▽ Editorial

Postdural Puncture Headache: Mechanisms, Treatment, and Prevention

Postdural puncture headache (PDPH) fascinates both clinical and academic anesthesiologists. Numerous studies on its mechanism(s), prevention, and treatment are published. A study appearing in this issue of *Regional Anesthesia and Pain Medicine* suggests a new technique for the prevention of headache after dural puncture,[1] and we will review the proposed mechanisms, treatment, and prevention of PDPH.

One of the theories proposed for headache after dural puncture is decreased subarachnoid pressure from the leakage of cerebrospinal fluid (CSF) through the dural rent. Proponents suggest the decrease in subarachnoid pressure causes the brain to sag when the patient assumes the upright position, resulting in traction on the pain-sensitive structures (blood vessels) of the brain. A correlation between decreased subarachnoid pressure and headache has been documented.[2] However, not all patients who develop headache after dural puncture have decreased subarachnoid pressure. Marshall[3] performed a second lumbar puncture whether the patients developed a headache or not and found no correlation between the occurrence of headache and the subarachnoid pressure. The 6 patients who had postural headache had opening pressures of 0, 0, 35, 60, 105, and 150 mm H_2O. Of the patients who did not have headache, 1 had an opening pressure of 0 mm H_2O, 1 had 5 mm H_2O, while 7 had pressures of 50 mm H_2O or less.

The injection of epidural saline or blood causes an immediate increase in epidural and subarachnoid pressures and typically an immediate relief of headache.[4,5] The ability of epidural saline to relieve the headache has been attributed not only to the increase in epidural pressure, but also to the increase in subarachnoid pressure, normalizing the pressure gradient between the CSF, the intracranial vessels, and other intracranial structures. However, the pressure increase is short-lived (minutes after the epidural saline injection)[4,5]; therefore, a sustained return of CSF pressure to its baseline value is not likely the dominant mechanism of headache relief.

The degree of CSF leak, or CSF volume, per se, does not correlate with the degree of headache after dural puncture. Iqbal et al.[6] measured the amount of CSF leak after a dural puncture. Of the 4 patients who developed headache, the amount of CSF leak was small (<10 mL) in 2 patients, moderate (10 to 110 mL) in 1 patient, and large (>110 mL) in another patient. Finally, 1 patient had continued evidence of CSF in the paraspinous area for 7 days but never developed a headache.[6]

Relief of headache after an epidural blood patch often occurs within 1 hour after the blood injection, a time that is not adequate to fully replace the CSF deficit. Raskin[7] proposes that *sudden* alteration in CSF volume is the primary mechanism of headache after dural puncture. He theorizes the loss of CSF volume and the change in pressure differential across intracranial venous structures result in venous dilatation. He bases this on the inconsistent relationship between

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subarachnoid pressure or the intracranial pressure and the occurrence of headache,[3] and the worsening of the headache when jugular venous compression is applied, even though the technique increases the intracranial pressure (jugular venous compression results in venous dilatation).[7]

The graded withdrawal of CSF in baboons results in reduction of CSF pressure and increased cerebral blood flow.[8] This compensatory intracranial vasodilation has been proposed by Sechzer as the mechanism for the PDPH. Sechzer believes that the low CSF pressure is a correlate but not the basic cause of the headache.[9] Cerebral vasoconstrictors, such as caffeine and sumatriptan, have been found to be effective in relieving dural puncture headache in some studies.

Block of the brain adenosine receptors by caffeine and theophylline results in vasoconstriction.[7] Acute increases in epidural and subarachnoid pressures seen after epidural saline[4] or epidural blood patch[5] may deactivate the adenosine receptors relieving the headache. The compression of the dural sac by the blood or saline may have an additional effect. The dura is rich in adrenergic, cholinergic, and peptidergic fibers,[7] and the effects of xanthines on these systems are well known.

It is also possible that PDPH has several mechanisms and that a combination of these mechanisms play a role in each patient. It is also possible that our treatments correct one or more of these mechanisms.

Current treatment for PDPH includes oral or intravenous caffeine, theophylline, sumatriptan, adrenocorticotrophic hormone, epidural saline, and epidural blood patch. Several techniques have been tried in an attempt to reduce the incidence of PDPH. The maintenance of the supine position after dural puncture is not effective, whereas prophylactic epidural blood patch may be effective.[10] The incidence of PDPH after continuous spinal anesthesia is low; however, it is not clear whether placement of a subarachnoid catheter after dural puncture with a large-bore needle decreases the incidence of headache.[11,12]

The study by Charsley and Abram[1] is an intriguing technique to minimize the development of PDPH. The beneficial effect of their technique may be due to an increase in subarachnoid pressure or correction of the altered CSF pressure induced by the CSF leak, normalization of the pressure gradients between the intracranial contents, and deactivation of the adenosine receptors. The intrathecal saline may also favor approximation of the cut dural fibers, a mechanism specifically proposed by Charsley and Abram. Most studies on PDPH lack scientific rigor in that they were not prospective, randomized, or blinded, and follow-up and outcome criteria were not standardized. We agree that the risks of subarachnoid injection of sterile saline are minimal; the definitive role of this novel technique should be examined in prospective, randomized, and blinded studies.

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