While most surgeons strive for the hemostasis that assures bleeding has been stopped, undesired clotting in the form of a venous thromboembolism (VTE) remains a prevalent yet insidious process, affecting medical and surgical patients alike. The term VTE encompasses deep vein thrombosis (DVT) and pulmonary embolism (PE). Although many patients receive various forms of therapy to prevent VTE, prophylaxis guidelines haven’t advanced to assure that patients at risk for clots are always protected. Of equal concern are patients who seemingly have no risk factors for VTE yet develop a clot. While there are known modifiable risk factors, such as obesity, smoking, and oral contraceptive use, there are many more nonmodifiable risk factors prevalent in hospitalized patients. Consistent correlations with VTE have been established among certain populations, including: surgical patients (particularly orthopedic surgery patients), trauma victims, the immobile, those with cancer undergoing therapy, patients who have venous compression or a history of DVT, older adults, those with a genetic predisposition for clotting, and parturients. OR staff should be familiar with the systemic effects and resuscitation of patients experiencing a PE, as much of the literature regarding prophylaxis and interventions involves surgical patients. This article will review the prevalence and initial response to VTE with a particular focus on the critical sequelae of pulmonary thromboemboli and other specific PE.

**Definition and prevalence**

Despite the prevalence of DVT prophylaxis, there are still 1 to 3 DVTs per 1,000 patients every year. PE is likely underdiagnosed, but according to one landmark study, it occurs in 23 patients per 100,000 each year. The primary theory regarding development of a VTE revolves around Virchow’s Triad. This theory has prevailed since its development in the 1800s by the German pathologist Rudolf Virchow. His three tenants for development of VTE are: impaired blood flow; injury to a vessel wall; and a hypercoagulable state. While this theory is still accepted, there’s an emerging concept that correlates VTE with atherothrombosis. This concept indicates a likely relationship among diffuse cardiovascular pathologies, including coronary, carotid, and peripheral vascular disease and their correlation with VTE patients. In fact, PE remains the third most common cardiovascular disease following myocardial infarction and stroke. Because thrombosis occurs at the site of injury to a vessel wall, it’s considered an...
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Inflammatory process. A thrombus may develop to the extent that it occludes a vessel, and if a thrombus detaches and travels to the lung, it’s then described as a pulmonary embolus (see Pulmonary emboli). While the genesis of a thrombus develops at the microvascular level, the systemic effect of a PE is an abnormality in gas exchange known as dead space ventilation. This defect in respiratory function paired with a mechanical obstruction of flow into the left side of the heart results in an ominous impairment to both systemic oxygenation and perfusion.

**Signs, symptoms, and diagnosis**

There are several classic symptoms of both DVT and PE. Most commonly, a DVT will present with pain in the calf, swelling of the affected extremity, increased temperature locally, and tenderness with redness at the site. As for PE, a conscious patient will often complain of pleuritic chest pain, dyspnea, hemoptysis, palpitations, apprehension, and, sometimes, a history of syncope. Syncope is associated with PE in 8% to 19% of patients and is primarily attributed to the decrease in hemodynamic reserve. One aspect of PE symptoms that makes clinical diagnosis so difficult is the complex yet nonspecific symptoms that mimic so many other pathologies. Collectively, systemic symptoms of PE follow a pattern of impaired gas exchange and altered circulation.

Diagnosis of a DVT most often involves an ultrasound exam of the extremity. Several advantages of this type of tool are the noninvasive nature, rapid interpretation, high specificity, and prevalent availability of the equipment. For PE, several tools are often employed to determine the presence of an embolus. The chest computed tomography (CT) remains the primary diagnostic tool; however, other methods exist, such as angiography and ventilation-perfusion studies, though they’re less frequently used due to limitations on availability and interpretation. For patients with PE presenting in the early stages with an unclear diagnosis, arterial blood gas testing will reveal significant hypoxemia, decreased carbon dioxide (CO₂), and respiratory alkalosis.

One difficulty in recognizing a PE in the OR is that so many of the symptoms are subjective and masked while under anesthesia. Under such conditions, there may be few predictive clues before hemodynamic collapse (see Signs of PE). The d-dimer is a lab test often used to help evaluate coagulation abnormalities. This test evaluates the specific degradation process of fibrin. For a patient with acute thrombus formation, this test will be positive and won’t necessarily diagnose a PE, as other abnormalities may cause a similarly abnormal d-dimer. However, because of its near 100% sensitivity, the absence of an abnormal d-dimer is helpful to rule out an acute PE. Symptomatically, a patient under general anesthesia may demonstrate hypotension, tachycardia, and hypoxia, despite otherwise adequate oxygen supply. The airway CO₂ monitor,
used with every general anesthetic, may aid in identifying an acute PE, which would display an acute decrease in exhaled (end-tidal) CO₂ (ETCO₂). Another diagnostic and management tool is the ECG. Although changes specific to PE aren’t as readily identified as with other devices, these clues may be added to the overall clinical picture to help form a diagnosis. Changes that may be observed include P pulmonale, right bundle branch block, right-axis deviation, sinus tachycardia, and ST-segment/T-wave changes common to ischemia. It’s helpful to keep in mind that symptoms may appear quite subtly in patients with a suspected PE. Some patients may even appear normal despite significant obstruction to pulmonary flow and suddenly suffer cardiovascular collapse. Healthcare providers must keep in mind that patients with significant PE don’t always present with severe symptoms, although their pathology may indeed be life threatening.

**Unique aspects of pulmonary emboli: varieties**

In addition to a venous thromboembolus, the OR nurse should be aware of other types of emboli in the lung (see Sources of emboli). One of the ominous varieties is the amniotic fluid embolus (AFE) affecting the parturient. Since 70% of AFE events occur in labor and delivery, what would otherwise be an uncomplicated process can quickly become a complex and difficult resuscitation. There’s no diagnostic test for this syndrome, and autopsy remains the only method of definitive diagnosis for AFE despite having been studied extensively. The incidence of AFE is 1 in 800 to 1 in 8,000 with a high mortality, comprising the etiology of 10% of maternal deaths. Initial presentation frequently includes seizure activity, respiratory failure from acute respiratory distress syndrome, and cardiovascular collapse. Hypotension has been associated with all cases, yet other less-lethal causes of hypotension may initially mask the true etiology. Hypoxia, followed quickly by pulmonary edema, is a characteristic sign of this ominous syndrome. Besides the inflammatory and hemodynamic presentation, disseminated intravascular coagulation remains an additional burden on the providers, as it occurs in as much as 83% of patients. Initial management includes therapy similar to other significant embolic events. Intubation shouldn’t be delayed, and cardiac support, including increased cardiac preload and inotropic support, should be given. Correction of coagulopathy with blood products should be provided. In extreme cases when oxygenation and perfusion are severely compromised and such services are available, extracorporeal membrane oxygenation or cardiopulmonary bypass can be instituted. Besides maternal concerns, there’s also a high incidence of fetal mortality.

In addition to thrombotic genesis of PE, in which the inflammatory cascade initiates obstruction, other nonbiological material can become intravascular and result in mechanical occlusion of the pulmonary arteries. For example, during the vertebroplasty procedure, bone cement is absorbed into the venous system as often as 72% of cases. Other emboli include the venous uptake of suture or particles of I.V. catheters. Emboli of this nature don’t result in the inflammatory changes consistent with thrombotic events; however, their mechanical obstruction to
pulmonary flow and associated hemodynamic and oxygenation complications can be equally devastating.

An additional and more prevalent type of embolus is the gas embolus, which may be air, CO₂, or another agent to which the patient’s vasculature is exposed. Nurses frequently adjust pumps and syringes to remove air from the fluid before an I.V. injection to avoid an air embolus. Interestingly, the lethal amount of air has been shown to be between 100 to 150 mL, which is more than the priming volume of most entire I.V. infusion sets (see Air bubbles seen on echocardiogram).17 This type of embolus is often less severe and likely more recoverable. One reason for this is the resolvable nature of gas within the vasculature. As opposed to mechanical obstruction with an inflammatory response, gas can be physically removed through a central venous line or absorbed and exhaled via uptake in the lungs. Early recognition of a gas embolus is key to preventing cardiovascular collapse. Echocardiography is an ideal diagnostic tool, though loss of ETCO₂ is a sensitive indicator for problematic volumes of gas in the heart. Until the gas is extracted or absorbed, resuscitation focuses on supporting hemodynamics with inotropes, keeping the heart and vasculature full with I.V. volume resuscitation, stopping the influx of gas into the veins, and placement of the patient into the left lateral Trendelenburg position. This position facilitates the movement of the gas into a pocket of the right atrium or ventricle where it doesn’t impede blood flow and allows the heart to pump fluid rather than attempting to pump air.

Certain surgical positions are more prone to allow entraining of air into the venous system. Classically, the sitting craniotomy is a high-risk category. Due to the exposure of the venous sinuses and the surgical site being above the heart, this position has more conditions that are conducive to venous air embolus (VAE). Subatmospheric conditions within the venous system, surgical sites above the heart, or pressurization of a tissue or cavity such as laparoscopic procedures all have a risk of facilitating a VAE. The prevalence of laparoscopic surgery increases the likelihood of CO₂ embolus due to this particular gas being preferred for cavity insufflation. Two methods of reducing the incidence of VAE have been suggested: increase central venous pressure (CVP) by aggressive I.V. hydration and for patients unable to tolerate this or when impractical, elevate legs to increase CVP. Though not universally accepted, the addition of positive end-expiratory pressure to the mechanical ventilation sequence can increase CVP. Caution is advised with this maneuver in the presence of a patent foramen ovale. Entrained air could move from the manually increased pressure of the right atrium into the left atrium, resulting in exposure of air to the coronary and carotid circulation.19 Should air be entrained into the heart, the OR nurse may be required to provide external chest compressions despite a pulsatile cardiac rhythm. This will help augment movement of medications to the heart and migrate a large bolus of air into the lung where it can be absorbed.19

Generalized treatment and resuscitation
Treatment of a diagnosed VTE involves several aspects. The in situ PE must be addressed, especially if it’s causing hemodynamic compromise, recurrence must be prevented, and long-term sequelae such as postthrombotic syndrome must be considered.3 For a patient who’s unstable, rapid fibrinolysis is recommended to resolve symptoms and restore circulation to the lungs.4,11 If a patient persists with an unresolved PE despite emergent fibrinolysis, has deteriorated in condition, or when such anticoagulation is contraindicated, emergent surgical intervention is warranted, including procedures such as a
Although DVT and PE are not uncommon among the hospitalized patients, massive acute pulmonary emboli are infrequent. Given their infrequency along with the lack of broad consensus for initial treatment, resuscitative efforts remain largely symptomatic until the actual fibrinolysis can take place. If left-sided heart failure is observed, this is often an ominous finding resulting from right-sided heart failure. As pulmonary blood flow becomes dampened due to mechanical obstruction by the embolus, pulmonary vasoconstriction from hypoxia further impedes the flow of blood to the left side of the heart. The lack of blood to the left ventricle results in a systemic hypotension that’s often refractory to vasopressors. The American Heart Association’s Advanced Cardiovascular Life Support (ACLS) guidelines list PE as one of the 10 common causes of asystole and pulseless electrical activity (PEA). In addition to the sequelae of left-sided heart failure, the pulmonary blood flow changes associated with significant PE result in a characteristic strain and distension of the right ventricle. Right ventricular dysfunction and failure, tricuspid valve regurgitation, and venous congestion are all associated with severe PE, and when observed, correlate with an increased mortality. Pharmacologic interventions are aimed at increasing the perfusion pressure to the right and left ventricle, increasing contractility, and decreasing the pulmonary vascular resistance. Because the management of severe PE is often the symptomatic treatment of the left- and right-sided heart failure, valvular dysfunction, volume changes, and cardiac ischemia, the use of echocardiography is helpful as an adjunct for real-time evaluation and intervention guidance.

**Echocardiography**

Echocardiography is a key tool in the management of patients with PE. While it isn’t the most sensitive test for diagnosis, it’s specific in the capability for evaluating other aspects of care. In particular, systemic volume, cardiac contractility, and valvular function are directly assessed. Approximately 50% of patients will demonstrate a pronounced right ventricular dysfunction on transesophageal echocardiography (TEE), which correlates with a high short-term mortality. Partially explaining the significance of right ventricular failure is the realization that 30% of pulmonary vasculature must be occluded to develop ventricular changes. Additionally, while TEE exam may not have as high a sensitivity to detect a PE when compared to
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CT, the availability within the OR and the utility for identifying significant PE sequelae, such as right-sided heart failure, provide more than sufficient indication for use.1 For differential diagnosis assessment, TEE is more sensitive than either ECG or the pulmonary artery catheter for diagnosing cardiac ischemia. Additionally, significant cardiac events such as aortic dissection, air embolus, and valvular dysfunction are readily identified with a trained user.

Prevention
One of the focal dilemmas for surgeons is deciding when to institute pharmacologic DVT prophylaxis (see ACCP guidelines: Information for VTE prophylaxis). The risk of intraoperative bleeding often warrants the delay of anticoagulants until after surgical hemostasis is certain, and thus, develops the debate in optimum timing for medication administration.2 While the surgeons have developed a rather specific set of guidelines to assist in the prevention of VTE, nurses should anticipate and prepare for prophylaxis in certain high-risk populations, such as any major surgery, trauma patients, hip fractures, in patients with significant paralysis or spinal cord injury, or in patients with a malignancy.23

ACCP guidelines: Information for VTE prophylaxis20

General guidelines:
• Mechanical thromboprophylaxis primarily for patients at high risk for bleeding.
• Aspirin not recommended as sole agent for prophylaxis.
• Low-risk general surgery without risk factors, no specific therapy except early and frequent ambulation.

Additional guidelines
• For major general surgical procedures, thromboprophylaxis recommended until discharge from hospital.
• Specific recommendations are provided for specific surgical categories, including patients receiving laparoscopic, bariatric, orthopedic, vascular, and spine surgery.
• Acutely ill medical patients should receive pharmacologic prophylaxis unless contraindicated.
• Long-distance traveler’s guidelines are provided, including suggested calf muscle exercise, mechanical calf compression devices, and pharmacologic therapy for indicated patients.

One nursing responsibility for prophylaxis involves the placement of mechanical compression devices. Although one theory of the pneumatic compression boot suggests that manual compression of the calf can promote venous flow, manufacturers vary in fitting and effectiveness of this boot. This variation of compression can alter the efficacy of the boot in venous circulatory augmentation. Despite the seeming inferiority of pneumatic devices when compared to pharmacologic interventions for DVT prophylaxis, one notable advantage for surgical candidates is the lack of increased risk for bleeding during implementation.20 Although they are considered inferior to anticoagulation, when compared to no prophylaxis at all, compression devices reduce VTE incidence by 60%.1 One reason for the decreased value of the compression devices is the limited study data evaluating their effectiveness, both as risk reduction tools and as a comparison among the varieties and styles.20 Therefore, no additional recommendations are provided by the American College of Chest Physicians regarding the use of the devices with regard to style or mode of operation except to optimize sizing and proper application.20 The actual utilization and proper application appear more important than the specific type of device.25 Additionally, mechanical compression hose or stockings have been shown to help relieve symptoms of postthrombotic syndrome. These symptoms include leg swelling, feelings of leg heaviness, and leg pain.21

Moving forward
Due to the ongoing prevalence of VTE, clinicians must be vigilant in the prophylactic therapy aimed at preventing this potentially life-threatening syndrome. OR staff must be particularly alert to the subtle signs of a PE due to the frequently unassuming nature of its presentation. The nonlinear correlation between presenting symptoms and severity of the obstruction requires rapid intervention and careful planning to prevent catastrophic cardiac and pulmonary failures. Research will need to continue in this field to further identify individuals at risk for VTE as well as methods of preventing occurrence.

REFERENCES


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The author and planners have financial relationships related to this article.

DOI: 10.1097/01.ORN.0000431585.98335.02