

Gas Embolism in Obstetrics and Gynecology

A Review

Amir Weissman, M.D., Shahar Kol, M.D., and Bezalel Alik Peretz, M.D.

OBJECTIVE: To review gas embolism in the field of obstetrics and gynecology, with an emphasis on the pathophysiology, clinical presentation and treatment options.

STUDY DESIGN: A review of the world literature on gas embolism.

CONCLUSION: Gas embolism is an unusual complication and has increased in frequency since the introduction of new invasive procedures. Since the clinical presentation of gas embolism has many faces, it is important to identify it as early as possible: timely treatment may be life saving, while a delay may have serious consequences. (J Reprod Med 1996;41:103-111)

Keywords: air embolism.

Introduction

The increased use of invasive techniques in modern medicine has led to a parallel rise in the number of cases of iatrogenic gas embolism. Although not frequently encountered by obstetricians and gynecologists, there are occasional reports in the literature

of fatalities or serious morbidity resulting from this complication. Modern obstetrics and gynecology involves numerous procedures that can cause gas embolism. Since physicians are often unfamiliar with its clinical presenta-

tion, treatment may be delayed. The last comprehensive review of gas embolism in obstetrics and gynecology was published more than three decades ago,¹ and since treatment modalities have been developed since that time, we present an update on this important topic.

Historical Aspects

The first clinical documentation of arterial gas embolism in humans is probably one by Morgagni, in his treatise, *The Seats and Causes of Disease*, published in 1769,² although there have been sporadic reports of gas embolism since ancient times. Early in the 19th century, Barlow³ documented a case in which a sudden hissing sound was heard during removal of a neck tumor, and the patient immediately died. Only 30 years later was it recog-

Gas embolism may be encountered more frequently than was previously thought, and its consequences can be grave.

From the Department of Obstetrics and Gynecology, Rambam Medical Center, Haifa, Israel.

Address reprint requests to: Amir Weissman, M.D., Department of Obstetrics and Gynecology, Rambam Medical Center, 31096 Haifa, Israel.

nized that this event had been a case of gas embolism. In 1827 Magendie⁴ recorded a similar incident during an attempt to remove a shoulder tumor by detaching part of the clavicle. In 1864 Roger described iatrogenic gas embolism as a result of open-

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ing and irrigating empyema cavities, a commonly employed procedure at the time.⁵ The true nature of these events was not understood, and the etiology was thought to be neurologic. (The term *pleural epilepsy* was used to describe these cases.) Later, as underwater diving became more popular, cases of gas embolism were encountered more frequently.

With the great progress made in medical practice over recent decades and with the advent of new, invasive medical techniques, iatrogenic gas embolism has become increasingly common, and cases have been reported in almost every field of medicine.

The first reports of gas embolism associated with obstetrics were presented by Cormack in 1837 in his thesis *The Presence of Air in the Organs of Circulation*.⁶ Numerous other cases resulting from different obstetric or gynecologic procedures have been reported since then.

Incidence

The true incidence of gas embolism is unknown and, for a number of reasons, is probably underestimated. First, while most cases of diagnosed fatal gas embolism are documented correctly, some are misdiagnosed and not reported as such. Many minor, nonlethal cases are not reported for the same reason. Second, illegal abortions, a major source of iatrogenic gas embolism, are rarely reported. Third, most cases of embolism are included under the general heading of "pulmonary embolism," without specifying the embolus type. In a recent report analyzing the causes of maternal mortality in New York City in the years 1981-1983,⁷ pulmonary embolism was found to be one of the leading ones. Although only one case was attributed to air embolism, other cases could not be excluded because of incomplete reporting and incorrect diagnosis, as mentioned above. Since gas embolism is not en-

countered frequently in obstetrics and gynecology and obstetricians are not familiar with it as a possible complication of many of the procedures they perform, its true incidence in this field remains unknown.

Pathophysiology

Any invasive procedure is a potential portal of entry of gas into the circulatory system. Gas embolism occurs when a noncollapsible vein or artery is opened or severed and a positive pressure gradient is built up between the vessel and the ambient pressure or a source of positive pressure. In the obstetric and gynecologic setting, gas most commonly enters the circulatory system through the pelvic venous plexi. Gas emboli are usually filtered by the pulmonary capillary bed, leaving the patient asymptomatic. Considerable volumes of gas may pass through the pulmonary filter without demonstrable abnormalities. However, a patent foramen ovale is not a rare phenomenon even in healthy adults; an autopsy study of 965 "normal" hearts found a 25% incidence of patent foramens ovale.⁸ These patients are at greater risk of gas embolism due to the possibility of arterialization of a venous air embolism at the level of the atria.

The lung's capacity to act as a filter has been tested in animal models. In pigs, air infused directly into the right ventricle at a rate of 0.05 mL/kg/min was completely filtered and remained undetected by transesophageal echocardiography.⁹ An increase in pulmonary vascular pressure may decrease the filtration capacity by spilling bubbles into the arterial circulation via noncardiac physiologic shunts.¹⁰ In addition to this, as mentioned above, 25% of adult patients have been reported to have a patent foramen ovale, which facilitates the passage of gas into the left side of the heart and the systemic arterial circulation.¹¹ Because of their buoyancy, gas bubbles will be distributed throughout the systemic circulation, depending on the patient's position. If the head is lowered, gas may be trapped in the left ventricle and further embolization delayed. Coronary artery embolism is likely to occur if the patient remains supine. In this case, even 0.5 mL of gas may cause fatal arrhythmia.¹²

Since the head is normally higher than the heart, the brain is the main target organ of gas embolism. Animal experiments have questioned the importance of bubble buoyancy in the arterial circulation.¹³ Some data suggest that the head-down position does not prevent cerebral embolism and may

even prove detrimental as far as neurologic outcome is concerned. Gas in the cerebral circulation will spread through the arterial tree until it blocks arteries 30–60 μm in diameter,¹⁴ also producing an immediate change in vascular permeability. The ischemic region becomes hyperemic as the gas-filled arteries dilate and lose their capacity for autoregulating blood flow. An inflammatory reaction occurs during the immediate postischemic period, with granulocyte accumulation, coagulation and thrombosis.¹⁵ As gas enters the cerebral vasculature, there is an immediate rise in cerebrospinal fluid pressure.¹⁶

Small bubbles ($15 \pm 5 \mu\text{m}$ [mean \pm SD]) are generally assumed to be harmless since they do not arrest the cerebral circulation. However, Hills and James¹⁷ demonstrated, in an animal model, that even microbubbles impair blood-brain barrier integrity. This finding suggests that besides blocking blood vessels, air has a direct effect on brain tissue. In an area of focal ischemia, dead cells are surrounded by others that maintain their integrity but not their function; this is termed the "ischemic penumbra."¹⁸

Other pathologic changes that occur in the immediate postembolization period (30–180 minutes) include disruption of the blood-brain barrier, astrocytic and neuronal swelling, vacuolation and ischemic neuronal changes.¹⁹ Prompt oxygenation may restore neuronal function, minimizing neurologic impairment.

Much research has been done in an attempt to determine the highest nonlethal dose of air that can be introduced into the circulation. Since this work was performed on nonhuman animals, the conclusions drawn must be regarded with caution. Extensive reviews of these studies have been published by Nelson¹ and Peirce.²⁰ Embolism was generally noted at doses $>1 \text{ mL/kg/min}$ and death at rates $>3 \text{ mL/kg/min}$. Lower flow rates (0.5 mL/kg/min) caused permanent brain damage. The small amount of human data reported shows that oxygen given intravenously at flow rates of $0.1\text{--}0.4 \text{ mL/kg/min}$ has not been fatal.¹

Diagnosis

The clinical presentation of gas embolism is highly variable and depends on several factors: (1) the patient's position, (2) the flow rate of the gas, (3) the type of gas, (4) the total volume of gas introduced, (5) the size of the bubbles, (6) the weight of the patient, and (7) the physical condition of the patient

prior to the insult. With the patient in the supine position, the gas is likely to cause coronary artery embolization, with resultant cardiac arrhythmias, chest pain and myocardial infarction due to ischemia. In the Trendelenburg position, gas bubbles may be trapped in the splanchnic circulation, liver or left ventricle for a long time and only later pro-

Gas embolism must be borne in mind whenever a previously healthy patient develops cardiorespiratory or neurologic symptoms....

duce symptoms of gas embolism. In the standing or sitting position, the gas bubbles usually migrate to the brain, causing a variety of neurologic symptoms, such as seizures, unconsciousness or altered sensorium, paresthesias, paralysis, visual disturbances or cortical blindness. Some of the symptoms are caused by air bubbles trapped in the right ventricle. These produce a mixture of gas and blood that is turned into foam by each heart beat, resulting in an "air trap" that prevents blood flow to the pulmonary tree. Pressure in the right side of the heart increases, and there is a marked decrease in blood flow from the left side of the heart. Some patients may exhibit the typical "mill wheel" murmur on cardiac auscultation. Skin marbling, blanching of arteriole segments in the nail beds, pallor of mucous membranes (i.e., areas of pallor on the tongue, or Liebermeister's sign) and even air bubbles in retinal arteries are also occasionally found. Gas bubbles trapped in the microcirculation may cause late manifestations, such as disseminated intravascular coagulation, tissue ischemia and necrosis, and gastrointestinal bleeding due to mucosal damage, all of which are caused by blockage of blood vessels and destruction of adjacent tissue.

Suspicion of gas embolism may be aroused by the sudden development of acute cardiorespiratory failure or neurologic symptoms in an otherwise healthy patient, usually following an invasive procedure. One simple method of detecting cardiac air emboli is by using a stethoscope.²¹ More accurate diagnosis of air embolism requires facilities not routinely found in the general clinical setting. Doppler ultrasound can be used to investigate the occurrence of gas embolism.^{22–24} This technique is based on the frequency shift in relation to the velocity of

moving particles (e.g., air, amniotic fluid or thrombi). Doppler monitoring is therefore a sensitive but nonspecific method of detecting air emboli. Echocardiography, however, can distinguish between air and other emboli. A prospective study of 49 parturients undergoing cesarean section demon-

Prompt diagnosis and treatment in a high-pressure chamber could be life saving.

strated an excellent correlation between the embolic events detected by Doppler ultrasound and by echocardiography. It was concluded that venous emboli detected by Doppler monitoring were indeed air emboli, not amniotic fluid or thromboemboli.²⁵

Another means of detecting air embolism is the end-tidal carbon dioxide monitor, which is claimed to be more reliable for this purpose than precordial Doppler ultrasound.^{26,27} Computed tomography of the brain would be an appropriate diagnostic method in the event of massive cerebral air embolism.²⁸ A definitive diagnosis of air embolism may be obtained by demonstrating gas bubbles in the heart or circulation by aspiration or on postmortem examination by opening the heart under water to detect gas bubbles.

Treatment

Control of gas embolism lies in its prevention. If an embolism does occur, immediate measures should be taken to cut off the gas supply. Further emergency care includes administration of 100% oxygen by mask as well as intravenous fluids to counteract hemoconcentration, which increases blood viscosity. The use of corticosteroids has been recommended,²⁹ although convincing evidence for their therapeutic efficacy is lacking. Osmotic diuretics should be avoided since they affect normal brain tissue but not ischemic zones³⁰ and may aggravate hemoconcentration. Vasodilators should also be avoided since they do not affect vessels in areas of ischemia and may in fact lead to pooling of blood in normal zones. Heparin is not recommended since the gas may cause a hemorrhage in infarcted areas.¹⁴ Aspirin has also been considered; however, by inhibiting both thromboxane and prostacyclin, aspirin

may have an unpredictable effect on the cerebral vasculature affected by the gas and therefore requires further evaluation.

It is widely held that the patient with an arterial gas embolism should immediately be placed in a steep head-down position (the Trendelenburg position). This maneuver is of benefit in the treatment of venous gas embolism because it leads to gas trapping in the right atrium, thus reducing the amount of gas distributed throughout the pulmonary vasculature.³¹ Based on similar considerations of the buoyancy effect, it was suggested earlier in this century that the Trendelenburg position may protect the brain from arterial embolization.³² The therapeutic effect of the head-down position has not been studied in humans. However, Dutka et al¹³ found, in a carefully designed animal model, that the head-down position may have a deleterious effect, mainly due to an increase in intracranial pressure. They recommended that patients with suspected gas embolism be transferred to the nearest hyperbaric treatment facility in a supine or sitting position. All these measures serve to stabilize the patient's condition. However, the specific and single most effective therapy for gas embolism is hyperbaric oxygenation (HBO). HBO is based on the mechanical compression of gas bubbles according to Boyle's law: the volume of a gas is inversely proportional to the pressure to which it is subjected. Hence, compression to 6 atmospheres absolute (ATA) will reduce the size of an intravascular gas bubble to one-sixth its original volume. This helps relieve the vascular obstruction and restore perfusion. Other therapeutic effects of HBO are acceleration of bubble resorption; vasoconstriction, with a reduction in brain edema and swelling of ischemic cells; and enhanced oxygenation of ischemic brain tissue.³³

Hyperbaric oxygenation was introduced in the 1930s for the treatment of pulmonary barotrauma-associated air embolism in divers.³⁴ However, there have been no prospective, randomized, controlled studies that have proven the effectiveness of HBO in humans, and it would probably be unethical to conduct such studies. Animal studies, supported by data from submarine escape training accidents, provide firm evidence in favor of hyperbaric treatment. The HBO treatment profile is determined by the type of gas breathed, its pressure and the exact time interval during which the patient breathed the gas. Due to the possibility of central nervous system oxygen toxicity, the partial pressure of oxygen in

I. OBSTETRIC CAUSES

- A. During pregnancy
 - 1. Intravaginal/intrauterine insufflation of powder, liquid or gas
 - a) Powder insufflation for the treatment of trichomoniasis
 - b) Induction of abortion (illegal)
 - c) During sexual intercourse
 - 2. Induction of abortion by intraamniotic instillation of hypertonic saline
 - 3. During cerclage procedures
- B. During labor and delivery
 - 1. Uncomplicated labor or delivery
 - 2. Complicated labor or delivery
 - a) Rupture of uterus
 - b) Placenta previa
 - c) Inversion of uterus
 - 3. Operative delivery
 - a) Forceps/vacuum delivery
 - b) Cesarean section
 - c) Intrauterine manipulations
- C. During puerperium
 - 1. Postpartum exercise (knee-chest exercise)

II. GYNECOLOGIC CAUSES

- A. Diagnostic procedures
 - 1. Rubin test
 - 2. Laparoscopy
 - 3. Hysteroscopy
- B. Operative procedures
 - 1. Hysterectomy
 - 2. Sterilization
 - 3. Operative hysteroscopy

Figure 2 Obstetric and gynecologic causes of gas embolism.

Legal. There are occasional reports of air embolism after legal abortion.^{1,50,51} In one case, air embolism complicated abortion induced by intraamniotic instillation of hypertonic saline. HBO therapy administered soon after the incident almost completely reversed the neurologic insult.⁵²

Orogenital Sex

Numerous reports have described gas embolism resulting from orogenital sex during pregnancy. A review of 11 such cases was published by Bray et al in 1983,⁵³ and many others have been documented since then.⁵⁴⁻⁵⁹ During pregnancy the vagina is extremely distensible and can accommodate up to 2 L of air under pressure. The pressurized air may dissect through the amniotic membranes, gaining access to the subplacental sinuses and entering the systemic circulation. Sudden collapse during or after orogenital sex has been followed frequently by death.

Labor and Delivery

Gas embolism as a complication of normal labor and delivery has also been reported.^{60,61} Although the exact mechanism could not be determined, it was speculated that partial separation of the placental edge allowed air to gain access to the uterine sinuses. Under uterine contractions, the air was forced into the systemic circulation.

Complicated and Operative Delivery

There are many case reports describing gas embolism in complicated deliveries: during manual removal of retained placental fragments, in placenta previa or accreta, in placental abruption, in forceps and vacuum delivery, in a ruptured uterus and after breech delivery.^{1,62-64} Cesarean section has often been implicated as a cause of air embolism.^{22,24,65-68} In a prospective study, precordial Doppler ultrasound was used to detect air embolism during cesarean section in 78 patients. Doppler changes consistent with venous air embolism were recorded in 51 patients (65%), 37 of whom showed a decrease in arterial O₂ saturation.²⁴ This confirmed that a large proportion of patients undergoing cesarean section are at risk of developing gas embolism; some actually have sublethal subclinical or clinical gas embolism. Gas embolism was found to occur between uterine incision and delivery and to be less common with general anesthesia than local. Both ruptured membranes and a protracted interval between uterine incision and delivery were found to predispose to embolism.

Air Embolism in the Puerperium

A number of publications describe air embolism following the "knee-chest" exercise recommended in the past for puerperal women, mainly for the treatment of retroversion and subinvolution of the uterus.⁶⁹⁻⁷³ Air entered the vagina in the "knee" position but was prevented from escaping on return to the original position with the approximation of the thighs and labia, which created a one-way valve mechanism. The pressure thus built up in the vagina pushed air to the uterus and through the uterine sinuses to the systemic circulation. Since this exercise is probably no longer practiced, no new cases have been reported over the past two decades.

Fetal Reduction

Air embolism may be employed for the selective termination of multiple pregnancies.^{74,75} A small

volume of air is injected into the fetus intracardially or via the umbilical vein, under sonographic or fetoscopic guidance, until fetal death is documented. Although no maternal effects are noted, we mention this procedure because air under pressure is intentionally introduced into the body as a medical procedure.

Gynecology

Diagnostic Procedures (Rubin Test)

Transcervical insufflation of air was introduced by Rubin in 1913 and gradually gained popularity as a diagnostic and therapeutic procedure. Today, however, newer methods have been adopted, and the Rubin test is employed less frequently. Several cases of fatal air embolism have been reported following the use of this test. In one of them, curettage had been performed beforehand, providing grounds for suspecting a port of air entry.⁷⁶ In another, no clear source of entry was found.⁷⁷ Further cases were published in later years.^{78,79}

Laparoscopy

The direct introduction of gas under pressure into body cavities is more likely to cause air embolism than any other procedure. There are several reports of cardiovascular collapse during or after laparoscopy,⁸⁰⁻⁸³ and this procedure is thought to be one of the major causes of clinical and subclinical gas embolism.

Hysteroscopy

Several cases of gas embolism during hysteroscopy or hysteroscopy-related procedures have been reported in recent years. It was found that about 10% of patients undergoing hysteroscopy with a carbon dioxide laser manifested heart sounds typical of gas embolism.²¹ Baggish and Daniell reported on five patients, of whom four died and one survived with neurologic deficits following endometrial ablation with an Nd-YAG laser.⁸⁴ These and other reports⁸⁵ confirm that patients undergoing hysteroscopy are at risk of gas embolism. We recently reported a fatal outcome after operative hysteroscopy in which the clinical diagnosis was suspected air embolism.⁸⁶

Postpartum Sterilization

One case of postpartum gas embolism following surgical sterilization has been reported.⁸⁷ The mechanism was thought to be the abrupt change from the Trendelenburg to the supine position, with

approximation of the thighs and trapping of air under pressure in the vagina; the air subsequently was forced through the raw uterine sinuses into the systemic circulation.

Hysterectomy

Air embolism has been reported to occur during radical hysterectomy.⁸⁸ It was speculated that in the Trendelenburg position, the pressure in the venous plexus may be subatmospheric, particularly if central venous pressure is low. In this situation, large amounts of air may enter the exposed, thin-walled veins and spread throughout the circulation.

Cervical Cerclage

There is one reported case of proven paradoxical gas embolism following cervical cerclage.⁸⁹ Despite resuscitation attempts, the patient died.

Discussion

Gas embolism may be encountered more frequently than was previously thought, and its consequences can be grave. There is probably no obstetric or gynecologic procedure that is safe from gas embolism. The possibility of gas embolism must be borne in mind whenever a previously healthy patient develops cardiorespiratory or neurologic symptoms during or after an operation or other invasive procedure. Prompt diagnosis and treatment in a high-pressure chamber could be life saving.

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