eter. Another risk is the possibility of perforating a vessel during the positioning of the catheter, which fortunately did not happen in our series.

We choose the constant infusion of local anesthetic for children, because intermittent doses would have required injecting very low volumes (0.5–1 ml), thus risking underdose (ineffectiveness) or overdose (toxicity). The basic problem still is the assessment of the efficacy of the method. Pain is a difficult phenomenon to quantify. Its intensity varies for the same type of surgery, and the way in which it is accepted differs from individual to individual. In the adult it is difficult enough to assess the adequacy of analgesia by direct means; in the young or retarded child it is impossible. What seemed remarkable in our series was that the children, when unaware that they were being observed, were calm and showed no particular signs of discomfort. "Blind" pricking with the needle did not in itself provoke crying, although, of course, a child would cry because he or she was immobilized or saw someone approaching. In particular, the child accepted respiratory physiotherapy. Clapping on the back and chest was tolerated, and coughing, when provoked, produced expectoration. Breathing rhythm and capacity remained unchanged from what they were before surgery or improved. In our series, the catheter was withdrawn on the second or third day, that is, when bowel movements resumed. At that point, pain intensity for the adult is reduced considerably,[5] from which a similar experience may be extrapolated for the child.

The method of epidural analgesia thus would appear to be reliable in the child with respiratory disability or insufficiency. It represents an interesting alternative to postoperative controlled ventilation, which is often necessary to similar cases. If the risks inherent to this technique seem acceptable, the role of epidural analgesia in the child with no respiratory disorders and undergoing major surgery still remains to be defined.

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Hemiparesis Following Dural Puncture

DENISE J. WEDEL, M.D.,* and MICHAEL F. MULROY, M.D.†

A known complication of epidural anesthesia is dural puncture, spinal anesthesia, and postlumbar puncture headache. Clinical reports of subdural hematoma, acute and chronic, as well as intracerebral hemorrhage following postlumbar puncture headache have been described.[1–3] Mantia[4] reported a case in which an intracerebral hemorrhage occurred 5 days after development of a postlumbar puncture headache. The following case describes the occurrence of an intracerebral hematoma coincident with dural puncture with a 19-gauge needle during attempted epidural anesthesia.

Report of a Case

A 37-year-old female physician presented for a left femoral hernia repair. She had no history of medical problems, previous surgery, childbirth, headache, or medications. Routine preoperative studies included chest roentgenogram, complete blood count, and urinalysis, all of which were normal. On admission to the hospital, arterial blood pressure was 100/72 mmHg, heart rate 72 beats/min, and temperature 37.2° C. She was interviewed by an anesthesia resident and after discussing risks and complications, an epidural anesthetic was chosen. The following morning diazepam 10 mg po and morphine sulfate 10 mg im was given prior to arriving in the anesthesia induction room. After being positioned with the left side down, local infiltration was done at the L_{2-3} interspace with 2 ml bupivacaine 0.75% using a 30-gauge needle for the skin wheal and 22-gauge 1½-inch needle for deeper infiltration. Three attempts were then made with a 19-gauge Quincke tip spinal needle to locate the ligamentum flavum using a midline approach. During these attempts, a 3-ml syringe of bupivacaine 0.75% with 1:200,000 epinephrine was attached to the needle and

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* Instructor in Anesthesiology, Mayo Clinic and Mayo Medical School, Rochester, Minnesota.

† Staff Anesthesiologist. The Mason Clinic.

Address reprint requests to Dr. Wedel: Department of Anesthesiology, Mayo Clinic, 200 First Street SW, Rochester, Minnesota 55905.

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small intermittent injections totaling 3 ml were given while exploring for ligament. Although no cerebrospinal fluid was seen or aspirated, the patient noted a feeling of warmth in her feet accompanied by numbness, especially on the left side. The technique was abandoned as the patient began to complain of nausea and progressive numbness; the needle was removed and she was turned to the supine position. Systolic arterial blood pressure was palpated at 80 mmHg, and ephedrine 12.5 mg iv and 25 mg im were given. Systolic blood pressure then was palpated at 140 mmHg with a heart rate of 120 beats/min and the patient complained of a throbbing headache. She was alert and lucid throughout and when stable was moved to the operating room. In the operating room, arterial blood pressure was 130/70 mmHg and her sensory level of anesthesia was at T_1 when examined by pinprick. Her left arm was positioned by her side throughout the hernia repair. The headache resolved shortly after she was moved to the operating room, and vital signs continued to be stable with the arterial blood pressure ausculated at 100–110/60–70 mmHg. Diazepam 5 mg and thiamylal 200 mg were given iv in increments of 40 mg for sedation. A left femoral hernia repair was completed in 90 min. After transfer to the recovery unit, arterial blood pressure was auscultated at 100/56 mmHg, heart rate 76 beats/min, respiration $16 \cdot min^{-1}$ and sensory level T_5 bilaterally. The patient was sleepy but oriented and comfortable. The recovery room nurse recorded limb movement as a "+1," which represents "moves two limbs" on the recovery room record. No comment was written specifically concerning upper extremity movement. The possibility of postdural puncture headache was discussed, and she was discharged from the recovery room with a T_7 sensory level bilaterally.

Eight hours later, the patient commented to a nurse on the floor that she had been unable to move her left side since being in the recovery unit. Review of the floor nursing notes revealed the comment "left lower extremity and left arm without sensations" when the patient arrived on the floor. There was no comment about motor activity. Evaluation revealed left hemiparesis, increased tone of the left arm and leg, and a normal sensory examination. A neurologist later confirmed spastic left hemiparesis sparing the face, which was felt to be a residual effect from the injection of local anesthesia into the subarachnoid space. However, no improvement occurred, and a CT scan performed on the first postoperative day revealed a right frontoparietal intracerebral hematoma. The patient was observed for 24 h in the ICU without further progression of her neurologic deficit. She received physical and occupational therapy, showing daily improvement, and was discharged 18 days after the surgery, walking with a cane. Follow-up examinations show continued improvement with some residual left-sided weakness 3 weeks after the initial incident. A tentative diagnosis of ruptured arteriovenous malformation was made, based on the anatomic location of the hematoma.

Discussion

A variety of intracranial events have been related causally or temporally to lumbar puncture. Acute and chronic subdural hematomas,[1,2,5] intracerebral hemorrhage following diagnostic lumbar puncture,[3] cerebral infarction following spinal anesthesia,[6] and exaccerbation of preexisting neurologic disease[7,8] have been reported. Acute intracerebral hemorrhage is a known complication of lumbar puncture in the patient with a mass lesion,[3] however, no relationship has been established between a lumbar puncture and intracerebral hemorrhage in the neurologically asymptomatic patient. Review of a large series of subarachnoid hemorrhages (6,368) showed that symptoms from arteriovenous malformation rupture were associated with surgery in 0.51% and with parturition in 0.51% of the cases—in contrast to 36% occurring during sleep.[9] Mantia[4] recently reported a case of intracerebral hemorrhage 2–5 days after delivery in a previously healthy woman. He suggests that pregnancy-associated hypertension may have been a contributing factor.

Our patient complained of a throbbing headache immediately following the iv administration of ephedrine 12.5 mg. Systolic arterial blood pressure had risen from 80 to 140 mmHg after the ephedrine was given, both values within a common range of blood pressure in this age group. Although she was awake and apparently oriented throughout the episode, she did not indicate any motor weakness in her left upper extremity until several hours later. We speculate that the hemorrhage probably occurred after the dural puncture and administration of vasopressor when she complained of a severe headache. Rupture of an intracerebral aneurysm has been reported under anesthesia associated with wide fluctuations of blood pressure (140/90 to 240/160 mmHg),[10] but the low range of pressure and mild symptoms in our patient did not suggest this cause immediately. Exact onset and evolution of the neurologic symptoms are very difficult to ascertain. Limb movements are recorded on the recovery room record with a code of +2, +1, or 0, representing "moves 3, 2, or 0 limbs," respectively. This patient had a T_7 sensory level upon discharge to the ward and was not moving her lower extremities. The absence of any code for "moves 1 limb" attests to the rarity with which this event occurs. It is possible that the left arm was involved and this was not observed in this sleepy patient or that the hemiparesis progressed as the patient was transferred to the ward. Her recall of events is perhaps somewhat clouded by the sedation that she received as well as change in mentation related to the intracerebral event. The absence of cranial nerve findings further obscured the underlying abnormality initially, though computerized tomography findings confirmed the diagnosis of intracerebral hemorrhage, and a clinical diagnosis of ruptured occult AVM was made by the neurologist.

This case report describes a rare but devastating complication of dural puncture and spinal anesthesia and reiterates that intracerebral hemorrhage can occur in patients without previous suggestive history or symptoms. Intracerebral hemorrhage has been reported following dural puncture, however, the use of vasopressors with a sudden rise in blood pressure also may have been a contributing factor, even though systolic pressure did not exceed 140 mmHg. As healthy individuals can tolerate moderate hypotension for the short term, incremental doses of vasopressor can be administered to titrate the effect in a more gradual fashion. While sudden headache can be caused by administration of vasopressors, careful

observation and documentation of neurologic status in the intraoperative and postoperative period may reveal signs of an intracerebral event. This case demonstrates that even a medically trained patient can be unwilling or unable to supply appropriate information during this period.

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Intrathecal Morphine for Relief of Labor Pain in a Parturient with Severe Pulmonary Hypertension

THERESE K. ABBOUD, M.D.,* JULIO RAYA, M.D.,† RABIA NOUEIHED, M.D.,‡ JAMES DANIEL, M.D.§

In women who have primary pulmonary hypertension,[1] maternal mortality is more than 50%[2] usually during labor or puerperium.[3] The hemodynamic features of this condition consist of pulmonary artery pressure higher than 30/15 mmHg,[2] right ventricular hypertrophy, and eventually failure and a low fixed cardiac output. Most complications arise from a decrease in systemic vascular resistance and reduction in venous return. For this reason, spinal or epidural anesthesia often is avoided in these patients.[4] Hyperbaric morphine injected intrathecally provides excellent analgesia during labor without any significant autonomic or motor effects.[5] We describe the labor and delivery in a patient with severe pulmonary hypertension who received intrathecal morphine analgesia.

* Associate Professor of Anesthesiology, Director of OB Anesthesia Research.
† Anesthesia Resident.
‡ Research Fellow in Obstetrical Anesthesia.
§ Assistant Professor of Clinical Anesthesia.

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Address reprint requests to Dr. Abboud: Department of Anesthesiology, LAC/USC Medical Center, P.O. Box 12, 1200 North State Street, Los Angeles, California 90033.

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Report of a Case

A 34-year-old woman, 155 cm, 67 kg, gravida 6, para 5, with primary pulmonary hypertension, was admitted at 38 weeks of gestation because of hemoptysis, dyspnea, and orthopnea. The patient had a chronic case of alcoholism and had a history of addiction to iv methylphenidate (Ritalin®) years ago, which was no longer an active problem, with the last use being 2 years before admission. She had no cardiopulmonary symptoms until 2 years before admission, which was also about 2 years after her last delivery. Symptoms began with decreased exercise tolerance, which progressed to orthopnea and later hemoptysis. Physical examination disclosed a grade IV/VI holosystolic murmur, best heard at the pulmonic area and apex. ECG showed right ventricular hypertrophy, right axis deviation, and left ventricular strain pattern. Chest roentgenogram revealed a prominent pulmonary artery. Right-sided cardiac catheterization revealed a pulmonary artery pressure (PAP) of 111/38 mmHg, with a mean of 64 mmHg, right ventricular pressure of 115/2 mmHg, and right atrial pressure (RAP) of 33 mmHg. While breathing room air, pH_a was 7.48, Pa_{O_2} 50 mmHg, Pa_{CO_2} 26 mmHg, and BE −3 mEq/l. Hematocrit was 39%, serum Na 139 mEq/l, and K 3.7 mEq/l.

Digoxin, diuretics, and potassium supplements were given by mouth and oxygen via nasal prongs. Spontaneous labor started 6 days after admission. PAP and RAP were measured using a flow-directed catheter. Cardiac output (CO) was measured by thermodilution. Systemic blood pressure was measured using an automated blood pressure monitor; blood gas and acid base variables and respiratory rate also were recorded. When the patient became uncomfortable (cervix 4 cm dilated) 1 mg of preservative-free morphine sulfate in 7.5% dextrose was injected at L_3-L_4 interspace using a 25-gauge spinal needle, with the patient in the lateral position. After injection, the patient was turned supine with her head elevated 30 degrees. The onset of analgesia occurred within 15 min, and within 30 min she was comfortable as