Post-dural puncture headache: pathophysiology, prevention and treatment

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Post-dural puncture headache (PDPHA) has been a vexing problem for patients undergoing dural puncture for spinal anaesthesia, as a complication of epidural anaesthesia, and after diagnostic lumbar puncture since Bier reported the first case in 1898. This Chapter discusses the pathophysiology of low-pressure headache resulting from leakage of cerebrospinal fluid (CSF) from the subarachnoid to the epidural spaces. Clinical and laboratory research over the last 30 years has shown that use of small-gauge needles, particularly of the pencil-point design, is associated with a lower risk of PDPHA than traditional cutting point needle tips (Quincke-point needles).

A careful history can rule out other causes of headache. A positional component of headache is the sine qua non of PDPHA. In high-risk patients (e.g., age > 50 years, post-partum, large-gauge-needle puncture), patients should be offered early (within 24–48 h of dural puncture) epidural blood patch. The optimum volume of blood has been shown to be 12–20 ml for adult patients. Complications of autologous epidural blood patch are rare.

Key words: headache; post-dural puncture; anaesthesia; spinal; complications; treatment.

Post-dural puncture headache (PDPHA) is a possible complication of both spinal and epidural anaesthesia. The first report of PDPHA was described after Professor August Bier and his Surgical Resident, Dr Hildebrandt, performed spinal anaesthesia on each other in 1898, using cocaine.¹ When Dr Hildebrandt could not attach the syringe with the local anaesthetic to the needle placed in the subarachnoid space of Professor Bier, a considerable amount of cerebrospinal fluid (CSF) was lost. Not wanting to disappoint Professor Bier, Dr Hildebrandt volunteered subsequently to receive a spinal anaesthetic himself. Both men developed significant postural cephalalgia following the experiment, Professor Bier’s lasting for 9 days and Dr Hildebrandt’s commencing the evening of...
the procedure and associated with at least two episodes of vomiting. Bier suggested that it was the loss of CSF that resulted in his condition, and further postulated that the loss of CSF might be minimized by the use of fine needles and the placement of a finger into the hub of the needle, as expeditiously as possible, to prevent further loss. Although Bier may have been the first to speculate on the relationship between low CSF pressure and PDPHA, the first publication of this theory was made by MacRobert in 1918. Thus, PDPHA and its cause have been known since the advent of spinal anaesthesia, and it remains one of the most perplexing limitations of this anaesthetic technique.

It was not until the reports of Gormley in 1960 and DiGiovanni and Dunbar in 1970 that a definitive treatment was known for the syndrome. DiGiovanni and Dunbar described the epidural injection of autologous blood in 50 patients suffering from PDPHA. This proved to be an effective measure to stop the process of CSF leakage and postural cephalalgia. PDPHA remains the most common postoperative complication of spinal anaesthesia. Its diagnosis and treatment should be considered part of the standard armamentarium of the regional anaesthesiologist.

INCIDENCE

The incidence of PDPHA following spinal anaesthesia has been reported to vary from 0.2 to 24%. The generally quoted incidence is ~3%. PDPHA is more frequently noted in pregnant women receiving spinal anaesthesia, and in patients less than 50 years of age. In obstetric patients, a lowering of intra-abdominal pressure after delivery of the fetus may lower epidural pressure, and thus theoretically increase CSF leakage from a dural hole. Additionally, hormonal changes at the time of delivery may make the cerebral vasculature especially reactive and predispose parturients to PDPHA. There is a definite decrease in the incidence of PDPHA in older adults. After the age of 50 years, the risk of PDPHA declines appreciably.

There is considerable controversy regarding whether non-pregnant women are at any increased risk of developing PDPHA following dural puncture. A study by Lybecker et al included no parturients, and found no gender differences in the incidence of PDPHA following spinal anaesthesia. The literature on obstetrics suggests an incidence of PDPHA of up to 75% following dural puncture using 17- or 18-gauge needles. Norris demonstrated that orientating the epidural needle bevel parallel to the fibres of the dura decreases the incidence of PDPHA in labouring women following inadvertent dural puncture from 75 to 31%. For reasons yet unexplained, morbid obesity may decrease the incidence of PDPHA following epidural needle puncture of the dura. Thus, there are several potential patient-related factors which can affect the incidence of PDPHA.

In addition to patient-related factors, there are several technical factors which can affect the incidence of PDPHA. The incidence of PDPHA after dural puncture with epidural anaesthesia is markedly less when saline is used for the loss of resistance technique versus air (10 versus 65%). While the incidence of unrecognized dural puncture may occur in up to 1.5% of attempted epidural punctures, the incidence of PDPHA following attempted epidural block may be as high as 81–91%. Rotating an epidural needle once inside the epidural space may increase the risk of dural puncture (and hence PDPHA) and is therefore not recommended.
The most important technical factor in determining the incidence of PDPHA is undoubtedly the size of the dural hole produced during puncture. There are two major relevant sub-topics to this issue: needle size, and design of the needle point. There is a direct relationship of PDPHA to spinal needle tip diameter. The larger the diameter of the needle, the more frequent and more severe the headache. There is an incidence of >80% with 16-gauge needles versus 5% with 26-gauge needles; the overall incidence was 11% of 10,098 spinals in one large study. Use of small-gauge pencil-point spinal needles has reduced the incidence to about 0.02–1.5%. This latter factor is thought to be due to the smaller dural hole produced by a pencil-point needle compared with that produced by a cutting (Quincke) point needle, resulting in less leakage of CSF leakage.

For example, Flaatten et al compared the incidence of PDPHA after spinal anaesthesia using either 27-gauge pencil-point (non-cutting) versus 27-gauge Quincke (cutting) needles. There were 301 patients in the study; 153 in the pencil-point group, and 148 in the Quincke group. There were three PDPHAs in the pencil-point group, versus 12 in the Quincke group. A meta-analysis of other studies totalling 1131 patients showed a relative risk of developing PDPHA of 0.38 (95% confidence interval, 0.19–0.75) in the pencil-point (non-cutting) group compared with the Quincke group. However, there is an increased failure rate of spinal anaesthesia associated with 27-gauge spinal needles. PDPHA risk is increased if repeat dural puncture occurs after failed attempts at spinal block, even when needles of fine gauge are used. Multiple small dural holes can result in the same loss of CSF as from one large hole. As very small-gauge needles have an increased failure rate, a reasonable compromise is a 24-gauge pencil-point needle, which is currently the favourite spinal needle used by the present authors.

Although the highest incidence of PDPHA may occur in obstetric patients, as many as 40% of parturients who do not receive any neuraxial anaesthesia whatsoever complain of headache in the peripartum period. This emphasizes the need to rule out other causes of headache in any patient, including tension headache, migraine headache, caffeine withdrawal headache, sinusitis and pre-eclampsia. One must also consider the possibility of headache due to a pneumocephalus (from using the loss of resistance technique with air to identify the epidural space). In the case of pneumocephalus, usually the headache is worse in the recumbent position, and it typically resolves over several hours. Consideration must also be given to the possibility of subarachnoid haemorrhage, intracranial haemorrhage, subdural haematoma, or cortical vein thrombosis. Typically, these other causes of headache can be differentiated from PDPHA by lack of a postural component to the headache.

Not all headaches following spinal anaesthesia are due to leakage of CSF. Gurmanik proposed that the mild headache occasionally following spinal anaesthesia might result from a chemical irritation or chemical meningitis due to povidone-iodine antiseptic used to prepare the skin prior to performing the block.

PDPHA may result not from epidural or spinal needle puncture, but rather from epidural catheter puncture of the dura mater. From the anaesthesia records of about 9000 epidural techniques for obstetrical analgesia, Kalas and Hehre identified 19 epidural catheter punctures resulting in PDPHA in six of these patients (31.6%); in this series, there were 99 epidural needle punctures of the dura, resulting in 48 PDPHA (48.5%). In a larger series (26,490 labouring patients receiving epidural analgesia), two out of six (33%) patients with documented catheter-induced dural puncture developed PDPHA. Following continuous spinal anaesthesia using 20-gauge catheters, the incidence of PDPHA seems to be low (about 1%). Intrathecal catheters (20-gauge)
placed through 18-gauge needles did not have a higher incidence of PDPHA than smaller continuous catheters placed through 20-gauge needles or compared with 'single-shot' spinal anaesthesia using 22-gauge pencil-point needles. The reason for this low incidence of PDPHA after continuous spinal anaesthesia is not known. Some have speculated that an inflammatory reaction around the catheter reduces the loss of CSF via the dural hole. There is little direct evidence to support this hypothesis.

PDPHA infrequently occurs following combined spinal/epidural anaesthesia (CSE). Inadvertent dural puncture does occur with similar frequency following CSE compared with lumbar epidural anaesthesia. There are several possible explanations for this observation. First, the reduced incidence of PDPHA following CSE may be due to the epidural needle acting as an introducer for the spinal needle, reducing the number of attempts to puncture the dura using the spinal needle. Second, 25- to 27-gauge spinal needles are used for CSE, and the incidence of PDPHA following their use is low (< 2%). Third, the injection of local anaesthetic (LA) into the epidural space during CSE reduces leakage of CSF through the dura secondary to increased epidural pressure.

Fourth, epidural opioids may provide a prophylactic effect against PDPHA. Again, direct evidence for these possible explanations is lacking at present.

CLINICAL FEATURES OF PDPHA

A history of dural puncture and a postural component of the headache are essential factors in making a diagnosis of PDPHA. PDPHA usually occurs within a minimum of a few hours following dural puncture. The onset of headache usually occurs within the first or second day after dural puncture but may occur 5 days or more later. For the first 24–48 h following dural puncture, loss of CSF exceeds production. Eventually, the dural rent is repaired by fibroblasts forming a fibrin seal. However, there are reports of chronic PDPHA occurring up to 5 months following dural puncture. Bed rest following dural puncture has not been shown to prevent, but merely to postpone, the development of PDPHA. In almost all cases, including chronic PDPHA, a history of worsening the headache when assuming the sitting or standing position, due to a lowering of CSF pressure, can be elicited.

The typical location of the headache is bifrontal and/or occipital. Occasionally, symptoms involve the neck and upper shoulders. Intensity ranges from mild to excruciating. Classically it is worse in the upright position or during coughing or straining. Symptoms usually subside with recumbency. There may be associated nausea, loss of appetite, photophobia, and changes in hearing acuity and tinnitus, as well as depression. In severe cases, there may be diplopia and cranial nerve dysfunction and nerve palsies secondary to traction on those nerves. All of these symptoms can be explained by low CSF pressure (see below).

PATHOPHYSIOLOGY

Any theory explaining PDPHA must account for the relationship of the headache to the loss of CSF. One theory states that the loss of CSF through a dural hole results in intracranial tension or traction on nerves and meningeal vessels. There is support for this theory from audiometric studies performed on patients who developed temporary hearing loss and PDPHA after dural puncture. These studies also documented
improvement in hearing acuity and resolution of the headache following epidural blood patch. The bimodal theory of the pathophysiology of this syndrome was first published by Kunkle et al in 1943. The bimodal theory suggests that there is a combination of both low CSF pressure and resultant cerebral vasodilation in reaction to the stretching of vessels.

In the normal human, 450–500 ml of CSF is produced per day (0.3 ml/min). The normal CSF volume is 150 ml, which is divided about equally between intracranial and spinal CSF. The major portion of CSF (90%) is produced from filtration of blood in choroid plexuses in the lateral, third and fourth ventricles of the brain and is subsequently absorbed back into blood by the arachnoid granulations found in the superior sagittal and transverse venous sinuses. The other 10% of CSF is derived from brain substance itself. There is a slow movement or circulation of CSF from the ventricles to the intracranial subarachnoid space and then to the venous sinuses. The spinal subarachnoid space is in effect a 'backwater' in which there is very little movement of CSF except by simple diffusion and postural changes. As the body generates its entire CSF volume three times per day, all that is necessary to treat a PDPHA is to stop the leakage of CSF.

Decreased brain ‘buoyancy’ owing to low intracranial pressure causes traction on structures supporting the brain and cranial nerves. A caudal shift of the brain occurs with a decreased volume of CSF, causing dural stretching and traction on pain-sensitive intracranial structures (cerebral vessels, falx cerebri and tentorium). The trigeminal, glossopharyngeal and vagus nerves are thought to be involved. Vagal traction may stimulate chemoreceptors in the medulla to produce nausea. Traction on the abducens and trochlear nerves can lead to ocular muscle paralysis, with strabismus and double vision resulting. Hearing loss has also been reported, due to low CSF pressure transmitted to the inner ear via the cochlear aqueduct. This decrement in hearing is fully reversible once normal subarachnoid pressure is restored.

Although there is no evidence that the body compensates for a loss of CSF by increasing its production, venous dilatation does occur, which partially compensates for loss of intracranial volume. This phenomenon may be responsible for the sensation of pressure that is experienced with PDPHA. When standing, the intracranial pressure decreases and intracranial venous distention increases, worsening the headache. When supine, equalization of intracranial cisternal and lumbar CSF pressures takes place, so no expansion of the venous system occurs, and the headache improves. Neck pain may result from tension on cervical nerves 1–3. Neck pain may develop into a myofascial-like pain. Trigger point injections are sometimes helpful in treatment.

Intracranial subdural haematoma is a very rare complication of prolonged reduction of CSF pressure. This has been associated with a 14% mortality rate, and a high rate of persistent neurological complications in those who survive, emphasizing the need to diagnose and treat persistent PDPHA expeditiously.

The amount of CSF loss depends upon the size and shape of the dural hole and the pressure difference between the subarachnoid and epidural spaces. The design of the spinal needle tip and orientation of a cutting needle bevel are both factors in determining the rate of CSF loss (see below). In one in vitro study performed using human postmortem thoracolumbar dura mater (Figure 1), the median loss of CSF volume in 5 min was significantly less with a 22-g Whitacre needle than with a 22-g Quincke. There was a 21% reduction in leakage of CSF if the Quincke needle bevel was parallel to the large axis of the vertebral column. Orienting the Quincke needle bevel in the longitudinal axis promotes separation instead of cutting of dural fibres. However, this cadaver study has been disputed by anatomical and in vitro analyses. A classic
clinical study by Mihic demonstrated that the incidence of PDPHA is lower when a Quincke needle bevel is oriented parallel to the longitudinal fibres. The size of a dural hole relates to outside diameter of a given spinal needle, as well as to bevel configuration. Cutting fewer fibres of the dura reduces the size of the dural hole. Needle tips that stretch dural fibres should reduce the size of a resulting hole, in comparison to tips that cut fibres; hence the rationale for using pencil-point needles rather than cutting needles. The dura mater consists of tough fibrous tissue that varies in thickness, even in the same individual. Fibres run longitudinally, but also transversely. In 1988, Dittmann et al reported that dural fibres are not uniformly parallel, and that the thickness of the dural sac varies. They described the so-called ‘tin can lid’ phenomenon: needle perforation of the dura with a Quincke needle resembles the top of an almost completely opened tin can with the lid hinged at its base. The hole tends to be ellipsoidal when the needle is inserted through a thick section of dura. By contrast, when a needle of the same gauge and design perforates a thinner section of dura, the resultant orifice is larger, and it closes much more slowly. A hole left in the dura resembles an opened tin can rather than a neat punched out hole. The resulting flap may fall back into place, virtually sealing the hole (Figures 2–4). Quincke needles produce an oval or ellipsoidal hole, as opposed to Whitacre or Sprotte needles, which produce a more rounded hole.

Figure 1. MRI confirmation of cerebrospinal fluid collection following dural puncture at L2–L3. The arrows demonstrate the fluid mass (sagittal spin echo proton density). (Reproduced with permission from Reference 61.)
Figure 2. Scanning electron micrograph of a dural puncture hole made by a 25-gauge Quincke (cutting) needle. View is from the inner (intrathecal) side of the dura mater. Note the ‘open-tin-can’ configuration. (Photograph courtesy of Drs Mark Johnson and Vladimer Bittner)(Reproduced with permission from Reference 61).

Figure 3. Scanning electron micrograph of a dural puncture hole made by a 25-gauge Quincke (cutting) needle. View is from the outer (epidural) side of the dura. (Photograph courtesy of Drs Mark Johnson and Vladimer Bittner)(Reproduced with permission from Reference 61).
The arachnoid mater is closely applied to the dura mater, but is not attached to it. For CSF to leak, the hole in the dura must be closely aligned to a hole in the arachnoid mater. An angled approach to the subarachnoid space, such as a paramedian approach, is associated with a lower incidence of PDPHA, although at least one study does not support this contention. In summary, pencil-point needles produce smaller dural holes, a reduced loss of CSF, and lower rates of PDPHA than do Quincke point needles of the same size. When using a Quincke point needle, a paramedical approach produces a decreased loss of CSF and a lower incidence of PDPHA. A lower incidence of PDPHA is seen when the bevel of the Quincke needle is oriented parallel to the longitudinal axis of the body.

**TREATMENT**

Based on the theories of PDPHA discussed above, it should seem readily apparent that the goal is to restore CSF pressure. It should be kept in mind that the natural history of PDPHA is one of resolution with time, with up to 90% resolving within 10 days of onset. It is essential to consider other possible diagnoses of headache prior to embarking upon treatment. Failing to recognize other potential causes might result in treatment failures, or worse, in misdiagnosis of other life-threatening causes of cephalalgia.

In cases where the headache appears to be related to the dural puncture, i.e. is postural in nature, conservative measures may be attempted for headache. These

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**Figure 4.** Scanning electron micrograph of a dural puncture hole made by a 25-gauge Whitacre (non-cutting) needle. View is from the inner (intrathecal) side of the dura mater. Note that the fibres are ‘torn’ rather than ‘cut’, resulting in an ellipsoidal configuration. (Photograph courtesy of Drs Mark Johnson and Vladimer Bittner) (Reproduced with permission from Reference 61).
include the use of non-steroidal anti-inflammatory drugs (NSAIDs), acetaminophen, bed rest (to avoid symptoms, not to prevent onset), and possibly weak opioid analgesics. In cases where the patient is unlikely to lie flat, such as a mother who has just delivered a baby, and who will have an over-riding need to get out of bed to care for her newborn, conservative measures are not likely to be effective. Abdominal binders force more venous blood through the epidural venous plexus, and although not advocated by the present authors, might have a place in very mild cases or in cases where the patient wishes to exhaust all non-invasive modalities prior to considering epidural blood patch. Aggressive hydration of patients (to try to stimulate CSF production) is of no proven benefit. Additionally, this manoeuvre will result in a brisk diuresis and will subsequently interfere with instructions to lie flat.

Caffeine, a methylxanthine, has a cerebral vasoconstrictor effect, and may have some efficacy in the management of PDPHA; however, caffeine is also a diuretic. Caffeine is inexpensive, widely available, and most importantly is associated with minimal risk. Camann et al evaluated 40 postpartum patients with headache in a randomized, double-blind, placebo-controlled study. Caffeine 300 mg orally versus placebo resulted in a 300% reduction in the visual analogue pain score at 4 h after administration, but there was no difference at 24 h. Caffeine may also be administered parenterally. For intravenous use, caffeine is combined with sodium benzoate to enhance solubility. Sechzer and Abel performed a randomized, double-blind, placebo-controlled study consisting of 41 patients with PDPHA who received either placebo or intravenous caffeine 500 mg. All 41 patients subsequently received intravenous caffeine 500 mg, regardless of initial treatment or outcome, and 71% ultimately achieved relief of headache. Their conclusion was that intravenous caffeine is a safe and effective method for treating PDPHA. However, placebo-controlled studies of either oral or intravenous caffeine have not shown a reduction in the number of patients subsequently requiring epidural blood patch.

Other intravenous agents that have been used in an attempt to relieve PDPHA include sumatriptan and adrenocorticotropic hormone; both have demonstrated some efficacy, but have limited evidence to support their use. Sumatriptan, a 5-HT agonist, is further limited by its 2-h plasma half-life.

EPIDURAL INJECTIONS

Autologous epidural blood patch (AEBP) has become the ‘gold standard’ in the treatment of PDPHA. As there is some risk of infection when injecting blood into the epidural space, we will discuss the efficacy of some other aqueous agents which have been injected into the epidural space to treat PDPHA. Prior to considering the use of epidural injections of blood or other substances to relieve the symptoms of PDPHA, there needs to be a clearly negative history of sepsis and coagulopathy. HIV infection is not considered to be a contraindication to AEBP (see below).

Dextran and 0.9% NaCl (saline) injections into the epidural space function by the same basic mechanism, i.e. to transiently increase pressure in the epidural space, which subsequently decreases the leakage of CSF and restores subarachnoid pressure. Unfortunately, this effect is only transient. Dextran, being of large molecular weight, stays in the epidural space longer than saline and provides increased pressure for longer periods of time. Bolus injection of epidural dextran 20 ml has relieved headache refractory to bed rest, hydration or caffeine, with long-lasting relief occurring within
10 min of injection. Unfortunately, the success rate is moderate. Anaphylaxis has been reported following the use of dextran for this purpose.\textsuperscript{83,84} Injection of 0.9\% NaCl (saline) into the epidural space after unintended dural puncture dates back to 1950, when Rice and Dobbs reported immediate relief of headache in 99.5\% of cases with a 54\% recurrence rate.\textsuperscript{85} Usubiaga et al and Baysinger et al showed that intermittent injections of saline 10–30 ml relieved headache, without reported re-occurrence.\textsuperscript{86,87} It is believed that the mechanism of action of both saline and dextran involves an increase in epidural pressure which reduces outflow of CSF, while a fibrin clot forms over the dural puncture site.\textsuperscript{63,88} Other studies show very limited effects of prophylactic epidural saline infusions on the prevention of PDPHA, particularly in postpartum patients.\textsuperscript{22,24,89}

**AUTOLOGOUS EPIDURAL BLOOD PATCH**

The autologous epidural blood patch was first described by Gormley in 1960, and later popularized by DiGiovanni et al.\textsuperscript{3,4,90} The suspected mechanism of action of AEBP is by tamponade of the dural leakage while raising the subarachnoid pressure. Elevation of subarachnoid and epidural pressures remains so only for about 20 min.\textsuperscript{86,91} MRI evidence confirms a mass effect after injection of epidural blood, with gradual resolution over about 7 h (Figure 5 A–C).\textsuperscript{92} Unlike saline, dextran or other fluids, blood is not removed quickly from the epidural space\textsuperscript{93}, and it potentially exerts a tamponade effect for much longer periods of time. The autologous blood is thought to form a fibrin clot over a dural rent, allowing CSF volume (and hence, pressure) to normalize as new CSF is generated.\textsuperscript{94}

Abouleish et al summarized 524 cases of AEBP reported by 11 centres.\textsuperscript{95} Persistent symptomatic relief of PDPHA following epidural blood patch was >95\%, particularly when using volumes of blood >15 ml. In this review, using volumes of blood greater than 20 ml offered no advantage as it is known that 20 ml spreads about 9–10 spinal segments when administered to patients in the sitting position.\textsuperscript{12}

Some studies have demonstrated lower success rates, with only 61–75\% of patients demonstrating sustained benefit. These lower success rates may reflect dural puncture occurring with large-bore epidural needles versus smaller-gauge spinal needles.\textsuperscript{22,63,96,97} In obstetrical studies, the success rate of epidural blood patch for PDPHA is lower, because the dural hole made by 17-gauge Tuohy needles results in a large leakage of CSF, necessitating a second blood patch in as many as 29\% of patients.\textsuperscript{22,23,25}

**THE TECHNIQUE OF AUTOLOGOUS EPIDURAL BLOOD PATCH**

The procedure is performed only after a careful history to exclude other causes of headache. Contraindications to blood patch include infection at the skin puncture site or untreated sepsis. Informed consent is obtained and risks, benefits and alternatives are explained. Strict sterile technique is mandatory. While some authors recommend the administration of prophylactic antibiotics for the procedure, the present authors do not. The patient is placed in either the sitting position (for obese patients to facilitate identification of the midline) or the lateral decubitus position (particularly if the headache is severe). Rarely, if fluoroscopy is being utilized, the prone position may be selected. After carefully identifying a prominent vein in an upper extremity, a sterile skin
prep and drape is performed. Concomitantly, a skilled anaesthesiologist chooses either the previous level of puncture (if this can be discerned following multiple needle attempts), or, alternatively, one level below the previous injection site. These are the preferred interspaces to inject into, because blood preferentially rises cephalad following its injection into the lumbar epidural space with a patient in the sitting position.92,93,98 Once the anaesthesiologist has gained entrance into the epidural space at the selected level, the phlebotomy should proceed with the withdrawal of at least 20 ml of venous blood. It is important to wait until epidural entry has been successful, because the venous blood might otherwise clot if it is withdrawn too early. The blood is carefully, and aseptically, transferred to the anaesthesiologist, who injects it slowly through the epidural needle until one of several endpoints occurs: (a) if the patient complains of backache or worsening headache during the performance of the epidural

Figure 5. (A) MRI demonstrating autologous blood in the epidural space. Blood is primarily seated at L2–L3, but extends as high as T12 and as caudal as L4. (Reproduced with permission from Reference 61). (B) MRI demonstrating T2-weighted image of autologous blood in the epidural space. In this view, there is less contrast between the blood and other anatomical structures including bone and soft tissues. The arrows indicate the mass of blood primarily seated at L2–L3. (Reproduced with permission from Reference 61). (C) MRI demonstrating adjacent slice to Figure 5(A) and (B). Here, the regional morphology of the spinal cord, blood pooling along the thecal sac and its anterior layering (arrow) is shown. (Reproduced with permission from Reference 61).
injection, or (b) once at least 15 ml have been successfully injected without complaint by the patient. Patients are typically placed supine for up to 30–60 min following the procedure to reduce the leakage of CSF, out from the dural hole. A period of 30–60 min is long enough for the fibrin clot to form. The patient is advised to avoid straining, bending or heavy lifting for 2–3 days to allow the dural hole to heal.

As regards the optimum volume of autologous blood to be injected epidurally, Taivainen et al found that using 10 ml standard in all patients was equivalent to 10–15 ml variably administered based upon height; there was no significant difference in early or late success rates between groups in this one study. Others have advocated more generous volumes. Crawford found that 20 ml was associated with 96% success, versus 70% success using 6–15 ml. The ideal time to perform an epidural blood patch is within 24 h of puncture. Treatment failure after blood patch may reflect continued transdural leak; in this case, the blood patch should be repeated while keeping the patient flat for 24 h afterwards to reduce the flow of CSF through the dural hole.

Complications following AEBP include the following: backache (35%), neck ache (0.9%) and transient temperature elevations (5%) lasting 24–48 h. Bleeding, infection, repeat dural puncture, and arachnoiditis from blood injected into the subarachnoid space have been reported. There have been at least two cases of facial nerve paralysis reported following autologous blood patch, both of which resolved spontaneously.
Lowe and McCullough suggested that the aetiology is ischaemia of the 7th nerve resulting from decreased blood supply after an increase in intracranial pressure due to the injection of blood in the epidural space. There has also been at least one case of intractable dizziness, vertigo, tinnitus and ataxia. Blood patch has occasionally been associated with vasovagal syncope.

Subsequent epidural anaesthesia is probably not affected by a previously performed epidural blood patch; however, there exists considerable controversy regarding this subject. Ong et al identified 29 parturients who had epidural anaesthetics after previous blood patch; there was a 59% success rate in this group, compared with an 88–92% success rate in individuals who did not have a previous blood patch. These authors also reviewed 17 patients who had dural puncture without subsequent blood patch; in only 65% was subsequent epidural anaesthesia successful. A study by Hebl et al failed to demonstrate any difference in success of subsequent epidural anaesthesia between patients who received epidural anaesthesia following blood patch (29 cases) versus individuals who had not undergone blood patch procedures. This corresponds to findings of other investigators. Thus, we believe that there is little reason not to offer epidural anaesthesia to a patient who has previously undergone epidural blood patch. Despite concerns of meningitis, arachnoiditis or CNS spread of HIV infection occurring after a patch, to date these have not been demonstrated to occur following an AEBP.

PREVENTION

Using small-gauge, pencil-point spinal needles for spinal anaesthesia minimizes the likelihood of PDPHA. When using a Quincke point needle, aligning the spinal needle bevel longitudinally so as to separate the longitudinal dural fibres, rather than shearing the fibres, further minimizes dural leakage of CSF. The use of a paramedian approach to the subarachnoid space has been touted as a means of reducing PDPHA. Avoiding multiple puncture holes in the dura mater is also recommended to avoid PDPHA. There is little advantage in using small (25–29 gauge) Quincke point needles compared with 24-gauge pencil-point needles. In summary, most authorities suggest using smaller-gauge, non-cutting needles, minimizing the number of puncture attempts, and aggressive follow-up of the patients to detect PDPHA when dural puncture (intentional or unintentional) does occur, so that early treatment can be instituted.

PROPHYLACTIC BLOOD PATCH

Some have suggested that blood patch be performed as a prophylactic measure (i.e. prior to the development of a headache) in cases of unintended dural puncture occurring after the insertion of 17–18-gauge epidural needles into the subarachnoid space, particularly when there has been loss of considerable quantities of CSF. To date, there have been no large, prospective studies to advocate for this practice, although it is widely practiced. Existing studies regarding prophylactic epidural blood patch are limited by small patient numbers. Also, there have been limitations suggested by the small volumes of blood injected in some reviews. If one chooses to perform prophylactic AEBP, some caveats are in order; one should avoid prophylactic AEBP
immediately following LA epidural top-off dose administration, because the resultant high epidural pressure has resulted in at least one case of total spinal block.\textsuperscript{119} Also, the presence of LA in the epidural space may theoretically interfere with subsequent blood clot formation.\textsuperscript{120} There are strong advocates for prophylactic blood patch, particularly if an epidural catheter is in place, avoiding the need for another epidural puncture.\textsuperscript{121–123} Given the low additional risk, as long as strict sterile technique is employed, prophylactic epidural blood patch in high-risk patients (e.g. young, postpartum, large-gauge needle puncture) would seem to be worthwhile and is certainly justifiable. However, this decision needs to be individualized to the patient.

### Summary and Conclusions

Post-dural puncture headache (PDPHA) continues to be a vexing problem for patients and anaesthesiologists. Avoidance of this problem is the best treatment strategem. Using small-gauge (e.g. 24- or 25-gauge) pencil-point needles has been shown to minimize PDPHA. Early diagnosis, by eliciting a history of a postural headache following a lumbar puncture, and by ruling out other causes of headache, and early treatment with AEBP has been shown to be the most effective treatment of PDPHA. This is most important in patients younger than 50 years of age, postpartum patients, and those whose dural puncture was performed with large ($\geq$22-gauge) needles. Although the natural history of untreated PDPHA is gradual resolution of the headache over 5–7 days, chronic PDPHA has been reported. The authors recommend using 24-gauge pencil-point needles for spinal anaesthesia and minimizing the number of dural punctures to reduce the risk of PDPHA in patients at high risk for this complication.

### Practice Points

- Using small-gauge pencil-point needles can minimize the risk of PDPHA. The authors recommend a 24-gauge Sprotte or Whitacre needle. The paramedian approach may have a lower risk of PDPHA than a midline approach.
- If a headache occurs in a patient following dural puncture, a careful history can rule-in or rule-out PDPHA. The sine qua non of a PDPHA is a positional change in the headache. In high-risk patients (young, postpartum, large-gauge dural puncture), an AEBP should be performed within 24–48 h of onset of headache.
- In a patient with an epidural catheter in place, consideration should be given to prophylactic epidural blood patch.
- Alternatives to AEBP are not nearly as effective, although short-term relief from headache is often seen. The optimum volume of epidural blood appears to be 15–20 ml. Treatment failures do occur, and chronic PDPHA has been reported. Therefore, follow-up of the patient is important.

### Research Agenda

- The optimum timing of epidural blood patch with respect to dural puncture has not been determined.
- The optimum treatment of chronic PDPHA has not been determined.

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