Post-dural puncture headache: The worst common complication in obstetric anesthesia

Adam Sachs, MD, and Richard Smiley, MD, PhD*

Columbia University College of Physicians and Surgeons, 630 W 168th St PH5, New York, NY 10032

ARTICLE INFO

Keywords:
headache
PDPH
spinal
dural
post-dural

ABSTRACT

Ever since the first spinal anesthetic in the late 19th century, the problem of "spinal headache" or post-dural puncture headache (PDPH) has plagued clinicians, and more importantly, patients. It has long been realized that the headache and other symptoms that often occur after the entry of a needle into the subarachnoid space is somehow related to fluid loss, although the exact pathophysiology of the headache has really never been defined. With the introduction of pencil-point spinal needles for spinal anesthesia in pregnant women over the past 2 decades, the problem of PDPH in obstetrics has been more associated with accidental dural puncture during attempted epidural procedures. Accidental puncture probably occurs in about 1% of procedures, so with over 60% of pregnant women receiving epidural analgesia for labor, there are probably 20,000–50,000 obstetric patients with PDPH in the United States each year. In this article, we will discuss the current state of knowledge in this area, suggesting that the PDPH syndrome is more severe and often more long-lasting, with some potentially life-threatening complications (cerebral hemorrhage) than usually appreciated or admitted. While prevention and treatment options are still limited, with the only clearly effective treatment being the epidural blood patch, recognition of the PDPH syndrome in postpartum women by anesthesiologists and obstetricians, with aggressive follow-up and treatment, may help limit the associated morbidity and mortality.

© 2014 Elsevier Inc. All rights reserved.

Incidence

In the United States, there were 4 million births in 2012,1 and 61% of singleton vaginal births in 2008 received a spinal, epidural, or combined spinal–epidural (CSE) procedure for labor analgesia. Extrapolated data suggests that greater than 2 million women will receive labor analgesia this year. The overall incidence of PDPH following spinal, epidural, or CSE is approximately 1% with typical obstetric anesthesia practices. This reflects estimates of a 0.5–2% incidence of headache after planned spinal anesthesia/analgesia with small, pencil-point needles. In addition, when attempting epidural procedures with 16–18-gauge (G) epidural needles, accidental entry into the subarachnoid space (wet tap) occurs 0.5–4% of the time, with a resulting headache rate of 45–80%.2 Therefore, we can conservatively expect greater than 20,000 postpartum women will experience a PDPH in the US in 2014.

*Corresponding author.
E-mail address: rms7@cumc.columbia.edu (R. Smiley).

http://dx.doi.org/10.1053/j.semperi.2014.07.007
0146-0005/© 2014 Elsevier Inc. All rights reserved.
The containment of cerebrospinal fluid (CSF) in the subarachnoid space. If in fact PDPH occurs within 72 h after meningeal puncture in 90% of patients and is evident by a headache typically worsening within 20 s of standing or sitting and resolving within 20 s of recumbency, although the International Headache Society (IHS) defines it as occurring within 15 min of standing and resolving within 30 min of recumbency. While the diagnosis of PDPH is predominantly clinical, radiologic imaging can be useful to rule out other pathology or corroborate an unclear presentation. PDPH confirmation from magnetic resonance imaging consists of enhancement of the pachymeninges, decreased size of subarachnoidal cisterns and cerebral ventricles, downward displacement of the brain, and subdural collections. Alternate studies may include CT scan (with or without myelography), transcranial Doppler, or cerebral angiography. If the diagnosis is uncertain, or clinical features worrisome, neurologic consultation can be utilized before or after imaging is obtained. Unfortunately, a diagnostic spinal tap, which is often utilized in these scenarios, may worsen a preexisting PDPH, or cause a new PDPH, and delay or confuse diagnosis and treatment of the original problem. In our practice, we try to discourage a diagnostic LP until after imaging.

Diagnosis

Headache after childbirth is extremely common, occurring in 30% of women, ranging from benign to severe in severity and varying as much in etiology. The majority of postpartum headaches are caused by lack of sleep, caffeine withdrawal, tension, or migraine headaches. When compared to the general population, postpartum women without meningeal puncture are at increased risk of serious causes of headache, including subarachnoid hemorrhage. This occurs at a rate of 6 per 100,000 deliveries and may be related to increased plasma volume, vascular tone, alterations in the fibrinolytic system or coagulation cascade, or disseminated intravascular coagulation DIC. Other serious etiologies for postpartum headache include cortical vein thrombosis, sagittal sinus thrombosis, preeclampsia/eclampsia, posterior reversible encephalopathy syndrome (PRES), and vasculopathy.

Pathophysiology

The pathophysiology of PDPH almost certainly involves the leakage of cerebrospinal fluid (CSF) out of the intrathecal space. MRIs conducted on patients with PDPH show decreased CSF volume, sagging of intracranial structures, and meningeal enhancement attributed to vasodilation of vessels secondary to intracranial hypotension. Despite MRI evidence of decreased CSF volume and pressure as the etiology, the exact mechanism of head pain remains unclear. Intrathecal hypotension may result in caudal excursion of the brain, which results in headache through traction on pain-sensitive areas of the brain and meninges. Alternatively, CSF loss may cause increased cerebral blood flow and vascular dilation, resulting in a pathophysiology similar to vascular headaches. Recent investigational literature has examined the benefits of treatments designed and used for vascular and migraine headaches, including caffeine, theophylline, triptans (sumatriptan), and methylergonovine (as discussed in the section Treatment), although none of these therapies have been found to be universally effective in controlled studies. There is some evidence linking the pain to substance P and neurokinin 1 receptors.

The spinal cord is bathed in CSF, and both are contained within the intrathecal sac, which is bordered by the meninges. The meninges are made up of three layers (dura, arachnoid, and pia mater), which are responsible for containment of CSF. CSF is located between the arachnoid and pia mater, with the dura superficial to the arachnoid mater. The term post-dural puncture headache (PDPH) is quite likely erroneous because a tear in the arachnoid matter, and not the dura, is the likely explanation for the clinical symptoms experienced. The dura is composed of predominantly collagen fibers, and the relatively large molecular distances between fibers allow drug and fluid movement. By contrast, the arachnoid mater consists of tiers of cells attached by tight junctions and occluding junctions. This phenomenon results in the impedance of drug passage into the CSF and the containment of CSF in the subarachnoid space. If in fact PDPH is caused by CSF leakage out of the intrathecal space, it seems logical that a tear in the arachnoid meninges is more important than the dura since this layer is primarily responsible for the containment of CSF around the spinal cord and brain. A more appropriate name would therefore seem to be post-arachnoid puncture headache, or spinal headache (less specific), although the actual importance of this distinction may not be clinically relevant. While acknowledging that the dura may not be the important barrier, in this review, we will use the term “dural puncture” and PDPH, as they are the familiar designations for the syndrome and almost universally used.

Clinical course and complications

While meningeal puncture is often not considered the most serious cause of headache following pregnancy, it is not a
benign complication. These headaches can be severe and debilitating, preventing ambulation and limiting interaction between mother and baby during the postpartum period, in addition to prolonging hospitalization and increasing health care costs. Indeed, 39% of patients with PDPH experience greater than 1 week of impairment in activities of daily living.5 Furthermore, it has been implicated in permanent disability, including cranial nerve palsy, chronic headache (sometimes necessitating surgical closure), reversible cerebral vasoconstriction syndrome (RCVS), subdural hematoma, intracerebral bleeding, cerebral venous sinus thrombosis, or aneurysmal rupture.14–16

Chronic headache following meningeal puncture is a serious problem that is probably underreported.17 When a meningeal defect occurs, either from intentional or accidental dural puncture (ADP), it is usually repaired by the body’s own restorative mechanisms. Occasionally, this does not occur, and a chronic headache can result from perpetual intracerebral hypotension. Although there are multiple case reports detailing chronic CSF leak and headache from previous meningeal puncture, many patients do not receive treatment due to a lack of disclosure, limited information, or lack of follow-up by practitioners.

According to the American Society of Anesthesiologists closed claims project, a national registry that examines closed malpractice claims from multiple insurers, the third most common cause of lawsuits against anesthesiologists by obstetric patients was headache (approximately 12%). Currently, more obstetric anesthesiologists are being sued for headache than maternal death, which may demonstrate the increased safety in obstetric anesthesia care, the litigious nature of our society, or the severity of these headaches.18

Another possible consequence of PDPH is reversible cerebral vasoconstriction syndrome (RCVS), of which postpartum cerebral angiopathy (PCA) is a subtype. RCVS is characterized by a reversible, multifocal vasoconstriction of the large- and medium-sized cerebral arteries in the anterior and posterior circulation, which may be evident by a pearl and string sign on cerebral imaging. Cerebral vasoconstriction can result from mechanical stimuli, such as caudal brain migration from CSF loss, in addition to chemical, neurogenic, or electrical causes.19,20

Subdural hematoma is one of the more serious complications from meningeal puncture and results from tearing intracerebral bridging veins due to caudal brain migration after intraspinal CSF loss. Symptoms (worsening headache and focal neurologic symptoms) can present hours to weeks after spinal puncture depending on the rate of blood accumulation. Mortality from this complication is high at 13% so early diagnosis is crucial.21 Unfortunately, the majority of these patients have already presented with severe headache so identification of the new/additional pathophysiology is sometimes difficult. Some patients may have vomiting and a non-postural headache or change in the characteristic of their headache (especially less of the postural component). Early or prophylactic blood patching (discussed in the section Treatment) may prevent the development of subdural hematoma because it decreases CSF loss, intracranial hypertension, and perhaps traction on the bridging veins. However, if epidural blood patch (EBP) is performed in the presence of a significant subdural hematoma, intracranial hypertension and further neurologic sequelae can result.21 When evaluating a patient with postpartum headache, sudden onset, vomiting, seizures, changes in the nature of the symptoms, decreased level of consciousness, or neurological exam abnormalities should all prompt immediate consideration for imaging and neurologic consultation.22

**Risk factors**

The rate of PDPH following meningeal puncture is extremely variable (ranging from 1% to 75%), due to the large number of factors that affect its incidence.22–24 While all adults aged 18–30 years are at high risk for development of PDPH, young pregnant women with a low BMI seem to constitute the highest risk group.25 The literature suggests that less distensible meninges secondary to atherosclerosis or mechanical changes may be the cause of the particularly low incidence in elderly individuals.25,26 PDPH rarely occurs in patients younger than 10 years for unknown reasons, although under-reporting probably occurs. Performance of an epidural blood patch has been reported in a 4-year-old child.27

The literature clearly indicates that factors associated with larger defects in the meninges cause a greater egress of CSF and result in a drastically higher incidence of PDPH. Size and type of spinal needle are therefore two of the most important factors for decreasing the risk of PDPH.22,24,28 In fact, cutting needles (Quincke) are associated with at least a five-fold increase in CSF loss and PDPH when compared with blunt/pencil point (e.g., Sprotte, GM®, Whitacre) of the same gauge. Similarly, a larger bore 22-G needle results in a 6-fold increase in CSF loss and PDPH when compared to a 25-G needle. Interestingly, scanning electron microscope evaluation comparing cutting and pencil-point/blunt needles shows that pencil-point needles result in what appears to be more dural trauma. Without the evaluation of the effect of different needle sizes and shapes on the arachnoid mater, only limited conclusions can be drawn from this data. If arachnoid and dura mater traumas are congruent after meningeal puncture, this suggests that greater meningeal trauma may result in a more rapid repair, although this is speculative.16

Another factor that results in a decreased incidence of headache is insertion of cutting spinal needles or epidural needles with the bevel longitudinal/parallel to meningeal fibers, presumably from a decreased disruption of meningeal fibers. Studies show that the development of PDPH after inadvertent dural puncture is 3-fold higher when the needle bevel is oriented perpendicular to the spinal axis compared to parallel.29 Contrary to previous teaching that attempted to explain the above finding, contemporary imaging reveals that dural collagen fibers are not oriented parallel to the spinal column whereas the arachnoid are. This may further suggest that PDPH is caused by arachnoid puncture and not dural puncture, or at least that we do not understand the etiology as well as we might think we do.

When considering the summation of these factors, the opportunity for practitioners to alter the development of PDPH based on choice of spinal needle becomes evident. A 20-G cutting needle with bevel directed perpendicular to the
dura (e.g., opening in the cranial direction) has an incidence of PDPH greater than 60%, whereas the risk with a 27-G pencil-point needle is around 1%. Some practitioners, especially outside of anesthesiology, continue to claim/report that adequate CSF flow can only be achieved with spinal needles of 22G or larger, which constitutes the predominant reason for their continued use. This has not been the authors’ experience; we have been able to obtain >10 ml of cerebrospinal fluid consistently in research subjects using 25-G Whitacre needles (R.M.S., unpublished observations). There is probably rarely an indication for a spinal needle larger than 25G in women of childbearing age, and almost never a need for a needle size greater than 22G in any patient.

An additional way to possibly decrease the incidence of PDPH relates to the technique used by anesthesiologists during epidural placement. Typically, a blind technique utilizing a “loss of resistance” (LOR) to either air or saline is used. The pressure difference between the epidural space (0.13 kPa) and ligamentum flavum (9.2 kPa) allows the practitioner to feel when the epidural space is entered. If the practitioner does not sense the change as the needle passes from the ligamentum flavum into the epidural space because of a soft ligamentum flavum, malfunction of the plunger, or inexperience, then an inadvertent dural/arachnoid puncture can occur as the epidural space is traversed by the anesthesiologist without stopping the needle. If this occurs while using the LOR to air technique then pneumocephalus can result. A headache from pneumocephalus presents immediately after arachnoid puncture and occurs when the patient changes from a lateral to upright position. It often is severe, temporarily relieved with the supine position, improves with oxygen, and usually of brief duration. In porcine models, the continuous LOR technique was more accurate when compared to the intermittent LOR technique for detecting pressure differences between the ligamentum flavum and epidural space. When comparing the midline with the paramedian approach, no significant difference was found. Therefore, the continuous LOR to saline technique, whether midline or paramedian, theoretically offers the practitioner a decreased risk of pneumocephalus and ADP, offering a greater opportunity to accurately identify the epidural space. It should be noted that there are no good randomized studies supporting a difference in outcome with different techniques, so the choice of method remains one of practitioner’s preference.

Another area where obstetric anesthesiologists may be able to alter the incidence of PDPH involves whether a combined spinal-epidural (CSE) or epidural technique is chosen to provide labor analgesia. The techniques are similar and involve placement of a needle, then a catheter through the needle into the epidural space to provide a continuous infusion of local anesthetic solution to decrease the pain of labor. However, a CSE involves placing a spinal needle (typically 25-27-G pencil-point type) through the epidural needle (before catheter placement) into the subarachnoid space, detecting CSF, and then injecting local anesthetic and an opioid intrathecally. This allows parturients to obtain pain relief more quickly and may result in higher satisfaction. Because this technique involves intentionally puncturing the meninges, it has been suggested that this would lead to a higher incidence of PDPH. The data thus far has shown this to be untrue with similar rates of PDPH (approximately 1%) and epidural blood patch between the two techniques. The reasons are probably twofold. The risk of headache from puncturing the meninges with a 27-G needle is relatively low and probably only slightly increases the incidence of PDPH. In addition, performance of the CSE technique may reduce the risk of ADP with the epidural needle. Norris et al. found in a residency training program that the accidental meningeal puncture rate was 4.2% when the epidural technique was used and 1.7% when a CSE technique was used. When practitioners encounter a challenging epidural catheter placement and the exact position of the epidural needle, in relation to the epidural space, is unclear, the CSE technique allows the practitioner to use the spinal needle to obtain additional information. If the spinal needle is placed through the epidural needle and CSF is obtained, then the intrathecal sac is directly ahead and the epidural needle should not be advanced further. By contrast, when performing a standard epidural technique, the practitioner cannot use a spinal needle to assist in determining position. Which technique is ultimately safer for preventing PDPH remains a matter of debate, and a definitive conclusion will probably not be evident in the near future.

Treatment

When a PDPH occurs, there are no accepted algorithms for treatment, but the nature of the defect (needle size suspected of making the puncture) and the presence and severity of symptoms are important considerations. Severe symptoms warrant immediate intervention assuming alternate causes of headache have been excluded. The gold standard for the treatment of severe PDPH is epidural blood patch because it has the highest benefit-to-risk ratio and is the most effective treatment to date (as discussed below). Patients with larger meningeal defects probably warrant more aggressive follow-up and earlier intervention. It is now evident that untreated PDPH, especially when significant intrathecal hypotension is present, may lead to a higher incidence of morbidity and mortality, including chronic headache syndromes. The following therapeutic treatment options are divided into conservative, medical, and invasive sections.

Conservative

Despite decades of patients being told by nurses, anesthesiologists, and obstetricians to “lie flat for a day” there is no evidence that bed rest, patient positioning (prone, supine, head-up, head-down, etc.), immobilization, or supplemental IV fluid administration following meningeal puncture helps prevent PDPH. Obviously, the symptoms of the PDPH do tend to be ameliorated in the supine position, but there is no evidence for prevention or faster recovery.

Medical therapy

While multiple medical modalities have been tried, there is a dearth of high-quality evidence-based literature on the topic.
Methylxanthine derivatives, including caffeine or theophylline, have been used and have shown some benefit in the treatment of PDPHs. The mechanism of action may involve blocking adenosine receptors, which results in cerebral vasoconstriction, countering the cerebral vasodilation that can occur from CSF leakage and intrathecal hypotension. While proof of benefit from high-grade studies is lacking, the current clinical literature shows some continued support for caffeine use, particularly in mild to moderate cases. IV or oral current clinical literature shows some continued support for caffeine use, particularly in mild to moderate cases. IV or oral caffeine (300–500 mg) once or twice daily causes a faster resolution of symptoms in patients with documented PDPH and may prevent the development of PDPH when given prophylactically after inadvertent dural puncture or intentional puncture in high-risk populations. For comparison, a cup of coffee contains 50–100 mg while a soft drink has 35–50 mg of caffeine. Unfortunately, most of the studies suggest that the improvement from caffeine administration is temporary and there is no reduction in the rate of epidural blood patch administration. Theophylline has been shown to decrease PDPH pain up to almost 60% although CNS stimulation and cardiac problems have restricted its clinical use.

Gabapentin is a structural analogue of gamma-aminobutyric acid (GABA), and although its exact mechanism is unknown, it likely increases the concentration or rate of synthesis of GABA in the brain. Studies have shown that preoperative gabapentin may be efficacious in reducing postoperative pain, which has been extrapolated to the treatment of PDPH. Initial reports have been encouraging, with studies showing that gabapentin (300 or 400 mg) administered to patients with documented PDPH, every 8 h for 4 days, decreases VAS scores for 5 days and epidural blood patch necessity without any evidence of significant side effects. Similarly, pregabalin may be part of a potential therapeutic regimen to reduce the severity of PDPH when 50 mg is given every 8 h over a course of 3 days.

Sumatriptan and other “triptans” are tryptamine drugs that function as 5HT1B/1D receptor agonists and are frequently used in the treatment of migraine and cluster headaches. They cause constriction of intracranial blood vessels, and their use has been investigated for the treatment of PDPH. Sumatriptan, the most well-studied triptan, has been largely ineffective for PDPH when controlled studies are considered. Frovatriptan, an orally administered triptan, has a high selectivity for cerebral vasculature and long half-life/therapeutic action making it a drug perhaps worth considering. Although administration of Frovatriptan 2.5 mg once a day for 5 days after meningeal puncture showed benefit, more investigation is clearly necessary.

Adrenocorticotropic hormone (ACTH) is another drug, which is being investigated for therapeutic effects for treating PDPH. It is hypothesized that ACTH may increase CSF production through a sodium active transport mechanism or increase the pain threshold by raising beta-endorphin levels. The results regarding ACTH have been conflicting, but treatment regimens that have shown benefit include 60 units of ACTH intramuscularly or 1.5 units/kg IV over an hour (both can be repeated 24 h later for headache recurrence). A recent study using cosyntropin (an ACTH analogue) reported remarkable efficacy but has yet to be independently confirmed.

Hydrocortisone, which is intimately tied to ACTH in the hypothalamic-pituitary-adrenal axis, has also been examined for treatment of PDPH because of its use in pain modulation and anti-inflammatory properties. One study showed that intravenous hydrocortisone 200 mg, then 100 mg TID for 48 h, when given to postpartum patients with evidence of PDPH decreased VAS scores by as much as 50% 6 h later and 75% 24 h after treatment.

Overall, however, medical therapies have been disappointing, probably because most therapies rely on cerebral vasoconstriction as a therapeutic modality. This reasoning assumes the pathophysiology of PDPH is due to cerebral vascular dilation, which is probably only partially correct, or too simplistic. In addition, therapies that may be of some benefit for “small-needle” (i.e., spinal needle) headaches may be ineffective for PDPH resulting from an ADP with a 16–18-G epidural needle.

Invasive

Since its first description in 1960, EBP has become the treatment of choice for moderate to severe PDPH. The procedure involves the injection of up to 30 ml of autologous blood into the epidural space. Therapeutic EBP results in a lower incidence of PDPH when compared to conservative treatment or a sham procedure. In fact, up to 95% of patients will exhibit immediate short-term relief, with up to 70% headache free several days later. This is probably the result of a dual mechanism. The long-term process can be explained by MRI and postmortem studies which show EBP blood extends 3–9 spinal levels and tightly adheres to the thecal sac resulting in clot formation at the meningeal puncture site for greater than 18 h. The immediate relief cannot be explained by fistula closure because CSF is produced at a rate of 0.5 ml/min, which is inadequate to quickly replenish the amount of extravasated CSF which can be as much as 200 ml/day. MRI studies have shown that extradural blood injection causes an initial hematoma that constricts the dural sac and nerve roots for up to 3 h. Therefore, the sudden relief experienced by patients after EBP, similarly seen with saline injection, is probably caused by immediate compression that temporarily restores normal intrathecal and intracerebral pressures. Even though EBP blood extends multiple spinal levels, its distribution is not even, with a tendency toward cephalad migration. MRI studies have shown that blood migrates, on average, 3.5 intervertebral spaces above and 1 intervertebral space below the site of injection, after 20 ml of lumbar epidural blood is administered. This suggests that EBP should be performed below the level of puncture and not above if possible, although the interspace that appears technically easiest to access the epidural space is often used. While the complications associated with EBP are similar to epidural placement, there is an increased risk of lower limb paresthesia, epidural infection, and backache (secondary to nerve root and perhaps muscular irritation from the blood), which can last up to 5 days. In addition, if blood is accidentally injected intrathecally, instead of epidurally, it can cause arachnoiditis, meningitis, cauda equina syndrome, and permanent nerve damage. To prevent this occurrence, prior to epidural blood administration, some practitioners
recommend an epidural test dose in addition to negative CSF aspiration through the catheter. By contrast, many believe that the absence of CSF flowing out of the Tuohy after entering the epidural space is enough evidence of epidural placement, and it is not our practice to routinely perform an epidural test dose during blood patching.

Although EBP is largely effective, up to 30% of patients will require a second EBP due to return of symptoms, which is probably especially true when ADP occurs with larger bore needles. This is likely a result of dislodgement of the clot or failure of clot formation at the defect. If two blood patches have been completed and the patient’s headache still persists, then imaging is probably warranted in most situations to confirm that the proper diagnosis has been made, although if each EBP is successful, there is a strong possibility PDPH is the diagnosis and the EBP is simply failing to provide permanent relief. With respect to the quantity of blood injected, 20–30 ml of blood results in lower pain scores and a higher probability of permanent relief when compared to smaller amounts, but patients also experience a higher incidence of temporary back pain.

While there is clear evidence that therapeutic EBP is effective, there is some evidence that earlier EBP (within the first 24–48 h after ADP) may be less effective. In fact, the recurrence of headache can be 50% higher if EBP is completed within the first 24 h. This phenomenon is perplexing and may be related to several causes. If in fact the therapeutic benefit of EBP is secondary to clot formation at the site of meningeal puncture, then factors hindering clot formation will likely result in a lower therapeutic benefit. After meningeal puncture, there is a large pressure gradient between intrathecal and epidural spaces, which promotes the outflow of CSF. If an EBP is performed soon after ADP, before significant intrathecal hypotension has occurred, then clot formation at the site of the meningeal hole may be impossible because the flow of CSF through it is too great. Another alternative is that residual local anesthetic solution remains in the epidural space when prophylactic EBP is performed and this solution mixes with the patient’s blood, thereby preventing clot formation at the meningeal defect through a dilutional or chemical mechanism. A third possibility relates to the amount of time needed for the meningeal hole to close. If EBP is performed early, then clot degradation may occur prior to the body’s ability to close the defect. For these reasons, some practitioners recommend waiting at least 48–72 h after known meningeal puncture prior to EBP consideration. This opinion and practice is controversial, and others (including these authors) believe that early treatment or at least offering an EBP for severe symptoms makes medical (possibly decreasing severe complications), humanitarian (relieving pain and improving functioning in the early postpartum period), and perhaps legal sense, even if a repeat patch is needed 24–72 h later. Although EBP is a standard effective treatment for recent PDPH, it has also been shown to be effective in treatment-resistant cases that have persisted for months or years despite conservative management.

Practitioners have attempted to inject various amounts of saline, at various time intervals, either intrathecally or epidurally after ADP to prevent PDPH. This can be accomplished by intermittent saline bolus or continuous infusion intrathecally prior to removal of the Tuohy needle/intrathecal catheter, or epidurally after epidural catheter replacement.

In all scenarios, short-term improvements in headache are likely secondary to increased intrathecal CSF volume, or epidural fluid volume that transmits to the intrathecal space, resulting in decreased cerebral and meningeal traction. Although it seems logical this would only delay the development of symptoms, as saline likely has no role in closing the meningeal defect (as blood is believed to), certain studies have shown as much as a 50% reduction in the development of PDPH and the need for EBP. While the rate of CSF creation and egress during Tuohy puncture make this drastic reduction in the incidence of symptoms unlikely, these interventions are low risk, and any benefit is probably worth the attempt until better evidence is available to the contrary. If desired, 10 ml of saline can be administered intrathecally or 20 ml epidurally, prior to Tuohy or catheter removal.

The injection of colloid solutions (modified fluid gelatin, hydroxyethylstarch, or 40% Dextran) instead of autologous blood into the epidural space is another treatment option. The increased viscosity of colloid solutions, when compared to saline, results in slower migration from the epidural space. This should result in a longer period of increased epidural pressure, a decreased gradient for CSF flow, and more time to seal the defect. Colloid solutions are particularly attractive for the treatment of PDPH when EBP with autologous blood is undesirable. Examples include a Jehovah’s Witness patient who will not accept autologous blood that is not in a continuous circuit with the body or HIV/leukemia patients who may have an increased risk of central nervous system spread. Another beneficial aspect of colloid solutions is they produce a decreased inflammatory response when placed epidurally compared to autologous blood.

Injecting morphine into the epidural space has also been a potential therapeutic modality instead of blood. In one randomized, blinded study, 3-mg epidural morphine (a dose very similar to that given for post-cesarean analgesia) significantly reduced the incidence of PDPH and epidural blood patch after inadvertent dural puncture. Epidural or spinal morphine is often administered for parturients who undergo cesarean delivery, but not usually after uncomplicated vaginal births. Patients who undergo ADP and then subsequently deliver vaginally may benefit from preservative-free epidural morphine administration, although a confirmatory study would be very useful.

For patients whose PDPH recurs despite several epidural blood patches, fibrin glue injected epidurally has shown promise. Fibrin glue is frequently used in neurosurgery and consists of two components, including fibrin and a thrombin solution, which stimulates the last step of the coagulation cascade when mixed together. Fibrin glue complications include the transmission of infection because it is derived from pooled human plasma, immune reactions, anaphylaxis, and a theoretical risk of spinal cord or nerve root compression through mass effect. According to the Cochrane reviews, acupuncture is beneficial for the treatment of migraine and tension headaches. It has been suggested that migraine headaches and PDPHs may similarly activate the pain-sensitive trigeminovascular system of the brain causing a subsequent reaction of
intracranial vessels. Investigation into acupuncture for the treatment of PDPH has showed moderate success (limiting the duration of headache) although the evidence is limited. Several authors have suggested its use prior to EBP for mild to moderate PDPH because of its less invasive nature or in patients who refuse EBP.65,66

Prophylactic blood patch vs. intrathecal catheter

The best course of action to be taken at the time of ADP with a large-bore epidural needle remains unclear. When CSF is dripping or flowing rapidly through the Tuohy needle, the practitioner is left with two options: thread an epidural catheter intrathecally or remove the Tuohy and reattempt epidural placement. Performing a new procedure to site the catheter properly in the epidural space usually at a different intervertebral space was the usual option until about a decade ago, because this allows the usual dosing regimens to be used for analgesia and anesthesia. There is a small risk of complications, however, including the morbidity associated with an additional epidural procedure or an exaggerated block due to drug traversing the large hole created between epidural and intrathecal spaces. An additional advantage, however, is the possibility of injecting blood via the properly placed epidural catheter after labor (or cesarean delivery) is complete but before the patient develops a PDPH from CSF leakage (a so-called “prophylactic epidural blood patch”).

With regards to prophylactic blood patches, some studies have shown a decrease in the incidence of PDPH compared to no treatment, conservative treatment, or epidural saline treatment. Unfortunately, for proponents of its use, a randomized controlled blinded study compared prophylactic EBP to a sham procedure and found no reduction in the incidence of PDPH or therapeutic blood patch. Therefore, over the past decade, after ADP it has become more common for anesthesiologists to place the catheter into the intrathecal space, where it can be used for analgesia or anesthesia, without a new procedure. When pooled data is considered, prolonged intrathecal catheter placement reduces PDPH from 66% to 51%, and the need for epidural blood patch is reduced from 50% to 33%. The effect after leaving the catheter for an additional 24 h may be secondary to an inflammatory process that assists closure of the dura subsequent to catheter removal. Although this data is largely not based on randomized controlled trials, many practitioners recommend threading the catheter intrathecally because reattempting placement may result in subsequent ADP, probably further increasing the risk of PDPH. The arguments against proceeding with an intrathecal catheter are the possible increased risk of infection with a catheter inserted directly into the spinal fluid, the possible increased risk of nerve damage secondary to neurotoxic effects of local anesthetic, and the small but serious risk of a dosing error leading to high or total spinal anesthetic. If an epidural dose is administered into a spinal catheter, respiratory depression/arrest, hypotension, and shock can occur.

With no clear evidence of a benefit, and the possible success of intrathecal catheters limiting PDPH, most practitioners now reserve prophylactic EBP for unusual circumstances or severe cases. The pendulum may be turning again, however, because a very recent randomized controlled study compared prophylactic epidural blood patch to conservative treatment with a therapeutic epidural blood patch if conservative treatment failed. Patients who received a prophylactic blood patch had a 4-fold decreased risk of developing a PDPH. Proponents of prophylactic EBPs will point to this study, suggesting that in addition to reducing the incidence of headache and blood patch, prophylactic patching might help prevent the occurrence of more serious complications, such as subdural hematoma (mechanism mentioned above). Patterns of practice regarding whether to perform prophylactic blood patches instead of placing intrathecal catheters after ADP may change if further research corroborates this study.

Whichever of the above paths is chosen, the anesthesiologist is left with the decision of how long to leave the catheter in place (intrathecally or epidurally) and whether any additional medications or fluids should be given through the catheter prior to removal. There is no evidence that prolonged epidural catheter placement results in any reduction in PDPH occurrence. As mentioned above, there is some evidence that prolonged intrathecal catheter placement may be beneficial in patients with ADP, and if this strategy is adopted, we recommend intrathecal placement for at least 24 h if feasible. It cannot be emphasized enough, however, that if a spinal catheter is present, either in labor or postpartum, it must be clearly labeled and everyone who might care for the patient be notified to ensure no confusion exists. An epidural dose of local anesthetic delivered intrathecally can result in a complete spinal (apnea and cardiovascular collapse), and there is at least a theoretical infection risk. If there is no plan to inject saline, morphine, or other medications (see above) through the catheter, we typically tie the catheter in a knot to prevent inadvertent injection of medications (or spinal fluid leak) before sending a patient with a spinal catheter off the labor floor.

Follow-up

Because PDPH typically develops 24–48 h after meningeal puncture, patients are usually in the postpartum unit or even at home when symptoms occur. Because of the delay in onset of symptoms, it is especially important that the potential to develop a headache is mentioned to all parturients undergoing neuraxial anesthesia and the anesthesiologist can be contacted for follow-up. There are multiple case reports and lawsuits from patients who developed PDPH and did not seek, or could not obtain, treatment because they were never informed, or had no means, of contact. PDPH is a treatable condition, and much of the morbidity and mortality associated with this complication can be avoided with early intervention.

The role of the obstetrician

Obstetricians have a unique opportunity to limit the severity of morbidity and mortality experienced by parturients with a PDPH. As the primary care givers for peripartum women, they
frequently are the first to be notified about the presence of anesthetic complications. The importance of timely, appropriate notification of an anesthesiologist when a peripartum woman experiences a headache cannot be understated as imperative in correcting the problem. While many postpartum headaches are NOT due to meningeal puncture, PDPH may not present for days to months, and obstetricians should consider this diagnosis in the postpartum unit, at weekly/monthly follow-up appointments, and even more remote meetings. If a patient presents with new-onset headaches and received neuraxial analgesia for delivery, the possibility of a PDPH should at least be considered, especially if a documented “wet tap” occurred, and appropriate personnel notified if the index of suspicion warrants it. While the further diagnostic and therapeutic steps are probably going to be governed by the anesthesiology or neurology consultants, an appreciation of the severity of the syndrome, its potential long duration, and the possibility of rare but not unheard of severe complications should prompt the clinical team to be aggressive in diagnosis and treatment.

REFERENCES


