patients receiving epidural morphine, patient satisfaction with the relief obtained was comparable. Factors responsible for the lack of correlation between apparent pain and increased pain tolerance and satisfaction may be related to a more uniform level of analgesia, a lack of troublesome side effects, independence, and an awareness that reliable analgesia will be provided quickly as required. Patients utilizing PCA morphine did not achieve total analgesia, but settled at a level in which comfort was balanced by troublesome side effects, *i.e.*, increasing sedation, nausea, and dizziness. It may well be that morphine is not the optimal narcotic for use in PCA. An agent with faster onset, less sedation, and fewer adverse effects might further increase patient satisfaction and increase levels of analgesia to levels achieved with epidural narcotics.

In summary, two newer modalities of providing postoperative pain relief offered either superior analgesia or higher patient satisfaction than traditional im dosing. Epidural morphine offers the best possible analgesia if one is willing to accept higher morbidity and risks of delayed respiratory depression. In settings where epidural narcotics cannot be administered, the high patient satisfaction and uniform analgesia offered by PCA provides an attractive alternative.

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Venous Air Embolism during Removal of Tissue Expander in a Supine Child

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Venous air embolism (VAE) is a well-known complication of neurosurgical procedures, especially those done in the sitting position.[1,2] Additionally, isolated case reports have also appeared in the literature describing VAE in adults and children having a variety of extra-cranial procedures performed.[3-11] We describe a case in which venous air embolism occurred in a child having a large scalp flap raised after it had been delayed and enlarged with a tissue expander.

CASE REPORT

A 14-month-old boy had a tissue expander placed in a sub-galeal pocket to delay and expand a scalp flap in preparation for removal of a giant nevus. Anesthesia consisted of halothane, N_2O, and O_2. A 4.5-mm ID oral endotracheal tube was inserted during paralysis from

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pancuronium, 0.1 mg/kg iv and ventilation was mechanically controlled. The anesthesia and operation lasted 70 min and was uneventful.

At age 17 months, this child returned to the operating room for removal of the tissue expander, excision of the scalp nevus, and closure of wound with flap advancement. The child weighed 12 kg and had an estimated blood volume of 900 cc. Monitoring included ECG, blood pressure cuff, chest stethoscope, rectal temperature, and inspired oxygen concentration. Anesthesia consisted of 1% halothane, N_2O, and O_2 *via* mask, followed by iv pancuronium 0.1 mg/kg, orotracheal intubation, and mechanical ventilation. The child was placed in the supine position. His head was draped in a manner to allow side-to-side movement. The site of the incision was level with the heart at its lowest point and 7 cm above the heart at its highest. Surgery proceeded uneventfully for 1 h. At this time, the nevus had been excised, the tissue expanders removed, and flaps raised in the subgaleal plane. Moist laps were placed under the delayed flaps for hemostasis. The periosteum had not been incised or elevated. The estimated blood loss was 100 ml. Five per cent dextrose in lactated Ringer's solution had been infused. Suddenly arterial blood pressure (BP) decreased from 90 mmHg systolic to become unobtainable; the ECG showed a sinus rhythm increasing from 110 bpm to 125 bpm. No heart tones were heard and a pulse was not palpable. The presence of breath sounds was confirmed bilaterally. All anesthetics were discontinued immediately, 100% O_2 administered, chest compression begun, and epinephrine, 200 μg, was administered iv. BP returned to 80 mmHg systolic and later to 110 mmHg while the heart rate increased to 170 bpm.

An arterial line and precordial Doppler were placed. Arterial blood gas revealed a pH_a of 7.66, Pa_{CO_2} of 15 mmHg, a Pa_{O_2} of 508 mmHg, BE of 1.6 mEq/1, and a Hct of 25%. Packed red blood cells, 60 ml, were administered iv. Anesthesia and surgery was then resumed with 1% halothane, air, and O_2 to allow the scalp wound to be closed. As the surgeon lifted the scalp flap, air sounds were immediately heard on the Doppler and the BP suddenly became unobtainable again. Oxygen, 100%, was administered, chest compressions begun, and 200 μg of epinephrine was administered iv. The head of the patient was lowered below the level of the heart while the surgeon flooded the field with saline. BP immediately returned to 100 mmHg while the heart rate increased to 170 bpm. The scalp was quickly closed while the patient was in the Trendelenburg position. The child was awakened and observed overnight in the Intensive Care Unit. A follow-up neurologic examination and CAT Scan of the brain were normal.

DISCUSSION

This is the first reported case of VAE occurring with the removal of a tissue expander from under a delayed scalp flap. In general, air enters the vessel when venous pressure at the surgical site is lower than atmospheric pressure and the vessel is patent. Vessel patency is often maintained when vessels are imbedded in bony or fibrous structures that prevent retraction and collapse of the cut end of the vessel, thus explaining the association between VAE and craniotomies. Children are especially at risk for unexpected reductions in venous pressure, since acute blood loss is more difficult to estimate. Seemingly small amounts of blood loss may represent a significantly larger proporion of estimated blood volume, and may result in undetected low venous pressure.

Previously, a variety of operations have been associated with an increased risk for VAE, the most common being those in which the patient is undergoing a craniotomy in the sitting position.[12] VAE has occurred in extra-cranial procedures, such as mastectotomy[3] and surgery for head and neck tumors,[4] diagnostic and therapeutic air injections,[5,6] venous catheter placement,[7,8] and chest injury,[9,10] and during total hip replacement.[11] Small children with their proportionately larger head size and abundant venous channels are especially prone to VAE during intraacranial surgery, even while in the supine position.[13,14] Usually, the surgical procedures associated with an increased risk for VAE involve elevation of bony flaps, large patent blood vessels, and elevated extravascular pressure.

Our patient may have been at risk for VAE for multiple reasons. First, blood loss of approximately 100 ml (10–15% of estimated blood volume) occurred from the scalp incision. While blood replacement was felt to be adequate, venous pressure may have been lower than expected. Second, the placement of a tissue expander with rapid stretching and growth of the scalp may have promoted enlargement of previously present venous channels, as well as the development of new ones. An increased blood flow occurs in the microcirculation of flaps that have been delayed in pigs.[15] Indeed, examining human skin histologically has revealed capillaries and small vessels that are dilated in the dermis and an established vascular layer in the capsule filled with dilated blood vessels and newly formed small vessels.[16] Furthermore, the tough fibrous capsule that develops under an expanded flap may inhibit vessel retraction and contraction when it is cut. Emissary veins originating intracranially and perforating the skull to drain into the scalp venous system probably provided an entry point for VAE.[17] Finally, surgical manipulation of the tissue flap may have elevated an open vessel in relation to the level of the heart, producing a further drop in intravascular pressure at the surgical site, as well as mechanically stenting open the venous channels.

With the increasing use of tissue expansion in the head and neck region of children, an awareness of the increased risk of VAE is essential. Adequate hydration, blood replacement,[18] and being cognizant of the level of surgical manipulation in relation to the heart can decrease the risk of VAE. For immediate recognition of VAE, non-invasive monitoring is well justified. This should include ECG, esophageal stethoscope, precordial Doppler, and measurement of end-tidal CO_2 concentration. The roll of a CVP remains controversial at this time.[19,20]

Compressing the jugular veins can increase venous pressure at the site of the incision, limiting the entrance of air into the venous system. The application of PEEP, although somewhat controversial because of the risks of elevated right atrial pressure, can also raise venous pressure, impeding the accumulation of air in the venous system.[21] Most importantly, however, is early recognition of air embolism to allow for rapid and effective treatment and to minimize postoperative sequelae.

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Effect and Interaction of *p*H and Lidocaine on Epinephrine Absorption

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To achieve optimal local hemostasis in patients undergoing surgery, a dilute solution of epinephrine is often injected. We previously demonstrated that lidocaine added to an epinephrine solution accelerates the transfer of epinephrine to the blood in patients undergoing craniotomy.[1] Sosis *et al.* suggested that the *p*H of the solution might have been responsible for the variation in epinephrine uptake attributed to lidocaine,[2] because the *p*H of commercially available lidocaine with epinephrine is made acidic to achieve stability of the added epinephrine. Therefore, in this study, we investigated the effect of the *p*H of the solution on the absorption of locally injected epinephrine into the blood stream.

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MATERIALS AND METHODS

After obtaining approval of the committee for the protection of human subjects and informed consent, we studied 40 (18 males and 22 females) ASA I and II adult