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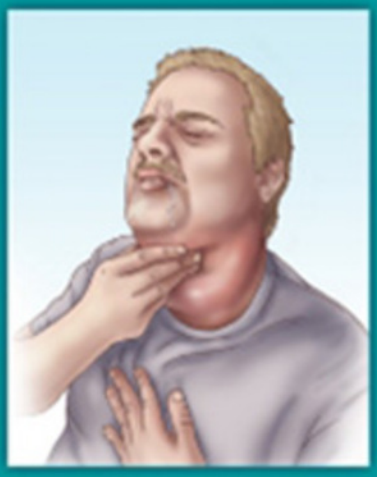

Atlas of COMMON PAIN SYNDROMES

Steven D. Waldman

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Atlas of COMMON PAIN SYNDROMES

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PREFACE

Recently, a medical student told me that, after several weeks of being really sick and being treated for myriad respiratory ills, she was finally diagnosed with pertussis. Now keep in mind that we are located in Kansas City, not Bangladesh. In trying to figure out what went wrong in the care of my student, I asked her several questions: "Were you immunized as a child?" Yes. "Had you recently traveled abroad?" No. "What was the pertussis like?" Horrible!

Having never seen a case of pertussis, I then asked the most obvious question. "How was it diagnosed?" The student initially thought that she had picked up a bad case of bronchitis on her pediatrics rotation. She took a Z-Pak without improvement and then completed a course of moxifloxacin. She went to the student health service on two separate occasions, and both times the doctor concurred with the working diagnosis of bronchitis or early pneumonia. A subsequent trip to the local emergency department yielded the same diagnosis. Her admitting diagnosis to the intensive care unit late one night was for respiratory failure. Antibiotics were given, and breathing treatments administered, yet diagnosis remained elusive. Finally, a second-year medical student suggested that perhaps all this coughing was the result of whooping cough, which the student had just read about in her medical microbiology class. At first, everyone laughed and rolled their eyes… two beats… silence, and then… the correct diagnosis was made.

You may be wondering why I include this story in the preface to a book about pain management. It seems to me that we, as medical practitioners, tend to limit ourselves to specific, personalized constructs that we devise to simplify the diagnosis of painful conditions. Within these constructs are the frequent admonition against hunting for zebras when ever we hear hoof beats and pressures to move toward the center of the bell curve, to cleave to evidence-based medicine, etc. However, if taken to extremes, these parameters severely limit how we process our patients' histories as well as the scope of our diagnoses. It is my hope that the fourth edition of *Atlas of Common Pain Syndromes* will help clinicians recognize, diagnose, and treat painful conditions they otherwise would not have even thought of, and as a result, provide more effective care for their patients who are in pain.

ACKNOWLEDGMENT

I want to give a special thanks to my editors at Elsevier, Michael Houston and Kathryn DeFrancesco, for their keen insights, great advice, and amazing work ethic.

Steven D. Waldman, MD, JD

CONTENTS

1

Acute Herpes Zoster of the First Division of the Trigeminal Nerve

ICD-10 CODE B02.22

THE CLINICAL SYNDROME

Herpes zoster is an infectious disease caused by the varicella-zoster virus (VZV). Primary infection with VZV in a nonimmune host manifests clinically as the childhood disease chickenpox (varicella). Investigators have postulated that during the course of this primary infection, the virus migrates to the dorsal root or cranial ganglia, where it remains dormant and produces no clinically evident disease. In some individuals, the virus reactivates and travels along the sensory pathways of the first division of the trigeminal nerve, where it produces the characteristic pain and skin lesions of herpes zoster, or shingles.

Why reactivation occurs in some individuals but not in others is not fully understood, but investigators have theorized that a decrease in cell-mediated immunity may play an important role in the evolution of this disease by allowing the virus to multiply in the ganglia, spread to the corresponding sensory nerves, and produce clinical disease. Patients who are suffering from malignant disease (particularly lymphoma) or chronic disease and those receiving immunosuppressive therapy (chemotherapy, steroids, radiation) are generally debilitated and thus are much more likely than the healthy population to develop acute herpes zoster. These patients all have in common a decreased cell-mediated immune response, which may also explain why the incidence of shingles increases dramatically in patients older than 60 years and is relatively uncommon in those younger than 20 years.

The first division of the trigeminal nerve is the second most common site for the development of acute herpes zoster, after the thoracic dermatomes. Rarely, the virus attacks the geniculate ganglion and results in hearing loss, vesicles in the ear, and pain (Fig. 1.1). This constellation of symptoms is called Ramsay Hunt syndrome and must be distinguished from acute herpes zoster involving the first division of the trigeminal nerve.

SIGNS AND SYMPTOMS

As viral reactivation occurs, ganglionitis and peripheral neuritis cause pain that may be accompanied by flulike symptoms. The pain generally progresses from a dull, aching sensation to dysesthetic or neuritic pain in the distribution of the first division of the trigeminal nerve. In most patients, the pain of acute herpes zoster precedes the eruption of rash by 3 to 7 days, and this delay often leads to an erroneous diagnosis (see "Differential Diagnosis"). However, in most patients, the clinical diagnosis of shingles is readily made when the characteristic rash appears. As with chickenpox, the rash of herpes zoster appears in crops of macular lesions that rapidly progress to papules and then to vesicles (Fig. 1.2). Eventually, the vesicles coalesce, and crusting occurs (Fig. 1.3). The affected area can be extremely painful, and the pain tends to be exacerbated by any movement or contact (e.g., with clothing or sheets). As the lesions heal, the crust falls away, leaving pink scars that gradually become hypopigmented and atrophic.

In most patients, the hyperesthesia and pain resolve as the skin lesions heal. In some patients, however, pain persists beyond lesion healing. This common and feared complication of acute herpes zoster is called postherpetic neuralgia, and older persons are affected at a higher rate than is the general population suffering from acute herpes zoster (Fig. 1.4). The symptoms of postherpetic neuralgia can vary from a mild, self-limited condition to a debilitating, constantly burning pain that is exacerbated by light touch, movement, anxiety, or temperature change. This unremitting pain may be so severe that it completely devastates the patient's life; ultimately, it

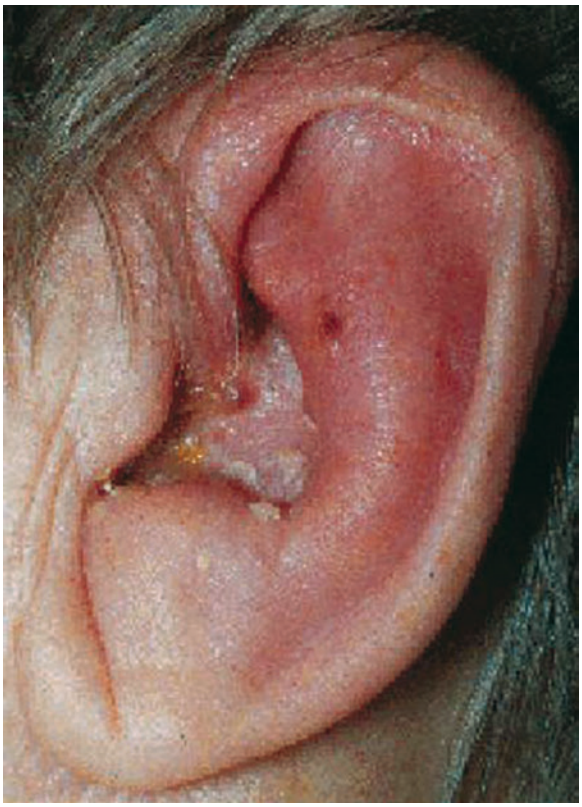

FIG 1.1 Ramsay Hunt syndrome.

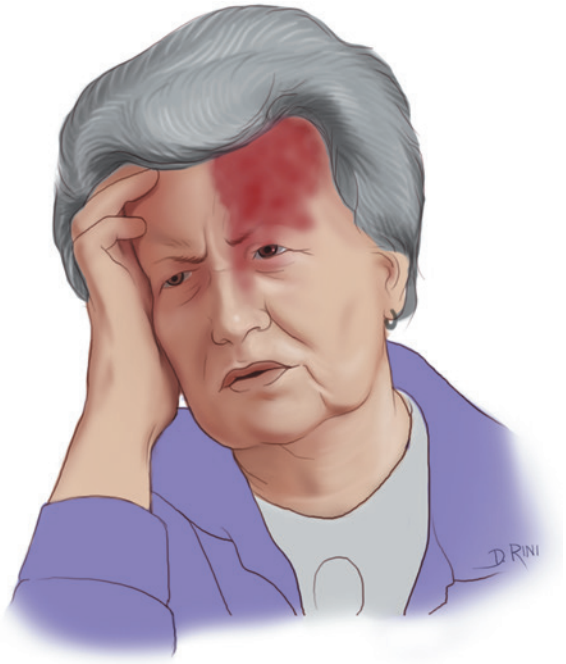

FIG 1.2 The pain of acute herpes zoster of the trigeminal nerve often precedes the characteristic vesicular rash.

can lead to suicide. To avoid this disastrous sequela to a usually benign, self-limited disease, the clinician must use all possible therapeutic efforts in patients with acute herpes zoster of the trigeminal nerve.

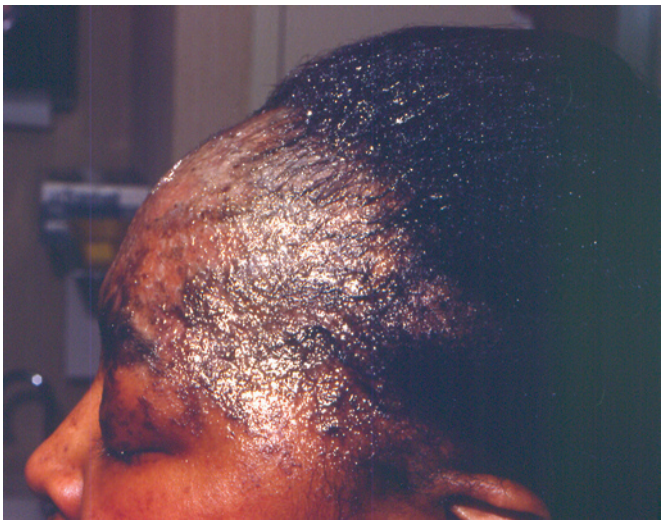

FIG 1.3 Acute herpes zoster involving the ophthalmic division of the left trigeminal nerve. (From Waldman SD. *Pain management*. Philadelphia: Elsevier; 2007.)

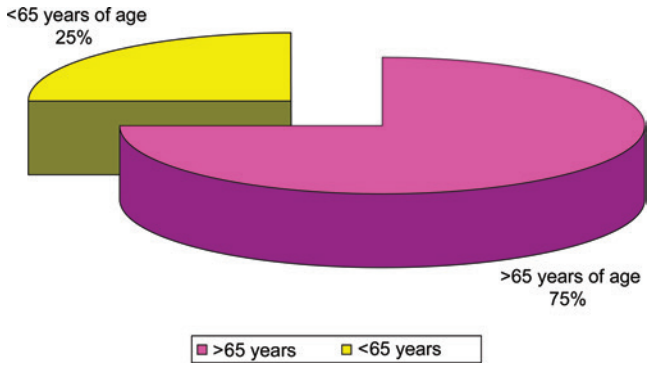

FIG 1.4 Age of patients suffering from acute herpes zoster.

TESTING

Although in most instances the diagnosis is easily made on clinical grounds, confirmatory testing is occasionally required. Such testing may be desirable in patients with other skin lesions that confuse the clinical picture, such as in patients with acquired immunodeficiency syndrome who are suffering from Kaposi sarcoma. In such patients, polymerase chain reaction testing and immunofluorescent antibody testing can rapidly identify herpes zoster virus and distinguish it from herpes simplex infections (Fig. 1.5). In uncomplicated cases, the diagnosis of acute herpes zoster may be strengthened by obtaining a Tzanck smear from the base of a fresh vesicle; this smear reveals multinucleated giant cells and eosinophilic inclusions (Fig. 1.6). However, this inexpensive bedside test does not have the ability to distinguish between lesions caused by the varicella-zoster virus and herpes simplex infections.

DIFFERENTIAL DIAGNOSIS

A careful initial evaluation, including a thorough history and physical examination, is indicated in all patients suffering

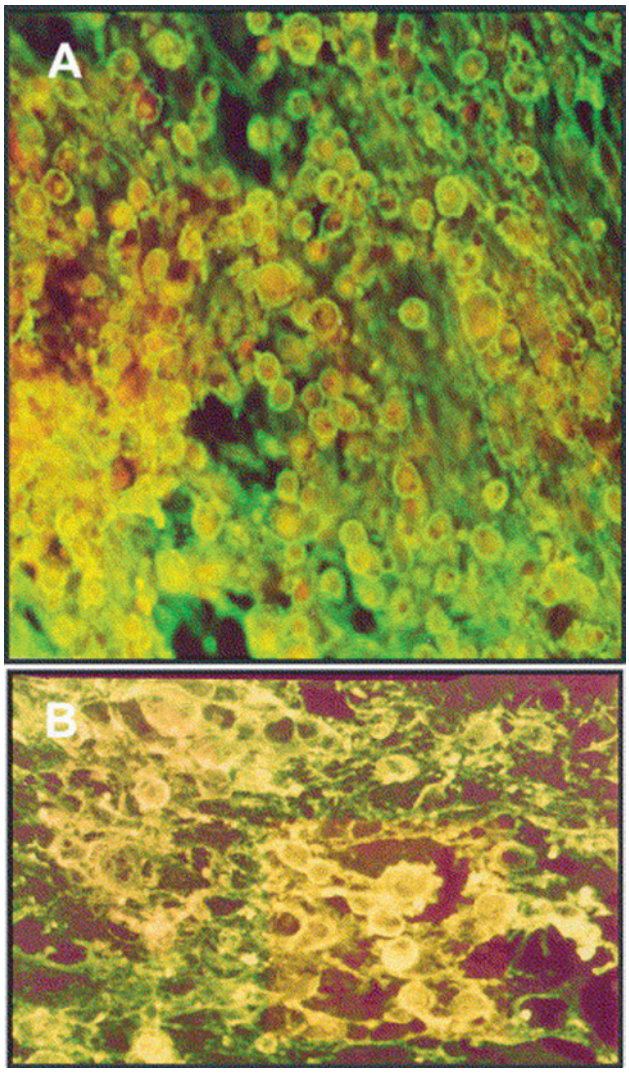

FIG 1.5 Detection of anti-varicella-zoster virus IgG by the fluorescent antibody to membrane antigen assay. (A) Positive result and (B) negative control. (From Sauerbrei A, Färber I, Brandstädt A, Schacke M, Wutzler P. Immunofluorescence test for sensitive detection of varicella-zoster virus-specific IgG: an alternative to fluorescent antibody to membrane antigen test. *J Virol Methods*. 2004;19(1):15-30.)

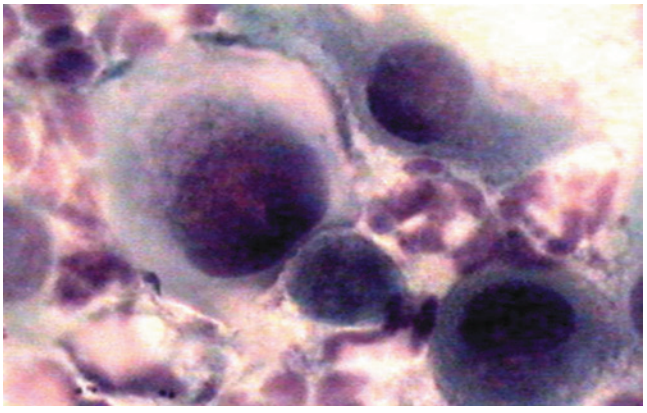

FIG 1.6 Tzanck smear showing giant multinucleated cell. (Courtesy Dr. John Minarcik.)

from acute herpes zoster of the trigeminal nerve. The goal is to rule out occult malignant or systemic disease that may be responsible for the patient's immunocompromised state. A prompt diagnosis allows early recognition of changes in clinical status that may presage the development of complications, including myelitis or dissemination of the disease. Other causes of pain in the distribution of the first division of the trigeminal nerve include trigeminal neuralgia, sinus disease, glaucoma, retro-orbital tumor, inflammatory disease (e.g., Tolosa-Hunt syndrome), and intracranial disease, including tumor.

TREATMENT

The therapeutic challenge in patients presenting with acute herpes zoster of the trigeminal nerve is twofold: (1) the immediate relief of acute pain and symptoms and (2) the prevention of complications, including postherpetic neuralgia. Most pain specialists agree that the earlier treatment is initiated, the less likely it is that postherpetic neuralgia will develop. Further, because older individuals are at the highest risk for developing postherpetic neuralgia, early and aggressive treatment of this group of patients is mandatory.

Nerve Block

Sympathetic neural blockade with local anesthetic and steroid through stellate ganglion block is the treatment of choice to relieve the symptoms of acute herpes zoster of the trigeminal nerve, as well as to prevent postherpetic neuralgia. As vesicular crusting occurs, the steroid may also reduce neural scarring. Sympathetic nerve block is thought to achieve these goals by blocking the profound sympathetic stimulation caused by viral inflammation of the nerve and gasserian ganglion. If untreated, this sympathetic hyperactivity can cause ischemia secondary to decreased blood flow of the intraneural capillary bed. If this ischemia is allowed to persist, endoneural edema forms, thus increasing endoneural pressure and causing a further reduction in endoneural blood flow, with irreversible nerve damage.

These sympathetic blocks should be continued aggressively until the patient is pain free and should be reimplemented if the pain returns. Failure to use sympathetic neural blockade immediately and aggressively, especially in older patients, may sentence the patient to a lifetime of suffering from postherpetic neuralgia. Occasionally, some patients do not experience pain relief from stellate ganglion block but do respond to blockade of the trigeminal nerve.

Opioid Analgesics

Opioid analgesics can be useful to relieve the aching pain that is common during the acute stages of herpes zoster, while sympathetic nerve blocks are being implemented. Opioids are less effective in relieving neuritic pain, which is also common. Careful administration of potent, long-acting opioid analgesics (e.g., oral morphine elixir, methadone) on a time-contingent rather than an as-needed basis may be a beneficial adjunct to the pain relief provided by sympathetic neural blockade.

Because many patients suffering from acute herpes zoster are older or have severe multisystem disease, close monitoring for the potential side effects of potent opioid analgesics (e.g., confusion or dizziness, which may cause a patient to fall) is warranted. Daily dietary fiber supplementation and milk of magnesia should be started along with opioid analgesics to prevent constipation.

Adjuvant Analgesics

The anticonvulsant gabapentin represents a first-line treatment for the neuritic pain of acute herpes zoster of the trigeminal nerve. Studies suggest that gabapentin may also help prevent postherpetic neuralgia. Treatment with gabapentin should begin early in the course of the disease; this drug may be used concurrently with neural blockade, opioid analgesics, and other adjuvant analgesics, including antidepressants, if care is taken to avoid central nervous system side effects. Gabapentin is started at a bedtime dose of 300 mg and is titrated upward in 300-mg increments to a maximum of 3600 mg given in divided doses, as side effects allow. Pregabalin represents a reasonable alternative to gabapentin and is better tolerated in some patients. Pregabalin is started at 50 mg three times a day and may be titrated upward to 100 mg three times a day as side effects allow. Because pregabalin is excreted primarily by the kidneys, the dosage should be decreased in patients with compromised renal function.

Carbamazepine should be considered in patients suffering from severe neuritic pain who fail to respond to nerve blocks and gabapentin. If this drug is used, strict monitoring of hematologic parameters is indicated, especially in patients receiving chemotherapy or radiation therapy. Phenytoin may also be beneficial to treat neuritic pain, but it should not be used in patients with lymphoma; the drug may induce a pseudolymphoma-like state that is difficult to distinguish from the actual lymphoma.

Antidepressants may also be useful adjuncts in the initial treatment of patients suffering from acute herpes zoster. On a short-term basis, these drugs help alleviate the significant sleep disturbance that is commonly seen. In addition, antidepressants may be valuable in ameliorating the neuritic component of the pain, which is treated less effectively with opioid analgesics. After several weeks of treatment, antidepressants may exert a mood-elevating effect, which may be desirable in some patients. Care must be taken to observe closely for central nervous system side effects in this patient population. In addition, these drugs may cause urinary retention and constipation, which may mistakenly be attributed to herpes zoster myelitis.

Antiviral Agents

A few antiviral agents, including valacyclovir, famciclovir, and acyclovir, can shorten the course of acute herpes zoster and may even help prevent the development of postherpetic neuralgia. They are probably useful in attenuating the disease in immunosuppressed patients. These antiviral agents can be used in conjunction with the aforementioned treatment modalities. Careful monitoring for side effects is mandatory.

Adjunctive Treatments

The application of ice packs to the lesions of acute herpes zoster may provide relief in some patients. Application of heat increases pain in most patients, presumably because of the increased conduction of small fibers; however, it is beneficial in an occasional patient and may be worth trying if the application of cold is ineffective. Transcutaneous electrical nerve stimulation and vibration may also be effective in a limited number of patients. The favorable risk-to-benefit ratio of these modalities makes them reasonable alternatives for patients who cannot or will not undergo sympathetic neural blockade or cannot tolerate pharmacologic interventions.

Topical application of aluminum sulfate as a tepid soak provides excellent drying of the crusting and weeping lesions of acute herpes zoster, and most patients find these soaks soothing. Zinc oxide ointment may also be used as a protective agent, especially during the healing phase, when temperature sensitivity is a problem. Disposable diapers can be used as absorbent padding to protect healing lesions from contact with clothing and sheets.

COMPLICATIONS AND PITFALLS

In most patients, acute herpes zoster of the trigeminal nerve is a self-limited disease. In older patients and in immunosuppressed patients, however, complications may occur. Cutaneous and visceral dissemination may range from a mild rash resembling chickenpox to an overwhelming, life-threatening infection in those already suffering from severe multisystem disease. Myelitis may cause bowel, bladder, and lower extremity paresis. Ocular complications of trigeminal nerve involvement may range from severe photophobia to keratitis with loss of sight.

CLINICAL PEARLS

Because the pain of herpes zoster usually precedes the eruption of skin lesions by 3 to 7 days, some other painful condition (e.g., trigeminal neuralgia, glaucoma) may erroneously be diagnosed. In this setting, an astute clinician should advise the patient to call immediately if a rash appears, because acute herpes zoster is a possibility. Some pain specialists believe that in a few immunocompetent patients, when reactivation of VZV occurs, a rapid immune response attenuates the natural course of the disease, and the characteristic rash of acute herpes zoster may not appear. In this case, pain in the distribution of the first division of the trigeminal nerve without an associated rash is called zoster sine herpete and is, by necessity, a diagnosis of exclusion. Therefore other causes of head pain must be ruled out before this diagnosis is invoked.

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2

Migraine Headache

ICD-10 CODE G43.109

THE CLINICAL SYNDROME

Migraine headache is a periodic unilateral headache that may begin in childhood, but almost always develops before age 30 years. Attacks occur with variable frequency, ranging from every few days to once every several months. More frequent migraine headaches are often associated with a phenomenon called analgesic rebound. Between 60% and 70% of patients who suffer from migraine are female, and many report a family history of migraine headache. The personality type of migraineurs has been described as meticulous, neat, compulsive, and often rigid. They tend to be obsessive in their daily routines and often find it hard to cope with the stresses of everyday life. Migraine headache may be triggered by changes in sleep patterns or diet or by the ingestion of tyramine-containing foods, monosodium glutamate, nitrates, chocolate, wine, or citrus fruits. Changes in endogenous and exogenous hormones, such as with the use of birth control pills, can also trigger migraine headache as can the ingestion of nitroglycerine for angina. The typical migraine headache is characterized by four phases: (1) the prodrome; (2) the aura; (3) the headache; and (4) the postdrome (Fig. 2.1). Some migraineurs will experience a premonition or warning that a migraine may be on the horizon. This premonition or warning is known as a prodrome and may manifest as mood changes, food cravings, frequent yawning, changes in libido, and constipation. Approximately 20% of patients suffering from migraine headache also experience a neurologic event before the onset of pain called an aura. The aura most often takes the form of a visual disturbance, but it may also manifest as an alteration in smell or hearing; these are called olfactory and auditory auras, respectively. Following a migraine headache, some patients will experience a period of confusion, dizziness, weakness, or elation known as a postdrome.

SIGNS AND SYMPTOMS

Migraine headache is, by definition, a unilateral headache. Although the headache may change sides with each episode, the headache is never bilateral at its onset. The pain of migraine headache is usually periorbital or retro-orbital. It is pounding, and its intensity is severe. The time from onset to peak of migraine pain is short, ranging from 20 minutes to 1 hour. In contradistinction to tension-type headache, migraine headache is often associated with systemic symptoms, including nausea and vomiting, photophobia, and sonophobia, as well as alterations in appetite, mood, and libido. Menstruation is a common trigger of migraine headache.

As mentioned, in approximately 20% of patients, migraine headache is preceded by an aura (called migraine with aura). The aura is thought to be the result of ischemia of specific regions of the cerebral cortex. A visual aura often occurs 30 to 60 minutes before the onset of headache pain; this may take the form of blind spots, called scotoma, or a zigzag disruption of the visual field, called fortification spectrum (Fig. 2.2). Occasionally, patients with migraine lose an entire visual field during the aura. Auditory auras usually take the form of hypersensitivity to sound, but other alterations of hearing, such as sounds perceived as farther away than they actually are, have also been reported. Olfactory auras may take the form of strong odors of substances that are not actually present or extreme hypersensitivity to otherwise normal odors, such as coffee or copy machine toner. Migraine that manifests without other neurologic symptoms is called migraine without aura.

Rarely, patients who suffer from migraine experience prolonged neurologic dysfunction associated with the headache pain. Such neurologic dysfunction may last for more than 24 hours and is termed migraine with prolonged aura. These patients are at risk for the development of permanent neurologic deficit, and risk factors such as hypertension, smoking, and oral contraceptives, must be addressed. Even less common than migraine with prolonged aura is migraine with complex aura. Patients suffering from migraine with complex aura experience significant neurologic dysfunction that may include aphasia or hemiplegia. As with migraine with prolonged aura, patients suffering from migraine with complex aura may develop permanent neurologic deficits.

Patients suffering from all forms of migraine headache appear systemically ill (Fig. 2.3). Pallor, tremulousness, diaphoresis, and light sensitivity are common physical findings. The temporal artery and the surrounding area may be tender. If an aura is present, results of the neurologic examination will be abnormal; the neurologic examination is usually within normal limits before, during, and after migraine without aura.

TESTING

No specific test exists for migraine headache. Testing is aimed primarily at identifying occult pathologic processes or other

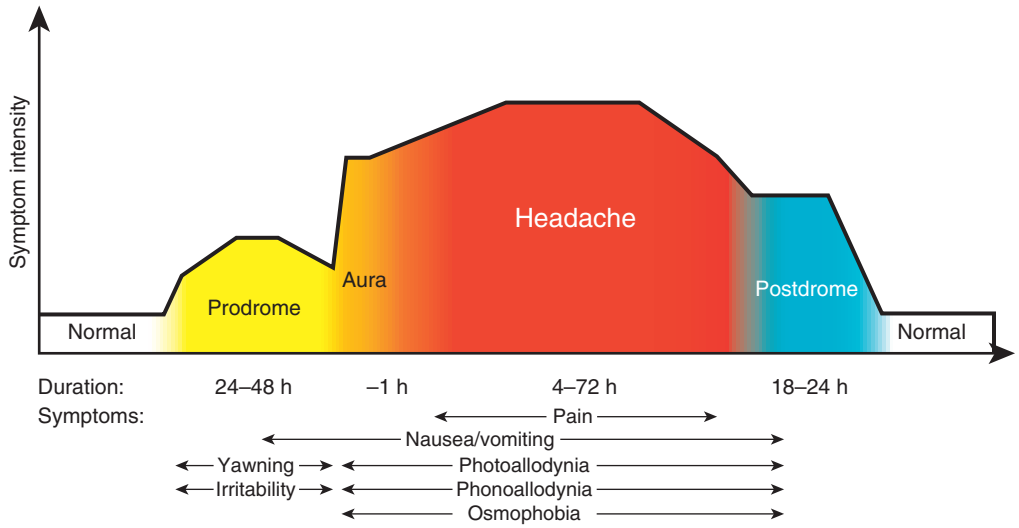

FIG 2.1 The Four Phases of Migraine. (Redrawn from Burgos-Vega C, Moy J, Dussor G. Meningeal afferent signaling and the pathophysiology of migraine. *Prog Mol Biol Transl Sci.* 2015;131:537-564.)

FIG 2.2 Artist's depiction of zigzag fortification spectrum visual aura. (From Podoll K, Ayles D. Sarah Raphael's migraine with aura as inspiration for the foray of her work into abstraction. *Int Rev Neurobiol.* 2006;74:109-118.)

FIG 2.3 Migraine headache is an episodic, unilateral headache that occurs most commonly in female patients.

diseases that may mimic migraine headache (see "Differential Diagnosis"). All patients with a recent onset of headache thought to be migraine should undergo magnetic resonance imaging (MRI) of the brain. If neurologic dysfunction accompanies the patient's headache symptoms, MRI should be performed with and without gadolinium contrast medium (Fig. 2.4); magnetic resonance angiography should be considered as well. MRI should also be performed in patients with previously stable migraine headaches who experience an inexplicable change in symptoms. Screening laboratory tests, including an erythrocyte sedimentation rate, complete blood count, and automated blood chemistry, should be performed if the diagnosis of migraine is in question. Ophthalmologic evaluation is indicated in patients who experience significant ocular symptoms.

DIFFERENTIAL DIAGNOSIS

The diagnosis of migraine headache is usually made on clinical grounds by obtaining a targeted headache history. Tension-type headache is often confused with migraine headache, and this misdiagnosis can lead to illogical treatment plans because these two headache syndromes are managed quite differently.

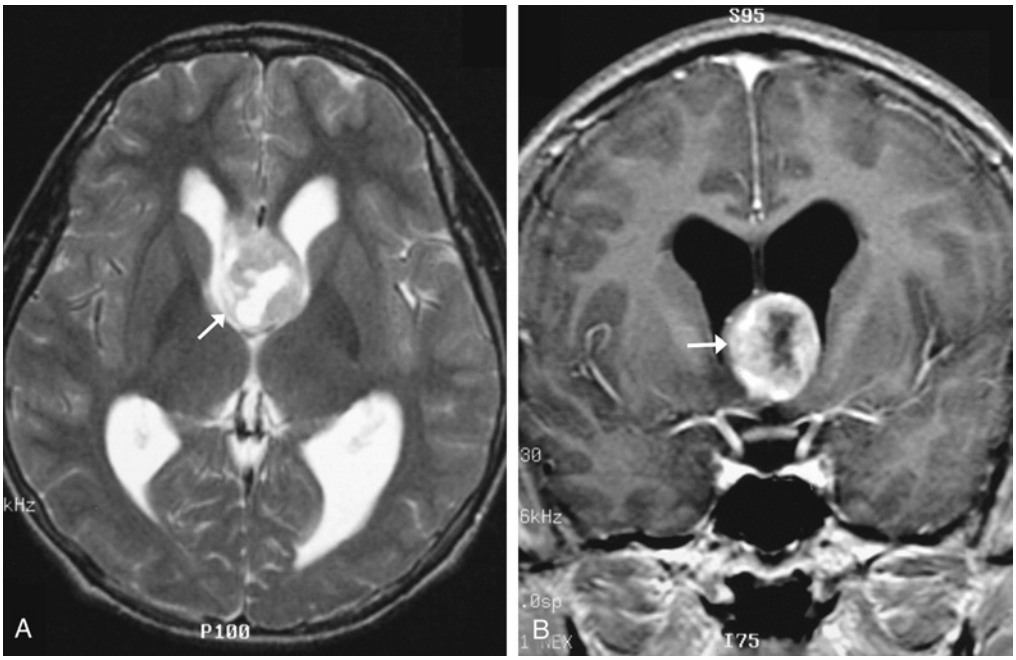

FIG 2.4 Glioblastoma multiforme involving the septum pellucidum. **A,** Axial T2-weighted magnetic resonance imaging (MRI) through the inferior aspect of the frontal horns of the lateral ventricles. An ovoid, heterogeneously hyperintense mass *(arrow)* arising from the inferior aspect of the septum pellucidum indents and partially occludes the frontal horns bilaterally. Note the irregularly marginated intratumoral hyperintensity, suggesting central necrosis. **B,** Following intravenous administration of gadolinium, coronal T1-weighted MRI demonstrates intense contrast enhancement *(arrow)* of the thick peripheral rind, with nonenhancement of the central cavity. (From Haaga JR, Lanzieri CF, Gilkeson RC, eds. *CT and MR imaging of the whole body*. 4th ed. Philadelphia: Mosby; 2003:140.)

Table 2.1 distinguishes migraine headache from tension-type headache and should help clarify the diagnosis.

Diseases of the eyes, ears, nose, and sinuses may also mimic migraine headache. The targeted history and physical examination, combined with appropriate testing, should allow the clinician to identify and properly treat any underlying diseases of these organ systems. The following conditions may all mimic migraine and must be considered when treating patients with headache: glaucoma temporal arteritis; sinusitis; intracranial disease, including chronic subdural hematoma, tumor (see Fig. 2.4), brain abscess, hydrocephalus, and pseudotumor cerebri; and inflammatory conditions, including sarcoidosis.

TABLE 2.1 Comparison of Migraine Headache and Tension-Type Headache

	Migraine Headache	Tension-Type Headache
Onset-to-peak interval	Minutes to 1 hr	Hours to days
Frequency	Rarely >1/wk	Often daily or continuous
Location	Temporal	Nuchal or circumferential
Character	Pounding	Aching, pressure, bandlike
Laterality	Always unilateral	Usually bilateral
Aura	May be present	Never present
Nausea and vomiting	Common	Rare
Duration	Usually <24 hr	Often days

TREATMENT

When deciding how best to treat a patient suffering from migraine, the clinician should consider the frequency and severity of the headaches, their effect on the patient's lifestyle, the presence of focal or prolonged neurologic disturbances, the results of previous testing and treatment, any history of previous drug abuse or misuse, and the presence of other systemic diseases (e.g., peripheral vascular or coronary artery disease) that may preclude the use of certain treatment modalities.

If the patient's migraine headaches occur infrequently, a trial of abortive therapy may be warranted. However, if the headaches occur with greater frequency or cause the patient to miss work or be hospitalized, prophylactic therapy is warranted.

Abortive Therapy

For abortive therapy to be effective, it must be initiated at the first sign of headache. This is often difficult because of

the short interval between the onset and peak of migraine headache, coupled with the problem that migraine sufferers often experience nausea and vomiting that may limit the use of oral medications. By altering the route of administration to parenteral or transmucosal, this situation can be avoided.

Abortive medications that can be considered in patients with migraine headache include compounds that contain isometheptene mucate (e.g., Midrin); the nonsteroidal anti-inflammatory drug (NSAID) naproxen; ergot alkaloids; the triptans including sumatriptan, rizatriptan, almotriptan, naratriptan, zolmitriptan, frovatriptan, and eletriptan; and intravenous lidocaine combined with antiemetic compounds. The inhalation of 100% oxygen may abort migraine headache, and sphenopalatine ganglion block with local anesthetic may be effective. Caffeine-containing preparations, barbiturates, ergotamines, triptans, and opioids have a propensity to cause a phenomenon called analgesic rebound headache, which may ultimately be more difficult to treat than the original migraine. The ergotamines and triptans should not be used in patients with coexistent peripheral vascular disease, coronary artery disease, or hypertension.

Prophylactic Therapy

For most patients with migraine headache, prophylactic therapy is a better option than abortive therapy. The mainstay of prophylactic therapy is β-blocking agents. Propranolol, metoprolol, timolol, and most other drugs in this class can control or decrease the frequency and intensity of migraine headache and help prevent auras. An 80-mg daily dose of the long-acting formulation is a reasonable starting point for most patients with migraine. Propranolol should not be used in patients with asthma or other reactive airway diseases.

Valproic acid, calcium channel blockers (e.g., verapamil), clonidine, tricyclic antidepressants, the angiotensin-converting enzyme inhibitor lisinopril, and NSAIDs have also been used for the prophylaxis of migraine headache. Onabotulinum toxin A may also provide migraine prophylaxis in selected patients. Each of these drugs has advantages and disadvantages, and the clinician should tailor a treatment plan that best meets the needs of the individual patient.

COMPLICATIONS AND PITFALLS

In most patients, migraine headache is a painful but not life-threatening disease. However, patients who suffer from migraine with prolonged aura or migraine with complex aura are at risk for the development of permanent neurologic deficits. Such patients are best treated by headache specialists who are familiar with these unique risks and are better equipped to deal with them. Occasionally, prolonged nausea and vomiting associated with severe migraine headache may result in dehydration that necessitates hospitalization and treatment with intravenous fluids.

CLINICAL PEARLS

The most common reason for a patient's lack of response to traditional treatment for migraine headache is that the patient is actually suffering from tension-type headache, analgesic rebound headache, or a combination of headache syndromes. The clinician must be sure that the patient is not taking significant doses of over-the-counter headache preparations containing caffeine or other vasoactive drugs, such as barbiturates, ergots, or triptans, that may cause analgesic rebound headache. Until these drugs are withdrawn, the patient's headache will not improve.

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3

Tension-Type Headache

ICD-10 CODE G44.209

THE CLINICAL SYNDROME

Tension-type headache, formerly known as muscle contraction headache, is the most common type of headache that afflicts humankind. It can be episodic or chronic, and it may or may not be related to muscle contraction. Significant sleep disturbance usually occurs. Patients with tension-type headache are often characterized as having multiple unresolved conflicts surrounding work, marriage, and social relationships, and psychosexual difficulties. Testing with the Minnesota Multiphasic Personality Inventory in large groups of patients with tension-type headache revealed not only borderline depression but somatization as well. Most researchers believe that this somatization takes the form of abnormal muscle contraction in some patients; in others, it results in simple headache.

SIGNS AND SYMPTOMS

Tension-type headache is usually bilateral, but can be unilateral; it often involves the frontal, temporal, and occipital regions (Fig. 3.1). It may present as a bandlike, nonpulsatile ache or tightness in the aforementioned anatomic areas (Fig. 3.2). Associated neck symptoms are common. Tension-type headache evolves over a period of hours or days and then tends to remain constant, without progression. It has no associated aura, but significant sleep disturbance is usually present. This disturbance may manifest as difficulty falling asleep, frequent awakening at night, or early awakening. These headaches most frequently occur between 4 and 8 AM and 4 and 8 PM. Although both sexes are affected, female patients predominate. No hereditary pattern to tension-type headache is found, but this type of headache may occur in family clusters because children mimic and learn the pain behavior of their parents.

The triggering event for acute, episodic tension-type headache is invariably either physical or psychological stress. This may take the form of a fight with a coworker or spouse or an exceptionally heavy workload. Physical stress, such as a long drive, working with the neck in a strained position, acute cervical spine injury resulting from whiplash, or prolonged exposure to the glare from a cathode ray tube, may precipitate a headache. A worsening of preexisting degenerative cervical spine conditions, such as cervical spondylosis, can also trigger a tension-type headache. The pathologic process responsible for the development of tension-type headache can produce temporomandibular joint dysfunction as well.

TESTING

No specific test exists for tension-type headache. Testing is aimed primarily at identifying an occult pathologic process or other diseases that may mimic tension-type headache (see "Differential Diagnosis"). All patients with a recent onset of headache that is thought to be tension type should undergo magnetic resonance imaging (MRI) of the brain and, if significant occipital or nuchal symptoms are present, of the cervical spine. MRI should also be performed in patients with previously stable tension-type headaches who have experienced a recent change in symptoms. Screening laboratory tests consisting of a complete blood count, erythrocyte sedimentation rate, and automated blood chemistry should be performed if the diagnosis of tension-type headache is in question.

DIFFERENTIAL DIAGNOSIS

Tension-type headache is usually diagnosed on clinical grounds by obtaining a targeted headache history. Despite their obvious differences, tension-type headache is often incorrectly diagnosed as migraine headache. Such misdiagnosis can lead to illogical treatment plans and poor control of headache symptoms. Table 3.1 helps distinguish tension-type headache from migraine headache and should aid the clinician in making the correct diagnosis.

Diseases of the cervical spine and surrounding soft tissues may also mimic tension-type headache. Arnold-Chiari malformations may manifest clinically as tension-type headache, but these malformations can easily be identified on images of the posterior fossa and cervical spine (Fig. 3.3). Occasionally, frontal sinusitis is confused with tension-type headache, although individuals with acute frontal sinusitis appear systemically ill. Temporal arteritis, chronic subdural hematoma, and other intracranial disease such as tumor may be incorrectly diagnosed as tension-type headache.

TREATMENT

Abortive Therapy

In determining the best treatment, the physician must consider the frequency and severity of the headaches, their effect on

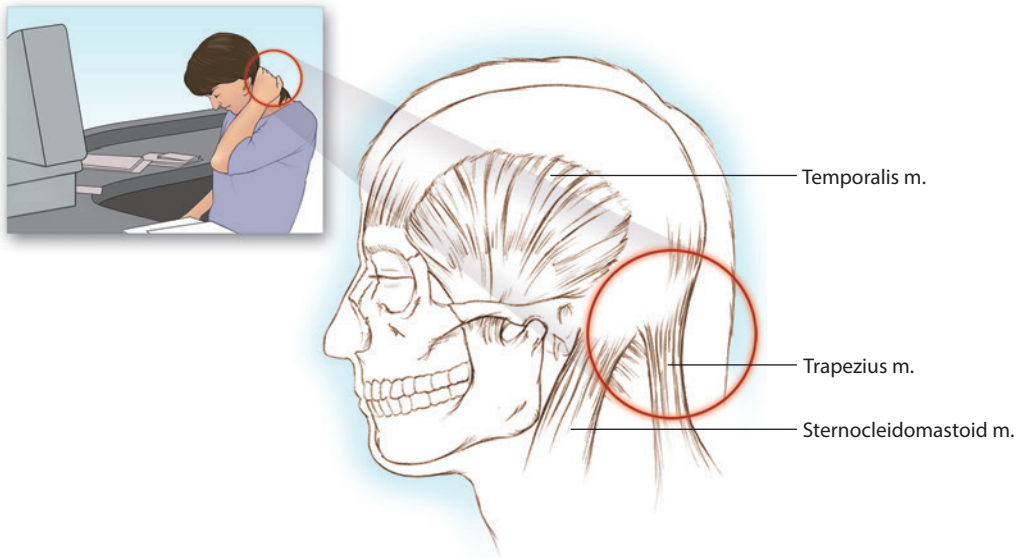

FIG 3.1 Mental or physical stress is often the precipitating factor in tension-type headache.

FIG 3.2 Tension-type headache presents as a bandlike, nonpulsatile ache or tightness in the forehead, temples, neck, and occipital regions. (Redrawn from Kaufman DM. *Kaufman's clinical neurology for psychiatrists*. 7th ed. Philadelphia: Elsevier; 2013:F9-1.)

TABLE 3.1 Comparison of Tension-Type Headache and Migraine Headache

	Tension-Type Headache	Migraine Headache
Onset-to-peak interval	Hours to days	Minutes to 1 hr
Frequency	Often daily or continuous	Rarely >1/wk
Location	Nuchal or circumferential	Temporal
Character	Aching, pressure, bandlike	Pounding
Laterality	Usually bilateral	Always unilateral
Aura	Never present	May be present
Nausea and vomiting	Rare	Common
Duration	Often days	Usually <24 hr

the patient's lifestyle, the results of any previous therapy, and any prior drug misuse or abuse. If the patient suffers an attack of tension-type headache only once every 1 or 2 months, the condition can often be managed by teaching the patient to reduce or avoid stress. Analgesics or nonsteroidal antiinflammatory drugs (NSAIDs) can provide symptomatic relief during acute attacks. Combination analgesic drugs used concomitantly with barbiturates or opioid analgesics have no place in the treatment of patients with headache. The risk of abuse and dependence more than outweighs any theoretic benefit. The physician should also avoid an abortive treatment approach in patients with a prior history of drug misuse or abuse. Many drugs, including simple analgesics and NSAIDs, can produce serious consequences if they are abused.

Prophylactic Therapy

If the headaches occur more frequently than once every 1 or 2 months or are so severe that the patient repeatedly misses work or social engagements, prophylactic therapy is indicated.

Antidepressants

Antidepressants are generally the drugs of choice for the prophylactic treatment of tension-type headache. These drugs not only help decrease the frequency and intensity of headaches but also normalize sleep patterns and treat any underlying

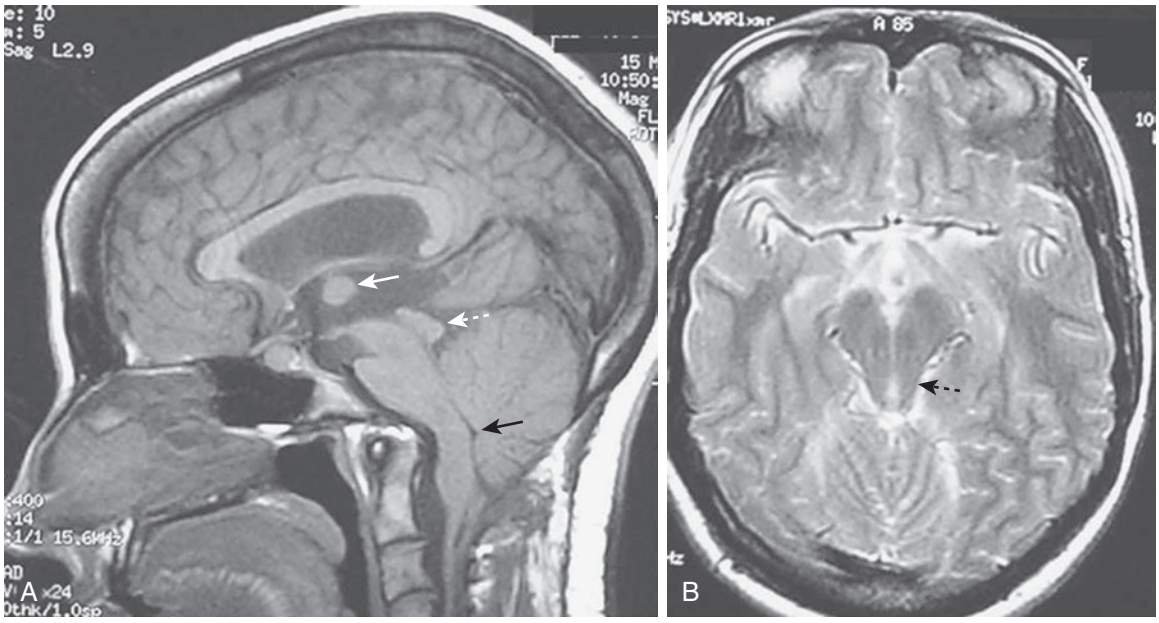

FIG 3.3 A, Sagittal T1-weighted magnetic resonance imaging (MRI) of an adult patient with Arnold-Chiari type II deformity. The posterior fossa is small with a widened foramen magnum. Inferior displacement of the cerebellum and medulla with elongation of the pons and fourth ventricle *(black arrow)* is evident. The brainstem is kinked as it passes over the back of the odontoid. An enlarged massa with intermedia *(white arrow)* and beaking of the tectum *(broken white arrow)* are visible. **B,** Axial T2-weighted MRI shows the small posterior fossa with beaking of the tectum *(broken black arrow)*. (From Waldman SD, Campbell RSD. *Imaging of pain*. Philadelphia: Saunders; 2011:30.)

depression. Patients should be educated about the potential side effects of this class of drugs, including sedation, dry mouth, blurred vision, constipation, and urinary retention. Patients should also be told that relief of headache pain generally takes 3 to 4 weeks. However, normalization of sleep occurs immediately, and this may be enough to provide a noticeable improvement in headache symptoms.

Amitriptyline, started at a single bedtime dose of 25 mg, is a reasonable initial choice. The dose may be increased in 25-mg increments as side effects allow. Other drugs that can be considered if the patient does not tolerate the sedative and anticholinergic effects of amitriptyline include trazodone (75 to 300 mg at bedtime) or fluoxetine (20 to 40 mg at lunchtime). Because of the sedating nature of these drugs (with the exception of fluoxetine), they must be used with caution in older patients and in others who are at risk for falling. Care should also be exercised when using these drugs in patients who are susceptible to cardiac arrhythmias, because these drugs may be arrhythmogenic. Simple analgesics or longer-acting NSAIDs may be used with antidepressant compounds to treat exacerbations of headache pain.

Biofeedback

Monitored relaxation training combined with patient education about coping strategies and stress-reduction techniques may be of value in some tension-type headache sufferers who are adequately motivated. Patient selection is of paramount importance if good results are to be achieved. If the patient is significantly depressed, it may be beneficial to treat the depression before trying biofeedback. The use of biofeedback may allow the patient to control the headaches while avoiding the side effects of medications.

Cervical Epidural Nerve Block

Multiple studies have demonstrated the efficacy of cervical epidural nerve block with steroid in providing long-term relief of tension-type headaches in patients for whom all other treatment modalities have failed. This treatment can also be used while waiting for antidepressant compounds to become effective. Cervical epidural nerve block can be performed on a daily to weekly basis, depending on clinical symptoms.

COMPLICATIONS AND PITFALLS

A few patients with tension-type headache have major depression or uncontrolled anxiety states in addition to a chemical dependence on opioid analgesics, barbiturates, minor tranquilizers, or alcohol. Attempts to treat these patients in the outpatient setting are disappointing and frustrating. Inpatient treatment in a specialized headache unit or psychiatric setting results in more rapid amelioration of the underlying and coexisting problems and allows the concurrent treatment of headache. Monoamine oxidase inhibitors can often reduce the frequency and severity of tension-type headache in this subset of patients. Phenelzine, at a dosage of 15 mg three times a day, is usually effective. After 2 to 3 weeks, the dosage is

tapered to an appropriate maintenance dose of 5 to 10 mg three times a day. Monoamine oxidase inhibitors can produce life-threatening hypertensive crises if special diets are not followed or if these drugs are combined with some commonly used prescription or over-the-counter medications. Therefore their use should be limited to highly reliable and compliant patients. Physicians prescribing this potentially dangerous group of drugs should be well versed in how to use them safely.

CLINICAL PEARLS

Although tension-type (muscle contraction) headache occurs frequently, it is commonly misdiagnosed as migraine headache. By obtaining a targeted headache history and performing a targeted physical examination, the physician can make a diagnosis with a high degree of certainty. The avoidance of addicting medications, coupled with the appropriate use of pharmacologic and nonpharmacologic therapies, should result in excellent palliation and long-term control of pain in most patients suffering from this headache syndrome.

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4

Cluster Headache

ICD-10 CODE G44.009

THE CLINICAL SYNDROME

Cluster headache derives its name from the headache pattern—that is, headaches occur in clusters, followed by headache-free remission periods. Cluster headache is a primary headache that is included in the group of headaches known as the trigeminal autonomic cephalgias. Unlike other common headache disorders that affect primarily female patients, cluster headache is much more common in male patients, with a male-to-female ratio of 5:1. Much less common than tension-type headache or migraine headache, cluster headache is thought to affect approximately 0.5% of the male population. Cluster headache is most often confused with migraine by clinicians who are unfamiliar with the syndrome; however, a targeted headache history allows the clinician to distinguish between these two distinct headache types easily (Table 4.1).

The onset of cluster headache occurs in the late third or early fourth decade of life, in contradistinction to migraine, which almost always manifests by the early second decade. Unlike migraine, cluster headache does not appear to run in families, and cluster headache sufferers do not experience auras. Attacks generally occur approximately 90 minutes after the patient falls asleep. This association with sleep is reportedly maintained when a shift worker changes from nighttime to daytime hours of sleep. Cluster headache also appears to follow a distinct chronobiologic pattern that coincides with seasonal changes in the length of the day. This pattern results in an increased frequency of cluster headache in the spring and fall.

During a cluster period, attacks occur two or three times a day and last for 45 minutes to 1 hour. Cluster periods usually last for 8 to 12 weeks, interrupted by remission periods of less than 2 years. In rare patients, the remission periods become shorter and shorter, and the frequency may increase up to 10-fold. This situation is termed chronic cluster headache and differs from the more common episodic cluster headache described earlier.

SIGNS AND SYMPTOMS

Cluster headache is characterized as a unilateral headache that is retro-orbital and temporal in location. The pain has a deep burning or boring quality. Physical findings during an attack of cluster headache may include Horner's syndrome, consisting of ptosis, abnormal pupil constriction, facial flushing, and conjunctival injection (Fig. 4.1). Additionally, profuse lacrimation and rhinorrhea are often present. The ocular changes may become permanent with repeated attacks. Peau d'orange skin over the malar region, deeply furrowed glabellar folds, and telangiectasia may be observed.

Attacks of cluster headache may be provoked by small amounts of alcohol, nitrates, histamines, and other vasoactive substances, as well as occasionally by high altitude. When the attack is in progress, the patient may be unable to lie still and may pace or rock back and forth in a chair. This behavior contrasts with that characterizing other headache syndromes, during which patients seek relief by lying down in a dark, quiet room.

The pain of cluster headache is said to be among the worst pain a human being can suffer. Because of the severity of the pain, the clinician must watch closely for medication overuse or misuse. Suicide has been associated with prolonged, unrelieved attacks of cluster headache.

TESTING

No specific test exists for cluster headache. Testing is aimed primarily at identifying an occult pathologic process or other diseases that may mimic cluster headache (see "Differential Diagnosis"). All patients with a recent onset of a headache thought to be a cluster headache should undergo magnetic resonance imaging (MRI) of the brain. If neurologic dysfunction accompanies the patient's headache symptoms, MRI should be performed with and without gadolinium contrast medium (Fig. 4.2); magnetic resonance angiography should be considered as well. MRI should also be performed in patients with previously stable cluster headache who experience an inexplicable change in symptoms. Screening laboratory tests, including an erythrocyte sedimentation rate, complete blood count, and automated blood chemistry, should be performed if the diagnosis of cluster headache is in question. Ophthalmologic evaluation, including measurement of intraocular pressures, is indicated in patients who experience significant ocular symptoms.

DIFFERENTIAL DIAGNOSIS

Cluster headache is usually diagnosed on clinical grounds by obtaining a targeted headache history. Migraine headache is

TABLE 4.1 Comparison of Cluster Headache and Migraine Headache

	Cluster Headache	Migraine Headache
Gender	Male 5:1	Female 2:1
Age of onset	Late 30s to early 40s	Menarche to early 20s
Family history	No	Yes
Aura	Never	May be present (20% of the time)
Chronobiologic pattern	Yes	No
Onset-to-peak interval	Seconds to minutes	Minutes to 1 hr
Frequency	2 or 3/day	Rarely >1/wk
Duration	45 min	Usually <24 hr

FIG 4.1 Horner's eye findings. Classic clinical eye findings are demonstrated in this patient with a right Horner syndrome (ptosis of the upper eyelid, elevation of the lower eyelid, and miosis). (From Reede DL, Garcon E, Smoker WR, et al. Horner's syndrome: clinical and radiographic evaluation. *Neuroimaging Clin N Am*. 2008; 18(2):369-385.)

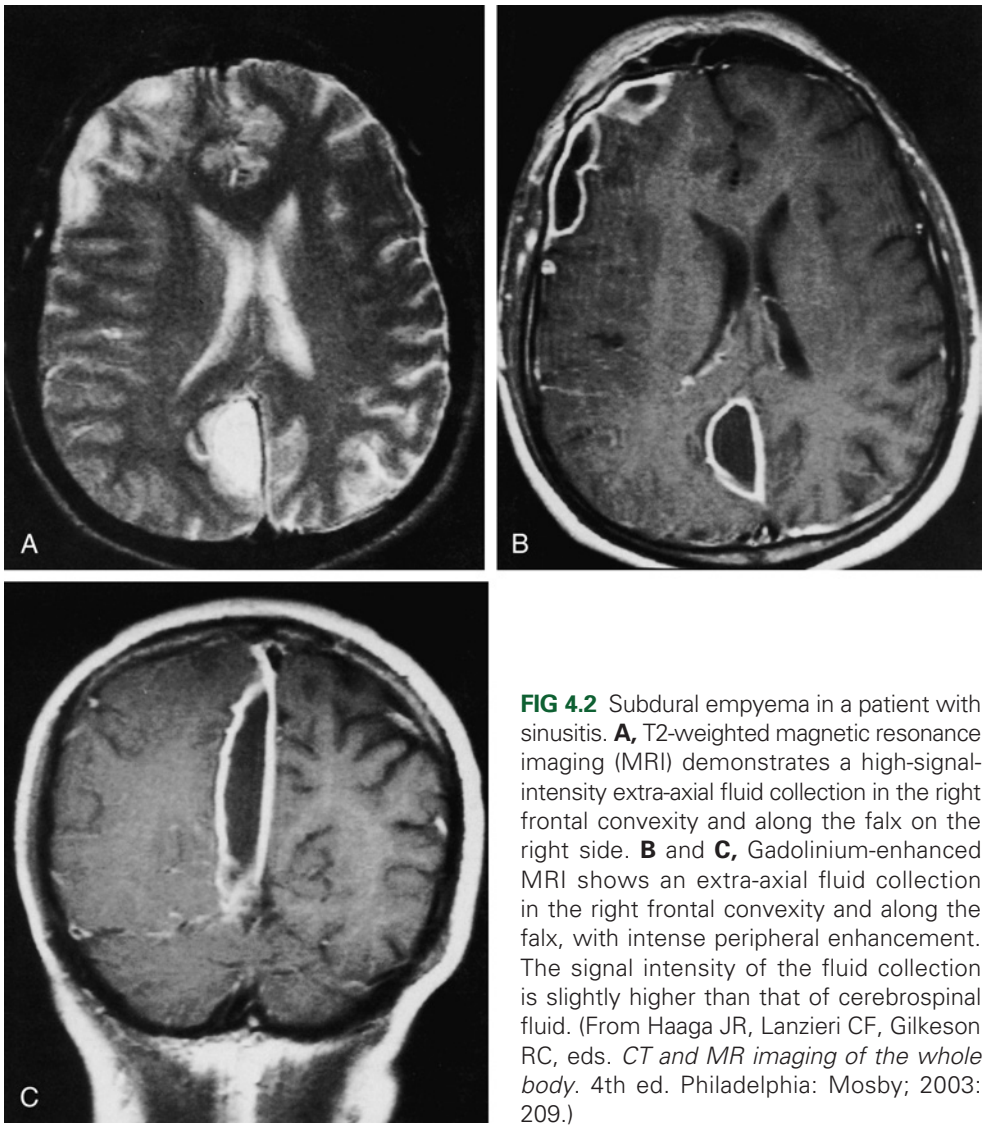

FIG 4.2 Subdural empyema in a patient with sinusitis. **A,** T2-weighted magnetic resonance imaging (MRI) demonstrates a high-signal-intensity extra-axial fluid collection in the right frontal convexity and along the falx on the right side. **B** and **C,** Gadolinium-enhanced MRI shows an extra-axial fluid collection in the right frontal convexity and along the falx, with intense peripheral enhancement. The signal intensity of the fluid collection is slightly higher than that of cerebrospinal fluid. (From Haaga JR, Lanzieri CF, Gilkeson RC, eds. *CT and MR imaging of the whole body*. 4th ed. Philadelphia: Mosby; 2003: 209.)

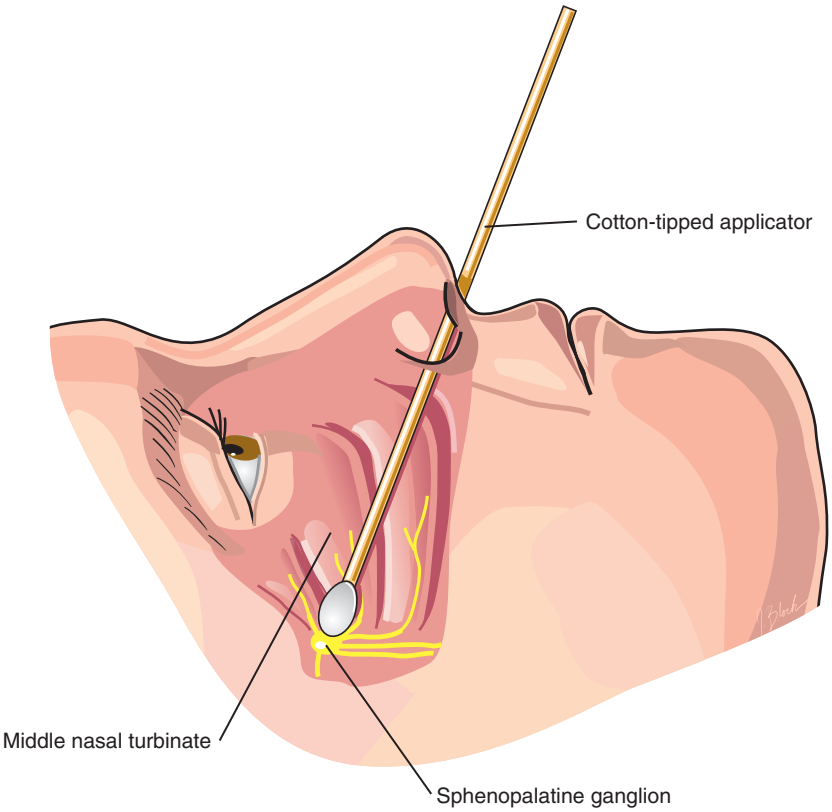

FIG 4.3 Sphenopalatine ganglion block is a useful treatment in the management of cluster headache. (From Waldman SD. *Atlas of interventional pain management*. 4th ed. Philadelphia: Elsevier; 2015.)

often confused with cluster headache, and this misdiagnosis can lead to illogical treatment plans because the management of these two headache syndromes is quite different. Table 4.1 distinguishes cluster headache from migraine headache and should help clarify the diagnosis.

Diseases of the eyes, ears, nose, and sinuses may also mimic cluster headache. The targeted history and physical examination, combined with appropriate testing, should help an astute clinician identify and properly treat any underlying diseases of these organ systems. The following conditions may all mimic cluster headache and must be considered in patients with headache: glaucoma; temporal arteritis; sinusitis (see Fig. 4.2); intracranial disease, including chronic subdural hematoma, tumor, brain abscess, hydrocephalus, and pseudotumor cerebri; and inflammatory conditions, including sarcoidosis.

TREATMENT

Whereas most patients with migraine headache experience improvement with β-blocker therapy, patients suffering from cluster headache usually require more individualized therapy. Initial treatment is commonly prednisone combined with daily sphenopalatine ganglion blocks with local anesthetic (Fig. 4.3). A reasonable starting dose of prednisone is 80 mg given in divided doses and tapered by 10 mg/dose per day. If headaches are not rapidly brought under control, inhalation of 100% oxygen through a close-fitting mask is added. Octreotide, a synthetic form of somatostatin, may also be useful in aborting acute attacks of cluster headache.

If headaches persist and the diagnosis of cluster headache is not in question, a trial of lithium carbonate may be considered. The therapeutic window of lithium carbonate is small, however, and this drug should be used with caution. A starting dose of 300 mg at bedtime may be increased after 48 hours to 300 mg twice a day. If no side effects are noted after 48 hours, the dose may be increased again to 300 mg three times a day. The patient should stay at this dosage for a total of 10 days, after which the drug should be tapered over a 1-week period. Other medications that can be considered if these treatments are ineffective include methysergide and sumatriptan and sumatriptan-like drugs.

In rare patients, the aforementioned treatments are ineffective. In this setting, given the severity of the pain of cluster headache and the risk of suicide, more aggressive treatment is indicated. Destruction of the gasserian ganglion either by injection of glycerol or by radiofrequency lesioning may be a reasonable next step. Case studies suggest that deep brain stimulation may play a role in the treatment of intractable cluster headache.

COMPLICATIONS AND PITFALLS

The major risk in patients suffering from uncontrolled cluster headache is that they may become despondent owing to the unremitting, severe pain and commit suicide. Therefore if the clinician has difficulty controlling the patient's pain, hospitalization should be considered.

CLINICAL PEARLS

Cluster headache represents one of the most painful conditions encountered in clinical practice and must be viewed as a true pain emergency. In general, cluster headache is more difficult to treat than migraine headache and requires more individualized therapy. Given the severity of the pain associated with cluster headache, multiple modalities should be used early in the course of an episode of cluster headache. The clinician should beware of patients presenting with a classic history of cluster headache who request opioid analgesics.

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