Tension Headaches: An Investigation into Their Causes from a Naturopathic Perspective

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A thorough understanding of the physiological processes that underlie muscle tension is foundational to any discussion on environments and substances that alter such a process. The fascial web, an interconnection of the various fascia, extends throughout the entire body and acts to link every area of the body together and connect external and internal structures. It is the fascial tissue that transmits forces locally (i.e., between muscle and bone or between muscle and ligament) and distally, thereby creating muscle contraction in interconnected but distant areas of the body. Fascia is composed of various cell types, fibers (elastin, collagen, and reticular), and a fluid-like ground substance that is rich in proteoglycans. Fascial tissue responds both acutely and chronically to its environment via adaptations in both collagen and proteoglycan structure. Acutely, this results in the normal contraction of muscles and resultant movement, such as looking down at one’s feet while walking, but chronically it can result in chronic pain syndromes, including tension headaches, due to tenegravity (tensional integrity) changes in the fascial framework. This can occur as a response to repetitive strain or acute injury. The purpose of this discussion is to provide an overview of some of the many influences on muscle tension from the perspective of a naturopathic doctor. I will use tension headaches as an example, though the principles discussed here may be extended to many different chronic pain syndromes.

Introduction

Tension headaches, also known as tension-type or stress headaches, are the most common type of primary headache with as many as 90% of adults being affected (Taylor & Davis, 2015). Headaches are characterized by at least two of the following symptoms (Singh, 2014):

- Pressing or tightening pain on both sides of the head
- Mild or moderate intensity head pain

• Not aggravated by routine physical activity
• No nausea or vomiting
• Possible sensitivity to light or sound, but not both

Tension headaches may be episodic (at least 10 headache episodes lasting 30 minutes to 7 days, occurring fewer than 180 times per year) or chronic (average frequency of 10 days per month, or 180 days a year; Singh, 2014).

The etiology of tension headaches is not well understood; however, if we appreciate that a tension headache usually results via contracture of fascial tissue and thereby muscle tissue an understanding of the various triggers and mechanisms for headache can be more easily understood.

Any patient presenting with a headache should receive a full work-up including complete history and physical examination. Most patients do not require a head CT scan or MRI unless there are abnormal findings on neurological exam or the patient’s clinical picture does not match one of primary headache disorder (Blanda, 2015). Red flag signs and symptoms suggest a secondary cause of headache and must be referred for emergent care. These include:

- New onset headache
- Abrupt onset headache
- Progressive symptoms
- Abnormal neurological signs
- Headache with exertion
- Change with head position
- Change with Valsalva maneuver, such as cough, sneeze, or strain
- Symptoms consistent with trigeminal autonomic cephalalgia (TAC) diagnosis (Eller & Goadsby, 2013)

Once emergent situations have been ruled out, a deeper look into the role of the following factors will help to elucidate the underlying cause of tension headaches in the presenting patient.
Dehydration

Dehydration results when water loss exceeds water intake, resulting in net movement of water out of the cell and into the bloodstream. Dehydration can be acute (such as in excessive sweating, hyperthermia, vomiting, diarrhea, diuretic use, or electrolyte disturbances) or chronic (from any of the above or from ongoing inadequate water intake). When the ground substance of fascia becomes dehydrated it compromises the tissue’s ability to dissipate compression forces, lubricate surrounding tissues, and transfer nutrients between capillaries and cells (Muscolino, 2011). When muscle tissue becomes dehydrated, its myelin tubes develop an undulating contracted formation, which presents as hypertonicity of the muscles and results in pain (Chen, Schmidt, Olmsted, & MacKintosh, 2001). Ensuring adequate fluid intake for the weight, age, activity level, and environment of the patient is a vital consideration in the setting of tension headache. A sample calculation for water requirement is illustrated in Table 1.

Please note that this is a rough estimate and actual amounts will vary depending on age, environment, gender, activity levels, and health conditions (Perry, 2012).

Electrolyte Imbalance

Electrolytes are minerals in the body that carry an electrical charge. They affect water balance, blood acidity, muscle function, and many other processes in the body (Dugdale, 2013). Electrolytes in the human body include sodium, potassium, chloride, calcium, magnesium, and phosphorus. When electrolyte levels move outside of physiologically normal levels in the blood, a patient may experience headache, nausea, fatigue, muscle contractions, mental status changes, and even death. Although all electrolytes serve a vital role in osmotic balance and thereby, hydration status in the body, two particular electrolytes stand out as nutritional considerations when it comes to tension headaches: calcium and magnesium.

Table 2 outlines the mechanism of muscle contraction and relaxation highlighting the role of calcium and magnesium in this process. Figure 1 illustrates the same process graphically.

Low calcium levels can result from increased calcium loss, decreased calcium intake, high phosphate, low albumin or magnesium, low vitamin D, medications, or hypoparathyroidism (Suneja, 2015; Tortora, 2006). Symptoms of low calcium include numbness and tingling in the fingers, hyperreflexia, muscle cramps, tetany, convulsions, fractures, muscle spasms, and death through laryngeal muscle spasm (Tortora, 2006). Elevated calcium is also troublesome, resulting from hyperparathyroidism, certain cancers, excessive calcium intake, excessive vitamin D intake, or Paget’s disease (Tortora, 2006). Symptoms include lethargy, weakness, anorexia, nausea, vomiting, polyuria, pruritus, bone pain, depression, confusion, paresthesia, stupor, and coma (Tortora, 2006). Calcium is an electrolyte that must be within normal physiological ranges within the body and also in proper balance with magnesium in order for muscle contractility to be optimal. When muscles are hypotonic or hypertonic, tension headaches can result. Supplementation with appropriate forms of calcium, balanced with magnesium and vitamin D can help to regulate optimal muscle tone.

Low levels of magnesium are implicated in both tension and migraine headaches (Pizzorno, Murray, & Joiner-Bey, 2008). Altura and Altura (2001) suggest that muscle tension and scalp tension are more associated with tension headache than any other type of headache, whereby magnesium intake and metabolism in the body may account for as much as 40%–50% of headache attacks. Furthermore, in females of childbearing age, tension headaches can occur

<table>
<thead>
<tr>
<th>Table 1. Sample Formula to Calculate Daily and Hourly Water Requirements</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pounds of body weight</strong></td>
</tr>
<tr>
<td><strong>Water requirement from above (calculate 75% of the body’s pound weight, and plan to drink that many ounces/day)</strong></td>
</tr>
<tr>
<td><strong>Add for dryness of climate</strong></td>
</tr>
<tr>
<td><strong>Add for strenuous exercise</strong></td>
</tr>
<tr>
<td><strong>Total per day</strong></td>
</tr>
<tr>
<td><strong>Divide by the number of hours you are awake to find your hourly water requirement:</strong> 144.6 / 16 =</td>
</tr>
</tbody>
</table>

Note. Please note that this is a rough estimate and actual amounts will vary depending on age, environment, gender, activity levels, and health conditions (Perry, 2012).

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more frequently during the premenstrual phase; most of these women present with low levels of magnesium (Altura & Altura, 2001). Having adequate levels of magnesium, along with proper metabolism within the body, is vital for the relief of these type of headaches.

Magnesium is a cofactor in over 300 metabolic reactions in the body including playing a vital role in muscle contraction (Jahnen-Dechent & Ketteler, 2012). As described in Table 2, during muscle contraction, magnesium facilitates calcium reuptake by the calcium-activated ATPase of the saccroplasmic reticulum, thereby clearing calcium from the myofibrils causing muscle contraction to cease (Jahnen-Dechent & Ketteler, 2012).

Adequately diagnosing low magnesium in a patient requires testing either red blood cell magnesium or ionized Mg2+ due to the fact that most magnesium in the body is intracellular, rendering measures of serum magnesium rather meaningless as they will only indicate an end-stage deficiency (Pizzorno et al., 2008). Low magnesium levels can result from insufficient dietary intake, chronic stress, diabetes mellitus, alcoholism, diuretic treatment (including caffeine), excessive carbohydrate intake, or loss through urine and stool (Tortora, 2006). Symptoms of low magnesium include weakness, irritability, tetany, delirium, convulsions, confusion, anorexia, nausea, vomiting, paresthesia, and cardiac arrhythmias (Tortora, 2006).

A common treatment in the naturopathic field is intravenous nutrient therapy. One study showed moderate effectiveness of intravenous magnesium sulfate (MgSO4) in acute headache. The authors found partial or complete pain relief in 56% of patients treated with IV magnesium, irrespective of the patient’s presenting serum magnesium level (Ginder, Oatman, & Pollack, 2000).

Although acquiring a clinically relevant analysis of a patient’s electrolyte levels and assessing metabolic activity of calcium and magnesium within the body can offer insight into their role in tension headaches, patients may benefit from supplementation of these nutrients (orally or

### Table 2. General mechanism of muscle contraction and relaxation with roles of calcium and magnesium highlighted (Guyton & Hall, 2006).

<table>
<thead>
<tr>
<th>Action potential in motor nerve</th>
<th>Reaches muscle fiber causing release of acetylcholine (ACh)</th>
<th>Local muscle fiber responds to ACh by opening ACh-gated channels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium ions stimulate attractive forces between actin and myosin in muscle fibers, causing them to move alongside each other causing muscle contraction</td>
<td>Muscle membrane depolarizes causing sarcoplasmic reticulum to release large amounts of calcium</td>
<td>Sodium ions diffuse into muscle fiber causing an action potential at the local membrane</td>
</tr>
<tr>
<td>Less than a second later, the calcium is pumped back into the sarcoplasmic reticulum (facilitated by magnesium) where it is stored until another action potential arrives</td>
<td>Removal of calcium from myofibrils causes muscle to relax</td>
<td></td>
</tr>
</tbody>
</table>

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Tension Headaches from a Naturopathic Perspective
Figure 1. Contraction of a muscle fiber. A cross-bridge forms between actin and the myosin heads triggering contraction. As long as Ca$^{++}$ ions remain in the sarcoplasm to bind to troponin, and as long as ATP is available, the muscle fiber will continue to shorten. “Wikimedia Commons Contraction New” by OpenStax College is licensed under CC by 3.0.
intravenously in the appropriate form), regardless of their initial levels.

**Hypertension**

The connection between high blood pressure and headaches has been a topic of much debate for several decades. The prevalence of both of these conditions is high, and a mechanism of their association has yet to be elucidated. Several theories have been proposed as to why headaches are slightly more prevalent in hypertensives than in normotensives, including a higher incidence of stress and anxiety in hypertensives that may be the cause of headache, a higher incidence of sleep apnea in hypertensive patients that may offer an explanation for morning headaches, and a higher incidence of headaches in patients who were aware of their hypertension (suggesting a psychosomatic response; Pickering, 2000).

One study involving 2,673 patients in seven double-blind, placebo-controlled trials assessed the effectiveness of irbesartan, an angiotensin receptor blocker, in headache symptoms in mild to moderate hypertensive patients. In both the placebo group and the treatment groups, headache was positively correlated with gender (female), age (>50 years), and last diastolic blood pressure reading. Active drug treatment was a significant independent predictor of decreased headache, whereby the irbesartan group had a lower incidence of headache compared to the placebo group (17% vs. 22%; \( p = .003 \)). The authors concluded that headache may be one symptom of mild to moderate hypertension, which may be reduced with antihypertensive therapy (Hansson, Smith, Reeves, & Lapuerta, 2000).

In another study of 150 patients evaluated for hypertension (many of whom were medicated), headache was a symptom reported in journals of 30% of patients. The authors of this study did not find a significant relationship between headache and blood pressure, and observed that many patients did not report symptoms of headache when blood pressure was highest (Pickering, 2000).

The results of these two studies highlight the controversy that still exists around the role of hypertension in headaches. To complicate the picture even more, it is important to note that headaches are a common side effect of several antihypertensive medications, most notably calcium channel blockers and direct vasodilators such as hydralazine (Yeung, 2006). Furthermore, patients with hypertension are commonly taking other prescription medications such as nitrates or statins, both of which can cause headaches (Yeung, 2006).

Again we are reminded of the multifactorial nature of tension headaches when we observe that many hypertensive patients’ headaches improve when their blood pressure is normalized, but that the mechanism of this improvement may be attributed to a healthier lifestyle, decreased stress, less anxiety, a healthier diet, increased exercise, and/or the psychosomatic relief of knowing that their blood pressure is controlled.

**Food Intolerance**

Food intolerance, or non-IgE-mediated food hypersensitivity, refers to difficulty digesting certain foods due to a delayed immunologic reaction. Food intolerances are distinct from food allergies in that they stimulate the production of IgG rather than IgE antibodies. IgE antibodies, which are produced during an allergic reaction, have an immediate and short-lived response, often presenting as an anaphylactic reaction with symptoms such as hives, trouble breathing, tightness in the throat, hoarse voice, nausea, vomiting, diarrhea, and abdominal pain. IgG antibodies, on the other hand, which are produced during an intolerance or sensitivity reaction, often have a delayed onset and can last for months in the body.

As described by Rocky Mountain Analytical (RMA), a well-known Calgary-based medical lab, once a reactive food is ingested, IgG antibodies attach themselves to the allergen creating an antibody–allergen complex, which is normally removed from the body by macrophages. However, if the number of complexes outnumbers macrophage resources, or if the reactive food is still being consumed, the antibody–allergen complexes accumulate and are deposited in various body tissues. Once implanted, these complexes release inflammatory chemicals, which are known to contribute to a number of chronic illnesses.

The trigger(s) for this type of immune reaction and symptom picture are unique to each individual and proper blood testing is crucial for diagnosis. Some of the most common intolerances include dairy, wheat and gluten, corn, soy, eggs, and nuts. Presenting symptoms most commonly affect the skin (especially eczema), brain (mood, memory, behavior), lungs (bronchitis and asthma), musculoskeletal system (joint pain, stiffness, swelling), and digestion (nausea, vomiting, diarrhea, constipation, bloating, abdominal pain). Symptoms can also by systemic, including fatigue, chills, fever, sweating, weakness, and lethargy (Rocky Mountain Analytical, 2014). The link between food intolerance and migraines has been well-established, but the link to tension headaches may be equally strong. The Australian New South Wales Food Authority lists headache as one of the most common symptoms of food intolerance, along with bloating, migraines, cough, runny nose, feeling...
under the weather, stomach ache, irritable bowel, and hives (Nordqvist, 2014).

The mechanism of food intolerance is multifaceted. Several theories have been proposed as to why some people react to foods and others do not. These include:

- Lack of proper digestive enzymes
- Food chemicals (including amines, sulfites, caffeine, and salicylates)
- Food toxins (including aflatoxins)
- Naturally occurring histamine in foods
- Food additives (including artificial coloring, artificial flavoring, emulsifiers, preservatives, and sweeteners; particularly problematic are MSG, nitrates, and sulfites) (Nordqvist, 2014).

The mechanism of headache etiology may only be speculated. It may be related to the incidence of muscle tension in food intolerance, presenting in neck, shoulder, and temporomandibular joint hypertonicity. It may be related to nutrient deficiencies owing to compromised digestive function. It may be a consequence of chronic constipation caused by food intolerance. Finally it may be related to sinusitis and sinus congestion that often accompanies food intolerances. Further studies are needed to fully elucidate this relationship.

**Eyestrain and Computer Vision Syndrome**

In a world where much of our time is spent looking at a screen of some sort, often for hours at a time, the prevalence of eyestrain has risen dramatically. Eyestrain can be caused by poor lighting, fluorescent lighting, long periods of driving, extended hours looking at a computer, and reading (especially in poor lighting). The human eye is constantly adapting in order to focus objects at near and far distances. This is achieved via enlarging or minimizing the size of the pupil, by shortening or lengthening the lens, and/or by contracting and relaxing the extraocular muscles to coordinate the two eyes (Yan, Hu, Chen, & Lu, 2008). When this activity becomes repetitive for multiple hours, these muscles begin to fatigue.

Computer vision syndrome (CVS) is quickly becoming a modern epidemic. According to the American Optometry Association, CVS is defined as “the complex of eye and vision problems related to near work which are experienced during or related to computer use” (Yan, 2008 et al., p. 2026). The major symptoms of CVS can be divided into three main categories: (a) eye-related symptoms (dry, watery, irritated, or burning eyes); (b) vision-related symptoms (eyestrain, eye fatigue, headache, blurred vision, double vision); and (c) posture-related symptoms (sore neck, shoulders or back; Yan et al., 2008).

If we go back to the physiology of muscle contraction, muscle fatigue, and fascial tension, it becomes apparent how repetitive strain on the ocular muscles soon translates into tension headaches. Under normal conditions, the fascia around the eyes, along the forehead and covering the neck and shoulders will contract to facilitate proper vision. If, however, this activity occurs repetitively, often for months on end, the fascia will adjust its proteoglycan and collagen structure, thereby altering the contractile forces on surrounding muscles and resulting in chronic muscle tension and tension-type pain in the head and neck.

A few strategies for avoiding CVS and eyestrain include:

- Maintaining a distance of 20 inches (50.8 cm) between you and your screen
- Adjusting the computer’s viewing angle to 15 degrees lower than horizontal
- Employing the 20/20/20 rule: after 20 minutes of computer viewing, focus on something 20 feet away for at least 20 seconds
- Taking frequent breaks from the computer
- Monitoring screen and room lighting and glare
- Maintaining proper posture during computer work
- Keeping your eye exams and prescriptions up to date
- Wearing your glasses or contact lenses if needed

If using a computer for >3hrs per day, try warm eyelid massages daily can help prevent CVS (Yan et al., 2008).

**Temporomandibular Disorder**

Temporomandibular disorder (TMD) is a term that encompasses several dysfunctional patterns in the process of mastication: muscle disorders, temporomandibular joint dysfunction, and dysregulation of associated structures. According to a study by Mitrirattanakul et al. (2006) headache is a symptom present in 73% of TMD patients, whereas other dental patients have only a 32% prevalence of headache (Karibe, Goddard, & Okubo, 2014). In TMD patients, there is frequently hypertonicity of the muscles of mastication, due to clenching, grinding, injury, physical dental structure, and/or some disease processes. Again, if we revisit the physiology of muscle contraction and the consequent adaptations to chronic tension, we see fascial changes that result in chronic pain syndromes such as headache.

Chewing habits are also of paramount importance when analyzing TMD. One often overlooked antecedent to chronic tension of the jaw muscles is excessive use of chewing gum. Watemberg, Matar, and Har-Gil (2014)
investigated the role of excessive chewing gum use in teenagers and its influence on headache prevalence and severity. Although a small study, his results showed a decrease in headache symptoms upon chewing discontinuation in 26 out of 30 patients and complete resolution of headache in 19 out of 30 patients. After a period of discontinuation, 20 patients resumed their chewing gum habit and all reported symptom relapse within days of reintroducing the habit (Watemberg et al., 2014).

A thorough investigation of jaw function, dental health, and chewing habits is vital in any patient with headache, along with referral to a specialist when warranted for assessment and treatment of TMD.

**Sinusitis**

Headache is a common symptom of many febrile illnesses including sinus infections, influenza, and the common cold, and usually resolves at the resolution of the illness. Severe headache following a cold, however, may be due to a secondary bacterial infection of the frontal or maxillary sinuses and must be investigated with appropriate radiography. Sphenoid sinusitis, although rare, is a potentially life-threatening cause of headache (often presenting as a “thunderclap” headache in adults), and is easily missed upon examination. The potential of this serious scenario also warrants radiography when sinusitis is suspected.

Chronic sinusitis is a common complaint and is frequently attributed to structural abnormalities of the facial tissues, disturbances to mucociliary clearance, pathogens (such as low-grade infection or fungal colonization), inflammatory factors (including food intolerances), dry climates, allergies, and/or inhaled particles (particularly dust and mould; Hamilos, 2000). The symptom picture typically includes nasal congestion (73%), postnasal drip (65%), headache (48%), cough (47%), facial pressure (42%), anosmia or hyposmia (39%), wheezing (34%), hypogeusia (29%), and throat clearing (29%; Hamilos, 2000). The average duration of symptoms is 14 years (Hamilos, 2000).

When inflammation occurs in the sinus cavities, there is an accumulation of mucus, which can block the small drains of the sinuses; this results in a build-up of pressure within the sinus cavity. In severe cases this results in pain, most commonly headache. Properly assessing a headache patient for sinus involvement, both subjectively in office and objectively through radiography, is crucial. Furthermore, identifying the etiology of the sinusitis and treating accordingly has the potential to alleviate chronic headaches in that patient.

**Hormones**

Changes in female hormones are commonly associated with migraine headaches; however, premenstrual tension headaches are a common complaint in women of childbearing age. What is unclear at this point is whether it is the hormone changes themselves that incite headaches or the electrolyte shifts that accompany these hormonal changes. It may be that the fall in estrogen that accompanies the premenstrual period is more closely associated with migraine headaches, whereas consequent shifts in serum magnesium levels may be responsible for tension-type headaches in this population.

Altura and Altura (2001) has found that most women who present with premenstrual tension headaches have low serum magnesium levels. Furthermore, there is recent evidence to suggest that the ovulatory and luteal phases of the menstrual cycle are correlated with decreases in serum magnesium and consequent increases in the calcium/magnesium ratio (Altura & Altura, 2001). It is the falling estrogen level that is believed to modulate the serum magnesium changes in both pre- and postmenopausal women (Altura & Altura, 2001). As discussed in the electrolyte section, such changes in calcium and magnesium ratios alters both the contractility and the ease of relaxation of muscle fibers, resulting in muscle tension and associated pain syndromes. Oral supplementation or intravenous magnesium may prove helpful in these scenarios.

**Caffeine Dependency and Withdrawal**

Caffeine in the form of coffee, tea, and cola-type drinks, is one of the most widely used and socially accepted neurostimulants of our time. Excessive caffeine intake creates physiological dependency, which upon withdrawal usually presents as headaches, irritability, decreased mental focus, drowsiness, decreased contentment, and social withdrawal (Evans & Griffiths, 1999).

Cerebral blood flow can decrease as much as 22%–30% after a 250 mg dose of caffeine through its activity on vascular smooth muscle (Addicott et al., 2009). Chronic caffeine intake results in adaptive changes to the vascular adenosine receptor system, which is perhaps a response to this vasoconstrictive effect (Addicott et al., 2009). Caffeine competitively antagonizes adenosine A2A and A2B receptors, which normally act on vascular smooth muscle to produce vasodilation by opening ATP-dependent potassium channels and decreasing calcium conductance (Addicott et al., 2009). It is believed that chronic caffeine intake (and perhaps even in low doses) can result in an upregulation of adenosine receptors, often yielding
tolerance to the physiological effects of caffeine (Addicott et al., 2009).

A study conducted by Evans and Griffiths (1999) looked at the effects of caffeine withdrawal in 76 participants in a total of four experiments (interval of caffeine dosing, caffeine maintenance dose, suppression of caffeine withdrawal, and duration of caffeine exposure). All participants were analyzed after administration of placebo after maintenance on 300 mg/day caffeine. Their results showed a significant increase in headache, headache with poor mood, tiredness, fatigue, confusion or bewilderment, and total mood disturbance, along with a decrease in activity and/or alertness, vigor, and friendliness upon substitution of placebo for caffeine (Evans & Griffiths, 1999).

Upon withdrawal of caffeine, this upregulation of adenosine receptors becomes notable as we observe an increase in cerebral blood flow following 20–24 hours without caffeine (Addicott et al., 2009). This response may be due to the increased number of adenosine receptors on the vascular muscles that are no longer antagonized by caffeine. This results in headaches along with other caffeine withdrawal symptoms discussed above.

**Toxic Effects—Chemical Sensitivity**

Another common factor to consider in the etiology of headaches is the toxic effects of drugs, chemicals, strong odors, pollutants, alcohol, and other noxious substances, collectively referred to as xenobiotics. The reactions to such substances can vary in form and severity depending on the person and range from a mild intolerance to certain chemicals, to a severe intolerance to many biological, chemical, or physical agents in a condition called Multiple Chemical Sensitivity (MCS). The symptoms can include headache, nausea, dizziness, cough, difficulty breathing, fatigue, muscle pains, poor focus, and mood changes.

An extensive list of the chemicals and substances that can stimulate a toxic reaction is beyond the scope of this discussion, but some of the more common triggers that we encounter on a daily basis include alcohol, cigarette smoke, carbon monoxide (car exhaust), industrial and household cleaners, cologne and perfume, industrial chemicals (such as solvents, paints, and lubricants), chlorine, and chemically treated products (such as new furniture, carpets, and toys).

There are several physiological responses to consider when analyzing how these substances affect the body and why some people are more strongly affected than others. These responses include first, the role of desensitization that appears dysfunctional in many patients with MCS; second, the role of phase I and phase II detoxification in the liver whereby total toxic burden and genetic detoxification single nucleotide polymorphisms (SNPs) play a role; and third, the role of genetic SNPs in the methylation cycle influencing the body’s ability to detoxify.

In 2012, Linus Andersson published his dissertation on a theoretical framework for chemical intolerance. Andersson (2012) discusses three existing theories that we have for chemical intolerance, namely neural sensitization, classical conditioning, and neurogenic inflammation. Neural sensitization suggests that certain neural networks in the brain, upon repeated exposure to a low to medium stimulus or a single strong stimulus, develop a permanent heightened response to that stimuli, which is expressed upon subsequent exposures to that substance or a cross-reactive substance (Andersson, 2012). In classical conditioning, a neutral substance is associated with a stressful or noxious event, which then elicits an unconditioned response. Once the mind has associated these two stimuli, upon future exposure the previously neutral stimulus is then reacted to as harmful (Andersson, 2012). In neurogenic inflammation, chemical intolerance is believed to elicit a similar inflammatory process as an allergic reaction; however, instead of producing IgE antibodies, there is abnormal sensitivity to chemicals of low molecular weight. This process may be one of adaptation occurring in several phases or of a proliferation of sensory c-fibers which up-regulate the inflammatory response (Andersson, 2012).

In his research, Andersson (2012) investigated the neural activity of both chemical-intolerant and chemical-tolerant individuals using EEG and fMRI technology and found that all the previous theories are merely different perspectives on the same physiological system and that there may be both a psychological and a physical basis to this condition. He found that the chemical-intolerant individuals did not become accustomed to a strong smell through a decrease in brain activity after an hour of exposure, whereas the control subjects showed a lessening of brain activity, indicating a sort of desensitization to the odor. Furthermore, in line with the neurogenic inflammation theory, he also found that the mucus membranes of chemical-intolerant individuals were more reactive than the controls in response to strong smells (Andersson, 2012). Perhaps our understanding of the process of chemical intolerance is still evolving into a single physiological mechanism, or perhaps there are several simultaneous reactions going on. Regardless of the mechanism, it appears that some individuals are far more sensitive to environmental substances than others.
The second process to consider in chemical intolerance is the functioning of phase I and phase II detoxification systems in the body. This is a process that naturally clears xenobiotics (products that are foreign to our biological system) from the body. Much of our research on these pathways has centered on drug actions, metabolism, and interactions, but the process extends much wider to include all foreign agents that must be properly eliminated from the body.

Phase I detoxification is a process that modifies xenobiotics through the action of various cytochrome enzymes. This starts the detoxification process by altering the chemical structure of toxins, rendering them sometimes more and sometimes less toxic than their original forms. Phase II detoxification involves conjugating the metabolites from phase I, usually to render them more hydrophilic for excretion. Conjugation pathways include acetylation, methylation, sulphation, glucuronidation, glutathione conjugation, and glycine conjugation.

There are several mechanisms that can decrease the effectiveness of detoxification, including nutrient status and genetic mutations. Almost all of the metabolic pathways involved in detoxification are dependent on nutrient cofactors (minerals and vitamins) whose supply will limit the rate of detoxification. Ensuring a patient is ingesting, digesting, and absorbing these nutrients is a vital consideration for adequate detoxification to occur.

Genetic mutations are also under thorough investigation for their role in detoxification. Of particular note are the pathways of acetylation and methylation. N-acetyltransferase is an enzyme coded by the NAT gene and is responsible for a large portion of phase II detoxification. Due to genetic variations, many people have a decreased activity of this enzyme; these individuals are referred to as slow acetylators. There is much variability between ethnic groups in terms of the slow acetylator phenotype: 40%–70% of Caucasians and African Americans are affected, along with 10%–20% of Japanese and Canadian Inuit, and more than 80% of Egyptians and some Jewish groups (Ma, Woo, & McLeod, 2002). The slow acetylators have been most studied in terms of drug metabolism, but the detoxification pathways of many nondrug chemicals are also affected by a decrease in acetylation. It is postulated that this can be one factor in the increased sensitivity of some individuals to chemicals and substances in the environment.

The second important pathway to consider is the methylation cycle. Through a similar mechanism to acetylation, many people have genetic single nucleotide polymorphisms (SNPs) in one or more of the enzymes in the methylation cycle. The repercussions of such polymorphisms can be vast, and vary in severity depending on the SNP and the number of abnormal copies that the individual carries. Methyltetrahydrofolate reductase (MTHFR) is one enzyme that is central to the processing of the methylation cycle; it is responsible for converting 5,10-methylenetetrahydrofolate into the activated form 5-methyltetrahydrofolate, or put more simply for activating folate for use in the body. There are several genes responsible for producing the MTHFR enzyme; mutations to the C677T gene appear to be the most severe in terms of biological consequences. MTHFR enzyme deficiencies have been associated with increased risk of myocardial infarction, stroke, venous thrombosis, several forms of cancer, congenital defects, inflammatory bowel disease, autism, infertility, many neuropsychiatric conditions, and impaired detoxification (Garilli, 2012). Ben Lynch, a pioneer in the field of genetic research, cites as many as 64 conditions that are associated with MTHFR defects (Lynch, 2011). C677T mutations again vary among ethnic groups: the prevalence of homozygous mutations (two abnormal copies) among Caucasians is 6%–14%, Africans 2%, Hispanics 21%, Chinese 18%, and Japanese 12% (Garilli, 2012).

A thorough discussion of all the health implications of methylation SNPs is beyond the scope of this discussion, but most notable with regard to tension headaches is the role of the methylation cycle in phase II detoxification. When genetic abnormalities exist in this cycle, the system is functioning at a reduced capacity, the severity of which is dependent upon affected genes. This hypofunction causes the body to be more easily overburdened by toxic substances, analogous to a plugged drain pipe. In such a scenario, slow methylators can appear to be functioning optimally when they are consuming a whole foods diet rich in fresh fruits and vegetables, sleeping sufficient hours, inhaling uncontaminated air, and are not exposed to xenobiotics (such as prescription or recreational drugs, alcohol, carbon monoxide, and cleaning products). However, once these individuals are exposed to an environment in which one or more of these conditions is altered, their methylation cycle can become overburdened and symptoms arise, including emotional reactions, symptoms of toxicity, and, of course, long-term development of the chronic diseases mentioned above.

Headaches are a common side effect of many prescription medications, and a thorough naturopathic workup will include an investigation into the contributory role of a
patient’s medications. Sometimes these side effects are simply due to the drug’s mechanism of action, sometimes they are due to nutrient depletions caused by medications, and sometimes they are due to the type of chemical sensitivity and inadequate detoxification mechanisms discussed here.

To summarize, there are a number of factors that can influence an individual’s ability to detoxify, including inadequate desensitization to environmental substances (especially inhaled substances), genetic defects, overburdened detoxification pathways, and nutrient depletions. These alterations in the biochemistry can result in a systemic toxic burden, a complex clinical picture with headache as one of the primary symptoms.

**Conclusion**

The physiology of muscle contraction is a complex process with many contributory factors. This article has aimed to serve as an introductory discussion on several of the processes that a naturopathic doctor will investigate when identifying the underlying cause (or causes) of a patient’s muscle hypertonicity and/or tension headaches. This is in no way an exhaustive list of contributory factors, but is intended to serve as a reminder to clinicians of the multifactorial nature of muscle tension and to offer a guide to help them navigate a patient to relief through the correction of the root cause of dysfunction when one (or several) factors can be elucidated.

**References**


Tension Headaches from a Naturopathic Perspective


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