Modern management modalities are vital to treating headaches effectively.

HEAD PAINS

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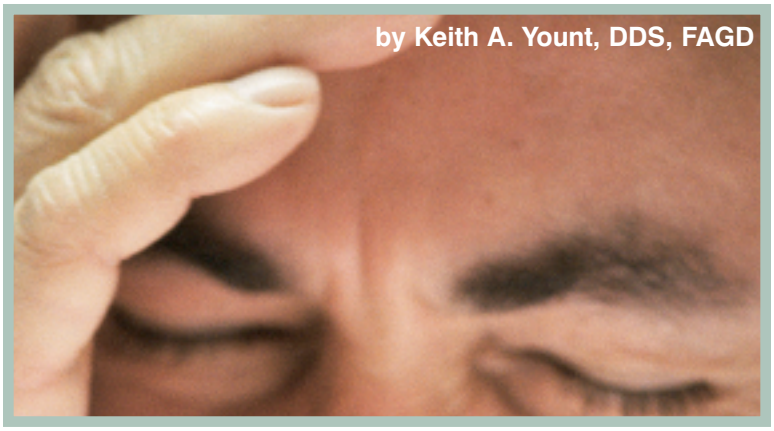

Until recently, scientific literature on headaches did not seem to answer a myriad diagnostic questions related to caring for patients. Prior to the last five years, dedicated research yielded astonishingly little conclusive information. This information was often presented in such a fragmented way as to cause further confusion.

The pathophysiology is still so full of conflicting and complicated theories that even the most earnest researcher is tempted to give up seeking the answers. Despite the paucity of training most medical professionals received in college regarding headaches, most clinicians remain committed to helping patients. Headaches are a problem that medical specialists encounter every day.

Headaches are far and away the greatest cause of human suffering. Consider this:

- Most people do not seek care for their headaches and yet four percent of all patients claim headaches as their primary concern and almost a third of all patients claim it as their secondary concern.[1]
- The estimated prevalence rate for all types of headaches is 93 percent of all males and 99 percent of all females.[2]
- Forty-five million Americans suffer a headache every day of their lives with the annual cost of migraine treatment topping one billion dollars in health care cost and 13 billion in lost workdays.[3]
- The pediatric headache rate has increased from 14 percent in 1972 to 50 percent in 1992.[4]
- Still, more than 90 percent of people with headache problems have never seen a specialist for their pain.[5]

Headache Types

Of the many types of headaches, the tension headache is by far the most prevalent — with 38 percent of all males and 46 percent of all females suffering from this form.[6] Seymour Diamond describes the tension headache in his chapter "Muscle Contraction Headache" in *Practicing Physician Approach to Headache*, as "chronic, nonpulsatile, tightness, band-like pain in the temporal area, unilateral or bilateral, associated with stress."

The next most prevalent is the migraine — claimed by six percent of all males and 18 percent of all females.[7] In the 1980s, the prevalence of migraines increased by 60 percent. Stephen Silberstein describes the migraine headache as "a severe episodic unilateral throbbing headache with genetic inheritance aggravated by exercise and associated with nausea, phonophobia, and photophobia."[8]

Other less frequent but still important forms of headaches are a result of sinus infections, aneurysms, tumors or growths, temporal arteritis and trigeminal autonomic cephalgia's (CPH, cluster, etc). This miscellaneous headache group is too complex to fully define, has less probability of occurring, and is too diffuse a group to review for the purposes of this article. Still, all forms must be considered in any headache differentiation.

This diagnostic picture is painful and depressing. With suffering all this time, why has science been so unfruitful on such a plague to society? The good news is much has been accomplished in the last five years.

Early Headache Management Theories

Thankfully, the management of headaches has come a long way since the days of leeches, bloodletting, and spirit removal. The early scientific management ideas resulting from the anecdotal evidence of single practitioners yielded many of the treatments used today.

Pharmacies are now full of migraine treatments. Much of the first effective migraine management came with the introduction of ergotoamines DHE. This was followed by heart medicine beta-blockers and calcium channel blockers, and later the tryptans. Unfortunately, hard science has had little to offer tension headache sufferers. Tension headaches have failed the polypharmacy, imaging, dental, and blood tests.

Oddly enough, up till now, practitioners have largely ignored the behavioral sciences as a means of managing headaches — especially migraines. Now, with the emergence of the most recent scientific literature, medicine has begun to embrace behavior modification techniques for use with both the tension headache and migraine therapies.

The complex pathophysiology of the tension headache and migraine has long challenged researchers seeking an etiology. The discovery of the etiology lies in the identification of the complex set of initiators, aggravators, and perpetuators — not a single-cause viewpoint.

Headache and Inflammation

The latest research on neurogenic inflammation in migraine and tension headaches provides a better understanding of the patho-physiology of both diseases. At last, there are the necessary tools to relieve headache suffering.

Neurogenic Inflammation

One of the most intriguing areas of

headache research concerns the effects of neurogenic inflammation. In his presentation at the AHS headache symposium in Scottsdale Arizona in 1999, Michael Cutrer of the Harvard Medical School stated that, "under steady-state conditions, the brain vigorously maintains the equilibrium of its extacellular environment."

When an appropriate amount of inflammation chemicals travel to the brain, glial cells, neurons, lymphatics, and vascular system rapidly take up the neurogenic chemicals. When an overload of chemicals travel to the brain, a headache results. What causes the overload is an increase in neural transmission in the trigeminal afferent system, cervical afferents, and even the sympathetic system. Other inflammation chemicals like lactic acid, prostaglandins, cytokines, nerve growth factor, and other inflammation by-products are also to blame. These neurotransmitters can cause the nociceptors in the trigeminal vessel walls to become sensitive which lowers the stimulation threshold.

Two neurotransmitters, CGRP and SubP, stimulate mast cells to degranulate which in turn spurs a release of inflammation chemicals. According to Weiler's analysis of PET scans, the neurogenic inflammatory chemicals seem to trigger the migraine generator in the corpus.[9]

The neuropeptide, Sub P, initiates a release of histamine from the mast cells, which activates H1 endothelial receptors inducing formation of nitric oxide (NO).[10] The nitric oxide, a gaseous neurotransmitter, rapidly diffuses into the vascular smooth muscle which activates CGNP and relaxes the muscle thus causing vasodilatation.[11]

The neurotransmitter CGRP exhibits vasodilatation properties of its own. Sub P also initiates release of cytokine from monocytes. The neuro-transmitters CGRP and Sub P alter the function and chemotactic activity of immune cells. It is likely that the release of these neuropeptides in large quantities overrides the brain's ability to manage the steady state of balance thereby initiating a cascade of biochemical changes.

Inflammation and the Central Nervous System

The classic analogy of the nervous system is a set system with simple transmission of signals. According to recent research, this analogy is far from correct.

First, the nervous system changes in response to inflammation. One such change involves the number and location of sodium channels. The sodium channel has different type of subclasses. One of the subtypes is the PN3 channel primarily expressed on nociceptors. In the presence of inflammation, it is preferentially transported from the cell body to the peripheral terminals following the nerve injury. This sodium channel is resistant to local anesthetics and may be a clue to some of the resistant neuropathic pains.

Another change concerns the large diameter fibers A Beta, which normally transport touch sensations. In the presence of inflammation, these fibers can begin expressing the neurotransmitter Sub P. These fibers have very few opioid receptors on them. The A beta fibers have also been shown to sprout in the dorsal horn and nucleus caudalis forming connections from lamina III and IV to lamina I.

When the nervous system is reactive to all the causes of stimulation, inflammation, and activation of the pain systems, addressing the original sources of inflammation is a vital key to managing headaches.

Inflammation and the Sympathetic System

The sympathetic system is intimately related to the creation of inflammation when it becomes stimulated by perceived or real external environment challenges, dangers, or invasions. This "fight or flight" reaction by the body causes the release of adrenalin, which activates energy systems in muscles as well as the gamma efferent pathways to muscle spindles. This, in turn, increases muscle tone, activation of the mitochondria and energy systems to habitual muscle groups.

These gamma afferents tend to develop varying pain patterns from person to person. For example, one person may feel the painful effects in the neck; another in the back. Still, another may feel effects in the form of a tension headache in the temple. So, at the same time that the adrenalin is regulating the muscles of the whole body, the gamma afferents are adding an additional demand on the muscles.

All this extra muscle activity produces isometric contraction of smooth muscle and often the skeletal muscle. This contraction is always accompanied by lactic acid build-up and its subsequent inflammation. Remember, this isometric contraction is on top of the contraction that results from everyday activities such as talking, chewing gum, eating, laughing, and frowning.

The muscles of the trigeminal and cervical systems produce a great deal of inflammation when over-stimulated by stress. Even a restraint stress of laboratory rats creates a release of neurogenic inflammatory chemicals from 70 percent of the intracranial mast cells.[12]

Still another plastic change is the sprouting of the sympathetic system in response to inflammation. The sympathetic system sprouts in basket-like connections around sensory neurons and C fibers.[13] This gives the sympathetic system control over the pain system.

Inflammation and Trigeminal Muscle Physiology

Using muscles for everyday activities creates a small amount of manageable inflammation. It's only when the trigeminal and cervical muscles are requested to provide additional activity for non-essential activities that the increase of inflammation and use of neurotransmitters overload the system. Non-essential activities include teeth-grinding or clenching, gum chewing, constant frowning, bad posture, and squinting.

The parafunctional use of the trigeminal muscles for clenching, bruxing, tooth bracing, muscle bracing, or tongue biting is one of the biggest contributors to neurogenic inflammation in the trigeminal muscles. In other words, when a patient presents with pain in the temple muscle or jaw pain in the masseter and medial pterygoid, parafunction should always be considered a culprit. Nearly two-thirds of tension headache suffers present clenching or grinding as an aggravating factor in their tension headaches.[14]

In addition, a poorly functioning temporomandibular joint with internal derangement can add to the inflammation chemicals in the trigeminal muscles. This often occurs with the high velocity trauma of a disc displacing and recapturing on every movement.[15] This is why nearly 70 percent of patients diagnosed with TMD are also reported to suffer from headaches.[16]

The osteoarthritic tempromandibular joint creates even more inflammation. In that case, fibrocartilage is lost along with the disc that creates a great deal of inflammation chemicals with just normal, everyday movement.[17] Damaged joints create a "sprained ankle effect" where muscle splinting adds to the muscle inflammation.

Inflammation and Cervical Muscle Physiology

The cervical muscles are also capable of producing large amounts of inflammation. These muscles, which must support the equivalent of a five-pound bowling ball, are stressed by poor physical posture, computer posture, forward shoulder posture, phone posture, and high-traffic driving tension. The overuse of these muscles accompanied by the stimulation of the nervous system, and the activation of the sympathetic system usually result in "a pain in the neck." In fact, poor posture alone causes muscle splinting, stress to ligaments, abuse of connective tissue, loaded function of the "zap" joints of the vertebrae, compression of blood vessels, and compressed nerves.

Remember there are three layers of cervical muscles, many synovial joints of vertebra, and many specialized tissues. Each of these is activated by gravity, posture, and function of the head and neck. These structures deliver their inflammation chemicals mostly into the vertebral artery and some into the carotid artery, which in turn, bombards the brain with more irritants. Even under normal conditions, the brain exerts tremendous energy to maintain homeostasis. That's why it is so easy for increased neurogenic inflammation chemicals to overrun the brain and cause headaches.

Inflammation and Estrogen

For many years, doctors have noted a relationship between increased levels of estrogen and an increase in headaches. Still, there is very little evidence of the pathophysiology of this relationship. It is known that:

1) Hormone replacement often exacerbates headaches;

2) Oral contraceptives change the character and frequency of migraines;

3) Headache frequencies change at puberty and menopause;[18]

4) Migraines tend to worsen during the first trimester and improve in the second and third trimesters.[19]

One of the most fascinating findings in recent years concerns the estrogen receptor on the female mast cell. The mast cell plays the role of "storage bin" for neurogenic chemicals and is the predominate cell in the inflammation process. Unfortunately, a woman's mast cell receptor is genetically coded with an inflammation enhancement that is not present in men.

When the estrogen levels and blood estrogen levels flux such as with the use of contraceptives or hormone replacement drugs, the estrogen couples with the mast cell receptor making it more sensitive to an inflammation stimulus.[20] Therefore, the mast cell releases neurogenic chemicals more quickly causing even greater numbers of mast cells to respond. A female during the flux will get more chemicals, more inflammation, more pain, and more swelling than a male reacting to the same stimulus.

One study, in particular, substantiates the mast cell's role in the pathophysiology of the hormone/headache relationship. A clinical trial of Tamoxifen, an estradiol receptor antagonist, was used on laboratory animals and was found to substantially reduce mast cell secretions and pain behavior.[21]

Obviously, the mast cell does not account for the whole picture of the female predominance toward headaches, but it is definitely a part of the female's predisposition to suffer chronic pain.

Modern Management Modalities

Improving the Function of the Sympathetic System

Therapies that directly and indirectly improve the function of the sympathetic system effectively reduce the pain of migraines and tension headaches.

Many therapies conventionally used for tension headache have also been found to work on migraines. Some of these include:

- biofeedback
- progressive muscle relaxation
- stress management
- anxiety management
- cognitive therapy
- Thi Chi
- Yoga
- cardiovascular exercise
- deep breathing

A few migraine headache therapies

CASE STUDY

The cervical connection: My wife's story.

Before I returned to post-doctoral school, my wife was plagued for many years with chronic daily headaches tension, migraine, and sinus and cervical dysfunction. The prevailing diagnosis seemed to be, "You're a stressed out female. It's all in your head, and you will just have to live with it."

After getting no relief from the headache community, I began a series of myofascial pain therapies. I provided her with a muscle relaxation orthotic for her mouth and prescribed muscle relaxants and NSAID. Some days the therapy worked great, and some days it didn't. It hurts when you are not a hero in your own home! If the pathology was the same and the treatment the same, why weren't the results the same?

Finally, researchers at the University of Florida discovered the key. We can't the treat the head while ignoring the neck. The neck is almost always a major player in myofascial pain. When we combined her MPD therapy with cervical therapy, we got phenomenal results.

We must no longer subject patients, particularly female patients, to the "stressed-out person" myth or make them feel as if their pain has resulted from a psychological pathology. By using MPD and cervical therapy simultaneously, we can now effectively reduce and perhaps totally eliminate most common headache pain.

also help in the treatment of tension headaches. Two of these include:

- TCA's[22]
- Sumatriptan[23]

Managing Muscle Physiology

We do know that the management modalities that have shown the most success with tension headaches are ones that directly or indirectly affect muscle physiology.

Direct measurements of muscle contraction have been elusive, but the measurement of muscle tenderness has been proven to have a direct relationship to the pain severity of the headache.[24]

A surgical study by David Hubbard indicates that the damaged muscle fiber trigger point) of trigeminal and cervical muscle pathology is a damaged muscle spindle.

In another study,[25] the trigger points were most effectively treated with sympathetic-ominetics related to the sympathetic system) such as phenylephrine, phenoxybenzamine, and noradrenalin but not botox or curare, which work on skeletal muscle. In this same study, EMG activity was measured from the exact trigger point and then from a small distance away. It was found that the EMG activity is directly correlated with the trigger point and point tenderness. Siegfried Mense, in a paper to the XII International Research Symposium in June of 1990, indicated that neurogenic inflammation sensitizes skeletal muscle nociceptors causing them to react at lower thresholds of stimulation.

It follows that effective treatment of tension and migraine headaches often includes some combination of the following:

- trigger point therapy
- physical therapy
- acupressure
- acupuncture
- chiropractic therapy
- massage
- ice and stretch
- ultrasound
- TENS

Conclusion

Recent research provides caring practitioners with many new insights and tools to help reduce their patient's pain and suffering from headaches. The medical community has combined years of research and clinical successes to develop the management modalities used today. The combination of Eastern and Western sciences, the combination of drug and behavior modalities, and the combination of personal resource development with medical science provides clinicians with the tools needed to effectively reduce the majority of headache pain.

The latest information on neurogenic inflammation will herald a new team concept with all members of the medical community working together to reduce headache pain. This team might include some of the following:

- Internist
- Orofacial Pain Specialist
- ENT
- Neurologist
- Rheumatologist
- Anesthesiologist
- Physical Therapist
- Psychologist
- Sympathetic Modulation Training (SMT) specialist
- Chiropractor
- Other members of the medical profession.

It's true that medical professionals are still at the beginning of the discovery journey for headache cure and prevention, but patients no longer have to sit at home suffering. With the latest research, patients and clinicians can all enjoy an improved quality of life. ■

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