

## CHAPTER 3

### Headache and Face Pain

Headache (HA) is one of the most common medical symptoms and should receive the physician's close attention. HA can be the initial and only symptom of serious intracranial disease such as brain hemorrhage, meningitis or brain tumor. Because of the potential seriousness of HA, the physician's first obligation is to rule out these life-threatening conditions.

Fortunately, most HA are "benign"; clinical features do not suggest the need for neuroimaging studies. Certain patients with "benign" HA features undergo unnecessary neuroimaging studies and are found to have incidental pathology, e.g., brain cysts, intracranial calcification; these have no pathophysiological role in the head pain. The symptoms of this "benign" HA however, can be quite disabling, and the physician should carefully characterize the HA and establish a treatment plan to relieve the head pain. Other HA has "malignant" or worrisome features, e.g., awaken patient from sleep, accompanying diplopia, fever or constitutional features; these patients require neuroimaging studies to exclude serious structural condition.

#### HISTORY

The clinical history is most important in analyzing the cause of HA. Failure to explore the historical details can lead to unfortunate errors. Too many patients with bleeding aneurysms or infectious meningitis are sent home from emergency departments with prescriptions for analgesic medication, only to return comatose the following days. All patients with new onset HA or chronic HA, but which has changed pattern, require neuroimaging studies.

The first question in the history should establish whether the patient is experiencing "new" or "unusual" HA or is seeking help for chronic unchanged HA. Severity of HA does not define HA type - some patients refer to "regular" or "usual" HA and others as "severe" or "migraine". This is incorrect, as migraine is defined by clinical features - some migraines are severe, others are mild. The patient who has never previously experienced HA and consults the physician for first-time HA demands careful attention. This pattern is referred to as "first and worse headache" and these require neuroimaging studies. HA of recent onset is much more likely to be the symptom of serious intracranial disease than is chronic HA.

The second feature of the history that will prove invaluable is a complete, step-by-step, temporal profile of the HA. Onset, location, and progress of pain all should be meticulously recorded. This temporal head pain profile is very important in determining the cause of the pain. [Box 3-1](#) can be used as a guide in history taking. Severe onset of headache that reaches maximal severity within minutes (thunderclap) suggests subarachnoid hemorrhage (SAH) or intracranial hypertension. In patients with chronic recurrent HA, it is important to obtain life history. Information that should be obtained is listed in [Box 3-2](#).

Box 3-1

1. Were there any antecedent or associated factors present, such as emotional or physical stress (including such factors as exercise or sexual intercourse); concomitant medical illness (e.g., hypertension); glaucoma; infection of sinuses, ears, or eyes; head trauma; or recent dental work? If the patient is female, is she currently using birth control pills? Does headache occur most frequently during ovulation or during menstrual periods? Does any food (e.g., monosodium glutamate used in Chinese food, alcohol, cheese, wine) or medication (e.g., vasodilators, drugs containing histamine or tyramine) precipitate headache?
2. Were any symptoms noticed immediately before onset of headache (aura), such as wavy lines in front of eyes, loss of vision in part of the field, scotoma (holes) in the vision with or without scintillating margins, weakness of one side, numbness, or diplopia?
3. Did pain come on suddenly (thunderclap) or build up gradually? How long did it take to reach maximal intensity? Does headache awaken patient from sleep? What time of day does headache usually occur (e.g., on awakening, does headache awaken patient from sleep (hypnic), late in day, at work, and so on)?
4. Where was pain? Unilateral? Occipital? Temporal? Frontal? Generalized? Does headache begin unilaterally and become bilateral?
5. What was character of pain? Throbbing? Sharp and stabbing? Tight, steady, and aching? Band-like?
6. How long did it last?
7. Was pain associated with nausea, vomiting, or sensitivity to light or sound?
8. Does anything exacerbate pain, such as coughing, sneezing, leaning over, or exerting physical effort?
9. Did pain change during the course of the headache? Unilateral to bilateral?
10. Did pain suddenly leave, gradually leave, or still present?
11. What medication was tried, and how successful was it?
12. Complete review of neurologic and medical symptoms should also be taken. This portion of history is very important in diagnosing headaches associated with intracranial disease. For example, transient arm weakness at onset of a unilateral throbbing headache suggests migraine, whereas persistent weakness and prolonged headache is more suggestive of brain hemorrhage or tumor.

**Box 3-2**

1. Age at onset
2. Frequency of headaches (an important factor in treatment planning)
3. Medications used and their efficacy
4. Periods in life during which headaches were either frequent or absent (e.g., college, marriage, menopause)
5. Relation to mental state (e.g., depression, stress)
6. Family history of headaches
7. History of motion sickness as a child (finding in some patients with migraine)

Chronic HA is frequently caused by emotional stress; migraine occurs during letdown periods (weekends, vacations, holidays) and tension HA occur during periods of emotional stress. It is important to point this out tactfully to the patient and then briefly ask about some aspects of the patient's personal life. Questions can be asked about family relationships, job situations, financial stability, and, if appropriate, sexual relations. Frequently, life stress is not unpleasant or emotionally significant but merely the product of a very demanding lifestyle in which many factors must balance perfectly if life is to run smoothly. For example, the single working mother may do well as long as the car works, children stay well, and she can leave work at her routine time. Keeping to such tight schedules impose enormous stress by virtue of ever-present possibility that one element can tip the delicate balance. Finally, it is wise to ask specifically about any feelings or signs of depression (e.g., sadness, sleeping or eating problems, crying spells, and lack of energy) because the patient may not mention these spontaneously. In addition to specialized HA history, complete neurological examination should be performed. Aspects of examination that should receive special note are listed in [Box 3-3](#). The extent of laboratory evaluation will depend on how strongly the physician suspects significant intracranial disease; this evaluation is based on chronicity of problem and findings from the history and physical examination.

## **DESCRIPTION OF THE CLINICAL SYNDROMES**

The discussions that follow help understand the various diseases that should be considered in differential diagnoses of head pain. The appropriate tests required to establish or rule out each are outlined in each section. The most common HA syndromes are discussed in decreasing order of prevalence. Major features of each are listed in [Table 3-1](#). The boundaries between primary HA types are quite indistinct; many patients experience features of both tension and migraine concurrently. In HA pathophysiology, the following unifying hypothesis has been proposed. The model called vascular-myogenic proposes a complex interaction between pain fibers in muscles

and blood vessels as well as with nuclei in the brain stem. HA process can begin in any of three structures and then affect the other two.

### **Box 3-3**

Fundusoscopic examination: papilledema and subhyaloid hemorrhage (seen in subarachnoid hemorrhage)

Level of consciousness: lethargy suggests an intracranial lesion or systemic disease

Pupillary asymmetry: aneurysm or herniation from mass lesion

Extraocular muscle weakness: aneurysm or herniation from mass lesion may compress ipsilateral oculomotor nerve to cause medial rectus paresis especially on near gaze; intracranial hypertension can cause abducens nerve paresis with lateral rectus paresis and diplopia especially on far gaze

Reflex asymmetry and lateralizing motor findings: unilateral brain lesion

Imbalance and abnormal cerebellar signs: posterior fossa tumors (cerebellum, brain stem)

Nuchal rigidity: meningitis and subarachnoid hemorrhage

## **Tension-Type Headache (Muscle Contraction Headache or Psychogenic Headache)**

Everyone has experienced a tension-type HA at some time in their life. Brought on by stress or depression, it is usually promptly relieved by mild analgesics, rest, and stress removal. For some people, however, tension HA are a severe recurrent problem that does not respond to simple measures; usually these patients seek medical assistance. In these patients, head pain occurs almost daily. Tension HA can be brief and episodic or continuous. The HA may not be associated with chronic muscle contraction.

The pain is often caused by steady contraction of muscles attached to skull (i.e., frontalis, temporalis, trapezius, paraspinal); this leads to tissue ischemia. There is abnormal neuronal sensitivity and pain facilitation with hypersensitivity of trigeminal nucleus. This indicates central rather than peripheral (muscle contraction) mechanism. The sustained muscle contraction causes lactic acid accumulation and this can trigger vasodilatation to induce vascular component. It seems that day-to-day stress of coping with busy life, often coupled with underlying anxiety, suppressed anger, hostility, and frustration, causes patients to tense these muscles for long periods of time, as though they were animals preparing to fight or flee (fight or flight response).

The pain is most commonly described as bilateral tightness starting in occiput, brow, or temples. When dull aching non-pulsatile HA is located in different regions, they are multiple differential considerations: frontal suggests "sinus"; temporal suggests "temporomandibular dysfunction"; occipital suggests "cervicogenic mechanism". HA often spreads to encircle the head such as a tight hatband or vise. The pain starts without warning. It is mild at first but builds in intensity over a period of hours. The pain characteristically lasts several hours or until patient sleeps or takes adequate analgesic medication. In some patients, pain is very severe, can last for

days, and can eventually throb or pulsate simulating migraine. When severe throbbing pain occurs, tension HA is labeled migraine. In keeping with new HA model, it is not difficult to see that active interaction of cranial musculature, cranial vessels, and central nervous system structures can produce overlapping symptoms. With or without medication, pain recedes gradually as it began. Tension HA most commonly occur in the afternoon but can be present on arising. Anxiety-producing dreams can produce nocturnal HA that awaken patient; therefore the belief that nocturnal HA are always an ominous sign (e.g., brain tumor) is not always correct.

Tension HA can occur at any age. Women are most commonly afflicted than men and exacerbations typically occur at stressful times of life. Physical examination demonstrates tightness or tenderness in affected muscles. In patients with typical tension HA and normal neurological examination, expensive laboratory evaluation is unjustified. There can be biochemical basis of tension HA such as low platelet serotonin level or elevated gamma-amino butyric acid levels.

Treatment is not always simple and requires sympathetic concern of the physician. Therapeutic success rests largely on establishing good physician-patient rapport. Important and useful steps are listed in [Box 3-4](#). Because most tension HA is caused by patient's lifestyle, treatment plan must take this into account. Many patients do not recognize the origin of their problem, and frank discussion about their lifestyle situation is often helpful. Other patients are

#### **Box 3-4**

Physician should acknowledge that real pain is present; too many patients are brushed off or merely handed a prescription and told to live with their problem.

Because many patients fear they may have brain tumor or other serious disease, it is inadvisable to reassure them that no evidence of a serious condition has been found; however, certain patient require a "therapeutic" CT/MRI to reassure them of absence of intracranial pathology.

A realistic goal should be set with patient. The number and intensity of headaches can probably be reduced, but not necessarily eliminated completely.

aware of their emotional status and welcome the opportunity to express their feelings openly. In either case, being able to talk about their situation with help from their physician, many patients can gain new perspectives. If major psychiatric problems are present, referral to a psychiatrist is warranted. Other avenues of treatment should also be used. Gentle neck massage and muscle relaxation can be helpful, as can taking a few minutes out of a busy day to lie down. Relaxation can be aided by biofeedback techniques. In biofeedback, patients are responsible for their treatment. Medications are another approach for abortive and prophylactic HA. Three basic classes of drugs are used: muscle relaxants, psychotropics (antidepressants or tranquilizers), and analgesics. Muscle relaxants such as methocarbamol (Robaxin) (750 mg, 4 to 8 pills daily) and tizanidine (Zanaflex) 2 mg tid help relax tightened muscles in some patients. Low doses of tricyclic antidepressants (e.g., amitriptyline, doxepin, or nortriptyline, 10 to 50 mg at bedtime) have been very effective in treating chronic HA. In patients whose HA is part of depression, full therapeutic dose of antidepressant medication is often helpful.

## Chronic Daily Headache (CDH)

Some patients have CDH which occur more than 15 days per month, and these are not related to systemic illness or structural lesion. These CDH are divided into 2 types based upon duration - long duration (longer than 4 hours) and short duration (shorter than 4 hours). Long duration CDH includes: (1) chronic (transformed) migraine, (2) chronic tension, (3) hemicrania continua. Short duration CDH include: (1) chronic cluster, (2) paroxysmal hemicrania, (3) hypnic (sleep), (4) idiopathic stabbing head pain, (5) short lasting unilateral neuralgic HA with conjunctival injection and tearing. CDH usually occurs in patients who initially had episodic migraine but later HA become more constant and has less migraine features. Transformation of episodic migraine to CDH usually occurs in association with medication overuse. CDH may also occur in patients who initially had episodic and later develop daily HA; frequently this change occurs in the setting of analgesic overuse. Hemicrania continua is a unilateral HA which may fluctuate in intensity but not completely disappear and is associated with autonomic dysfunction, e.g., ptosis, miosis, tearing, eye redness, sweating. Head pain is characterized by jabs, jolts and stabbing pain. This is usually indomethacin responsive HA.

Patients with CDH may overuse non-opiate or opiate analgesics, ergots, triptans, as the patient has become dependent on medication and develop "rebound" HA when medication is withdrawn. It is in the management of CDH that the HA specialists earn their living. The source of pain probably involves peripheral excitation of afferent sensory fibers and enhanced central sensitization with impaired pain modulation. When episodic HA has transformed to CDH, periodic reevaluation is warranted to exclude unrecognized systemic or structural disorder.

Treatment of CDH includes: (1) discontinuation of over-used medication (substitute phenobarbital for butalbital; methadone or clonidine for opioids, and substitute nonsteroidal anti-inflammatory medications for analgesics as the patient is detoxified), (2) hydrate patient adequately, (3) control nausea and pain with antiemetics, (4) intravenous dihydroergotamine with antiemetics, (5) intravenous corticosteroids, (6) treat coexisting psychiatric comorbid conditions, (7) utilize prophylactic condition (tricyclic antidepressant, divalproex, topiramate, gabapentin, beta blockers, calcium channel blockers), (8) patient education.

## Migraine

To many persons any severe HA is a "migraine". This is not always the case! Migraine has specific clinical features; severity is only one. Six basic diagnostic features strongly suggest migraine include: (1) typical sensory phenomena which are usually, but not necessarily, visual (2) unilateral pounding head pain, (3) HA worsened by exercise or activity, (4) nausea and vomiting early in a HA before medicine is taken, (5) distinct onset and termination of HA, (6) relatively specific response to ergot or triptan preparations. There are unique phases to migraine: (1) prodrome – early change in behavior or mental state, e.g., irritability, restlessness, difficulty concentrating, food craving, thirst, fluid retention; (2) aura - scintillating scotoma, phosphenes, micropsia, metamorphopsia; (3) head pain - throbbing quality with gradual buildup and should follow and not precede sensory aura; (4) resolution - HA resolves and patient feels irritable, listless, fatigued with impaired concentration. There are multiple migraine comorbid disorders - hypertension, stroke, mitral valve prolapse, anxiety-panic disorder, depression, epilepsy, vertigo, asthma. Frequency of attacks varies but tends to increase during times of stress or major life changes. Fatigue, hunger, certain tyramine-containing foods, contraceptive pills, change in hormones during menstruation or menopause, red wine, smoking, monosodium glutamate,

smelling certain odors, and emotional factors can bring on migraine. As in tension HA, there is an increased prevalence of migraine in females; there is also a strong familial tendency. Onset is usually in the 20s, but onset in childhood is not uncommon.

Migraine is disorder of young people. Early onset and strong familial association suggest genetic factors. Neuronal calcium and magnesium channel dysfunction can alter 5-hydroxytryptamine release; this triggers migraine attacks. Migraine is a primary neuronal disorder with secondary vascular effects causing reduced cerebral blood flow; this reduced-perfusion migrates from occipital cortex forward at 2-3 mm per minute. This oligemia does not correspond to specific vascular territories and is not due to arterial vasoconstriction. It is due to neuronal dysfunction. Migraine is associated with chronic state of neuronal hyperexcitability and instability of multiple brain chemicals - serotonin, dopamine, gamma-amino-butyric acid, calcium, magnesium, glutamate. During migraine episode, platelet serotonin levels drop and urinary excretion of 5-hydroxyindoleacetic acid (main serotonin metabolite) increase. Also, serotonin depletion precipitates migraine attack and serotonin receptor agonists have become important medications in aborting acute migraine. Migraine pain can be related to sterile neurogenic inflammation and this can be aborted by serotonin receptor agonists and possibly explain the role of nonsteroidal anti-inflammatory medications in reducing migraine pain. The migraine patient has some type of episodic central autonomic imbalance that causes changes in cerebrovascular tone. Serotonin receptor agonists have vasoconstrictive properties, e.g., effect on coronary and cerebral vessels, but their effect on migraine relief involves neural structures and altered neurotransmitter function.

#### *Classic Migraine (Migraine with Aura)*

In this migraine, patient experiences distinct neurological aura. This is due to spreading cortical depression, not vasoconstriction. The aura is most often visual (flashing lights, blurring of vision, wavy lines, spreading scotoma with scintillating margins, or hemianopia), although hemiparesis, ophthalmoplegia, hemisensory symptoms, aphasia, and occasionally confusional behavior do occur. The spreading cortical depression occurs at 2 to 3 mm/minute. When focal neurologic signs appear, such as hemiparesis, the term *complicated migraine* is often applied. The aura typically lasts between 5 and 45 minutes and usually precedes the head pain. If HA precedes the aura, strongly consider an alternative diagnosis to migraine. The HA then builds up over several hours. It is unilateral, pounding, and very severe. It is usually associated with an uneasy feeling in the stomach, nausea, vomiting (sick headache), and sensitivity to sound and light. During the migraine, neurological deficits usually fade so that when patient is seen by physician, neurological aura has faded and examination is normal. Pain lasts from several hours to all day and can recede rapidly or slowly. Attacks occur from once in a lifetime to daily. Each attack starts unilaterally but can spread. Most patients have a usual side for migraine pain, but it is characteristic that on occasion HA will begin on opposite side. If patient has had all migraine on a single side, physician must consider possibility that an underlying focal brain lesion, e.g., vascular malformation, may be present. Migraine like HA that switch sides overwhelmingly suggest the diagnosis of migraine. Vasomotor changes can occur in migraines, which can be responsible for patient's head feeling warm and hands icy cold. Also, syncope can occur in migraine patients as a result of vasomotor changes, pain, or medication effect. Medications used to abort migraine are listed in [Table 3-2](#).

### *Common Migraine (Migraine without Aura)*

In migraine without aura, a distinct aura is not present, but prodromal symptoms such as an uneasy feeling, depression, or malaise can be present for hours or days before the migraine. Onset of pain can be somewhat more gradual than in classic migraine. Pain is severe, usually unilateral at first, but often spreads. Nausea, vomiting, and photophobia are less common. Pain is intermittently pounding and lasts longer than does classic migraine. This type of migraine is sometimes difficult to differentiate from tension HA, which starts to throb as it increases in intensity. There is, in fact, a significant overlap of tension HA and common migraine. Some patients with common migraines can, over the years, develop chronic daily HA that has features of daily tension-type HA with escalation to migraine-like pain intensity and character on frequent occasions. Such patients tend to overuse analgesics, have depression, have significant life stresses, suffer sleep disturbance, have a greater than expected problem with substance abuse, and often have a family history of a similar disorder. These are the chronic daily HA.

### *Cluster Headache*

Cluster is an uncommon head pain syndrome. These are classified as trigeminal autonomic cephalalgias; these have cyclical pattern of cranial autonomic phenomena (ptosis, miosis, sweating, lacrimation, conjunctival injection). Cluster is more common in males; usually begin between the ages of 20 and 40, but often appear initially in middle age. The head pain is characterized by sudden, very severe, steady, boring, or throbbing pain. They often occur at night and awaken the patient from sleep at same time each night. The pain is exclusively unilateral and usually is located in or around the eye. The pain lasts between 15 and 90 minutes and is often associated with autonomic phenomena. The scalp can also feel sore or painful. The pain does not shift sides with attacks. The pain occurs in clusters over period of days to months and then disappears for 1 to several years. The pain can be triggered by alcohol consumption or smoking. As contrasted with migraine episode in which patient wants to remain still and undisturbed in a quiet room, cluster patient can not sit still and paces and may even bang their head against the wall. The pathophysiology of cluster involves activation of ophthalmic branch of trigeminal nerve, cavernous sinus, parasympathetic, or sympathetic nerves.

There is a variant of cluster in which occurrence is seen equally in males than females, as contrasted to female predominance of migraine and cluster in men. There can be bilateral pain that is more frontotemporal than periorbital; pain is not as severe; autonomic response (e.g., lacrimation) is infrequent; and background HA and jabbing head pain are experienced. Most cluster attacks are episodic with periods of remission; however, some patient's experience attacks without remission (chronic cluster). Another variant is the occurrence of short-duration attacks many times each day (chronic paroxysmal hemicrania).

Treatment of acute cluster includes: (1) 100% nasal oxygen at flow rate of 8 to 10 liters per minute via nasal cannula, (2) sumatriptan by nasal or subcutaneous route, (3) dihydroergotamine by intramuscular or intravenous route. For prophylaxis of cluster, these medications have been utilized - prednisone, methysergide, calcium channel blockers, lithium, divalproex.

### *Migraine Equivalents*

In some persons episodic aura or prodromata occur without accompanying head pain. Autonomic disturbances such as tachycardia, nausea, or visceral pain can be experienced as well as neurologic signs. Classic visual aura with spreading scotoma is occasionally experienced

initially in middle or old age without HA. Differential diagnosis from TIA or focal epilepsy can be quite difficult. Most of these patients do not have any demonstrable cerebral vascular disease to explain them. The reason for the appearance of the migraine equivalents at this age is unclear. These isolated migraine equivalents are at times reported by migraine patients but are also experienced by people who have never had an associated HA

### *Laboratory Evaluation*

If the patient has had only a few HA and they are all on one side or if the patient has either abnormalities on the neurologic examination or suspicious factors in the history, full evaluation is advisable. In addition, if HA precedes neurologic aura or if patient is seen during first ever attack, neuroimaging studies are warranted. In patients with typical history of chronic recurring HA, extensive evaluation seems unnecessary. EEG will often show focal abnormalities that are not significant clinically and only serve to worry both patient and physician about possibility of epilepsy. Routine CT or MRI seems unjustified in most HA patients.

### *Management of Migraine*

Treatment of migraine is abortive or prophylactic. The choice of medication depends on character of migraine and frequency. In patients with occasional migraine with a distinct aura or prodrome, abortive treatment with ergots or triptans should be used at onset of symptoms (Box 3-5). They are quite safe and can be used even in complicated migraines. If nausea prevents oral intake, suppositories can be used. Ergot must be started early in course of attack because it is rarely effective once severe migraine has started. Triptans have become more popular as they can be used effectively at any stage. If patient has no warning of HA onset or awakens patient at night, ergot derivatives are usually ineffective, and only effective treatment is triptans. After obtaining HA relief, migraine can rebound, and second treatment with triptans can be given. Place the patient in dark and quiet room. Sedation can relieve migraine and this can be the mechanism of opioid effect in migraine. Patients can become dehydrated due to vomiting and rehydration with adequate fluid load is important. Gastric paresis can cause nausea and vomiting and make oral medication ineffective. Utilize metoclopramide, promethazine, prochlorperazine, domperidone, droperidol, or dansetron to combat vomiting. Simple (aspirin, acetaminophen) and combination analgesics and non-steroidal anti-inflammatory medications can be effective in mild or moderate migraine and are certainly less expensive alternative to triptans. Remember response to triptans does not establish diagnosis of migraine!

In patients with daily or severe weekly migraine, daily prophylactic medication seems justified. The medications in [Table 3-3](#) have been shown to be effective. The length of therapy depends on patient and type of HA. In classic or common migraine, it is suggested patients remain on medication for 6 to 12 months; taper medication until HA recur. Breakthroughs from prophylactic therapy do occur and are treated as regular migraine.

## **Headaches Associated with Hypertension**

A significant number of hypertensive patients complain of head pain. Interestingly, this is more common in patients who are aware they have hypertension. This suggests that knowing about hypertension and possible sequelae, such as brain hemorrhage, bring on HA. Hypertensive HA is not particularly distinctive. It is often, although not always, throbbing. It is frequently localized

in occipital or frontal region and can lateralize to one side. It is present in mornings in approximately one-half of patients, particularly those with throbbing pain and scattered throughout the day in others. In some cases, pattern is migrainous, whereas in others it is not. Treatment of hypertension helps; thus it seems the increased arterial pressure in vessels can be related to HA in some patients. In hypertensive encephalopathy, HA is more persistent and is direct result of cerebral edema and increased intracranial pressure.

## **Sinus Headache**

Inflammation of mucosa and ostia of perinasal sinuses can give rise to localized head pain and facial pain. When sinus is blocked, intense pain can be caused by vacuum created by absorption of trapped air. Pain is steady, dull, and aching, and localized over area of affected sinus. [Table 3-4](#) shows location of pain based on sinus infected. The pain is aggravated by coughing, sneezing, bending, or straining. Tearing is often seen, and there is usually evidence of nasal inflammation. The bone over the sinus is often tender. In acute sinusitis, pain is quite severe; whereas in chronic sinusitis, pain is less intense and less associated with nasal symptoms. Sinus HA is often misdiagnosed as tension HA, especially if pain occurs in frontal region. Allergic rhinitis can occasionally be associated with sinus HA, but tension HA is much more common in allergy sufferers than sinusitis.

The diagnosis of sinusitis and sinus HA is usually made on history, examination of nose, and sinus imaging studies. Treatment with decongestants, antibiotics, and analgesics can be effective. The sphenoid sinus is located within sphenoid bone and is located within middle cranial fossa. It is contiguous to sella-pituitary gland and cavernous sinus. Sphenoid sinusitis causes HA which awakens patient from sleep, is worsened by activity, e.g., walking, bending, coughing. Pain can be at vertex or behind the eyes. Severe, new-onset HA that interferes with sleep and does not respond to analgesics or opioid should raise the possibility of sphenoid sinusitis. Examination shows no sinus tenderness. Diagnosis is established by CT or MRI; they show soft tissue in usually aerated sinus; however, physician must be certain CT/MRI adequately images the sphenoid sinus. Since sphenoid is intracranial sinus, failure to detect sphenoid sinusitis can lead to meningitis or focal suppurative intracranial infection. Treatment includes decongestants and high-dose intravenous antibiotics for 14 days.

## **Chronic Pain from Cervical Spine Disease**

Degeneration of intervertebral disks in cervical region can be source of occipital HA. The syndrome is principally seen in laboring middle-aged males and after whiplash types of injuries. Pain can be caused by local degenerative changes or pressure on cervical nerve roots. Pain is steady; exacerbated by neck motion, and can spread into shoulder or arms, particularly when root impingement is present. Examination usually reveals only minor spasm of upper trapezius with reduced range of motion and loss of cervical lordosis. Most occipital HA is tension type pain, triggered by cervical abnormality, but careful neurological examination for signs of nerve root (radiculopathy) and spinal cord (myelopathy) compression should be carried out. The majority of patients over age of 40 have MRI evidence of disc desiccation and other signs of cervical spondylosis. It is important not to attribute occipital HA to cervical spondylosis, simply because of radiographic abnormalities. Treatment, in absence of neurological deficit, is conservative: heat to area, simple analgesics, occasional muscle relaxants, cervical traction, and intermittent use of a soft collar are most helpful.

## Posttraumatic Headaches

Posttraumatic HA can be chronic and recurrent. These HA usually occur immediately after head injury and seen after mild and severe injuries. Three patterns of HA have been described: (1) constant, band-like, diffuse HA; (2) superficial tenderness at the local injury site; (3) episodic, throbbing, unilateral HA. Head pain can be intermittent or constant, worsened by head movement, and associated with dizziness and difficulty concentrating. Migraine with or without aura can be triggered by accident or may begin at some time related to trauma. It is essential to perform careful neurological examination to exclude structural trauma-related lesion. If this examination is normal, CT/MRI is not usually necessary. The majority of posttraumatic HA resolve within several weeks or months after head injury; however, some are prolonged and intractable. Treatment depends on HA pattern and if symptoms suggest predominantly migraine or muscle contraction component. When HA begins to increase in severity 2 to 4 weeks after trauma, obtain CT or MRI to exclude chronic subdural hematoma. Since the head bone is connected to neck bone, cervical-occipital and shoulder pain can be associated with posttraumatic HA.

## Headache Associated with Brain Tumor and Other Lesions Causing Increased Intracranial Pressure

### *Mass Lesions*

The brain does not have pain fibers; pain from mass lesions comes from distortions and traction on pain sensitive structures such as dura, dural sinuses, and large vessels at brain base. This type of HA has several important clinical features. There is seldom a long history; patient usually has no prior HA or there is prior head pain history. There are new characteristics and pattern of the HA. Pain is a dull, steady, aching feeling; usually much less intense than migraine. The pain is usually worse in morning and is acutely exacerbated by rapid assumption of upright position. Coughing, sneezing, and straining can also cause acute worsening of pain. Pain is often localized to area of the lesion. In subtentorial tumors, HA is very common and primarily in occipital region. Supratentorial tumors in temporal lobe are most likely to cause pain.

History of HA developing gradually over several months is a possible indication of mass lesion. If history is associated with other neurological symptoms (seizure, decreased level of awareness, vomiting, language impairment, visual disturbance such as bumping into objects due to bitemporal defect of chiasmal-pituitary lesion, double vision on far gaze due to lateral rectus palsy secondary to intracranial hypertension), the likelihood of tumor increases. It is unlikely for HA with an entirely normal neurological examination to be only symptom of brain tumor. If mass is suspected, it is imperative to perform neurodiagnostic evaluation. *Spinal puncture should never be performed when intracranial mass lesion is suspected, prior to performing CT or MRI.*

### *Hydrocephalus*

Hydrocephalus (expansion of cerebral ventricles because of obstruction of flow) results in dull frontal and occipital pain. This can be very severe if there is acute obstruction that can occur in posterior fossa and midline, e.g., ventricular tumors. Abnormalities of CSF production, circulation and reabsorption lead to intracranial hypertension. CT/MRI detect the presence of hydrocephalus, location of obstruction as well as delineating presence of a mass causing obstruction. Shunting of cerebrospinal fluid (CSF) shunting is mainstay of treatment.

### *Idiopathic Intracranial Hypertension (Pseudotumor Cerebri)*

Idiopathic intracranial hypertension is a condition that occurs primarily in young, overweight females. Symptoms are primarily HA; intermittent blurred vision (obscuration) and diplopia can accompany head pain. HA is an intermittent, generalized pain, worse in the morning and exacerbated by coughing or sneezing. On examination, the patient is alert, has papilledema, and occasionally show lateral rectus weakness, reduced visual acuity, and enlarged blind spots. Evaluation should include CT/MRI to rule out mass or hydrocephalus, then lumbar puncture (LP). CSF pressure is elevated but there must be normal CSF content. Following LP, pressure should be reduced to 50% of elevated opening pressure. Magnetic resonance venogram (MRV) must be done to exclude venous sinus obstruction. If this is etiology, anticoagulation is appropriate treatment. If cause is known (e.g., anemia), correction of basic problem will reverse condition. When no cause is known, often initial LP decreases pressure. Repeated therapeutic taps (every other day) have been used although without theoretic basis to reduce intracranial pressure. Corticosteroids (prednisone, 80 mg daily) and (acetazolamide [Diamox], 1 to 3 g daily) have also been used. In rare cases in which these measures have failed and vision is in jeopardy, optic nerve sheath decompression or lumbar peritoneal shunt may be necessary. Most cases resolve without permanent visual loss, but some cases (less than 10%) become recurrent or chronic and present severe management problem.

### *Intracranial Hypotension*

Intracranial hypotension is divided into two groups: (1) spontaneous, with no evidence of CSF leak or systemic illness, (2) symptomatic and associated with CSF leakage. HA is most common symptom. It is dull or throbbing, worsened by standing, relieved by recumbent position and exacerbated by coughing or sneezing. CT/MRI shows diffuse meningeal enhancement; CSF shows low pressure of 0 to 60 mm H<sub>2</sub>O and is otherwise normal. The most common cause is prior LP or following surgery or trauma to result in dural tear. HA is believed due to low CSF volume and pressure due to fluid leakage. Radioisotope cisternography can identify CSF leak. Treatment includes bed rest, volume expansion, abdominal binder, caffeine, blood patch, and lastly, surgical leak repair. Presence of orthostatic HA relieved by recumbency is characteristic of intracranial hypotension.

### *Subarachnoid Hemorrhage*

When blood suddenly invades subarachnoid space, it stretches, distorts and irritates the pain-sensitive vessels and meninges. This produces acute and severe head pain. The patient can usually report exact time of pain onset and will frequently exclaim it was "worst headache I have ever had." Such history should immediately alert physician to possibility of serious intracranial disaster, very probably subarachnoid hemorrhage. HA usually strikes without warning. It is severe, steady, and generalized. There can be an immediate alteration in level of consciousness, and some patients become comatose within seconds. The alert patient usually complains of neck stiffness, photophobia, dizziness, and nausea. With more serious hemorrhages, focal neurological signs can be present. On examination, nuchal rigidity is usually present. Subhyaloid hemorrhages in retina, oculomotor nerve paralysis, and other neurological signs can also be present. Evaluation should begin with CT; this can show hematoma formation as well as subarachnoid blood. If no clot or subarachnoid blood is demonstrated, LP must be performed. If blood is

present, four-vessel arteriogram should be performed and specific treatment dictated by these findings.

## **Meningitis**

This is frequently heralded by HA. Pain originates in inflamed meninges. HA is usually occipital but can be present in other locations. Stiff neck and fever are almost always present and suggest meningitis. Other neurological signs can be present, including altered level of consciousness, cranial nerve palsies, and major motor abnormalities. Diagnosis is made by LP. One note of caution concerning occipital HA and nuchal rigidity: these disorders can be caused by herniation of cerebellar tonsils in posterior fossa masses. A careful search should be made for lower cranial nerve abnormalities and nystagmus before attempting LP. If patient is afebrile with questionable diagnosis of meningitis, CT/MRI should be performed prior to LP.

## **Temporal Arteritis**

Temporal arteritis is a type of diffuse granulomatous arteritis; the majority of patients experience HA. The disease most commonly affects ophthalmic artery and branches of external carotid artery (superficial temporal, occipital, facial, maxillary). The condition is rare in persons under age 60. HA is most common feature. Classically, pain is steady, localized, and strong, with intermittent sharp, stabbing pains. Initial clinical manifestations include fever, myalgia, jaw claudication, arthralgias (polymyalgia rheumatica). This is not always the case and diagnosis is often delayed. Unilateral or bilateral rapid (12 to 48 hours) *irreversible* visual loss can occur. Jaw claudication (pain with chewing) from involvement of maxillary artery is also common. A variety of signs of generalized illness can be seen (e.g., malaise, fever, depression, anorexia), and condition is often associated with polymyalgia rheumatica (aching pains in the joints without objective signs of arthritis). On examination, temporal artery can be tender or at times pulseless and hard secondary to thrombosis. The scalp is often tender. In patients with impaired visual acuity, funduscopy shows narrowing of retinal arteries consistent with anterior ischemic optic neuropathy. The most specific laboratory test is erythrocyte sedimentation rate, which is elevated in more than 90%, often to very high levels. Temporal artery biopsy can also be performed to establish diagnosis pathologically.

Treatment should be started immediately to preserve vision; prednisone, 60 to 80 mg, given daily for 4 to 6 weeks and gradually tapered to 5 to 10 mg. Initiate corticosteroid therapy early before visual loss occurs because treatment can prevent, but not reverse, visual loss. Treatment at low doses should be continued for at least 1 year. Periodic erythrocyte sedimentation rates will document effectiveness.

## ***Headache Associated with Vascular Disease***

Remember, migraine is due to primary neuronal instability state and is not primary vascular disorder; however, migraine can be risk factor for ischemic stroke in certain patients. Migraine aura can mimic transient ischemic attack (TIA). Migraine usually causes “positive” symptoms and TIA causes “negative” symptoms. In stroke of hemorrhagic type (ICH, SAH), HA may be prominent feature, but there are usually other accompanying neurological deficits. Migraine can cause stroke – migraine infarction. In this disorder, patient with aura develops fixed permanent neurological deficit; CT or MRI shows ischemic-infarction pattern, usually in posterior cerebral artery territory. Also, migraine can be one of many risk factors for stroke. Migraine can

precipitate stroke in women using oral contraceptive medication; this is most common in hypertensives, smokers, older women, and have those having mitral valve prolapse. Stroke can occur as consequence of migraine due to migraine-induced oligemia (reduced cerebral blood perfusion) and platelet dysfunction as well as occurring in comorbid conditions including antiphospholipid antibody syndrome, hypertension, mitral valve prolapse, cerebral autosomal dominant arteriopathy with subcortical infarcts, and leukoencephalopathy. Some patients with structural CNS disease such as arteriovenous malformation can mimic “migraine with aura”; however, HA can precede the aura and neurological abnormalities can persist into HA phase .

Sudden onset of severe HA suggests SAH. Severe unilateral HA can occur following carotid endarterectomy (CEA). This can be trigeminal neuralgia-like pain with autonomic features or HA due to hyperperfusion syndrome in post-CEA patients. HA due to intracranial hypertension can occur with cerebral venous thrombosis and diagnosis is established by MRV. In patients who have had head and neck trauma, carotid or vertebral dissection must be considered if HA and neck pain persist. Diagnosis is established by vascular imaging procedures. Angitis of CNS can be primary or complication of systemic disorder. Diagnosis is established by vascular imaging procedure. Benign angiopathy of CNS causes acute onset of HA; angiography shows multiple reversible segmental areas of stenosis. It occurs in young healthy patients and most commonly in pregnant patients. This resolves spontaneously, and nimodipine (calcium channel blocker) has been utilized in some patients.

### *Pain from Structures in the Head*

When taking history and examining HA patient, it must be remembered that many structures in the head can be responsible for pain. Ocular pathology (particularly glaucoma), severe refraction errors, and masses or inflammation either within or behind orbit are also sources of pain. Unless there is marked refractive error or eye appears clinically involved (e.g., red or swollen), eye is not source of HA. Abscessed teeth, middle ear infections, and mastoid infections can also cause HA.

A final fairly common source of HA is temporomandibular joint (TMJ) dysfunction. When there is malocclusion of teeth, unbalanced force is transmitted to this point. This results in pain radiating into temple, occiput, face, teeth, or neck. The joint is often painful to palpation; this sign will confirm the diagnosis. This condition frequently occurs after dental work. When diagnosis is made, referral to a dentist should be made. Treatment with indomethacin, 25 mg three times a day is often useful in treatment.

### **External and Cough Headaches**

Benign sudden head pain of brief duration (lasting less than one minute) can be precipitated by cough, sneezing, bending, weight lifting, straining at bowel movement. HA can also be precipitated by sexual activity. HA precipitated by these activities is brief and short-lived and resolves with termination of activity. This is a benign disorder. If HA persists after activity is stopped, consider subarachnoid hemorrhage due to ruptured aneurysm, Chiari formation or posterior fossa mass lesion. Treatment of benign exertional HA with indomethacin has been effective.

## Hypnic Headaches

This is bilateral throbbing HA without autonomic features. This awakens patient from sleep and duration is 15 to 30 minutes and can occur multiple times per night; they occur at the same time each night. They usually occur in elderly patients, often during rapid eye movement (REM) sleep. Because of nocturnal pattern, these can be confused with cluster. Also, since they occur mostly in elderly patients, consider temporal arteritis. Hypnic HA responds to Lithium, but this should be used carefully in elderly patients due to potential renal toxicity.

## Headache Associated with Other Systemic Diseases

HA is often an accompanying symptom of systemic disease such as infection, endocrine disorders, allergic, and immunologic disorders and can be prominent secondary symptom in patients with chronic illness.

## Face Pains

### *Trigeminal Neuralgia*

There are several syndromes of severe face pain. The most common is tic douloureux, or trigeminal neuralgia. Age at onset is usually 50, and disorder is more common in females. Cardinal symptom is lightning-like (lancinating), excruciating, unilateral face pain that radiates down one or combination of fifth (trigeminal) nerve branches. Syndrome has also been described in ninth (glossopharyngeal) and tenth (vagus) nerves. Some patients may describe similar type of pain, occurring in back of throat and can radiate to the ear. These are stabbing or electric-like pains that can be precipitated by swallowing or yawning. This pain is characteristic of glossopharyngeal neuralgia. The pains frequently come in bursts over a few seconds to minutes with soreness present with resolution of pain. Pain can be triggered by touching face, eating or drinking hot or cold substances, talking, jarring the head in any way. Pain is frequently experienced in distribution of mandibular and maxillary divisions 36%; mandibular in 19%; all divisions 15%; maxillary 14%; maxillary and ophthalmic 11%; and ophthalmic 3%. Initially these pains occur in bouts over a few days and then remit for months; however, bouts lengthen and remission shortens.

On examination, there can be little or no abnormality in sensory examination over affected areas. The presence of significant facial hypalgesia or other cranial nerve abnormality (e.g., loss of corneal reflex or weakness) usually indicates structural lesion either in the nerve, such as a neurofibroma, or in posterior fossa, such as a multiple sclerosis plaque, cerebellar tumor, acoustic neuroma. In the absence of any positive neurological findings, the only advisable laboratory study would be dental roentgenograms to rule out apical abscess or other similar conditions. The pathophysiology of pain is not known; however, the two main theories are pressure from a blood vessel lying on nerve as it exits from brain stem and chronic infection with herpes simplex virus.

Treatment can be medical or surgical. It is best to use conservative therapy with medication initially. The medications prevent pain by stabilizing nerve membranes. Most effective are carbamazepine (Tegretol, 200 mg bid; increasing by 200 mg daily every 3 to 4 days until pain stops or patient is taking 1600 mg daily), phenytoin (Dilantin, 300 to 400 mg daily), and baclofen (Lioresal, 20 to 60 mg daily). If medication fails, alcohol injection into nerve or percutaneous radiofrequency trigeminal gangliolysis is effective but can produce anesthesia and

residual hyperpathic pain. Retromastoid craniectomy with dissection of vessels off nerve is also successful procedure.

### *Atypical Face Pain*

The second important face pain syndrome is atypical facial pain. This condition is seen in young as well as older adults and characterized by rather diffuse, nondescript aching pain. It is often quite widespread and can involve half or occasionally the entire head and spread to neck. Pain is quite persistent and can last for years.

Many pathologic conditions, e.g., sinus tumor, tooth abscess, and TMJ dysfunction can be present with face pain and must be ruled out. If no specific lesion is found, diagnosis of idiopathic or atypical facial pain is made. Cause is not known, but psychotropic medications (particularly antidepressants or minor tranquilizers) have been successful treatment.

## **SUMMARY**

There is a spectrum of head pain from classic migraine to tension HA at the other. The history of the clinical characteristics of head pain will place HA on appropriate location in that spectrum. Specific medications can then be prescribed based on the character of HA and its frequency.

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## Tables

Table 3-1 Headache Profiles

<b>Condition</b>	<b>Character of Pain</b>
Tension headache (circumferential)	Tightness; gradual buildup (occipital, frontal or temporal, or
Classic migraine (migraine with aura)	Visual aura then unilateral throbbing pain with nausea and vomiting
Common migraine (migraine without aura)	No aura; unilateral headache at first but then generalized but then generalized throbbing; can have nausea
Cluster headache	Strong, periorbital, boring, steady or throbbing pain; frequently awakens patient; rapidly reaches maximal intensity
Subarachnoid hemorrhage	Severe generalized headache of sudden onset
Tumor	Dull morning pain that is increased by coughing, sneezing, or Exertion
Idiopathic intracranial hypertension	Dull, steady pain frequently with visual complaints including diplopia and visual obscurations
Systemic arterial hypertension	Patient awakens with dull or throbbing pain which diminishes during course of the day and may be associated with dizziness
Sinus headache	Steady localized pain over sinus and headache is worse when patient is recumbent and decreases when patient gets out of bed
Temporal arteritis	Patients over 60; unilateral orbital pain; may be associated with polymyalgia and jaw claudication
Trigeminal neuralgia	Lancinating face pain
Atypical face pain	Dull, steady, diffuse face pain

**Table 3-1 Headache Profiles Continued**

<b>Duration</b>	<b>Recurrence</b>	<b>Treatment</b>
1 hour to many days	Tends to fluctuate with	Simple analgesics, amitriptyline, biofeedback, and psychological support
30 minutes to several hours	More frequent during or directly after times of stress	Ergotamine preparations, $\beta$ -blockers, amitriptyline, Bellergal (ergotamine, phenobarbital), calcium channel blockers, methysergide, analgesics and most recently, triptans for abortive therapy
Hours to days	Similar to classic migraine	Same as classic migraine
15-90 minutes	Occurs in clusters over a month or two; varying intervals between clusters	Prednisone and ergotamine, calcium channel blockers during cluster times, subcutaneous or nasal sumatriptan or high flow nasal oxygen (8 to 10 liters per minute)
Days	No	Evaluation and often surgical Treatment
Weeks to months	No	Surgery
Days to weeks corticosteroids, acetazolamide, and weight reduction	In some cases	Repeated spinal puncture,
Several hours, lessens during morning encephalopathy)	Related to arterial pressure	Antihypertensive medication (see also hypertensive
Days to weeks	Will recur	Antibiotics and antihistamines
Days to weeks	Can recur	Corticosteroids
Seconds	Recur throughout life	Carbamazepine, baclofen phenytoin, or surgery
Hours	Recur throughout life	Amitriptyline, sedative, anti-

anxiety or antidepressant medication

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Table 3-2

### Medication to Abort Migraine Episode

- Acetaminophen
  - Aspirin
  - Butalbital, caffeine and ASA/acetaminophen
  - Caffeine/APAP/ASA (Excedrin Migraine)
  - Isometheptene
  - Non-steroidal anti-inflammatory medications
  - Opioids
  - Ergots (tablet, suppository)
    - Cafelgot (Tablets, Suppository)
    - Migraine (Tablets, Suppository)
  - Dihydroergotamine (intravenous, intramuscular, nasal)
  - Triptans - tablets most
    - Almotriptan (Axert)
    - Eletriptan (Relpax)
    - Frovatriptan (Frova)
    - Naratriptan (Amerge)
    - Rizatriptan (Maxalt)
    - Sumatriptan (subcutaneous, nasal) (Imitrex)
    - Zolmitriptan (nasal) (Zomig)
-

<b>Table 3-3 Prophylactic Medications</b>		
<b>Trade Name (Generic Name)</b>	<b>Dose</b>	<b>Side Effects and Precautions</b>
<b>Migraine</b>		
<b>Beta Blockers</b>		
Inderal (propranolol) or Corgard (nadolol)	Start with Inderal 60 mg LA or Corgard 20 mg; increase to 240 mg maximum	Because of ( $\beta$ -blocking properties, it is not safe in patients with asthma or heart failure. Can slow heart rate.
<b>Tricyclic Antidepressants</b>		
Elavil (amitriptyline), Sinequan	10 to 25 mg at bedtime; can increase as needed	Nervousness, drowsiness, dry mouth, and other anticholinergic side effects.
<b>Calcium Channel Blockers</b>		
Verapamil	180 to 240 mg sustained	Can cause cardiovascular effects such as hypotension and arrhythmias, including heart block. Do not use with $\beta$ -blockers.
Sansert (methysergide)	2 mg twice daily up to four times a day	Retroperitoneal, pulmonary, and cardiac valve fibrosis. This can almost always be prevented by not giving the drug longer than 6 months without a month drug holiday. Never give more than 8 mg daily.
<b>Nonsteroidal analgesics</b>		
Bellergal-S (phenobarbital,	Two to four tablets daily	May cause stomach problems. Anaprox (naproxen) is particularly useful in catamenial (menstrual) migraine when used before and throughout the menstrual period. Drowsiness plus other side effects of ergot and

ergotamine, belladonna)		belladonna.
Periactin (cyproheptadine)	4 mg, two to four tablets daily	Drowsiness and antihistamine effects.

***Anticonvulsants***

Tegretol	Must titrate
Depakote	Must titrate
Topiramate	
Neurontin	

**Cluster headache**

Calcium channel blocker	As above	
Prednisone plus ergotamine	10 mg four times a day for 7 days, then taper off to four tablets daily for 7 days, then taper off	Can reinstitute full treatment regimen if headaches break through while tapering medications.
Sansert	As above	
Periactin	As above	Watch blood level
Lithium (chronic cluster)	300mg tid	Confusion, tremor, polyuria, nausea, diarrhea, weakness

**Cluster variant**

Indomethacin	25 mg three times a day	Monitor complete blood count.
Aspirin	Eight 5-grain tablets daily	Gastric distress

Table 3-4

<i>Sinus infected</i>	<i>Location of pain</i>
Frontal	Forehead above eyebrows
Ethmoidal	Bridge of nose
Maxillary	Below eye and in upper teeth
Sphenoidal	Behind eye and at vertex