



Integrated Medical and Nursing Perspectives in the Emergency Management of Venous Gas Embolism: A Multidisciplinary Clinical Approach

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Abstract

Background: Venous Gas Embolism (VGE) is a critical condition characterized by the entrainment of gas into the venous system, often due to iatrogenic causes like central venous catheterization and neurosurgery. It can lead to right ventricular outflow obstruction, cardiovascular collapse, and, via paradoxical embolism, systemic end-organ ischemia.

Aim: This article aims to synthesize an integrated, multidisciplinary approach to the emergency management of VGE, emphasizing the collaborative roles of medicine and nursing in rapid diagnosis, stabilization, and treatment to improve patient outcomes.

Methods: The review examines the pathophysiology, etiology, and clinical presentation of VGE. It evaluates diagnostic modalities, including continuous capnography and echocardiography, and details a structured management protocol involving immediate source control, patient repositioning, hemodynamic support, and aspiration of intracardiac air.

Results: VGE presents with a spectrum of symptoms, from subtle changes in end-tidal CO₂ to fulminant cardiovascular collapse. Key interventions—administering 100% oxygen, placing the patient in the left lateral decubitus position, and aspirating air via a central venous catheter—are time-critical. Hyperbaric oxygen therapy is a crucial adjunct for severe cases, particularly those with neurological involvement.

Conclusion: Successful management of VGE hinges on a high index of suspicion and immediate, coordinated action by a multidisciplinary team. A protocolized response that integrates medical and nursing expertise is essential to mitigate this life-threatening emergency's high morbidity and mortality.

Keywords: Venous Gas Embolism, VGE, Emergency Management, Multidisciplinary Team, Hyperbaric Oxygen, Right Heart Failure.

Introduction

Venous gas embolism (VGE) is defined as the entry and intravascular persistence of gas within the systemic venous circulation, where intraluminal bubbles behave as embolic particles capable of obstructing pulmonary outflow and provoking endothelial injury, ventilation-perfusion mismatch, and hemodynamic instability [1]. The pathophysiology centers on a pressure gradient that favors gas entrainment across a disrupted or open venous interface—such as a nonoccluded catheter hub

or surgical venous sinus—followed by bubble coalescence and transit to the right heart and pulmonary arterial tree. Consequences range from subclinical elevations in pulmonary artery pressure to fulminant cardiovascular collapse when the bubble load overwhelms pulmonary filtration or induces acute cor pulmonale and arrhythmias [1][2]. While the pulmonary vasculature is the usual sink for venous bubbles, right-to-left shunts—patent foramen ovale or intrapulmonary vascular communications—permit paradoxical embolization with end-organ ischemia,

thereby expanding the clinical spectrum to neurologic and myocardial injury even when the source is venous [2]. Epidemiologically, VGE is predominantly iatrogenic, with prototypical scenarios including central venous cannulation and manipulation, neurosurgical and otolaryngologic procedures performed above the level of the heart, thoracentesis, hemodialysis circuit breaches, and exposure to high airway pressures during mechanical ventilation that transiently elevate intrathoracic gradients and favor venous air ingress [1]. Traumatic mechanisms—blunt or penetrating chest injuries—can similarly disrupt venous integrity and serve as portals for gas entry, and clinically relevant VGE is also recognized after radiocontrast injection for computed tomography, most often when injection pressures or catheter positioning facilitate intravascular air entrainment [2]. Outside the hospital, diving-related barotrauma remains an established precipitant, in which decompression generates intravascular inert-gas bubbles that seed the venous system and impose a cardiopulmonary burden proportional to the ascent profile and tissue gas load [1]. Although a substantial proportion of episodes remain clinically silent due to efficient pulmonary clearance, symptomatic VGE constitutes a medical emergency: rapid recognition and immediate interventions—air source control, patient repositioning, cardiopulmonary stabilization, and consideration of hyperbaric therapy—are essential to forestall progressive right ventricular failure, refractory hypoxemia, and death [1][2].

Etiology

Venous gas embolism (VGE) is a multifactorial clinical event that arises whenever there is a direct communication between the venous system and an external gas source coupled with a pressure gradient favoring gas entry. Approximately 90% of all VGE cases are iatrogenic, underscoring the importance of meticulous procedural technique and preventive vigilance in hospital and surgical environments [1]. The majority of these incidents stem from medical or surgical interventions that transiently expose the venous circulation to atmospheric air or medical gases. The risk is particularly elevated in operations where the operative site is situated above the level of the right atrium, such as procedures performed in the sitting or semi-sitting (Fowler) position, because gravitational gradients facilitate air entrainment into open veins [2]. Within neurosurgical practice, posterior fossa and cervical spinal surgeries performed in the sitting position represent the prototypical high-risk settings. During such operations, venous pressure within dural sinuses can fall below atmospheric levels, permitting substantial air entry if venous sinuses or emissary veins are inadvertently opened. Indeed, neurosurgery accounts for a significant proportion of reported VGE cases, particularly in posterior cranial fossa interventions, where the incidence can reach 40–45% when sensitive intraoperative Doppler monitoring is used [3].

Minimally invasive neurosurgical procedures such as deep-brain stimulation, stereotactic biopsies, and endoscopic cranial approaches can also precipitate VGE, albeit often in smaller volumes, because even brief exposure of cortical veins to negative pressure is sufficient for air entrainment. Similarly, craniostomy repair, particularly in infants and young children, is a frequent pediatric scenario given their small blood volume and fragile venous networks [4].

Beyond the brain, a wide array of surgical and interventional specialties carry procedural risk. In orthopedic surgery, cemented hip arthroplasty and spinal instrumentation are known to generate intramedullary pressure surges that can force air or fat into open venous channels. In obstetric and gynecologic practice, placental removal, cesarean delivery, and pregnancy termination can introduce air into disrupted uterine sinuses, while laparoscopic interventions employing carbon dioxide insufflation under positive pressure may allow gas migration into damaged venous plexuses or through microtears in the peritoneum. Even relatively delicate procedures, such as vitrectomy and ophthalmic microsurgery, have been implicated, as venous connections in the orbital region can serve as potential conduits for air under certain intraoperative pressure conditions [4]. Catheter-related events represent another dominant etiologic category. Insertion, manipulation, or removal of central venous catheters (CVCs) remains one of the most frequent medical causes of VGE, often linked to preventable lapses in technique. When the catheter hub or needle port is open to the atmosphere, air can be entrained rapidly, particularly if the patient takes a deep inspiration or is positioned upright, further lowering intrathoracic venous pressure [5]. Disconnections in pressurized infusion or contrast injection systems, as seen during contrast-enhanced CT scans, are increasingly recognized iatrogenic causes. The volume of air required to induce hemodynamic compromise is relatively small—estimated at 100 to 300 mL in adults—making meticulous attention to line integrity and connection security imperative [5].

In addition, positive-pressure ventilation can contribute to the development of VGE, especially when alveolar overdistension or high positive end-expiratory pressures disrupt alveolar–capillary interfaces, permitting microbubbles to enter the pulmonary venous system. Intraoperative irrigation with hydrogen peroxide is another potential cause, as its catalytic decomposition liberates oxygen gas capable of intravascular migration. Likewise, penetrating and nonpenetrating thoracic injuries, including blast or missile wounds, can facilitate direct air entry into major veins or the right heart chambers when venous integrity is compromised [5]. Outside of iatrogenic and traumatic contexts, environmental and occupational exposures, most notably deep-sea diving, remain an important etiologic domain. During descent,

inert gases such as nitrogen dissolve in the bloodstream and tissues under high ambient pressure. If ascent occurs too rapidly, the declining environmental pressure exceeds the rate at which dissolved nitrogen can be safely eliminated through respiration, resulting in intravascular bubble formation within the venous system [6–10]. These gas emboli, initially microscopic, may coalesce into larger bubbles that obstruct pulmonary arterioles, impair gas exchange, and trigger an inflammatory cascade involving endothelial activation and platelet aggregation. The condition, a central feature of decompression sickness, exemplifies the same mechanical and physiologic principles as iatrogenic VGE, albeit driven by ambient pressure changes rather than direct air entry.

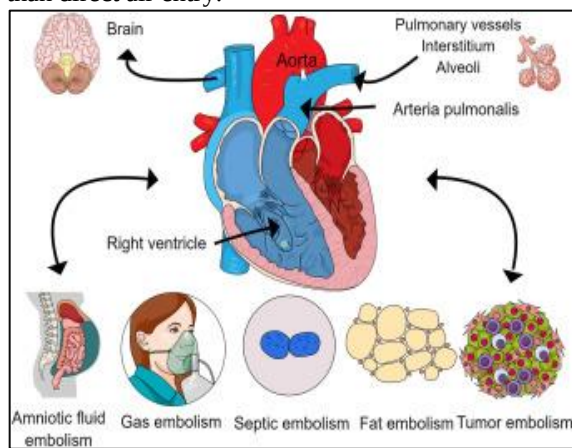


Figure-1: Gas embolism.

Pathophysiologically, all these mechanisms share two prerequisites: (1) a communication between a gas source and the venous circulation, and (2) a pressure gradient that drives gas into the vessel lumen. Once introduced, gas bubbles can expand or migrate depending on ambient pressure, body position, and respiratory mechanics, potentially crossing to the arterial side through right-to-left shunts such as a patent foramen ovale, creating paradoxical embolism. Moreover, the hemodynamic consequences depend on the volume, rate of entry, and gas composition. Large volumes of air or carbon dioxide entering rapidly overwhelm pulmonary filtration capacity, causing acute right ventricular outflow obstruction, while smaller, chronic microembolic loads may remain subclinical yet still provoke subtle hypoxemia or endothelial dysfunction. In summary, the etiology of venous gas embolism is dominated by iatrogenic factors associated with modern medical and surgical interventions, though traumatic and environmental exposures remain relevant contributors. The condition's pathogenesis underscores the delicate balance between venous pressure dynamics and procedural technique. Preventive strategies must therefore focus on controlling patient positioning, maintaining closed intravascular systems, vigilant line management, and adherence to decompression protocols in divers. Understanding the diverse

etiologic mechanisms not only facilitates timely recognition and management but also informs preventive practices that can substantially reduce morbidity and mortality associated with this potentially catastrophic event [1–10].

Epidemiology

The epidemiology of venous gas embolism (VGE) reflects both the expansion of invasive medical practice and the increased sensitivity of contemporary monitoring. As the volume and complexity of procedures involving central venous access, endovascular manipulation, laparoscopic insufflation, and advanced neurosurgical positioning have risen, so too has the opportunity for gas entrainment into the venous circulation. Parallel improvements in intraoperative surveillance—particularly continuous end-tidal carbon dioxide (EtCO₂) monitoring and precordial or transesophageal Doppler ultrasonography—have augmented case ascertainment, revealing subclinical or transient embolic episodes that would previously have escaped notice [9]. Consequently, reported rates vary widely across settings and study designs, with detection heavily influenced by the monitoring modality employed, the definition of clinically significant events, and the rigor of postoperative surveillance [10]. Importantly, these methodological differences complicate attempts to define a singular incidence figure; most episodes remain occult because small embolic loads are rapidly filtered by the pulmonary circulation and produce no overt hemodynamic or respiratory symptoms, thereby generating a large “iceberg” of undetected VGE beneath the clinically apparent cases [9]. Risk is not uniformly distributed across clinical domains. Neurosurgical procedures consistently exhibit the highest detection rates, a pattern attributable to several convergent anatomic and physiologic factors. Upright or semi-sitting positioning lowers intracranial venous pressures below atmospheric levels, creating a favorable pressure gradient for air entrainment through noncollapsed venous channels; the cranial venous system also lacks valves and is relatively noncompressible compared with peripheral veins, further facilitating continuous ingress when venous sinuses are exposed [10]. The close vertical relationship of the operative field to the right atrium amplifies this gradient, such that even minor lapses in hemostasis or brief exposure of emissary veins can yield detectable Doppler signals and, in some cases, clinically relevant declines in EtCO₂ or oxygenation [9][10]. By contrast, specialties in which patients are supine and venous structures are easily compressible typically report lower VGE frequencies unless additional drivers—such as high insufflation pressures, pressurized injectates, or large-bore venous access—are present [9].

Population characteristics and procedural context add further nuance to epidemiologic patterns.

Pediatric cohorts may exhibit higher detection sensitivity during craniofacial and craniostylosis surgery because of smaller circulating volumes and the proximity of emissary veins to the operative field, while adult populations accrue risk through cumulative exposures to catheter-based interventions and complex oncologic or vascular procedures [10]. Outside the operating room, surveillance in intensive care has identified sporadic VGE linked to central line manipulation and high-pressure ventilation, although many of these events are brief and self-limited when recognized promptly. In nonmedical environments, the epidemiology in diving communities remains closely tied to ascent profiles and adherence to decompression protocols, with modern dive computers and training programs mitigating, but not eliminating, venous bubble loads detectable by Doppler after routine dives [9]. Taken together, current evidence suggests that the apparent rise in VGE incidence reflects a combination of genuine exposure growth and enhanced diagnostic vigilance, while the true burden remains underestimated due to the predominance of subclinical events. Ongoing standardization of monitoring thresholds, reporting criteria, and postoperative follow-up is therefore essential to refine incidence estimates and enable meaningful comparisons across institutions and procedural categories [10].

Pathophysiology

The pathophysiology of venous gas embolism (VGE) is governed by a triad of essential conditions: the presence of a gas source, a patent communication between the gas and the venous circulation, and a pressure gradient that favors gas entrainment into the vasculature [11]. When these elements coexist, air is drawn into open venous channels where the intraluminal pressure falls below atmospheric pressure. Factors that accentuate this process include hypovolemia, which lowers central venous pressure; upright positioning exceeding 45°, which amplifies the hydrostatic pressure gradient between the surgical site and the right atrium; and positive-pressure mechanical ventilation, which can intermittently generate large intrathoracic pressure shifts that promote bubble formation and retrograde flow of air [12]. Experimental studies have demonstrated that even a subatmospheric gradient as small as 5 cm H₂O is sufficient to initiate venous air ingress [13]. Once air enters the venous circulation, its physiological impact depends primarily on the rate and total volume of gas introduced, as well as the anatomic location of entry relative to the heart. Small volumes of air may be filtered harmlessly by the pulmonary capillary bed, whereas rapid entrainment of large volumes produces abrupt hemodynamic collapse. Symptomatic VGE generally occurs when air volumes exceed 5 mL/kg, while injection of as little as 1–2 mL into the central nervous system (CNS) circulation can prove fatal. Similarly, intravascular air as small as 0.5 mL entering the coronary arteries can precipitate

myocardial ischemia and ventricular fibrillation by mechanically obstructing perfusion to conduction tissues [14]. When air gains direct access to the central venous circulation, particularly via large-bore catheters or open venous sinuses near the heart, it travels rapidly to the right atrium and ventricle. Larger boluses—typically between 50 and 100 mL—can obstruct right ventricular output by forming an air lock, in which a frothy admixture of blood and air accumulates near the pulmonary outflow tract, impeding right ventricular ejection and drastically reducing cardiac output. Volumes exceeding 300 mL are usually fatal due to total circulatory arrest [14]. Smaller volumes that escape complete obstruction nevertheless become trapped within the pulmonary arterioles, where they increase pulmonary vascular resistance (PVR), resulting in pulmonary arterial hypertension, right ventricular strain, and eventual acute right-sided heart failure.

On a cellular level, gas bubbles are not inert. They trigger a cascade of biochemical and inflammatory reactions, including endothelial activation, platelet aggregation, and complement-mediated injury. Microbubbles tend to aggregate with neutrophils, fibrin, erythrocytes, and lipid particles, generating a prothrombotic microenvironment that disrupts normal capillary perfusion and increases alveolar-capillary membrane permeability. This culminates in interstitial pulmonary edema, diffuse alveolar damage, and impaired gas exchange, further compromising systemic oxygenation. Additionally, free radicals released during bubble-endothelium interactions exacerbate endothelial dysfunction and perpetuate vascular leakage. A particularly serious consequence of venous air entry is paradoxical embolism, which occurs in approximately 14% of documented cases [15]. This phenomenon arises when air crosses from the right to the left side of the circulation through a patent foramen ovale (PFO), ventricular septal defect (VSD), or pulmonary arteriovenous malformation (AVM). Once on the arterial side, air emboli can occlude cerebral, coronary, or mesenteric arteries, leading to stroke, myocardial infarction, or intestinal ischemia [16][17]. Even in the absence of a structural shunt, elevated pulmonary arterial pressure caused by VGE can open latent intrapulmonary anastomoses, further promoting right-to-left gas migration. The resulting arterial hypoxemia and hypercapnia are due to increased alveolar dead space and ventilation-perfusion mismatch, which severely impair oxygen delivery to vital organs [18]. When the air burden exceeds the filtration capacity of the pulmonary circulation, arterial gas embolism may occur, even in the absence of anatomic shunts. The extreme distension of pulmonary capillaries and overpressurization may rupture alveolar septa, allowing bubbles to pass directly into the pulmonary veins and systemic arterial tree [19]. Once arterialized, gas bubbles propagate distally until they lodge within arterioles and

capillaries, obstructing perfusion and eliciting localized ischemia and inflammatory microvascular damage.

Moreover, gas bubbles exert systemic hematologic effects by activating the coagulation cascade and promoting microthrombosis. Platelet degranulation releases vasoactive and procoagulant mediators such as thromboxane A₂ and serotonin, which further elevate pulmonary vascular tone and amplify hypoxia. These pathophysiologic mechanisms can culminate in disseminated intravascular coagulation (DIC) in severe or sustained cases. Concurrently, reflex pulmonary vasoconstriction and reduced venous return precipitate systemic hypotension and end-organ hypoperfusion, producing a rapidly progressive cycle of hemodynamic failure. In addition, under specific conditions, gas can travel retrogradely through the jugular venous system due to the low specific gravity of air and the presence of incompetent venous valves. This retrograde migration enables intracranial venous air emboli to form, with potentially devastating neurological consequences, including seizures, altered consciousness, and coma. Even small volumes of air entering the cerebral venous sinuses can obstruct drainage, elevate intracranial pressure, and compromise cerebral perfusion, leading to focal ischemic injury. In summary, the pathophysiological cascade of VGE evolves from a mechanical event—gas entry under a pressure gradient—into a complex hemodynamic, inflammatory, and coagulative disorder. The severity of clinical manifestation depends on the volume and rate of gas entry, the site of introduction, and the presence of intracardiac or intrapulmonary shunts. Ultimately, the resulting combination of mechanical obstruction, vascular inflammation, coagulopathy, and tissue ischemia underpins the potentially catastrophic outcomes of venous gas embolism [11–19].

History and Physical

Venous gas embolism (VGE) is principally a clinical diagnosis, with presentations spanning from clinically silent episodes—estimated in roughly 10% of events—to fulminant, life-threatening crises reported in up to 20% of cases. The breadth of this spectrum is governed by the volume and rate of air entrainment, the anatomic site of entry, and host factors such as cardiopulmonary reserve and the presence of right-to-left shunts. A defining feature is the tight temporal linkage between symptom onset and a precipitating maneuver or exposure, such as intravascular catheter manipulation, neurosurgical positioning, or rapid decompression. Because early manifestations may be nonspecific, recognition requires a high index of suspicion and careful attention to peri-procedural context. The history should elicit the exact timing of invasive steps, patient position, episodes of deep inspiration during line handling, and any abrupt deterioration in hemodynamics or ventilation. Concurrent review of monitoring traces,

including abrupt end-tidal CO₂ decline or oxygen desaturation, can reinforce the diagnostic impression when paired with new cardiopulmonary symptoms. The physical examination often reveals a multisystem pattern reflecting embolic load and distribution. Cardiovascular signs range from new tachyarrhythmias or bradyarrhythmias, jugular venous distension, hypotension, and acute right ventricular failure to anginal chest pain and, in severe bolus entrainment, a characteristic machinery-like mill-wheel precordial murmur. Pulmonary findings include sudden cough, dyspnea, tachypnea, wheeze, rales, cyanosis, mild hemoptysis, and episodic apnea; in larger embolic burdens, rising pulmonary vascular resistance may precipitate profound hypoxemia. Central nervous system involvement spans headache, confusion, seizures, transient focal deficits, ischemic strokes, and coma, particularly when paradoxical embolization occurs via a patent foramen ovale or intrapulmonary shunt. Bedside clues are augmented by supportive signs such as acute drops in end-tidal CO₂, widening alveolar–arterial gradients, and echocardiographic visualization of right-sided intracardiac bubbles or outflow tract obstruction when available. Clinical grading with the Tübingen scale provides a structured method to stratify severity, guide immediate resuscitative priorities, and inform the threshold for adjunctive interventions such as hyperbaric oxygen therapy [20].

Evaluation

The diagnostic evaluation of venous gas embolism (VGE) rests on the synthesis of clinical context with targeted physiologic and imaging data, recognizing that no single test captures the entire spectrum of disease. Continuous capnography provides an early, widely available signal; a sudden end-tidal carbon dioxide (EtCO₂) decline exceeding 3 mm Hg should prompt consideration of VGE when temporally linked to a precipitating maneuver. Because entrained air increases alveolar dead space and impairs pulmonary perfusion, EtCO₂ typically falls while arterial PaCO₂ may rise, producing a widening PaCO₂–EtCO₂ gradient. Measurement of nitrogen in expired air during administration of 100% oxygen adds specificity: persistence or elevation of end-tidal nitrogen implies ongoing venous entrainment or incomplete clearance. End-tidal nitrogen monitoring is highly sensitive and can detect VGE earlier than EtCO₂; however, it may miss minute air loads and can misleadingly suggest resolution as bubbles redistribute or measurement conditions change, underscoring the need to interpret trends rather than isolated values. Bedside examination remains informative. Precordial auscultation may reveal the classic “mill-wheel” murmur during substantial air loads. In massive events, palpable crepitus can occasionally be detected over superficial veins. Invasive hemodynamic monitoring often shows abrupt rises in central venous pressure and pulmonary

artery wedge pressure, reflecting right ventricular outflow obstruction and acute pulmonary hypertension. If a central venous catheter is in place, cautious aspiration of air from the right atrium or superior vena cava can provide immediate therapeutic benefit and additional diagnostic confirmation, although a negative aspiration does not exclude VGE [21]. Laboratory and adjunctive tests frequently demonstrate the cardiopulmonary consequences of embolization. Electrocardiography may show right ventricular strain patterns, new conduction disturbances, or ischemic changes when myocardial perfusion is compromised. Chest radiography, though nonspecific, can reveal interstitial or alveolar edema in larger embolic burdens. Arterial blood gas analysis typically discloses hypoxemia, hypercapnia, and a metabolic acidosis from systemic hypoperfusion. Fundoscopic examination, when feasible, may visualize intraretinal gas bubbles, lending supportive evidence of systemic embolization in severe cases. Cross-sectional imaging assists in defining the extent and consequences of embolization but must be timed thoughtfully given rapid gas resorption. Computed tomography (CT) with lung windows or minimum intensity projection reconstructions can depict vascular air in the chest and, occasionally, air in dural venous sinuses; magnetic resonance imaging (MRI) may reveal watershed infarctions or other parenchymal sequelae of paradoxical embolization. Because the visibility of intravascular gas is governed by both bubble volume and local blood flow velocity, vascular air may be absent at imaging despite compelling clinical and physiologic evidence. CT's speed and accessibility favor its use when imaging is indicated to evaluate complications or exclude mimics [21].

Echocardiography provides the most direct visualization of intravascular gas. Transthoracic echocardiography demonstrating air within the right atrium, right ventricle, or right ventricular outflow tract is considered pathognomonic for VGE and is among the most sensitive modalities, albeit semi-invasive in the perioperative context [21]. Precordial or transesophageal echocardiography can confirm the diagnosis, reveal acute right ventricular dilation and interventricular septal flattening, and estimate pulmonary artery pressures. Bedside point-of-care ultrasound offers rapid, repeatable assessment in unstable patients. Despite their value, precordial and transesophageal Doppler techniques are operator dependent and logistically demanding, limiting universal deployment. Conversely, EtCO₂ trends are nonspecific and correlate imperfectly with embolic volume and clinical severity. Institutions can improve detection by integrating automated threshold alarms for sudden EtCO₂ declines, centralized physiologic signal monitoring, and standardized response protocols that prompt immediate verification, catheter aspiration when appropriate, patient repositioning, and

escalation to advanced imaging or echocardiography as dictated by clinical stability [21].

Treatment / Management

Initial Management and Stabilization

The immediate priorities in venous gas embolism (VGE) are to secure the airway, support ventilation, and stabilize circulation, recognizing that hypoxemia, acute right ventricular (RV) failure, and systemic hypotension can evolve within minutes. High-flow 100% oxygen should be delivered at once to mitigate hypoxemia and to accelerate the washout of nitrogen from intravascular bubbles, thereby shrinking their volume through diffusion gradients and improving ventilation-perfusion matching. Concomitantly, meticulous attention must be paid to halting further air ingress: the operative field should be flooded with isotonic saline to occlude open venous channels; any exposed venous sinuses should be promptly sealed; and insufflation sources, as in laparoscopy, must be discontinued to eliminate the driving pressure for gas entry. Positioning interventions that lower the surgical site relative to the right atrium reduce the hydrostatic gradient that favors venous entrainment, while brief Valsalva maneuvers or manual jugular venous compression can raise central venous pressure transiently and blunt further air ingress when a venous breach cannot be immediately repaired. Early hemodynamic assessment with invasive monitoring, when available, guides titration of fluids and vasoactive agents to sustain coronary perfusion and RV preload in the face of rising pulmonary vascular resistance. In parallel, any use of nitrous oxide should be terminated because its high blood-gas solubility allows rapid diffusion into preexisting bubbles, enlarging their volume and worsening RV outflow obstruction. These measures are undertaken while coordinating definitive actions with the operative team to address the inciting lesion and with critical care teams to maintain cardiopulmonary stability during the vulnerable early phase [22][23][24].

Interventions to Reduce Embolus Volume

The second therapeutic objective is deliberate reduction of bubble size to relieve microvascular obstruction and restore forward flow. Administering 100% oxygen is foundational, both to correct hypoxemia and to accelerate nitrogen egress from embolized gas through alveolar-capillary diffusion, thereby shrinking bubbles in the venous and pulmonary arterial trees. When implemented early, hyperbaric oxygen therapy (HBOT) further exploits Boyle's and Henry's laws to compress bubbles and supersaturate plasma with oxygen, enhancing nitrogen reabsorption and oxygen delivery to ischemic tissues. Although the optimal window is within the first 4 to 6 hours, clinically meaningful benefit may still accrue beyond that period in severe cases with neurological deficits or refractory cardiopulmonary compromise. At the surgical field, continuous irrigation with saline helps disperse surface-adherent bubbles and reduces

the chance of recurrent entrainment by forming a liquid seal over exposed vein. Intermittent bilateral jugular venous compression can be applied in short cycles to increase central venous pressure and discourage additional ingress; however, this maneuver should be balanced against the risk of raising intracranial pressure in neurosurgical patients. Adjustments in ventilatory strategy—careful elevation of positive end-expiratory pressure (PEEP) to maintain alveolar recruitment without provoking alveolar overdistension—may improve oxygenation and reduce shunt fraction, although excessive PEEP can impede venous return and worsen RV preload, necessitating vigilant hemodynamic monitoring. Concurrently, the discontinuation of nitrous oxide is imperative because it diffuses into bubbles and expands them, whereas 100% oxygen contracts them. These physiologic manipulations, combined with field control of the gas source, are synergistic in reducing embolus burden and improving perfusion at the capillary level [22][23].

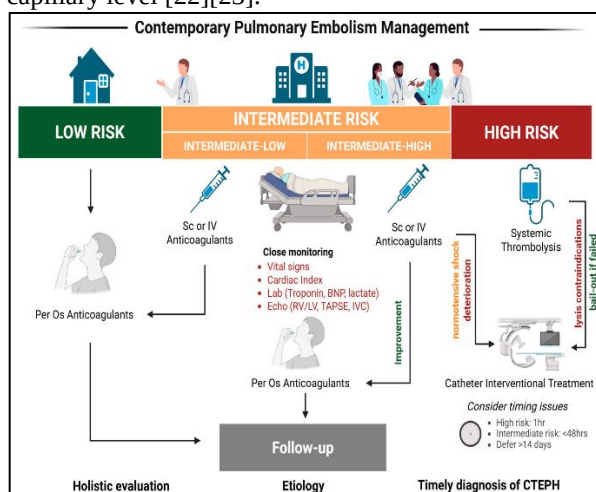


Figure-2: Management of venous gas embolism.

Techniques to Overcome Embolic Mechanical Obstruction

When a bolus of intravascular air accumulates within the right heart, it can create a functional “air lock” that blocks RV ejection and threatens circulatory arrest. Patient positioning into the left lateral decubitus (Durant) and Trendelenburg orientations aims to trap air in the right atrium and RV apex, moving it away from the right ventricular outflow tract (RVOT) and pulmonary valve, thereby restoring an ejection pathway. This maneuver is particularly useful when embolization is recognized intraoperatively and can be executed rapidly before hemodynamic collapse. If a multi-orifice central venous catheter is already positioned with its tip in the right atrium or superior vena cava, gentle aspiration should be attempted to evacuate entrained air; this measure can immediately reduce RVOT obstruction and improve preload. Emergent placement of a new central line in unstable patients, however, is not supported when one is not already present, as

procedure-related delays and additional venous breaches can exacerbate the problem; moreover, pulmonary artery catheters are generally too narrow for effective aspiration of foamy air–blood mixtures [22][23][24]. Pharmacologic augmentation of RV performance is often necessary while mechanical strategies take effect. Inotropic agents such as dobutamine or epinephrine can enhance contractility and support cardiac output against the backdrop of elevated pulmonary vascular resistance, while vasopressors such as norepinephrine may be required to maintain systemic perfusion pressure and coronary flow. Judicious crystalloid or colloid infusion can optimize RV preload, though overzealous volume loading risks septal shift and further compromise of left ventricular filling. If hemodynamic collapse ensues, high-quality chest compressions provide dual benefit: they generate forward flow to sustain organ perfusion and physically fragment or displace air from the RVOT and main pulmonary artery, potentially re-establishing partial flow. In extreme cases refractory to conventional measures, rescue cardiopulmonary bypass (CPB) or surgical thoracotomy with direct pulmonary artery aspiration or hilar clamping may be lifesaving, particularly when a large discrete air burden is identified; these approaches, however, carry significant morbidity and historically low survival rates, and thus are reserved for selected centers and cases with immediate surgical access [22][23][24].

Adjunctive therapies may modulate downstream injury. Lidocaine infusion has been recommended by the European Consensus Conference on Hyperbaric Medicine as a neuroprotective and antiarrhythmic adjunct in arterial gas embolism and may have a role in VGE with neurological involvement. Anticoagulation or antiplatelet therapy can be considered to mitigate thrombus formation on bubble interfaces, given the procoagulant milieu induced by endothelial activation and platelet aggregation, though decisions must balance bleeding risk in operative fields. Ventilatory management should target normoxia and normocapnia, avoiding excessive tidal volumes and PEEP that could worsen RV loading conditions. Continuous hemodynamic observation—arterial line pressure, central venous pressure trends, echocardiographic reassessment when feasible—guides titration of inotropes and vasopressors as pulmonary vascular resistance evolves during bubble resorption. Across settings, the effectiveness of these interventions is amplified by structured protocols that formalize the sequence of recognition and response: immediate oxygen and source control; rapid positioning and, if available, right atrial aspiration; escalation to vasopressor–inotrope support; consideration of HBOT based on neurological and cardiopulmonary status; and multidisciplinary coordination among anesthesiology, surgery, critical care, and hyperbaric teams. Post-event surveillance should continue for several hours to days

given the risk of delayed neurological deterioration from paradoxical embolization or evolving pulmonary inflammation. Secondary prevention—line handling checklists, avoidance of open catheter hubs, field flooding in high-risk neurosurgical positions, and simulation-based team training—reduces recurrence and shortens time to intervention in future cases. Ultimately, timely synthesis of physiologic principles with disciplined execution of positioning, aspiration, oxygenation, and hemodynamic support remains the cornerstone of VGE management, while selective deployment of HBOT and, in rare instances, surgical rescue augments recovery in the most severe presentations [22][23][24].

Differential Diagnosis

The differential diagnosis of venous gas embolism (VGE) is broad and varies with the dominant organ system involved at presentation, necessitating a contextual synthesis of peri-procedural events, physiological monitoring trends, and targeted imaging to avoid delays in definitive care. When respiratory manifestations predominate—sudden dyspnea, tachypnea, hypoxemia, wheeze, or hemoptysis—clinicians should consider acute pulmonary embolism, tension pneumothorax, bronchospasm, aspiration, and cardiogenic or noncardiogenic pulmonary edema, all of which can produce abrupt ventilation–perfusion mismatch and end-tidal carbon dioxide (EtCO₂) decline similar to VGE. Distinguishing features include pleuritic chest pain and D-dimer elevation in thromboembolism, unilateral hyperlucency with mediastinal shift in tension pneumothorax, and diffuse alveolar opacities with elevated natriuretic peptides in cardiogenic edema; yet the temporal linkage to a precipitating event (e.g., central line manipulation, neurosurgical positioning, laparoscopic insufflation) remains the most discriminating clinical clue for VGE. Cardiovascular presentations—hypotension, mill-wheel murmur, jugular venous distension, acute right ventricular (RV) failure, and malignant arrhythmias—may mimic cardiogenic shock, acute myocardial failure from ischemia or myocarditis, neurogenic pulmonary edema, or septic shock. Here, rapid correlation with monitoring signatures, such as a stepwise EtCO₂ fall, widening PaCO₂–EtCO₂ gradient, and echocardiographic visualization of right-sided intracardiac air, can prioritize VGE over alternative etiologies. Neurological syndromes—headache, altered mental status, seizure, transient focal deficits, or coma—demand urgent differentiation from transient ischemic attack and acute ischemic stroke, particularly when paradoxical embolization is plausible through a patent foramen ovale or intrapulmonary shunt; diffusion-weighted MRI patterns, vascular imaging, and a contemporaneous cardiopulmonary trigger favor VGE-related events. Across these domains, structured use of the Tübingen severity scale, integration of capnography and precordial Doppler when available, and attention to

the operative or interventional timeline enable accurate separation of VGE from competing diagnoses and expedite the initiation of targeted therapy [20].

Prognosis

Prognostic trajectories after VGE span from complete recovery in clinically silent or minimally symptomatic cases to severe, persistent disability or death when large gas volumes enter the venous system or when arterialization occurs through right-to-left shunts. Most patients experience no complications when only minute amounts of air are entrained and efficiently filtered by the pulmonary capillary bed; by contrast, even small volumes entering the central nervous system (CNS) can be devastating, and air trapped within end-arterial territories may precipitate irreversible ischemic injury despite timely intervention. Outcomes are primarily driven by initial mental status, the presence and extent of neurological signs, the rapidity of hemodynamic stabilization, and patient age, with older individuals demonstrating less physiological reserve. Mortality rates following trauma-associated VGE are substantial, with reports ranging from 30% to 80% in severe chest injury where large volumes of air may access central veins under high pressure; this spectrum underscores the importance of immediate recognition and aggressive supportive care to interrupt the hemodynamic spiral of RV outflow obstruction and hypoxemia. Procedure-related factors also influence prognosis. Surgeons employing the Fowler or sitting position, or using carbon dioxide insufflation, should maintain heightened vigilance because VGE remains a recognized complication in neurosurgery and laparoscopy; prompt source control, oxygenation, and positioning maneuvers materially alter outcomes when implemented early [9][10]. Long-term sequelae are most often neurological and include cognitive impairment, focal deficits, and seizure disorders; cardiopulmonary consequences such as chronic thromboinflammatory remodeling and reduced exercise tolerance may persist after massive events. In aggregate, prognosis reflects the interaction of embolus volume and rate, shunt anatomy, timeliness of therapy—including hyperbaric oxygen when indicated—and comorbid host factors, with asymptomatic patients generally doing well and those with CNS involvement facing the greatest risk of permanent deficit [20].

Complications

Although uncommon relative to the volume of invasive procedures performed, VGE carries a meaningful risk of life-threatening complications when diagnosis is delayed or when embolic load is large [25]. The immediate cardiopulmonary hazards are mechanical and inflammatory: air lock at the right ventricular outflow tract, acute pulmonary hypertension with RV failure, refractory hypoxemia from increased dead space, and arrhythmias culminating in pulseless electrical activity or ventricular fibrillation. Secondary inflammatory

cascades—endothelial activation, platelet aggregation, and complement-mediated injury—promote microthrombosis, increase alveolar–capillary permeability, and can precipitate noncardiogenic pulmonary edema. Paradoxical embolization via a patent foramen ovale, ventricular septal defect, or intrapulmonary shunt extends risk to the arterial circulation, with ischemic stroke, myocardial infarction, spinal cord ischemia, and mesenteric infarction among the most feared outcomes. Procedure-specific data highlight neurosurgical vulnerability, where mortality has been reported as high as 21% in select series, reflecting both the frequency of air entrainment in the sitting position and the limited compressibility of cranial venous sinuses [25]. Late complications include persistent neurological deficits, post-hypoxic cognitive syndromes, seizure disorders, and in rare cases, chronic thromboembolic pulmonary hypertension when recurrent or massive microembolic loads provoke maladaptive vascular remodeling. Iatrogenic sequelae may follow central venous catheter manipulation—hematoma, catheter-related thrombosis, or infection—particularly when emergent access is attempted during resuscitation. The cumulative profile of complications emphasizes the need for rapid oxygenation, source control, hemodynamic support, and consideration of hyperbaric oxygen therapy in severe or neurologically complicated cases to curtail the transition from mechanical obstruction to inflammatory organ injury [20][25].

Deterrence and Patient Education

Prevention of VGE rests on anticipatory systems design, vigilant intraoperative monitoring, and patient-specific risk mitigation, implemented through interprofessional collaboration. In high-risk surgical candidates, especially those scheduled for procedures in the sitting or semi-sitting position, screening for right-to-left shunt may be informative; contrast echocardiography for patent foramen ovale and bedside saline injection with transcranial Doppler to detect microbubbles in the middle cerebral artery provide rapid, noninvasive assessments that can influence positioning and monitoring plans. Intraoperatively, minimizing the vertical distance between the operative field and the right atrium reduces the hydrostatic gradient that favors entrainment; fluid loading, judicious jugular venous compression during high-risk steps, and maintenance of positive end-expiratory pressure around 10 mm Hg can further elevate central venous pressure and blunt air entry, balanced carefully against cerebral and RV physiology [26]. Continuous EtCO₂ and precordial Doppler offer sensitive, readily deployable surveillance, while transesophageal echocardiography remains the most specific tool when available and appropriate for the setting. Catheters can serve diagnostic and therapeutic roles: right atrial aspiration

via a multi-orifice central venous catheter is occasionally lifesaving, but best-practice catheter management is essential—Trendelenburg positioning during insertion and removal, Valsalva or breath-hold maneuvers, scrupulous air-lock precautions, closed hubs, frequent connection checks, and immediate purging of infusion lines. At the bedside, avoidance of hyperventilation in upright or seated neurosurgical patients, lowering the head relative to the legs to generate positive pressure in dural sinuses, and deferring seated positioning in those with known shunts reduce risk. Central venous access should be deferred in hypovolemic patients when possible, and patients should receive clear instructions to avoid deep inspiration during catheter manipulation; all infusions must be air-free, and any suspicion of VGE mandates immediate cessation of infusions, source control, and clinician notification. Education of patients and caregivers about early warning signs—sudden dyspnea, chest pain, confusion—supports rapid escalation after discharge from interventional suites [26].

Enhancing Healthcare Team Outcomes

Optimizing outcomes in VGE depends on prevention, early recognition, and rapid, protocolized management executed by an interprofessional team. Because clinical presentations are heterogeneous and monitoring changes (e.g., EtCO₂ drops) are nonspecific, a high index of suspicion and closed-loop communication are essential to trigger timely responses. Institutions should implement standardized pathways that integrate role-assigned actions: immediate 100% oxygen, source control at the field, Durant/Trendelenburg positioning, consideration of right atrial aspiration if a catheter is present, hemodynamic support with fluids, vasopressors, and inotropes, and early consultation with hyperbaric medicine when neurological or cardiopulmonary instability persists [27]. Team composition typically spans anesthesiologists, surgeons, internists, cardiologists, hyperbaric specialists, neurologists, and intensive care nurses, with clear escalation criteria and debrief processes to refine performance. Simulation-based training that rehearses recognition of EtCO₂ alarms, Doppler signatures, and echocardiographic findings can shorten time-to-intervention and reduce cognitive overload during crises. Post-event care is primarily supportive—oxygenation, hemodynamic stabilization, vigilant surveillance for evolving complications—and outcomes correlate with the volume and rate of gas entrainment and the presence of neurological involvement; asymptomatic patients generally fare well, whereas those with CNS manifestations risk persistent deficits despite therapy, and large gas volumes are frequently fatal [28]. Ultimately, aligning prevention checklists, real-time monitoring, and decisive, team-based interventions with continuous quality improvement creates a

resilient safety net that lowers VGE incidence and mitigates its most serious consequences [26][27][28].

Conclusion:

In conclusion, Venous Gas Embolism (VGE) is a potentially catastrophic event whose successful management is critically dependent on swift recognition and a coordinated, multidisciplinary response. The prognosis is highly variable, ranging from full recovery in minor cases to severe disability or death when large volumes of air entrain or paradoxical embolism occurs. The cornerstone of treatment involves immediate life-support measures: securing the airway with 100% oxygen, halting the source of gas entry, and repositioning the patient to trap air in the right heart. Concurrently, hemodynamic stabilization with fluids and vasoactive agents, along with the aspiration of air via a central venous catheter if one is present, is vital. For cases with neurological compromise or refractory cardiopulmonary instability, hyperbaric oxygen therapy serves as a definitive treatment to reduce bubble size and improve oxygen delivery. Ultimately, optimizing outcomes requires not only individual clinical expertise but also a systematized, team-based protocol that ensures seamless collaboration across medical, nursing, and surgical disciplines, from prevention and early detection through to definitive care and post-event surveillance.

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