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Infusion-Related Air Embolism

ABSTRACT

Vascular air embolism as a medically induced complication may be associated with numerous treatments and therapies. In infusion therapy, the risk is associated with venous and arterial catheterization as well as various other invasive procedures and much of the equipment used for them. The manner of air entry and the presentation of symptoms may vary greatly. Appropriate treatment options are dependent on air entry routes. Nurses need to be aware of the common and seldom-considered causes of air embolism to be able to guard against this complication, yet adequately support the patient if it occurs.

Key words: air embolism, bolus entry, IV catheter, microbubble, patent foramen ovale, Trendelenburg, Valsalva

The fatal potential for vascular air embolism was first described in 1667 by Italian scientist Fransesco Redi.[1] Air embolism as a complication of intravenous (IV) therapy has been recognized since the 19th century. Unfortunately, modernization of medicine has not diminished or eliminated this concern. The overall incidence of air embolism is considered low, and though the true frequency of occurrence is unknown, it has been estimated at a high of 1:47 catheterization events to a low of 1:3000. Overall, episodes of verified air embolism are associated with high mortality (30% or greater) or devastating morbid events.[2] This prompted The Joint Commission (then the Joint Commission on the Accreditation of Healthcare Organizations) to include air embolism as 1 of the 34 sentinel events recognized in a 1995 risk-management tool.[3] In 2008, the Centers for Medicare and Medicaid Services included it as 1 of the original 8 costly, preventable and non-reimbursable events.[4]

Air embolism can occur in conjunction with any entry into the vascular system. Implicated instruments include central or peripheral catheters, arterial lines, intraosseous devices, or any specialty vascular catheter used for treatment or diagnosis. Events may be associated with insertion, use, maintenance, or removal. Failure or improper use of equipment or devices has also been implicated. Confirmed embolism has been noted in association with surgical and nonsurgical procedures. No vascular procedure is exempt from the risk, and air embolism has been identified in relation to blood administration, therapeutic phlebotomy, lab draws, and a multitude of invasive procedures.

GENERAL PATHOLOGY

Two conditions must be simultaneously present for air to enter the vascular system: There must be a pressure gradient between the vascular space and atmospheric air, and there must be a direct line of access to the blood vessel. The severity of the embolism depends on the volume of air that enters the vessel, the rate of entry, and the patient's position at the time of entry. Patient-dependent considerations are age, size, and existing disease process.[2]

There is not an exact volume of air that is significant, but, in general, greater than 50 mL is considered potentially lethal. Case histories have demonstrated that 20 mL or less of rapid air intake may result in fatal embolism.[5] The rate of entry affects the potential for and severity of resulting morbidity or mortality. Rapid bolus injection may result in precipitous cardiovascular collapse, whereas gradual accumulations (microbubbles) may go unnoticed.

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The pressure gradient that allows for air entry may be active (forced) or passive. When the access vessel is above the level of the heart and open to the atmosphere, conditions are optimal for passive air entry. For example, patients seated in an upright position during central venous catheter removal are at much greater risk for passive air entry than those in a supine position.[6] Active, or forced, air entry can occur with the patient in any position. Occurrences are often related to the use of pressure bags, improperly primed IV tubing, or syringes that have not been entirely purged of air.

Air entry into the vascular system is not always easily and intuitively recognizable. In some cases, the route of entry is so insidious that diagnosis is based on autopsy findings; the actual route of entry may never be determined.

Microbubbles

Although microbubbles are the less likely culprit of a serious air embolism, the risk is significant enough that it merits discussion. *Microbubbles*, as the term implies, are tiny bubbles of air that enter circulation, migrate through the right chambers of the heart, and enter the pulmonary artery. Because of their tiny size, they are able to travel through the pulmonary artery directly to the pulmonary vascular bed. Rarely does this present a significant event because the lungs are a superb filter of small bubbles.[7] Rapid entrapment or a large number of bubbles, however, overloads the system. Associated mortality is related to the number and size of the bubbles and the rate of accumulation. The age and size of the patient as well as underlying diagnoses will also affect the outcome.

The phenomenon of bubble formation in tubular devices, such as IV tubing, has been recognized since the late 1800s and can be researched by reviewing Reynolds number or Hagen-Poiseuille's law. Infusion-related microbubbles are most commonly formed in extracorporeal tubings such as those used for dialysis and external perfusion. The risk of formation in these lines seems to be related to pressure and/or fluid temperature.[8] Any rapid infusion, however, may result in the creation of bubbles. The basic concept is that flow through IV tubing begins as laminar flow as fluid exits the drip chamber, with all particles maintaining essentially straight lines. As the fluid advances down the tubing, the lines of particles begin to angle, causing the fluid to bounce off the sides of the tubing. This provides temporary areas on the sides of the IV tubing that are free of force and in which bubbles can form and become attached.[9] Larger bubbles are buoyant enough to rise to the drip chamber, but smaller bubbles become trapped in the flow and are swept into the bloodstream.[8] Turbulence increases in relation to the length of the tubing and rate of flow. The risk of significant air bubble entrapment within the lungs cannot be predicted, and parameters for rate and fluid amount are not available.

Turbulent flow is also created when IV flush solutions are administered using the push-pause technique. The amount of fluid administered is usually insignificant to generate enough bubbles to form an embolism. Likewise, the "bubble" test, which is used as a diagnostic or verification technique, uses amounts of fluid and air too small to cause significant risk.[10] This test involves the rapid instillation of a mixture of 5 to 10 mL of saline with 1 mL or less of air that has been agitated to form bubbles; the injected bubbles aid in visualization of certain parameters within the heart chambers.

Should the collection of microbubbles in the lungs become significant, the time frame in which the problem is apparent will depend on the amount of damage incurred to the parenchyma and major organs. Initially, bubbles are evacuated through normal lung filtration. But when accumulation exceeds filtration rate, these bubbles mingle with one another, merging into larger bubbles. These large bubbles become trapped in the pulmonary capillaries, forming emboli in the vascular bed. The following sequence of events then occurs, which may be devastating, depending on the size and number of emboli that develop.[1,8]

The bubble initially presses against the endothelial capillary wall, stripping the cells and forcing gaps to appear between the cells. Intravascular fluid leaks into surrounding tissue, resulting in interstitial edema. Back pressure forms behind the bubbles, causing increased pulmonary artery pressure. Furthermore, tissue ischemia of the pulmonary walls occurs in relation to the positioning of the bubbles. Neutrophils recognize the bubbles as foreign and begin to surround them, initiating the inflammatory response and forming clumps, which extend the embolus. Pulmonary membranes become more permeable to fluid, and interstitial pulmonary edema develops.

As a continuation of the immune response, complement is activated, stimulating mast cells to release histamine. Secondary to complement activation, granulocytes are triggered to release cytotoxic substances, which further damage tissue. As a part of the inflammatory response, platelets and blood protein surround the bubble. The coagulation cascade is activated as platelets aggregate on the bubble surface. Even though the bubble may have dissolved by this time, the resulting thrombus further plugs the vessel and extends tissue damage.

One issue with microbubbles is that removal or repositioning of the air is not an option. Treatment is aimed at supporting systems and attempting to reduce the onset of severe pulmonary edema.

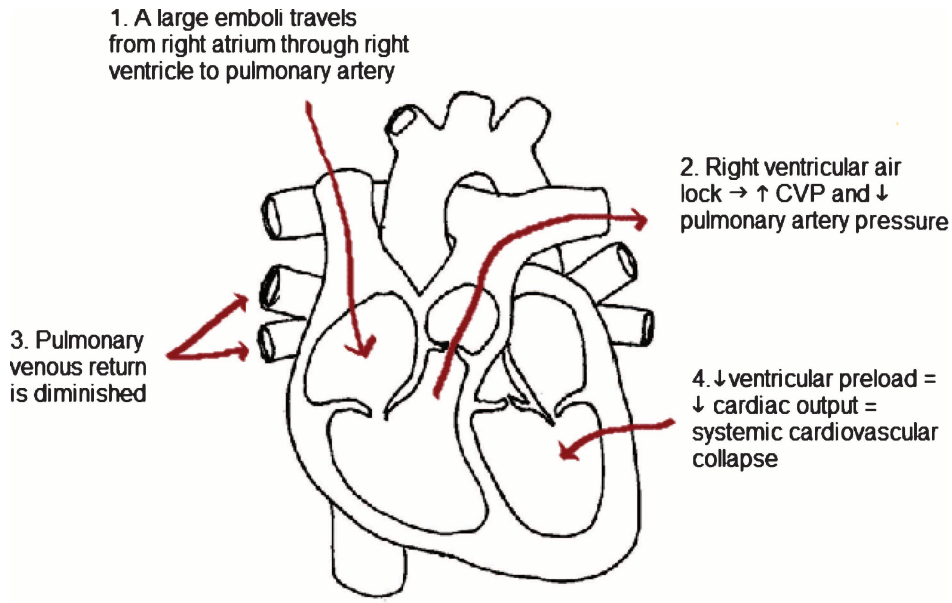

Figure 1. Dynamics of a bolus air embolism.

Bolus Influx

Most commonly, when air enters the vascular system, it does so as bolus influx or injection. The literature is dotted with examples of air being actively introduced, the most common culprits being connection of the catheter to an unprimed tubing or syringe. Passive entry, however, is the more typical scenario.

The risk of passive air entry is greatest during the inspiratory phase of spontaneous respiration. During inspiration, the external intercostal muscles move the rib cage up and out. This creates negative pressure within the intrathoracic space and allows for expansion of the lungs. Simultaneously, compression on the right atrium is released, resulting in decreased right atrial (central venous) pressure and allowing venous blood to flood the chamber.[11,12]

At this time, there is a potential difference between the pressure in the right atrium and that of the atmosphere. If exposure to air occurs during this time of increased venous return, a significant embolism can occur. The potential increases further with deep respirations, with coughing, in the presence of dyspnea or hypovolemia, and when the patient's head is elevated. Because of further decreases in central venous pressure (CVP) in these latter situations, embolism can occur even during expiration.[13]

Of interest, ventilated patients who are receiving positive end-expiratory pressure (PEEP) are at decreased risk for passive air embolism because inspiration is associated with a positive, rather than negative, pressure gradient.[10] Therefore, there is no corresponding decrease in CVP, and venous return is diminished. Although the practice is controversial, some invasive surgical procedures that have a high risk of air embolism are performed with the application of PEEP to reduce the risk of air embolism.[10,14]

The passive entry of air may occur at any time from placement of the catheter until several days after removal. A 14-gauge (5 French) catheter 5 cm in length can allow for the influx of 100 mL of air per second, so even the briefest exposure to atmospheric air can result in lethal embolism.[15-17] On removal of the catheter, an open tract up to 2.5 cm in length may be briefly present; this tract can allow for the passage of up to 200 mL of air per second. Air entry has even been noted when only the guidewire is in place.[18]

Pathology of Events

A bolus of air can travel rapidly through the right chambers of the heart. Air enters the right atrium and passes into the right ventricle, developing an air lock that prevents passage of blood into the pulmonary artery. On one side of the air lock, CVP increases as blood becomes trapped and accumulates in the right atrium. On the other side of the air lock, pulmonary artery pressure decreases because of diminished blood supply. The impaired blood supply to the lungs results in decreased pulmonary venous return. This, in turn, results in diminished left ventricular preload, which decreases cardiac output and, finally, results in systemic cardiovascular collapse (Figure 1).[5]

Patent Foramen Ovale

The severity of air embolism is increased in the presence of a patent foramen ovale (Figure 2). In fetal circulation, because the lungs are not used, blood bypasses pulmonary circulation. Instead, it is shunted from the right

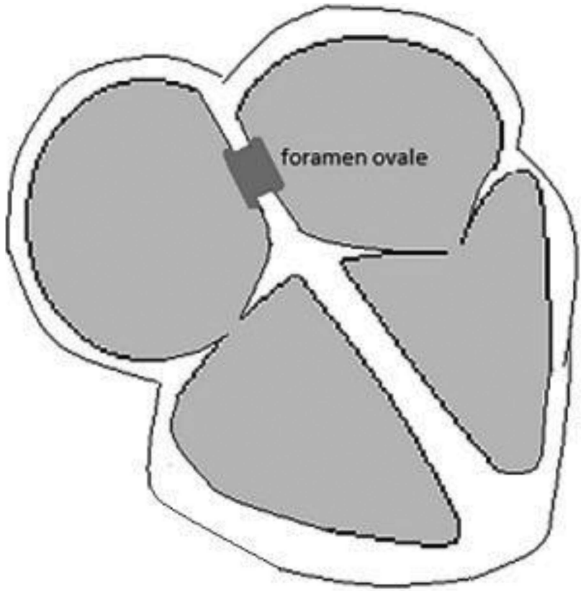

Figure 2. Patent foramen ovale.

atrium to the left through an opening known as the foramen ovale. After birth, a flap of skin in the left atrium closes over the opening and becomes permanently attached, forcing venous blood into the pulmonary vasculature.[19] However, persistence of a patent foramen ovale, in which the flap does not permanently adhere, has been reported in anywhere from 10% to 15%[19] of the population to as high as 35%.[20] Any time right atrial pressure exceeds left atrial pressure, nonoxygenated blood is pushed through this opening into arterial circulation. In the event of air embolism, air in the right chamber is able to cross over to the left atrium and enter systemic circulation. The condition is known as a paradoxical air embolism.[2] Blockage may occur in any organ but is most likely to be noted in the coronary arteries (with resulting myocardial infarction) or the brain (with resulting stroke).[19,21]

Signs and Symptoms

The most common symptoms of bolus air embolism are sudden dyspnea, lightheadedness, shoulder and chest pain, and, possibly, nausea.[2,22] The resulting dyspnea may stimulate a gasp reflex as the patient attempts to pull in air. This reflex results in a short, forced inspiration that increases negative thoracic pressure and pulls additional air through the open system.[18] Agitation, irritability, or anxiety, often expressed as feelings of impending doom, are not uncommon.

The most common signs associated with bolus air embolism are tachypnea, tachycardia, and hypotension.[22] Neurological manifestations may emulate stroke.

A splashing auscultatory sound, referred to as a mill-wheel murmur, is indicative of a large right ventricular air embolism.[2,10,15] This unique sound provides definitive diagnosis of bolus air embolism, but, unfortunately, it is a rare presentation and, even when present, may be difficult to recognize. More commonly, a harsh systemic murmur may be noted, but often no changes in heart sounds can be detected.

Diagnosis

Because the route for air entry is not always identifiable, the diagnosis may not be straightforward. Numerous differential diagnoses are associated with similar signs and symptoms (Table 1).[23] Although bolus injections of air are not necessarily immediately fatal, prompt diagnosis and intervention will decrease the potential morbidity and mortality.

Laboratory tests are neither sensitive nor specific for air embolism. Although some tests may be useful in evaluating resulting end-organ injury, none are definitive for the initial diagnosis.[24]

A number of radiological techniques have shown some benefit in determining diagnosis. Transthoracic echocardiography may be performed at the bedside and provide prompt diagnosis and intervention.[23] Precordial ultrasonography is more sensitive in detecting air and is capable of detecting as little as 0.25 mL.[9] The benefit of these techniques, however, is limited to occasions when air embolism can be anticipated and planned for, such as certain invasive or surgical procedures. They seem to hold no value when the embolism is unanticipated.[24]

Treatment

The initial symptoms of air embolism may be subtle, and the problem may not be fully recognizable until cardiovascular collapse occurs. For that reason, treatment should begin if embolism is suspected even though symptoms have not yet fully manifested (Table 2).

Turn the patient onto the left side and place him or her in the Trendelenburg position. If the patient will not tolerate the Trendelenburg position, the left lateral decubitus (left side, head flat) position should be used. (See Table 3 for positioning recommendations.) Trendelenburg is optimal because it decreases the gradient between atmospheric air and the vasculature while left-sided positioning holds the entrapped air in the apex of the right atrium to prevent occlusion of the pulmonary artery.[25] To a lesser degree, left lateral decubitus provides these benefits. Fowler's and semi-Fowler's position (back lying, head elevated) should be avoided unless the patient has a known elevation of intracranial pressure. In this case, maintain the head at a 30° position to reduce cerebral herniation.[5]

If the route of passive air entry is obvious, attempts should be made to immediately occlude the opening.[26(pp117-119)] Maintaining sterility of the catheter or site should not be the primary concern. Consider the case of Ida, a long-term sickle cell patient who accidently cut off all 3 lumens of her catheter when

TABLE 1

Differential Diagnosis for Air Embolism[22]

Pulmonary thromboembolism
Acute bronchospasm
Pneumothorax
• Spontaneous
• Traumatic
• Procedure related
Myocardial infarction
Pericardial tamponade
Pulmonary edema
Shock
• Cardiogenic
• Septic
• Hypovolemic
Stroke
Hypoglycemia

TABLE 2

Immediate Treatment of Suspected Air Emboli

1. Place patient in left-sided Trendelenburg position if not contraindicated.
2. Occlude entryway of passive air.
3. Administer oxygen at 100%.
4. If applicable, attempt to aspirate air from catheter.
5. Monitor vital signs.
6. Notify physician for further instructions.

trimming her hair. The remaining lumen portions could not be clamped, and there was no time for catheter removal. The nurse applied a wet cloth over the exposed lumens to prevent further air entry.

At all times, monitor and support vital signs. If the patient is not in an acute care setting, emergency medical services should be notified and transfer should be arranged. Administer oxygen via mask at 100%.[10] The high concentration of oxygen works directly to reduce the size of the air bubble. Because the bubble is composed of atmospheric air, it contains a high nitrogen concentration. Nitrogen does not readily release, so absorption of the air bubble is slow. When superconcentrated oxygen is administered, nitrogen diffuses out of the bubble and oxygen diffuses in. The resulting high-oxygen bubble is more readily absorbed into the system.

If a central catheter is in place, attempts to withdraw the air may provide some relief from symptoms. It is unlikely, though, that all air will be removed, so other supportive treatment should continue. Closed-chest cardiac massage has sometimes been found beneficial in breaking apart the emboli to facilitate diffusion.[22,25]

Arterial Embolism

Having discussed the potential that, through a patent foramen ovale, venous air can enter arterial circulation, keep in mind that air can directly enter the arterial system through an arterial catheter. The reported incidence is small and seems more commonly related to routine blood pressure monitoring.[27] However, incidence related to specialty procedures, such as cardiac catheterization, has been reported.[28] A common culprit is flushing the arterial catheter with an improperly primed line. An alternate cause is sudden release of pressure against the bag, which causes abrupt expansion of air volume.[29(p35)]

The infusion of a large air embolism is usually disastrous. Symptoms will indicate end-artery occlusion, and tissue ischemia and necrosis will be rapidly obvious in the affected area. Although any organ may be affected, embolism to the heart or brain will yield particularly deleterious results, leading to potentially fatal stroke or infarction. There is little that can be done to reduce or eliminate the embolism, although hyperbariatric therapy may be of some use.

Small infusions of air may be clinically silent. The exact amount of air needed to cause a significant embolism has been estimated in tests with animal models. Studies have shown that less than 2 mL resulted in tissue ischemia.[28]

CASE HISTORIES AND PREVENTION STRATEGIES

Catheter-related bolus air embolism is considered an always-preventable event. However, the opportunities for air entry are diverse, and subtle transgressions in care can often lead to lethal air influx. (See Table 4 for strategies to reduce risk of air entry.)

There are challenges regarding the recognition and treatment of air embolism. The reason for air entry may not be intuitively recognized. The diagnosis itself may not be intuitive because symptoms can mimic so many

TABLE 3

Uses and Exclusions of Patient Positioning for Treatment and Prevention of Air Embolism

Trendelenburg—head-down position (preferred)
Supine—back lying, face up
Left lateral decubitus—back lying, left side
• Reduces gradient between atmospheric air and central vasculature
• Not recommended in the presence of increased intracranial pressure, after eye surgery, or in severe pulmonary or heart disease
Other exclusions may apply
Semi-Fowler's—back lying, head at 15°-30°
Fowler's—back lying, head at 45°-60°
High Fowler's—back lying, head at 80°-90°
• Increase gradient between atmospheric air and central vasculature
• In known presence of elevated intracranial pressure, semi-Fowler's is preferred for treatment and prevention of air embolism
• Avoid Fowler's and high Fowler's when
○ Inserting or removing lines
○ Providing maintenance care that may open line to air

other disorders. Even when air embolism is suspected, the Trendelenburg position may not provide resolution of symptoms. Alternate forms of treatment may not be timely or effective.

Therefore, recognizing practice issues that have been associated with air embolism is important to reduce the risk. The following case histories may provide insight into specific causes, looking at both common and rare etiologies and preventive strategies.

Central Catheter Insertion

A 7-month-old girl was sent to the operating room for insertion of a Broviac catheter. The patient was taken out of the Trendelenburg position prior to insertion of the catheter into the peel-away sheath. As soon as the dilator was removed from the sheath, air was audible entering the pathway. Over the next several seconds, the girl's blood pressure plummeted to 38 mm Hg, and oxygen saturation fell to 17%. Advancement of the Broviac catheter was completed, and the line was used to withdraw air from the right atrium. She was placed back into the Trendelenburg position and, after several minutes of additional therapy, recovered fully.[30]

Air embolism during central catheter insertion is associated with 0.13% to 0.5% incidence. The risk seems to be most significant when placing tunneled catheters and occurs particularly when the peel-away sheath is exposed for insertion of the catheter. Mortality at this junction has been reported at 23% to 50%.[16]

For other catheters, the internal jugular (IJ)[6] or subclavian approach[31] has significant risk, whereas femoral insertions and peripherally inserted central catheters are less prone, in the latter case because of the relative positioning of the vessel below the thoracic cavity. However, absence of case histories does not equate with absence of occurrence because air embolism is not always readily diagnosed.

Positioning and monitoring are keys to safe central line insertion. If the IJ or subclavian approach is used, Trendelenburg is the recommended position throughout the procedure. By positioning the head down, there is no longer a pressure gradient between the intrathoracic space and atmospheric air, so the risk of air embolism is greatly reduced. Patients with elevated intracranial pressure are unlikely to tolerate the Trendelenburg position, however. Horizontal or any head-down positioning to establish central venous access puts these individuals at risk of cerebral herniation.[6] Placing these patients at a 30° dorsal position is recommended, but the increased risk for embolism must be noted.

Despite positioning, at all critical junctions, a positive air gradient must be ensured. The greatest risks for air entry occur when there is exposure to atmospheric air, which occurs during dilation, connection of a syringe, or insertion of the catheter into the peel-away sheath or over a guidewire. Health care workers need to minimize these exposure times and take precautions. For anesthetized patients ventilated by use of a bag-valve device, positive pressure is obtained on inspiration.[32]

If the patient is awake and capable of assisting, Valsalva's maneuver is recommended. Valsalva's maneuver is an attempt to expel air with a closed glottis (airway) and is performed by attempting to forcibly exhale while keeping the mouth and nose closed. The technique can be used to clear ears when they become clogged, and the phrase *bear down* is often used to describe the process. The strain phase of Valsalva's maneuver increases intrathoracic pressure, which decreases right atrial filling so that air cannot be readily drawn into the chamber. As soon as Valsalva's maneuver is stopped, however, blood rushes to fill the right atrium; so, premature release during critical junctions can be devastating. If Valsalva's maneuver is to be used, patient teaching should be done before starting the procedure to ensure the patient understands how to perform the technique and is capable of doing so.[33]

TABLE 4

Strategies for the Prevention of Catheter-Related Air Embolism

Note: Disease-specific considerations may contraindicate some recommendations.
Catheter insertion and removal
Positioning—supine or Trendelenburg position if tolerated
Alternative: 30° head up may be required in some conditions
Breathing—have patient hold breath or perform Valsalva's maneuver during insertion, when catheter is open to air, and during removal.
• Valsalva's maneuver may be contraindicated for some conditions.
• If patient is receiving positive-pressure ventilation, risk of air entry is decreased during expiration.
Place a petroleum-based gauze or gel (eg, triple antibiotic) over insertion immediately after removal to ensure that dressing is air occlusive.
• Be sure this equipment is at the bedside in the event of unanticipated line dislodgment.
Maintain air-occlusive dressing until epithelialization is complete (24+ hours).
Catheter use
Expel all air from IV systems (tubing, syringes) before attaching to the patient.
Do not leave tubing attached to fluid but unprimed at the bedside if it is intended to be connected.
When connecting subsequent fluid bag to IV tubing, purge residual air from primary or secondary tubing.
Close roller clamps before puncturing fluid bag to prevent inadvertent air entry into the bag.
Avoid using open vented tubing or alternate forms of venting with collapsible fluid bags. Recognize that vented, rigid containers are at risk for air entry.
Use luer-lock connections to reduce accidental disconnection of tubing.
Avoid the use of evacuated containers during phlebotomy; use gravity-flow bags instead.
Examine all equipment for cracks or leaks that may allow for ingress of air. Cracks on the catheter hub are a common source of air entry.

Abbreviation: IV, intravenous.

A study by Wysoki[34] verified that positive pressure is more difficult to produce when methods other than Valsalva's maneuver are used for increasing intrathoracic pressure. Only 75% of test subjects attained positive pressure by simply holding their breath without closing the glottis; 80% were successful while humming. But Valsalva's maneuver was associated with nearly 100% success in attaining positive pressure. The technique should be avoided, however, in patients with severe coronary artery disease, a history of recent myocardial infarction, or a severe reduction in blood volume.

Removal of a Central Line

A 36-year-old, previously healthy man being treated for a gunshot wound received therapy through a right subclavian catheter. The course of his recovery was noneventful. The catheter was removed on the third day with the patient in a semierect position, and a nonocclusive gauze dressing was placed over the insertion site. Within 10 minutes, the patient complained of severe dyspnea and demonstrated respiratory and cardiac decline. Despite vigorous resuscitative efforts, he died.[20]

Air embolism during or after removal of a central line is one of the most common air embolism events documented. Two factors dominate literature on causation: failure to place the patient in a supine or Trendelenburg position during removal and failure to provide an occlusive dressing over the site.

Removal precautions are similar to insertion precautions because of the risk of air entry at the critical junction when the cannulated vessel is exposed to air. The Trendelenburg position is recommended when tolerated, and Valsalva's maneuver should be employed as the catheter is removed and the dressing is applied. A study by Ely et al[20] verified poor compliance in a large intensive care unit despite a onetime education session provided as part of that study. Because the recognized incidence is low, basic safe practice techniques were rarely in place despite the subspecialty of the physician or the education and experience of the nurse.

The second area of concern relates to the subcutaneous tract that forms as the catheter resides in the vessel. With brief placement of a central line, removal is accompanied by a collapse of surrounding tissue, which seals the entry. As the length of dwell increases, a

fibrinous tract forms that conjoins the vein lumen and atmosphere.[35] Tract formation may occur as soon as 24 hours after placement.[15] In the absence of an occlusive dressing, air entry into the tract can occur. The tract left by a 14-gauge catheter may allow for entry of 200 mL of air per second.[17] Conditions that increase the likelihood of this are placement in a sitting position, deep breathing, and coughing.[15,35] Even if the tract is partially epithelialized, a strong cough can separate the tissue and allow for air ingress.[15]

An occlusive dressing is unlikely to be formed with only the use of gauze and tape. Most medical adhesives on the market, including paper, silk, foam, adhesive, pink, and plastic tapes, do not possess occlusive qualities. Transparent, semipermeable membranes are also not air occlusive. One of the few medical adhesives that boasts this quality is Blenderm (3M Medical). If tape is being used as the predominant means of forming an occlusive dressing, be sure the tape is capable of performing the task.

The use of gel-based antibiotics or petroleum jelly-based gauze at the insertion site will also ensure an occlusive dressing. These products are not always readily available on the unit. It is advised that the products needed to make an occlusive dressing be ordered prior to line insertion and that they be placed in an easy-access location for use in the event of premature catheter dislodgment.

Peripheral IV Access

A 4-week-old, previously healthy newborn was started prophylactically on IV fluids for a fever of 39°C. Vital signs were otherwise normal with no indications of respiratory distress or hemodynamic impairment. Health care workers inserted an IV catheter and started fluids via slow gravity drip. Approximately 1 minute after the infusion was started, the infant became cyanotic, and grunting respirations were noted. Heart rate and respiratory rate decreased, and the patient's blood pressure could not be measured. It was simultaneously noted that the IV tubing had not been primed prior to connection; the tubing was removed, flushed, and reattached. Oxygen and closed cardiac massage were initiated. After several minutes, symptoms resolved, and the infant recovered fully.[36]

Although the passive ingress of a large air bolus is unexpected through small-bore catheters, forced entry can occur despite catheter size. Priming errors are a common cause of air entry. The amount of fluid required to purge tubing depends on the tubing type and manufacturer, but it may exceed 20 mL. Best practice is to always purge tubing, even if the fluid is not to be immediately attached to the patient. It is easy to become distracted and forget this important step if it is left for later.

Peripheral IV insertion is a rare cause of air embolism because the catheter is usually inserted with the limb below chest level. Keep in mind that a 14-gauge catheter can allow for entry of 100 mL of air per second. Smaller catheters can also allow for ingress of lethal air amounts. If the catheter is positioned above the heart, the risk is increased. When removing the stylet or changing the tubing, syringe, or needleless connector, maintain pressure at the distal tip of the catheter.

External jugular catheters are considered peripheral access and are at greatest risk for air entry of all peripheral lines. The same precautions for insertion and removal of central lines should be considered.

Intraosseous Infusion

A 7-month-old girl, known to the hospital for issues surrounding prematurity, was admitted to the emergency room. She was nonresponsive on admission, and attending staff made a tentative diagnosis of food aspiration. An intraosseous device was placed during resuscitation to gain venous access. Despite rigorous resuscitative attempts, the child died. Although the cause of death was inconclusive, the probability of fatal air embolism secondary to the intraosseous needle was strongly assumed on the basis of autopsy results.[37]

Intraosseous infusion has become increasingly popular in recent years to provide fluid resuscitation when peripheral or central access is limited. It is particularly popular in children, and the overall complication rate is reported at 1%.[37] Access to the marrow is obtained using an intraosseous infusion needle or Jamshidi bone marrow needle, which is inserted into an approved bone (often the tibia, fibula, or iliac crest) to the cortex. Because the bone marrow cavity is continuous with venous circulation, fluids and medications rapidly enter the system.[38] The risk, presentation, and treatment for air embolism are the same as with any venous access device.

Inadvertent Disconnection

A 60-year-old female had immediate respiratory and cardiovascular deterioration when the tubing connected to her IJ catheter was inadvertently separated.[39] A 26-year-old female suffered bilateral vision loss and hemiparesis after her catheter became disconnected from parenteral nutrition.[40] A 62-year-old female on total parenteral nutrition died when her catheter disconnected as she positioned herself in bed.[41]

By far the greatest incidences of air embolism surround the issue of normal catheter use and unintentional disconnection. One of the most publicized cases occurred in 1991. A 39-year-old female underwent surgery for ulcer repair and received fluids postoperatively through an IJ line with friction- (slip-) tipped connectors. When

the line became dislodged, she suffered a seizure, resulting in brain damage. She died 4 years later, never having regained consciousness. The resulting lawsuit was settled in 2002 and had a profound effect on the selection of luer-lock fittings over the use of tape and other connectors for securement.[42]

Luer-lock technology was patented in 1925 by Fairleigh Dickinson Sr, cofounder of Becton Dickinson Company.[43] It was decades, however, before the technology became universally available. Even after luer-lock technology became more accessible, the caveats of using friction- (slip-) tip technology were not fully recognized, and the luer-lock design was underused, probably because of cost. Today, it is the standard of practice to use luer-lock fittings on all patients.[44]

Use of Collapsible Bags

A 39-year-old male experienced sudden collapse when his IV bag was changed while he sat in a chair. Because the roller clamp remained open during the bag change, air traveled down the tubing and into his catheter.[45]

A 56-year-old male had sudden-onset bradycardia, hypotension, and hypoxia during surgery for kidney transplantation. Fluid and medication administration was by means of a right IJ catheter. During resuscitation, it was noted that the IV tubing was full of air, and an abnormally large amount of air was also present in the saline bag. Health care workers realized that the bag of saline had been removed for the addition of mannitol and was then reconnected. Such a significant amount of air had entered the saline bag during the disconnection that pressure within the bag was greater than atmospheric pressure, and air was able to continue to flow after the fluid had emptied.[46]

Flexible, closed-system IV containers are designed to hold a specific amount of fluid along with a small amount of prefiltered air, which assists in drainage. To avoid entry of atmospheric air to the container, the bag should be punctured with the IV roller clamp closed; the use of opened, vented tubing or alternate methods of venting should be avoided. If the container is not manipulated during use, the amount of air should remain steady.

Tubing should always be purged of air between bags if the drip chamber has been allowed to empty. Residual air will be pushed into circulation as the new infusion begins. Also keep in mind that if a pierced bag is open to air before infusion is complete, it will reexpand. In this event, residual air may be sufficient for significant embolism potential. The use of infusion pumps with air-detection alarms and/or air-eliminating filters reduces the risk of air entry but does not eliminate the need for caution.

Air may also enter the system when bags are "piggybacked" together for sequence or concurrent infusion. If 2 bags are suspended the same distance off the floor, then as 1 bag empties the remaining air will be gradually sucked into the adjacent bag and infused.[47] Note that this does not occur when administering a bolus that is positioned higher than the primary bag. In this case, air will flow from the empty secondary bag into the tubing but only to the level of the primary fluid. Then, the primary fluid bag, with the advantage of gravity, will resume flow.

Equipment Failure

A 60-year-old man was recuperating well from multiple trauma sustained in an automobile accident. Shortly after attending personnel began weaning him from positive-pressure ventilation, the patient's condition started to deteriorate. Clinical examination revealed air entering the patient's circulation via a fracture in the hub of the central venous catheter that had been in place for 10 days.[41]

Despite care in the insertion and removal of IV catheters, air embolism can occur because of the design of tubing and bags, secondary to unrecognized cracks, fractures, or leaks, or any time there is a breach in the closed system. Fluid containers should always be visibly inspected prior to addition, with the bags gently squeezed to discern invisible fractures. All equipment should be examined periodically to ensure that connections are intact and that no unexplained leaks or bubbles are detected.

Blood Administration

A 70-year-old female with rectal carcinoma received a rapid infusion of 1 unit of packed cells via a right subclavian triple-lumen catheter. To facilitate flow, the blood was placed within a manually inflated pressure bag. The patient's oxygen saturation suddenly decreased to 50%, and she became hypotensive. It was noted that the blood bag was empty and the entire tubing to the central line was full of air. She was immediately resuscitated successfully, and her remaining hospital course was uneventful.[48]

Although blood is stored in air-free containers, atmospheric air can enter the system when the bag is punctured for use or when the drip chamber is squeezed. The amount of air is likely to increase if a back prime method is used to dilute the blood with saline prior to administration. (Note: The practice of diluting blood prior to infusion is not endorsed by the author and is mentioned only because the practice is known to exist and may have repercussions of air embolism.)

The use of pressure to infuse blood may add to the risk of air embolism if any air has been introduced to the bag. According to the laws of thermodynamics, the amount of air within the bag will remain constant as

long as the pressure remains constant. However, when pressure is suddenly negated, as occurs when the blood bag empties, air expands and the volume increases. (This will be seen with pressurized fluid administration as well.) As long as the air pressure within the bag is greater than the air pressure in the atmosphere, air will continue to flow into the tubing.[29(p35)] The risk of significant air embolism is dependent on the amount of air in the bag.

Another threat for potential air entry during transfusion is with the use of blood warmers.[49] Warming bladders may have a capacity to hold up to 75 mL. Although the tubing has provision for air elimination, improper priming or use can result in significant air embolism.

Phlebotomy

A patient undergoing therapeutic phlebotomy presented with unremarkable vital signs at the onset of the procedure. An evacuated bottle was used for the draw. Phlebotomy proceeded well for the first 75 to 100 mL of blood, but then flow stopped. As instructed, the patient began to open and close his fist to enhance flow, but this was unsuccessful. He then repositioned himself in his chair, and the tourniquet loosened from his arm. Reverse flow from the bottle and tubing began, and he became immediately symptomatic. Resuscitation was unsuccessful. On autopsy, 40 mL of air was measured in his brain.[50]

Evacuated containers were developed in 1936 by the Baxter Corporation and were used for fluid and blood administration as well as for blood collection.[51] By the 1940s, the risk of air embolism secondary to phlebotomy was well documented, and the mechanism of air entry had been determined.[52] Donating blood using an evacuated container carried a serious risk of death or impairment. The invention of plastic bags in 1953 by the Fenwall Company made blood collection and storage safer and more sophisticated for blood banking purposes, but the use of evacuated containers for therapeutic phlebotomy continued.

Overall, data on blood donation safety are provided by the AABB (formerly American Association of Blood Banks) and are specific to allogeneic donors. Understandably, with the invention of plastic containers, the reported cases of air embolism virtually disappeared, and the risk today is ignored in discussions of the legal aspects of blood banking and of proper phlebotomy room techniques.[51] Many facilities, however, continue to use evacuated containers for therapeutic phlebotomy. Medical experts have been unable to cite an advantage to the use of evacuated containers, and the risk of air embolism is unchanged in today's environment.

When using evacuated containers, the vacuum can be inadvertently broken during normal procedures. As blood enters a non–vacuum-sealed bottle, air pressure builds up. The significance of the pressure depends on the size of the bottle, the amount of blood that has been withdrawn, the distance of the bottle below the arm, and the patient's blood pressure.[52] Reverse flow (ie, air from bottle retrogrades into the needle) can be achieved even with the tourniquet in place, but the risk becomes explosive if the tourniquet is released while the needle remains open and in the patient's arm. In a 1982 letter to the editor of the *New England Journal of Medicine*, Chwirut[50] asked, "Why are glass bottles still used for therapeutic phlebotomy? Given the potential for accidents or negligent misuse, is the use of evacuated bottles for phlebotomy appropriate?"

CONCLUSION

Most nurses will never report seeing any air embolism events during their careers. Yet, the fact that such an event has not been reported does not mean that one has not occurred—only that it has not been recognized. Any entry into the vascular system causes an at-risk condition for air embolism. Nurses must recognize potential signs and symptoms and be familiar with recommended interventions. Should air embolism be suspected, they will need to act quickly and decisively. Early intervention will save lives and reduce negative outcomes.

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