The sensation of headache is rarely due to injury to the brain parenchyma itself. Rather, head pain results from tension, traction, distention, dilation, or inflammation of pain-sensitive structures external to the skull, portions of the dura mater, and blood vessels. Each of these mechanisms is probably mediated by a final common biochemical pathway that results in pain; therefore, a favorable response to analgesics should not be used to judge the cause of an individual headache.

**PATHOPHYSIOLOGY**

**PRESENTING SIGNS AND SYMPTOMS**

EPs should develop a logical, practical, and accurate approach to identification of patients with serious pathology. A comprehensive organizational scheme developed by the International Headache Society has recently been updated (Table 101.1); however, this scheme is cumbersome in emergency practice. For practical purposes, headaches can be divided into “benign” and “cannot miss” categories (Table 101.2).

Treatment of pain should occur in parallel with the history and physical examination. Appropriate analgesia is all that most patients require, and comfortable patients are more willing to undergo tests and procedures (e.g., lumbar puncture [LP]). Immediate pain control results in greater patient satisfaction and more rapid disposition. That said, a given patient’s response to analgesics should not alter the diagnostic strategy, so there is no reason to withhold treatment.

Evaluation should focus on signs and symptoms that can differentiate a benign headache from one requiring emergency work-up and treatment. For example, although location of the headache is often considered significant, unilateral headache is a hallmark of both primary (migraine, cluster) and secondary (intracerebral hemorrhage, glaucoma) headaches, thus limiting its usefulness in diagnosis. In contrast, fever and neck stiffness are uncommon with primary headache and are therefore very useful.

**TIMING AND DURATION**

Identifying the timing and duration of the headache is useful. Questions such as “What brings you here today rather than any other day?” can help focus patients on the timing of their symptoms. Worrisome features include a new acute headache...
or a subacute headache that is increasing in severity. An abrupt or “thunderclap” onset suggests intracranial hemorrhage or cerebral venous sinus thrombosis. If the maximum intensity of the pain occurred at the onset, aneurysmal bleeding should be considered. Very fleeting headaches, termed “jabs and jolts,” that last seconds are typically benign. In abrupt-onset headaches, the activity at the onset sometimes suggests the cause, such as with coital headache or benign exertional headache. However, even though a history of these activities can be a sensitive indicator of the corresponding diagnoses, specificity is poor. Therefore, subarachnoid hemorrhage cannot be excluded on the basis of activity before the headache.

LOCATION
Location of the pain is not very helpful in diagnosing headaches because of the significant overlap between benign and serious causes. Some recommend work-up of patients whose headaches always occur on the same side.

SEVERITY
Severity of the pain also has limitations in differentiating benign from serious headaches. Although the “worst-of-my-life” headache suggests a more serious problem, most severe headaches seen in the ED have benign causes. Patients without a previous history of similar, severe headaches should be evaluated for a secondary cause of the headache. In patients with a history of previous headaches, ask about details and consider evaluating those whose headaches are clearly increased in severity or different in quality.

QUALITY
The quality of the patient’s pain is critical. Most secondary headaches are qualitatively unique, unusual, or distinctly different from prior headaches. Diagnostic work-up of patients with chronic headaches that have new or unusual features should be strongly considered.

ASSOCIATED SYMPTOMS
Specific associated symptoms can provide important clues to a dangerous cause of the headache. Fever and neck stiffness suggest meningitis. Syncope, seizure, or any focal neurologic symptoms or new signs associated with a headache should prompt an evaluation. Diplopia suggests a mass, cerebral aneurysm, or elevated intracranial pressure (ICP).

Unfortunately, migraine headaches can produce an array of associated symptoms traditionally associated with secondary
headaches. Although nausea, vomiting, and photophobia can occur with increased ICP or infection, they are also associated with migraines. A helpful differentiating factor in migraineurs is whether these symptoms accompanied previous migraines or whether the finding is new. Visual abnormalities are associated with migraine headaches but also with idiopathic intracranial hypertension, temporal arteritis, and pituitary apoplexy.

EXACERBATING AND ALLEVIATING FACTORS
Determining exacerbating and alleviating factors is occasionally helpful. Post-LP headache tends to worsen on standing upright, and headache from sinusitis often worsens on bending forward with the head dependent. In contrast, the classic history of a headache caused by a brain tumor—worse on awakening—is neither specific nor sensitive because it is also seen in patients with hypercapnic chronic lung disease (in which the headache worsens during sleep). In terms of alleviating factors, diagnostic significance should not be ascribed to pain relief, even with over-the-counter medications.

OTHER FACTORS
Age is useful as a consideration because new-onset headache at older ages suggests a secondary cause such as giant cell arteritis, tumors, subdural hematoma, and side effects of medications. Environmental considerations include winter season and common-source clusters, which can indicate carbon monoxide poisoning.

PAST AND FAMILY HISTORY
Predisposing factors for a secondary cause of headache should be determined. For example, poorly treated hypertension may lead to hypertensive encephalopathy, vascular risk factors can result in stroke, and a past or family history of cerebral aneurysm increases the likelihood of subarachnoid hemorrhage. Environmental considerations include winter season and common-source clusters, which can indicate carbon monoxide poisoning.
patients with hemophilia are at higher risk for bleeding. Obesity suggests idiopathic intracranial hypertension (pseudotumor cerebri), especially in women. A history of cancer can raise suspicion for brain metastasis, and patients infected with human immunodeficiency virus or taking immunosuppressive medicines are at higher risk for infection.

**PHYSICAL EXAMINATION**

The physical examination (Box 101.2) is critical in guiding the differential diagnosis and appropriate work-up.

**GENERAL APPEARANCE AND VITAL SIGNS**

General appearance can be deceiving. For example, shielding one’s eyes from the light is seen with both migraine and meningeal irritation. Fever is not a symptom of migraine and suggests infection or a several-day-old subarachnoid hemorrhage. Hypertension suggests hypertensive encephalopathy, stroke, or other secondary causes but also can be due to pain or stress. The EP should have a low threshold for brain imaging (and possibly LP) in patients with headache and persistent hypertension. However, it should be remembered that patients with secondary headaches can appear well and neurologically intact.

**HEAD AND NECK**


**BOX 101.1 Critical Features of the History in the Emergency Department**

<table>
<thead>
<tr>
<th>Timing</th>
<th>Abrupt onset, “thunderclap” (pain rapidly reaches maximal intensity)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Location</td>
<td>Nonspecific with regard to the differential diagnosis</td>
</tr>
<tr>
<td>Severity</td>
<td>Worst of my life, most severe; have never been to an emergency department before for headache</td>
</tr>
<tr>
<td>Quality</td>
<td>New type, qualitatively different from previous headaches</td>
</tr>
<tr>
<td>Associated Symptoms</td>
<td>Fever, neck stiffness, Seizure, syncope, Focal neurologic complaints, Visual abnormalities (diplopia, decreased or altered vision)</td>
</tr>
<tr>
<td>Exacerbating or Alleviating Factors</td>
<td>Worse with cough or lying down suggests increased intracranial pressure (but is not specific)</td>
</tr>
<tr>
<td>Past Medical History</td>
<td>Stroke, vascular disease, cancer, Immunocompromised status, Hypercoaguable states or bleeding diathesis</td>
</tr>
<tr>
<td>Family History</td>
<td>Hypercoaguable states, bleeding diathesis, cerebral aneurysm</td>
</tr>
</tbody>
</table>

**BOX 101.2 Critical Features (Potential Diagnoses) of the Physical Examination**

| Vital Signs | Fever (meningitis, encephalitis, abscess), Elevated blood pressure (stroke or problems associated with elevated intracranial pressure) |
| Head | Vesicles on the scalp (herpes zoster of the upper two cervical roots or the root of the fifth trigeminal nerve), Tender temporal artery (giant cell arteritis), Tender sinuses (sinusitis) |
| Eyes | Red, edematous (acute angle–closure glaucoma), Proptosis (cavernous sinus thrombosis), Papilledema (increased intracranial pressure) |
| Nose | Vesicles on the tip of the nose (herpes zoster of the root of the fifth trigeminal nerve) |
| Neck | Meningismus with positive jolt accentuation, Kernig or Brudzinski sign (infection, subarachnoid hemorrhage) |
| Neurologic Examination | Change in mental status (increased intracranial pressure, infection, carbon monoxide poisoning), Decreased visual acuity (giant cell arteritis, acute angle–closure glaucoma), Visual field cut (mass lesion, pituitary apoplexy), Third nerve palsy (subarachnoid hemorrhage, cavernous sinus thrombosis), Sixth nerve palsy (increased or decreased intracranial pressure, basilar meningitis), Direction-changing nystagmus (cerebellar or brainstem stroke), Lower motor neuron seventh nerve palsy (Bell palsy, Ramsay Hunt syndrome), Eighth nerve palsy (diminished hearing or vertigo, Ramsay Hunt syndrome), Gait ataxia (cerebellar stroke), Any focal sensory or motor deficit (mass lesion, stroke) |
horizontally in alternate directions (as though rapidly shaking one’s head “no”) two to three times per second, the baseline headache increases in intensity.

**NEUROLOGIC EXAMINATION**

A complete neurologic examination of all patients with the chief complaint of headache should be performed and documented. Although some patients with migraine headaches may have neurologic deficits, the presence of new neurologic abnormalities should trigger a work-up beyond the history and physical examination. Abnormalities may suggest a diagnosis and the location of a mass lesion or cerebrovascular accident. Abnormal mental status with a new headache suggests increased ICP, a diffuse process such as meningitis, or carbon monoxide poisoning.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

Following the history and physical examination, the EP must determine whether further diagnostic testing is necessary. Patients with new abnormal findings on physical examination clearly need further evaluation. Similarly, patients with a reassuring history and normal physical examination findings may require only appropriate analgesia and follow-up arrangements. Diagnostic dilemmas usually arise with patients who have normal findings on physical examination but some worrisome aspect of the history. No well-studied and validated decision rules have been published; for the most part, experience, judgment, and careful attention to the clinical examination and differential diagnosis guide further testing.

**COMPUTED TOMOGRAPHY**

Computed tomography (CT) is often the first neuroimaging test because it is both rapid and widely available. A non-contrast-enhanced CT scan is extremely sensitive for acute intraparenchymal bleeding and very sensitive for subarachnoid bleeding (Fig. 101.1), but small or less acute subarachnoid bleeding may not be visible. Although some small tumors and abscesses are not visible on a non–contrast-enhanced scan, some abnormal finding will usually be seen on such scans in patients with masses large enough to cause a significant headache or focal neurologic findings (Fig. 101.2). Any focal neurologic signs or symptoms should be conveyed to the radiologist reading the CT scan so that appropriate attention can be directed to the anatomic site in question.

Which patients require CT scanning is a matter of some debate. Hard and fast rules do not exist, but in general, high-risk factors indicate the need for CT (Box 101.3). The American College of Emergency Physicians has a clinical policy about the use of CT in some situations.

The type of CT to perform depends on the specific differential diagnosis under consideration. Imaging of a mass or an

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**BOX 101.3 Indications for Computed Tomography**

<table>
<thead>
<tr>
<th>History</th>
</tr>
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<tbody>
<tr>
<td>New or qualitatively different type of headache, thunderclap headache</td>
</tr>
<tr>
<td>Hemophilia or other coagulopathy</td>
</tr>
<tr>
<td>Blunt trauma (especially in the elderly)</td>
</tr>
<tr>
<td>Immunocompromised status (human immunodeficiency virus infection, chemotherapy)</td>
</tr>
<tr>
<td>Elderly</td>
</tr>
<tr>
<td>Fever with neurologic findings</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Physical Examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glasgow Coma Scale score less than 15 with no clear explanation</td>
</tr>
<tr>
<td>Any new focal neurologic finding</td>
</tr>
<tr>
<td>Signs of increased intracranial pressure</td>
</tr>
</tbody>
</table>

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![Fig. 101.1 Subarachnoid hemorrhage.](image1)

![Fig. 101.2 Subdural hematoma.](image2)
abscess can be improved with intravenous infusion of a contrast agent. CT angiography (CTA) can be performed with multidetector scanners. Depending on the number of detectors, the software, and the skill of the neuroradiologist, CTA can approach conventional angiography in direct visualization of the cerebral vasculature. For patients in whom an arteriovenous malformation or aneurysm is suspected, CTA is a useful modality, although the standard diagnostic algorithm is still CT followed by LP.\textsuperscript{2}–\textsuperscript{4} CT venography can be useful in the diagnosis of cerebral venous sinus thrombosis.

**MAGNETIC RESONANCE IMAGING**

In general, magnetic resonance imaging (MRI) is superior to CT, especially in evaluating vascular and neoplastic lesions and infections and pathology at the cervicomedullary junction and in the posterior fossa. Brain tumors, abscesses, ischemia, and pituitary apoplexy are easily visible. Recent studies suggest that MRI may even identify small cerebral hemorrhages that are not detected by CT scanning. Arterial and venous blood vessels can be evaluated by magnetic resonance angiography. Carotid and vertebral artery dissections, cerebral aneurysms, and cerebral venous sinus thrombosis can be diagnosed with magnetic resonance angiography and magnetic resonance venography.

Given the expense and scarcity of this resource, it is important that the EP carefully evaluate its necessity. For example, a headache patient with known thrombophilia or signs of cerebellar ischemia may require MRI evaluation for venous sinus thrombosis or a cerebellar stroke. Similarly, any newly documented neurologic deficit must be explained, and if contrast-enhanced CT scanning is not sufficient to find the cause, the EP should consider arranging for MRI, with the urgency depending on the clinical situation.

**LUMBAR PUNCTURE**

LP remains an important diagnostic tool for headache patients. In patients with subarachnoid hemorrhage, the EP must be aware that CT scanning may be nondiagnostic and that LP is the next step necessary.\textsuperscript{2} LP can establish the diagnosis of suspected meningitis with nearly 100% sensitivity. Elevated opening pressure suggests idiopathic intracranial hypertension or cerebral venous sinus thrombosis. However, as with all tests in medicine, even LP has limitations. In particular, patients taking prednisone, who are otherwise immunocompromised, may not have the elevated cerebrospinal fluid white blood cell count expected. LP to rule out subarachnoid hemorrhage can be traumatic as well, thus making interpretation difficult. In such cases, an elevated opening pressure may help identify pathology such as subarachnoid hemorrhage.

**LABORATORY STUDIES**

Routine laboratory studies are rarely helpful and not recommended in the work-up of patients with headache. In a few very specific circumstances, targeted laboratory tests can be helpful. Such tests include an erythrocyte sedimentation rate if giant cell arteritis is suspected, a toxicology screen for cocaine or other sympathomimetics in cases of intracranial hemorrhage in which they might play a role, and a carboxyhemoglobin level in patients with suspected carbon monoxide poisoning.

**TONOMETRY**

Tonometry is a critical test in the ED for patients with headache, eye complaints, and findings that suggest acute narrow angle–closure glaucoma.

**TEMPORAL ARTERY BIOPSY**

Temporal artery biopsy helps diagnose giant cell arteritis. When this diagnosis is suspected, high-dose oral steroid therapy should be started and arrangements made for the patient to see a surgeon for biopsy within the next week or so.

**NEUROLOGY CONSULTATION**

One final issue to be considered is neurology consultation. The timing of consultation can vary, depending on the differential diagnosis and the duration of the headache. In patients with a new, definite neurologic finding on physical examination and normal results on brain imaging, the physician should consider neurologic consultation on an urgent or emergency basis.

**TREATMENT**

Airway protection is always paramount in a critically ill patient. Patients with impending herniation from a mass lesion or intracranial bleeding may require intubation. Although neurologic examination is important in the acute phase of the patient’s hospitalization, short-term paralysis for rapid-sequence intubation can and should be used to achieve the optimal intubation conditions. Lidocaine and fentanyl are sometimes advocated to blunt the transient rise in ICP that accompanies tracheal intubation. If feasible, a quick neurologic examination should be performed first.

Once the airway is secure, sedation should be adequate (to avoid elevations in ICP), but oversedation should be avoided to provide the best possible serial neurologic examination (propofol is a short-acting sedative that is useful for this purpose). If airway management precedes imaging, emergency neuroimaging should rapidly follow intubation.

For patients with signs or symptoms of acute bacterial meningitis, a critical early decision is whether to perform a CT scan or proceed directly to LP (Box 101.4). Administration of antibiotics should not be delayed in patients who have signs of acute meningitis. Performing LP directly in an alert, neurologically intact patient with no medical history is usually safe, especially in those with normal venous pulsations on funduscropy.
**Giant Cell Arteritis (Temporal Arteritis)**

Giant cell arteritis is classically manifested as a sudden, severe temporal headache in patients older than 50 years. A major differentiating feature of this disease is the presence of ischemic symptoms such as jaw claudication, scalp tenderness, or visual loss. In the ED, giant cell arteritis is a clinical presumptive diagnosis; therefore, high-dose prednisone should be administered empirically immediately. Temporal artery biopsy should be scheduled within a week or so to establish a definitive diagnosis, although even a negative biopsy result does not definitively exclude this disorder. The decision whether to continue steroid therapy following negative biopsy findings should be made by the primary care physician in consultation with appropriate specialists.

**Acute Angle-Closure Glaucoma**

Patients with acute angle-closure glaucoma typically have a recurrent unilateral headache behind the eye associated with blurred vision and erythema; there will often be a prolonged course of symptoms before appropriate diagnosis. Although the diagnosis is usually based on clinical findings, tonometry can establish the condition. Intraocular pressure in the affected eye will be elevated up to 40 to 80 mm Hg. In addition to appropriate pain control, any or all of the following therapies may be considered:

1. Acetazolamide, 500 mg intravenously followed by 500 mg orally
2. Timolol, 0.25% to 0.5% applied topically
3. Prednisolone, 1 to 2 drops onto the affected eye
4. Pilocarpine, 2% applied topically
5. Isosorbide, 1.5 g/kg orally, or glycerin, 1 to 2 g/kg orally
6. Mannitol, 1.5 to 2 g/kg intravenously
7. Antiemetics and analgesia as needed
8. Anterior chamber paracentesis

An ophthalmologist should be consulted on an emergency basis for definitive peripheral iridectomy or laser iridotomy.

**Sinusitis-Related Headache**

A diagnosis of sinus-related headache should be made with caution because paranasal sinus mucosal thickening is a common incidental finding on CT and does not imply causality. Pain control is the cornerstone of treatment. Nonsteroidal agents and decongestants can be provided. Oxymetazoline nasal spray should be used for no more than several days. The Centers for Disease Control and Prevention recommends that the diagnosis of bacterial sinusitis be made only after 7 days of symptoms and that amoxicillin should be a first-line agent for mild sinusitis in patients with no previous antibiotic use.

**Migraine Headache**

Migraine headache is classically defined as a throbbing unilateral headache with associated symptoms, including photophobia, phonophobia, nausea, and vomiting. The headache can be preceded by an aura, such as scintillating scotomata, jagged lines, or other visual abnormalities. Neurologic signs, including hemiparesis, paresthesias, ophthalmoplegia, and aphasia, can complicate migraine. Patients will usually have a history of similar headaches and frequently a family history of migraines.

Migraine is most confidently diagnosed by a history of at least five similar headaches with several specific criteria. New-onset headaches or those of a different or unusual quality often require further work-up. One cannot definitively...
TENSION HEADACHE

Tension headache is usually characterized by throbbing pain that radiates bilaterally from front to back and to the neck muscles. As with migraine headaches, one cannot firmly diagnose tension headache after a single episode, and this diagnosis requires more than nine previous episodes. Pain control consists of nonsteroidal antiinflammatory drugs, antiemetics, and perhaps caffeine (Box 101.7). Butalbital-containing agents (as with migraine headaches) may be used with caution, given the risk for dependency and rebound headache.

Disposition is entirely a function of the cause of the headache and the need for further treatment or, in some cases, pain control.

Box 101.5 summarizes the various agents that are useful in the management of acute migraine. Although opiates are used frequently in the ED, they should not be first-line treatment and should be reserved for rescue therapy in patients who do not respond to the initial medications—and even in this situation they should be used sparingly.

CLUSTER HEADACHE

Cluster headache is typically a severe unilateral headache that can be accompanied by conjunctival injection, lacrimation, ptosis, miosis, rhinorrhea, and nasal congestion. Attacks can occur up to eight times a day and are severe but short-lived; the autonomic symptoms are typically unilateral and ipsilateral to the pain. Recognition is important because this headache subtype is uniquely sensitive to oxygen. Mainstays of emergency management include administration of oxygen and subcutaneous sumatriptan (Box 101.6).
Complications and Pitfalls
Just because a patient states that he or she has had “migraine” (or “tension” or “sinus”) headaches does not mean that the headaches ever formally met these criteria or have ever been evaluated or that this headache is the same as the previous headaches. This is especially true for recent-onset headaches.

If head computed tomography is performed to evaluate for subarachnoid hemorrhage, it should always be followed by lumbar puncture to evaluate for xanthochromia and the presence of blood.

Visualization of mucosal thickening in the paranasal sinuses on computed tomography is a common incidental finding and should not be used to diagnose acute sinusitis.

A favorable response to analgesics has little or no diagnostic significance and should not be used to exclude a secondary cause of headache.

REFERENCES AND SUGGESTED READINGS
References and suggested readings can be found on Expert Consult @ www.expertconsult.com.
SUGGESTED READINGS

De Luca GC, Barlleson JD. When and how to investigate the patient with headache. Semin Neurol 2010;30:131-44.
Edlow JA. What are the unintended consequences of changing the diagnostic paradigm for subarachnoid hemorrhage after brain computed tomography to computed tomographic angiography in place of lumbar puncture? Acad Emerg Med 2010;17:991-5; discussion 996-997.
McCormack RF, Hutson A. Can computed tomography angiography of the brain replace lumbar puncture in the evaluation of acute-onset headache after a negative noncontrast cranial computed tomography scan? Acad Emerg Med 2010;17:444-51.
REFERENCES

3. Edlow JA. What are the unintended consequences of changing the diagnostic paradigm for subarachnoid hemorrhage after brain computed tomography to computed tomographic angiography in place of lumbar puncture? Acad Emerg Med 2010;17:991-5; discussion 996-997.