the large section of the liver. These intrahepatic veins exposed during resection are thin walled and anatomically difficult to collapse. During resection, the IVC was manipulated or compressed, which narrowed the portion of the IVC under the junction of hepatic veins. The air might have been drawn inward via the large number of small hepatic veins open to the atmosphere, aided by a Venturi effect (fig. 1)

In summary, these cases demonstrate that VAE can occur during hepatic resection in patients in the supine position even if the opening of a large vein does not occur. This study does not define the frequency of VAE during hepatic resection, but it does suggest that this is not a rare event. Therefore, the possibility of VAE must always be considered during hepatic resection and appropriate monitoring is recommended.

REFERENCES


Tracheal Intubation Is Not Invariably Confirmed by Capnography

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Capnography is a highly reliable method for confirming tracheal intubation. Continued presence of a CO₂ waveform almost invariably confirms tracheal intubation, and absence of a capnogram strongly suggests esophageal intubation. We could find only one previous report in which a CO₂ waveform was absent despite proper tracheal tube placement and subsequent adequate ventilation.¹ This occurrence related to inadvertent application of PEEP to a loosely fitted (uncuffed) tracheal tube that caused expiratory gases to escape around the tube. The incidents reported here describe two markedly different problems in which decisions based on capnography alone would have led to a misdiagnosis. In each situation, patient safety required expeditious intubation and was jeopardized by unexpected difficulties both in laryngoscopy and subsequent confirmation of intubation.

CASE REPORTS

Case 1. An obese 67-yr-old woman (weight, 188 kg; height, 1.55 m) underwent embolectomy of a clotted femoral popliteal arterial graft after induction of spinal anesthesia (10 mg hyperbaric tetracaine with 0.2 mg epinephrine). Sensory anesthesia to the T6 dermatomal level was produced with only a transient decrease in blood pressure that responded quickly to a fluid bolus (200 ml) and ephedrine (5 mg). Twenty minutes after surgical incision, heparin (5,000 U) was injected intravenously to prevent thrombosis of the artery during application of the cross-clamp. After removal of the clamp, the surgeons requested that the patient be given 20 mg protamine. A 1-mg test dose of prot-
amine was administered. Within 3 min, the patient’s systolic blood pressure decreased from 120 to 55 mmHg, and she became apneic and unresponsive. Ventilation of the lungs via a face mask and oral airway (100% oxygen) was difficult; the pulse oximeter revealed a rapid decline in hemoglobin oxygen saturation (SpO₂) to 84% and thereafter failed to provide readings. Laryngoscopy was extremely difficult; the vocal cords could not be visualized, and a tracheal tube was passed blindly beneath the epiglottis. Ventilation seemed impossible. Inspiratory pressure exceeded 50 cmH₂O, and breath sounds were absent. Reintubation was contemplated due to the lack of a CO₂ waveform on the capnograph (Pulse Oximeter/ETCO₂ Monitor Model 7000, Novametrix Medical Systems, Wallingford, CT). Ventilation, however, quickly began to improve, bronchial wheezing became audible bilaterally, and a capnographic tracing appeared after an iv epinephrine (0.3 mg) injection that was administered for a suspected anaphylactic reaction to protamine. The pulse oximeter regained its signal, and SpO₂ increased to 94% with an FiO₂ of 1.0. The patient survived, and the only complication was rethrombosis of the femoral graft.

Case 2. An obese 64-yr-old woman (weight, 104 kg; height, 1.6 m) with aortic stenosis and gastroesophageal reflux presented for urgent drainage of an infected hip prosthesis. Her preoperative vital signs were as follows: blood pressure, 140/80 mmHg; pulse, 105 beats per min; respiratory rate, 18 breaths per min; and temperature, 37.8° C. She had had nothing to eat or drink during the preceding 7 h. As a further precaution against aspiration, she had received ranitidine (50 mg) and meclopramide (10 mg) intravenously. Anesthesia equipment check, as usual, included calibration of the capnometer. After the patient breathed oxygen for 5 min, cricoid pressure was applied; anesthesia was rapidly induced (100 µg fentanyl, 16 mg etomidate, and 100 mg succinylcholine); and laryngoscopy was performed. The vocal cords could not be visualized, and the tracheal tube (7.0 mm) was placed without seeing the glottis. The cuff was inflated, and subsequent auscultation over the thorax and upper abdomen revealed muffled sounds of air movement. Although no capnographic tracing was evident, the pulse oximeter showed an SpO₂ of 98%, and vital signs were unchanged. We therefore proceeded to reevaluate capnometer function despite lingering uncertainty about correct tracheal tube placement. The anesthesiologist removed the cuvette from the breathing circuit, exhaled through it, and noted the absence of a capnogram. After placing a new cuvette in the breathing circuit, a normal CO₂ waveform appeared. The remainder of the anesthetic proceeded uneventfully over the next several hours.

Discussion

Rapid confirmation of proper tracheal tube placement is one of the most important tasks confronting anesthesiologists. Clinical evaluation is not always reliable, and at least one report has documented the inability of several examining physicians to detect prolonged esophageal intubation in a critically ill patient with respiratory failure. Similarly, pulse oximetry may be misleading; despite esophageal intubation, minutes may elapse before oxygen desaturation occurs in patients whose lungs have been denitrogenated with 100% O₂. Also, despite proper tracheal tube placement, SpO₂ may be decreased in patients with severe cardiopulmonary dysfunction. For the above reasons, a capnographic CO₂ waveform is emerging as the standard for rapid detection of esophageal intubation. Capnography, however, has important limitations, as emphasized by the incidents presented here.

In the first case, severe bronchospasm and hypotension were associated with the absence of a CO₂ waveform. A waveform appeared almost immediately after epinephrine therapy partially relieved bronchospasm. We believe that the lack of a CO₂ waveform in this case resulted from complete airways obstruction. Although our patient was severely hypotensive during the anaphylactic reaction to protamine, it is unlikely that the decrease in blood pressure caused the capnogram to be absent; capnograms, albeit with markedly diminished amplitudes, have even been reported shortly after cardiac arrest when airways remain unobstructed.

In the second case, mechanical failure caused the capnogram to be absent. The problem, related to a hardware anomaly of our mainstream capnometer (Novametrix Pulse Oximeter/ETCO₂ Monitor Model 7000), could not be detected by internal calibration (using two reference signals) of the device. In this case, the device failed to detect the display of a CO₂ waveform. However, because some light transmission occurred, there was no error message to alert the anesthesiologists to a malfunction.

This lack of a capnogram before intubation was not interpreted as a mechanical failure but rather as an expected consequence of our anesthetic management. During denitrogenation of the lungs, high flows of oxygen (8 l/min) were used, and the mask was applied loosely for reasons of patient comfort. Such conditions often preclude the display of a CO₂ waveform. In addition, due to concerns about regurgitation and aspiration of gastric contents, mask ventilation was not performed after iv induction of anesthesia. Thus, our first attempt to interpret information from the capnograph followed a difficult intubation. The absence of a capnogram merely added to our uncertainty regarding proper tracheal tube placement. As a result of this experience, we believe that a complete evaluation of capnometer function should include exhalation through the breathing circuit (by the anesthesiologist or the patient) to demonstrate a CO₂ waveform before the induction of anesthesia.

Clinicians who routinely employ capnometers during anesthesia have come to rely on these devices to confirm proper intubation of the trachea. A normal capnogram virtually ensures tracheal intubation while its absence strongly suggests esophageal intubation. However, decisions based on capnography alone may lead to misdiagnosis when severe cardiopulmonary dysfunction is present or mechanical failures occur. Thus, rapid evaluation of tracheal tube placement should be based on information from monitors in combination with careful clinical assessment.

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Total Spinal Anesthesia Following Early Prophylactic Epidural Blood Patch

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The use of an epidural blood patch (EBP) to treat post-lumbar puncture headache (PLPH) when conservative measures have failed is well established.1,2 Complications are infrequent and usually transient.1–3 The occurrence of headache is most frequent when the dura has been accidentally punctured during attempts to establish epidural anesthesia due to the large size and shape of the needle employed. In such cases, it has been suggested that a blood patch be used prophylactically4,5 via a successfully placed epidural catheter before the catheter is withdrawn at the end of the operation. This practice, however, remains controversial.6

This is a report of an unpredicted and previously unreported response to the use of a prophylactic EBP administered at the end of surgery but before the block had receded.

CASE REPORT

A 27-yr-old, 84-kg, 170-cm man was scheduled for the repair of a torn anterior cruciate ligament. He had previously had uneventful epidural anesthesia for arthroscopy of the knee. He agreed to epidural anesthesia again but requested that he be well sedated throughout the procedure. He was in otherwise excellent health.

After preanesthetic medication with 10 mg im morphine and 50 mg hydroxyzine, he was alert but calm. With the patient in the lateral position, the first attempt at placing an 18-G epidural needle resulted in accidental penetration of the dura. The epidural space was then successfully identified one space lower at L4–5. An epidural catheter was inserted and threaded 4 cm with the needle tip pointing cephalad. After a negative test dose, satisfactory anesthesia was established with a total of 20 ml lidocaine 1.5% with 1200,000 epinephrine. Three supplemental doses of 10 ml each were given during the procedure to maintain a level at T4–6. To assist with postoperative analgesia, 5 mg morphine was added to the final dose. The patient also received iv sedation with 2 mg midazolam and 7.5 mg nalbuphine intraoperatively. He was rousable but would fall asleep readily. Pulse and blood pressure remained in the normal range, and oxygen saturation (SpO2) was 100% with oxygen 4 l/min via nasal prongs.

The surgery was completed sooner than expected after the last dose of lidocaine. The analgesic level was T4 at this time, which was 250 min after the initial dose and 35 min after the final dose that contained the morphine. Before withdrawing the epidural catheter, it had been decided to give an epidural blood patch. Fifteen milliliters of blood were drawn from an arm vein and injected through the epidural catheter while the surgical dressing was being applied. Two minutes later, spontaneous respiration abruptly ceased, and in response to commands the patient could move only his facial muscles. The lungs were ventilated via mask with 100% oxygen as the block rapidly ascended. The trachea was intubated without neuromuscular relaxants, but 125 mg thiopental was given to provide amnesia. His blood pressure decreased transiently to 100/60 mmHg before returning spontaneously to 120/70 mmHg. Naloxone (0.1 mg × 2) was given without any response. His SpO2 remained normal throughout, but his pupils became widely dilated and unresponsive to light.

On arrival in the postanesthesia care unit, the patient was deeply unconscious and required controlled ventilation of his lungs. Initially, his blood pressure increased to 167/107 mmHg and his pulse to 106 beats per min, but they returned to normal levels after he was given 5 mg labelatal and 5 mg hydralazine. The patient regained consciousness and cranial nerve function 70 min after the EBP (115 min after the last dose of lidocaine). The level of block receded steadily over the next 90 min, and the trachea was extubated when satisfactory spontaneous respiration resumed. He required one dose of im morphine for surgical pain after discharge to the ward. Subsequently, he developed no signs of meningeval irritation, back pain, or headache. He was totally amnestic for the whole perioperative period.

DISCUSSION

The sequence of events in this case strongly suggest that the occurrence of acute total spinal anesthesia was temporally and directly related to the injection of 15 ml autologous blood via the epidural catheter. The timing of the event makes it highly unlikely that the previous dose of lidocaine, on its own, coincidentally resulted in

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