

olution and multiplanar imaging capabilities without patient exposure to ionizing radiation.¹¹ For these reasons, many investigators feel that MRI is useful for the investigation of disorders of the upper aerodigestive tract; specifically, the larynx and hypopharynx.

This case demonstrates how difficult it can be to predict in which patients endotracheal intubation will be difficult, even when they have been assessed using the numerous criteria reported. The epiglottis must not be overlooked as one of the factors which can cause difficulty with intubation. Since there is certainly not one cause for all difficult intubations, airway assessment must be as comprehensive as possible, while producing the least risk to the patient. In this regard, MRI offers a unique tool in that it is not invasive and produces no ionizing radiation exposure, yet it provides excellent detail of airway structures. For these reasons, MRI provides the means for prospectively and retrospectively assessing the difficult airway.

REFERENCES

1. Bannister FB, Macbeth RG: Direct laryngoscopy and tracheal intubation. *Lancet* 2:651-654, 1944
2. Cass NM, James NR, Lines V: Difficult direct laryngoscopy complicating intubation for anaesthesia. *Br Med J* 1:488-489, 1956
3. Mallampati SR, Gatt SP, Gugino LD, Desai SP, Waraksa V, Freiburger D, Liu PL: A clinical sign to predict difficult tracheal intubation: A prospective study. *Can Anaesth Soc J* 32:429-434, 1985
4. Salem MR, Mathrubhutham M, Bennet EJ: Difficult intubation. *N Engl J Med* 295:879-881, 1976
5. White A, Kander PL: Anatomical factors in difficult direct laryngoscopy. *Br J Anaesth* 47:468-474, 1975
6. Nichol HC, Zuck D: Difficult laryngoscopy—The "anterior" larynx and the atlanto-occipital gap. *Br J Anaesth* 55:141-143, 1983
7. Bachman AL: Benign, non-neoplastic conditions of the larynx and pharynx. *Radiol Clin North Am* 16:273-290, 1978
8. Tucker JA, Tucker G, Vidic B: Clinical correlation of anomalies of the supraglottic larynx with the staged sequence of normal human laryngeal development. *An Otol Rhinol Laryngol* 87:636-644, 1978
9. Gregor RT, Michaels L: Computed tomography of the larynx: A clinical and pathologic study. *Head Neck Surg* 3:284-296, 1981
10. Gamsu G, Webb WR, Shallit JB, Moss AA: CT in carcinoma of the larynx and pyriform sinus: Value of phonation scans. *Am J Roentgenol* 136(3): 577-584, 1981
11. Baker HL, Berquist TH, Kispert DB, Reese DF, Houser OW, Earnest F, Forbes GS, May GR: Magnetic resonance imaging in a routine clinical setting. *Mayo Clin Proc* 60:75-90, 1985

Anesthesiology
68:142-145, 1988

Identification of Inadvertent Intravenous Placement of an Epidural Catheter in Obstetric Anesthesia

A. VAN ZUNDERT, M.D., PH.D.,* L. VAES, M.D.,† M. SOETENS, M.D.,† A. DE WOLF, M.D.*

One of the complications of epidural analgesia and anesthesia is an unrecognized puncture of an epidural vessel by the needle or the epidural catheter, followed by accidental intravascular (iv) injection of the local anesthetic. Aspiration before injecting is recommended, but may not be entirely reliable.^{1,2}

We recently described an effective and safe test dose,

which could be very useful in obstetric epidural anesthesia,³ especially if combined with an improved technique to monitor maternal heart rate (MHR) using a cardiotocograph.⁴ We found that a test dose, consisting of 12.5 mg of bupivacaine plus 12.5 µg of epinephrine in 10 ml of physiologic saline, injected in a peripheral arm vein over 30 s, gives a predictable and easily detectable increase in heart rate and arterial blood pressure, provided that the patient is adequately monitored.³ We speculated that a similar result would be seen if the test dose were injected in an epidural vein in a pregnant patient. We now report two cases of obstetric epidural analgesia, where the test dose was administered through an epidural catheter located in an epidural vein; intravascular injection was demonstrated clearly.

REPORT OF TWO CASES

Case 1. A 21-yr-old healthy primigravida (height 159 cm, weight 67 kg) presented at term for delivery under epidural analgesia. Because

* Staff Anesthesiologist, Department of Anesthesiology, Catharina Hospital.

† Staff Anesthesiologist, Department of Anesthesiology and Reanimation, St-Elisabeth Hospital.

Received from the Department of Anesthesiology, Catharina Hospital, Eindhoven, The Netherlands; and the Department of Anesthesiology and Reanimation, St-Elisabeth Hospital, 2300 Turnhout, Belgium. Accepted for publication August 17, 1987.

Address reprint requests to Dr. Vaes: Department of Anesthesiology and Reanimation, St-Elisabeth Hospital, 2300 Turnhout, Belgium.

Key words: Anesthetic techniques: epidural; regional. Complications: intravascular injection.

she was already moderately distressed by the painful contractions early in labor (cervical dilatation 2 cm), an epidural catheter was inserted at the L3-L4 interspace. It soon became clear that the catheter had entered an epidural blood vessel, because blood was aspirated freely. This catheter was left *in situ* and a second epidural catheter was inserted one segment lower. A blood sample was aspirated through the first epidural catheter for blood gas analysis, revealing venous blood: pH 7.36, p_{CO_2} 37 mmHg, HCO_3^- 21 mmol \cdot l $^{-1}$, pO_2 43 mmHg, and O_2 Sat 78%. After obtaining informed consent from the patient, our test dose⁵ (10 ml of bupivacaine 0.125% plus epinephrine 1:800,000) was injected over 30 s, while monitoring the MHR⁴ and arterial blood pressure. Within 20 s after the end of the injection, a steep increase in the beat-to-beat MHR lasting 40 s was seen on the cardiocorograph HP 8030A (fig. 1), confirming the iv position of the epidural catheter. Arterial blood pressure increased from 100/60 to 130/90 mmHg 1 min after injection of the test dose and returned gradually to normal within 5 min. The only subjective symptom was a short-lived period of palpitations. Obviously, no analgesia developed. Then the same test dose was given through the second epidural catheter after a negative aspiration test for blood or cerebrospinal fluid. No signs of an accidental iv or spinal injection were seen, and analgesia developed within 15 min. This second epidural catheter was used for intermittent administration of bupivacaine 0.125% with epinephrine 1:800,000, and, 5 h after the start of the epidural analgesia, a healthy boy (weight 2800 g) was delivered spontaneously (expulsion time 4 min). The patient received a total of 60 mg of bupivacaine plus 60 μ g of epinephrine.

Case 2. A 28-yr-old multipara (height 178 cm, weight 80 kg) presented at term for delivery of her second child under epidural analgesia. Her medical and obstetrical history was uneventful. An epidural catheter was inserted at the L3-L4 interspace early in labor. A test dose (12.5 mg bupivacaine plus 12.5 μ g epinephrine) was injected after a negative aspiration test for blood and cerebrospinal fluid while monitoring continuously the MHR using the fetal cardiocorograph HP 8040A.⁴ After 20 min, sensory analgesia was detected from T9-L2. A continuous epidural technique was then chosen administering 10 ml per hour of the same solution (bupivacaine 0.125% plus epinephrine 1:800,000) using a Treonic IP3[®] (Vickers Medical, Hants, England) volumetric pump. The level of the sensory block was evaluated hourly. Arterial blood pressure was monitored intermittently. Excellent analgesia was obtained during several hours, but, 5 h after the start of the epidural analgesia, the patient began to complain of painful contractions. The level of the sensory block was determined by pinprick, and was found to be from T12-L5. Neither malfunction of the equipment, nor disconnection could be found. As an aspiration test was negative, we decided to give a bolus of 10 ml, containing 12.5 mg of bupivacaine plus 12.5 μ g of epinephrine (which is the same dose as our test dose), injected over 30 s. Within 1 min, the patient complained of short-lived palpitations. On the cardiocorograph, a steep rise in the MHR from 100 bpm to 130 bpm and a temporary decrease in the uterine contractions were seen (fig. 2). The arterial blood pressure rose from 105/70 to 140/85 mmHg at about 1 min after the injection, returning towards baseline values within a few minutes. These signs demonstrated the intravascular migration of the catheter. After disconnecting the bacterial filter (Portex[®] 100/386/010 0.2 μ m), aspirating through the epidural catheter revealed that the epidural catheter indeed had entered an epidural vein, since, this time, blood could be aspirated. A blood gas analysis (pH 7.40, p_{CO_2} 33 mmHg, HCO_3^- 20.1 mmol \cdot l $^{-1}$, BE -3.7 mmol \cdot l $^{-1}$, pO_2 49 mmHg, and O_2 Sat 85%) indicated venous blood. The epidural catheter was removed and another catheter was inserted at the same site. The further progress was uneventful, and, 11 h after the start of the epidural analgesia, a healthy boy (weight 4100 g, Apgar scores at 1 and 5 min 8 and 10, respectively) was delivered with the help of a vacuum extractor (expulsion time 14 min). In total, 160 mg of bupivacaine was given.

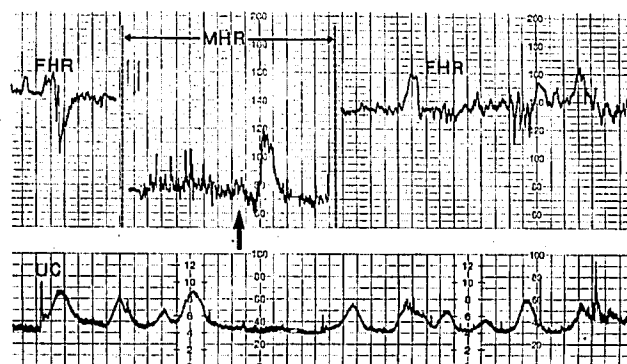


FIG. 1. A clear increase in maternal heart rate (MHR) is seen after injection of the test dose, confirming the intravenous location of the epidural catheter. The arrow shows the start of injection. Thirty seconds is represented between each pair of vertical lines (paper speed is 1 cm/min). FHR = fetal heart rate; UC = uterine contractions.

DISCUSSION

Accidental iv injection of local anesthetics during epidural analgesia and anesthesia is not uncommon, and may be potentially hazardous and even fatal, especially when bupivacaine or etidocaine is used.⁵ The reported incidence of blood vessel puncture of a catheter into the lumbar epidural space in obstetric practice appears to vary between 1 and 10%.⁶ Therefore, as a safeguard against accidental subarachnoid or intravenous injection, a test dose containing local anesthetic and epinephrine is recommended before performing an epidural blockade.^{1-3,7} However, controversy still exists about the use of epinephrine in the test dose in obstetric epidural anesthesia.⁸ First, maternal heart rate may vary considerably during active labor, and increases in heart

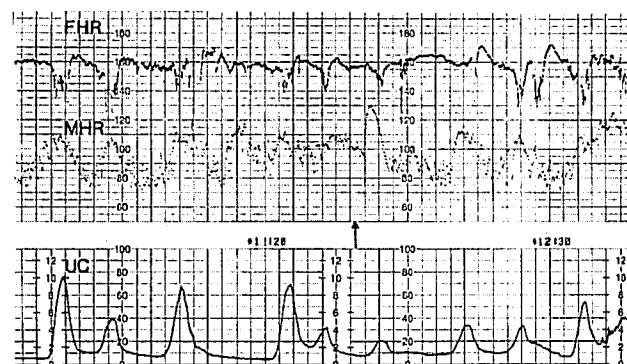


FIG. 2. Although there is a highly variable maternal heart rate (MHR), injecting the test dose through the epidural catheter demonstrates the intravenous migration of this catheter. The arrow shows the start of injection. Thirty seconds is represented between each pair of vertical lines (paper speed is 1 cm/min). FHR = fetal heart rate.

rate can be seen even after injecting plain bupivacaine.⁹ But the iv epinephrine effect on MHR can be distinguished easily when a cardiotocograph is used to monitor MHR, even in the presence of wide swings in MHR (fig. 2).^{3,4} It is important to monitor MHR very closely during and after injecting the test dose; otherwise, the brief increase in heart rate could be missed clinically. Second, it has been argued not to use epinephrine in a test dose because, if injected iv, it could decrease uteroplacental blood flow, especially when pre-eclampsia is present. Indeed, there is evidence now that iv injection of epinephrine decreases uterine blood flow in animals.^{10,11} However, it is not clear if the same thing occurs in humans, since species differences are certainly present (in contrast to humans, all animals demonstrated bradycardia after iv epinephrine). The animal studies^{10,11} measured total uterine blood flow, and did not differentiate between myometrial and placental perfusion. Although we realize that uteroplacental blood flow is already reduced in patients with pre-eclampsia, we feel that adding a small dose of epinephrine to the test dose to detect an accidental iv injection is justified to prevent a possible catastrophe, even and, perhaps, especially in the presence of complicated labor, until another reliable and, possibly, safer marker of iv injection is developed.

Because iv or intrathecal migration of the catheter is always possible, and reliance on aspiration of the catheter is not an entirely dependable way of avoiding subarachnoid or iv injection, the test dose should also be given before each top-up dose when an intermittent epidural technique is used.⁷ When using a continuous infusion technique, hourly testing of the level of anesthesia will give warning in time in case of transdural migration, since it has been demonstrated that an infusion of bupivacaine $12.5 \mu\text{g} \cdot \text{h}^{-1}$, if given accidentally intrathecally, would need to continue for 2 h before creating a blockade higher than T9–T10.¹²

Recently, we proposed that injecting 12.5 mg of bupivacaine plus $12.5 \mu\text{g}$ of epinephrine in 10 ml of physiologic saline in a peripheral arm vein over 30 s in pregnant patients gives a predictable and easily detectable increase in heart rate and blood pressure.³ However, we were not yet absolutely sure if injecting the test dose in an epidural vein would be equally recognizable. During pregnancy, there is a partial or total obstruction of the inferior vena cava, and blood return from the lower body has to go *via* the paravertebral (epidural) veins emptying into the azygos system.¹³ Lateral position partially relieves the obstruction. The obstruction results in pooling of venous blood,¹³ which, theoretically, could increase circulation time from an epidural vein to the

heart. In turn, this could make the effects of epinephrine in the test dose less clear. However, as with intravenous injection through an arm vein, within less than 1 min we observed a transient tachycardia and a temporary increase in arterial blood pressure, together with a temporary decrease in contraction intensity or contraction frequency in the two reported cases. Clinically, the time between injection of the test dose and the onset of tachycardia was similar in our cases, compared to the intravenous injections in a peripheral arm vein,³ so that stasis of venous blood in the epidural space is unlikely. Therefore, an iv injection of a test dose containing epinephrine into a catheter which is located in an epidural vein can be detected easily if the mother is adequately monitored. If one is still uncertain about the position of the epidural needle or catheter after close observation of MHR and arterial blood pressure, the test dose should be repeated.

In conclusion, these two cases suggest that the previously proposed test dose (12.5 mg of bupivacaine plus $12.5 \mu\text{g}$ of epinephrine in 10 ml of physiologic saline)³ is effective in detecting accidental iv location of an epidural catheter in the clinical setting of a pregnant patient undergoing epidural analgesia. Furthermore, our experience during the second case suggests that removal of the bacterial filter before aspirating might make the aspiration test more reliable.

REFERENCES

1. Abraham RA, Harris AP, Maxwell LG, Kaplow S: The efficacy of 1.5% lidocaine with 7.5% dextrose and epinephrine as an epidural test dose for obstetrics. *ANESTHESIOLOGY* 64:116–119, 1986
2. Crawford JS: Some maternal complications of epidural analgesia for labour. *Anaesthesia* 40:1219–1225, 1985
3. Van Zundert A, Vaes L, Soetens M, De Vel M, Van Der Aa P, Van Der Donck A, Meeuwis H, De Wolf A: Every dose given in epidural analgesia for vaginal delivery can be a test dose. *ANESTHESIOLOGY* 67:436–440, 1987
4. Van Zundert AA, Vaes LE, De Wolf AM: ECG monitoring of mother and fetus during epidural anesthesia. *ANESTHESIOLOGY* 66:584–585, 1987
5. Albright GA: Cardiac arrest following regional anesthesia with etidocaine or bupivacaine (editorial). *ANESTHESIOLOGY* 51:285–287, 1979
6. Verniquet AJW: Vessel puncture with epidural catheters. Experience in obstetric patients. *Anaesthesia* 35:660–662, 1980
7. Moore DC, Batra MS: The components of an effective test dose prior to epidural block. *ANESTHESIOLOGY* 55:693–696, 1981
8. Leighton BL, Norris MC, Sosis M, Epstein R, Chayen B, Larjani G: Limitations of epinephrine as a marker of intravascular injection in laboring women. *ANESTHESIOLOGY* 66:688–691, 1987
9. Cartwright PD, McCarroll SM, Antzaka C: Maternal heart rate changes with a plain epidural test dose. *ANESTHESIOLOGY* 65:226–228, 1986

10. Hood DD, Dewan DM, James FM: Maternal and fetal effects of epinephrine in gravid ewes. *ANESTHESIOLOGY* 64:610-613, 1986
11. Chestnut DH, Weiner CP, Martin JG, Herrig JE, Wang JP: Effect of intravenous epinephrine on uterine artery blood flow velocity in the pregnant guinea pig. *ANESTHESIOLOGY* 65:633-636, 1986
12. Bogod DG, Rosen M, Rees GAD: Extradural infusion of 0.125% bupivacaine at $10 \text{ ml} \cdot \text{h}^{-1}$ to women during labour. *Br J Anaesth* 59:325-330, 1987
13. Cheek TC, Gutsche BB: Maternal physiologic alterations during pregnancy, Anesthesia for Obstetrics, 2nd edition. Edited by Shnider SM, Levinson G. Baltimore, Williams & Wilkins, 1987, pp 3-13

Anesthesiology
68:145-146, 1988

Reduction Cranioplasty and Severe Hypotension

DAVID PARSONS, M.D., F.R.C.P.(C.),* STANLEY I. SAMUELS, M.B., F.F.A.R.C.S.,†
GARY STEINBERG, M.D., PH.D.,‡ LAWRENCE SHUER, M.D., F.A.C.S.§

Reduction cranioplasty is an extremely rare operation performed in children when hydrocephalus has been neglected or ineffectually treated. We report the anesthesia problems encountered with removal of the cranium and the resultant loss of a massive amount of cerebrospinal fluid (CSF).

CASE REPORT

A 5-yr-old, 18-kg woman with congenital hydrocephalus presented with a head circumference of 90 cm (normal 51 cm) and the inability to hold her head securely enough to ambulate. She had developed slowly but was able to communicate in two languages. She had undergone several prior ventricular peritoneal shunt revisions because of shunt malfunction, infection, and chronic subdural hematomas. At the time of surgery, she had a functioning shunt in place, was alert, and was without focal neurological signs. The surgical plan was to identify the sagittal sinus by digital subtraction angiography in order to avoid massive hemorrhage intraoperatively, and then to undertake a reduction cranioplasty.

Without premedication, anesthesia was induced in the radiology suite at 0730 h with nitrous oxide, oxygen, and halothane without incident. Once an intravenous line was established, endotracheal intubation was facilitated by the administration of pancuronium bromide

1.5 mg iv. Monitoring included ventilation-oxygen monitors, as well as electrocardiogram, precordial stethoscope, nasal temperature, automated blood pressure device, and pulse oximetry.

During the 3½-h radiological procedure, anesthesia was maintained with nitrous oxide, oxygen, and halothane, and pancuronium was used for muscle relaxation. Of note was a decrease in heart rate commensurate with the decrease in temperature from an initial 35.6° to 34.5° C. Following the radiological procedure, the child was transferred to the operating room, where a number of changes in anesthetic management became necessary. The patient's position was changed to semi-sitting and, thus, the nitrous oxide was discontinued and end-tidal carbon dioxide and nitrogen were monitored by mass spectrometry. A precordial Doppler was placed and tested with an aspirated saline bubble embolus. A left external jugular line was placed and passed centrally. A left radial arterial line was also inserted. A peripheral nerve stimulator and urinary catheter were also utilized. The anesthetic technique was as changed to a narcotic-based (morphine) one, with the anticipation of postoperative controlled ventilation.

The reduction in temperature was treated with warming lights, a warming blanket, and a heated humidifier placed in the circle system. Fluid management consisted of $3.0 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ maintenance and $4.0 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ for third space losses. Blood loss was replaced to account for measured and estimated losses.

Five and one-half hours later, as the cranial cap was being removed, there was a sudden and marked loss of CSF estimated at 1000-1500 ml. At the same time, the arterial blood pressure dropped progressively over a 1-2-min period from a mean of 70 mmHg to 35 mmHg. There was no change in ventilatory status, and no recent drug administration had occurred. The blood loss at that time was estimated to be 300 ml, which had been fully replaced.

Intravenous fluids were administered as blood and crystalloid. Vasopressors, including ephedrine and epinephrine, as well as calcium chloride, were used with little effect except for transient increases in arterial blood pressure. Additional lines for intravenous fluids were established and external cardiac compressions were begun when mean arterial pressures declined below 35 mmHg.

Continued low blood pressures mandated a change in position to Trendelenburg, but this action markedly accelerated the blood loss from sagittal sinus bleeding, making resuscitation more difficult. Blood loss following the change in position was estimated at 2000 ml during the resuscitation period. Resuscitation continued for 55 min

* Resident in Anesthesia, Department of Anesthesia.

† Associate Professor of Anesthesia (Clinical), Department of Anesthesia.

‡ Assistant Professor of Neurosurgery, Division of Neurosurgery.

§ Assistant Professor of Neurosurgery (Clinical), Division of Neurosurgery.

Received from the Department of Anesthesia and the Division of Neurosurgery, Stanford University Medical Center, Stanford, California. Accepted for publication August 18, 1987.

Address reprint requests to Dr. Parsons: The Vancouver General Hospital, Department of Anaesthesia, 855 West 12th, Vancouver, British Columbia, Canada V5Z 1M9.

Key words: Anesthesia; neurologic; pediatric. Complications: hypotension; hypovolemia.