Complications in Foot and Ankle Surgery

Management Strategies

Michael S. Lee
Jordan P. Grossman
Editors

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Editors
Michael S. Lee
Capital Orthopedics and Sports Medicine
Clive, IA, USA

Jordan P. Grossman
Akron General Orthopedics, Inc.
Akron, OH, USA

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Foreword

Complication-

> "The unfavorable evolution or consequence of a disease, a health condition or a therapy."
>
> "An unanticipated problem that arises following, and is a result of, a procedure, treatment, or illness. A complication is so named because it complicates the situation."

Surgical procedures of the foot and ankle carry the potential for adverse events and complications. Dealing with the sequelae of complications and errors that arise in the course of normal practice is part and parcel of a surgeon's working life. It is crucial to focus on the needs of patients and their families when complications occur. Surgeons must respond to the challenge of providing effective patient care and may also need to deal with the reactions of the patient's family, the judgment of colleagues, and, in some cases, with disciplinary or legal action. Complications can keep us awake at night, undermine our confidence and ability to function, and even affect our enjoyment of life. The ability to manage complications is therefore paramount to what we do.

We have witnessed a stunning evolution in the advancement of foot and ankle surgery. The volume of research that has resulted in evidence-based medicine, as well as technological advancements in our field, has been remarkable. Furthermore, the dissemination of information through all sorts of learning vehicles has exploded. This information is at our fingertips and can be easily accessed. We have huge volumes of information to assist and guide us in our decision-making. Surgeons can develop a plan for virtually any procedure with precision and accuracy. However, there is often a paucity of information when it comes to dealing with complications of foot and ankle surgery. It can be difficult to find information to help us deliver effective patient care in these situations. Obtaining information to help us deal with complications often requires cumbersome searches that provide little guidance. This textbook on complications of foot and ankle surgery is the first of its kind and is long overdue. It will serve as a resource to help us properly evaluate and institute appropriate therapy for complications.

This textbook by Michael Lee and Jordan Grossmann is a great starting point for those of us looking to deal with complications in a practical and effective manner. The text provides insight on how complications develop and evolve. It discusses major perioperative complications such as deep venous thrombosis, nonunions, incisional problems, surgical infections, and adverse effects associated with comorbid conditions. The text also reviews complications following specific surgical procedures. The authors in each section discuss evaluation and management of specific complications, as well as potential pitfalls with each procedure. Drs. Lee and Grossman have gathered a diverse group of authors who share their experience and techniques.

I am honored and privileged to provide the foreword for this complete, comprehensive and timely textbook on complications of foot and ankle surgery. I commend Drs. Lee and Grossman, recognized leaders in our profession, as well as my former residents and dear friends, for their dedication to this project. This will become an authoritative textbook on complications. Thanks for making me proud!

We must dedicate ourselves to deliver responsible, patient-centered care in a safe environment that includes equitable treatment and full disclosure to all patients. We must address surgical complications by providing effective, evidence-based patient care. These principles represent the way forward in our specialty and will define how well we are able to truly advance our profession. As such, it is imperative that we develop the requisite skills necessary to deal with complications. This textbook is a great STARTING point.

Alan Catanzariti

Acknowledgments

We would like to thank our *friends* and colleagues for their time, dedication, and efforts toward the completion of this book. This list of contributors is representative of the hardworking and thoughtful portion of our profession that have and will continue to advance the field of foot and ankle surgery. Completing a chapter is a daunting task as there are always other projects and expectations that manage to consume our day. We are grateful and appreciate these authors for finding the time to complete their respective chapters.

To that end, and equally important, we'd like to thank our own families for allowing us to find that precious time to dedicate to this worthy project. Your love and support has never wavered, and for that we are thankful. Finally, thanks to Alan Catanzariti, for his mentoring and friendship of both of us. Cats' dedication to the profession, his patients, his residents, and his family is an example for everyone—we are forever thankful and incredibly proud to be products of his West Penn family.

Contents

Contributors

Eric A. Barp, DPM, FACFAS Foot and Ankle Surgery, The Iowa Clinic, West Des Moines, IA, USA

Michelle Butterworth, DPM Pee Dee Foot Center, Kingstree, SC, USA

Glenn Weinraub Department of Orthopedic Surgery, Kaiser Permanente, San Leandro, CA, USA

Alan Catanzariti, DPM, FACFAS West Penn Hospital, The Foot and Ankle Institute, Pittsburgh, PA, USA

Gage M. Caudell Fort Wayne Orthopedics, Fort Wayne, IN, USA

Thomas J. Chang, DPM Redwood Orthopedic Surgery Associates, Inc., Santa Rosa, CA, USA

Luke D. Cicchinelli, DPM Centro Clinico Quirurgico, Vigo, Spain

J. Randolph Clements, DPM Carilion Clinic Orthopaedics, Virginia Tech Carilion School of Medicine, Roanoke, VA, USA

Richard Derner, DPM, FACFAS Past President, American College of Foot and Ankle Surgeons, Department of Orthopedics INOVA Fairfax Medical Campus, Falls Church, VA, USA

Brian T. Dix Sanford Health, Thief River Falls, MN, USA

Lawrence A. Di Domenico, DPM Ankle and Foot Care Centers, Youngstown, OH, USA

Kent State University, College of Podiatric Medicine, Cleveland, OH, USA

Mindi Feilmeier, DPM School- Des Moines University, College of Podiatric Medicine and Surgery, Des Moines, IA, USA

Justin J. Fleming, DPM Aria 3B Orthopaedics, Philadelphia, PA, USA

Lawrence A. Ford, DPM, FACFAS Department of Orthopedics and Podiatric Surgery, Kaiser Permanente Medical Center, Oakland, CA, USA

Kaiser San Francisco Bay Area Foot and Ankle Residency Program, Oakland, CA, USA

Michael Gentile, DPM Westside Foot and Ankle Specialists,Tigard, OR

Sean T. Grambart, DPM, FACFAS Carle Physician Group, Champaign, IL, USA

David Granger OSS Health, York, PA, USA

Graham A. Hamilton, DPM, FACFAS Department of Orthopedics and Podiatric Surgery, Kaiser Permanente Medical Center, Antioch, CA, USA

Kaiser San Francisco Bay Area Foot and Ankle Residency Program, Oakland, CA, USA

Matthew J. Hentges, DPM The Foot and Ankle Institute of Western Pennsylvania, Pittsburgh, PA, USA

Mark H. Hofbauer, DPM Monongahela Valley Hospital, Monongahela, PA, USA

Byron Hutchinson, DPM Department of Ortho, CHI/Fransiscan Health System, Spine and Foot and Ankle, Saint Francis Hospital, Federal Way, WA, USA

Christopher F. Hyer, DPM, MS, FACFAS Orthopedic Foot and Ankle Center, Westerville, OH, USA

Allen M. Jacobs, DPM St. Louis, MO, USA

Meagan M. Jennings, DPM Orthopedics and Podiatry, Palo Alto Foundation Medical Group, Mountain View, CA, USA

Erin E. Klein, DPM, MS Department of Surgery, Weil Foot and Ankle Institute, Des Plaines, IL, USA

Kwasi Y. Kwaadu, DPM Temple University School of Podiatric Medicine, Philadelphia, PA, USA

Bradley M. Lamm, DPM Foot and Ankle Deformity Center, Paley Orthopedic and Spine Institute, St. Mary's Medical Center & Palm Beach Children's Hospital, West Palm Beach, FL, USA

Guido LaPorta, DPM, FACFAS LaPorta and Associates, PC, Dunmore, PA, USA

George Tye Liu, DPM Department of Orthopaedic Surgery, University of Texas Southwestern Medical Center, Dallas, TX, USA

Frank A. Luckino Ankle and Foot Care Centers, Youngstown, OH , USA

Chrisitne P. Miller, DPM, DMM Department of Orthopaedics-Division of Foot and Ankle, University of Florida College of Medicine-Jacksonville, Jacksonville, FL, USA

Kyle Moore, DPM West Penn Hospital, The Foot and Ankle Institute, Pittsburgh, PA, USA

Trevor Payne, DPM Augusta Foot and Ankle, Evans, GA, USA

Ellianne M. Nasser, DPM Geisinger Health System, Scranton, PA, USA

Kyle S. Peterson, DPM Suburban Orthopaedics, Bartlett, IL, USA

Jason A. Piraino, DPM, MS, FACFAS Department of Orthopaedics-Division of Foot and Ankle, University of Florida College of Medicine-Jacksonville, Jacksonville, FL, USA

Rachael Poore Podiatric Medicine and Surgery, Unity Point Health-Des Moines, Des Moines, IA, USA

Christopher L. Reeves, DPM Orlando Foot and Ankle Clinic, Teaching Faculty, Florida Hospital East Orlando Residency Program, Orlando, FL, USA

Laurence G. Rubin, DPM, FACFAS Mechanicsville, VA, USA

D. Scot Malay, DPM, MSCE, FACFAS Director Podiatric Education and Staff Surgeon, Penn Presbyterian Medical Center, Philadelphia, PA, USA

Amber M. Shane, DPM, FACFAS Orlando Foot and Ankle Clinic, Teaching Faculty, Florida Hospital East Orlando Residency Program, Orlando, FL, USA

Naohiro Shibuya, DPM, MS, FACFAS Texas A&M University, Health Science Center, College of Medicine, Temple, TX, USA

Surgical Services, Central Texas VA Health Care System, Veterans Hospital, Temple, TX, USA

Department of Surgery, Baylor Scott and White Health Care System, Temple, TX, USA

Alan Sue Orthopedics and Podiatry, Palo Alto Foundation Medical Group, Mountain View, CA, USA

James Thomas, DPM The Orthopaedic Clinic, Auburn, AL, USA

Joshua D. Thun Monongahela Valley Hospital, Monongahela, PA, USA

Nicholas Todd, DPM Orthopedics and Podiatry, Palo Alto Foundation Medical Group, Mountain View, CA, USA

Lowell S. Weil, DPM Department of Surgery, Weil Foot and Ankle Institute, Des Plaines, IL, USA

Collin M. Zdenek, DPM Mountain View Center, Palo Alto Medcal Foundation, Mountain View, CA, USA

"Anatomy" of Complications

1

Allen M. Jacobs

Introduction

Complications are an inevitable occurrence in the performance of foot and ankle surgery. With reference to surgery, a variety of definitions have been offered to define the term "complication."

Sokol and Wilson [1] defined a complication as "any undesirable, unintended, and direct result of surgery affecting the patient which would not had occurred had the surgery gone as well as could reasonably be hoped." Classification systems for evaluation of foot and ankle surgical clinical outcomes, such as those advocated by the American College of Foot and Ankle Surgeons [2] or the American Orthopedic Foot and Ankle Society [3], examine postoperative results in terms of function, overall health of the patient, quality of life, and activity scores. Sink et al. [4] noted that no standard exists for reporting complication rates, and as a result, reported outcomes of surgical procedures such as surgery are incomplete without a standardized, objective, complication grading scheme applied concurrently with reporting outcome studies. As a result, the lack of an established basis for grading and reporting complications renders comparisons between studies ineffective. As an example, the impact of a nonunion following attempted arthrodesis of an interdigital joint for hammertoe surgery is considerably different from the impact of a nonunion following ankle joint arthrodesis.

Generally, clinical outcome studies describe complications in subjective terms, such as mild, moderate, or severe, without a standardized classification systems or consistency in definition.

A.M. Jacobs, DPM & Associates (✉)
6400 Clayton Road, Suite 402, St. Louis, MO 63117, USA
e-mail: drjacobs0902@sbcglobal.net

Definitions

Surgical complications should be differentiated from other postoperative occurrences such as failure of treatment (i.e., failure to cure), operative sequela, or surgical error. As an example, a patient continuing to demonstrate pain or limited range of motion following a cheilectomy or implant arthroplasty of the great toe performed for hallux rigidus is a failure to cure, or a procedure cure, but is not a complication. A failure of the operative intervention to provide a satisfactory outcome is per se not a complication, but rather a failure of the surgical procedure to have produced the desired outcome. Conversely, nonunion of a great toe arthrodesis for the correction of a hallux rigidus deformity is a complication and not a "failure to cure."

Postoperative sequelae are known, generally predictable, possible consequences of surgery. For example, the development of osteoarthritis of the ankle following subtalar joint fusion or triple arthrodesis is a known potential sequela of the arthrodesis, and is not a complication. The requirement for hardware removal, following ORIF of an ankle fracture, is a known potential sequela of the surgical procedure.

Surgical errors, or mistakes, may occur even when care is rendered by an outstanding foot or ankle surgeon. Surgical error may be associated with medical negligence in some circumstances. A mistake is negligence only when a reasonably careful practitioner would not have made the same mistake under similar circumstances [5]. A relationship may exist between complications and surgical error. In a review of 9830 surgical procedures, a complication rate of 3.4% was noted which included an identifiable error in 78.3% of those patients incurring a complication. The errors included most commonly error in surgical technique, judgment error, and attention to detail, or an incomplete understanding of the patient's clinical status [6].

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Complication Classification Systems

At the present time, there is no generally recognized classification system for complications following foot and ankle surgery. Complications are accepted in the surgical literature as an important outcome measure and useful indicator for measuring quality. Efforts have been made to classify complications, such as general surgical complications, following abdominal surgery, as well as some orthopedic surgical procedures.

With reference to general surgery, one of the most commonly employed classification systems is that of Clavien-Dindo [7]. A modification of this classification system has been described for some orthopedic interventions [4], gastroenterology surgery [8], urologic surgery [9], and nephrology [10].

Presently, there are no accepted classification systems or postoperative complications subsequent to foot and ankle surgery. The lack of such classification systems results in unreliability when comparing clinical outcomes reported for the same or comparative procedures. For example, nonunion following first MPJ arthrodesis, or following first metatarsal-cuneiform arthrodesis, is frequently associated with little or no symptomatology. The nonunion rate reported for the Lapidus procedure may be greater than the nonunion rate for alternative surgical interventions, such as the Austin or scarf procedures utilized in the correction of hallux valgus and bunion deformity. However, the clinical impact of a nonunion of a procedure other than the Lapidus employed as an alternate procedure may be of greater impact on quality of life and may be associated with a greater need for operative intervention.

Table 1.1 contains a proposed adaptation of the Clavien-Dindo system for the classification of complications following foot and ankle surgery.

Table 1.1 Classification of complications following foot and ankle surgery (Jacobs-Babette)

Grade I
Any deviation from the normal postoperative course without the need for additional medical therapy, physical therapy, or surgical interventions. Such complications require no treatment and have no significant clinical relevance *Examples:* • Nonsymptomatic persistent edema following digital surgery • Nonpainful hypertrophic scar formation • Nonsymptomatic limited motion following bunion surgery
Grade II
A deviation from the normal postoperative course requiring unplanned physical therapy, or pharmacologic therapy *Examples:* • Symptomatic limited motion following total ankle joint replacement requiring physical therapy • Postoperative infection requiring antibiotic therapy • Transfer metatarsalgia requiring orthotic or shoe therapy
Grade III
A deviation from the normal postoperative course requiring surgical intervention *Examples:* • Postoperative infection requiring incision and drainage • Osteotomy malalignment requiring revision • Revision of a symptomatic arthrodesis or osteotomy nonunion • Symptomatic nerve entrapment requiring operative intervention A. Not requiring general anesthesia B. Requiring general anesthesia
Grade IV
A deviation from the normal postoperative course which is limb threatening *Examples:* • Necrotizing fascial infection requiring extensive incision and debridement • Acute vascular compromise to the foot or leg • Compartment syndrome requiring fasciotomies
Grade V
A deviation from the normal postoperative course which is threatening to quality of life *Examples:* • Postoperative complex regional pain syndrome • Significant limb shortening following total ankle joint infection and revision
Grade VI
A deviation from the normal postoperative course which is potentially life-threatening *Examples:* • DVT ± pulmonary embolism • Malignant hyperthermia • Organ failure (e.g., renal failure, cardiac failure)

Complication and the Surgical Patient

A ubiquity of complications may occur in association with foot and ankle surgery. There are virtually no surgical procedures without the potential for complication. Ingrown toenail correction, for example, can be associated with persistent pain or swelling, chemical burns, contact allergy, primary irritant sensitivity, infection, regrowth of the nail, spiculization, or acosmetically displeasing result.

Not infrequently, complications may result in a request for medical records by the patient and result in an allegation of malpractice. It is imperative that all patients have a clear understanding of the potential for complications to occur even when the best care has been rendered.

In some cases, the evaluation and treatment of complications following surgery will result in significant unanticipated inconvenience to the patient and considerable increased cost associated with care, again, unanticipated by the patient. These factors are superimposed on what may be a less than optimal outcome than had been perceived by the patient.

It is important that the surgeon appropriately recognize complications and appropriately treat such complications. It is equally important that the surgeon communicate with the patient regarding the nature of the complication and potential impact of the complication and express to the patient an

understanding of the effects of additional delay in healing and financial burdens.

An acknowledgment of the complication should be made by the surgeon to the patient/family. Studies have demonstrated that a patient is more likely to seek medical-legal action against a provider if that physician does not disclose the complication and effectively communicate this information to the patient [11]. It should always be recalled that in most instances, a complication does not imply negligent care. However, when a complication does occur, the question of negligence arises when such complications are not recognized in a timely manner and treated in an appropriate manner.

In the case of surgical error or complication, an acknowledgment of the patient's emotions and increased physical as well as financial burden is helpful in maintaining the ability to assist the patient in resolving such complications.

In discussing the etiology of malpractice litigation, Nisselle discussed three major factors: poor patient rapport, unmet expectations, and a "big bill" [12]. Similarly, in a review of those factors leading patients to sue their doctors, insensitive handling of the incident, poor communication following the incident, and a less than satisfactory explanation are the most commonly cited factors [13]. Studies have demonstrated that a problematic doctor-patient relationship, a sense that the surgeon was abandoning the patient in the face of a complication, a devaluation of the patient and family views, a poor informational delivery, and a failure of the surgeon to understand the perspective of the patient and family are recognized as causes a patient with complications to seek litigation [14]. It is critical that faced with a complication, the surgeon communicate with and work closely with the patient.

Complications and Surgical Consent Forms

Because complications, failure to achieve the desired surgical outcome, or known common sequela may occur following any surgical procedure, the process of informed consent is required for surgical procedures.

The process of informed consent is more than obtaining a signature on a routine consent form. It is a process of communication between the patient and surgeon.

Informed consent should include a verbal or written explanation to the patient of the most common possible complications and sequela of surgical procedures. Generally, with reference to complications, surgeons rely on a standard consent form, frequently not unique or specific to the surgical procedure to be performed.

The American Medical Association has noted that forms which serve mainly to satisfy all legal requirements (e.g., "all material risks have been explained to me") may not preclude a patient from asserting that the actual disclosure did not include risks that the patient unfortunately discovered after treatment. Consequently, such consent forms may not prove to be sufficient in a court of law.

In discussing the potential for complications, the severity of potential complications relative to each patient should be considered. For example, osteomyelitis as a complication of great toe implant arthroplasty may have significantly greater impact on a competition ballroom dancer than an individual who is sedentary. Another common example is the patient who is a runner, or otherwise very athletically active, in whom the complication of avascular necrosis following bunionectomy could be much more significant than in a less athletic individual. In discussing complications, reasonable information should be provided to any patient given their individual circumstances. With regard to complications, informed consent disclosure should be tailored to the patient's individual situation thus requiring a combination of good judgment and communication skills.

The likelihood of complications occurring should also be discussed with the patient. For example, a significant nonunion rate is associated with ankle arthrodesis in the patient who smokes. Similarly, elective surgery performed in the diabetic patient, particularly in the presence of neuropathy, vascular disease, or elevated hemoglobin A1c, is associated with a much greater risk of complications. Therefore, the need for patient compliance in overall healthcare as well has postoperative care to reduce the incidence of complications is important to discuss with the patient. Such discussions ideally are documented in the patient's chart. For some procedures, such as tarsal tunnel surgery, excision of a Morton's neuroma, Lapidus procedure, Austin bunionectomy, and ankle arthrodesis, complication rates such as delayed or nonunion are established and should be related to the patient.

Good surgical judgment, together with an integration of patient beliefs and values, is critical in avoiding complications or perceived complications. The specific goals for any surgical procedure should be agreed upon by the patient, family, and surgeon. For example, reduction of a deformity associated with recurring neuropathic ulceration may not require as a goal of the surgery a perfectly functioning or cosmetic result, but rather elimination of pressure. Surgical management of a tarsal coalition may be directed at relief of pain as well as increased ambulatory tolerance, but may not include as a goal restoration of the "normal arch." The concept of limited results, and not a perfect end result, should be accepted by the patient.

Reducing the Risk of Complications

There is no certain manner with which the possibility of complications may be eliminated. Conversely, there are both surgeon and patient factors which can increase the likelihood

of a complication associated with surgical intervention. The surgeon should be competent and knowledgeable with procedure to be performed. It is important that the surgeon recognize the limits of his or her professional competence and make appropriate referrals when necessary. The surgeon must not overestimate his or her ability and demonstrate some humility being aware of their individual strengths and weaknesses. Cases should be referred to a colleague when these are beyond his or her capability.

Prior to surgery, the surgeon should ensure that all needed equipment is present and functioning and that the surgeon is familiar with the use of the equipment. This is particularly important in the utilization of new fixation devices which, when mal-applied, can result in an increased risk of complications.

It is important that the surgeon continue to update their individual knowledge to allow patients to benefit from new surgical techniques with improved outcomes and lower complication rates. Thus, continuing professional development and maintenance of surgical competence are important factors in reducing the risks of surgical complications.

Complications and the Noncompliant Patient

Noncompliance is one of the most common causes for treatment failure. It is largely unrecognized, as patients infrequently volunteer that they had failed to comply with the directions of the surgeon. The effects of noncompliance range from trivial to catastrophic and include a variety of behaviors such as failure to take medications prescribed, failure to offload or remain non-weight-bearing, and failure to interdict smoking.

Noncompliance is a common occurrence in the management of the medical/surgical patient. For example, Jowett et al. have demonstrated that noncompliance with bracing is the most common cause of clubfoot relapse following initial correction [15]. Other common examples are the failure of a patient to utilize prophylactic compression stockings to prevent DVT [16] or failure of patient to perform prescribed home physical therapy for the treatment of Achilles tendinosis [17].

Because noncompliance is so common and may result in significant postoperative complications, instructions given to patients for postoperative care should be included in the medical record. The opportunities to do so include the progress notes, the operative note, and the discharge summary. In addition, discharge instructions to the patient should include specifics regarding weight-bearing status, activity level, and other significant factors such as medication use. Compliance with postoperative orders, or the failure to comply with postoperative orders, should be included in follow-up notes. The surgeon should ask about compliance, document the potential effects of noncompliance, and alert the patient to the increased risks of complications as a result of their noncompliance. The patient should be reinstructed regarding their postoperative care when necessary.

When confronted with noncompliance, the surgeon should consider whether the patient understands their problem. An attempt should be made to clarify to the patient the purpose of treatment and have the patient understand the potential effects and consequences of continued therapy noncompliance.

Smoking and Surgical Complications

It is well established that smoking is associated with increased risks of problematic soft tissue healing, increased risk of postoperative infection, increased risk of delayed union and nonunion, and increased risks of thrombosis, platelet adhesions, vasoconstriction, and ischemia [18, 19]. Smoking cessation should be discussed with the patient, and a discussion should be documented. The potential adverse effects of smoking in the perioperative period should also be discussed and documented. Not infrequently, patients demonstrate poor recall of preoperative complication risk discussion and therefore the need for good documentation. In one particular study of foot surgery patients, 11 potential complications were discussed. At which patient recall ranged from zero to a maximum of four complications was discussed. [20]

Summary

Complications may occur following any surgical procedure. Most often, complications are not the result of medical negligence. Complications should be discriminated from failure to cure, from medical error, and from sequela that may occur following any foot or ankle surgical procedure.

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Part I

Perioperative Complications

Venous Thromboembolism Associated with Foot and Ankle Surgery

2

D. Scot Malay

A vast array of information is available to surgeons to aid in the prevention and management of venous thromboembolism (VTE) [1–4], and readers are encouraged to use these and other sources and to stay abreast of the ever-changing body of knowledge related to these conditions. VTE includes deep vein thrombophlebitis (DVT) and pulmonary embolism (PE), and an appreciation of the clotting cascade can serve as a foundation for understanding clot formation and its prevention and treatment (Fig. 2.1). It is also important for foot and ankle surgeons to maintain a high index of suspicion for VTE, since the condition is prevalent and potentially deadly, and even when it is identified and properly treated, the sequelae of post-thrombotic syndrome (PTS) (Fig. 2.2a–d), which occurs in approximately 30% of VTE patients [5], and chronic pulmonary thromboembolic hypertension (CPTH), which occurs in approximately 4% of PE patients [6], are debilitating. PTS is associated with profound lower extremity edema, chronic stasis dermatitis, and cutaneous ulceration in 5–10% patients that suffer with lower extremity DVT. Still further, it has been estimated that 45,000–75,000 patients in the United States die annually as a result of PE [7], whereas <10% of patients with PE die if timely treatment is administered [8].

VTE develops in response to a mixture of acquired and hereditary exposures that promote hypercoagulability or thrombophilia. Virchow's classic triad of venous stasis, damage to the vein wall, and activation of the clotting cascade serve as the foundation for venous thrombosis. Clots that develop in veins are composed primarily of red and white blood cells combined with platelets and fibrin and are particularly prone to localize in the stagnant blood in the perivalvular segments of the veins of the lower extremity. Venous thrombi that remain fixed in the calf or thigh veins eventually undergo thrombolysis and recanalization, whereas those that break free can migrate with the return of venous blood to the pulmonary arteries where they occlude blood flow to the lungs. Unfortunately, VTE can be difficult to identify, and it can be recurrent. Almost 90% of VTE occur in the lower extremities, and the more proximal the site of VTE in the lower extremity, the greater is the risk of PE. In fact, VTE occurring in the femoral or popliteal veins is associated with a 50% risk of PE if left untreated, whereas the risk of PE is approximately 20–25% for VTE localized to the calf, and, overall, about 15–30% of lower extremity VTE result in PE [9]. PE, in turn, demands compensatory right ventricle inotropism in order to force the blood through the occluded pulmonary artery, which leads to pulmonary artery hypertension and subsequent right heart failure, especially in patients with preexisting cardiac and/or lung disease.

The prevalence of VTE has been estimated to be approximately one million cases per year in the United States [10], and approximately 67% of these cases occur in association with hospitalization, and about half of these patients die as a result of the disease [11]. Risk factors for VTE are present in many hospitalized patients and include comorbidities such as diabetes mellitus, hypertension, hypercholesterolemia, and cigarette smoking, as well as infection, cancer, age >75 years, obesity, and a history of previous VTE [12, 13], surgery, or trauma (Table 2.1) [14]. Following acute myocardial infarction and cerebral vascular accident, VTE is the most common cardiovascular disease [15]. Hereditary conditions, such as protein C and S and antithrombin deficiencies, factor V Leiden, and prothrombin gene mutation, also increase the likelihood of developing VTE.

Diagnosis of Venous Thromboembolism

Clinical Examination

The diagnosis of DVT and/or PE can often be made, or at least strongly suggested, based on the results of the historical review and clinical examination, and combinations of

D. Scot Malay (✉)
Director Podiatric Education and Staff Surgeon, Penn Presbyterian Medical Center, Philadelphia, PA, USA
e-mail: editorjfas@gmail.com

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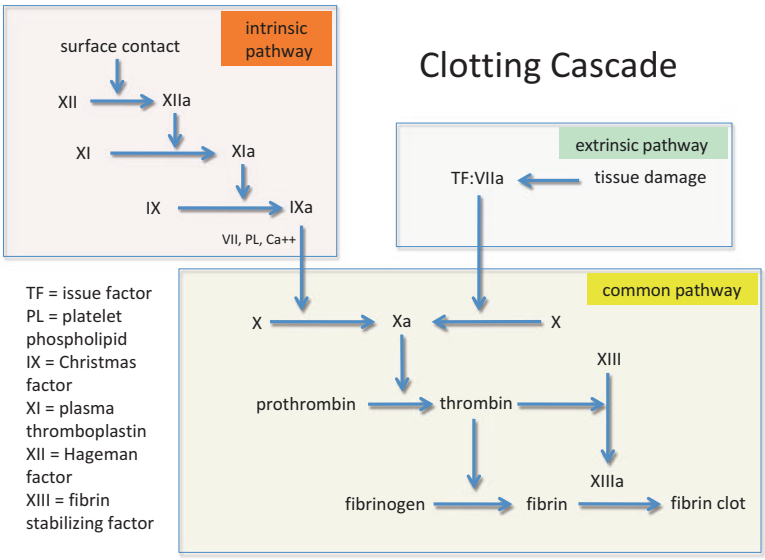

Fig. 2.1 The clotting cascade

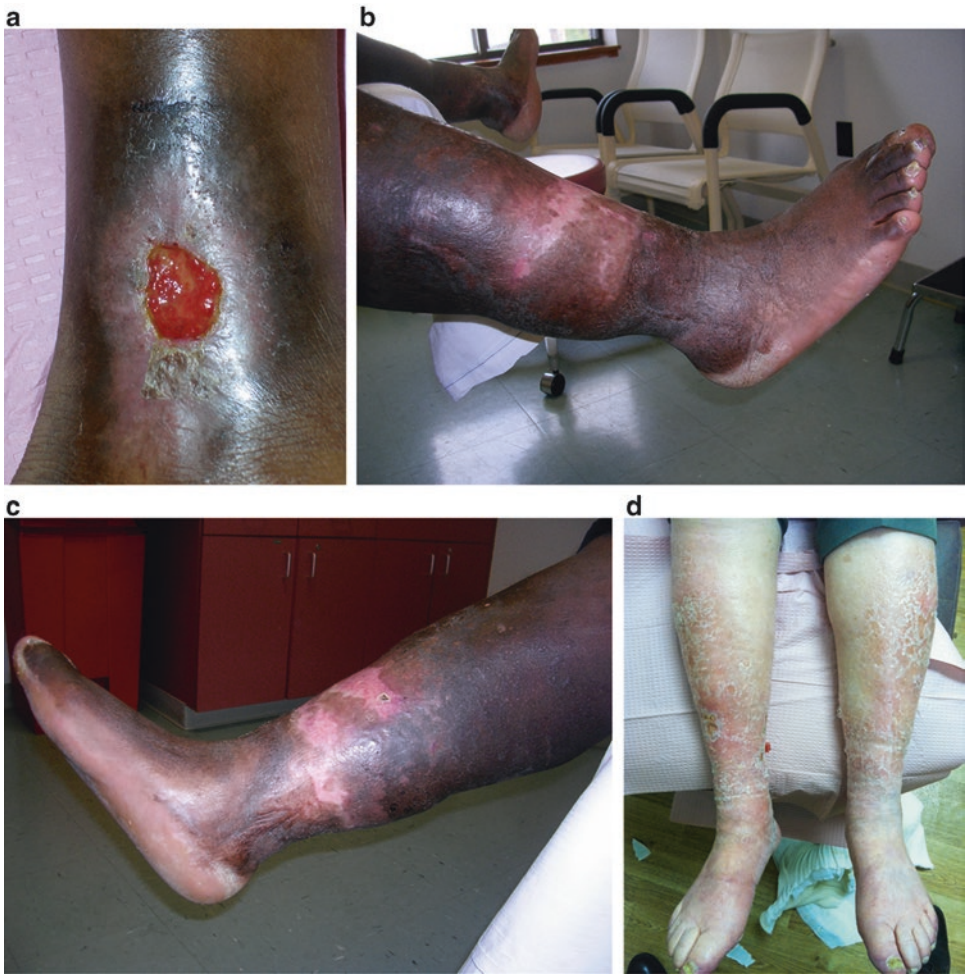

Fig. 2.2 (**a**) Chronic right lateral lesser saphenous venous stasis ulcer in patient with post-thrombotic syndrome. (**b**) Chronic post-thrombotic stasis dermatitis and scarification right lower extremity, lateral view. (**c**) Chronic post-thrombotic stasis dermatitis and scarification right lower extremity, medial view (same patient as viewed in **b**). (**d**) Bilateral postphlebitic stasis dermatitis with post thrombotic syndrome

diagnostic criteria have been shown to be more or less suggestive of DVT (Table 2.2) [14, 16]. Clinically, DVT of the lower extremities is commonly associated with pain localized to, and swelling in the extremity distal to, the site of the thrombus. The involved extremity can also be warm, with cutaneous erythema, and regional varicosities may be evident. Homan's sign, which is pain in the calf upon dorsiflexion of the ankle, is commonly thought to be evidence of

Table 2.1 Risk factors for venous thromboembolism

Inherited risk factors	Antithrombin deficiency
	Dysfibrinogenemia
	Elevated levels of factor VIII
	Factor V Leiden mutation
	Hyperhomocysteinemia
	Protein C or S deficiency
	Prothrombin gene mutation
Acquired risk factors	Air travel
	Antiphospholipid syndrome
	Body mass index >30
	Cancer or certain cancer treatments
	Cardiovascular risk factors (smoking, hypertension, hyperlipidemia, diabetes mellitus)
	Heparin-induced thrombocytopenia
	Immobilization[a]
	Indwelling central venous catheter or pacemaker
	Inflammatory bowel disease
	Medical illness (heart failure, chronic obstructive pulmonary disease)
	Myeloproliferative disorder
	Pregnancy, oral contraceptive use, hormone replacement therapy
	Presence of an inferior vena cava filter
	Previous episode of venous thromboembolism
	Surgery[a]
	Trauma[a]

[a]Acquired risk factors related to most, if not all, foot and ankle surgical patients

Table 2.2 The Wells [14, 16] diagnostic criteria suggestive of deep vein thrombosis[a]

Risk factor criteria	Points
Active cancer	1
Recently bedridden >3 days or major surgery within 4 weeks	1
Calf swelling >3 cm compared to contralateral calf measured 10 cm distal to tibial tuberosity	1
Presence of collateral non-varicose superficial veins	1
Entire ipsilateral lower extremity (leg) swollen	1
Ipsilateral tenderness localized to deep venous system	1
Pitting edema greater in the symptomatic lower extremity	1
Paralysis, paresis, or recent immobilization of symptomatic lower extremity	1
History of previously documented DVT	1
Alternative diagnosis to DVT as or more likely	-2

[a]Interpretation: A score ≥ 2 indicates that the probability of DVT is likely, whereas a score < 2 indicates that probability of DVT is unlikely

calf DVT; however, this has been shown to be an unreliable assessment for calf DVT [17]. Multiple thrombosed deep and collateral veins in an extremity can result in a severely edematous, inflamed extremity known as phlegmasia cerulea dolens, which can be limb threatening due to ischemia, and may be associated with prothrombotic disorders such as heparin-induced thrombocytopenia (HIT), myeloproliferative disease, factor V Leiden mutation, polycythemia vera, and paroxysmal nocturnal hemoglobinuria (PNH). As for PE, signs and symptoms include chest pain; tachypnea and dyspnea, along with a sense of impending doom; tachycardia; hyperpyrexia; cough; hemoptysis; syncope; and, frequently, evidence of an associated DVT. Unfortunately, DVT can be clinically silent until the clinical signs of PE become evident.

Diagnostic Laboratory Tests for Venous Thromboembolism

Coagulation Tests

The *partial thromboplastin time* (PTT) is a reliable coagulation screening test, although it may not be sensitive enough to detect subtle coagulopathies. The PTT is also used to monitor heparin anticoagulation therapy, although it is not suitable for monitoring factor VII or platelet factors. The normal range for the PTT is 25–35 s, and it remains normal in von Willebrand's disease, platelet dysfunction, and thrombocytopenia. The PTT is prolonged by defects in clotting factors I, II, V, VIII, IX, X, XI, and XII. The *prothrombin time* (PT) can be used to monitor long-term warfarin anticoagulation therapy. The normal range for the PT is 11–16 s, and it is prolonged with defects in factors I, II, V, VII, and X, as well as in vitamin K deficiency, fat malabsorption (steatorrhea, colitis, jaundice), salicylate or warfarin therapy, and advanced hepatic disease. The *bleeding time* is a simple clinical examination used to assess the overall ability to stop bleeding following a cutaneous prick, and it is particularly sensitive to platelet defects. The normal range (Duke) for the bleeding time is 1–4 minutes, and it is prolonged in thrombocytopenia, abnormal platelet function, and von Willebrand's disease. The *clotting time* is a nonspecific, in vitro screening test used to determine the presence of a major clotting deficiency. The normal range (Lee-White) clotting time is 3–6 min in a capillary tube and 6–17 min in a test tube. The *international normalized ratio* (INR) is a test that was established by the World Health Organization (WHO) and the International Committee on Thrombosis and Hemostasis so that the results of blood clotting tests could be reported by any lab by virtue of the fact that all of the results are standardized with the international sensitivity index for the particular thromboplastin reagent and instrument combination used to perform the test. For warfarin, the usual optimal therapeutic prothrombin time is INR = 2–3.

Platelet Count

The normal range for the *platelet count* is 140,000–340,000/mm^3, and platelets are commonly diminished in pregnancy, leukemia, myelodysplasia, hepatic cirrhosis, aplastic anemia, iron deficiency, vitamin B12 deficiency, HIV/AIDS, Epstein-Barr virus infection, chicken pox, and various toxicities (chemotherapy, alcohol, radiation, other chemicals),

hypersplenism, autoimmune diseases, septicemia, idiopathic or thrombotic thrombocytopenic purpura, hemolytic uremia, and disseminated intravascular coagulation. Thrombocytosis, which can predispose to VTE, can be caused by acute hemorrhage and blood loss, surgery, trauma, burn wounds, allergic reactions, cancer, chronic kidney disease, exercise, myocardial infarction, coronary artery bypass, infection, iron deficiency, vitamin deficiency, splenectomy, hemolysis, systemic inflammatory disease (rheumatoid arthritis, inflammatory bowel disease, celiac disease), pancreatitis, and certain medications, including epinephrine, tretinoin, vincristine sulfate, and heparin sodium.

D-Dimer Test

As a result of fibrinolysis, some of the fibrin in a thrombus degrades to form the D-dimer protein, which consists of two cross-linked D fragments of fibrin and which is elevated in the blood in the presence of VTE, rheumatoid arthritis with elevated rheumatoid factor, myeloproliferative disorders, infection, hemorrhage, trauma, and surgery. So, the D-dimer assay is not very specific for VTE, since it is also elevated in several other conditions; nonetheless, when combined with a symptomatic leg and even more so a symptomatic leg and venous Doppler ultrasound imaging of the involved extremity, elevation of the D-dimer can be very specific. The normal value for D-dimer units is ≤250 ng/mL or ≤0.5 mcg/mL fibrinogen equivalent units. As a rule, patient with a low pretest probability of DVT or PE with a negative D-dimer test should undergo further testing, typically imaging evaluation, if VTE is still suspected.

Imaging Studies for Venous Thromboembolism

Duplex Doppler Ultrasound (DDUS)

Duplex Doppler ultrasound (DDUS) imaging of the lower extremity veins is the mainstay diagnostic imaging examination used to identify lower extremity DVT. DDUS is noninvasive, usually readily available and, in comparison to other imaging modalities, relatively inexpensive in terms of the crude cost of the study. Venous DDUS is highly sensitive (95%) and specific (98%) for the diagnosis of DVT in the symptomatic lower extremity, although it is less sensitive in the asymptomatic extremity and in very obese patients [18, 19].

Other Venographic Methods

Contrast, magnetic resonance, and computerized axial tomographic venography can also be used to identify DVT in the lower extremity. The routine use of contrast venography has dwindled over the past 20–30 years, due to the potentially hazardous nature of invasive contrast media (nephrotoxicity), limitations related to inadequate deep vein filling with the contrast dye, and the steady improvement and availability of noninvasive magnetic resonance venography (MRV) and computerized axial tomographic venography (CATV).

Diagnostic Tests for Pulmonary Embolism

When PE is suspected, either with or without a prior diagnosis of DVT, a number of diagnostic laboratory, imaging, and functional tests can be helpful. As previously mentioned above, elevation of the D-dimer units present in the blood can be suggestive of VTE and is most useful in terms of specificity. Hypoxia secondary to diminished pulmonary perfusion related to PE results in an arterial blood gas with a $PaO_2 \leq 80$ mmHg in about 84% of patients with confirmed PE and no previous cardiopulmonary disease [20]. Measured levels of urine prothrombin fragment F1 + 2 can also potentially be used to assess the individual risk of vascular thrombotic complications, including VTE, following total hip arthroplasty and to test for noninvasive detection of sustained coagulation activation [21]. Right heart failure secondary to PE and pulmonary artery hypertension can also lead to elevation of cardiac troponin, as well as elevation of brain natriuretic peptide in patients without renal failure [22]. Radiographic images of the chest might reveal nonspecific signs of atelectasis, consolidation, and pleural effusion, in association with PE; electrocardiography might reveal several findings commonly associated with PE, including the $S_1Q_3T_3$ waveform pattern [23]; and transthoracic as well as transesophageal echocardiography can reveal evidence of right heart failure as a result of PE. The use of a ventilation-perfusion (V-Q) scan, where radiopharmaceutical (a gamma ray emitting xenon or technetium compound) is injected and lung ventilation and perfusion monitored, can be helpful in cases where contrast medium cannot be administered due to allergy or kidney disease or in certain obese or pregnant patients, and it is strongly suggestive of PE when it is abnormal and observed in conjunction with strongly indicative clinical findings. Pulmonary angiography, by means of contrast medium injection or magnetic resonance angiography (MRA), can also be useful, although computerized tomographic pulmonary angiography with contrast, while being expensive and invasive in terms of contrast medium and radiation exposure, has been shown to be more sensitive for PE than VQ scanning [24] and may be more sensitive and more specific than classic contrast pulmonary angiography [25].

Methods of VTE Prophylaxis and Treatment

Patient Education, Mechanical Prophylaxis, and Non-general Anesthesia

Prevention of VTE is a worthwhile and potentially lifesaving endeavor. The basic elements of VTE prophylaxis include patient education, mechanical methods that promote venous blood flow in the extremities, chemoprophylactic measures

that inhibit thrombus formation, and variations in anesthesia and fluid management. Nurses [26] and surgeons, as well as house and office staff, can play an important role in teaching patients the basic physiology and warning signs of VTE, both DVT and PE, and this can go a long way in regard to compliance with prophylactic measures and even early diagnosis should a complication develop. As a rule, combined prophylactic modalities decrease significantly the incidence of VTE, in particular DVT [27]. Of course, lower extremity movement, in particular ambulation with active contraction and relaxation of the crural musculature and resultant knee and ankle motion, is an important deterrent to VTE. In fact, most VTE prophylaxis protocols in otherwise healthy individuals are discontinued once regular knee and ankle motions are resumed following foot and ankle surgery. When lower extremity range of motion cannot be implemented, such as when complete bed rest is a required element of treatment or when the extremity is immobilized, then graduated compression stockings (GCS), and intermittent pneumatic compression (IPC) devices on one or both extremities can be employed (Fig. 2.3). A 2010 review of the literature pertaining to the use of GCS revealed that their use reduced the incidence of DVT from 26% to 13% ($p < 0.0001$) in comparison to patients treated without GCS [28]. IPC devices applied to any extremity can also provide beneficial fibrinolytic activity, as evidenced by elevated blood-borne fibrinolytic activity in assays procured from sites distant to the location of the IPC device [29, 30], and this could be particularly useful to foot and ankle surgical patients when both lower extremities have to be in the surgical field [31]. In general, when foot and/or ankle surgery is undertaken on one lower extremity, an IPC device is typically applied to the contralateral lower extremity during the operative procedure and throughout the course of hospitalization.

In regard to anesthesia and its potential adverse influence on venous stasis and the development of VTE, it is generally considered favorable to avoid cessation of the calf muscle pump influence on venous return from the lower extremities, and, therefore, avoidance of skeletal muscle paralysis and general anesthesia, when possible, is likely to decrease the risk of DVT [32–35]. In fact, the American Association of Plastic Surgeons has recommended that when possible, the use of non-general anesthesia, such as monitored anesthesia care, local anesthesia with sedation, or neuraxial anesthesia instead of general anesthesia, should be used in order to diminish the risk of VTE [31].

Pharmacological Prophylaxis (Chemoprophylaxis) and Treatment of VTE

Naturally occurring, physiologic anticoagulants such as antithrombin III and activated protein C prevent widespread thrombosis and localize clot formation to sites of vascular injury. The clotting cascade is also balanced by plasmin-mediated fibrinolysis, resulting in the formation of D-dimers and other fibrin degradation products. When the body's intrinsic anticoagulation system requires bolstering to prevent or treat VTE complicating surgery or medical care, a wide range of therapeutic agents are available (Table 2.3), including unfractionated heparin (UFH), which is typically administered subcutaneously (SC) or via intravenous (IV) infusion; low-molecular-weight (fractionated) heparins (LMWHs), which are administered via subcutaneous injection or IV infusion and include agents such as enoxaparin, dalteparin, and tinzaparin; vitamin K antagonists like warfarin, which can be administered orally or via IV infusion; factor Xa inhibitors, which are administered via subcutaneous injection or IV infusion and include agents such as fondaparinux, rivaroxaban, apixaban, and edoxaban; direct thrombin inhibitors like dabigatran, which can be administered orally; and combined factor Xa and thrombin inhibitors like danaparoid, which can be administered via subcutaneous injection or IV infusion. The decision to choose one anticoagulant over another varies by indication and patient-specific factors, and surgeons are always encouraged to use their clinical judgment in order to tailor evidence-based guidelines for VTE prophylaxis or treatment to the particular needs of their individual patients. Useful guidelines for VTE prophylaxis and treatment are available, and readers are encouraged to review these [31, 36–38]. Surgeons are also encouraged to read the clinical pharmacology

Fig. 2.3 Intermittent pneumatic compression (IPC) device on the left lower extremity of a patient about to undergo right foot surgery

Table 2.3 Anticoagulants used for venous thromboembolism prophylaxis and treatment

Category	Agent	Prophylaxis[a, b]	Treatment[a]
Unfractionated heparin (UFH)	Heparin	5000 units SC 2 h preoperative	80 u/kg bolus followed by 18 u/kg/hr IV infusion, or initial IV bolus of 5000, followed by 17,500 u SC twice daily
Low-molecular-weight heparin (LMWH)	Enoxaparin	40 mg SC 2 h preoperative	1.5 mg/kg/day SC once daily or 1 mg/kg SC every 12 h
	Dalteparin	2500 u SC starting 4–8 h after surgery, then 5000 u daily; or 2500 u SC starting 2 h preoperative, then 2500 u SC 4–8 h postoperative on the day of surgery, then 5000 u SC daily; or, 5000 u SC 10–14 h presurgery, then 5000 u SC 4–8 h postoperative on the day of surgery, then 5000 u SC daily	200 u/kg SC daily or 100 u/kg SC every 12 h
	Tinzaparin	50–75 u/kg 2 h preoperative, then 50 u/kg daily for 7–10 days, or 75 u/kg daily postoperative for 7–10 days	175 units/kg SC for 6–7 days
Factor Xa inhibitor	Fondaparinux	2.5 mg SC once daily starting 6–8 h postoperative, for 5–32 days after surgery	5 mg (body weight < 50 kg), 7.5 mg (body weight 50–100 kg), or 10 mg (body weight > 100 kg) SC once daily for 5 days and until a therapeutic oral anticoagulant effect is established
	Rivaroxaban	10 mg orally once daily with or without food	The 15 mg and 20 mg tablets are taken with food, whereas the 10 mg tablets can be taken with or without food. For the treatment of DVT, PE, and reduction in the risk of recurrence of DVT and of PE, 15 mg orally twice daily for the first 21 days for the initial treatment of acute DVT or PE. After the initial treatment period, 20 mg orally once daily with food for the remaining treatment
	Apixaban	2.5 mg orally twice daily	10 mg taken orally twice daily for 7 days, followed by 5 mg taken orally twice daily, for treatment of DVT and PE, or 2.5 mg taken orally twice daily for reduction in the risk of recurrent DVT and PE following initial therapy
	Edoxaban	Not indicated for prophylaxis	60 mg once daily or, if creatinine clearance, 15–50 mL/min or body weight less than or equal to 60 kg or who use certain *P*-glycoprotein inhibitors, 30 mg once daily
Direct thrombin inhibitor	Dabigatran	For patients with creatinine clearance >30 mL/min, 110 mg orally first day, then 220 mg once daily	For patients with creatinine clearance >30 mL/min, 150 mg orally, twice daily after 5–10 days of parenteral anticoagulation and for reduction in the risk of recurrence of DVT and PE for patients with creatinine clearance >30 mL/min, 150 mg orally twice daily after previous treatment
Combined factor Xa, heparinoid thrombin inhibitor	Danaparoid	For nonvascular surgery, if ≤90 kg, 750 u SC 1–4 h preoperative repeated ≥6 h postoperative then 750 u SC twice daily for 7–10 days starting the first postoperative day; and if >90 kg, then 750 u SC 1–4 h preoperative repeated ≥6 h postoperative then 1250 u SC twice daily or 750 u SC three times daily for 7–10 days starting the first postoperative day. In patients with current HIT, body weight ≤ 90 kg, 750 u SC two or three times daily for 7–10 days, following initial IV bolus of 1250 u SC; if >90 kg, then 1250 u SC two or three times daily for 7–10 days after an initial. In patients with past HITT, ≤90 kg, 750 u SC two or three times daily for 7–10 days; and if >90 kg then 1250 u SC two or 750 u SC three times daily for 7–10 days	For a thrombosis <5 days old and weight ≤ 55 kg, 1250–1500 u IV bolus then 400 u/h next 4 h then 300 u/h next 4 h then 150–200 u/h for 5–7 days or maintenance of 1500 u SC twice daily for 4–7 days; if weight 55–90 kg, 2250–2500 u IV bolus then 400 u/h next 4 h then 300 u/h next 4 h then 150–200 u/h for 5–7 days or maintenance of 2000 u SC twice daily for 4–7 days; and if weight > 90 kg, then 3750–2500 u IV bolus then 400 u/h next 4 h then 300 u/h next 4 h then 150–200 u/h for 5–7 days or maintenance of 1750 u SC twice daily for 4–7 days. For thrombosis ≥5 days old and weight ≤ 90 kg, 1250 u IV bolus the 750 u SC two or three times daily; if >90 kg, then 1250 u IV bolus then 750 u SC three times daily or 1250 u SC two or three times daily

(continued)

Table 2.3 (continued)

Category	Agent	Prophylaxis[a, b]	Treatment[a]
Vitamin K antagonist	Warfarin	Warfarin is available as scored tablets of 1, 2, 2.5, 3, 4, 5, 6, 7.5, or 10 mg and as a vial of 5 mg of reconstituted lyophilized powder for injection. Individualized dosing of warfarin is administered orally (or IV) when heparin or heparinoid therapy is already in effect, and it can usually begin (or be resumed if a 5-day overlap, bridging protocol is in effect) on the night of surgery, after which therapy is guided by the INR for at least 10 days and continued up to 4–6 weeks depending on patient-specific factors. Patients that have discontinued their maintenance warfarin therapy preoperatively can typically resume their usual dose beginning the night of surgery or the first postoperative day	After initiation of oral (or IV) administration, aiming for an INR of 2–3, warfarin is usually continued for 3–6 months
Cyclooxygenase inhibitor	Aspirin (acetylsalicylic acid)	325 mg daily for 4–6 weeks, starting the night of surgery, always in combination with physical measures such as GCS and ICD, as well as patient education and avoidance of GA whenever possible	Not indicated for treatment

Abbreviations: *GA* general anesthesia; *GCS* gradient compression stockings; *ICD* intermittent compression device; *IV* intravenous; *SC* subcutaneous, *u* international units

[a]Duration of prophylaxis is usually continued until the involved extremity is mobilized and the duration of treatment is typically 3–6 months post identification of the clot

[b]Prophylaxis entails chemoprophylaxis combined with patient education, mechanical methods, and avoidance of general anesthesia when possible; surgeons and anesthesiologists need to use caution if neuraxial (spinal, epidural) anesthesia is to be used since the risk of spinal or epidural hematoma increases with VTE prophylaxis; and dosage recommendations are based primarily or VTE prophylaxis associated with hip and/or knee surgery or general medical patient care

information contained in the package insert specific to each of these medications in order to review the details related to their proper use for VTE prophylaxis, treatment, and reversal (which is beyond the scope of this text). Surgeons are also encouraged to recruit the medical expertise of other experienced clinicians in the management of acute DVT and/or PE, since the care of such patients can be complicated and requires a wide range of expertise and intervention. Although the primary adverse effect of anticoagulant pharmacological agents is hemorrhage, parenteral administration and patient and surgeon nonadherence to treatment and guidelines are important limitations of their use [39].

Prophylaxis is generally continued until the risk factors are such that VTE is not likely, and this is typically at a time when lower extremity immobilization is discontinued and surgeons need to individualize the duration of prophylaxis based on the needs of each individual patient. The duration of treatment for confirmed DVT and/or PT depends to a large degree on the risk of recurrence. Patients at a high risk for recurrence include those with idiopathic DVT or PE, malignancy, antiphospholipid syndrome, an inferior vena cava filter, obesity, the elderly, and males. As always, the risk of prolonged anticoagulation is hemorrhage.

Anticoagulants that are commonly used for VTE prophylaxis include unfractionated heparin, fractionated heparins such as dalteparin and enoxaparin, the factor Xa inhibitor fondaparinux, and the heparinoid danaparoid, which is particularly useful in patients with a history of heparin-induced thrombocytopenia (HIT). Aspirin can also play a role in prophylaxis [40], although it is not considered to be a sole method of prophylaxis. Warfarin can also be used and is often initiated after surgery using a bridging protocol that also employs heparin or another anticoagulant.

The main goals of treatment for DVT include prevention of PE, postphlebitic syndrome, and recurrent thrombosis. Once VTE is suspected, anticoagulation should be started immediately unless there is a contraindication. In their review of the treatment of VTE, Wells et al. [4] divided therapies into acute (first 5–10 days), long-term (from the end of acute to 3–6 months), and extended (beyond 3–6 months) phases. And, despite the potentially lethal and acutely morbid nature of DVT and PE, they found that low-molecular-weight heparin (LMWH) along with vitamin K antagonists or the use of two oral agents without LMWH, along with ambulation and other physical measures, allows for outpatient management of most cases of DVT and some cases of PE, in the acute phase. Unless there is a specific contraindication, anticoagulation should be initiated as soon as VTE is diagnosed. Beyond the use of LMWH and/or oral therapies combined with physical measures, the use of thrombectomy