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PSYCHOLOGICAL AND SLEEP QUALITY DIFFERENCES BETWEEN CHRONIC DAILY HEADACHE AND TEMPOROMANDIBULAR DISORDERS PATIENTS

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ABSTRACT OF THESIS

PSYCHOLOGICAL AND SLEEP QUALITY DIFFERENCES BETWEEN CHRONIC DAILY HEADACHE AND TEMPOROMANDIBULAR DISORDERS PATIENTS

The aim of this study was to investigate whether chronic daily headache (CDH) and temporomandibular disorders (TMD) patients present with different psychological and sleep quality characteristics. Sixty seven patients diagnosed with CDH according to Silberstein et al.'s classification criteria were matched by age, sex, pain intensity, and pain duration with 67 patients who had a primary diagnosis of myofascial pain (MP), and 67 patients with a primary diagnosis of TMJ intracapsular pain (IC) according to the Research Diagnostic Criteria for TMD. The CDH group was comprised of three mutually exclusive diagnostic groups, that is transformed migraine (n=35), chronic tension-type headache (n=26), and "other CDH" (n=6). All CDH sub-groups showed similar psychological and sleep quality profiles. All patients completed a battery of psychological and sleep quality questionnaires. The CDH and MP groups revealed higher levels of psychological distress than the IC group on most psychological domains. The MP group also revealed numerically higher levels of psychological distress in most psychological domains than the CDH group, although these differences where not statistically significant. We did not find statistically significant differences between the three groups on post traumatic stress symptoms either. Sleep quality was significantly worse in the MP group than in the CDH and IC groups. These results are discussed in the context of multimodal patient evaluation and treatments that are necessary for successful clinical management.

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THESIS

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The Graduate School
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THESIS

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1. Introduction

Temporomandibular disorders (TMD) represent a collection of problems related to craniocervical structures, in which pain, temporomandibular joint (TMJ) dysfunction, and masticatory muscle dysfunction are three of the most common complaints. Joint dysfunction is frequently described as an achy, sharp pain, localized in the preauricular area, accompanied by clicking, crepitation, and catching or locking of the TMJ. Masticatory muscle pain is described as a diffuse, dull pain in and around the jaw-muscle area. TMD pain is generally aggravated by jaw function. Other symptoms such as otalgia, tinnitus, cervical pain and headaches are also frequently reported by TMD patients.

Headaches are extremely frequent in the general population, and represent one the leading complaints among patients seeking treatment in facial pain clinics (Abramson, Hopp et al. 1980;D'Alessandro, Benassi et al. 1988;Linet, Stewart et al. 1989b;Rasmussen, Jensen et al. 1991b). While headache prevalence in the general population is approximately 20%, several studies have reported a primary headache prevalence of up to 70% in TMD patients. (Magnusson and Carlsson 1978;Wanman and Agerberg 1987). Similarly, some studies have detected TMD symptomatology in almost 60% of headache patients. Several authors have suggested that diverse forms of nociception, such as TMD, may play a critical role in headache pathogenesis (Olesen 1991c;Olesen, Tfelt-Hansen et al. 2000a). In addition, it is important to remember that the IHS classification categorizes TMD as a secondary form of headache (1988). In this paper, however, we will use the term “headache” when referring to primary headache disorders (i.e. migraine).

2. Purpose of the Study

Despite the extensive amount of studies addressing headache and TMD comorbidity (Reik, Jr. and Hale 1981;Lous and Olesen 1982;Forssell and Kangasniemi 1984a;Forssell, Kirveskari et al. 1985;Wanman and Agerberg 1986;Jaeger 1989;Mennell 1989;Schokker, Hansson et al. 1990;Mohlin, Pilley et al. 1991;Jensen, Rasmussen et al. 1992;Haley, Schiffman et al. 1993;Jensen, Rasmussen et al. 1993;Jensen, Bendtsen et al. 1998;Pettengill 1999;Aaron, Burke et al. 2000;Liljestrom, Jamsa et al. 2001;Ciancaglini and Radaelli 2001;Graff-Radford 2001), studies investigating the possible somatic and psychological differences between TMD and primary headache disorders are scarce in the literature (Mongini, Ciccone et al. 2000). Therefore, the management of patients experiencing headache and TMD may benefit from studies that shed light on this unanswered question. The aim of this study will be to investigate the psychological and sleep quality differences between TMD (myofascial pain and TMJ pain) and chronic/daily primary headache patients.

3. Literature Review

3.1 Temporomandibular disorders

3.1.1 Presentation and prevalence

Temporomandibular disorders signs and symptoms are extremely prevalent in the general population. Several studies have reported that 65% to 75% of the population experience at least one sign of TMD, and that 30% to 45% experience at lest one symptom (Agerberg and Carlsson 1973;Helkimo 1974c;Solberg, Woo et al. 1979;Rugh and Solberg 1985;Locker and Slade 1988;Pullinger, Seligman et al. 1988;de Kanter, Truin et al. 1993;Schiffman, Haley et al. 1995). Signs of TMD may include TMJ joint noises (50%) and mouth opening limitations (5%), while TMJ and masticatory muscle pain may be considered TMD symptoms.

Despite the high prevalence of TMD signs and symptoms, treatment needs appear to be around 5% to 7% of the general population according to the available literature (Rugh and Solberg 1985;Dworkin, Huggins et al. 1990;Schiffman, Haley, Baker, and Lindgren 1995)(de Kanter), with pain being the chief complaint in the vast majority of patients. Women represent the majority of TMD patients, with a male-female ratio of approximately 8:1 (Rugh and Solberg 1985;Dworkin, Huggins, LeResche, Von Korff, Howard, Truelove, and Sommers 1990;de Leeuw, Steenks et al. 1994). There seems to be a discrepancy, however, regarding the prevalence of women experiencing signs and symptoms of TMD when comparing patient and non-patient populations. Indeed, most epidemiological studies found the prevalence of signs and symptoms of TMD to be almost equal in men and women in the general population (Agerberg and Carlsson 1972;Helkimo 1974a;Helkimo 1974b;Salonen, Hellden et al. 1990). Some authors have attributed the higher prevalence of women in TMD patient populations to a greater propensity of women to seek medical care. Indeed, according to the report compiled by the CDC's National

Center for Health Statistics based on doctor visits in 1997 and 1998, women were twice as likely as men to visit a doctor for annual examinations and preventive services. Nevertheless, further research is needed in order to explain the higher numbers of women who seek care for TMD.

The majority of studies in the literature suggest that TMD signs and symptoms do not present an age-disease progression, but rather in the majority of cases it is self-limiting, or fluctuating over time (Dworkin, Huggins, LeResche, Von Korff, Howard, Truelove, and Sommers 1990;Salonen, Hellden, and Carlsson 1990;Osterberg, Carlsson et al. 1992;Egermark, Carlsson et al. 2001). Some have suggested, however, a higher incidence of TMD pathology in patient populations between the ages of 15 and 45 years, which generally corresponds with the highest activity period of an individual's life (Okeson 2002b).

Temporomandibular disorders can be roughly divided into masticatory muscle disorders and TMJ disorders. During the last decades, one of the main problems encountered by researchers in the field of TMD has been the lack of a standardized diagnostic classification for TMD. The development in 1992 of the Research Diagnostic Criteria (RDC) for TMD was the first step taken by the scientific community to establish a recognized research classification in the field of TMD (Dworkin and LeResche 1992). In spite of its value for research purposes, most clinicians may encounter problems when using the RDC for the diagnosis of myogenous TMD patients, since masticatory muscle disorders are basically grouped as one single entity. This lack of specificity is mainly due to a disagreement in the scientific community regarding the clinical presentation and classification of the different masticatory muscle pain disorders. Nevertheless, the RDC are still a valuable and important tool for researchers in the area of TMD.

3.1.2 Pathogenesis

Temporomandibular disorders are frequently described as a multifactorial entity, primarily involving pain and dysfunction arising in and from the musculoskeletal structures of the masticatory system. As mentioned before, most classifications divide TMD into masticatory muscle pain disorders and TMJ disorders. Since these disorders are associated with distinct pathophysiological mechanisms, the relevant literature for each will be reviewed separately.

3.1.2.1 Masticatory muscle disorders

Unquestionably, the most frequent complaint given by patients suffering TMD is masticatory muscle pain, although both TMJ disorders and muscle disorders may coexist frequently in the same patient (Truelove, Sommers et al. 1992;Okeson 2002b). The majority of patients describe muscle pain, as a diffuse, achy pain, that aggravates with functional activities, such as chewing, swallowing and speaking, and/or with palpation of the masticatory muscles. Restricted mandibular function is common, although this feature may be also present in patients suffering from TMJ disorders.

Much is unknown about muscle pain pathogenesis. At this moment, no general consensus regarding its pathophysiology and pain mechanisms has been reached. Nevertheless, numerous authors have postulated different theories to explain pain originating from muscle without evident pathology. Increased muscle activity has been blamed as the most common cause for muscle pain. Several researchers have claimed that muscle hyperactivity may cause muscle pain, which ultimately leads to muscle dysfunction, and initiating a "vicious cycle" that maintains pain. Mense (Mense 1993) postulated that muscle pain could be initiated by a direct trauma or muscle hyperactivity causing ruptures of muscle cells and leading to the release of

vasoneuroactive substances such as, bradykinin, substance P (SP) and calcitonin gene related peptide (CGRP). The release of these substances then provokes edema and subsequently decreased venous flow. This in turn will cause muscle hypoxia leading to a change in muscle metabolic activity. A failure in the contraction mechanisms of the muscle (calcium pump impairment) will provoke an increased muscle contraction leading to more ischemia, thus initiating a "vicious cycle". The available literature, however, has failed to support consistently an increased EMG activity in painful muscles, thus weakening the pain-muscle hyperactivity concept (Abraham 1977;Lund, Donga et al. 1991;Svebak, Anjia et al. 1993;Carlson, Okeson et al. 1993). Indeed, several authors demonstrated that the activity of agonist muscles is often reduced by pain, even when the pain does not arise from the muscle itself (Lund, Donga, Widmer, and Stohler 1991). Other studies have also demonstrated a decreased muscle contraction capacity in muscle pain patients (Clark, Beemsterboer et al. 1984), thus making it unlikely that any change in muscle activity could have a significant effect on the development of pain. In view of these findings, Mense suggested that local muscle contractures in the painful muscle could be electrically silent, thus suggesting a limitation of EMG recordings in the muscle pain diagnosis (Mense 1993). Although it is unlikely that muscle pain may provoke muscle hyperactivity, there is scientific evidence that muscle hyperactivity may , in certain occasions, cause muscle pain. Indeed, several lines of evidence support the concept that heavy muscle work can lead to both delayed and immediate muscle pain (Newham, Jones et al. 1987;Jones, Newham et al. 1987;Newham and Mills 1999). Nevertheless, more research is still needed to determine the validity of the majority of concepts postulated in the "vicious cycle" theory.

Several authors have also suggested that chronic masticatory muscle pain may be caused by central hyperexcitability and plasticity in trigeminal neurons (Dubner and Ruda 1992;Arendt-

Nielsen, Graven-Nielsen et al. 1997;Svensson 1997). According to this concept, a sustained barrage of nociceptive information from myogenous tissues may induce central changes in spinal and trigeminal neurons, leading to an increased depolarization mediated by excitatory aminoacids and activated NMDA receptors. This course of events may eventually lead to excitotoxicity, cell dysfunction, and loss of inhibitory pain mechanisms, which will cause increased levels of muscle pain. The triggers for this sequence of events remain to be determined.

Recent theories also suggest that muscle pain may be the result of a reduced capacity to regulated or inhibit nociceptive transmission (Maixner, Fillingim et al. 1995). Maixner et al. (1995) proposed that a dysfunction of the central nervous system (CNS) ascending reticular activation system (ARAS) would cause a disinhibition of nociceptive myogenous stimuli. This region of the CNS regulates different somatosensory functions, emotional responses, somatomotor output, and autonomic reactions and, in addition, interacts with the endogenous pain-regulatory systems. Therefore, chronic myogenous trigeminal pain could cause central sensitization and neuroplastic changes at the level of the trigeminal spinal tract nucleus (TSTN), resulting in ARAS impairment. The prominent cortical representation, and dense innervation of orofacial structures, may explain the higher pain propensity in craniofacial structures. However, at this point in time the mechanisms of ARAS dysfunction remain mostly unknown.

Other authors have recently proposed that muscle pain and fatigue may be strongly linked to sympathetic nervous system arousal through limbic system stimulation by myofascial tissue nociception. Bertrand (Bertrand 2001) postulated that metabolic fatigue and tissue damage of the myofascial structures initiate nociceptive transmission, that if sufficiently intense may reach the thalamo-cortical-basal ganglia circuits (TC-BG) in the CNS. These brain circuits are involved in

descending motor commands that shape behavioral responses to pain, thus altering cranial nerve (i.e. trigeminal) mediated physiology. Generally fatigue and tissue damage impulses share common CNS pathways, and may change patterns of motor unit recruitment and vascular perfusion in order to maintain efficient motor behavior. When fatigue becomes centrally mediated, it may be associated with perceived functional (i.e. motor) difficulties until the brain adjusts motor behavior to accomplish functional demands. Increasing levels of pain and fatigue impulses reaching the TC-BG circuits may activate the CNS limbic system due to its intimate physiologic connection with the TC-BG circuits. The limbic system is known to be strongly involved in emotional aspects of pain, such as fear, anger and threat. Consequently, anxiety and depression may follow intense limbic system activation provoking an up-regulation of the sympathetic nervous system (SNS). Autonomic nervous system arousal may in turn stimulate the release of norepinephrine (NE) in somatic peripheral tissues (i.e. muscle tissues), thus facilitating reflexive behaviors (i.e. parafunctional activities) that bombard the CNS with more fatigue and pain impulses. Consecutively, this intense barrage of fatigue-pain impulses may modify CNS patterns of motor unit recruitment and vascular perfusion in order to maintain efficient motor behavior, thus initiating a self-perpetuating cycle.

3.1.2.2 TMJ disorders

A common condition observed in the TMJ is internal derangement (ID), which represents a broad term used by many authors to describe functional and histological alterations of the TMJ disc-condyle complex. Arthralgia is frequently reported, but dysfunction (i.e. mouth opening limitations) is the most common finding in patients suffering from TMJ dysfunction (Okeson 2002a). Many TMJ patients report a catching, clicking sensation of the joint during mandibular

excursions, which is most often related to an anterior displacement of the TMJ disc. The loss of normal disc position can occur with elongation of TMJ disc ligaments and the inferior retrodiscal lamina. The most common cause for TMJ disc displacement is trauma. (Pullinger and Seligman 1987;Pullinger and Seligman 1991). In spite of the morphological alterations, pain is not always present in patients with TMJ disorders. Indeed, numerous studies have shown a large percentage of asymptomatic subjects with an abnormal TMJ disc position (Kircos, Ortendahl et al. 1987;Huber and Hall 1990;de Leeuw, Boering et al. 1995). Additionally, higher degrees of TMJ morphological alterations are not necessarily correlated to higher levels mandibular dysfunction (Schiffman, Anderson et al. 1992;de Leeuw, Boering et al. 1993;de Leeuw, Boering et al. 1994). Therefore, TMJ ID is not necessarily related to pain or dysfunction.

Osteoarthritis (OA), which is frequently present in patients suffering from TMJ disc disorders, is described as a noninflammatory degenerative joint disease characterized by a degeneration of the articular surfaces of the joint condyle and fossa. The processes of OA are widely unknown, although three main concepts concerning its etiopathogenesis have been postulated, based on cartilaginous or extracartilaginous factors. Several authors have postulated that the degenerative changes associated with OA may occur at any time if the joint is loaded over the intrinsically adaptive capacity of the articular cartilage (Stegenga, de Bont et al. 1989).

The second concept regarding OA pathogenesis implies a failure of the chondrocyte-controlled internal remodeling system. This theory suggests that a primary insult, whether mechanical, biochemical, inflammatory, or immunologic in nature, may disrupt the balance between synthesis and degradation of the extracellular matrix of the articular cartilage. As a result of this, the chondrocytes release a higher amount of proteolytic enzymes resulting in rapid breakdown of the extracellular matrix (Dijkgraaf, de Bont et al. 1995).

The third concept suggests that the combination of different factors such as subchondral bone microfractures, the reduction of the quality and/or quantity of synovial fluid, vascular changes, and/or changes in the synovial membrane, may lead to cartilage degradation. Ref: Dijkgraaf? All the same, these three hypotheses regarding the pathogenesis of OA are not mutually exclusive since one may influence the other. Other factors, such as age, joint hypermobility and alterations in the chondrocyte metabolic activity may also play a role in the genesis, initiation or perpetuation of OA (Dijkstra, de Bont et al. 1992).

Osteoarthritis is also frequently associated with TMJ disc displacements. Whether OA leads to disc displacement or disc displacement causes OA remains uncertain. Several lines of evidence suggest that a displacement of the TMJ disc may lead to a breakdown of the retrodiscal tissues, hence initiating the destruction of the articular surfaces (Katzberg, Keith et al. 1983). Conversely, other studies have observed osteoarthritic changes in the surfaces of TMJ with normal disc-condyle relationships, thus suggesting that osteoarthritic changes may precede disc displacement (de Bont, Boering et al. 1986).

3.1.3 Comorbidity

Evidence associating TMD with other somatic and psychological alterations besides headache disorders is not overwhelming in the literature. Nonetheless, there seems to be some evidence for the presence of sleep disturbances, cervical pain, otalgia, headaches, and other somatic disorders in TMD patients (Magnusson, Egermark et al. 2000;Kamisaka, Yatani et al. 2000;Lam, Lawrence et al. 2001;Moldofsky 2001;Arima, Svensson et al. 2001). Several studies also suggest that depression, anxiety, and other psychological disorders are highly prevalent in TMD populations (Stockstill and Callahan 1991;Shiau and Chang 1992;Dahlstrom 1993;Rugh,

Woods et al. 1993;Parker, Holmes et al. 1993;List and Dworkin 1996;Dahlstrom 1998;Cimino, Michelotti et al. 1998;Michelotti, Martina et al. 1998;Ohrbach and Dworkin 1998;Glaros 2000;Sipila, Veijola et al. 2001a;Turner, Dworkin et al. 2001b;Sipila, Veijola et al. 2001b;Dworkin, Huggins et al. 2002;Yap, Chua et al. 2002).

3.1.3.1 Somatic conditions

Otalgia and other aural symptoms are frequently observed in patients with TMD (Ciancaglini, Loreti et al. 1994;Parker and Chole 1995;Bush, Harkins et al. 1999). Lam et al. (2001) in a retrospective study assessed the prevalence of aural symptoms in 776 orofacial pain patients and investigated the potential association between temporomandibular disorders (TMD) and aural health, while controlling for covariates known to be associated with TMD or auditory dysfunction. They reported that of the 344 subjects who had TMD, 59.9% complained of aural symptoms (otalgia, tinnitus, vertigo, or perceived hearing loss), versus 29.2% of the 432 patients without TMD. They concluded that TMD is significantly correlated to aural health, although no cause-and-effect relationship has yet been demonstrated. Aural symptoms were also found to have a measurable impact on the patient's quality of life.

Although the interaction between sleep and craniofacial pain has generated considerable interest during the past decades, it is still unknown if TMD is the cause or effect of poor sleep. Poor sleep has been reported in TMD populations in a broad number of studies (Magnusson, Egermark, and Carlsson 2000;Riley, III, Benson et al. 2001), although to the best of our knowledge none of these studies have clarified the exact role of sleep in the genesis of TMD. Several authors, however, have published excellent review articles on the topic of sleep and TMD (Bailey 1997;Lavigne, Goulet et al. 1999;Bailey and Attanasio 2001), proposing that poor

sleep in TMD patients may be positively correlated to pain density (frequency, intensity and duration) (Lavigne, Goulet, Zuconni, Morrison, and Lobbezoo 1999). These authors, on the other hand, have also suggested that although a linear cause-and-effect relationship is plausible between pain density and sleep quality, other variables such as concomitant fatigue, mood disturbance or depression, and the presence of chronic poor health could also play an important role in the genesis of sleep disturbances.

3.1.3.2 Psychological conditions

The relationship between TMD and psychosocial disturbances has been extensively studied during the last decade. There are numerous studies that report an association between depression, anxiety, stress and other psychological disturbances in TMD populations (Stockstill and Callahan 1991;Shiau and Chang 1992;Dahlstrom 1993;Rugh, Woods, and Dahlstrom 1993;Parker, Holmes, and Terezhalmy 1993;List and Dworkin 1996;Dahlstrom 1998;Cimino, Michelotti, Stradi, and Farinaro 1998;Michelotti, Martina, Russo, and Romeo 1998;Ohrbach and Dworkin 1998;Glaros 2000;Turner, Dworkin et al. 2001a;Sipila, Veijola, Jokelainen, Jarvelin, Oikarinen, Raustia, and Joukamaa 2001a;Sipila, Veijola, Jokelainen, Jarvelin, Oikarinen, Raustia, and Joukamaa 2001b;Dworkin, Huggins, Wilson, Mancl, Turner, Massoth, LeResche, and Truelove 2002;Yap, Chua, and Hoe 2002). Parker et al. (1993) evaluated 110 patients complaining of nondental orofacial pain of more than 3 months duration using the Minnesota Multiphasic Personality Inventory (MMPI). The results of the study revealed four distinct personality profiles: psychophysiologic reaction, in 52% of the patients; depressed reaction, in 11% of the patients; defensive reaction, in 12% of the patients; and "no diagnosis" (normal), in

24% of the patients. The authors concluded that chronic orofacial pain patients may present personality characteristics that are similar to those of other chronic pain patients.

Michelotti et al. (1998) analyzed psychological profiles of chronic pain patients affected with temporomandibular disorders (TMD), by means of the MMPI test. Fifty consecutive TMD patients were examined and were then divided into two subgroups: 1. myofascial pain (MP) and 2. TMJ articular disorders. Sixty-two percent of the whole sample presented pathological MMPI scores. Both subgroups presented similar profiles with alteration of the neurotic triad (hypochondriasis, depression, hysteria), and pathological values of hypochondriasis and hysteria ("V" configuration). These authors also concluded that chronic TMD patients present personality characteristics similar to those of other chronic pain patients according to the MMPI.

Sipila et al. (2001) evaluated the association between symptoms of TMD and depression in a large population sample of young adults. Questionnaire information concerning TMD symptoms was collected from a sample of 5,696 subjects. Depression was measured with the aid of a question about reported depression (diagnosed by a doctor) and with the Symptom Checklist depression subscale (SCL-25 DS). Of the TMD symptoms, those related to pain had the most significant relations to indicators of depression. In both genders, the proportion of depression indicated with the SCL-25 DS was significantly higher in subjects with pain-related symptoms of TMD, i.e., facial pain and "pain at jaw rest", and in men with "pain on jaw movement", compared with non-pain subjects ($p<0.05$). Among women, the prevalence of recognized depression was also significantly higher in subjects with pain-related symptoms of TMD, compared with subjects with no pain ($p<$ or $=0.05$). The authors concluded that depression was associated with TMD symptoms, especially those related to pain.

Several lines of evidence support a greater probability of emotional problems among masticatory MP patients compared to those diagnosed with TMJ disorders (Eversole, Stone et al. 1985;Harness, Donlon et al. 1990;Glaros 2000). Harness et al. (1990) studied 150 patients meeting diagnostic criteria for MP, TMJ pain, and atypical facial using the MMPI. The scores from 95 subjects were compared with self-report measures of depression and anxiety. Psychopathological factors were found to be more significant among MP patients than TMJ pain patients. Other studies, however have not found psychological differences between MP and TMJ disorders (Michelotti, Martina, Russo, and Romeo 1998). Overall, there seems to be stronger evidence in favor of greater psychological disturbances in MP patients.

3.2. Headaches

3.2.1 Introduction

Headache disorders are extremely common in the general population (Abramson, Hopp, and Epstein 1980;D'Alessandro, Benassi, Lenzi, Gamberini, Sacquegna, De Carolis, and Lugaresi 1988;Linet, Stewart et al. 1989a;Rasmussen, Jensen, Schroll, and Olesen 1991b) and represent one of the more common reasons why people seek care from ambulatory care centers in the United States. Rasmussen et al. (1991) in a lifetime, point prevalence study of 740 patients reported a total lifetime headache prevalence of 96%. The prevalence of headaches, howeve,r is still a point of controversy in the medical community. The lack of reliable laboratory markers and its diagnostic subjectivity has led to methodological discrepancies, and therefore contradictory results among headache studies. Population bias and measurement technique differences may also be possible causes for the contradictory results between studies.

Numerous studies have also revealed age and sex prevalence differences for headache in the general population. Several lines of evidence suggest a declination of headache prevalence after middle age (Waters 1974;Philips 1977;Ziegler, Hassanein et al. 1977;Newland, Illis et al. 1978;Nikiforow and Hokkanen 1979). An increased mortality among the elderly, and an increased incidence among the younger population may be possible explanations of this low headache prevalence with advanced age (Waters 1974;Philips 1977;Ziegler, Hassanein, and Couch 1977;Nikiforow and Hokkanen 1978;Rasmussen, Jensen, Schroll, and Olesen 1991b). Several lines of evidence have also revealed an increased prevalence of headaches among women (Waters and O'Connor 1971;Johannes, Linet et al. 1995;Pettengill 1999). Female hormones are thought to be one of the possible causes of sex prevalence differences in headache studies (Rasmussen 1993).

Headaches are commonly classified as primary (not related to an organic problem) or secondary (related to organic diseases). Migraine headache and tension-type headache (TTH) are examples of primary headaches, while head trauma, TMD, and vascular and non-vascular intracranial disorders are examples of secondary headaches. Primary headaches can also be classified as episodic or chronic-daily. Chronic daily headache (CDH) is a descriptive term rather than a specific diagnosis. Even though relatively infrequent in general practice, CDH is the major reason for consultation in headache specialty clinics. CDH is estimated to affect 2% to 3% of the general population and defines a group of patients that require distinctive diagnostic and management needs (Silberstein, Lipton et al. 1996;Silberstein, Lipton et al. 1998). Although not life threatening, CDH can be considerably incapacitating with a substantial impact on the patient's social life and work performance.

3.2.2 Migraine

3.2.2.1 Presentation and prevalence

Migraine is a recurrent, idiopathic form of headache lasting 4-72 hours. Its typical clinical characteristics are unilateral location; pulsating quality; moderate to severe intensity; associated nausea, photo-and/or phonophobia, and increased pain intensity with physical activity. Migraine headaches can be subdivided into migraine with and without aura, which is a cluster of neurological symptoms that precede the migraine attack (Olesen, Tfelt-Hansen et al. 2000b)**.** Migraine headache affects approximately 16% of the general population, with a higher prevalence in women (25%) compared to men (8%). Sexual hormones may be related to this higher prevalence of migraine in the female population (Silberstein and Merriam 1991;Rasmussen 1993;Silberstein and Merriam 1999).

3.2.2.2 Genetics

Genetic factors may play an important role in the development of migraine headaches. On occasion, anyone can have a migraine attack without being considered necessarily a migraine patient. What defines a migraine patient is not the attack itself, but the frequency of the attacks. Genetic factors are commonly implicated in migraine pathogenesis. A well known monogenic subtype of migraine is familial hemiplegic migraine (FHM), which consists of a mutation in a gene on chromosome 19 coding for a subunit of voltage dependent P- and Q- Ca2+ channels. Some families may also carry alterations in chromosome 1, X and elsewhere (Ducros, Joutel et al. 1997;Nyholt, Curtain et al. 2000).

Several authors have proposed a model for migraine as a complex genetic disorder. According to this model, genetic load can be cumulative and determines the probability for a

given person suffering migraine attacks. Genetic factors seem to set the individual threshold for migraine attacks, although both endogenous and exogenous factors also modulate migraine disorders. (Russell, Iselius et al. 1995;Russell and Olesen 1995;Ferrari 1998;Gervil, Ulrich et al. 1999;Ulrich, Gervil et al. 1999).

3.2.2.3 Pathogenesis

Neuronal and vascular components are probably interrelated in migraine pathogenesis. Some of the neuronal structures involved are the cerebral cortex, the brain stem, and both the peripheral and central components of the trigeminovascular system. The role of these structures in the pathogenesis of migraine is still controversial. During the 1940s, several theories such as the "vascular theory" and the "spreading depression" theory were proposed as possible explanations of the underlying mechanisms of migraine pain. More recently, however, it has been demonstrated that the vascular changes observed to be associated with migraine pain were the result and not the cause of the pain (Okeson and Bell 1995). Current studies support the concept that migraine is a neurovascular pain disorder that appears to be related to a trigeminovascular reflex involving peripheral and central mechanisms, though cortical and brainstem alterations may also be related. An increase of central or peripheral afferent input at the level of the nucleus caudalis of the spinal tract nucleus, and its thalamic connections, may activate a trigeminovascular reflex, producing a neurogenic inflammation at the level of the intracranial vessels as a result of the release of excitatory neurotransmitters by primary afferents. This begins a cascade of events that produce migraine pain (Sanchez and Moskowitz 2000).

Since Laeo (Laeo 1944) proposed the theory of cortical spreading depression (CSD) to explain the mechanism of migraine with aura, a substantial number of literature reviews have

been published concerning this phenomenon (Buzzi, Carter et al. 1991;Hoskin, Kaube et al. 1996;Leysen, Gommeren et al. 1996).This theory states that the threshold of neuronal excitability is altered, resulting in neuronal dysfunction. Neurons and glial cells depolarize during CSD, giving rise to an intense but transient spike activity. Neuronal silence immediately follows, lasting for a few minutes; evoked potentials usually take 15-30 minutes to recover. In experimental animal studies, a noxious stimulus resulted in suppression of neuronal activity that spread slowly across the brain surface at a rate of 2-4 mm/min. The neurochemical basis of spreading depression is the release of potassium or the excitatory amino acid glutamate from neural tissue; this depolarizes adjacent tissue, which, in turn, releases more neurotransmitters, thus propagating the CSD. Calcium influx triggered by NMDA receptor activation, a subtype of glutamate receptor, also activates nitric oxide (NO) synthase activity. Nitric oxide is known to promote central sensitization of trigeminal nociceptors (Lauritzen 2000). During the last decade, the CSD theory has received important support from neuroimaging and positron emission tomography (PET) studies (Olesen 1991a;Diener 1997;Fuentes, Diez et al. 1998;Lauritzen 2000;Olesen and Hans-Christopher 2000;Baron 2000).

Olesen (Olesen 1991b) proposed a model of migraine pain perception, called the vascular-supraspinal-myogenic (VSM) model, which addresses the theoretical relationship of internal and external factors for triggering and expressing the migraine attack. This model not only includes the source of pain, but also its central modulation. Neurons present in the subnucleus caudalis of the spinal tract nucleus (STN) of the trigeminal nerve (TN) integrate afferent input from multiple intracranial and extracranial tissues. This area of the spinal cord also receives supraspinal inhibitory and excitatory inputs that modulate the afferent information into the STN. The sum of all these inputs projects to the thalamus and onto the cortex. Even if

migraine pain is primarily vascular or dural, additional nociception from muscles and other cranial structures may also contribute to pain. Vascular input from the migraine attack is normally sufficient to fire these neurons, but in some occasions it may only cause a partial depolarization of the neurons. Additional afferent input from pericranial muscles and other cranial tissues may increase the likelihood of neuronal depolarization.

3.2.2.4 Comorbidity

There are abundant studies in the literature addressing the comorbidity of headaches and certain physiological and psychological conditions. Indeed, numerous clinical series studies, case-control studies and epidemiological surveys have addressed migraine headache comorbidity with contradictory results (Markush, Karp et al. 1975;Gamberini, D'Alessandro et al. 1984;Chen, Leviton et al. 1987;Couch and Hassanein 1989;Welch and Levine 1990;Lipton, Ottman et al. 1994). The variability between these studies may be explained by differences in methodology, which may limit the conclusions drawn by the authors.

3.2.2.4.1 Somatic conditions

Several lines of evidence have found significant associations between migraine and cardiovascular disorders, hypotension, stroke, epilepsy, gastrointestinal disorders, sleep disorders, and respiratory disorders (Markush, Karp, Heyman, and O'Fallon 1975;Gamberini, D'Alessandro, Labriola, Poggi, Manzoni, Carpeggiana, and Sacquegna 1984;Featherstone 1985;Chen, Leviton, Edelstein, and Ellenberg 1987;Chen and Leviton 1990;Sahota and Dexter 1990;Lipton, Ottman, Ehrenberg, and Hauser 1994;Paiva, Batista et al. 1995;Sorbi, Maassen et al. 1996;Carolei, Marini et al. 1996;Merikangas, Fenton et al. 1997;Bruni, Fabrizi et al.

1997;Hering-Hanit, Yavetz et al. 2000;Happe, Zeitlhofer et al. 2001). Merikangas et al. (Merikangas, Fenton, Cheng, Stolar, and Risch 1997;Merikangas and Rasmussen 2000) studied migraine comorbidity using data from a large-scale *National Health and Nutrition Examination Survey* in the U.S. adult population. Baseline and first follow-up data were used to investigate cross-sectional and longitudinal associations between migraine and different medical conditions. After controlling for confounding factors, significant associations were found between migraine and cardiovascular disorders, hypotension, stroke, epilepsy, and gastrointestinal and respiratory disorders.

Carolei et al. (1996) in a case-control study of 308 patients aged 15-44, with either transient ischaemic attack (TIA) or stroke, and of 591 age- and sex-matched controls prospectively recruited in seven university hospitals, reported that history of migraine was more frequent in patients than in controls (14.9% vs 9.1%; adjusted odds ratio 1.9, 95% confidence interval 1.1-3.1). In the prospectively designed subgroup analyses, a history of migraine reached the highest odds ratio (3.7, 95% CI 1.5-9) and was the only significant risk factor in women below age 35 (p=0.003). They concluded that there was a rare association between migraine and cerebral ischaemia, limited to women below age 35. Therefore, according to these studies there seems to be a clear comorbidity of migraine headache and certain medical conditions.

Other studies, in contrast, have failed to find associations between migraine and other medical disorders (Chen, Leviton, Edelstein, and Ellenberg 1987;Rasmussen and Olesen 1992;Rasmussen and Olesen 1994;Sternfeld, Stang et al. 1995). Sternfeld et al. (1995) examined the relationship between migraine, chest pain, and risk of myocardial infarction (MI) in a retrospective cohort study of 79,588 enrollees. Both migraine and chest pain were ascertained by questionnaire, and follow-up began at the time of each participant's medical examination and

continued until the earliest occurrence of hospitalization for MI, death, termination of enrollment, or end of study period. There was a strong relationship between migraine and chest pain, but, in general, no significant association was found between migraine and risk of MI except among women with a family history of MI in whom a self-reported physician diagnosis of migraine was related to a greater than two-fold increase in risk. Despite the contradictory results of the different studies addressing migraine comorbidity, most lines of evidence suggest that several disorders, including allergies, mitral valve prolapse, hypertension and stroke, are strongly associated with migraine headaches. Differences across studies may be the result of methodological differences, a lack of standardized diagnostic definitions, sampling differences, and or/a lack of statistical power.

The relationship between migraine and sleep disturbances is well established in the literature. Several studies have reported significantly higher levels of sleep disturbance in migraine patients when compared to non-headache populations (Bruni, Fabrizi, Ottaviano, Cortesi, Giannotti, and Guidetti 1997). Bruni et al. (1997) performed a survey to determine the prevalence of sleep disturbances in children with migraine and tension-type headache. A questionnaire of history and clinical data and of sleep disturbances was given to parents of 283 headache subjects (164 with migraine and 119 with TTH), and their results were compared to a group of 893 normal healthy subjects with a normal sex and age distribution. Their results revealed that migraine subjects showed a higher prevalence of sleep disturbances during infancy. In both headache groups, more parents had sleep disturbances and there was a higher occurrence of co-sleeping and napping. A higher frequency of sleep disturbances involving sleep quality, night awakenings, nocturnal symptoms and daytime sleepiness was also reported in the headache group. No statistical differences were found in the prevalence of sleep disturbances between

migraine and tension-type headache. Nevertheless, the migraine group reported to have "disturbed sleep" more often when compared to the tension-type headache group. Therefore, according to this study there seems to be a higher prevalence of sleep disturbances in “children-migraineurs” than in non-headache populations.

Several lines of evidence, however, also support the hypothesis that sleep disturbances in adult headache populations may be the result of medication overuse. Indeed, several studies have shown a positive correlation between medication withdrawal and sleep quality improvements (Paiva, Batista, Martins, and Martins 1995;Hering-Hanit, Yavetz, and Dagan 2000). Nevertheless, more randomized controlled studies are needed in order to clarify the role of sleep disturbances in headache pathogenesis.

3.2.2.4.2 Psychological conditions

Over the past decade, several controlled prospective studies have addressed the association between migraine and psychological factors. The role of personality factors and negative emotions has been studied thoroughly in the literature (Drummond 2000). Neuroticism has been suggested to predispose to migraine headaches, though recurrent migraine headaches could also be responsible to heightened neuroticism scores (Breslau and Andreski 1995).The prevalence of stress, depression and anxiety have also been investigated in migraine populations (Arena, Blanchard et al. 1984;Sorbi, Maassen, and Spierings 1996). The associations between migraine, depression and anxiety may reflect a predisposition to these disorders, due to a common neurotransmitter (NT) or receptor disturbance in the brain stem or midbrain (Merikangas and Stevens 1997). An activation of the pituitary adrenocortical axis by administration of the serotonin receptor agonist piperizine provokes a release of cortisol,

followed several hours later by a migraine like headache. This suggests that an endogenous release of cortisol in response to psychological stress could set migraine headaches in motion (Drummond 2000).

Sorbi et al. (1996) prospectively compared daily hassles, mood changes, and sleep quality in the three days preceding migraine attacks to migraine-free control days in nineteen female migraine patients. Their results indicated increased hassles, particularly in the 24 pre-migraine hours; psychological arousal (increased irritability, annoyance, and tenseness), predominantly from 60 to 24 hours before the attack; repeated fatigue in the 60 pre-migraine hours, with a peak immediately before the attack; and a sharp decrease in sleep quality in the night preceding the attack. Stewart et al. (Stewart, Breslau et al. 1994) using data from a population-based study of more than 10,000 respondents studied whether individuals with a history of panic attacks were at greater risk of having specific headaches in the week preceding an interview. During the study, four types of headache were defined. Of these, only migraine was strongly associated with panic attacks.

3.2.3 Tension-type headache

3.2.3.1 Presentation and prevalence

Tension-type headache is one of the most frequent sources of cranial pain. This type of headache is often described by the patient as a dull, tight, oppressive pain, that feels like a tight headband. According to the International Headache Society (IHS) criteria, the pain is usually mild or moderate in severity. The pain should thus hinder but not prohibit daily activities. This type of headache is predominantly bilateral, and is usually located in the occipital parietal, temporal and frontal regions (Iversen, Langemark et al. 1990;Rasmussen, Jensen et al. 1991a).

The symptomatology of TTH is not distinct, since many disturbances can cause the previously mentioned symptoms. Although the International Headache Society (IHS) classifies TTH into a number of subforms, all these subforms really are believed to be variants of the same disorder. TTH can be subdivided into episodic (>15 episodes/month) and chronic (<15 episodes/month). The reason for this subdivision is purely practical, since chronic tension-type headache (CTTH) patients constitute the majority of patients seeking care in headache clinics, whereas episodic tension-type headache (ETTH) patients rarely consult their general physician (Olesen 2000). The IHS also subdivided TTH into associated with pericranial muscles and unassociated with pericranial muscles. This subdivision was an attempt by the IHS, to encourage scientists to study the impact of pericranial muscle tenderness in TTH in a more systematic way. According to the IHS criteria, no vomiting and no more than one of the symptoms (nausea, photophobia, phonophobia) occur in TTH.

Despite being one of the main complaints of patients seeking care in headache clinics, the medical community has drawn little attention to TTH epidemiology. TTH affects 2-3% of the general population on a daily basis (Hollnagel and Norrelund 1980;Rasmussen, Jensen, Schroll, and Olesen 1991b;Gobel, Petersen-Braun et al. 1994;Schwartz, Stewart et al. 1998), and has a 1 year prevalence that ranges from about 30% to about 80% (Nikiforow 1981;Rasmussen, Jensen, Schroll, and Olesen 1991b;Gobel, Petersen-Braun, and Soyka 1994).

Most studies confirm that the prevalence of TTH is slightly higher in women, with a male-to-female ratio of 1:1.3. Rasmussen et al. (Rasmussen, Jensen, Schroll, and Olesen 1991b) recruited one thousand 25-64 year old men and women for a study of specific headache entities using the operational diagnostic criteria of the IHS. All participants were invited to a general health examination focusing on headache and including: a self-administered questionnaire

concerning sociodemographic variables, a structured headache interview and a general physical and neurological examination. Their results indicated a 1 year point prevalence of TTH in 63% of males compared to 86% of females. Hormonal factors as in migraine headaches may also play a role in the higher incidence of TTH in the female population.

Most studies confirmed that TTH peaks between the ages of 30 and 39 years and then declines with age. Due to the cross-sectional nature of most headache epidemiological studies, it is difficult to support conclusions about how TTH evolves over time. The lower prevalence in older groups could be explained by spontaneous remission of headaches with advancing age or an increased incidence in the younger age groups (Rasmussen 1993). Distinguishing a real effect of aging requires longitudinal follow-up studies. The impact of medication induced remission in the older age group also needs further investigation by the medical community.

3.2.3.2 Genetics

TTH may have a familial aggregation, though data supporting this assumption are scarce in the literature. Only a single genetic epidemiologic study has investigated familial aggregation of chronic tension-type headache. Ostergaard et al. (Ostergaard, Russell et al. 1997) examined the familial occurrence of CTTH in spouses and first degree relatives of probands with CTTH in order to evaluate its possible genetic background. Neurology residents interviewed 122 consecutive probands, who met the International Headache Society's criteria for CTTH. The spouses and first degree relatives aged 18 years or above were interviewed by telephone. The risk of familial occurrence was assessed by estimating the population relative risk of the disease in specified groups of relatives. The risk was calculated as the probability that a relative is affected given that the proband is affected, divided by the probability that a random member of

the population is affected. A family aggregation is implied when this risk ratio significantly exceeds 1. The results of the study revealed that in comparison to the general population, first degree relatives had a significantly increased risk of CTTH, while spouses had no increased risk of CTTH. The authors concluded that multifactorial inheritance may alternatively reflect the genetic heterogeneity of CTTH.

3.2.3.3 Pathogenesis

For many years the scientific community has directly related TTH to muscle tension. Several different terms, such as muscle contraction or muscle tension-type headache, have been used in the literature when referring to TTH. Several authors, however, have questioned the validity of this assumption when observing normal electromyographic (EMG) activity of pericranial muscles in TTH patients (Pikoff 1984). Moreover, several studies have demonstrated that therapies aiming at a reduction of pericranial muscle EMG activity are not effective in reducing TTH symptoms (Richman and Haas 1994;Rollnik, Karst et al. 2001). Although some studies do suggest an increase of EMG in TTH patients when submitted to emotional stressors compared to controls, this increase may actually be a physiologic response to pain and not its cause (Schoenen, Gerard et al. 1991;Schoenen and Bendtsen 2000). The contradictory results between studies may be attributable to the different recording conditions. Taken together, however, the available scientific literature suggests that there is no causal relationship between TTH and surface EMG activity, although EMG activity in certain muscles may be higher in TTH patients compared to controls.

Olesen et al. (Olesen and Schoenen 2000) proposed that TTH is the result of a dynamic interaction between peripheral nociceptive and mechanoceptive second-order neurons and their

descending control systems. Peripheral mechanisms may dominate in episodic TTH, whereas central changes may become the predominant pathogenic mechanism in chronic TTH. Physical stress and lack of sleep may increase nociceptive activity of Aδ and C fibers, sensitizing spinal tract nucleus neurons. This increased nociceptive activity under normal circumstances is counterbalanced by the descending inhibitory mechanisms of the CNS. These descending inhibitory mechanisms involve numerous CNS nuclei and brainstem efferent pathways, including the amygdala, periaqueductal gray matter (PAG), dorsolateral pontine tegmentum (DLPT) and rostral ventral medulla (RVM) (Moreau and Fields 1986;Tortorici and Vanegas 1995;Kalyuzhny and Wessendorf 1998;Fields and Basbaum 1999;Calejesan, Kim et al. 2000;Fields 2000;Odeh and Antal 2001). Through different descending projections (mainly serotonergic and noradrenergic), these pain modulatory "circuits" control both spinal and trigeminal dorsal horn pain transmission neurons and mediate both opioid and stimulation produced analgesia (SPA) in humans. These inhibitory systems, however, are also theoretically capable of generating or enhancing perceived pain intensity in certain circumstances. Consequently, an inadequate function of these inhibitory systems due to stress, anxiety and emotional disturbances may favor the development of TTH. Muscle tension and activation of brainstem on-cells (facilitate nociception) may also increase by an up-regulation of limbic system due to increased emotional disturbances.

If the peripheral nociceptive activity is prolonged over time, central changes may occur within the trigeminal system. Myofascial factors are likely to perpetuate pain over time, since they are more effective inducing central sensitization than cutaneous stimuli. When central sensitization reaches a certain threshold the pain may become chronic. A cycle may be initiated by which incoming peripheral stimuli may provoke abnormal reactions that maintain the state of

central sensitization, even if the original causative stimulus or stressor has resolved. Therefore, preventing the transition from episodic to chronic tension-type headache is crucial when managing headache patients.

3.2.3.4 Comorbidity

3.2.3.4.1 Somatic conditions

Data supporting an association between TTH and different somatic conditions is scarce in the literature. Nevertheless, there are still a few studies addressing a link between TTH and diverse somatic disorders. Chronicity may be related to the development of comorbid somatic disorders, though this is not a unique characteristic of TTH (Silberstein and Lipton 2000;Lainez and Monzon 2001). Lack of sleep is a common complaint of patients suffering TTH. In the general population, subjects with TTH (both episodic and chronic forms) had a significantly greater number of sleeping problems compared with migraineurs (Rasmussen 1993). Chronic TTH has sometimes been suggested to be localized part of fibromyalgia syndrome because impaired sleep, widespread chronic pain and recurrent headaches are among the most frequent complaints in fibromyalgia patients (Aaron, Burke, and Buchwald 2000).

3.2.3.4.2 Psychological conditions

Tension-type headache has variously been labeled psychogenic headache, depression headache, stress headache, conversion headache and psychomyogenic headache. The relationship between TTH and psychosocial factors has been addressed by different studies with contradictory results (Lous and Olesen 1982;Blanchard, Kirsch et al. 1989;Merikangas, Merikangas et al. 1993;Arena, Bruno et al. 1997;Wittrock and Myers 1998;Puca, Genco et al.

1999;Andrasik and Passchier 2000). Puca et al. (1999) investigated in a multicenter study carried out in 10 Italian headache centers the prevalence of psychosocial stress and psychiatric disorders listed by the IHS classification as the "most likely causative factors" of TTH. Two hundred and seventeen TTH adult outpatients consecutively recruited underwent a structured psychiatric interview (CIDI-c). The assessment of psychosocial stress events was carried out using an ad hoc questionnaire. Diagnoses were made according to DSM-III-R criteria. At least one psychosocial stress event or a psychiatric disorder was detected in 84.8% of the patients. Prevalence of psychiatric comorbidity was 52.5% for anxiety, 36.4% for depression, and 21.7% for headache as a delusion or an idea. Psychosocial stress was found in 29.5% of the patients and did not differ between patients with and without psychiatric comorbidity. Generalized anxiety disorder (83.3%) and dysthymia (45.6%) were the most frequent disorders within their respective psychiatric group. The authors concluded that the results of this study emphasize the need for a systematic investigation of psychiatric comorbidity in TTH patients.

Venable et al (Venable, Carlson et al. 2001) studied the role of anger in headaches, and its relationship to anxiety, depression, and daily life stressors. Participants were 65 young adult women who suffered from recurrent headaches (tension-type and “mixed” headache). The sample was obtained in a large-scale screening of young adult women using the Headache Symptoms List to identify those with recurrent headache. Those individuals reporting headaches completed a battery of psychological questionnaires including the State-Trait Anger Expression Inventory, the Mood and Anxiety Symptoms Questionnaire, and the Hassles Scale. Their results revealed a statistically significant relationship between anger suppression and depression as well as anger expression and anxiety for those with headache. Patients with headaches also experience more general, nonspecific distress rather than symptoms indicative of anxiety and depression. In

addition, the mixed headache group scored high on both general and specific measures of depression and on anger suppression. These results suggest that mixed headache patients might experience more psychological distress than those with TTH, although both groups present significantly higher psychological distress levels when compared to normal individuals.

Other studies have not found differences in psychological profiles between normal controls and TTH patients (Merikangas, Merikangas, and Angst 1993). Merikangas et al. (1993) studied the association between psychiatric disorders and headache syndromes in a longitudinal epidemiologic sample of young adults who were selected from the general population of Zurich, Switzerland. Headache syndromes were defined according to the newly introduced diagnostic criteria of the International Headache Society in 1988. The prevalence rates of psychiatric disorders, according to specific headache subtypes, were examined both cross-sectionally and longitudinally. Their results revealed that subjects with TTH did not differ from controls with respect to any of the effective or anxiety disorders in both the cross-sectional and longitudinal data. Penzien et al. (Penzien, Rains et al. 1993) when reviewing the available headache literature reporting psychological data found that 71% of the studies presented increased psychological test scores for the headache patients when compared to normal controls. In the majority of these studies TTH patients showed the most heightened psychological scores. The authors concluded, however, that the psychological scores were clinically significant in only 5-15% of the headache patients.

Despite the contradictory results between studies, most lines of evidence support an association between TTH and different psychological disorders. Nevertheless, it remains to be determined whether the psychological distress observed in most TTH patients is mainly caused by a pain-filled history or reveals true psychological differences between TTH and normal

individuals. Indeed, several lines of evidence support a positive association between headache density (frequency and intensity) and levels of psychological distress (De Benedittis and Lorenzetti 1992;Siniatchkin, Riabus et al. 1999). De Benedittis et al.(De Benedittis and Lorenzetti 1992) investigated the relationship between minor life events (i.e. daily hassles) and personality patterns from selected scales of MMPI in the persistence of primary headache in 83 patients. Their results indicated that between headache subgroups TTH patients are much more likely than those with migraine to have experienced high level of microstress (hassles density), with mixed headache in between. There were no significant differences due to sex, age, headache history and status, except for the headache density. The authors concluded that it is likely that high-stress levels are due, at least in part, to greater density of pain, rather than to discrete headache syndromes. Consequently, since psychological distress seems to be positively associated with pain density, its exact role in the pathogenesis TTH remains unclear.

3.2.3.5 Migraine and tension-type headache comorbidity

Migraine and TTH coexist frequently in the same person. Tension vascular headache, combination headache, mixed headache, or vascular and muscle-contraction headache are examples of terms used in the literature when referring to mixed TTH and migraine headache disorders. Clinical observations indicate that many migraine patients suffer also TTH episodes (Newland, Illis, Robinson, Batchelor, and Waters 1978;Langemark, Olesen et al. 1988;Iversen, Langemark, Andersson, Hansen, and Olesen 1990;Johannes, Linet, Stewart, Celentano, Lipton, and Szklo 1995). The IHS recommends an independent diagnosis for each type of headache in patients suffering from “mixed headache” disorders.

3.2.4 Chronic daily headache

The International Headache Society (IHS) classification published in 1988 has been one of the most important disease classifications. Rapid advancement in headache knowledge during the last decade, however, has made a revision necessary (Olesen 2001). Changes in the TTH group are likely to be small in this new edition, while several changes have been proposed for migraine classification. While the IHS classification distinguishes between chronic and episodic variants for most primary forms of headache, it does not provide for a frequent form of migraine. During the last decades, the terms transformed migraine and CDH have been commonly used in the literature when referring to the frequent form of migraine, although there has not been consensus regarding their exact meanings. Both terms are profusely used in the literature to describe a chronic form of migraine, while other authors also used them with a generic connotation that group different forms of daily headaches. Transformed migraine and CDH are also frequently used in the literature as a form of headache associated with analgesic overuse. Different authors have used the term substance "rebound headache" when describing CDH.

Most authors have historically considered migraine and TTH distinct entities. The pathogenesis of migraine was assumed to be vascular, whereas TTH was believed to have a myogenous origin. Additionally, CDH was considered a "subform" of TTH. Consequently, those headaches that presented a constant or daily pattern, with a bilateral location, a mild to moderate pain intensity, and no worsening with physical activity were diagnosed as CTTH. In addition, CTTH was associated with only one of the following symptoms: nausea, photophobia or phonophobia. Patients with CDH, however, often experience significant variation in their headache symptoms, experiencing a periodic disabling headache superimposed on the baseline headache pattern that fluctuates in intensity. Some of these headache "outbursts" may have

migraine characteristics (i.e transformed migraine) while others may not. In addition, these headache patterns may also be associated with medication overuse and or psychological distress (Juang, Wang et al. 2000;Wang and Juang 2002). Therefore, investigating how the headache pattern began and evolved is critical when distinguishing between transformed migraine and CTTH. Consequently, in 1994 Silberstein et al. recommended revising the IHS criteria for chronic, frequent primary headache disorders and proposed adding several headache subtypes to the current IHS classification (Silberstein, Lipton et al. 1994). Their intention was to make the IHS criteria more comprehensive by providing a biological niche for transformed migraine. Two years later, however, these authors modified their own "revised classification" facilitating the classification criteria for transformed migraine (Silberstein, Lipton, and Sliwinski 1996). According to their results during a field trial of 150 consecutive patients with CDH, 43% and 25% of the patients could not be classified using the IHS criteria and their 1994 criteria respectively. However, using their new criteria, they were able to classify 100%. Seventy-eight percent had transformed migraine, 15.3% had CTTH, and 6.7% had "other" headache disorders, including new daily persistent headache and hemicrania continua. For reasons of symmetry, however, transformed migraine will be referred to as "chronic migraine" in the revised IHS classification (Olesen 2001;Goadsby and Boes 2002). Consequently, the term chronic migraine should be used when referring to patients who have a frequent migrainous headache.

New daily persistent headache as described by Silberstein et al. refers to a heterogeneous group of headache disorders that begin as daily headaches with no previous history of migraine, TTH, or head trauma (Silberstein, Lipton, Solomon, and Mathew 1994). Simultaneously, patients may sometimes recall the day and time that the headache began. This group of headache patients requires diagnostic evaluation unless the etiology of the headache can be clearly defined. Several

authors have postulated that new daily persistent headache may be caused by a form of systemic viral infection. Indeed, several studies have observed a high prevalence of Epstein-Barr virus infections in patients with new daily persistent headache (Diaz-Mitoma, Vanast et al. 1987;Hamada, Ohshima et al. 1991).

Hemicrania continua is a *rare, indomethacin-responsive headache disorder characterized by a continuous but fluctuating, moderately-severe, unilateral headache* (Silberstein, Lipton, Solomon, and Mathew 1994). Hemicrania continua is accepted as a clinical entity by the vast majority of the scientific community despite not being included in the 1988 IHS classification. It will, however, be incorporated in the upcoming 2003 IHS classification.

3.3 TMD and headache comorbidity

Pericranial forms of nociception have been strongly involved in the pathophysiological mechanism of both migraine and TTH (Olesen and Schoenen 2000). Since TMDs are one of the most common sources of nociceptive input in the trigeminal system, they may be frequently involved in the pathophysiology of migraine and TTH. Headaches have been reported to be as frequent as 70% in a TMD population, representing one of the most challenging complaints for the clinician treating TMD (Magnusson and Carlsson 1978;Wanman and Agerberg 1987). As mentioned before, the IHS classification includes temporomandibular disorders as a cause of headaches (Points 2.1.1, 2.2.1, 11.6 and 11.7) (1988). Pericranial muscle tenderness is a common complaint in patients suffering tension type and/or migraine headaches. Consequently the IHS subdivides ETTH and CTTH into “associated with disorder of pericranial muscles” and “unassociated with disorders of pericranial muscles”.

There are numerous studies in the literature supporting an association between TMD and different forms of headaches (Reik, Jr. and Hale 1981;Lous and Olesen 1982;Forssell and Kangasniemi 1984a;Forssell, Kirveskari, and Kangasniemi 1985;Wanman and Agerberg 1986;Jaeger 1989;Mennell 1989;Schokker, Hansson, and Ansink 1990;Mohlin, Pilley, and Shaw 1991;Jensen, Rasmussen, Pedersen, Lous, and Olesen 1992;Haley, Schiffman, Baker, and Belgrade 1993;Jensen, Rasmussen, Pedersen, and Olesen 1993;Jensen, Bendtsen, and Olesen 1998;Pettengill 1999;Aaron, Burke, and Buchwald 2000;Liljestrom, Jamsa, Le Bell, Alanen, Anttila, Metsahonkala, Aromaa, and Sillanpaa 2001;Ciancaglini and Radaelli 2001;Graff-Radford 2001). Pettengill (1999) compared a TMD group of patients with a non-TMD group for recent headache symptoms, TMD symptoms and gender differences. The author found that the TMD groups had a greater severity of headache symptoms than the non-TMD group. Ciancaglini et al. (2001) found a significant relationship of headache with TMD in a personal interview survey on 483 adult subjects in the general population of Segrate, Italy. Haley et al. (1993) when comparing 56 sequential headache patients with a group of TMD patients and an asymptomatic population group, found that migraine and TTH patients had significantly more pericranial and neck muscle tenderness than a general population. These and other studies support the role of muscle and joint nociception as triggers of tension-type and migraine headaches.

Mongini et al. (Mongini, Ciccone, Ibertis, and Negro 2000) analyzed the personality characteristics and anxiety levels of 243 consecutive patients by using the MMPI and the Spielberger State and Trait Anxiety Inventory (STAI). Four different MMPI clusters (depressive, conversive, emotional, coper) were also considered. After history taking and clinical examination the patients were assigned to one of the following diagnostic groups: TMJ intracapsular disorder (n = 71), tension-type headache (n = 52), migraine (n = 68), chronic daily headache (CDH, n =

26), or facial pain disorder as somatoform disorder (n = 26). Their results revealed that the TMJ group had significantly lower scores of several MMPI and of state anxiety. The TMJ group also revealed significantly lower odds ratio values < 1 for all symptoms except phobias and for emotional, conversive, and depressive MMPI profiles. The facial pain and CDH groups, conversely, had higher MMPI and STAI scale elevations. The authors concluded that some types of headache and facial pain seem to correlate with the presence of a number of accompanying symptoms and with some changes in personality. These changes are particularly significant in CDH and facial pain disorder populations. On the contrary, patients with TMJ intracapsular disorders tended to show a low prevalence of accompanying symptoms and a normal personality profile. Nevertheless, the results of this study, although important, should be interpreted with caution since the study design did not control for pain duration and intensity between the different diagnostic groups.

3.4 Conclusions

The number of studies addressing the relationship between headaches and somatic and psychological conditions is profuse in the literature. Consequently, during the last decades several lines of evidence have demonstrated a strong comorbidity between headache disorders and several forms of craniofacial nociception. Moreover, several authors have postulated that different pericranial forms of nociception (i.e. MP) may play a crucial role in TTH and migraine genesis (Olesen 1991c;Olesen, Tfelt-Hansen, and Welch 2000a). Indeed, several studies have shown a strong correlation between TMD and headache disorders, therefore supporting the potential role of craniofacial pain in the genesis of headaches (Reik, Jr. and Hale 1981;Lous and Olesen 1982;Forssell and Kangasniemi 1984b;Forssell and Kangasniemi 1984c;Forssell,

Kirveskari, and Kangasniemi 1985;Wanman and Agerberg 1986;Jaeger 1989;Haley, Schiffman, Baker, and Belgrade 1993;Jensen, Rasmussen, Pedersen, and Olesen 1993;Schiffman, Haley, Baker, and Lindgren 1995;Jensen and Rasmussen 1996;Jensen, Bendtsen, and Olesen 1998;Ciancaglini and Radaelli 2001;Graff-Radford 2001). However, studies addressing somatic and psychological differences between TMD and headache patients are scarce in the literature (Mongini, Ciccone, Ibertis, and Negro 2000). Therefore, more studies are necessary to determine whether TMD patients present unique physiological and psychological alterations when compared to headache patients.

Sleep disturbances are frequently observed in headache and TMD patients, although their exact role in the pathogenesis of both primary headache and TMD disorders remains unknown (Rasmussen 1993;Bruni, Fabrizi, Ottaviano, Cortesi, Giannotti, and Guidetti 1997;Magnusson, Egermark, and Carlsson 2000;Krymchantowski and Moreira Filho 2000;Riley, III, Benson, Gremillion, Myers, Robinson, Smith, Jr., and Waxenberg 2001;Spierings, Ranke et al. 2001). While the majority of studies addressing sleep and headache disorders found no statistically significant difference between the different primary headache populations, a detailed analysis of the available TMD literature revealed that myofascial pain patients report poorer sleep quality than TMJ pain patients (Lindroth, Schmidt et al. 2002). Nevertheless, a great number of authors believe that sleep disturbance seems to be a consequence or cause of pain *per se*, not being necessarily related to a specific pain disorder (Yatani, Studts et al. 2002).

3.5 Hypothesis

The association between headache and TMD is well documented in the literature (Reik, Jr. and Hale 1981;Lous and Olesen 1982;Forssell and Kangasniemi 1984b;Forssell and

Kangasniemi 1984c;Forssell, Kirveskari, and Kangasniemi 1985;Haley, Schiffman, Baker, and Belgrade 1993;Jensen, Rasmussen, Pedersen, and Olesen 1993;Schiffman, Haley, Baker, and Lindgren 1995;Jensen and Rasmussen 1996;Jensen, Bendtsen, and Olesen 1998;Ciancaglini and Radaelli 2001;Graff-Radford 2001). Numerous studies have reported a strong comorbidity between headaches and TMD, therefore suggesting a role of TMD in the pathogenesis of headache disorders.

A number of studies have studied the relationship between psychological factors and TMD (Eversole, Stone, Matheson, and Kaplan 1985;Harness, Donlon, and Eversole 1990;Glaros 2000). Several lines of evidence support a higher probability of emotional problems among masticatory MP patients compared to TMJ pain patients, although the reason for these differences requires further investigation. The more diffuse nature of muscle pain and its higher capacity to generate central excitatory effects may account for some of the psychological differences observed between these two pain populations.

We postulate that CDH and MP patients would report significantly higher psychological distress levels than TMJ pain patients. Additionally, we postulate that CDH and MP would present similar psychological profiles. Our hypothesis is based on our belief that psychological distress levels may be partly related to the nature of pain itself and not necessarily related to a difference in pain intensity and/or duration among the different study populations. Since CDH and MP populations present a more poorly localized pain pattern, with a higher capacity to generate central excitatory effects than TMJ pain patients, we postulate that CDH and MP patients would present higher psychological distress levels than TMJ pain patients. Hence, MP patients and CDH patients would present similar psychological profiles. Our hypothesis that MP and CDH patients would present similar levels of psychological distress agrees with other

previously published studies (Mongini, Ciccone, Ibertis, and Negro 2000). Our hypothesis that masticatory MP and CDH patients would present higher levels of psychological distress than TMJ pain patients is also in accordance with other previously published studies (Eversole, Stone, Matheson, and Kaplan 1985;Harness, Donlon, and Eversole 1990;Glaros 2000;Mongini, Ciccone, Ibertis, and Negro 2000;Lindroth, Schmidt, and Carlson 2002). We also postulate that MP patients will present similar sleep disturbance levels than CDH patients. Both groups, however, will present significantly higher sleep disturbance levels than TMJ pain patients.

4. Material and Methods

During the study period, a total of 100 consecutive adult headache patients were seen at the Orofacial Pain Center of the University of the Kentucky Dental School. Prior to the initial examination, all patients completed an orofacial pain questionnaire and a battery of psychological questionnaires. The psychological questionnaires included the Symptom Check List-90-Revised (SCL-90-R; Derogatis, 1979), the Pittsburg Sleep Questionnaire (PSQ; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), the PCL questionnaire (Blanchard, Jones-Alexander et al. 1996), and the Multidimensional Pain Inventory (MPI; Kerns, Turk, & Rudy, 1985). These questionnaires cover a wide range of symptoms and behaviors that are important in the development of a comprehensive treatment/management plan for the patient.

The orofacial pain examination began with a detailed history of the patient's chief complaint(s), associated symptoms, TMJ noise, mandibular dysfunction, para-functional habits, past facial trauma, past medical history, family medical history, previous treatments/consultations for their chief complaint(s), previous and present medications for their chief complaint(s), as well as their psychosocial history. The subsequent physical examination included measurements of vital signs (blood pressure and heart rate), cranial nerve examination, cervical and mandibular range of movements with notation of associated pain, TMJ palpation and auscultation, cervical and masticatory muscle palpation with notation of associated pain and/or presence of myofascial trigger points, and intraoral examination.

The examinations were conducted by dentists with advanced training in the diagnosis of orofacial pain conditions. No formal reliability data were collected, but all examiners were trained in the Orofacial Pain Center of the University of Kentucky within the guidelines of the American Academy of Orofacial Pain (American Academy of Orofacial Pain and Okeson 1996).

The information obtained provided the basis for primary and secondary diagnoses. The IHS classification criteria (1988) were used for headache diagnoses except in those patients who fulfilled the criteria for chronic daily headache (CDH) (number of headache days was >15 days/month, and >4 hours/day). Correspondingly, patients who fulfilled the criteria for CDH were diagnosed according to the revised IHS criteria for CDH proposed by Silberstein et al. (Silberstein, Lipton, and Sliwinski 1996). The patients were classified, on the basis of their history and clinical examinations, as suffering from CDH (n=67) and "other" headaches (n=33). Consequently, only patients fulfilling a diagnosis of CDH were selected for the study. CDH patients were diagnosed into three mutually exclusive diagnoses, that is, transformed migraine (n=35), chronic tension-type headache (n=26), and "other CDH" (n=6). The "other CDH" group was comprised of patients who did not fulfill the criteria of either transformed migraine or chronic tension-type headache.

The CDH patients were matched by age, sex, pain intensity, and pain duration with 67 patients seen in the Orofacial Pain Center with a primary diagnosis of myofascial pain (MP), and 67 patients with a primary diagnosis of TMJ intracapsular pain (IC) according to the Research Diagnostic Criteria (RDC) for TMD (Dworkin and LeResche 1992). Consequently, The IC group was composed of patients diagnosed with disc displacement with reduction (RDC group IIa), disc displacement without reduction with or without limited mouth opening (RDC group IIb and IIc), arthralgia (RDC group IIIa), and/or osteoarthritis (RDC group IIIb). All patients with a diagnosis of disc displacement were also diagnosed with arthralgia or osteoarthritis as a secondary diagnosis. The MP group was composed of patients diagnosed with MP without limited mouth opening (RDC Ia), and MP with limited opening (Ib). MP patients with a secondary diagnosis of TMJ pain, and TMJ pain patients with a secondary diagnosis of MP, were

not selected for this study. Patients with a secondary and/or tertiary diagnosis of primary headache were also excluded from the study. The patient's data were collected following the same clinical and diagnostic procedures as for the headache patients. The overall patient population was therefore comprised of 201 subjects (CDH: n=67; MP: n=67; IC: n=67).

Physical Measures

The physiological measures of Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), and heart rate (HR) were recorded using a Paramed 9200 automated blood pressure cuff. The cuff was placed on the patient's left arm.

Psychological Measures

The SCL-90-R (Derogatis 1979) is a 90-item multi-dimensional self-report inventory that measures nine dimensions of psychological functioning, including somatization (SOM), obsessive-compulsive behavior (OC), interpersonal sensitivity (IS), depression (DEP), anxiety (ANX), hostility (HOS), phobic anxiety (PHOB), paranoid ideation (PAR), and psychoticism (PSY). Test-retest reliabilities range from r=0.78 to 0.90 for non-patient samples, and internal consistencies range from 0.77 to 0.90.

The MPI (Kerns, Turk et al. 1985) assesses pain severity, social and physical activities, affective distress, social support, and feelings of life control. It also classifies respondents into one of six categories: adaptive coper, dysfunctional, interpersonally distress, hybrid, anomalous, and unanalyzable. Test-retest reliabilities of scale scores range from r= 0.68 to 0.86, and internal consistencies range from 0.73 to 0.90.

The PSQI (Buysse, Reynolds et al. 1989) requests information regarding the number of hours spent in bed and asleep, frequency and reasons for awakening, and difficulty returning to

sleep after awakening. The PSQI has been shown to be a valid and reliable assessment to overall sleep quality and disturbance (Buysse, Reynolds, Monk, Bernman, and Kupfer 1989;Carpenter and Andrykowski 1998), with good test-retest reliability (r=0.85) and internal consistency (alpha= 0.83).

The PCL-C (Blanchard, Jones-Alexander, Buckley, and Forneris 1996) questionnaire is a 17-item measure designed to assess symptoms of Post-Traumatic-Stress-Disorder (PTSD). Patients are asked to report problems or complaints they may have experienced in the last month in response to a stressful situation. Responses include, but are not limited to, flashbacks, distressing dreams, hyper-vigilance, impaired concentration, and avoidance behaviors. The PCL has exhibited test-retest stability ($\underline{r}$ = 0.96), good overall internal consistency ($\underline{\alpha}$ = 0.92), and provides a valid and reliable assessment of the presence of PTSD symptoms.

The orofacial pain questionnaire asks patients to describe their pain experience using descriptors from the McGill Pain Questionnaire (Melzack 1979). These descriptors are divided into sensory and affective classifications. The sensory category contains terms such as throbbing, shooting, stabbing, and aching, while the affective category contains terms such as sickening, exhausting, and punishing. The questionnaire also asks patients about the number and types of specialists seen, treatments pursued in previous attempts to eliminate their pain, including massage, surgery, counseling, or medication(s), and diagnostic tests they had received.

Statistical Analysis

Analysis of variance (ANOVA) was used to analyze the differences between the three groups on pain severity, pain duration, affective and sensory pain descriptors. Chi-square tests were used for analyzing group differences on MPI profile classifications, life stressors, and use

of alternative treatments. Additionally, differences between the three groups on psychological and sleep quality characteristics, as measured by the SCL-90-R, MPI, PCL-C, PSQI subscales, were tested with multivariate analysis of covariance (MANCOVA), controlling family wise error by adopting a stringent alpha level ($\alpha = 0.01$).

TABLE 1
Proposed IHS Classification for Chronic Tension-Type Headache; Silberstein et al. 1996.

A. Average headache frequency more than 15 days/month (180days/year) with average duration of 4 hours/day (if untreated) for 6 months fulfilling criteria B-D listed below.
B. At least two of the following characteristics:
    1. Pressing/tightening (non-pulsatile) quality
    2. Mild or moderate intensity (may inhibit but does not prohibit activities
    3. Bilateral location
    4. No aggravation by physical activity
C. History of episodic tension-type headache in the past
D. Both of the following:
    1. No vomiting.
    2. No more than one of the following: nausea, photophobia or phonophobia.
E. At least one of the following:
    1. History, physical, and neurological examinations do not suggest one of the disorders listed in group 5-11
    2. History and/or physical and/or neurological examinations do suggest such disorder, but it is ruled out by appropriate investigations
    3. Such disorder is present, but tension-type headache does not occur for the first time in close temporal relation to the disorder

TABLE 2
Proposed IHS Criteria for Transformed Migraine; *Silberstein et al. (1996).*

E. Daily or almost-daily (>15 days/month) head pain for >1 month.
F. Average headache duration >4 hours/day (if untreated).
G. At least one of the following:
    1. History of episodic migraine meeting any IHS criteria 1.1 to 1.6.
    2. History of increasing headache frequency with decreasing severity of migrainous features over at least 3 months.
    3. Current headache meets IHS criteria for migraine 1.1 to 1.6.
H. At least one of the following
    1. There is no suggestion of one of the disorders listed in group 5-11
    2. Such disorder is suggested, but it is ruled out by appropriate investigations.
    3. Such disorder is present, but first migraine attacks do not occur in close temporal relation to the disorder.

5. Results

Preliminary analysis

The CDH sub-populations (transformed migraine, chronic tension-type headache, and "other CDH") that make up the CDH group for this study were analyzed on a number of domains to determine if there were any significant differences between these diagnostic categories that might preclude grouping them together for further comparison with the MP and IC diagnostic groups. There were no significant differences between the CDH sub-populations on all physiological and sleep quality domain variables, including pain severity, pain duration, affects and sensory pain descriptors, psychological characteristics (SCL-90-R dimension scores, MPI scale scores), sleep quality scores, and post traumatic stress symptoms.

Participants

The patients in the study were 201 individuals seen at the Orofacial Pain Center at the University of Kentucky College of Dentistry between January of 1997 and August of 2002. The patients were between 18 and 69 years old with a mean age of 36.5 years (SD = 11.4). The overall sample was 81 percent female, with 54 females and 13 males in each group (CDH, MP, IC). There was no significant difference in age between the three diagnostic groups ($F=.018$, $p > 0.05$). Demographic characteristics regarding the three diagnostic groups are displayed in Table 3.

Pain Severity and Duration

Differences in pain severity were determined by comparing scores on the MPI pain severity scale between the groups. Mean pain severity for the groups was as follows: CDH

group mean = 41.8, SD = 11.6; MP group mean = 43.7, SD = 10.8; IC group mean = 38.6, SD = 11.1. There was an overall significant difference between the three groups on pain severity (F=3.68, p=.027). Post-hoc comparisons showed a significant difference between the MP and IC groups (p=.024), with no significant difference between the CDH group and the other two groups.

Pain duration was similarly tested between the groups. Mean pain duration for the groups was as follows (in months): CDH group mean = 74.7, SD = 96.2; MP group mean = 66.2, SD = 78.8; IC group mean = 42.1, SD = 54.2. There was a significant difference between the three groups on pain duration (F=3.13, p=.046). Post-hoc comparisons showed a significant difference between the CDH and IC groups (p=.05), with no significant difference between the MP group and the other two groups. Due to the significant differences between groups on pain severity and duration, these variables were controlled for in the remaining multivariate analyses by treating them as covariates.

Affect and Sensory Pain Descriptors

The descriptive terms used to differentiate between sensory pain experiences and affective pain experiences from the MPQ in the medical/dental history questionnaire were used to determine if there was a significant difference in how the three diagnostic groups classified their experience of pain. The sums of the number of affective descriptors and sensory descriptors endorsed by each patient were calculated and a comparison was made between the groups. The CDH group endorsed more affective descriptors of their pain experience (M [Mean] =.94, SD = 1.1) than the IC group (M = .36, SD = .60), p = .001. There was no significant difference between the number affective descriptors endorsed between the MP group (M = .72,

SD = .88) and the other two groups. When the groups were compared on the sum of sensory descriptors of pain, the MP group endorsed more sensory descriptors (M = 3.3, SD = 2.2), than the IC group (M = 2.3, SD = 1.5), p = .008. There was no significant difference between the number of sensory descriptors endorsed between the CDH group (M = 2.9, SD = 1.8) and the other two groups.

Psychological Characteristics

When patient psychological symptoms were compared, there were statistically significant differences (p's < 0.01) between the three groups on four of the nine dimensions of the SCL-90-R (somatization, obsessive-compulsive, depression, and anxiety). The groups also showed a statistically significant difference on the Global Severity Index (GSI; p<.01), which is a composite measure of overall distress. The GSI combines information concerning the number and intensity of symptoms reported (Derogatis,1979). These results are presented in Table 4. Post-hoc comparisons showed significant differences between the MP and IC groups on all four of the dimensions (all p's<.01), with the MP reporting more psychological distress in each case. The CDH group was significantly different than the IC group on somatization (p<.05). There was also a significant difference between the MP and IC groups on the GSI (p<.01). There were no other significant differences between the CDH group and the other groups on the SCL-90-R dimensions. Figure 1 depicts a graphical description of SCL-90-R mean T-scores for the three diagnostic groups.

There were significant differences between the three groups on the life interference and life control subscales of the MPI (see Table 5). Post-hoc comparisons showed significant differences between all three groups on life interference (p's<.01) with the CDH group reporting

the most interference and the IC group reporting the least. There was also a significant difference between the IC and the other two groups on life control and affective distress, with the IC group reporting more control (p's < .01) than both the CDH and MP groups. There were no other statistically significant differences noted between the groups on the subgroups of the MPI.

The groups were also compared on the number of patients who met criteria for the different profile classifications on the MPI (Table 6). The distribution of MPI profile classifications is shown in Table 6. Chi-square analysis indicated a significant difference between the groups on the number of patients in each profile classification ($X^2 = 21.6$, $p = .017$). Specifically, there were more dysfunctional and interpersonally distressed profiles in both the CDH and MP groups than in the IC group.

Sleep Quality

The PSQI gives a total score, representing overall sleep quality, as well as several subscale scores. The three groups showed statistically significant differences on the PSQI total score, sleep duration, sleep disturbances, use of sleep medication, and daytime dysfunction (Table 7). Post-hoc comparisons indicated more overall sleep dysfunction in the MP group, than in the IC and CDH groups (p's<.01). The MP and the CDH groups reported significantly more daytime dysfunction (p<.01) than the IC group. Compared to the CDH group, the MP group also reported significantly poorer sleep duration (p <.01. The CDH group reported significantly more daytime dysfunction than the IC group (p<.01). There were no other significant differences between the groups on the PSQI scores.

Post Traumatic Stress Symptoms

The total score obtained from the PCL was used to determine if there was a significant difference in PTSD symptoms between the three diagnostic groups. There was a significant difference between the number of patients reporting serious life stressors between the three groups ($\chi^2 = 7.16$ p=.028) with the CDH and MP group reporting more life stressors than the IC group. There was no significant difference, however, in the PCL total score, or on the PCL subscales (reexperiencing, avoidance, and arousal) between the three groups in the multivariate context (p's>.01), indicating no difference in traumatic experiences or PTSD symptoms between the groups (Table 8 and 9).

Physical Measures

During the initial visit to the Center, diastolic blood pressure (DBP), systolic blood pressure (SBP), and heart rate (HR) were taken and recorded. There were no significant difference found between the three diagnostic groups for SBP (CDH group: M =130.1, SD = 23.2; MP group: M = 132.3, SD = 22.3; IC group: M = 128.9, SD = 18.1), DBP (CDH group: M =73.3, SD = 14.7; MP group: M = 74.9, SD = 13.0; IC group: M = 70.7, SD = 14.1), or HR (CDH group: M =78.6, SD = 13.9; MP group: M = 75.5, SD = 11.7; IC group: M = 76.7, SD = 11.0).

Alternative Specialists, Treatments, and Tests

The medical/dental history questionnaire also included information regarding various types of treatment modalities, specialists seen, and diagnostic tests the patients had employed for symptom relief prior to seeking treatment in the Orofacial Pain Center. There was no significant

difference in the number of alternative treatments received between the groups (F=1.63, p=.20; CDH group: M =3.3, SD = 3.0; MP group: M = 2.7, SD = 3.0; IC group: M = 2.4, SD = 3.0). The only specific treatment that the CDH group used significantly more often than the IC or MP groups was injections (X^2 = 10.3, p = .006). There was marginal support for traction (X^2 = 7.2, p = .027) and chiropractic treatment (X^2 = 6.3, p = .044).

Overall, there were no significant differences in the number of diagnostic tests the patients had received (F=1.97, p=.14; CDH group: M =1.4, SD = 1.2; MP group: M = 1.0, SD = 1.4; IC group: M = 1.0, SD = 1.3), or the number and types of specialists seen prior to coming to the Center between the groups (F=1.34, p=.27; CDH group: M =3.4, SD = 2.2; MP group: M = 2.8, SD = 2.1; IC group: M = 2.9, SD = 2.7). The only specific diagnostic test that the CDH group used significantly more often than the IC or MP groups was CT scan (X^2 = 10.6, p = .005), although there was also a marginal support for MR scan (X^2 = 6.1, p = .048). Additionally, the CDH group reported more use of their family physician (X^2 = 11.6, p = .003), and had visited a neurologist more often (X^2 = 30.0, p = .000) than the other two groups. The IC group reported more visits to an oral surgeon (X^2 = 11.3, p = .004) than the CDH and MP groups.

TABLE 3
Demographic Characteristics by Diagnostic Group.

	CDH Group	MP Group	IC Group
Marital Status			
Married	33	37	29
Single	15	9	14
Divorced/ Separated	5	8	4
Missing	14	13	20
Employment Status			
Full time	30	31	24
Part time	5	3	11
Unemployed	9	10	8
Disabled	5	6	3
Retired	0	2	2
Student	1	1	0
Missing	15	16	19

CDH= Chronic Daily Headache. MP= Myofascial Pain. IC= Intracapsular Pain.

TABLE 4
SCL-90-R Symptom Dimension Means and Standard Deviations.

	CDH Group (n=64) Mean (SD)	**MP Group (n=65) Mean (SD)**	**IC Group (n=67) Mean (SD)**	**F(2,191)[a]**	**p**
Somatization	62.7[b] (7.6)	65.2[b] (10.7)	58.4[c] (9.0)	6.29	.002
Obsessive-compulsive	59.7[b,c] (11.8)	63.0[b] (9.6)	55.9[c] (11.3)	4.83	.009
Interpersonal Sensitivity	56.5 (10.9)	59.5 (11.9)	53.6 (10.4)	3.51	.032
Depression	58.5[b,c] (11.1)	61.8[b] (9.9)	54.5[c] (10.5)	5.71	.004
Anxiety	56.9[b,c] (11.7)	60.2[b] (11.5)	52.9[c] (11.4)	4.68	.010
Hostility	54.8 (10.3)	58.4 (10.8)	52.8 (9.9)	3.37	.036
Phobic anxiety	51.2 (9.7)	54.2 (11.0)	49.9 (8.8)	2.18	1.16
Paranoid ideation	51.6 (11.2)	55.1 (11.3)	50.3 (9.5)	2.31	.102
Psychoticism	55.8 (11.7)	58.2 (11.8)	52.5 (10.8)	2.72	.069
Global Symptom Index	59.7[b,c] (10.5)	63.4[b] (9.8)	56.2[c] (10.4)	5.82	.004

Note. [a]Associated with analysis of covariance, with pain severity and pain duration as covariates. [bc]When superscripts are the same between two groups on a measure, post-hoc comparison indicate no significant difference in group means. When superscripts are different, post-hoc comparison indicate significant difference between group means at $p<.01$. SCL-90-R = Symptom CheckList 90 – Revised. CDH= Chronic Daily Headache. MP= Myofascial Pain. IC= Intracapsular Pain.

TABLE 5
Multi-dimensional Pain Inventory Means and Standard Deviations.

	CDH Group (n=67) Mean (SD)	**MP Group (n=67) Mean (SD)**	**IC Group (n=67) Mean (SD)**	**F(2,199)[a]**	**p**
Life interference	36.4[b] (14.7)	32.7[b] (14.9)	25.5[c] (13.7)	9.8	.000
Life control	49.6[b] (6.7)	49.3[b] (7.1)	53.0[c] (6.5)	6.20	.002
Affective distress	46.4[b,c] (8.6)	49.0[b] (9.3)	43.4[c] (10.6)	5.71	.004
Support	48.1 (10.5)	49.1 (9.9)	47.2 (10.3)	.498	.608
Punishing Responses	46.3 (8.8)	47.4 (8.9)	44.0 (6.2)	2.40	.094
Soliciting responses	50.7 (9.7)	50.7 (10.5)	47.3 (10.4)	1.91	.151
Distracting responses	47.4 (9.4)	49.5 (9.8)	47.2 (9.9)	1.01	.366
Household chores	55.2 (7.9)	55.0 (9.5)	54.8 (11.6)	.038	.963
Outdoor work	53.6 (9.2)	53.4 (11.2)	57.2 (12.9)	2.51	.084
Activities away from home	54.1 (10.1)	52.1 (10.4)	54.1 (10.2)	.832	.437
Social activities	51.4 (8.7)	51.9 (9.9)	53.1 (9.5)	.587	.557
General activity level	55.0 (8.0)	54.4 (10.1)	56.6 (9.8)	.944	.391

Note. [a]Associated with analysis of covariance, with pain severity and pain duration as covariates. [bcd]When superscripts are the same between two groups on a measure, post-hoc comparison indicate no significant difference in group means. When superscripts are different, post-hoc comparison indicate significant difference between group means at $p<.01$. CDH= Chronic Daily Headache. MP= Myofascial Pain. IC= Intracapsular Pain.

TABLE 6
Multi-dimensional Pain Inventory Profile Classification Distribution Between the Diagnostic Groups.

	CDH Group	**MP Group**	**IC Group**
Dysfunctional	14 $_{a}$	14 $_{a}$	8 $_{b}$
Interpersonally distressed	8 $_{a}$	7 $_{a}$	1 $_{b}$
Adaptive Coper	20	25	23
Hybrid	7	4	1
Anomalous	7	10	18
Uninterpretable	11	7	16
	Value		**p**
Pearson Chi-Square	21.6		.017

Means sharing common superscripts were not found to be significantly different.
CDH= Chronic Daily Headache. MP= Myofascial Pain. IC= Intracapsular Pain.

TABLE 7
PSQI Means and Standard Deviations.

	CDH Group (n=65) Mean (SD)	**MP Group (n=59) Mean (SD)**	**IC Group (n=63) Mean (SD)**	**F(2,182)[a]**	**P**
PSQI total score	9.0[b] (4.0)	11.1[c] (4.5)	7.9[b] (4.4)	5.52	.005
Subjective sleep quality	1.5 (.69)	1.7 (.86)	1.3 (.83)	2.26	.107
Sleep latency	1.4 (1.1)	1.6 (.96)	1.3 (1.1)	1.05	.353
Sleep duration	1.0[b] (.83)	1.5[c] (.97)	1.1[b,c] (1.0)	3.62	.029
Sleep efficiency	.72 (.94)	1.2 (1.2)	.75 (1.1)	2.53	.082
Sleep disturbances	1.7 (.59)	1.9 (.73)	1.6 (.59)	3.46	.034
Use of sleep medication	1.2 (1.4)	1.7 (1.4)	.97 (1.3)	3.25	.041
Daytime dysfunction	1.5[b] (.87)	1.4[b] (.75)	.94[c] (.76)	6.02	.003

Note. [a]Associated with analysis of covariance, with pain severity and pain duration as covariates. [bcd]When superscripts are the same between two groups on a measure, post-hoc comparison indicate no significant difference in group means. When superscripts are different, post-hoc comparison indicate significant difference between group means at $p<.01$. PSQI = Pittsburgh Sleep Quality Index. CDH= Chronic Daily Headache. MP= Myofascial Pain. IC= Intracapsular Pain.

TABLE 8
Life Stressor Criteria

	CDH Group	MP Group	IC Group
No	22	23	28
Yes	36[a]	36[a]	20[b]
Missing	9	8	16

	Value	p
Pearson Chi-Square	7.16	.028

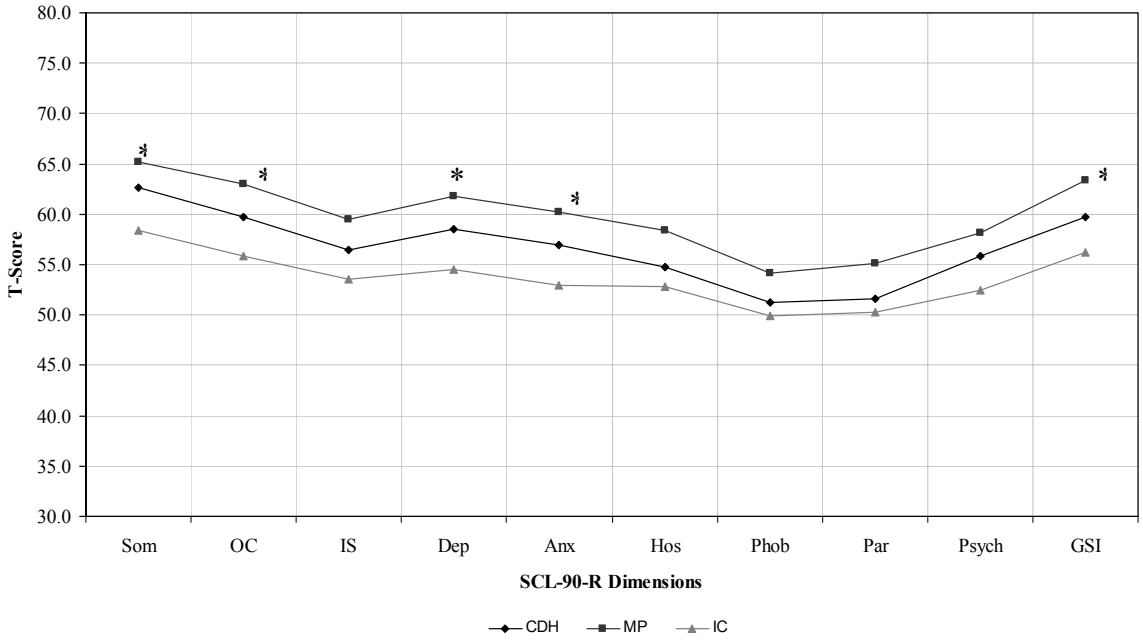
CDH= Chronic Daily Headache. MP= Myofascial Pain.
IC= Intracapsular Pain.

TABLE 9
PCL-C Means and Standard Deviations for patients endorsing a significant stressor.

	CDH Group (n=36) Mean (SD)	**MP Group (n=36) Mean (SD)**	**IC Group (n=20) Mean (SD)**	**F(1,92)[a]**	**P**
PCL Sum	30.8 (12.4)	35.4 (16.7)	31.9 (14.6)	1.69	.161
Avoidance	11.6 (5.2)	13.3 (7.2)	11.6 (5.2)	1.73	.152
Reexperiencing	8.6 (4.0)	10.9 (6.0)	9.8 (5.3)	1.27	.290
Arousal	10.7 (5.1)	11.2 (5.3)	10.6 (5.3)	1.65	.169

Note. [a]Associated with analysis of covariance, with pain severity and pain duration as covariates. PCL-C= Postraumatic Check List-Civilian. CDH= Chronic Daily Headache. MP= Myofascial Pain. IC= Intracapsular Pain.

FIGURE 1
SCL-90-R Dimension Mean T-scores by group



* Indicates statistically significant difference between groups on these dimensions ($p<.01$). SCL-90-R = Symptom CheckList 90 – Revised. CDH= Chronic Daily Headache. MP= Myofascial Pain. IC= Intracapsular Pain.

6. Discussion

Chronic daily headache is the predominant diagnosis among patients visiting headache centers, and represents a major challenge for headache specialists (Solomon, Lipton et al. 1992). Even so, studies analyzing psychological differences between CDH and TMD patients are sparse in the literature (Mongini, Ciccone, Ibertis, and Negro 2000). The present study revealed higher levels of psychological distress among CDH and MP groups than among the IC group on most psychological domains. Although the MP group revealed numerically higher levels of psychological distress on most psychological domains than the CDH group, these differences were not statistically significant.

Our findings are generally consistent with the results of Mongini et al. (Mongini, Ciccone, Ibertis, and Negro 2000) who also found lower levels of psychological distress in TMJ pain patients than in facial pain and CDH patients. Since the Mongini et al. (2000) study did not control for pain duration and intensity in the different diagnostic groups, our findings strengthen their results. In the present sample, we also found statistically significant differences in life stressors with the CDH and MP groups reporting more life stressors than the IC group. There were no statistically significant differences, however, between TMD and CDH patients on the PCL total score or on the PCL subscales, including reexperiencing, avoidance, and arousal. Additionally, none of the patient groups overall scored over 39 on the PCL total score which is generally considered the cut-off point for PCL-C clinical significance (Blanchard, Jones-Alexander, Buckley, and Forneris 1996). These findings suggest that only a small number of patients may be affected by post-traumatic stress in the present sample. This finding is

rather surprising given the evidence from other chronic pain samples that indicate a high prevalence of significant life stressors on these populations (Geisser, Roth et al. 1996;Sherman, Turk et al. 2000;Asmundson, Coons et al. 2002).

Several lines of evidence indicate that PTSD symptoms are most prevalent among chronic pain patients with dysfunctional profiles compared to those with adaptive coper or interpersonally distressed profiles (Asmundson, Bonin et al. 2000). Our study results support this hypothesis since both PCL-C scores and dysfunctional profiles were numerically higher in the MP and CDH groups, than in the IC group. Additionally, only a small percentage of the overall sample presented with dysfunctional profiles, and this may explain the low prevalence of PTSD symptoms.

Research has indicated that the severity of anxiety disorders and life interference may be positively correlated with the severity of PTSD symptoms in chronic pain patients (Fedoroff, Taylor et al. 2000;Asmundson, Coons, Taylor, and Katz 2002). Furthermore, other studies suggest that patients with persistent headache pain and those with musculoskeletal pain who present with dysfunctional profiles tend to have elevated anxiety sensitivity relative to other pain populations (Asmundson, Bonin, Frombach, and Norton 2000). In the present study those patient groups with higher PCL-C scores, that is CDH and MP groups, also revealed the highest levels of anxiety, life interference, and affective distress, the lowest life control scores, and the highest number of dysfunctional profiles. Therefore, anxiety disorders and PTSD symptoms seem to be highly prevalent in patients with dysfunctional patterns of pain behaviors.

Our study also revealed significant differences between the MP and IC group on four of the nine dimensions of the SCL-90-R (somatization, obsessive compulsive,

depression, and anxiety). These groups were also statistically different on the Global Severity Index, which is a composite measure of overall distress. Our results are in agreement with other studies in the field of TMD that indicate higher levels of psychological distress in MP patients than in TMJ pain patients (Eversole, Stone, Matheson, and Kaplan 1985;Harness, Donlon, and Eversole 1990;Lindroth, Schmidt, and Carlson 2002). Michelotti et al. (Michelotti, Martina, Russo, and Romeo 1998), on the other hand, did not observe psychological differences between MP and IC patients , although this may be due to methodological differences such as sample selection criteria, and pain duration and intensity discrepancies between the different study populations .

There is evidence that high levels of pain duration and intensity may be partially responsible for the higher levels of depression and anxiety observed in chronic pain patients (Haythornthwaite, Sieber et al. 1991;Dohrenwend, Raphael et al. 1999;Sokka, Kankainen et al. 2000;Auerbach, Laskin et al. 2001). Therefore, the psychosocial differences observed between MP and TMJ pain patients in previous studies could be attributed directly to a difference on pain intensity and duration among the different pain populations. In light of the current findings and those of Lindroth et al (Lindroth, Schmidt, and Carlson 2002), both of which controlled for pain intensity and duration, there seems to be compelling evidence for higher levels of psychological distress among MP patients as compared to IC patients regardless of pain intensity and pain duration.

It is noteworthy that only the MP group scored 63 or more on two of the SCL-90-R dimensions (somatization and obsessive compulsive behavior). Scores greater than 63-T are considered by most authors as the “cut-off” point for clinical significance (Monsen and Havik 2001). Also neither the CDH nor the IC group scored over 63 on any of the

SCL-90-R dimensions. All groups, however, scored higher than the average general population means in all SCL-90-R dimensions. Given these findings, it is important to screen for major psychological distress when evaluating these chronic pain populations because there is the possibility that such issues may be significant for developing a comprehensive treatment plan.

Sleep disturbances are extremely prevalent among pain populations (Magnusson, Egermark, and Carlsson 2000;Riley, III, Benson, Gremillion, Myers, Robinson, Smith, Jr., and Waxenberg 2001). Few studies, however, have provided information about sleep quality differences between TMD and CDH populations. Our study results showed more overall sleep dysfunction in MP group than in the CDH and IC groups. Our sleep findings are generally consistent with Mongini et al. (Mongini, Ciccone, Ibertis, and Negro 2000) who also observed a higher prevalence of sleep disorders, as identified by the MPI questionnaire, among facial pain patients when compared to primary headache and TMJ pain patients. Their results did not reveal no sleep quality differences between primary headache and TMJ pain patients. To the best of our knowledge, our study is the first to evaluate sleep quality differences between TMD and CDH populations by using a specialized sleep quality questionnaire (PSQI). Consequently, a detailed analysis of our data showed that the MP group also had more daytime dysfunction compared to the IC group. Additionally, the CDH group had significantly poorer sleep duration than the MP group. Furthermore, since our study design controlled for pain intensity and duration, the sleep quality differences observed between our study groups could not be attributed to a difference in pain intensity or duration among the different pain populations. These findings regarding sleep differences may be, therefore, the result of true

pathophysiological differences between IC, MP, and CDH patients. The more diffuse nature of pain and its higher capacity to generate central excitatory effects in CDH and MP patients, may account for some of the psychological differences observed between these pain populations and patients diagnosed with IC pain.

It is possible that sleep disturbances may be a consequence of pain *per se* and are not necessarily related to a specific pain disorder (Yatani, Studts, Cordova, Carlson, and Okeson 2002). Some investigators have reported that while pain intensity and duration may be important in the development of sleep disturbances, other variables such as concomitant fatigue, medication overuse, mood disturbance or depression, may also play an important role in sleep disturbance pathogenesis (Lavigne, Goulet, Zuconni, Morrison, and Lobbezoo 1999;Hering-Hanit, Yavetz, and Dagan 2000). Our study results support this hypothesis since most sleep disturbance and psychological distress scores were significantly higher in the MP and CDH group, than in the IC group.

Sleep disturbance may be a key factor in MP and CDH pathophysiology. It is well established that the balance between hypothalamic growth hormone-releasing hormone (GHRH) and corticotropin-releasing hormone (CRH) plays a key role in normal and pathological sleep regulation. GHRH stimulates deep sleep (stages 3 and 4) and growth hormone (GH) secretion but inhibits cortisol release, whereas CRH disrupts deep sleep and decreases GH secretion. During normal aging and during depression, the GHRH:CRH ratio is distorted in favor of CRH, resulting in disturbances in sleep endocrine activity (Hartman, Veldhuis et al. 1993;Gardi, Obal, Jr. et al. 1999). Since GH is known to play a crucial role in skeletal muscle synthesis and repair, deep sleep deprivation, by altering GH synthesis, may compromise muscle healing in MP patients.

Further research is warranted to examine the effects of GH deficiencies in the pathogenesis of MP disorders.

Hypothalamic dysfunction has also been implicated in transformed migraine pathogenesis. Several authors have reported abnormal patterns of hypothalamic hormonal secretion in transformed migraine patients when compared to normal individuals, including increased cortisol concentrations, and lower melatonin concentrations (Peres, Sanchez et al. 2001). Furthermore, several lines of evidence suggest that a central serotonergic system dysfunction, which has been implicated in pain control and sleep regulation, may be strongly related to hormonal hypothalamic-pituitary-adrenal axis (HPA) alterations in CTTH patients (Rainero, Valfre et al. 2002). Several lines of research also revealed that depression and anxiety disorders may be associated with HPA axis and autonomic nervous system (ANS) up-regulation, and inhibition of vegetative processes likely to impede survival during a life threatening situation (i.e sleep and endocrine programs for growth) (Valdivieso, Duval et al. 1996;Gold and Chrousos 2002). Therefore, psychological distress and sleep disturbances in MP and CDH patients may be both the cause and/or result of ANS and HPA axis dysfunction.

7. Summary

This study confirms and extends previous reports addressing psychological differences between TMD and CDH populations. Although CDH patients revealed lower levels of psychological distress than MP patients, these differences were not statistically significant. Chronic daily headache patients, however, showed higher levels of psychological distress and sleep disturbance than IC patients. However, most of these differences, although sometimes statistically significant, remained within one standard deviation of one another, and this raises questions about their ultimate clinical significance, in this sample at least. Therefore, further research is needed to determine whether CDH, MP, and IC populations require distinct psychological management as a part of a multidimensional treatment approach.

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