Headache

 Each of us is likely to have experienced headache either sporadically or chronically. Indeed, it is estimated that 40% of the worldwide population suffers with severe, disabling headache at least annually. It is well that such a common ailment usually has a benign course, but headache may be the presenting symptom of life-threatening disease. Given this, and the frequency that the complaint is encountered in ENT practice, we as otolaryngologists should be comfortable with the evaluation, diagnosis and treatment of headache and facial pain.

 Brain parenchyma itself is not pain sensitive, but the meninges and supporting structures are heavily innervated. Pain may be elicited when these structures are inflamed or distorted. In the case of migraine, it is believed that activation of trigeminovascular structures is the source of the headache. Processes inside the cranial vault may produce pain referred to areas of the jaw, neck, face and scalp. Pathology within the anterior or middle cranial fossae may elicit pain that is referred to the scalp or face anterior to the coronal suture. Lesions in the posterior fossa cause pain in the more posterior portions of the head and upper neck. Pain arising from processes in the sphenoid or sella is commonly referred to the vertex.

 Much attention has been given to the sinuses and nose as a source of acute and chronic head and face pain. It is therefore worth a brief review of sinonasal innervation. The general sensory innervation of the mucosa of the nose and paranasal sinuses is from the **ophthalmic and maxillary branches of the trigeminal nerve**, with minor contributions from the greater superficial petrosal **branch of the 7th nerve**. Pain arising from processes within the sinuses is therefore frequently referred to the corresponding cutaneous dermatome innervated by the 5th nerve, or to the auricular or periauricular region due to the contributions of the 7th nerve. The mucosa of the sinuses is rather insensitive to pain, and the turbinates are more sensitive than the nasal septum. The region of the ostia of the sinuses is the most sensitive of any of the nasal structures.

**Tension-type headache (TTH)**

 Is the most common type of headache. It occurs in 69% of men and 88% of women over a lifetime and the annual prevalence is 63% in men and 88% in women. TTH can be further distinguished as “episodic” TTH (ETTH) or “chronic” TTH (CTTH). The distinction is made largely on frequency of occurrence – less than 15 days a month for ETTH and greater than 15 days a month for CTTH.

 Headaches last from 30 minutes to 7 days. They are often described as **pressing or** **tightening (non-pulsating)** in quality. The intensity is mild to moderate and may limit but not prohibit activities. Its location may be bilateral or variable. There is no aggravation with physical activity, nausea and vomiting is rare, and photophobia or phonophobia may occur though not simultaneously.

**Treatment :**

 Patients who acknowledge the role of stress in the etiology of their headaches, especially those with ETTH, are frequently well managed by biofeedback and stress reduction techniques. Posture correction and physical exercises should be prescribed as indicated. Patients with bruxism may benefit from a dental splint.

 For patients with ETTH, medications may be avoided, but when needed, the patient may do well with low dose benzodiazepines or amitriptyline once daily in a short course spanning several weeks. Pharmaceuticals are more likely to be necessary in the patient with CTTH.

 Abortive medications include aspirin, acetaminophen, aspirin-caffeine-butalbital or phenacetin combinations or short half-life non-steroidal anti-inflammatory medications (NSAIDs).

 Preventive medications include daily antidepressants, muscle relaxants and long half-life NSAIDs. Opiates and benzodiazepines may be effective but prolonged use is contraindicated. Daily NSAID use should be limited to less than one week. The treatment regimen employed must be individualized based upon the triggering factors elucidated in the history and physical exam findings.

**Migraines**

 Are perhaps the most studied of the headache syndromes. This is due in part to the high incidence and significant loss of productivity and limitation on quality of life suffered by those with the syndrome. It is estimated that 17% of females and 6% of males have migraine headaches. Onset is usually in the second or third decade. Migraines are characterized by headaches of moderate to severe intensity located **unilaterally with a pulsating quality**. Attacks last from 4-72 hours and are aggravated by routine physical activities. In order to meet diagnostic criteria, there must be nausea, vomiting, photophobia or phonophobia. Migraines may occur with or without aura. Migraine with aura is less common. Vision complaints are the most common manifestation of aura, but patients may experience paresthesia, aphasia, nausea and vomiting prior to the onset of headache. These findings are completely reversible and precede the headache by no more than 60 minutes.

 Migraines seem to have a triggering event that precipitates a sterile inflammatory response around intracranial vessels that is mediated by the trigeminovascular system.

 **Triggering factors** may include stress, menses, pregnancy and oral contraceptive pills, infection in the head and neck, trauma or surgery, red wine, aged cheeses, vasodilating medications, strong odors, irregular diet or sleep and bright sunlight or flickering lights.

 Recent studies have discovered serotonin receptor subtypes in the central nervous system that play significant roles in the neurologic changes and intracranial blood vessel change. Newly available treatments such as sumatriptan target these receptors. Several other neuropeptides have been identified as pro-inflammatory and are believed to play a significant role in migraine development.

**Treatment :**

 The treatment of migraine headaches may be approached using several strategies: aborting the attacks at their onset, controlling the pain once is fully evolved and reducing the frequency of attacks.

 Therapies aimed at **aborting an attack** should be started as soon as the premonitory or warning signs are noted. Abortive therapy has been revolutionized with the introduction of 5-hydroxytryptamine (5-HT) receptor agonists. These include sumatriptan, naratriptan ,rizatriptan and zolmitriptan. These medications have allowed the migraine sufferer to quickly and effectively treat attacks several times a month with minimal side effects. Other medications used to abort headaches include ergotamine tartrate, Butorphanol is a mixed narcotic agonist/antagonist available by nasal spray. It does have potential for abuse and chronic use is contraindicated. Many NSAIDs have been shown to be effective in migraine headaches. Lidocaine administered intranasally in 4% spray, either singly or in combination with nasal decongestants, has been shown to be effective, although of short duration.

 If abortive therapy fails, management should be aimed at **reducing the intensity of the pain** and controlling associated symptoms such as nausea and vomiting. It is desirable to avoid opiates for the treatment of migraine. Finkel *et. al.* recommend several treatment regimens:

 (1) prochlorperazine IV push that may be repeated in 20 minutes if no effect,

(2) Dihydroergotamine IV push followed by IV prochlorperazine,

(3) Chlorpromazine IV push, may repeat in 20 minutes if needed

 Patients experiencing 2 or more attacks per month should be started on a **prophylactic regimen**. Appropriate first steps are to limit the activities or factors that trigger the headaches. This may be effective by itself, but medical prophylaxis is often needed as well. Multiple antidepressant medications have been shown to be effective in the prevention of migraine headache. These include amitriptyline, nortriptyline, doxepin, trazodone, imipramine and desipramine. The newer selective serotonin re-uptake inhibitors (SSRI) including *Prozac* and *Zoloft*, have not been shown to be effective in migraine therapy. NSAIDs have some usefulness in the prevention of attacks as well as the treatment of the acute headache. β-blockers have been used with some success but are contraindicated in patients with depression, asthma or diabetes.Calcium channel blockers (verapamil, nifedipine, nimodipine) have shown some effectiveness preventing migraine attacks as well.

**Cluster headache (CH)**

 It is characterized by intensely severe pain (sometimes termed *suicide headache*) with boring **or burning qualities** located unilaterally in the orbit, supraorbital or temporal area. Attacks last from 15 to 180 minutes. The headache is associated with at least one symptom of autonomic hyperactivity: conjunctival injection, lacrimation, nasal congestion, rhinorrhea, forehead and facial sweating, miosis, ptosis or eyelid edema. Attacks occur between one every two days to eight per day. At least 5 such attacks must occur to meet the diagnostic criteria. Nausea and vomiting is uncommon and there is no aura. Onset is usually in the second to fifth decades. Cluster headache is the only headache syndrome with a male preponderance. It is associated with alcohol use and intolerance, and during an active phase or “cluster”, alcohol may precipitate an attack.

 **Treatment** is aimed at preventing an attack during a cluster. Once an effective therapy is discovered, it is continued for 6 to 8 weeks and then gradually tapered. Options for treatment include calcium channel blockers (nifedipine, nimodipine, verapamil), low dose daily ergotamine (*Bellergal*) and lithium carbonate (especially in chronic forms of CH).

**Temporal arteritis**

Itis characterized by daily headaches of moderate to severe intensity, scalp sensitivity, fatigue and various non-specific complaints with a general sense of illness. 95% are over 60 years old. The pain is usually **unilateral**, although some cases of bilateral or occipital pain do occur. The pain is **a continuous ache** with superimposed sharp, shooting head pains. The pain is similar to and may be confused with that of CH, but CH tends to occur in younger patients. The two may also be distinguished on physical exam when dilated and tortuous scalp arteries are noted. The erythrocyte sedimentation rate (ESR) is markedly elevated in temporal arteritis as well.

 Definitive diagnosis is made by artery biopsy from the region of the pain, although negative biopsy may be due to the spotty nature of the disease. High dose steroid therapy usually precipitates a dramatic decrease in head pain. Failure to respond to steroid therapy with a negative biopsy should call the diagnosis into question. If the diagnosis seems likely based on history and physical examination, steroids should be started immediately to avoid vision loss, the most common complication of the disorder occurring in 30% of untreated cases. The biopsy remains positive for 7-10 days from starting steroid therapy. Steroids may be tapered to an every other day maintenance schedule when the pain resolves and ESR normalizes. The disease is usually active for 1-2 years, during which time steroids should be continued to prevent vision loss.

**Trigeminal neuralgia**

 (formerly also known as tic doloureux) is characterized by **paroxysmal** pain attacks lasting from a **few seconds to less than two minutes**. The pain is severe and distributed along one or more of the branches of the trigeminal nerve with a sudden, sharp, intense stabbing or burning quality. Between attacks the patient is completely asymptomatic without neurological defects (facial numbness, loss of corneal reflex, change in taste or smell). The pain may be precipitated from trigger areas or with certain daily activities such as eating, talking, washing the face or brushing the teeth. The syndrome is most common in patients over 50. The course may fluctuate over many years and remissions of months or years are not uncommon.

 Medical treatment of the disorder includes carbamazepine, gabapentin, baclofen, phenytoin, sodium valproate. Tricyclic antidepressants (TCA) and NSAIDs may be used as adjuvant therapy. Opiates are usually ineffective. Surgical treatment is occasionally necessary when medical therapy fails to control the pain attacks.

**Glossopharyngeal neuralgia**

 is characterized by pain attacks similar to those in trigeminal neuralgia, but located unilaterally in the distribution of the glossopharyngeal nerve. Pain is most common in the posterior pharynx, soft palate, base of tongue, ear, mastoid or side of the head. Swallowing, yawning, coughing or phonation may trigger the pain. Management is similar to that for trigeminal neuralgia.

**Dental pain** Offending tooth usually tender on percussion.

**Temporomandibular disorders** **(TMD)**

 It includes a heterogeneous group of processes all with a similar clinical presentation. Common symptoms of TMD include temporal headache, earache, facial pain limited jaw opening or joint noise. The majority of TMD originate spontaneously with only 40% able to recall a specific event, usually trauma, preceding the onset of pain.

Pain that truly originates in the TMJ is rare and characterized by tenderness to palpation of the condyle and pain with joint movement.

 The vast majority of patients, 60%-70% have combined muscle and joint pain with muscle pain dominating the clinical picture. These patients usually have tenderness to palpation of the muscles of mastication.

**Sinus headache**

 While acute sinusitis is widely accepted and recognized as a cause of headache with pain referred to the skin or intracranial structures also innervated by the nerve branches providing the sinuses, chronic sinusitis or sinonasal abnormalities as a cause of headache has been more controversial. Chronic sinusitis is not validated as a cause of headache unless relapsing into an acute phase. Migraine and tension-type headache are often confused with true sinus headache because of similarity in location.

 The pain associated with acute sinusitis is commonly described as constant, **dull** and aching. Occasionally the pain is sharp and may be **worsened by** jarring of the head, bending forward or stooping. Although the discomfort is most often located over the acutely inflamed sinuses, the pain may be referred to other areas of the head or face based upon the anatomic structures sharing innervation with affected sinuses as described previously. In most instances, the pain will be **accompanied by** such symptoms as purulent nasal discharge, malaise and congestion.