Postpartum headache is the complaint of cephalic, neck, or shoulder pain occurring during the first 6 weeks after delivery. The overall incidence of postpartum headache has not been determined in a prospective study. However, information is available from a prospective evaluation of women during the first week postpartum and from a prospectively collected database that recorded symptoms during pregnancy and the first week after delivery. Among 985 women in the prospective evaluation, the incidence of headache in the first week postpartum was 38.7%. The median time from delivery to onset of symptoms was 2 days, and the median duration of headache was 4 hours. Benhamou et al. examined information collected on pregnant women who delivered at their institution during a 2-year period; exclusion criteria included recognized dural puncture, preterm delivery, multiple gestation, and/or elective cesarean delivery. Headache was reported by 12% of 1058 patients who had epidural anesthesia without dural puncture and by 15% of 140 patients who delivered without epidural anesthesia.

Post–dural puncture headache (PDPH) is one of the most common postpartum complications of neuraxial anesthesia. However, physicians and nurses should be aware that dural puncture is only one of many causes of postpartum headache (Table 31-1). Difficult diagnostic problems may require the opinion of a neurologist. The purpose of this chapter is to discuss the differential diagnosis of postpartum headache and of PDPH in particular.

Primary Headaches

The postpartum patient can present with a recurrence of her known primary disorder or with the first manifestation of a primary condition. Patients with a history of headache disorders typically are diagnosed with one of four major types of primary headaches. The most common postpartum headaches are tension-type and migrainous headaches, which account for almost two thirds of headaches during this period. Tension-type headaches are often circumferential and constricting, can be associated with scalp tenderness, and are usually of mild to moderate severity.

Migraine

Migrainous headaches (or migraines) are defined as recurring cranial pain lasting 4 to 72 hours, often with typical features such as pulsating pain in a unilateral location,
### TABLE 31-1 Differential Diagnosis of Postpartum Headache

<table>
<thead>
<tr>
<th>Headache Etiology</th>
<th>Percentage of Postpartum Headaches*</th>
<th>Primary Symptoms/Signs</th>
<th>Diagnostic Modality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tension headache</td>
<td>39</td>
<td>Mild to moderate headache, lasting 30 minutes to 7 days</td>
<td>History and physical examination</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Often bilateral, nonpulsating, and not aggravated by physical activity</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Associated with neck and/or shoulder pain</td>
<td></td>
</tr>
<tr>
<td>Migraine</td>
<td>11-34</td>
<td>Recurrent moderate to severe headache, lasting 4 to 72 hours</td>
<td>History and physical examination</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Often unilateral, pulsating, and aggravated by physical activity</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Associated with nausea, photophobia, and phonophobia</td>
<td></td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>11-15</td>
<td>Mild to moderate headache accompanied by neck and/or shoulder pain</td>
<td>History and physical examination</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preeclampsia/Eclampsia</td>
<td>8-24</td>
<td>Hypertension and/or HELLP (hemolysis, elevated liver enzymes, low platelet count)</td>
<td>Laboratory evaluation (alanine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>syndrome</td>
<td>aminotransferase [ALT], aspartate</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Headache often bilateral, pulsating and aggravated by physical activity</td>
<td>transaminase [AST], uric acid,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>platelet count, urine protein)</td>
</tr>
<tr>
<td>Post–dural puncture headache</td>
<td>5-16</td>
<td>Headache within 5 days of dural puncture</td>
<td>History and physical examination</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Worsens within 15 minutes of sitting or standing</td>
<td>Possible MRI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Associated with neck stiffness, tinnitus, photophobia, and nausea</td>
<td></td>
</tr>
<tr>
<td>Cortical vein thrombosis</td>
<td>3</td>
<td>Nonspecific headache that may have a postural component</td>
<td>History and physical examination</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Often accompanied by focal neurologic signs and seizures</td>
<td>CT or MRI</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>1</td>
<td>Abrupt onset of an intense and incapacitating headache</td>
<td>History and physical examination</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Often unilateral</td>
<td>CT without contrast or MRI (FLAIR</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Accompanied by nausea, nuchal rigidity, and altered consciousness</td>
<td>sequence)</td>
</tr>
<tr>
<td>Posterior reversible</td>
<td>Unknown</td>
<td>Severe and diffuse headache with an acute or gradual onset</td>
<td>History and physical examination</td>
</tr>
<tr>
<td>(leuko)encephalopathy (PRES) syndrome</td>
<td></td>
<td>Possible focal neurologic deficits and seizures</td>
<td>MRI</td>
</tr>
<tr>
<td>Brain tumor</td>
<td>Unknown</td>
<td>Progressive and often localized headache</td>
<td>History and physical examination</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Often worse in the morning</td>
<td>CT or MRI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Aggravated by coughing/straining</td>
<td></td>
</tr>
<tr>
<td>Subdural hematoma</td>
<td>Unknown</td>
<td>Headache usually without typical features</td>
<td>History and physical examination</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Often overshadowed by focal neurologic signs and/or altered consciousness</td>
<td>CT or MRI</td>
</tr>
<tr>
<td>Cerebral infarction/</td>
<td>Unknown</td>
<td>Moderate headache accompanied by focal neurologic signs and/or altered consciousness</td>
<td>History and physical examination</td>
</tr>
<tr>
<td>ischemia</td>
<td></td>
<td></td>
<td>CT or MRI (showing angiographic “string of beads” appearance)</td>
</tr>
</tbody>
</table>

CSF, cerebrospinal fluid; CT, computed tomography; MRI, magnetic resonance imaging; FLAIR, fluid-attenuated inversion recovery.

nausea, and photophobia. Headache with aura is a subtype of migraine that is characterized by focal neurologic symptoms preceding the headache. The prevalence of migraine is approximately 17% in the female population (three times more common than the prevalence in the male population) and is more common in patients between 30 and 50 years of age. Pregnancy has an ameliorating effect on migraine frequency in the majority of sufferers. However, symptoms may recur soon after delivery, with reports of 34% recurrence within the first week postpartum and 55% within the first month. Generally the symptoms are similar to their typical pattern, although often milder and less often unilateral. It is rare for a migraine to manifest for the first time during the postpartum period. There appears to be an association between migraine and preeclampsia, which may reflect an underlying predisposition to cerebral ischemic injury.

### Secondary Headaches

A common secondary headache in the postpartum period is the musculoskeletal headache, exacerbated by the maternal physical exertion of labor and associated sleep deprivation. This headache has accompanying neck and shoulder pain.

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**TABLE 31-1—cont’d Differential Diagnosis of Postpartum Headache**

<table>
<thead>
<tr>
<th>Headache Etiology</th>
<th>Percentage of Postpartum Headaches</th>
<th>Primary Symptoms/Signs</th>
<th>Diagnostic Modality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pseudotumor cerebri/Benign intracranial hypertension</td>
<td>Unknown</td>
<td>Progressive nonpulsating headache Aggravated by coughing/straining Associated with increased CSF pressure and normal CSF chemistry</td>
<td>History and physical examination Lumbar puncture</td>
</tr>
<tr>
<td>Spontaneous intracranial hypotension</td>
<td>Unknown</td>
<td>No history of dural trauma Diffuse, dull headache worsening within 15 minutes of sitting or standing Associated with neck stiffness, nausea, tinnitus, and photophobia CSF opening pressure &lt; 60 mm H₂O in the sitting position</td>
<td>History and physical examination Lumbar puncture Radioisotope cisternography CT myelography</td>
</tr>
<tr>
<td>Sinusitis</td>
<td>Unknown</td>
<td>Frontal headache with accompanying pain in the face Development of headache coincides with nasal obstruction Purulent nasal discharge, anosmia, and fever</td>
<td>History and physical examination Nasal endoscopy CT or MRI</td>
</tr>
<tr>
<td>Meningitis</td>
<td>Unknown</td>
<td>Headache is most frequent symptom Often diffuse Intensity increases with time Associated with nausea, photophobia, phonophobia, general malaise, and fever</td>
<td>History and physical examination Lumbar puncture</td>
</tr>
<tr>
<td>Pneumocephalus</td>
<td>Unknown</td>
<td>Frontal headache Often an abrupt onset immediately following dural puncture Symptoms can worsen with upright posture</td>
<td>History and physical examination CT</td>
</tr>
<tr>
<td>Caffeine withdrawal</td>
<td>Unknown</td>
<td>Onset of headache within 24 hours of cessation of regular caffeine consumption Often bilateral and pulsating Relieved within 1 hour of ingestion of caffeine 100 mg</td>
<td>History and physical examination</td>
</tr>
<tr>
<td>Lactation headache</td>
<td>Unknown</td>
<td>Mild to moderate headache associated temporally with onset of breastfeeding or with breast engorgement</td>
<td>History and physical examination</td>
</tr>
</tbody>
</table>

The ICHD-II criteria state that caffeine-withdrawal headache occurs upon cessation of ≥ 200 mg daily caffeine consumption for more than 2 weeks. However, others have suggested that caffeine-withdrawal headache may occur after as little as 3 days’ exposure to 300 mg/day, or 7 days’ exposure to 100 mg/day.
BOX 31-1 International Classification of Headache Disorders (ICHD-II) Classification

Primary
- Migraine
- Tension-type headache
- Cluster headache and other trigeminal autonomic cephalalgias
- Other primary headaches

Secondary
- Headache attributed to:
  - Head and/or neck trauma
  - Cranial or cervical vascular disorder
  - Nonvascular intracranial disorder
  - A substance or its withdrawal
  - Infection
  - Disorder of homeostasis
  - Disorder of the cranial structures (e.g., eyes, ears, nose, sinuses, teeth, mouth)
  - Psychiatric disorder
- Cranial neuralgias and central causes of facial pain
- Other headache, cranial neuralgia, central or primary facial pain


without a history of dural puncture. Approximately 11% of postpartum headaches are diagnosed as musculoskeletal. Other causes of secondary headache are discussed in the following paragraphs.

HYPERTENSION
Gestational hypertension is commonly associated with headache. Eclampsia is a form of hypertensive encephalopathy characterized by headache, visual disturbances, nausea, vomiting, seizures, stupor, and sometimes coma. Seizures may occur in the absence of severe hypertension. Headache is a serious premonitory sign, being present in over 50% of women in whom eclampsia develops. Eclampsia first manifests postpartum in 11% to 44% of affected women. Other hypertensive disorders, with or without superimposed pre-eclampsia, may also cause postpartum headache and lead to encephalopathy.

POSTERIOR REVERSIBLE LEUKOENCEPHALOPATHY SYNDROME
Posterior reversible (leuko)encephalopathy syndrome (PRES) was described in 1996 following recognition of a consistent symptom presentation in a diverse group of patients. Conditions associated with PRES include pre-eclampsia, uremia, hemolytic-uremic syndrome, and exposure to immunosuppressant drugs. Approximately 25% of cases of PRES occur in pregnant patients. PRES symptoms include headache, seizures, altered mental status, visual changes, and, occasionally, focal neurologic deficits.

The neuroradiologic features of PRES include symmetric areas of cerebral edema, predominantly involving the white matter regions of the posterior circulation (i.e., occipital lobes, posterior parietal, and temporal lobes) (Figure 31-1). The pathophysiology of PRES is similar to that of hypertensive encephalopathy, in that altered cerebrovascular regulation causes loss of blood-brain barrier integrity. The accompanying vasogenic edema can be reversed by prompt recognition and supportive therapy (i.e., cessation of provocative medications, aggressive treatment of hypertension, seizure prophylaxis). However, irreversible cytotoxic edema with permanent neurologic damage can occur if the initial disorder is not diagnosed early. This syndrome often manifests in the postpartum period, usually in conjunction with preeclampsia. Diagnosis may be delayed if other potential causes of headache are also present (e.g., dural puncture). Typical features that distinguish PRES from other postpartum headaches include seizures and focal neurologic deficits, such as temporary loss of vision.

SUBARACHNOID HEMORRHAGE
The incidence of subarachnoid hemorrhage is increased during pregnancy, being estimated at approximately 20 per 100,000 deliveries. It usually occurs in patients with a cerebral aneurysm, arteriovenous malformation, or hypertensive encephalopathy. The classic presentation consists of the sudden onset of a severe headache that is unlike any previous headache. Pregnancy may increase the risk of bleeding because of increased blood volume and hormonal changes that affect arterial integrity. Other factors associated with subarachnoid hemorrhage during pregnancy include cigarette smoking, genetic diseases (e.g., polycystic kidney disease), and nulliparity. Suspicion of subarachnoid hemorrhage necessitates urgent investigation by computed tomography (CT), because nonsurgical therapies (e.g., endovascular ablation) are available and long-term sequelae can be minimized.

CORTICAL VEIN THROMBOSIS
The incidence of cerebral cortical vein thrombosis is increased in pregnancy, and is estimated to be 10 to 20 per 100,000 deliveries in developed countries. The incidence appears higher in developing countries (e.g., 450 per 100,000 deliveries in India). Often it is difficult to distinguish cortical vein thrombosis from PDPH, because the headache of cortical vein thrombosis may have a postural component. Preceding dural puncture has been reported in several cases, and it has been hypothesized that the reductions in cerebrospinal fluid (CSF) pressure and cerebrovascular vasodilation that accompany dural puncture predispose to thrombosis development. Associated features include focal neurologic signs, seizures, and coma. Cerebral infarction may ensue if diagnosis is delayed. Diagnosis is best confirmed by magnetic resonance imaging (MRI), because CT appears to identify only a third of cases. Treatment of cortical vein thrombosis largely is symptomatic, with the aim of preventing seizures. Some studies have suggested that anticoagulation therapy may improve outcome; however, additional data are needed.

CEREBRAL INFARCTION/ISCHEMIA
Cerebral arterial insufficiency is one cause of stroke in pregnancy, with an estimated incidence of 19 per 100,000 deliveries. Approximately half of the events occur in the peripartum period, and the clinical presentation often
comprises a sudden onset of headache, vomiting, seizures, and focal neurologic deficits. Postpartum cerebral angiopathy has been detected with the aid of cerebral angiography, in which a characteristic “arterial beading” indicative of arterial spasm is evident. Several case reports have described the delayed diagnosis of cerebral infarction because providers assumed a diagnosis of PDPH.\(^{25,26}\) The key feature was a nonpostural headache. Initial CT and MRI findings are often normal, and intracranial Doppler or angiographic investigations may be necessary to diagnose ischemia or infarct.

**SUBDURAL HEMATOMA**

In rare instances, dural puncture is associated with the subsequent development of a subdural hematoma. In several case reports, the identification of the subdural hematoma was preceded by symptoms of a PDPH.\(^{27-29}\) Dural puncture results in leakage of CSF and decreased intracranial pressure (ICP). Presumably, the reduction in ICP causes stress on bridging cerebral vessels, thereby precipitating bleeding. Neurologic signs of subdural hematoma are variable but include evidence of increased ICP (e.g., headache, somnolence, vomiting, confusion) and focal abnormalities.

**PNEUMOCEPHALUS**

The subdural or subarachnoid injection of air used for identification of the epidural space may be associated with the sudden onset of severe headache, sometimes accompanied by neck pain, back pain, or changes in mental status.\(^{30}\) Headache symptoms can mimic those of PDPH, in that they are worse in the sitting position and may be relieved by lying down. Radiologic studies confirm the presence of intracranial air, and the headache typically resolves over the first week.

**MENINGITIS**

The severe headache of meningitis typically manifests within the first several postpartum days (see Chapter 32). It is accompanied by fever, nuchal rigidity, and the presence of Kernig and Brudzinski signs. Lethargy, confusion, vomiting, seizures, and a rash may also occur. Usual pathogens include streptococci of the viridans type.\(^{31}\) The diagnosis is confirmed by examination and culture of the CSF.

**PSEUDOTUMOR CEREBRI/BENIGN INTRACRANIAL HYPERTENSION**

Parturients with pseudotumor cerebri (i.e., increased ICP in the absence of a mass lesion) present with headache and visual disturbances, usually in the antepartum period. The features of the postpartum headache of pseudotumor cerebri mimic the usual chronic headache symptoms experienced by the patient. The diagnosis largely is one of exclusion (see Chapter 49). Treatment involves reduction of CSF pressure through the use of glucocorticoids, carbonic-anhydrase inhibitors, diuretics, or serial lumbar punctures.

**BRAIN TUMOR**

Intracranial tumors may manifest as postpartum headache.\(^{32-34}\) Headache that is dull rather than throbbing in character may be an early symptom of a brain tumor. Nausea, vomiting, seizures, and/or focal neurologic signs may be present. Neurologic examination may reveal evidence of increased ICP. Case reports suggest that atypical presentation of headache, either with persisting headache

---

**FIGURE 31-1** Posterior reversible (leuko)encephalopathy syndrome (PRES). **A,** Areas of hypodensity in the parieto-occipital white matter (black arrows). **B,** Abnormal hypodensity is also seen in the cerebellum (white arrows), with the abnormality larger on the left (L) than the right (R). (From Hinchey J, Chaves C, Appignani B, et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med 1996; 334:494-500.)
symptoms in the supine position or exacerbation following epidural blood patch, should prompt further neuroradiologic investigations.32-34

SPONTANEOUS INTRACRANIAL HYPOTENSION
Spontaneous intracranial hypotension develops because of CSF leakage secondary to dural tears. The tears usually occur at the thoracic spinal level and are not associated with prior neuraxial procedures.35 Diagnosis requires radioisotope cisternography and CT myelography, which may also identify the level of the leak. Presentation of this disorder is identical to that of PDPH, as the pathophysiology is the same. The only difference is the lack of a prior neuraxial procedure in spontaneous intracranial hypotension. One case report has described the development of a postural headache 4 days after a spontaneous vaginal delivery without neuraxial anesthesia.36 The patient was found to have a cervical-thoracic dural leak.

SINUSITIS
Headache caused by inflamed paranasal sinuses is associated with purulent nasal discharge and, occasionally, fever. Pain may be unilateral or bilateral, depending on the extent of the disease, and the skin over the affected sinus may be tender. Frontal sinus infection causes headache in the frontal region. Ethmoidal and sphenoidal sinus infections cause periorbital pain, and maxillary sinus infection may cause diffuse facial discomfort. The sinuses fill overnight, and pain typically is worse on awakening. Pain improves in the upright position, which assists drainage.37

CAFFEINE WITHDRAWAL
The withdrawal of caffeine may lead to headache, increased fatigue, and anxiety. Caffeine withdrawal headaches have been reported in the postoperative period and may occur after as little as 3 days’ exposure to 300 mg/day or 7 days’ exposure to 100 mg/day of caffeine.39 Normal-sized caffeinated drinks usually contain 50 to 100 mg of caffeine per serving. Women often decrease their caffeine intake in the puerperium, and although caffeine withdrawal headache has not been confirmed as a cause of postpartum headache, the diagnosis should be considered.

LACTATION HEADACHE
Askmark and Lundberg40 reported episodes of intense headache during periods of breast-feeding in a woman known to suffer from migraine. Onset of headaches occurred within the first few minutes of breast-feeding, and the headaches resolved after cessation of nursing. The headaches were associated with an increase in plasma vasopressin concentration. Headaches have also been described in women with breast engorgement who either have elected not to breastfeed or have reduced the frequency of breast-feeding.41

POST–DURAL PUNCTURE HEADACHE

Incidence

Post–dural puncture headache (PDPH) may occur after intentional dural puncture with a spinal needle or unintentional dural puncture with an epidural needle.
A meta-analysis of studies of PDPH in obstetric patients (n = 328,769) calculated a pooled risk of unintentional dural puncture with any epidural needle of 1.5% (95% confidence interval [CI], 1.5% to 1.5%). Following a dural puncture with an epidural needle, the risk of PDPH was 52.1% (95% CI, 51.4% to 52.8%) (Figure 31-2). The rate of PDPH after dural puncture with spinal needles ranged between 1.5% and 11.2%, depending on the needle size and type of needle (see later) (Table 31-2). Although PDPH is often considered a “minor” complication of dural puncture, it was the cause of 14% of obstetric claims in the American Society of Anesthesiologists (ASA) Closed-Claims Project database (see Chapter 33).

**Symptoms**

 Patients typically experience headache pain in the frontal and occipital regions. Pain often radiates to the neck, which may be “stiff.” Some women have a mild headache that permits full ambulation. In others, pain is severe and incapacitating. Symptoms are worse in the upright position and are usually relieved in the horizontal position. Abdominal compression may relieve pain in some patients. The ICHD-II defines PDPH as occurring within 15 minutes of a patient’s moving to an upright position (sitting or standing) and resolving within 15 minutes of the patient’s moving to the supine position.3 The ICHD-II criteria for PDPH require one of the following symptoms to be present: neck stiffness, tinnitus, hypacusis, photophobia, and nausea. Lybecker et al.43 reported the incidence of these symptoms in a prospective study of 75 nonobstetric patients with PDPH (Table 31-3). Cranial nerve palsy, thought to be secondary to nerve traction due to low CSF volume, is occasionally associated with PDPH. The sixth cranial nerve is most susceptible to traction during its long intracranial course. The traction results in failure of the involved eye to abduct, and patients may have diplopia. Hearing loss is usually in the low-frequency range and is related to reduction of CSF pressure and alteration of hair cell position in the inner ear. The risk of hearing loss appears to be higher with advanced age (more than 40 years) and dural puncture with larger-gauge needles.

Rarer symptoms associated with PDPH include seizures, abdominal pain, and diarrhea. Shearer et al.45 reported postpartum seizures in eight women with severe PDPH without convincing evidence of preexisting hypertension. Cerebral vasospasm was thought to be the etiologic factor. Vercauteren et al.46 described a woman who had received epidural analgesia and experienced postpartum seizures with a severe headache. The CSF was blood-stained, but a CT scan showed no source of hemorrhage. This patient improved dramatically after the performance of a blood patch. A 20-year-old man experienced PDPH after administration of spinal anesthesia with a 25-gauge Quincke needle.47 Twelve days after the anesthetic administration, he experienced abdominal pain and diarrhea, which persisted despite the lack of evidence of mucosal or organic disease. One month following spinal anesthesia, an epidural blood patch procedure was performed, which resulted in prompt resolution of headache and gastrointestinal symptoms. The investigators speculated that this patient had noninfectious arachnoiditis with lumbosacral root involvement, which resulted in visceral hyperalgesia and dysfunction.

**Onset and Duration**

Headache typically occurs on the first or second day after dural puncture; by ICHD-II criteria, it must appear within 5 days of dural puncture.3 Ninety-five percent of PDPH headaches last less than a week. The National Obstetric Anaesthetic Database project of the Obstetric Anaesthetists’ Association of the United Kingdom demonstrated that 75% of 975 women with PDPH suffered from headache-associated difficulty in performing the activities of daily living.48 Rarely, symptoms may persist for months or even years.49

### TABLE 31-2 Frequency of Post–Dural Puncture Headache in Obstetric Patients According to Spinal Needle Design

<table>
<thead>
<tr>
<th>Needle Design</th>
<th>Gauge</th>
<th>n/N</th>
<th>Frequency of PDPH (%)</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quincke†</td>
<td>24</td>
<td>15/238</td>
<td>11.2</td>
<td>10.2-12.2</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>90/1624</td>
<td>6.3</td>
<td>6.3-6.3</td>
</tr>
<tr>
<td></td>
<td>26</td>
<td>139/2467</td>
<td>5.6</td>
<td>5.6-5.7</td>
</tr>
<tr>
<td></td>
<td>27</td>
<td>28/1007</td>
<td>2.9</td>
<td>2.8-3.0</td>
</tr>
<tr>
<td>Atraucan‡</td>
<td>26</td>
<td>16/350</td>
<td>4.6</td>
<td>2.6-7.3</td>
</tr>
<tr>
<td>Whitacre†</td>
<td>22</td>
<td>1/68</td>
<td>1.5</td>
<td>1.2-2.8</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>103/6366</td>
<td>2.2</td>
<td>2.2-2.2</td>
</tr>
<tr>
<td></td>
<td>27</td>
<td>10/668</td>
<td>1.7</td>
<td>1.6-1.8</td>
</tr>
<tr>
<td>Sprotte‡</td>
<td>24</td>
<td>57/1767</td>
<td>3.5</td>
<td>3.5-3.5</td>
</tr>
<tr>
<td>Polymedic†</td>
<td>25</td>
<td>22/292</td>
<td>6.6</td>
<td>5.9-7.4</td>
</tr>
<tr>
<td>BD‡</td>
<td>26</td>
<td>205/2560</td>
<td>5.8</td>
<td>5.6-5.9</td>
</tr>
</tbody>
</table>

n, number of headaches; N, total number of procedures; PDPH, post–dural puncture headache.

*Meta-analysis of estimated pooled event rate using a random effects model.

†Data from reference 42.

‡Data from references 71-73.

### TABLE 31-3 Symptoms Associated with Post–Dural Puncture Headache

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Incidence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nausea</td>
<td>60</td>
</tr>
<tr>
<td>Vomiting</td>
<td>24</td>
</tr>
<tr>
<td>Neck stiffness</td>
<td>43</td>
</tr>
<tr>
<td>Ocular*</td>
<td>13</td>
</tr>
<tr>
<td>Auditory†</td>
<td>12</td>
</tr>
</tbody>
</table>

*Ocular symptoms include photophobia, diplopia, and difficulty in accommodation.

†Auditory symptoms include hearing loss, hypacusis, and tinnitus.

Imaging

Imaging investigations are not routinely recommended for the postpartum patient with a PDPH unless the symptoms suggest other diagnoses and the diagnosis of PDPH is in doubt. Contrast-enhanced MRI is the method of choice to study the meninges and has revealed characteristic findings of PDPH.50,51 These findings include marked, diffuse contrast enhancement with thickening of the dura mater, occasional extradural fluid collections, and enhancement and expansion of the superior sagittal sinus. The enlarged venous sinus may represent compensatory venous expansion in response to low CSF pressure.51

Pathophysiology

Debate continues regarding the precise etiology of PDPH symptoms. The original theory was that pain-sensitive nerve fibers were stimulated by a downward shift of the brain secondary to a loss of CSF volume. German surgeon August Bier52 is credited with the first description of PDPH after his pioneering work on spinal anesthesia with cocaine. Bier and an assistant performed spinal anesthesia on each other, using blows to the shin with an iron hammer and application of a burning cigar to the skin to demonstrate sensory blockade.52 Both experienced severe PDPH. The assistant forced himself to work the next day, but Bier stayed home for 9 days. Bier suggested that the PDPH might be caused by CSF loss. Today there is no doubt that leakage of CSF initiates the syndrome. Kunkele et al.53 consistently produced PDPH by draining 20 mL of CSF from volunteers. Symptoms were relieved immediately by subarachnoid injection of saline to restore initial CSF pressure.

Total CSF volume is estimated to be 150 mL, and the production rate is approximately 0.35 mL/min, or a daily production rate of 500 mL. The rate of CSF leakage through a dural hole may exceed the rate of CSF production. If this occurs, low CSF pressure results in a loss of the cushioning effect provided by intracranial fluid.

CSF pressure during labor is normal between contractions but increases significantly during painful contractions and expulsive efforts.54 Effective epidural analgesia attenuates the development of headache.55 However, with development of headache symptoms, the mean epidural pressure measurements were found to be decreased significantly.

Not all parturients with PDPH have decreased CSF pressure, and not all parturients with a significant CSF leak experience symptoms.56 The pain of PDPH may be caused, in part, by an increase in cerebral blood flow (and cerebral vasodilation) as a consequence of low CSF pressure or volume. This phenomenon has been observed in animals.57,58 The inverse relationship between intracranial blood volume and CSF volume reflects the body’s effort to maintain a constant intracranial volume.59 The lumbar CSF compartment is a dynamic structure that acts as a reservoir for intracranial CSF volume adjustment. The occurrence of cerebral vasodilation may explain the relief of headache symptoms with vasoconstrictors such as caffeine, theophylline, and sumatriptan.

Risk Factors

In a classic study of 10,098 spinal anaesthetic procedures published in 1956, Vandam and Dripps60 noted three demographic factors associated with PDPH: age, gender, and pregnancy. The analysis did not allow determination as to whether these factors were independent risk factors. Subsequently, other risk factors for development of PDPH have been identified.

AGE

Extensive evidence supports the observation that PDPH is uncommon in patients older than 60 years and is most common in patients younger than 40 years. In the elderly, the dura may be inelastic and less likely to gape after puncture. CSF leakage may be impeded by adhesions and calcification. The cerebrovascular system also may be less reactive in older patients. Further, this group is less active physically, and older patients may be less likely to complain.

GENDER

Vandam and Dripps60 observed a twofold higher incidence of PDPH in women than in men (14% versus 7%, respectively). This difference may be related to differences in cerebral vascular reactivity, because it is well known that migraine occurs predominantly in females and is influenced by hormonal changes. Women may have enhanced vascular reactivity, or perhaps changes in cerebral blood flow are more likely to produce pain in women than in men. A meta-analysis of randomized clinical trials identified a twofold higher risk for PDPH in nonpregnant females than in males.61

VAGINAL DELIVERY

Vandam and Dripps60 also observed a high incidence of PDPH (22%) after vaginal delivery. This higher incidence may be a result of the mechanical consequences of expulsive efforts during the second stage of labor and/or postpartum hormonal changes in cerebrovascular reactivity. Expulsive efforts during the second stage of labor may increase CSF leakage. This possibility has prompted some physicians to restrict maternal pushing after unintentional dural puncture and to use forceps to shorten the second stage of labor. In a 20-year retrospective review of 460 parturients who experienced unintentional dural puncture during labor at the Birmingham Maternity Hospital in the United Kingdom, Stride and Cooper62 did not identify a lower incidence of PDPH in women who underwent prophylactic forceps delivery than in those who had spontaneous vaginal delivery. In contrast, in a retrospective review of the records of 33 laboring women who had experienced unintentional dural puncture, Angle et al.63 found that women who were allowed to push were much more likely to experience PDPH (relative risk, 7.4; 95% CI, 1.1 to 48.2) and also were more likely to require an epidural blood patch.

MORBID OBESITY

Some evidence suggests that morbidly obese patients are less susceptible to PDPH and are also less likely to receive an epidural blood patch for treatment of PDPH,64 suggesting either a reduced severity of PDPH or anesthesia provider reticence to perform an epidural blood patch in this patient population. Possible but unproven explanations for the lower incidence and severity of PDPH in obese
patients include increased abdominal pressure (which may reduce the extent of CSF leak) and/or reduced physical activity of these patients. Other confounding factors, such as differences in the mode of delivery (higher rate of cesarean delivery) and neuraxial opioid administration, may also play a role.

AIR TRAVEL
PDPH has been reported during air travel after spinal anesthesia. Presumably, the headache is precipitated by a change in the gradient between the subarachnoid space and the epidural space due to decreased atmospheric pressure.

HISTORY OF PREVIOUS POST–DURAL PUNCTURE HEADACHE
A history of PDPH after previous spinal anesthesia is associated with the development of PDPH with subsequent spinal anesthesia. A cohort of nonobstetric women with a history of previous spinal anesthesia were monitored prospectively after a second spinal anesthesia procedure. Those with a previous history of PDPH were 2.3 times more likely (95% CI, 1.0 to 5.1) to have a second PDPH than women without a history of headache (24.2% versus 10.6%, respectively). This finding suggests that certain individuals are predisposed to the development of PDPH.

MULTIPLE DURAL PUNCTURES
Seeberger et al. found that multiple dural punctures significantly increased the risk for PDPH. Surgical patients who received a second spinal injection owing to failure of the initial spinal injection had a 4.2% incidence of PDPH, compared with a 1.6% incidence among patients who had a single dural puncture.

NEURAXIAL ANESTHETIC TECHNIQUE
Technical factors related to the neuraxial technique influence the incidence of PDPH.

Epidural Needle Size/Design
The high rate of PDPH following unintentional dural puncture with an epidural needle has led investigators to alter the epidural needle design or size in an attempt to reduce headache incidence or severity. Data on the success of this endeavor are conflicting. In an in vitro study using cadaver dura, no differences were found in fluid leak rate among punctures made with Hustead, Tuohy, Crawford, and Sprotte epidural needles. In contrast, in an in vivo study, Morley-Forster et al. observed a lower incidence of PDPH with use of an 18-gauge Sprotte needle than with use of the standard 17-gauge Tuohy needle, despite there being no difference in the unintentional dural puncture rate. A pilot study has examined the utility of a 19-gauge Tuohy needle with a 23-gauge epidural catheter.

Spinal Needle Design
Historically the beveled, cutting-tip Quincke needle (Figure 31-3) has been the most widely used needle for dural puncture for both diagnostic and anesthetic purposes (see Chapter 12). A modification of the Quincke needle, the Atraucan needle, has a cutting tip and a double bevel, which are intended to cut a small dural hole and then dilate it. Clinical experience with the Atraucan needle has been generally good, although studies appear to suggest that the use of this needle is associated with a higher risk of PDPH than the use of a noncutting, pencil-point needle (see Table 31-2).

In 1951, Hart and Whitacre introduced a solid-tipped, pencil-point spinal needle with a lateral injection port, which is now known as the Whitacre design (see Figure 31-3). They believed that their needle would stretch and separate rather than cut the dural fibers, resulting in a lower incidence of PDPH. Currently, both 25- and 27-gauge Whitacre needles are very popular, and studies have confirmed the anticipated low incidence of PDPH. A randomized comparison of 27-gauge Quincke and pencil-point needles found a significantly lower incidence of PDPH in the pencil-point needle group. In vitro evidence suggests that the rate of fluid leak is lower through the dural puncture site after use of a pencil-point needle than after use of a beveled needle.

With the recognition that pencil-point spinal needle tips reduce the incidence of PDPH, other tip designs have appeared. In 1987, Sprotte et al. reported experience with the use of a needle that was designed to reduce the risk of neural and dural trauma (see Figure 31-3). The Sprotte needle has a solid oval tip and a longer orifice than the Whitacre needle. The incidence of PDPH was 0.02% with its use in a heterogeneous population of almost 35,000 patients. Subsequent studies have shown that the incidence of PDPH with the Sprotte needle is lower than that with Quincke needles of smaller gauge (see Table 31-2). Other noncutting needle products are the Gertie Marx needle and the Polymedic needle.

Spinal Needle Size
With the Quincke needle, the incidence and severity of PDPH are directly related to the size of the needle. The incidence of PDPH appears to be lower with a 27-gauge needle than with 25- and 26-gauge needles (see Table 31-2). A similar relationship may exist with pencil-point needles. When needles smaller than 27-gauge are used, the incidence of PDPH is very low, but technical problems with needle insertion and failure to produce adequate

![Figure 31-3 Designs of spinal needle tips (not to scale).](image-url)
anesthesia are more common.\textsuperscript{79} Locating the epidural space before insertion of the spinal needle (e.g., using the epidural needle as an introducer needle) may improve the rate of success with fine-gauge needles. The current popularity of spinal anesthesia in obstetric patients is largely a result of new needle technology, which has led to a reduction in the incidence of PDPH. Because of the morbidity associated with PDPH, every effort should be made to use a needle associated with a low incidence of PDPH (e.g., a small-gauge, noncutting needle). There are times when urgency or body habitus dictates the use of a larger needle, but there is seldom justification for using a Quincke needle larger than 27-gauge.

Direction of Bevel of the Quincke Needle

Studies have confirmed that puncturing the dura mater with the Quincke needle bevel parallel to the long axis of the spine is associated with an incidence of PDPH 70% lower than that associated with a perpendicular orientation.\textsuperscript{80} An early study by Franksson and Gordh\textsuperscript{81} demonstrated that orientation of the bevel of a Quincke spinal needle parallel to the long axis of the spine produced less dural trauma than occurred when the bevel was inserted perpendicularly. These investigators thought that the dural fibers were predominantly longitudinal in direction. Electron microscopy has shown that the dural structure is far more complex than originally was supposed. Fink and Walker\textsuperscript{82} noted that the dura consists of multidirectional interlacing collagen fibers and both transverse and longitudinal elastic fibers. They suggested that the insertion of the needle with the bevel parallel to the long axis of the spine most likely results in less tension on the dural hole, and therefore, a smaller aperture with less CSF leak. \textit{In vitro} studies of bevel orientation and fluid leak have provided conflicting results.\textsuperscript{83,84} However, despite confusing anatomic evidence, clinical experience strongly supports insertion of the Quincke needle with the bevel parallel to the long axis of the spine.

Direction of the Bevel of the Tuohy Needle

Norris et al.\textsuperscript{85} examined two groups of women who received epidural anesthesia with a Tuohy needle. In one group the needle was inserted with the bevel perpendicular to the long axis of the spine. In the other group the needle entered the epidural space with the bevel parallel to the long axis and was rotated 90 degrees before insertion of the catheter. The investigators observed a lower incidence of PDPH in the latter group. However, some anesthesia providers argue that rotation of the needle within the epidural space may increase the risk of unintentional dural puncture. Richardson and Wissler\textsuperscript{86} randomly assigned laboring patients to a cephalad or lateral orientation of the Tuohy bevel during epidural needle insertion. The needle was not rotated before insertion of the epidural catheter. There was no difference in dural puncture rates, but catheter insertion was easier with a cephalad orientation of the bevel.\textsuperscript{86}

Midline or Paramedian Approach

There is conflicting evidence as to whether the spinal needle approach affects the incidence of PDPH. Hatfalvi\textsuperscript{87} reported no cases of PDPH in a retrospective survey of 4465 spinal anesthesia procedures. This investigator used a paramedian approach with a 20-gauge Quincke needle, and the skin was punctured 3 cm from the midline. He suggested that tangential dural puncture creates a dural flap and prevents PDPH. In contrast, Viitanen et al.\textsuperscript{88} prospectively monitoring obstetric patients after administration of single-shot spinal analgesia for labor (27-gauge Quincke needle), observed PDPH in 3 of 85 (3.5%) patients in whom the midline approach was used, compared with 15 of 127 (11.8%) patients in whom the paramedian approach was used. Using a rigid paper cylinder model of the dura, Kempen and Mocek\textsuperscript{89} studied midline and paramedian punctures with a 22-gauge Quincke needle in different orientations. With midline punctures, all entry and exit holes were of uniform size regardless of bevel orientation, and no dural flaps were seen. After paramedian punctures, flaps formed when the needle bevel faced the cylinder surface with a near-tangential angle of perforation.

Skin Preparation

In a nonrandomized, nonblinded study reported in a letter, Gurmaršik\textsuperscript{90} found that the removal of dried povidone-iodine from the skin before placement of the spinal needle was associated with a lower incidence of PDPH (6% versus 0%). This investigator recommended removal of the povidone-iodine before insertion of the spinal needle, and suggested that chemical meningismus resulting from povidone-iodine introduced into the intrathecal space by the spinal needle contributed to PDPH. This finding has not been confirmed by other investigators. (Editors’ Note: We do not recommend removal of povidone-iodine before initiating neuraxial analgesia, because povidone-iodine works by desiccating bacteria, and removing the povidone-iodine reduces its antibacterial effect.)

Air versus Saline Method of Locating the Epidural Space

The method of locating the epidural space may influence the incidence of PDPH.\textsuperscript{91} Many anesthesia providers have adopted the loss-of-resistance-to-saline technique in the belief that it is associated with a lower incidence of unintentional dural puncture and PDPH than the use of air.\textsuperscript{82,92} However, the data are inconsistent, and not all studies have found a difference.\textsuperscript{92,94} In a study of epidural steroid injections (at all spinal levels) in a pain clinic, a single practitioner performed more than 3700 procedures, alternating weekly between use of the loss-of-resistance to air and saline methods.\textsuperscript{95} There was no difference between groups in the incidence of unintentional dural puncture. Although the incidence of postprocedure headache was lower in the saline group, the definition of headache was not rigorous, and it was not possible to differentiate between headache from pneumocephalus and classic PDPH.\textsuperscript{95}

Choice of Local Anesthetic Drug for Spinal Anesthesia

Naulty et al.\textsuperscript{96} reported that the use of bupivacaine-glucose or lidocaine-glucose for spinal anesthesia was associated with a higher incidence of PDPH than use of tetracaine-procaine. They postulated that osmotic, cerebral (meningeal) irritant and/or cerebrovascular effects of the glucose could be responsible for these findings.

Continuous Spinal Anesthesia

A multicenter trial published in 2008 reexamined the safety and utility of 28-gauge microcatheters for spinal labor analgesia.\textsuperscript{97} There was no difference in the incidence of
PDPH (9% versus 4%, respectively) or epidural blood patch (5% versus 2%, respectively), between women randomly assigned to receive an intrathecal catheter and those who received an epidural catheter; however, the study was insufficiently powered to assess these outcomes. Spinal microcatheters currently are not commercially available in North America.

**Combined Spinal-Epidural Anesthesia**

Combined spinal-epidural (CSE) analgesia/anesthesia is widely used for labor analgesia and, to a lesser extent, for cesarean delivery. Intuitively, it seems that the incidence of PDPH should be identical to that after single-shot spinal anesthesia with the same size and type of needle. Many anesthesia providers believe that the intentional dural puncture with the CSE technique is associated with a higher incidence of PDPH than epidural anesthesia alone. However, the available evidence, primarily from observational studies, suggests that the risk of PDPH is not increased with the CSE technique. \(^{98-100}\) PDPH rates for the CSE technique in these three studies were 1.7%, 0.43%, and 1.4%, respectively, compared with 1.6%, 0.45%, and 0.8% for the epidural technique. \(^{98-100}\) Initial placement of the epidural needle facilitates precise dural puncture, and the subsequent increase in epidural space pressure after the epidural injection of local anesthetic may reduce CSF leakage. If the anesthesia provider is in doubt about correct epidural needle placement, a needle-through-needle dural puncture might resolve the issue and prevent unintentional dural puncture with a large-gauge epidural needle.

**Complications**

The immediate problems associated with PDPH include (1) the inability to perform the activities of daily living, such as providing care for the newborn; (2) an extended duration (almost one full day) of hospitalization; and (3) a higher number of emergency room visits, with almost 40% of patients returning for at least one visit.\(^ {101}\) Although these complications are very bothersome to the patient, they are short-lived and do not result in long-term morbidity. However, rare but serious complications may occur after dural puncture and PDPH.

Zeidan et al.\(^ {102}\) reviewed the published reports of subdural hematoma after dural puncture. They found that subdural hematoma was associated with new neurologic symptoms in addition to changing headache characteristics. The proposed mechanism of subdural hematoma development is ongoing intracranial hypotension leading to caudal movement of the brain and rupture of fragile, bridging subdural veins. These cases have been managed with an epidural blood patch as well as with neurosurgical decompression.

**Dural sinus thrombosis** has been documented after unintentional dural puncture and treatment of PDPH with an epidural blood patch.\(^ {18,20}\) Responsible factors may be cerebral venous dilation (associated with decreased ICP) and the hypercoagulability that occurs during pregnancy. Therapy may include anticoagulation.

**Diplopia** or **hearing loss** after dural puncture, secondary to cranial nerve dysfunction, may be permanent, even after successful treatment of the PDPH with an epidural blood patch. A review of 95 cases of neurapraxia or axonotmesis of the ocular cranial nerves concluded that symptoms may last from 2 weeks to 8 months but that almost 90% of patients recover.\(^ {103}\)

Two surveys from a single institution have attempted to estimate long-term morbidity arising from unintentional dural puncture or spinal anesthesia in obstetric patients.\(^ {104,105}\) Women delivering between 1978 and 1985,\(^ {104}\) and between 1991 and 1996,\(^ {105}\) were asked to recall symptoms beginning after their deliveries, including back pain and headache. The responses of women with unintentional dural puncture or PDPH following spinal anesthesia were compared with those of women who had uneventful procedures. The first study found that 18% of women with unintentional dural puncture had complaints of frequent headaches or neck ache, compared with only 7% of women experiencing uneventful neuraxial procedures.\(^ {104}\) The later study did not confirm a higher risk of headache with prior unintentional dural puncture but, instead, found a higher rate of backache symptoms.\(^ {105}\) These results have not been confirmed in a prospective study or by other investigators and likely suffer from the limitations of surveys that rely on patient recall.

**Prevention**

A 2005 survey of British obstetric anesthetists identified the frequency of practices and maneuvers that many believe will reduce the likelihood of a PDPH after unintentional dural puncture with an epidural needle.\(^ {106}\) These include encouraging postpartum fluid intake (91%) and regular analgesia administration (83%), and placement of an intrathecal catheter (15%) at the time of the unintentional dural puncture. Older practices, such as avoiding pushing during the second stage, restricting postpartum mobility, and prophylactic epidural administration of saline or autologous blood, appear to be declining in use.

**POSTURE**

In a Cochrane systematic review, Sudlow and Warlow\(^ {107}\) reviewed the evidence for reducing the incidence of PDPH by use of bed rest rather than early mobilization (usually within 6 hours of dural puncture). The review included the only randomized trial of obstetric patients, and results were consistent for all patient types; there was no benefit for bed rest over early mobilization (PDPH incidence 31% versus 27%, respectively). It is important to encourage early ambulation during the puerperium. Pregnant women are hypercoagulable and at higher risk for deep vein thrombosis and pulmonary embolism, and immobility increases this risk (see Chapter 38).

**HYDRATION**

Despite the widespread practice of encouraging women to increase oral fluid intake after unintentional dural puncture, there is little evidence that greater hydration prevents PDPH. The Cochrane review identified only one randomized trial of 100 nonobstetric patients.\(^ {107}\) There was no difference in the incidence of PDPH in patients randomly assigned to receive either 3 L or 1.5 L of fluid per day.

**ABDOMINAL Binder**

In a 1975 study, the use of a tight abdominal binder placed immediately after delivery and continued until discharge
home apparently was successful in reducing the incidence of PDPH. The study included parturients who received single-shot spinal anesthesia through 22-gauge cutting needles, which are more likely to lead to PDPH than modern-day spinal needles. However, the application of this technique to prevent PDPH after unintentional dural puncture with an epidural needle has not been evaluated. Abdominal binders are seldom used today.

CAFFEINE
Two clinical trials in nonobstetric patients have evaluated the efficacy of oral caffeine to prevent PDPH, but neither study showed a reduction in the incidence of headache. At this time, prophylactic caffeine is not advocated for prevention of PDPH.

INTRASPINAL OPIOIDS
Earlier studies suggested that prophylactic neuraxial administration of a hydrophilic or lipophilic opioid does not reduce the incidence of PDPH after spinal anesthesia or unintentional dural puncture. However, in a randomized blinded trial published in 2008, 50 obstetric patients with unintentional dural puncture and subsequent epidural analgesia received epidural morphine 3 mg or saline-placebo after delivery and again at 24 hours before removal of the epidural catheter. The incidence of PDPH was 48% in the saline-placebo group and 12% in the morphine group. Although no complications were reported, we would caution against routine administration of epidural morphine in these circumstances until this finding is confirmed and further safety studies are undertaken. The movement of morphine across the dura is increased by the presence of a large-gauge needle puncture, possibly increasing the risk of respiratory depression.

INTRATHECAL CATHETER
Placing a 19- or 20-gauge epidural catheter into the intrathecal space after an unintentional dural puncture with an epidural needle has become an increasingly popular technique. The immediate benefits of an intrathecal catheter are reliable, low-dose labor analgesia and rapid-onset surgical anesthesia should it be required. Some authorities have speculated that the intrathecal catheter may reduce the immediate CSF leak into the epidural space by mechanical obstruction and induce an inflammatory fibrous reaction in the dura, which facilitates closure of the puncture site after removal of the catheter. Currently available studies are mostly retrospective and observational and lack rigorous outcome definitions and follow-up. Data from these studies are conflicting but suggest that intrathecal catheters do not significantly reduce the incidence of PDPH unless they are left in place for 24 hours after delivery (Table 31-4). The safety of this practice has not been well studied.

Randomized clinical trials to assess the effect of intrathecal catheters on the incidence of PDPH are desirable but would likely require multicenter participation owing to the infrequent occurrence of unintentional dural puncture.

**PROPHYLACTIC EPIDURAL/INTRATHECAL SALINE**
Trivedi et al. randomly assigned patients with unintentional dural puncture to receive a prophylactic epidural saline bolus (40 to 60 mL) or a prophylactic epidural blood patch (15 mL) given just prior to epidural catheter removal, or conservative therapy without a saline bolus or blood. The incidence of PDPH was no different between the saline and control groups (88% versus 67%, respectively). Shah studied 17 patients who received an epidural saline infusion (at a rate of approximately 40 mL/hr) for 24 to 36 hours after unintentional dural puncture. Four patients complained of severe interscapular pain, which resolved when the infusion rate was reduced. Severe PDPH developed in 47% of patients after the infusion was stopped. Despite these discouraging results, prophylactic epidural saline infusions continue to be popular in some centers. The saline infusion should not be initiated, however, until residual local anesthetic effects have resolved.

In a nonrandomized, nonblinded study of patients with unintentional dural puncture, Charsley and Abram

### TABLE 31-4 Rate of Post–Dural Puncture Headache after Unintentional Dural Puncture and Prophylactic Intrathecal Catheter Placement

<table>
<thead>
<tr>
<th>Study*</th>
<th>Study Design</th>
<th>Spinal Catheter, n/N (%)</th>
<th>No Spinal Catheter, n/N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Norris &amp; Leighton</td>
<td>Retrospective cohort; catheter left in place for 2 hr</td>
<td>19/35 (54)</td>
<td>11/21 (52)</td>
</tr>
<tr>
<td>Cohen et al.</td>
<td>Retrospective cohort: Catheter discontinued immediately after delivery</td>
<td>8/17 (47)</td>
<td>5/15 (33)</td>
</tr>
<tr>
<td>Dennehy &amp; Rosaeg</td>
<td>Catheter left in place for 24 hr</td>
<td>0/13 (0)</td>
<td>—</td>
</tr>
<tr>
<td>Paech</td>
<td>Case series; catheter left in place for 13-19 hr</td>
<td>0/3 (0)</td>
<td>—</td>
</tr>
<tr>
<td>Rutter et al.</td>
<td>Prospective cohort; catheter discontinued immediately after delivery</td>
<td>21/24 (87)</td>
<td>60/76 (79)</td>
</tr>
<tr>
<td>Ayed et al.</td>
<td>Retrospective cohort: Catheter left in place for unknown duration</td>
<td>24/34 (71)</td>
<td>30/37 (81)</td>
</tr>
<tr>
<td></td>
<td>Catheter discontinued immediately after delivery</td>
<td>18/35 (51)*</td>
<td>34/37 (92)</td>
</tr>
<tr>
<td></td>
<td>Catheter left in place for 24 hr</td>
<td>2/31 (6)*</td>
<td>—</td>
</tr>
</tbody>
</table>

*Different than no-catheter, P < .05.
reported that intrathecal injection of 10 mL of normal saline immediately before needle or catheter withdrawal resulted in a lower incidence of headache and the need for blood patch in patients so treated than in a non-randomized control group. The intrathecal injection of saline after unintentional dural puncture deserves further study.

**PROPHYLACTIC BLOOD PATCH**

Interest in the use of prophylactic epidural blood patch prior to removal of the epidural catheter arose after early observational studies suggesting that the incidence of PDPH was lower with such treatment. A 2001 Cochrane review identified two randomized studies involving 65 women, which demonstrated a reduction in the risk of any postpartum headache after unintentional dural puncture (odds ratio, 0.06; 95% CI, 0.02 to 0.15). However, this review highlighted the difficulty in estimating risk and benefit with small numbers of patients and PDPH events. In 2004 Scavone et al. reported a double-blinded trial in which 64 parturients with unintentional dural puncture were randomly assigned to receive 20 mL of autologous blood (prophylactic epidural patch) or a sham prophylactic blood patch. Both groups had a 56% incidence of PDPH; however, the duration of headache was shorter in the prophylactic blood patch group.

Because unintentional dural puncture with a 16- or 18-gauge epidural needle results in a high incidence of PDPH, some anesthesia providers believe that a prophylactic blood patch is always justified. Others argue that with such an approach, a significant number of patients would receive unnecessary treatment and that a blood patch is not devoid of complications. These latter anesthesia providers call attention to the potential for epidural catheters to become contaminated after prolonged use. The injection of blood through a contaminated epidural catheter may be associated with a higher risk of infection than injection through an epidural needle placed de novo for a therapeutic blood patch. A case report of a parturient who received a prophylactic epidural blood patch and was subsequently diagnosed with streptococcal septicemia highlights the potential risk for maternal infection during the puerperium.

If performed, a prophylactic blood patch procedure should be delayed until residual neuroblockade has resolved, for several reasons. First, the patient should have full return of sensation because the occurrence of pain is a signal for the anesthesia provider to stop the injection of blood. Second, evidence suggests that lidocaine may inhibit coagulation. Leivers reported a case of total spinal anesthesia after the epidural injection of 15 mL of blood before epidural anesthesia had regressed. This investigator speculated that residual lidocaine in the lumbar CSF was transferred to the brain as a consequence of an increase in lumbar CSF pressure produced by the patch. (Editors’ Note: One of us [D.H.C.] has observed one case of transient, total blindness after the rapid, bolus injection of 30 mL of epidural saline following vaginal delivery in a patient who had experienced unintentional dural puncture during labor. The blindness resolved after approximately 15 to 20 minutes, and subsequent ophthalmologic and neurologic findings were normal. The etiology of the transient blindness was unclear. Nonetheless, it seems prudent to delay administration of prophylactic epidural saline or blood until the nerve block has regressed and to avoid rapid epidural administration of blood or saline at any time.)

It is important to avoid the direct intrathecal injection of blood. Aldrete and Brown reported a case of intrathecal hematoma and arachnoiditis with prolonged neurologic sequelae after prophylactic blood patch. Nineteen milliliters of blood were injected through an epidural catheter that, in retrospect, was positioned in the subarachnoid space. There was considerable resistance to injection of the blood, and severe lower back pain with tinnitus accompanied the procedure.

**PROPHYLACTIC DEXTRAN PATCH**

Salvador et al. reported the prophylactic epidural injection of 20 mL of dextran-40 in 17 patients who had experienced unintentional dural puncture with a 17- or 18-gauge needle. Three of the patients were parturients, and none of the 17 patients experienced PDPH. This injection was performed before regression of the local anesthetic effect. No additional studies of this technique have been reported, so its safety and efficacy remain unclear.

**Treatment**

Early treatment of PDPH is indicated. Not only does it avert the vicious cycle of immobility, weakness, and depression, but also it may help prevent the rare case of subdural hematoma or cranial nerve palsy in the patient with persistent PDPH.

**PSYCHOLOGICAL SUPPORT**

The patient is aware that PDPH is an iatrogenic problem, and she may be angry and resentful as well as depressed and tearful. Headache makes it more difficult to provide care for the newborn and to interact with other family members. Severe PDPH may delay discharge from the hospital and may also have economic consequences. Unlike patients who have PDPH after nonobstetric surgery, postpartum patients typically are healthy and do not expect to feel ill. Two patients have eloquently described their own miserable experiences with postpartum PDPH. Not surprisingly, a retrospective study of 43 obstetric patients with PDPH showed that this complication leads to a negative attitude toward epidural anesthesia.

It is essential that anesthesia providers visit the patient at least once daily to explain symptoms and prognosis, give support, and offer therapeutic options. If feasible, the patient’s partner should attend these discussions. Nurses can help the patient by ensuring that analgesics are given on a regular schedule and by teaching alternative breastfeeding techniques, such as the lateral horizontal position. The anesthesia providers and nurses should write detailed notes in the patient’s record. After discharge, follow-up telephone conversations should be documented. Headache associated with neuraxial anesthesia was the third most common reason for litigation among obstetric cases in the American Society of Anesthesiologists Closed-Claims Project database (see Chapter 33), after maternal death and newborn brain damage. This fact should dispel any notion that postpartum PDPH is a trivial complaint.
POSTURE
The diagnosis of PDPH requires demonstration of a postural component. Significant relief should occur when the patient assumes the horizontal position. The prone position relieves PDPH in some patients, presumably because increased intra-abdominal pressure results in an increase in CSF pressure. Unfortunately, this position is not comfortable for many patients, especially those who had a cesarean delivery.

HYDRATION
Enhanced oral hydration remains a popular therapy initiated by most anesthesia providers for parturients with PDPH, but there is no evidence that vigorous hydration has any therapeutic benefit in a patient with normal fluid intake. However, no patient with PDPH should be allowed to become dehydrated, because of the fluid requirements associated with breast milk and CSF production.

ABDOMINAL BINDER
A tight abdominal binder increases intra-abdominal pressure, possibly leading to an increase in CSF pressure. This method of treatment has never undergone proper assessment.

PHARMACOLOGIC TREATMENT
In the past, a variety of drugs have been used to treat PDPH, including steroids, vasopressin, alcohol, and ergotamine. A safe and effective oral drug therapy for PDPH would be very useful, even if relief is transient. Blood patch therapy is not appropriate or effective in all patients.

Caffeine
Caffeine has been used to treat PDPH for many years, despite lack of clear evidence of its efficacy. A systematic review of the literature identified a single randomized trial performed by Camann et al. In that trial, 40 postpartum women with PDPH received a one-time dose of oral caffeine 300 mg or placebo. Headache scores (visual analog scale [VAS]) were lower in the caffeine group at 4 hours compared to the placebo group (33 ± 6 versus 49 ± 7 mm, respectively), but there was no difference at 24 hours. It appears that the beneficial effect of caffeine is transient.

The caffeine content of a 150-mL cup of drip coffee is approximately 150 mg. Caffeine is a cerebral vasoconstrictor, and one study has demonstrated a reduction of cerebral blood flow after intravenous administration of caffeine sodium benzoate for the treatment of PDPH. Caffeine is also a potent central nervous system stimulant. There are published case reports of seizures after intravenous administration of caffeine for the treatment of PDPH. Bolton et al. described a seizure shortly after combined blood patch and intravenous caffeine therapy. Paech reported seizures in a patient who received oral caffeine (1000 mg during a 23-hour period). Transient atrial fibrillation complicated intravenous caffeine therapy for PDPH in a 71-year-old man. Carter and Pasupuleti reported the management of a patient with intractable PDPH who had already received three epidural blood patches; cosyntropin 0.5 mg in 1 L lactated Ringer’s solution was infused over 8 hours, and the headache was completely and permanently relieved.

To date the only randomized clinical trial of ACTH involved 18 postpartum women who had PDPH after either spinal anesthesia or unintentional dural puncture. There was no difference in the severity of headache or the requirement for epidural blood patch between women who received cosyntropin acetate 1 mg intramuscularly and those who received saline-placebo. Oliver and White described three patients who experienced PDPH after administration of epidural analgesia during labor and who subsequently had seizures. All had received two or three doses of tetracosactrin acetate (1 mg intramuscularly), and one had also received sumatriptan.

Adrenocorticotropic Hormone
The use of adrenocorticotropic hormone (ACTH) for the treatment of PDPH was first reported in a 1994 letter to the editor. Subsequently, anecdotal reports have described different regimens of either intramuscular or intravenously administered ACTH or the synthetic drugs cosyntropin and tetracosactrin acetate. Mood elevation, anti-inflammatory effects, increased endorphin levels, and augmented intravascular volume are postulated as possible modes of action in the relief of headache with ACTH. Kshatri and Foster described a curative response to ACTH in two patients with PDPH. The dose used was 1.5 U/kg in 250 mL of normal saline, infused intravenously over 30 minutes. Gupta and Agrawal assessed the effect of ACTH 60 U intramuscularly in 48 patients with PDPH. Complete and permanent relief was obtained in 40 patients, without any side effects. Carter and Pasupuleti reported the management of a patient with intractable PDPH who had already received three epidural blood patches; cosyntropin 0.5 mg in 1 L lactated Ringer’s solution was infused over 8 hours, and the headache was completely and permanently relieved.

Sumatriptan
Sumatriptan is a serotonin receptor agonist that affects predominantly type 1D receptors. Possessing cerebral vasoconstrictor properties, this agent is used in the treatment of migraine. It is given by subcutaneous injection. Side effects include pain at the injection site and, uncommonly, chest tightness. Sumatriptan may cause coronary artery vasospasm and should not be used in patients with Prinzmetal’s angina or known coronary artery disease. Carp et al. reported that administration of sumatriptan 6 mg resulted in complete resolution of PDPH in four of six patients. Paech et al. reported relief of PDPH in one of seven patients treated with sumatriptan. Connelly et al. studied 10 patients with severe PDPH scheduled for epidural blood patch who were randomly assigned to receive sumatriptan 6 mg or placebo. After 1 hour, only one patient in each group had significant relief, and the investigators concluded that sumatriptan was of no value.
No epidural blood patch procedures were performed. CT scans showed or suggested infarction in two of these subjects. The investigators stated that before the occurrence of these three cases, ACTH had been used regularly to treat PDPH in their institution but that subsequently they had discontinued the administration of ACTH as a therapy for PDPH. No conclusion can be drawn about the possible contribution of ACTH therapy to the observed seizure activity. With the evidence to date, it appears that ACTH therapy cannot be recommended as first-line treatment of PDPH but may be considered for cases that are not amenable to epidural blood patch therapy.

**Miscellaneous Medications**

Other agents evaluated for their effectiveness in reducing PDPH symptoms are gabapentin, methylergonovine, and hydrocortisone. Perhaps the most compelling evidence is for the use of intravenous hydrocortisone (200-mg loading dose followed by 100 mg three times daily for 2 days). Noyan Ashraf et al. evaluated 60 parturients who experienced PDPH after spinal anesthesia and were randomly assigned to receive intravenous hydrocortisone or conventional therapy, which consisted of bed rest, hydration, and scheduled acetaminophen with meperidine. Patients who received hydrocortisone had a 50% reduction in headache severity as assessed by VAS scores at 6 to 48 hours. A criticism of this study is the lack of blinding of the study participants, which may have influenced their perception of headache pain.

Use of gabapentin or methylergonovine has been reported only in case series. Efficacy and side effects (maternal and neonatal) are unclear. All three drugs require further study before they can be recommended for therapy of PDPH.

**EPIDURAL BLOOD PATCH**

**Efficacy**

The epidural blood patch procedure, regarded by many as the gold standard therapy for PDPH, was first described by Gormley in 1960. He reported relief of PDPH symptoms in seven patients after epidural administration of 2 to 3 mL of blood. However, this report was largely ignored until 1970, when DiGiovanni and Dunbar described the immediate and permanent cure of PDPH in 41 of 45 patients in whom 10 mL of autologous blood was injected into the epidural space. Their success led to the widespread adoption of this technique for the relief of PDPH. An excellent review of the history of PDPH and the development of the blood patch was written by Harrington.

In early case series, the reported success rate of epidural blood patch therapy for PDPH was between 89% and 91%. Subsequent studies have not confirmed this high rate of success. Taivainen et al. studied 81 patients with PDPH after spinal needle puncture. Initially symptoms were relieved in 88% to 96% of patients; however, a permanent cure was achieved in only 61%. Safa-Tisseront et al. reviewed the experience with blood patch therapy at their institution over a 12-year period (n = 504, including 78 obstetric patients). Complete relief of PDPH was obtained in 75%, partial relief occurred in 18%, and treatment failed in 7% of patients. The investigators noted a significantly higher failure rate of blood patch after large-gauge needle puncture of the dura. The difference in early reports and more modern audits of PDPH and epidural blood patch therapy success may be related to differences in the duration of follow-up, or perhaps to other differences in management after blood patch therapy, such as delayed mobilization.

In studies limited to obstetric patients, the published success rates of the epidural blood patch have been even less encouraging. Stride and Cooper noted complete and permanent relief of PDPH in 64% of patients after one blood patch procedure. Williams et al. prospectively monitored 100 patients with unintentional puncture during an epidural procedure or after spinal anesthesia with a 25-gauge needle were assigned to receive a 30-mL epidural saline bolus in the lumbar region or a 10-mL lumbar epidural blood patch. Forty-two patients had dramatic relief of their symptoms in the first hour following the intervention; however 12 of the 21 (57%) patients who received saline had recurrence of the PDPH in the next 24 hours.

Prolonged epidural saline infusion may provide better therapy for PDPH symptoms than therapy with a single bolus. Two case reports described the use of epidural saline infusion for parturients with an unintentional dural puncture, whose PDPH symptoms returned after epidural blood patch therapy. The rate of infusion (15 to 25 mL/hr) was limited by the onset of pain in the back, legs, and eyes. A comparative study of epidural saline bolus versus infusion to treat PDPH is needed to determine whether either modality continued over 24 hours would provide better results than conservative therapy. This option might be considered for patients who have a contraindication to epidural blood patch therapy.

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dural puncture. Fifty-eight patients received a therapeutic blood patch; the treatment completely failed in 3 patients, and 17 patients had recurrence of moderate or severe headache requiring further therapy. These observational studies also describe the use of repeated epidural blood patch procedures for parturients with a recurrence of PDPH.

A 2002 Cochrane systematic review\textsuperscript{124} identified only one small randomized trial\textsuperscript{168} evaluating the efficacy of epidural blood patch therapy. This trial involved 12 heterogeneous patients with PDPH who were randomly assigned to undergo epidural blood patch therapy with 10 to 20 mL of blood or a sham patch procedure. Five of 6 patients receiving a blood patch had complete relief of headache symptoms at 24 hours, and none of the sham procedure patients did. Subsequently two additional randomized trials have been reported.\textsuperscript{169,170} Sandesc et al.\textsuperscript{169} reported 32 obstetric and nonobstetric patients who had PDPH symptoms for a minimum of 24 hours, who were randomly assigned to receive either conservative therapy or an epidural blood patch. The primary outcome was headache VAS scores at 2 and 24 hours. At 2 hours the mean VAS score for the conservative therapy group was 8.2 ± 1.4 cm compared with 1.0 ± 0.18 cm for the group receiving a blood patch (P < .001). This difference remained evident at 24 hours. In the largest trial to date, 40 subjects who had PDPH for 1 to 7 days were randomly assigned to receive either conservative therapy or an epidural blood patch using 15 to 20 mL of autologous blood.\textsuperscript{170} The primary outcome was headache 24 hours after intervention, but patients were followed up for 1 week after therapy. The incidence of headache at 24 hours was 58% in the blood patch group compared with 90% in the conservative therapy group (relative risk, 0.64; 95% CI, 0.43 to 0.96). At 1 week the difference widened, with 16% incidence of headache in the blood patch group versus 86% in the conservative group. In summary, administration of an autologous epidural blood patch, while not perfect, often dramatically relieves this debilitating symptom, but patients were followed up for 1 week after therapy. The incidence of headache at 24 hours was 58% in the blood patch group compared with 90% in the conservative therapy group (relative risk, 0.64; 95% CI, 0.43 to 0.96). At 1 week the difference widened, with 16% incidence of headache in the blood patch group versus 86% in the conservative group.

The optimal timing for administration of a blood patch has not been studied adequately. Observational studies suggest that failure is more likely if the blood patch is performed within 24 hours of the dural puncture.\textsuperscript{166,178} It is unlikely that the increase in CSF pressure is sustained or that the blood acts as a mechanical plug to block CSF leak for a prolonged duration. The blood applied to the hole in the dura may initiate an inflammatory reaction that facilitates puncture site repair and closure. It is possible, and even likely, that an epidural blood patch ameliorates PDPH by several mechanisms.

**Timing**

The optimal timing for administration of a blood patch has not been studied adequately. Observational studies suggest that failure is more likely if the blood patch is performed within 24 hours of the dural puncture.\textsuperscript{166,178} It is unclear, however, whether this observation is a result of selection bias. Early-onset PDPH (often resulting from dural puncture with a large-gauge needle) is likely to be more severe and more difficult to treat. Alternatively, a large CSF leak may displace the clot. Partial healing of the dura may have already occurred if a blood patch procedure is delayed, a possibility that may explain the better outcome of a delayed patch procedure.

**Technique for Blood Patch**

The anesthesia provider should thoroughly explain the risks and benefits of the blood patch procedure to the patient.
and the patient should give written informed consent for the procedure. An epidural blood patch can be accomplished on an outpatient basis. Contraindications to the administration of an epidural blood patch are related to complications of placing a needle in the central neuraxis or the administration of blood into the epidural space; they include (1) known coagulopathy, (2) local cutaneous infection or untreated systemic infection, (3) increased ICP due to a space-occupying lesion, and (4) patient refusal. Transient bradycardia has been observed after administration of an epidural blood patch, and some anesthesia providers may choose to establish intravenous access and monitor the electrocardiogram (ECG) in selected patients.

The blood patch procedure should employ sterility measures equivalent to those used in the performance of any neuraxial procedure. The lateral position is more comfortable than the sitting position for patients with severe PDPH. If the anesthesia provider is uncertain about the location of the dural puncture, the more caudad interspace should be chosen. The epidural space is identified in the usual manner. Using meticulous sterile technique (including skin preparation and draping, and donning of a mask and sterile gloves), an assistant withdraws the desired volume of blood into a syringe, usually 10 to 20 mL. This autologous blood is injected slowly through the epidural needle, but the injection is terminated if severe back, leg, or neck pain or pressure occurs. Sometimes slowing the injection rate leads to resolution of the back pain. Blood patch procedures can be administered to Jehovah’s Witness patients with a technique designed to keep blood in continuity with the circulation.

Occasionally, a few drops of CSF are encountered upon entry of the needle into the epidural space, leading to doubt about correct needle placement. In this situation a small test dose of a local anesthetic agent may be administered, sufficient to cause a rapid onset of spinal anesthesia if the needle tip is in the intrathecal space. If no neuroblockade occurs, a blood patch can be performed.

After the procedure the patient should rest quietly in the horizontal position for 1 to 2 hours. Subsequently the patient may resume ambulation, but she should avoid vigorous physical activity for several days. It would be wise for the patient to avoid the Valsalva maneuver and heavy lifting. A stool softener should be prescribed. Most patients report almost instantaneous relief of headache symptoms but may continue to have neck and back fullness over the next 24 hours. Patients should be counseled to immediately report fever, severe back pain, or radiating
lower extremity pain. The anesthesia provider should contact the patient daily for several days after the blood patch procedure.

The blood patch procedure may be repeated if the first one fails to relieve pain. Often the second procedure is successful. The diagnosis should be reconsidered if headache persists after two failed blood patch procedures. A neurology consultation is desirable when a PDPH fails to respond to two blood patch procedures, and should definitely be requested if there is any doubt about the diagnosis. Imaging of the head should be considered to exclude other causes of headache.

Complications of Blood Patch Procedures

Ong et al. reported that the success of neuraxial anesthesia was impaired in women with a prior history of unintentional dural puncture with or without epidural blood patch therapy. However, this conclusion has been refuted by follow-up studies in both obstetric and non-obstetric patients. In both of these retrospective studies, a history of blood patch therapy had no apparent effect on the quality of subsequent epidural anesthesia. Loughrey et al. described a patient who experienced PDPH after diagnostic lumbar puncture at term gestation. A blood patch was administered and the headache was relieved, but 6 hours later, urgent cesarean delivery was required. Spinal anesthesia was administered via the same intervertebral space as the blood patch procedure. The anesthetic course and postoperative period were uneventful, and the patient had no recurrence of headache.

Although epidural blood patch therapy is the most reliable method of relieving PDPH symptoms, adverse outcomes are associated with the procedure. These adverse events can be categorized into two broad groups, infectious/hematologic and neurologic.

Infectious/Hematologic Complications

Conventional wisdom holds that the patient should be afebrile at the time of a blood patch procedure. Many anesthesia providers believe that it is wise to avoid the epidural injection of blood in the presence of systemic infection. Meningitis has been reported after a blood patch. After conservative measures have failed, the optimal treatment of a febrile patient with severe, persistent PDPH is controversial. Epidural infusion of saline involves the use of an indwelling epidural catheter for many hours, which may also be undesirable in a febrile patient. A patch using dextran-40 may be an alternative in febrile patients, but further experience in healthy pregnant patients is needed before this technique can be recommended. The presence of high fever or other evidence of sepsis contradicts the performance of a blood patch procedure. However, we do not believe that a low-grade fever of known etiology is an absolute contraindication to epidural blood patch therapy, provided that the patient is receiving appropriate antibiotic therapy. Management should be individualized, and the known benefits of blood patch therapy should be weighed against the unknown risk of infection.

The risk of epidural blood patch therapy in the presence of human immunodeficiency virus (HIV) infection has been debated (see Chapter 44). However, the central nervous system is infected with HIV at the time of primary infection; therefore, it seems unlikely that injection of autologous blood into the epidural space would alter progression of the disease. There are published reports of the successful use of epidural blood patch therapy, without sequelae, in patients who have acquired immunodeficiency syndrome (AIDS) or who are HIV-positive.

The incidence of cancer during pregnancy is increasing secondary to advancing maternal age. The development of PDPH in this population has raised a theoretical concern about seeding the neuraxial space with neoplastic cells if a blood patch is performed. This concern should be discussed with the patient and her oncologist before the procedure. Bucklin et al. reported the conservative management of a woman with acute leukemia and PDPH; the investigators discussed the therapeutic options for this immunocompromised patient. The use of epidural fibrin glue was reported in a nonobstetric patient with metastatic breast cancer and PDPH, whereas a blood patch procedure was performed for PDPH in a young women with rhabdomyosarcoma.

Neurologic Complications

Serious or permanent problems after epidural blood patch therapy are rare. Diaz et al. wrote an excellent review of case reports of adverse neurologic complications after epidural blood patch procedures. These authors identified 26 reports published between 1966 and 2004 and stratified the complications into neurologic, neurovascular, or inflammatory events. The events occurring in obstetric patients included lumbovertebral syndromes (defined as low back pain with neurologic impairment of the lower extremities), subdural hematoma, arachnoiditis, radicular back pain, pneumocephalus, seizures, and acute meningeal irritation. Compression complications (i.e., lumbovertebral syndrome, subdural hematoma, cauda equina syndrome) were associated with a larger blood patch volume (mean of 35 mL) than the non-compression complications (17 mL). Cranial nerve palsy symptoms that were present prior to the blood patch procedure did not uniformly resolve. The delay in administering the blood patch may have been a significant factor. Two patients who were treated within 4 days of the onset of PDPH recovered within 6 weeks, whereas three patients treated on days 9 to 11 had palsy that persisted for 3 to 4 months. Two obstetric patients experienced new cranial nerve palsies (involving cranial nerve VII), which manifested as facial weakness after administration of an epidural blood patch.

Abouleish et al. reported the results of the long-term evaluation of 118 patients who had received an epidural blood patch. Back pain was the most common complication; it occurred during the first 48 hours in 35% of patients and persisted in 16% of patients, with a mean duration of 27 days. These investigators also noted some cases of neck pain, lower extremity radicular pain, and transient temperature elevation.

The development of an inflammatory reaction to epidural blood can cause acute arachnoiditis, an entity that can manifest several days after the successful cure of a PDPH with a blood patch. This phenomenon is believed to be secondary to free radical damage to spinal nerve roots in the intrathecal space after hemoglobin degradation. There are case reports of obstetric patients presenting with this entity and requiring analgesic therapy for prolonged periods. The diagnosis is made from a presenting history of severe back pain, often with radicular pain, and...
characteristic MRI findings such as nerve root clumping in the intrathecal space and adhesions between nerve roots.

The occurrence of new neurologic symptoms after an epidural blood patch procedure should prompt consideration of the presence of other intracranial pathology. Such symptoms may include (1) mental deterioration due to increased ICP from an intracranial tumor and (2) seizures due to late-onset eclampsia.26,32,195

Diaz196 described a woman in whom permanent paraparesis and cauda equina syndrome developed after an epidural blood patch with 30 mL of blood injected slowly and without symptoms. Low back pain and leg pain developed after the blood patch procedure, and later the patient also experienced incontinence. Twelve days after the procedure, a subdural hematoma at L2 to L4 was diagnosed and surgically treated. Six months later the patient still had significant symptoms. Although a larger volume of blood than usual was injected, the technique appears to have been within normal practice standards. Other long-term sequelae reported in obstetric patients include a postpartum cerebral ischemic event after two epidural blood patch procedures that resulted in permanent hemianopsia197 and a calcified epidural blood patch leading to chronic back pain.198

Dextran/Gelatin Patch
Dextran-40 and gelatin-based solutions, including Gelfoam and Plasmion, have been substituted for blood in epidural patches.130,199,200 These were chosen as alternatives to blood owing to relative contraindications to injection of blood. The use of alternative solutions appears to be more common in countries outside North America. In an observational study of 56 patients, Barrios-Alarcon et al.201 reported that epidural administration of 20 to 30 mL of dextran-40 was safe and effective for the relief of PDPH; all headaches were relieved permanently. The only side effect was a transient discomfort or burning sensation at the time of injection in 6 patients. Some physicians have treated intractable PDPH successfully by administering a dextran-40 patch followed by epidural infusion of dextran at 3 mL/hr for 5 to 12 hours.202,203

Information on the neurotoxicity of these materials is scant.204 Chaninov et al.204 did not identify neurotoxicity after infusion of dextran-40 or polygel (a gelatin powder) into the rat intrathecal space. However, further information is needed before these materials can be widely adopted for epidural patches. From MRI studies of patients with blood patches, we can anticipate that some dextran will enter the subarachnoid space. The small but definite risk of anaphylaxis after the injection of dextran also must be considered, although the risk appears minimal with dextran-40.

Fibrin Sealant Patch
Fibrin sealant is composed of fibrinogen and thrombin. Several commercial products are prepared from human pooled plasma. Products may also contain antifibrinolytics, such as animal aprotinin.205 When injected, these products form a firm, nonretractable fibrin clot. Epidural injection of fibrin glue in rats produces a sustained increase in CSF pressure comparable to the increase that occurs after injection of blood.176 Fibrin sealant has been evaluated for its effectiveness in preventing dural leaks after spinal surgery.206 Epidural fibrin glue patch procedures have been used successfully to treat recurrent PDPH,207 spontaneous intracranial hypotension,208 and CSF leak after long-term intrathecal catheterization.209 In the future, fibrin glue may have a role in patients with intractable PDPH, but further study is required before it can be recommended for routine use.

Surgery
There are rare reports of curative surgical closure of a dural rent for intractable PDPH. In one case, the interval between dural puncture and surgery was 5 years.210

SUMMARY OF TREATMENT
The parturient with PDPH should be actively managed with scheduled analgesics, and she should receive psychological support as she cares for her newborn and manages her symptoms. If the headache is severe, the physician may either try caffeine or proceed directly to epidural blood patch. Epidural administration of fluids other than blood, such as saline or dextran, typically is not first-line therapy but may be considered when there are contraindications to the epidural injection of autologous blood. The accuracy of PDPH diagnosis must always be considered when atypical symptoms are present or when therapy fails.

Unanswered Questions
Important information about PDPH is still lacking. A large, detailed prospective study of PDPH, with and without blood patch therapy, with a long follow-up period (e.g., 1 year) is needed in obstetric patients. What are the long-term effects of both PDPH and blood patch therapy? How common are residual back pain, neurologic symptoms, and auditory/visual symptoms, and do they interfere with everyday life? Answers to these questions are needed to enable anesthesia providers to give patients reliable information, establish a sound basis for informed consent, and administer the best possible care. The Obstetric Anaesthetists’ Association and the Association of Anaesthetists of Great Britain and Ireland211 have recommended that each facility providing obstetric anesthesia services have institution-specific protocols for the management of PDPH, in order to facilitate the identification of parturients with this complication and to provide consistent care.

KEY POINTS
- Dural puncture is only one of many causes of postpartum headache, although many are quick to blame postpartum headaches on dural puncture. A detailed history and physical examination along with indicated neuroimaging in selected cases should ensure diagnostic accuracy.
- A patient with post–dural puncture headache experiences an exacerbation of symptoms when she moves from the horizontal to the upright position, possibly owing to decreased intracranial pressure and secondary cerebral vasodilation, which affect pain-sensitive intracranial structures.
- Anesthesia providers should use small-gauge (<24-gauge), noncutting (pencil-point) spinal needles whenever possible to decrease the risk of post–dural puncture headache.
The initial therapy for post–dural puncture headache consists of psychological support, bed rest in the horizontal position, and scheduled oral analgesics. Although dehydration should be avoided, no evidence supports a role for vigorous hydration for prophylaxis or therapy of post–dural puncture headache.

The "gold standard" for therapy of post–dural puncture headache is autologous epidural blood patch. A second blood patch procedure may be performed—and typically is successful—if the first one fails. If the second procedure fails, alternative diagnoses should be excluded. Other therapies have not proved as effective and safe as the epidural blood patch for treatment of post–dural puncture headache.

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