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The Injection of Intrathecal Normal Saline Reduces the Severity of Postdural Puncture Headache

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Background and Objectives: We investigated whether the injection of 10 mL of normal saline into the subarachnoid space following accidental dural puncture reduced the incidence of postdural puncture headache (PDPH) and the need for epidural blood patch (EBP).

Methods: Twenty-eight patients who experienced accidental dural puncture with an epidural needle had 10 mL of normal saline injected into the subarachnoid space. In 22 patients, the injection was performed immediately through the epidural needle. In 6 patients who had intrathecal catheters placed through the epidural needle, the saline was injected through the catheter before removal. All other patients who experienced wet taps during the same period that the study was in progress but did not receive the saline injection served as a control group, 26 in number. Patients with severe or persistent PDPHs were treated with EBP.

Results: Of those patients who received intrathecal normal saline immediately through the epidural needle, 32% developed a headache compared with 62% of controls. Of these, 1 patient who received saline required EBP compared with nine in the control group ($P = .004$). Of those patients who had intrathecal catheters placed, there were no headaches in the saline group of 6 compared with 3 in the control group of 5, 1 of whom was treated with EBP ($P > .05$).

Conclusions: The immediate injection of 10 mL intrathecal normal saline after a wet tap significantly reduced the incidence of PDPH and the need for EBP. When an intrathecal catheter had been placed following a wet tap, injection of 10 mL of normal saline before its removal effectively prevented PDPH. *Reg Anesth Pain Med 2001;26: 301-305.*

Key Words: Postdural puncture headache, Intrathecal normal saline injection, Wet tap, Epidural blood patch.

There is considerable variability in the incidence of headache following accidental dural puncture. With a 17-gauge needle, this incidence can be as high as 76% to 85%.[1] It is generally accepted that the headache is due to the leak of cerebrospinal fluid (CSF), resulting in diminished hydraulic support for intracranial structures. Tension on these structures results in headache and occasionally oculomotor or trigeminal paresis.[2] Hearing loss has also been observed.[3] In addition, it has been postulated that compensatory vasodilatation of cerebral vessels[4] or cerebral vasoconstriction[5] is the mechanism for postdural puncture headache (PDPH). More serious consequences such as seizures[5] and even rupture of an intracranial vessel[6] may occur. We investigated a simple, low-risk technique for the prevention of this complication.

Currently, attention is primarily focused on the treatment of PDPH and not on prevention. There is a sound rationale for this given that PDPH is not an inevitable consequence of dural puncture and most interventions incur some risk, or even significant cost. There have been, however, some methods

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proposed for headache prophylaxis in the population at highest risk, i.e., the obstetric population in which accidental dural puncture has occurred with a 16- to 18-gauge needle. A prophylactic epidural blood patch (EBP) with the injection of 15 to 20 mL of autologous blood within 14 hours of the puncture has been recommended.[7,8] Results are encouraging, but there is some concern about risk of infection associated with injection of blood through a catheter that has been in place for some time.

A retrospective study by Cohen et al.[9] found a decreased incidence of headache using continuous postoperative intrathecal analgesia in patients who had experienced accidental dural puncture following attempts at epidural anesthesia for cesarean delivery. Perhaps it was the maintenance of intrathecal volume rather than the narcotic that influenced their result. This technique is not easily implemented for most patients with PDPH and could be quite costly.

Historically, when PDPH was originally discovered to be the result of a CSF leak, there were various attempts made to replace the lost fluid. Ahearn[10] and Pickering[4] injected saline solution into the subarachnoid space in patients suffering from PDPH with some positive results. The first successful EBP, performed by Gormley in 1960,[11] was preceded by the injection of 15 mL of normal saline into the intrathecal space. Only 2 mL of autologous blood was used, raising the possibility that the intrathecal saline rather than the epidural blood was responsible for the relief of symptoms. The technique proposed in this study is the immediate injection of 10 mL of preservative-free normal saline intrathecally immediately before the removal of the needle or the catheter. The cost of this technique is negligible, the risk of infection is minimal, and cost savings from reduced hospital stay and reduced treatment costs could be substantial.

Methods

The study was approved by the Human Research Review Committee at the University of New Mexico School of Medicine. Informed consent was obtained from 28 patients who experienced dural puncture with an epidural needle. In 22 patients, 10 mL of normal saline was injected into the subarachnoid space and the needle was removed. In 6 patients, an intrathecal catheter was placed through the epidural needle and was used for spinal analgesia. Before removal of the catheter, 10 mL of normal saline was injected into the subarachnoid space. Twenty-six patients who suffered a postdural puncture during the same period and did not receive normal saline intrathecally (September 1997 through May 1999) served as controls. This was a random, though not controlled, occurrence. Of the multiple anesthesia providers, some were not aware of the study and did not enroll patients. In 21 of these patients, the needle was removed and the epidural was performed at another level. In 5 of these patients, intrathecal catheters were placed after dural puncture, but no saline was administered before catheter removal. Patients in whom an increase in the CSF volume would be likely to cause a problem, e.g., patients with decreased intracranial elastance or increased intracranial pressure (ICP),[12] were excluded from the study. There were no significant differences in age or weight between the 2 groups. Patients were seen the day after the procedure and assessed for presence or absence of a postural headache. Patients were called at home 2 weeks later and asked about the presence or absence of a postural headache during the previous 2 weeks. All patients complaining of a PDPH were offered conservative treatment including intravenous and oral fluids, caffeinated beverages, rest, analgesics, and sumatriptan. All patients were offered EBP if the headache was severe or if conservative therapy failed and the headache persisted.

Patients who consented to treatment with intrathecal saline were treated as follows: When dural puncture occurred, CSF flow was immediately stopped by occluding the end of the needle with a sterile gloved thumb, and the stylet was replaced in the needle. Ten milliliters of preservative-free normal saline was drawn up through a filter straw and injected slowly through the epidural needle. The needle was then withdrawn, and the epidural needle and catheter were placed at a different level. In those patients in whom a catheter was placed intrathecally, 10 mL of preservative-free normal saline was injected through the catheter just before its removal. The patients were not given specific instructions as to posture but were advised to stay hydrated.

The treatment and control groups were compared for incidence of headache and for the number of patients requiring EBP using Fisher's Exact test.

Results

There was no difference between patients who received intrathecal saline versus those who did not with respect to age or weight. Patients in the study group were all obstetrical patients, with the exception of 1 female pain clinic patient and 1 female patient who had orthopedic surgery. The controls were all obstetrical patients except for 1 male orthopedic patient.

Table 1 shows 21 patients who had a dural punc-

Table 1. Comparison Between Patients Treated Immediately Through the Needle With Intrathecal Saline and Controls

	Saline Group	No Saline Group
No. of patients	22	21
No. with no headache	15	8
No. with headache	7	13
No. requiring EBP	1	9

NOTE. None of these patients had an intrathecal catheter placed.

ture but no injection of saline. Thirteen of these patients (62%) experienced PDPH and 9 (43%) required EBP. Twenty-two patients received intrathecal saline through the needle. Seven of these patients (32%) experienced PDPH (Fisher's Exact P value = .07) and 1 (5%) required EBP (P = .004).

Table 2 shows 5 patients who had intrathecal catheters placed but did not have saline injections. Three of these patients experienced PDPH and 1 required EBP. Six patients had intrathecal catheters placed and were administered intrathecal saline before catheter withdrawal. None of these patients experienced PDPH. These differences were not significant because the subject numbers were small.

Table 3 shows the results for the entire group, combining the patients who had intrathecal catheters with those who did not. The headache incidence was significantly lower for the saline group (P = .01), as was the need for EBP (P = .002).

Discussion

PDPHs are still a problem after 100 years of spinal anesthesia, and there are many unanswered questions regarding them.[13] In our study, the intrathecal injection of normal saline was associated with a significantly reduced incidence of PDPH and a reduced need for EBP. One explanation for the beneficial effect of intrathecal saline is that the increased CSF pressure may result in approximation of the dura and arachnoid at the puncture site, thus sealing the defect. This may be an oversimplification, however, and there may be other mechanisms.

Table 2. Patients Who Had Intrathecal Catheters Placed Through the Epidural Needle After the Postdural Puncture

	Saline Group	No Saline Group
No. of patients	6	5
No. with no headache	6	2
No. with headache	0	3
No. requiring EBP	0	1

NOTE. The numbers here are too small to show significance and are included merely for clarity.

Table 3. Comparison Between All Patients Treated With Intrathecal Saline and Controls

	Saline Group	No Saline Group
No. of patients	28	26
No. with no headache	21	10
No. with headache	7	16
No. requiring EBP	1	10

The most widely accepted theory concerning the cause of PDPH is based on the concept of loss of CSF through a dural tear, and indeed this is the initiating event. However, questions still remain. Why does the headache often take 24 to 72 hours to become evident? Why are some patients susceptible to this complication and others resistant? Why do certain remedies, including caffeine,[14-17] sumatriptan,[18,19] theophylline,[20] and adrenocorticotropin hormone[21,22] appear to help the condition when they do nothing to reduce the loss of CSF? Raskin[23] speculates that the sudden decrease in CSF volume that occurs after lumbar puncture activates adenosine receptors, thus producing venous and arterial vasodilation and the resulting syndrome of PDPH. Others have found a decreased incidence of PDPH when the patient is placed in the head-down position immediately after the puncture.[24] Perhaps this maneuver also prevents activation of adenosine receptors by maintaining the CSF volume in the cranium. By the injection of 10 mL of normal saline into the intrathecal space immediately after the dural puncture and by making every effort to limit the loss of CSF volume, we may be able to prevent the activation of adenosine receptors and thereby prevent or reduce the severity of the symptoms.

There are many variables in the pathophysiology of PDPH. CSF pressure and its relationship to headache are not uniform.[25] Marshall[26] has shown that CSF hypotension is not found consistently in patients with PDPH. Carpenter et al.[27] published an analysis of the volume of CSF fluid and related this to the variation in extent of spinal anesthesia. The relationship of CSF volume and the incidence of PDPH have not been explored. The original volume of CSF in patients suffering from subsequent PDPH is unknown. CSF density is also a variable, which could have a bearing on headache incidence. Richardson and Wissler[28] have shown that CSF density in pregnant and postpartum women, who are particularly susceptible to PDPH, is significantly lower. Naulty et al.[29] found that the use of hyperbaric bupivacaine and lidocaine was associated with a higher incidence of PDPH compared with an isobaric technique. Other studies of hyperbaric versus isobaric local anesthetic mixtures show a higher incidence of spinal headache when higher glucose

concentrations were used.[30,31] When fentanyl is mixed with hyperbaric bupivacaine, the density of the injectate is changed. Johnson et al.[32] found a 50% reduction in incidence of PDPH when fentanyl was added to the hyperbaric bupivacaine. We have not studied the effect of the injection of normal saline on the density of our patients' CSF.

Posture after the dural tap has also been a subject of controversy. Historically, patients were advised to lie supine, the theory being that this would minimize the CSF pressure in the area of the puncture and hopefully decrease CSF loss. Patients also were more comfortable in this position. Recumbence, however, has not been found to alter the incidence of the headache.[33,34] Thornberry and Thomas[35] reported a lower incidence of severe spinal headaches in patients who were randomized to early mobilization versus bed rest. Some believe that the erect position hastens recovery by allowing the increased lumbar CSF pressure to approximate the dura to the rigid structures of the spinal canal and thereby to seal the leak.[16] Our study patients were not advised to remain supine. We did not discourage ambulation. The majority of our patients went on to deliver infants vaginally in the semi-sitting position with the consequent increases in CSF and epidural pressure brought on by pushing through the second stage of labor, in itself considered to be an additional risk factor for PDPH.

Our numbers are small and our study suffers from a lack of randomization. Initially begun as a pilot study with a limited number of patients and no controls, this study expanded as larger numbers became available. All patients who experienced wet taps were being monitored for the development of PDPH. This assessment, though not blinded, was not necessarily done by the provider who had performed the epidural. There were a large number of providers: CRNAs, residents, and attending anesthesiologists, including per diem staff. The specific providers were not tracked, although residents, CRNAs, and attendings provided care for both the controls and the study group. The results may conceivably have been affected by the technique or management of the specific provider. After the injection of the saline, however, study patients were not assigned to different treatments. Other parameters concerning our patients, such as number of dural punctures, history of migraine, blood pressure, family history, etc., were not addressed. In addition, there is a widely variable incidence of PDPH in pregnant patients, which could have a bearing on our results. Finally, there is the great variation in patients' tolerance to pain and other independent factors, which affect the decision to opt for an EBP. Our study had multiple providers whose approach to the patient and whose presentation of the alternatives would inevitably differ. Larger numbers than this, in addition to randomization and blinded assessment, are required to adequately compensate for such variables.

In summary, injection of 10 mL of normal saline before the removal of an intrathecal needle or catheter and the avoidance of the supine position, appear to reduce the severity of PDPH following accidental dural puncture. Further studies are needed to confirm these findings and to determine the mechanism of the beneficial effect.

References

1. Brownridge P. The management of headache following accidental dural puncture in obstetric patients. *Anaesth Intens Care* 1983;11:4-15.
2. Lee JJ, Roberts RB. Paresis of the fifth cranial nerve following spinal anesthesia. *Anesthesiology* 1978;49: 217-218.
3. Lybecker H, Anderson T. Repetitive hearing loss following dural puncture treated with autologous epidural blood patch. *Acta Anesthesiol Scand* 1995;39:987-989.
4. Pickering GW. Experimental observations on headache. *Br Med J* 1939;1:907-912.
5. Shearer VE, Jhaveri HS, Cunningham FG. Puerperal seizures after post-dural puncture headache. *Obstet Gynecol* 1995;85:255-260.
6. Pavlin DJ, McDonald JS, Child B, Rusch V. Acute subdural hematoma—An unusual sequela to lumbar puncture. *Anesthesiology* 1979;51:338-340.
7. Quaynor H, Corbey M. Extradural blood patch—Why delay? *Br J Anaesth* 1985;57:538-540.
8. Colonna-Romano P, Shapiro BE. Unintentional dural puncture and prophylactic epidural blood patch in obstetrics. *Anesth Analg* 1989;69:522-523.
9. Cohen S, Amar D, Pantuck EJ, Singer N, Divon M. Decreased incidence of headache after accidental dural puncture in caesarean delivery patients receiving continuous postoperative intrathecal analgesia. *Acta Anaesthesiol Scand* 1994;38:716-718.
10. Ahearn RE. Management of severe postlumbar puncture headache. *NY State J Med* 1948;48:1495-1498.
11. Gormley JB. Treatment of postspinal headache. *Anesthesiology* 1960;21:565-566.
12. Sperry RJ, Gartrell A, Johnson JO. Epidural blood patch can cause acute neurologic deterioration. *Anesthesiology* 1995;82:303-305.
13. Liu SS. Why are postdural puncture headaches still a problem? *Reg Anesth Pain Med* 2000;25:347-349.
14. Sechzer, PH, Abel, L. Post-spinal anesthesia headache treated with caffeine. Evaluation with demand method. Part I. *Curr Ther Res* 1978;24:307-312.
15. Sechzer PH. Post-spinal anesthesia headache treated with caffeine. Part II: Intracranial vascular distention, a key factor. *Curr Ther Res* 1979;26:440-448.
16. Baumgarten RK. Should caffeine become the first

line treatment for postdural puncture headache? *Anesth Analg* 1986;65:913-914.
17. Jarvis AP, Greenawalt JW, Fagraeus L. Intravenous caffeine for postdural puncture headache. *Anesth Analg* 1986;65:316-317.
18. Carp H, Singh PJ, Vadhera R, Jayaram A. Effects of the serotonin-receptor agonist sumatriptan on postdural puncture headache: Report of six cases. *Anesth Analg* 1994;79:180-182.
19. Hodgson C, Roitberg-Henry A. The use of sumatriptan in the treatment of postdural puncture headache. *Anaesthesia* 1997;52:808.
20. Feuerstein TJ, Zeides A. Theophylline relieves headache following lumbar puncture. *Klin Wochenschr* 1986;64:216-218.
21. Kshatri MA, Foster PA. Adrenocorticotropic hormone infusion as a novel treatment for postdural puncture headache. *Reg Anesth* 1997;22:432-434.
22. Gupta S, Agrawal A. Postdural puncture headache and ACTH. *J Clin Anesth* 1997;9:258.
23. Raskin NH. Lumbar puncture headache: A review. *Headache* 1990;30:197-200.
24. Broker RJ. Technique to avoid spinal-tap headache. *JAMA* 1958;168:261-263.
25. Fay T. A new test for the diagnosis of certain headaches: The cephalalgiogram. *Dis Nerv System* 1940;1: 312-315.
26. Marshall J. Lumbar-puncture headache. *Neurol Neurosurg Psychiatr* 1950;13:71-74.
27. Carpenter RL, Hogan QH, Liu SS, Crane B, Moore J. Lumbosacral cerebrospinal fluid volume is the primary determinant of sensory block extent and duration during spinal anesthesia. *Anesthesiology* 1998;89:24-29.
28. Richardson MG, Wissler R. Density of human cerebrospinal fluid. *Reg Anesth* 1996;21(suppl 2):29.
29. Naulty SJ, Hertwig L, Datta S, Weiss JB, Ostheimer GW. Influence of local anesthetic solution on postdural puncture headache. *Anesthesiology* 1985;63: 3A,454.
30. Axelsson K, Widman B. Clinical significance of specific gravity of spinal anesthetic agents. *Acta Anaesthesiol Scand* 1979;23:427-434.
31. Chambers WA, Edstrom HH, Scott DB. Effect of baricity on spinal anesthesia with bupivacaine. *Br J Anaesth* 1981;53:279-282.
32. Johnson MD, Hertwig L, Vehring PH, Datta S. Intrathecal fentanyl may reduce the incidence of spinal headache. *Anesthesiology* 1989;71:3A,911.
33. Carbaart PAT, van Crevel H. Lumbar puncture headache: Controlled study on the preventive effect of 24 hours' bed rest. *Lancet* 1981;2:1133-1135.
34. Hilton-Jones D, Harrad RA, Gill MW, Warlow CP. Failure of postural manoeuvres to prevent lumbar puncture headache. *J Neurol Neurosurg Psychiatry* 1982;45:743-746.
35. Thornberry EA, Thomas TA. Posture and post-spinal headache. A controlled trial in 80 obstetric patients. *Br J Anaesth* 1988;60:195-197.