12

Headaches over the Age of 50

Randolph W. Evans

The prevalence of headache decreases with older age (Table 12-1) (1). Although 90% of headaches in younger patients are of the primary type, only 66% of those in the elderly are primary (2).

There are numerous causes of new-onset headaches in those over 50 years of age (Table 12-2) (3). Although new-onset tension-type headaches are fairly common, migraine and cluster-type headaches uncommonly begin after 50 years of age. Temporal arteritis, hypnic headache, and headache of Parkinson's disease are secondary headaches occurring with much greater frequency in this population.

Causes of secondary headache disorders with onset or more common in later life include the following: neoplasms; subdural and epidural hematomas; head trauma; cerebrovascular disease; temporal arteritis; trigeminal neuralgia; postherpetic neuralgia; medication-related headache, including those caused by specific medications and medication rebound; systemic disease, such as infections, acute hypertension, hypoxia, or hypercarbia; and other metabolic disorders, such as hypercalcemia, severe anemia, hyponatremia, and chronic renal failure; diseases of the cranium, neck, eyes, ears, and nose, including cervicogenic headache, glaucoma, otitis, sinusitis, and dental infections; Parkinson's disease; and angina (also see Chapter 13), which may rarely present with exertional headache without chest pain.

In a study of 193 patients 65 years of age and over seen by a neurology service with new-onset headaches, the most frequent diagnoses were tension type (43%) and trigeminal neuralgia (19%) (4). Only one patient met migraine criteria. Fifteen percent had secondary headaches due to conditions such as stroke, temporal arteritis, or intracranial neoplasm. The risk of serious disorders causing headache increased 10 times after age 65, compared with younger patients.

This chapter reviews the primary headaches and some of the secondary headaches, including temporal arteritis, postherpetic neuralgia, and Parkinson's disease. The other secondary headaches are covered in Chapters 6 (medication related), 9 (subdural and epidural hematomas), 14, and 15 (trigeminal neuralgia).

PRIMARY HEADACHES

Migraine

Only 2% of migraineurs have the new onset after 50 years of age. Migraine prevalence decreases with older age. The prevalence is about 5% in women and 2% in men past 70 years of age. When migraine criteria are met, computed tomography (CT) or magnetic resonance imaging (MRI) scans have a very low yield in this population (5).

Medication use presents a variety of problems in older patients. Ergotamine, dihydroergotamine (DHE), and triptans should not

Table 12-1. Prevalence of headaches at various ages

Age	Women	Men
21–34	92%	74%
55–74	66%	53%
75+	55%	22%

Table 12-2. New-onset headaches occurring over 50 years of age

Primary headaches
Migraine
Tension
Cluster
Hypnic
Secondary headaches
Neoplasms
Subdural and epidural hematomas
Head trauma
Cerebrovascular disease
Temporal arteritis
Trigeminal neuralgia
Postherpetic neuralgia
Mediation related
Systemic disease
Diseases of the cranium, neck, eyes, ears, and nose
Parkinson's disease
Exertional headache due to angina

be used in patients with coronary artery disease, cerebrovascular disease, or peripheral vascular disease. Some patients without such a history may require screening, especially if they have risk factors such as diabetes, a positive family history, smoking, or hyperlipidemia. Older patients may be more sensitive to anticholinergic, hypotensive, sedative, cognitive, and cardiac side effects of preventive medications. For example, patients with prostatism, glaucoma, or cardiac arrhythmias may need to avoid tricyclic antidepressants, whereas those with congestive heart failure, diabetes, or bronchial asthma may need to avoid beta-blockers. Drugs used for other indications, such as estrogen replacement therapy or nitrates, may trigger migraines. A variety of other medications can cause headaches, including nifedipine, phosphodiesterase inhibitors for erectile dysfunction, and nonsteroidal anti-inflammatory drugs (NSAIDs) (also see Chapter 3).

Late-Life Migrainous Accompaniments

Fisher described late-life migrainous accompaniments (6,7), which are transient visual, sensory, motor, or behavioral neurological manifestations that are similar or identical to the auras of migraine with aura (8). Headache is associated with only 50%

Table 12-3. Late-life migrainous accompaniments

1. Gradual appearance of focal neurological symptoms with spread or worsening over a period of minutes.
2. Headache is only present in 50% of cases and may be mild.
3. Positive visual symptoms such as scintillating scotoma, flashing or bright lights.
4. A history of similar episodes associated with a more severe headache.
5. Serial progression from one accompaniment to another (e.g., from flashing lights to paresthesias, paresis, or dysphasia).
6. Diagnosis facilitated with the occurrence of two or more identical episodes.
7. A duration of 15 to 25 minutes.
8. A characteristic "flurry" of accompaniments.
9. A usually benign natural history, without permanent sequelae.
10. Another cause not shown by diagnostic testing that is performed when indicated.

of cases and may be mild. These accompaniments occur more often in men than in women with a prevalence of about 1%.

Table 12-3 provides the features of this disorder. The complaints occur as follows, from most to least common: visual symptoms (transient blindness, homonymous hemianopsia, and blurring of vision); paresthesias (numbness, tingling, pins-and-needles sensation, or a heavy feeling of an extremity); brain stem and cerebellar dysfunction (ataxia, clumsiness, hearing loss, tinnitus, vertigo, and syncope); and disturbances of speech (dysarthria or dysphasia).

Other causes of transient cerebral ischemia should be considered, especially when the patient is seen after the first episode or if there are unusual aspects. The usual diagnostic evaluation for transient ischemic attacks (TIAs) or seizures (such as CT scan, MRI and magnetic resonance angiography of the brain, carotid ultrasound, electroencephalography, cardiac evaluation, and blood studies) is performed.

The following features help distinguish migraine from TIAs: a gradual buildup of sensory symptoms, a march of sensory paresthesias, serial progression from one accompaniment to another, longer duration (90% of TIAs last for less than 15 minutes), and multiple stereotypical episodes.

If the episodes are frequent, preventive treatment can be considered with medications such as topiramate, divalproex sodium, and verapamil. Beta-blockers should be avoided because of the potential for worsening vasospasm. For acute treatment, ergotamine, DHE, and triptans should be avoided because of the risk of increasing cerebral vasospasm.

Tension-Type Headaches

About 10% of those with tension-type headaches have an onset after 50 years of age. When new-onset tension-type headaches occur, the diagnosis is one of exclusion. Over the age of 65 years,

the prevalence of tension-type headaches is about 27%. The patient and physician should be aware of the potential for medication-overuse headaches.

Medications for tension-type headaches are the same as those for younger people. It is often prudent to start with lower doses and to be cautious about an increased susceptibility to side effects in the elderly. Physical therapy can be helpful for cervicogenic headache.

Chronic Daily Headache

About 4% of the population age 65 years or older has chronic daily headache with a higher prevalence in women than in men. Medication overuse is a concern as a cause of rebound headache just as in younger patients.

Cluster Headaches

Cluster headaches are a rare disorder with a 5:1 male-to-female preponderance. Although the age of onset is typically between the ages of 20 and 50 years, onset can occur in the 70s.

Hypnic Headache

Hypnic headache is a rare disorder originally described by Raskin in 1988 (9), which has since been reported in men and women from the ages of 36 to 83 years. The headache only occurs during sleep when the sufferer is awakened at a consistent time. Nausea is infrequent and autonomic symptoms are rarely associated. The headache can be unilateral or bilateral, throbbing or nonthrobbing, and mild to severe in intensity. The headache can last 15 minutes to 6 hours and can occur frequently, as often as nightly, for many years. Medications that may be effective include caffeine (1 or 2 cups of caffeinated coffee or a 40- to 60-mg caffeine tablet before bedtime), lithium carbonate (300 mg at bedtime), indomethacin, atenolol, cyclobenzaprine, melatonin (6 mg at bedtime), and flunarizine (not available in the United States) (10,11).

The diagnosis is one of exclusion because secondary causes of nocturnal headaches include drug withdrawal, temporal arteritis, noctural headache-hypertension syndrome, sleep apnea, oxygen desaturation, pheochromocytomas, primary and secondary neoplasms, communicating hydrocephalus, subdural hematomas, and vascular lesions (12).

Migraine, cluster, and chronic paroxysmal hemicrania are other primary headaches that can cause awakening from sleep. Migraine typically has associated symptoms and occurs very uncommonly only during sleep. Cluster headaches have autonomic symptoms and may occur during the day as well as during sleep. Chronic paroxysmal hemicrania occurs both during the day and at night, lasts for less than 30 minutes, and occurs 10 to 30 times a day.

SECONDARY HEADACHES

Temporal Arteritis

Epidemiology

Temporal (giant cell) arteritis (TA) is a systemic panarteritis that selectively involves arterial walls with significant amounts of elastin. Approximately 50% of patients with TA have polymyalgia

rheumatica, and about 15% of patients with polymyalgia rheumatica have TA. Both conditions occur almost exclusively in patients over the age of 50, with a mean age of onset of about 70. The ratio of women to men is 3:1. The disorder is more common in Scandinavia and the northern United States, and it is more common in whites than in other ethnic groups. The annual incidence is about 18 in 100,000 over 50 years of age.

Clinical Manifestations (13,14)

Headaches are the most common symptom reported by 60% to 90% of patients. The pain is most often throbbing, although many patients describe a sharp, dull, burning, or lancinating type of pain. The pain may be intermittent or continuous and is more often severe than moderate or slight. For some patients, the pain may be worse at night when lying on a pillow, while combing the hair, or when washing the face. Tenderness or decreased pulsation of the superficial temporal arteries is present on physical examination in about half of the patients with TA.

The location of the headache is variable. In one series, 65% of patients presented with temporofrontal headache (15). In another series, the following percentages of patients reported the distribution of pain in these categories: 25%, only the temple; 54%, the temple, either exclusively or inclusively; 29%, not involving the temple at all; and 8%, generalized (16). When headaches were limited to or included the temple, the headaches were bilateral in 50% of the patients. Intermittent jaw claudication was reported by 38%. Other studies have reported one-sided pain in 2%; 8% reported pain affecting the face or neck.

Temporal arteritis can present as occipital neuralgia. In a study of 46 patients with biopsy-proven TA, 17% of the patients had an initial presentation with occipital pain, which was unilateral in 38% (17). Two of the patients with unilateral pain exactly similar to greater occipital neuralgia had normal sedimentation rates but abnormal superficial temporal artery biopsies. Tenderness over the greater occipital nerve can be explained by inflammation of the occipital artery, which is adjacent to the greater occipital nerve in the suboccipital region. TA should be considered in patients over 50 years of age who present with new-onset "occipital neuralgia," even with normal sedimentation rates, especially when they do not respond to the usual treatments.

Neurological manifestations of TA are common. One series found evidence of neurological disease in 31% of the patients, including ophthalmological findings, 20%; mononeuropathies and peripheral neuropathies, 14%; carotid distribution transient ischemic events or stroke, 7%; vertebrobasilar distribution transient ischemic events or stroke, 2%; otological findings, 7%; tremor, 4%; psychiatric findings, 3%; tongue numbness, 2%; and transverse myelopathy, less than 1% (18). Depression and confusion can be associated with temporal arteritis.

There is a broad spectrum of neuroophthalmological manifestations of TA (19). Visual loss may occur because of anterior and posterior ischemic optic neuropathy; central and branch retinal artery occlusion; anterior segment ischemia; and prechiasmal, perichiasmal, and postchiasmal field defects. Ophthalmoparesis

Table 12-4. Criteria for the diagnosis of TA of the American College of Rheumatology

Three out of the following five criteria should be satisfied:
1. Age at least 50 years
2. New onset of localized headache
3. Temporal artery tenderness or decreased pulse
4. Erythrocyte sedimentation rate of at least 50 mm/h
5. Positive histology

can be due to the following: oculomotor, abducens, and trochlear nerve palsies; orbital constriction resulting from orbital cellulitis and cavernous sinus thrombosis; and oculomotor synkinesis. Autonomic dysfunction may be caused by Horner's syndrome and parasympathetic pupillary light dysfunction/near dissociation. Rarely, complex visual hallucinations occur after infarction of the tertiary visual association cortex.

Diagnostic Evaluation

According to the American College of Rheumatology 1990 criteria, the diagnosis of TA can be established by fulfilling three out of five criteria (Table 12-4) (20). The presence of three or more of the five criteria is associated with a sensitivity of 93.5% and a specificity of 91.2%.

The diagnosis is based on clinical suspicion that is usually but not always confirmed by laboratory testing (21,22). The three best tests are the Westergren erythrocyte sedimentation rate (ESR), the C-reactive protein (CRP), and temporal artery biopsy. Elevation of plasma viscosity has a sensitivity and specificity similar to that of the ESR. Mild normochromic normocytic anemia, elevation of liver enzymes (especially alkaline phosphatase), and decreased alpha$_2$-globulin levels are fairly common.

Color duplex ultrasonography of the superficial temporal arteries may show a dark halo around the lumen of the superficial temporal arteries due to edema of the artery wall (23). However, the ultrasound may only modestly improve the probability of biopsy-proven TA and not improve the diagnostic accuracy of a careful physical examination (24).

For elderly patients, the ESR range of normal may vary from less than 20 mm/hour to 40 mm/hour. A formula for the upper limits of normal for the ESR that includes 98% of healthy people is as follows: age in years divided by 2 for men and age in years plus 10 divided by 2 for women (25). Elevation of the ESR is not specific for TA. Elevation can be seen in any infectious, inflammatory, or rheumatic disease. The level can even be affected by the length of time between the venipuncture and the laboratory testing (26). TA with a normal ESR has been reported in 10% to 36% of patients. Repeating the ESR may be helpful in some cases in which the ESR is initially normal and then rises. When abnormal, the ESR averages 70 to 80 and may reach 120 or even 130 mm/hour. When the ESR is elevated at the time of diagnosis, it can be followed to help guide the dosage of corticosteroid medication.

CRP is an acute-phase plasma protein from the liver. As with the ESR, elevation is nonspecific and can be seen with numerous disorders. The CRP is not influenced by various hematological factors or age and is more sensitive than the ESR for the detection of TA. The ESR and CRP combined give the best specificity, 97%. Levels of interleukin-6 are also a sensitive indicator of active disease.

The diagnosis is made with certainty when the superficial temporal artery biopsy demonstrates necrotizing arteritis characterized by a predominance of mononuclear cell infiltrates or a granulomatous process with multinucleated giant cells. The false-negative rate of temporal artery biopsies in various series ranges from 5% to 44%. Biopsy-negative cases may have a more benign course than biopsy-positive cases (27).

Possible reasons for negative temporal artery biopsies include noncontinuous pathological findings or skip lesions, choice of site and length of the biopsy, examination of an incomplete number of sections, involvement of other vascular territories, and initiation of corticosteroid therapy prior to the biopsy. When the biopsy is negative, a biopsy of the contralateral superficial temporal artery increases the positive yield by 5% to 15%. Pathological evidence of TA persists for at least 4 to 5 days after the start of corticosteroid treatment.

Because temporal artery biopsy is a simple, low-risk procedure, a case can be made for obtaining a biopsy in every suspected patient. However, when three or four of the American College of Rheumatology criteria are met (Table 12-4), a strong argument can be made for treatment without biopsy. The result of the CRP and/or color duplex ultrasonography may also influence the decision.

In patients in which the clinical presentation is somewhat suspicious or when there is a very high probability of corticosteroid side effects (e.g., a type I diabetic), a biopsy should be obtained. In the occasional patient with a normal or only slightly elevated ESR and a negative biopsy, the same consideration may apply in a decision to biopsy the contralateral artery.

Management

When contraindications are not present, treatment is typically started with prednisone at a dosage of 40 to 80 mg/day or 1 mg/kg/day (28,29). The headache will often improve within 24 hours. The initial dose is maintained for about 4 weeks and then slowly reduced over many months by a maximum of 10% to 20% of the total daily dose each week or every 2 weeks, depending on the clinical effect, the ESR, and occurrence of side effects. Because TA is active for at least 1 year and an average of 3 to 4 years in some series, long-term treatment is usually required. About 30% to 50% of patients have an exacerbation, especially during the first 2 years of treatment, independent of the corticosteroid regimen. There are numerous complications of long-term steroid treatment. Calcium and vitamin D supplementation should be given along with corticosteroids to help prevent osteoporosis. In patients with reduced bone mineral density, bisphosponates are also indicated. Adjunctive use of methotrexate with corticosteroids is not beneficial to control disease activity or

to decrease the cumulative dose and toxicity of corticosteroids (30). Infliximab (antitumor necrosis factor onoclonal antibody) might be effective in patients unresponsive to or intolerant of corticosteroids.

Postherpetic Neuralgia (31,32)

Epidemiology

Acute herpes zoster occurs when the dormant varicella zoster virus (from a previous chickenpox infection) is reactivated in the trigeminal, geniculate, or dorsal root ganglion. The annual incidence of acute herpes zoster is approximately 400 in 100,000. The incidence dramatically increases with older age. According to various studies, the incidence ranges from 40 to 160 in 100,000 for those under 20 years of age to 450 to 1,100 in 100,000 for those 80 years of age or older. There are more than a million cases per year in the United States, most in the elderly. The lifetime risk of developing acute herpes zoster for those who live into their 70s and 80s is as high as 40%. The lifetime risk of a second or third attack in healthy people is about 5%.

Postherpetic neuralgia (PHN) is the most common neurological complication of varicella zoster infection and occurs in about 10% to 15% of those with acute zoster. PHN develops in some 50% of those older than 50 years of age and in 80% of those older than 80. Up to 200,000 people in the United States have PHN, which can persist for years. Zoster involving the face nearly doubles the risk of developing PHN, which lasts longer than PHN in other locations.

Clinical Manifestations

Radicular pain is the most common complication of zoster and may precede the eruption of grouped vesicles (shingles) by days to weeks. Zoster occurs in a trigeminal distribution, usually in the ophthalmic division, in 23% of cases. Uncommonly, an extraocular muscle paresis may be associated as a result of involvement of the third, fourth, or sixth cranial nerves. Reactivation of virus in the geniculate ganglion can result in vesicles in the external auditory canal and a facial palsy known by the eponym of Ramsay-Hunt syndrome. Occasionally, pain occurs without a rash (zoster sine herpete). The pain is usually sharp or stabbing. Typically, the vesicles crust, the skin heals, and the pain resolves within 3 to 4 weeks of the onset of the rash. However, in many people the pain can persist.

Postherpetic neuralgia is the persistence of pain after the initial rash for more than 1 to 6 months. (There are different opinions about the definition in the literature.) The involvement of the head is typically unilateral in the distribution of the ophthalmic or maxillary divisions of the trigeminal nerve or at the occipitocervical junction. Three types of pain may be present: a constant burning or deep aching; an intermittent spontaneous pain with a jabbing or lancinating quality; and a superficial, sharp, or radiating pain or itching provoked by light touch (allodynia). The types of pains vary from person to person. Allodynia is present in 90% of individuals with PHN. The pain often interferes with sleep.

Management

For the treatment of acute zoster, oral corticosteroids (prednisone starting at 60 mg/day and tapering off over 2 weeks) may reduce acute pain but may not reduce the risk of PHN. Corticosteroids should be used in combination with antiviral medication. Use of oral acyclovir (800 mg every 4 hours, 5 times daily, for 7 to 10 days) may decrease the acute pain but only modestly decreases the risk of PHN. Famciclovir (33) (500 mg every 8 hours for 1 week) and valacyclovir (1 g every 8 hours for 1 week) are more effective in reducing the incidence and duration of PHN. (The doses of both drugs are reduced in renal insufficiency.) The earlier antiviral therapy is initiated the greater the likelihood of response because in the clinical trials, antivirals were started within 72 hours of the onset of skin lesions. Although nerve blocks are a highly effective treatment for acute pain, it is uncertain if this approach will reduce the risk of PHN.

A variety of treatments are available for PHN with varying efficacies (34). Tricyclic antidepressants, including amitriptyline, nortriptyline, and desipramine, are effective treatments. Start with a low dose and then slowly increase to an optimal dose with the most pain relief and tolerable side effects. Up to 61% of patients may have pain relief with tolerable side effects. For those who can not tolerate tricyclics, venlafaxine may be more tolerable and may also be effective. Some patients may benefit from the addition of a phenothiazine medication such as fluphenazine. Gabapentin (35) and pregabalin are also effective in the treatment of pain and sleep interference associated with PHN. Anecdotally, other antidepressants such as fluoxetine may also be effective. Keppra may also be effective.

Topical agents, including capsaicin, lidocaine 5%, aspirin, and NSAIDs, may be useful. The 0.075% capsaicin cream may be more effective than lower concentrations. Some patients may have burning pain on application of the cream, which limits use.

Opioids such as sustained-release oxycodone (10 mg every 12 hours, slowly increasing as necessary to 30 mg every 12 hours) (36) and oral levorphanol may be effective when other drugs fail or cannot be tolerated. Transcutaneous electrical nerve stimulation (TENS), with the electrodes placed above and below the involved area, may help about one third of patients. Because efficacy has not been demonstrated in properly designed studies, neural destructive treatments such as neurolytic nerve blocks, nerve sectioning, and dorsal root entry zone lesions are not recommended.

Cervicogenic Headache

Cervical spondylosis and muscle spasm may cause headache in older patients. The headache may be unilateral or bilateral. Digital pressure in the suboccipital area may reproduce the headache. The headache may be due to occipital neuralgia, myofascial pain with trigger points, or referred from neck structures such as the upper cervical facets (Chapter 9). Beneficial treatments include NSAIDs, muscle relaxants, tricyclic antidepressants, and physical therapy. Occipital nerve blocks and trigger point injections may be helpful in appropriate cases.

Parkinson's Disease

Headache associated with muscle rigidity may occur more often in Parkinson's disease. Tricyclic medications such as amitriptyline or nortriptyline may be effective. Amantidine and L-Dopa, drugs for the treatment of Parkinson's, can cause headaches in some people.

REFERENCES

1. Waters WE. The Pontypridd headache survey. *Headache* 1974; 14:81–90.
2. Solomon GD, Kunkel RS, Frame J. Demographics of headache in elderly patients. *Headache* 1990;30:273–276.
3. Geriatric headache. In: Silberstein SD, Lipton RB, Goadsby PJ, eds. *Headache in clinical practice*, 2nd ed. London: Martin Dunitz, 2002:269–283.
4. Pascual J, Berciano J. Experience in the diagnosis of headaches that start in elderly people. *J Neurol Neurosurg Psychiatry* 1994;57:1255–1257.
5. Cull RE. Investigation of late-onset migraine. *Scott Med J* 1995;40:50–52.
6. Fisher CM. Late-life migraine accompaniments as a cause of unexplained transient ischemic attacks. *Can J Neurol Sci* 1980; 7:9–17.
7. Fisher CM. Late-life migraine accompaniments: further experience. *Stroke* 1986;17:1033–1042.
8. Purdy RA. Late-life migrainous accompaniments. In: Gilman S, ed. *MedLink neurology.* San Diego: Arbor, 2004. Available at www.medlink.com.
9. Raskin NH. The hypnic headache syndrome. *Headache* 1988; 28:534–536.
10. Dodick DW, Mosek A, Campbell JK. The hypnic ("alarm clock") headache syndrome. *Cephalalgia* 1998;18:152–156.
11. Evers S, Goadsby PJ. Hypnic headache: clinical features, pathophysiology, and treatment. *Neurology* 2003 25;60(6):905–909.
12. Gould JD, Silberstein SD. Unilateral hypnic headache: a case study. *Neurology* 1997;49:1749–1750.
13. Myklebust G, Gran JT. A prospective study of 287 patients with polymyalgia rheumatica and temporal arteritis: clinical and laboratory manifestations at onset of disease and at the time of diagnosis. *Br J Rheumatol* 1996;35:1161–1168.
14. Wyand CM, Goronzy JJ. Giant-cell arteritis and polymyalgia rheumatica. *Ann Intern Med* 2003;16;139(6):505–515.
15. Jonasson F, Cullen JF, Elton RA. Temporal arteritis: a 14-year epidemiological, clinical and prognostic study. *Scot Med J* 1979;24:111–117.
16. Solomon S, Cappa KG. The headache of temporal arteritis. *J Am Geriatr Soc* 1987;35:163–165.
17. Jundt JW, Mock D. Temporal arteritis with normal erythrocyte sedimentation rates presenting as occipital neuralgia. *Arthritis Rheum* 1991;34:217–219.
18. Caselli RJ, Hunder GG, Whisnant JP. Neurologic disease in biopsy-proven giant cell (temporal) arteritis. *Neurology* 1988;38:352–358.
19. Mehler MR, Rabinowich L. The clinical neuro-ophthalmologic spectrum of temporal arteritis. *Am J Med* 1988;85:839–844.

20. Hunder GG, Bloch DA, Beat AM, et al. The American College of Rheumatology 1990 criteria for the classification of giant cell arteritis. *Arthritis Rheum* 1990;33:1122–1128.
21. Lee AG, Brazis PW. Temporal arteritis: a clinical approach. *J Am Geriatr Soc* 1999;47:1364–1370.
22. Smetana GW, Shmerling RH. Does this patient have temporal arteritis? *JAMA* 2002;287:92–101.
23. Schmidt WA, Kraft HE, Voker L, et al. Color duplex ultrasonography in the diagnosis of temporal arteritis. *N Engl J Med* 1997;337:1336–1342.
24. Salvarani C, Silingardi M, Ghirarduzzi A, et al. Is duplex ultrasonography useful for the diagnosis of giant-cell arteritis? *Ann Intern Med* 2002;137(4):232–238.
25. Miller A, Green M, Robinson D. Simple rule for calculating normal erythrocyte sedimentation rate. *BMJ* 1983;286:266.
26. Hayreh SS, Podhajsky PA, Raman R, et al. Giant cell arteritis: validity and reliability of various diagnostic criteria. *Am J Ophthalmol* 1997;123:285–296.
27. Duhant P, Pinede I, Bornet H, at al. Biopsy proven and biopsy negative temporal arteritis: differences in clinical spectrum at the onset of the disease. *Ann Rheum Dis* 1999;58:335–341.
28. Nesher G, Rubinow A, Sonnenblick M. Efficacy and adverse effects of different corticosteroid dose regimens in temporal arteritis: a retrospective study. *Clin Exp Rheumatol* 1997;15: 303–306.
29. Salvarani C, Cantini F, Boiardi L, et al. Polymyalgia rheumatica and giant-cell arteritis. *N Engl J Med* 2002;347(4):261–271.
30. Hoffman GS, Cid MC, Hellmann DB, et al. A multicenter, randomized, double-blind, placebo-controlled trial of adjuvant methotrexate treatment for giant cell arteritis. *Arthritis Rheum* 2002;46(5):1309–1318.
31. Gnann JW Jr, Whitley RJ. Herpes zoster. *N Engl J Med* 2002; 347:340–346.
32. Cluff RS, Rowbotham MC. Pain caused by herpes zoster infection. *Neurol Clin* 1998;16:813–832.
33. Dworkin RH, Boon RJ, Griffin DR, et al. Postherpetic neuralgia: impact of famciclovir, age, rash severity, and acute pain in herpes zoster patients. *J Infect Dis* 1998;178[Suppl 1]:S76–S80.
34. Pappagallo M, Haldey EJ. Pharmacological management of postherpetic neuralgia. *CNS Drugs* 2003;17(11):771–780.
35. Stacey BR, Glanzman RL. Use of gabapentin for postherpetic neuralgia: results of two randomized, placebo-controlled studies. *Clin Ther* 2003;25(10):2597–2608.
36. Watson CP, Babul N. Efficacy of oxycodone in neuropathic pain: a randomized trial in postherpetic neuralgia. *Neurology* 1998;50: 1837–1841.

HANDBOOK OF HEADACHE
Author Queries
Chapter 12

AQ1 In ref 3, please fill in author(s) of chapter.