AORN Guideline for Prevention of Venous Stasis

Introduction

Deep venous thrombosis (DVT) and pulmonary embolus (PE) are major risk factors for patients undergoing surgical or other invasive procedures, and thromboprophylaxis should be considered for all these patients.[1,2] Prevention of venous thromboembolism (VTE), a combination of DVT and PE, is more effective than treatment and is an important aspect of patient care before, during, and after surgery.[3] Identification of risk factors should be used as a basis to determine if pharmacological and/or mechanical thromboprophylaxis should be initiated.

Deep vein thromboembolisms and PE may be asymptomatic and difficult to detect. Patients with DVT may exhibit symptoms such as local tenderness or impaired drainage of blood vessels distal to the obstruction.[4] In addition to producing localized symptoms, DVT can break off and travel to the lungs, resulting in PE. The incidence of PE is greater when the DVT is close to the heart.[4] Studies indicate as many as 80% of orthopedic surgical patients may develop DVT. Without preventive treatment 4% to 10% of these patients will develop PE.[5] Deep venous thrombosis and subsequent PE remain the most common cause for emergency readmission and death following joint replacement surgery, even when preventive therapies are used.[6]

The National Quality Forum, the Joint Commission on Accreditation of Healthcare Organizations, the Centers for Medicare and Medicaid Services,[7] the American College of Obstetricians and Gynecologists,[8] and many other organizations have created protocols or recommendations on the prevention of venous stasis/DVT/PE. The American College of Chest Physicians recommends that every hospital develop a formal strategy or protocol addressing the prevention of thromboembolic complications.[9] The Surgical Care Improvement Project developed two process measures recommending the presence of orders for venous thromboembolism prophylaxis for surgical patients and that patients receive prophylaxis for DVT within 24 hours before or after surgery.[7]

Use of protocols for prevention of DVT might save the lives of two-thirds of the patients who die from PE. The protocols known to be effective for the management of PE have been severely underused.[10] Estimates indicate that 1% of patients admitted to hospitals die because of PE and at least one-half of these at-risk patients might be saved with effective prophylaxis use.[3] Perioperative nurses should be knowledgeable about venous stasis and should participate in multidisciplinary teams to develop policies, procedures, and protocols to reduce the risk of venous stasis and assist in preventing DVT and PE.

The purpose of this guideline is to provide a framework that perioperative registered nurses and others can use to develop and implement policies, procedures, and protocols for prevention of venous stasis and DVT/PE. This document does not cover the long-term effects of venous stasis, such as venous stasis ulcers or their postoperative treatment.

The "AORN guideline for prevention of venous stasis" is based on current available research and expert opinion. It is assumed that ongoing research will result in new knowledge, procedures, and medical and nursing interventions for prevention of DVT. This guideline may not apply to every individual and may require modification based on the specific needs of a given patient. Review of this document has been solicited from content experts considered knowledgeable in the prevention of venous stasis and included representatives from academia, medicine, perioperative nursing, and clinical nurse specialists.

Definitions

For purposes of this document the following definitions apply.

Anticlotting factors: Naturally occurring proteins that, when activated, prevent blood clot formation.[11]

Anticoagulant: Substance that prevents or delays blood from clotting.[11]

Antiembolism stockings: Elastic stockings that reach from the foot to just below the knee or to the thigh. The stockings are tighter at the ankles than at the knee. The varying tightness of compression stockings helps circulate blood that collects in the calves.[10]

Central access venous catheters: A catheter designed for continuous access to the venous system. Frequently implanted into the internal jugular, antecubital, or subclavian vein.

Clotting factors: Naturally occurring proteins which, when activated, cause blood clot formation.[11]

Deep vein thrombosis: Development of a thrombus (ie, clot) in one of the deep veins of the body, frequently the iliac or femoral veins or major upper-extremity veins. These clots can break off from the vein, travel through the heart, and lodge in the arteries of the lungs, causing a potentially fatal PE.[10]

Intermittent pneumatic compression device (IPC): A mechanical device that uses intermittent pneumatic compression, applied via a cuff that inflates and deflates sequentially or uniformly. The cuff may be applied to the foot, calf, or thigh.[1]

Post-thrombotic syndrome or postphlebitic syndrome: A chronic inflammation of a vein that destroys the valves after an episode of thrombophlebitis or the presence of a thrombus.[12,13]

Pulmonary embolism: A thrombus that breaks free from a vein, travels through the veins, reaches the lungs, and lodges in a pulmonary vessel. A pulmonary embolism is a potentially fatal condition that may cause death within minutes to hours.[10]

Thrombolytic medication: A medication used for dissolving existing blood clots.

Thrombophlebitis: The formation of a venous clot accompanied by inflammation of the vein. This may be the result of trauma to the vessel wall; hypercoagulability of the blood; infection; chemical irritation; intra- or postoperative venous stasis; prolonged sitting, standing, or immobilization; or a long period of IV catheterization.[10]

Venous stasis: A condition that occurs when the normal blood flow though a vein is slowed or halted.[10]

Venous thromboembolism: A condition including DVT and PE.[14]

Venous thrombosis (phlebothrombosis): A condition that occurs when a clot forms within a vein without inflammation.[10]

Virchow's triad: A descriptor listing the three main causative factors in DVT formation—endothelial damage, hypercoagulability, and venous stasis.[15]

Prevalence

The prevalence of thromboembolic events varies by the type of event. The following classifications describe the most common types of thromboembolic phenomenon.

Venous Thromboembolism

This combination of DVT and PE affects more than 1 in 1,000 adults annually in the United States, causing discomfort, suffering, and occasionally death.[16] More than 2.5 million people annually are diagnosed with DVT.[17] Deep vein thrombosis factors into approximately 600,000 hospital admissions annually, and 50,000 to 200,000 of these patients will suffer from PE. In the United States, PE is the third most common cause of death. Pulmonary embolism occurs most frequently in patients admitted to the hospital for a diagnosis other than PE or DVT.[1,18] A DVT formed in the leg is not life-threatening, but if the clot breaks loose and moves to the lungs, a life-threatening PE can result.[17] Pulmonary embolism is a medical emergency affecting both the cardiovascular and respiratory systems and has mortality rates that may be as high as 25%.[17]

Pulmonary embolism is reported to be the number-one preventable hospital-acquired complication. Autopsies reveal an estimated 10% to 25% of all deaths in hospitals involve a PE.[3] Some studies have shown that in 60% of inpatient deaths the patient had a PE, and as many as 70% of these were not diagnosed.[18]

One study has shown that 8.83 surgical patients with risk factors per 1,000 develop DVT. The study included all surgical patient discharges defined by specific diagnosis-related groups and coded for an operating room procedure according to the *International*

Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) classification system. Patients who were excluded

- had ICD-9-CM codes for DVT or PE at time of admission,
- were obstetrical patients in Major Diagnostic Category 14,
- were patients where a procedure for interruption of the vena cava was the only operating room procedure, or
- the procedure performed occurred before or on the same day as the first operating room procedure.

In this study, the day of the operative procedure was not available on some data files, creating a limitation in the study. This may have led to an incidence rate slightly lower than if that information had been available.[19]

Without prophylaxis, the incidence of objectively confirmed, hospital-acquired DVT is approximately 10% to 40% among medical or general surgical patients and 40% to 60% following major orthopedic surgery.[9] In one study, 42% of hospitalized patients with a diagnosis of DVT and PE did not receive prophylaxis.[20,21]

Approximately 300,000 patients are newly diagnosed with DVT yearly in the United States, and possibly three to four times this many may occur without obvious symptoms and thus are not detected.[16] About 80% of DVT are thought to be silent, with no signs or symptoms. There is a direct correlation between asymptomatic DVT and future development of a symptomatic VTE. One study of critical care patients found that patients with asymptomatic DVT had a significantly greater rate of PE development during their index hospitalization compared to those patients with symptomatic DVT (ie, 11.5% vs 0% respectively; $P = .01$).[9]

In the United States, published studies estimate that 200,000 to 600,000 patients receive a diagnosis of DVT and PE annually. These two diagnoses are thought to contribute to 60,000 to 200,000 deaths. If simple preventive measures were instituted, many of these deaths could be prevented. Patients need to be warned about DVT risks and assessed for risk factors, and measures need to be used.[22]

Many hospital-acquired VTE are not evident until after discharge, due to the increased number of same-day surgery procedures and the decreased patient length of stay.[9] Lack of VTE prevention often leads to readmission, a rise in anticoagulation therapy complications, an increased risk of long-term morbidity from post-thrombotic syndrome, and a higher rate of future recurrent thrombosis.[9] An estimated $2.9 billion are spent annually in the United States for the treatment of DVT and PE.[9] The most serious outcome resulting from hospital-acquired VTE is death from an undiagnosed PE.

Superficial Venous Thrombophlebitis (SVT)

Generally, thrombophlebitis is used to describe superficial thrombophlebitis that occurs in the veins located near the surface of the body, but the term often is used synonymously with DVT.[23] Superficial thrombophlebitis occurs annually in 400 patients out of 100,000.[24] Cases of SVT occur more frequently in women. The average age of occurrence for women is 58 years. In men, SVT occurs in 35% to 46% of surgical patients. The average age of occurrence for men is 54 years.[24,25] Ninety percent of thrombophlebitis occurs in the leg, with 10% occurring in the arms and other parts of the body. As many as 60% to 80% of the cases of SVT arise in patients with varicose veins. These patients frequently experience the thrombophlebitis after minor trauma such as a bruise to the varicosity.[24,25]

Post-Thrombotic or Postphlebitic Syndrome (PTS)

This is a chronic inflammation of a vein that occurs after an episode of thrombophlebitis or the presence of a thrombus and that destroys the valves. Long-term venous thrombosis is associated with PTS syndrome. The prevalence of PTS has been estimated to be as high as 2% in the general population and occurs in 50% to 67% of subjects who sustain proximal vein thrombosis.[12,17,26]

Pathophysiology

To understand how DVT and PE phenomena can be prevented, practitioners need an understanding of the pathophysiology of the venous system and clotting mechanisms.

Gross Anatomy of the Venous System

The venous system has superficial and deep veins whose function is to return blood to the heart and lungs. Superficial veins are located just under the skin. These superficial veins connect to the deep veins located in the large muscles by the collecting (ie, communicating) veins. Deep veins located in the muscles are largely responsible for returning blood flow to the heart. The venous system is a low-pressure system that acts against gravity; therefore, it acts in a reverse pressure gradient. Valves within the veins prevent backflow in a healthy venous system. The pressure of blood flow against a normal functioning valve is what opens the valve.[17]

Most collecting veins are suprafascial and can dilate to accept large quantities of blood at any one time without interfering with the normal function of the venous system. From the collecting veins, venous blood travels to the deep veins largely located in the lower extremities. The movement of blood from the feet to the right atrium depends on a complex system of muscle pumps. These pumps propel the blood against gravity and are located in the arch of the foot, calf, and thigh. The contraction of the muscle pumps increases the velocity of the blood and forces the blood upward, which in turn increases the pressure against the valve cusps within the vein, causing them to open and push the blood forward. Healthy, functioning muscle pumps and venous one-way valves move the deoxygenated blood toward the right atrium and prevent pooling or backflow in the vascular system.[17]

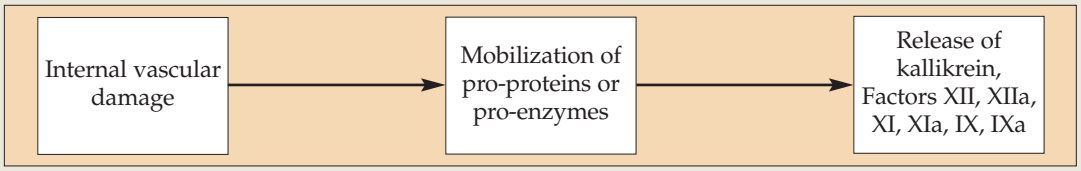
Deep vein thrombosis develops frequently in the proximal deep veins, with 90% of DVTs occurring in the deep veins of the leg, usually in the femoral vein.[3] In the upper extremities, the increasing use of indwelling venous access catheters is associated with a greater number of DVT cases. Mortality rates for DVT occurring in the legs range from 13% to 21%, and up to 48% in the arms.[15] If the thrombus partially or completely blocks the flow of blood through the vein, blood begins to pool and build up below the site, potentially resulting in chronic swelling and pain. The valves in the blood vessels also may be damaged, leading to venous hypertension.[16]

Clotting Cascade

The clotting cascade is an intricate system of proteins and enzyme actions that must occur in sequence to produce clots, inhibit further clot formation, or cause destruction of an existing clot. Initiation of one of two systems in the clotting cascade (ie, the intrinsic or extrinsic system) begins the actual clot formation process. The following is a simplified explanation of the complex blood clotting progression.[5,27]

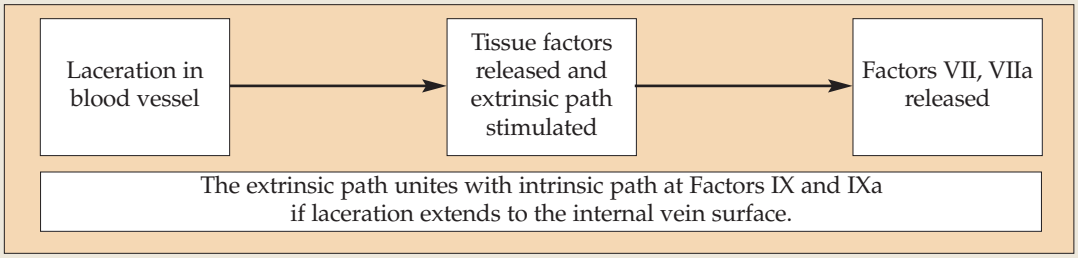
Intrinsic clotting path

Internal vascular irritation or lesions initiate the intrinsic system with the mobilization of pro-proteins and pro-enzymes to stimulate the transport of kallikrein, Factors XII, XIIa, XI, XIa, IX, and Xa to the site of the irritation.



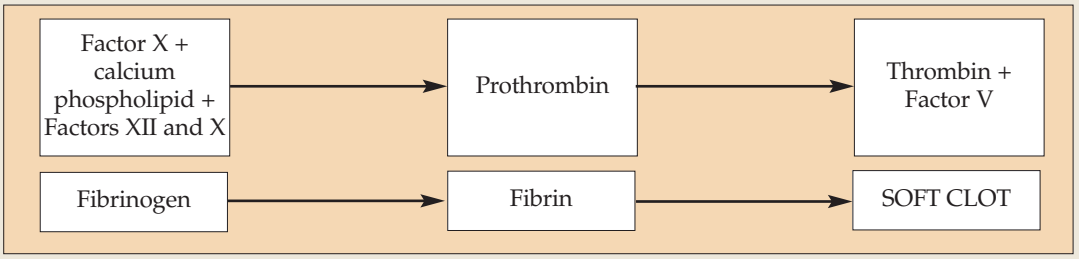
Extrinsic clotting path

If a laceration occurs to the blood vessel the extrinsic system is activated, releasing tissue factor proteins from the damaged cells as well as mobilizing Factors VII and VIIa. The intrinsic system also is stimulated into action if the damage extends to the internal surface of the vein. The two systems unite at IX and IXa and continue the clotting process.



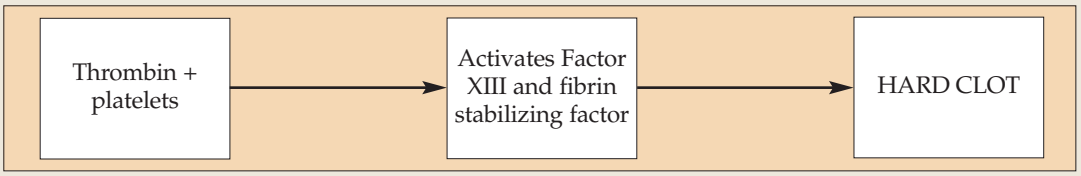
Soft clot formation

The next step is soft clot formation, which occurs when Factor X and calcium phospholipids unite with prothrombin, yielding thrombin formation. Thrombin is anchored to the phospholipids, limiting thrombin to the site of the injury or irritation. In the presence of Vitamin K, thrombin and Factor V unite, initiating the development of fibrinogen, which yields fibrin to form a mesh-like structure called a soft clot. **Requires the Presence of Vitamin K.**



Hard clot formation

After soft clot formation, thrombin activates Factor XIII, a fibrin-stabilizing factor. This factor along with the fibrin-soft-clot-matrix, traps platelets, aggregating the platelets at the injury site and resulting in a hard clot.



Clotting termination

At some point, the clotting cascade must terminate, and eventually the clot dissolves. The termination process begins with release of Factors V, VII, VIII, XIII, and Protein C and Protein S. The actual release of Protein C signals the clotting cascade to dismantle. Protein C targets Factors V and VII to slow and eventually stop clot formation.

Factors V, VII, VIII, and XIII	+	Protein S and Protein C	=	Signal to stop clot formation

Inhibition of clotting process

Antithrombin III is produced to inhibit thrombin. Tissue damage or irritation also stimulates mast cell production. Mast cell production releases heparin and other proteases that function as mild antithrombin-type substances and other antithrombin III enhancers. These actions stop clot formation.

Antithrombin III + Heparin + Other inhibitor = Termination of clotting process

Clot dissolution (Fibrinolysis)

Fibrinolysis, or clot dissolution, requires plasminogen and tissue plasminogen activator to release plasmin. Plasmin, along with heparin, has a binding affinity to the fibrin, digesting the clot.

Plasminogen + Tissue Plasminogen Activator + Heparin = Clot dissolution

Pathogenesis of Deep Vein Thrombosis and Pulmonary Embolism

Factors that influence venous thrombus formation can be explained by a theory developed by Rudolf Karl Virchow in 1856.[15] This theory, known as Virchow's triad, describes three conditions that contribute to the formation of DVT. Deep vein thrombosis is caused by an abnormality in one or all three of the components of Virchow's triad.[4]

Virchow's Triad

Virchow's triad describes the three main causative factors in DVT formation: endothelial damage, hypercoagulability, and venous stasis.[1,3,5,15,18] The following conditions all can contribute to these causative factors and result in DVT.

Vessel wall injury. Any vessel wall injury that does not include the interior vessel wall initiates the extrinsic pathway of the clotting cascade. When the endothelium (eg, interior) of a vessel is damaged, causing roughening of the vessel wall, the intrinsic pathway of the clotting cascade is initiated, resulting in platelet adhesion and aggregation that promotes blood coagulation at the site of the injury.

Trauma and surgical procedures or interventions cause instantaneous injury to vessel walls, both interior and exterior, and create a risk for thrombosis. Venipunctures, intravenous therapies, venous access catheters, and heart valves cause a constant irritant and also add to the potential for DVT occurrence.

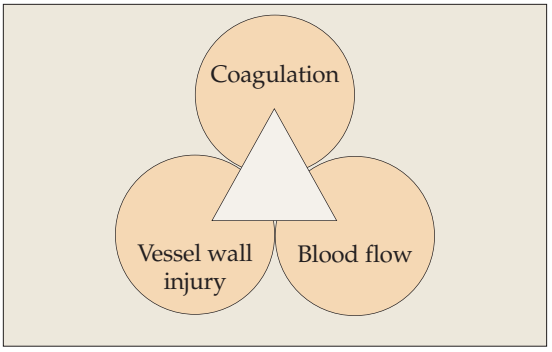
Blood flow alteration. Alterations in blood flow (ie, venous stasis) allow blood to pool within the veins, causing clot production. These clots increase in viscosity and do not get washed away rapidly related to the stasis. They may sit and enlarge and then may be propelled into the remainder of the venous system. Blood flow alteration may be caused by venous stasis, which can occur in sedentary or bedridden patients or pregnant women, especially those with varicosities. Patients with blood dyscrasias that contribute to blood pooling, such as proliferative polycythemia, erythrocytosis, or some malignancies, also can lead to stasis problems.

Blood flow also can be altered due to venous obstruction. A population at risk due to pooling of venous blood includes patients with pelvic tumors, those having long abdominal or pelvic laparoscopic surgical procedures, and patients with congestive heart failure.

Smoking, another cause of blood flow alteration, is a chemical irritant that causes vasoconstriction and thus reduces the oxygen carried in the blood. This alters the blood flow and leads to retention of blood in the vascular system.

Alterations in coagulation. Any increase in the coagulation profile (ie, hypercoagulability) of the blood can result in DVT formation. Factors increasing the risk of blood clot formation can be due to any of the following:

- an increase in the number or amount of clotting factors released (eg, surgery, burns, trauma);
- a decrease or lack of factors that stop the clotting cascade;
- a decrease in the fibrinolysis action of the clotting system caused by medications (eg, estrogen), inflammatory diseases (eg, inflammatory bowel disease), or chronic systemic diseases (eg, lupus erythematosus);
- absence of coagulation modulators that signal clot formation to stop (eg, protein C, protein S, and antithrombin III, and changes in the activity of the fibrolytic system or presence of tissue plasminogen activator, plasmin, or heparin); and
- age. As people age, the presence of additional venous thrombus risk factors within Virchow's triad (below) escalate the possibility and occurrence of DVT formation.[22]



Superficial Venous Thrombophlebitis

Superficial thrombophlebitis occurs when one of the elements of Virchow's triad—ie, vessel wall injury, blood flow, and coagulation—stimulates the clotting process. Superficial thrombophlebitis causes an acute inflammatory reaction that triggers the thrombus to adhere to the vein wall.[25]

Post-Thrombotic or Postphlebitic Syndrome

This syndrome is thought to be caused by venous hypertension resulting from venous valve destruction or persistent obstruction due to thrombosis. The high pressure present renders the perforating veins of the calf incompetent. Blood flow is then directed into the superficial system, leading to edema and, when severe, impaired viability of subcutaneous tissues, leading to venous stasis ulceration.[13,17,26]

Assessing for Risk Factors

It is imperative for perioperative practitioners to identify the risks factors for DVT and/or PE in their nursing assessments.[15,28,29] Assessment of risk factors begins with the initial patient assessment. It continues throughout the patient's surgical/interventional procedure and postoperative recovery until he or she is discharged from care. The process of assessing patients for DVT or PE begins with the identification of risk factors. Risk factors are grouped into three general categories.

Age. Risks for DVT increase in frequency as a person advances in age, beginning at age 40.[3]

Primary admitting medical diagnosis. The primary admitting medical diagnosis (ie, admitting diagnosis, primary diagnosis) is the patient's diagnosis when presenting to the health care facility. The perioperative nurse should develop a plan of care based on increased risk for DVT when this diagnosis includes any of the following:

- trauma;[9]
- orthopedic fractures (eg, hip, major bone, pelvis, knee);[14,22]
- burns;[15,30]
- planned surgery or interventional procedures where
  - patient positioning includes flexion and rotation of a joint that may cause constriction of a vessel leading to vascular damage or[28,31]
  - surgery may cause an interruption of circulation (eg, tourniquet usage);[28]
- use of central venous indwelling catheters;[9,32] and
- procedures lasting longer than 30 to 45 minutes.[15]

Past medical history. The patient's past medical history may include or be independent from the current admitting diagnosis. The medical history assessment is based on the patient's past, current, and familial medical history. It should include an assessment of

- any recent surgery (especially orthopedic, abdominal, or thoracic);[7]
- history or family history of thrombosis, coagulopathy, blood clots, blood-clotting disorders, DVT, or PE;[9]
- history of cancer (ie, malignancy with or without treatment);[9]
- varicosities or leg swelling;[9]
- obesity;[9]
- smoking;[9]
- chronic obstructive pulmonary disease;[9]
- estrogen or hormone therapy;[9]
- nephrotic syndrome;[9]
- pregnancy or postpartum period of less than one month;[9]
- recent myocardial infarction;[9]
- congestive heart failure;[9]
- history of atrial fibrillation;[9]
- inflammatory bowel disease;[9]
- sedentary/nonambulatory lifestyle greater than 72 hours;[10]
- immobility, including that related to casts, braces, or splints;[9]
- stroke;[9]
- infection or sepsis;[9]
- dehydration or hypovolemia;[15]
- recent travel (eg, prolonged air travel or riding in a vehicle);[33] and
- ethnicity (ie, greater risk in Caucasians and African Americans).[15]

The presence of any of these in the patient's history indicates a need for increased surveillance for DVT.

Special Considerations for High-Risk Patients

Pediatric Patients

Today more children survive diseases previously considered incurable. Along with this increased survival, the number of children experiencing DVT is increasing (eg, approaching 0.2% in North America). A second factor for increasing the rate of childhood DVT is the increasing use of central venous access catheters.[9,34]

The epidemiology of DVT in children differs from adults related to the location of the thrombi. Venous thromboembolism in children usually is located in the upper body venous system, primarily because this is the preferred location for central venous access catheter placement.[32] One study found that approximately 40% of thrombi in children occur in the upper venous system, but in adults this occurs only approximately 1% of the time.[35] The greatest risk factor for DVT in children is the placement of central venous access catheters or arterial catheters.[36]

General Surgery

The risk of thromboembolic complications in the general surgery population may be elevated due to more extensive surgical procedures being carried out on older and more critically ill patients, the use of preoperative chemotherapy, and the shorter duration of prophylaxis related to decreased lengths of stay in the hospital. The risk for DVT is decreased by earlier mobilization and increased use of thromboprophylaxis.[9]

Gynecologic Surgery

The rates of VTE after gynecologic surgery are similar to the rates of general surgery patients. Gynecologic patients frequently have a combination of risk factors that make them more susceptible to VTE. Laparoscopic gynecological procedures appear to have a lower risk of VTE compared to open procedures but can result in impaired venous return from the legs and activation of the factors involved in coagulation.[9]

Laparoscopic Surgery

The potential for a longer surgical time for laparoscopic operations leads to a greater risk of VTE. Venous stasis is created in these patients related to both pneumoperitoneum and the use of the reverse Trendelenburg position. The length of stay associated with laparoscopic procedures leads to a decrease in the length of the thromboprophylaxis time, and mobility after discharge is difficult to control.[9]

Urologic Surgery

The rates of VTE in urological patients are decreasing, but VTE remains an important nonsurgical complication. The increased risk factors for patients undergoing major urologic surgery are very similar to those undergoing gynecologic procedures, including the use of the lithotomy position and open procedures resulting in longer operative times.[9]

Orthopedic Surgery

Patients having major orthopedic surgery, including hip and knee arthroplasty and hip fracture repair, are considered to be at high risk of developing VTE. The rates of DVT in patients having surgery for injuries below the femur are poorly studied. Many of these patients have a decreased number of risk factors (eg, younger age, overall healthier status) compared to those having fractured femurs, thus reducing some of their risk factors.[9]

Neurosurgery

Patients undergoing neurosurgery (ie, nonspine) are considered to be in the moderate risk category for the development of postoperative VTE. Some of the risk factors for this population include decreased leg strength and the length of the operative procedure.[9]

Trauma, Spinal Cord Injury, Burns

Trauma patients are at the highest risk (ie, greater than 50%) among hospitalized patients with a risk of having DVT. Pulmonary embolism is the third leading cause of death in those trauma patients who do not receive prophylaxis and who survive the first 24 hours after injury. Trauma patients with spinal cord injury are at greatest risk, with 60% to 100% reported as having DVT.[9]

Serious burns result in a patient with profound systemic hypercoagulabilty. This factor, combined with prolonged bed rest, multiple surgical procedures, femoral venous catheter insertion, and recurrent bouts of sepsis, increases the risk of DVT.[9]

Malignancy

Venous thromboembolism is a common complication seen in patients with cancer, accounting for nearly 20% of all patients with a new VTE diagnosis. Comparing those with and without cancer, those with cancer have a sixfold increased risk of developing VTE. When a cancer patient has a surgical procedure, the risk of postoperative DVT is more than twice that of a noncancer

patient, and the risk of a fatal PE is more than three times that of a noncancer patient undergoing a similar procedure.[9]

Critical Care

Patients in a critical care setting have multiple risk factors for VTE (eg, recent surgery, trauma, sepsis, malignancy, immobilization, stroke, advanced age, heart or respiratory failure, previous VTE, and pregnancy), many occurring before the admission. The critical care patient also has additional risk factors such as immobilization, use of pharmacologic agents for paralysis or sedation, central venous access catheters, surgical procedures, sepsis, mechanical ventilation, vasopressor use, heart failure, renal dialysis, and depletion of endogenous anticoagulants during their stay in critical care unit.[9]

Nursing Process Application

Application of the nursing process when caring for patients at risk for DVT or PE includes the use of the Perioperative Nursing Data Set (PNDS). The PNDS vocabulary is a clinically relevant and empirically validated standardized nursing language. It is related to the delivery of care in the perioperative setting. This standardized language consists of a collection of data elements and includes perioperative nursing diagnosis, interventions, and outcomes. In 1999, the American Nurses Association Committee on Nursing Practice recognized the PNDS information infrastructure as a data set useful in the practice of nursing.

The Perioperative Patient Focused Model provides the conceptual framework for the PNDS and the model for perioperative nursing practice.[37] The patient and his or her family members are the core of the PNDS model. The model depicts perioperative nursing in four domains and illustrates the relationship between the patient, family members, and the care provided by the professional perioperative nurse. The patient-centered domains are:

- **D1** Safety
- **D2** Physiological Responses
- **D3-A** Behavioral Responses—Patient and Family: Knowledge
- **D3-B** Behavioral Responses—Patient and Family: Ethics
- **D4** Health System

A unique identifier represents each data element in the PNDS. Domains are represented by the letter "D," followed by numbers one through four to indicate the particular domain being addressed. Nursing diagnoses are represented by the letter "X" and a number unique to the diagnosis. Interventions are represented by the letter "I" and a number. These designations are used in this document as appropriate. Desired outcomes are represented by the letter "O."[37]

The following is a partial list of outcomes, nursing diagnoses, and interventions adapted from the PNDS vocabulary that may be associated with the individual with a potential for or having a diagnosis of venous stasis, DVT, or PE. The outcomes, nursing diagnosis, and interventions are grouped by domains.

Domain/Outcome/Nursing Diagnosis/Interventions			Preop	Intraop	Postop
DOMAIN 1: SAFETY					
O5	**The patient is free from signs and symptoms of injury related to positioning.**				
X40	Perioperative positioning injury, risk for impairment of tissue perfusion, or length of surgical procedure.				
X61	Tissue perfusion: Ineffective.				
	I77	Implements protective measures to prevent skin/tissue injury due to mechanical sources.	X	X	X
	I64	Identifies physical alterations that require additional precautions for procedure-specific positioning.	X		
	I96	Positions the patient.		X	
	I38	Evaluates for signs and symptoms of injury as a result of positioning.	X	X	X
	I152	Evaluates for signs and symptoms of physical injury to skin and tissue.	X	X	X
	I138	Implements protective measures prior to operative or invasive procedure.			
	I77	Implements protective measures to prevent skin/tissue injury due to mechanical sources.	X	X	X
	I15	Assesses factors related to risks for ineffective tissue perfusion.	X	X	X

Domain/Outcome/Nursing Diagnosis/Interventions			Preop	Intraop	Postop
O9	**The patient receives appropriate medication(s), safely administered during the perioperative period.**				
X29	Injury, risk due to positioning and inadequate mobility.				
	I8	Administers prescribed medications and solutions.	X	X	X
	I51	Evaluates response to medications.	X	X	X
DOMAIN 2: PHYSIOLOGIC					
O11	**The patient has wound/tissue perfusion consistent with or improved from baseline levels established preoperatively.**				
X18	Fluid volume, risks related to inadequate circulation.				
X28	Infection, risk of circulation or tissue perfusion.				
	I60	Identifies baseline tissue perfusion.	X	X	
	I15	Assesses factors related to risks for ineffective tissue perfusion.	X	X	
	I46	Evaluates postoperative tissue perfusion.		X	X
	I3	Administers care to invasive device sites.	X	X	X
O14	**The patient's respiratory function is consistent with or improved from baseline levels established preoperatively.**				
X7	Breathing pattern: Ineffective due to respiratory compromise.				
X21	Gas exchange: Impaired due to inadequate respiratory exchange.				
X55	Ventilation: Impaired spontaneous.				
	I87	Monitors changes in respiratory status.	X	X	X
	I121	Uses monitoring equipment to assess respiratory status.	X	X	X
	I110	Recognizes and reports deviation in arterial blood gas studies.	X	X	X
	I45	Evaluates postoperative respiratory status.		X	X
O15	**The patient's cardiovascular status is consistent with or improved from baseline levels established preoperatively.**				
X8	Cardiac output: Decreased related to impaired tissue perfusion.				
X41	Peripheral neurovascular dysfunction: Risk for.				
	I59	Identifies baseline cardiac status.			
	I120	Uses monitoring equipment to assess cardiac status.	X	X	X
	I44	Evaluates postoperative cardiac status.	X	X	
O30	**The patient's neurological status is consistent with or improved from baseline levels established preoperatively.**				
X11	Confusion: Acute related to physiological conditions.				
X1	Activity intolerance: Related to multiple conditional factors.				
	I146	Evaluates postoperative neurological status.	X	X	
	I111	Recognizes and reports deviations in diagnostic study results.	X	X	X
DOMAIN 3-A: BEHAVIORAL RESPONSES—PATIENT AND FAMILY: KNOWLEDGE					
O19	**The patient demonstrates knowledge of medication management.**				
X71	Health maintenance: Ineffective.				
	I104	Provides instruction about prescribed medications.			
O21	**The patient participates in the rehabilitation process.**				
X34	Physical modality: Impaired due to injury or disease process.				
	I106	Provides instructions based on age and identified needs.	X		X
	I50	Evaluates response to instructions.	X		X

Domain/Outcome/Nursing Diagnosis/Interventions			Preop	Intraop	Postop
DOMAIN 3-B: BEHAVIORAL RESPONSES—PATIENT AND FAMILY: ETHICS					
O23	**The patient participates in decisions affecting his or her perioperative plan of care.**				
X35	Noncompliance due to knowledge deficit or patients' rights.				
X71	Health maintenance: Ineffective.				
	I80	Includes patient and family members in discharge planning.	X		X
	I79	Includes family members in preoperative teaching.	X		

Additional interventions should be considered by the perioperative nurse when developing an individualized plan of care for the patient with a potential of venous stasis, DVT, or PE. Interventions should be selected according to the procedure to be performed, and they should address the potential prevention of venous stasis, DVT, or PE. The interventions below are grouped by location of care. Some of the interventions are repeated because this intervention should be carried out in all three areas. An intervention listed in only one area does not mean that it may not be appropriate for other areas depending upon the situation and the setting.[38,39]

PREOPERATIVE.

- Assess all patients to determine risk factors for venous stasis, DVT, or PE. Data collection involves patients and their significant others. A tool such as Table 1 or Appendix A may be used to classify the patient's risk level. This risk level can be used in determining the appropriate method of prophylaxis.
- The choice of therapy is a medical decision, but the perioperative registered nurse should collaborate with surgeons, anesthesia care providers, and other health care professionals involved regarding initiating the established protocol orders for DVT or PE prevention.[9]
  - If antiembolism stockings are the treatment of choice, the preoperative nurse should measure the patient for proper fit and apply as ordered. If applied incorrectly, stockings may cause DVT, arterial ischemia, gangrene, and necrosis and result in amputation by constricting blood flow. If the stockings roll down, there is the potential that a tourniquet effect will be created.[31]
  - Ensure that IPC devices are applied properly, in a timely manner per manufacturer's recommendation, if ordered.[31]
  - Ensure that the devices do not hinder ambulation.[9]
  - Ensure that prophylactic medications are given as ordered.[9]

INTRAOPERATIVE.

- Confirm that the antiembolism stockings and IPC devices are placed on the patient properly and that medications are given as ordered when performing the preoperative assessment.[9,31]
- After the patient is transferred to the OR bed, the IPC device should be turned on before the beginning of induction of general anesthesia or before regional anesthesia has been administered.[1]
- Instruct the surgical assistant or scrub nurse to avoid extreme degrees of flexion and internal rotation of hip and knee. Preventing endothelial damage due to abnormal leg positioning (eg, during hip surgery when the acetabular head is dislocated and the leg brought across the body, causing kinking of the femoral vein) is an important responsibility shared by the surgery team and the circulating nurse.[28]
- Be aware that unnecessarily high tourniquet pressures and prolonged periods of inflation should be avoided, if possible, when a tourniquet is used.[28]
- Avoid the reverse Trendelenburg position whenever possible because of a potential reduction in blood flow rate to the legs.[28]
- Check antiembolism stockings to ensure that they have not rolled down during movement to the OR bed or during positioning.[31]
- Ensure that the IPC devices work properly during the surgical procedure.[31]
- Continue the established protocol orders for DVT or PE prevention.[9]

POSTOPERATIVE.

- Ensure that the IPC devices do not hinder ambulation.[9]
- Ensure that the device is removed for only a very short period of time.[1]
- Ensure that the IPC device is turned on and working properly and confirm proper functioning and application with the nurse assuming care, if ordered.[1]

TABLE 1
Classification of Risk Levels[1-3]

Risk level	Surgical parameters	Prophylactics
Low	Uncomplicated minor surgery in patients younger than 40 years with no clinical risk factors requiring general anesthesia less than 30 minutes.	• Early and aggressive ambulation • No specific prophylaxis
Moderate	Surgical patients aged 40 to 60 years with no additional risk factors. Major surgery in patients younger than 40 years with no additional risk factors requiring general anesthesia longer than 30 minutes. Minor surgery in patients with risk factors.	• Low-dose unfractionated heparin every 12 hours • Low-molecular-weight heparin (< 3,400 units daily) • Graded compression stockings or intermittent pneumatic compression (IPC)
High	Major surgery in patients older than 60 years without additional risk factors. Major surgery in patients aged 40-60 years with additional risk factors. Patients with history of myocardial infarction. Medical patients with risk factors.	• Low-dose unfractionated heparin; 5,000 units every 8 hours • Low-molecular-weight heparin (> 3,400 units daily); elastic stockings or IPC
Highest	Major surgery in patients older than 40 years with multiple risk factors (eg, prior venous thromboembolism, malignant disease, hypercoagulable state). Patients with elective major lower extremity orthopedic surgery, hip fracture, stroke, multiple trauma, or spinal cord injury.	Low-molecular-weight heparin (> 3,400 units daily); fondaparinux, oral vitamin K antagonists (international normalized ratio 2-3); low-dose unfractionated heparin or low-molecular-weight heparin; IPC or graded compression stockings.

1. Anderson FA, Audet AM. Best practices: preventing deep vein thrombosis and pulmonary embolism. DVT.org. Available at: http://www.outcomes-umassmed.org/dvt/best_practice. Accessed June 18, 2006.
2. Wilson Tang WH. Perioperative DVT prophylaxis. WebMD. Available at: http://emedicine.com/med/topic3164.htm. Accessed July 5, 2006.
3. Arcelus JI, Kudma JC, Caprini JA. Venous thromboembolism following major orthopedic surgery: what is the risk after discharge? Orthopedics. *2006;29:506. Available at: http://www.orthosupersite.com/print.asp?rID=17193. Accessed July 30, 2006.*

- Instruct the patient in the importance of moving and ambulation in preventing postoperative complications per the surgeon's postoperative orders.[1]
- Continue the established protocol orders for DVT or PE prevention.[9]
- Educate patients, caregivers, and health care professionals about prevention of DVT. Education should include common signs and symptoms such as leg pain, swelling, and unexplained shortness of breath.[22]

TREATMENT AND PREVENTION

A protocol for the prevention of DVT or PE, detailing the primary and secondary prophylactic therapy methods, should be developed by each facility.[22]

PRIMARY PREVENTION

Perioperative or primary prophylactic therapy in patients with risk factors for DVT or PE involves their prevention, if possible. If preventive measures are unsuccessful, the goal is to reduce the consequences of DVT or PE. The two main strategies are use of nonpharmacologic interventions and pharmacologic interventions. The Seventh American College of Chest Physicians Consensus Conference on Antithrombotic Therapy recommends that patients be classified as low, moderate, high, or very high risk for the development of DVT or PE, and that prophylactic regimens be used according to risk stratification. Examples of the recommended risk stratification are described in Table 1 and Appendix A.[9] A protocol outlining the preventive measures to be taken for each risk classification should be

developed by every health care organization.[22] The pharmacologic and nonpharmacologic interventions can be used together or separately depending upon the patient's risk classification.

NONPHARMACOLOGIC INTERVENTIONS. Low-risk patients should receive nonpharmacologic prophylaxis during the perioperative period until ambulation can be initiated.[20] These measures include elastic stockings, IPC devices, and early ambulation, and are especially useful when heparin therapy is contraindicated.

Compression stockings (either thigh-high length or calf-high length) frequently are used after surgery and during airplane rides to promote circulation.[33] Compression stockings may be uncomfortable, but their effect on blood circulation helps to reduce the potential for DVT. Calf-length elastic stockings are effective for patients who undergo low-risk procedures and are relatively free of complications.[1,20]

Intermittent pneumatic compression (IPC) is a nonpharmacologic prophylactic method used to reduce stasis and improve venous return from the lower extremities. Intermittent pneumatic compression is effective in patients with moderately high risk. Devices are available in various designs (eg, foot pumps [arteriovenous impulse system], intermittent uniform pneumatic compression).[1,9]

Caution should be taken in using IPC on patients who have been on bed rest or immobilized for more than 72 hours because of the risk of disrupting newly formed clots.[17,20] Contraindications for IPC include any local leg condition or venous flow compromise that the sleeves would interfere with, such as

- dermatitis,
- gangrene,
- severe arteriosclerosis or other ischemic vascular disease,
- massive leg edema,
- pulmonary edema with congestive heart failure,
- extreme leg deformity,
- preexisting deep vein thrombosis, and
- injuries or surgical sites located in the lower extremities.[26,40]

Early ambulation is the most important and effective nonpharmacologic approach in the prevention of DVT and PE.[3,14,20,38] Vena cava filters can be used in patients who cannot tolerate pharmacologic interventions or in those for whom alternative treatments have been unsuccessful. The primary limitation of these filters is that they become ineffective after one to two years.[5]

PHARMACOLOGIC INTERVENTIONS. Pharmacologic interventions are anticoagulant medications that inhibit the blood from clotting. The pharmacologic regimen consists of medications such as coumarin, synthetic pentasacchride (ie, fondaparinux), low-molecular-weight heparin, and low-dose heparin. The route of administration is dependent on the medication. The platelet active drugs and coumarin are administered orally; heparin and fondaparinux are given by injection.[4,5]

Complications of anticoagulant therapy include bleeding, hematoma formation, compartment syndrome, and heparin-induced thrombocytopenia. The bleeding may be major or minor. Major bleeding can include hemorrhage that alters the patient's treatment or outcome.[5]

Anticoagulant therapy is contraindicated in patients experiencing hemorrhage or with multiple traumas that are in an unstable condition, pregnancy, head trauma, or patients with a spinal catheter in place. Anticoagulants should be given with caution in patients with a history of cerebral or gastrointestinal hemorrhage, thrombocytopenia, or coagulopathy.[5]

SECONDARY PREVENTION

Secondary prevention is screening of high-risk postoperative patients. This screening leads to the early detection and therefore timely treatment of subclinical DVT. This is particularly important in those patients in whom primary prophylaxis is either contraindicated or ineffective. Routine ultrasonography screening at discharge or during outpatient follow-up is not affordable or available in many facilities and therefore is not recommended in asymptomatic patients.[3,38]

EVALUATION OF OUTCOMES

The perioperative nurse evaluates the patient's progress toward attainment of outcomes. This evaluation should be systematic, ongoing, and documented using the PNDS.

Outcome indicators will vary according to specific patient outcomes. These indicators may include

- physiological indicators (eg, cardiovascular status);
- cognitive indicators (eg, asking appropriate questions);
- affective indicators (eg, willingness to comply with treatment regimens); and
- supportive resources (eg, family member participation in care planning and delivery).

Patient Satisfaction

Evaluation of the patient's progress is based on observation of the patient's response to nursing interventions and the effectiveness of these interventions in moving the patient toward the preferred outcomes. Preferred patient outcomes, nursing interventions, and potentially applicable nursing diagnoses are articulated in the standardized perioperative nursing vocabulary, which provides the basis for documentation of perioperative nursing practice. Ongoing assessments should be used to revise diagnoses, outcomes, and the plan of care as needed. Revisions in diagnoses, outcomes, and the plan of care should be documented. The patient and his or her significant others, working in conjunction with the health care provider, should be involved in the process.

Quality Assessment

Health care organizations that develop a protocol should evaluate the protocol on a periodic basis. The protocol should be evaluated against current research and research-based established guidelines. The effectiveness of the protocol should be evaluated using information supplied by physicians and examination of records of postoperative readmissions for venous stasis or its complications.

Summary

The assessment for patient risk and the prevention of DVT and PE are of prime importance in all patients. Assessment and prevention are especially important in the patient undergoing a surgical intervention. Perioperative nurses can use this document to increase their awareness of and skill at detecting patients at increased risk and to improve their skills in caring for these patients.

References

1. Van Wicklin SA, Ward KS, Cantrell SW. Implementing a research utilization plan for prevention of deep vein thrombosis. *AORN J.* 2006;83:1353-1362.
2. Horlocker TT. New guidelines for antithrombotic therapy: making blood thinner than water. *ASA Newsletter.* 2005;69. Available at: http://www.asahq.orgNewsletters/2005/05-05/horlocker05-05.html. Accessed July 3, 2006.
3. Anderson FA, Audet AM. Best practices: preventing deep vein thrombosis and pulmonary embolism. DVT.org. Available at: http://www.outcomes-umassmed.org/dvt/best_practice. Accessed December 18, 2006.
4. Quader MA, Stump LS, Sumpio BE. Treatment of thrombosis. *J Am Coll Surg.* 1998;187:641-658. Available at: http//www.facs.org/jacs/articles/sumpioend.html. Accessed July 12, 2006.
5. Ennis RS. Deep vein thrombosis prophylaxis in orthopedic surgery. WebMD. Available at: http://emedicine.com/orthoped/topic600.htm. Accessed August 23, 2006.
6. Deep vein thrombosis. American Academy of Orthopaedic Surgeons. Available at: http://orthoinfo.aaos.org/fact/printer_page.cfm?topcategory=Knee&Thread_ID+264. Accessed July 28, 2006.
7. Joint Commission on Accreditation of Healthcare Organizations. National data report: VTE measures and consensus standards under development. *Jt Comm Benchmark.* May/June 2006;8(3):10-11.
8. American College of Obstetricians and Gynecologists. Clinical management guidelines for obstetricians–gynecologists. *ACOG Prac Bull.* 200;21:1-10. Available at: http://www.acog.org/acog_districts/dist_notice.cfm?recno=14&bulletin=1955. Accessed July 3, 2006.
9. Geerts WH, Pineo GF, Heit JA, et al. Prevention of venous thromboembolism: the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest.* 2004;126 [Suppl]:338S-400S. Available at: http://www.chestjournal.org/cgi/content/full/126/3suppl/338S. Accessed June 13, 2006.
10. *Mosby's Dictionary of Medicine, Nursing & Health Professions.* 7th ed. St Louis, Mo: Mosby; 2006:115-116, 335, 520, 1454, 1849, 1949.
11. Deep venous thrombosis (DVT). Doctors Corner Internet Group, Inc. Available at: http://your-doctor.com/healthinfocenter/medical-conditions/cardiovascular/cardiac-conditions/vascular-dz/dvt.html. Accessed June 30, 2006.
12. Barnes C, Newall F, Mongale P. Post-thrombotic syndrome. *Arch Dis Child.* 2002;86:221-214. Available at: http://adc.bmjjournals.com/cgi/reprint/86/3/212. Accessed July 3, 2006.

13. Büller HR, Agnelli G, Hull RD, Hyers TM, Prins MH, Raskob GE. Antithrombotic therapy for venous thromboembolic disease: the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest.* 2004;126 [Suppl]:401S-428S.
14. O'Donnell M, Weltz JI. Thromboprophylaxis in surgical patients. *Can J Surg.* 2003;46:129-135.
15. Merli GJ. Reducing patient risk. *RN suppl.* June 2004:6-8.
16. Goldhaber SZ, Fanikos J. Cardiology patient pages. Prevention of deep vein thrombosis and pulmonary embolism. *Circulation.* 2004;110:e445-e447. Available at: http://circ.ahajournals.org/cgi/content/full/110/16/e445. Accessed June 13, 2006.
17. Sharma S. Pulmonary embolism. WebMD. Available at: http://www.emedicine.com/med/topic1958.htm. Accessed June 20, 2006.
18. Day MW. Recognizing and managing deep vein thrombosis. *Nursing.* 2003;33:36-41.
19. Agency for Healthcare Research and Quality. Postoperative pulmonary embolism or deep vein thrombosis: rate per 1,000 surgical discharges. *AHRQ Quality Indicators: Guide to Patient Safety Indicators* [version 2.1, revision 3; January 17, 2005]. AHRQ Pub #03-R203. Available at: http://www.qualitymeasures.ahrq.gov/summary/summary.aspx?ss=1&doc_id=6025. Accessed July 18, 2006.
20. Goldhaber SZ, Tapson VF. A prospective registry of 5,451 patients with ultrasound-confirmed deep vein thrombosis. *Am J Cardiol.* 2004;93:259-262. Available at: http://www.sciencedirect.com/science. Accessed August 22, 2006.
21. Goldhaber SZ, Dunn K, MacDougall RC. New onset of venous thromboembolism among hospitalized patients at Brigham and Women's Hospital is caused more often by prophylaxis failure than by withholding treatment. *Chest.* 2000;118:1680-1684. Available at: http://www.chestjournal.org/cgi/content/full/118/6/1680. Accessed August 22, 2006.
22. American Public Health Association. Deep vein thrombosis: advancing awareness to protect patients' lives. *APHA White Paper.* February 26, 2003:1-12.
23. Thrombophlebitis. In: *Medline Plus Medical Encyclopedia.* Medline. Available at: http://www.nlm.nih.gov/medlineplus/ency/article/001108.htm. Accessed July 10, 2006.
24. Superficial thrombophlebitis. Vascular Unit. Available at: http://www.freevas.demon.co.uk/paper1.htm. Accessed July 19, 2006.
25. Ascher E, Hingorani A. Superficial venous thrombophlebitis. American Venous Forum. Available at: http//www.venous-info.com/handbook/hbk07a.html. Accessed February 10, 2006.
26. Oakley MJ, Wheelwright EF, James PJ. Pneumatic compression boots for prophylaxis against deep vein thrombosis: beware occult arterial disease. *BMJ.* 1998;316:454-455. Available at: http://bmj.bmjjournals.com/cgi/content/full/316/7129/454. Accessed July 7, 2006.
27. Intrinsic pathway of blood coagulation. General Practice Notebook. Available at: http://www.gpnotebook.co.uk/simplepage.cfm?ID=26869801&linkID=1002&cook=no. Accessed September 4, 2004.
28. Arnold A. DVT prophylaxis in the perioperative setting. *Br J Perioper Nurs.* 2002;12:294-297.
29. Leeper KV. Detecting and treating pulmonary embolism. *Venous Thromboembolism: An Update, RN suppl.* June 25, 2004:10-11.
30. Wahl WL, Brandt MM, Ahrns KS, et al. Venous thrombosis incidence in burn patients: preliminary results of a prospective study. *J Burn Care Rehabil.* 2002;23:97-102.
31. Arnold A. DVT prophylaxis in the perioperative setting. *Br J Perioper Nurs.* 2002;12:326-332.
32. Male C, Chait P, Andrew M, Hanna K, Julian J, Mitchell L. Central venous line-related thrombosis in children: association with central venous line location and insertion technique. *Blood.* 2003;101:4273-4278. Available at: http://www.bloodjournal.org/cgi/reprint/101/11/4273. Accessed July 3, 2006.
33. Deep vein thrombosis. MDtravelhealth.com. Available at: http://www.mdtravelhealth.com/illness/deep_vein_thrombosis.html. Accessed July 20, 2006.
34. Levy ML, Granville RC, Hart D, Meltzer H. Deep venous thrombosis in children and adolescents. *J Neurosurg.* 2004;101(Suppl):32-37.
35. Pediatric hemostasis: decidedly different. McMaster University Medical Center and the Hospital for Sick Children. Available at: http://www.beckman.com/literature/ClinDiag/mcmaster.pdf#search='dvtchildren'. Accessed June 30, 2006.
36. Monteleone PM. Thromboembolism. WebMD. Available at: http://www.emedicine.com/ped/topic2239.htm. Accessed July 3, 2006.
37. Beyea SC, ed. *Perioperative Nursing Data Set.* 2nd ed. Denver, Colo: AORN, Inc; 2002:81-184.
38. Wilson Tang WH. Perioperative DVT prophylaxis. WebMD. Available at: http://emedicine.com/med/topic3164.htm. Accessed July 5, 2006.
39. Arcelus JI, Kudrna JC, Caprini JA. Venous thromboembolism following major orthopedic surgery: what is the risk after discharge? *Orthopedics.* 2006;29:506. Available at: http://www.orthosupersite.com/print.asp?rID=17193. Accessed December 7, 2006.
40. Auguste KI, Quiñones-Hinojosa A, Berger MS. Efficacy of mechanical prophylaxis for venous thromboembolism in patients with brain tumors. *Neurosurg focus* [electronic resource]. 2004;17:1-5. Available at: http://www.aans.org/education/journal/neurosurgical/oct04/17-4-3.pdf. Accessed September 27, 2006.

Approved by the AORN Board of Directors, November 2006.

APPENDIX A
Venous Thromboembolism Risk Factor Assessment

Patient's Name _____ Age: _____ Sex: _____ Wgt: _____ lbs

Choose all that apply

Each risk factor represents 1 point

- ❒ Age 41–60 years
- ❒ Minor surgery planned
- ❒ History of prior major surgery
- ❒ Varicose veins
- ❒ History of inflammatory bowel disease
- ❒ Swollen legs (current)
- ❒ Obesity (BMI > 30)
- ❒ Acute myocardial infarction (< 1 month)
- ❒ Congestive heart failure (< 1 month)
- ❒ Sepsis (< 1 month)
- ❒ Serious lung disease, including pneumonia (< 1 month)
- ❒ Abnormal pulmonary function (COPD)
- ❒ Medical patient currently at bed rest
- ❒ Leg plaster cast or brace

Each risk factor represents 3 points

- ❒ Age over 75 years
- ❒ Major surgery lasting 2-3 hours
- ❒ BMI > 50 (venous stasis syndrome)
- ❒ History of SVT, DVT/PE
- ❒ Family history of DVT/PE
- ❒ Present cancer or chemotherapy
- ❒ Positive Factor V Leiden
- ❒ Positive Prothrombin 20210A
- ❒ Elevated serum homocysteine
- ❒ Positive Lupus anticoagulant
- ❒ Elevated anticardiolipin antibodies
- ❒ Heparin-induced thrombocytopenia (HIT)
- ❒ Other thrombophilia

  Type _____

Choose all that apply

Each risk factor represents 2 points

- ❒ Age 60–74 years
- ❒ Major surgery (> 60 minutes)
- ❒ Arthroscopic surgery (> 60 minutes)
- ❒ Laparoscopic surgery (> 60 minutes)
- ❒ Previous malignancy
- ❒ Central venous access
- ❒ Morbid obesity (BMI > 40)

Each risk factor represents 5 points

- ❒ Elective major lower extremity arthroplasty
- ❒ Hip, pelvis, or leg fracture (< 1 month)
- ❒ Stroke (< 1 month)
- ❒ Multiple trauma (< 1 month)
- ❒ Acute spinal cord injury (paralysis) (< 1 month)
- ❒ Major surgery lasting over 3 hours

For women only (each risk factor represents 1 point)

- ❒ Oral contraceptives or hormone replacement therapy
- ❒ Pregnancy or postpartum (< 1 month)
- ❒ History of unexplained stillborn infant, recurrent spontaneous abortion (= 3), premature birth with toxemia or growth-restricted infant

Total Risk Factor Score_____

Please see following page for prophylaxis safety considerations. Revised July 23, 2006

APPENDIX A, CONTINUED
Venous Thromboembolism Risk Factor Assessment

VTE Risk and Suggested Prophylaxis for Surgical Patients				
Total Risk Factor Score	**Incidence of DVT**	**Risk Level**	**Prophylaxis Regimen**	**Legend**
0-1	< 10%	Low Risk	No specific measures; early ambulation	**ES** – Elastic Stockings **IPC** – Intermittent Pneumatic Compression **LDUH** – Low Dose Unfractionated Heparin **LMWH** – Low Molecular Weight Heparin **FXaI** – Factor X Inhibitor **Use for major orthopedic surgery*
2	10-20%	Moderate Risk	ES, IPC, LDUH (5000 U BID), or LWMH (< 3400 U)	
3-4	20-40%	High Risk	IPC, LDUH (5000 U TID), or LMWH (> 3400 U)	
5 or more	40-80% 1-5% mortality	Highest Risk	Pharmacological: LDUH, LMWH (> 3400 U)*, Warfarin*, or FXaI* alone or in combination with ES or IPC	

Prophylaxis Safety Considerations: Check Box If Answer Is "YES"
Anticoagulants: Factors Associated With Increased Bleeding
❒ Is patient experiencing any active bleeding?
❒ Does patient have (or has patient had history of) heparin-induced thrombocytopenia?
❒ Is patient's platelet count < 100,000/mm^3?
❒ Is patient taking oral anticoagulants, platelet inhibitors (eg, NSAIDS, Clopidogrel, Salicylates)?
❒ Is patient's creatinine clearance abnormal? If yes, please indicate value _____
If any of the above boxes are checked, the patient may not be a candidate for anticoagulant therapy and you should consider alternative prophylactic measures: elastic stockings and/or IPC.
Intermittent Pneumatic Compression (IPC)
❒ Does patient have severe peripheral arterial disease?
❒ Does patient have congestive heart failure?
❒ Does patient have an acute superficial/deep vein thrombosis?
If any of the above boxes are checked, then patient may not be a candidate for intermittent compression therapy and you should consider alternative prophylactic measures.

Examiner _____ **Date**_____

Based on: Geerts WH, et al. Prevention of Venous Thromboembolism. Chest. *2004;126(suppl 3):338S-400S; Nicolaides AN, et al.* 2001 International Consensus Statement: Prevention of Venous Thromboembolism, Guidelines According to Scientific Evidence*; Arcelus JI, Caprini JA, Traverso CI. International perspective on venous thromboembolism prophylaxis in surgery.* Semin Thromb Hemost. *1991;17(4):322-325; Borow M, Goldson HJ. Postoperative venous thrombosis. Evaluation of five methods of treatment.* Am J Surg. *1981;141(2):245-251; Caprini JA, Arcelus I, Traverso CI, et al. Clinical assessment of venous thromboembolic risk in surgical patients.* Semin Thromb Hemost. *1991;17(suppl 3):304-312; Caprini JA, Arcelus JI, et al. State-of-the-art venous thromboembolism prophylaxis.* Scope. *2001;8:228-240; Caprini JA, Arcelus JI, Reyna JJ. Effective risk stratification of surgical and nonsurgical patients for venous thromboembolic disease.* Seminars in Hematology. *April 2001;38(2)(suppl 5):12-19; Caprini, JA. Thrombosis risk assessment as a guide to quality patient care.* Dis Mon. *2005; 1:70-78; Oger E. Incidence of venous thromboembolism: a community-based study in western France.* Thromb Haemost. *2000:657-660; Turpie AG, Bauer KA, Eriksson BI, et al. Fondaparinux vs. enoxaparin for the prevention of venous thromboembolism in major orthopedic surgery: a meta-analysis of 4 randomized double-blind studies.* Arch Intern Med. *2002;162(16):1833-1840; Ringley, et al. Evaluation of intermittent pneumatic compression boots in congestive heart failure.* American Surgeon. *2002;68(3):286-289; Morris, et al. Effects of supine intermittent compression on arterial inflow to the lower limb.* Archives of Surgery. *2002;137(11):1269-1273; Sugarman HJ, et al.* Ann Surg. *2001;234(1):41-46.*