DEEP VENOUS THROMBOSIS AND PULMONARY EMBOLISM

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INTRODUCTION

In 1845, Virchow began lecturing on the pathogenesis of venous thrombosis and embolism, which was a major step in the understanding of this disease process. Over the next few years, he continued his research, and described three predisposing factors which lead to the development of venous thrombosis, which is now termed "Virchow's Triad." This now serves as the basis of our understanding of thromboembolic disorders.

Deep vein thrombosis (DVT) is a well-recognized problem in hospitals today. The events leading to DVT are not as well understood as those leading to thromboses in the arterial vasculature. Damage to a vessel wall may be the underlying etiology, although there are usually no recognizable lesions on the endothelium. Submicroscopic endothelial injury has been hypothesized, possibly as a result of local tissue response to the stasis and turbulence found in the venous vasculature.

Although DVT has been well studied throughout the literature, an accurate assessment of the incidence and prevalence is hard to ascertain. However, recent literature gives the following gross estimates:

- 2,500,000 cases of deep venous thrombosis (DVT) per year
- 600,000 episodes of pulmonary embolism (PE) per year
- 150,000 deaths per year contributed to by PE
- 50,000 deaths per year caused by PE
- 11,000 postoperative PE deaths per year
- 30-40% of postoperative patients will develop DVT
- 15% deaths in acute care hospitals are due to PE

These figures are gross estimates only, since autopsies are not routinely performed on every death. The inaccuracy in diagnosis is probably due to an underestimation of the magnitude of the problem. In addition, a large number of patients with this disease are overlooked due to an absence of symptoms.

The population of patients with the greatest risk of developing DVT are those following surgery. The postoperative patient, in particular, is predisposed to developing DVT due to events which occur during and after surgery. Furthermore, many podiatric surgical patients have additional risk factors which include age, weight, social habits, or genetics.

Virchow's triad consists of stasis of blood flow, intimal vessel damage, and hypercoagulability. The postoperative patient, due to immobilization during and after surgery, fulfills the first requirement of this triad, stasis. Intimal damage, the second factor, occurs during surgery (or sepsis) due to the disruption of normal anatomy. And finally, hypercoagulability can result from the decreased blood volume associated with surgery. Hypercoagulability can also occur secondary to cancer, dehydration, or malnutrition. These three situations, which make up Virchow's triad, increase the risk for developing DVT.

DVT RISK FACTORS

The risk factors for DVT have been broken down into high, moderate, and low risk categories by various authors.

High risk patients include anyone over the age of 40, who also possesses secondary risk factors. Secondary risk factors include CHF, obesity, malignancy, varicosities, estrogen use, malnutrition (deficiency of Anti-thrombin III, protein C and F) and paralysis. Pelvic and abdominal surgical patients are also included here. These factors, coupled with a history of DVT, constitute the high risk category of patients.

The moderate risk category includes any patient in the high risk classification without a known history of DVT.

Low risk surgical patients include those undergoing surgery of less than 30 minutes in length, and without secondary risk factors or a positive history.

PREVENTION OF DVT

Venous thrombosis with subsequent pulmonary embolism (venous thromboembolic disease) is considered one of the most common cardiopulmonary illnesses in North America and Europe. Although it is much easier and less expensive to prevent DVT than it is to diagnose and treat it, DVT prophylaxis continues to be under-utilized. This is a disturbing fact, as effective prophylaxis is readily available through both pharmacological and nonpharmacological methods.

It has been shown that the production of thrombi depends on the presence of activated clotting factors in areas of stasis. Thrombus formation does not occur as a result of activation of these factors or stasis alone, but through a combination of the two. Due to this fact, addressing either the hypercoagulable state (pharmacological methods) or stasis (non-pharmacological methods) has proven successful in DVT prevention.

PHARMACOLOGIC METHODS OF DVT PROPHYLAXIS

There are several pharmacological methods available for effective DVT prophylaxis. These include heparin, low molecular weight (LMW) heparin, dextrans, and coumadin.

Heparin

Low-dose (or mini-dose) heparin is properly utilized for DVT prophylaxis by administering 5000 U subcutaneously 2 hrs. preoperatively, followed by the same dose every 12 hrs. postoperatively. Due to the short half-life of this drug, the aPTT (activated partial thromboplastin time) will not be noticeably affected at this dose.

Heparin, which acts to inhibit the intrinsic coagulation cascade, must be administered preoperatively to be effective. Likewise, administration of heparin postoperatively has no effect on dissolving already formed clots. Patients on low dose subcutaneous heparin can potentially have a higher incidence of wound hemorrhage and hematoma formation, although this has not been shown to be statistically significant. This form of prophylaxis is suitable for orthopedic surgery, where a tourniquet is used for hemostasis. However, this is not acceptable in opthalmic, spinal or neurosurgery, where even minor hemorrhaging is deleterious. Although effective in DVT prophylaxis, the presence of its potential side effects has propagated further research with newer products.

Low Molecular Weight (LMW) Heparin

Low molecular weight heparin has three major potential advantages over unfractionated heparin therapy: lower frequency of bleeding, effective prophylaxis with once-a-day dosing, and greater efficiency.

LMW heparin is formed by the depolymerization of standard heparin, through a variety of procedures. The end result is an isolated fraction with a molecular weight of around 5000 Daltons. With reference to the clotting cascade, LMW Heparins possess diminished activity against Factor Xa and IIa, both of which are thought to be associated with a lower bleeding potential. In addition, standardized heparin has a half-life of 1 to 1.5 hrs., while LMW Heparin has a half-life of 3 to 4 hrs. A more convenient once-daily administration of LMW Heparin would be advantageous. In a double-blind study by Kakkar, comparing standard unfractionated versus LMW heparin for DVT prophylaxis in 295 patients, patients receiving standard heparin were three times as likely to develop DVT.

Dextran

Dextrans impair platelet function, thereby causing a decrease in platelet aggregation. Dextrans are available in two molecular weights, 40 and 70. The use of dextran, however, is not completely benign. Anaphylactoid reactions have been reported in 0.1-0.25% of patients. Volume overload and subsequent heart failure is a concern in patients with a marginal cardiac reserve. Also, caution should be exercised in patients with renal insufficiency, since further decrease in renal function may result from excessive diuresis.

Coumadin

Coumadin (warfarin) is a low molecular weight anti-coagulant which inhibits the effects of Vitamin K in the synthesis of clotting factors II, VII, IX, and X. Coumadin affects the extrinsic clotting cascade and will prolong the PT (prothrombin time). Typically, prophylaxis starts two to three days preoperatively with a dose of 2.5 mg/day. The PT is monitored daily until a therapeutic level is reached, which is 1.3-1.5 times the control value.

In contrast to Heparin, the risk of bleeding and hematoma formation is well documented. Physicians should also be aware of the rare but potential complication of "coumadin skin necrosis," which occurs in less than 0.25% of cases. Skin necrosis, which is localized to areas of increase subcutaneous tissue, has been observed in mild to moderately obese females who began therapy with loading doses greater than 20 mgs.

MECHANICAL METHODS OF DVT PROPHYLAXIS

Mechanical devices for DVT prophylaxis encourage muscular activity within the leg and thigh, in an attempt to maintain venous flow in immobilized patients. The muscular activity of the calf, during normal ambulation, helps to pump blood back to the heart. In immobilized patients, these devices simulate the body's own "calf pump." The action of mechanical devices on this pump is analogous to the actions of dobutamine on the left ventricle.

Graduated Compression Stockings

Studies have shown that graduated compression stockings can reduce the frequency of venous thrombosis by greater than one half, when compared with no treatment. Compression is greatest at the ankle, and gradually decreases proximally up to the thigh. These stockings should be considered first line prophylaxis against DVT/PE in hospitalized patients. Caution is advised when using these stockings in patients with peripheral vascular disease, as the condition may be aggravated by vascular compression.

Pneumatic Compression Stockings

Pneumatic compression stockings have proven to be an even more effective mechanical method for DVT/PE prophylaxis than other devices. (Figure 1) The physiologic reason for their success is twofold. Pneumatic stockings increase the velocity of venous flow at the time of compression, and increase fibrinolytic activity, both at the site of compression as well as systemically. Numerous authors have reported a significant decrease in thrombosis utilizing these devices prophylactically.

Pneumatic stockings can be either single or multi-chamber by design. Single chamber stockings are usually inflated from 35 to 40 mm Hg, while multiple chambered devices produce pressures of 35, 30, and 20 mm Hg at the ankle, calf, and thigh, respectively. Although both have proven effective, the intermittent sequential compression devices appear to be superior. Kamm and Shapiro demonstrated that when using the single chamber devices, veins in the popliteal region were occluded before the calf veins were

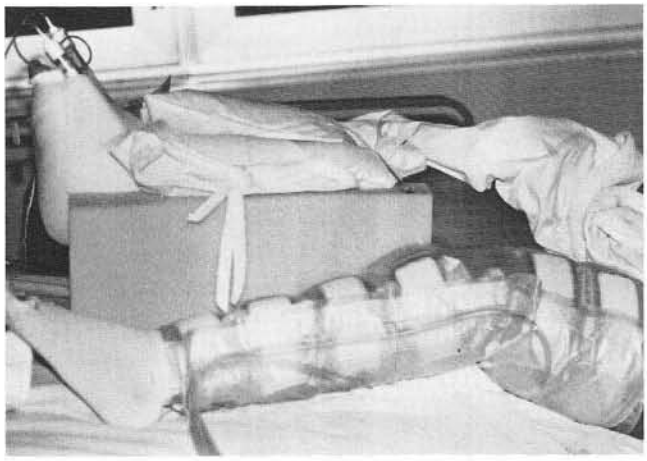

Figure 1. Example of multi-chamber pneumatic intermittent compression stockings as used perioperatively. These stockings are started preoperatively and continued until the patient is out of bed. Note the Ted hose gradient stocking also used with this device.

adequately emptied. This caused a decrease in the amount of blood which propelled forward, when compared with the flow produced by the milking action of the sequential devices. In comparison, the peak velocity flow at the thigh, during the time of compression, was measured to be 50% greater using the sequential device.

The use of intermittent stockings also increases the level of fibrinolysis within the circulation. With vessel stimulation, the local release of plasminogen activator occurs. This will act locally and systemically to convert plasminogen to plasmin and counteract the hypercoagulable state. Tarnay also demonstrated that the greater the volume of tissue compressed, the greater the fibrinolysis effect. Thigh-length boots showed increased results compared to calf-length boots.

DIAGNOSIS OF DVT

The diagnosis of deep vein thrombosis is essential so that timely treatment can be initiated, thus preventing the sequelae of this disease. Unfortunately, the diagnosis is often difficult to establish because the clinical symptoms and signs of DVT cannot be relied upon solely. Many studies have attempted to correlate clinical signs and symptoms of pain, swelling, tenderness, temperature changes, Homan's sign, and Lowenberg's test, to the confirmation of DVT through diagnostic modalities. Even in the group in which the diagnosis was most highly suspected, and might be considered clinically certain, they found the accuracy of physical examination to be only 55%.

Due to the absence of accurate clinical signs which correlate highly with the diagnosis, physicians rely heavily on diagnostic testing to establish a definitive diagnosis. These tests can be divided into two types: hemodynamic testing and anatomic testing. Hemodynamic tests measure specific flow parameters of the venous system, and use this information to diagnose DVT.

Plethysmography and Doppler studies are examples of tests which assess venous hemodynamics. A false positive study can potentially occur when any mass surrounding the venous architecture causes a partial or complete obstruction. Reduced arterial inflow (PVD) causes decreased outflow and may also yield false positive results. Finally, patient positioning may also hinder normal venous return within the extremity being tested.

Anatomic studies allow one to visualize the venous system directly, and are slightly more sensitive and specific to diagnosing DVT. Small asymptomatic thrombi, with equal potential for propagation and embolization, can only be detected with anatomic exams. Real-Time B-mode ultrasonography, Duplex Scanning, and Contrast Venography are examples of anatomic studies.

Hemodynamic Tests

Plethysmography

Plethysmography was the first non-invasive test developed for the diagnosis of DVT. A thigh tourniquet is utilized to effect hemodynamic changes within the venous volume, as blood pools and empties out of the deep leg veins. These tests are sensitive to volume changes within the calf veins, caused by outflow obstructions in the popliteal, femoral, or iliac veins. Measurement of calf circumference can be performed with a strain gauge (SGP) or impedance electrodes (IPG). The sensitivity and specificity of plethysmographic techniques fall within the 95% range.

Doppler Ultrasound

Doppler studies obtain a blood flow velocity waveform when placed directly over the vein being studied. There are five characteristic properties of a vein which can be studied with a doppler: spontaneity, phasicity, augmentation, compression, and pulsatility. In DVT, the normal properties of phasicity, with respiration and augmentation of the venous signal, may be absent, indicating absence of blood flow in the obstructed venous segment. Although the exam is relatively easy to perform at bedside, the evaluation is rather subjective and requires technical experience for accuracy. In competent hands, reports have suggested that this technique may exceed 95% sensitivity in detecting deep vein thrombosis, at or above the level of the knee, and major calf veins.

Anatomic Tests

Real-time Ultrasonography

This study utilizes a transducer to obtain an anatomic image of the venous lumen, in both the longitudinal and transverse planes. The ultrasound echo of this technique is sensitive to any

thrombus present within the venous circulation. Non-compressibility of the vein's lumen by the transducer and non-visualization of blood flow are characteristic of DVT. The ability to image structures outside of the vein's lumen provides information that cannot be obtained by even contrast venography. Masses and other structures causing obstruction to the venous circulation can be visualized when ultrasound examination is normal.

Duplex Scanning

Duplex scanning combines the anatomic imaging capability of real-time ultrasonography and the hemodynamic capabilities of doppler ultrasound, for a complete evaluation of venous thrombosis. This technique was first described for the diagnosis of DVT in 1982. The following parameters need to be documented for a normal venous exam: compressibility, anechoic lumen (absence of thrombogenic echo), blood flow with normal valve motion, and normal doppler signals. In recent years, the addition of color flow and lower frequency probes have improved this technique in previously "hard to detect" areas in the lower extremity (isolated calf and iliac thrombi). Pidala et al. reported a specificity and sensitivity of 93-100%, and this modality is quickly becoming the "gold standard" for non-invasive testing. Experienced technicians are able to quickly approach 100% accuracy.

Contrast Venography

Contrast venography is still the "gold standard" in the diagnosis of DVT, although Duplex Scanning is running a close second. The major disadvantage of venography is the invasive nature of the procedure. Reported complications of venography include chemical phlebitis, infection, hypersensitivity, and local skin necrosis due to extravasation of the contrast dye. In addition, finding a suitable vein in the foot is not always possible. Due to these potential problems, newer, non-invasive techniques have undergone significant development within the past three decades.

TREATMENT

Before the treatment options for deep venous thrombosis and pulmonary embolism are discussed, one must first define the terms of treatment. Whether or not DVT should be treated at all, and at what anatomic level (DVT above or below the knee) should treatment begin, has been widely debated in the past.

Before the introduction of Heparin, the death rate among patients with DVT was 10%, primarily due to pulmonary embolism. Patients with untreated pulmonary embolisms had a 25% mortality and a 50% recurrence rate. With **adequate** Heparin anticoagulation, the risk of thrombophlebitis extension or pulmonary embolism decreases to less than 5%. Therefore, it is standard therapy to treat DVT with anticoagulation. The question arises as to whether or not one should treat DVT above or below the knee, due to the decreased incidence of embolization that is seen in patients with DVT below the knee.

Proximal DVT is defined as that which occurs at the popliteal vein level, and can extend as far as the inferior vena cava (IVC). Distal DVT is defined as that which occurs in the tibial veins, or the upper level of thrombus extends to the popliteal vein. Proximal involvement is seen in 80% of patients with DVT without any evidence of pulmonary embolism. Of the patients with a silent asymptomatic pulmonary embolism, 95% of these patients were discovered to have a proximal DVT. In patients with an obvious pulmonary embolism, 68% were discovered to have a proximal DVT. Distal DVT rarely causes a pulmonary embolism (incidence = 0.8%).

In general, all patients with proximal DVT are treated with anticoagulation unless there are absolute contraindications. Patients with distal DVT are treated with anticoagulation when there are a high number of risk factors for thrombus formation present. Patients with distal DVT are not treated with anticoagulation if there are relative contraindications to Heparin use, or if the clot is so small and so distal that is of minimal concern. However, for the patient not to be treated, they must not possess any risk factors. In addition, frequent follow-up with venous duplex examination should be performed.

Heparin anticoagulation is the mainstay of DVT and pulmonary embolism therapy. The use of Heparin significantly reduces the associated morbidity and mortality of this disease process. Heparin anticoagulation functions through the binding of Heparin to antithrombin III. Antithrombin III then catalyzes thrombin inactivation and

other intrinsic pathway factors. Therefore, patients with antithrombin III deficiency are resistant to Heparin anticoagulation.

If there are no contra-indications to anticoagulation, Heparin is initially given as a bolus IV dose, 100 units per kilogram of body weight. Thus, the usual starting bolus is between 5,000 and 10,000 units. Prior to the initiation of Heparin anticoagulation, base line blood studies are drawn, and include: PT, PTT, CBC, and SMA 7. Continuous Heparin infusion is then given at a rate of 1,000 units per hour, with a follow-up PTT level assessed in 4 to 6 hours. Infusion adjustments are made to keep the PTT level at 1.5 to 2 times normal. PTT and platelet counts are monitored daily, and a CBC and SMA 7 are drawn every other day.

If no invasive procedures or tests are planned, Coumadin therapy can be started on the same day that Heparin is initiated. Coumadin inhibits the synthesis of clotting factors II, VII, IX, and X, and anticoagulant proteins C and S. Therefore, it takes several days to achieve an adequate level, and several days to return to baseline. Patients on Coumadin are monitored with PT levels, which should be maintained at a level which is 1.2 to 1.5 times the normal PT level. If one uses the INR classification level (international normalized ratio), the PT level is maintained between 2 and 3 times normal. The usual loading dose of Coumadin is 10 mg per day times 3 days, then 5 mg per day as a maintenance level. PT levels are then drawn on a weekly or bi-monthly basis, depending upon the stability of the patient's level.

Patients are treated for an average of 3 to 4 months, depending upon the severity of the DVT or PE. Patients with recurrent thromboembolic disease, who posses high risk factors for subsequent thrombosis, may continue treatment for 6 months. Those with continued risk factors, such as malignancy or protein C deficiency, should be maintained on Coumadin indefinitely.

Thrombolytic enzymatic agents, such as urokinase and streptokinase, are not generally recommended for the treatment of DVT, as the indications for their use is still widely debated. Currently, the use of these lytic agents is reserved for the younger patient with large proximal DVT where treatment can be started within 48 hours of the initial event, or if the patient has a life-threatening, large, pulmonary embolism and the only recourse is surgery. Lytic therapy has not been shown to improve survival or shorten hospitalization. While studies have demonstrated earlier dissolution of the clot, no clear long-term benefit has been shown. There are many different regimens offered for lytic therapy. In general, whenever there are multiple recommendations for any given treatment protocol, the actual benefit to the treatment and superiority of one protocol over another is questionable.

Surgical intervention, which involves venous thrombectomy, is no longer the standard recommendation for treating proximal DVT. The recurrence rate and incidence of pulmonary embolism is much higher following thrombectomy than it is with Heparin and/or lytic agent therapy. Venous thrombectomy is now reserved only for a few select situations, such as phlegmasia cerulea dolans. This occurs when venous hypertension develops due to massive venous thrombosis, e.g. iliofemoral and greater saphenous vein thrombus without collateral drainage from the leg. It produces an increased compartmental pressure with a concomitant reduction in arterial inflow, resulting in ischemia to the lower extremity. This is an emergent situation requiring immediate removal of the venous thrombus. Venous thrombectomy is used in this situation in conjunction with lytic and anticoagulation therapy, and filter placement. Fortunately, this is a rare occurrence.

SEQUELAE

The most detrimental sequela to DVT is pulmonary embolism, which can be life threatening. There are other possible sequela which are not as life threatening, but still cause a great deal of morbidity. These sequela are collectively referred to as the post-phlebitic syndrome. Post-phlebitic syndrome includes persistent lower extremity edema, stasis dermatitis, stasis ulcerations, varicosities, pain, and recurrent DVT. Chronic venous insufficiency and venous stasis ulcers are often the result of a past history of proximal deep venous thrombosis.

POST-PHLEBITIC SYNDROMES

Thrombus Formation

In the development of DVT, a thrombus becomes attached to the endothelial lining of the vein wall, as well as the valve cusps. This can occur in a time frame of 72 hours, which corresponds to the first two to three days of postoperative bed rest. Afterwards, the thrombus is no longer friable or easily detached from the vein wall, as it mostly consists of a fibrous tissue (fibrin). Resolution of the clot is initiated by the invasion by fibroblasts, platelets, and mast cells. By day 14, the thrombus almost entirely consists of fibrin, and this is the reason why lytic agents are no longer effective when a clot is present for more than 14 days. Thereafter, the effectiveness of lytic agents continues to decrease with time. Therefore, lytic agents work best when a thrombus is fresh (less than 72 hours old), and primarily formed of red blood cells.

Recanalization

The disease process invokes an inflammatory response within the vessel, which results in vein wall thickening and destruction of the very friable and thin valve cusps. The thrombosed vein is eventually recanalized by the body's own natural lytic system, through the resorption of the fibrous material by macrophages. Once blood flow is restored through the recanalized vein, further recanalization occurs by the velocity of blood flowing past this area within the vein. This process may take 8 to 12 weeks to occur, and is the reason why most patients are on Coumadin therapy for 3 to 4 months.

Collateralization

By this point of time (3-4 months), leg edema is decreased secondary to the formation of venous collaterals, which carry blood around the thrombosed vein. However, when the primary pathway opens via recanalization, the blood will flow preferentially through the main vein due to lower resistance. Once additional blood begins to flow via the main vein, flow via the collaterals decreases. Due to this decrease in flow, the collaterals decrease in size.

Venous Congestion and Edema

Venous valvular cusps act as "check valves" and are responsible for the unidirectional flow of blood against gravity. In addition, they allow flow from distal to proximal and superficial to deep. Once the valves are destroyed, there is no mechanism to prevent retrograde flow from the deep to the superficial system. This phenomenon is referred to as an incompetent perforator.

The eventual result is venous pooling in the lower extremity, which begins distally and spreads proximally. Venous congestion occurs in the leg, and this can cause a heavy feeling in the leg which aches after periods of dependency (standing). Chronic venous insufficiency can continue for months to years, and if left untreated can cause the formation of venous stasis dermatitis and ulceration.

Chronic Venous Stasis and Ulceration

Stasis dermatitis and ulceration, which can serve as a nidus for infection, are thought to be caused not only by increased pressure on the skin from the underlying edema, but also from the shunting away of oxygenated arterial blood. Arteriovenous shunts result in shifting the arterial blood toward the venous side of the capillary beds. Increased venous pressure, from the poor evacuation of venous blood, is therefore associated with vascular and valvular incompetence.

Due to the increased venous pooling of blood, venous hypertension results, causing a leakage of fluid and molecules into the interstitial tissue, as based on the Starling principle. The increase in fluid and molecules in the interstitial tissue blocks oxygen diffusion, producing tissue ischemia and necrosis, resulting in venous ulcer formation. Furthermore, extremities with chronic venous insufficiency develop a classic brownish discoloration of the skin secondary to hemosiderin and melanin deposition.

Phlegmasia Cerulea Dolans

Phlegmasia cerulea dolans, another complication of DVT, occurs when venous hypertension develops secondary to iliofemoral and greater saphenous vein thrombosis. Once again, inadequate venous collateral drainage is implicated. The lack of collateral circulation can result in a massive venous thrombosis. It produces an increased com-

partmental pressure, with a concomitant reduction in arterial inflow, resulting in ischemia. This is an emergent situation requiring immediate removal of the venous thrombus by venous thrombectomy or aggressive lytic therapy. If untreated, venous hypertension results in an increased compartmental pressure syndrome, which can cause a total loss of arterial inflow, resulting in gangrenous changes of the entire lower extremity. This can occur within 6 hours of presentation.

CLASSIFICATION OF VENOUS DISEASE

A classification scheme was adopted by the Ad Hoc Committee For Reporting Standards of the Society for Vascular Surgeons, and the International Society for Cardiovascular Surgery. This classification scheme is based on clinical signs and symptoms. It includes patients with primary valvular insufficiency and post-thrombotic syndrome. Class 0 represents extremities without clinical evidence of chronic venous insufficiency. Class I represents mild disease, with the extremities typically demonstrating mild edema or dilation of the superficial veins. Class II signifies moderately severe disease, with the presence of significant edema or hyperpigmentation. Class III characterizes extremities with the most severe manifestations of chronic venous insufficiency, including patients with active or healed venous ulcers.

DIFFERENTIATING ARTERIAL AND VENOUS DISEASE

Most venous ulcers are located proximal to the ankle, and in the medial aspect of the leg. They have a characteristic shallow base that bleeds easily. This is quite different from an arterial ulcer, which is usually more distal on the lower extremity. In contrast, arterial ulcers are pale and do not bleed easily. A WORD OF WARNING BEFORE TREATING LOWER EXTREMITY ULCERS: BE SURE TO DOCUMENT THAT THERE IS ADEQUATE ARTERIAL FLOW TO THE LOWER EXTREMITY.

Venous claudication (pain) occurs in patients with an obstruction of the deep venous system. This is distinct from the typical mild aching sensation experienced at rest in patients with chronic venous insufficiency. It presents as cramping in the calf during ambulation, similar to that which is seen in patients with arterial insufficiency. These patients must also stop and rest for a period of time to allow the venous congestion to resolve. This is primarily seen in patients who have total iliofemoral thrombosis without adequate collateral formation. One must be sure that the arterial inflow is adequate, and if the patient is diagnosed with DVT, it usually indicates the lack of adequate venous collateralization. Patients with sufficient collateral venous flow, around the iliofemoral occlusion, usually do not experience venous claudication. The most frequent type of leg pain associated with chronic venous insufficiency is a heaviness or aching sensation that occurs after prolonged standing, and is usually felt over the calf area. This pain is eased by walking or lying down, particularly with elevation of the extremity.

PULMONARY EMBOLISM

Pulmonary embolism primarily occurs in patients with a proximal DVT. Patients with free floating thrombi in the iliofemoral segments are particularly prone to developing a pulmonary embolism. Depending on how diligently one searches for PE's, approximately 50% of all patients with a proximal DVT will have some degree of a pulmonary embolism. (Figure 2)

Heparin is the mainstay of therapy in patients with PE's. Once Heparin anticoagulation is initiated, it reduces the mortality and recurrence rates significantly.

A large pulmonary embolus in a hypotensive patient is life-threatening, and these patients are in need of immediate dissolution of the clot, either by surgery or lytic therapy. Pulmonary artery embolectomy, in this setting, is associated with a high mortality rate, and is reserved for patients in which lytic therapy has failed. Lytic agents are especially beneficial in this setting, however there is still an associated mortality rate of 50%. The routine use of lytic agents for the typical patient with a PE is still an area of great debate. Another option includes the placement of an inferior vena cava filter. There are many different filters available, including a Bird's nest, Nitinol, Greenfield, Adams-Deweese clip, Moretz, and Mobin-uddin filter. However, the Greenfield filter has the longest track record, and is the most familiar to vascular surgeons. (Figure 3)

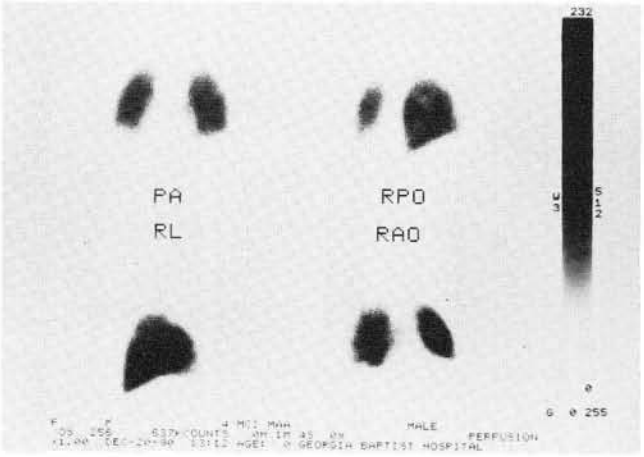

Figure 2A. Example of a positive ventilation perfusion scan. Note the decreased perfusion in this mild case.

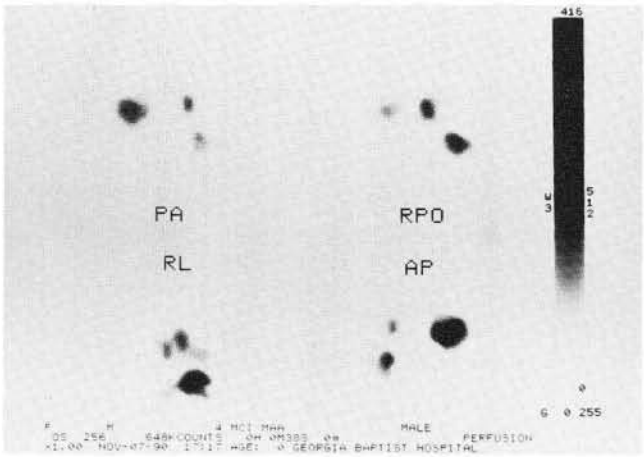

Figure 2B. Example of a positive ventilation perfusion scan. Note the almost complete loss of perfusion from PE in this severe case.

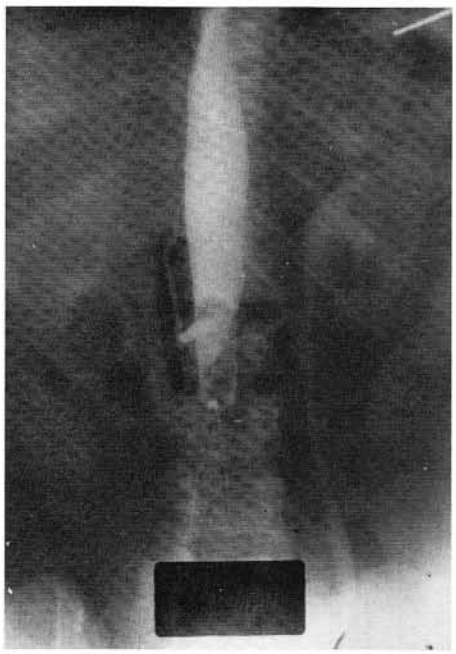

Figure 3. Radiograph of the proper placement of Greenfield filter in the inferior vena cava.

Indications for filter placement, in a patient with a pulmonary embolism and lower extremity DVT, includes a contraindication to anticoagulation, or a history of recurrence while on an *adequate* dosage regimen of anticoagulants. The filter is placed in the infrarenal position via the right internal jugular or the right femoral vein. These filters can now be placed via a percutaneous technique.

Venous filters, which are intended to prevent a PE, do not prevent DVT formation. In addition, 2% of patients with filters can have a recurrent PE. This occurs when either a thrombus slips along the side of the filter, via transportation through gonadal veins which bypass an infrarenal filter, or from the formation of a thrombus superior to the filter. Therefore, continued anticoagulation is recommended, even after filter placement, if there are no contraindications. The key element to remember is that an ounce of DVT prevention is worth a pound of cure for a chronically venous insufficient leg.

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