tance” testing. Usui andgi has demonstrated that slow injections of local anesthetic solutions elevate the pressure in the epidural space 10 to 60 cmH₂O. This rise in pressure was more marked in pregnant patients. A rapid injection of air, although more compressible than a liquid, may sharply elevate air pressure in the epidural space to several times the venous pressure. If an opening in the vein has been created during the puncture, a bolus of air may be forced into the vein. It is conceivable that a sharp elevation of pressure may in fact disrupt venules and allow air embolization.

A precordial Doppler device is capable of detecting very small emboli (0.1 ml), but cannot quantify the amount of air which enters the circulation. All the emboli we have noted were of brief duration and were clinically insignificant. Adornato noticed significant symptoms in dogs when air was infused at rates of approximately 0.5 ml·kg⁻¹·min⁻¹. It is unlikely that such volumes of air would be introduced during epidural cannulation. However, paradoxical embolization of small amounts of air into the systemic arterial circulation (e.g. via a patent foramen ovale) may produce organ infarction and serious sequelae. Epidural anesthesi is used in cases of congenital heart disease with right-to-left intracardiac shunts, which could allow ready passage of air from the venous to the systemic arterial circulation, and in these patients, particular care should be taken to avoid the possibility of air embolism. This may be accomplished by careful hydration, performance of the puncture in the right lateral decubitus position, and using a loss of resistance test with saline or local anesthetic rather than air.

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Anesthesiology
57:412–414, 1982

Neonatal Sepsis Presenting as Delayed Emergence from General Anesthesia

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Reasons for delayed emergence from general anesthesia in the newborn include hypothermia, metabolic disturbances, and severe iatrogenic causes such as an overdose with muscle relaxants or anesthetics. Delayed emergence from general anesthesia is an unique sign of neonatal sepsis which may include variations in temperature, apnea, metabolic acidosis, seizures, respiratory distress, and metabolic derangements. The following case report stresses the importance of considering neonatal sepsis as a factor contributing to delayed emergence in a previously healthy full-term infant.

REPORT OF A CASE

This was a 2,750-g 39-week gestation female infant born to a 31-year-old healthy woman after an uneventful pregnancy. Labor was complicated by prolonged rupture of membranes for 19 hours and arrest of labor. The infant was delivered by cesarean section with spinal anesthesia with an ope and five minute Apgar score of 2 and 7. With a FiO₂ of 1.0, ventilation as controlled via a mask and bag. Resuscitation was successful, and the infant was referred to our hospital

0003-0322/82/1100-0042 $00.95 © The American Society of Anesthesiologists, Inc.
for repair of a myelomeningocele. The gastric juices were negative for bacteria on Gram stain.

On arrival in the operating room, at 17 hours of age, the infant was breathing room air, and appeared to be in good health. She was active, pink, and crying with vital signs showing a heart rate (HR) of 140/min, respiratory rate (f) of 40/min, and a systolic blood pressure (BP) of 70 mmHg. We intubated the trachea while the infant was awake without difficulty and then anesthetized her with 1.0–1.5% halothane, N₂O, O₂, and air with no muscle relaxants. Vital signs remained stable during the 90 min of surgery with a rectal temperature of 36.7°C, HR 140–160/min, controlled respirations of 40–50/min, and BP of 50–70 mmHg. Just before skin incision, 75 mg oxacillin was given iv which is routine for our neurosurgical care.

At the conclusion of surgery, the halothane and N₂O were discontinued. Vital signs remained stable; however, the patient remained apneic and lethargic requiring controlled ventilation. The infant opened her eyes after 25 min, and we observed horizontal nystagmus; however, she remained apneic and the trachea remained intubated.

In the recovery room, she still required controlled ventilation. Blood (1.5 ml) shortly thereafter appeared in the endotracheal tube. Generalized tonic-clonic seizures followed and the infant remained lethargic, apneic, and with poor peripheral perfusion. Within a FTOH of 0.4, pH was 7.25, PaO₂ 177 mmHg and PaCO₂ 25 mmHg. Dextrotox revealed a blood glucose level of 180 mg/dl, prothrombin time 19.9 s (control 12.5 s), partial thromboplastin time 50.1 s (control 31.2 s), while white blood cell count of 8700 cells/mm³ (43% polys, 26% bands, 27% lymphs), platelets 264,000 cells/mm³, and blood cultures were sent. With a FTOH of 0.4, ventilation was controlled, f was 20/min, peak inspiratory pressure 20 cmH₂O, and positive end-expiratory pressure 5 cmH₂O. Phenobarbital, 5 mg/kg, iv, fresh frozen plasma, and a regimen of antibiotics that included ampicillin, gentamicin, and oxacillin were given.

A diagnosis of neonatal sepsis was made. A blood culture drawn in the recovery room subsequently grew Group B Streptococcus. Eventually, both gastric aspirate and an ear swab from the delivery room grew Group B Streptococcus, defining this as a case of "early onset" Group B Streptococcal sepsis.

The infant subsequently had required further therapy for seizure control, and eventually required a ventriculo-peritoneal shunt for hydrocephalus, but survived this septic episode.

DISCUSSION

If we specifically consider neonates that come to surgery who are less than 24 hours of age and become septic, "early onset" Group B Streptococcal infection should be highly suspected. The pathogenesis of Group B Streptococcal sepsis usually is associated with maternal or host risk factors. It has been estimated that as many as 25% of adult women carry Group B Streptococcus as an organism in their vaginal flora. During vaginal delivery, the newborn probably is colonized with Group B Streptococcus in the pharynx and on the skin. With the risk factor of PROM, as in our patient, an ascending pathway is present for the entrance of bacteria into the usually aseptic intrauterine environment, resulting in a colonized or infected newborn.

Sepsis neonatorum includes bacterial infection in the bloodstream with or without meningitis, and mortality rates of 20 to 75 per cent. There are many obstetric complications, including prolonged premature rupture of membranes that increase the risk of neonatal sepsis, but neonatal host-defense mechanisms have also been shown to be abnormal, further increasing neonatal susceptibility to various bacterial infections. Some of these abnormal defense mechanisms include decreased leukocyte bactericidal activity, inadequate supply of neutrophils, monocyte and neutrophil chemotaxis impairment, and a deficiency of C3 complement factor. The effect of anesthetics on the immune system may also be an additional risk factor predisposing the neonate to sepsis. Investigations have demonstrated decreased leukocyte motility and impaired extravascular leukocyte migration in animals who were exposed to bacterial infection while anesthetized with halothane. When reticuloendothelial system function under general anesthesia was examined in humans, significant reductions in phagocytosis rates were seen. The implications of clinical experience are uncertain, but some elements of immune system paralysis by anesthetics may be important, especially in an already immunologically compromised host such as the neonate.

The mechanism by which neonatal sepsis may delay emergence from general anesthesia is unclear as is the lethargy seen in the septic neonate not undergoing anesthesia. Sepsis may lead to a decreased cardiac output and, thereby, a slower fall in brain partial pressure of halothane on emergence. There also may be altered hepatic function in neonatal sepsis leading to decreased hepatic clearance of halothane, which may be significant during the "tail" of anesthetic recovery. In addition, sepsis-induced changes in the blood-brain barrier may result in an alteration in brain solubility or lead to increased interstitial diffusion in the brain between gray and white matter for the inhaled anesthetics. It is more likely that a combination of these factors could account for the delayed emergence observed in the septic newborn.

In summary, this case report brings to attention the consideration of neonatal sepsis as a factor for delayed emergence from general anesthesia in the full-term newborn. Specifically, "early onset" Group B Streptococcal sepsis should be highly suspected in the septic newborn under 24 hours of age. Anesthesiologists who care for newborns should be aware of the possibility that neonatal sepsis may account for an otherwise unexplained episode of delayed awakening from anesthesia.

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An Unusual Complication of Esophageal Obturateur Airway (EOA)

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The esophageal obturateur airway (EOA) is recognized as part of emergency airway treatment, especially by paramedical personnel outside the hospital. Improper utilization of the EOA may result in trauma to the soft tissues of the airway or esophagus. Inadequate oxygenation has occurred secondary to the airway being located in the trachea instead of the esophagus. Decreased ventilation has been reported with the EOA and its mask as compared with using an anesthesia mask and oropharyngeal airway.

We describe an unusual airway complication during the use of an EOA resulting in gastric placement of the device and difficulty with ventilation secondary to soft tissue obstruction.

REPORT OF A CASE

A 75-year-old man was admitted to the emergency department of the hospital with the diagnosis of cardiac arrest at home approximately 40 min prior to admission. Cardiopulmonary resuscitation (CPR) was started at home prior to the arrival of the paramedics. The diagnosis of ventricular fibrillation was followed by ventilation and insertion of an EOA with CPR being continued. Drugs and defibrillation were administered via radio command.

The ventilation was reported difficult due to poor face seals with the standard EOA mask. Therefore, a bag/mask unit of conventional type was applied over the EOA providing a better seal with the patient’s face. Upon arrival at the emergency department, the paramedic reported increased difficulty in ventilating the patient. An endotracheal tube was inserted without difficulty. The EOA was not observed except for the pilot tube, which protruded from the hypopharynx. Ventilation of the lungs then was accomplished successfully, as evidenced by bilateral breath sounds by auscultation.

A roentgenogram was obtained, and the tip of the endotracheal tube was in an appropriate position above the carina and within the trachea. The EOA was noticed with the tip located in the stomach below the diaphragm (fig. 1). The upper limit of the EOA was noted to be somewhere in the area immediately behind the larynx with the proximal end curving into the soft tissues on the left side of the esophagus and pharynx. The pilot tube was visible at the corner of the mouth, although the main body of the EOA was not visualized with the aid of a laryngoscope.

DISCUSSION

The esophageal obturateur airway has become an accepted part of airway management for paramedics throughout the United States. When used properly, it provides occlusion of the esophagus as well as access to the pharynx for ventilation. The esophageal occlusion is accomplished by a blind-ended tube with inflatable cuff which is inserted into the esophagus and the cuff inflated. A specially designed face mask is attached to the upper portion of the tube and latches into position over the esophageal obturator, preventing descent of the tube during ventilation.

Since the mask has a rigid plastic form with an inflatable plastic rim, effective seals with the face are nec-