Headaches:

An optometrist must be prepared for many different presentations.

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Abstract: Many optometric patients will complain of headaches. This paper focuses on the three most common headaches: migraine, tension, and cluster. Migraine and cluster can both have visual manifestations. The optometrist must be able to recognize these headaches, and know the warning signs of headaches requiring immediate medical attention.

Headaches are probably the most common complaint known to humans. Virtually anybody, young or old can experience headaches. Though reports conflict, one study found that 90 percent of the population are prone to headaches, with males and females about equally affected.[1] The effects of headaches are staggering. The U.S. loses 157 million workdays a year and spends more than $2 billion a year on over-the-counter painkillers for treatment.[2] A recent study says that chronic headache sufferers are more disabled, their lives more upended, than people with arthritis, diabetes, or back problems.[3] Many optometric patients present with headache as a chief or secondary complaint because headaches are often associated with pain around the eyes. Some headaches even produce visual phenomenon such as scintilating scotoma associated with migraine headaches or the intense lacrimation of cluster headaches. While it is true that headaches comprise part of the symptomatology of health problems within the diagnostic scope of optometry (e.g. refractive errors, oculomotor imbalances, glaucoma), it is a fact that most headaches are caused by nonvisual and nonocular factors.[4] It is up to the optometrist to have a thorough background knowledge of headaches in order to recognize those that warrant optometric treatment, those which deserve a reassuring hand, and those requiring immediate medical attention.

The key to dealing with headaches is taking a detailed history of the nature of the headache. The history will be the primary tool through which the most useful diagnostic and therapeutic information will be derived. It is the purpose of the history to establish a pattern profile of the headache, so it may be classified according to one of the 13 types of headache that has been identified by the International Headache Society. (See table 1). The following points should be emphasized during the case history when exploring headache related complaints: duration, frequency, time of onset, character of pain, location, associated signs and symptoms, and any known precipitating factors.

Table 1. New International Headache Society classification of headache (abbreviated)

1 **Migraine**
- Migraine without aura
- Migraine with aura
- Retinal migraine
- Ophthalmoplegic migraine
- Basilar migraine

2 **Tension type headache**
- Episodic tension type headache
- Chronic tension type headache

3 **Cluster headache**

4 **Miscellaneous not associated with structural lesion**
- Cold stimulus headache
- Benign cough headache
- Headache associated with sexual activity

5 **Headache associated with head trauma**

6 **Headache associated with vascular disorders**
- Acute ischemic cerebrovascular disorder
- Subarachnoid hemorrhage

7 **Headache associated with nonvascular intracranial disorder**
- High CSF pressure
- Intracranial neoplasm

8 **Headache associated with substances or their withdrawal**

9 **Headache associated with noncephalic infecton**
- Viral infection
- Bacterial infection

10 **Headache associated with metabolic disorder**
- Hypoxia
- Hypoglycemia

11 **Headache or facial pain associated with disorder of cranium, neck, eyes, ears, nose sinuses, teeth, mouth, or other facial or cranial structures.**
- Temporomandibular joint disease

12 **Cranial neuralgias, nerve trunk pain, and deafferentation pain**
- Trigeminal neuralgia
- Glossopharyngeal neuralgia

13 **Headache not classifiable**

Migraine.

A fully developed migraine attack can be divided into four (sometimes overlapping) phases, each of which may have ocular manifestations: (1) premonitory symptoms (hours to days before a migraine attack), which may include heightened perceptions of taste and smell, arousal, exhilaration, sleep disorders, hypoactivity, abdominal discomfort, depression, craving for special foods, dulled mentation, yawning, and syncope; (2) aura (may be absent), a complex of focal neurologic symptoms immediately preceding and sometimes accompanying the headache; (3) headache; and (4) postheadache alteration of mood or mentation, including drowsiness, lethargy, duresis, or epiphora.[5]

Migraine is characterized by severe, unilateral headaches which are usually associated with nausea, vomiting and photophobia. This disorder often begins in childhood, adolescence, or the early twenties. The frequency of this recurring headache may range from one or more a month to once a year. It may occur at any time of the day, sometimes awakening the patient from sleep, but most often it is present when the patient awakens in the morning. The typical migraine lasts 2 to 3 hours and rarely more than 12 hours. The pain is usually frontal and temporal, but it may radiate into the face or neck and shoulders posteriorly. The pain is initially dull and crescendos into a severe, throbbing, pulsatile pain. This headache affects women three times as often as men. There is a tendency for the headaches to occur during the period of premenstrual tension and fluid retention. The immediate family history is positive for migraine in over 60 percent of cases.[6] Migraine may occur with a sudden increase or decrease in stress level. For instance, a person entertaining an unwelcome guest, or a hardworking executive who begins an overdue vacation may experience a migraine. Many other items are believed to be "triggers" for migraine including birth control pills and foods containing nitrites. (See Table 2).

Table 2. POSSIBLE TRIGGERS OF MIGRAINE HEADACHES

Beverages: Alcohol, Caffeine
Dairy: Butter, Sour Cream, Buttermilk, Chocolate Milk, Most Cheeses
Soups: Canned (MSG)
Breads and Cereals: Yeast Breads, Cheese Breads, Sour Dough Bread, Doughnuts, Granola Breads, Granola Cereals
Sweets: Chocolate
Fruits and Nuts: Avocado, Raisins, Peanuts, Sunflower Seeds, Pumpkin Seeds
Miscellaneous: Pizza, Soy Sauce, MSG Substances, Brewer's Yeast, Pickled Foods

A satisfactory theory of the pathophysiology of migraine has eluded clinical investigators. Certain facts appear indisputable; the symptoms of migraine are associated with changes in cerebral blood flow, presumably secondary to changes in vessel caliber. The premonitory symptoms and aura are accompanied by cerebral arteriolar constriction and decreased blood flow

in the cerebral cortex, and the headache that follows is related to increased pulsation and vasodilation of extracranial vessels. In addition there are associated changes in circulating vasoactive agents such as serotonin, norepinephrine, prostaglandins, bradykinin and histamine. The exact role of these vasoactive substances remains undetermined.

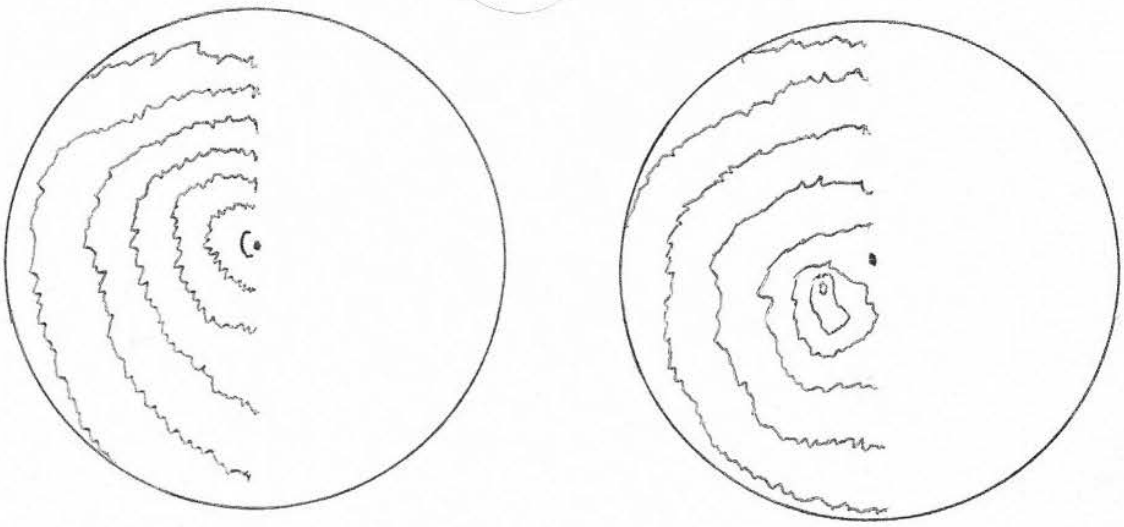
Migraines used to be called either *classic* or *common* depending on the presence or absence of aura. Classic migraine is now called *migraine with aura* and common migraine is now called *migraine without aura*. Aside from the aura, the differences between them are small.

The aura is a usually a 20-30 minute interval preceeding the headache. The aura is marked by neurological dysfunction that is a red flag that the headache wil be coming soon. Some aura victims experience unilateral parathesia or unilateral numbness, but for most the primary feature is the visual aura. The visual aura may include photopsia (flashing lights and colors), teichopsia (bright shimmering or wavy lines), fortification spectrum (zigzag pattern), scotoma (blind spot), hemianopsia (partial visual field loss), and metamorphopsia (illusions of distorted size or shape). Visual changes are typically bilateral and present in homonymous portions of the field. While many variations occur, the following description by Richards[7] summarizes the most typical of visual phenomenon (Fig. 1]:

> The visual disturbance usually precedes the headache... [it] begins near the center of the visual field as a small gray area with indefinite boundaries. If this area first appears during reading, as it often does, then the migraine is first noticed when words are lost in a region of "shaded darkness." During the next few minutes the gray area slowly expands into a horseshoe with bright zigzag lines appearing at the expanding outer edge. These lines are small at first and grow as the blind area expands and moves outward toward the periphery of the visual field.

One important aspect of the visual disturbance just described is that it expands slowly, over a period of 10 to 20 minutes. The episode is complete typically in 20 to 30 minutes. Symptoms slowly fade and vision returns to normal. When visual acuity defects or visual field defects persist after the headache passes, a cerebrovascular accident or tumor should be suspected.

Fig 1. Successive arcs expand across half of visual field, as shown in two diagrams. The spectra may take 10 to 20 minutes to expand from a fuzzy gray area near the fixation point (dot) to the outer linit of the visual field.



The diagnosis of migraine is made primarily by history; physical findings are absent, and laboratory evaluation is not helpful. When the attacks are atypical, particularly when neurological disability is severe or prolonged, CT scans may be required to rule out structural lesions of the brain or its vasculature. However, such instances are rare.

There are three rare but important subcategories of migraine. These include retinal migraine, ophthalmoplegic migraine, and basilar migraine.

Retinal Migraine

Retinal migraine is a rare type of migraine that presents with only the visual symptoms typical of migraine. The headache is absent. The sufferer of this type of migraine experiences an aura which can also include monocular blackouts in severe cases. The vision loss usually lasts 30 minutes to an hour though in some cases it has persisted for more than seven hours followed by complete recovery of vision.[8] The pathogenesis of such episodes is not well understood. It would appear in most instances that the visual disturbances are due to constriction in either the central retinal artery or the ophthalmic artery, with resultant ischemia of the optic nerve and retina. Diagnosis of retinal migraine is not made at the initial presentation and other causes of transient monocular blindness must be carefully excluded. An embolic etiology is the most important "rule out." Order carotid duplex scanning, echocardiogram and complete blood assessment. If these tests are negative, only then can retinal migraine be diagnosed.

Ophthalmoplegic Migraine

Ophthalmoplegic migraine is exceedingly rare and consists of repeated attacks of headache and temporary oculomotor paralysis in early life. It should be considered last in the differential diagnosis of painful or painless ophthalmoplegia. Structural lesions must always be ruled out by appropriate diagnostic tests. In the typical case, a young child develops a severe migraine headache which is followed by a sudden third nerve palsy leading to ophthalmoplegia. The third nerve is the cranial nerve most commonly affected, but the sixth and seventh cranial nerves have also been affected on occasion.[9] The headache, which may be associated with nausea and vomiting, is unilateral and ipsilateral to the paralysis. The headache may persist for days before ophthalmoplegia sets in. With the involvement of the third cranial nerve, the patient may have a blown pupil and accommodative loss. Recovery is gradual and tends to be less complete after repeated attacks. Ideally, prophylactic therapy would prevent both the occurence of repeated episodes and the development of permanent eye muscle palsies, but reports suggest that therapy has met with only limited success. A trial with propranolol or methysergide to try and stop multiple headaches would certainly appear warranted if the attacks are frequent.

Basilar Migraine

This rare headache occurs primarily in young girls and usually has a

benign prognosis. Commonly it is associated with menstration. The areas supplied by the basilar artery, including the brainstem are commonly affected. This results in profound aura in a bilateral presentation, followed by a severe throbbing headache usually in the occipital region. The visual symptoms described include vivid flashes of light throughout the entire visual field, intense enough to obscure vision completely, and sudden bilateral vision loss occuring over seconds and persisting up to 15 minutes, with a gradual return of vision to normal.[10] This extreme type of migraine usually evolves into more benign presentations with age.

Tension Headache

Tension headache is perhaps the most common of all headaches but has no ophthalmic manifestations. The typical tension type headache sufferer experiences mild to moderate dull aching, nonpulsatile sensation or tightness around the head like that produced by a hatband. The pain is often in the back of the head and neck, extending into the temples, with a concomitant increase in contraction of the muscles of the neck and scalp. Tension type headaches affect males about as often as females. Forty percent of patients have the onset of symptoms by the age of 20.[11] As a result many patients who present to the optometric practice will complain of years of headache. The headache can last anywhere from minutes to weeks in length. Associated with the headache is increased tenderness in the pericranial muscles. Tension headaches can be precipitated by many factors such as fatigue or anxiety. A tension headache occurs with greater frequency than migraine, and unlike migraines, vomiting is rare with tension headaches. These headaches can occur anytime during the day, but most commonly occur during the afternoon or evening and rarely interrupt sleep. A chronic type of tension headache may develop. Chronic tension type headache is classified as occuring 15 or more days per month.

The etiology and pathogenesis of tension type headaches is largely unknown. Until recently most researchers believed that the sustained contraction of skeletal muscles of the head and neck was a major cause of this headache. This sustained contraction was believed to induce ischemia and in turn cause pain. However, electromyographic studies have shown a variable correlation between muscle contraction and actual headache.[12] Vascular studies have also failed to find ischemia during tension type headache.[13] Some researchers believe that tension type headache is only a mild type of migraine. It is believed that when the pain is mild, the headache is labled tension type; when the pain becomes more severe it is called a migraine without aura and when the headache is associated with focal neurologic symptoms, it is called migraine with aura. It remains to be proven whether the same biochemical mechanism is at work in both of these.

Cluster headaches

The etiology of cluster headaches also remains undetermined. Cluster headaches are believed to have a neuronal component, particularly the

trigeminal nerve. Like migraine, cluster headache is considered a vascular headache.

The pain of cluster headache is very excruciating, penetrating and usually nonthrobbing. Pain reaches its peak about 10 to 15 minutes after onset and generally lasts for 45 to 60 minutes. Attacks occur at a frequency of one to three times per day. In 80 percent of patients, the attacks occur in clusters that last 4 to 8 weeks.[14] Onset usually occurs in the late twenties, but may occur at any age. No familial tendency has been established. It involves men more often than women by a ratio of 5.6 to 1.[15] Cluster headache pain is unilateral and is not associated with an aura. Periodicity is the main feature of cluster headache. The average length of remission between cluster periods is about 2 years, although remission may be as short as 2 months or as long as 20 years. Cluster headaches are often precipitated by alcohol, and many sufferers tend to be heavy smokers. Cluster headaches are commonly accompanied by either excessive or deficient neural activity ipsilateral to the pain. Associated features include ipsilateral lacrimation, injection of conjunctiva and nasal stuffiness, and in about 20% of those afflicted, a modified Horners Syndrome consisting of unilateral transient ptosis and miosis.[16]

Though cluster headaches present with characteristic symptoms, neuroimaging should be obtained to rule out a potential intracranial lesion.

Table 3. Characteristics of the three most common headaches

Feature	Migraine*	Tension	Cluster
Age of onset	1st or 2nd decade	Any age	Late twenties
Duration	2 - 12 hours	Minutes to weeks	45 - 60 minutes
Frequency	1/month - 1/year	Weekly-daily	Periods of 3/day
Time of onset	Upon awakening	Evenings	During sleep
Character of pain	Throbbing	Bandlike	Sharp, stabbing
Location	Unilateral	Bilateral	Unilateral
Severity	Moderate-severe	Mild-moderate	Severe
Those affected	Female > male	Female = male	Male > female
Family history	Often present	Present	Not present
Associated Symptoms	Nausea, vomiting, photophobia	Uncommon	Ipsilateral lacrimation, rhinorrhea

*Migraine with aura and migraine without aura

The emergency headache

All headaches should be taken seriously but any of the following presentations should cause particular concern: (1) "first or worst" headache, particularly with acute onset or associated neurologic symptoms; (2) headache with subacute onset that worsens progressively over days or weeks; (3) headache associated with fever, nausea, and vomiting that cannot be

explained by a systemic disorder; (4) headache associated with focal neurologic findings, papilledema, changes in consciousness or cognition (e.g. difficulty in reading, writing or thinking), or stiff neck. A patient with any of these presentations should be referred immediately for neurologic consultation including neuroimaging.

Clinical Examination

Besides the history, which will probably provide the most helpful information, the optometrist needs to thoroughly examine the patient. This includes, of course, ruling out refractive error and uncorrected presbyopia. Blood pressure should be checked for elevation, the sinuses for focal tenderness, and the visual fields for defects which are suggestive of lesions along the visual pathway. The temporal artery should be palpated for tenderness. The eyes also need to be examined for glaucoma, uveitis, and papilloedema. The optometrist should also be aware of any signs of neurological dysfunction. Neuro-imaging and blood tests should be ordered when warranted.

Treatment

A good place to begin in the management of headaches is in trying to identify any potential "triggers." The patient should be encouraged to keep a daily log of activities, foods and beverages that preceeded the headache and potentially precipitated it. Elimination of one or more of these may bring about a reduction in the frequency or severity of the headaches.

Other possible options to consider in the treatment of headaches before pharmacotherapy is started, or to supplement it, include: relaxation techniques, biofeedback, acupuncture, massage, sleep, hot baths and cold showers. General excerise such as swimming and calisthenics have helped some patients by improving the patients general health and sense of well being.

Table 4. Preventive therapy for headaches

NSAIDS	Beta Blockers	Calcium Channel Blockers
Ibuprofen	Propranolol	Verapamil
Naproxen	Atenolol	Diltiazem
Aspirin	Naldol	Nifedipine

Antidepressants	Anticonvulsants
Amiitriptyline	Phenytoin
Doxepin	Carbamazepine
Fluoxetine	Divalproex sodium

The pharmacological management of headaches usually centers on two approaches: preventive and abortive therapy. (See Tables 4 and 5). Preventive

therapy, designed to reduce the frequency, duration, and intensity of attacks, can be accomplished through a wide variety of medications. The most commonly used are beta-blockers, but calcium channel blockers, antidepressants, anticonvulsants, and nonsteroidal anti-inflammatory agents have all been used with success. Agents used in abortive therapy treat the intensity and duration of pain, after the onset of the headache. Abortive therapy usually begins with simple analgesics and NSAIDS followed by narcotic analgesics combinations. When these fail to offer relief, vasoactive compounds such as ergotamine and ergotamine with caffeine are useful. The newest and perhaps the most effective abortive drug available for migraine therapy is sumatriptan. Sumatriptan is a serotonin-recptor agonist. Antiemetics used as adjunctive treatment, particularly with migraine, can be essential in controlling the nausea and vomiting. When other medicatons have failed or are contraindicated, corticosteroids, such as prednisone and dexamethasone, may be used. With corticosteroids as well as the other medications, patients should be monitored for potential side effects.

Table 5. Abortive therapy for headaches

Analgesics	Vasoactive Compounds	Antiemetics
Acetominophen	Ergotamine	Metoclopramide
Aspirin	Ergotamine and caffeine	Prochlorperazine
Acetominophen or aspirin w/ codeine		

Corticosteroids	Seratonin Agonists
Dexamethasone	Sumatriptine
Prednisone	Dihydroergotamine

Conclusion. The optometrist must be ready to handle their patient's complaints of headache. The optometrist does this through a thorough history, examination, and observation of the patient for neurologic dysfunction. By being knowledgeable of the most common types of headache and by being aware of the warning signs of headaches that require medical attention, the optometrist is able to provide the highest standard of care for their patients.

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