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Venous Thromboembolism Prophylaxis in Foot and Ankle Surgery: A Literature Review

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Venous thromboembolism (VTE) is a well-known entity and one with potentially fatal consequences. However, there is very little data in the literature on VTE prophylaxis relative to foot and ankle surgery. The overwhelming majority of orthopedic VTE literature involves major orthopedic surgery, which has distinct differences in comparison to foot and ankle surgery. We review the recent recommendations and studies that may provide useful information on this vastly important subject for foot and ankle surgeons.

Key words: VTE prophylaxis, DVT

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The incidence of venous thromboembolism (VTE), which includes both deep vein thrombosis (DVT) and pulmonary embolism (PE), has been estimated at 1,350,000 cases each year in the United States alone.[1] These events can have potentially grave consequences. Clinically overt massive or fatal PE occurs in the presence of proximal vein thrombosis (thromboses at or above the knee).[2] Because 10% of proximal DVT's embolize massively or fatally, clinically occult DVT is very dangerous.[2]

Symptomatic proximal DVT has a 40-50% rate of PE if left untreated.[3] In addition, calf vein thrombosis propagates and then becomes proximal in 30% of patients.[2] The basal rate of thromboembolic disease in the general population has been reported to be 0.076%.[4] The VTE risk for patients undergoing surgery, however, is determined by the combination of individual predisposing factors and the specific type of surgery.[5] Risk factors for VTE include previous VTE, increasing age, female gender, thrombophilia, obesity, malignancy, heart failure or recent myocardial infarction, and stasis, all of which are known to increase the risk of VTE twofold to ninefold.[3]

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Surgery
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Estrogen-containing oral contraception
Hormone replacement therapy
Selective estrogen receptor modulators
Acute medical illness
Respiratory failure
Inflammatory bowel disease
Nephrotic syndrome
Myeloproliferative disorders
Paroxysmal nocturnal hemoglobinuria
Smoking
Varicose veins
Central venous catheterization

Table 1 Additional risk factors that may cause VTE.[6]

Commonly unrecognized VTE risk factors also include sepsis, severe chronic obstructive pulmonary disease, sickle cell disease, spinal cord injury, and stroke with impaired mobility.[1] Postoperative DVT usually starts in the calf, or less commonly, at the site of venous trauma.[7] It is important to consider that postoperative DVT of the lower limb is often asymptomatic, and that fatal PE is the first clinical manifestation of postoperative VTE in many patients.[5]

Unfortunately, there is a true paucity of information in the literature on VTE prophylaxis relative to foot and ankle surgery. The overwhelming majority of VTE literature in orthopedics involves "major orthopedic surgery" which includes hip replacement, knee replacement, and hip fracture surgeries.[5,6] Patients undergoing these type of procedures are considered to be at particularly high risk, and VTE prophylaxis with vitamin K antagonists, low-molecular-weight heparin (LMWH), or fondaparinux sodium (Arixtra®) is recommended.[5,6]

Factors contributing to the high rate of VTE in these patients include direct vessel trauma, venous stasis of the limb, and a population that is already at high baseline risk due to age and immobility.[3] Routine thromboprophylaxis has been the standard of care for major orthopedic surgery for over 18 years.[6] One study found that the rate of deep vein thrombosis (DVT) without prophylaxis is 45-57% for hip replacement surgery and 40-84% in knee replacement surgery.[8]

The incidence of PE in major orthopedic surgery in the absence of prophylaxis is around 8.6%.[9]

The incidence of fatal PE without thromboprophylaxis in these major orthopedic procedures ranges from 0.1% to 12.9%.[9] Even with the use of prophylaxis, the rate of clinically overt VTE in patients undergoing major orthopedic surgery remains almost 3%.[5]

The only multicenter study on VTE following foot and ankle surgery, to our knowledge, is that of Mizel, et al, from 1998.[4] The authors evaluated 2733 patients for preoperative risk factors and postoperative thromboembolic events. The use of postoperative VTE prophylaxis, if any, was determined by the surgeon, and medication, dose, duration, and other pertinent information was not provided in the article. The incidence of DVT was 0.22%, and that of nonfatal PE, 0.15%. No patient experienced a fatal PE, but the authors reported that the frequency of fatal PE after foot and ankle surgery appears to be less than 0.037%. Based on these results, the authors concluded that routine prophylaxis for thromboembolic events after foot and ankle surgery is probably not warranted. They also found that the only statistically significant relationships with thromboembolic events were postoperative regimens of non-weightbearing and cast immobilization. All patients with thrombotic events had been treated postoperatively with immobilization and nonweightbearing, with a thromboembolism incidence of 6 of 1150 (0.52%).

Because all of the patients who were nonweightbearing also were immobilized, the two effects could not be separated from each other. No relationship was found between VTE occurrence and age, weight, history of previous DVT, steroid use, diabetes mellitus, cancer, cardiac disease, hypertension, or any medical condition. Tourniquet use was also not predictive of a thromboembolic event.

The incidence of DVT after foot and ankle surgery was also studied by Solis and Saxby.[10]

The study involved 201 patients, none of whom received DVT prophylaxis, and underwent bilateral calf duplex ultrasound at their first postoperative visit. Deep calf clots were found in 3.5%, but none showed progression on follow-up ultrasound or extension proximal to the calf. None of these patients had any clinical symptoms. These authors also concluded that the rate and progression of DVT after foot and ankle surgery is low and does not require routine prophylaxis, even in high risk patients. They also noted that postoperative immobilization ($p=0.053$), hindfoot surgery ($p=0.02$), increased tourniquet time ($p=0.03$), and advancing age ($p=0.051$) were associated with risk of DVT formation. The article did not specify what constitutes increased tourniquet time or advancing age.

Medical prophylaxis in major orthopedic surgery

As noted previously, vitamin K antagonists, low-molecular-weight heparin (LMWH), or fondaparinux sodium (Arixtra®) is recommended as VTE prophylaxis for patients undergoing major orthopedic surgery.[5] The use of aspirin as VTE prophylaxis is controversial.[11] The guidelines from the Seventh American College of Chest Physicians Conference on Antithrombotic and Thrombolytic Therapy recommend against the use of aspirin *alone* as thromboprophylaxis for any patient group.[6]

Among other reasons, they cited that the inferior efficacy of aspirin compared to other methods of VTE prophylaxis has been demonstrated in clinical trials, and that aspirin use is associated with a small but significant increased risk of major bleeding, especially if combined with other anti-thrombotic agents.[6]

Antiplatelet therapy trials for VTE prevention have often been inconclusive, but a meta-analysis of their results indicated reductions in DVT and PE risks in various high-risk groups.[12] The Pulmonary Embolism Prevention (PEP) trial, a large randomized placebo-controlled trial, was undertaken to analyze this further.

The trial evaluated the effect of 160 mg of aspirin per day begun preoperatively and continued until postoperative day 35. The study consisted of 13,356 patients undergoing hip fracture surgery at 148 hospitals in five countries, and 4088 patients undergoing elective hip or knee arthroplasty at 22 hospitals in New Zealand. There were proportional reductions in PE of 43% and in symptomatic DVT of 29% in the hip fracture group receiving aspirin. Aspirin also prevented 4 fatal PE per 1000 patients, representing a proportional reduction of 58%, with no apparent effect on deaths from any other vascular or non-vascular cause. The proportional effects of aspirin in elective arthroplasty were comparable to that of the hip fracture group. The authors stated that this trial, along with results of the previous meta-analysis, demonstrated that aspirin reduces the risk of PE and DVT by at least a third throughout a period of increased risk, and noted that the risks of PE and other vascular events may continue for many weeks or months postoperatively. They concluded that there is good evidence for considering aspirin routinely in a wide range of surgical and medical groups at high risk of VTE, and for continuing it throughout the period of increased risk.[12] However, it is difficult to extrapolate the results of this study to the foot and ankle, as these results are taken from data on the larger and more proximal joints of the lower extremity.

It also does not take into consideration the combined effects of aspirin and mechanical prophylactic measures, as none of the latter were employed in this study.

Kearon states that the PEP trial findings are consistent with the results of the Antiplatelet Trialists' Collaboration, in which antiplatelet therapy reduced postoperative DVT by 26% and PE by 63% in an overview of 60 studies.[7] Kearon therefore estimates that after an initial 7-10 days of LMWH or warfarin, a month of aspirin therapy (81 to 325 mg/day) will reduce the frequency of post discharge symptomatic VTE by one third.[7]

Questions remain regarding the risks and benefits of starting VTE prophylaxis preoperatively or postoperatively and of continuing for several months, since most bleeding complications occur in the first few days after surgery, while the risk of thromboembolism may persist for some weeks or months after hospital discharge.[12] Agnelli states that the results of several studies support extended prophylaxis after discharge in high-risk surgical patients.[5] Schiff, et al, note that prolonged prophylaxis (up to 35 days) has been demonstrated to significantly decrease the frequency of venographically proven VTE in orthopedic surgery and likely decreases symptomatic VTE as well.[3]

There is also controversy over the optimal time for initiation of VTE prophylaxis. Agnelli states that in general, perioperative prophylaxis given between 2 hours before and 4 hours after surgery is more effective; however, it is associated with an increased risk of bleeding.[5] Thus, perioperative prophylaxis should be given to those at high risk for DVT and low risk of bleeding.[5] For major orthopedic surgery, the Seventh Chest Physicians Conference guidelines recommend that a decision about the timing of prophylaxis initiation be based on the efficacy-to-bleeding tradeoffs for that particular agent.[6] For low molecular weight heparin (LMWH), starting preoperatively or postoperatively are both acceptable.[6]

Medical prophylaxis and bleeding complications

In the PEP trial, aspirin caused six bleeding episodes that were thought to require transfusion per 1000 patients undergoing hip fracture surgery, but there was no increase in fatal, cerebral, or disabling bleeding episodes.[12] Hematemesis or melena associated with transfusion occurred in only one more patient per 1000 given aspirin, but there was a definite excess of nine per 1000 with less severe gastrointestinal bleeding. Aspirin was not associated with significant excess of wound hematoma evacuation or suction drainage volume.

A small, but statistically significant extra fall in mean perioperative hemoglobin concentration of 0.2 g/dL occurred in the aspirin group.[12]

Agnelli states that when pharmacological prophylaxis is used properly, the risk of bleeding complications is low.[5] Provided patients with contraindications are not treated, Kearon further remarks that extended prophylaxis with LMWH does not appear to be associated with increased major bleeding after major orthopedic surgery, although minor bleeding is approximately 50% more common (3.4% vs. 2.7%).[7] In a pooled analysis of randomized controlled trials reporting on the incidence of venous thrombosis and bleeding with LMWH prophylaxis in elective hip surgery, the rate of major bleeding ranged from 1.4% to 6.3%.[13] In this study, the group with preoperative initiation of LMWH prophylaxis, defined as beginning at least 12 hours before surgery, had a 1.4% major bleeding rate. The postoperative group, defined as those who began prophylaxis 12-48 hours after surgery, had a 2.5% major bleeding rate. The perioperative group, which included all trials that began LMWH prophylaxis 2 hours before or up to 4 hours after surgery, had a 6.3% major bleeding rate.[13]

Warfarin inhibits carboxylation of natural anticoagulant proteins C and S, resulting in a rapid decline of their levels.[14] This poses a theoretical risk for a venous thromboembolic event in patients who have a deficiency of proteins C and S at baseline or who have a hypercoagulable state.[14] However, bleeding is the primary concerning adverse event with warfarin.[15] Estimates of bleeding rates vary widely depending on study design, with annual incidences of 0.6% for fatal bleeding, 3.0% for major bleeding, and 9.6% for major or minor bleeding.[15] One meta-analysis of randomized trials in major orthopedic surgery found that vitamin K antagonist therapy was associated with a significantly higher risk of bleeding, mostly wound hematoma.[9] However, the authors noted that this risk appears acceptable since, in contrast to PE, the bleeding was rarely life-threatening.

In 538 patients, there was one fatal major hemorrhage with vitamin K antagonists compared with none administering placebo or no treatment.[9]

Many different factors, such as concurrent use of interacting medications, can increase a patient's risk for bleeding.[15] Literally hundreds of drugs can increase the risk of hemorrhage in patients taking warfarin. These include some nonprescription drugs widely perceived as innocuous.[16] Drug interactions are largely avoidable, yet can precipitate bleeding that is often unheralded and sometimes life-threatening. Among others, drugs that interact with warfarin and increase the risk of hemorrhage include antiplatelet agents, anti-inflammatory agents, acetaminophen, most antibiotics (however rifampin is associated with a decreased hemorrhage risk), antifungals, antidepressants, amiodarone, and many alternative remedies such as Gingko biloba.[16] Furthermore, patients should maintain regular dietary habits while on warfarin, and take precautions with vitamin K-rich foods, cranberry juice, and green tea.[17]

Warfarin is characterized by a narrow therapeutic window, a marked inter-individual variation in dose requirements, and excessive risk of adverse events with over- and underdosing.[18]

A recently published study estimated that over 175,000 emergency department visits for adverse drug events occur yearly among U.S. patients aged 65 and older.[19] This study also found that warfarin accounted for more of these visits, 17.3%, than any other drug. Of the adverse events attributed to warfarin, 73% involved clinically evident bleeding and hospitalization was required in 44.2%.[19]

Also of interest to surgeons is the fact that that operative blood loss transiently elevates post-operative INRs rather than affect long-term warfarin requirements.[18] Patients lose clotting factors with blood loss during surgery that are not replaced via transfusions of packed red blood cells. This loss temporarily inflates INR values and may explain why some postoperative patients are particularly sensitive to warfarin.[18]

Risk factors with warfarin use include a history of prior stroke or gastrointestinal bleeding, renal and hepatic disease, diabetes mellitus, uncontrolled hypertension, excessive alcohol use, noncompliance, and an age of 65 years or older.[17] Warfarin initiation carries a high risk of adverse events.[18] Consequently, it is far from being considered a benign form of therapy.

Non-pharmacological means of VTE prophylaxis

Patients who are at low risk for VTE (such as those who are ambulatory or undergoing a same-day procedure) or at high risk for bleeding (including those with severe renal impairment) are candidates for nonpharmacologic prophylaxis.[11] Mechanical methods of prophylaxis, which include graduated compression stockings (GCS), intermittent pneumatic compression (IPC) devices and venous foot pumps (VFP), increase venous outflow and/or reduce stasis within the leg veins.[6] When used in the surgical setting, mechanical devices should be placed on the patient before inducing anesthesia.[6] Their primary attraction is the lack of bleeding potential.[6]

Although all three of these mechanical methods have been shown to reduce DVT risk in a number of patient groups, they have been studied much less intensively than anticoagulant-based options and are generally less efficacious than anticoagulants for DVT prevention.[6]

Compression stockings have been shown to prevent DVT compared with placebo, but the effect is only modest and most studies enrolled only low-risk patients.[11] Also, these garments should be used with caution in patients with arterial insufficiency.[6] While intermittent pneumatic compression was shown to be effective in two studies on patients with head injuries, a number of other studies and a meta-analysis were unable to show any significant benefit in DVT reduction with IPC versus no prophylaxis.[6] Sequential compression devices must also be worn nearly 90% of the day to be effective.[11]

The efficacy of the VFP was challenged by a randomized clinical trial in which the rate of DVT was three times greater with these devices than with IPC, and by another study of 100 trauma patients with a 57% rate of venographically screened DVT despite prophylaxis with bilateral VFPs.[6] Of significance, no mechanical prophylaxis option has been shown to reduce the risk of death or PE.[6] Thus, the Seventh Chest Physicians Conference guidelines recommend that mechanical prophylaxis be used primarily in patients who are at high risk of bleeding or as an adjunct to anticoagulant-based prophylaxis.[6]

Trauma and Venous Thromboembolism

Trauma patients fall into the high-risk category for thromboembolic complications when they do not receive prophylaxis.[20] Geerts, et al, prospectively evaluated patients for VTE with objective diagnostic testing and found that 58% had lower extremity DVT after major trauma, including 69% of those with lower extremity orthopedic injuries.[21]

Thrombi were found in 80% of patients with femoral fractures, 77% with tibial fractures, 74% with ankle fractures, and 61% with pelvic fractures. VTE prophylaxis was not employed in this study. Older age, blood transfusion within the first 24 hours after admission, surgery, femur or tibia fracture, and spinal cord injury were all found to be independent risk factors for DVT. The Injury Severity Score was not associated with an increased thrombosis risk, suggesting that a specific type of injury plays a greater part in the genesis of venous thrombosis than does the severity of trauma. Only 1.5% of patients with DVT had clinical characteristics suggestive of thrombosis. Three patients died in this study due to pulmonary embolism. Not one of these patients had clinical features suggestive of VTE before their sudden deaths ranging from 15-18 days after the trauma. The authors noted that fatal PE in trauma patients may occur without warning, in the absence of clinical evidence of DVT and despite noninvasive screening.

Also of note, the incidence of DVT in this study was 46% in patients under the age of 30. The authors additionally found that even some of the fully mobile patients with single site injuries had extensive proximal DVT, and therefore consider all patients with major trauma to be at high risk for thromboembolic complications.

The Seventh Chest Physicians Conference guidelines recommend that all trauma patients with at least one risk factor for VTE receive thromboprophylaxis.[6] Geerts, et al, also note that although DVT risk increases with age, thromboprophylaxis should not be withheld simply because of young age. Also of interest, trauma patients with multiple injuries or lower limb fractures have a higher risk of VTE than those with single-system, nonorthopedic injuries.[6]

Lower limb fractures are especially vulnerable to DVT, and thrombi may occur in up to 61% of patients with pelvic fractures, 80% with femoral fractures, and 77% with tibial fractures.[8]

Abelseth, et al, found a high incidence of distal thrombosis in a study done to determine the incidence of venographically proven DVT in patients who had early operative fixation of long bone fractures distal to the hip.[2] The overall incidence of DVT was 28%, and all were clinically occult. Four inpatients had clinical evidence of PE. Forty-three percent of patients with tibial plateau fractures had DVT, versus 40% of those with femoral shaft fractures, 22% with tibial shaft fractures, and 12.5% of patients with tibial plafond fractures. Patients with fractures of the patella, foot, or ankle were excluded from the study. Three variables significantly predicted DVT occurrence and were thus referred to as risk factors: age greater than 60 years, operating time over 105 minutes, and time from injury to operation greater than 27 hours. The authors recommend that given the high incidence of DVT with femoral and tibial plateau fractures, lower-limb trauma in older patients, longer operating times, and high-energy injury, anticoagulation prophylaxis should be administered or these patients should be screened for DVT within two weeks of their injury.

VTE risk factors after isolated lower extremity injury include advanced age, fractures, and obesity.[6] It is not clear whether operative repair itself is a risk factor.[6] The risk of DVT appears to increase with the proximity of the fracture to the knee, such that tibial plateau fractures pose the highest risk, followed by tibial shaft fractures and then ankle fractures.[6] Interestingly, the risk of DVT after lower extremity tendon ruptures appears to be at least as high as that following lower extremity fracture.[6]

Patients with below-knee injuries have a 10 to 40 percent risk of asymptomatic DVT.[6] LMWH prophylaxis is considered a standard of care in some areas of Europe, and reduces the frequency of asymptomatic DVT, especially in those with tendon ruptures. However, the Seventh Chest Physicians Conference guidelines do not recommend routine thromboprophylaxis in isolated lower extremity injuries, since it is uncertain whether prophylaxis similarly reduces the risk of clinically important VTE, or is cost-effective.[6]

This particular Seventh Chest Physicians Conference recommendation guideline pertains to patients with fractures, ligament and cartilage injuries of the knee and ankle, and Achilles tendon ruptures.[6]

Hjelmstedt and Bergvall studied the incidence of thrombosis in 76 patients with 79 tibial shaft or metaphyseal fractures phlebographically or on autopsy. They found that thrombosis of varying extent occurred in 44.7%.[22] More widespread thrombosis with or without embolism, or minor thrombosis with embolism, occurred in 15.8%. These patients did not receive VTE prophylaxis. The authors concluded that this suggests prophylaxis should be administered to all patients with tibial fracture who are 39 years of age or older. Of note, however, there were no malleolar fractures included in this study.[22]

Immobilization and Venous Thromboembolism

Presently, there are no generally accepted recommendations for VTE prevention in patients requiring immobilization after leg injury.[23] However, multiple studies have shown that these patients are at increased risk for VTE. Kujath, et al, studied DVT in 253 outpatients with lower limb injuries who were immobilized in a plaster cast.[24] This was a prospective randomized study, and half received Fraxiparin injected subcutaneously daily until the cast was removed, and the other half received no thromboprophylaxis. DVT occurred in 16.5% of patients not given VTE prophylaxis compared to only 4.8% in the Fraxiparin group.[8,24] The diagnosis was made after an average of 17.1 days. Thrombosis was more common in patients with fractures than in those with ligamentous and other soft tissue injuries. Side effects such as hematoma, acute bleeding, and thrombocytopenia did not occur in these patients. The authors concluded that LMWH is recommended for all patients with injury of the lower limb being immobilized by a plaster cast, irrespective of age.[22]

Lassen, et al, evaluated the use of the LMWH or Reviparin in preventing DVT in 371 patients immobilized in a plaster cast or brace for at least five weeks after a leg fracture or Achilles tendon rupture.[23] DVT occurred in 9% of patients receiving Reviparin and 19% of those receiving placebo. The two cases of PE were both in the placebo group. There were no differences in adverse events between the two groups. They concluded that DVT is common in patients with leg injury requiring prolonged immobilization, and Reviparin appears to be safe and effective in reducing the risk of DVT.

Kock, et al, did a randomized prospective study of the effect of LMWH on the incidence of DVT in 339 patients with minor injuries immobilized in a plaster cast.[25] Patients in the prophylaxis group received daily subcutaneous injections of 32 mg of LMWH, while the control group received no prophylaxis. There was no incidence of DVT in the prophylaxis group and 4.3% in the control group. The mean duration of immobilization in the control group was 18.8 days, and DVTs were found after a mean immobilization of 11.4 days.

No severe side effects of LMWH were observed, and the authors concluded that LMWH administered once daily is effective in reducing the risk of DVT in outpatients with plaster-cast immobilization.

In a randomized, assessor-blinded, multicenter study, Jorgensen, et al, investigated the incidence of DVT in 205 patients immobilized in a plaster cast and the possible efficacy of prophylaxis with LMWH.[26] The treatment group received 3.5 IU anti-Xa of tinzaparin subcutaneously once daily, and the control group received no prophylaxis. The average patient age was 49 in the treatment group and 46 in the control group. Ascending venography was performed after the cast was removed at a mean of 5.5 weeks. DVT was found in 10 of 99 patients in the treatment group and 18 of 106 patients in the control group. This difference is not statistically significant. No bleeding complications occurred. The authors concluded that DVT after plaster casting is a problem, with an incidence of almost 20% in this young untreated population. They concluded that this prophylaxis regimen was not sufficient to prevent DVT.

Discussion

The vast majority of VTE prophylaxis literature discusses major orthopedic surgery. These prophylaxis recommendations have not been adopted in foot and ankle surgery, which seems appropriate given that there are several major differences between these procedures and those of the foot and ankle. First and foremost, the incidence of VTE events is much greater after major orthopedic surgery. The risk of postoperative DVT in the absence of prophylaxis is greatest in major orthopedic surgery, with knee surgery being 65% and hip surgery 50%, in comparison to neurosurgery (29%), general surgery (20%), gynecologic surgery (19%), and prostate surgery (11%.)[11] In contrast, the rate of postoperative DVT without prophylaxis in foot and ankle surgery was found by Solis and Saxby to be 3.5%.[10]

This incidence is similar to that of clinically overt VTE in major orthopedic surgery *with* the use of prophylaxis (nearly 3%).[5] Additionally, hip fracture surgery has a fatal PE rate of 5% without prophylaxis.[11] By comparison, the frequency of fatal PE after foot and ankle surgery appears to be less than 0.037%.[4]

Major orthopedic procedures are considered to be in the "very high" risk category, while the "very low" risk category involves "minor, same-day surgery."[11] Aggressive ambulation has been suggested as prophylaxis for the latter category, something that is not always possible in same-day foot and ankle surgeries.[11]

Perhaps another large difference between major orthopedic surgeries and those of the foot and ankle is the patient population itself. Major orthopedic surgery patients are said to already be at high baseline risk for VTE due to age and immobility.[3] Additionally, a significant number of these patients are greater than 70 years of age, a group for which there is a trend toward concurrent cardiovascular disease, a major contributor to VTE.[27]

One important similarity shared by major orthopedic surgery and foot and ankle surgery is that VTE may occur weeks or months postoperatively. Mizel, et al, found that clinically detectable symptoms of thromboembolism occur an average of 34.8 days after surgery in foot and ankle patients, with a range of 3-70 days.[4,8] Geerts, et al, state that the VTE risk after major orthopedic surgery continues to be higher than expected for at least two months after surgery.[6] Two definite peaks in DVT initiation have been demonstrated in patients undergoing total hip replacement.[28] The first is at postoperative day number four, and the second is at postoperative days thirteen and fourteen.[28] Wang, et al, noted that DVT often develops weeks after a surgical patient has been discharged, and that the danger of DVT or PE persists up to five or six weeks following surgery.[8]

They conclude that the use of extended prophylaxis postoperatively in the outpatient setting may also be beneficial for ankle surgery patients who present with additional VTE risks factors.[8]

Clearly, more information on VTE prophylaxis relative to foot and ankle surgery would be very helpful, as would prophylaxis recommendations for cast immobilization. There are no recommendations in the Seventh Chest Physicians Conference guidelines for foot and ankle surgery.[6] Furthermore, Geerts, et al, related being unable to generate evidence-based recommendations to help clinicians decide which patients with isolated lower extremity injury, if any, might benefit from prophylaxis, or the type, dose, or duration of prophylaxis.[6] They state that pending further data on these patients, clinicians may choose to provide no prophylaxis, in-hospital prophylaxis, or prophylaxis that is continued after hospital discharge.[6] Thus, particularly because VTE is such an important concern and one with potentially fatal consequences, the foot and ankle community would surely benefit from studies evaluating VTE prophylaxis in its cases, both elective and non-elective in nature, since a protocol is only as good as the evidence on which it is based.

References

1. Steier KJ, Singh G, Ullah A et al: Venous Thromboembolism: Application and Effectiveness of the American College of Chest Physicians 2001 Guidelines for Prophylaxis. J Am Osteopath Assoc 106: 388, 2006.
2. Abelseth G, Buckley RE, Pineo GE: Incidence of Deep-Vein Thrombosis in Patients with Fractures of the Lower Extremity Distal to the Hip. J Ortho Trauma 10: 230, 1996.
3. Schiff RL, Kahn SR, Shrier I et al: Identifying Orthopedic Patients at High Risk for Venous Thromboembolism Despite Thromboprophylaxis. Chest 128: 3364, 2005.
4. Mizel MS, Temple HT, Michelson JD et al: Thromboembolism After Foot and Ankle Surgery. Clinical Orthopaedics and Related Research 348: 180, 1998.
5. Agnelli G: Prevention of Venous Thromboembolism in Surgical Patients. Circulation 110: IV-4, 2004.
6. Geerts WH, Pineo GF, Heit JA et al: Prevention of Venous Thromboembolism: The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. Chest 126: 338, 2004.
7. Kearon C: Duration of Venous Thromboembolism Prophylaxis After Surgery. Chest 124: 386, 2003.
8. Wang F, Wera G, Knoblich GO et al: Pulmonary Embolism Following Operative Treatment of Ankle Fractures: A Report of Three Cases and Review of the Literature. Foot Ankle Int 23: 406, 2002.
9. Mismetti P, Laporte S, Zufferey P et al: Prevention of Venous Thromboembolism in Orthopedic Surgery with Vitamin K Antagonists: a Meta-analysis. Journal of Thrombosis and Haemostasis 2: 1058, 2004.
10. Solis G, Saxby T: Incidence of DVT Following Surgery of the Foot and Ankle. Foot Ankle Int 23: 411, 2002.
11. Michota FA: Preventing Venous Thromboembolism in Surgical Patients. Cleveland Clinic Journal of Medicine 73: S88, 2006.
12. Pulmonary Embolism Prevention (PEP) Trial Collaborative Group: Prevention of Pulmonary Embolism and Deep Vein Thrombosis with Low Dose Aspirin: Pulmonary Embolism Prevention (PEP) Trial. Lancet 355: 1295, 2000.
13. Strebel N, Prins M, Agnelli G et al: Preoperative or Postoperative Start of Prophylaxis for Venous Thromboembolism with Low-Molecular-Weight Heparin in Elective Hip Surgery? Arch Intern Med 162: 1451, 2002.
14. Begelman SM: Venous Thromboembolism. The Cleveland Clinic Disease Management Project. May 29, 2002.
15. Dahri K, Loewen P: The Risk of Bleeding with Warfarin: A Systematic Review and Performance Analysis of Clinical Prediction Rules. Thromb Haemost 98: 980, 2007.
16. Juurlink DN: Drug Interactions with Warfarin: What Clinicians Need to Know. Canadian Medical Association Journal 177: 369, 2007.
17. Dharmarajan L, Sajjad W: Potentially Lethal Acetaminophen-Warfarin Interaction in an Older Adult: An Under-recognized Phenomenon? JAMDA 545, October 2007.
18. Lenzini PA, Grice GR, Milligan PE et al: Optimal Initial Dose Adjustment of Warfarin in Orthopedic Patients. Ann Pharmacother 41: 1798, 2007.
19. Budnitz DS, Shehab N, Kegler SR et al: Medication Use Leading to Emergency Department Visits for Adverse Drug Events in Older Adults. Ann Intern Med 147: 755, 2007.
20. Harrington DT, Mozingo DW, Cancio L et al: Thermally Injured Patients Are at Significant Risk for Thromboembolic Complications. J Trauma 50: 495, 2001.
21. Geerts WH, Code KI, Jay RM et al: A Prospective Study of Venous Thromboembolism after Major Trauma. N Engl J Med 331: 1601, 1994.
22. Hjelmstedt A, Bergvall U: Incidence of Thrombosis in Patients with Tibial Fractures. Acta Chir Scand 134: 209, 1968.
23. Lassen MR, Borris LC et al: Use of the Low-Molecular-Weight Heparin Reviparin to Prevent Deep-Vein Thrombosis After Leg Injury Requiring Immobilization. N Engl J Med 347: 726, 2002.
24. Kujath P, Spannagel U et al: Incidence and Prophylaxis of Deep Venous Thrombosis in Outpatients with Injury of the Lower Limb. Haemostasis 23: (suppl 1) 20, 1993.
25. Kock HJ, Schmit-Neuerburg KP, Hanke J et al: Thromboprophylaxis with Low-Molecular-Weight Heparin in Outpatients with Plaster-Cast Immobilisation of the Leg. Lancet 346: 459, 1995.
26. Jorgensen PS, Warming T, Hansen K et al: Low Molecular Weight Heparin (Innohep) as Thromboprophylaxis in Outpatients with a Plaster Cast. Thrombosis Research 105: 477, 2002.
27. Enyart JJ, Jones RJ: Low-Dose Warfarin for Prevention of Symptomatic Thromboembolism After Orthopedic Surgery. Annals of Pharmacotherapy: 39: 1002, 2005.
28. Ennis RS: Deep Venous Thrombosis Prophylaxis in Orthopedic Surgery. eMedicine. February 7, 2005.