CHAPTER 26

# Headache and Facial Pain

*Gregory P. Van Stavern*



Headache and facial pain are common complaints and represent a diverse range of etiologies, from benign to life- and vision-threatening. Headache, migraine in particular, has been described in the popular and medical literature for over 3000 years (1). Trepanation, a sign of neurosurgery, has been seen on Neolithic skulls dating from 7000 B.C. (Fig. 26.1). The Ebers papyrus, an ancient Egyptian prescription dating back to 1200 B.C., mentions migraine, neuralgia, and shooting head pains, and is thought to be based on earlier medical documents from 1550 B.C. (2). Indeed, it is estimated that over 90% of individuals have noted at least one headache over their entire life (3). Patients with headache may present initially to a neurologist or ophthalmologist, and are often misdiagnosed initially. Since correct diagnosis facilitates appropriate treatment, it is important for the clinician to be familiar with common causes of head and facial pain. This chapter will focus primarily on those causes of headache and facial pain most relevant to neuro-ophthalmologists.

## APPROACH TO HEADACHE AND FACIAL PAIN

Headache and facial pain result from disorders that affect the pain-sensitive structures in the head and neck, such as meninges, blood vessels, and muscles. Pain-sensitive structures responsible for head and facial pain are listed in Table 26.1.

A **history** is the first and most important step in the evaluation of head and facial pain; the examination and any ancillary tests serve to confirm or exclude what is already suspected. In many patients with headache and facial pain, the general neurologic and ophthalmologic examination is normal, and the correct diagnosis rests upon a thorough and accurate history. Most, but by no means all, of the more serious and life-threatening causes of headache present with acute onset of pain: a patient with his or her ''first or worst'' headache has a greater likelihood of harboring an ominous cause for the head pain than someone with a 10-year history of recurrent, low-grade headache that has not changed in character. Although a chronic and recurrent headache is more likely to represent a benign condition, patients with a headache distinct from previous headaches (in terms of location or quality of pain) are also more likely to harbor a serious underlying illness.

The **location** of the pain may prove helpful. Unilateral headache is an invariable feature of cluster headache and occurs with most migraine attacks. Patients with tension-type headaches generally describe bilateral pain. In patients with ocular pain, a primary ophthalmic etiology should be excluded, while paranasal pain localized to the sinuses might suggest sinus obstruction. A new temporal headache in an elderly patient might raise suspicion of giant cell arteritis (GCA). Figure 26.2 depicts regional pain in common primary headache syndromes. However, since most head and


**Figure 26.1.** Neolithic skull showing trepanation hole (ca. 7000 B.C.) (Courtesy of Nationalmuseet, Copenhagen. From Headache in Clinical Practice. Oxford, UK: Isis Medical Media, 1998.)

**Table 26.1**
**Pain-Sensitive Structures of the Head and Neck**

| Extracranial |
|---|
| Skin and blood vessels of the scalp |
| Head and neck muscles |
| Second and third cervical nerves |
| Periosteum of the skull |
| Eyes, ears, teeth, sinuses, oropharynx |
| Mucous membranes of nasal cavity |
| **Intracranial** |
| Dura mater |
| Anterior and middle meningeal arteries |
| Trigeminal (V), glossopharyngeal (IX), and vagus (X) nerves |
| Proximal portions of internal carotid artery and branches near the circle of Willis |
| Periaqueductal gray matter |
| Sensory nuclei of the thalamus |

facial pain is ultimately mediated by the trigeminal nerve, referred pain may result in false localization.

A complete headache history should include a description of the **quality** of the pain. Migraine headache is typically reported as throbbing; tension-type headache is often described as dull and aching, with a band-like sensation around the head. The pain produced by intracranial mass lesions is often dull and steady. A sharp, lancinating quality suggests neuralgic pain. The **tempo** and **evolution** of the pain may suggest a specific diagnosis, since some headache syndromes have fairly characteristic temporal patterns (Fig. 26.3).The onset and duration of the attacks, including time

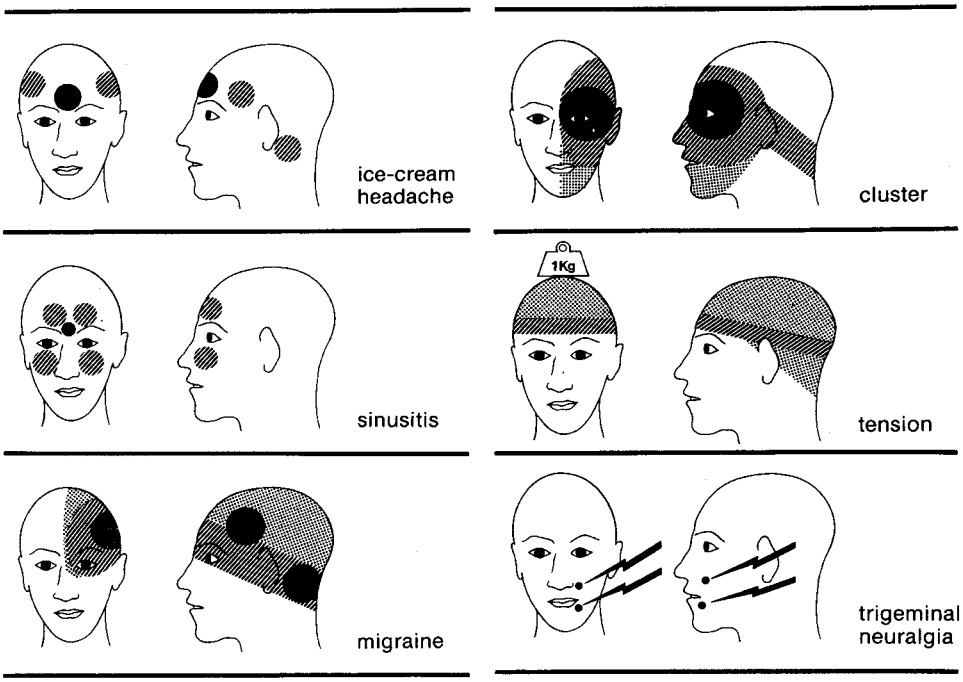


**Figure 26.2.** Sites of common head and facial pain syndromes. (From Lance JW. Migraine and Other Headaches. New York: Charles Scribner's Sons.)

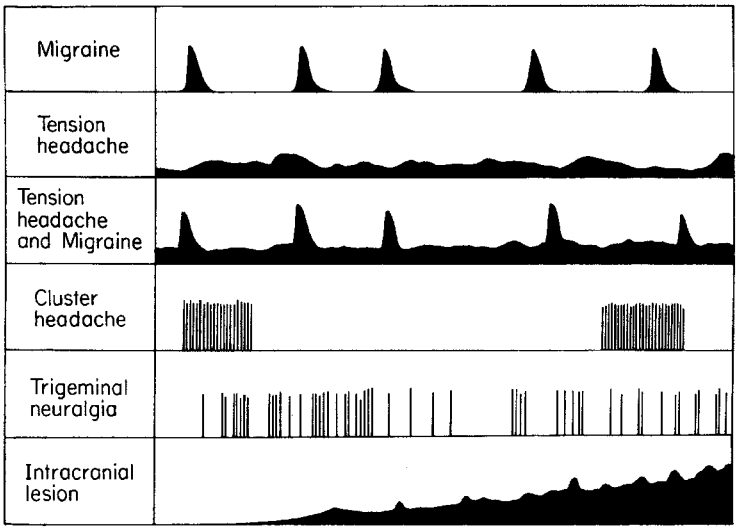


**Figure 26.3.** Temporal patterns of headache. (From Lance JW. Mechanism and Management of Headache. Ed 5. Oxford, UK: Butterworth-Heinemann, 1993.)

to peak intensity, should be determined. The frequency of attacks should be ascertained; a change in frequency of a preexisting headache raises concern about a secondary headache syndrome superimposed upon previous primary headaches.

**Exacerbating** and **relieving** factors may provide clues about etiology. Migraine is often provoked by certain foods, worsened by routine physical activity, and may be worse during menses. A positional headache might suggest either increased or decreased intracranial pressure. Pain provoked by chewing or eating might raise suspicion of giant cell arteritis or temporomandibular joint dysfunction. Precipitation of headache with alcohol is common with cluster headache. Migraine headaches are frequently relieved by darkness and sleep.

**Associated focal neurologic symptoms** which do not meet criteria for migraine with aura might merit further investigation. The general medical history is valuable, as well. Previous head injury might suggest post-traumatic headache. Medical co-morbidities such as hypertension may be relevant when choosing pharmacologic treatments. Family history should be reviewed, as some headache syndromes have a genetic component. A detailed medication history is important, since analgesic overuse may complicate many chronic headaches. Medication history also may help guide future medical treatment.

The **general examination** may help identify an underlying systemic illness. Fever might suggest an intracranial or meningeal infection, but headache can accompany nearly any systemic infectious illness. Inspection of the skin may reveal cutaneous lesions associated with a particular diagnosis (e.g., vesicular lesions in patients with herpes zoster). Blood pressure measurement is important. Although chronic hypertension does not cause headache, an acute rise in arterial pressure might cause sudden head pain. Stroke and subarachnoid hemorrhage (both of which cause headaches) are both associated with an acute rise in blood pressure. Palpation of the temporal arteries should be performed in all cases where giant cell arteritis is suspected: focal tenderness over the artery, a palpable cord, or obliteration of the pulse increase the likelihood of the disease. Meningeal signs should be sought in patients with an acute onset of headache. Recall that meningeal irritation causes nuchal rigidity in the anterior-posterior rotation, while cervical spine disorders restrict movement in all directions.

The neurologic and neuro-ophthalmic examination is aimed primarily at confirming or excluding a focal deficit, or findings localizable to a specific cranial structure. Such finding would then prompt appropriate further evaluation. The **neuro-ophthalmologic examination** is essential in the evaluation of a patient with headache or facial pain. Funduscopic examination should be performed systematically looking for papilledema; the finding of a Horner syndrome in a patient with unilateral facial or head pain may suggest cluster headache or internal carotid dissection; a 6th nerve palsy or comitant esotropia may raise the possibility of raised intracranial pressure. Finally, ocular causes of facial pain should not be overlooked and a red eye or orbital symptoms should prompt a detailed ophthalmologic examination.

In many patients, the examination is entirely normal, and the physician must decide whether or not to obtain neuroimaging. Since computerized tomography (CT) and magnetic resonance imaging (MRI) are noninvasive and have become widely accessible, many patients with headache will be imaged, whether or not there are clear indications. This is, in part, fostered by the medicolegal climate in the United States. However, neuroimaging is expensive and inconvenient for many patients. In addition, if neuroimaging is required, it is preferable to order the correct study the first time (e.g., MRI or MRA rather than CT). The American Academy of Neurology position paper, based on an analysis of 16 CT and MRI studies, concluded that the routine use of CT or MRI was not warranted in patients whose head-

aches fit a broad definition of recurrent migraine and who have had no recent change in headache pattern and have no focal neurologic deficits on examination (4). The U.S. Headache Consortium guidelines evaluated the utility of neuroimaging studies in patients with headache. Significant intracranial lesions were found at a rate of only 0.18% in patients with migraine and normal neurologic examinations (5). Some patients with migraine may have non-specific abnormalities on MRI (6,7). Igarashi and colleagues (6) found that 31% of migraineurs had small foci of T2 hyperintensity on MRI; the signal changes were most commonly seen in the centrum semiovale and frontal white matter (Fig. 26.4). A significantly lower number of matched controls without migraine had similar signal changes on brain MRI. There was no correlation between MRI changes and migraine type; ergotamine use; or frequency, duration, or intensity of migraine. These MRI findings are of uncertain clinical significance.

Forsyth and Posner (8) evaluated 111 patients with primary and metastatic brain tumors, and identified headache in 48%. Many of these patients had headaches that were similar to previous headaches, but now were associated with seizures, prolonged nausea, confusion, or other neurologic findings. The authors concluded that a change in headache symptoms is an indication for neuroimaging. In most cases, MRI is superior to CT scan for evaluation. There are a number of disorders causing headache that may be missed by CT scanning, particularly if contrast is not administered (Table 26.2).

There are few published studies evaluating the value of neuroimaging in some of the less common headache disorders. The decision to image in those circumstances should be made on a case by case basis. Patients with chronic eye pain and a normal neuro-ophthalmic examination are a too-common source of referral to ophthalmologists and neuro-ophthalmologists. These patients are often classified as having ''atypical facial pain'' and in many cases have a substantial component of analgesic overuse and rebound. The value of imaging in these patients is uncertain. Golnik et al. (9) reviewed 101 patients with unilateral eye and facial pain and noted no findings on examination that would account for the pain. Neuroimaging was normal in 93% of patients; in those with abnormal imaging it was unclear whether the pain was attributable to the lesions found. They concluded that the yield of neuroimaging in a patient with unilateral eye pain and a normal examination is low. The decision to image should be made on a case by case basis. Table 26.3 lists reasonable indications for neuroimaging.

For many patients the etiology of headache and facial pain can usually be ascertained after a careful history and examination. An awareness of relevant neuroanatomy as well as typical presentation for common headache syndromes is essential. If neuroimaging is indicated, the type if image ordered should be the one most likely to confirm or exclude certain diagnoses. For example, CT scanning has high sensitivity for bony lesions and acute hemorrhage and is the test of choice for acute head trauma and neurologic deficits with abrupt onset. MRI has much higher sensitivity

**Table 26.2**
**Lesions that May Be Missed by CT Scan**

| |
|---|
| Low-grade glial tumors |
| Vascular malformations (particularly in noncontrast CT) |
| Venous sinus thrombosis |
| Cavernous sinus and sellar lesions |
| Meningeal disease |

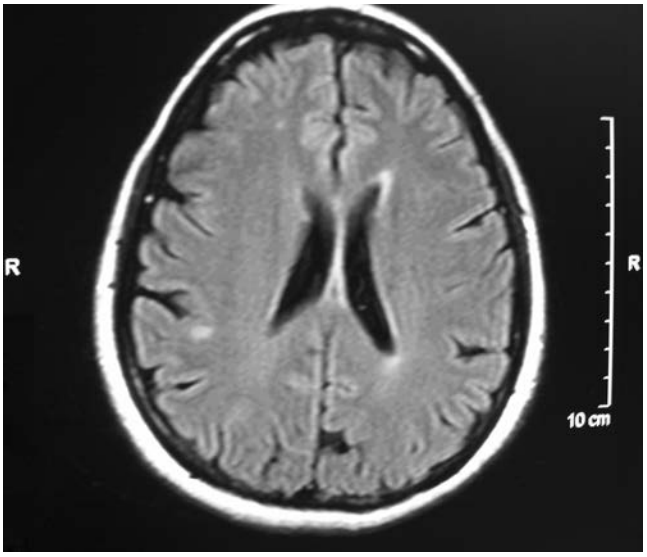


**Figure 26.4.** MRI of a 53-year-old woman with a long history of migraine. FLAIR sequences. The size and location of the white matter lesions are relatively nonspecific and are atypical for demyelinating disease.

**Table 26.3**
**Indications for Neuroimaging in Patients with Head and Facial Pain**

| History |
|---|
| The first or worst headache of the patient's life, particularly if abrupt onset |
| Change in frequency, severity, or clinical features of headache attack |
| Neurological symptoms that **do not** meet criteria for migraine with aura |
| Hemicrania that is **always** on the same side and is associated with contralateral neurologic symptoms |
| Positional headache |
| Lack of improvement with conventional therapy |
| **Examination** |
| Localizable deficits on neurologic or neuro-ophthalmologic exam |
| Persistent neurologic or neuro-ophthalmologic deficits |

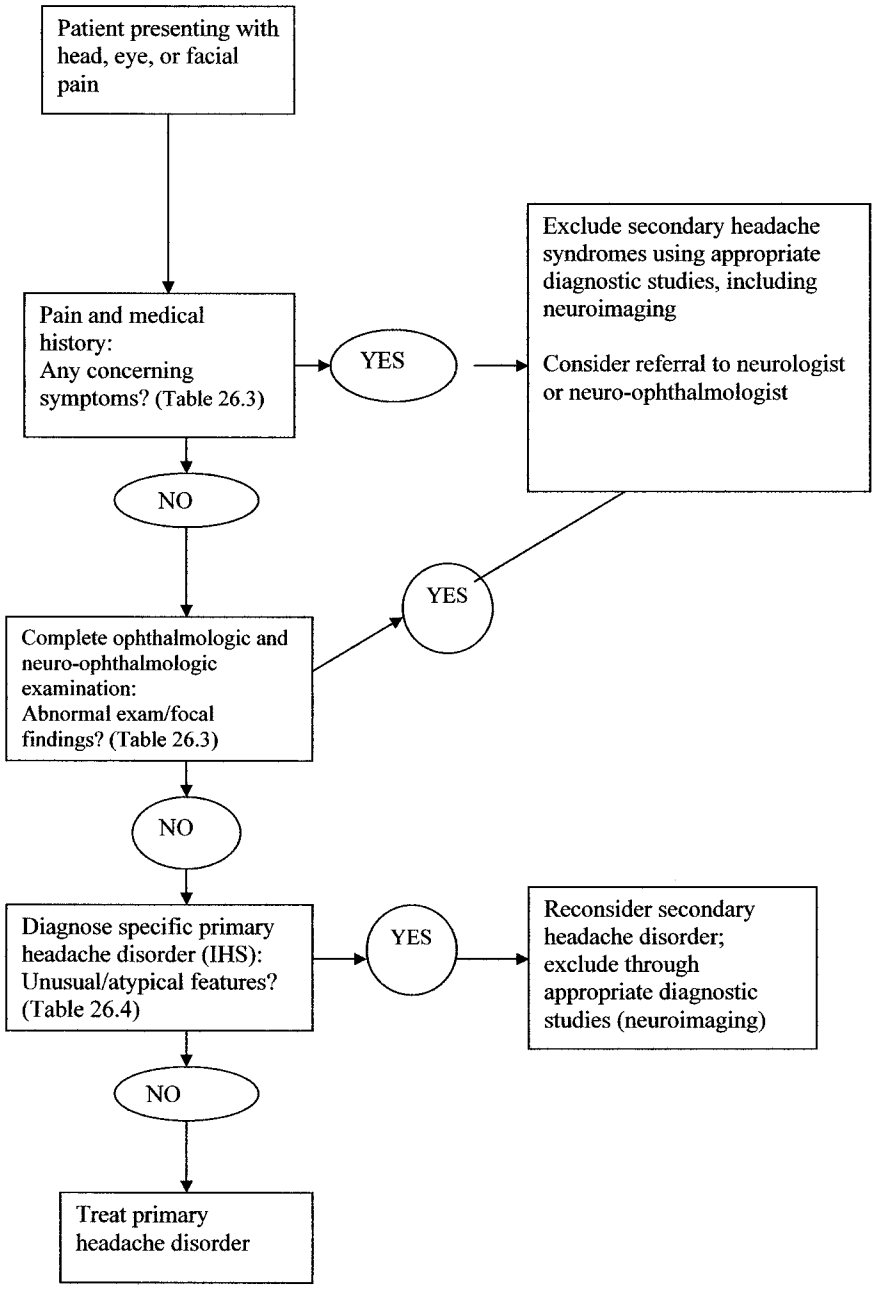


**Figure 26.5.** Algorithm for management of patients with headache.

for posterior fossa, cavernous sinus, and sella turcica lesions. The MRI may miss orbital pathology unless fat-suppressed orbital sequences (in which the bright T1 signal from orbital fat is eliminated) are specifically ordered. Magnetic resonance angiography and venography are specialized MRI sequences that may be ordered when arterial (e.g., dissection, aneurysm) or venous (e.g., cerebral venous thrombosis) disease is suspected. Figure 26.5 presents a reasonable algorithm for evaluating patients presenting with head and facial pain. It is not intended to be all-inclusive, but rather provide a framework for managing patients with headache.