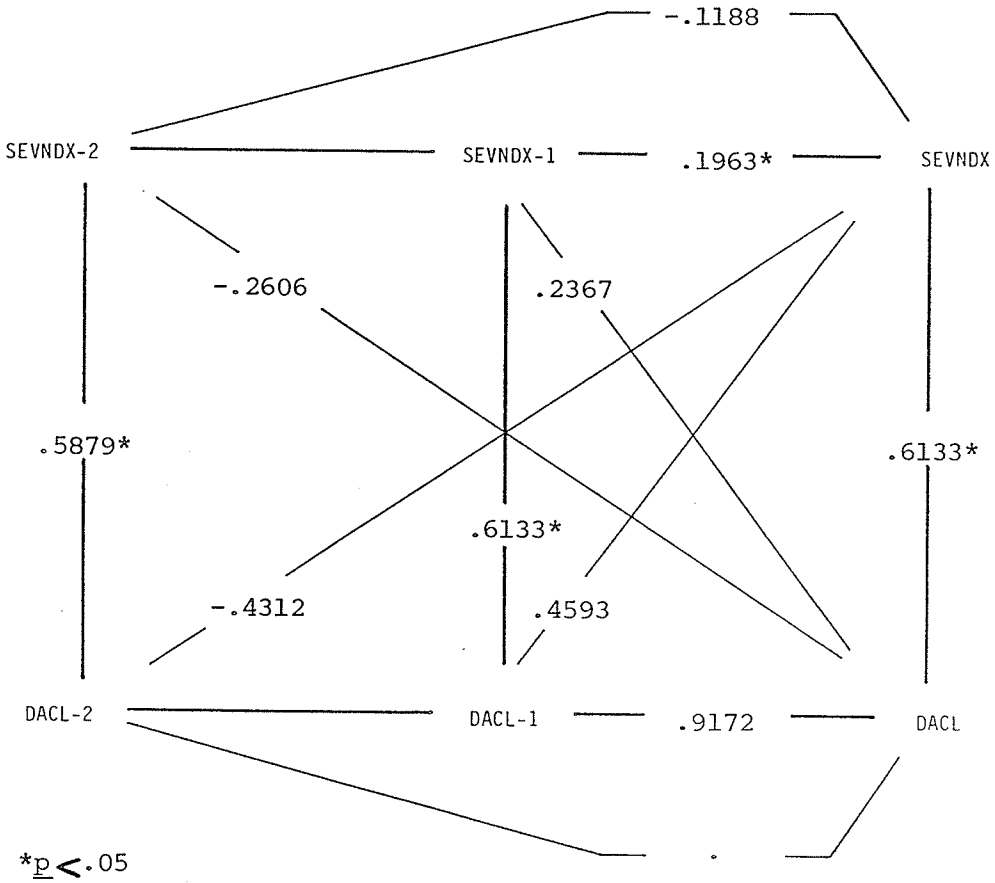
S# 148 (F)



*p<.05

	M	SD
DACL	10.619	2.688
SEVNDX	1.385	0.925

S# 161 (M)

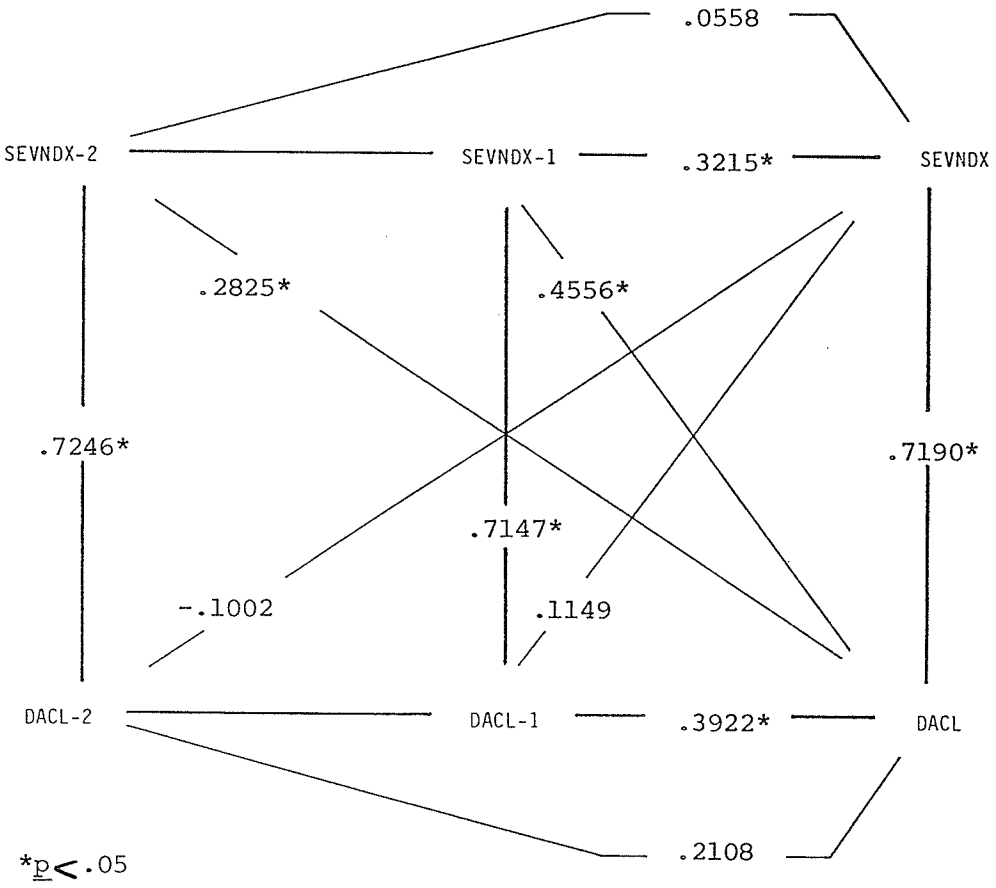


*p < .05

. correlation could not be computed

	M	SD
DACL	10.500	1.739
SEVNDX	1.214	0.964

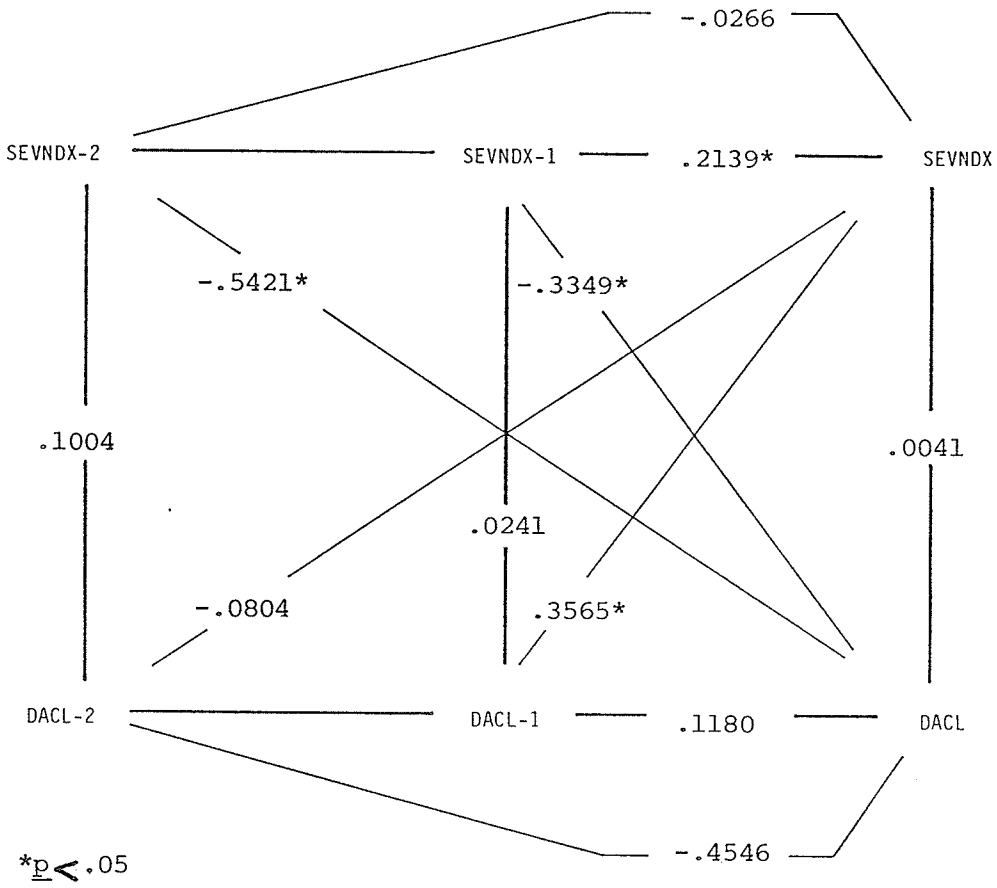
S# 168 (F)



*p<.05

	M	SD
DACL	11.238	5.249
SEVNDX	2.390	1.272

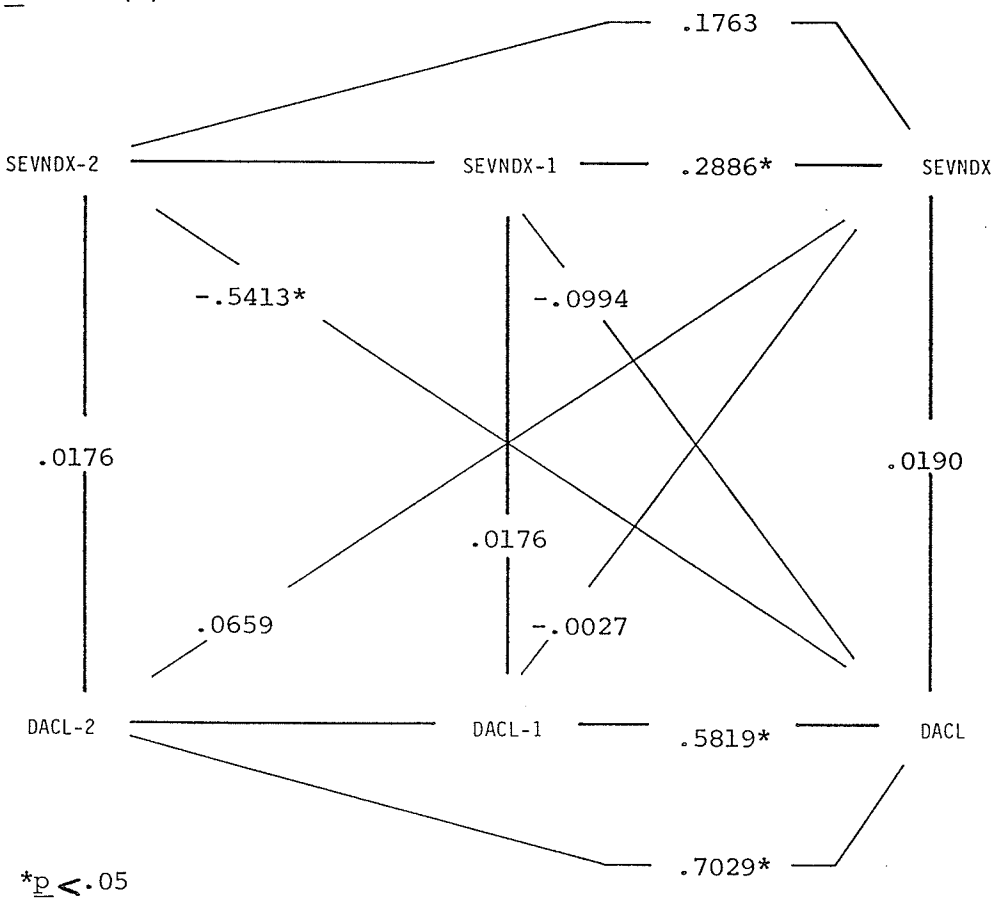
S# 247 (F)



*p < .05

	M	SD
DACL	9.548	3.534
SEVNDX	1.593	1.007

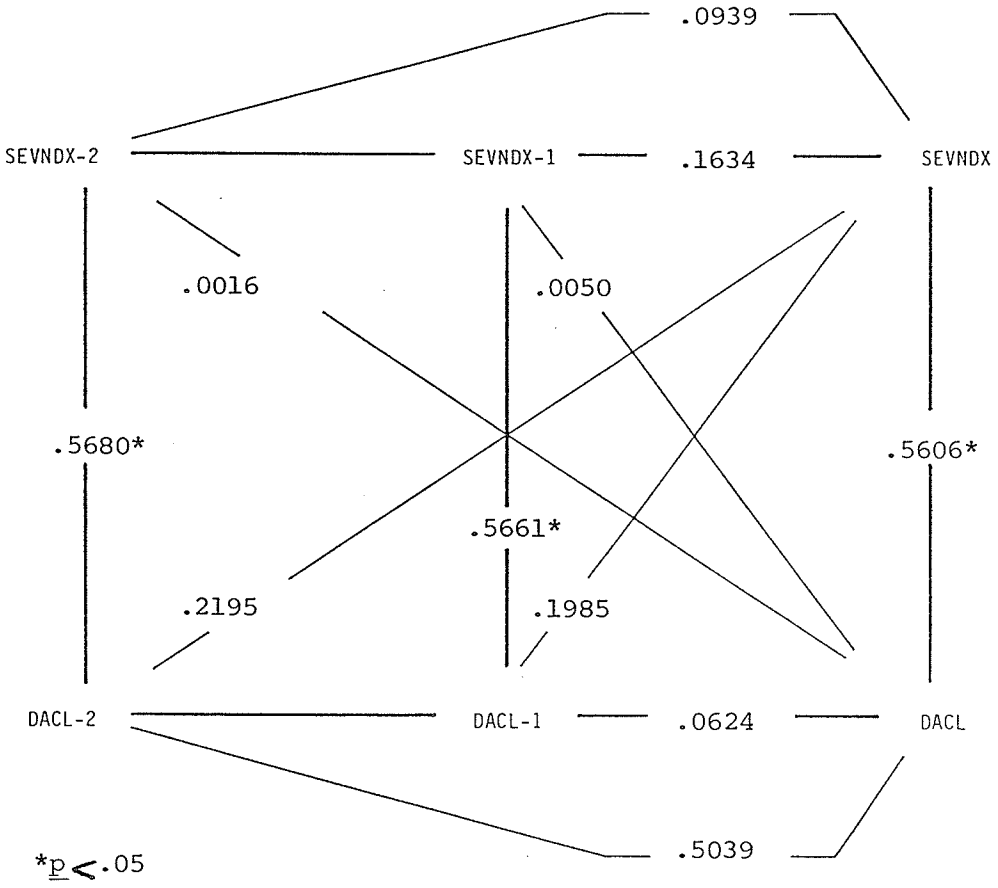
S# 257 (F)



*p < .05

	M	SD
DACL	10.039	3.394
SEVNDX	1.474	0.691

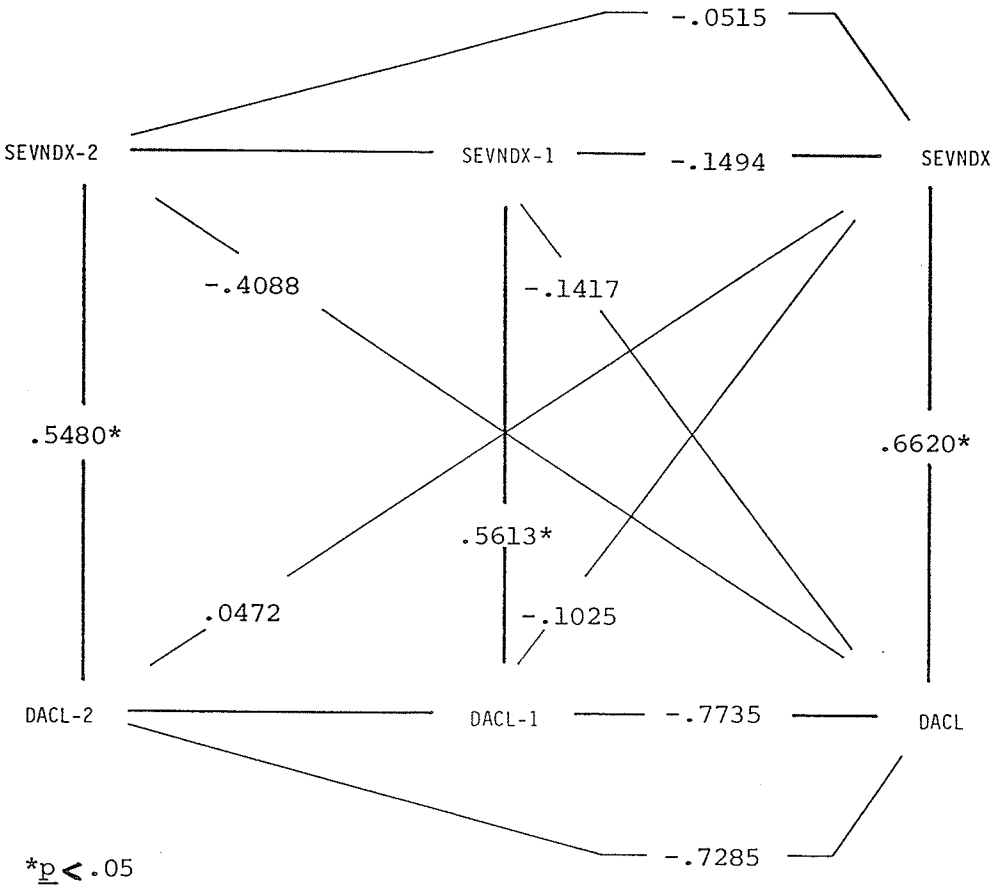
S# 266 (F)



*p < .05

	M	SD
DACL	9.349	4.538
SEVNDX	2.201	0.949

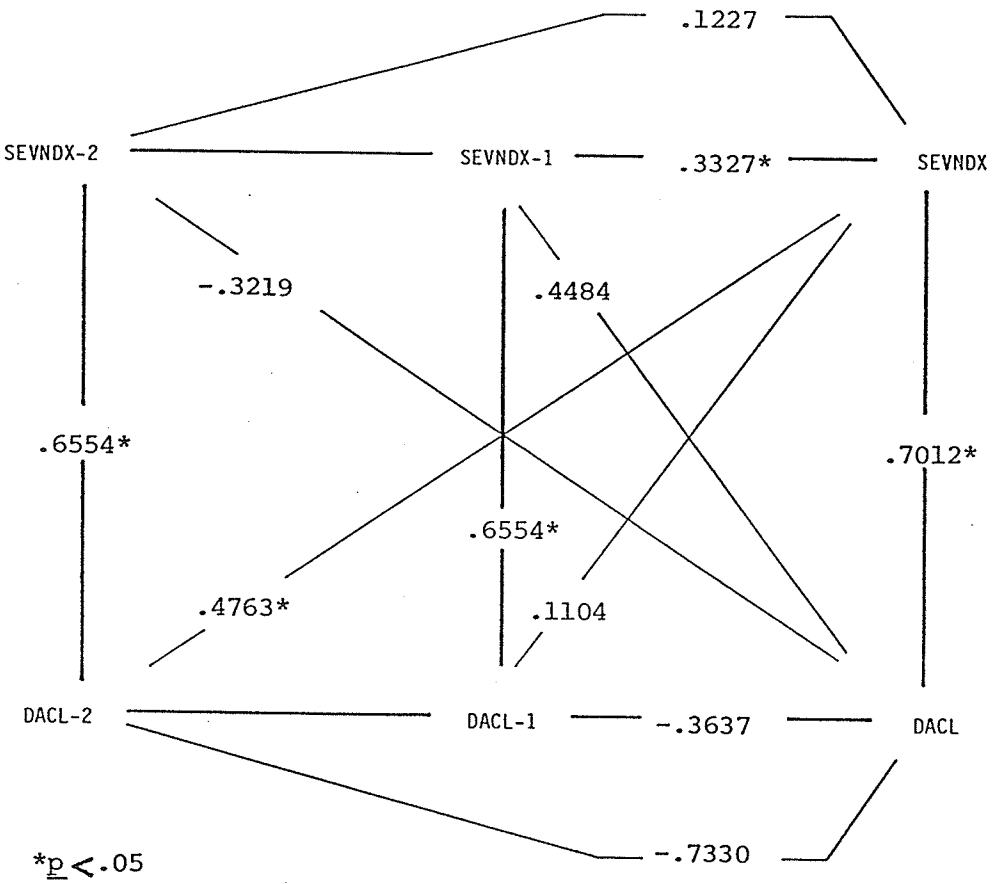
S# 305 (M)



*p < .05

	M	SD
DACL	7.637	4.715
SEVNDX	3.735	2.068

S# 53 (M)



*p < .05

	M	SD
DACL	8.500	2.753
SEVNDX	3.750	2.928

to .653 (p <.015).

3. One subject (247) showed a negative association (r = -.335, p <.044) between current mood status and headache severity at lag 1.
4. One subject (168) showed a positive association (r = .283, p <.045) between current headache severity and mood status two measurement periods earlier (lag 2).
5. Three subjects (247, 257, 305) showed a negative association between current headache severity and mood status at lag 2, with correlations in the range of -.409 (p <.052) to -.542 (p <.022).
6. Two subjects (145, 247) showed a positive association between current mood status and headache severity at lag 1 (r = .357, p <.037; r = .621, p <.037).
7. One subject (53) showed a positive association (r = .476, p <.050) between current mood status and headache severity at lag 2.

In order to determine whether observed headache-mood associations might be attributable to identifiable factors,[5] separate 2-way ANOVA's were conducted employing variables from the HHQ. Subjects who evidenced a headache-mood association were not differentiated from the rest of the Study 2 subsample by sex, F (1,22) = 0.028, p >.05, age, F (7,22) = 1.042, p >.05, chronicity, F (4,21) = 1.959, p

[5] A basic assumption of the cross-lagged panel correlation method is that the variables correlated should be regarded as, "symptoms of a common or third variable rather than as specific causes" (Cook & Campbell, 1979, p. 310).

>.05, frequency, F (2,21) = 0.368, p >.05, or severity, F (3,23) = 0.462, p >.05, but were differentiated on family prevalence of headache, F (3,23) = 3.104, p <.046, with the 12 subjects examined above showing more extensive family prevalence.

Discussion

The research reported in Study 2 was inspired initially by Diamond's (1983) interpretation of a clinically-observed association between headache and depression, particularly in light of Sternbach's (1974, 1976) view of mood disturbance in such cases as a secondary consequence of chronic pain. While undertaken ostensibly as a screening procedure, Study 1 did yield data beyond its limited intentions. Over one third of those who completed the HHQ reported being prone to severe or recurrent headache, on a weekly or more frequent basis, for no less than 6 months prior to the study. These episodes, though not severe on average, were characterized by bilateral throbbing head pain, perceived as triggered by stress, exacerbated by noise or bright light, and relieved by rest -- a profile most consistent with a non-specific vascular or combined form of headache (Ad Hoc Committee, 1962).

Consistent with earlier reports, no subject, irrespective of chronicity, frequency, or severity, had formally undergone medical or other treatment for headache in the

recent past (Andrasik, Holroyd, & Abel, 1979; Banks, Beresford, Morrell, Waller, & Watkins, 1975; Waters & O'Connor, 1975). This finding could not be taken as further evidence of the under-reporting of headache complaint, however, since current treatment was an exclusionary criterion. That nearly two thirds of the sample did not answer the severe or recurrent headache item positively suggests that it may have been interpreted as assessing whether or not they perceived themselves as "troubled" or "bothered" by headache -- i.e., whether headache interfered with routine activities on an at least occasional basis. Clearly, most subjects experienced relatively frequent headache, but did not view typical episodes as particularly bothersome.

Contrary to predictions derived from both Diamond (1983) and Sternbach (1974, 1976), neither headache chronicity nor frequency were predictive of depression on the BDI. Factor analysis of HHQ data did, however, suggest the predominance of duration of headache complaint in two of four derived dimensions. These in turn were predictive of BDI scores. It could be argued that the HHQ and BDI presented different task demands, with the former relying on memory for pain phenomena, which is typically poor (Oleson, 1978), and the latter involving evaluation of current or recent feelings and events. This cannot, however, adequately account for the association between headache severity and mood status

documented in Study 1; severity emerged as a consistent predictor of mood status in both studies.

Greater than 90% of the subsample who completed Study 2 reported being prone to severe/recurrent headache on the HHQ, up to several times weekly -- some, on a daily or near-daily basis -- for no fewer than 6 months prior to the study. Generally, their headaches approximated closely the collective profile of the larger Study 1 sample, with particular emphasis on headache of possibly longer duration (i.e., beginning in the morning and worsening through the day). Among this group, both severity and frequency, but not chronicity, were predictive of depression scores on the BDI. By selecting on the basis of reported frequency, Study 2 sought to focus on those subjects who were more likely to perceive headache as troublesome or problematic. The finding of a severity-mood association was consistent with Sternbach's (1974, 1976) notion of pain density as it might apply to affective response to pain. Both Sternbach and Diamond (1983) would, however, predict a greater role for headache chronicity.

Consistent with the first hypothesis of Study 2, the four intensity or severity measures on the modified HFR were highly intercorrelated. Andrasik et al. (1981) argued for the inclusion of some measure of the reactive dimension of pain experience during headache assessment. The findings of Study 2 suggest that, in practice, subjects may simply

regard like-scaled intensity or severity measures as equivalent. The inclusion of verbal definitions for numerical points on the visual analogue intensity scale (HFR item 5) may have served in the present case to standardize interpretation of scale values across subjects, leading to some attenuation of systematic differences that might have otherwise been obtained among measures of perceived intensity and affective response. Additionally, the use of identically-scaled (6-point) measures may have influenced response in the direction of treating these as more or less identical or interchangeable. In any event, it appears that headache severity can be adequately assessed through a relatively simple rating procedure comprising both visual analogue and verbal scale features.

Computed as a simple mathematical index, headache severity on the modified HFR was predictive of mood status on the DACL, confirming the second hypothesis of Study 2. Consistent with the findings of Study 1 and earlier investigations (Arena, Andrasik, & Blanchard, 1984; Harrigan, Kues, Ricks, & Smith, 1984; Harvey & Hay, 1984), perceived pain severity emerged as a critical variable in influencing -- or being influenced by -- mood status. No association was found between severity and total number of symptoms reported on the modified HFR, contrary to one prediction derived from Featherstone's (1985) severity-continuum model. It may be that headaches were, on

average, not sufficiently intense to comprise the expected range of associated symptoms.

Contrary to predictions derived from Diamond (1983) and Weatherhead (1980), no association was found among the severity index, DACL scores, and a symptom profile suggestive of tension-like or combined headache. This finding may in part be attributable to the relatively brief duration of headache complaint among the sample. It may be that the expected association holds only among more long standing cases, such as those who typically present in specialized pain clinics. Certainly, there is evidence that chronic headache and other pain patients differ from other clinic and non-clinic groups along several dimensions (Arena, Andrasik, & Blanchard, 1985; Belar, 1979; Crook, Rideout, & Browne, 1984; Friedman, 1979; Merskey, 1980; Sternbach & Timmermans, 1975; Woodeforde & Merskey, 1972). If one regards a strong pain-mood association as indicative of an increasing predominance of functional control over pain experience, then the role of chronicity in the association seems clear. The psychobiological model (Bakal, 1975, 1980), on the other hand, would predict greater autonomy of the physiological mechanisms underlying headache over time, with a consequent reduction in perceived headache-mood association. Further research is needed to establish the temporal course of this relationship.

The basic rationale of Study 2 concerned the temporal association between headache and mood status, and posed two related questions: (a) is there an association between headache (severity index) and depressed affect (DACL scores)?; and (b) if so, is one more "predictive" of the other? There is ample evidence of a pain-mood association among patients who present clinically (Diamond, 1983; Harvey & Hay, 1984; Martin, 1978; Sternbach, 1974, 1976), but little data bearing on this phenomenon in non-clinic pain groups, or among those in earlier stages of pain complaint. With respect to directionality, Diamond's (1983) formulation would predict some degree of mood disturbance as a precursor or partial determinant of perceived pain severity; Sternbach (1974, 1976) and others might expect depressed affect secondary to the experience of pain.

The findings of Study 2 were somewhat equivocal: Some patients showed evidence of a severity-mood association, but no consistent directional pattern emerged. Where association was evident, it was not strong. These findings may in part reflect a restricted range of scores on both the severity index and the DACL, though this was not determined statistically. There is some evidence that the magnitude of the correlation among two or more measures can be attenuated somewhat by a restricted range of scores (Barnes, 1984). The question naturally arises as to what factors might have differentiated among those who did and did not evidence the

association. The present investigation attempted to evaluate this question through reference to key HHQ variables -- sex, age, headache chronicity, frequency, severity, and family prevalence, only the last of which differentiated subjects.

It may be that those who reported more extensive family prevalence of headache had "learned", over time and through repeated interactions with parents, siblings, or relatives with histories of problem headache, to perceive their own developing headache disorder in terms of antecedents and consequences of pain complaint. Yen and McIntyre (1971) argued for the assessment of behavioural antecedents and consequences in the study of headache. There is mounting evidence of shared illness "patterns" within families that cannot be readily explained in terms of biological vulnerabilities (Coyne & Holroyd, 1982). If, as Andrasik et al. (1981) have suggested, affective response to pain is a critical factor in individual headache presentation, then greater attention will need to be directed at clarifying the influence of family and other interpersonal dynamics on pain expression (e.g., Roy, 1987).

As is suggested above, present findings on headache-mood association may be in part attributable to the relatively low chronicity of headache within the sample studied. Chronicity alone, however, cannot fully account for individual differences in directionality of association. Beyond family dynamics, more immediate differentiating

factors might be identified through, for example, more extensive psychological characterization of subjects. Completion of the full MMPI (Hathaway & McKinley, 1943) might have yielded more potential variables or patterns of interest, some of which -- the relative elevations of Scales 1 to 3, for example -- might then serve to differentiate among those who did and did not evidence association between headache severity and depressed affect.

Review of recent literature on headache and depression revealed only two intensive studies to date (Harrigan, Kues, Ricks, & Smith, 1984; Harvey & Hay, 1984), only the latter of which employed a research design comparable to that of the present investigation. Harvey and Hay (1984) had 10 migraineurs monitor headache intensity, frequency, and mood status using the DACL and a visual analogue mood scale, twice daily (morning, evening) over 30 consecutive days. Subjects averaged a total of 13.1 "headache days" during the study, with DACL scores in the range of 9.65 to 13.50 -- somewhat higher than "normal", but within the range of a normative sample (Lubin, 1967) of non-depressed psychiatric patients. Mood was not significantly more depressed on headache than on headache-free days, with the exception of days immediately preceding headache occurrence, for which DACL scores were lowest -- subjects reported feeling "less depressed" on days prior to headache.

These findings were not replicated in the present investigation. On average, DACL scores in Study 2 were lower than those reported by Harvey and Hay (1984), and well within the normal non-depressed range. As such, the headache-mood association observed among the 12 subjects examined in Study 2 could not be taken as evidence of a link between headache and depression per se, at least not with regard to how the latter is conceived clinically. The present investigation defined depression operationally in terms of scores on both trait (MMPI-D, BDI) and state[6] (DACL) measures of depression or depressed affect. As was noted in Chapter 3 of the preceding review, depression is a multidimensional construct (Cancro, 1985; Shaw, Vallis, & McCabe, 1985; Turk, Rudy, & Steig, 1987), comprised not only of depressed affect or "mood-tone" (Hinsie & Campbell, 1970), but of biological and psychosocial factors as well. It was reasoned that the most sensitive and appropriate measure of the construct as a whole -- irrespective of severity -- would be afforded by some measure of depressed mood. The DACL was employed because of its brevity, ease of completion, availability of multiple forms, and extensive normative data base. Nevertheless, even a more extensive

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[6] The use of the terms "trait" and "state" in this context is not meant to imply that the former class of measure is impervious to change over time or as a consequence of intervening processes (e.g., treatment, spontaneous remission), but rather that test-retest reliability is generally better for such measures than it is for those designed to evaluate transient or phasic changes. Generally speaking, the BDI can be regarded as a more appropriate measure of chronic enduring depression than the DACL.

headache-mood association would need to be interpreted with caution, as obtained scores on the DACL were not, on average, in the depressed range. This finding was not surprising, however, in that the sample employed was selected in part on the basis of at least moderate headache frequency, with no current history of treatment for either headache or depression.

Further comments are warranted on the general adequacies and inadequacies of the measures selected. Among headache questionnaires generally, the HHQ is well standardized (Thompson & Collins, 1979), and, while somewhat weighted toward "migrainous" items, seemingly well-suited to the task of screening and subject selection. Normative data are available for those items rating the frequencies of various headache features (items 7 to 26). On average, HHQ ratings in Study 1 were somewhat lower than those of the normative group which, demographically at least, closely resembled that of the present investigation. Lower ratings in Study 1 may in part reflect the effects of co-administration of the HHQ and BDI. Low average severity ratings and DACL scores could not be attributed to either diurnal effects (e.g., attenuation of daily average DACL by low morning scores, for example), or habituation over repeated measurements (day of study).

The range of scores on the BDI was considerable (0 to 49), but on average, within the low end of the mild range of

depression (M 10.528; SD 1.093). Similarly, average MMPI-D (M 24.258; SD 5.508) and DACL scores (M 9.494; SD 4.192) were within normal ranges While the BDI and MMPI-D were moderately correlated, similar associations did not hold among the BDI or MMPI-D and the DACL, perhaps reflecting the conceptual distinction between the former as trait measures, and the latter as a state measure. There is some evidence that the BDI yields unstable findings in the mild to moderate ranges of depression, particularly among young and college-aged respondents (Deardorff & Funabiki, 1985; Hatzenbeuhler, Parpat, & Mathew, 1983; Zimmerman, 1986). Despite the numerous advantages of the MMPI noted in Chapter 2 of the preceding review, there are problems in administering scales independently (Graham, 1987), most notably the loss of individual validity data. Further, there is evidence that individual clinical scales may be elevated among subjects or patients with a variety of medical illnesses or complaints (Harrison, 1975). In the present investigation, the MMPI-D was intended mainly as a supplementary measure to the BDI, and was not employed in the evaluation of major hypotheses.

Andrasik, Holroyd, and Abel (1979) reported a relatively high prevalence of recurrent headache in a large college student sample. While it was not feasible to estimate prevalence in the present investigation, subject recruitment was facilitated by high student interest in the topic, and

the response to requests for qualified subjects was generally positive. A college student sample cannot, of course, be assumed representative of the larger population of headache sufferers. At the same time, the problems of extrapolating from clinical headache samples, outlined in Chapter 2 of the preceding review, suggest the need for more extensive study of headache symptom patterns in a variety of groups, particularly those in earlier stages of headache complaint.

If the notion of a progressive predominance of functional control in chronic pain is valid, then it would seem wise to identify such factors early in the development of pain complaint. If, for example, headache were reliably predicted by depressed mood, then individuals could be instructed to identify mood "predictors" of oncoming headache, and perhaps apply preventive self-control measures (e.g., relaxation). Chronicity data in the present investigation suggested that the sample more or less conformed to the profile of the "pre-chronic" headache sufferer.

Conclusions and Suggestions for Further Study

The present investigation demonstrated the importance of headache severity as a potential determinant of mood status in recurrent or severe headache. A temporal association was observed among headache severity and mood status in some subjects, typically those reporting higher family prevalence

of headache. Most often, episodes resembled combined headache. Consistent with earlier reports (Anderson & Franks, 1981; Bakal & Kaganov, 1979; Cohen, 1978; Kaganov, Bakal, & Dunn, 1981), the majority of subjects reported both vascular and tension-like features at one time or another during the course of headache self-monitoring. Mood scores and headache symptom ratings were uniformly low throughout the study.

Future research on the headache-mood association appears warranted, and should address the following:

1. The present investigation would have benefitted from a selection procedure comprising both headache frequency and general level of depression (i.e., BDI score). This would have permitted the tracking of headache and mood status in individuals who displayed both frequent or severe headache and mood disturbance -- a group perhaps more likely to evidence reliable association between headache and depression.
2. The investigation would further have benefitted from a simpler, briefer mood measure, such as a visual analogue mood scale (e.g., Harvey & Hay, 1984), and a single, simple measure of headache severity, incorporating visual analogue and verbal scale properties.
3. Some measure of headache duration might have proven useful. It is not clear to what extent mood status

might be affected by length of headache episode. Study 2 treated each instance of reported head pain at a particular measurement time as a discrete event.

4. One potential influence on headache-mood covariation is menstrual cycle. Many female headache patients perceive some precipitating, aggravating, or relieving role of their cycle. Future studies employing female subjects should control for, or co-monitor, menstrual period during extended self-monitoring of headache. This could have been achieved in the present evaluation through addition of "menstrual cycle" to the perceived cause list of the modified HFR.
5. Further identification is needed of demographic, personality, and other factors that might serve to differentiate among subjects who do and do not evidence headache-mood association. This end might have been achieved in the present investigation by incorporating a more extensive psychological (e.g., full MMPI) and health (e.g., general health questionnaire) characterization of subjects.
6. Because anxiety is frequently cited as co-symptomatic of certain forms of headache complaint (Crisp, Kalucy, McGuinness, Ralph, & Harris, 1977; Ziegler, 1979; Ziegler, Rhodes, & Hassanein, 1978), and is relatively simple to assess empirically (e.g., STAI), a design incorporating concurrent evaluation of

anxiety and depression or other affective disturbance would be useful.

7. The present investigation demonstrated the utility of a self-monitoring strategy involving the tracking of headache and mood status under relatively naturalistic conditions. Further study is needed to aid in determining appropriate frequency and duration of recording, which may in turn be derived from reported frequency and duration of headache.

8. Given the relatively high prevalence of headache reported in college student samples (Andrasik, Holroyd, & Abel, 1979), it would seem that these and similar groups could be employed more extensively in the study of non-chronic headache, the development of the chronic pain profile among headache sufferers, and the covariation of head pain and mood status or some other variable(s). As was noted in Chapter 2 of the preceding review, the severity-continuum model of headache (Bakal, 1975; Featherstone, 1985) both permits and invites further research on non-clinic groups.

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Appendixes

Appendix A Headache History Questionnaire (HHQ)

HEADACHE HISTORY QUESTIONNAIRE (HHQ)

HH/DI Subject# _____ Date: _____

INSTRUCTIONS: The following questions are concerned with your personal headache history. Please try to answer each with your "typical" headache episode in mind, by circling the appropriate number next to the item.

1. Are you prone to severe or recurring headaches?
   0 yes
   1 no

2. If you answered "yes", then about how long would you say you've been experiencing these headaches?
   0 less than one month
   1 one to six months
   2 six months to a year
   3 one to five years
   4 five to ten years
   5 more than ten years

3. About how often do you have headaches?
   0 less than once every 2 or 3 months
   1 about one every 2 or 3 months
   2 once a month
   3 once a week
   4 once a day
   5 more than once a day, or nearly always

4. Which of the following best describes the amount of pain you feel during a typical headache episode?
   0 no noticeable pain
   1 the pain is there, but I can easily ignore it
   2 I can't ignore it, but it doesn't interfere with things
   3 severe enough to interfere with my concentration
   4 severe enough to interfere with most activities
   5 so severe that I feel sick and need to lay down

5. Do you ever experience any of the following symptoms during a headache? (check one or more)
   _ dizziness
   _ nausea or vomiting
   _ visual problems or distortions
   _ loss of appetite
   _ need to avoid bright light
   _ mood changes

6. Are any of your family members or relatives bothered by headaches? (check one or more)
   _ none
   _ mother or father
   _ brother or sister
   _ grandmother or grandfather
   _ other relative(s)

7. Does your typical headache begin on <u>one side</u> of the head?
   0 never
   1 very seldom
   2 about half the time
   3 most of the time
   4 always

8. When you have head pain on <u>both</u> sides of the head, does it ever seem to be stronger on one side than the other?
   0 never
   1 very seldom
   2 about half the time
   3 most of the time
   4 always

9. Does noise or bright light seem to make your headache worse?
   0 never
   1 very seldom
   2 about half the time
   3 most of the time
   4 always

10. Does your headache pain seem to "throb" or "pulse"?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

11. Does your headache pain ever seem to move from one side of the head to the other?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

12. Do you ever feel sad or depressed during a headache?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

13. Do your arms or legs ever feel cold during a headache?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

14. Do your eyes ever water, itch, or burn during a headache?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

15. Do you ever have nausea or stomach pains during a headache?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

16. Do you ever lose your appetite because of a headache?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

17. Does your headache pain ever occur equally on <u>both sides</u> of the head?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

18. Does your headache pain ever feel like a tight band around your head?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

19. Does your headache ever seem to get worse if you move your head?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

HEADACHE HISTORY QUESTIONNAIRE (HHQ) -- CONT.

HH/DI Subject# _____ Date: _____

20. Do you ever wake up with a headache?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

21. Are you ever woken during the night by a headache?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

22. Does your headache ever get better if you lay down?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

23. Do you ever get headaches that start in the morning and seem to get worse as the day goes on?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

24. Do you ever have headaches because of changes in the weather?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

25. Do you ever have headaches because of tension or stress?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

26. Do your headaches ever interfere with your regular activities?
    0 never
    1 very seldom
    2 about half the time
    3 most of the time
    4 always

Appendix B Beck Depression Inventory (BDI)

BECK DEPRESSION INVENTORY (BDI)

HH/DI Subject# _____ Date: _____

INSTRUCTIONS: On this questionnaire are groups of statements. Please read each group of statements carefully. Then pick out the one statement in each group which best describes the way you have been feeling the past week, including today! Circle the number beside the statement you picked. If several statements in the group seem to apply equally well, then circle each one. Be sure to read all the statements in each group before making your choice.

1. 0 I do not feel sad
   1 I feel sad
   2 I am sad all the time and I can't snap out of it
   3 I am so sad or unhappy that I can't stand it

2. 0 I am not particularly discouraged about the future
   1 I feel discouraged about the future
   2 I feel I have nothing to look forward to
   3 I feel that the future is hopeless and things can't improve

3. 0 I do not feel like a failure
   1 I feel I have failed more than the average person
   2 As I look back on my life, all I see is a lot of failures
   3 I feel I am a complete failure as a person

4. 0 I get as much satisfaction out of things as I used to
   1 I don't enjoy things the way I used to
   2 I don't get real satisfaction out of anything anymore
   3 I am dissatisfied or bored with everything

5. 0 I don't feel particularly guilty
   1 I feel guilty a good part of the time
   2 I feel quite guilty most of the time
   3 I feel guilty all of the time

6. 0 I don't feel I am being punished
   1 I feel I may be punished
   2 I expect to be punished
   3 I feel I am being punished

7. 0 I don't feel disappointed in myself
   1 I am disappointed in myself
   2 I am disgusted with myself
   3 I hate myself

8. 0 I don't feel I am any worse than anybody else
   1 I am critical of myself for my weaknesses or mistakes
   2 I blame myself all the time for my faults
   3 I blame myself for everything bad that happens

9. 0 I don't have any thoughts of killing myself
   1 I have thoughts of killing myself, but would not carry them out
   2 I would like to kill myself
   3 I would kill myself if I had the chance

10. 0 I don't cry any more than usual
    1 I cry more now than I used to
    2 I cry all the time now
    3 I used to be able to cry, but now I can't even if I want to

11. 0 I am no more irritated now than I ever am
    1 I get annoyed or irritated more easily than I used to
    2 I feel irritated all the time now
    3 I don't get irritated at all by the things that used to irritate me

12. 0 I have not lost interest in other people
    1 I am less interested in people than I used to be
    2 I have lost most of my interest in other people
    3 I have lost all of my interest in other people

13. 0 I make decisions about as well as I ever could
    1 I put off making decisions more than I used to
    2 I have greater difficulty in making decisions than before
    3 I can't make decisions at all any more

14. 0 I don't feel I look any worse than I used to
    1 I am worried that I am looking old or unattractive
    2 I feel that there are permanent changes in my appearance that make me look unattractive
    3 I believe that I look ugly

15. 0 I can work about as well as before
    1 It takes extra effort to get started at doing something
    2 I have to push myself very hard to do anything
    3 I can't do any work at all

16. 0 I can sleep as well as usual
    1 I don't sleep as well as I used to
    2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep
    3 I wake up several hours earlier than I used to and cannot get back to sleep

17. 0 I don't get more tired than usual
    1 I get tired more easily than I used to
    2 I get tired from doing almost anything
    3 I am too tired to do anything

18. 0 My appetite is no worse than usual
    1 My appetite is not as good as it used to be
    2 My appetite is much worse now
    3 I have no appetite at all any more

19. 0 I haven't lost much weight, if any, lately
    1 I have lost more than 5 pounds
    2 I have lost more than 10 pounds
    3 I have lost more than 15 pounds
    _ I have purposely been trying to lose weight (check)
    _ I have not been trying to lose weight (check)

20. 0 I am no more worried about my health than usual
    1 I am worried about physical problems such as aches and pains, or upset stomach, or constipation
    2 I am very worried about physical problems and it's hard to think of much else
    3 I am so worried about my physical problems that I cannot think about anything else

21. 0 I have not noticed any recent change in my interest in sex
    1 I am less interested in sex than I used to be
    2 I am much less interested in sex now
    3 I have lost interest in sex completely

22. What is your: Age ____, Sex ____

Appendix C MMPI-D Scale

MMPI-D (DEPRESSION) SCALE

MDI Subject# _____ Date: _____

INSTRUCTIONS: Beside each of the following items, please check either "T" (true) or "F" (false), according to whether or not you feel that the statement applies to you generally. Please answer all items, but do not spend too much time on any one item.

T	F		
___	___	1.	I have a good appetite
___	___	2.	I am easily awakened by noise
___	___	3.	My daily life is full of things that keep me interested
___	___	4.	I am about as able to work as I ever was
___	___	5.	I work under a great deal of tension
___	___	6.	I am very seldom troubled by constipation
___	___	7.	I am troubled by attacks of nausea and vomiting
___	___	8.	At times I feel like swearing
___	___	9.	I find it hard to keep my mind on a task or job
___	___	10.	I seldom worry about my health
___	___	11.	At times I feel like smashing things
___	___	12.	I have had periods of days, weeks, or months when I couldn't take care of things because I couldn't "get going"
___	___	13.	My sleep is fitful and disturbed
___	___	14.	My judgement is better than it ever was
___	___	15.	I am in just as good physical health as most of my friends
___	___	16.	I prefer to pass by school friends, or people I know but have not seen for a long time, unless they speak to me first
___	___	17.	I am a good mixer
___	___	18.	I have often had to take orders from someone who did not know as much as I did
___	___	19.	I sometimes keep on at a thing until others lose their patience with me
___	___	20.	I wish I could be as happy as others seem to be
___	___	21.	I sometimes tease animals
___	___	22.	I am certainly lacking in self-confidence
___	___	23.	I usually feel that life is worthwhile
___	___	24.	It takes a lot of argument to convince most people of the truth
___	___	25.	I go to church almost every week
___	___	26.	I believe in the second coming of Christ
___	___	27.	I don't seem to care what happens to me
___	___	28.	I am happy most of the time
___	___	29.	I seem to be about as capable and smart as most others around me
___	___	30.	I have never vomited blood or coughed up blood
___	___	31.	I do not worry about catching diseases
___	___	32.	Criticism or scolding hurts me terribly
___	___	33.	I certainly feel useless at times
___	___	34.	At times I feel like picking a fist fight with someone
___	___	35.	Most nights I go to sleep without thoughts or ideas bothering me
___	___	36.	During the past few years I have been well most of the time
___	___	37.	I have never had a fit or convulsion
___	___	38.	I am neither gaining nor losing weight
___	___	39.	I cry easily
___	___	40.	I cannot understand what I read as well as I used to
___	___	41.	I have never felt better in my life than I do now
___	___	42.	My memory seems to be all right
___	___	43.	I am afraid of losing my mind
___	___	44.	I feel weak all over much of the time
___	___	45.	Sometimes, when embarassed, I break out in a sweat which annoys me greatly
___	___	46.	I do not have spells of hay fever or asthma
___	___	47.	I enjoy many different kinds of play and recreation
___	___	48.	I like to flirt
___	___	49.	I have at times stood in the way of people who were trying to do something, not because it amounted to much but because of the principle of the thing
___	___	50.	I brood a great deal
___	___	51.	I dream frequently about things that are best kept to myself
___	___	52.	I believe I am no more nervous than most others
___	___	53.	Sometimes without any reason or even when things are going wrong I feel excitedly happy, "on top of the world"
___	___	54.	I have difficulty in starting to do things
___	___	55.	I sweat very easily even on cool days
___	___	56.	When I leave home I do not worry about whether or not the door is locked and the windows are closed
___	___	57.	I do not blame a person for taking advantage of someone who lays himself open to it
___	___	58.	At times I am full of energy
___	___	59.	Once in a while I laugh at a dirty joke
___	___	60.	I have periods in which I feel unusually cheerful without any special reason

MODIFIED HEADACHE FREQUENCY RECORD (HFR) INCORPORATING DEPRESSIVE ADJECTIVES CHECKLIST (DACL) FORM A

HFR-1 Subject# _____

Date: _____

Please complete this form at the time circled below:

10:00 am 4:00 pm 10:00 pm

1. During my headache, I experienced the following symptoms (check one or more):

__ throbbing or pulsating pain
__ tightness and pressure in my head
__ visual disturbances
__ dull and aching pain
__ nausea or stomach pain
__ vomiting
__ light sensitivity

2. From each of the 2 lists below, circle the number that you feel best describes the severity of your headache pain:

1	2
0 just noticeable	0 bearable
1 mild, weak	1 uncomfortable
2 strong, moderate	2 distracting
3 severe, intense	3 miserable
4 extremely strong	4 intolerable
5 excruciating	5 agonizing

3. I believe that my headache was probably caused by (check one or more):

__ I'm not sure
__ an allergy or allergies
__ hunger
__ fatigue or tiredness
__ alcohol or drug use
__ something I ate/drank
__ stress
__ poor sleep
__ anger
__ depression or sadness
__ anxiety or tension
__ an illness (e.g., cold)

4. When it was at its strongest peak, my headache was (check one):

0 I had no headache
1 very mild; I was hardly even aware of it
2 mild; it could be ignored at times
3 moderate; the pain was noticeably present all the time
4 severe; it was hard to work or concentrate with the pain
5 extremely intense; I couldn't do anything because of it

5. Please rate the severity or intensity of your headache pain along the scale below by circling the appropriate number:

0 1 2 3 4 5

-- mild moderate severe --

6. Did you use any medications to control your headache?

__ no __ yes; identify: _____

7. INSTRUCTIONS: Below you will find words which describe different kinds of moods and feelings. Check the words which describe how you feel right now. Some of words may sound alike, but please check all the words that describe your feelings. Work rapidly and check all of the words which describe how you feel right now.

__ wilted	__ safe	__ gloomy	__ miserable
__ dull	__ gay	__ sad	__ low-spirited
__ unwanted	__ fine	__ down-cast	__ broken-hearted
__ failure	__ afflicted	__ active	__ enthusiastic
__ strong	__ tortured	__ listless	__ sunny
__ destroyed	__ wretched	__ broken	__ light-hearted
__ criticized	__ grieved	__ dreamy	__ oppressed
__ hopeless	__ joyous	__ weary	__ droopy

Appendix D Modified Headache Frequency Record (HFR)

MODIFIED HEADACHE FREQUENCY RECORD (HFR) INCORPORATING DEPRESSIVE ADJECTIVES CHECKLIST (DACL) FORM B

HFR-2 Subject# _____

Date: _____

Please complete this form at the time circled below:

10:00 am 4:00 pm 10:00 pm

1. During my headache, I experienced the following symptoms (check one or more):

__ throbbing or pulsating pain
__ tightness and pressure in my head
__ visual disturbances
__ dull and aching pain
__ nausea or stomach pain
__ vomiting
__ light sensitivity

2. From each of the 2 lists below, circle the number that you feel best describes the severity of your headache pain:

1	2
0 just noticeable	0 bearable
1 mild, weak	1 uncomfortable
2 strong, moderate	2 distracting
3 severe, intense	3 miserable
4 extremely strong	4 intolerable
5 excruciating	5 agonizing

3. I believe that my headache was probably caused by (check one or more):

__ I'm not sure
__ an allergy or allergies
__ hunger
__ fatigue or tiredness
__ alcohol or drug use
__ something I ate/drank
__ stress
__ poor sleep
__ anger
__ depression or sadness
__ anxiety or tension
__ an illness (e.g., cold)

4. When it was at its strongest peak, my headache was (check one):

0 I had no headache
1 very mild; I was hardly even aware of it
2 mild; it could be ignored at times
3 moderate; the pain was noticeably present all the time
4 severe; it was hard to work or concentrate with the pain
5 extremely intense; I couldn't do anything because of it

5. Please rate the severity or intensity of your headache pain along the scale below by circling the appropriate number:

0 1 2 3 4 5

-- mild moderate severe --

6. Did you use any medications to control your headache?

__ no __ yes; identify: _____

7. INSTRUCTIONS: Below you will find words which describe different kinds of moods and feelings. Check the words which describe how you feel right now. Some of words may sound alike, but please check all the words that describe your feelings. Work rapidly and check all of the words which describe how you feel right now.

__ cheerless	__ animated	__ blue	__ lost
__ dejected	__ healthy	__ bad	__ discouraged
__ despondent	__ free	__ uneasy	__ despairing
__ peaceful	__ grim	__ whole	__ distressed
__ bouyant	__ weak	__ optimistic	__ tormented
__ low	__ deserted	__ burdened	__ wonderful
__ crushed	__ somber	__ joyless	__ interested
__ lucky	__ chained	__ crestfallen	__ pessimistic

MODIFIED HEADACHE FREQUENCY RECORD (HFR) INCORPORATING DEPRESSIVE ADJECTIVES CHECKLIST (DACL) FORM C

HFR-3 Subject# _____

Date: _____

Please complete this form at the time circled below:

10:00 am 4:00 pm 10:00 pm

1. During my headache, I experienced the following symptoms (check one or more):

__ throbbing or pulsating pain
__ tightness and pressure in my head
__ visual disturbances
__ dull and aching pain
__ nausea or stomach pain
__ vomiting
__ light sensitivity

2. From each of the 2 lists below, circle the number that you feel best describes the severity of your headache pain:

1	2
0 just noticeable	0 bearable
1 mild, weak	1 uncomfortable
2 strong, moderate	2 distracting
3 severe, intense	3 miserable
4 extremely strong	4 intolerable
5 excruciating	5 agonizing

3. I believe that my headache was probably caused by (check one or more):

__ I'm not sure
__ an allergy or allergies
__ hunger
__ fatigue or tiredness
__ alcohol or drug use
__ something I ate/drank
__ stress
__ poor sleep
__ anger
__ depression or sadness
__ anxiety or tension
__ an illness (e.g., cold)

4. When it was at its strongest peak, my headache was (check one):

0 I had no headache
1 very mild; I was hardly even aware of it
2 mild; it could be ignored at times
3 moderate; the pain was noticeably present all the time
4 severe; it was hard to work or concentrate with the pain
5 extremely intense; I couldn't do anything because of it

5. Please rate the severity or intensity of your headache pain along the scale below by circling the appropriate number:

0 1 2 3 4 5
+---+---+---+---+---+

-- mild moderate severe --

6. Did you use any medications to control your headache?

__ no __ yes; identify: _____

7. INSTRUCTIONS: Below you will find words which describe different kinds of moods and feelings. Check the words which describe how you feel right now. Some of words may sound alike, but please check all the words that describe your feelings. Work rapidly and check all of the words which describe how you feel right now.

__ lively	__ unfeeling	__ alone	__ downhearted
__ unhappy	__ alive	__ terrible	__ poor
__ forlorn	__ alert	__ exhausted	__ heartsick
__ bright	__ glum	__ desolate	__ composed
__ clean	__ moody	__ pleased	__ dispirited
__ dead	__ bleak	__ light	__ sorrowful
__ morbid	__ grey	__ mashed	__ heavy-hearted
__ hopeful	__ unlucky	__ melancholy	__ easy-going

RESEARCH PARTICIPATION ATTENDANCE SHEET

Experiment _____ Dates covered by this sheet _____
Name from Sign-up Booklets

Researcher(s)_____ Faculty Advisor _____

Student's Name PRINTED	Student's Signature	Student No.

TO BE FILED WITH STUDENT ADVISOR IN P203 AT CONCLUSION OF STUDY.

Appendix E Study 1: Forms and Instructions

SUBJECT INSTRUCTIONS: STUDY 1

Instructions to Subjects in Study 1

Hello, I'm Robert Woods. I would like to thank you all for showing interest in this study. What I would like you to do is to complete the questionnaire in front of you. It contains 48 items, and should take you no more than 60 minutes at the most to complete. Please read the instructions carefully before starting. Answer each item carefully, and do not go back to any item once you have completed it, unless you are certain you have made an error. When you have completed the questionnaire, please leave the booklet on your desk. You may then leave if you wish. You'll notice in the instructions that I will be conducting a second study on headache, and if you think you might be interested, then please print your name and telephone number on the front sheet, and detach it from the rest of the questionnaire. Are there any questions? Please note that your responses to all items in this questionnaire will be kept completely confidential.

INSTRUCTIONS FOR COMPLETION OF HHQ/BDI PACKAGE

HH/DI Subject# _____ Date: _____

INSTRUCTIONS: The attached questionnaire is divided into two sections, and consists of a total of 48 (26+22) items. Please read the instructions for each section carefully before answering the items, and be sure to answer all of the items to the best of your ability. Some of the items may seem personal or unusual to you, but please attempt to respond honestly to each one. The use of the pre-assigned subject number (above) will ensure that your answers are kept confidential.

If after having completed the questionnaire you feel that you would be interested in participating further in the study for more experimental credits, then please print your name and telephone number in the space provided below. Do not sign unless you think that you would like to participate further. Then, detach this sheet from the others and return it to me separately. If you are eligible to participate in the second part of the study, then I will contact you at the number you leave with further details. The second part would be more involved, but similarly would involve completing questionnaires.

Please turn this page and complete the questionnaire. You will receive a total of 1experimental credit for your participation in this part of the study.

I would like to participate in the second part of the study, and agree to being contacted by telephone by the experimenter. I understand that all questionnaire materials will be kept confidential, and are coded by subject number only.

Name (please print): _____

Telephone number: _____

Best time to reach me at this number is: _____

_____

SUBJECT INSTRUCTIONS: STUDY 2

Instructions to Subjects in Study 2

Hello, I'm pleased to see you all here. I would like to thank you once again for your interest in this study. This part of the study will begin in much the same way as the last. What I'd like you to do to begin is complete the questionnaire in front of you. This contains 60 true/false items, and should take no more than an hour to complete. Following this, at _____ (time), we'll take a 15-minute break. If you finish quickly, however, please feel free to start the break earlier. After the break, at _____ (time) I want to explain to you what you'll be doing during the rest of the study. As I mentioned earlier, it will also involve completing questionnaires, but the actual procedure will be a little more complicated than it has been up to now. Although I certainly hope you'll want to complete the entire study, you are of course not obligated to do so. If you do decide during today's break not to continue, please let me know. You will still receive 1 experimental credit for completing the questionnaire, and 5 more if you decide to complete the entire study. Again, all of your responses to the questionnaire items will be kept completely confidential. Are there any questions? Let's begin. Please go through the 60 items sequentially, and don't return to earlier items.

(MMPI-D administered, then 15 minute break)

Welcome back. I'm glad you've decided to continue in the study. This part will involve what's called "home self-monitoring". In other words, I'll be giving you materials to complete at home at specified times, 3 times a day for 28 consecutive days beginning on _____ (date). What you see on top of the file folder in front of you is a single day's questionnaire package. There are 4 forms in each package, and 7 packages in each of 4 manila envelopes in the file folder. The package in front of you is for "practice".

First, let's go over the instructions on the inside cover of your file folder.

(turn on overhead diagram of HFR, and read "Instructions for HFR-DACL package)

Keep these instructions handy for reference, and make sure you fully understand them. In fact, keep all of the materials in this folder. Each of the 4 manila envelopes in the folder contains 1 week's worth of questionnaires. At the end of each week, please return these in the envelope to _____ (departmental mailbox). During the first week of self-monitoring, I'll be calling you at home once a day to see how things are going and answer any questions you have about the procedure. During the second to fourth weeks, I'll be calling you twice a week.

If you complete the entire self-monitoring phase, you'll receive a total of 5 experimental credits. The longer you participate, the more credits you can receive. You'll receive 1 per week for each of the first 3 weeks you complete, and 2 for completing the final week of self-monitoring.

If you wish, I will mail to your home address a brief letter explaining the purpose of the study a few weeks after you've completed. In order to receive this, please complete and sign the address card included in the manila envelope for the last week of the study, and return it with the questionnaire materials for that week.

Appendix F Study 2: Forms and Instructions

INSTRUCTIONS FOR MODIFIED HEADACHE FREQUENCY RECORD

HFR Subject# _____

INSTRUCTIONS: The enclosed questionnaire materials are to be completed as follows: Each page contains 7 items, having to do with headache severity, symptoms, and any medications used to control headache pain. Please begin by recording the date in the space provided. Below this, you will note that one of 3 times is circled: 10:00 am, 4:00 pm, or 10:00 pm. Complete the form at the time specified. You are asked to complete 3 forms per day over the next 28 days. Please do these at the times specified; do not attempt to complete forms by memory. Your folder contains a total of 84 forms. You will find that these become very easy to complete with practice, and will never take you more than a few minutes to do.

If you are not experiencing headache at a particular time, then completing the form will be very easy. In such cases, you need only note in item 4 that you had no headache at the specified time. Even if you do not have a headache, please complete item 7! This step is crucial. So, no matter whether you experience a headache or not, please complete item 7 at the specified time, 3 times a day.

I will be contacting you periodically by telephone to see how you're doing with the forms, and to answer any questions you may have about the procedure. You will receive a total of 5 experimental credits for full participation in this part of the study, at a rate of 1 credit per week for the first 3 weeks, with 2 credits given for completion of the fourth and final week of the study.

TELEPHONE CONTACTS BEFORE AND DURING STUDY 2

Telephone Contact of Subjects for Study 2

Hello (Mr./Ms.) _____, this is Robert Woods calling, from the Psychology Department. I'm calling about the headache study that you've expressed interest in. What the study will involve will mainly be completing a small number of brief questionnaires daily at home over 28 consecutive days. Each day's work will never take you more than 20-30 minutes to complete, and you will receive a total of six experimental credits if you successfully complete the entire study. If you are interested in participating I must first get your answer to the following question: Are you currently undergoing any medical or other supervised treatment for your headaches?
(if yes then)
I'm very sorry, but I can only ask people to participate who are not being treated for their headaches. Thank you very much for your time and cooperation.
(if no then)
I will be meeting with all of the subjects as a group on _____ (date) at _____ (time), in _____ (location). Would you be able to make this time? Thank, I'll see you then.

Telephone Contact of Subects During Self-Monitoring

Hello (Mr./Ms.) _____, this is Robert Woods. I'm calling to see how things have been going with the self-monitoring. Do you have any questions about the procedure? Please remember to follow the instructions carefully. I'll talk to you again _____ (time).

POST-STUDY DEBRIEFING LETTER

Dear (Mr./Ms.) _____,

Let me first take this opportunity to thank you once again for your cooperation in my doctoral dissertation research project on headache. As promised, I am writing to you to briefly describe what the study was all about.

Basically, the study was designed to compare two competing theories in the headache literature. Many researchers have reported a correlation, in some people, between headache and moods, particularly depressed mood. One theory says that headache is a physical expression of depression in these people. The other theory, based on observations of chronic pain patients, says that depression is simply a natural consequence of living with the chronic pain of recurrent headache. The outcome of my study has certain practical implications. If the second theory is true, then the study really doesn't tell us anything new. But, if the first theory is true, then it might be possible to help people learn the warning signs of headache by noting changes in their moods, and taking preventive measures before headache begins.

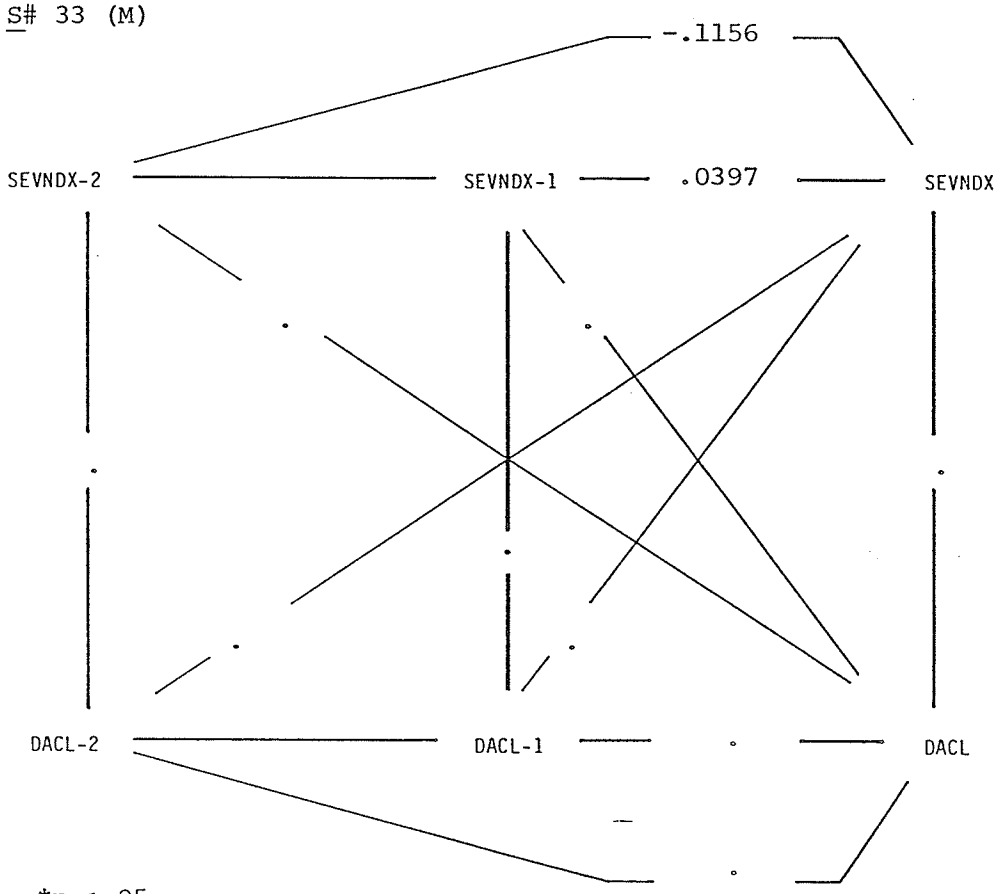
If you are still troubled by headache, I would suggest that you first see your family doctor for a complete physical examination, just to make sure there's no medical problem. You might then consider contacting someone who specializes in the behavioural treatment of headache or other pain using relaxation or biofeedback. These approaches have been shown to be safe and effective treatments. There are professionals here at the university at the Psychological Service Centre and Department of Social Work, and at both St. Boniface Hospital and the Rehabilitation Hospital at the Health Sciences Centre.

Thank you once again for your time and cooperation.

Sincerely,

Robert B. Woods

S# 33 (M)



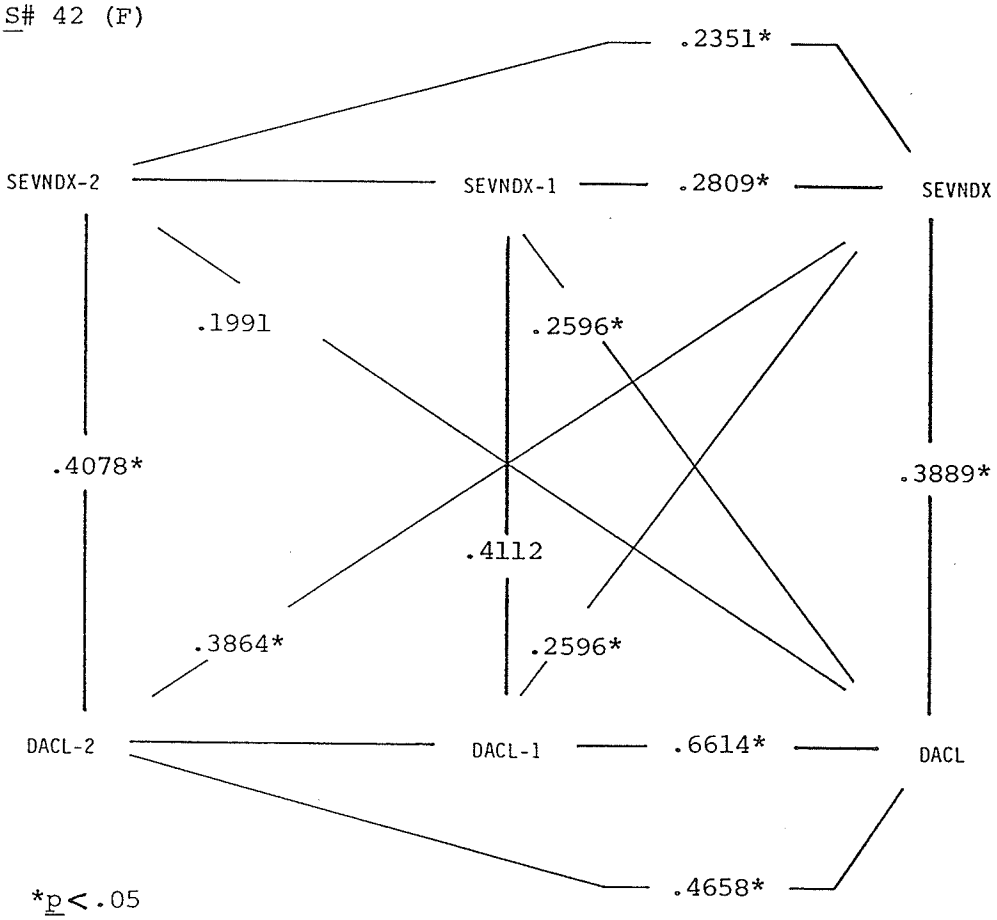
*p < .05

. Correlation could not be computed

	M	SD
DACL	10.177	1.364
SEVNDX	.	.

Appendix G Individual Data on Headache-Mood Association

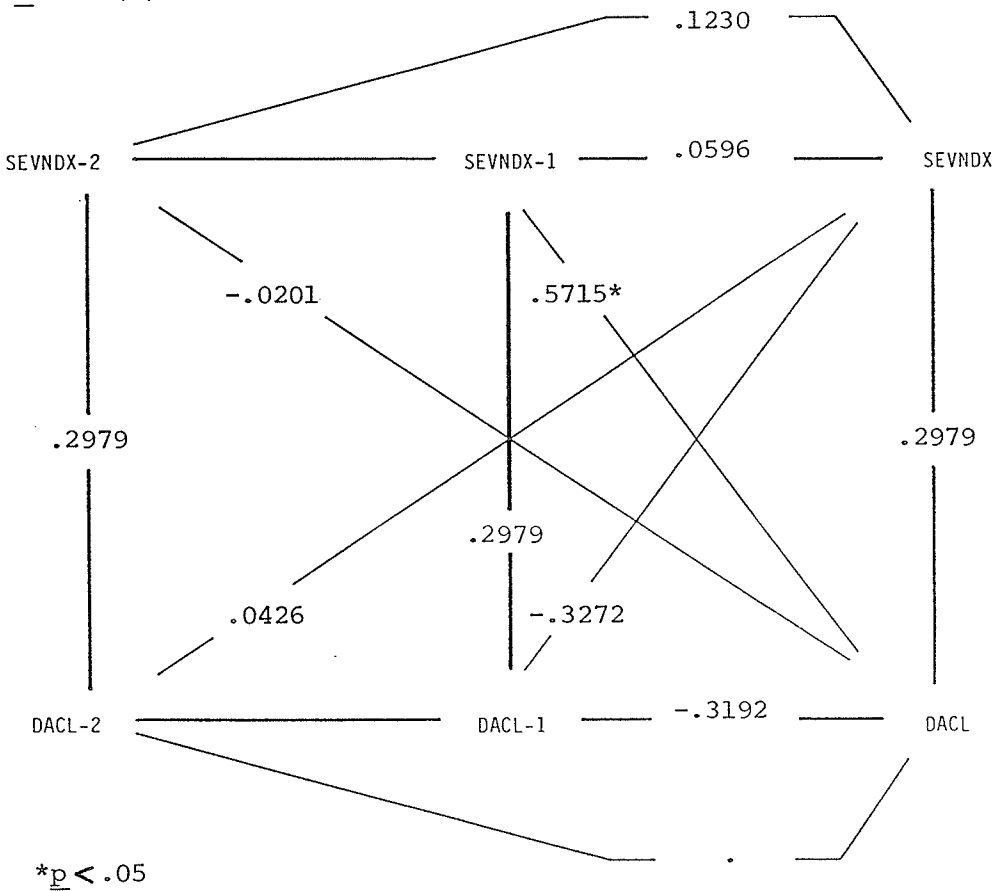
S# 42 (F)



*p < .05

	M	SD
DACL	4.263	1.741
SEVNDX	1.411	0.634

S# 46 (F)

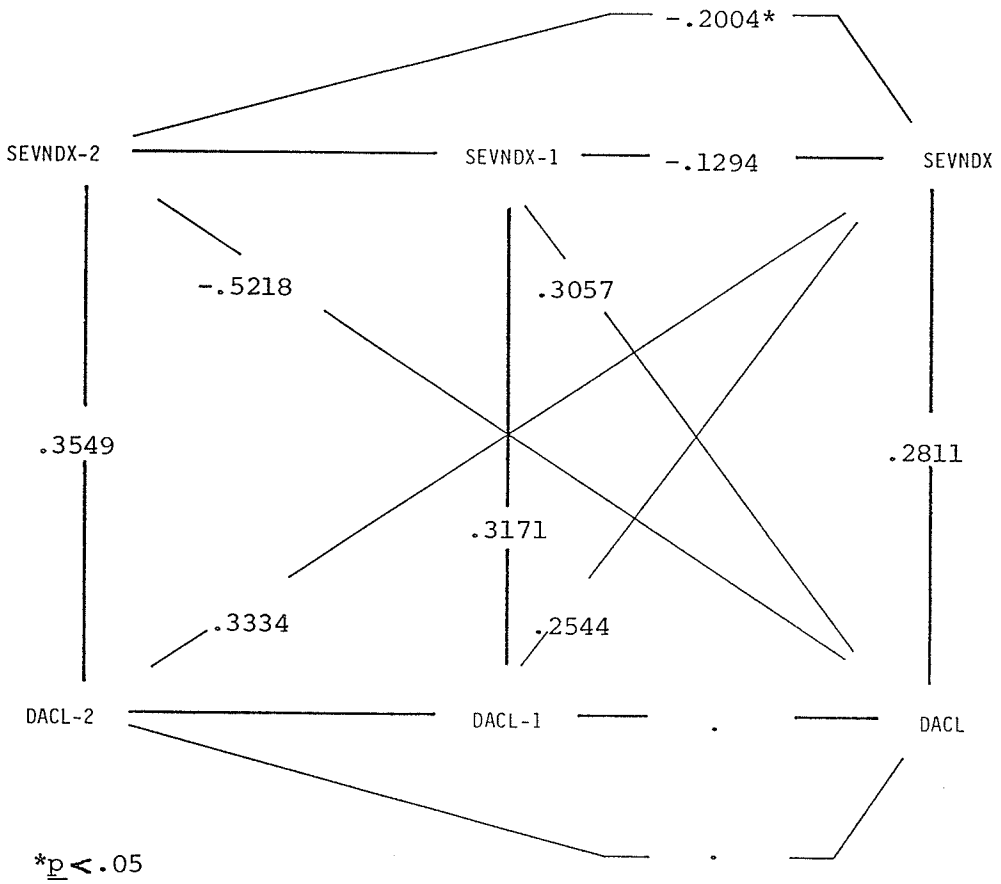


*p < .05

. Correlation could not be computed

	M	SD
DACL	8.798	3.722
SEVNDX	1.142	0.636

S# 56 (F)

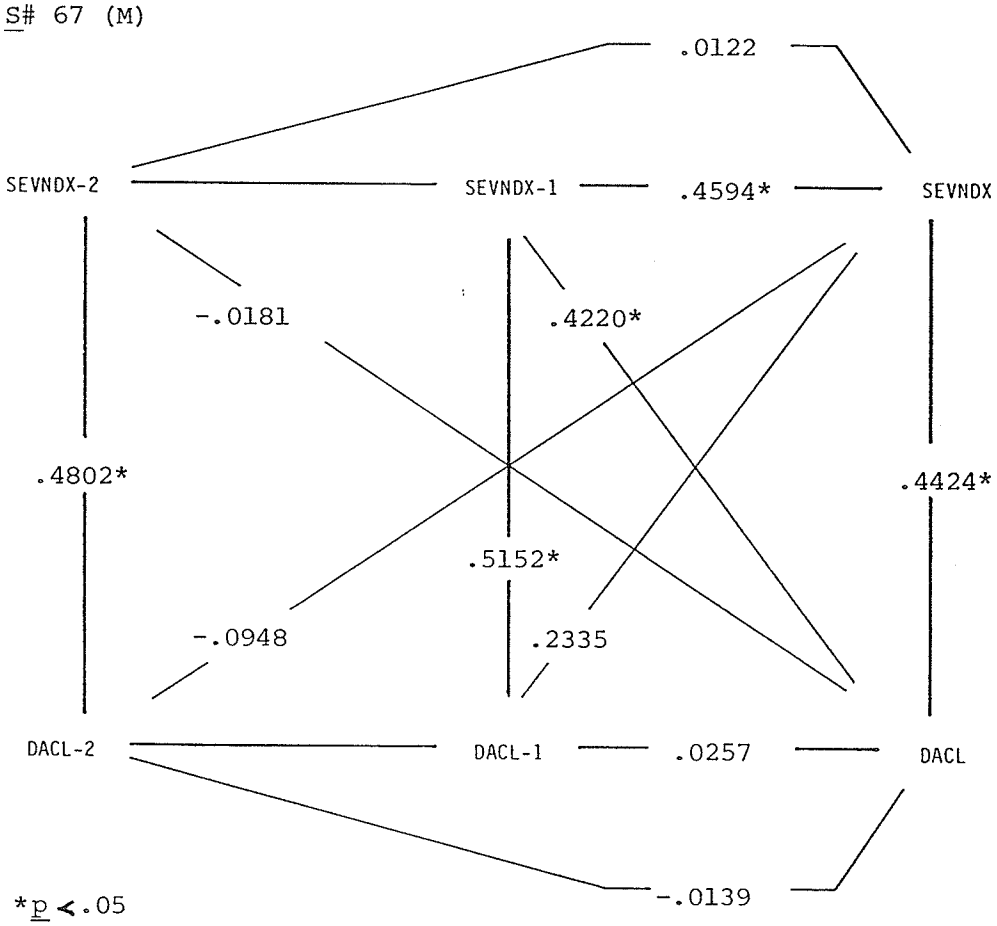


*p < .05

. Correlation could not be computed

	M	SD
DACL	7.397	4.708
SEVNDX	1.304	0.676

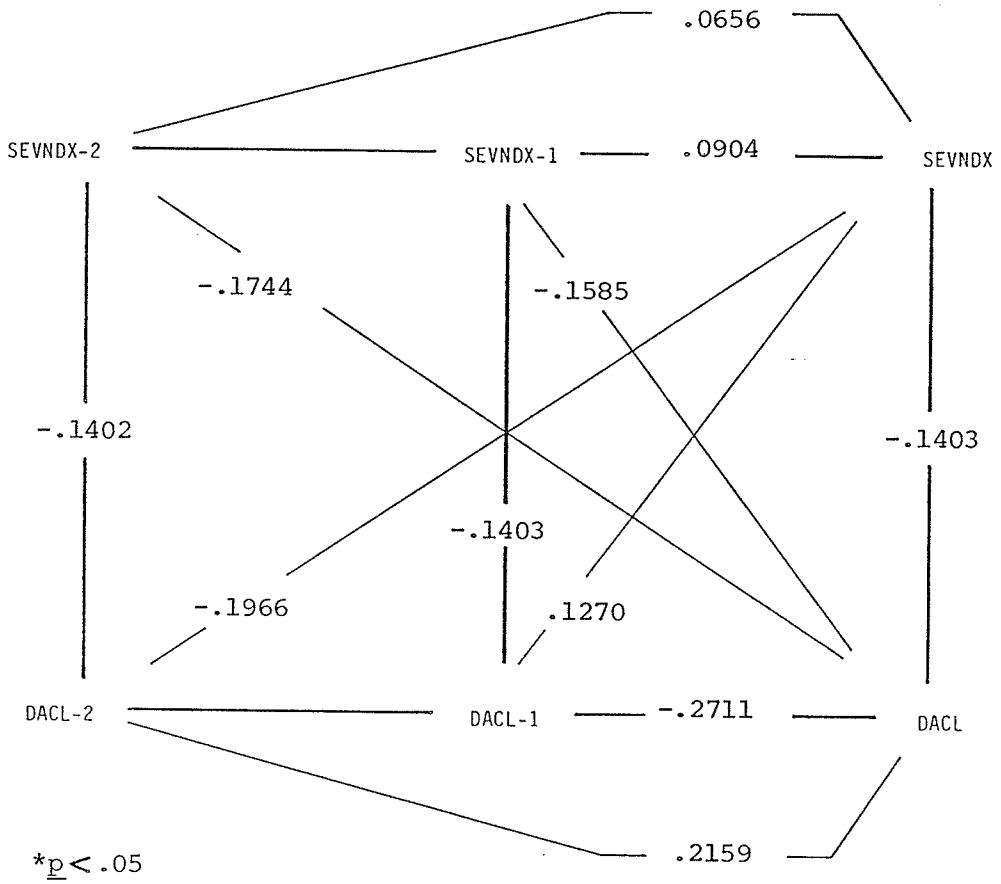
S# 67 (M)



*p < .05

	M	SD
DACL	9.013	6.248
SEVNDX	1.063	0.883

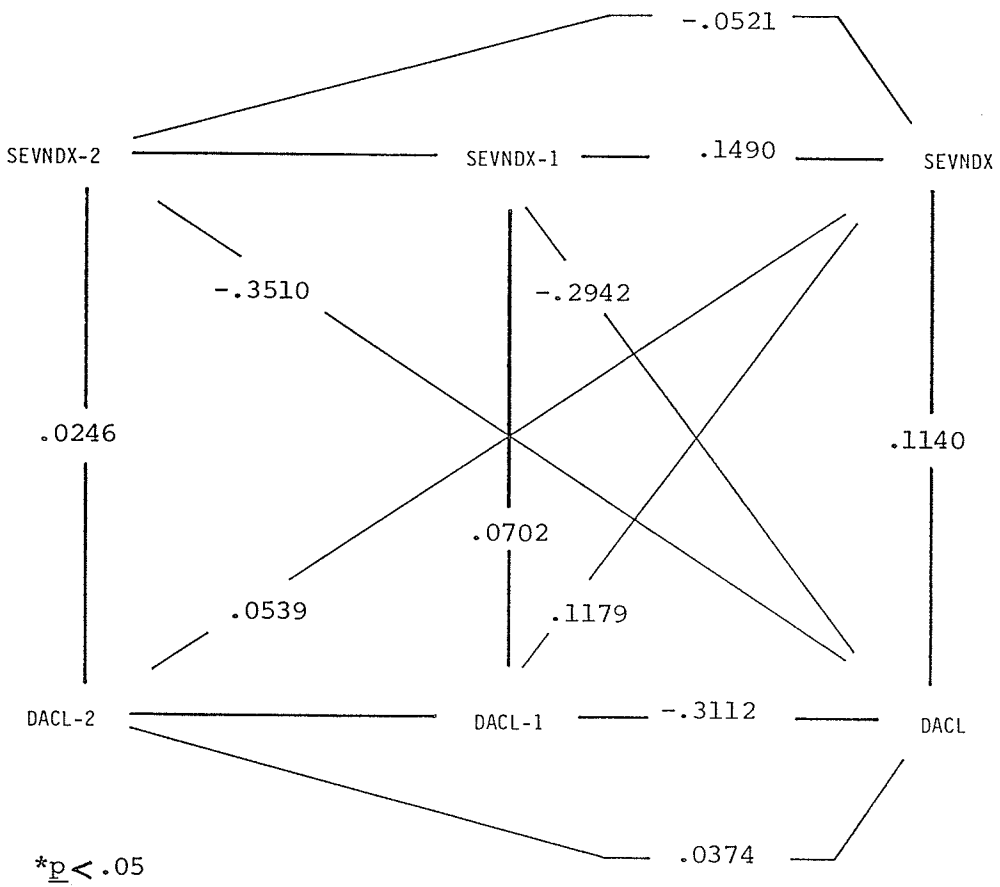
S# 80 (F)



*p<.05

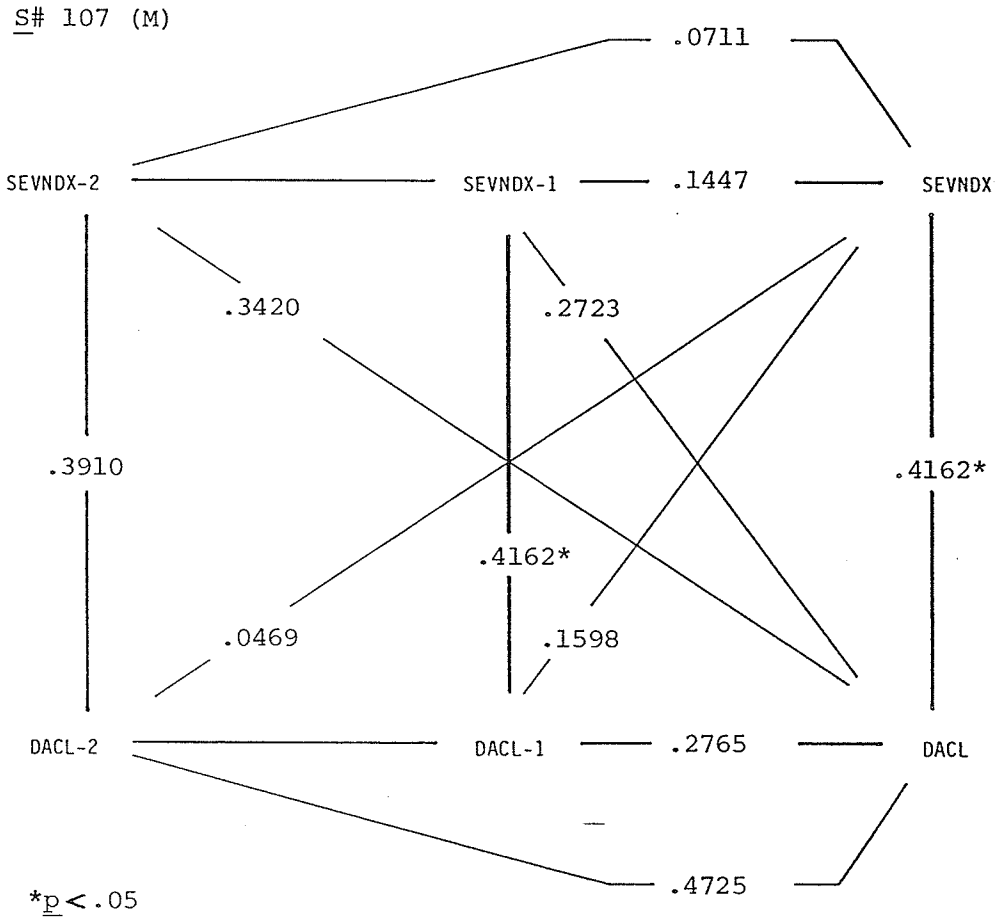
	M	SD
DACL	8.863	2.941
SEVNDX	1.170	0.876

S# 81 (M)



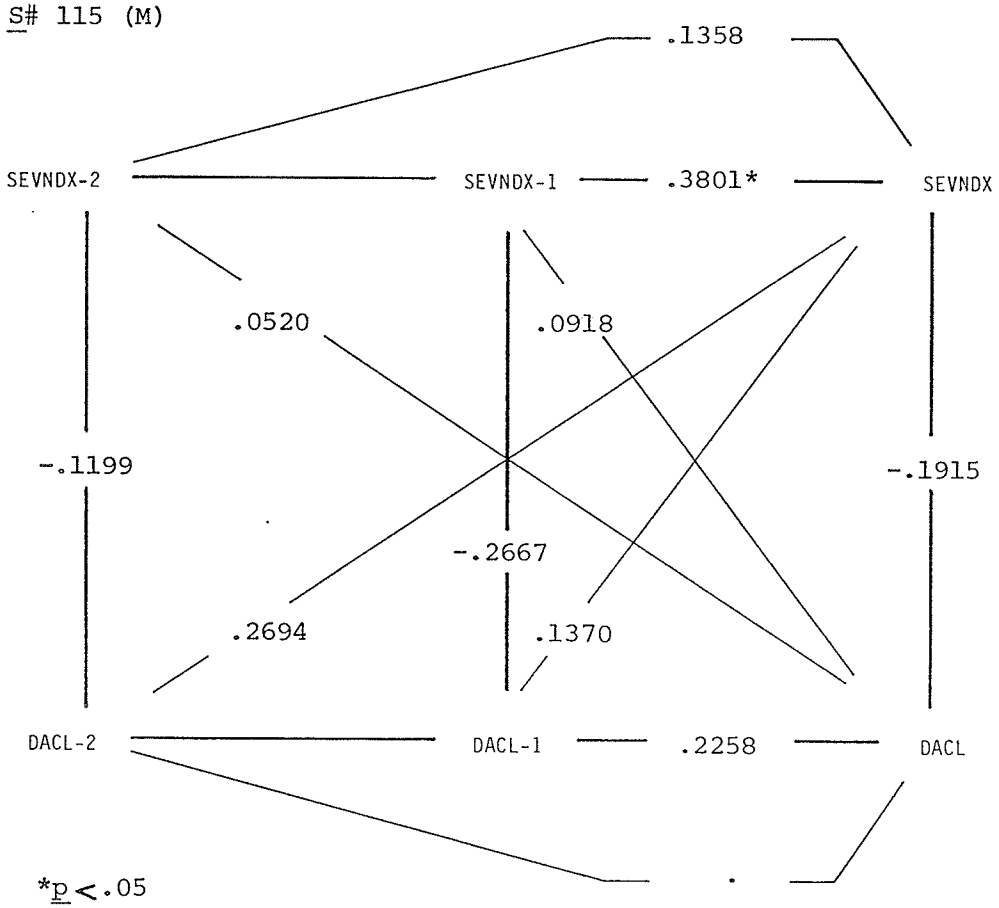
*p < .05

	M	SD
DACL	11.679	2.396
SEVNDX	1.086	0.711



	M	SD
DACL	8.272	2.555
SEVNDX	2.471	1.896

S# 115 (M)

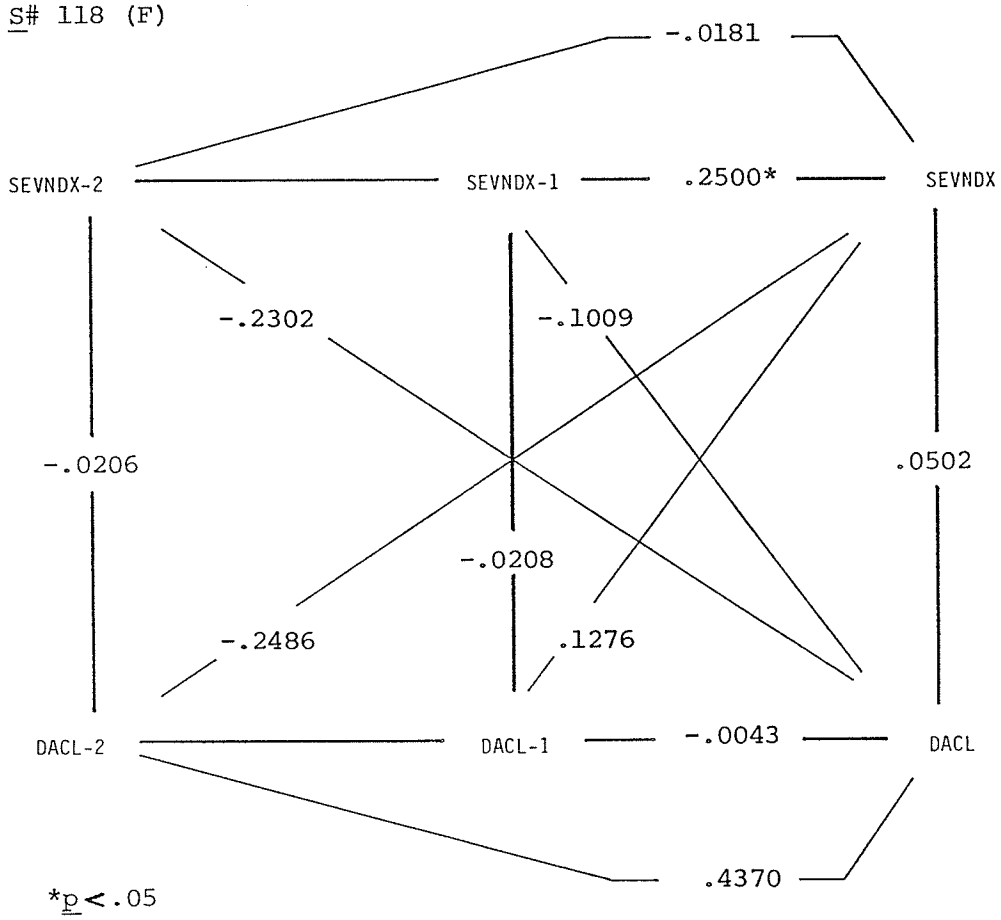


*p < .05

. Correlation could not be computed

	M	SD
DACL	9.417	6.634
SEVNDX	1.021	0.793

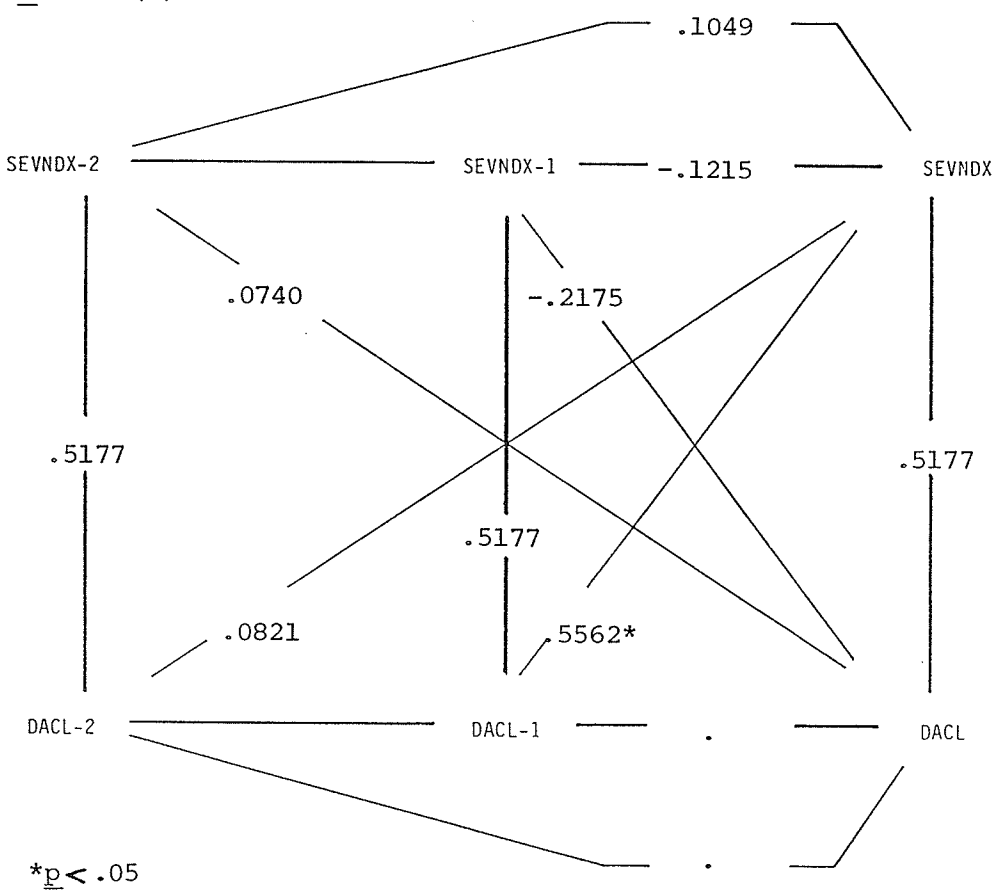
S# 118 (F)



*p < .05

	M	SD
DACL	9.955	3.653
SEVNDX	3.524	2.623

S# 135 (M)

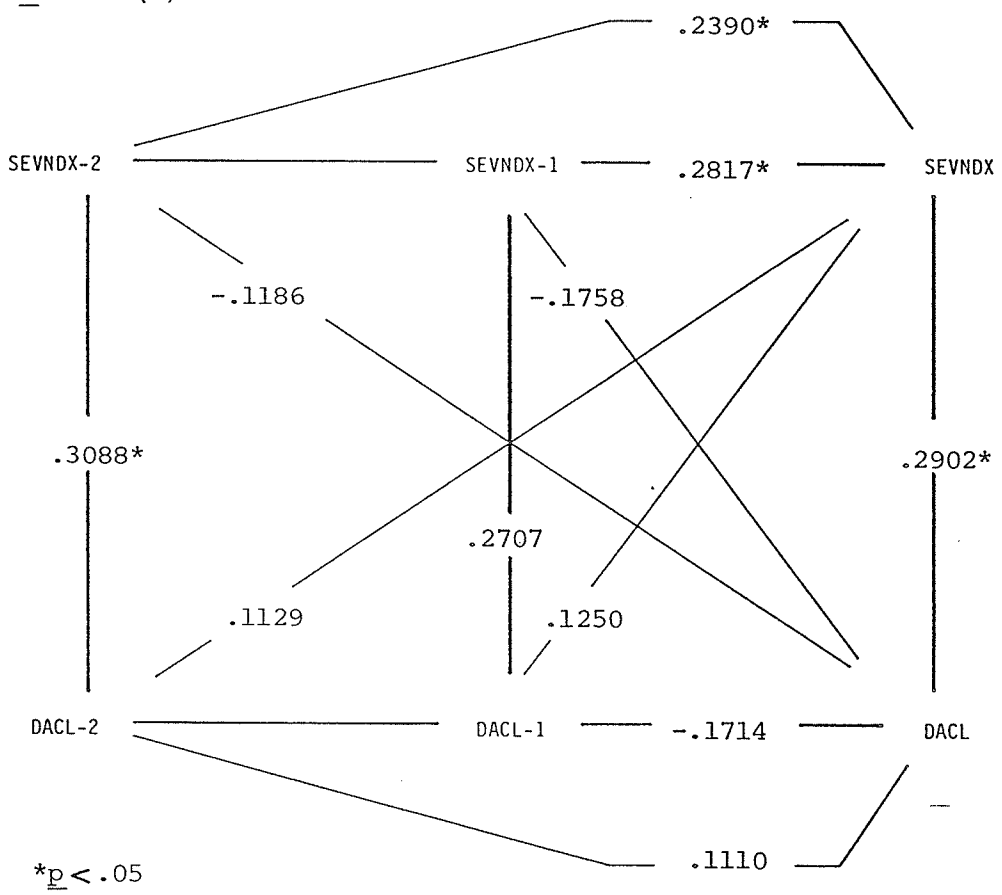


*p < .05

. Correlation could not be computed

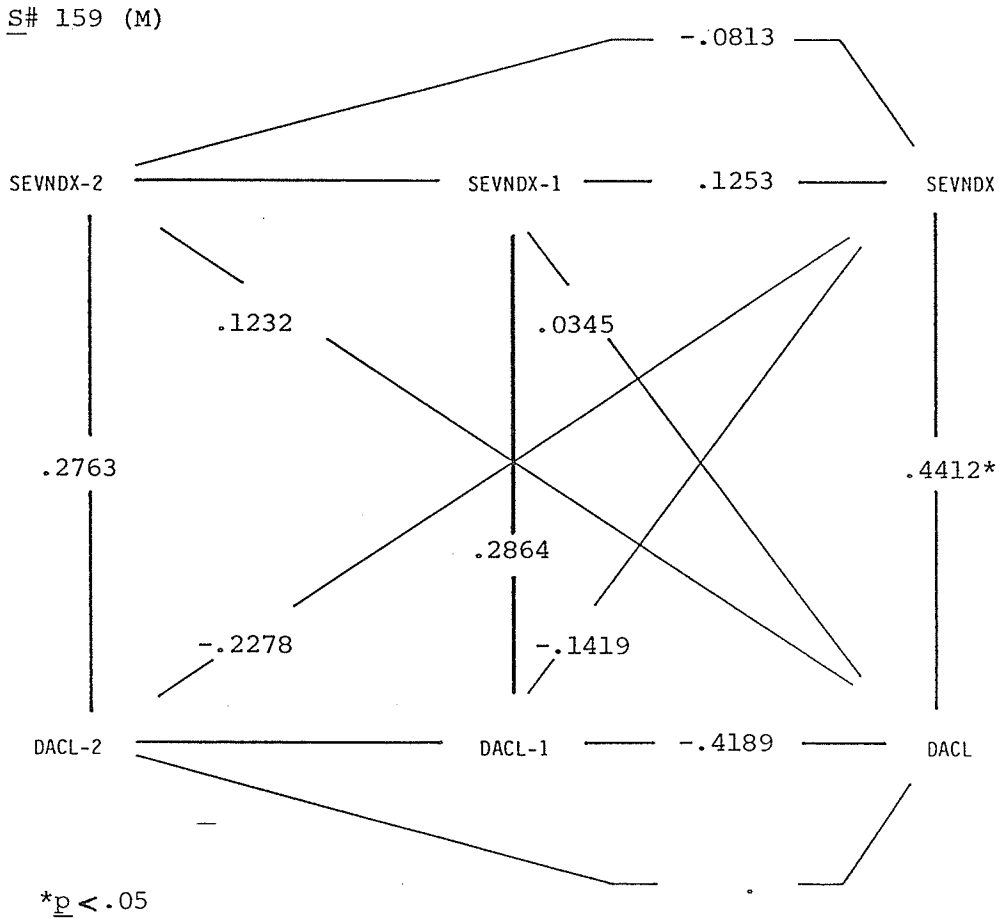
	M	SD
DACL	9.202	2.104
SEVNDX	1.181	0.511

S# 146 (F)



*p < .05

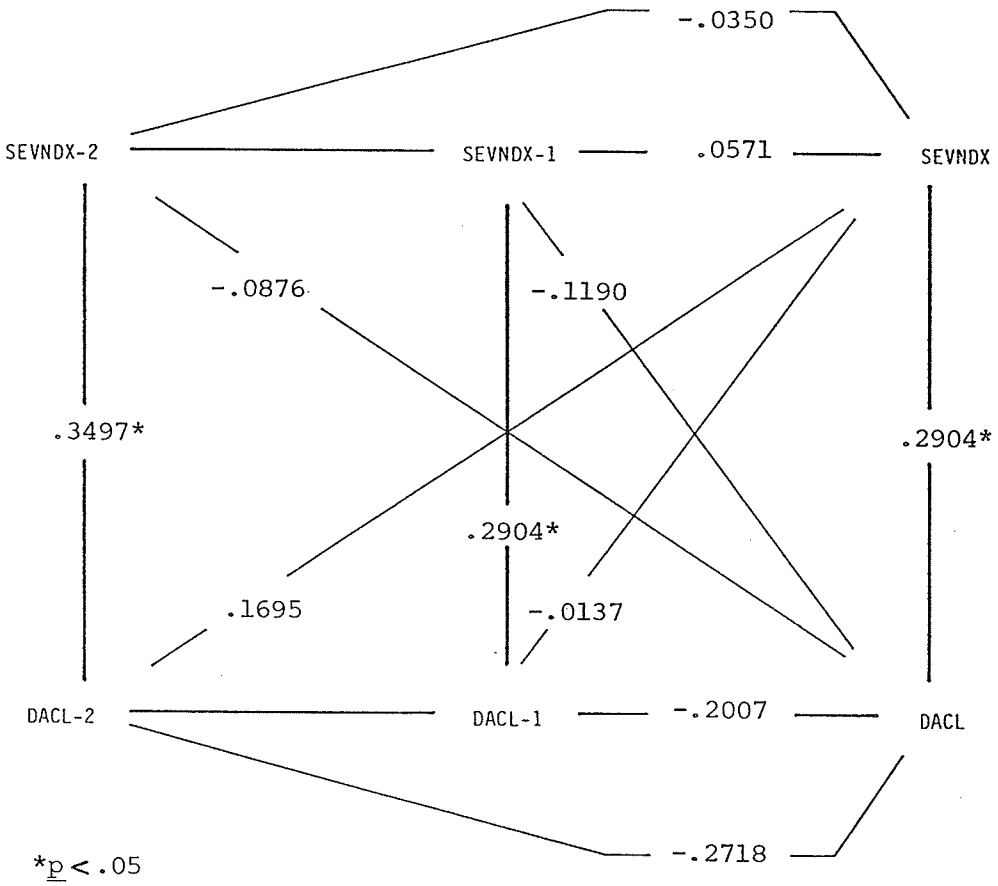
	M	SD
DACL	10.000	3.953
SEVNDX	2.090	1.246



. Correlation could not be computed

	M	SD
DACL	10.024	2.513
SEVNDX	1.048	0.712

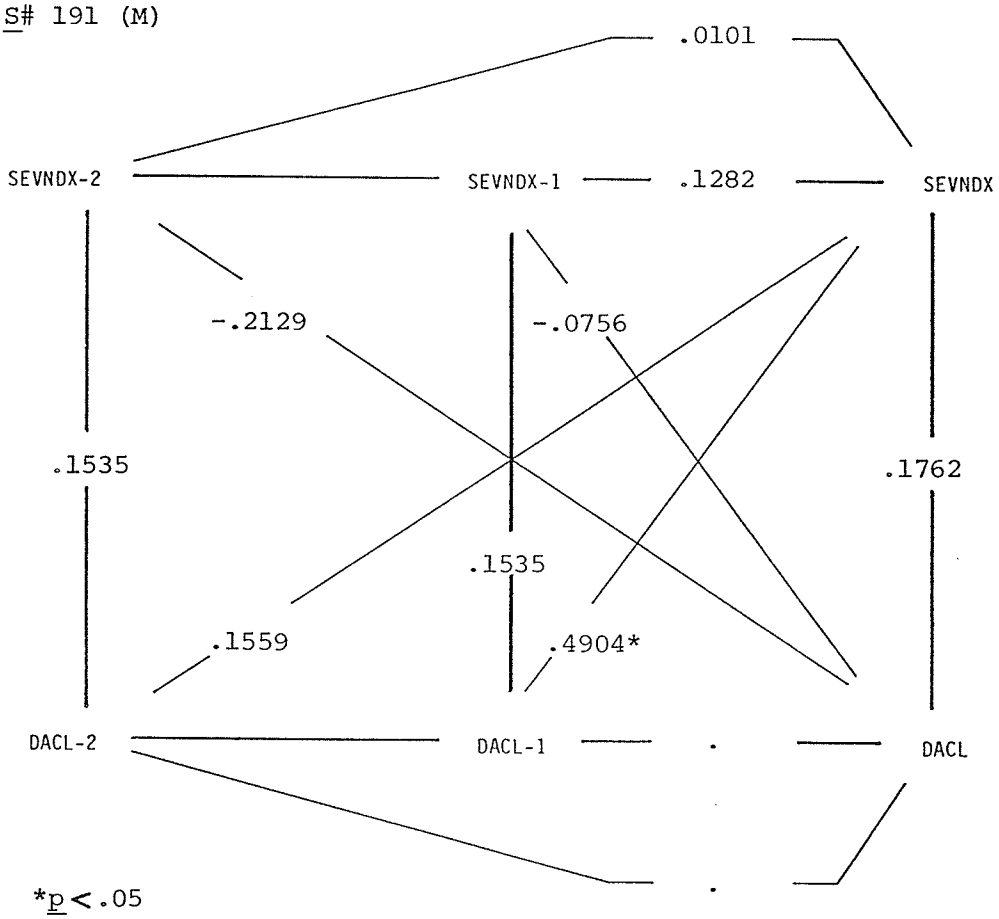
S# 162 (F)



*p < .05

	M	SD
DACL	9.643	4.193
SEVNDX	0.957	0.637

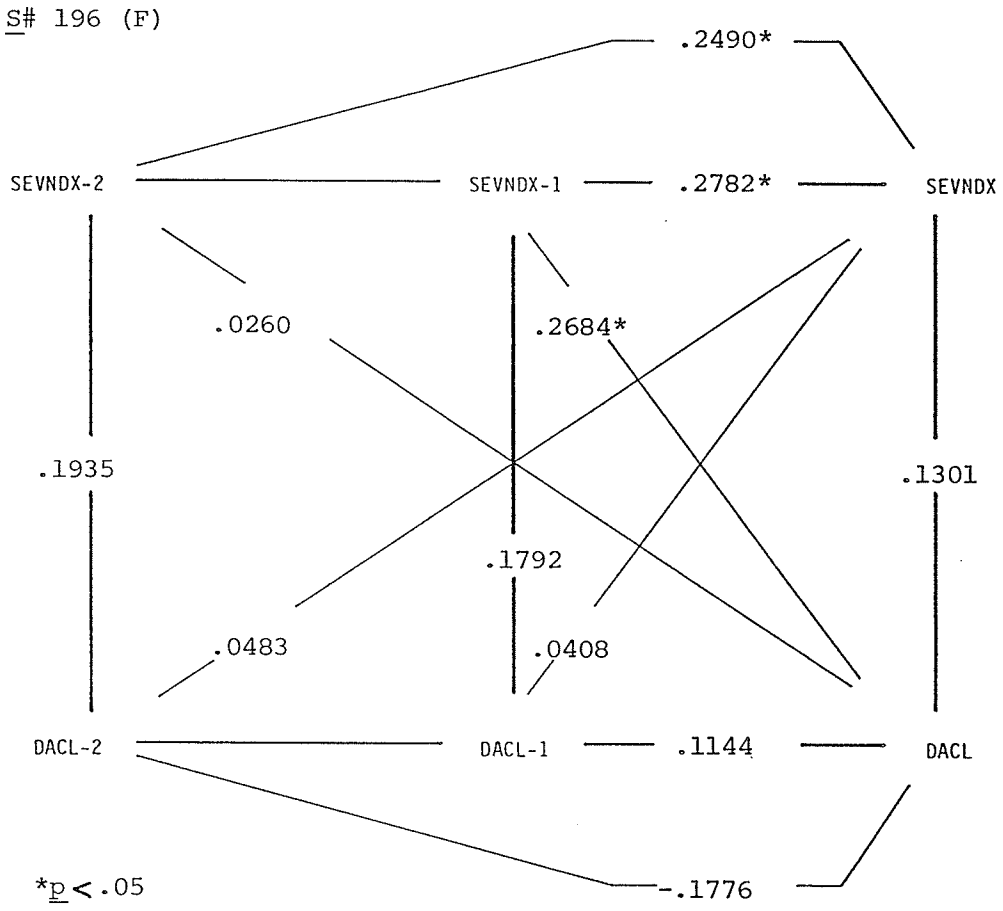
S# 191 (M)



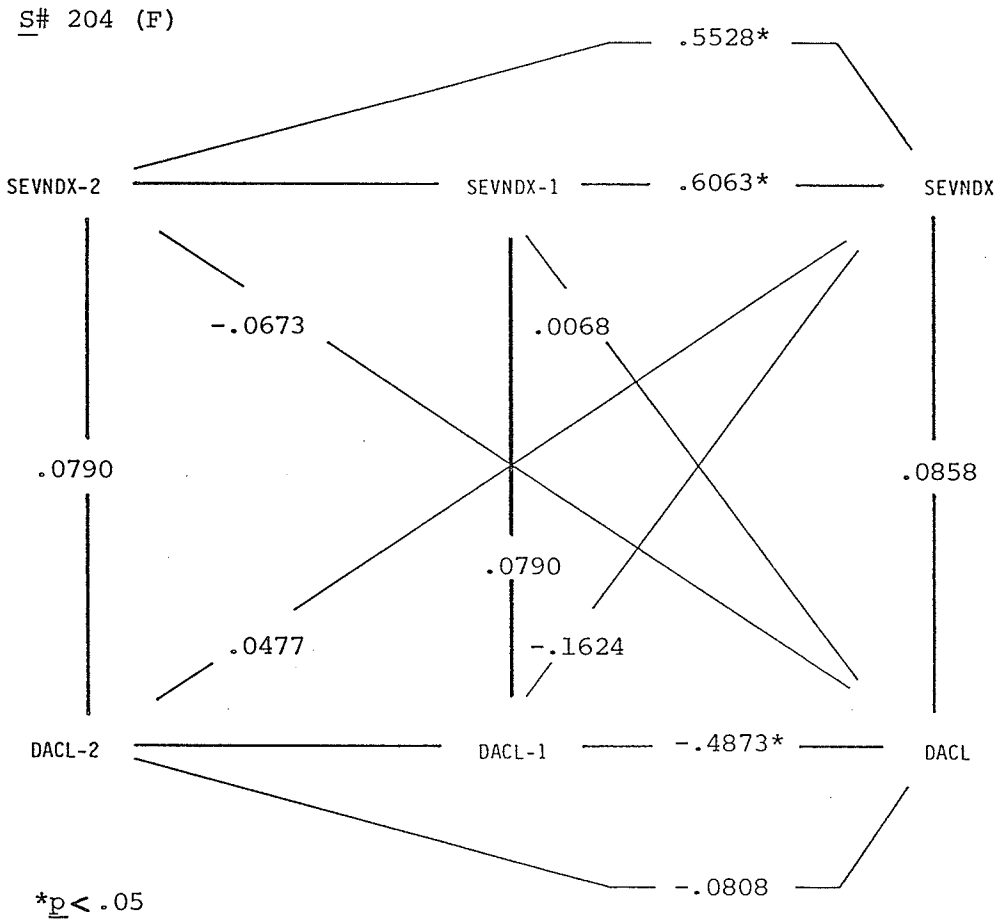
*p < .05

. Correlation could not be computed

	M	SD
DACL	11.012	6.300
SEVNDX	3.000	1.954

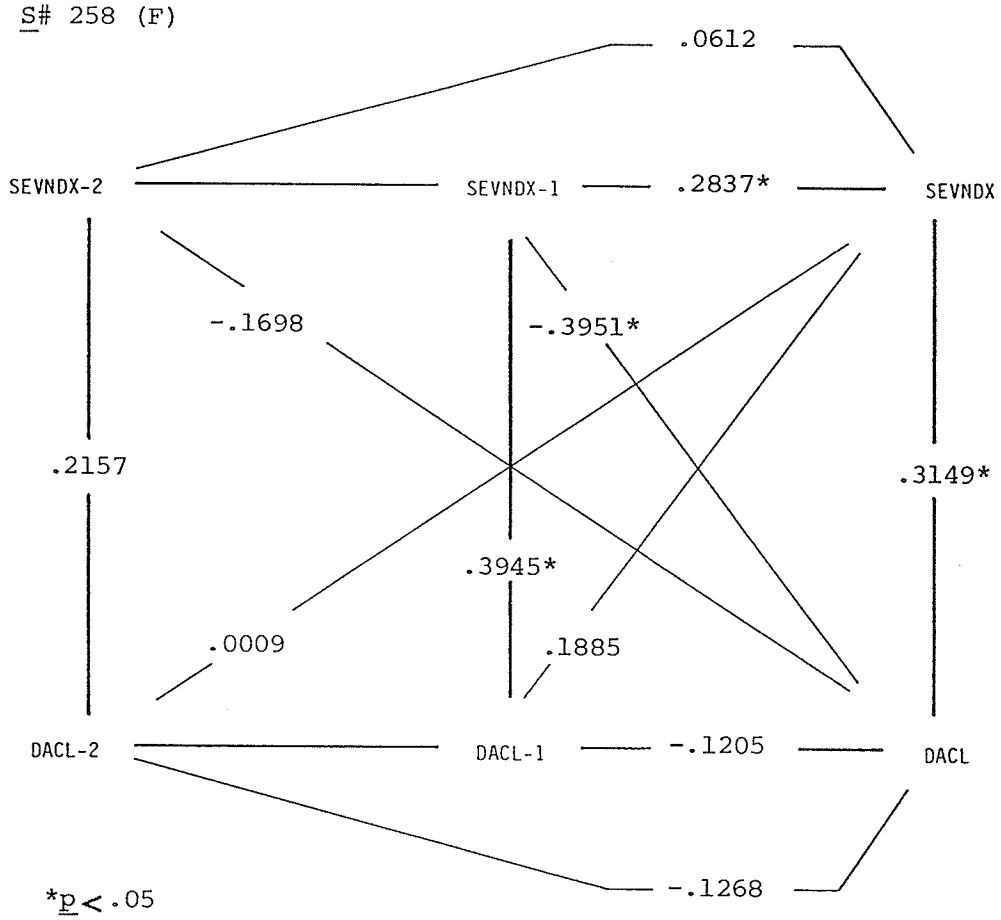


	M	SD
DACL	8.936	2.362
SEVNDX	2.477	2.325



	M	SD
DACL	6.605	3.347
SEVNDX	1.130	0.828

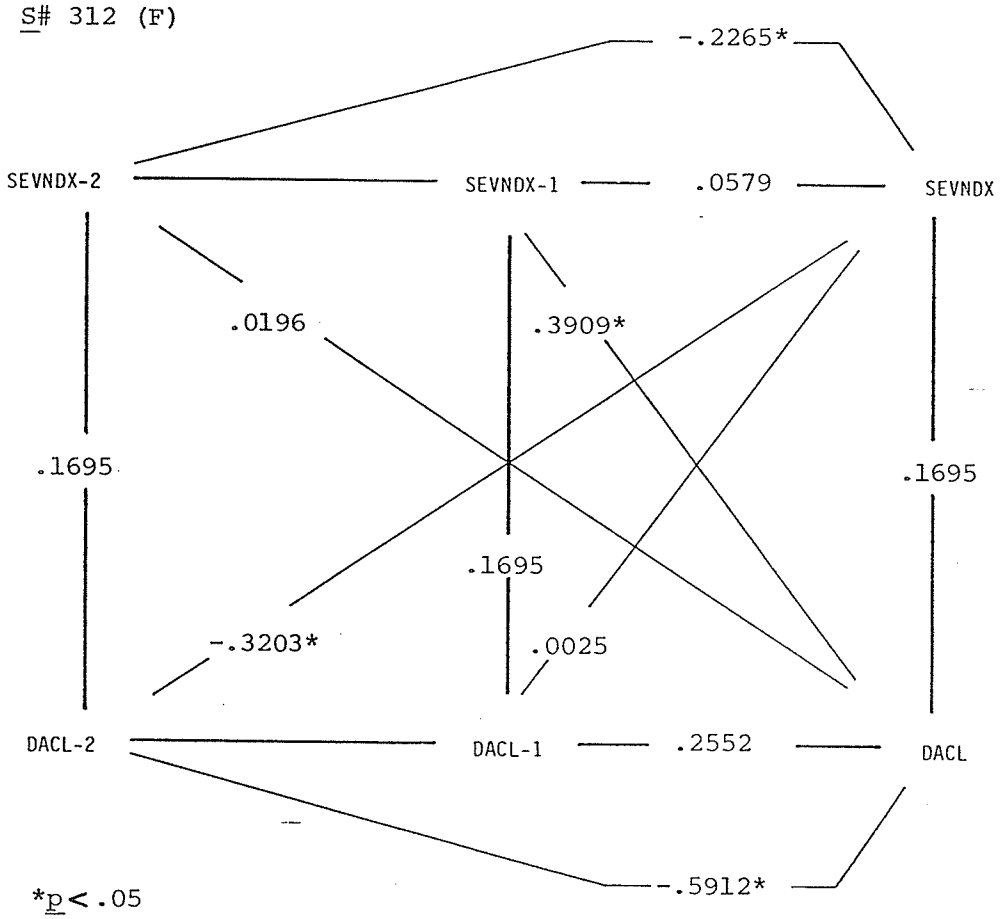
S# 258 (F)



*p < .05

	M	SD
DACL	9.941	2.283
SEVNDX	1.013	0.753

S# 312 (F)



*p < .05

	M	SD
DACL	13.305	7.277
SEVNDX	1.998	1.270