tion of the degree of respiratory obstruction. Fiberoptic tracheobronchoscopy is essential for direct preanesthetic evaluation of dynamic airway anatomy and for accurate endotracheal tube placement at induction of anesthesia. This technique also facilitates assessment of the integrity of the entire tracheobronchial tree at the close of surgery. Appropriate measures may then be taken to assure airway patency in the postoperative period. A careful, deliberate approach to the induction and maintenance of anesthesia in such cases may avoid the need for emergent rigid bronchoscopy or cardiopulmonary bypass to alleviate precipitous respiratory obstruction.

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REFERENCES

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Postpartum Seizure after Epidural Blood Patch and Intravenous Caffeine Sodium Benzoate

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Postlumbar-puncture (PLP) headache after accidental dural puncture is a well-known complication of epidural analgesia for labor and delivery. The use of conservative therapy and epidural blood patch for relief of PLP headache have been described previously.1–3 We present a case of postpartum seizures temporally related to an epidural blood patch given to a patient for a PLP headache. This patient had failed conservative therapy, including epidural saline and iv caffeine sodium benzoate (CSB).

CASE REPORT
A 24-yr-old woman (GpPsAb) was admitted while in active labor at 41 weeks gestation. Her prenatal course was uncomplicated. Her medical/social history was positive only for a brief episode of iv drug abuse 3 years prior. All prenatal laboratory work was normal. Baseline arterial blood pressure (BP) was 120/70 mmHg. Her admission examination showed a cervix 90% effaced and 3–4 cm dilated, BP 138/96 mmHg, reflexes 2+ and equal, no edema, and urinalysis negative for protein. She was given a 3.0 g magnesium sulfate iv bolus and started on a 2.0 g/hr iv infusion. Her labor was also augmented with oxytocin.
The anesthesiologist on call was consulted regarding management of labor pain. In the placement of a continuous lumbar epidural with an 18-G Hustle needle, a wet tap resulted at the L3-4 interspace. A catheter placed at L2-3 provided adequate labor analgesia and subsequent anesthesia for a cesarean section. The patient delivered a 4,388-g male infant with Apgars of 8 and 9 at 1 and 5 min, respectively. Magnesium sulfate was continued for 24 h postoperatively. The patient was without any complaints, ambulating, and had a stable BP at 120/70 mmHg. To prophylactically treat a potential headache, the epidural catheter was left in place for 24 h, and in a technique described by Smith, epidural saline was injected. Forty-eight hours postoperatively, the patient complained of neck and shoulder discomfort, headache in the temporal regions, mild chest tightness, and shortness of breath. The obstetric resident was consulted at this time and his examination showed the following: BP 160/90 mmHg, heart rate 80 beats/min, and respiratory rate 14 breaths/min. Physical examination was normal except for neck tenderness with palpation. Chest radiograph, electrocardiogram, and arterial blood gases were all normal. The patient was treated for musculoskeletal neck pain with ibuprofen from which she received her symptoms.

On the third postpartum day the patient again complained of continuing headache. The anesthesiologist who had performed the original lumbar epidural evaluated the patient and found her alert and oriented but elicited a description of frontal/temporal headache that resolved when supine. BP was 130/68 mmHg, heart rate 90 beats/min, and respiratory rate 24 breaths/min. The diagnosis of a PLP headache was made. Treatment started with a protocol of CSB, 500 mg added per liter of lactated Ringer's solution administered iv over 1 h. Subsequent evaluation revealed the patient's headache had resolved. CSB (500 mg) was then added to the next liter of lactated Ringer's solution and administered over the next 2 h. One hour after this liter of crystalloid the patient again complained of a postural headache that was worse than that prior to treatment. The decision was made to proceed with an epidural blood patch. She was transferred from the postpartum ward to the recovery room for the procedure. Vital signs upon arrival in the recovery room were as follows: BP 150/100 mmHg, heart rate 92 beats/min, and respiratory rate 24 breaths/min. All vital signs had increased over baseline presumably secondary to pain. Ten milliliters of autologous blood was injected epidurally at the L3-4 interspace through an 18-G Hustle needle without difficulty. Twenty minutes after the epidural blood patch was given the patient again complained of an increasing headache. While evaluating the patient, and as she attempted to assume a position of comfort by sitting up, she developed a tonic clonic seizure of approximately 30 s duration. She was administered oxygen (FiO\textsubscript{2} of 1.0 via Ambu bag and spontaneous ventilation), diazepam 5 mg, and magnesium sulfate 4.0 g iv. The patient was lucid after being in a postictal state for 10 min. Twenty minutes later a second seizure occurred for which she received an additional 2.0 g of magnesium, along with midazolam 5.0 mg iv, which resulted in resolution of the seizure after which she had a normal neurologic examination. Magnesium infusion was restarted at a rate of 5.0 g/h. The patient was then sedated for computerized axial tomography (CAT) where she had a third seizure while moving herself from the gurney to the scanner. Magnesium sulfate 5.0 g, midazolam 5.0 mg, and pethynotin 1.0 g were administered iv. The CAT scan was normal. Electrolytes were also normal. Arterial blood gases during the seizures showed a metabolic acidosis, which resolved quickly. An electroencephalogram (EEG) performed after resolution of seizure activity was normal.

Magnesium sulfate therapy was continued and maintained at a blood level in the therapeutic range (6-8 mEq/l) for 2 days. Phenytoin was kept at a therapeutic level by oral administration. The patient was discharged on the seventh postoperative day and continued on pethynotin for 1 month. At a 5-month follow-up the patient had remained seizure-free.

**Discussion**

A headache in the parturient is a common complaint occurring in 25% of patients not receiving any anesthetic intervention. Others have found that of the headaches that occur after a dural puncture, up to 26% appear to be coincidental to the anesthetic. Because dural puncture does not always cause headache, and headache is a common postpartum complaint, the onset of a headache should be evaluated thoroughly. Headaches have been classified by many different authors. Major classifications are vascular, muscle contraction, traction, and inflammatory headaches, with PLP headache probably being a traction or traction–vascular type headache. A proper diagnosis depends on a complete history and physical examination along with a review of the anesthetic record. The characteristics of a PLP headache have been described since the first spinal anesthetic, and include a number of unique features that help in the diagnostic evaluation: 1) headache is usually postural in nature with pain increasing in the upright position; 2) headache is most commonly occipital (25%), frontal (22%), or occipital and frontal (25%); 3) the majority of headaches start on the first or second postoperative day; and 4) 90% of patients have associated symptoms including neck ache, neck stiffness, backache, nausea, vomiting, blurred vision, or tinnitus. In the current case the original assessment of the patient's headache did fit the description of a PLP headache. However, when questioning the patient after the resolution of her seizures, she stated that her headache first presented with increasing pain while in a supine position and she had experienced relief from the headache while standing. These symptoms are more consistent with a hypertensive headache than with a PLP headache. In a patient with a PLP headache a number of conservative treatments have been described. Two such treatments are in common use in our hospital. One is the injection of saline through a functioning epidural catheter after a wet tap as a prophylactic attempt to reduce the incidence of PLP headache. Craft et al. described a reduction in the incidence of PLP headache from 76.5% to 12.5% by this technique. A second conservative regimen is the use of CSB for treatment of PLP headache as described by Sechzer and Abel. They described an 85% success rate with a 500 mg bolus of CSB for relief of PLP headache. However, we have been using a similar method described by Jarvis et al. in which 500 mg of CSB is given over a 1-h period in 1 l of fluid. We found this protocol to be successful for referrals with presumed PLP headache. Two-thirds of our patients require no further treatment, whereas one-third of our patients require an epidural blood patch for complete headache relief. The only reported complications of treatment regimens with CSB were transient dizziness or flushing.
no side effects or complications using our caffeine sodium benzoate regimen prior to our current case.

Many reports on the use of epidural blood patches have addressed the issue of technique, volume of blood, and complications,5,10 We have been using an 18-G Hustead needle at the site of the prior wet tap, finding the epidural space with a loss of resistance technique. Ten to 12 milliliters of autologous blood is used for the patch. Back pain, dizziness, cranial nerve paresis, paresthesia, and radicular pain have been the reported complications of epidural blood patches.15 We found no report describing seizures as a complication. An explanation for an epidural blood patch causing a seizure may be difficult to formulate. Even if the blood had been injected subarachnoid, this should not precipitate seizure activity. Although patients with a subarachnoid hemorrhage are at an increased risk of seizures, they do not seize from the presence of blood in the cerebrospinal fluid.9

In the parturient who presents with new onset seizures, an extensive differential diagnosis must be considered, including cerebrovascular accidents, arteriovenous malformation, hypertensive disease, space occupying central nervous system lesions, infectious processes, metabolic diseases, epilepsy, and, of course, eclampsia.19 In the obstetric literature some authors view late postpartum eclampsia with skepticism and do not consider the appearance of convulsions after 48 h as true eclampsia.20 The pathophysiology of eclampsia remains unknown, and there are no signs or symptoms in patients diagnosed as preeclamptic that are predictive of eclampsia.21 However, the appearance of late postpartum eclampsia has been described in a few review articles,22,23 and Sibai et al.22 concluded that any “seizure which occurs after delivery, even after 48 hours should be considered as eclampsia until proven to the contrary.”

Initially, because of an elevated BP on admission, our patient had been diagnosed as having mild pregnancy-induced hypertension (PIH) and, therefore, was treated with iv magnesium sulfate. Because the BP returned to normal and no other manifestations of PIH developed, therapy was discontinued at 24 h postpartum. After her seizures evaluation was negative for metabolic abnormalities, and her CAT scan and EEG were normal. Therefore, the initial diagnosis of PIH, in light of a negative seizure workup, could lead to the conclusion that her seizures were indeed that of a late postpartum eclampsia.

Is there a possible relationship between the use of iv CSB and the onset of our patient’s seizure? Methylxanthines are known to be central nervous system stimulants, with caffeine being the most potent.24 At high doses they can cause generalized focal convulsions, with theophylline being more profound than caffeine.24 Animal studies have supported the hypothesis that caffeine convulsion activity occurs at benzodiazepine receptors.25 However, when caffeine is given in a therapeutic dose there is a question as to whether the drug will unmask seizures.26 Davis et al.27 described two infants treated for apnea with iv caffeine who developed seizures after an iv infusion of 20 mg/kg of caffeine. They theorize that the caffeine may have unmasked the seizures by a reduction in seizure threshold. Twenty other infants receiving the same therapy did not develop any seizure activity. Caffeine has been considered contraindicated for epileptics, but others have found that coffee often relieves attacks.28 In two studies in the use of caffeine to lengthen seizures during electroconvulsive therapy, both found that iv caffeine up to 2.0 g did not lower seizure threshold but did lengthen the seizure achieved.29,30 However, these findings may have been influenced by the use of barbiturate for the anesthetic. Additionally, Mueller and Solow31 describe a 17-yr-old girl 2 weeks postpartum who developed tonic-clonic seizures after ingesting a high dose of a caffeine-containing stimulant. Finally, in a number of studies reported by Oser and Ford,32 the metabolic half-life of caffeine was found to be 4-6 h, but a doubling of this time occurs during pregnancy.33 It is interesting to speculate whether the prolonged half-life of caffeine in our patient resulted in a drug level high enough to cause a seizure, to unmask an underlying seizure disorder, or to facilitate a predominantly epileptic seizure. Or was the seizure one of eclampsia totally coincidental to our treatments? We cannot provide a definitive answer. However, it would be prudent to avoid the use of CSB for treatment of PLP headache in patients with a diagnosis of PIH or a seizure disorder.

REFERENCES

4. Smith BE: Prophylaxis of epidural “wet tap” headache. ANES-
THEOLOGY 51:304, 1979
5. Grove LH: Backache, headache and bladder dysfunction after del-
6. Brownridge P: Spinal anesthesia revisited: An evaluation of sub-
arachnoid block in obstetrics. Anaesthesia 34:34-342, 1984
446-450, 1980
9. Diamond S, Dalesio DJ: The Practicing Physician’s Approach to
Headache, 3rd edition. Baltimore, Williams & Wilkins, 1982
Edited by Goldstein PJ. New York, Futura, 1986, pp 247-263
Chir 51:361, 1899
Caudal Epidural Anesthesia in an Infant with Epidermolysis Bullosa

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Epidermolysis bullosa (EB) refers to several hereditary diseases characterized by separation of the epidermis and/or dermis following shear forces. Patients with EB present with bullae, blister, and erosion formation following seemingly minor trauma; these may heal completely or may lead to scarring, deformity, malnutrition, chronic infection, and death. Because of the skin contact involved, many aspects of general anesthesia, especially manipulation of the airway, expose patients with EB to severe risk.5–8

Reviews of the anesthetic management of patients with EB have cautioned against the use of regional anesthesia; concerns were raised that scarring and contractures would obscure landmarks; chronic infections would make block placement unsafe; and injection of local anesthetics might lead to bullae formation.2,5,6 Despite these concerns, there have been recent reports of the use of regional anesthesia in patients with EB; these include two reports of the use of brachial plexus block in children undergoing reconstructive hand surgery,6,7 and two reports of subarachnoid and epidural block in adults having gynecologic, obstetric, and abdominal procedures.8,9 There have been no reports of the use of regional techniques in infants with EB; we therefore present this report of an infant with the simplex form of EB who received a caudal epidural anesthetic for circumcision.

CASE REPORT

A 6-wk-old, 5-kg male infant presented for elective circumcision. He was the 5.8-kg product of an uncomplicated gestation and delivery;