CHAPTER **54**

# DEEP VENOUS THROMBOSIS: A Practical Review with Current Recommendations

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Deep venous thrombosis (DVT) and pulmonary embolism (PE) can be the most devastating post-operative complications encountered by a surgeon. The incidence of pulmonary embolism is estimated between 300,000 and 600,000 cases per year.[1] Pulmonary emboli account for approximately 100,000 fatalities per year[2,3] and are the fourth leading cause of death in the United States.[4] A large percentage of pulmonary emboli are silent,[5,6] and although fatality in symptomatic pulmonary emboli usually occurs in the first 30 minutes,[2] four to eight thousand deaths a year could be prevented with simple prophylaxis.[2,4] Most pulmonary emboli originate from thrombosis within the veins of the lower extremity.[5] Proximal iliofemoral thrombosis accounts for a higher percentage of embolic events than does distal or calf thrombosis. Other significant sequelae from a deep venous thrombosis are the pain and discomfort which may be experienced at initial onset, and the possible development of post-phlebitic syndrome which occurs due to valvular incompetence subsequent to thrombosis.

Multiple orthopaedic studies have been performed to assess the risk factors, prophylactic measures, diagnostic modalities, and treatment regimens associated with deep venous thrombosis of the lower extremity.[7-41] Other medical specialties have also noted the necessity for DVT prophylaxis [42-45] and have shown a substantial decrease in the incidence of DVT when adequate prophylaxis is implemented. Marshall states that all general surgical patients undergoing major operative procedures should receive peri-operative DVT prophylaxis.[46] However, it is clear that many patients who need prophylactic measures do not receive them.[47-49] In one study, a review of hospitalized patients showed that only 32% of the patients categorized in the high risk group received appropriate protection against DVT.[47]

Most of the preventative measures against DVT are performed in the hospital. With the advent of economic-medical reform, hospital stays for both medical and surgical patients will potentially be much shorter. This will have an effect on the delivery and efficacy of prophylactic protocols against DVT. Even more alarming is the realization that most patients still have significant risk factors after discharge from the hospital. Although most thrombus formation begins during surgery or within 48 hours after surgery, several studies have documented that the development of DVTs can occur well into the postoperative course.[50-52] Kakkar observed that 23% of DVTs occurred between the 15th and 25th postoperative day.[52] Johnson reviewed approximately 8000 total hip arthroplasties and found that 36.1% of all fatal pulmonary emboli occurred between the third and fifth post-operative week.[53] Recently, Trowbridge et al. followed 38 total hip arthroplasties monthly with duplex ultrasound and venography for a total of three months. At the time of discharge from the hospital, patients were presumed to be thrombosis free after ultrasound or venographic evaluation. Four of the 38 patients developed post-hospitalization DVT; 2 of the 4 occurred during the first postoperative month while the other two developed during the second postoperative month.[35] This finding may support the recommendations of many orthopaedic surgeons who recommended extended, post-hospitalization coumadin prophylaxis for up to 12 weeks following discharge.[3,11,32,34,54]

A review of the major textbooks of surgery of the foot and ankle will reveal little if any discussion concerning the peri-operative prevention of deep venous thrombosis.[55-58] Because a majority of surgery performed on the foot and ankle is not considered major surgery, it would seem that foot and ankle surgeons are relatively immune to the

complication of deep venous thrombosis. However, major fusions, complex fracture repairs, and multiple osteotomies usually entail an operative time of over two hours, induction of general anesthesia, the use of a thigh tourniquet, and some type of postoperative immobilization of the operative extremity. Certain reconstructive procedures of the foot and ankle certainly qualify as major surgery and merit careful attention to additional risk factors for deep venous thrombosis. This realization, coupled with shorter hospitalization, an aging public, and the need for postoperative immobilization, has convinced the authors to be more aggressive with prophylaxis against DVT.

## RISK FACTORS

Thorough assessment of the variables involved with a specific surgery is first priority. Duration and anatomical location of surgery, type of anesthesia, use of a tourniquet, and postoperative immobilization are all primary considerations in deciding whether a patient will need peri-operative DVT prophylaxis. It becomes quite obvious that a patient undergoing routine forefoot surgery on an outpatient basis possesses far less risk than a patient who is scheduled for a triple arthrodesis. Based on the analysis of the procedure, many foot and ankle surgical patients will be eliminated from prophylactic consideration. However, in patients who will have major reconstructive surgery of the foot and ankle, analysis of common risk factors will help place patients into low, medium, or high risk groups for the development of thrombosis. Based on this classification, a decision on the need for prophylaxis can be made.

Many authors place all of the risk factors into one group (Table 1);[4,48,59-61] however, it is essential to identify the common or primary risk factors for proper classification. The three most common risk factors for DVT among hospitalized patients are recent major surgery, age, and obesity.[48] Even though there is no scientifically proven mechanism, several studies show that increasing age, especially greater than 65 years, predisposes the patient towards the development of DVT.[2,46,48,49,59-64] A significant number of the patients involved in foot and ankle surgery are older than 40 years. Also, obesity is not an uncommon finding, especially in patients who have had a dysfunctional foot and/or ankle which has subsequently curtailed previous activity levels. When evaluating all possible risk factors and applying the classification system used by Hull et al., surprisingly, many of the reconstructive surgical patients fall into the moderate and even the high risk groups.[65] (Table 2)

**Table 1**

### RISK FACTORS FOR DVT/PE

1. Recent surgery (increased with orthopaedic surgery)
2. Prolonged surgery (especially greater than 2 hours)
3. History of previous DVT
4. Inherited or acquired coagulopathies
5. Cancer
6. Immobilization or bed rest
7. Short or long leg cast following major surgery.
8. Congestive heart failure
9. Obesity
10. Age (especially greater than 65)
11. Oral estrogen use
12. Serious burns
13. Varicose veins
14. Tourniquet use

(Modified from Baker and Bick[69])

**Table 2**

### CLASSIFICATION OF RISK FOR DVT/PE

**HIGH RISK**

1. Age >40
2. Prolonged Surgery
3. Previous DVT or PE
4. Secondary Risk Factors*
5. Hereditary or acquired coagulopathies

**MODERATE RISK**

1. Age >40
2. Prolonged Surgery
3. Secondary Risk Factors*

**LOW RISK**

1. Minor Surgery
2. No secondary Risk Factors*

*Risk factors 6-14 in Table 1
(Modified from Merli59)

In some respects, major surgery of the foot and ankle may involve even more danger than predicted by any risk group analysis or classification system, due to the ambiguity of immobilization throughout the literature. Immobilization has not been properly and clearly defined with regards to foot and ankle surgery. Most definitions of immobilization actually are describing a non-ambulatory situation. It is clear that a patient may be ambulatory postoperatively but still have an entire extremity immobilized in a short or long leg cast. This differs greatly from the postoperative course of total hip or knee arthroplastic procedures. Weight bearing and motion are progressively encouraged during the early postoperative course of the above mentioned orthopaedic procedures. Motion activates muscle pump blood return and diminishes stasis. Clearance of blood from the lower extremity is largely dependent upon and augmented by contraction of the calf muscles. A patient in a short or long leg cast loses the benefit of active ankle joint motion and is at increased risk for developing a DVT, specifically a calf DVT.

Deep venous thrombosis occurring in the soleal or tibial veins has been given little attention due to the reported low incidence of fatal PE and the relative infrequency of post-phlebitic syndrome. Fatal pulmonary emboli are commonly thought to originate from large thrombi within the iliofemoral venous segment. Moser and LeMoine followed thirty-six DVTs to evaluate for subsequent pulmonary emboli with respect to location of the DVT. Twenty-one of the thirty-six thrombotic events occurred in and were isolated to the calf veins; whereas, fifteen DVTs were classified as proximal DVTs. Eight of the fifteen proximal DVTs demonstrated pulmonary embolism via a ventilation-perfusion scan. Only one of the eight patients had clinical symptomatology. None of the calf DVTs had a positive screening for a pulmonary embolism.[66] Even though this study down-plays the importance of calf thrombosis, indirectly it illustrates a common theme which can be seen throughout the literature. Many readers and researchers automatically conclude that only the proximal thrombotic event should be treated. However, the possibility of proximal extension from a popliteal thrombus to include the proximal iliofemoral complex was not specifically addressed by Moser and LeMoine.

Although less than 1% of all fatal pulmonary emboli are thought to originate from calf thrombosis, 20-30% of calf DVTs extend proximally into the popliteal space. Kakkar et al. followed 132 postoperative patients, and detected forty cases of deep venous thrombosis; thirty-nine of these were confirmed with phlebography. Fourteen of the forty demonstrated small thrombi less than five centimeters in length located in the soleal or tibial veins. All of these underwent spontaneous lysis within seventy-two hours, demonstrated by an absence of activity using $^{125}$I-labelled fibrinogen. Increased counts were present after seventy-two hours via $^{125}$I-labelled fibrinogen and remained localized to the calf in seventeen of the remaining twenty-six patients. Confirmation of location was achieved by repeat phlebography. Nine of the forty cases showed extension of calf thrombosis into the popliteal and/or iliofemoral veins via phlebography. Four of the nine cases developed pulmonary embolism.[67] Philbrick and Becker concluded that an acute calf DVT is not a benign event, and that propagation of a calf DVT varies from 5.6-23%.[68] There is no clear evidence that an isolated, acute calf DVT should be treated with anticoagulation; however, follow-up diagnostic studies to evaluate for proximal extension should be mandatory. Recently, Baker and Bick recommended treating distal thrombosis in the same fashion as a proximal thrombosis on an acute basis, followed by coumadin or heparin for three to six weeks, then aspirin for three months.[69]

How to treat an acute calf DVT remains controversial. From the previous discussion it should be clear that the prevention of a calf DVT is crucial due to the possibility of proximal extension and subsequent increased chances of pulmonary emboli. This is especially important if the patient will have a short or long leg cast with increased chances of stasis in the calf venous system.

Another important risk factor which cannot be overemphasized is the history of previous DVT. These patients should be treated as high risk, and strong consideration should given towards an aggressive prophylactic approach. In the presence of a previous DVT, an accurate history of prior surgery and/or trauma should be evaluated along with any other risk factors. If there is not a reasonable explanation for the DVT, then there should be suspicion of other predispositions towards the development of DVT such as hereditary or

acquired coagulopathies (antithrombin III or protein C or S deficiencies). Baker and Bick also suggest taking an extensive family history to look for DVT or predisposing coagulopathies.[69]

## PATHOGENESIS

It is clear from the literature that any major surgical candidate over the age of forty, potentially could develop a DVT. However, a brief overview of the pathogenesis of venous thrombosis will illustrate the same potential for formation in major surgery of the foot and ankle. In 1856, Virchow described a triad of pathophysiologic events which herald the evolution of venous thrombosis.[61] The effects of any surgical procedure on venous blood flow, endothelial integrity of a vessel, and viscosity or hypercoagulabilty of blood are directly related to the development of DVT.

Venous stasis is affected by both the type of anesthesia and the position of the patient and extremity during anesthesia. General anesthesia is associated with greater occurrence of DVT than spinal anesthesia. This is possibly due to the peripheral vasodilatory effect from general anesthetic agents which results in pooling or stasis of blood in the soleal sinusoids.[70-72] Paralyzing agents used during endotracheal intubation also contribute to venous stasis by inactivating the calf pump.[60] Venographic studies have also shown a decreased venous return in patients in the supine position.[73,74]

Excessive and prolonged rotation of the hip has been shown to diminish venous return. Three separate studies, utilizing intra-operative venographic techniques, documented a narrowing effect on the femoral vein with excessive internal and external rotation of the hip for the placement of a total hip implant.[17,75,76] Although there is not nearly as much hip repositioning during major surgery to the foot and ankle, a surgeon may elect to maximally internally or externally rotate the lower extremity at the hip joint to aid in the lateral or medial dissection of the rearfoot or ankle. This probably has a negligible effect on venous stasis and endothelial damage, but could be contributory during a long procedure utilizing a tourniquet with the patient under general anesthesia and in the supine position. In essence, there is a cumulative effect by each of these seemingly minor factors.

An intact endothelial surface is essential for the prevention of thrombous formation.[4] Exposed subendothelial collagen is a nidus for clot formation. Trauma or surgery in the area of the venous return may result in endothelial damage. Excessive manipulation of the extremity and prolonged angular positioning (e.g. flexion of the knee) can also produce intimal damage. The most common cause of endothelial pathology during foot and ankle surgery would be the use of a thigh tourniquet. Not only can the tourniquet cause direct effects on the intima of the vein with prolonged use, but the tourniquet can also cause damage in an indirect fashion. Anoxia produced by the tourniquet is postulated to effect the endothelium at cusp sites, where the endothelial cells are either rendered malfunctional or are shed due the decreased oxygen tension.[60,77] Multiple studies have shown stagnation of blood flow at the cusp sites in the soleal venous complex. An area with pooled blood and an absence of endothelium is at high risk for clot formation.

Some degree of hypercoagulability may be present in patients who undergo major orthopaedic surgery.[59] Fibrinogen has been show to be increased following surgery.[60] This may be due to an increase in the production of fibrinogen, but most likely is secondary to a shutdown of the fibrinolytic system.[39,74] This, coupled with the documented decrease of anti-thrombin III in post-operative orthopaedic patients,[22] produces hypercoagulability.[79] Some orthopaedic surgeons use supplemental antithrombin III with heparin as prophylaxis against DVT.[22]

## PROPHYLAXIS

There are two approaches for the prophylaxis of venous thromboembolism. The first approach would be considered primary prophylaxis and entails pharmacologic and mechanical methods administered prior to surgery in order to prevent thrombosis formation. The second method utilizes diagnostic screening tests (e.g. venography) for the early detection of deep venous thrombosis, and institution of treatment for the prevention of an embolism. The second method is called secondary prophylaxis. The significant risk of failure in primary prophylaxis is the development of a DVT, while the risk of failure in secondary prophylaxis is embolism. Obviously, an embolism is a much greater concern than a deep venous thrombosis, because an embolism carries with it the immediate risk of death. It is always preferential to prevent a

disease process rather than make an early diagnosis and then treat the pathology. In addition, a DVT which does not produce a clinically significant embolism is not without potential sequelae (e.g. post-phlebitic syndrome). It is highly recommended to use primary prophylaxis whenever possible to prevent the development and possible sequelae of a deep venous thrombosis.[4]

A plethora of literature has been published concerning prophylaxis against the development of deep venous thrombosis. There are no controlled studies demonstrating a significant incidence of DVT with subsequent pulmonary emboli in unprophylaxed patients undergoing foot and ankle surgery. However, it is quite evident that major reconstructive surgery of the foot and ankle should have prophylactic measures for the prevention of venous thrombosis when clinically warranted. The authors recommend that a physician assess the risks and benefits of multiple treatment options before deciding on a particular regimen. For proper assessment, a thorough history and physical is essential. This will help identify patients who are candidates for prophylaxis, as well as those who may be intolerant of a particular prophylactic modality (e.g. a patient with a low-normal platelet count preoperatively may not do well with adjusted dose heparin due to its potential side effect of thrombocytopenia). It is also highly recommended that a team approach be used in regard to DVT prophylaxis. This should optimize patient care and minimize risks. The following is a brief synopsis of the most common methods used for DVT prophylaxis.

According to Raskob and Hull, an ideal agent for prophylaxis is efficacious, well-tolerated by the patient, and accepted by health care providers. The prophylactic modality should also be economical, easily administered, and require minimal monitoring.[61] Prophylaxis can either be administered in a mechanical or pharmacologic manner. There are multiple forms of medical prophylaxis including but not limited to unfractionated heparin, low molecular weight heparin, coumadin, dextran, and aspirin. Mechanical prophylactic modalities include early ambulation, elevation of extremities, gradient compression stockings, and intermittent pneumatic compression devices.

## PHARMACOLOGIC METHODS

### Heparin

Heparin is the most widely-used and studied form of prophylaxis in the surgical and non-surgical patient, and is considered by some the benchmark for thrombosis prophylaxis.[69,80,81] Heparin is a glycosaminoglycan derived from either porcine intestinal mucosa or beef lung. The main utility of heparin for the prophylaxis and treatment of DVT lies in its ability to inhibit thrombin formation. This is accomplished primarily by inhibiting factor Xa and potentiating the effects of antithrombin III. Clotting factor Xa helps convert prothrombin to thrombin. The capability of a specific heparin preparation to inhibit factor Xa is directly proportional to in vivo anticoagulation.[69] Heparin serves as a cofactor and accelerates the formation of a molecular complex between antithrombin III and coagulation factors (XII, XI, IX, and X). This increases inhibition of thrombin formation.[82] Thrombin has a positive feedback effect on the clotting cascade, specifically on factors V and VIII. The presence of thrombin can result in the generation of large quantities of more thrombin. Some feel that the most important effect of heparin is the avoidance of this positive feedback mechanism by the prevention of thrombin formation.[82,83]

Thus, in order for heparin to be effective as a prophylactic agent, it must be given prior to surgery as it works by inhibiting the formation of thrombin. Heparin does not dissolve a formed thrombus. Fixed low-dose heparin is usually given 5000 units two hours preoperatively, followed by 5000 units either every eight or twelve hours. This regimen is commonly referred to as mini-dose heparin or fixed low-dose heparin.

Heparin is clearly an effective prophylactic agent in moderate risk general surgical and medical patients. Browse reviewed 24 studies involving 4932 general surgery patients. Heparin given subcutaneously every 8-12 hours following a 2 hour preoperative dose was shown to reduce the incidence of DVT from 30% to 9%, compared to patients who did not receive heparin.[60] There is a debate as to whether the twice a day or three times a day dosing schedule is superior. There is no controlled study which has unequivocal evidence supporting one over the other. However, Clagett reviewed 29 studies which included over 8000

patients. In patients treated with heparin dosed every 12 hours, there were 289 DVTs out of 2446 patients, or 11.8%. Patients who were dosed every eight hours developed DVTs in 159 cases out of a possible 2039 patients, which equates to a 7.5% incidence. Bleeding and wound hematomas were also compared between the two dosing regimen. Although slightly more bleeding complications occurred with the Q 8 hour dose (20/1142 vs. 36/3839) statistically there was not a significant difference. The development of wound hematomas was almost identical between the two groups.[84]

Even though it has been shown that prophylaxis decreases the incidence of DVTs in orthopaedic surgery,[85] most authorities do not recommend low dose heparin for the prophylaxis of high risk patients, especially those with total knee or hip implants or hip fractures.[4,86,87] Mohr's review of DVT prophylaxis for hip and knee implants at the Mayo Clinic showed that only 9 out of a possible 409 cases were chosen for the use of heparin as a prophylactic measure.[29] This reluctance to use low dose heparin in high risk patients is supported by its ineffectiveness when compared to other regimens.[88,89] Because of this, physicians have used an adjusted dose of heparin for high risk patients.[12,90] The heparin is adjusted postoperatively to keep the partial thromboplastin time within 4 seconds of high normal. Increased bleeding complications have been noted with this type of heparin dosing, but it is an accepted alternative for the prophylaxis of high risk patients, and appears more effective than regular mini-dose heparin.

In general, heparin should not be used for eye surgery, neurosurgery, and spinal surgery due to the significant consequences of bleeding. Low dose heparin does not need to be monitored; however, a baseline platelet count is recommended and should be followed if patients have a low normal count, or if patients will be on extended outpatient use. Thrombocytopenia is an observed side effect in up to 10% of patients with long term heparin use.[91]

### Heparin with Dihydroergotamine

Low dose heparin with dihydroergotamine (DHE) has been shown to be more effective than plain low dose heparin for the prophylaxis of high risk patients.[92-94] Dihydroergotamine increases the velocity of venous return by decreasing the diameter of the vessel. Its vasoconstrictive effects are due to alpha adrenergic activity on the smooth muscle cell. Dihydroergotamine is given at a dose of 0.5mg subcutaneously two or three times per day. The effect of dihydroergotamine may act synergistically with heparin and improve the prophylactic effect of heparin; however, DHE used alone is not reported to have a significant effect on the prevention of calf DVTs.[60] With an arithmetical study comparison, Browse showed that a combination of heparin and dihydroergotamine had a 6% incidence of DVT compared to the 10% rate with heparin alone. However, this may not be statistically significant when evaluating the number of patients along with the percentages.[60] Significant vasospastic side effects have placed the use of DHE in disfavor. It is not recommended in the presence of coronary artery disease or peripheral arterial ischaemia. Currently, the combination is not being marketed in the United States due to the above stated side effects.[2]

### Heparin with Antithrombin III

Heparin has also been combined with supplemental antithrombin III. Postoperatively, there is a decrease in the plasma concentration of circulating antithrombin III. Antithrombin III has anticoagulant properties due to its ability to inactivate enzymes involved in the coagulation cascade. Francis et al. compared heparin and antithrombin III to dextran 40 for prophylaxis against DVT in hip implant arthroplasty. Antithrombin III levels were monitored postoperatively and found to be much closer to preoperative levels as compared to the dextran group. Francis hypothesized that this translated into the decreased rate of DVT in the antithrombin III group. Only 3 of 41 patients in the antithrombin III group were diagnosed with DVT via ascending venography, while 12 of 41 patients in the dextran group were diagnosed with DVT by the same method.[22] Although the cost of antithrombin III therapy may be prohibitive compared to equally effective methods, it may prove cost-effective if a substantial decrease in DVT can be shown. Additional clinical studies are needed to further outline the potential benefits of antithrombin III, alone or in conjunction with heparin.

### Low Molecular Weight Heparin

During the last few years, low molecular weight heparin (LMW heparin) has become an attractive alternative for DVT prophylaxis. LMW heparin has a similar mechanism of action as standard heparin,

but, proportionally, has a greater effect on factor Xa. As the molecular weight of heparin decreases, the effect on the activated partial thromboplastin time diminishes, but the effect on factor Xa remains significant.[69,95] This translates into adequate antithrombotic activity with less of a chance of bleeding when dosed properly. LMW heparin is reported to have greater bioavailability and a longer biological half life.[69,81,95] This allows once a day dosing. LMW heparin also has decreased effects on platelets when compared to unfractionated heparin, and subsequently causes less thrombocytopenia.

Several studies have demonstrated the efficacy of LMW heparin for prophylaxis against DVT.[13,96-98] Specifically, LMW heparin has been shown to be effective in orthopaedic surgery.[14,17,95,96,99,100] In a meta-analysis performed by Bergqvist using 32 randomized trials, LMW heparin was shown to be more efficacious than standard low-dose heparin. In the LMW heparin group, there were no fatal pulmonary emboli out of 5027 patients; whereas, in the heparin group, there were 6 fatal pulmonary emboli from 4873 patients.[94] The optimal time for initial administration has not yet been established. Three different regimens currently exist: 10-12 hours preoperatively, 2 hours preoperatively, or postoperatively.[94] LMW heparin has been primarily used outside of the United States for the past fifteen years. Results from ongoing clinical trials should increase the availability of this modality, as it appears to be an efficacious, cost effective prophylactic agent.

### Coumadin

Oral anticoagulation via coumadin is a first line choice for prophylaxis in high risk orthopaedic patients [2,4,60,61] Coumadin also has been shown to be effective in gynecologic[101] and general surgery patients.[102] Coumadin works by its inhibition of vitamin K dependent coagulation factors II, VII, IX, X, protein C, and protein S. There are many accepted ways to institute coumadin therapy, but almost all start with a dose given sometime preoperatively. The three most common ways to institute coumadin prophylaxis are listed in Table 3.

**Table 3**

**METHODS OF COUMADIN PROPHYLAXIS FOR DVT/PE**

**METHOD 1**

Evening before surgery - 10mg
Evening of surgery - 5mg
Adjust PT to 16-18s

**METHOD 2**

Begin 12-14 days preoperatively
Maintain PT 1.5-3.0 sec. above control
POD #1 Adjust PT to 16-18s

**METHOD 3**

Evening of surgery 10mg
No Coumadin POD #1
POD #2 adjust PT to 16-18s
(Modified from Merli[59])

There are a few disadvantages which make coumadin less attractive than other prophylactic modalities. Monitoring must be performed to ensure appropriate dosing. Even with the INR between the recommended levels (2.0-3.0), a 6.0% incidence of major bleeding has been reported.[69] The time before the anticoagulant takes effect is at least 36 hours. The risk of hemorrhage is greater than with other pharmacologic methods. There are also many drug interactions with comadin which may increase the risk complications. Before instituting coumadin prophylaxis, the physician should review the particular patient's medications to look for possible interactions with the coumadin.

Often, oral anticoagulation is continued until the patient is fully ambulatory. Post-discharge coumadin administration has been advocated to last between 4[3] and 12 weeks.[10] Paiement et al. reviewed 268 patients who were treated with adjusted low-dose coumadin for the prevention of venous thromboembolism. Patients were given 10 milligrams of coumadin the night before surgery, and adjusted coumadin postoperatively to maintain a PT between 14 and 16 seconds. The patients continued prophylaxis for 12 weeks postoperatively. There were no fatal pulmonary emboli and only two non-fatal pulmonary emboli, both of which occurred prior to discharge. All ten major bleeding

episodes occurred during hospitalization. Sixteen patients experienced minor bleeding episodes, none of which required treatment.[10]

## MECHANICAL MODALITIES

Mechanical modalities for DVT prophylaxis includes leg elevation, early ambulation, gradient compression stockings, and intermittent pneumatic compression devices. Early ambulation and postoperative leg elevation clearly have been shown to be effective in the prevention of DVT.[103] However, these methods should not be relied upon solely in the presence of moderate to high risk patients.[4,63] Gradient compression stockings (GCS) aid in venous return and may prevent trauma to the vessel.[104] GCS has a compressive effect which commonly starts at 18mmHg at the ankle and tapers to 8mmHg at the popliteal fossa. There is 10mmHg of pressure at the low thigh, which decreases to 8mmHg at the midthigh.[63] Jeffery and Nicholaides report that GCS reduce the rate of DVT by 60% when compared to control groups. This effect is increased to 85% when an additional modality is implemented. The same authors recommend GCS as a first line modality in all hospitalized patient with low risk.[104] GCS are used for moderate and high risk patients, but the addition of another mechanical or a pharmacologic method is mandatory.[63,104]

The most common, effective form of mechanical prophylaxis is intermittent pneumatic leg compression, which will help prevent thrombosis in two ways. First, the compression device will increase blood return from the lower extremity and help prevent stasis. However, in lower leg surgery the operative extremity does not benefit from this and may still be predisposed to venous stasis and subsequent DVT. The second effect of the compression device is that it will increase the activity of the fibrinolytic system. Increased levels of prostacyclin and fibinolytic byproducts have been demonstrated with the use of IPC boots.[105,106] This is a systemic effect of the compression, and is not dependent on where the device is placed; thus, the operative extremity still will receive some benefit from the increased fibrinolysis. The effect on the fibrnolytic system is directly proportional to the surface area of tissue compressed; therefore, thigh-high IPC boots are recommended. Graded sequential IPC boots are preferred over uniform IPC boots. Optimally, the IPC boots are placed on the non-operative extremity prior to surgery.

Mechanical prophylaxis is opted for when there is a contraindication for heparin and/or coumadin in the face of a potential bleeding complication. There is definite evidence that IPC boots have a significant reduction in the formation of distal DVT; however, there is some concern with proximal DVT.[84,107] Francis et al. found a proximal DVT rate of 12% with the use of IPC boots, compared to a 3% occurrence rate with coumadin prophylaxis in major orthopaedic procedures. The authors concluded that coumadin was significantly more effective than IPC for the prophylaxis of DVT in hip replacement patients. The high incidence of proximal DVT with IPC only, caused termination of the study.[21] In contrast, Hull and associates have shown that full-length IPC boots lowered the incidence of iliofemoral DVT.[108]

IPC boots are especially useful in patients who cannot tolerate pharmacologic prophylaxis due to potential bleeding complications (e.g neurosurgical patient).[109] Intermittent pneumatic compression can be used solely or in conjunction with pharmacologic agents, and has been used in many trials with excellent success. The devices are applied prior to surgery and are mechanically inflated intermittently throughout the surgery. This is continued after the surgery until the patient is ambulatory. The only downside is that the devices may be cumbersome for the patients.

## DISCUSSION

Any patient who is scheduled to undergo reconstructive surgery of the foot and/or ankle and is over forty years of age should be evaluated for additional risks factors for the development of deep venous thrombosis. By most, foot surgery or podiatric surgery is considered minor surgery. However, by using criteria from other specialties it is quite evident that a sector of podiatric surgery is indeed major surgery. For instance, a patient who is scheduled to undergo a major rearfoot, ankle, or pantalar arthrodesis will most likely experience general anesthesia in a supine position with the utilization of a thigh tourniquet. Most of these procedures take greater than two hours to complete successfully. All of these factors have been shown to increase the predilection for a patient to develop a DVT, especially in the presence of additional known risk factors, such as obesity, cigarette smoking, estrogen supplementation, diabetes, and immobilization.

Additionally, a majority of these patients will be non-weight bearing in a leg cast for at least six to eight weeks. The literature suggests that immobilization is no longer a risk factor once ambulation begins. However, it may be prudent to view patients who are non-weight bearing in a cast as having a greater risk for the development of DVT, although the patients are ambulatory on the contralateral extremity with crutches or a walker.

The authors routinely evaluate major surgical cases with respect to thrombotic risk factors. Patients who have an additional risk factor for developing a DVT are considered at moderate risk. Moderate risk patients carry a 10-40% chance of calf DVT, a 2-10% chance of a proximal DVT, and 0.1-0.7% chance of a pulmonary embolism.[59,69] These patients are given heparin 5000 units subcutaneously two hours preoperatively, followed by 5000 units every eight hours. In addition, the authors usually employ a mechanical method for prophylaxis as well.

A potential drawback to the administration of preoperative heparin is the fear of hemorrhage and subsequent postoperative wound complications due to uncontrolled bleeding. There is no significant literature to support the contention that low dose heparin causes bleeding. The authors have found that meticulous dissection, intra-operative hemostasis, and judicious use of drains have a much greater effect on bleeding and wound healing than the use of preoperative heparin.

Gradient compressive stockings or pneumatic compression devices are added preoperatively. Gradient compressive stockings are used in moderate risk patients while pneumatic compressive devices are utilized in the high risk patients. Care must be taken in obese patients when applying gradient compression stockings, as the proximal aspect may not have a large enough diameter to encompass the entire thigh. The compression stocking may actually produce a tourniquet effect in this instance.

With the realization that patients have a significant risk of forming a DVT after discharge from the hospital, moderate and high risk patients are considered for outpatient thrombotic prophylaxis. Patients who do not have a contraindication to coumadin are placed on 2.5 milligrams of coumadin on postoperative day number one. Sequential INR values are obtained and compared to the preoperative value. An INR stands for the international normalized ratio and represents a standardized or corrected prothrombin time. The authors attempt to keep the INR at about 2 or the PT between 13 and 15 seconds. Other surgeons have recommended the use of postoperative coumadin prophylaxis ranging from one milligram[109] to 5 milligrams per day.[10] Lotke et al. recently gave strong recommendation for the continued prophylaxis of patients on an outpatient basis for up to 3 months.[7]

Currently, the authors continue the 2.5 milligram per day dose on an outpatient basis for the entire non-weight bearing time period. For the first 4 weeks a repeat INR is obtained on a weekly basis and is communicated to the attending surgeon and the co-admitting physician. Any adjustments can be made from this value. Coumadin is a highly protein bound drug which may interact with other drugs that a patient may be taking. Some medications cause altered activity of coumadin due to protein displacement (e.g. NSAIDs). Patients must be instructed to be aware of signs of bleeding.

Other simple measures can be performed to help decrease risk factors for the development of DVT. A Jones compressive dressing which encompasses the gastroc-soleal complex may help to increase venous return. Elevation of the affected extremity with early range of motion exercises of the contralateral limb can also be beneficial. Although there is no specific documentation, isometric exercises for the operative extremity while casted may improve venous blood return. Early mobilization of the patient postoperatively is essential to decrease of the formation of thrombosis.

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