Staff Development Special

Follow this guide to lead a staff discussion on deep vein thrombosis.

Get the edge on deep vein thrombosis

Head off progression of this deadly condition by knowing when to assess and what to look for during patient screening.

Teaching Guide

GENERAL PURPOSE: To provide registered professional nurses with an overview of the prevention, identification, and treatment of deep vein thrombosis.

LEARNING OBJECTIVES: After reading this article and taking this test, you should be able to:

1. Identify the clinical manifestations and interventions related to deep vein thrombosis and venous thromboembolism.
2. Discuss various diagnostic and laboratory tests used in the identification and treatment of deep vein thrombosis and venous thromboembolism.
3. Describe the purpose and adverse effects of the medications prescribed for deep vein thrombosis and venous thromboembolism.

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Staff Development CE

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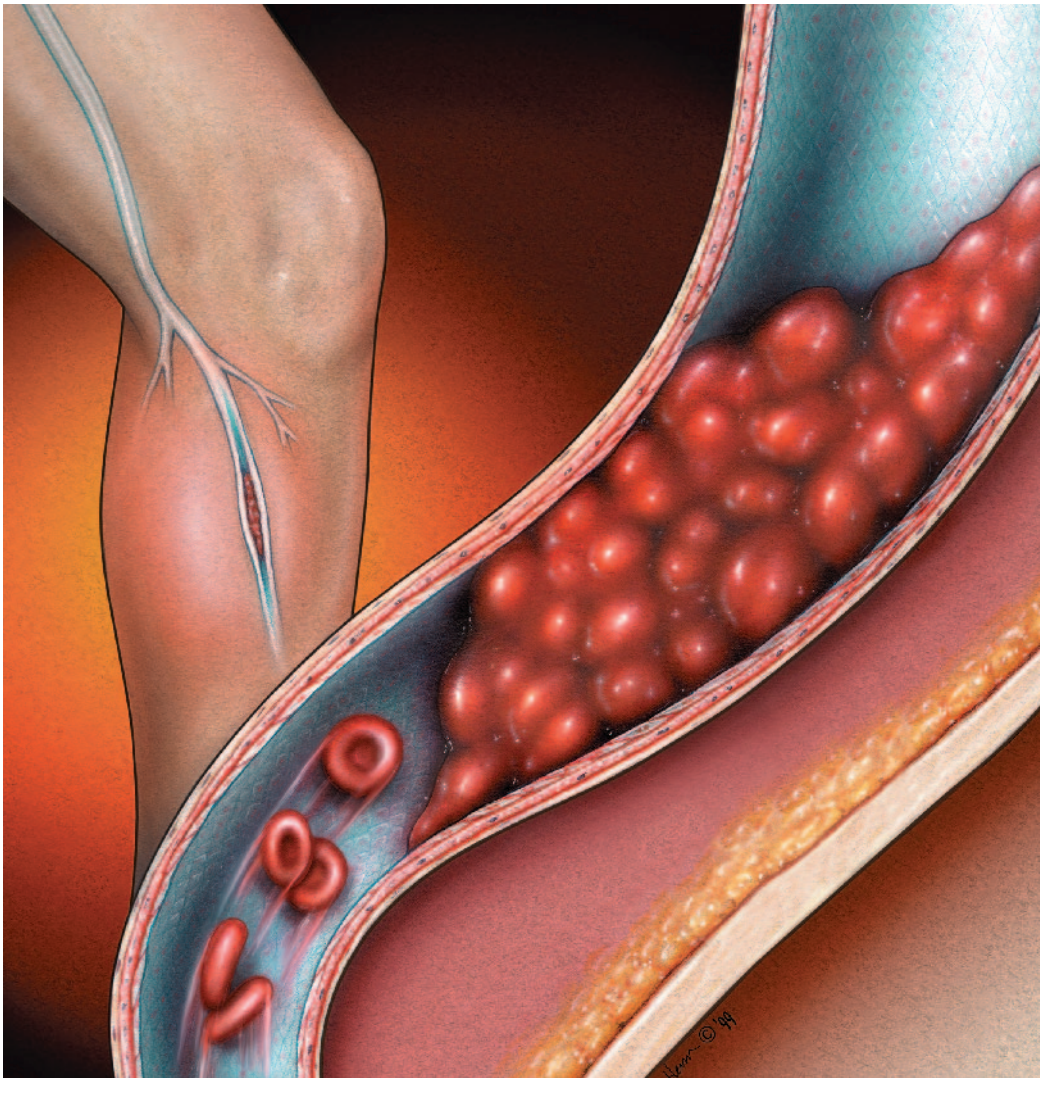

Head off progression of this deadly condition by knowing when to assess and what to look for during patient screening.

Wayne Heim

CE 1.5 ANCC/AACN CONTACT HOURs
Rx 1.0 ANCC PHARM. CONTACT HOUR

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Abstract: Review practical DVT screening considerations to enhance assessment skills. [Nurs Manage 2004:35(1):21-30]

Deep vein thrombosis (DVT), or thrombophlebitis, affects approximately 1.6 of every 1,000 Americans annually, with more than 200,000 first-time DVT episodes diagnosed each year.[1] The exact incidence is unclear, due to variations in patient symptomatology, study populations, and diagnostic accuracy.

The process of thrombus formation in the veins and arteries is similar, but venous thrombi are much more common. Thrombus development can occur in either the superficial or the deep veins of the extremities. Superficial vein thrombosis of the saphenous veins or their tributaries is usually treated with anti-inflammatory agents like ibuprofen. Although DVT develops most often in the legs, its occurrence in the upper extremities is becoming more common due to increased use of subclavian venous catheters.[2] Mortality from DVT varies: 13% to 21% for lower extremity DVT, and as high as 48% for those with upper extremity DVT.[3]

In 1856, German pathologist Rudolph Virchow became the first to identify the three factors necessary for DVT development:

1. venous stasis or obstruction
2. overt vessel injury
3. intravascular conditions such as intimal irritation; roughening, inflammation, infection, and abnormalities of coagulation or fibrinolysis.[4-7]

DVT patients remain at risk for pulmonary embolism (PE), a life-threatening complication that may contribute to between 60,000 and 200,000 deaths each year.[8] The incidence of PE increases with the number of risk factors. Ninety percent of all PEs originate from the lower extremities. One study documented that 56% of deaths from PE occurred in patients not receiving DVT prophylaxis. When properly diagnosed and treated, mortality declines to 8% to 9%.[9] Signs and symptoms of PE include sudden dyspnea, chest pain that worsens "with inspiration," and hemoptysis.[10]

A majority of those with proximal DVT also have PE, whether symptomatic or not. *Venous thromboembolism* (VTE) is the term that includes both DVT and PE, as they're each part of the same condition.[11]

VTE development

A meshwork of protein strands made of fibrin, a *thrombus* forms when a chemical stimulus from either the intrinsic or extrinsic coagulation pathway activates. Initially, platelets adhere, then activate, and then aggregate to form a plug. This initiates a complex chain of chemical reactions known as the *coagulation cascade*. In stage one of this cascade, either the intrinsic or extrinsic pathway ends with the formation of prothrombin-converting factor. The remainder of the coagulation cascade uses one common pathway.

In stage two, prothrombin-converting factor begins the series of chemical interactions that slowly converts prothrombin to thrombin. Once formed, thrombin accelerates its own rate of formation. Fibrinogen interacts with thrombin in stage three to form fibrin. Other cells, such as erythrocytes, phagocytes, and microorganisms, also collect at the site to complete thrombosis development. (See "Clotting cascade.")

Assessment basics

DVT causes more than 600,000 hospitalizations in the United States annually.[12] It's vital to screen all patients carefully for DVT, identifying any predisposing factors. For example, more than 50% of those undergoing orthopedic surgeries (particularly of the knee and hip), 10% to 40% of those receiving abdominal or thoracic operations, and 10% to 20% of those with overt cancer develop DVT.[13] *Secondary DVT* occurs in the setting of a known risk factor, while *primary* or *idiopathic DVT* occurs in the absence of risk factors. (See "Additional predisposing factors.")

A DVT patient may be asymptomatic, particularly if the thrombus exists in a vein of the calf (distal DVT). Additionally, many patients with venous thrombosis in the calf present with a completely normal physical exam. Often, the only abnormality is pain. The classic findings of DVT, present in about 50% of those afflicted, include unilateral leg edema, warmth, erythema, dilated superficial vein, or a palpable cord. Tenderness along the course of the vein, increased tissue turgor, and prominent venous collaterals may also exist. If edema causes soft tissue to compress local nerves, neuralgia will develop and may become perma-

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nent. Don't rely on a positive Homan's sign (pain or soreness in calf on dorsiflexion of the foot) for diagnosis, because it's often not present. Proximal thrombus extends to the popliteal, femoral, or iliofemoral vessels in approximately 20% of patients. When this happens, the clot is more likely to break away from the vessel lumen and cause PE.[14]

DVT diagnosis

The following tests may help diagnose DVT:

♦ *Venous duplex ultrasonography* has emerged as the diagnostic method of choice for DVT, offering better accuracy than indirect measures of blood flow.[15] Venous duplex ultrasonography provides two-dimensional imaging and pulse wave doppler interrogation, with high sensitivity and specificity for proximal DVT, but less for visualization of distal DVT.

If ultrasonography is positive for distal DVT, repeat the test one week later, or serially, to reassess for proximal extension. Routine duplex testing in asymptomatic total hip or knee replacement surgery patients isn't recommended.[16]

♦ *Plethysmography techniques* indirectly measure venous capacitance during physiologic maneuvers by either impedance testing with electrodes (impedance plethysmography) or leg volume testing with air-filled cuffs (phleborheography).[17]

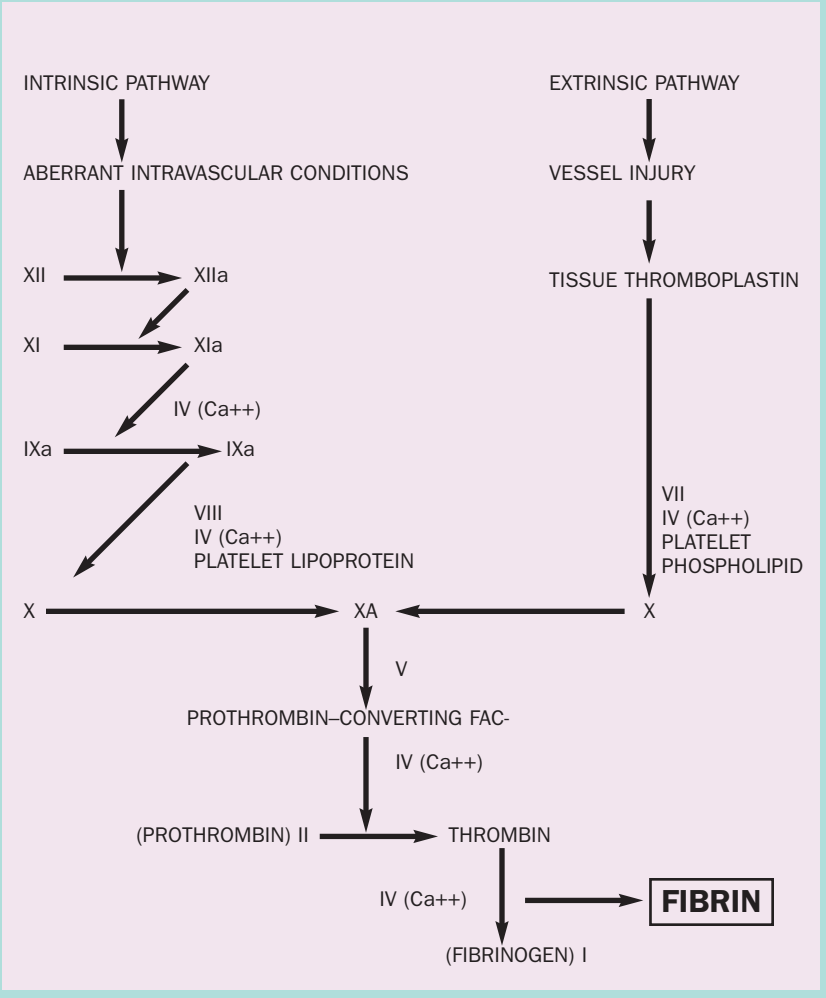
♦ *I-fibrinogen scanning* uses an isotope which, when injected, is taken up by thrombi. This technique is sensitive for distal venous thrombosis but is less specific for DVT in proximal veins.[18]

♦ *Venography injection of contrast medium* into the foot's superficial vein is directed through the venous system with tourniquets to detect filling defects in the deep veins. This process remains the diagnostic reference standard.[19,20]

♦ *Magnetic resonance direct thrombus imaging* is a noninvasive procedure for accurate diagnosis for distal and proximal DVT.[21] Some believe that magnetic resonance venography is superior to other diagnostic tests for detection of pelvic and proximal deep vein thrombosis.[22]

♦ The *D-Dimer assay* is a serum test that can help to exclude DVT. The normal value is less than 500 fibrin equivalent units μ/L. When DVT exists, D-Dimer in serum elevates due to

Clotting cascade



The extrinsic pathway is activated when tissue trauma occurs, causing factor III to interact with factor VII and factor IV to form factor X. Factor X and factor IV interact to form factor V, which is needed to form prothrombin-converting factor.

The intrinsic pathway is activated under conditions such as stress, anxiety, or fear, and in the absence of external tissue injury. Factor VII in the plasma comes into contact with subendothelial substances during platelet aggregation to form factor XI. Factor XI interacts with factor IX, which interacts with factor VIII. This factor—in the presence of factor IV—activates substances that form factor X. As above, factor X—in the presence of factor IV—interacts to form factor V, needed for the formation of prothrombin-converting factor.

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Additional predisposing factors

Other predisposing factors of DVT include:

- advanced age
- trauma
- spinal cord injury
- immobilization
- myocardial infarction
- heart failure
- stroke
- previous thromboembolic disease
- thrombotic abnormalities
- obesity
- pregnancy
- constricted clothing
- homocystinuria
- systemic lupus erythematosus
- inflammatory bowel disease
- central venous catheter use
- oral estrogen use

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increased fibrinolysis that's triggered during thrombogenesis. Using a combination of D-Dimer values and risk stratification by clinical examination can improve diagnosis of DVT.[23,24]

♦ *Genetic blood testing* may help identify inherited thrombotic disorders. Screening yields best results when venous thrombosis occurs in those ages 45 or younger; when a venous thrombus occurs at an unusual location, such as gastrointestinal region, brain, or the arm; and when there's a positive immediate family history of DVT.[25]

Primary VTE prevention

Venous thromboembolism is silent by nature and leads to increased health care costs and morbidity and mortality, particularly when left untreated. Hospitalized patients prone to VTE require primary prevention.

According to the American College of Chest Physicians' *Consensus Guidelines on Antithrombotic Therapy* (2001), nonpharmacologic measures for low-risk populations include leg exercises and early ambulation to lessen venous stasis. Those at moderate or greater risk should use devices that reduce venous stasis and improve fibrinolytic activity, such as elastic compression stockings or intermittent pneumatic compression devices.[26]

Pharmacological prophylaxis

International studies document that compliance with DVT prophylaxis standards need improvement. Current pharmacological guidelines recommend that patients without contraindications receive individualized pharmacological prophylaxis before and after any procedure or event with an increased VTE risk.[27]

Low-dose unfractionated heparin (UFH) or low molecular weight heparin (LMWH), combined with nonpharmacologic primary preventative measures, is indicated for moderate or high-risk groups. Patients receive these medications in lower doses for prophylaxis than for DVT treatment.[28] They're contraindicated with severe active bleeding, hypersensitivity, and heparin-induced thrombocytopenia (HIT). Aspirin and antiplatelet drugs aren't recommended for prophylaxis.

UFH binds to antithrombin III (AT), accelerating the rate at which it inactivates factors IX, X, XI, XII, and thrombin, thus blocking the formation of fibrinogen and fibrin. Less heparin is needed to inhibit the activation of factor X than is needed to prevent the formation of fibrin. Therefore, clinicians use lower doses more effectively in prophylactic therapy than in DVT treatment.[29] Heparin binds to platelets, endothelial cells, and von Willebrand factor, variably affecting bleeding.

Because heparin isn't absorbed from the gastrointestinal tract, clinicians must administer it subcutaneously or intravenously, avoiding intramuscular injection due to the potential for hematoma formation. The I.V. route provides immediate onset of action, while onset with the subcutaneous route is slower, peaking between 2 to 4 hours. The half-life of heparin is between 30 and 180 minutes and increases with higher doses; it's prolonged in liver disease.

Frequent laboratory measurements of activated partial thromboplastin times (aPTT) and dose adjustments are needed to keep the aPTT ratio between 1.5 and 2.5.[30] Heparin use requires frequent patient and laboratory monitoring and dose titration, requiring administration only in a hospital setting.

Avoid heparin use in patients experiencing spinal or epidural anesthesia, spinal cord injury, total hip replacement surgery, and knee replacement surgery, although you may use it as an alternative approach in hip fracture surgery. Note that the usual subcutaneous dose is 5000 units every 12 hours (every 8 hours for extensive gynecologic surgery), beginning one to two hours prior to surgery.[31]

Administer low-dose UFH prophylactically to patients without a greater than usual risk of bleeding and with moderate to high risk of DVT who are undergoing general surgery, gynecological surgery, open urologic surgery, and some neurosurgery procedures.

DVT prophylaxis with low molecular weight heparin

	Agent	Dose	Initiation	Duration
General surgery, moderate risk	enoxaparin tinzaparin dalteparin	20 mg sc daily 3500 IU sc daily 2500 IU sc daily	1 to 2 hours before surgery 2 hours before surgery 1 to 2 hours before surgery	7 days or until fully ambulating or discharged from hospital
General surgery, high risk	enoxaparin tinzaparin dalteparin	40 mg sc daily — 5000 IU sc daily	1 to 2 hours before surgery — 8 to 12 hours before surgery	
Orthopedic surgery Major trauma	enoxaparin tinzaparin dalteparin	30 mg sc q12 hours or 40 mg sc daily 75 IU/KG sc daily or 4500 IU sc q12 hours 5000 IU sc daily or 2500 IU sc daily	12 to 24 hours after surgery or 10 to 12 hours after surgery 12 to 24 hours after surgery or pre-op and daily post-op 8 to 12 hours pre-op and daily post-op (beginning 12 to 24 hours post-op) or 6 to 8 hours after surgery	7 to 10 days or extended beyond hospitalization for high-risk cases
Acute spinal cord injury	enoxaparin	30 mg sc q12 hours	12 to 36 hours if hemodynamically stable	not described
Medical conditions	enoxaparin	30 mg sc q12 hours	—	
	enoxaparin tinzaparin dalteparin	40 mg sc daily — 2500 IU sc daily	upon arrival to facility — upon arrival to facility	6 to 11 days — not described

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Also consider low-dose UFH appropriate for acute myocardial infarction patients, ischemic stroke patients, and other medical patients with risk facters for DVT.[32]

LMWH contains small molecular fragments, about one third the size of UFH. These drugs inhibit thrombin generation owing to reduced factor IIa activity, but with enhanced inhibition of factor Xa and thrombin by binding to and accelerating antithrombin III activity.

LMWHs are less bound to plasma proteins, offering a more predictable dose response, making blood monitoring in most cases unnecessary. Additionally, they're cleared by the kidneys and have a two- to four-fold longer half-life than UFH, making them the preferred agent in many VTE patients. Administer LMWHs by subcutaneous injection.

For DVT prophylaxis, LMWH is the anticoagulant of choice after major orthopedic surgery, including total hip replacement surgery, knee replacement surgery, and hip fracture surgery.[33] It's also preferred in major trauma patients due to a lower risk of bleeding than with UFH. LMWHs may have an incidental effect of inhibiting metastasis and angiogenesis necessary for tumor growth. (See "DVT prophylaxis.")

VTE treatment

UFH or LMWH are the agents of choice for rapid anticoagulation desired for VTE treatment. When using intravenous UFH, measure the aPTT at baseline, every six hours after an I.V. bolus dose, and regularly after hanging the maintenance I.V. drip. Adjust dosing based on the result; keep the plasma heparin level of 0.2 to 0.4 international units(IU)/mL by protamine sulfate assay. Note that weight-based nomograms, developed to help guide dosing, are facility-specific, tailored to individual laboratory reagents.

Generally, patients receive heparin for at least four days, or up to ten days for massive PE. Optimal duration of I.V. heparin therapy is five to seven days to avoid heparin-induced thrombocytopenia and the potential for bone loss that can occur from heparin-induced activation of osteoclasts.[34]

Alternatively, initial subcutaneous heparin dosing begins after a small I.V. bolus of between 3,000 and 5,000 units, followed by 17,500 units given subcutaneously every 12 hours. Adjust the dose to a plasma heparin level of 0.2 IU/mL within an hour of the next scheduled dose, and follow it closely for the first few days, after which a daily level should suffice.[35]

Most studies demonstrate comparable outcomes with LMWH and intravenous heparin for VTE recurrence, effectiveness, and safety. Some studies find that LMWH is superior for reducing VTE recurrence rates, hemorrhagic complications, and mortality. Additionally, studies document that health care costs are lower with LMWH use because there's no need for persistent laboratory testing and because many patients can use this agent at home, saving hospital-associ-

Oral anticoagulent drug interactions

Drugs that *increase* anticoagulant effect of warfarin	Drugs that *decrease* anticoagulant effect of warfarin
acetaminophen	ascorbic acid
androgens	dicloxacillin
beta-blockers	ethanol
chlorpropamide	ethchlorvynol
clofibrate corticosteroids	griseofulvin
cyclophosphamide	nafcillin
dextrothyroxine	sucralfate
disulfiram	trazodone
erythromycin	aminoglutethimide
fluconazole	barbiturates
gemfibrozil	carbamazepine
glucagons	etretinate
hydantoins	glutethimide
influenza virus vaccine	rifampin
isoniazid	cholestyramine
ketoconazole	contraceptives, oral
miconazole	estrogens
moricizine	thiopurines
propoxyphene	spironolactone
quinolones	thiazide diuretics
streptokinase	vitamin K
sulfonamides	
tamoxifen	
thioamines	
thyroid hormones	
urokinase	
amiodarone	
chloramphenicol	
cimetidine	
ifosfamide	
lovastatin	
metronidazole	
omeprazole	
phenylbutazones	
propafenone	
quinidine	
quinine	
sulfonamide-trimethoprim	
sulfinpyrazone	
chloral hydrate	
loop diuretics	
nalidixic acid	
aminoglycosides	
mineral oil tetracyclines	
vitamin E	
cephalosporins	
diflunisal	
NSAIDs	
penicillins	
salicylates	

Source: Hebel, S., Kastrup, E., and Killion, K.: Drug Facts and Comparisons, *56th ed. St. Louis: Wolters Kluwer, 2002.*

ated expenses of therapy, particularly in high-risk patients requiring long-term prophylaxis. Candidates for outpatient LMWH therapy include those with stable proximal DVT or PE, normal vital signs, low risk for bleeding, absence of renal insufficiency, and a practical home system for medication administration, appropriate monitoring, and treatment of recurrent VTE and bleeding complications.[36-40]

Used as an alternative to heparin in patients with heparin-induced thrombocytopenia and in those with severe systemic allergies to heparin, *hirudin* is a direct inhibitor of thrombin, independent of AT activity. *Warfarin sodium* (brand name Coumadin) is an oral anticoagulant used for long-term anticoagulation therapy. It inhibits the synthesis of vitamin K-dependent coagulation factors (II, VII, IX, and X) as well as protein C and protein S. Onset of action is 90 minutes and the half-life is 36 to 42 hours.

It takes several days of warfarin administation to clear coagulation factors from plasma. Factor VII and protein C have short half-lives, clearing rapidly from the plasma, while other coagulation factors take longer to deplete. Warfarin is metabolized by the liver and excreted in the urine. It's contraindicated with severe active bleeding, hypersensitivity, and pregnancy, but not during lactation.

Large loading doses of warfarin have the potential to cause coagulation rather than anticoagulation, so administer moderate ones.[41,42] Note that the usual loading dose is 5 mg started on day 1 after surgery. Use warfarin for long-term anticoagulation, initiating administration concurrently with heparin, and continuing it for 5 to 6 days, until achieving a therapeutic anticoagulation effect. Then administer the usual maintenance dose of 2 to 5 mg daily to maintain international normalized ratio (INR) range between 2.0 and 3.0, with a target of 2.5.

Treat patients for at least three months if there's low risk of recurrence, up to 6 months with higher risk, and for 1 year or longer if long-term risk is identified.[43] Factors influencing blood levels of warfarin include certain medications, vitamin K rich foods, genetic factors, variations in laboratory testing, patient nonadherence to prescribed therapy, and miscommunication with providers regarding dosing. (See "Oral anticoagulant drug interactions.")

Prothrombin time, a commonly used diagnostic assay to assess warfarin effects, may produce varying results due to differences in laboratory testing reagents. Clinicians report more consistent results when employing the INR assay that internationally standardizes reagents for testing. An INR range of between 2.0 and 3.0, with a target of 2.5, is therapeutic for treatment of DVT and PE.[44]

Other DVT treatment strategies

Use *thrombolytics* in hemodynamically unstable patients with massive PE who aren't prone to bleeding. These agents activate plasmin in the fibrinolytic system, which degrades any thrombus in its presence. Because use of these drugs is highly individualized, screen patients for risk of hemorrhage: There's a 1% to 2% risk of intracranial bleeding from the drugs.[45]

You may also consider using *vena cava filters* to reduce the PE rate in patients with high risk of VTE who have contraindications to or complications of anticoagulation. Vena cave filters have been used for patients with extensive trauma, visceral cancers, those undergoing hip or knee surgery, and those with recent DVT. Contraindications include pregnancy, venous anatomic abnormalities, and thrombosis location proximal to standard device point of placement. There's a tendency toward more recurrent DVT in patients who've received a filter.[46]

Pulmonary embolectomy is performed only in emergent situations with hemodynamically unstable patients when angiographically documented PE exists, and when conservative measures have failed to stabilize them. Mortality rates vary from 10% to 75%.[47]

Complications

Anticoagulation therapy often causes hemorrhagic complications. Intravenous heparin poses a bleeding risk of less than 3%, but this increases with higher doses.[48,49] *Protamine sulfate* is a heparin antagonist: Each mg neutralizes approximately 100 units of heparin. Calculations include only the last several hours of heparin infusion. Administer protamine over 3 minutes, not exceeding 50 mg over 10 minutes to avoid side effects like bradycardia and hypotension. The drug has a quick onset; its effects last for 1 to 2 hours.

Because anaphylaxis is associated with previous exposure to the drug, patients may require steroids prior to protamine injection. Vitamin K (phytonadione) is the warfarin antagonist, given for warfarin overdose. It promotes hepatic synthesis of clotting factors, reducing prothrombin depression. Use the smallest effective dose—2.5 mg to 10 mg orally, repeated 12 to 24 hours as needed, or 0.5 mg to 10 mg subcutaneously or intramuscularly, repeated in 6 to 8 hours as needed. Administer vitamin K intravenously in an emergency only, and at a rate of 1 mg/min, up to a 10-mg maximum.[50]

Post-thrombotic syndrome may occur in 29% to 79% of patients. After DVT, ambulatory venous hypertension, venous valve incompetence, and recurrent DVT can lead to chronic pain, edema, hyperpigmentation, and ulceration. Exercise may help these patients by improving their calf flexibility.[51]

Because HIT is an antibody mediated adverse reaction of heparin, measure platelet counts between day 3 and day 5 of therapy, between day 5 and day 7, and again on day 14. When the platelet count drops rapidly in a sustained way by half or to under 100,000, discontinue heparin immediately. Place a vena cava filter immediately when HIT is present to prevent PE from paradoxical thrombosis development. Heparin resistance occurs in approximately a quarter of DVT patients. This occurs when a patient requires more than 35,000 units/24 hours of heparin to achieve minimal therapeutic anticoagulant effect.[52]

Nursing implications

Nurses can employ numerous measures to prevent VTE in patients, including patient education regarding leg exercises, early ambulation, avoidance of prolonged inactivity and constricted clothing, hydration maintenance, and use of *graduated compression stockings*, which patients can wear before a surgical procedure and after, until fully ambulatory. This will improve venous flow and reduce swelling.

Other devices appropriate for patients not experiencing an acute DVT episode, large open wounds, skin grafts, or cancer of the extremity, include:

- *intermittent pneumatic compression devices*, which compress the lower extremities, thus increasing venous flow. Patients wear a fitted sleeve around the extremity that contains chambers that perform a compression/relaxation pumping action. Successful usage hinges on proper sizing and application.
- *venous plexus foot pump*, which mimics the action of walking by intermittently compressing and relaxing the sole of the foot, thus causing muscle contraction and improving venous flow. This device demonstrates great success in orthopedic patients.

Staff members must also serially monitor patients for bleeding and laboratory results. When discussing patient education, nurses should emphasize the importance of notifying care providers in the event of nose or gum bleeding or hematuria. Patients should also follow up with all ordered laboratory tests.

Provide patients with a list of medications and foods that can alter INR. Natural dietary intake of vitamin K should remain consistent, so that warfarin dose response ratio remains stable. Clinicians should remain aware of which medications adversely influence the dose-response ratio for warfarin. To avoid drug interactions, patients taking warfarin should notify their prescriber before starting any other medication.

VTE is a common and potentially devastating disease. Prevention is the best medicine. Nurses are well prepared to help patients achieve optimal outcomes with preventative measures and early treatment. NM

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Get the edge on deep vein thrombosis

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Get the edge on deep vein thrombosis

GENERAL PURPOSE: To provide registered professional nurses with an overview of the prevention, identification, and treatment of deep vein thrombosis.

LEARNING OBJECTIVES: After reading the preceding article and taking this test, you should be able to: **1.** Identify the clinical manifestations and interventions related to deep vein thrombosis and venous thromboembolism. **2.** Discuss various diagnostic and laboratory tests used in the identification and treatment of deep vein thrombosis and venous thromboembolism. **3.** Describe the purpose and adverse effects of the medications prescribed for deep vein thrombosis and venous thromboembolism.

1. Which of the following patients has the greatest risk of developing a deep vein thrombosis (DVT)?
a. a 15-year-old post appendectomy
b. a 20-year-old post pulmonary wedge resection
c. a 50-year-old post cholecystectomy
d. a 60-year-old post hip replacement

2. Which of the following is a classic assessment finding in a patient who has a DVT in the leg?
a. tachycardia
b. absent pedal pulse
c. coolness of the extremity
d. pain in the extremity

3. Which of the following is the preferred diagnostic procedure to identify a DVT?
a. I-fibrinogen scan
b. magnetic resonance direct thrombus imaging
c. D-Dimer assay
d. venous duplex ultrasonography

4. Which of the following diagnostic test can be used to detect filling defects in a deep vein?
a. venous duplex ultrasonography
b. venography with contrast media
c. magnetic resonance direct thrombus imaging
d. D-Dimer assay

5. To prevent a venous thromboembolism (VTE) in a low-risk patient, the nurse should expect to use which of the following preventive measures?
a. encourage ambulation
b. apply elastic compression stockings
c. use an intermittent pneumatic compression device
d. administer heparin

6. In which of the following patients would the half-life of heparin be prolonged?
a. a 55-year-old post total knee replacement
b. a 60-year-old with a history of cirrhosis
c. a 65-year-old with multiple sclerosis
d. a 70-year-old with a history of MI

7. In a patient receiving UFH, the activated partial thromboplastin time ratio should be maintained at
a. 0.5 to 1.5. c. 2.5 to 3.5.
b. 1.5 to 2.5. d. 3.5 to 4.5.

8. Heparin would be recommended for a patient who has had a
a. pulmonary lobectomy.
b. total hip replacement.
c. total knee replacement.
d. spinal cord injury.

9. Low molecular weight heparin (LMWH) must be administered via which route?
a. by mouth
b. subcutaneous injection
c. intramuscular injection
d. intravenous infusion

10. A patient receiving warfarin therapy should be advised not to increase consumption of foods that are rich in
a. vitamin A. c. vitamin C.
b. vitamin B. d. vitamin K.

11. For which of the following patients is it safe to administer warfarin?
a. A patient who has a bleeding gastric ulcer.
b. A hemophiliac patient who has hemarthrosis.
c. A woman who's pregnant.
d. A woman who's lactating.

12. One advantage of using LMWH for a VTE recurrence is that
a. daily blood work can be done in the home.
b. it's administered orally.
c. it may not require hospitalization.
d. treatment lasts for 3 days.

13. A patient may require a higher dosage of warfarin if she's also receiving
a. nafcillin. c. metronidazole.
b. ketoconazole. d. tamoxifen.

14. Which of the following laboratory tests is used to assess the therapeutic effect of warfarin in a patient who has a VTE?
a. platelet count
b. activated partial thromboplastin time
c. international normalized ratio
d. prothrombin time

15. Which of the following international normalized ratio values indicates a therapeutic maintenance level of warfarin?
a. 1.0 to 2.0 c. 3.0 to 4.0
b. 2.0 to 3.0 d. 4.0 to 5.0

16. Which of the following medications is used as a heparin antagonist?
a. phytonadione
b. vitamin K
c. protamine sulfate
d. AquaMEPHYTON

17. Administration of protamine sulfate in less than 3 minutes may lead to which of the following side effects?
a. bradycardia
b. hypertension
c. tachypnea
d. elevated temperature

18. Anaphylaxis to protamine sulfate is often associated with
a. an increased heparin dosage.
b. a decreased white blood count.
c. previous exposure to the drug.
d. a prolonged prothrombin time.

19. Post-thrombotic syndrome may be helped by
a. administering steroids.
b. stopping heparin administration.
c. limiting foods high in vitamin K.
d. encouraging exercise.

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2.	❍	❍	❍	❍	6.	❍	❍	❍	❍	10.	❍	❍	❍	❍	14.	❍	❍	❍	❍	18.	❍	❍	❍	❍
3.	❍	❍	❍	❍	7.	❍	❍	❍	❍	11.	❍	❍	❍	❍	15.	❍	❍	❍	❍	19.	❍	❍	❍	❍
4.	❍	❍	❍	❍	8.	❍	❍	❍	❍	12.	❍	❍	❍	❍	16.	❍	❍	❍	❍					

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