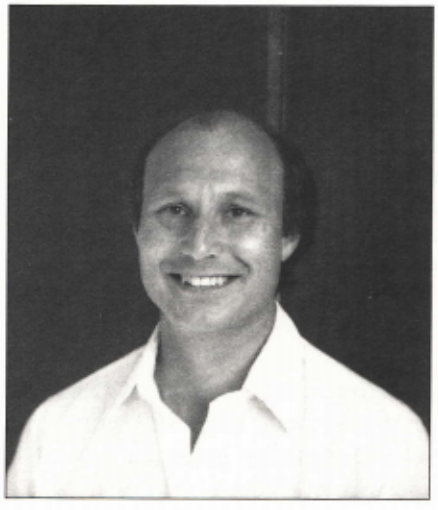
The Nature and Meaning of Tension Headaches — RJ Henbest



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Curriculum vitae

Ronald J Henbest was born in Edmonton, Alberta (Canada) where he qualified in 1974 with a BSc in Maths and Psychology and in 1978 with an MD from the University of Alberta. He then completed two years postgraduate study (residency) in Family Medicine with the Department of Family Medicine at the University of Western Ontario (Canada) and obtained his CCFP from the College of Family Physicians of Canada. Ron joined the Department of Family Medicine at Medunsa in 1980. He has a particular interest in the doctor-patient interaction and its importance for healing. He returned to the University of Western Ontario in 1984 to take their Master of Clinical Science Degree in Family Medicine (MCISc), which emphasizes patient care, teaching and learning, and research. His thesis on Patient-Centred Care involved the development of a method for measuring patient-centredness and testing it against patient outcomes. In 1989, Ron returned to his home city, Edmonton, for a period of 21 months where he was engaged as an associate professor in the Department of Family Medicine at the University of Alberta. During this time, he also completed further training in systemic family therapy. In October 1990, Ron returned, with his wife Judy and four year old son Benji, this time as associate professor and deputy head of the Department of Family Medicine at Medunsa.

Summary

Headache is among the commonest symptoms experienced by mankind. During the past 50 years, a wide range of laboratory and clinical studies have increased our knowledge of this fascinating and complex subject. This paper examines the literature with respect to tension headache, including its classic description, its revised classification and recently agreed-upon diagnostic criteria, and the controversy about its pathogenesis involving muscle spasm, ischaemia, the autonomic nervous system, platelet activation, psychological factors, and the role of stress. It concludes with an attempt to integrate the foregoing information in a discussion on the meaning of tension headache.

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Introduction

Headache is both an old and a common problem. In spite of this, the vast majority of headaches are still considered idiopathic and are still usually classified as tension or migraine headaches even though there is now a fair amount of controversy about whether these two commonest types of headache represent separate and distinct entities, or whether they are part of a continuum.

This is not because too little attention has been paid to headache. In fact, a great deal of important research has been done. Rather, it is a matter of complexity – one of those situations where it seems that the more we know, the more we realize how much more there is to know.

This paper comments on the revised international classification of headache, presents a description of and diagnostic criteria for tension-type headaches, reviews in some detail the current understanding of the pathogenesis of tension headaches, including both physiological and psychological factors and concludes with a discussion on the meaning of tension headaches.

Classification

The basis of almost all classifications of headache for the past thirty years has been that published by the Ad Hoc Committee on Classification[1] in 1962. This classification was a major achievement at the time in that it provided the first truly comprehensive classification of headache and was based on the mechanisms of head pain as determined from the extensive research of Wolff and his colleagues,[2,3,4] dating from the late 1930s. Wolff's work has stood the test of time remarkably well and still provides a great deal of valuable information that has yet to be improved upon.

However, many clinicians have found the very comprehensiveness of the Ad Hoc Committee's classification with its 15 major categories to be impractical for daily use, and those who do much teaching about headaches, often make modifications of the classification to suit their individual conceptualizations.[5,6,7] In addition, the classification provided descriptions rather than definitions, and lacked specific diagnostic criteria.

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This left major categories of headache open to interpretation, causing considerable difficulty both in research and clinical work.

Thus, the new classification published in 1988 by the International Headache Society (IHS)[8] after three years of extensive international collaboration, was a welcome addition to the literature.

... the more we know, the more we realize how much more there is to know!

This classification contains significant differences from the previous one, reflecting a changing understanding of a number of headache entities. In particular, the new classification differs with respect to the most common types of headache encountered in family practice: tension and migraine.

Muscle contraction headaches, as they were labelled by the Ad Hoc Committee, along with the various psychogenic headaches, are now called "tension-type" headache in recognition of the uncertainty and controversy regarding the pathophysiology involved.

Tension-type headaches are categorized as being either "episodic" (less than 180 days/year) or "chronic" (180 days or more/year) and then subdivided into those "associated with disorder of pericranial muscles" and those "unassociated with disorder of pericranial muscles." There is a third category for tension-type headaches not fitting the criteria for the above categories.

Description and Diagnostic Criteria

The classic description for tension headache since 1962, has been that given by the Ad Hoc Committee for muscle contraction headaches, namely: "An ache or sensations of tightness, pressure, or constriction, widely varied in intensity, frequency, and duration, sometimes long lasting, and commonly occipital."[1] The two key features are a dull aching discomfort or a tight, pressure or band-like sensation. These headaches are usually global or bilateral, but some are localized with discomfort either on one side of the head or across the frontal or occipital regions. The neck and trapezius muscles often feel tight or stiff. Less commonly, the discomfort may be localized to the preauricular area due to increased muscle contraction around the temporomandibular joint.

Tension-type headaches may go on for days, weeks, or even months, but are not usually described as being of great intensity. They are nonpulsatile,

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Tension-type headache: more pronounced pericranial tenderness than headache-free controls

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and although occasionally described as thumping or throbbing, this is usually short-lived and not a major feature. These headaches are not accompanied by an aura nor to any extent by photophobia, phonophobia, nausea or vomiting, but dizzyness and fatigue are often mentioned.

Two main patterns have often been recognized.[9] The first is repeated, long, dull aching headaches occurring one to three times per week and thought to be related to situational stress. The second pattern is daylong headaches occuring daily and thus amounting to an almost continuous headache. This second

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Sustained contraction of the scalp and neck muscles seems to be the major cause of discomfort

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pattern is thought to be associated with depressive illness. These two patterns can be recognized in the revised classification of headache in its categorization of tension-type headaches as either episodic or chronic.

As mentioned earlier, one of the problems with the previous classification was a lack of accompanying diagnostic criteria. The IHS Classification Committee has provided operational diagnostic criteria for all categories of headache, using information readily available to the clinician.

The criteria for episodic tension-type headache are as follows: at least 10 previous headache episodes totalling less than 180 days per year with the headaches lasting from 30 minutes to 7 days, no nausea or vomiting (anorexia may occur), photophobia and phonophobia are absent, or one but not the other is present, and at least 2 of the following 4 pain characteristics are present: pressing/tightening (nonpulsating) quality,

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mild or moderate intensity, bilateral location, and no aggravation by walking, climbing stairs, or similar routine physical activity.[8] The criteria for chronic tension-type headache require headache episodes totalling 180 days or more per year. The diagnostic criteria for the subgroup "associated with a disorder of pericranial muscles" is exaggerated pressure tenderness of pericranial muscles and/or increased electromyographic activity.

On all measures the muscle contraction headache group were more vasoconstricted than controls

A recent study[10] done to determine the symptoms of tension headaches involved one hundred and forty-eight patients (92 women and 56 men) with tension headache at least 10 days per month, recruited from a headache clinic, a private neurology clinic and a neurological hospital department. The findings were as follows: 63% of the patients had daily headaches (note the entrance criteria of at least 10 days/month); 63% described their headaches as pressing, 27% as pressing and pulsating mixed, and 10% as pulsating only; 51% had global headaches, another 23% bilateral, and 26% unilateral headaches; the intensity of their usual headaches did not interfere with daily activity in 51%, hampered their activity in 42%, and was inconsistent with normal daily activity in 7%; associated symptoms occurred but were only infrequently described as common: photophobia 13%, phonophobia 7%, anorexia/nausea 8%, and visual disturbance 3%.

Pathophysiology

Research into the pathophysiology of tension-type headaches has never equalled that for migraine. Perhaps migraine headache has always had a certain mystique and perhaps also, it has been assumed that the mechanism of tension headaches was well understood. But there has been renewed interest in the study of tension headaches sparked partly by uncertainty about the muscle contraction hypothesis, partly by the suggestion that migraine and tension headaches may be more similar than previously thought (perhaps even having a common underlying mechanism), and partly by the development of more sophisticated measurement techniques.

Presented below are fairly detailed reviews of the research evidence for the two major theories concerning the pathophysiology of tension-type headaches, namely: (1) the muscle contraction theory, and (2) the ischaemic theory. As well, a brief mention is made of the possible role of the autonomic nervous system and

Patients with tension headaches have lower 5-HT platelet concentrations during headaches than non-headache controls

platelet activation. This review concerns tension-type headaches; that is, those headaches previously referred to as primary muscle contraction and psychogenic headaches. It is well recognised that head pain from any cause, including conditions of the cervical spine, may cause reflex contraction of the skeletal muscles of the head and neck[4] (secondary muscle contraction headaches), but these are not dealt with in this paper.

The Muscle Contraction Theory/ Electromyography

It is well known that muscle spasm exists and that it can produce the kind of pain experienced with tension-type headaches.[11-13] Recent studies have demonstrated that

How we feel greatly colours our experience of life as a whole

patients with tension-type headaches have significantly more pericranial muscle tenderness than headache-free controls[10,14] and it has also been shown that patients with tension headaches have more pronounced pericranial tenderness than patients with both tension and migraine headaches, who in turn, have more tenderness than patients with migraine alone.[14]

Electromyographic (EMG) studies have documented excessive contraction of scalp and neck muscles accompanying headache. The studies done by Wolff and his colleagues[4] remain the most comprehensive. They studied electromyographs of the pericranial muscles both in clinical conditions (such as sinusitis and anxiety) and in experiments with head pain induced by various means (noxious stimuli to the scalp and histamine injections). Wolff concluded from these studies that sustained skeletal muscle contraction

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muscle activity (frontalis) during stress task involvement in tension headache patients.[19,20] Third, significantly higher neck tension has been demonstrated repeatedly in headache patients during headache free periods compared with controls[18,21,22] (usually even higher in migraine headache patients than tension headache patients).[18,21] The same has been shown for frontalis

Daily hassles are more likely than major life stresses to trigger tension-type headaches

and temporalis muscle groups in patients with tension-type headaches.[22] Fourth, greater pericranial muscle (frontalis) activity has been shown in patients with a high frequency of tension headache compared to those with a low frequency of headache.[19] The only exception to the above findings that I am aware of are those of an older study by Bakal and Kaganov (1977) who measured only the EMG potentials from the frontalis muscles and failed to find any differences between a muscle contraction headache group and controls.[23]

A recent study by Pritchard[18] (1988) was rather unique in that the sample consisted of non-medical volunteers sought from the local press who were subsequently assessed at home, in their natural environment, rather than in hospital or clinic. This should have reduced the selection bias inherent in patient samples attending headache clinics and allowed before and during headache measurements without moving the participants, while in pain, to a laboratory for examination. Pritchard found, as have others, high neck tension in headache (both tension and migraine) sufferers even while headache free, as compared with controls. He quite appropriately postulated that this may represent general bodily tension, that is, a "whole-body reaction"[18] to stress, rather than a source of pain. In addition, he found differences between tension and migraine sufferers only while they suffered (that is, when headache was present). The difference was that the output of the occipitalis muscle was significantly higher in those with tension headaches than those with migraine. As a result, Pritchard postulated that occipitalis output may be a potential marker for arousal as its output is relatively uninfluenced by voluntary effort or body movement. We shall come back to the theme of arousal later in this paper.

I shall conclude this section on muscle contraction by noting something familiar to most clinicians. In many, and perhaps most patients, sustained contraction of the scalp and

Tension headache may be part of a defensive response to adversity

neck muscles seems to be the major cause of their discomfort. However, there are patients who describe the same type of pain who do not demonstrate muscle tenderness nor obvious muscle spasm. EMG studies have shown the same – that there are patients without EMG abnormalities who report typical tension-type headaches of similar severity to those with increased EMG activity.[22]

Clearly, there is more than muscle contraction involved in the pathogenesis of tension-type headaches.

The Ischaemic Theory/Vasomotor Reflexes

In contrast to the considerable amount of research on the vasomotor responses accompanying migraine headaches, there has been very little comparable study of tension headache. Cohen,[15] in his 1978 review of vasomotor reflexes in headache, found only two such studies. I have found only one more.

Tension headache sufferers are hyper-responsive to stimuli

The ischaemic theory of pain in tension headache was first proposed by Tunis and Wolff[17] as a result of their now classic study published in 1954. They compared the temporal artery pulse wave of 10 muscle contraction headache sufferers and 10 non-headache controls. The headache patients were measured during and in-between headaches. On all measures, the muscle contraction headache group were more vasoconstricted than controls. Moreover, the headache patients demonstrated greater vasoconstriction during headache than when pain free as evidenced by average pulse wave heights of 4,6 mm and 8,3 mm respectively, and in contrast to the 12,0 mm average for controls. Wolff also presented evidence that the arteries supplying the pericranial muscles responded to generalized noxious stimuli by vasoconstriction and hypothesized that the pain of sustained skeletal

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muscle spasm is enhanced by the presence of ischaemia in the muscle induced by this vasoconstriction.[4] This conclusion was supported by the demonstration of relief of pain by vasodilators and lack of effect or worsening of pain by vasoconstrictors.[4] Many years later, Bakal and Kaganov[23] demonstrated temporal artery vasoconstriction in muscle contraction headache patients to 80 dB white noise in contrast to vasodilation in controls subjected to the same stimulus. Of interest, they found the same response (vasoconstriction) in migraine patients.

More recently, Langemark et al[24] set out to test the ischaemic theory by measuring temporal muscle blood flow using the xenon 133 clearance technique in 40 patients with chronic tension-type headache and 13 controls. They failed to find any differences between headache patients and controls in any of the blood flow parameters, including resting blood flow and measurements made during isometric work, where blood flow increased approximately fivefold in both groups. Understandably, their conclusion was that it was unlikely that temporal muscle ischaemia is the cause of muscle tenderness in patients with chronic tension-type headaches. This study is an important one in that it causes us to at least think about a long accepted theory that has received precious little challenge, but I have a number of concerns about this study and the conclusion drawn from it. The first concern is that 35 of the 40 patients making up the headache sample had daily headaches, making it quite a select group. The findings may well have been different for patients with episodic tension headaches rather than continuous ones, because of compensatory

measures that may take place. Second, 15 of the 40 headache patients also suffered from migraine. Migraine patients have been shown to have an opposite response during headache (vasodilation)[15] to that of tension headache patients, and thus, including these patients could increase the average blood flow for the headache group, making it more

Tension headache patients appraise ambiguous events as undesirable; controls view them as desirable

similar to the controls. Third, the blood flow was not measured when patients were headache free to allow detection of a change in flow from headache free to headache present periods (36 of the patients had headache at the time of the study). Fourth, and most important, the actual results may be more significant than the authors realize. The median resting blood flow for headache patients was calculated to be 3,7 ml/100g/minute compared with 5,0 ml/100g/minute in the control subjects.

This difference was found not to be statistically significant, which can be accounted for by the small sample size of 13 for the controls (confidence limits would help). A more important question is whether this 26% difference in blood flow between headache patients and controls is clinically significant? It certainly could be: 8 headache patients had subsequent hyperperfusion compared to 1 control.

Thus, the ischaemic theory could well still be in the running. Sustained muscle contraction may cause decreased blood flow through the muscular arterioles and lead to muscle ischaemia and consequent biochemical changes which may add further to the pain. But, there is another major system that affects the state of the vessels, the autonomic nervous system.

The Role of the Autonomic Nervous System

Dexter, an authority on sleep, describes that, "We live our lives between the states of excitation and relaxation."[25] Excitation is the sympathetic state; relaxation is the 'let down', the parasympathetic state. The sympathetic state is characterized by tachycardia, elevated blood pressure, cutaneous vasoconstriction, muscular vasodilation, and increased muscle tone. Dexter hypothesizes that tension headache is a "sympathetic headache, in that it is related to that end of the spectrum."[25] This is in contrast to the parasympathetic state

Head pain is a personal experience influenced by previous experiences and one's own understanding of pain

of migraine, "... where bradycardia, bowel hyperactivity, nausea, vomiting, cutaneous dilation, muscular vasoconstriction and decreased muscle tone predominate."[25]

Dexter's conceptualization is thus similar to that of Pritchard's[18] arousal and dearousal mechanisms. Pritchard states that the pain in tension headache, "may signal the danger of arousal", and thus have "the status of an adaptive alerting signal,"[26] in contrast to migraine which, "... may be experienced in relation to dearousal mechanisms."[26]

The Role of Platelet Activation

If adequately stimulated, platelets can be activated resulting in a change of shape and a release of various substances contained in specific platelet granules into the plasma. Dense bodies release 5-hydroxytryptamine (5-HT, serotonin), nucleotides and calcium; alpha granules release Beta-thromboglobulin (BTG), platelet factor four (PF4) and others. Patients

Tension headache is a defensive response – protecting oneself against friends, threats, family, and even intimacy

with tension headaches have been found to have significantly lower 5-HT platelet concentrations during headaches than nonheadache controls,[27] and significantly elevated plasma levels of BTG, PF4 and 5-HT while headache free compared to controls.[28] In addition, plasma BTG levels have been found to be significantly higher and PF4 and 5-HT nonsignificantly higher in migraine and tension headache patients. Thus, these studies provide good evidence both for platelet activation during tension headaches and for continuous platelet activation in both tension and migraine headache patients. The platelet activation may be milder in the tension headache patients. The possible implication of these studies

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These earlier assumptions were questioned as subsequent research failed to demonstrate specific psychopathology for either tension or migraine headache patients[33-36] and it was postulated that headaches in general may be either the response of an inadequate personality to ordinary stress, or the response of an adequate personality to extraordinary stress.[37]

Gradually a shift in thinking began – a shift away from a linear, specific cause and effect model towards a systemic, multifactoral one.[38]

Two recent studies have provided important confirmation for two of the intuitive understandings held by many headache sufferers as well as those dealing with them: firstly, that one's perception of people and events greatly affects how we feel about them, and secondly, that how we feel greatly colours our experience of life as a whole.

Holm et al[39] studied 117 recurrent tension headache sufferers (having at least 3 severe headaches per week characterized by bilateral dull aching pain) and 174 controls (who did not consider headache a problem, had less than 6 headaches per year and had no migraine symptoms), selected from a sample of 1 486 college students who had completed a screening questionnaire. Young, otherwise healthy students were chosen in order to avoid the influence of longterm treatment and chronic pain. The test measures used were the Life Events Inventory adapted from Cochrane and Robertson,[40] the Hassles Scale,[41] a Cognitive Appraisal Inventory adapted from Hammen and Mayol,[42] and the Coping Strategies Inventory.[43] In contrast to controls, headache sufferers: (1) reported a greater number of chronic everyday stresses or daily hassles versus similar numbers and types of stressful life events, (2) rated the life events and daily hassles more negatively, (3) employed less effective coping strategies, and (4) appraised ambiguous events as undesirable. Thus, stressors that trigger and aggravate tension-type headaches are likely to be better understood by assessing daily hassles than major stressful life events. Some people are more inclined to view both major life events and daily occurrences more negatively than others. No doubt, negative cognitive appraisal is associated with negative feelings such as anxiety and anger, and these feelings seem to be important factors in tension headache as shown in the following study.

Stress acts as a trigger which arouses the sympathetic nervous system to action, producing muscle spasm which causes the pain

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Donias et al[44] studied the frequency of precipitation of headache attacks by different emotional states in 90 consecutive patients with tension-type headache and 50 consecutive migraine subjects at an outpatient headache clinic. Anxiety and anger were significantly more frequently identified by tension headache sufferers than by migraine patients, as precipitating headache attacks. In addition, patients with tension-headache reported a graded frequency of attack precipitation, by individual emotional states, with anxiety significantly more frequently associated with attacks than anger, which was significantly more frequently associated with attacks than depression and positive emotional states (which were equally frequently associated with an attack). Only 3,3% of the tension headache group and 8% of the migraine patients did not report association of attacks to any emotional state. Thus it would indeed seem that the most frequent precipitation of tension-type headaches involves negative emotional arousal and that persons prone to having tension headaches are more likely to experience negative emotional arousal, perhaps in part, due to a pessimistic outlook (inclination towards negative cognitive appraisal) on life.

The Meaning of Tension Headache

It seems appropriate to conclude this paper by addressing perhaps the very heart of this matter concerning the head – the meaning of tension headache. I shall present one basic proposition and provide support for it from a number of different perspectives and sources. The proposition is that tension headache is part of a defensive response to adversity or perceived adversity.

This theme has been eloquently developed by John Graham, one of the giants of headache study, in a talk given to the 55th Harvard College Reunion in 1986, entitled: Headache as Cranial Angina.[45] Graham observed that we protect our heads both from physical and emotional threats. He describes how, when walking in a northeast snow storm through Harvard Yard, you tuck your head down in the collar of your overcoat and pull your hat down to your eyebrows against the threatening snow. This analogy has great meaning for Canadians and

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Russians; here in South Africa we might think of a strong northwest wind accompanied by rain in Cape Town. Graham goes on to describe how the same reflex causes us to tighten our head, neck and face muscles against blinding headlights on a wet night's drive and how the same painful muscle contractions occur when we are faced with other types of adversity - anger, fear of failure, guilt, deadlines, difficult problems at home or work, financial threats, and anticipated future problems.

It is normal, in fact, essential, to have appropriate defensive responses to protect ourselves from adversity, and perhaps, the occasional tension headache is an appropriate response - warning of danger or threat. However, hyperresponsivity, hypersensitivity or over-reaction, producing a warning signal or symptom when none is required, when the threat is not that great, may lead to recurrent tension headaches that have become a problem rather than a helpful message - an enemy rather than a friend.

This paper has provided substantial evidence to support the contention that recurrent and/or chronic tension headache sufferers are hyper-responsive to a variety of stimuli.

First, in keeping with Graham's picture of walking, huddled in one's coat and collar through a snowstorm, Langemark et al[10] found that patients with chronic tension headache not only had greater pericranial muscle contraction than controls, but, also that those who indicated influence by environmental factors (including weather changes and indoor climate as well as poor sleep, exercise, and psychological stress), had the highest pericranial tenderness scores.

Second, consider again the finding, by Bakal and Kaganov,[23] that headache patients and non-headache controls responded differently to 80 dB white noise: tension headache patients with vasoconstriction, controls by vasodilation. Sokolov's theory of orientating and defensive behaviour[46] predicts that a person will have cephalic vasodilation to orientating stimuli and vasoconstriction to noxious stimuli. If true, then this data suggests that tension headache patients interpret stimuli differently from controls - more negatively. Sokolov considered that an 80 dB tone was of an intensity expected to produce an orientating response (a non noxious stimulus), and thus Bakal and Kaganov's findings would support the theory that headache patients are hypersensitive, interpreting rather innocuous stimuli as dangerous.

> Activation of the sympathetic nervous system also results in pericranial vasoconstriction, producing muscle ischaemia which adds to pain

Third, Holm et al[39] found that, in addition to experiencing stressful events more negatively than controls, tension headache sufferers also appraised ambiguous events as undesirable, in contrast to controls who viewed them as desirable. Thus defensive, as opposed to orientating behaviour might be employed. In fact, negative appraisal of a situation can, itself, be seen as a defensive response.

Fourth, the study by Donias et al[44] on the differential emotional precipitation of migraine and tension-type headaches also makes the connection between negative cognitive appraisal and a defensive response in persons suffering from tension headaches. Donias et al suggest that distinct "cognitive schemata" ("organized structures of stereotypic knowledge derived from previous experience and modulating the individual's action and experience") exist in both tension and migraine headache patients. They go on to state that these distinct cognitive schemata "... may function either over-effectively, as screening mechanisms processing emotional arousal information in an inflexible manner, or defectively because of learned vulnerabilities to the specific emotional information processed at the time".[47] They postulate that... "selectively assessing negative emotional arousal as signals for alertness and/or counterattack in tension headache, could start off the neuromuscular reaction as a quasiprotective mechanism".[47]

Finally, Dexter's conceptualization of tension headaches as being "sympathetic headaches",[25] and Pritchard's[18] association of tension headache with arousal are both in keeping with the hypothesis that tension headaches represent a defensive response; for it is, of course, the sympathetic nervous system that is primarily responsible for arousal and for mobilization of the body's defenses.

In concluding this section on meaning, I would like to make two further comments. The first is to recognize that all pain, including head pain, is a highly personal

experience that is influenced by our previous experiences and our understanding of the pain, its implications, and the meaning we attach to the situation in which we find ourselves. There is good evidence to suggest that pain involves both a perceptual experience and an integrative process involving sensory, emotional, and cognitive elements.[48]

The second comment is that systems theory[38] has a valuable contribution to make to our understanding of tension headache. Our medical training taught us to look beyond the symptom in order to diagnose and treat the underlying disease. Systems thinking also asks us to go beyond the symptom, but stresses the importance of conceptualizing human problems and their resolution in interactional rather than individualistic ways.

To recognize tension headache as a defensive response, allows one to ask, "What is being defended against?" As has been referred to earlier, we may sometimes need to protect ourselves from the environment and even from the 'darker' side of ourselves, but perhaps most often, we defend ourselves against others, including family members, friends, and those with whom we work. Certainly, there is widespread breakdown of relationships, especially those of marriage and family, with people both pitted against and defending themselves from one another. We defend ourselves against both real and perceived threats to self and perhaps ironically we often defend ourselves against the very things we desire most. Intimacy would seem to be a prime example here, and with respect to the topic of this paper, I am reminded of the particularly apt cliche, "Not tonight dear, I have a headache".

Conclusion

Headache in general, and tension headache in particular, is perhaps the commonest symptom known to mankind and is amongst the commonest symptoms presented to family practitioners.

The term 'tension-type' headache has been introduced in the recently revised international headache classification and specific diagnostic criteria agreed upon. During the past 50 years, a wide range of laboratory and clinical studies have increased our knowledge of this most fascinating and highly complex phenomenon.

Tension headache may be viewed as a defensive response to adversity. Occasional tension-type headaches, in response to real threat or danger, can then be seen as a normal healthy protective mechanism. On the other hand, recurrent and chronic tension type headaches, as a result of over-responsiveness to relatively innocuous stimuli, can be seen as a maladaptive defensive response.

The pathogenesis of tension-type headaches is still being elucidated and there is no longer a common consensus. However, it would seem that some form of stress or adversity acts as a trigger that arouses the sympathetic nervous system to action, producing muscle contraction or spasm which would seem to be the major cause of pain in most patients suffering from tension-type headache. In addition, it appears that activation of the sympathetic nervous system also results in pericranial vasoconstriction which can produce muscle ischaemia and may further add to the pain. Platelet activation would also seem to be involved.

In spite of the advances, there is still much more to learn before we fully understand the nature of headache.

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