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A Forensic Analysis of Pulmonary Embolism And Its Missing Link: Causation

William Price

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I. Introduction

This paper explores the use of forensics outside of the criminal context, specifically in regard to pulmonary emboli.[1] This paper will analyze what pulmonary embolism are, how they interact with the rest of the body, what causes them, and the legal considerations that arise because of them. Pulmonary embolism is common and deadly, making them a subject in a vast amount of medical malpractice litigation. This paper aims to educate the public and attorneys as to how this condition can be prevented and how to prove pulmonary embolism in civil suits regarding claims of malpractice.

II. The Vascular System

The circulatory system in the human adult is comprised of an astounding 60,000 miles of blood vessels. The vessels serve as a continuous pipe-like conduit for the flow of blood throughout the body, pumped by the heart. Arteries, arterioles, and capillaries convey blood from the heart to the tissues and organs of the body, while venules and veins serve to transport blood from the body back to the heart. The heart pumps six quarts of blood per minute via the blood vessels (360 quarts per hour).[2]

The heart (see Figure 1) is the key component of the vascular system. It is approximately the size of a human fist and is situated in the upper mid-chest articulated slightly to the left. The heart is a hydraulic pump that sends blood via the vascular system both to and from the tissues and organs of the body. The aorta, the heart's large artery, sends out oxygen and nutrient rich blood that travels through arteries and then arterioles that connect with the network of capillaries. The small capillaries conduct at the cellular level the transfer of oxygen and nutrients to enrich

[1] Pulmonary Embolism: blood clots which travel to the lungs.
[2] Leahy, Monique C.M. Medical Malpractice Liability for Blood Clot, Deep Vein Thrombosis, Pulmonary Embolism, or Clot-Provoked Strike Causing Injury or Death, 135 Am. Jur. Trials 207 (Aug. 2016).

the organs of the body and exchange that for waste such as carbon dioxide to be moved back through the system.[3]

The superior vena cava is the large vein that carries blood from the upper body to the right atrium. This major vein is formed from tributaries draining the head, neck, and arms. (See Figure 2.) The inferior vena cava carries blood from the legs, back, and abdomen to the right atrium. (See Figure 3.)[4]

The internal iliac vein drains pelvic organs, the pelvic wall, and part of the thigh, and joins the external iliac vein to form the common iliac vein. The common iliac veins from each side join to form the inferior vena cava. Later, in its course within the abdomen, the inferior vena cava receives the renal veins. In addition, the venous drainage from the stomach, spleen, and intestine is carried by the portal vein to the liver. Blood leaving the liver flows through the hepatic veins, which enter the inferior vena cava as it passes through the diaphragm.[5]

Veins are wide and relatively thin-walled channels which offer little resistance to the flow of blood and are much more easily compressed than arteries. Like artery walls, vein walls are composed of three layers: the intima lining the lumen,[6] the media, and the outer adventitia. The intima consists of a single layer of endothelial cells (flat cells that line the blood vessels) and the sub-endothelium,[7] which contains basement membrane.[8]

All but the largest and smallest veins contain valves. Valves consist of a number of semicircular folds of the intima projecting into the lumen. Valves prevent the flow of blood away

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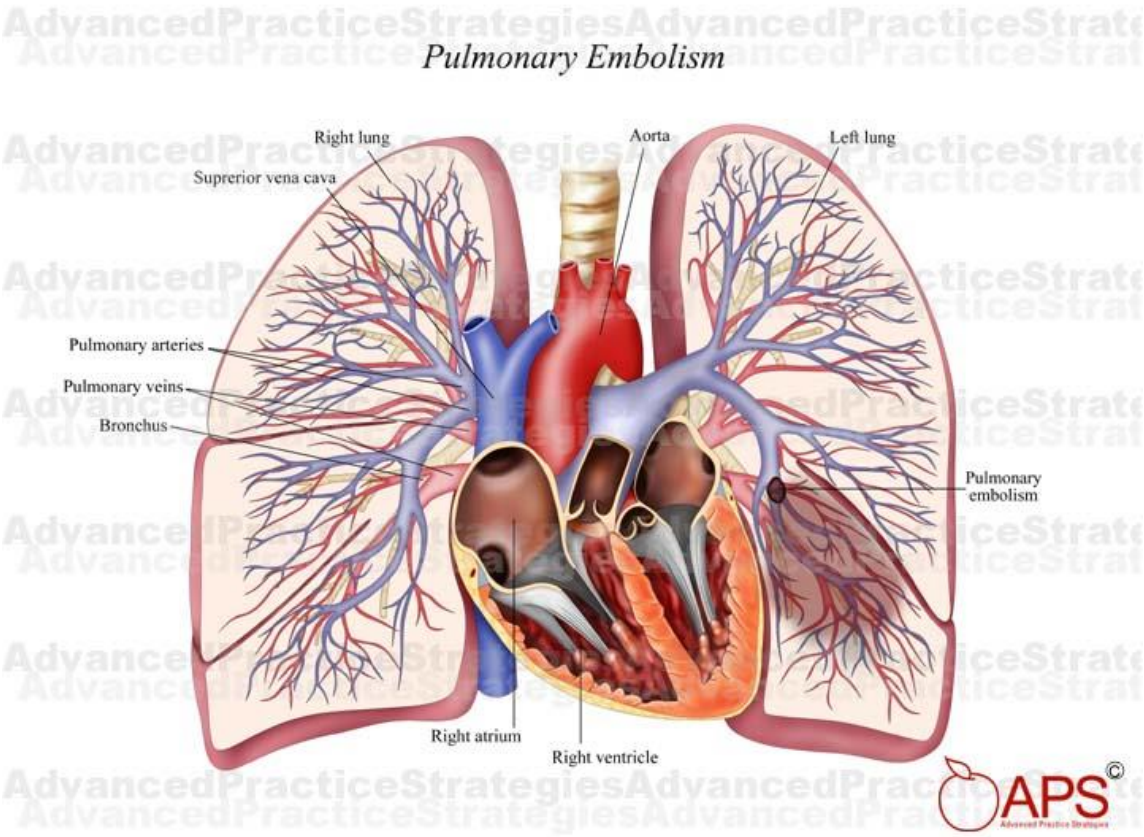
[3] Russ, Freeman and McQuade, Attorneys Medical Advisor §§ 5:1 to 5:14.
[4] Mann, C. Katherine. Thrombophlebitis, 10 Am. Jur. Proof of Facts 3d 369 (Sept. 2016).
[5] Id.
[6] Lumen: The passageway within a vein.
[7] Subendothelium: The lining of the blood vessels made up of flat cells.
[8] Basement membrane: is a thin layer of delicate noncellular material of a fine filamentous texture underlying the endothelium. American Jurisprudence Proof of Facts 3d, Taber's Cyclopedic Medical Dictionary (16th ed.).

from the heart. When skeletal muscles contract, the veins are compressed and the blood within their lumens is forced towards the heart.[9]

Figure 1: The Heart and Lungs[10]



[9] Ogston, Venous Thrombosis: Causation and Prediction 4–7 (1987)
[10] Pulmonary Elmbolism Following Laproscopic Gastric Bypass, Illustrated Verdict, http://www.illustratedverdict.com/projectreview/IV/IV_V1_2012web/1650w3lg.jpg

Figure 2. Major veins of the upper body[11]

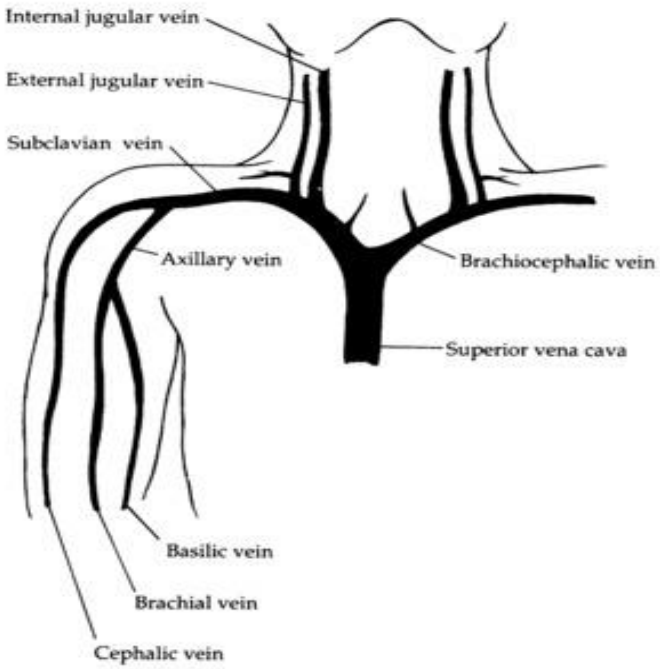
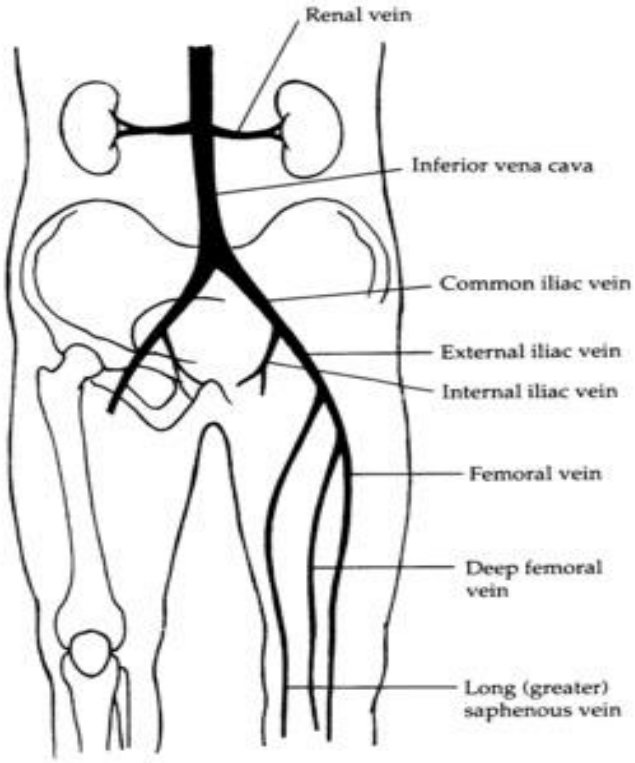


Figure 3. Major veins of the lower body[12]



[11] 10 Am. Jur. Proof of Facts 369 at 13.
[12] Id at 14.

Blood, in turn, nourishes the cells and essential organs of the body by the intricate transmission of oxygen and nutrients which it carries, and pumps away "dirty blood" containing waste products, which are then filtered and eliminated through the liver and kidneys.[13]

Blood consists of plasma containing semisolid red and white blood corpuscles, lymph corpuscles, and platelets. The red corpuscles transport oxygen and nutrients to individual cells. White and lymph corpuscles carry waste products away from the cells. Platelets serve as a coagulant.[14]

a. Pulmonary Embolism and Venous Thrombosis

Pulmonary embolism occurs when a blood clot clogs one of the pulmonary arteries, blocking blood flow to a part of the lung[15] with often fatal results. Pulmonary embolism is estimated to cause 50,000 deaths per year in the United States. The larger the embolism, the more likely it is to be fatal. An embolism large enough to block both pulmonary arteries at once is called a “saddle” embolus. The usual cause of the clot is a deep vein thrombophlebitis[16] in a leg vein or in the pelvic veins. This accounts for 70% of pulmonary embolisms.[17] Deep vein thrombosis, commonly referred to as "DVT," occurs when a blood clot or thrombus, develops in the large veins of the legs or pelvic area. Some DVTs may cause no pain, whereas others can be quite painful. With prompt diagnosis and treatment, the majority of DVT's are not life threatening. However, a blood clot that forms in the invisible "deep veins" can be life

[13] DeBakey, The New Living Heart 36 (Adams Media Corp., 1997).
[14] Coagulation: The process by which the blood forms clots; Penofsky, Daniel J. Litigating Vascular Medical Malpractice Cases. 10 Am. Jur. Trials 1, 38 (2007)
[15] Diagnosing and Treating Pulmonary Embolism, 8 Medico-Legal Watch 17.
[16] Thrombophlebitis: inflammation of the vein coupled with thrombosis
[17] Tennenhouse, Dan J. Pulmonary Embolism, 2 Attorneys Medical Deskbook § 24:5.

threatening. A clot that forms in the large, deep veins is more likely to break free and travel through the vein. It is then called an embolus.[18]

The term "thrombophlebitis" has come into general use to designate two closely associated conditions: clot formation in a vein and inflammation of the vein wall. Thrombophlebitis may be defined as a partial or complete closing of a vein by a clot with preceding or secondary inflammation of the wall of the vein.[19] There are two types of emboli: arterial and venous. Arterial emboli usually originate in the left side of the heart in persons with heart disease. These emboli can travel to the brain, kidneys, spleen, or the extremities.[20] Venous emboli usually originate in the veins of the lower extremities and travel through the venous system through the right side of the heart and into the lungs, where they can produce a pulmonary embolism or a pulmonary infarction,[21] or both. A pulmonary infarction is usually fatal.[22]

Traumatic thrombophlebitis of the superficial veins is more likely to develop as a result of direct trauma than is thrombophlebitis of the deep veins. In the deep veins the condition most often develops as a secondary reaction to trauma elsewhere. Thrombophlebitis in the deep veins is particularly treacherous because it is likely to develop without visible inflammation of the vein walls. A clot may break loose without warning and lodge in the lung region. Venous thromboembolic disease is a major source of morbidity and mortality, especially among

[18] Deep Vein Thrombosis, Vascular Disease Foundation, 2012.
[19] 10 Am. Jur. Proof of Facts 369 at 12.
[20] Id.
[21] Pulmonary Infarction: A condition in which one or more arteries in the lungs become blocked by a blood clot.
[22] Shafer, Traumatic Thrombophlebitis, 10 Lawyer's Medical Journal 2d 648, 651 (1982).

hospitalized persons. Because of the lack of uniform diagnostic methods, the incidence of deep vein thrombosis is difficult to estimate.[23]

In the case of thrombosis of the deep veins, a distinction is sometimes drawn between thrombophlebitis and phlebothrombosis. The former is used to describe a thrombus which is adherent to an inflamed vein wall; the latter is used to describe a fresh thrombus free from, or just loosely adherent to, a normal, non-inflamed vein wall. It is recognized, however, that the presence of a thrombus in the lumen of a vein elicits an inflammatory reaction. Phlebothrombosis may be regarded, therefore, as an earlier phase of thrombophlebitis. The terms thrombophlebitis and venous thrombosis are used, generally, to cover both superficial thrombophlebitis and deep vein thrombosis.[24]

b. Clot Forming Process

The body maintains the integrity of its circulatory system following an injury through the process of hemostasis.[25] This process allows blood to continue circulating while permitting the blood to clot in necessary areas, such as the site of a cut. Blood loss prevention is vital and occurs when several elements work together to form a blood clot, which acts as a barrier to further blood loss. The basic components involved in the clotting process are the blood vessel (which constricts when injured), the platelet, and coagulation or clotting factors that circulate in the bloodstream. When there is a cut or damage, the blood vessel wall constricts, minimizing blood loss even before clotting begins.[26] Although the initiating factors may be quite variable,

[23] Id.
[24] Ogston, Venous Thrombosis: Causation and Prediction 3 (1987).
[25] Hemostasis: The arrest of bleeding or circulation.
[26] 135 Am. Jur. Trials at 9.

the clot-forming process, once established, is generally similar in all cases no matter what the cause.[27]

Platelets play an important role in blood clot formation. When a vessel wall is injured, platelets adhere to each other and to the edges of the injury site and form a plug that covers the area. The adhesion of platelets to the vessel wall and the aggregation of platelets to each other is followed by the deposition of fibrin,[28] which serves to stabilize the plug. The fibrin, formed by the coagulation system, is deposited as fine interlacing filaments in which red and white blood cells and platelets are entangled.[29] When enough platelets collect at the damaged site, a plug or white thrombus forms. If the damaged area is very small, the platelet plug may be enough to stop the bleeding. If not, the blood clotting mechanisms are triggered.[30]

c. Initiation of Thrombosis

Three initiating factors have been recognized in the formation of thrombi in the veins: changes in the vessel wall, changes in blood flow, and changes in blood coagulability. These factors are known as Virchow's triad.[31]

i. Changes in the Vessel Wall

The intact lining of the vessel prevents the formation of a thrombus on the vessel wall under normal circumstances. Platelets do not adhere to normal lining, but injury resulting in exposure of sub-lining structures is followed by the accumulation of platelets and the buildup of

[27] 10 Am. Jur. Proof of Facts 369 at 16
[28] Fibrin: A whitish, threadlike protein formed by the action of the enzyme thrombin on the protein fibrinogen. This process is the basis of blood clotting.
[29] Id.
[30] Russ, Freeman and McQuade, Attorneys Medical Advisor §§ 6:9, 6:10; Thrombophlebitis (Venous Thrombosis), 10 Am. Jur. Proof of Facts 3d 369.
[31] 10 Am. Jur. Proof of Facts 3d 369 at 17.

a platelet mass extending into the vessel lumen.[32] The importance of damage to the vein in the development of venous thrombosis is controversial. It is probable that the majority of venous thrombi in the legs are not the result of local trauma; exceptions are cases of femoral vein thrombi associated with hip replacement surgery and cases of femoral neck fractures.[33]

ii. Changes in Blood Flow

While stasis by itself is not likely to result in thrombosis, such reduction in blood flow clearly has an important influence on the development of thrombosis. This importance probably lies in the role of stasis in reducing the washing away of loose masses of platelets at the margins of the developing thrombosis, and in slowing the rate at which activated coagulation factors are removed, thereby facilitating local accumulation. Additionally, slowing of the blood flow leads to an increase in viscosity, which itself leads to a further reduction in flow.[34]

While there is doubt about whether stasis alone can induce venous thrombosis, there is abundant evidence that stasis is frequently associated with deep vein thrombosis of the lower limb and is likely to be an important contributory factor in the production of thrombosis in a number of situations, such as during and after surgery. Such evidence includes a report that a decrease in velocity of venous blood flow in the leg occurs in the majority of persons during surgical operations.[35]

iii. Blood Coagulability

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[32] Erskine, "Blood Vessels and Lymphatics," in Krupp, Chatton & Tierney, eds, Current Medical Diagnosis and Treatment (1986).
[33] Ogston, Venous Thrombosis: Causation and Prediction 54 (1987)
[34] Malone & Morris, The Sequestration and Margination of Platelets and Leucocytes in Veins During Conditions of Hypokinetic and Anaemic Hypoxia, 12 Journal of Pathology 119 (1978); Morrison et al., The Effects of Anoxia on the Morphology and Composite Metabolism of the Intact Aortic Intima-Media Preparation, 59 Journal of Clinical Investigation 1027 (1977).
[35] Doran, Drury & Sivyer, A Simple Way to Combat the Venous Stasis Which Occurs in the Lower Limb During Surgical Operations, 51 British Journal of Surgery 486 (1964).

The role of altered blood coagulability in the production of venous thrombosis has been widely debated.[36] Altered blood coagulability can take the form of an increased level of coagulation factors, the presence of activated clotting factors, or altered fibrinolytic capacity to remove forming fibrin. There can be little argument that these factors contribute to the growth and ultimate size of a thrombus, but few authorities would claim that the factors are responsible for the initiation of venous thrombosis. Likewise, venous stasis alone is not likely to be a sufficient stimulus for thrombus formation. However, the combination of stasis and the presence of activated coagulation factors has been shown repeatedly under experimental conditions to be sufficient for the production of venous thrombosis. It has been proposed that the activated clotting factors may be formed locally or generated at a distant site, but the precise mode of initial activation is the subject of speculation. What is clear is that once thrombin is formed, platelets are activated. In the absence of effective neutralization of the thrombin, a feedback mechanism leads to the further generation of thrombin. The result is the buildup of a fibrin-platelet mass.[37]

III. Predisposition and Preexisting Conditions

Physicians have long understood some of the precipitating causes of deep venous thrombosis: injury to the vessel wall, venous stasis, and changes in coagulation. Thus, physicians know that a whole host of conditions can contribute to the formation of deep venous thrombosis: injury to vessel walls of the lower extremities caused by trauma, such as fractures or burns; venous stasis due to obesity, congestive heart failure, peripheral vascular disease, pregnancy, prolonged bed rest, or traveling; and increased coagulability of the blood, brought on by malignancy, surgery, aging, use of birth control pills, pregnancy, and abnormalities in the

[36] Ogston, Venous Thrombosis: Causation and Prediction 56 (1987).
[37] Id.

blood.[38] Obviously, if there is a combination of factors, the risk increases. For example, pregnancy is accompanied not only by venous stasis due to the pressure of the enlarged uterus on the inferior vena cava, but also by subtle changes in the coagulation system that lead to increased coagulability of the blood. This combination results in a greater risk of thrombosis than the risk presented by stasis or increased coagulability alone.[39]

Trauma, illness, pregnancy, and childbirth can be complicated by thrombosis, especially if the affected person is of advanced age; has had a previous episode of deep vein thrombosis; has heart disease or malignant disease; has varicose veins; is obese; uses birth control pills; or has abnormalities in the blood, such as polycythaemia,[40] thrombocytosis,[41] paroxysmal nocturnal hemoglobinuria,[42] sickle cell disease,[43] abnormal coagulation factors, a raised plasma fibrinogen, or defective fibrinolysis. In many persons these risk factors play a very small part; the ultimate trigger of the coagulation chain reaction is the hypercoagulability induced by surgery or injury, coupled with the reduced venous blood flow that occurs because the ill or injured patient cannot exercise. Nevertheless, all the conditions that predispose to thrombosis must be considered as risk factors of the condition.[44]

The risk of thromboembolism following a fracture of a lower limb has long been recognized. Thrombosis following fracture of the upper end of the femur has received particular attention. Pulmonary embolism is a frequent cause of death following fracture of the hip. The

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[38] Shafer, Traumatic Thrombophlebitis, 10 Lawyer's Medical Journal 2d 648, 649 (1982).
[39] Id.
[40] Polycythaemia: Excess of red blood cells.
[41] Thrombocytosis: Increase in number of blood platelets.
[42] Paroxysmal Nocturnal Hemoglobinuria: Breakdown of red blood cells.
[43] Sickle Cell Disease: Abnormal red blood cells.
[44] Browse, Burnand & Thomas, Diseases of the Veins: Pathology, Diagnosis and Treatment 301 (1988).

incidence of thromboembolism after fractures of the lower portions of the leg is also substantial.[45]

A number of autopsies have shown that the incidence of venous thromboembolic disease is markedly increased in persons with cardiac disease and, in particular, those with congestive cardiac failure. A variety of factors probably contribute to such an increase. For example, venous stagnation and decreased cardiac output is present in many victims of heart disease, and bedrest with consequent immobility is frequently a component in creating venous stasis. Changes in blood viscosity and hemostatic components have been reported in persons with cardiac disease; such changes may also contribute to the production or persistence of venous thrombi.[46]

Malignant disease and venous thromboembolic disease are linked in a number of ways. Thrombosis is likely to develop when a vein is surrounded or infiltrated by a tumor, and is common in the terminal, wasting stages of malignant disease. Of long-standing interest has been the relationship between thrombophlebitis migraines (recurring attacks in various sites) and visceral carcinoma (cancer of the abdominal organs). Thrombophlebitis migraines may antedate the clinical appearance of cancer by months or years and may be regarded as an indicator of the possible presence of hidden malignant disease.[47] The cause of the increased risk of thromboembolism in persons with malignant disease is not fully explained. Clearly, a number of other predisposing factors frequently coexist with malignant disease such as, advancing years, prolonged bedrest, and surgery.[48]

a. Immobilization

[45] Fitts et al., An Analysis of 950 Fatal Injuries, 56 Surgery 663 (1964); Hjelmstedt & Bergval, Incidence of Thrombosis in Patients with Tibial Fractures: A Phlebographic Study, 134 Acta Chirurgica Scandinavica 209 (1968).
[46] Ogston, Venous Thrombosis: Causation and Prediction 93 (1987).
[47] Id. at 94.
[48] Id. at 96.

Elderly patients and others with preexisting heart or lung problems are at especially high risk of pulmonary embolism while immobilized. The possible end result has been described as follows: "The suddenness of death may be extremely dramatic. A patient may stand up after a period of confinement to bed and suddenly topple over silently with a massive embolus."[49]

Venous thrombosis and pulmonary emboli constitute a hazard to any patient who is immobilized, even if only one leg is placed at rest. The elderly are at particularly increased risk because they are more likely to have varicose veins and other venous abnormalities that interfere with the return of blood from the legs and thereby promote the development of thrombi. The elderly are also, of course, more likely to have preexisting heart conditions that reduce the heart's ability to pump against increased resistance.[50]

Deep vein thrombi may or may not cause noticeable symptoms, such as pain and muscle tightness. The presence of leg thrombi can be demonstrated on X-rays after contrast material has been injected into the veins, but they frequently go undetected until pulmonary embolism occurs. For this reason, efforts are made to prevent leg thrombosis in high risk patients. For example, venous blood flow is increased by elevating the legs during surgery and exercising them soon after surgery.[51] Anticoagulants are also administered. Alternative surgical procedures include tying off, clipping, folding, or otherwise interrupting the lowest portion of the inferior vena cava, or implantation of an "umbrella" to intercept emboli. These preventive procedures are reserved

[49] S. Robbins & R. Cotran, Pathologic Basis of Disease 129 (2d ed 1979)
[50] Hair, Robert E. et. al., Complications Due to Immobilization, 39 Am. Jur. Proof of Facts 2d 545, 18 (1984).
[51] J. Ludbrook & G. Jamieson, "Disorders of Systemic Veins," in Davis-Christopher Testbook of Surgery 1829–1830 (11th ed Sabiston 1977)

for high-risk patients with whom anticoagulants cannot be employed or have proven ineffective.[52]

b. Prevention of Complications

The three most important measures in preventing physical disuse phenomena are: (1) active exercise, when possible; (2) passive mobilization (having medical personnel or others move the patient's joints through their full range of motion whenever active motion is impossible); and (3) frequent change of position (including the standing position for patients able to stand or use a tilt table or standing board).[53]

Unfortunately, these simple measures are commonly ignored. Also, with a severely injured person, these measures are frequently impossible until considerable recuperation has occurred. A healthy person does not remain in one position, even for 30 minutes at a time, whether awake or asleep. The trauma victim immobilized in a bed or chair or by a cast, corset, or brace, particularly when in pain or under the influence of anesthetics or certain medications, is likely to spend long periods of time with little or no motion.[54] Pulmonary embolism and blood clots in the legs are typically a complication of immobilization that can be prevented through proper prophylactic measures.[55]

c. Cardiovascular Effects

Immobilization produces significant progressive cardiovascular changes.[56] These changes stem in large part from impairment of the autonomic control of the heart and the peripheral

[52] Merck Manual 649–651.
[53] 39 Am. Jur. Proof of Facts 545 at 9.
[54] Id.
[55] Robbins, Pathological Basis of Disease
[56] Kottke, 196 JAMA 825, 829.

circulation. Signs of complications arising out of immobilization include an increase in pulse rate and fall in blood pressure.[57]

The rapid fall of blood pressure when the patient is placed in an upright position, is caused by dilation of the blood vessels in the abdomen and the lower extremities under the weight of the blood volume accumulating in the lower portions of the body when the patient is upright. While the blood vessels normally have enough strength to contract reflexively under this pressure and reduce the volume, they lose this strength during prolonged disuse.[58]

IV. Vascular Medical and Surgical Considerations

Vascular surgery is that specialized branch of medicine which is concerned with the diagnosis and surgical treatment of diseases and disorders of the vascular system, exclusive of the intracranial (head and brain) vessels, and the arteries and veins of the heart. Vascular surgical treatment encompasses: (1) vascular diagnoses; (2) the performance of vascular surgical procedures, generally in a vascular surgeon's treatment room, under local or regional anesthesia; and (3) the performance of vascular surgery, generally in-hospital, under general anesthesia.[59]

Vascular surgery, performed by a vascular surgeon following the diagnosis of a vascular disease or disorder, can alleviate and cure many of the diseases and disorders. In fact, each year, more than 200,000 vascular procedures and surgeries are performed in the U.S.[60] For the most part, vascular diagnoses are accurate and vascular surgeries are generally performed without incident or complication, with the patient regaining normal vascular function following a reasonably short recuperative period. Most such successful surgeries allow the patient to regain

[57] Taylor et al., Effects of Bedrest on Cardiovascular Function and Work Performance, 2 (No. 5) Journal of Applied Physiology 223 (Nov 1949).
[58] Id. at 237.
[59] Penofsky, Daniel J., Litigating Vascular Surgery Malpractice Cases, 106 Am. Jur. Trials 1, 37 (2007).
[60] Id. at 38.

strength, mobility, physical endurance, and they promote a healthful and normal lifespan. However, there have been glaring examples of vascular misdiagnosis attributable to malpractice on the part of the diagnosing vascular surgeon, for which liability has been imposed.[61]

Vascular surgery malpractice cases are complex. They mandate a consummate knowledge of the vascular system; the nature and cause of the particular vascular disease or disorder which has afflicted the artery or vein in question; the accuracy of the vascular surgeon's diagnosis; the type of vascular surgery that was performed by the vascular surgeon to treat the diseased or disordered artery or vein; whether the complication and physical injury were proximately caused by a malpractice act or omission on the part of the vascular surgeon in diagnosis or the performance of the vascular surgery; and whether the vascular surgeon has breached the applicable standard of vascular medical and surgical care in diagnosis or surgical treatment.[62]

a. Types of Vascular Diseases and Disorders[63]

Varicose Veins: the most frequently diagnosed venous disease or disorder affecting the legs. Varicose veins are superficial veins that have become swollen, distorted and ineffective. They may occur anywhere in the body, but the veins most commonly involved are the saphenous veins of the legs. Varicose veins in the leg present as thready linear or bumpy protrusions just beneath the skin.

Chronic Venous Insufficiency: an obstruction or incompetence of a vein in the deep venous system of the leg, because of a thrombosis or blood clot(s) within the vein. The thrombosis blocks the venous flow of blood from the leg to the heart. This condition is

[61] Id.
[62] Id.
[63] See Davies, Venous Disease 33 (TFM Pub., 2006).

characterized by progressive edema, a reddish-brown hyper-pigmented appearance in the lower leg just above the ankle, recurrent skin ulceration, and poor wound healing.

Thrombosis: a blood clot condition within a vein, which blocks the venous flow of blood within the afflicted vein to the heart. Thrombosis may affect peripheral and deep veins, and if an embolus breaks off, it can be carried to a distant part of the circulatory system, where it may lodge in another blood vessel.

Phlebitis: an inflammation of a vein. When coupled with thrombosis, the condition is called thrombophlebitis. The thrombosis and inflammation may clinically present as superficial, deep, or both, creating the risk of pulmonary embolism.

Venous Ulcer: an anatomical break and overgrowth of fibrous tissue on the skin and subcutaneous layer, often on the leg, ankle or foot, provoked by venous reflux. The condition is frequently chronic.

Venous Valvular Incompetence: a disorder of a vein which incapacitates the valves of the vein and permits venous reflux, or the backflow of venous blood.

b. Vascular Evaluation

The vascular evaluation or examination conducted by a vascular surgeon proceeds upon the medical premise that many vascular diseases or disorders, especially those which affect the legs, are provoked by an underlying systemic disease, such as diabetes. Accordingly, not only must the presenting vascular disease or disorder be diagnosed and treated, but also any underlying provoking systemic disease as well.[64]

The primary goal of the vascular evaluation is to make a tentative diagnosis of any vascular disease or disorder afflicting the patient as well as any underlying systemic disease,

[64] Davies, Vascular Surgery 9 (Springer-Verlag, 2006).

confirmed by diagnostic testing and imaging studies, and then to proceed upon a course of nonsurgical or surgical treatment to alleviate or cure the presenting vascular disease or disorder. Where any underlying systemic disease is treatable by the vascular surgeon, this is accomplished as well. Where any underlying systemic disease is non-treatable by the vascular surgeon but it is diagnosed, referral of the patient to an appropriate medical specialist such as a cardiologist is made.[65]

c. Presenting Signs and Symptoms

The clinical diagnosis of deep vein thrombosis can be extremely difficult because many persons have no signs or symptoms in the affected limb. When present, the signs and symptoms that are commonly produced by deep vein thrombosis are pain, swelling, and a faint blue-red discoloration of the skin. Deep vein thrombosis may also present as a fever of unknown origin or with the features of pulmonary embolism without any evidence of the disease in the legs. The clinical features of pulmonary embolism are chest pain, coughing up of blood, and shortness of breath.[66]

The most common but most misleading indication of deep vein thrombosis is tenderness on compression of the calf muscles, or tenderness over the course of the main veins of the thigh. However, only half of those persons with calf muscle tenderness have a thrombosis.[67] Deep vein thrombosis often causes mild pitting edema of the ankle. This is a significant clinical sign and is a true indicator of thrombosis in seventy percent of cases, especially if the edema is unilateral.[68]

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[65] 106 Am. Jur. Trials 2d 1 at 54.
[66] Browse, Burnand & Thomas, Diseases of the Veins: Pathology, Diagnosis and Treatment 475 (1988).
[67] Flanc, Kakkar & Clark, The Detection of Venous Thrombosis of the Legs Using I-125 Labelled Fibrinogen, 55 British Journal of Surgery 742 (1968).
[68] Browse, Burnand & Thomas, Diseases of the Veins: Pathology, Diagnosis and Treatment 475 (1988).

The calf muscles may feel "woody" hard if there is extensive intramuscular thrombosis. If there is significant venous outflow obstruction, the skin of the leg may feel warm, and the superficial veins may be distended and fail to collapse when the limb is elevated. Distended veins in the affected groin of a leg with a thrombosis indicate a major degree of venous obstruction.[69]

Systemic signs such as restlessness, fever, tachycardia,[70] and changes in blood pressure may be the only signs of a deep thrombophlebitis. A pulse rate over ninety has been observed in seventy-five percent of the cases and fever has been observed in eighty percent of the cases.[71] Thus, when these signs occur following surgery or childbirth, and in the absence of any other localized condition, thrombophlebitis should be seriously suspected.[72]

d. Diagnosis

The imprecision of the clinical diagnosis of deep vein thrombosis is now generally accepted. In some patients, a thrombosis of a deep vein of the lower limb produces no symptoms or signs that might give forewarning of possible pulmonary embolism, while a considerable proportion of persons with clinical features suggestive of deep vein thrombosis are ultimately found not to have thrombotic disease. Accurate diagnostic techniques are essential for the proper institution of preventative measures and therapeutic regimes, and so that the application of such measures and regimes can be limited to those persons who require them.[73]

There are certain conditions that can mimic active thrombosis, thus a diagnosis is generally referred to as a differential diagnosis. A differential diagnosis is made by comparing

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[69] Id. at 476.
[70] Tachycardia: an abnormal rapidity of heart action. American Jurisprudence Proof of Facts 3d, Taber's Cyclopedic Medical Dictionary (16th ed.).
[71] Pratt, Surgical Management of Venous Clotting, Surgical Clinics of North America 341 (April 1948).
[72] Shafer, Traumatic Thrombophlebitis, 10 Lawyer's Medical Journal 2d 648, 660 (1982).
[73] 10 Am. Jur. Proof of Facts 3d at 28.

and contrasting available information of disease signs, symptoms, physical findings, examination results, laboratory data, and radiologic and other imaging studies, that are possibly responsible for the patient's illness, then narrowing down the determination of disease or illness afflicting the patient to two or three possible vascular diagnoses, and then *excluding* all possible diagnoses except for the one thought most likely to be the cause of the patient's symptomatology. [74]

In descending order of prevalence, the conditions to be distinguished from thrombosis include congestive heart failure, trauma, cancer, cellulitis (spreading inflammation of tissue), lymphangitis (inflammation of lymphatic channels), arthritis, and paresis (partial paralysis).[75] In addition, thrombophlebitis may mimic the symptoms of acute arterial ischemia (deficiency of blood supply), or systemic diseases such as edema from the nephrotic syndrome (kidney disease), and myositis ossificans (inflammation and conversion of muscle tissue to bone).[76]

Thrombophlebitis must be distinguished from a large number of clinical conditions in which symptoms and signs are located in the vicinity of the calf muscles. Among these conditions are ruptured popliteal (Baker) cyst, muscle strain due to hematoma formation (hemorrhage into the limb), tennis leg, and muscle hernia (rupture). Other conditions to be considered are superficial thrombophlebitis located under the skin in the vicinity of the calf muscles, dermatitis and chronic or acute cellulitis in the same site, lymphedema with cellulitis, and rupture of the plantaris tendon in the calf or Achilles tendonitis (inflammation of the tendon).[77]

e. Treatment

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[74] Davies, Venous Disease 141 (TFM Pub., 2006); American Jurisprudence Proof of Facts 3d, Taber's Cyclopedic Medical Dictionary (16th ed.).
[75] Shafer, Traumatic Thrombophlebitis, 10 Lawyer's Medical Journal 2d 648, 699 (1982).
[76] Browse, Burnand & Thomas, Diseases of the Veins: Pathology, Diagnosis and Treatment 478 (1988).
[77] Shafer, Traumatic Thrombophlebitis, 10 Lawyer's Medical Journal 2d 648, 700 (1982).

As a general rule of vascular medical practice, conservative nonsurgical treatment of a diagnosed vascular disease or disorder is initiated first, if at all possible, before surgical treatment is contemplated. A variety of nonsurgical vascular treatment modalities exist, depending upon the diagnosed vascular disease or disorder.[78]

In cases of thrombosis in the deep veins of the leg, or phlebitis or thrombophlebitis, conservative nonsurgical treatment includes bed rest, elevation of the afflicted leg, and anticoagulation with the drug heparin. Some patients may require thrombolytic therapy, which involves the intravenous injection of a substance such as urokinase, streptokinase, or tissue plasminogen activator to dissolve the thrombus.[79] Specific mechanical methods of thromboprophylaxis, which include graduated compression stockings (GCS), intermittent pneumatic compression (IPC) devices, and the venous foot pump (VFP), increase venous outflow and/or reduce stasis within the leg veins. As a group, mechanical thromboprophylaxis modalities have important advantages and limitations. The primary attraction of mechanical thromboprophylaxis is the lack of bleeding potential. These modalities, therefore, have advantages for patients with high bleeding risks. While all three of the mechanical methods of thromboprophylaxis have been shown to reduce the risk of DVT in a number of patient groups, they have been studied much less intensively than anticoagulant-based approaches and they are generally less efficacious than anticoagulant thromboprophylaxis, such as above-mentioned heparin.[80]

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[78] DeBakey, The New Living Heart 182 (Adams Media Corp., 1997).
[79] Id.; Erskine, "Blood Vessels and Lymphatics," in Krupp, Chatton & Tierney, eds, Current Medical Diagnosis and Treatment (1986).
[80] Geerts, William H. Prevention of Venous Thromboembolism. American College of Chest Physicians Evidence – Based on Clinical Practice Guidelines (8th Edition) (June, 2008 Supplement).

Where conservative nonsurgical treatment over time fails to alleviate or cure a diagnosed vascular disease or disorder, surgical intervention in the form of the performance of a vascular procedure or vascular surgery is considered. In the case of thrombosis in the deep veins of the leg, or phlebitis or thrombophlebitis, venous thrombectomy - direct surgical removal of the venous thrombosis - may be required.[81]

Thrombosis in a deep vein occludes its lumen and destroys its valves; therefore, restoration of complete normality to the affected vein requires the removal of the thrombus before it has destroyed the valves. If a thrombus cannot be removed, its ultimate effect is, at present, beyond medical control. All that can be done is to prevent further thrombosis by giving anticoagulants, or to stop complications, such as pulmonary embolism, by performing surgical venous blockade[82].[83]

There are two methods for removing thrombi: thrombectomy, which is the removal of the thrombus by surgical means, and pharmacological thrombolysis, which is the breaking up of the thrombus through drug therapy. Both methods are most effective when the thrombus is fresh and non-adherent to the vein wall, a stage at which a thrombus is often symptomless. By the time symptoms appear, the thrombus has often become adherent and old, two features which make its removal or dissolution difficult. Whenever possible, pharmacological lysis (dissolution or decomposition) of thrombi is preferred to surgical thrombectomy because lysis will affect all the

[81] Id.
[82] Venous blockade: A procedure (ligation) by which a thread or wire is used to tie a vein in order to constrict it to prevent the passage of emboli through the venous system to the lungs.
[83] Browse, Burnand & Thomas, Diseases of the Veins: Pathology, Diagnosis and Treatment 501 (1988).

thrombi in a limb, some of which are inaccessible to the surgeon. Clearly, pharmacological lysis also avoids the risks and complications of surgery.[84]

The decision to perform a thrombectomy is based upon the nature and extent of the thrombus seen through diagnostic tests, specifically a venography, and the severity of the clinical symptoms and signs. If a fresh non-adherent thrombus is completely removed and the person is given anticoagulants, fatal embolism is eliminated and minor embolism rarely occurs. However, in cases in which the operation fails or is only partially successful, recurrent embolism is common - more common than in cases in which the patient was given anticoagulants alone. Therefore, except when it is thought to be the only way to save the limb, thrombectomy is advised only when the venography indicates that all of the thrombus can be removed. If the thrombus cannot be completely removed, a venous blockade procedure may be coupled with the thrombectomy.[85]

Effective anticoagulation prevents the propagation of existing thrombi by stopping the formation of new thrombi, but such treatment has no effect on the behavior of existing thrombi. Thrombus fragmentation, retraction, or adhesion will still occur, and therefore anticoagulants do not prevent embolism or deep vein damage. Heparin is usually given as the initial treatment and can be replaced by an oral anticoagulant when the circumstances causing the hypercoagulable state begin to recede.[86]

Heparin therapy does not prevent recurrent embolism.[87] And there is no clinical evidence that heparin causes or accelerates thrombolysis.[88] Nor do studies give any indication that

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[84] Id.
[85] Id. at 512, 517, 518.
[86] Id. at 523.
[87] Browse et al., Prevention of Recurrent Pulmonary Embolism, 3 British Medical Journal 382 (1969); Plate, Ohlin & Eklof, Pulmonary Embolism in Acute Iliofemoral Venous Thrombosis,

anticoagulants reduce the incidence of the post-phlebitic syndrome. All that can be expected from anticoagulants is the prevention of further thrombosis. Thus, anticoagulants will save a life or prevent later symptoms in a limb on only a few occasions.[89]

f. Prognosis

Of patients who are diagnosed with PE and start treatment, ~5% die of the initial PE or another PE within the next 7 days. However, although the risk of dying of PE differs markedly among patients, no validated risk prediction tool is available. Risk of dying of PE is estimated to be ~70% if cardiopulmonary arrest occurs (~1% of patients at presentation), 30% if there is shock requiring inotropic support (~5% of patients), and ~2% in patients who are not hypotensive. In the presence of normal systemic arterial pressure, prognosis can also differ, depending on (1) clinical evaluation; (2) cardiac biomarkers such as troponin or brain natriuretic peptide; and (3) assessment of right ventricular size and function.[90]

V. Legal Considerations

Generally, medical malpractice is considered to be the negligent or unskillful performance by a physician of the duties that are devolved and incumbent on the physician on account of his or her relations with the patient or of a want of proper care and skill in the performance of a professional act.[91]

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72 British Journal of Surgery 912 (1985); Basu et al., A Prospective Study of the Value of Monitoring Heparin Treatment with the Activated Partial Thromboplastin Time, 287 New England Journal of Medicine 324 (1972).

[88] Elliot et al., A Comparative Randomized Trial of Heparin Versus Streptokinase in the Treatment of Acute Proximal Venous Thrombosis, 66 British Journal of Surgery 838 (1979); Widmer, The Treatment of Venous Thrombosis: Angiological Aspects, 16 Triangle 47 (1977).

[89] Young, Lea & Browse, Comparison Between Sequelae of Surgical and Medical Treatment of Venous Thromboembolism, 4 British Medical Journal 127 (1974).

[90] Kearon, Clive, et. al. Antithrombotic Therapy for VTW Disease, Chest 2012 December 1:142(6): 1698.

[91] Am. Jur. 2d, Physicians, Surgeons, and Other Healers §§ 183 to 230.

In vascular surgery diagnosis and treatment, the patient may reasonably expect that the vascular surgeon will diagnose and surgically treat the patient in accordance with the applicable standard of vascular medical and surgical care.[92] The patient may also reasonably expect that the vascular surgeon will bring to bear his or her full learning, skill, care, and experience in diagnosing and surgically treating the patient's diagnosed vascular disease or disorder.[93]

Accordingly, a bad result of a vascular diagnosis, procedure or surgery, standing alone, will not result in the imposition of malpractice liability on the part of the vascular surgeon performing the diagnosis, procedure or surgery. However, where a bad result of a vascular diagnosis, procedure or surgery occurs as a proximate result of a provable malpractice act of omission or commission on the part of the vascular surgeon performing the diagnosis, procedure or surgery, then malpractice liability may be imposed on the vascular surgeon in the particular case.[94]

[92] Jordan v. Bogner, 844 P.2d 664 (Colo. 1993).
[93] *Coleman v. Wilson*, 85 N.J.L. 203 (N.J. Ct. Err. & App. 1913). (A physician who elects to provide care in a field beyond his or her expertise is held to the standard of care for the applicable specialty); *Aves By and Through Aves v. Shah*, 997 F.2d 762 (10th Cir. 1993).
[94] Id. at 87.

a. Standard of Care

The negligence duty or standard of care or conduct may be prescribed by statute, regulation, ordinance or another written standard which then replaces or supersedes the general negligence, "reasonable person" standard of care; the violation of such a statute constitutes a deviation from the standard of care and supplies the breach of duty element of a negligence cause of action.[95]

In the case of a duty not being prescribed by statute, it is the generally accepted rule that physicians who hold themselves out to be specialists in a particular field of medicine are bound to bring to the discharge of their professional duties as a specialist that degree of skill, care, and learning ordinarily possessed by specialists of a similar class, having regard to the existing state of knowledge in medicine. This is known as the medical specialist standard of care. The question of whether a physician is a medical specialist is one of fact, and is primarily for that physician's own determination; if the physician holds himself or herself out as such, the physician must bring to his or her patients that degree of skill which a specialist in the particular medical field assumes to possess.[96]

Accordingly, a vascular surgeon who holds him or herself out as a specialist in the practice of vascular surgery is held accountable as a specialist in vascular surgery for his or her professional acts, including vascular diagnosis and the performance of nonsurgical and surgical treatment, according to what any reasonable and prudent vascular surgeon from anywhere in the U.S. would do in the same situation. It is not sufficient that the vascular surgeon, in the rendition of a vascular diagnosis or the performance of nonsurgical or surgical treatment, acted to the limits of his or her potential, acted in good faith, or did what was considered "usual" or

[95] Palo, Catherine. Deep Vein Thrombosis and Air Travel, 95 Am. Jur. Trials 1, 14 (2005).
[96] 106 Am. Jur. Trials 1, at 88.

"customary." The test or standard of vascular medical and surgical care is what another responsible vascular surgeon exercising due care and skill in vascular diagnosis or vascular surgical treatment would have done under the identical factual circumstances. This is a higher standard of care than that which is imposed upon the general medical practitioner.[97]

By now the locality rule has been virtually abrogated in most jurisdictions by general acceptance of the proposition that the prevailing standard of care applicable to medical specialists should be based upon a realistic assessment of proficiency rather than geographic proximity. Accordingly, application of a national rather than a local standard of care to gauge the skill and competency of a vascular surgeon seems entirely appropriate. This is buttressed by the rapid dissemination nationwide of vascular diagnostic and surgical procedure methodologies in peer-reviewed medical journal articles, and standardization across the U.S. in the methodology of vascular diagnosis and treatment.[98]

Patients undergoing major orthopedic surgery, represent a group that has a particularly high risk for VTE, and routine thromboprophylaxis has been standard of care for more than 20 years. Randomized clinical trials have demonstrated that the rates of venographic DVT and proximal DVT 7 to 14 days following major orthopedic surgery in patients who received no thromboprophylaxis are approximately 40 to 60% and 10 to 30%, respectively. With the routine use of thromboprophylaxis in these patients, fatal PE is now uncommon, although symptomatic VTE continues to be reported in 1.3 to 10% of patients within 3 months after surgery.[99]

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[97] *Fusilier v. Dauterive*, 764 So. 2d 74 (La. 2000); *Franklin v. Toal*, 19 P.3d 834 (Okla. 2000), as corrected, (Feb. 26, 2001); *Douglas v. Children's Hosp.*, 69 So. 3d 434 (La. Ct. App. 4th Cir. 2010), writ denied, 51 So. 3d 10 (La. 2010) and writ denied, 51 So. 3d 15 (La. 2010).

[98] *Shumaker v. Johnson*, 571 So. 2d 991 (Ala. 1990); *State Bd. of Medical Examiners v. McCroskey*, 880 P.2d 1188 (Colo. 1994*); Francoeur v. Piper*, 146 N.H. 525, 776 A.2d 1270 (2001); *Macy v. Blatchford*, 330 Or. 444, 8 P.3d 204 (2000).

[99] Geerts, et al. Prevention of Venous Thromboembolism, at 404S.

Accordingly, every trauma unit should develop a management guideline fin the prevention of VTE, and every trauma patient should he assessed for his or her VTE risk and should be prescribed optimal thromboprophylaxis consistent with thromboembolic and bleeding risks. The use of LMWH,[100] started once primary hemostasis has been achieved, is the most efficacious and simplest option for the majority of moderate-risk and high-risk trauma patients.[101]

For example, the evidence in a malpractice action supported the circuit court's finding that the estate of a patient, who died of a pulmonary embolism shortly after a hysterectomy, failed to establish a nationally recognized standard of care as to the use of sequential compression devices. The court noted that the burden was on the estate of the patient to establish a nationally recognized standard of care as to the use of sequential compression devices and the breach of that standard in the malpractice action. In this case, even though the estate's expert felt that the doctor did not follow the standard of care for patients who were at low risk of developing deep vein thrombosis or pulmonary embolism, the expert offered no peer-review literature to support his position and he offered no literature or studies to support that his position was the nationally recognized standard of care.[102]

Further, the evidence was sufficient to show that a treating physician's failure to diagnose a pulmonary embolism and the patient's continued treatment for pneumonia based on the information available to him did not fall below the standard of care, as required to support the medical malpractice action after the patient died from the pulmonary embolism. The physician was informed by a radiologist that the lung scan showed a low probability for embolism, the

[100] LMWH: Low molecular weight heparin (an anticoagulant).
[101] Id. at 421S.
[102] *Ervin ex rel. Wrongful Death Beneficiaries v. Delta Regional Medical Center*, 55 So. 3d 190 (Miss. Ct. App. 2010), cert. denied, 56 So. 3d 574 (Miss. 2011).

patient's symptoms improved with the treatment for pneumonia, the patient's white blood cell count was consistent with pneumonia, and an invasive pulmonary angiogram to investigate a possible embolism would have been inappropriate due to the risks associated with the procedure, in view of the patient's recent surgery, age, and lack of physical activity.[103]

While the identification of the applicable standard of care in a medical malpractice action is a question of law, the ultimate determination of whether a party deviated from the standard of care, and was therefore negligent, is a question of fact. To resolve the issue, a finder of fact must determine what conduct the standard of care would require under the particular circumstances presented by the evidence and whether the conduct of the alleged tortfeasor conformed with that standard.[104]

Generally, expert testimony on the standard of care is required. To qualify as an expert witness in a medical malpractice action, a witness who does not practice the same specialty as the defendant must demonstrate that the witness is familiar with the standard of care applicable to the defendant's school or specialty and that his or her familiarity is sufficient to enable the witness to give an expert opinion as to the conformity of the defendant's conduct to those particular standards and not to the standards of the witness's school and, or, specialty if it differs from that of the defendant. An estate's expert, who was a vascular surgeon, presented significant evidence that the standard of care for the diagnosis of deep vein thrombosis (DVT) did not vary based on whether the patient presented herself to a family practitioner, an emergency room physician, or a specialist in vascular disease. Accordingly, the expert's lack of recent experience in emergency medicine did not render him unqualified to testify as to the standard of care required of an emergency room physician in a medical malpractice action brought by the estate

[103] *Gee v. Treece*, 365 Ill. App. 3d 1029, 851 N.E.2d 605 (5th Dist. 2006).
[104] *Murray v. UNMC Physicians*, 282 Neb. 260 (2011).

of the patient who died as a result of a pulmonary thromboembolism. The physician was asked to "rule out" DVT much as the surgeon had been asked to do in the surgeon's practice.[105]

b. Elements and Requisites of Vascular Surgery Malpractice

A breach of duty by the defendant and a causal connection between the defendant's breach of duty and the resulting harm to the plaintiff are essential elements of a cause of action in negligence. A defendant's negligence is "a cause" of a plaintiff's injury or damage if it was a substantial factor in producing the injury or damage. The element of proximate cause can be established through a chain of circumstances from which the ultimate fact required to be established is reasonably and naturally inferable.[106]

The elements of damages in an action involving thrombophlebitis are the same as the elements of damages in personal injury actions generally. The disabling effects of thrombophlebitis hold major implications for future earning power. In workers' compensation and personal injury actions, it is essential to establish the physical dysfunctions that exist, and the degree to which they restrict the victim's movements, strength, and endurance. In personal injury actions, it is important to demonstrate how those permanent injuries translate into economic loss. Counsel should consider the value of expert testimony by a vocational expert on the issue of physical restrictions and the potential for rehabilitation, and by an economic expert on the financial impact of the disability and the adaptations it requires.[107]

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[105] *Schutte v. Mooney,* 165 Ohio App. 3d 56, 844 N.E.2d 899 (2d Dist. Montgomery County 2006).

[106] *Sheridan v. St. Luke's Regional Medical Center*, 135 Idaho 775 (2001); *Neade v. Portes*, 193 Ill. 2d 433 (2000); *Merriam v. Wanger*, 2000 ME 159, 757 A.2d 778 (Me. 2000); *Francoeur v. Piper*, 146 N.H. 525 (2001); *Powell v. Margileth*, 259 Va. 244 (2000); *Paul v. Skemp*, 2001 WI 42 (2001).

[107] *Vermilya v Nationwide Mut. Ins. Co*. 280 Pa Super 504, 421 A2d 835 (1980).

Future and past medical treatment are important items of damages in a personal injury action for thrombophlebitis. If the trial is held before all medical treatment is completed, the court should be presented with detailed evidence as to the need for such treatment, and of the best possible estimate of the cost of the future procedures. The cost of monitoring the plaintiff's condition for a reasonable period of time is a readily ascertainable element of such future damages. Early settlement of the plaintiff's claim can be risky in cases where there is a significant possibility of delayed onset of thrombophlebitis or its subsequent complications.[108]

VI. The Missing Link: Causation

In the vascular surgery malpractice action, an essential element which must be established by a preponderance of the trial evidence is proof of causation, or proximate cause. The trial evidence must show that plaintiff patient's physical injuries were actually or proximately caused by a malpractice act or omission in the defendant's rendition of a vascular diagnosis, or the performance of a vascular procedure or surgery. Actual cause in this context may be expressed as follows: But for the negligent conduct of the vascular surgeon in diagnosis or vascular surgical treatment, the physical injuries suffered by plaintiff patient would not have occurred.[109]

In contrast, proximate cause is that in which the physician or heath care provider's act or omission was a substantial factor in bringing about the loss or damage. Both proximate and actual cause is difficult to prove and is often where plaintiffs fail in proving their case. Breach of the standard of care can come in many forms: failure to promptly diagnose and treat patient; failure to take adequate patient history including current symptoms; failure to take adequate

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[108] *Nolan v. Union College Trust of Schenectady*, 858 N.Y.S.2d 427 (3d Dep't 2008), leave to appeal denied (N.Y. Sept. 11, 2008); *Freeman v Kirkland,* 584 NYS2d 828 (1992, 1st Dept); *Mink v Metro-North C. R. Co.,* 583 NYS2d 837(1992, 1st Dept).
[109] 106 Am. Jur. Trials 1, at 95.

history of patient risk factors (example: prior blood clotting problems, family history of clotting disorders, smoking, obesity, etc.); failure to conduct adequate physical examination; failure to order imaging diagnostics; failure to review or interpret imaging properly; failure to order other laboratory tests (example: blood test, etc.); failure to record data in medical record; failure to admit patient for emergency care, but none of this matters unless one can determine that this was at least a substantial factor in creating or causing the injury suffered.

Establishing causation of thrombophlebitis can be complicated by the fact that the precipitating factors in the disease are intricately interrelated. Any one of these factors has the potential for constituting a cause different from the one being claimed by the plaintiff. Because of this, if it is supported by the facts, the defense should propound the theory that the major contributing cause of the plaintiff's condition was a cause unrelated to defendant's conduct. Also, a defendant physician may be able to show that the patient had a preexisting condition or was predisposed to thrombophlebitis.[110] The defendant may concede that the condition complained of did not exist before the incident, while at the same time showing that the alleged condition developed from, or was caused by, a preexisting abnormality or proclivity, making the injury inevitable regardless of the alleged negligence.

Preexisting condition can be an absolute defense if it is found that the defendant's conduct played no substantial role in the current manifestation or exacerbation of the disease. Predisposition can be effective in a similar way, if it is proven that the onset of the condition complained of was inevitable, thus negating the causal link that is the source of liability. For example, evidence of preexisting heart or kidney disease, cancer, or a predisposing condition

[110] See, for example, Witherell v. Weimer, 118 Ill. 2d 321 (1987) (the patient's thrombophlebitis was found to be a result of the patient's inactivity and long car rides, not the physician's prescription of birth control pills).

such as obesity or multiple pregnancies would tend to show that plaintiff's thrombophlebitis was not caused by defendant's conduct. In establishing preexisting condition, the defense should show that the plaintiff had symptoms of the condition prior to the incident complained of. An examination of the injured leg at the time of the injury is important in this regard, because the examination may reveal a preexisting abnormal condition of the blood vessels in the legs that would explain the otherwise unlikely development of such complications following the injury. Similarly, an examination at the time of injury, if negative for thrombophlebitis, may be an important means of refuting or minimizing the causal link between the injury and a related subsequent complication of thrombophlebitis.

The case law regarding pulmonary embolisms and claims of malpractice demonstrate the struggle plaintiffs in the past have had with this issue.

For example, although a university medical center breached the standard of care by failing to use anti-embolic stockings during the patient's breast reduction surgery, there was insufficient evidence that the non-use of the stockings was a proximate cause of the patient's death from a blood clot. In this case, no medical expert testified that it was more probable than not that the medical center's failure to use the stockings caused the patient's fatal pulmonary embolus, and while there was testimony that the anti-embolic stockings could help prevent formation of blood clots during surgery, there was no expert testimony that the stockings more likely than not would have prevented the patient's fatal pulmonary embolus.[111]

In *Lyon v. Bryan*,[112] the jury's finding that the physician's negligence did not cause the patient to suffer injuries was supported by adequate evidence. In this action, the patient alleged that the physician negligently failed to diagnose a postoperative blood clot condition that caused

[111] *Young v. University of Mississippi Medical Center*, 914 So. 2d 1272 (Miss. Ct. App. 2005).
[112] *Lyon v. Bryan*, 2011 UT App 256, 262 P.3d 1199 (Utah Ct. App. 2011).

the patient to suffer a pulmonary embolism. The court reasoned that the jury was free to disregard the expert witness's testimony on causation even though the expert's opinion was unchallenged. The jury verdict was not internally inconsistent based upon the finding that the physician was negligent but did not cause the patient's injuries. Thus, no new trial was warranted. Evidence was introduced that questioned the accuracy of the physician's notes, and since neither the jury instructions nor verdict form limited the jury's consideration of the physician's negligence, the jury could reasonably have concluded that the physician was negligent in failing to take and keep proper notes but that this failure in no way caused the patient's injuries.

In a medical malpractice action, the plaintiff failed to show that the absence of a specific discharge instruction from the emergency-room physician to the patient concerning avoidance of deep vein thrombosis during recovery from a small fracture of the greater trochanter of the right leg was a proximate cause of the development of the blood clot that dislodged, traveled to the heart, and caused the patient's sudden death. In this case, the widower did not show that the injury more likely than not resulted from the physician's alleged negligence in failing to instruct the patient to try to be as mobile as possible.[113]

Similarly, a patient who was prescribed a birth control patch failed to demonstrate that the patch manufacturer's alleged failure to warn a prescribing physician of the increased risk of blood clots associated with the use of hormonal contraceptives was a cause-in-fact and proximate cause of the patient's development of deep vein thrombosis (DVT) in a left calf vein. This was because no additional information regarding the patch would have changed the physician's

[113] *Guadagno v. Lifemark Hospitals of Florida, Inc*., 972 So. 2d 214 (Fla. 3d DCA 2007).

decision to continue the patient's use of the patch. In this case, the patient had been aware of the risks associated with use of the patch.[114]

Another complication involving causation is the patient itself. If it is negligence on the part of physician not to give the proper discharge instructions to a patient to prevent a clot, it is equally negligent for the patient not to follow those instructions. For example, a plaintiff who brought an action for medical malpractice alleging that negligence on the part of a physician in treating her broken leg had caused her to suffer thrombophlebitis, unsuccessfully contended that the trial court had abused its discretion in allowing the physician to present a comparative negligence defense - a defense based on the plaintiff's failure to follow postoperative instructions—to the jury. The court held that the plaintiff's testimony on cross–examination, coupled with the testimony of the defense experts was substantial, competent evidence to support the jury's finding of comparative negligence, and the trial court properly refused to disturb the jury's verdict finding that the plaintiff was 45% comparatively negligent. Specifically, the court pointed to the plaintiff's admission, on cross-examination, that during recovery and contrary to the physician's advice, she had not maintained her legs in an elevated position above her heart, and she had not quit smoking. Additionally, the court observed, the plaintiff testified that during the last three or four days preceding the attack of thrombophlebitis, she had no activity, despite the physician's instructions to do otherwise.[115]

The issue of causation in the legal context stems from the ultimate fact that the causation and formation of thrombosis and pulmonary embolism is hidden and underdeveloped. Medical researchers do not know exactly how the development of venous thrombosis is affected by alterations in blood flow (including stasis), vessel wall damage with exposure of sub-endothelial

[114] Legard v. Ortho-McNeil Pharmaceutical, Inc., 833 F. Supp. 2d 775 (N.D. Ohio 2011).
[115] Nordt v. Wenck, 653 So. 2d 450 (Fla. 3d DCA 1995).

structures, and altered blood coagulability. But it is likely that each of these factors contributes to thrombosis, although the contribution of each factor might vary under differing circumstances.[116] In any event, interest in the cause of venous thrombosis is now moving away from these relatively simple concepts. More attention is being focused on the balance between activator and inhibitor substances secreted by vessel walls, and between levels of procoagulants and anticoagulants circulating in the bloodstream. Many of these compounds exist in a delicate balance in which inhibition and activation are evenly matched. Alteration of this balance may encourage thrombosis. The development of methods capable of measuring minute quantities of activators and inhibitors helps researchers determine the role of these compounds in the development of thrombosis.[117] Until this research is completed and more is known about the formation of thrombosis, this element will be the hardest to prove in any plaintiff's malpractice claim. Outside clear cases of negligence on behalf of a vascular surgeon or medical professional, causation will be the missing link necessary for any recovery despite this horrible and harmful disease.

VII. Conclusion

In conclusion, blood clots and deep vein thrombosis are very serious conditions that must be met with equally serious medical and legal efforts in order to prevent them from becoming fatal. The medical ramifications are clear, while the legal considerations are highly inconclusive. Whether it is litigation arising out of a vascular diagnosis or treatment, DVT and pulmonary embolism create a vast number of issues that must be solved in order to reduce the incidence of this condition. One of the hurdles we must tackle as a medical and legal community is the issue

[116] Ogston, Venous Thrombosis: Causation and Prediction 56 (1987).
[117] Browse, Burnand & Thomas, Diseases of the Veins: Pathology, Diagnosis and Treatment 456 (1988).

of causation and its relationship to pulmonary embolism and blood clots generally. The initiation of blood clots can arise from a host of different sources, and understanding the timing of the onset or initiation of thrombi is critical. Better diagnostic tools and a better understanding of this conditions will improve the ability of the scientific, medical and legal communities to determine the potential signs and symptoms that accompany this condition. These will also help determine what preventative measures both citizens and physicians can take to stop clots from reaching the lethal points in our bodies. Helping to understand the legal ramifications surrounding this condition can only better the understanding of how this condition can be prevented, and this forensic analysis of pulmonary embolism aims to do just that.