**100 Questions & Answers About Deep Vein Thrombosis and Pulmonary Embolism**

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The Foundation for Women & Girls with Blood Disorders is pleased to provide excerpts from *100 Questions & Answers about Deep Vein Thrombosis and Pulmonary Embolism*, written by leading experts at Duke University, Drs. Andra James, Thomas Ortel and Victor Tapson.

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*100 Questions & Answers about Deep Vein Thrombosis and Pulmonary Embolism* is a comprehensive resource of questions and answers about clotting disorders and how to manage patient issues. The following are 10 questions from this book. Questions will be updated bi-monthly. Complete copies of the book can be ordered on Jones & Bartlett Learning [[http://www.jblearning.com/catalog/9780763757670/]] or Amazon.com.

**BASIC INFORMATION**

**1. What is blood?**

Blood is a fluid that transports essential substances around the body. It is composed of proteins, other molecules and cells. The main type of cell in the blood is the **red blood cell**. Red blood cells contain hemoglobin, a molecule that has the unique ability to pick up oxygen in areas of the body where the concentration of oxygen is high (the lungs), carry it to other parts of the body, and release it where the concentration of oxygen is low (arms, legs, and organs other than the lungs). Oxygen combines with energy sources derived from food to provide “fuel” to individual cells within the tissue of organs and extremities ensuring that the cells can survive and function. The process produces carbon dioxide which must be then carried away. Hemoglobin also has the ability to pick up carbon dioxide where the concentration of carbon dioxide is high (arms, legs and organs other than the lungs) and release it where the concentration is low (the lungs). In summary, the red blood cells carry oxygen to the cells within the tissue of organs and extremities (arms and legs) and carry carbon dioxide away.

The other types of cells found in blood are white blood cells and **platelets**. White blood cells fight infection. Platelets help prevent blood from leaking out of injured blood vessels.
2. What is the purpose of blood vessels?

The purpose of blood vessels is to carry blood to organs and extremities. Two major types of blood vessels exist—arteries and veins.

Arteries are thick, muscular vessels that carry blood away from the heart. Within organs and extremities, arteries branch into smaller vessels called arterioles; the arterioles in turn branch into capillaries. Capillaries are thin, tiny vessels that allow molecules, fluids, and even some cells to travel in and out of them. Veins carry blood back to the heart from organs and extremities. Capillaries branch into larger blood vessels called venules; the venules branch into veins. In contrast to the arteries, the pressure in the veins is very low. Because veins carry blood back to the heart under low pressure and against gravity, they contain valves that open with forward flow of the blood (when the heart contracts) and close to prevent back flow (when the heart relaxes).

Blood returns from organs and extremities to the right side of the heart through a very large vein called the vena cava. The “superior” branch of the vena cava carries blood from the upper part of the body and the “inferior” branch of the vena cava carries blood from the lower part of the body. The right side of the heart—particularly the right ventricle—then pumps blood through a very large artery called the pulmonary artery, to the lungs. The pulmonary artery branches into the left pulmonary artery (which carries blood to the left lung) and the right pulmonary artery (which carries blood to the right lung). Inside the lungs, the right and left pulmonary arteries branch into smaller arterioles and even smaller capillaries. Carbon dioxide passes out of the capillaries into the air sacs (alveoli) of the lungs and, simultaneously, oxygen passes out of the alveoli into the capillaries. The newly oxygenated blood travels from the capillaries through the venules and pulmonary veins into the left side of the heart, where it is pumped by the left ventricle through a very large artery, called the aorta, to the rest of the body.

3. How and why does blood clot?

Because normal blood flow is necessary to supply oxygen to organs and extremities and to carry carbon dioxide away from these tissues, damage to a blood vessel could jeopardize life-sustaining functions by allowing blood to leak out. All animals, including humans, have an inborn mechanism by which a possible leak at the site of blood vessel injury is plugged. This mechanism is called blood clotting or coagulation.

Blood vessels can be injured in many ways, including minor trauma, serious injuries, or surgery. Arteries can also be damaged by certain disease processes such as atherosclerosis, commonly called, “hardening of the arteries.” Ordinarily, blood vessels are lined by a smooth, slippery surface called the endothelium. When blood vessels are injured, the endothelium is damaged and the tissue underneath the endothelium, called the subendothelium, is exposed. The subendothelium is rough and sticky. As a consequence, platelets, which are the cells that prevent blood from leaking out of an injured blood vessel, stick or adhere to the subendothelium where it is exposed. In the process, the platelets change their shape from a disk to a globular shape (like an ameba). During this shape-changing process, certain internal structures or granules are disrupted and release substances that activate the platelets. Activated platelets have receptors on their surfaces that allow them to stick to one another (that is aggregate). Aggregated platelets form a plug at the site of a possible leak.

The plug remains just a clump of platelets until a mesh or net made of a substance called fibrin surrounds the cells. Fibrin is solid and is formed from fibrinogen, a specialized protein or clotting factor that is found in blood. When a blood vessel is injured, the exposed subendothelium causes a protein
known as “tissue factor” to be exposed to the blood. The tissue factor sets off a chain reaction, called the coagulation cascade that activates a whole series of clotting factors. The last step of this chain reaction is the conversion of fibrinogen into fibrin, which forms the mesh that holds the platelets firmly in place. The clot (also known as a thrombus), therefore, is made up of fibrin, platelets and other cells, particularly red blood cells that are trapped in the process.

Besides blood vessel injury, two other factors are associated with the development of blood clots: interruption of blood flow which results in slow, sluggish or nonexistent blood flow, and an increased tendency within the blood to form clots. The identification of these three factors is associated with the development of blood clots is attributed to the famous nineteenth century German pathologist, Rudolph Virchow (although he did not actually describe them). Virchow did describe the process whereby some clots detach from the subendothelium, travel through larger blood vessels, and become lodged in smaller, remote ones. Blood vessel injury, interruption of blood flow, and the increased tendency toward blood clotting are, therefore, commonly called Virchow’s triad.

Because clots are necessary to prevent blood from leaking out of blood vessels after injury, individuals who have too few platelets, abnormal platelets, platelets that do not function normally or deficiencies of clotting factors may not form normal clots and may suffer from excessive bleeding. Conversely, when clots form when and where they should not, serious consequences—including death—may occur. For instance, if a clot forms in the arteries supplying the heart (the coronary arteries), blood flow is blocked, the oxygen supply is cut off, and the cells in the heart begin to die, resulting in a myocardial infarction, heart attack. If a clot forms in the arteries supplying the brain, blood flow is blocked, the oxygen supply is cut off, and the cells in the brain begin to die, resulting in a cerebrovascular accident (stroke).

4. What is deep vein thrombosis?

Thrombosis is another word for blood clot. Deep vein thrombosis (DVT) refers to the formation of a blood clot in one of the deep veins of the body, usually a vein in the muscle of one of the legs. There are two types of thrombosis are distinguished: nonocclusive thrombosis which does not completely block a vein, and occlusive thrombosis, which does completely block the vein. A blood clot or thrombosis may not get noticed, or produce symptoms, until it completely occludes a vein.

5. How common is DVT?

The chances of developing DVT are about 1 in 1000 per year, although certain factors greatly increase this risk. Young people are less likely than older people to develop DVT. The cumulative chance of developing DVT over a lifetime is 2 percent to 5 percent. While certain conditions can provoke DVT (such as cancer, surgery, and being confined to bed), this condition may also occur spontaneously. Approximately 20 to 40 percent of people who develop spontaneous DVT have an inherited or acquired predisposition to thrombosis or thrombophilia (discussed later in this Q&A series). An estimated 300,000 first-time cases of DVT occur in the United States every year.

6. Where does DVT occur?

In a study of more than 5000 people with DVT, a blood clot occurred in one leg in 77 percent of cases and both legs in 12 percent of cases. In 11 percent of cases, the DVT occurred in an arm. In less than 1 percent of individuals, DVT occurred in the veins of the brain, neck, liver, or pelvis or inferior vena cava (the very large vein that carries blood back to the heart from the lower part of the body). Thus, while such cases are rare, blood clots due occur in sites other than the legs.
Dena’s story:
When I was 23 years old, I started having severe pains in my stomach—so bad that I couldn’t sit up straight. After waiting a few days, and with the pain not subsiding, I visited my family physician and he, unknowingly, gave me something for stomach bloating. Needless to say, that didn’t work, and after a couple of days, I went back to him again. He could not find out what was causing my pain so he sent me directly to a surgeon who specialized in laparoscopic procedures. The surgeon thought that maybe it was my appendix, so he decided to go ahead and do an exploratory laparotomy. He removed my appendix, which was normal; tested my lymph nodes for cancer, which were normal, performed tests for sexually-transmitted diseases, which were normal; and so on.

When I continued to get worse (my weight went from 119 to 156 in a matter of days), my mother was adamant that I be moved to another hospital. I was transported immediately to another hospital and a specialist was called in. After seeing the pain I was in and realizing the urgency of the situation, he performed emergency surgery. He found a huge section of my small intestine was dead and removed almost 3 feet of it. He said he was almost positive that a blood clot in my intestines had started this whole ordeal. He asked me if I had taken karate, been kicked in the stomach, or had any trauma to that area, which I hadn’t.

The first 48 hours after surgery were critical. The doctors were unsure if I would make it. If I did, they said I may have to wear a colostomy bag. I was very lucky, and all of my organs regained proper function. After surgery and during my 3-week stay in the hospital, I learned that a blood clot had formed in my superior mesenteric vein (a vein in the intestines) and small clots had formed in both common femoral veins (the veins in my groin).

Jennifer’s story:
My story started when I was 27 years old and was pregnant. I was nearing my due date when my doctor discovered my blood pressure was higher than it had been all through my pregnancy. My blood pressure had been normal up until this point. It continued to be high, so they sent me to the hospital to do a nonstress test, but everything appeared normal. Once I passed my due date, the doctor recommended that I be induced. I went in early in the morning on a Thursday. My husband and I were very excited. I gave birth later that night to our beautiful and healthy daughter. There were no problems with the delivery and we both went home three days later.

Monday afternoon, I started having a headache on the back top part of my head. Before I knew it, it was a pounding and I was in a lot of pain. I called my family physician and described the throbbing headache I was having. She told me to go to the emergency room. My husband took me, and when I talked to the doctor there, he said that I may be experiencing an after-effect headache from the epidural that I had had during my labor. Because the records didn’t indicate there had been any problems when the epidural was administered, he wasn’t sure. He also thought the headache could be due to preeclampsia. He prescribed Tylenol with codeine and advised me to come back in a few days if things didn’t get better.

The headaches continued, but the medicine helped to mask them a little bit over the next few days. When I woke up Friday morning, I was feeling better than I had all week. Since my mother was taking care of our daughter and I was anxious to get back into my regular routine, I went downstairs and started to wash dishes. As I was doing this, my left arm started to go numb and the next thing I knew it was shaking uncontrollably. This really made me nervous. Thank goodness my mother was there to calm
me down and call my family physician. My doctor thought I was having a seizure and sent me to a neurologist, whom I saw later that day.

The arm shaking happened again while I was having my appointment with the neurologist. He told me I was having a seizure and wanted to know if I had a history of having seizures or other medical problems. I told him I had never had any seizures or serious medical problems. The urologist told me he wanted me to have an MRI that evening to rule out a blood clot, a brain tumor, or an aneurysm. I was very nervous and was so glad my husband was with me because I didn’t understand why all this was happening. I went to an office downstairs from the neurologist’s office to have the MRI done. Once we went back up to his office, he told me nothing showed up on the MRI. Because of the arm seizures, he prescribed an antiseizure medication. He said it could take a few days to take full effect, so I should take it easy.

The next day my husband brought me some lunch as I was resting. All of a sudden, my head turned right around uncontrollably and I went into a full body seizure. I don’t remember having the seizure, but afterward I remember my husband on the phone with 911, asking them to send an ambulance. I was so scared. I had never ever experienced anything like this before.

Shortly after I was taken to the emergency room, I was admitted to the hospital and the neurologist became my main doctor. During my stay, I went through a series of tests to determine a diagnosis. The tests included an angiogram, an electroencephalogram (EEG), a spinal tap, an echocardiogram (a heart ultrasound) and a CT scan. Although the spinal tap showed some “old” blood was in the spine and brain, and the EEG showed some abnormalities, most of the tests came back normal. My doctor didn’t know what the problem was, but he thought it could be epilepsy or preeclampsia. The doctors did not discover the clot during all of this testing, but I continued to have symptoms. My headaches occurred daily, and I even lost motor skills on my -- the left side of my body. I experienced numbness in my arms and legs on my left side, and I had tingling above my lips. I did have a couple of seizures during my stay, but the seizures subsided as I continued to take the medication. After about two weeks, the doctor let me go home, even though I continued to have headaches and was not feeling well. There was still no diagnosis.

For the next three days I lay listless in my bed with constant pain in my head and no appetite. No over-the-counter medicine helped. My husband called the doctor each day, and because my condition was getting worse, on the third day, the doctor ordered another MRI. This time, a dye was injected into a vein that helps “light up” the veins in the brain. That is when they discovered that I had a blood clot in a vein in my brain.

DIAGNOSIS AND SYMPTOMS

7. What are the symptoms of DVT?

DVT usually occurs in a leg or, less often, an arm. Sometimes a clot is small or only partially obstructs a blood vessel, and there are no symptoms. The classic symptoms, however, are pain, swelling, tenderness to the touch along the course of the vein, redness, or in some cases, even bluish discoloration of the affected arm or leg. It is possible to have these symptoms without having DVT. In fact, a diagnosis of DVT is confirmed in only 25 percent of such cases. Other conditions that can cause the same or similar symptoms are an infection, a bruised muscle, or the rupture of one of the fluid sacs or “bursa” that cushion the tissue around the knee. This condition is called a ruptured Baker’s cyst.

Marvin’s story:
I have had four episodes of DVT or pulmonary embolism. In retrospect, the first episode, which occurred in my right leg, was never diagnosed and eventually got better on its own.

The second episode, which was the first recognized episode, began with a severe pain in the left calf muscle. It felt much like a cramp but did not yield to any kind of manipulation. The onset came 10 days after a gastroenterologist had removed a polyp from my colon. Wondering if there might be a connection, I asked the gastroenterologist. He thought not, but suggested I see my family physician. Because my family doctor had died recently, I had to see a doctor who was not known to me, a general practitioner. When I visited him, he bent the toes of my left foot back and because I didn’t wince, the doctor concluded the pain in my calf was not a clot but inflammation of the gastrocnemius muscle (a muscle in the calf), for which he prescribed a regimen of butazolidin (a nonsteroidal anti-inflammatory drug that is no longer on the market). It did not relieve the symptoms or alter the condition.

Nonetheless, because I had been reassured that my pain was only muscle inflammation, I kept my commitments which included an appearance on a television show. I drove 120 miles to New York City, stayed overnight, appeared for the taping, and drove home the next day. All the while the severe pain remained untouched by pain relievers. I checked back with the gastroenterologist and he asked me to come in immediately for an emergency visit. When he saw the swollen and painful leg, he ordered a gurney and had me transferred to the hospital, where I spent five days for the initial treatment of a DVT.

8. What is the difference between DVT and superficial vein thrombosis or superficial thrombophlebitis?

Superficial vein thrombosis is also called superficial thrombophlebitis. Symptoms include pain, swelling, and redness along the course of a superficial vein. While DVT occurs in a deep vein, a superficial vein thrombosis occurs in a superficial vein. Superficial veins, which are sometimes visible on the arms or legs, collect blood underneath the surface of the skin. They are not deep. They do not carry blood directly back to the heart, but instead transfer blood to the deep veins through small communicating veins.

Because superficial veins do not carry blood directly back to the heart, the consequences of a superficial vein thrombosis are not the same as those associated with DVT. Even if the clot or a piece of the clot breaks free, it cannot fit through the small communicating veins and, therefore, cannot travel to the heart and wedge itself into one of the pulmonary arteries or its branches, resulting in a pulmonary embolism (PE) (See Question 10). Therefore, superficial vein thrombosis does not necessarily require treatment with anticoagulation unlike DVT and PE do. Treatment usually consists of heat; non-steroidal anti-inflammatory drugs, such as ibuprofen (Motrin®, Advil®, Nuprin®) or naproxen (Aleve®, Orudis KT); fitted elastic compression stockings; and elevation of the affected extremity. Symptoms usually disappear in two to six weeks. Sometimes anticoagulation is used to help alleviate the symptoms. If the condition worsens or does not improve, DVT should be considered.

9. How is DVT diagnosed?

In the diagnosis of DVT, the physician considers the patient’s specific risk factors, the patient’s symptoms, the physical examination, other possible explanations for the symptoms and the results of objective tests, such as some method of imaging, that is, seeing the clot.

Duplex Ultrasound
The first method that is usually performed in an attempt to image the clot is ultrasound, specifically Duplex ultrasound. Duplex refers to the two parts of the process.

In the first part of the process, brightness modulation ultrasound (also known as B-mode ultrasound) is used to obtain an image or picture. The ultrasound machine creates high-energy sound waves (ultrasound) that are bounced off internal tissues and make echoes. The patterns of these echoes form an image, which is then shown on the screen of the machine. While imaging the deep veins of the leg, the sonographer (the person who operates the ultrasound machine) tries to collapse or compress the veins. If a vein cannot be compressed because a clot prevents the vein from collapsing, a DVT diagnosis is made. The ability to completely flatten a vein with compression is the most useful way to be certain that a clot is not present.

In the second part of the process, Doppler ultrasound is used to detect abnormalities of blood flow. Sound waves are bounced off the blood within a vein. Flowing blood changes the sound waves by the “Doppler effect.” The ultrasound machine can detect these changes and determine whether blood within a vein is flowing normally. Absence of blood flow confirms the diagnosis of DVT.

Duplex ultrasound successfully identifies 95 percent of deep vein thromboses that occur in the large veins above the knee. The ability of duplex ultrasound to detect DVT in the large veins above the knee is so good that when the test is positive, no further testing is necessary and treatment may be started. Conversely, if the test is negative, the chance that there is a DVT is so small that treatment may safely be withheld.

The technique is not as good at detecting DVT that occurs below the knee or in the calf veins, however. Duplex ultrasound successfully identifies only 60 to 70 percent of calf vein thrombosis when such a diagnosis is made, it is correct only 60 to 70 percent of the time. While calf vein thrombosis account for 20 percent of all DVT cases, only one in five of these thromboses ever grows in the first week or two after it is initially suspected. Also, calf vein thromboses are less likely to break free and travel to the lung or “embolize.” Therefore, if the ultrasound is negative, even though a DVT may be present in a calf vein, treatment may be withheld and the ultrasound repeated in five to seven days if the symptoms persist. Calf vein DVT may be treated like superficial thrombophlebitis. Most physicians prescribe anticoagulants in such cases, however, because a DVT in a calf vein can lead to larger, more proximal DVT that can break off and migrate to the lung.

**Duplex Ultrasound in Recurrent DVT**

Abnormalities of the veins are common after DVT, making it difficult to diagnose a recurrent clot. For instance, half of the time the results of the duplex ultrasound remain abnormal one year after the first episode of DVT. Consequently, if duplex ultrasound is being performed to determine whether a new clot has developed, lack of compression or absence of blood flow does not prove the existence of a new clot unless a new segment of the vein or a different vein is involved.

**Venography and Magnetic Resonance Imaging**

If the ultrasound is negative, yet the patient’s symptoms are severe or a DVT is strongly suspected, the next step is either a venogram (venography) or magnetic resonance (MR) imaging. Sometimes the ultrasound is negative because there is a clot in a vein in the pelvis, hidden from the ultrasound. Although isolated pelvic vein thrombosis is uncommon, it can occur in women who are pregnant or who have recently delivered a baby, in people who have had pelvic cancer, or in people who have had recent pelvic surgery.
Until recently, venography using x-rays was used to diagnose DVT. During venography, contrast dye (usually an iodine dye) which helps blood vessels show up clearly on x-ray, was injected into a vein in the foot. A series of x-rays of the veins was looking for blockages. Today, the use of x-ray venography has been almost entirely replaced by the use of ultrasound and magnetic resonance (MR) venography, because x-ray venography, is “invasive” and can be painful. The MR machine uses pulses of radio-frequency waves to cause hydrogen atoms to line up within tissues. When the pulse stopped, the hydrogen atoms return to their natural state. In the process, they give off a signal that the machine converts into an image. Different tissues give off different signals. Because clots give off different signals than flowing blood, MR can be used to detect a thrombosis.

MR venography does a better job of imaging the veins in the pelvis, abdomen and chest than ultrasound does. Because it does not require compression, this technology can be used to detect clots in limbs inside of plaster casts. Overall, MR may be superior to ultrasound, but it is a much more involved test and costs much more than ultrasound.

**D-dimer test**

After a blood clot starts to form, another series of reactions normally begins to dissolve (that is, lyse) the clot. Fibrin, which forms the mesh or net that holds the platelets firmly in place within a clot, is solid and is formed from fibrinogen, a specialized protein (clotting factor) that is found in blood (See Question 3). These fibrinogen molecules are strung together end-to-end and cross-linked with fibrin.

During the lysis process, fibrin is broken down or degraded by an enzyme called plasmin. Plasmin cuts the strands of fibrin on either side of what were the ends of the fibrinogen molecules. These ends are called “D” units. A dimer is a pair, and the D-dimer is a fragment of cross-linked fibrin that is a pair of two “D” units. D-dimers can be present in a variety of conditions, including the formation of a blood clot. While the presence of D-dimers does not guarantee that a blood clot is present, it is a clue that the clotting process has begun. If D-dimers are absent, however, it is very unlikely that a blood clot has begun to form. For that reason, a blood test for D-dimers is often performed to ensure that a blood clot is absent.

A number of tests for D-dimers exist. If such a test is intended to prove that a blood clot is absent, then the test should be a sensitive one (one that will detect D-dimers whenever they are present). Also, the test should be interpreted in the context of an individual’s situation.

Recent research suggests that testing for D-dimers when discontinuation of warfarin is being considered may help identify the best time to stop the warfarin. If the D-dimer test is normal, it might suggest that it is safe to stop anticoagulation and that the patient’s risk for experiencing a recurrent clot may be lower. More research is being done in this area.

**10. What are the consequences of DVT?**

After a blood clot starts to form, another series of reactions almost immediately begins to dissolve (lyse) the clot. The purpose of this process is to confine the clot to the injured area of the blood vessel, limit the size of the clot, and prevent it from growing too large. Sometimes DVT can be completely dissolved or lysed by the body’s own natural processes. Even under these circumstances, there is likely permanent damage to the vein and its valves. In fact, 5 to 30 percent of individuals who experience DVT develop a second or recurrent DVT within five years of the first episode.
Sometimes, a clot serves as a surface on which another clot forms and the clot continues to grow. As it increases in size, blood flow may be completely blocked within a vein. The clot may even extend into the larger vein to which the present vein is connected. In this case, circulation to a leg, arm or other organ may be so impaired that the limb, organ, or person's life may be threatened.

In one case out of every five cases, the clot (or a piece of it) breaks free and travels through progressively larger veins, through the vena cava, and through the right side of the heart, and then wedges into one of the pulmonary arteries or its branches, resulting in pulmonary embolism (PE). As many as 600,000 Americans are hospitalized each year for DVT and its primary complication, PE. In the United States, DVT and PE account for as many as 300,000 deaths per year.

Three to four percent of patients who experience PE, suffer chronic (persistent) obstruction of blood flow through the lungs - a condition known as chronic thromboembolic pulmonary hypertension (CTEPH). The obstruction of this blood flow increases the blood pressure in the pulmonary arteries, which carry blood from the right side of the heart to the lungs. CTEPH therefore strains the right side of the heart, causing symptoms of heart failure including shortness of breath.

**Glossary**

Blood: A bodily fluid that circulates in the arteries and veins. Blood consists of plasma (the liquid portion that contains proteins and other molecules) and cells. Blood cells include white blood cells, which fight infection; red blood cells, which carry oxygen to the tissues and carbon dioxide back to the lungs; and platelets, which are like little corks that plug up holes to stop bleeding.

Proteins: Essential molecules in the body that are made up of strings of amino acids. The genes in our DNA contain the necessary information to make all the proteins in our bodies. Examples of proteins include antibodies and blood clotting factors.

Red blood cell: A cell in the blood that carries oxygen to the tissues. Red blood cells are an important component of blood clots.

Hemoglobin: The protein molecule in red blood cells that carries oxygen from the lungs to the body's tissues and returns carbon dioxide from the tissues to the lungs. The iron contained in hemoglobin is responsible for the red color of blood.

Platelet: The smallest cell in the blood. Platelets are important for normal blood clotting; they prevent blood from leaking out of an injured blood vessel.

Artery: A vessel through which the blood passes away from the heart and to the various parts of the body.

Vein: A vessel through which blood passes from various organs back to the heart.

Vena cava: A large vein that carries blood from the tissues to the heart, and then on to the lungs to pick up oxygen. The “superior” branch of the vena cava carries blood from the upper part of the body; the “inferior” branch of the vena cava carries blood from the lower part of the body.

Pulmonary artery: The main blood vessel carrying blood from the right side of the heart (right ventricle) into the lungs to pick up oxygen. This large blood vessel divides into smaller and smaller branches deeper into the lung. The pulmonary arteries are where pulmonary emboli migrate to, and block off.
Coagulation: The process of blood clotting.

Endothelium: The lining of a blood vessel. Damage to the endothelium, such as from trauma (or a previous blood clot), makes a patient more susceptible to a blood clot.

Fibrin: A solid substance formed from fibrinogen, a specialized protein or clotting factor that is found in blood. Fibrin makes a clot more stable (harder to break up). It forms the mesh or net that holds blood platelets firmly in place.

Fibrinogen: A specialized protein or clotting factor that is found in blood. When a blood vessel is injured, another clotting factor, thrombin, is activated and converts fibrinogen to fibrin, which is the mesh or net that holds platelets firmly in place.

Tissue factor: A substance that is released from the blood vessel lining and initiates the clotting reaction.

Clot: A thrombus.

Thrombus: A blood clot.

Virchow’s triad: The three basic factors that increase a patient’s risk for deep vein thrombosis: (1) stasis (reduced mobility or immobility), (2) injury to a blood vessel, and (3) thrombophilia.

Thrombosis: The pathologic (abnormal) process whereby liquid blood forms a clot within a blood vessel or the heart.

Deep vein thrombosis (DVT): A condition in which a blood clot forms in the deep veins of the legs, pelvis, or arms. The treatment for DVT includes anticoagulant therapy.

Spontaneous DVT: A clot that forms when there are no obvious risk factors. Twenty to 40 percent of people who develop a spontaneous DVT have an inherited or acquired predisposition to thrombosis or thrombophilia.

Acquired: a condition that is not genetic (inherited) or congenital (present at birth); usually caused by environmental factors and/or other physical conditions.

Thrombophilia: A predisposition to the development of blood clots. It is sometimes referred to as “hypercoagulability.” Thrombophilia can be either inherited or acquired during a person’s lifetime.

Superficial thrombophlebitis: A blood clot or clots that form in the veins nearer to the surface.

Anticoagulation: A general term for a treatment that interferes with the ability of the blood to form a normal blood clot. Anticoagulant medicines include heparin and warfarin, and are sometimes referred to as “blood thinners.”

Fitted elastic compression stockings: Elastic stockings which ideally exert a pressure of at least 30 to 40 mm Hg at the ankle with less pressure at the knee. Fitted elastic compression stockings provide counter-pressure to veins and help return fluid that has leaked out of them back into the circulation.

Ultrasound: A test used to identify a number of medical conditions. When DVT is suspected, the inability to compress the leg veins with the ultrasound device indicates the presence of DVT. Abnormal blood flow can also be demonstrated when DVT is present.
Magnetic resonance imaging (MRI): A test that images clots in the body. While MRI does a better job of imaging the veins in the pelvis, abdomen, and chest than ultrasound does, ultrasound for the legs is usually adequate (and is cheaper). Neither test exposes a patient to radiation.

Lyse or lysis: To lyse a clot is to dissolve or destroy a clot. Lysis is the process whereby a clot is dissolved or destroyed. This process can occur naturally over time or can be accomplished by powerful, clot-busting drugs (thrombolytics).

D-dimer: A breakdown product of fibrin, which is present in a blood clot. D-dimers are not generally present in the blood unless a clot has begun to form, although the presence of D-dimers does not guarantee that a clot is present. If D-dimers are absent, it is very unlikely that a blood clot has begun to form.

Warfarin: A medicine given by mouth that interferes with blood clotting and is generally used for the prevention or treatment of blood clots. It is often referred to as a “blood thinner.”

Pulmonary embolism (PE): A blood clot from a deep vein, usually in the legs, pelvis, or arms, that breaks loose and travels through the veins and then through the heart before becoming lodged in the blood vessels in the lung. Large pulmonary emboli can be life-threatening and may need to be treated with thrombolytic drugs (“clot-busters”).

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