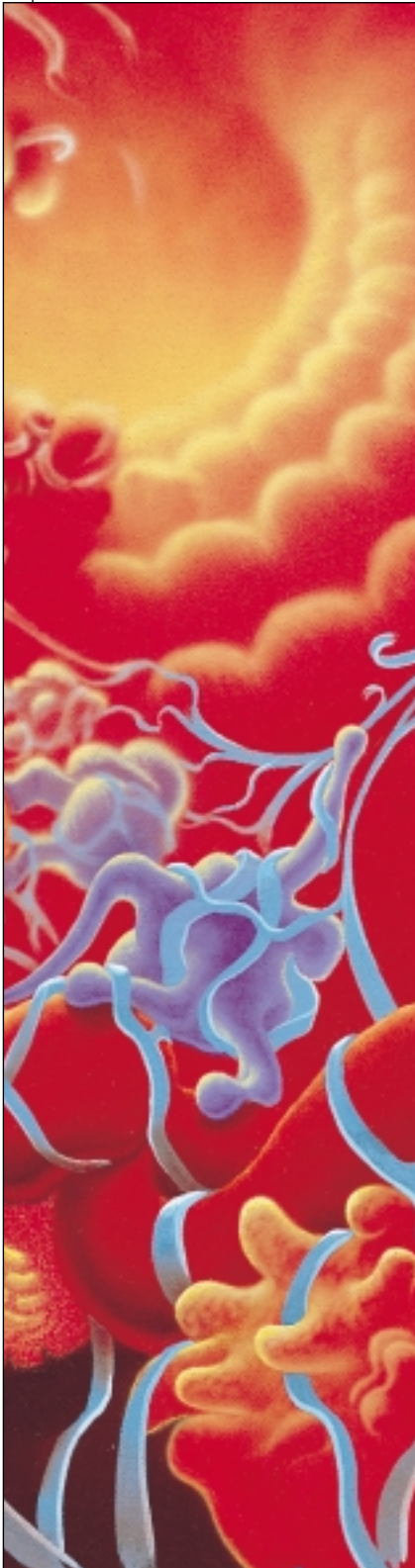


PAULA BREEN, RN, CRRN



Deep vein thrombosis often has no signs or symptoms, yet it can become life-threatening in seconds. That's why it's important to know how to prevent it—from drug therapy or stents to simple measures like repositioning and compression devices—and how to help patients recover when it does develop.

Deep vein thrombosis (DVT) is a common cause of morbidity and mortality.¹ It can develop in all sorts of patients—trauma, surgical, and cardiac, for example—and in healthy people, too, for no obvious reason.

We've all been taught to recognize unilateral leg pain, swelling, and tenderness as classic signs of DVT, but a large percentage of cases—perhaps as high as 80%²—don't produce these or any other clues. That's what makes DVT so perilous. There may be no indication that there's a problem until part of the clot dislodges and makes its way to the lungs, where it can become a fatal pulmonary embolism (PE).

Even if a PE never develops, research shows that patients treated for DVT in acute care hospitals may have a mortality rate as high as 30% three years after discharge.³ It's therefore important to know who's at risk for DVT and what pharmacologic and nursing inter-

ventions will decrease the chance of it developing.

These circumstances cause clots to form

Three factors—collectively known as Virchow's triad—are responsible for DVT: vessel wall damage, venous stasis, and hypercoagulability.

Vessel wall damage—which is most likely to occur in the pelvis

KEY WORDS

- ▶ deep vein thrombosis
- ▶ pulmonary embolism
- ▶ Virchow's triad
- ▶ anticoagulation therapy
- ▶ unfractionated heparin
- ▶ low molecular weight heparin
- ▶ warfarin (Coumadin)
- ▶ venography
- ▶ Doppler compression ultrasonography
- ▶ D-dimer assay
- ▶ activated partial thromboplastin time (APTT)
- ▶ international normalized ratio (INR)

PAULA BREEN is a nursing practice leader in the neurotrauma unit at Spaulding Rehabilitation Hospital in Boston.

STAFF EDITOR: Amy Slugg Moore

and legs—initiates the clotting mechanism. The vessel's lining of endothelial cells inhibits platelet adhesion and aggregation. An injury that breaks this surface exposes platelets in the bloodstream to collagen in the subendothelium. This, in turn, triggers the release of a substance that causes the platelets to collect at the injured site.

Venous stasis occurs when blood return from the legs is impaired. The blood pools in the legs, allowing clotting factors to reach levels high enough to initiate clotting. Like vessel wall damage, venous stasis occurs mainly in the lower extremities; however, it can develop in any immobilized limb.

Several conditions predispose a patient to hypercoagulability—the increased tendency for blood to clot. These and other risk factors for DVT are listed in the box on the facing page.

Proactive intervention: Pharmacologic options

Nurses can help prevent DVT by ensuring that moderate- and high-risk patients are started on anticoagulation therapy. That includes people age 40 and older who are having major surgery; those with a fracture of the hip, pelvis, or lower extremity; those with a history of DVT or PE; those who've undergone lower limb amputation; those whose legs are paralyzed; and those confined to bed for three or more days for any reason.⁴ Cancer patients who've undergone major abdominal or pelvic surgery are also at high risk.⁴

Preventive anticoagulation is accomplished with heparin, which works by binding with antithrom-

bin III (AT III) to form a complex that inactivates thrombin and several other clotting agents. There are two types: unfractionated and low molecular weight heparin (LMWH). When either type is used for DVT prophylaxis, it's given subcutaneously—unfractionated heparin, twice a day; LMWH, once or twice a day.

There are three LMWH compounds approved by the FDA for DVT prevention: enoxaparin (Lovenox), dalteparin (Fragmin), and ardeparin (Normiflo). Each one is approved for specific indications: enoxaparin after hip or knee replacement or abdominal surgery, dalteparin after hip replacement or abdominal surgery, and ardeparin after knee replacement.⁵

When anticoagulants can't be used—the patient is at high risk for hemorrhage, for example—a filter may be placed in the inferior vena cava. The filter will catch clots traveling from the legs before they enter the lungs.

Nursing actions can also help

There are other steps nurses can take to prevent DVT in patients at risk. To reduce venous stasis, frequently reposition the patient and raise his legs above the level of his heart. Tell him to regularly move and stretch his legs, particularly the calf muscles. And teach him how to perform deep breathing exercises, which will help the large veins—in the legs and elsewhere in the body—empty by increasing negative pressure in the thorax.

Elastic stockings or intermittent pneumatic compression devices may also be helpful. The stockings compress the superfi-

cial veins, which enhances venous return and circulation to the deep veins. Be sure they fit properly; if they roll at the top, they can act as a tourniquet. Briefly remove them twice a day, and inspect the skin for irritation and breakdown.

Intermittent pneumatic compression devices work by repeatedly squeezing the legs, which helps empty the leg veins. Because patients with altered levels of consciousness or confusion may try to remove them, or the device may not have been applied correctly in the first place, check frequently to be sure that they're on properly. Also check the compression system to ensure it's connected and set to deliver the correct amount of pressure.

A diagnosis is made with a sharp eye and tests

Because DVT can develop in any patient—not just those with known risk factors—it's important to know how to spot the problem. The most common physical finding is sudden swelling in just one extremity. Patients may complain of a dull ache in the calf that intensifies during walking, or they may say their leg feels heavy or tight.

You should also suspect DVT if a limb—especially the calf—becomes painful, warm, and reddish. It may also feel firm.

Don't rely on Homans' sign—pain in the calf after rapid dorsiflexion of the foot. Although it's suggestive of DVT, it's absent in nearly half of all known cases. And, Homans' sign doesn't always mean DVT; it may also be present in other conditions that cause lower extremity discomfort.

Risk factors for DVT

Vascular wall injury	Venous stasis	Hypercoagulability
Surgery	Immobilization for more than three days	Pregnancy
Trauma	Obstruction or compression of the iliac or femoral veins from large abdominal or pelvic tumors, obesity, pregnancy	Estrogen therapy
Indwelling IV catheters	Lengthy surgery (more than 30 minutes)	Malignancy
Injection of irritating substances	Congestive heart failure	Polycythemia rubra vera
IV drug abuse	Shock	Sickle cell disease
Prior DVT	Varicose veins	Inherited coagulopathies
		Antithrombin III deficiency
		Dehydration

A clinical suspicion of DVT must be confirmed by venography, Doppler compression ultrasonography (also called duplex scanning), or impedance plethysmography. A blood test for D-dimer—a product of fibrin degradation—may also be ordered. When a thrombus is present, plasma D-dimer concentrations are usually greater than 1,591 ng/ml.⁶

If the patient develops a clot

Once DVT is diagnosed, anticoagulant therapy is usually initiated or adjusted to prevent new clots from forming and keep the present one from growing. Traditionally, patients with DVT were given unfractionated heparin—an IV bolus, then a continuous infusion—followed by warfarin (Coumadin).

Now there's another option. The FDA has approved enoxaparin for inpatient treatment of acute DVT, with or without PE, when used in conjunction with warfarin. (It can be used to treat DVT without PE on an outpatient basis, too; this article covers inpatient treatment only.) It's given

subcutaneously, in amounts higher than the preventive dose, either once or twice a day.

Enoxaparin may be easier and safer to use than unfractionated heparin, which binds not only with AT III but with plasma proteins, macrophages, and endothelial cells resulting in inconsistent bioavailability and unpredictable anticoagulant effects. With unfractionated heparin, the infusion must therefore be titrated to ensure that the activated partial thromboplastin time (APTT) reaches and remains at a therapeutic level—usually at least 1.5 times the control, although this value can vary depending on the PTT method being used. All this translates to an inpatient hospital stay with serial blood drawing, frequent monitoring of lab results to titrate the heparin dose, and increased patient discomfort.

Enoxaparin, on the other hand, binds with few plasma proteins, so there's a more predictable dose response. The drug doesn't affect PTT, so there's no need to monitor this lab value. It also doesn't significantly change fibrinogen levels or significantly inhibit platelet aggregation at the site of

vascular injury, so it may cause less bleeding.¹ And it doesn't require IV infusion. (For a more detailed discussion of how enoxaparin works, see "Low molecular weight heparin" in the April 1998 issue of *RN*.)

No matter which type of heparin is used, you'll start the patient on warfarin (Coumadin) within 72 hours of heparin therapy. Continue the heparin until the patient's international normalized ratio (INR) is between 2.0 and 3.0, which usually occurs within five days.

If the risk is high that the clot will migrate, a filter may be used, too. Some centers may use thrombolytic agents, such as streptokinase (Kabikinase), to dissolve pulmonary emboli.

The thrombus may need to be surgically removed when anticoagulation and thrombolytic therapy are contraindicated, when there is an extremely high risk of fatal PE, or if venous drainage is so severely compromised that irreversible damage in the affected leg could result.

Getting the patient back on his feet

How long a patient with DVT is kept in bed depends on the physician. Some want their patients up and moving around as soon as possible to reduce venous stasis. At our hospital, patients stay in bed for five to seven days. This allows the thrombus to adhere to the vessel wall, reducing the risk of embolization.

No matter how long your patient is on bed rest, there are things you can do to make him more comfortable. Frequently apply warm compresses to the affected area. Elevate the limb as

much as possible to promote venous return and reduce edema. Also reposition the patient periodically. Do all this with extreme care so you don't dislodge the clot.

If the DVT is in a leg, apply elastic stockings, which also will help facilitate venous return. Whether you do this right away or after the traditional five to seven day waiting period will depend on your facility's protocol.

When the patient is permitted to move about, encourage him to walk, rather than sit or stand for extended periods. When in bed, the patient should perform calf exercises, such as dorsiflexion.

Be alert for signs of PE—sudden shortness of breath, pleuritic chest pain, increased respiratory rate, and hemoptysis. Don't use oxygen saturation as the sole numerical indicator of the patient's respiratory status; in two out of three PE patients, oxygen saturation will be greater than 90%.⁷

ABGs are a far more reliable indicator of your patient's status; in most cases of PE, they'll reveal mild to moderate hypoxemia with a low PaCO₂.

As you would with any patient on anticoagulant therapy, watch for signs of bleeding. Check stools for evident and occult blood, and check IV sites for oozing. Avoid IM injections. When preparing the patient for discharge, tell him to be alert for post-thrombotic syndrome, a complication of DVT. Also known as postphlebotic syndrome, it occurs when valves in the deep veins of the legs are damaged by a thrombus, resulting in chronic venous stasis.

Signs and symptoms include chronic edema, pain, altered pigmentation, and induration. Early recognition and prompt treatment are important, as difficult-to-heal skin ulcers can eventually develop.

What patients need to know about warfarin

Because patients on warfarin therapy are at risk for bleeding, they'll need to take precautions to protect themselves. Here's what to teach them:

- ▶ Have your blood drawn regularly, as instructed, so that the lab can measure the time it takes for your blood to clot. Doing so will help ensure that you have the right amount of the anticoagulant, warfarin, in your blood. Be sure to keep all lab appointments.
- ▶ Take the medication at the same time each day to keep the level in your blood consistent.
- ▶ Don't take any other medication or stop taking warfarin without speaking with your physician or nurse first.
- ▶ Don't drink alcoholic beverages or take aspirin; they can increase warfarin's effects.
- ▶ Before receiving treatment from another healthcare provider, such as a dentist, tell him that you're taking an anticoagulant, or blood thinner.
- ▶ Because vitamin K affects how your body reacts to warfarin, don't significantly increase or decrease the amount of vitamin K-rich foods that you eat. Examples of food products rich in vitamin K are broccoli, cabbage, lettuce, asparagus, spinach, turnip greens, and green tea.
- ▶ Be sure to report any faintness, dizziness, weakness, severe headache or stomach pain, excessive menstrual bleeding, red or black bowel movements, abnormal bruising, or other unusual bleeding.
- ▶ If long-term warfarin therapy is necessary, order and wear a medical alert ID bracelet or necklace that indicates you're taking an anticoagulant.

A patient who is started on warfarin in the hospital will most likely continue taking it at home. The length of therapy will depend on the patient's underlying illness, past history, and current risk factors—he may need to take it for weeks, years, or the rest of his life. Discharge instructions for patients taking warfarin are covered in the box at left.

Preventing DVT can spare patients a lot of pain and a prolonged hospitalization. Just as important, however, is knowing how to spot the problem and effectively care for the patient who develops it—which may just help save his life. □

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