

occlusion. These cases can occur in hypercoagulable states, including peripartum, in association with the combined oral contraceptive pill, and in association with antiphospholipid antibody syndrome. After secondary causes of IHH have been eliminated, the patient should have dietary counseling for weight loss. Carbonic anhydrase inhibitors (acetazolamide) and corticosteroids have proved useful in headache control. As a second-line agent, furosemide also acts to lower CSF production. Serial lumbar punctures are understandably unpopular with patients even though transient headache relief is obtained. CSF shunting procedures (ventriculoperitoneal shunt) are occasionally necessary. For patients with progressive visual loss, optic nerve sheath fenestration preserves or restores vision in 80% to 90% of cases and provides headache relief in a majority.

Idiopathic Intracranial Hypotension

Also known as *low pressure headache*, idiopathic intracranial hypotension is commonly encountered as a sequela of lumbar puncture, resulting from leakage of CSF through the dural sac. Low pressure headaches may also occur spontaneously as a result of rupture of subarachnoid cysts. The headache is initially characteristically positional, being severe on standing but relieved rapidly on lying down. Occasionally the headache is associated with focal or “false localizing” signs, especially abducens nerve palsies.

Post-Traumatic Headache

Headache following trauma has no specific quality and is associated with irritability, concentration impairment, insomnia, memory disturbance, and light-headedness. Anxiety and depression are present to variable degrees. Multiple treatment options are available, and amitriptyline and nonsteroidal anti-inflammatory agents are useful. Occasionally, muscle relaxants and anxiolytics are beneficial. Training is occasionally associated with the onset of typical migraine.

Giant Cell Arteritis

Headache occurs in 60% of patients with giant cell arteritis, a granulomatous vasculitis of medium and large arteries. Over 95% of patients are 50 years of age or older. Malaise, fever, weight loss, and jaw claudication occur early, in addition to headache. Poly-myalgia rheumatica, a syndrome of painful stiffness of the neck, shoulders, and pelvis, is found in half the patients (Chapter 131). Visual impairment secondary to ischemic optic neuritis may occur. The headache is usually described as aching and is exacerbated at night and after exposure to cold. The superficial temporal artery is frequently swollen, tender, and may be pulseless. The erythrocyte sedimentation rate is usually elevated; the mean is 100 mm/hr. Anemia is frequently present. Temporal artery biopsy usually confirms the diagnosis, but, because the arteritis is segmental, large or multiple sections may be required. Prednisone therapy is often dramatically effective and must be given promptly to preserve vision on the affected side.

Evaluation of the patient with acute headache

It is important to distinguish benign from ominous causes of headache. A detailed history (the quality, location, duration, and

time course of the headache) helps in determination of which patients have a symptomatic structural intracranial lesion (Table 111-7; Table 111-8; Table 111-4)). Pain intensity is not of much diagnostic value, except for the patient who complains of the acute onset of the worst headache of his or her life). The quality of pain (“throbbing,” “pressure,” “jabbing”) and the location may also be helpful, especially if the pain is of extracranial origin, such as temporal in temporal arteritis. Posterior fossa lesions cause occipitocervical pain, occasionally associated with unilateral retro-orbital pain. In general, multifocal pain usually implies a benign cause. It is most important to clarify the acuity of onset of the headache; patients who describe the onset of pain as “like being hit on the head with a bat” should be suspected of having the sentinel headache of subarachnoid headache. Equally important is to establish the time course of the headache. Is this paroxysmal, nonprogressive headache (typical of migraine or tension-type headache)? Or is the headache daily persistent (such as in temporal arteritis) or progressive (suggesting the presence of a structural brain lesion)? Patients should be asked about any known triggers for the headache, such as menses, particular foods, caffeine, alcohol, or stress. Positional headache (headache that is maximal in the upright position and disappears rapidly on lying down) is characteristic of intracranial hypotension (low pressure headache). Diurnal variation in headache severity may give a clue to cause; morning headache or headache that awakens a patient from sleep may indicate raised intracranial pressure or sleep apnea as a cause. The presence of associated symptoms such as visual disturbances, nausea, or vomiting should be noted. The history should include inquiries about medications, especially use of analgesics and over-the-counter remedies. Information regarding the patient’s past medical history as well as family history should also be taken into consideration. In the majority of patients with headache, the physical and neurologic examination findings are normal, although special attention may be directed toward examination of the eyes for papilledema, as well as the temporal arteries for a palpable nonpulsatile artery. Assessment of the patient with acute nontraumatic headache in the emergency room can be challenging; it is essential to establish how the headache evolved. Acute-onset severe headache should prompt investigation to exclude SAH, intracranial hemorrhage, acute obstructive hydrocephalus, and meningitis (Table 111-7). Appropriate initial investigations should include brain imaging with CT or magnetic resonance imaging (MRI). Patients with suspected meningitis without focal neurologic signs or impaired consciousness should not have their lumbar puncture delayed unnecessarily before imaging. All patients should have standard

TABLE 111-8 CLINICAL FEATURES OF HEADACHES SUGGESTING A STRUCTURAL BRAIN LESION

SYMPTOMS	SIGNS
Worst of the patient’s life	Nuchal rigidity
Progressive	Fever
Onset >50 years of age	Papilledema
Worse in early morning—awakens patient	Pathologic reflexes or reflex asymmetry
Marked exacerbation with straining	Altered state of consciousness
Focal neurologic dysfunction	