PART
IV

Physical Examination of Patients with Headache

CHAPTER 13

CHAPTER OUTLINE

Clinical Reasoning in the Diagnosis: History Taking in Patients with Headache

Peter A. Huijbregts, PT, MSc, MHSc, DPT, OCS, MTC, FAAOMPT, FCAMT

ROLE OF HISTORY TAKING IN THE CLINICAL EXAMINATION

In physical therapy, as in other health-care professions, there are five elements to patient management. The examination is followed by evaluation of the examination findings, establishing a diagnosis, producing a prognosis and developing a plan of care, and, finally, performing the interventions (American Physical Therapy Association [APTA], 2001). The examination element of this process of care usually consists of history taking, systems review, and tests and measures. Unique to professions such as physical therapy with a limited scope of practice, a systems review is a brief history and physical examination specifically reviewing the cardiopulmonary, integumentary, musculoskeletal, and neuromuscular systems, but also meant to get an impression of a patient's communication ability, affect, cognition, language, and learning style (APTA, 2001).

Generally, the role of the examination process is twofold. First, intended to be comprehensive with regard to screening and specific testing, this process ideally leads to diagnostic classification. A second important role of the physical therapy examination and related to the limited neuromusculoskeletal scope of practice of physical therapy is the identification of problems outside this scope of practice that require a

referral for medical or surgical diagnosis and, perhaps, comanagement. The systems review component of the examination plays an important role in detecting indications for such referral (APTA, 2001).

Obtaining a comprehensive history is paramount to any diagnostic process but perhaps even more so in patients presenting with headache. Bartleson (2006) noted that in patients with complaint of headache, history taking in combination with the general and neurologic examinations constitutes the diagnostic gold standard. Indeed, the primary headache disorders defined in the *International Classification of Headache Disorders* (International Headache Society [IHS], 2004) have no available confirming diagnostic tests or procedures. Welch (2005) further emphasized the importance of history taking by noting that most patients presenting with headache often have few signs on physical examination. In children, the role of the history is perhaps even more important. Dooley et al. (2003) reported that the history provided the correct diagnosis and management in 100% of 150 children presenting with headaches.

Although there is some variation in a worldwide context, physical therapy interventions are generally limited to manual therapy, education, exercise, and modality-based treatments, thereby excluding pharmacologic and surgical interventions. In this aspect, physical therapy is similar to other professions that might be involved in the diagnosis and management of patients with headache, such as chiropractic and massage therapy. As discussed in this chapter, this makes the diagnostic process in such professions a bit easier.

There is mounting research evidence (or at the very least a plausible pathophysiologic rationale) that manual therapy interventions and other modalities within the scope of practice of physical therapy are effective in the management of five distinct types of headaches. Of the primary headaches, the scientific literature indicates that tension-type headache, and to a lesser extent migraine, may have an underlying neuromusculoskeletal contribution (Fernández-de-las-Peñas et al., 2006a, 2006b, 2006c; Tuchin et al., 2000; Simons et al., 1999; Tuchin, 1999). Secondary headaches with a neuromusculoskeletal etiology that are, therefore, potentially amenable to interventions within the physical therapy scope of practice include cervicogenic headache, occipital neuralgia, and headache associated with temporomandibular disorder (Issa & Huijbregts, 2006; Bronfort et al., 2001; Jull et al., 2002; Okeson, 1996; Bogduk, 1986). It should be noted here that these headache types—and especially migraine headache—all may benefit from pharmacologic management as well, indicating the (potential) need for medical comanagement.

This limited number of headache types potentially amenable to physical therapy management means that one of the main objectives of the examination will be to identify whether the presenting headache complaint can in fact be classified as one of these five headache types. **Tables 13.1** to **13.5** provide the diagnostic criteria for these headache types. We should note that for cervicogenic headache there are two different sets of diagnostic criteria (**Table 13.3**). In addition to the criteria in the *International Classification of Headache Disorders* (IHS, 2004), Sjaastad et al. and the Cervicogenic Headache International Study Group (1998) established criteria that are in fact used more frequently in the clinical situation.

Granella et al. (1994) demonstrated that diagnosis of primary headache types using the *International Classification of Headache Disorders* had adequate interrater reliability for clinical use ($\kappa = 0.74$). However, before we become too confident in our ability to diagnose headaches using this classification system, we have to realize that this system may seem simple to use but that the absence of confirming imaging, electrophysiologic, or laboratory tests for the primary headache disorders means that the clinician is frequently in the position of needing to consider or rule out a secondary headache type that may be similar in appearance to the primary headache disorder based on history and examination findings alone (Bartleson, 2006). For an extended outline of headache diagnosis, see Chapter 3.

Some of these secondary headache disorders mimicking the headache types that therapists can, in fact, manage may be serious or even life threatening and require urgent medical or surgical referral. Because the diagnostic procedures confirming such secondary headaches are outside the diagnostic scope of practice of the therapist, the second main objective of the history—and of the examination process in general—becomes not only the identification of a headache (or other undiagnosed) disorder not amenable to physical therapy intervention but also and more specifically the identification of those headache emergencies that require urgent referral (Chapter 3). Within the context of history taking, the clinician should seek to identify red flag symptoms indicating serious pathology. The history component of the systems review may also yield indications of pathology that may or may not be related to the presenting complaint of headache but that still require referral.

Table 13.1 Tension-Type Headache Criteria

Type	Diagnostic Criteria
Infrequent episodic tension-type headache (2.1)	A. At least 10 episodes occurring on <1 day per month on average (<12 days per year) and fulfilling criteria B–D B. Headache lasting from 30 minutes to 7 days C. Headache has at least two of the following characteristics: 1. Bilateral location 2. Pressing/tightening (nonpulsating) quality 3. Mild or moderate intensity 4. Not aggravated by routine physical activity such as walking or climbing stairs D. Both of the following: 1. No nausea or vomiting (anorexia may occur) 2. No more than one of photophobia or phonophobia E. Not attributed to another disorder
Frequent episodic tension-type headache (2.2)	A. At least 10 episodes of occurring on ≥1 but <15 days per month for at least 3 months and fulfilling criteria B–D B. Headache lasting from 30 minutes to 7 days C. Headache has at least two of the following characteristics: 1. Bilateral location 2. Pressing/tightening (nonpulsating) quality 3. Mild to moderate intensity 4. Not aggravated by routine physical activity such as walking or climbing stairs D. Both of the following: 1. No nausea and/or vomiting (anorexia may occur) 2. No more than one of photophobia and phonophobia E. Not attributed to another disorder
Chronic tension-type headache (2.3)	A. Headache occurring on ≥15 days per month on average for >3 months and fulfilling criteria B–D B. Headache lasts hours or may be continuous C. Headache has at least two of the following characteristics: 1. Bilateral location 2. Pressing/tightening (nonpulsating) quality 3. Mild to moderate intensity 4. Not aggravated by routine physical activity such as walking or climbing stairs D. Both of the following: 1. No more than one of photophobia, phonophobia, or mild nausea 2. Neither moderate or severe nausea nor vomiting E. Not attributed to another disorder
Chronic tension-type headache associated with pericranial tenderness (2.3.1)	A. Headache fulfilling criteria A–E for 2.3 (*chronic tension-type headache*) B. Increased pericranial tenderness on manual palpation
Chronic tension-type headache not associated with pericranial tenderness (2.3.2)	A. Headache fulfilling criteria A–E for 2.3 (*chronic tension-type headache*) B. No increased pericranial tenderness

Modified from International Headache Society, 2004.

In summary, although mirroring the two main goals of the examination in general as outlined earlier (APTA, 2001), the three main objectives of history taking in patients with headache disorders are more specific:

1. Confirm the diagnosis of tension-type headache, migraine headache, cervicogenic headache, occipital neuralgia, or headache associated with temporomandibular disorder, according to

Table 13.2 Migraine Headache Criteria

Type	Diagnostic Criteria
Migraine without aura (1.1)	A. At least five attacks fulfilling criteria B–D B. Headache attacks lasting 4–72 hours (untreated or unsuccessfully treated) C. Headache has at least two of the following characteristics: 1. Unilateral location 2. Pulsating quality 3. Moderate or severe pain intensity 4. Aggravation by or causing avoidance of routine physical activity (e.g., walking or climbing stairs) D. During headache at least one of the following: 1. Nausea and/or vomiting 2. Photophobia and phonophobia E. Not attributed to another disorder
Typical migraine with aura (1.2.1)	A. At least two attacks fulfilling criteria B–D B. Aura consisting of at least one of the following, but no motor weakness: 1. Fully reversible visual symptoms including positive features (e.g., flickering lights, spots or lines) and/or negative features (i.e., loss of vision) 2. Fully reversible sensory symptoms including positive features (i.e., pins and needles) and/or negative features (i.e., numbness) 3. Fully reversible dysphasic speech disturbance C. At least two of the following: 1. Homonymous visual symptoms and/or unilateral sensory symptoms 2. At least one aura symptom develops gradually over ≥5 minutes and/or different aura symptoms occur in succession over ≥5 minutes 3. Each symptom lasts ≥5 and ≤60 minutes D. Headache fulfilling criteria B–D for 1.1 (*migraine without aura*) begins during the aura or follows aura within 60 minutes E. Not attributed to another disorder
Chronic migraine (1.5.1)	A. Headache fulfilling criteria C and D for 1.1 (*migraine without aura*) on ≥15 days/month for >3 months B. Not attributed to another disorder
Probable migraine without aura (1.6.1)	A. Attacks fulfilling all but one of criteria A–D for 1.1 (*migraine without aura*) B. Not attributed to another disorder

Modified from International Headache Society, 2004.

established diagnostic classification systems (IHS, 2004; Okeson, 1996; Sjaastad et al., 1998).
2. Identify diagnostic indicators of headache types not amenable to physical therapy management, and specifically red flag symptoms indicating serious pathology requiring urgent referral.
3. Identify the presence—but not the exact nature—of pathology not necessarily related to the presenting headache complaint that requires referral for medical or surgical evaluation.

This chapter presents a suggested format for history taking in patients presenting with headache (**Table 13.6**) with specific attention to these three main objectives. The following sections discuss the various items in this format.

DEMOGRAPHICS

In patients older than 50, a new onset of migraine headache is unlikely (Gladstein, 2006). In fact, if a new headache suddenly appears in a patient older than 50, it should be considered a secondary headache until causes such as tumor, cerebrovascular disease, or temporal arteritis are excluded (Gentile, 2005). Temporal or

Table 13.3 Cervicogenic Headache Criteria

International Classification of Headache Disorders Criteria	Cervicogenic Headache International Study Group Criteria
A. Pain, referred from a source in the neck and perceived in one or more regions of the head and/or face, fulfilling criteria C and D B. Clinical, laboratory, and/or imaging evidence of a disorder or lesion within the cervical spine or soft tissues of the neck known to be, or generally accepted as, a valid cause of headache C. Evidence that the pain can be attributed to the neck disorder or lesion based on at least one of the following: 1. Demonstration of clinical signs that implicate a source of pain in the neck 2. Abolition of headache following diagnostic blockade of a cervical structure or its nerve supply using placebo or other adequate controls D. Pain resolves within 3 months after successful treatment of the causative disorder or lesion	I. Symptoms and signs of neck involvement: a. Precipitation of head pain, similar to the usually occurring one: 1. By neck movement and/or sustained awkward head posturing, and/or 2. By external pressure over the upper cervical or occipital region on the symptomatic side b. Restriction of the range of motion (ROM) in the neck c. Ipsilateral neck, shoulder, or arm pain of a rather vague nonradicular nature or, occasionally, arm pain of a radicular nature II. Confirmatory evidence by diagnostic anesthetic blockades III. Unilaterality of the head pain, without side shift For a diagnosis of CGH to be appropriate, one or more aspects of Point I must be present, with Ia sufficient to serve as a sole criterion for positivity or Ib and Ic combined. For scientific work, Point II is obligatory, while Point III is preferably obligatory.

Modified from International Headache Society, 2004, and Sjaastad et al., 1998.

Table 13.4 Occipital Neuralgia Criteria

Type	Diagnostic Criteria
Occipital neuralgia (13.8)	A. Paroxysmal stabbing pain with or without persistent aching between paroxysms in the distribution(s) of the greater, lesser, and/or third occipital nerves B. Tenderness over the affected nerve C. Pain is eased temporarily by local anesthetic block of the involved nerve

Modified from International Headache Society, 2004.

Table 13.5 Criteria for Headache Attributed to Temporomandibular Disorder

Type	Diagnostic Criteria
Headache or facial pain attributed to temporomandibular joint (TMJ) disorder (11.7)	A. Recurrent pain in one or more regions of the head and/or face fulfilling criteria C and D B. X-ray, MRI, and/or bone scintigraphy demonstrates TMJ disorder C. Evidence that pain can be attributed to the TMJ disorder, based on at least one of the following: 1. Pain is precipitated by jaw movements and/or chewing of hard or tough food 2. Reduced range of or irregular jaw movements 3. Noise from one or both TMJs during jaw movements 4. Tenderness of the joint capsule(s) of one or both TMJs D. Headache resolves within 3 months, and does not recur, after successful treatment of the TMJ disorder

Modified from International Headache Society, 2004.

Table 13.6 Suggested Format for History Taking in Patients with Headache

Demographics
Location of pain
Onset and course of headache
Character and intensity of headache
Aggravating and easing factors
Neurologic symptoms
Otolaryngologic symptoms
Systemic symptoms
Medical history
Medication history
Family history
Previous diagnostic tests
Prognostic indicators
Systems review

giant cell arteritis can lead to blindness due to occlusion of the ocular arteries. Patients may report severe unilateral or bilateral throbbing pain in the temporal region that is worse at night. Patients may also report scalp tenderness, jaw claudication, visual complaints, fatigue or joint pain associated with polymyalgia rheumatica, weight loss, fever, and night sweats. They may also have noted a tender, red, thickened, and nonpulsatile temporal artery (Peters, 2004; Welch, 2005). The clinician should not get too fixated on an age limit of 50 years, because values reported in the literature as red flags for the onset of a new headache vary between 40 and 60 years (Bartleson, 2006).

Gender also plays a role in diagnosis in that various headaches are more prevalent in women. European and American studies have showed a 1-year prevalence of 6% to 8% in men but 15% to 18% in women for migraine headaches. In developed countries, tension-type headache affects two-thirds of men and over 80% of women (World Health Organization, 2004). Musculoskeletal pain due to temporomandibular disorder is nine times more common in women than in men (Türp et al., 2002). In contrast, cluster headaches are more common in men, most typically smokers in their late 20s (Welch, 2005).

LOCATION OF PAIN

Characteristic locations of pain for the five headache types amenable to physical therapy management are indicated to some extent in the diagnostic criteria provided in Tables 13.1 to 13.5. Myofascial trigger points have been suggested as a main etiologic factor in tension-type headache (Fernández-de-las-Peñas et al., 2006a, 2006b, 2006d, 2006e). This means that specific pain locations may be matched with established pain referral patterns from myofascial trigger points, which in turn will guide the subsequent palpatory examination. Headache location is matched to specific muscle trigger points in **Table 13.7** (Fernández-de-las-Peñas et al., 2005, 2006f; Simons et al., 1999).

Further, with cervical zygapophysial joints implicated as the major etiologic factor in cervicogenic headache, referral patterns of these joints as established by way of joint infiltration can also guide subsequent examination (Dwyer et al., 1990). **Figure 13.1** provides zygapophysial joint referral patterns.

Headache occurring exclusively on one side may indicate a need for referral. Note that unilateral headaches are also likely in patients with more innocuous headache types such as cervicogenic headache; occipital neuralgia; headaches due to temporomandibular disorder; cluster headaches; paroxysmal hemicrania; short-lasting, unilateral, neuralgiform headache attacks with conjunctival injection and tearing (SUNCT); and with migraine. However, in a patient with a new onset of unilateral headache that does not have the features of one of these headache disorders—and of course for all headache types not amenable to (sole) physical therapy management—referral for further medical diagnosis is required (Bartleson, 2006).

ONSET AND COURSE OF HEADACHE

Headache onset can be qualified as acute, subacute, and chronic. In addition, patients may report a course of intermittent, episodic, or continuous pain that is stable or progressive in intensity. The five headache types amenable to physical therapy intervention are generally insidious in their onset (with the exception of a first-ever migraine headache) and of an intermittent, nonprogressive nature. Cluster headaches are—as the name implies—episodic in nature. Especially if a constant headache is progressive and worsening or if the patient presents with an acute-onset, worst-ever headache, the clinician should be attentive to a serious underlying pathology. Mills et al. (1986) reported abnormal findings on computed tomography (CT) scan for 29% of patients presenting with sudden-onset, reported worst-ever, or severe persistent headaches.

Causes of acute-onset headache include intracranial or intracerebral hemorrhage, acute subdural or epidural hematoma, pituitary apoplexy, acute closed-angle

Table 13.7 Myofascial Trigger Point Pain Referral Areas

Location of Pain	Muscles Potentially Involved
Vertex of the head	Sternal portion of sternocleidomastoid Splenius capitis
Occipital headache	Trapezius Sternal and clavicular portions of sternocleidomastoid Semispinalis capitis Semispinalis cervicis Splenius cervicis Suboccipital muscles Occipitalis Digastric Temporalis
Temporal headache	Trapezius Sternal portion of sternocleidomastoid Temporalis Splenius cervicis Suboccipital muscles Semispinalis capitis Superior oblique Lateral rectus
Frontal headache	Sternal and clavicular portions of sternocleidomastoid Semispinalis capitis Frontalis Zygomaticus major Superior oblique Lateral rectus
Ear and temporomandibular joint pain	Lateral and medial pterygoid Masseter Clavicular portion of sternocleidomastoid
Eye and eyebrow pain	Sternal portion of sternocleidomastoid Temporalis Splenius cervicis Masseter Suboccipital muscles Occipitalis Orbicularis oculi Trapezius
Cheek and jaw pain	Sternal portion of sternocleidomastoid Masseter Lateral and medial pterygoid Trapezius Digastric Buccinator Platysma Orbicularis oculi Zygomaticus major

Modified from Simons et al., 1999; Fernández-de-las-Peñas et al., 2005, 2006f.

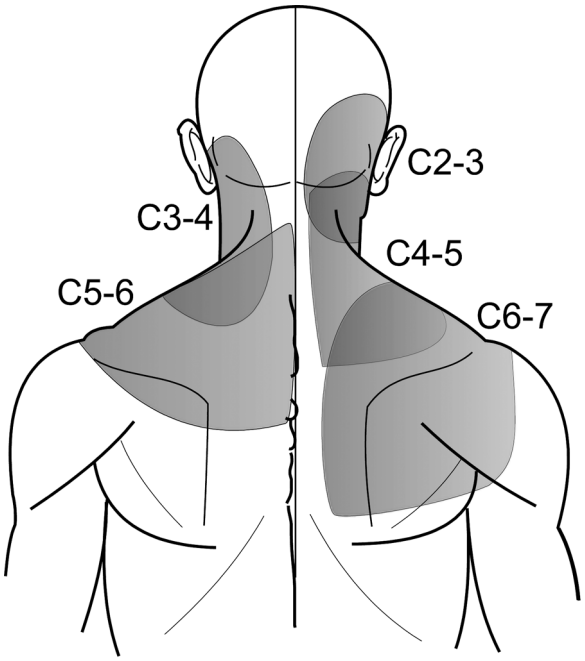

Figure 13.1 Zygapophysial Joint Pain Patterns

Modified from Lord SM, Barnsley L, Wallis BJ, Bogduk N. Chronic cervical zygapophyseal joint pain after whiplash. Spine 1996;21:1737–1745.

glaucoma, acute severe hypertension, internal carotid or vertebral artery dissection, head trauma causing a hemorrhage or cavernous sinus thrombosis, spontaneous low cerebrospinal fluid pressure headaches, acute obstructive hydrocephalus due to a tumor, benign central nervous system angiopathy, and idiopathic primary thunderclap headache. In contrast to all other causes listed here, primary thunderclap headache is innocuous, likely related to transient, and possibly recurrent, vasospasm (Bartleson, 2006; Peters, 2004).

Acute-onset headache, especially reported by the patient as a worst-ever headache, is also referred to as a thunderclap headache. Subarachnoid hemorrhage is the most common cause of this type of headache—usually located in the occipital region—and excluding this particular pathology takes priority in the diagnostic process (Bartleson, 2006; Gentile, 2005; Welch, 2005). Patients have compared the sensation of this type of headache to being hit across the back of the head with a blunt object (Welch, 2005). Thunderclap headache can also occur prior to aneurysmal subarachnoid hemorrhage in patients with an unruptured cerebral aneurysm (Bartleson, 2006). Relevant to the differential diagnosis from a first-ever migraine headache is that a thunderclap headache comes on within seconds, whereas a migraine builds up for over an hour (Gladstein, 2006). However, even a headache fitting the diagnostic criteria of recent-onset migraine may be following a small subarachnoid hemorrhage, indicating the need to take into consideration the whole clinical picture, and particularly patient age as discussed earlier (Gentile, 2005). Subarachnoid hemorrhage can be accompanied by a low-grade fever and neck stiffness: blood in the subarachnoid space may act as a local irritant and endogenous pyrogen (Gladstein, 2006).

Glaucoma constitutes another acute-onset headache emergency, as does cavernous venous thrombosis. Patients with glaucoma will report eye pain and vision changes. In case of cavernous venous thrombosis, patients usually have a history of ear, nose, or throat surgery or localized nasal infection, or they may report using medications that could cause a hypercoagulable state (Gladstein, 2006).

Secondary headaches with a subacute onset that may worsen rapidly but not suddenly include headaches caused by meningitis, encephalitis, sinusitis, cerebral vein thrombosis, ischemic cerebrovascular disease, and cerebral vasculitis (Peters, 2004).

Secondary headaches with a more gradual buildup may be related to pathologies such as a brain tumor, chronic subdural hematoma, brain abscess, temporal arteritis, idiopathic intracranial hypertension or pseudotumor cerebri, intracranial infection (such as those caused by Lyme disease, AIDS, or other systemic infections), and chronic sinusitis (Peters, 2004). Chronic progressive headache increasing slowly but steadily over the course of weeks to months may indicate a space-occupying lesion such as a brain tumor. Coughing, straining in the bathroom, sneezing, bending forward, and physical activity may exacerbate the headache related to a brain tumor. Pain is often worse in the morning and then gets a little better as the day progresses (Gentile, 2005; Gladstein, 2006). Often headache due to an intracranial mass lesion is accompanied by vomiting that is not preceded by nausea, especially in children (Gentile, 2005). Chronic subdural hematoma often will manifest weeks to months after a precipitating but often forgotten injury. In addition to headache worsened by sudden head movements, symptoms may include apathy, confusion, and inappropriate behavior (Peters, 2004).

CHARACTER AND INTENSITY OF HEADACHE

The diagnostic relevance of worst-ever, acute-onset, or thunderclap headache was discussed earlier. In studies on patients presenting at the emergency room for this type

of headache, the combined prevalence of significant intracranial pathology was 43% (95% CI: 20–68%) (Detsky et al., 2006).

The *International Classification of Headache Disorders* (IHS, 2004) classifies cluster headache as a primary headache. Cluster-type headaches are excruciating orbital and temporal headaches that come and go in a clusterlike pattern. Although this pattern is characteristic of the primary cluster headaches, it may also serve as an indicator of significant secondary headache types: the presence of cluster-type headaches carries a positive likelihood ratio (LR) of 10.7 (95% CI: 2.2–5.2) for the presence of serious intracranial abnormalities (Detsky et al., 2006).

Relevant to this portion of the history-taking process is that frequently headaches are of a mixed nature: migraine and tension-type headache frequently occur together, and they can be combined later in their course with headaches related to medication overuse. Of patients with migraine, 83% also have had tension-type headaches, whereas 23% of patients with tension-type headache also report migraine headaches (De Jongh et al., 2001). Every headache type requires a separate history (Welch, 2005). Tables 13.1 to 13.5 provide further details on the character and intensity of the headache types amenable to physical therapy management.

AGGRAVATING AND EASING FACTORS

Headaches amenable to physical therapy management should have underlying neuromusculoskeletal dysfunctions that can usually be aggravated or eased by mechanical factors such as those occurring with movement and position. Effects differ between these headache types: migraine may be differentiated from tension-type headache in that migraine headache is exacerbated by physical activity, whereas tension-type headache is not. Smetana (2000) established a positive LR of 3.7 (95% CI: 3.4–4.0) and a negative LR of 0.24 (95% CI: 0.23–0.26) for exacerbation by physical activity in the differential diagnosis of these two primary headache types. In fact, patients with tension-type headache may even seek out physical activity to decrease pain by way of distraction (Weeks & Weier, 2006). Patients with migraine tend to retreat to a dark and quiet environment, whereas patients with cluster headache are agitated and often will pace the room or rock back and forth holding their head (Gladstein, 2006; Weeks & Weier, 2006). However, some movement- and position-related effects on headache may be indicative of secondary headaches requiring (urgent) referral.

A more benign secondary headache is one related to cerebrospinal fluid hypotension. This type of headache is classically postural in that it occurs or worsens within 15 minutes of going from a lying to an upright position. It generally disappears within 30 minutes of again lying down. Usually bilateral and frontal in location, this headache is commonly associated with nausea, vomiting, dizziness, and tinnitus. One of the most common causes is a persistent cerebrospinal fluid leak after a lumbar puncture, but in the absence of such a medical history, it may be indicative of spontaneous low cerebrospinal fluid pressure syndrome (Bartleson, 2006; Peters, 2004).

Idiopathic intracranial hypertension usually causes a generalized frontal headache that may be unilateral or bilateral and is often worsened by bending over or straining. Patients may note a progressive loss of vision. This secondary headache is most common in obese women; average age of onset is 30 years (Peters, 2004).

It was noted earlier that the headache related to a brain tumor is exacerbated with coughing, straining in the bathroom, sneezing, bending forward, and physical activity. Pain is often worse in the morning and then gets a little better as the day progresses (Gentile, 2005; Welch, 2005; Gladstein, 2006). Coughing or posture changes may also cause the onset of headache if there is an underlying structural cause, such as Arnold-Chiari malformation type I, a colloid cyst of the third ventricle, or a tumor growing in the ventricular system (Gentile, 2005). Although exertional and cough headache can occur as an innocuous primary headache disorder, headache aggravated by exertion or a Valsalva-type maneuver carries a positive LR of 2.3 (95% CI: 1.4–3.8) for the presence of serious intracranial pathology (Detsky et al., 2006). In patients with a normal neurologic examination, headache aggravated by a Valsalva maneuver occurred significantly more often in those with secondary headaches than in patients with primary headaches (OR = 3.4) (Duarte et al., 1996). Bartleson (2006) noted that headache initiated by exertion or Valsalva maneuver (or cough) and associated neurologic symptoms or signs that are not consistent with the criteria for migraine with typical aura indicate the need to search for underlying intracranial disease.

Headache brought on by sexual activity can also occur as a relatively innocuous primary headache disorder. Similar to the exertional headache discussed earlier, it can be associated with migraine. Nevertheless, headache associated with sexual activity can also occur secondary to subarachnoid hemorrhage and arterial dissection and should always be considered a red flag

warning symptom requiring urgent referral (Bartleson, 2006).

Nonmechanical triggers, such as headache brought on by consuming certain food substances (caffeine, monosodium glutamate, chocolate, cheese, sulfites, citrus fruits, or alcohol), headache when relaxing, or headache brought on by alterations in sleeping, exercising, and eating habits all suggest migraine. Cluster headache is often brought on during a cluster period by drinking alcoholic beverages (De Jongh et al., 2006; Gladstein, 2006; Welch, 2005). The role of nonmechanical triggers as prognostic indicators is discussed later in this chapter.

NEUROLOGIC SYMPTOMS

Headache with an aura carries a positive LR of 3.2 (95% CI: 1.6–6.6) for serious intracranial pathology (Detsky et al., 2006). Of course, a visual or sensory aura is also part of the diagnostic criteria of typical migraine with aura (IHS, 2004). These auras generally have a gradual onset and diffuse slowly, taking almost 20 minutes to reach their widest distribution. This slow diffusion helps to distinguish them from auras associated with transient ischemic attacks, which have a quicker diffusion (Gentile, 2005). Because most studies used to establish the above LR did not provide information regarding the type of aura, interpretation of the diagnostic utility of the presence of an aura is somewhat limited (Detsky et al., 2006).

Seizures can occur in patients with brain tumors, cerebral vein thrombosis, and other causes of raised intracranial pressure (Peters, 2004; Welch, 2005). They may also occur in patients with meningitis (Welch, 2005). Focal neurologic deficits can occur in patients with pathologies that cause raised intracranial pressure, including cerebral vein thrombosis and intracranial hemorrhage (Davenport, 2002; Welch, 2005). Transient vision deficits occur in cases of increased intracranial pressure (Welch, 2005). Drowsiness, apathy, confusion, altered consciousness, or inappropriate behavior may be related to raised intracranial pressure due to hemorrhage and chronic subdural hematoma (Davenport, 2002; Peters, 2004). A (pre)syncope starting off an acute-onset, worst-ever headache is usually associated with a subarachnoid hemorrhage (Gentile, 2005).

Meningism or stiffness on neck flexion can occur in patients with meningitis but also in patients with subarachnoid hemorrhage (Gladstein, 2006; Welch, 2005). Rotation of the neck is usually not affected by true meningism, but it is limited in patients with cervical degenerative joint disease as implicated in cervicogenic headache, allowing for differential diagnosis (Davenport, 2002). However, it should be noted that nuchal rigidity has a sensitivity of only 30% for meningitis, making a negative test relatively meaningless when it comes to excluding serious causative pathology (Peters, 2004). In patients with brain tumors, the headache usually is preceded by neurologic symptoms, including, as noted earlier, seizure but also hemiparesis, ataxia, and cognitive or speech impairment (Peters, 2004).

Table 13.8 Brainstem Signs and Symptoms: Five D's and Three N's

Dizziness
Drop attacks
Diplopia (including amaurosis fugax and corneal reflux)
Dysarthria
Dysphagia (including hoarseness and hiccups)
Ataxia of gait
Nausea
Numbness (in ipsilateral face and/or contralateral body)
Nystagmus

Modified from Terrett, 2001.

Spinal manipulation has been associated with cervical artery dissection. Rubinstein et al. (2005) reported an OR of 3.8 for neck manipulation as a risk factor for cervical artery dissection. Perhaps this is why manual medicine practitioners, including physical therapists, chiropractors, medical and osteopathic physicians, and massage therapists, are hypervigilant with regard to the proposed ensuing classic brainstem signs and symptoms provided in **Table 13.8**. However, a variety of other pathologies can cause such symptoms. Specific to headache, during childhood and adolescence basilar migraine is a much more common cause than vertebral or internal carotid artery dissection (Gentile, 2005). **Table 13.9** summarizes the diagnostic criteria for basilar migraine (IHS, 2004).

With regard to a possible diagnosis of cervical artery dissection, it is important to realize that ischemic symptoms are not the only symptoms that may appear. Nonischemic symptoms usually develop first and are likely the result of deformation of nerve endings in the tunica adventitia of the affected artery and direct compression on local somatic structures (Kerry & Taylor, 2006). In fact, these nonischemic symptoms may occur hours to days and even a few weeks prior to the ischemic findings (Blunt & Galton, 1997). In the case of internal carotid artery dissection, this delay may even

Table 13.9 Diagnostic Criteria for Basilar Migraine

A. At least two attacks fulfilling criteria B–D
B. Aura consisting of at least two of the following fully reversible symptoms but no motor weakness: dysarthria, vertigo, tinnitus, hypoacusia, diplopia, visual symptoms simultaneously in both temporal and nasal fields of both eyes, ataxia, decreased level of consciousness, simultaneously bilateral paresthesia
C. At least one of the following: at least one aura symptom develops gradually over ≥5 minutes and/or different aura symptoms occur in succession over ≥5 minutes; each aura symptom lasts ≥5 and ≤60 minutes
D. Headache fulfilling criteria B–D from migraine without aura begins during the aura or follows the aura within 60 minutes
E. Not attributed to another disorder

Modified from International Headache Society, 2004.

be as long as years (Terrett, 2001). Ischemic findings develop in 30% to 80% of all dissections. Up to 20% of patients progress to a full cerebrovascular accident (Blunt & Galton, 1997). Nonischemic symptoms are unique to the exact pathology of the dissection, but ischemic neurologic symptoms can, of course, be expected to be similar for all underlying causes of cervical artery dysfunction.

In addition to the classic cardinal signs and symptoms summarized in Table 13.8, the literature has described further symptoms related to cervical artery dysfunction. **Table 13.10** provides ischemic and nonischemic signs or symptoms associated with cervical artery dissection (Albuquerque et al., 2002; Blunt & Galton, 1997; Guy et al., 2001; Kerry & Taylor, 2006; Taylor & Kerry, 2005; Terrett, 2001; Wojcik et al., 2003). Issues that are more relevant to the physical examination but that the patient may also report during the history taking

Table 13.10 Nonischemic and Ischemic Signs and Symptoms of Cervical Artery Dysfunction

	Vertebrobasilar System	Internal Carotid Artery
Nonischemic	Ipsilateral posterior neck pain Ipsilateral occipital headache Sudden onset and severe Described as stabbing, pulsating, aching, thunderclap, sharp, or of an unusual character: "a headache unlike any experienced before" Very rarely C5-C6 nerve root impairment (due to local neural ischemia)	Ipsilateral upper and midcervical spine pain Ipsilateral frontal-temporal or periorbital headache Sudden onset, severe, and of an uncommon character Horner syndrome Pulsatile tinnitus Cranial nerve palsies Ipsilateral carotid bruit Neck swelling Scalp tenderness Anhydrosis of face
Ischemic	Five **D**'s and three **N**'s (see Table 13.8) Vomiting Loss of short-term memory Vagueness Hypotonia and limb weakness affecting arm or leg Anhydrosis: lack of facial sweating Hearing disturbances Malaise Perioral dysesthesia Photophobia Clumsiness Agitation Cranial nerve palsies Hindbrain stroke: Wallenberg or locked-in syndrome	Transient ischemic attack Middle cerebral artery distribution stroke Retinal infarction Amaurosis fugax: temporary blindness Localized patchy blurring of vision: scintillating scotomata Weakness of extraocular muscles Protrusion of the eye Swelling of the eye or conjunctiva

Modified from Albuquerque et al., 2002; Blunt & Galton, 1997; Guy et al., 2001; Kerry & Taylor, 2006; Taylor & Kerry, 2005; Terrett, 2001; and Wojcik et al., 2003.

are the cranial nerve palsies that can occur with cervical artery dissection. Dissection of the internal carotid artery mainly causes cranial nerve IX to XII dysfunction, with the hypoglossal nerve initially affected and then the other three nerves; eventually, all cranial nerves except the olfactory can be affected (Blunt & Galton, 1997; Kerry & Taylor, 2006; Rubinstein et al., 2005).

Table 13.10 indicates that Horner syndrome is frequently associated with ipsilateral carotid artery dissection. The patient may have noted these physical signs and therefore may report these during the history taking. However, Horner syndrome is also common in cluster headaches. When the patient suffers symptoms and signs suggestive of a cluster headache, including Horner syndrome, but the pain is constant rather than intermittent and episodic, the possibility of arterial dissection needs to be considered, and, of course, more urgent referral is indicated (Gentile, 2005).

Arnold-Chiari type I malformation is a structural cause for headache with coughing or posture changes that requires surgical intervention once symptomatic (Gladstein, 2006). This pathology involves downward displacement of the cerebellar tonsils through the foramen magnum, causing symptoms of cerebellar involvement and brainstem compression. Ataxia in this malformation affects gait and is bilateral. Resultant hydrocephalus may also cause headache and vomiting. Brainstem compression can be associated with vertigo, nystagmus, and cranial nerve palsies (Simon et al., 1999). Other symptoms demonstrated in patients with compression at the level of the foramen magnum include suboccipital or neck pain (described as a tight collar) (in 65% of patients), often exacerbated by neck movement; pain in the hand (59%) or arm (55%), especially burning along the ulnar border of the contralateral arm in unilateral lesions; pain in the leg (26%) and face (7%); weak arm (40%) or leg (30%); hand clumsiness (27%); bladder dysfunction (22%); dysphagia (13%); dysarthria (3%); and paresthesia along the spine with trunk and neck flexion (positive Lhermitte sign) (3%). In addition to Horner syndrome occurring with internal carotid artery dissection and cluster headaches as discussed earlier, Arnold-Chiari malformation may also be the cause (Cross & Coles, 2002).

In summary, the neurologic symptoms associated with headaches can be varied and confusing. In general, we can state, though, that patients with nonmigrainous transient or persistent neurologic symptoms, seizures, or persistent neurologic signs most certainly require referral for medical diagnosis unless the neurologic symptoms and signs can be explained by another static and already medically diagnosed condition in the patient's medical history (Bartleson, 2006).

OTOLARYNGOLOGIC SYMPTOMS

Earlier we discussed how headache related to temporomandibular disorders could present with neuromusculoskeletal dysfunctions amenable to physical therapy. Temporomandibular disorders can present with any combination of headache, earache, or facial pain. The patient may also note limited opening of the jaw, joint noises, and localized muscle tenderness that is often unilateral (Cosenza, 2000; Türp et al., 2002). Patients may also report parafunctional habits such as teeth grinding, clenching, nail biting, and chewing gum (Royal College of Dental Surgeons of Ontario, 2002). This topic is addressed in detail in Chapter 17.

As discussed earlier, one of the pathologies resulting in a subacute-onset headache is acute sinusitis. Patients with sinusitis usually have purulent discharge from the nose, pain on palpation over the involved sinuses, cough, halitosis, and low-grade fever. However tempting to attribute all such symptoms to sinus pathology, we should assume that patients who present with lacrimation, rhinorrhea, sinus tenderness, and nasal congestion usually have migraine and not sinusitis (Gladstein, 2006; Peters, 2004). In case of autonomic symptoms and an acute and recurrent pattern, the likelihood of migraine headache is further increased. Of differential diagnostic importance is that sinus disease does not remit and return as migraine does (Gladstein, 2006).

SYSTEMIC SYMPTOMS

Nausea is a symptom helpful in distinguishing migraine from tension-type headache. Smetana (2000) reported a positive LR of 19 (95% CI: 15–25) and a negative LR of 0.19 (95% CI: 0.18–0.20). Photophobia and phonophobia also have good diagnostic utility for the purpose of distinguishing these two primary headache types, with a positive LR of 5.8 (95% CI: 5.1–6.6) and 5.2 (95% CI: 4.5–5.9) for the presence of migraine, respectively. The negative LR is 0.24 (95% CI: 0.23–0.26) and 0.38 (95% CI: 0.36–0.40) for photophobia and phonophobia, respectively (Smetana, 2000). Headache associated with vomiting has limited diagnostic utility for detecting serious intracranial pathology, with a positive LR of 1.8 (95% CI: 1.2–2.6) (Detsky et al., 2006). Vomiting, together with fatigue, diarrhea, and poor appetite, may

also be the presenting picture of meningitis, especially in elderly patients and infants (Welch et al., 2005).

Up to 30% of acute headaches may be secondary to viral infections (Brna & Dooley, 2006). This may be even more relevant for children presenting with headaches. Burton et al. (1997) reported that viral illness accounted for 39% of headaches in children presenting to a U.S. emergency room. Fever and headache may indicate headache due to viral syndrome and need cause little concern. However, headache and fever associated with neck stiffness increases the suspicion of meningitis (Gladstein, 2006). The clinician should keep in mind the low sensitivity for meningism in the diagnosis of meningitis, as discussed previously. Usually meningitis is also associated with photophobia and a rash (Welch, 2005). Headache associated with fever and a change in mental status requires evaluation for encephalitis. A low-grade fever may also occur in a subarachnoid hemorrhage (Gladstein, 2006). This chapter has already discussed the fever, weight loss, and night sweats associated with temporal arteritis (Peters, 2004).

Night wakening due to headache has a high likelihood ratio (LR = 98) for serious intracranial pathology (Bartleson, 2006). Classically, headache associated with a brain tumor will disturb sleep (Peters, 2004). However, night wakening is also common with less worrisome headaches such as migraine, paroxysmal hemicrania, or cluster headache. Therefore, night wakening needs to be assessed in the context of the patient's total presentation. However, the clinician should keep in mind that waking from sleep because of headache is more worrisome in children (Bartleson, 2006). History-based indications for further diagnostic testing in children include recent onset of headache, seizures, and night wakening, especially in the absence of a family history of migraine (Bartleson, 2006).

MEDICAL HISTORY

Red flags in the medical history include history of human immunodeficiency virus (HIV) infection or cancer, because these diagnoses increase the chance of secondary headaches due to brain tumor, meningitis, and opportunistic infections (Bartleson, 2006; Clinch, 2001; Detsky et al., 2006). However, even a patient with cancer or HIV infection may require no further medical diagnosis if the presenting headache complaint is long-standing, stable, and consistent with a primary headache disorder (Bartleson, 2006).

Uncontrolled hypertension may lead to spontaneous intracerebral hemorrhage. Cerebral vein thrombosis should be considered in patients with hypercoagulable states, trauma, or rheumatologic disorders (Peters, 2004). In patients with headache due to cavernous venous thrombosis, there is usually a previous medical history of ear, nose, and/or throat surgery, localized nasal infection, or the use of medications that could cause a hypercoagulable state (Gladstein, 2006). Temporal arteritis is more common in patients with polymyalgia rheumatica (Welch, 2005). Cerebrospinal fluid hypotension headache is most commonly caused by a persistent leak after lumbar puncture (Peters, 2004).

A patient reporting a history of head and neck trauma indicates the need to look for epidural, subdural, subarachnoid, or other intracranial hemorrhage and posttraumatic dissection of the carotid and vertebral arteries (Bartleson, 2006). Acute subdural hematoma usually comes on after head trauma and is seen commonly in alcoholic patients who have frequent falls (Peters, 2004). With subdural hematoma and arterial dissection, there is often a delay between the precipitating trauma and the onset of headache. A relatively minor head injury, which may have occurred weeks or even months before the onset of the presenting headache, may be forgotten but significant in an older patient or a patient on anticoagulant medication (Bartleson, 2006). Prior facial injury, jaw fracture, or stressful life situations can all indicate the presence of headache related to temporomandibular disorders (Cosenza, 2000).

MEDICATION HISTORY

Collecting information on medications taken by the patient and on their effect allows the therapist to double-check the medical history provided for its comprehensiveness and accuracy. Asking about recreational or illicit substances may uncover underlying causes of headaches. Alcohol, caffeine, cannabis, and cocaine are examples of substances implicated with regard to producing headaches (Katsarava et al., 2006).

Medications that cause hypercoagulable states have been implicated in the etiology of cavernous venous thrombosis. In contrast, anticoagulant medications (e.g., warfarin, heparin, or aspirin) may lead to hemorrhage and hematoma even after minor trauma (Bartleson, 2006; Gladstein, 2006). Oral contraceptives; vitamin A; antibiotics, including tetracycline, minocycline, trimethoprim and sulfamethoxazole, and nalidixic acid; corticosteroids; and other drugs (e.g., isotretinoin or

Accutane, tamoxifen, cimetidine or Tagamet) have all been implicated in the etiology of idiopathic intracranial hypertension (Brna & Dooley, 2006; Clinch, 2001).

Perhaps most relevant to the etiology of chronic daily headache is the fact that overuse of analgesic medication—including most medications prescribed for acute attacks of migraine or tension-type headache—puts patients at risk of developing a daily, oppressive, dull headache that is worse in the mornings and is unrelieved by analgesics. This type of headache is known as a medication-overuse headache (Welch, 2005). In the context of medication-overuse headache, taking an acute attack medicine more than twice a week should be considered overuse. Usually patients have a headache that is improved by the acute attack medicine, only to return (rebound headache) as the effects of the analgesic wear off. Patients take the medicine again and thus start a vicious cycle. The main medications implicated with regard to medication-overuse headache are over-the-counter analgesics that combine Tylenol or aspirin with caffeine or codeine phosphate, or both. Relevant prescription analgesics include caffeine opiate agonists, ergotamine, and triptans (Goadsby & Boes, 2002).

Other medications can produce a headache mimicking either migraine or tension-type headaches. Nitrates, phosphodiesterase inhibitors, and histamine can cause a bilateral, frontotemporal headache that is aggravated by physical activity similar to migraine. In contrast, atropine, digitalis, imipramine, nifedipine, and nimodipine produce generalized headaches similar to tension-type headaches (Katsarava et al., 2006).

FAMILY HISTORY

Migraine headaches usually have a strong genetic component (Gladstein, 2006). In addition, taking a family history may reveal rheumatologic, collagenous tissue, and cardiovascular disorders that have been implicated in the etiology of secondary headaches.

PREVIOUS DIAGNOSTIC TESTS

Imaging findings and laboratory test results may indicate the presence of an underlying pathology responsible for a secondary headache disorder. The *International Classification of Headache Disorders* (IHS, 2004) notes imaging findings of a disorder or lesion within the cervical spine or soft tissues of the neck known to be, or generally accepted as, a valid cause of headache as one of the diagnostic criteria for cervicogenic headache.

PROGNOSTIC INDICATORS

Perhaps most relevant to physical therapists are symptoms that indicate a good prognosis (or absence thereof) with interventions within the physical therapy scope of practice. Although likely already collected as part of a comprehensive history taking, such prognostic indicators deserve to again be highlighted here.

Niere (1998) reported that patients noting diet as an aggravating factor, those who used affective and autonomic pain descriptors (i.e., *sickening*, *fearful*, *nauseating*, *punishing*, *splitting*), those who had unilateral headaches, and those with a low headache frequency all had a negative response to manipulative physiotherapy treatment, the content of which was left to the discretion of the treating clinician. In contrast, Niere found that high headache frequency predicted a positive outcome.

Jull and Stanton (2005) aimed to identify those patients with cervicogenic headache who did or did not achieve a 50% to 79% or 80% to 100% reduction in headache immediately and 12 months postintervention. Only the absence of lightheadedness indicated higher odds of achieving either a 50% to 79% (OR = 5.45) or 80% to 100% (OR = 5.7) reduction in headache frequency in the long term. Of note was that headaches of at least moderate intensity, patient age, and headache chronicity did not preclude a successful outcome with physical therapy consisting of combinations of manipulation and specific exercise.

Fleming et al. (2007) found that increased patient age, provocation or relief of headache with movement, and being gainfully employed were all patient factors that were significantly related to improved outcomes with physical therapy consisting of manual therapy and specific exercise aimed at specific impairments identified during the examination in patients with cervicogenic headache.

Clinical prediction rules are decision-making tools that contain predictor variables obtained from patient history, examination, and simple diagnostic tests. They can assist in making a diagnosis, establishing prognosis, or determining appropriate management (Laupacis et al., 1997). Fernández-de-las-Peñas et al. (2008) have developed a clinical prediction rule to identify patients with chronic tension-type headache apt to have short-term benefit from manual trigger point therapy consisting of varied combinations of pressure release, muscle energy, and soft-tissue techniques combined with progressive, low-load deep cervical flexor and extensor muscle strengthening exercises. Relevant improvement

Table 13.11 Suggested History Component for Systems Review

System	Questions
Cardiovascular	Do you ever experience chest pain (angina)?
	Do you experience excessive unexplained fatigue?
	Do you have shortness of breath?
	Do you ever note chest palpitations?
	Have you noted lightheadedness?
	Have you ever fainted?
	Do you experience widespread leg pains?
	Have you noted swelling in the feet, ankles, or perhaps the hands?
Pulmonary	Do you ever experience chest pain?
	Do you have shortness of breath?
	Have you been coughing more lately?
	Have you noticed a change in your breathing?
	Do you have difficulty catching your breath when lying flat; do you have to sleep propped up on multiple pillows?
Gastrointestinal	Have you had difficulty swallowing?
	Have you had abdominal pain?
	Has your stool been black?
	Has your stool been different in consistency (diarrhea, tarry stool)?
	Have you been constipated?
Genitourinary	Any difficulty urinating?
	Have you noted blood in your urine?
	Have you noted an increased frequency with regard to urination?
	Have you noted an increased urgency with regard to urination?
	Have you noted an increased difficulty with initiating urination?
	Have there been episodes of impotence?
	Have there been any changes with regard to menstruation?
	Have you experienced pain with intercourse?
	Have you noted incontinence for urine and/or stool?

was defined in this study as an at least 50% reduction in headache intensity, frequency, or duration and an increase of 5 or more points on a 15-point global rating of change or patient satisfaction scale. Four variables constituted the clinical prediction rule for benefit at 1 week after discharge: headache duration less than 8.5 hours per day, headache frequency less than 5.5 days per week, SF-36 bodily pain domain score less than 47, and SF-36 vitality domain score less than 47.5. If the patient had three of these four variables, the positive LR for short-term benefit at 1 week postdischarge was 3.4 (95% CI: 1.4–8.0). If all four variables were present, the positive LR increased to 5.9 (95% CI: 0.8–42.9). Two variables made up the clinical prediction rule for benefit 1 month postdischarge: headache frequency less than 5.5 days per week, and SF-36 bodily pain domain score less than 47. With one of these two variables present, the positive LR was 2.2 (95% CI: 1.2–3.8); two variables present yielded a positive LR of 4.6 (95% CI: 1.2–17.9).

SYSTEMS REVIEW

As discussed earlier, a systems review is part of the physical therapy examination and probably of the examination in all professions with a similarly limited scope of practice with regard to diagnosis and management. Partly consisting of a history component and partly of physical tests, this review of organ systems differs from the specific pathology-based diagnosis done by the physician or other health-care professional with a more comprehensive scope of practice and serves to alert the clinician to undiagnosed conditions that may affect patient management or that indicate the need for referral (Boissonnault, 1995). The clinician should keep in mind that such conditions may or may not be related to the presenting headache complaint. **Table 13.11** provides one example of a history component to the systems review process. Doing the history component and also—but to a lesser extent—the physical examination

component of the systems review depends on the information obtained in the rest of the history-taking process, meaning that all questions are not mandatory but rather at the discretion of the clinician.

CLINICAL PREDICTION RULES FOR DIAGNOSIS

Research-based data on the diagnostic utility of the history-taking process in patients with headache is limited. Most of the evidence relevant to the diagnosis of patients with headaches comes from uncontrolled studies, case series, and expert opinion. The over 300 potential causes for headache and the benign nature of most headaches, especially the most common types of headache such as migraine and tension-type headache, may explain this dearth of research evidence (Bartleson, 2006). However, in their review of the literature, Detsky et al. (2006) identified one clinical prediction rule relevant to diagnosis. This clinical prediction rule consists of the following five questions:

1. Is it a pulsating headache?
2. Does it last between 4 and 72 hours without medication?
3. Is it unilateral?
4. Is there nausea?
5. Is the headache disabling (with disabling headaches defined as headaches that disrupt a patient's daily activities)?

When the patient answers yes to four or more of these five questions, the positive LR for a diagnosis of migraine headache is 24 (95% CI: 1.5–388). With a yes answer to three questions, the positive LR is 3.5 (95% CI: 1.3–9.2). For a yes answer to one or two of these criteria, the positive LR is 0.41 (95% CI: 0.32–0.52). The mnemonic POUNDing (*p*ulsating, *d*uration of 4–72 hours, *u*nilateral, *n*ausea, *d*isabling) may be helpful for clinicians when using this clinical prediction rule (Detsky et al., 2006).

OUTCOME MEASURES

The information discussed so far can serve to produce questions in the history-taking process that provide the therapist with diagnostic information. However, history taking also should include outcome measures. The intent of outcome measures is not the collection of data to help with diagnosis but rather to provide the therapist, the patient, and other interested parties (such as third-party payers) with a reliable, valid, and responsive gauge of the effect of treatment.

Pain Measures

The Numeric Pain Rating Scale (NPRS) and the Visual Analogue Scale (VAS) for pain both are reliable, valid, and easy-to-apply outcome measures with regard to headache pain parameters. The VAS may be the preferred measure because research into its responsiveness has greater external validity to headache patients than research available on responsiveness for the NPRS. Kelly (2001) determined the minimal clinically important difference (MCID) for the VAS in a varied population of patients presenting to the emergency department with pain as 10 to 12 (mm), depending on but not significantly different between groups for severity of reported pain.

Disability Measures

The Headache Disability Inventory (HDI) is a 25-item questionnaire that seeks to measure the self-perceived disabling effects of headache on daily life. The questionnaire contains two subgroups of questions, thereby creating emotional and functional subscale scores and a total score. Two additional items on the questionnaire ask patients to rate the severity of their headache as mild, moderate, or severe, and the frequency as less than or equal to one per month, more than one but less than four per month, or four or more times per month. The HDI has good internal consistency reliability; correlations between the emotional and functional subscale scores and the total score were both excellent ($r = 0.89$) (Jacobson et al., 1994). It also has good short-term (1-week) ($r = 0.93–0.95$) (Jacobson et al., 1995) and generally good long-term (2-month) test-retest reliability ($r = 0.83$) for the total scores. The HDI also exhibits good internal construct validity ($P < 0.001$) (Jacobson et al., 1994). A minimal detectable change (MDC_{95}) score at 1-week retest is 16 points. This value for the MDC_{95} indicates that a clinician can be 95% confident that a true change has occurred with a change in the HDI score of 16 or more points (Jacobson et al., 1995). Similarly, a 29-point score improvement constitutes the MDC_{95} over a 2-month time period (Jacobson et al., 1994). The HDI test is simple to administer and takes little time to complete.

With physical therapy interventions for the five headaches amenable to physical therapy management

Table 13.12 Red Flag Indicators in the History Taking of Patients with Headaches That Indicate the Need for Urgent Referral

History-Taking Category	Red Flag Symptoms
Demographics	New onset of headache or change in existing headache pattern in patients over 50
Location of pain	Persistent unilateral location of headaches
Onset and course of headache	New-onset headache
	Onset of a new headache type
	Unexplained change for the worse in pattern of existing headache
	Progressively worsening headache
	Abrupt, split-second onset of headache: thunderclap headache
Character and intensity of headache	New pain level, especially when described as worst ever
	Cluster-type headache
Aggravating and easing factors	Headache aggravated or brought on by physical exertion, coughing, sneezing, straining, or sexual activity
	Noted effect of position changes on pain
	No response to seemingly appropriate treatment
Neurologic symptoms	Seizures, confusion, changes in alertness, apathy, clumsiness, unexplained inappropriate behavior, brainstem symptoms, bowel and bladder symptoms, neck flexion stiffness, aura preceding the headache (especially one with quick diffusion), or weakness (not consistent with an existing diagnosis of migraine headaches or other pathology explaining these symptoms)
	Presyncope or syncope starting off headache
Otolaryngologic symptoms	Associated eye pain and simultaneous vision changes
Systemic symptoms	Fever, weight loss, temporal artery tenderness, profuse vomiting (especially when not associated with nausea), photophobia, phonophobia, or developing rash (not consistent with an existing diagnosis of migraine headaches)
	Headache that awakens a patient from night sleep (especially in children)
Medical history	Medical history of cancer and human immunodeficiency virus infection
	Head or neck injury
	Uncontrolled hypertension
Medication history	Use of anticoagulant medication in combination with even minor trauma
Family history	Absence of a family history of migraine in children with migrainelike symptoms

largely geared to affecting underlying neuromusculoskeletal impairments in the cervical spine, the Neck Disability Index (NDI) is potentially a useful outcome measure in that this 10-item questionnaire aims to measure the self-perceived disabling effects of neck pain on daily life. Interpretation is possible through scoring intervals as follows: 0–4 = no disability, 5–14 = mild, 15–24 = moderate, 25–34 = severe, and above 34 = complete disability (Vernon & Mior, 1991). To arrive at a percentage disability, the total score is multiplied by two. The NDI has moderate test-retest reliability (ICC = 0.68) (Cleland et al., 2006). Construct validity of the NDI as an outcome measure for neck pain has been demonstrated by comparing it with other tests or measures. Cleland et al. (2006) showed that a 7-point (14%) change in the NDI constituted a minimally clinically important difference for the NDI, albeit only in patients with cervical radiculopathy and not specifically in patients reporting headaches.

RED FLAGS

One of the main objectives of the physical therapy history-taking process in patients presenting with a complaint of headaches is to identify red flag findings that indicate the need for urgent referral. **Table 13.12** provides a list of such red flag indicators based on the information presented in this chapter.

CONCLUSIONS AND IMPLICATIONS OF HISTORY FINDINGS

The history-taking process in patients with headaches presents unique challenges, particularly to health-care

providers with a limited scope of practice, such as physical therapists, chiropractors, and massage therapists. The information provided in this chapter should allow the clinician to develop a history-taking process that allows him or her to meet the three main objectives noted in the beginning of this chapter, namely, diagnosing those headaches amenable to physical therapy interventions, identifying diagnostic indicators of headache not amenable to sole physical therapy management or even those that require urgent referral, and, finally, identifying undiagnosed pathology that may or may not be related to the presenting complaint of headache but that requires referral nevertheless for further diagnosis and management. History taking and systems review as discussed here are only part of the examination process, and other chapters address the other and interrelated portions in more detail.

Potentially most relevant to history taking and the diagnostic process is the identification of red flag indicators. The relevance and implications of finding such red flag indicators should always be considered in the context of the whole presenting clinical picture. However, the principle of *primum non nocere* clearly indicates the need at all times for referral in case of even minimal diagnostic uncertainty.

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