Introduction
Tension-type headache is the most common headache. Almost everyone has experienced one or more during their lifetime. Theories and mechanisms for tension-type headache differ. Some suggest a peripheral mechanism and others suggest it is a central process; some believe in a continuum of headaches [1]. Migraine with aura makes up one end and tension-type headache makes up the other. This suggests that the clinical presentation is a continuum and that there is a common mechanism that exists for all headaches [1]. Others think that tension-type headache is a distinct entity [2]. Precipitating factors such as stress, lack of sleep, anxiety, poor posture, jaw clenching and grinding, and cervical and temporomandibular abnormalities are linked to tension-type headache; it is debated if these symptoms are the cause of the headache. Tension-type headache and migraine can be localized to the occipital region or frontal regions. This often confuses the appropriate diagnosis with cervical disorders or temporomandibular disorders (TMD). Support for TMD and cervical dysfunction as perpetuating and aggravating factors, not as causative entities, is provided here.

Definition of Tension-type Headache
The International Headache Society (IHS) has divided tension-type headache into an episodic and a chronic form [3]. Episodic tension-type headache is defined as a headache that lasts from 30 minutes to 7 days, with a pressing or tightening (non-pulsatile) quality, mild to moderate in intensity, and bilateral in location. It is not aggravated by routine physical activity such as walking up or down a flight of stairs [3]. There is no nausea or vomiting, which are symptoms commonly associated with migraine. Photophobia or phonophobia are not common, although one may be present [3]. Physical activity tends to reduce these headaches. Tension-type headaches can be localized to any part of the head, but are often described as a tight band around the head. The IHS defines the episodic form as having greater then 10 lifetime attacks, but fewer then 15 attacks each month [3]. The chronic form occurs more than 15 times each month for at least 6 months [3]. Chronic tension-type headache is not associated with vomiting as it is in the episodic form, but patients can experience nausea, photophobia, or phonophobia [3]. Chronic tension-type headache is much less common then episodic tension-type headache [4]. There are few symptoms that separate episodic tension-type headache from migraine. The presence of pericranial tenderness has also been associated with tension-type headache. The cause of this tenderness is controversial. Many think that the pericranial tenderness provides the peripheral focus or cause; others think it is a manifestation of a central mechanism.

Epidemiology of Tension-type Headache
There are several large, general population, tension-type headache studies. In a population-based study, Schwartz et al. [5] showed that the overall prevalence of episodic tension-type headache during a 1-year period was 38.3%. In the same study, the prevalence of chronic tension-type headache was 2.2% during the same year. Women had a higher prevalence than men for both types. Another large, general population study was performed by Gobel et al. [6] in Germany. It showed an overall lifetime prevalence of tension-type headache to be 38%. Overall, general population
surveys of North America and western Europe show a 1-year prevalence of tension-type headache to range from 30% to 80%, with the majority of these headaches being episodic rather than chronic [4]. Definition differences and study differences may be the reason for this large variation. These studies show a large prevalence of tension-type headache, with a male to female ratio of 1:1.3 [4].

Pathophysiology of Tension-type Headache

The pathophysiology of tension-type headache is not understood. Most researchers think that tension-type headache is caused by local peripheral mechanisms; however, some think that there is a central mechanism to the pain [7]. Jensen [8] has postulated that episodic tension-type headache occurs through peripheral mechanisms and chronic tension-type headache occurs through central mechanisms. Pericranial muscle tenderness is associated with both forms of tension-type headache [9]. What is the relationship between this muscle tenderness and the headache? Does the headache cause the muscle tenderness, or vice versa? Unfortunately, we don’t know the answers to these questions. The continuum theory for headaches describes that there may be a common mechanism for most headache types [10]. Tension-type headache is at one end of the spectrum and migraine is at the other end. If there is a common mechanism, then tension-type headache would most likely be generated or affected by central mechanisms, which is hypothesized for migraine. Imaging and neurochemical studies have shown that migraine generation can likely be localized to the raphe nuclei, the locus ceruleus, and the periaqueductal gray matter [11,12•]. If the continuum theory of headache is correct, then these brain locations could also be involved in tension-type headache. These studies have not been done. Kellgren [13] was the first to indicate that there may be a correlation between muscle tenderness and pain. He injected an algesic substance (hypertonic saline) into a muscle and asked the patients to define the area in which they perceived pain. They mapped out patterns of referral similar to those seen in tension-type headache. He then injected local anesthetic into similar areas after the pain was initiated, abolishing it. These tender points became known as myofascial trigger points. Under what circumstances could referral take place in the patterns described? That is a question that needs to be answered.

Mense [14–16] described a hypothesis for muscle pain referral to other deep somatic tissues that are remote from the original muscle stimulation site. He criticizes the convergence-projection pain referral theory because there is little evident convergence at the dorsal horn from deep tissues. Mense’s hypothesis adds two new components to the convergence-projection theory. First, the convergent connections from deep tissues to dorsal horn neurons are opened after nociceptive inputs from muscle are activated. The connections that open after muscle stimuli are called silent connections. Second, the referral to muscle beyond the initially activated site results from central sensitization and spreads to adjacent spinal segments. The initiating stimulus requires a peripheral inflammatory process (neurogenic inflammation). In the animal model described by Mense [14–16], bradykinin was the noxious stimulus injected into the muscle. In the work by Kellgren [13], a hypertonic saline solution was used to trigger the referred pain. This seems to mimic what is seen in the animal model. It is unclear what triggers the muscle referral in the clinical setting in which there is usually no obvious inflammation-producing incident. Mense’s theory has been used by Simons [17] to discuss a neurophysiologic basis for trigger point pain. Simons [17] hypothesizes that there is a neurotransmitter release in the dorsal horn (trigeminal nucleus) when the tender area in the muscle is palpated, resulting in the activation of previously silent nociceptive inputs. This causes distant neurons to produce a retrograde-referred pain. This model accounts for most of the clinical presentations and therapeutic options seen in myofascial pain, but does not account for what initiates the peripheral tenderness that must be present to activate the silent connections. Perhaps a central nervous system-activated neurogenic inflammation, similar to migraine, stimulates nociceptors in muscle rather than around the blood vessel. Calcitonin gene-related peptide (CGRP), neurokinin A, and substance P have been used to demonstrate their contribution in myofascial pain [18]. Fields and Heinricher [19] have described a means in which the central nervous system may activate nociception. They describe the presence of "on" cells that, when stimulated, may produce activation of trigeminal nucleus nociceptors.

Tension-type Headache, Temporomandibular Dysfunction, and Cervical Dysfunction

Pain from tension-type headache may be located in the frontal, occipital, and parietal regions of the head. It is generally bilateral, but can be unilateral. These headaches are often described as pressing, tightening, aching, or a vice-like band around the head. This description may also apply to pain caused by cervical dysfunction and TMD.

The description of these three conditions can be similar and their pain pathways can overlap. The temporomandibular joints and their associated musculature are innervated by the trigeminal nerve. Headaches are also mediated through the trigeminal system. Thus, pain associated with TMD may be perceived as headache. The occipital portion of the head down through the neck is innervated by the C2–C7 spinal nerves. There is convergence of the upper cervical spinal nerves with the trigeminal nucleus [20•]. This convergence may result in headache from cervical dysfunction. Pain from TMD or cervical dysfunction may begin as peripheral phenomena, but central sensitization may occur with repeated afferent barrages of pain [21•]. This sensitization can result in expansion of the pain