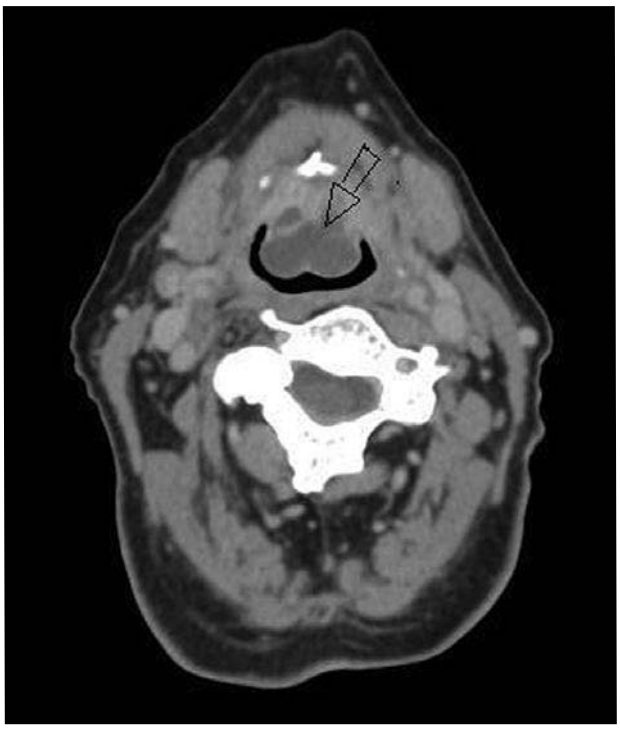

FIGURE 2 Computed tomography scan of the neck reveals an enlarged epiglottis (cross section view).

demonstrate signs of upper airway obstruction during the initial postoperative period, she complained of sore throat, hoarseness and dysphagia. Trauma to the epiglottis could have occurred during intubation or extubation of the trachea, possibly in association with suctioning of the oropharynx. Anticoagulant medications have also been identified as a possible risk factor in association with epiglottic hematoma.[3,4] This was unlikely to have been an issue in this case, as the patient's warfarin had been discontinued, and she had normal PT and APTT values preoperatively. It is uncertain if a residual anti-platelet effect of her non-steroidal anti-inflammatory drug (discontinued two days prior to surgery) may have been an associated risk factor in development of an epiglottic hematoma.

This report highlights that sore throat, hoarseness and dysphagia following endotracheal intubation could be presenting symptoms of the rare, but potentially serious complication of an epiglottic hematoma. This entity should be considered in the differential diagnosis of upper airway lesions when confronted with this triad of symptoms following anesthesia where endotracheal intubation was performed, especially in patients receiving concurrent anticoagulation therapy.

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Morbidity in a pediatric patient having alcohol ablation of an arteriovenous malformation

To the Editor:

An otherwise healthy 25-month-old, 11.8 kg male child was scheduled for alcohol embolization of an arteriovenous malformation on the back of his skull. The procedure was performed under general anesthesia. The duration of the case was approximately three hours and 45 min, and the patient remained hemodynamically stable throughout. After obtaining a cerebral angiogram, the arteriovenous malformation and fistula were treated with 11.5 mL of anhydrous alcohol administered in 2–3 mL increments over approximately one hour. The procedure was stopped after a dose of 1 $mL \cdot kg^{-1}$ of alcohol had been injected.

In order to monitor the metabolic effects of the injected alcohol, arterial blood gas chemistry and serum ethanol levels were obtained at the conclusion of the case. His arterial pH was 7.19, $PaC0_2$ 50 mmHg, $Pa0_2$ 121 mmHg, $HC0_3^-$ 19 $mmol \cdot L^{-1}$, BE -9, anion gap 31, Na^+ 133 $mmol \cdot L^{-1}$, K^+ 4.6 $mmol \cdot L^{-1}$, HCT 26% (preoperative 31.9%). The urine was serosanguinous at this time, and the serum alcohol level was 85.2 $mg \cdot dL^{-1}$ (legal intoxication is defined as > 80 $mg \cdot dL^{-1}$).

In response to the metabolic derangement, the patient received sodium bicarbonate 15 mEq *iv*. He was allowed to emerge from anesthesia, and his trachea was extubated in the catheterization labora-

tory. He was drowsy initially but arousable, and his breath smelled of ethanol. Shortly following admission to the pediatric intensive care unit (PICU), serum chemistry showed an improvement in his acid-base status. However, he was profoundly hypoglycemic, with a serum glucose of 14 $mg \cdot dL^{-1}$. While dextrose 50% increased his serum glucose to 112 $mg \cdot dL^{1}$, a peripheral blood smear showed "poc marks" on approximately 1% of erythrocytes. He was diagnosed with intravascular hemolysis secondary to the systemic absorption of anhydrous alcohol. The patient continued to be monitored in the PICU, where he was carefully hydrated and received a maintenance glucose infusion. His serum creatinine remained within the normal range, while serum alcohol concentration decreased to 7.5 $mg \cdot dL^{-1}$ within two hours. The remainder of his postoperative course was uneventful, and the patient was discharged from hospital two days following the procedure, in good condition.

Doses of alcohol up to 1 $mL \cdot kg^{-1}$ have been used for vascular ablation procedures. Multiple complications have been reported, including ethanol intoxication, intravascular hemolysis, hematuria, hyperthermia, elevated creatinine, hypoglycemia, pulmonary edema, pulmonary embolism, skin necrosis, and skin blistering.[1–3] The serum concentration of alcohol is proportional to the amount of alcohol injected.[1] This is the first reported case involving a young pediatric patient, and suggests that the effects of anhydrous alcohol are similar to those observed in older patients. However, our experience demonstrates several unique considerations in this population. Hypoglycemia in children is a non-dose dependent response to alcohol intoxication.[4,5] This patient had profound hypoglycemia which corrected quickly following dextrose administration.

The mechanism of metabolic acidosis in patients intoxicated with alcohol has not been clearly elucidated. In this setting, the base deficit and lactate levels do not correlate with serum alcohol levels.[3] The etiology of the acidosis in this patient did not appear to indicate poor perfusion. The acidosis resolved quickly with a small dose of bicarbonate. Laboratory analysis suggested the etiology of the hematuria was hemolysis. Reports suggest that the incidence of hemoglobinuria is not related to the alcohol level.[1] Up to 28% of patients have been reported to have hemoglobinuria during alcohol ablation.

In summary, this report describes the systemic effects of anhydrous alcohol injected in a child for treatment of a cerebral arteriovenous malformation. Vigilance for complications, and a continuous infusion of glucose with frequent glucose monitoring, should be strongly considered in any young child undergoing these procedures.

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Thromboelastographic® monitoring of the efficacy of recombinant factor VIIa administration in a parturient with factor VII deficiency

To the Editor:

A 35-yr-old parturient with factor VII deficiency presented at term with a factor VII level of less than 3%. Coagulation studies revealed a prothrombin time (PT) of 32.3 sec, international normalized ratio (INR) of 3.2, partial thromboplastin time (PTT) of 30.3 sec and hematocrit (Hct) of 37.2%. Two units of fresh frozen plasma (FFP) were transfused prior to epidural placement. A follow-up set of coagulation studies revealed a PT of 22.9 sec, INR of 2.0 and PTT of 30.3 sec. Thromboelastography® analysis revealed no difference in her coagulation status as compared to that prior to administration of FFP with the R-value remaining unchanged from 4.8 min [pre FFP: R = 4.8 min, measure of speed of clot strengthening (K) = 1.0 min, angle = 74.6°, measure of maximum strength of developed clot (MA) = 69.5 mm and post FFP: R = 4.8 min, K = 1.2 min, angle = 72.2°, MA = 67.7 mm]. Cesarean section was performed 16 hr later for failure