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CASE REPORT

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Infant Death Due to Air Embolism from Peripheral Venous Infusion

ABSTRACT: An otherwise healthy male infant was brought to the hospital because the mother suspected superficial infection at the operative site 5 days after an inguinal hernia repair. He was admitted to the pediatric unit overnight to be evaluated by his surgeon the next morning. When a venous infusion of maintenance fluids was started, the patient immediately went into cardio-respiratory arrest and was pronounced dead after resuscitation efforts failed. Subsequently, air collections were found in both venous and arterial circulations, including the splenoportal system. Detailed review of the clinical presentation and course, laboratory results, radiological, and pathological findings, along with a review of pertinent literature provides an explanation for the death by air embolism. Apparent inconsistent findings both radiographically and at autopsy are resolved. The mechanism of distribution of air to both systemic and splenoportal circulation is discussed. We believe this to be only the eighth case reported in English-language literature of infantile death from peripheral venous infusion. In all age groups, we find only six other cases in the English-language literature of gas found concomitantly in both the systemic and portal venous systems.

KEYWORDS: forensic science, air embolism, infantile death from peripheral air, embolism, splenoportal air

Case Report

Clinical Course

Five days after being discharged following unilateral inguinal hernia repair, an 11-week-old male was reported by his mother to have some reddening at the herniorrhaphy site with minimal clear brownish drainage and a fever of 100.7°F. After a couple of telephone conversations with the surgeon over a period of several hours, she was instructed to bring the child to the Emergency Department where the infant was found to be normally feeding and voiding. Initial vital signs were temperature, 99.5°F; pulse rate, 185/min; respiratory rate, 32/min; and oxygen saturation, 99%. Cultures of the blood and the wound were obtained; these were negative for any growth at 5 days. Upon consultation with the on-call surgeon, who suspected superficial wound infection, the patient was administered 100 mg of Ancef® (GlaxoSmithKline, Research Triangle Park, NC) with a syringe pump through a T-connector attached to a 24-gauge angiocatheter in the vein on the dorsum of the right hand. He was admitted to the hospital so that the surgical site could be evaluated by his surgeon the next morning.

Shortly after midnight, the patient was assigned and taken to a room in the pediatric unit. An admission physical was performed, which revealed temperature, 99.1°F; pulse rate, 145; respiratory rate, 40; blood pressure 89/47; and weight 4.45 kg.

Maintenance fluids had been ordered by the admitting physician. Consequently, before entering the infant's room, the admitting nurse set up a Baxter Colleague infusion pump with a 1000 cm^3 premixed bag of D51/2 NS+20 mEq K+(Baxter), a three-port Baxter interlink infusion tubing, and a Braun Safeline Clip Lock cannula. No air-eliminating filters were used. The infant was alert, awake, and being held head-high in his mother's lap. The mother was seated in a chair at the foot of the crib. The bag was hung at the head of the crib. The infant began crying as the nurse flushed the T-connector with normal saline. Immediately thereafter, the nurse started the infusion pump and returned to the head of the crib to record the time. Meanwhile, the baby's cries had turned into screams; he then coughed or gasped loudly; his back arched and his arms stiffened. He lost consciousness. The nurse returned immediately to him, suspected cardio-respiratory arrest, and pushed the code-alarm button. Resuscitation efforts began with chest compressions and several minutes of ventilation by means of an AMBU bag and mask. Subsequently, endotracheal and orogastric tubes were introduced, and manual ventilation was continued. The infant's status did not improve. The Pediatric Advanced Life Support (PALS) resuscitation protocol was followed, which included chest compressions, and intravenous epinephrine, calcium chloride, atropine, and amiodarone were administered through the Angiocath connector (distal to the IV tubing). Defibrillation was performed on four occasions. A portable chest X-ray examination was performed to determine endotracheal tube placement (Fig. 1). There was no improvement in the infant's status, and after 59 min of resuscitation he was declared dead. At the conclusion of the resuscitation, the surgeon who had performed the hernia repair arrived, examined the surgical site, and opined that it appeared to be healing normally and did not appear infected. The Risk Management Director (RMD) was contacted immediately upon the infant's arrest. The RMD came to the hospital and sequestered the bag, tubing, and alligator clip. However, inspection of this setup revealed bubbles in the tubing according

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FIG. 1—*"Portable" chest film obtained with a mobile X-ray unit during resuscitation. The image is of poor quality, underexposed, and shows the endotracheal tube only to the level of the second ribs posteriorly. The tracheobronchial tree and spinal segments are ill-defined. Intravascular or intracardiac air is not apparent. There are wires and monitoring leads superimposed on the chest image.*

to subsequent testimony of the RMD. Subsequently, the IV setup was never made available to the medical examiner for testing of the safety alarm or to determine the length of the tubing and its volume capacity.

Within 3 h of death, the body was removed from the hospital floor to a refrigerated morgue. Except for a brief interval for postmortem radiologic examinations in the hospital's radiology department, the body remained refrigerated until autopsy.

Past Medical History

The infant was premature (33 weeks, 5 days gestational age). He was reported to have a right inguinal hernia and a systolic murmur. The newborn period was uneventful and the patient thrived. A preoperative cardiology evaluation was obtained 3 weeks before the patient's death and was entirely normal for an infant of this age, revealing an innocent murmur of normal pulmonary artery turbulence and a 2.19 mm closing patent foramen ovale as measured by transthoracic echocardiogram. The preoperative chest radiograph was normal.

Laboratory Findings

Hematology studies drawn in the Emergency Department were consistent with expectations for a 5-day postoperative patient, including a white blood cell count of 18,000/μL. Along with the collection of blood for determination of blood gases and chemistries during resuscitation, a blood culture was taken, which showed no growth at 5 days.

Radiological Findings

The supine frontal view of the chest obtained during the resuscitative efforts (Fig. 1) was of poor technical quality, being underexposed, and the endotrachial tube is not visible below the second thoracic vertebral level. The spinal elements are barely apparent through the heart and mediastinal structures. No air is seen in vascular structures or the heart. A minimal amount of left upper quadrant gas is seen, probably representing the splenic flexure.

The post-mortem radiographs taken 12 h after death (Fig. 2) show air in the pulmonary and systemic circulation as well as air in the portal venous system beneath the diaphragm. There are other poorly defined mottled shadows over the heart that are further elucidated by the subsequent computed tomography (CT)

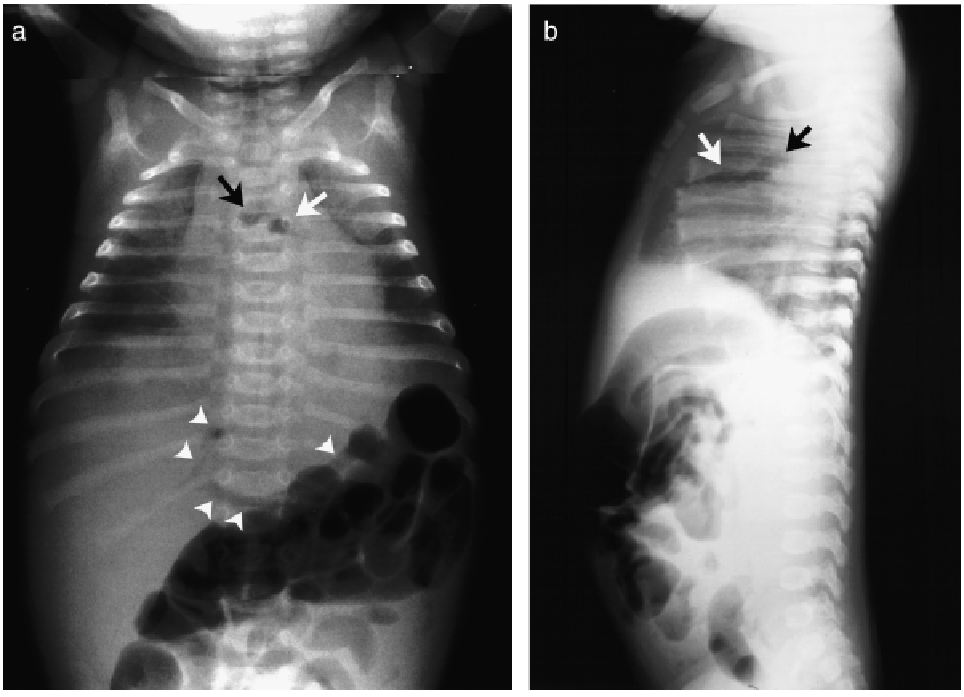

FIG. 2—*Post-mortem supine frontal (A) and lateral (B) radiographs of the chest obtained in the Radiology Department with fixed equipment. With no patient movement and no external manipulation, the frothy bubbles have begun to coalesce under gravitational influence in superior-most portions of vascular structures and cardiac cavities. The dark air collections are seen in the pulmonary artery (white arrows), the aortic arch (black arrows), and splenoportal venous complex (arrowheads). Considerable air is seen below the diaphragm in the stomach, small bowel, and colon.*

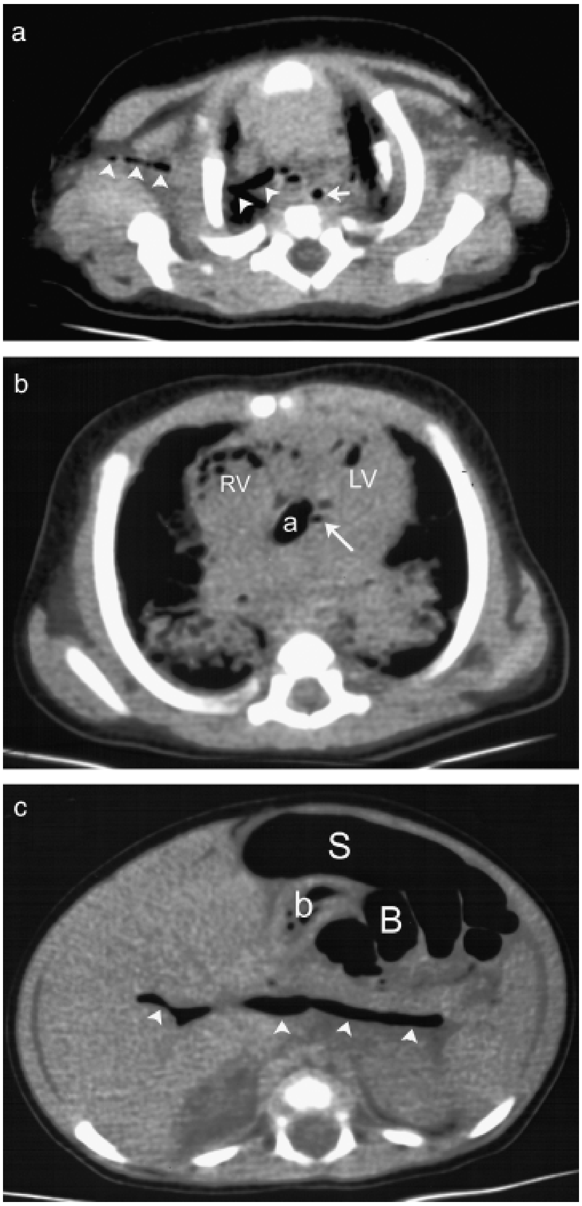

FIG. 3—*Post-mortem computed tomography of the chest and upper abdomen. In the axial images (A–C) (unlike diagnostic contrast-enhanced studies), the air contrast is discontinuous, making anatomic location of individual air collections more difficult. Representative selected image slices are shown. (Patient's right side is on viewer's left.) (A) Bony structures shown are at about the level of the manubrium sterni. The white arrow indicates the left vertebral artery, and the arrowheads follow the course of a right subclavian vein. The bulbous expansion at the most central arrowhead represents partial visualization of the superior vena cava. (B) Midcardiac level with air in both ventricles (RV and LV), aortic root (a) and probable origin of the left coronary artery (white arrow). The irregular-mottled densities posterior and lateral to the mediastinum represent post-mortem atelectasis in the lungs. (C) Level of the spenoportal veins (arrowheads) with air in the stomach (S) and large (B) and small (b) bowel loops.*

examination. A considerable amount of gas is present in the stomach and bowel.

CT of the chest was performed without contrast (Fig. 3). Axial sections enabled visualization of air in the right carotid artery, subclavian veins, left vertebral artery, the ascending aorta, and, probably, the origin of a left coronary artery. Bubbly air collections are seen in right and left ventricles. More peripheral collections at the margin of the heart shadow may represent either coronary vessels or epicardial veins. Axial views of the chest continued below the diaphragm demonstrate, again, air in the portal venous system, the stomach, and large and small bowel loops.

A CT of the brain (Fig. 4) revealed air in the left vertebral artery, bilateral carotid arteries and cavernous sinuses, venous sinuses, and probable subarachnoid air.

Additional chest radiographs taken at the Medical Examiners' office were of extremely poor quality, being poorly exposed, improperly processed, and marred by many handling artifacts. One could barely make out small collections of intravascular air greatly reduced in amount from earlier post-mortem studies.

It is important to note that on none of the radiological examinations was air or other gas found to be present in solid organs or superficial soft tissues.

Pathological Findings

Autopsy was performed by the Medical Examiner's office approximately 31 h after the infant's death. The examiner was not told of, or furnished, the venous access setup. Small amounts of blood-tinged fluid were present in the pleural cavities and pericardial sac, all felt to be attributable to the rigorous resuscitation efforts. A small amount of yellow–gray exudate and 21 cm^3 of fluid were present in the peritoneal cavity, believed to be attributable to the herniorrhaphy. Microscopic examination revealed no histopathological process. Drug screens were within normal limits. The foramen ovale was not probed, nor was the examination of the heart or major vessels performed under water. The cause of death was stated to be air embolism due to air introduced via the venous system.

Discussion

The distribution of intravascular gas in this case included both venous and arterial systems, right and left ventricles, and the splenoportal venous system. Venous air embolism in infants may occur in neurosurgical operations, cardiac surgery, epidural procedures, and umbilical vein catheterization (1–4). Documentation of air in the venous system following known or experimental introduction of air into arteries has implicated the cerebral circulation as a conduit for intravascular gas (5). There are fewer described causes of arterial gas embolism. In neonates receiving positive pressure ventilation, barotrauma can lead to systemic gas embolization. The pulmonary veins have been suggested as the point of entry of gas into the vasculature in such cases, allowing air to flow to the left heart and then to systemic arteries (6). Any systemic venous air may produce paradoxical arterial embolism by way of an intracardiac shunt or pulmonary arteriovenous malformation. Without a shunt pathway, venous air is usually dissipated or "filtered" in the pulmonary vasculature. However, if pressures are high, air can traverse the pulmonary vascular bed even without a demonstrable shunt (5,9,10). Precapillary arterial venous anastomoses have been described in many organs including the brain, liver, and spleen (5,9,10).

In this case, the peripheral venous infusion system was the avenue for air entry causing venous air embolism, and then paradoxical arterial air embolism. Clinical signs of significant venous air embolism include tachypnea and tachycardia. Right ventricular activity churns the mixture of air and blood into a bloody froth well known to forensic pathologists (5,11). Being compressible, the froth dampens the propulsive activity of ventricular contrac-

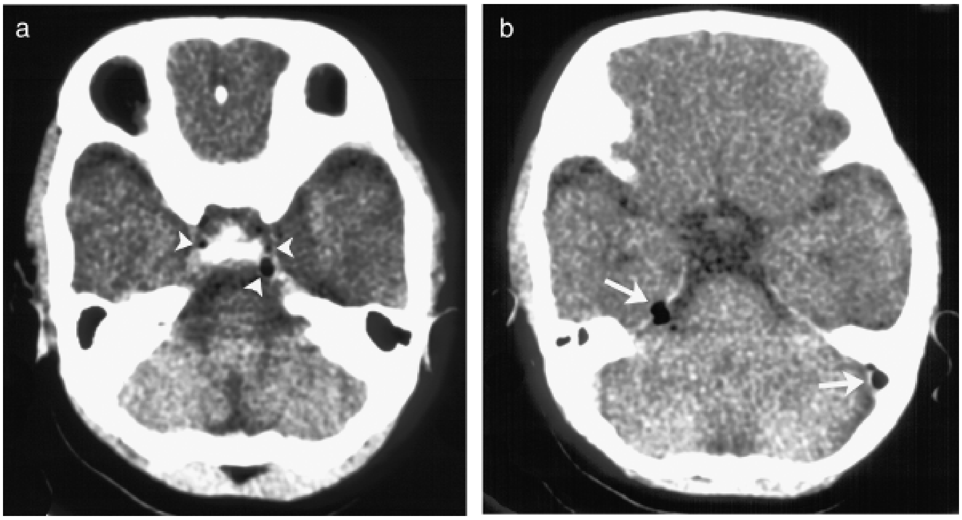

FIG. 4—*Post-mortem computed tomography of the head. (Patient's right side is on viewer's left.) (A) Level of the sella tursica. Air bubbles in the carotid arteries (bilaterally) and left cavernous sinus (arrowheads). (B) Level of the tentorium cerebelli. Air bubbles in venous sinuses (arrows).*

tion and can obstruct the right ventricular outflow tract. With obstruction of outflow, there is increased central venous pressure and decreased cardiac output with resultant hypotension and cyanosis. "Mill-wheel," or a rhythmic splashing, murmur has been described (12). Also described is a patient "gasp" with entry of air into the pulmonary circulation in both humans and experimental animal models (13,15). In young infants with a persistent patent ductus arteriosus or, as in this case, a patent foramen ovale, right-to-left shunting is enhanced by increased central venous pressure. Venous air becomes the more sinister systemic arterial emboli. All organs may be affected including the brain, kidneys, intestinal tract, and coronary arteries.

Signs of cerebral arterial gas embolus include seizure, focal neurological deficits, depressed consciousness, and lethal cardiac arrhythmias (16). In the case reported here, the infant appeared to gasp or cough seconds after the intravenous infusion began. Sudden convulsive straightening of the back and arms, that immediately preceded the cardiovascular collapse was consistent with seizure activity.

Air embolism in neonates and young infants due to peripheral intravenous infusion is exceedingly rare. We find only seven documented cases in the English-language literature (17–21). Only one of those involved a previously healthy infant as in this case (18). In adults, at least 54 cases of air embolism from peripheral IVs are found in the literature (22–28). Most of these patients (52 of 54) were asymptomatic, suggesting that venous air embolism can be well tolerated in adults. An infant with small organs, fewer alveoli, and a blood volume of about 1 L (80–90 mL/kg) cannot tolerate even tiny amounts of intravascular air. Symptomatic venous air embolism has been reported in infants with only 0.4 mL/kg introduced into the circulation (1). Moreover, the possibility of right-to-left shunting is much higher in neonates and infants when relative right and left atrial pressure differences are the lowest (29).

Finally, it has recently been shown that a shunt is not even necessary for venous gaseous emboli to result in intracranial systemic emboli. Air bubbles may rise in retrograde fashion against thoraco-cervical venous flow, depending on bubble size, central vein diameter, and cardiac output (30). Recall that the infant in the case presented was resting in the arms of his sitting mother, presumably in a head-high position.

In our case, intravascular or intracardiac air was not identified on the chest radiograph obtained during the resuscitation effort. However, this does not exclude the possibility, indeed the probability, that air was present within the vascular system although not detectable. Several factors may contribute to this dilemma. All collections of air may not be detected or may be obscured by normal overlying tissues. The exposure factors will contribute to this problem. The altered characteristics of air when it becomes frothy also change its visibility. Furthermore, collections of air or gas may be seen on CT studies that are not discernable on contemporary plain X-ray examination.

Movement or dispersion of vascular air also affects its visibility on radiographs. Dispersion of froth by chest compression moves the gas and creates smaller emboli. In one of Smith and Els' cases (17) of intracardiac air, "frothy/foamy" blood was withdrawn from the posterior tibial artery, showing that the froth can be widely dispersed by external manipulation. In this case, both chest compressions and mechanical ventilation were used, possibly lessening the damaging effects of air in the heart and lungs, but also making it less visible.

In our case, gas was also identified in the splenoportal venous system. Gas in this location was, for many years, considered an ominous sign and thought only to be due to ischemic bowel. In recent years, newer imaging modalities have enabled earlier detection of smaller collections of portomesenteric gas and this may improve the outcome in some cases (31,32). The most common factors eventuating in portomesenteric gas are intestinal wall alteration, bowel distention, and sepsis.

Shiotani et al. (33) showed a direct relationship between gastrointestinal distention and portal venous gas in 175 of 190 patients examined with postmortem CTs following cardiac arrest and cardiorespiratory resuscitation attempts. Our patient was ventilated by hand, at unknown pressures, with an AMBU bag, a likely cause of bowel distention.

Air embolism can be caused by the barotrauma of mechanical ventilation. However, in infantile cases of barotrauma, pulmonary interstitial emphysema, and/or pneumothorax are identified along with the catastrophic event. Neither was present in this case.

The finding of concomitant portal venous gas and systemic venous gas is rare. We can find only six cases on reviewing the English-language literature (34–38). None of these occurred in previously healthy infants. In one case of an infant with ischemic bowel, gas was actually seen on real-time ultrasonography to pass through the liver from portal to hepatic veins (37). A case of embolism death after laparoscopy delayed by air trapping in the portal circulation seems to show that air can pass from the portal system through the liver into the systemic venous circulation, i.e., the right heart (39). Edwards (40) writes that venous air embolism nearly always shows air in the liver on X-ray examination, presumably in hepatic veins. We know from experimental work years ago with carbon dioxide that there is easy egress of gas from the right atrium or inferior vena cava into the hepatic venous system (41). Can air go through the liver via hepatic veins to portal veins as well? Perhaps, but we have no indication of that in our case.

We surmise that ventilatory measures in our patient caused gastric distension, despite the orogastric tube, leading to portal venous gas. The stomach then decompressed post-mortem, revealing the portal gas, and thus explaining the lack of marked gastric distension on the post-mortem images.

The possibility that the gas observed on post-mortem images in the case reported here was a result of gas-forming organisms is refuted by no growth in blood cultures obtained before and after death. Putrefaction is dependent on temperature of the environment in which the body resides and on the antemortem state of health of the decedent; additionally, putrefaction develops more slowly in thin body types and in infants (42). After death, this formerly healthy infant's body was kept in a refrigerated morgue until autopsy except for a few minutes of post-mortem CT in the air-conditioned Radiology Department. Further, if putrefaction or production of gas by organisms existed, the amount of cardiovascular gas should have been increased on the film obtained in the medial examiner's office. Instead, the air was being resorbed and only small collections were still visible on this very bad radiograph.

Thus, this case is presented as one of tragic error: infant death by air embolism introduced through peripheral venous access with paradoxical systemic crossover. Venous air embolism may occur more often than is realized due to difficulty in diagnosis and the transient nature of clinical findings. It may not be identified at autopsy unless special care is taken to do so (43). In critically ill infants or in any patient under anesthesia, the symptoms may be completely undetected. In this previously healthy infant, clinical deterioration began immediately following an intravenous tubing change, and no other precipitating event explains the constellation of findings. Widely disseminated venous and arterial air collections in the thorax, abdomen, heart, and brain are the only radiographic or post-mortem abnormalities apart from agonal pulmonary edema.

"No medical tragedy is greater than the avoidable iatrogenic death. Perhaps saddest of all is the occurrence of venous air embolism, which is almost always the result of a therapeutic error or carelessness. . . .there is no instance in which a needle is placed in the venous system when the hazard of air embolism does not exist" (44).

References

1. Swartz N, Eisenkraft JB. Probable venous air embolism during epidural placement in an infant. Anesth Analg 1993;76:1136–8.
2. Erricsson JA, Gottlieb JD, Sweet RB. Closed-chest cardiac massage in the treatment of venous air embolism. N Engl J Med 1964;270:1353–4.
3. Hybels C. Venous air embolism in head and surgery. Laryngoscope 1980;90:946–54.
4. Toteishi H. Prospective study of air embolism. Br J Anaesth 1972;44: 1306–10.
5. Pate JW, Birdsong S. Carotid air embolism: an experimental study in cats. Arch Surg 1964;89:685.
6. Brown ZA, Clark JM, Jurg AL. Systemic gas embolus. A discussion of its pathogenesis in the neonate with a review of the literature. Am J Dis Chest 1977;131:994–1005.
7. Van Allen CM, Hrdina LS, Clark J. Air embolism from the pulmonary vein. Arch Surg 1929;19:567–99.
8. Menkin M, Swartzman RJ. Cerebral air embolism. Arch Neurol 1977;34: 168–70.
9. Hasegawa T, Ravens JR, Toole JF. Precapillary arteriovenous anastomoses: "thoroughfare channels" in the brain. Arch Neurol 1967;16: 217–24.
10. Fries CC, Levowitz B, Adler S, Cook AW, Karlson KE, Dennis C. Experimental cerebral gas embolism. Ann Surg 1957;145:461–9.
11. Larson CP. Venous air embolism: report of four cases. Suggested method of treatment. Am J Clin Path 1951;21:247–50.
12. Muth CM, Shank ES. Gas embolism. N Engl J Med 2000;342:476–82.
13. Adornato DC, Gildenberg PL, Ferrario CM, Smart J, Frost EAM. Pathophysiology of intravenous air embolism in dogs. Anesthesiology 1978;49: 120–7.
14. Laskey AL, Dyer C, Tobias JD. Venous air embolism during home infusion therapy. Pediatrics 2002;109(1):E15.
15. Maroon JC, Goodman JM, Horner TG, Campbell RL. Detection of minute venous air emboli with ultrasound. Surg Gynecol Obstet 1968; 157:1236–8.
16. Evans DE, Kobrine AI, Weathersby PK, Bradley ME. Cardiovascular effects of cerebral air embolism. Stroke 1981;12:334–9.
17. Smith J, Els I. Intracardiac air—the "hospital killer" identified? S Afr Med J 2003;93:922–7.
18. Levy I, Mosseri R, Garty B. Peripheral intravenous infusion—another cause of air embolism. Acta Pediatr 1996;85:385–6.
19. Wald M, Kirchner L, Lawrenz K, Amann G. Fatal air embolism in an extremely low birth weight infant: can it be caused by intravenous injections during resuscitations? Intensive Care Med 2003;29:630–3.
20. Willis J, Duncan C, Gottschalk S. Paraplegia due to peripheral venous air embolus in a neonate: a case report. Pediatrics 1981;67:472–3.
21. Peled N, Blaser SI, Moor A, Harwoo-Nash D. Computerized tomography appearance of accidental infusion of air into the venous sinuses. Pediatr Neurosurg 1991–92;17:251–3.
22. Susanti R IE, Rozans MH, Szerlip HM. Air embolism after intravenous injection of contrast material. Southern Med J 1999;92:930–3.
23. Groell R, Schaffler GJ, Rienmueller R. The peripheral intravenous cannula: a cause of venous air embolism. Am J Med Sci 1997;314: 300–2.
24. Abernathy CM, Dickinson TC. Massive air emboli from intravenous infusion pump: etiology and prevention. Am J Surg 1979;137:274–5.
25. Rubinstein D, Dangleis K, Thomas R. Venous air emboli identified on head and neck CT scans. J Comput Asst Tomogr 1996;20:559–62.
26. Woodring JH, Fried AM. Non-fatal venous air embolism after contrast-enhanced CT. Radiology 1988;167:405–7.
27. Adhikary GS, Massey SR. Massive air embolism: a case report. J Clin Anesth 1998;10:70–2.
28. Auroa T, Ward KR, Garza R, Rivers E. Iatrogenic venous air embolism. Am J Emerg Med 2000;18:255–6.
29. Chang AC, Wells W. Shunt lesions. In: Chung AC, Hanley FL, Wernovsky G, Wassell DL, editors. Pediatric cardiac intensive care. Philadelphia: Saunders, 2005:207–8.
30. Schlinp CJ, Loirimer T, Reiger M, Lederer W, Schmidts MB. The potential for venous air embolism ascending retrograde to the brain. J Forensic Sci 2005;50:906–9.
31. Sebastia C, Quiroga S, Espin E, Baye R, Alvarez-Castells A, Armengol M. Portomesenteric vein gas: pathologic mechanisms, CT findings, and prognosis. Radiographics 2000;20:1213–24.
32. Hou S-K, Chern C-H, How C-L, Chen J-D, Wang L-M, Lee C-H. Hepatic portal venous gas: clinical significance of computed tomography findings. Am J Emerg Med 2004;22:214–8.
33. Shiotani S, Kahno M, Ohashi N, Yamazaki K, Nakayama H, Watanabe K. Postmortem computed tomographic (PMCT) demonstration of relation between gastrointestinal (GI) distension and hepatic portal venous gas (HPVG). Radiation Med 2004;22:25–29.
34. Malin SW, Buthani VK, Ritchie WW, Hall ML, Paul D. Echogenic intravascular air and hepatic microbubbles associated with necrotizing enterocolitis. J Pediatr 1983;103:637–40.

35. Jones B. Massive gas embolism in *E. coli* septicemia. Gastrointest Radiol 1981;6:161–3.
36. Mallens WMC, Sckepers-Bok R, Nicolai JJ, Jacobs FAH, Heyerman HGM. Portal and systemic venous gas in a patient with cystic fibrosis: CT findings. Am J Radiol 1995;165:338–9.
37. Kreigshauser JS, Reading CC, King BF, Welch TJ. Combined systemic and portal venous gas: sonographic and CT detection in two cases. Am J Radiol 1990;154:1219–21.
38. Karaosmanoğlu D, Octar SÖ, Aruç M, Erba Ş. Portal and systemic venous gas in a patient after lumbar puncture. Br J Radiol 2005;78:767–9.
39. Root B, Levy MN, Pollack S, Lubert M, Pathak K. Gas embolism death after laparoscopy delayed by "trapping" in portal circulation. Anaesth Analg 1978;57:232–7.
40. Edwards DK III. The newborn infant with respiratory distress. In: Hilton SvW, Edwards DK III, editors. Practical pediatric radiology. 2nd ed. Philadelphia: Saunders, 1984:39.
41. Goldenberg DB, Brogdon BG. Hepatic venography with carbon dioxide. Maryland State Med J 1967;16:79–80.
42. Perper JA. Time of death and changes after death: part 1. Anatomical considerations. In: Spitz WU, Spitz DJ, editors. Medicolegal investigation of death. 4th ed.. Springfield, IL: CC Thomas, 2005:107–8.
43. Graham MA, Hutchins GM. Forensic pathology: pulmonary disease. Clinics Lab Med 1998;18:241–2.
44. Gottleib JD, Ercisson JA, Sweet RB. Venous air embolism. Anesth Analg 1965;44:773–9.

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