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Venous Embolism during Craniectomy in Supine Infants

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Venous air embolism (VAE) is a well-known complication of neurosurgical procedures. Its reported incidence ranges from 6-45% in seated adult neurosurgical patients,¹⁻³ and, occasionally, it is detected in the lateral, prone, or supine positions.³⁻⁵ VAE is thought to occur among seated pediatric neurosurgical patients with approximately the same frequency as among adults.^{6,7} Many neonates and small infants undergo neurosurgery, but the incidence of VAE among this population is unknown. Because several case reports have

documented VAE in supine infants having neurosurgery,^{5,8} we began a prospective study of VAE among infants undergoing craniectomy in the supine position.

MATERIALS AND METHODS

Following approval from our institution's Human Investigation Committee, 12 consecutive infants under 1 yr of age scheduled for elective repair of craniosynostosis were prospectively monitored for VAE. General anesthesia was induced with halothane, nitrous oxide, and oxygen by mask in 11 infants, and by intramuscular ketamine in one. Peripheral intravenous and arterial catheters were inserted, and oral or nasal endotracheal intubation was performed after administration of iv pancuronium bromide and fentanyl. Air filters were put on all of the intravenous lines to minimize microbubbles, but abandoned as ineffective after the first few patients had been studied. Infants were positioned supine with the head supported by a soft ring. Blood loss was estimated by the anesthesiologist, who administered crystalloid and blood products as needed.

Monitoring for VAE was planned with precordial Doppler and two-dimensional echocardiography

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(ECHO), but continuous Doppler was discontinued early in the study because of video interference from the Doppler signal. Continuous four-chamber real-time video recordings of the heart were performed using an echocardiographic imager (Cardiovue 100, Dasonics, Salt Lake) equipped with either a 5.0 or 7.5 M Hz transducer placed at the subxyphoid position. The operator was kept informed of all injections of drugs or infusions of volume. Visualized intracardiac reflectors following injection of drugs were deleted on film or documented as "injection artifact" by the operator. Videotapes were reviewed by pediatric cardiologists who were blinded to the operative course, together with the operating echocardiologist and the attending anesthesiologist. Intracardiac reflectors were diagnosed as VAE if they had the characteristic appearance of intravascular air and if no injections had been given prior to the sighting. VAE reflectors were treated according to established protocol. After presumptive diagnosis, the nitrous oxide was discontinued and the surgeon informed. If the reflectors stopped, if the patient remained stable, and, if the surgeon could not find a source of air, no other treatment was used and the nitrous oxide was resumed. Whenever reflectors persisted or the patient was hemodynamically unstable, 100% oxygen was administered and the surgeon was asked to pack the wound with saline-soaked sponges and to apply wax to open bone edges. Volume infusion or vasopressors were used to treat hypotension and the head was lowered if necessary. Statistical analysis of data was performed using unpaired *t* tests.

RESULTS

Craniectomies were performed for repair of craniosynostosis in 12 infants. The location of the surgical site is shown in table 1. Intravascular reflectors consistent with VAE were visualized in 66% (8/12) of the infants. Intracardiac reflectors were imaged either as small discrete groups of bubbles or as larger masses of bubbles which filled the cardiac chamber and "whited out" the right heart. Discrete bubbles passed through the heart of seven infants for up to 30 s without hemodynamic change (fig. 1). In some of the infants, this seemed to be correlated with the cranial osteotomy or hemorrhage; but, in others, they occurred without identifiable cause. No entry site for air was identified. One infant displayed a bolus pattern of intracardiac reflectors during a period of moderate blood loss. Premature arterial contractions, a nodal rhythm, and a 20 mmHg fall in blood pressure accompanied the sighting, but immediately responded to iv atropine. Reflectors continued to pass through the heart for 5 min before stopping. Extensive search of the surgical field revealed no obvious open vessels.

TABLE 1. Location of Synostosis and Embolism

Suture	VAE	No VAE	Total
Sagittal	5	1	6
Coronal	3	1	4
Metopic	0	2	2
Total	8	4	12

In one infant, ECHO identified an interseptal defect. This child had known tricuspid atresia with an atrial septal communication. No VAE was clearly demonstrated in this child, and no left-sided reflector was visualized in any of the other patients.

Table 2 depicts the characteristics of all patients compared by age, weight, blood loss, and fluid replacement. There were no statistically significant differences among age, weight, or intraoperative crystalloid replacement in the two groups. However, blood loss was significantly greater in the group without VAE.

DISCUSSION

The echocardiographic diagnosis of intracardiac reflectors consistent with VAE was made in 66% of infants undergoing craniectomy. This is higher than most reported incidences of VAE, and contrasts with the 14.6% reported by Albin among supine patients.³ Among seated children under 12 yr of age, Cucchiara⁶ reported a 33% incidence of VAE, while Katz⁷ reported a 60% incidence among seated dwarfs aged 4 months to 34 yr undergoing suboccipital craniotomy. We believe that the high incidence of VAE reported in this study is related to intraoperative ECHO imaging. Previous reports^{6,7} have relied heavily upon the Doppler diagnosis of VAE, but ECHO is a more sensitive monitor for

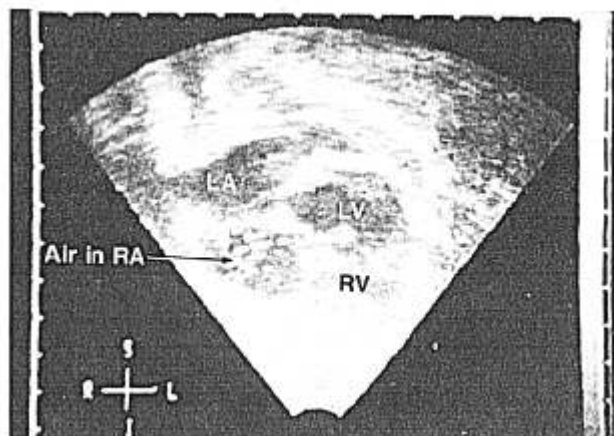


FIG. 1. Discrete bubbles in the right atrium.

TABLE 2. Profile of Patients

Age (Months)	Suture	Weight (kg)	Estimated Blood Loss (ml)	IV Fluid Administration	
				Blood (ml)	Crystalloid (ml)
With embolism					
1	Sagittal	4.7	90	80	265
2	Coronal	5.0	75	65	400
2	Sagittal	5.4	100	90	150
2.5	Coronal	6.1	80	120	130
3	Sagittal	6.5	60	115	240
4	Coronal	7.4	80	80	265
10	Sagittal	9.4	50	70	450
11	Sagittal	7.1	60	60	260
4.4 ± 1.3	Mean (±SD)	6.4 ± 1.5	74.3 ± 16.7	85 ± 22	270 ± 109
Without embolism					
2	Sagittal*	5.4	80	100	150
3.5	Coronal	6.8	250	330	310
8	Metopic	6.4	150	150	210
10	Metopic	7.3	120	100	265
5.6 ± 3.7	Mean (±SD)	6.3 ± 8	150 ± 72*	170 ± 109	233 ± 69

* Indicates statistically different from "with embolism" group ($P < 0.05$).

intravascular air. When positioned correctly over the right heart, Doppler can detect 0.24 ml/kg of intravascular air.⁹ In contrast, two studies of transesophageal ECHO have reported detection rates ranging from 0.02 mg/kg to 0.19 ml/kg.^{9,10} (While the imaging equipment employed in these studies was different from our model, we assume that sensitivities are similar and, therefore, superior to Doppler.) The Doppler diagnosis of VAE has other shortcomings. Sensitivity is affected by positioning over the right heart,¹¹ and sensitivity varies among different makes and models of Doppler (personal communication, Dr. Cucchiara).

ECHO has been shown to be extremely sensitive for intracardiac air, but it has shortcomings which limit its usefulness for clinical monitoring and research. The handheld transducer is bulky, is hard to position on the abdomen or chest without impeding venous return or causing pulmonary atelectasis, and requires the constant attention of a skilled operator. A pediatric transesophageal ECHO may alleviate some of these problems, although it could cause its own set of problems, such as vocal cord injury. The major drawback associated with ECHO is its lack of specificity. Spontaneously occurring echographic contrast has been reported on ECHO,¹² and bone marrow, fat, or microthrombi can all embolize to the heart and may be mistaken for VAE.¹³ Our prospective study of VAE in neonates and small infants encountered several technical difficulties. Most patients were too small to allow concurrent Doppler and ECHO monitoring, and data to confirm the diagnosis of VAE were not available from monitors with specificity for air embolism. End-tidal gas analysis could not be performed in every infant because of tech-

nical problems associated with the hospital's new mass spectrometer, and right atrial catheterization was performed only in the infant with the tricuspid atresia. In spite of these difficulties, we believe that the organized pattern of intravascular reflectors visualized on ECHO represent VAE. It is very unlikely that either the discrete reflectors or the larger mass of bolus reflectors traversing the heart represent spontaneous echographic contrast material. Likewise, we believe that communication between operating echocardiographer and anesthesiologist minimized the risk of mistaking injection microbubbles for VAE. However, it is virtually impossible for this study, or for most clinical studies of VAE, unequivocally to differentiate VAE from embolism resulting from other sources. Microthrombi or bone debris from surgery could have generated intracardiac turbulence, which ECHO would have misdiagnosed as air. Further study of this phenomenon is indicated.

Neonates and infants under the age of 1 yr may have a 66% incidence of intracardiac reflectors, because their anatomy and physiology make them more susceptible to VAE than older children or adults. All the children in this study had large, irregularly shaped heads, which, when positioned at surgery, lay from 5–10 cm above the level of the heart. Even normal infants have large heads in proportion to body size, and craniectomy is frequently associated with rapid hemorrhage. Relatively small quantities of blood represent a significant percentage of the blood volume of a small infant, and rapid blood loss could be accompanied by aspiration of air through small open sinusoids which are not visible from the operative field. However, as table 1 shows, total blood loss is not the critical variable with defines

infants at risk for VAE. Patients without VAE had twice the blood loss as those with VAE.

In summary, 66% of infants undergoing craniectomy in the supine position had echocardiographic evidence of embolism compatible with VAE. Only one child became symptomatic, and she responded promptly to iv atropine. No source for the air was identified in any child, and all children left the operating room in good condition. One child with tricuspid atresia had an interseptal defect, and no child had evidence of left-sided air. ECHO appears to be an extremely sensitive, but relatively nonspecific, monitor for VAE. Some of this lack of specificity was minimized by communication between echocardiographer and anesthesiologist. In spite of its shortcomings, ECHO offers valuable information about paradoxical embolism in infants with known or suspected interseptal defects.

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Ligneous Tracheobronchitis: An Unusual Cause of Airway Obstruction

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We recently were involved in the care of a child diagnosed with ligneous conjunctivitis, a rare illness of unknown etiology. The disease consists of growth of con-

junctival membranes having a fibrous or woody consistency, and has a variable course often requiring repeated surgical stripping of the membranes. Coexisting respiratory problems, especially frequent pneumonia in children, have often been noted. Our patient suffered from progressive airway obstruction associated with recurrent tracheobronchial growth of ligneous membranes. She required multiple anesthetics for stripping of both the conjunctival and tracheobronchial membranes. Management was frequently complicated by laryngospasm, bronchospasm, and tracheobronchial obstruction. Obstruction of distal airways may have occurred late in the course of her illness.

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