Primary Care

GAS EMBOLISM

CLAUS M. MUTH, M.D., AND ERIK S. SHANK, M.D.

GAS embolism, the entry of gas into vascular structures, is a largely iatrogenic clinical problem that can result in serious morbidity and even death.[1] Since gas embolism can result from procedures performed in almost all clinical specialties (Table 1), it is important for all clinicians to be aware of this problem. In most cases, gas embolism is air embolism, although the medical use of other gases, such as carbon dioxide, nitrous oxide, and nitrogen, can also result in the condition. There are two broad categories of gas embolism, venous and arterial, which are distinguished by the mechanism of gas entry and the site where the emboli ultimately lodge.

VENOUS GAS EMBOLISM

Venous gas embolism occurs when gas enters the systemic venous system.[2] The gas is transported to the lungs through the pulmonary arteries, causing interference with gas exchange, cardiac arrhythmias, pulmonary hypertension, right ventricular strain, and eventually cardiac failure. Physical preconditions for the entry of gas into the venous system are the incising of noncollapsed veins and the presence of subatmospheric pressure in these vessels. Noncollapsing veins include the epiploic veins, the emissary veins, and the dural venous sinuses. Air may enter these veins during neurosurgical operations, especially those performed with the patient in the sitting position.[3] The veins of the throat, and in some cases the veins in the coagulated operative field,[4] may also be entryways for air. Air may also enter veins through central venous and hemodialysis catheters[5,6] and may enter the veins of the myometrium during pregnancy and after delivery.[7,8]

From the Druckkammerzentrum Homburg, University Hospital Homburg, University of the Saarland, Homburg/Saar, Germany (C.M.M.); and the Department of Anesthesiology and Critical Care, Massachusetts General Hospital and the Department of Anesthesiology, Harvard Medical School — both in Boston (E.S.S.). Address reprint requests to Dr. Muth at the Druckkammerzentrum Homburg, Universitätskliniker des Saarlandes, 66424 Homburg/Saar, Germany, or at cmmuth@aol.com.

Pathophysiology

The most frequent form of venous gas embolism is the insidious venous aeroembolism, in which a series of gas bubbles resembling a string of pearls enters the venous system. Rapid entry or large volumes of gas put a strain on the right ventricle because of the migration of the emboli to the pulmonary circulation. The pulmonary arterial pressure increases, and the increased resistance to right ventricular outflow causes diminished pulmonary venous return. Because of the diminished pulmonary venous return, there is decreased left ventricular preload, resulting in diminished cardiac output and, ultimately, systemic cardiovascular collapse.[9] Tachyarrhythmias often develop, but bradycardias are possible as well. When large quantities of gas (over 50 ml) are injected abruptly, acute cor pulmonale, asystole, or both are likely to occur.[2] The alteration in the resistance of the lung vessels and the mismatch between ventilation and perfusion cause intrapulmonary right-to-left shunting and increased alveolar dead space, leading to arterial hypoxia and hypercapnia.

Diagnosis

To diagnose venous gas embolism, the physician should assess the clinical findings. The so-called millwheel murmur, a splashing auscultatory sound due to the presence of gas in the cardiac chambers and great vessels, is often present and can be auscultated by a precordial or esophageal stethoscope. A decrease in the end-tidal carbon dioxide levels, as determined by capnometry, suggests a change in the relation between ventilation and perfusion due to the obstruction of the pulmonary arteries.[10] Doppler ultrasonography is a sensitive and practical means of detecting intracardiac air, and it is often used during neurosurgical procedures,[3,11] procedures with the patient in the sitting position, and other procedures that entail a high risk of gas embolism. An even more sensitive and definitive method for detecting intracardiac gas is transesophageal echocardiography, although it requires training in performance and interpretation (Fig. 1).[2,3,12]

Treatment

When venous gas embolism is suspected, further entry of gas must be prevented. In certain cases, therapy with catecholamines is required, and, if necessary, aggressive cardiopulmonary resuscitation is performed. Adequate oxygenation is often possible only with an increase in the oxygen concentration of the inspired gas (up to 100 percent oxygen). Supplemental oxygen also reduces the size of the gas embolus

TABLE 1. MEDICAL SPECIALTIES WITH DOCUMENTED CASES OF GAS EMBOLISM.

SPECIALTY	MECHANISM OF GAS EMBOLISM
All medical specialties	Inadvertent entry of air through peripheral intravenous circuit
All surgical specialties	Intraoperative use of hydrogen peroxide, causing formation of arterial and venous oxygen emboli
Anesthesiology	Entry of air through disconnected intravascular catheter, inadvertent infusion of air through intravascular catheter
Cardiac surgery	Entry of air into extracorporeal-bypass pump circuit, incomplete removal of air from heart after cardioplegic arrest, carbon dioxide–assisted harvesting of peripheral veins
Cardiology	Entry of air through intravascular catheter during angiographic study or procedure
Critical care and pulmonology	Entry of air through disconnected intravascular catheter, pulmonary barotrauma, rupture of intraaortic balloon, entry of air into extracorporeal-membrane-oxygenator circuit
Diving and hyperbaric medicine	Pulmonary barotrauma, paradoxical embolism after decompression injury, entry of gas through disconnected intravascular catheter
Endoscopic and laparoscopic surgery	Entry of gas into veins or arteries during insufflation of body cavities
Gastroenterology	Entry of gas into veins during upper or lower endoscopy or endoscopic retrograde pancreatography
Neonatology and pediatrics	Pulmonary barotrauma in treatment of premature infants
Nephrology	Inadvertent entry of air through hemodialysis catheter and circuit on hemodialysis machine
Neurosurgery	Entry of air through incised veins and calvarial bone, especially during craniotomy with the patient in a sitting position
Obstetrics and gynecology	Cesarean section, gas insufflation into veins during endoscopic surgery, intravaginal and intrauterine gas insufflation during pregnancy
Otolaryngology	Nd:YAG laser surgery on the larynx, trachea, or bronchi*
Orthopedics	Gas insufflation into veins during arthroscopy, total hip arthroplasty, or spine surgery with the patient in a prone position
Radiology	Injection of air or gas as a contrast agent, inadvertent injection of air during angiography
Thoracic surgery	Entry of air into pulmonary vasculature during lung biopsy or video-assisted thoracoscopy, chest trauma (penetrating or blunt), lung transplantation
Urology	Entry of air during transurethral prostatectomy or radical prostatectomy
Vascular surgery	Entry of air during carotid endarterectomy

*Nd:YAG denotes neodymium:yttrium–aluminum–garnet.

by increasing the gradient for the egress of nitrogen from the bubble.[13] Rapid resuscitation with volume expansion is recommended to elevate venous pressure, thus preventing the continued entry of gas into the venous circulation.

Some authors recommend attempting to evacuate air from the right ventricle with the use of a central venous catheter (a multiorifice catheter may be more effective than one with a single lumen) or a pulmonary arterial catheter.[2,14,15] It may be possible to aspirate about 50 percent of the entrained air from an appropriately placed right atrial catheter,[2,14] but depending on the placement of the catheter and the position of the patient, a smaller effect is more likely.[2,15] Hyperbaric oxygen therapy is not a first-line treatment but may be a useful adjunct in severe cases. It should certainly be considered if there is evidence of neurologic changes. In this case, it should be assumed that a paradoxical embolism is present.

Paradoxical Embolism

A paradoxical embolism occurs when air or gas that has entered the venous circulation manages to enter the systemic arterial circulation and causes symptoms of end-artery obstruction. There are a number of mechanisms by which this can occur. One is the passage of gas across a patent foramen ovale into the systemic circulation. A patent foramen ovale, which is detectable in about 30 percent of the general population, makes possible right-to-left shunting of gas bubbles.[16] If there is a patent foramen ovale and if the pressure in the right atrium exceeds the pressure in the left atrium, right-to-left flow through the foramen ovale may occur.[17] Elevated pulmonary arterial pressure due to venous gas embolism may result in elevated right atrial pressure, making it possible for a bubble to be transported through a patent foramen. Furthermore, the decrease in left atrial pressure caused by controlled ventilation and the use of positive end-expiratory pressure may create a pressure gradient across the patent foramen ovale, favoring the passage of gas into the systemic circulation.[2,3]

In other situations, venous gas may enter the arterial circulation by overwhelming the mechanisms that normally prevent arterial gas embolism. Studies in animals suggest that either a large bolus of gas (20 ml or more) or small continuous amounts (11 ml per minute) introduced into the venous system may
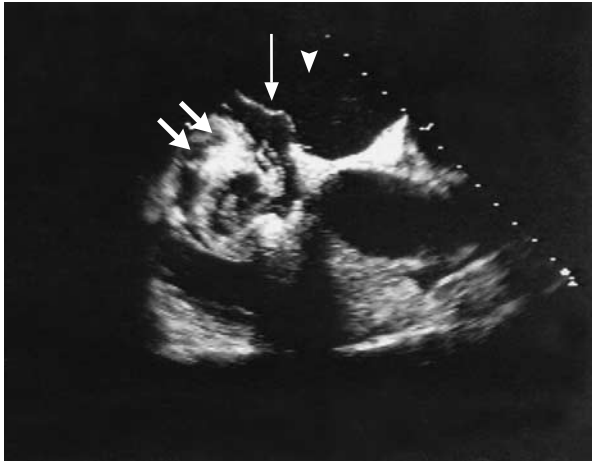
Figure 1. Transesophageal Echocardiogram from a Patient Evaluated for the Presence of a Patent Foramen Ovale.

Saline was agitated and injected rapidly into a central venous catheter. The bubbles appear as echodense areas in the right atrium (double arrows). If this patient had a patent foramen ovale, bubbles would be seen crossing the interatrial septum (thin arrow) and entering the left atrium (arrowhead). (Echocardiogram provided courtesy of S. Streckenbach.)

generate intraarterial bubbles.[18-20] There have been reports of fatal cerebral arterial gas embolism caused by a large venous gas embolus, although no intracardiac defects or shunt mechanisms could be demonstrated.[21] Various anesthetic agents diminish the ability of the pulmonary circulation to filter out gas emboli.[22] Studies in animals have shown that volatile anesthetics, specifically, may raise the threshold for a spillover of venous bubbles into systemic arteries. This finding may have relevance to surgical procedures that carry a substantial risk of venous gas embolism.

The treatment of paradoxical embolism is identical to that of primary arterial gas embolism (discussed below). Every venous gas embolism has the potential to evolve into an arterial gas embolism.

ARTERIAL GAS EMBOLISM

Arterial gas embolism is caused by the entry of gas into the pulmonary veins or directly into the arteries of the systemic circulation. Gas may enter the arteries as a result of overexpansion of the lung by decompression barotrauma or as a result of paradoxical embolism. Any cardiac surgical operation that uses extracorporeal bypass may also cause arterial gas embolism.[23] Even if only small amounts of gas enter the arterial system, the flow of gas bubbles into functional end arteries occludes these vessels. Although obstruction is possible in any artery, obstruction of either the coronary arteries or the nutritive arteries of the brain (cerebral arterial gas embolism) is especially serious and may be fatal because of the vulnerability of the heart and brain to short periods of hypoxia.[24]

Pathophysiology

The entry of gas into the aorta causes the distribution of gas bubbles into nearly all organs. Small emboli in the vessels of the skeletal muscles or viscera are well tolerated, but embolization to the cerebral or coronary circulation may result in severe morbidity or death.

Embolization into the coronary arteries induces electrocardiographic changes typical of ischemia and infarction; dysrhythmias, myocardial suppression, cardiac failure, and cardiac arrest are all possible, depending on the amount of gas embolized.[25] Circulatory responses may also be seen with embolization to the cerebral vessels.[26]

Cerebral arterial gas embolization typically involves the migration of gas to small arteries (average diameter, 30 to 60 μm).[25] The emboli cause pathologic changes by two mechanisms: a reduction in perfusion distal to the obstruction and an inflammatory response to the bubble (Fig. 2).[24]

Symptoms

The symptoms of cerebral arterial gas embolism develop suddenly. The clinical presentation, however, is determined by the absolute quantity of gas and the areas of the brain that are affected. Thus, there may be minor motor weakness and headache or moderate confusion; conversely, complete disorientation, hemiparesis, convulsions, loss of consciousness, and coma may occur.[27] Asymmetry of the pupils, hemianopia, and impairment of the respiratory and circulatory centers (manifested as bradypnea, Cheyne–Stokes breathing, cardiac arrhythmias, and circulatory failure)[25,26] are other well-known complications. In patients who have undergone surgical procedures that carry a risk of gas embolism, a delayed recovery from general anesthesia or a transitional stage of impaired consciousness may be a clue to the occurrence of cerebral arterial gas embolism. The diagnosis is not easy to establish in such patients, because complications of anesthesia, such as the central anticholinergic syndrome or the presence of residual anesthetic or muscle relaxant, can mimic mild cerebral arterial gas embolism.

Diagnosis

The most important diagnostic criterion is the patient's history, because the clinical suspicion of embolism is based on the initial neurologic symptoms and the direct temporal relation between these symptoms and the performance of an invasive procedure. The procedures that carry the greatest risk of venous or arterial gas embolism are craniotomy performed with the patient in the sitting position, cesarean section, hip replacement, and cardiac surgery with cardiopulmonary bypass. All these procedures have in common an incised vascular bed and a hydrostatic gradient favoring the intravascular entry of gas.

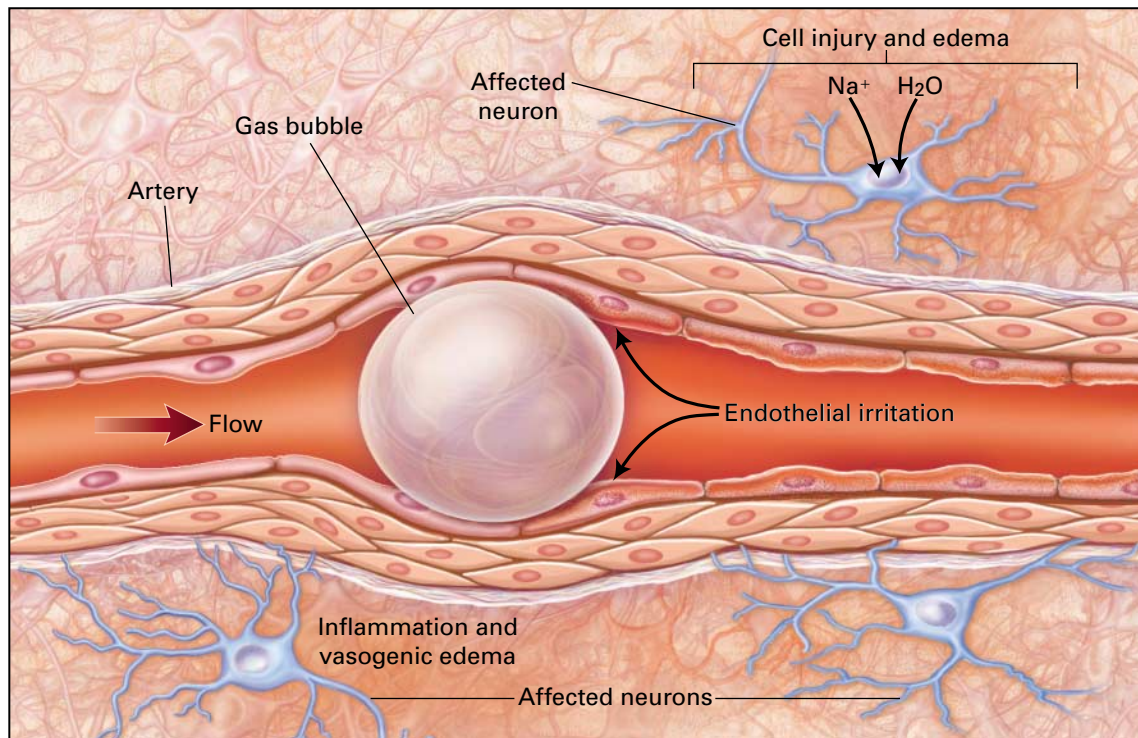Cerebral arterial gas embolism can sometimes be

Figure 2. Bubble Obstructing End-Arterial Flow in a Cerebral Vessel with a Diameter of 30 to 60 μm, Causing Distal Ischemia.
The obstruction causes the metabolic processes of neurons to fail. Sodium and water enter the vessel, and cytotoxic edema develops. The surface of the bubble generates a foreign-body response through cellular and humoral immune mechanisms. The bubble also mechanically irritates the arterial endothelium. Both processes result in vasogenic edema and greater impairment of perfusion. The neuronal injury extends beyond the area of obstruction.

distinguished from a cerebral infarct or intracerebral bleeding on a computed tomographic (CT) scan.[28] However, pathologic changes are sometimes very subtle and not well visualized on CT, and the diagnosis of cerebral arterial gas embolism must be considered early. Magnetic resonance imaging of the cerebrum can sometimes show an increased volume of water concentrated in the injured tissue. But this method is also not reliable and may fail to detect an embolism if the symptoms are mild.[24]

Gas bubbles in the vessels of the retina can occasionally be identified, but their absence does not rule out gas embolism.[24] Another finding that is nonspecific but that has been described in a number of cases is hemoconcentration with an increase in the hematocrit, possibly as a direct consequence of the extravascular shift of fluid into the injured tissues.[29]

Treatment

The primary goal of treatment is the protection and maintenance of vital functions. If necessary, cardiopulmonary resuscitation should be performed, since not only venous but also primary arterial gas embolism may lead to serious impairment of the cardiovascular system. Endotracheal intubation should be performed in a somnolent or comatose patient in order to maintain adequate oxygenation and ventilation. Oxygen should also be administered, at as high a concentration as possible.[30] Administration of oxygen is important not only to treat hypoxia and hypoxemia but also to eliminate the gas in the bubbles by establishing a diffusion gradient that favors the egress of gas from the bubbles.[13,24]

It is currently recommended that patients with arterial gas embolism be placed in the flat supine position.[31] The buoyancy of gas bubbles is not sufficient to counteract blood flow propelling such bubbles toward the head, even when the patient is placed in a head-down position. In addition, the head-down position may aggravate the cerebral edema that develops in these patients.[32]

Treatment of Generalized Seizures

Cerebral gas embolism often causes generalized seizures,[27] which may not respond to benzodiazepines. In these cases, it is recommended that the seizures be suppressed with barbiturates.[27,33] Although there is no proof that barbiturates provide cerebral

protection after cerebral ischemia, the use of barbiturates for ischemic brain lesions has certain advantages. They reduce cerebral oxygen consumption, intracranial pressure, the production of free radicals, and the release of endogenous catecholamines.[34,35] High doses of barbiturates depress respiration; ventilatory support must therefore be available when a patient is given barbiturates.

Hyperbaric-Oxygen Therapy

With hyperbaric-oxygen therapy, the patient breathes 100 percent oxygen at a pressure above that of the atmosphere at sea level. This therapy decreases the size of the gas bubble both by raising the ambient pressure (Fig. 3) and by causing systemic hyperoxia. An arterial partial pressure of oxygen greater than 2000 mm Hg is frequently achieved. The hyperoxia produces enormous diffusion gradients for oxygen into the bubble and for nitrogen out of the bubble.[31,32] The hyperoxia also allows much larger quantities of oxygen to be dissolved in the plasma and increases the extent of oxygen diffusion in tissues.[27] The improvements in the oxygen-carrying capacity of plasma and in the delivery of oxygen to tissues offset the embolic insult to the microvasculature.

Other benefits of hyperbaric oxygen have been proposed. It may help prevent cerebral edema by reducing the permeability of blood vessels while supporting the integrity of the blood–brain barrier.[36,37] Furthermore, experiments have suggested that hyperbaric oxygen diminishes the adherence of leukocytes to damaged endothelium.[38]

These benefits suggest that all patients with clinical symptoms of arterial gas embolism should receive recompression treatment with hyperbaric oxygen. Although immediate recompression produces the best response,[23] delayed treatment in a hyperbaric chamber may still be indicated to ameliorate the patient's condition.[39] Hyperbaric oxygen is the first-line treatment of choice for arterial gas embolism.[23,25,31,40] Thus, as soon as cardiopulmonary stabilization has been achieved, the patient should be transferred to a hyperbaric chamber.

Infusion Therapy

There is some evidence that gas embolism may cause hemoconcentration, which increases blood viscosity and impairs the already compromised microcirculation.[29] Therefore, normovolemia should be achieved to optimize the microcirculation. Hypovolemia is always tolerated less well than relative anemia. It is therefore acceptable to decrease the hematocrit, within certain limits. In animals, moderate hemodilution to a hematocrit of 30 percent reduces neurologic damage.[41] Colloid solutions are preferable to crystalloid solutions for hemodilution, because the latter may promote cerebral edema. Hypertonic solutions (e.g., 7.5 percent sodium chloride solution)
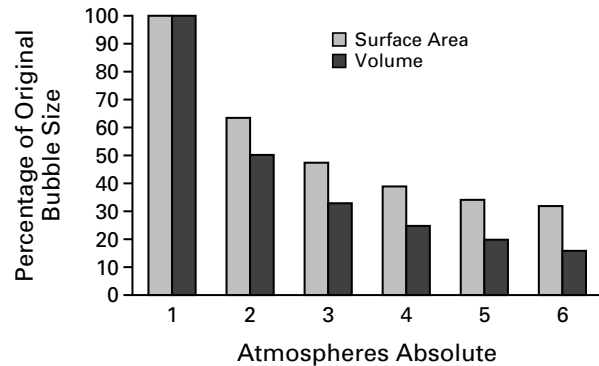

Figure 3. Relation between the Size of the Bubble and Pressure. The surface area and volume of the gas bubble are inversely proportional to pressure at a constant temperature (Boyle's law). Thus, as the patient is exposed to increasing ambient pressure, the gas bubbles shrink.

may become the first choice for volume replacement, but their use is still controversial and therefore cannot yet be recommended.

The goal of infusion therapy is normovolemia. Placement of a central venous catheter is strongly recommended to assess central venous pressure, which should be maintained at approximately 12 mm Hg. As a further method of ensuring adequate volume status, the urinary output should be monitored with a Foley catheter and maintained above 1 to 2 ml per kilogram of body weight per hour.

Anticoagulant Therapy

There is evidence that heparin may be beneficial in the treatment of gas embolism.[42] Studies have shown that the clinical course of arterial gas embolism is less severe if the patient has been treated with heparin before the embolic event occurs. An argument against heparin therapy is the risk it entails of hemorrhage into the infarcted tissue. At present, the use of heparin for the short-term treatment of cerebral arterial gas embolism is not generally recommended.

Corticosteroid Therapy

The use of corticosteroids in patients with arterial gas embolism remains controversial. Some authors recommend treatment with corticosteroids to combat the brain edema[43] that results from gas embolization in the cerebral arteries. Cerebral gas embolism initially induces the rapid development of cytotoxic brain edema, with diminished extracellular space and enlarged intracellular areas. This form of edema does not usually respond to corticosteroids.[44] Some authors report that corticosteroids aggravate ischemic injury after occlusion of the vessels.[45,46] Thus, since corticosteroids appear to offer no benefit in patients with cytotoxic edema and since these drugs may aggravate neuronal ischemic injury, we do not recommend them.

TABLE 2. TREATMENT OF GAS EMBOLISM.

TYPE OF TREATMENT	VENOUS GAS EMBOLISM	ARTERIAL GAS EMBOLISM
Prevention of further entry of gas	Measures to increase venous pressure (e.g., Valsalva maneuver or intravenous administration of fluids) Identification and shutting down of entryway for gas	Identification and shutting down of entryway for gas
Primary therapy	Supportive	Hyperbaric oxygen therapy as soon as patient's condition is stable enough for transfer to hyperbaric chamber
Supportive therapy	Oxygen, intravascular volume expansion, catecholamines	Oxygen, intravascular volume expansion, catecholamines
Positioning of patient	Supine, flat	Supine, flat
Evacuation of embolized gas	Aspiration with multiluminal central venous catheter (with patient in left lateral decubitus position)	Hyperbaric oxygen
Adjunctive therapy	Hyperbaric oxygen	Lidocaine, antiepileptic agents, physical therapy

---

Lidocaine Therapy

Although the results of clinical studies of lidocaine for the treatment of arterial gas embolism are not yet available, studies in animals suggest that lidocaine may be beneficial.[47-51] In animals given prophylactic doses of lidocaine, the depressant effects of gas embolism on somatosensory evoked potentials and the elevations in intracranial pressure caused by gas embolism were both reduced. In a clinical trial, lidocaine provided cerebral protection during cardiac surgery.[51] Therefore, a strong argument can be made for administering lidocaine in a bolus dose of 1.5 mg per kilogram and maintaining a therapeutic concentration, with continuous intravenous administration in patients with severe arterial gas embolism. However, an overdose of lidocaine may cause central nervous system depression, cerebral convulsions, and bradyarrhythmias.

CONCLUSIONS

The entry of gas into the venous or arterial system is a risk in virtually all areas of clinical care. Venous emboli may lead to cardiovascular collapse or to paradoxical arterial emboli. Arterial emboli may occlude end arteries throughout the body and may cause serious morbidity or death if they occlude cardiac or cerebral vessels.

Regardless of the mechanism responsible for the embolism, rapid and aggressive treatment is essential to preserve life and function (Table 2). For venous gas embolism, the mainstays of treatment are the prevention of further entry of gas, volume expansion, the administration of 100 percent oxygen, often with ventilatory support; positive inotropic support; and cardiopulmonary resuscitation, if necessary. For arterial gas embolism, hyperbaric oxygen is the treatment of choice, as soon as cardiopulmonary stabilization has been achieved.

REFERENCES

1. Murphy BP, Harford FJ, Cramer FS. Cerebral air embolism resulting from invasive medical procedures. Ann Surg 1985;201:242-5.

2. Palmon SC, Moore LE, Lundberg J, Toung T. Venous air embolism: a review. J Clin Anesth 1997;9:251-7.
3. Porter JM, Pidgeon C, Cunningham AJ. The sitting position in neurosurgery: a critical appraisal. Br J Anaesth 1999;82:117-28.
4. Donlon JV Jr. Anesthesia and eye, ear, nose, and throat surgery. In: Miller RD, ed. Anesthesia. 3rd ed. Vol. 2. New York: Churchill Livingstone, 1990:2001-23.
5. Halliday P, Anderson DN, Davidson AI, Page JG. Management of cerebral air embolism secondary to a disconnected central venous catheter. Br J Surg 1994;81:71.
6. Yu AS, Levy E. Paradoxical cerebral air embolism from a hemodialysis catheter. Am J Kidney Dis 1997;29:453-5.
7. Hill BF, Jones JS. Venous air embolism following orogenital sex during pregnancy. Am J Emerg Med 1993;11:155-7.
8. Weissman A, Kol S, Peretz BA. Gas embolism in obstetrics and gynecology: a review. J Reprod Med 1996;41:103-11.
9. Durant TM, Long J, Oppenheimer MJ. Pulmonary (venous) air embolism. Am Heart J 1947;33:269-81.
10. Shapiro HM, Drummond JC. Neurosurgical anesthesia and intracranial hypertension. In: Miller RD, ed. Anesthesia. 3rd ed. Vol. 2. New York: Churchill Livingstone, 1990:1737-89.
11. Gildenberg PL, O'Brien RP, Britt WJ, Frost EA. The efficacy of Doppler monitoring for the detection of venous air embolism. J Neurosurg 1981;54:75-8.
12. Mammoto T, Hayashi Y, Ohnishi Y, Kuro M. Incidence of venous and paradoxical air embolism in neurosurgical patients in the sitting position: detection by transesophageal echocardiography. Acta Anaesthesiol Scand 1998;42:643-7.
13. Van Liew HD, Conkin J, Burkard ME. The oxygen window and decompression bubbles: estimates and significance. Aviat Space Environ Med 1993;64:859-65.
14. De Angelis J. A simple and rapid method for evacuation of embolized air. Anesthesiology 1975;43:110-1.
15. Albin MS. Air embolism. In: Albin MS, ed. Textbook of neuroanesthesia with neurosurgical and neuroscience perspectives. New York: McGraw-Hill, 1997:1009-25.
16. Lynch JJ, Schuchard GH, Gross CM, Wann LS. Prevalence of right-to-left atrial shunting in a healthy population: detection by Valsalva maneuver contrast echocardiography. Am J Cardiol 1984;53:1478-80.
17. Gronert GA, Messick JM Jr, Cucchiara RF, Michenfelder JD. Paradoxical air embolism from a patent foramen ovale. Anesthesiology 1979;50:548-9.
18. Butler BD, Hills BA. The lung as a filter for microbubbles. J Appl Physiol 1979;47:537-43.
19. Vik A, Brubakk AO, Hennessy TR, Jenssen BM, Ekker M, Slørdahl SA. Venous air embolism in swine: transport of gas bubbles through the pulmonary circulation. J Appl Physiol 1990;69:237-44.
20. Spencer MP, Oyama Y. Pulmonary capacity for dissipation of venous gas emboli. Aerosp Med 1971;42:822-7.
21. Tommasino C, Rizzardi R, Beretta L, Venturino M, Piccoli S. Cerebral ischemia after venous air embolism in the absence of intracardiac defects. J Neurosurg Anesthesiol 1996;8:30-4.
22. Katz J, Leiman BC, Butler BD. Effects of inhalation anaesthetics on filtration of venous gas emboli by the pulmonary vasculature. Br J Anaesth 1988;61:200-5.
23. Ziser A, Adir Y, Lavon H, Shupak A. Hyperbaric oxygen therapy for

massive arterial air embolism during cardiac operations. J Thorac Cardiovasc Surg 1999;117:818-21.

24. Moon RE. Gas embolism. In: Oriani G, Marroni A, Wattel F, eds. Handbook on hyperbaric medicine. Milan, Italy: Springer, 1996:229-48.

25. Dutka AJ. A review of the pathophysiology and potential application of experimental therapies for cerebral ischemia to the treatment of cerebral arterial gas embolism. Undersea Biomed Res 1985;12:403-21.

26. Evans DE, Kobrine AI, Weathersby PK, Bradley ME. Cardiovascular effects of cerebral air embolism. Stroke 1981;12:338-44.

27. Tovar EA, Del Campo C, Borsari A, Webb RP, Dell JR, Weinstein PB. Postoperative management of cerebral air embolism: gas physiology for surgeons. Ann Thorac Surg 1995;60:1138-42.

28. Voorhies RM, Fraser RAR. Cerebral air embolism occurring at angiography and diagnosed by computerized tomography. J Neurosurg 1984; 60:177-8.

29. Smith RM, Van Hoesen KB, Neuman TS. Arterial gas embolism and hemoconcentration. J Emerg Med 1994;12:147-53.

30. Annane D, Troché G, Delisle F, et al. Effects of mechanical ventilation with normobaric oxygen therapy on the rate of air removal from cerebral arteries. Crit Care Med 1994;22:851-7.

31. Workshop Panel. Final summary of recommendations: diving accident workshop 1990. In: Bennett PB, Moon RE, eds. Diving accident management. Bethesda, Md.: Undersea and Hyperbaric Medical Society, 1990:366-9.

32. Moon RE, Dear GL, Stolp BW. Treatment of decompression illness and iatrogenic gas embolism. Respir Care Clin North Am 1999;5:93-135.

33. Bleck TP. Management approaches to prolonged seizures and status epilepticus. Epilepsia 1999;40:Suppl 1:S59-S66.

34. Patel PM, Drummond JC, Cole DJ, Kelly PJ, Watson M. Isoflurane and pentobarbital reduce the frequency of transient ischemic depolarizations during focal ischemia in rats. Anesth Analg 1998;86:773-80.

35. Hoffman WE, Charbel FT, Edelman G, Ausman JI. Thiopental and desflurane treatment for brain protection. Neurosurgery 1998;43:1050-3.

36. Miller JD, Ledingham IM, Jennett WB. Effects of hyperbaric oxygen on intracranial pressure and cerebral blood flow in experimental cerebral oedema. J Neurol Neurosurg Psychiatry 1970;33:745-55.

37. Mink RB, Dutka AJ. Hyperbaric oxygen after global cerebral ischemia in rabbits reduces brain vascular permeability and blood flow. Stroke 1995; 26:2307-12.

38. Thom SR, Mendiguren I, Hardy K, et al. Inhibition of human neutrophil beta2-integrin-dependent adherence by hyperbaric O2. Am J Physiol 1997;272:C770-C777.

39. Dexter F, Hindman BJ. Recommendations for hyperbaric oxygen therapy of cerebral air embolism based on a mathematical model of bubble absorption. Anesth Analg 1997;84:1203-7.

40. Peirce EC II. Specific therapy for arterial air embolism. Ann Thorac Surg 1980;29:300-3.

41. Reasoner DK, Ryu KH, Hindman BJ, Cutkomp J, Smith T. Marked hemodilution increases neurologic injury after focal cerebral ischemia in rabbits. Anesth Analg 1996;82:61-7.

42. Ryu KH, Hindman BJ, Reasoner DK, Dexter F. Heparin reduces neurological impairment after cerebral arterial air embolism in the rabbit. Stroke 1996;27:303-10.

43. Kizer KW. Corticosteroids in treatment of serious decompression sickness. Ann Emerg Med 1981;10:485-8.

44. Ganshirt H. Significance of hemorheology in diagnosis and therapy of ischemic cerebrovascular diseases: possibilities of therapy: antiedema procedures. Eur Neurol 1983;22:Suppl 1:78-82.

45. Sapolsky RM, Pulsinelli WA. Glucocorticoids potentiate ischemic injury to neurons: therapeutic implications. Science 1985;229:1397-400.

46. Dutka AJ, Mink RB, Pearson RR, Hallenbeck JM. Effects of treatment with dexamethasone on recovery from experimental cerebral arterial gas embolism. Undersea Biomed Res 1992;19:131-41.

47. Evans DE, Kobrine AI, LeGrys DC, Bradley ME. Protective effect of lidocaine in acute cerebral ischemia induced by air embolism. J Neurosurg 1984;60:257-63.

48. Evans DE, Catron PW, McDermott JJ, Thomas LB, Kobrine AI, Flynn ET. Effect of lidocaine after experimental cerebral ischemia induced by air embolism. J Neurosurg 1989;70:97-102.

49. Dutka AJ, Mink R, McDermott JJ, Clark JB, Hallenbeck JM. Effect of lidocaine on somatosensory evoked response and cerebral blood flow after canine cerebral air embolism. Stroke 1992;23:1515-20.

50. McDermott JJ, Dutka AJ, Evans DE, Flynn ET. Treatment of experimental cerebral air embolism with lidocaine and hyperbaric oxygen. Undersea Biomed Res 1990;17:525-34.

51. Mitchell SJ, Pellett O, Gorman DF. Cerebral protection by lidocaine during cardiac operations. Ann Thorac Surg 1999;67:1117-24.

CORRECTION

Gas Embolism

To the Editor: In their review, Muth and Shank (Feb. 17 issue)[1] discussed the pathophysiology, diagnosis, and treatment of gas embolism, a potentially fatal complication of various medical and surgical procedures. Although we found the review to be valuable, we are concerned that one of the treatment recommendations runs counter to the consensus opinion. Specifically, in Table 2, the authors indicate that the proper positioning of a patient with venous gas embolism is in the ``supine, flat´´ position. However, most authorities recommend that patients with suspected venous gas embolism be placed in a left lateral decubitus position with the head tilted downward (Trendelenburg's position).

Durant et al. demonstrated that dogs that were subjected in experiments to air emboli were more tolerant of air infusion, as indicated by measurement of hemodynamic variables, while lying on their left sides.[2] Durant et al. observed that this position placed the right ventricular outflow tract in a position inferior to the right ventricular cavity, allowing the air bolus to migrate superiorly and removing the obstruction to blood flow. In addition, Trendelenburg's position prevents the gas embolism from occluding the outflow tract by placing the right ventricular cavity in a more superior position.[3] These simple positional maneuvers should be performed in patients with a suspected venous gas embolism; they have been recommended by others who have written on the subject.[3,4,5]

Terrence D. Coulter, M.D.
Herbert P. Wiedemann, M.D.
Cleveland Clinic Foundation
Cleveland, OH 44195

References

1. Muth CM, Shank ES. Gas embolism. N Engl J Med 2000;342:476-482.

2. Durant TM, Long J, Oppenheimer MJ. Pulmonary (venous) air embolism. Am Heart J 1947;33:269-281.

3. Lambert MJ III. Air embolism in central venous catheterization: diagnosis, treatment, and prevention. South Med J 1982;75:1189-1191.

4. Orebaugh SL. Venous air embolism: clinical and experimental considerations. Crit Care Med 1992;20:1169-1177.

5. Coppa GF, Geuge TH, Hofstetter SR. Air embolism: a lethal but preventable complication of subclavian vein catheterization. JPEN J Parenter Enteral Nutr 1981;5:166-168.

To the Editor: … Figure 2 of the article by Muth and Shank is a misleading representation of the appearance of an intraarterial gas bubble in vivo. Our work demonstrates that bubbles are distorted into a sausage shape.[1] This difference in geometry in vivo means that the relations between bubble volume or surface area and ambient pressure shown for spherical bubbles in Figure 3 of the article do not apply. The altered shape of the bubble in vivo prolongs the absorption time, worsening the pathophysiologic consequences of an embolic event.[2] We have also demonstrated that microvascular gas bubbles provoke local arteriolar vasoconstriction. The depiction of the vessel wall bulging out around a spherical bubble in Figure 2 thus substantially understates the surface area of endothelium in contact with the bubble. The spatial and temporal effects of microvascular gas embolism are considerably worse than described by Muth and Shank.

David M. Eckmann, Ph.D., M.D.
University of Pennsylvania
Philadelphia, PA 19104

Annette B. Branger, M.S.
Northwestern University
Evanston, IL 60208

Daniel P. Cavanagh, Ph.D.
Bucknell University
Lewisburg, PA 17837

References

1. Cavanagh DP, Eckmann DM. Interfacial mechanics of stationary bubbles in flows in inclined tubes. J Fluid Mech 1999;398:225-44.

2. Branger AB, Eckmann DM. Theoretical and experimental intravascular gas embolism absorption dynamics. J Appl Physiol 1999;87:1287-1295.

To the Editor: In their excellent review of gas embolism, Muth and Shank did not refer to venous air embolism after the removal of a central venous catheter. The need to avoid air embolism during the insertion and manipulation of a central venous catheter is widely understood, but the risk associated with removal of the catheter is less widely known.[1] In a recent hospital survey, Ely et al.[2] found that 91 percent of 140 physicians placed patients in Trendelenburg's position when they were inserting a central venous catheter, but only 26 percent of the physicians knew that venous air embolism could occur after the removal of the catheter. Moreover, 14 percent removed catheters from patients who were placed in positions that could theoretically increase the incidence of this serious complication.

We have treated four patients with serious complications of gas embolism. Two cases occurred after the removal of a central venous catheter, and two occurred because of a delay in seeking care after the accidental removal of a central venous catheter.

Fibrin tracts consistently form around catheters, sometimes within 24 hours, creating a potential for portal venous air entry after the removal of a catheter[3] — even several minutes after the removal of the catheter.[4] Therefore, central venous catheters should be removed while the patient is either supine or in Trendelenburg's position, and the insertion site must immediately be covered with a sterile gauze, with firm manual pressure maintained until hemostasis is achieved. The insertion site must then be covered with an air-occlusive dressing. The dressing should remain in place for 24 to 72 hours.[1] It is also important to remember that urgent care is required to prevent gas embolism after the accidental removal of a central venous catheter.

Carlos Chamorro, M.D.
Miguel A. Romera, M.D.
Cándido Pardo, M.D.
Clínica Puerta de Hierro
Madrid 28035, Spain

References

1. Mennim P, Coyle CF, Taylor JD. Venous air embolism associated with removal of central venous catheter. BMJ 1992;305:171-172.

2. Ely EW, Hite RD, Baker AM, Johnson MM, Bowton DL, Haponik EF. Venous air embolism from central venous catheterization: a need for increased physician awareness. Crit Care Med 1999;27:2113-2117.

3. Hoshal VL Jr, Ause RG, Hoskins PA. Fibrin sleeve formation on indwelling subclavian central venous catheters. Arch Surg 1971;102:353-358.

4. Turnage WS, Harper JV. Venous air embolism occurring after removal of a central venous catheter. Anesth Analg 1991;72:559-560.

The authors reply:

To the Editor: Chamorro and colleagues point out the need for vigilance during the removal of a central venous catheter. We included this potential mechanism in Table 1 of our article: ``entry of air through disconnected intravascular catheter, inadvertent infusion of air through intravascular catheter.'' This is an important mechanism of gas embolism and deserves to be emphasized.

Eckmann and colleagues correctly suggest that an intraarterial gas bubble in vivo is more sausage-shaped than spherical — a point that helps explain why a bubble induces damage through complex interactions with the blood vessel's endothelium and the surrounding tissues. Our Figure 2 was designed to summarize the complex pathophysiology in a simple diagram.

Coulter and Wiedemann believe the left lateral decubitus position with the head tilted downward is the preferred position for the treatment of venous gas embolism, citing a study that was performed more than 50 years ago.[1] The results of that study suggested that dogs that received massive infusions of air were more likely to survive if they were in the left lateral decubitus position. More recent studies[2,3] indicate that hemodynamic responses are not improved by the use of alternative positions instead of a flat, supine position. These studies did demonstrate that intracardiac air relocates to more nondependent regions. If a transvenous catheter is present in the right side of the heart, placing the patient in the left lateral decubitus position before attempting to withdraw the air may have merits, as we mentioned in Table 2.

For most patients with venous gas emboli, placing them in the supine position affords physicians a better opportunity to administer supportive therapy, including ventilatory support and oxygen, to establish access for catecholamine delivery, and to perform cardiopulmonary resuscitation. Thus, since there are no demonstrated hemodynamic improvements associated with the use of the left lateral decubitus or head-down position, we believe that the supine position is the best choice.

Finally, the legend for Figure 2 should have read, ``Sodium and water enter the neurons, and cytotoxic edema develops,'' not ``Sodium and water enter the vessel . . . ,'' as printed.

Claus Martin Muth, M.D.
Universitätskliniker des Saarlandes
66424 Hamburg/Saar, Germany

Erik S. Shank, M.D.
Massachusetts General Hospital
Boston, MA 02114

References

1. Durant TM, Long J, Oppenheimer MJ. Pulmonary (venous) air embolism. Am Heart J 1947;33:269-281.

2. Mehlhorn U, Burke EJ, Butler BD, et al. Body position does not affect the hemodynamic response to venous air embolism in dogs. Anesth Analg 1994;79:734-739.

3. Geissler HJ, Allen SJ, Mehlhorn U, Davis KL, Morris WP, Butler BD. Effect of body repositioning after venous air embolism: an echocardiographic study. Anesthesiology 1997;86:710-717.