In this case report, the authors describe the occurrence of venous air embolism during nasal irrigation following an adenoidectomy. A literature review is presented of surgical procedures in which venous air embolism has been documented. The means of diagnosis, the pathophysiology, and the recommended treatment of venous air embolus also are presented.

The clinical signs and symptoms of air embolism are often nonspecific. Because of this, the importance of knowing the patient's history and the clinical settings in which air embolism can occur cannot be overemphasized. Air embolism may mimic an acute myocardial infarction or a cerebral vascular accident, and unless one thinks “air embolism,” the diagnosis may be missed.1

Air may enter the systemic veins in sufficient quantity to produce serious embolic manifestations under a number of circumstances. These may be summarized as follows:2

A. Surgical
   1. Operations involving veins of the neck
   2. Operations involving dural sinuses
   3. Uterine curettage

B. Diagnostic air injections
   1. Into the perirenal area—(Diagnosis of adrenal tumors)
   2. Into the peritoneal cavity
      a. Direct
      b. Transuterine—(When comparing the veins of the body, the uterine sinuses are probably the most vulnerable to the entrance of air)
   3. Into the urinary bladder
   4. Into the joints

C. Therapeutic air injections
   1. Maxillary sinus lavage
   2. Pneumoperitoneum
   3. Vaginal insufflation

D. Obstetrical
   1. During delivery of patients with placenta previa and abortion

E. Accidental entrance of air into the intravenous apparatus

Venous air embolism can be produced any time air is forced into the body deliberately. Any therapeutic or operative procedure about the head and neck which affords the possibility of air entering a vein has the potential of being extremely dangerous. Death has occurred during lavage of the nasal sinuses,5,7,8 during tracheostomy, and during thyroidectomy. The focus of this article is to present a situation in which air embolism was not recognized immediately. The pathophysiology, means of early diagnosis, and recommended treatment of air embolism will be reviewed.
The case

A two-year-old male child presented to the otolaryngologist with persistent nasal obstruction and chronic nasal drainage which did not clear with medication. Preoperative anesthesia assessment revealed that he had a history of pyloric stenosis and asthma which did not require hospitalization. Medications included metoclopramide hydrochloride (Reglan®), Septra® and Dime-tapp®. Metoclopramide was utilized to relax the pyloric sphincter and increase gastric motility. General anesthesia was planned and discussed with the parents. Premedication consisted of APND solution. Each milliliter of APND contains atropine sulfate 0.048 mg, promethazine (Phenergan®) 1.85 mg, and chloral hydrate 60 mg. The usual oral dose of APND is 0.2 ml per pound. The child weighed 28 pounds, so he received 2.75 ml of APND solution.

Upon admission to the operating room, the child was somewhat agitated and fussy; sedation effects appeared minimal. Monitoring devices included electrocardiogram, blood pressure cuff, a stethoscope placed over the left precordium, an axillary temperature probe, and an oxygen concentration monitor. After a No. 23 butterfly was started in the left hand, 50 mg of sodium thio-pental followed by 25 mg of succinylcholine were administered. After the child was ventilated with 100% oxygen, a 4.5 mm uncuffed endotracheal tube was inserted without difficulty. Breath sounds were auscultated and determined to be bilaterally present and equal. The child was maintained on a mixture of 2 liters oxygen, 3 liters N₂O, and 1½ to 2⅔% halothane.

Initially, bilateral myringotomies were performed without incident. Next, cotton swabs with 4% cocaine were placed in both nares to shrink the large inferior turbinates. An adenoidectomy was then performed, and wet packings were placed for hemostasis. The packings were removed, and a 19-gauge spinal needle was inserted into the left maxillary sinus. It was flushed with 30 cc water and then 40 cc air.

At this point, the anesthetist noted a change in the cardiac sounds from the precordial stethoscope. The new sound was described as swooshing and gurgling. Approximately 30 seconds later, the child's color began to appear cyanotic, with a heart rate drop from 140 to 100 beats per minute and a systolic blood pressure drop from 120 to 70 torr. At this point the patient was placed on 100% oxygen with controlled ventilation. Proper placement of the endotracheal tube again was verified. The blood pressure became unobtainable, and the pulse rate was 90. Atropine 0.4 mg and metaraminol (Aramine®) 0.5 mg were given intravenously. A right femoral intravenous line was started with a 20 gauge plastic cannula. An arterial blood gas reading obtained from the femoral artery revealed a pH of 7.18, a pCO₂ of 45, a PO₂ of 161, and an HCO₃ of 17. A systolic blood pressure of 110 and a heart rate of 170 were obtained six minutes after the initial insult. Initially, 12.5 mEq of sodium bicarbonate was given intravenously. This was repeated three minutes later with a 12.5 mEq dose. A second arterial blood gas reading revealed a pH of 7.25, a pCO₂ of 41, a PO₂ of 111, and an HCO₃ of 21.

Thin, frothy, pink fluid was suctioned from the endotracheal tube as the child began to respond and move all extremities. Lidocaine (Xylocaine®) 15 mg was given intravenously to decrease the child's reaction to the endotracheal tube. The child was taken to the recovery room and placed on a T-tube with 40% humidified O₂. At this time, the child had spontaneous respirations of 32-40 per minute, a systolic blood pressure of 110, and a heart rate of 178. Breath sounds were clear to auscultation. Postoperatively, a chest x-ray, electrocardiogram, and echocardiogram were obtained in the postanesthesia room. Consultation by a cardiologist and a neurologist revealed no apparent deficits from the incident. The child was observed overnight in the hospital. He was discharged the following day with no apparent problems.

Discussion

There is disagreement in the literature regarding the injection of air into the maxillary antrum after irrigating it with fluid. A review of otolaryngology literature reveals that most investigators advise against this practice and warn of the danger of air embolism. Durant postulated that injury from venous air embolism is caused primarily by obstruction of blood flow from the right to the left side of the heart. Additionally, the air-blood interaction causes the development of a network composed of air bubbles and fibrin strands interspersed with aggregates of platelets, red blood cells, and globules of fat. Air bubbles become less frequent as the pulmonary arterial vessels constrict, and the remaining material contains mainly fibrin. The pathophysiological alterations resulting from this interaction of air bubbles with blood constituents include an increase in airway resistance, pulmonary edema, a decrease in lung compliance, pulmonary hypertension, hypoxia, and myocardial ischemia.
The amount of air and the rate of delivery are the most important factors affecting the severity of an injury produced by venous air emboli. Dogs have tolerated as much as 1000 cc of air injected in 100 ml increments every five to ten minutes.

The position of the patient is another factor to consider. The right ventricle labors against the obstruction of air trapped in the pulmonary outflow tract when the position of the experimental animal is such that this area is the most superior portion of his right heart. This is the case when the animal is on his back or right side. When turned to the left side, the outflow tract assumes a position inferior to the body of the right ventricle. The air bolus rises above the now inferior position of the pulmonary outflow tract and therefore presumably is churned into a froth which is gradually mixed with blood in the right ventricle. This process relieves the obstruction to the circulation as the froth gradually is dispersed and transported with the blood by the pulmonary artery to the lungs where excretion can take place.

The symptoms of venous air embolism are nonspecific. The awake patient may feel dizzy and faint, express fear of death, and complain of substernal chest pain and/or dyspnea. The only specific sign attributed to venous air embolism is the mill wheel murmur. Typically, it is a loud, churning sound audible over the entire precordium. The sound results from the heart beating against a bubble of air in the right ventricle. Because the mill wheel murmur is one of the earliest signs of a venous air embolus, it is mandatory for a precordial stethoscope to be used whenever the possibility of air embolism exists.

Other presenting signs include wheezing resulting from acute bronchospasm and systemic hypotension in conjunction with elevated right heart pressures. These usually are late signs seen with large emboli.

Diagnosis of venous air embolism is facilitated by monitoring pulmonary artery pressure (P_{PA}) and the fraction of end-tidal CO\(_2\) (F_{ET}CO\(_2\)) in each expired breath. A fall in the F_{ET}CO\(_2\) is observed with air embolization because of an increase in dead space with resultant high ventilation-perfusion mismatch areas in the lungs. The use of a Doppler flowmeter placed over the right heart area is essential to detect air entry into the circulation. Hybels indicated that the use of the Doppler is the most sensitive method available because it can detect small emboli rapidly. Aspiration of air from a well-positioned right atrial or pulmonary artery catheter confirms the diagnosis of air embolism. Further aspiration from the catheter can reduce the size of the air bolus. This will limit the severity of the insult.

The standard electrocardiogram may demonstrate changes resulting from myocardial ischemia. The first sign of ischemia is inversion of the T waves. As the ischemia continues, this finding is followed rapidly by the elevation of the ST segment. These findings then may lead into a right branch bundle block pattern. ST segment depression as well as ventricular fibrillation have been reported. It should be noted that changes seen in
the electrocardiogram usually represent a late sign of air embolism and are often nonspecific.\textsuperscript{6,11}

In the clinical situation, Alvarana warns that vigorous resuscitation should be started within three minutes following massive venous air embolism to avoid irreversible cardiac and cerebral damage.\textsuperscript{4} Supportive measures consisting of vaso-pressors and 100\% oxygen were the main therapy until 1947 when Durant suggested treatment with the left lateral position (Figure 2).\textsuperscript{2} However, it often is difficult to change the patient’s position when the anesthetist suspects an air embolism. Because of this, another technique that has gained wide acceptance is closed chest cardiac massage. The success of this technique probably is the result of forcing air from the pulmonary outflow tract into the pulmonary arterial circulation where it becomes fragmented into smaller air bubbles. These air bubbles no longer totally obstruct the right ventricular outflow tract of blood, because of the increase in the surface area of the pulmonary arterial tree beyond the main pulmonary arteries.\textsuperscript{4}

It has been demonstrated that gas from the air emboli trapped in the pulmonary vasculature can diffuse readily into the alveoli. Air emboli which are comprised of 80\% nitrogen will decrease in size more rapidly, if the amount of nitrogen in the alveolus can be decreased. This can be achieved by the administration of 100\% oxygen.\textsuperscript{1}

Conclusion
Venous air embolism can occur during a variety of clinical situations. The anesthetist should be aware of these situations and be familiar with instituting treatment for suspected air embolism. Knowledge, vigilance, proper monitoring, and prompt treatment are the best defenses in preventing the tragedy of an undiagnosed venous air embolism.

REFERENCES
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