Postdural puncture headache: A literature review

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This is a review of literature from 1943 to mid-1989 on the postdural puncture headache. The article looks at the currently held thoughts on the cause, prevention, and treatments of this second most frequent side effect of spinal anesthesia. Postdural puncture headache (PDPH) is caused by vascular distension within the nondistensible cranium following the leakage of cerebral spinal fluid (CSF) into the epidural space. Prevention of PDPH can be accomplished by using small-gauge needles and possibly by using the lateral approach, as opposed to the midline approach. Luck plays a big part, because if the needle punctures a thicker portion of the dura, there is a reduced chance of PDPH.

Epidural saline injection is effective only if it is used as a continuous infusion for 24 hours. The usefulness of caffeine sodium benzoate with a 70-80% success rate and epidural blood patching with a 90%-plus success rate are discussed.

Key words: Cerebral spinal fluid, headache, postdural puncture headache.

What is a PDPH? Its most distinctive characteristic is its dependence upon posture. Diagnosis is made primarily by the presence of a headache that is most intense in the sitting or upright position and diminishes or disappears in the horizontal position or with flexion and extension of the neck.

Only rarely is PDPH unremitting. Fifty percent of patients experience frontal pain, and occipital pain occurs in 25% of the remainder. Pain in the parietal area was reported by 1.4% of patients, and the rest reported generalized, diffuse pain in the head and neck. The incidence of PDPH does not seem to be linked to the occurrence of parasthesias or a bloody tap. In one study, neither a bloody tap nor a paresthesia was elicited in 96% of the PDPHs studied.

Anatomy and physiology

The size of the dural hole and the speed with which it closes seem to be the most important factors determining the occurrence of PDPH. The absence of PDPH is probably the result of the prolapse of the arachnoid through the dural opening. Greene demonstrated in postmortem specimens that the dural sac was more firmly attached anteriorly than posteriorly, indicating that it moved upward with flexion of the vertebral column and neck.

The components of the sac—the thick dura mater and the thin inner arachnoid mater—slide nonuniformly with flexion (Figure 1). Therefore, the holes in both layers from the needle puncture are not usually in alignment when the patient
dural sac, which would cause a slit rather than a simple puncture in the two layers. Therefore, when the patient returns to a neutral position, the hole could overlap the layers, allowing the CSF to leak into the epidural space (Figure 3).

CSF leak would probably occur if there were adherence of the layers, failure to properly position the patient while performing the tap, or movement of the patient while the needle was piercing the dural sac, which would cause a slit rather than a simple puncture in the two layers. Therefore, when the patient returns to a neutral position, the hole could overlap the layers, allowing the CSF to leak into the epidural space (Figure 3).

In 1898, during the earliest experimentation with spinal anesthesia, Augustus Bier became one of the first recorded PDPH sufferers. He suggested that the etiology of his discomfort was leakage of CSF into the epidural space. With the patient in the upright position, there can be 40 to 50 cm of H₂O pressure gradient between the intradural CSF and the epidural space, making it possible for fluid to be lost into the low pressure space. While CSF production is about 0.35 mL/min, the rate of CSF reabsorption is proportionate to CSF pressure. Horizontal positions increase CSF pressure within the head, hastening the rate of reabsorption; vertical positions decrease CSF pressures, slowing the rate of reabsorption. As a result, decreasing the rate of reabsorption and increasing the rate of production cannot, in all instances, make up for the fluid lost into the epidural space within short periods of time. When the patient assumes the recumbent position to relieve the symptoms, the CSF pressures are equalized between the intercranial (IC), cisternal, and lumbar areas, thereby slowing the leakage and restoring CSF reabsorption to normal ranges.
Fink and Walker showed that leakage of fluid from the dural sac requires a critical pressure.\(^8\)
When the patient lies flat the CSF pressures of the lumbar, cisternal, and IC areas are equalized in the \(H_2O\) pressure range of 5 to 15 cm.\(^5\) However, in the standing position, these ranges change dramatically to \(-30\) cm of \(H_2O\) pressure in the superior IC area, 0 cm of \(H_2O\) in the cisternal area, and up to a maximum of 50 cm of \(H_2O\) in the lumbar area.\(^3\) The pressure gradient leads to a downward shift of the brain, with stretching of the dura, blood vessels, and nerves.\(^3\)\(^,\)\(^5\) The 9th and 10th cranial nerves (glossopharyngeal and vagus) and the three cervical nerves (C-1, C-2, C-3) below the tentorium cerebelli relay pain from the suboccipital regions and from the neck and shoulder areas. The second and third divisions of the fifth cranial nerve (trigeminal) above the tentorium cerebelli relay pain from the frontal area, while the sixth cranial nerve (abducens), which has the longest IC course, relays visual symptoms.\(^1\)\(^,\)\(^3\)\(^,\)\(^5\)\(^,\)\(^8\)

Current thought on IC pressure modulation holds that a reduction in volume of any IC component must be accompanied by an equalizing increase in volume in another IC structure. Therefore, dilatation of IC blood vessels occurs with decreases in CSF volume. Pain receptors in the venous sinuses, sinus tributaries, dural arteries, and cerebral arteries at the base of the brain and portions of the dura in this area are all stimulated. Therefore, the genesis of the PDPH is vascular in origin.\(^1\) Because of the neural distribution of the affected structures, cervical muscle spasm frequently occurs, leading to stiffness varying from a muscular tension headache to meningismus.

**Prevention**

Prevention of PDPH is contingent upon a number of factors of varying importance. There is an inverse relationship between PDPH and increasing age.\(^1\)\(^,\)\(^3\)\(^,\)\(^9\)\(^,\)\(^10\) This might be explained by decreasing pain sensitivity, loss of neural elements, and loss of distensibility of pain-sensitive structures. The incidence of PDPH is higher among females and is twice as frequent in the under-50 age group, even when patients who had spinal anesthesia for vaginal delivery are excluded.\(^1\)\(^,\)\(^3\)\(^,\)\(^5\)\(^,\)\(^8\)

The incidence of PDPH is highest in the parturient, because of a number of factors. Increases in CSF pressures from bearing down during vaginal delivery, dehydration secondary to nothing-by-mouth status, blood loss, postpartum diuresis, and decreases in intra-abdominal and peridural pressures postpartum can all contribute to increases in the incidence of PDPH in the parturient.\(^1\)\(^,\)\(^3\)\(^,\)\(^5\)

Hydration has long been thought to play a role in the prevention and treatment of PDPH. While volume preloading has reduced the incidence and magnitude of spinal anesthetic-induced sympathetic blockade, it does not seem to have a bearing upon the incidence of PDPH.\(^5\)\(^,\)\(^6\) The rationale behind increasing oral fluid intake was to increase CSF pressure by increasing production. Physiologically, CSF production cannot be directly increased by oral fluid intake.\(^6\) In fact, CSF loss is greatest in the upright position, so an increase in production would only lead to an increase in the loss of CSF. Increasing oral fluid intake does not influence the duration of PDPH, but underhydration could be a factor influencing the incidence of PDPH.\(^6\)

The needle bevel and orientation may have some small bearing on the incidence of PDPH. Greene stated that since the dural fibers are parallel in the longitudinal plane, a small needle inserted with the bevel oriented parallel to these fibers would separate rather than cut, thereby lessening the likelihood of a dural hole that remains open.\(^4\)\(^,\)\(^7\)

Light microscopy indicates that the direction of the fibers that comprise the dura are nonuniform.\(^8\) The lamellation of the dura is often concentric with the lumen of the canal, or it may be relatively homogeneous longitudinally. If it is longitudinal, then the increased CSF pressure associated with sitting would be more likely to cause the dural sac to gape from a longitudinal slit than from a transverse slit.\(^6\) A gaping longitudinal slit would tend to leak CSF more readily.

While most spinalis are done using the midline approach, the incidence of PDPH may be related to the angle at which the dura is punctured. In one study using 20-gauge needles and exclusively the lateral approach, the incidence of PDPH was 0% for 600 spinal anesthetics over a 5-year period.\(^9\) However, no criteria was provided for the diagnosis of PDPH, which might have skewed the results.

The Greene needle, with its smooth round form, has the Whitacre needle leaked 46% less than a 22-gauge Quincke needle (the standard needle point in most commercial spinal trays), when inserted with the bevel aligned across the fibers.\(^12\) The same study showed a 21% reduction in leakage if the Quincke needle bevel was parallel to the long axis of the vertebral column. A 22-gauge pencil-point needle produced a higher median but statistically insignif-
significant fluid loss when compared to a 26-gauge needle, with the bevel either parallel or transverse to the dural length.

The investigators found that punctures frequently left a "V" or crescent-shaped hole. These holes frequently have a flap similar to the lid of a can that has been opened, leaving only a small attachment point (Figure 4). With a "tin lid" flap, the dura can close holes produced by even the largest spinal needle.13

Almost all authors agree that needle size seems to have a direct correlation with the incidence of PDPH.1,5,10,12 The use of 29- and 30-gauge needles is associated with less than a 1% incidence of PDPH. However, use of these needles may lead to a higher rate of failure to achieve spinal anesthesia.12 The delayed appearance of CSF at the needle hub could be the contributing factor. It can take up to 61 seconds for CSF to appear at the hub of a 29-gauge needle.12 Vandam and Dripp give percentages of PDPH for each gauge of needle, while noting an 11% overall incidence of PDPH in 11,000 spinal anesthetics (Table I).7 Such percentages vary by author.

A 1989 report states that a double-blind study showed no significant difference in the incidence of PDPH in a 400-patient group when 20-gauge and 25-gauge needles were used. The mean age of the patients was 68.9.14 However, there was a significant difference in the incidence of PDPH in a corresponding group of patients whose mean age was 29.5 years.14

Even the size of needles vary by country of manufacture. The British Imperial Standard Wire Gauge (SWG), an older method of measure, is not the same as American measures. A U.S. standard 25-gauge needle is nominally .556 mm, while the SWG 25-gauge needle is .508 mm. Therefore, an American 25-gauge needle is equivalent to an SWG 24-gauge needle.15 This difference could be important to those doing missionary work abroad.

Until 1970, it was standard practice to have the patient lie flat for up to 24 hours after a lumbar puncture. This is still the standard in a few areas. (The author found this misinformation still being followed by the nursing service at his hospital.)

A number of studies published since 1970 have shown that restricting a patient to the horizontal position does not change the incidence of PDPH (Figure 5).2,3,16 Recumbency only delays the onset of symptoms, thereby possibly delaying the patient's release from the hospital. Chinese studies suggest that 3 days of recumbency would alleviate a high percentage of PDPH, but in the United States it is impractical in light of diagnosis-related groups (DRGs) and the use of ambulatory surgical centers.2,5 With the onset of symptoms, periodic recumbence is a sensible therapeutic measure for mild to moderate PDPH.

Although a continuous spinal, in which a catheter is placed through the dura, might be expected to result in an incidence of PDPH, in reality the incidence is less than 1%.17 This report suggests that a localized inflammatory edema develops in the dura and arachnoid membranes, thereby preventing the leakage and PDPH.17

Table I
Percentages of PDPH by needle size

<table>
<thead>
<tr>
<th>Needle Size</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>16-gauge</td>
<td>18%</td>
</tr>
<tr>
<td>19-gauge</td>
<td>10%</td>
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<tr>
<td>20-gauge</td>
<td>14%</td>
</tr>
<tr>
<td>22-gauge</td>
<td>9%</td>
</tr>
<tr>
<td>24-gauge</td>
<td>6%</td>
</tr>
<tr>
<td>26-gauge</td>
<td>5%</td>
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Adapted from Vandam1

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Figure 5
Headache rates for different lengths of time in recumbency

- % Headaches
- Hours recumbent

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Incidence

The severity of symptoms enables the classification of PDPH into three groups. Slight PDPH — 49% of the reported headaches — does not interfere with the patient's normal activities. Moderate PDPH, which accounts for 35% of the reported cases, is a headache bothersome enough to cause periodic returns to recumbency for pain relief. Severe PDPH — 15% of the reported headaches — is so painful that the patient cannot remain seated long enough to eat. \(^5\) The onset of PDPH may be immediate or delayed several days, presumably depending on the rate of CSF leakage required to outpace CSF production.

The duration of PDPH is usually self-limiting to approximately 3-5 days. It seldom lasts longer than 1 week, with 80% to 85% of cases being of less than 5 days' duration. \(^1\) The symptoms of PDPH start within 3 days of dural puncture in 90% of cases. \(^3\) One unusual case of a 19-month PDPH has been reported which was resolved with one autologous blood patch. \(^18\)

Ocular and auditory symptoms are reported with prolonged and/or severe PDPH, at an incidence of 0.4% for each. The ocular symptoms include blurred vision, double vision, and, infrequently, paralysis of lateral gaze. \(^3\) These symptoms are caused by pressure paralysis of the sixth cranial nerve, the motor nerve innervating the lateral rectus muscle. \(^19\)

Auditory symptoms include decreased hearing, dizziness tinnitus, unilateral deafness, and hyperacusitus, a painful sensitivity to sounds. When these symptoms are present, they are frequently accompanied by vertigo and ataxia, which are present because the semicircular canals and cochlea are in communication with the main CSF reservoir.

A recent study of patients with PDPH showed a decrease of 25% in hearing ability at a frequency of 125 Hz and an 18% reduction in the 250 Hz frequency range. The hearing loss was significantly reduced by using a 26-gauge needle as opposed to a 22-gauge needle. \(^20\) Although the hearing losses were small and of no clinical importance to the patients in this study, they demonstrate that audiometry may be a more sensitive measure of CSF leakage than PDPH. \(^1\) \(^20\)

Before a confirmed diagnosis of PDPH can be made, other causes of headache must be eliminated by physical examination and careful questioning. The other types of headache which must be excluded are migraine, cortical vein thrombosis, meningitis, and hematoma.

The migraine headache patient will usually have a long history of this type of headache. It is unilateral, pulsatile, and usually characterized by vasomotor symptoms. The key to differential diagnosis of PDPH and migraine headache is the failure of the supine position to provide any relief of the latter.

Cortical vein thrombosis (CVT) has a reported incidence of 1 in 6,000 to 1 in 600,000 parturients, with preeclamptic patients showing a greater propensity toward development of CVT. \(^21\) CVT is caused by obstruction of the superior longitudinal sinus or the cortical veins with thrombi. The thrombosis produces decreased CSF reabsorption and increased IC pressures. CVT is difficult to differentiate from PDPH, but there are subtle differences. CVT headaches are intensely throbbing and of increasing intensity. Frequently, CVT is accompanied by sweating, nausea, and vomiting. \(^3\) The parturient patient with these symptoms should be radiographically examined for CVT before epidural blood patching is initiated.

Strict aseptic techniques have made septic bacterial meningitis very rare in current anesthesia practice. Antibiotics remain the treatment for bacterial meningitis. Aseptic chemical meningitis may occur with the introduction of skin surface cleansing solutions. Symptomatic treatment is used for aseptic meningitis, which is self-limiting.

A hematoma — intradural, extradural, or subdural in origin — can occur after a significant loss of CSF, although it is very rare. A previous history of head trauma and overlooked anticoagulant therapy are the primary risk factors. A Swedish study shows a hematoma incidence of 1 in 500,000 to 1 in 1,000,000 for both spinals and epidurals. \(^5\)

Treatment

The treatment of PDPH depends upon its severity. Conservative therapy includes fluids, bed rest, analgesics, caffeine, and acupuncture. When talking with a patient, it is recommended that a definite number of days needed for the disappearance of mild to moderate symptoms not be provided. This will avoid implanting an expectation of a definite duration of symptoms in the patient's subconscious mind. Instead, the patient should be given an estimate of the usual range of time, while stressing the shorter time period.

As stated earlier, an increase in oral fluid intake will not increase CSF production but may be effective because of the self-limiting nature of PDPH or because of epidural vein distension. Underhydration is to be avoided in such patients. Bed rest as a treatment for the discomfort of PDPH is logical to temporarily relieve the symptoms. Early ambulation is recommended to alleviate postoperative surgical complications and to speed the body's return to normalcy. \(^1\) \(^3\) \(^5\) Analgesics, possibly in combin-
tion with sedatives, can alleviate the symptoms of mild to moderate PDPH while not influencing the physiological course of the dural rent.

Although very few anesthesia personnel are proficient and knowledgeable in acupuncture, there are reports of its successful use. It can be used to treat PDPH when the patient is not prepared to accept epidural blood-patching. The goal is to normalize vascular function within the head region. Analgesia is achieved by raising the pain threshold. Acupuncture uses both local and distal points. Most patients report improvement after the first treatment in a three- to four-sessions course.

Caffeine sodium benzoate treatment has a 70% to 80% effectiveness rate. The treatment consists of 500 mg of caffeine sodium benzoate in 1 liter of Ringer’s solution given over 1 hour and a second liter of 5% dextrose in Ringer’s solution given intravenously over the next 2 hours. Caffeine sodium benzoate seems to act by relieving painful IC vascular distension, causing contraction of the vessel size. It is less invasive than the remaining treatment modalities.

Epidural saline injection as a treatment for PDPH has a history of variable results, the reason for which is not readily apparent. An increase in the epidural pressure is probably why the PDPH is relieved for a period of 30 minutes to a few hours in a significant number of cases. When this treatment is effective, the probable cause is red cell destruction, which is the result of the introduction of a needle to the highly vascular epidural space, with possible rupture of a blood vessel and leakage of red blood cells. This could produce enough red cell destruction to initiate the clotting mechanism. The continuous infusion method, consisting of a 15-25 mL/hr infusion for 24 hours, seems to maintain epidural pressures at a sufficient level to stop CSF from escaping into the epidural space and allow for dural closure.

Two other forms of therapy for treatment for PDPH also entail epidural injections. The injection of 1.0 to 1.5 mL of dextran 40 per 10 cm of patient height has recently been used. The solution was injected over a period of approximately 2 minutes. A volume of 20 to 30 mL of the dextran solution relieved the PDPH in 100% of the cases, with the onset of relief varying from 5 minutes to more than 120 minutes. However, the injection of all exogenous substances carry the risk of allergic reactions.

The injection of autologous blood into the epidural space, first described by Cormley in 1960, is extremely effective in the treatment of PDPH. This relatively simple procedure consists of placing the patient in either a sitting or lateral recumbent position, while monitoring both an electrocardiogram and blood pressure. The lateral recumbent position would be less painful for the patient. An intravenous (IV) infusion line should be in place to ensure venous access. Two people are required to do the sterile procedure, one to perform the epidural puncture, and the other to perform the phlebotomy in the arm opposite the IV.

The original reports recommended injecting small amounts (2-3 mL) of blood, but these injections were associated with lower cure rates. The current recommendation is the injection of approximately 15 mL of blood into the epidural space. This approach alleviated symptoms in 90% to 98% of patients. A slow, measured injection of the blood into the epidural space is accomplished at a rate of 1.0 mL/3 sec. Injection should be slowed or stopped if the patient complains of pain in the back, buttocks, or legs. The patient should then remain supine for at least 30 minutes before being allowed to sit up and cautioned to rest and not strain for the next 24 hours.

The autologous blood injected into the epidural space spreads at an average of 1.6 mL per spinal segment. Blood tends to travel more cephalad than caudal. In most cases caudal spread stops at S-1 or upper S-2. The mean spread is six spinal segments upward and three segments downward. Use of the lowest interspace is recommended in cases of multiple punctures because of this preferential cephalad spread. The mechanism of action of an epidural blood patch seems to be the occlusion of the dural rent through the formation of a gelatinous tamponade. Subsequent surgeries in the area of the blood patch have not revealed the formation of adhesions. As with all invasive procedures, autologous blood injection carries possible hazards such as infections, pain, and death.

The most invasive treatment of PDPH is surgical closure of an open dural rent. Nelson reported passing a length of plain catgut suture through the needle and leaving it in the dural rent. While this procedure lowered the incidence of PDPH to 4.9%, it was associated with cauda equina syndrome in 50% of the patients.

**Summary**

The incidence of PDPH seems to depend upon a number of factors of varying importance. A decrease in the size of the needle lessens the incidence of PDPH. The anesthetist has little control over the next four factors: age, the thickness of the dura, the sex of the patient, and the power of suggestion. PDPH incidence decreases with increasing age.

The dura varies nonuniformly from 0.5 mm to almost 2.0 mm in the thickness. Thicker dura tends
to retract more rapidly than thin dura. Sex is also a noncontrollable factor, with females having a higher incidence.

The power of suggestion was demonstrated by Kaplan, whose study showed no significant difference in the incidence of headache between true lumbar puncture patients and patients who underwent sham punctures. The angle of insertion through the dura seems to be of greater importance than the type of needle point or its orientation to the dural tissue axis.

Finally, recumbency and hydration have no bearing on the incidence of PDPH, although both are used as a treatment for other aspects of spinal anesthesia, i.e., relief of symptoms and prevention of intraoperative hypotension.

The treatments for PDPH consist of:

1. Caffeine sodium benzoate infused intravenously.
2. The continuous infusion of normal saline for 24 hours.
3. The injection of dextran 40.
4. The injection of 12 to 15 mL of autologous blood into the epidural space—the current treatment of choice.

REFERENCES


AUTHOR
John Hess, CRNA, BA, received a diploma from Carraway Memorial Methodist Hospital School of Nursing, Birmingham, Alabama, in 1968. He was a graduate of Charlotte Memorial Hospital School of Nursing in 1985. After 12 years of private practice, he moved to Albuquerque and his present position with the Veteran Administration portion of the New Mexico Regional Federal Medical Center in 1985.