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

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 the University of Virginia and 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.

Diagnosis and Symptoms

11. What is the post-phlebitic or post-thrombotic syndrome?

Approximately 30 percent of people who experience DVT develop chronic symptoms as a consequence of permanent injury to veins and their valves. These chronic symptoms may not occur right away. In fact, after one to two years, only 10 to 20 percent of people experience these symptoms; after five years, however, 20 to 30 percent do.

Chronic symptoms include swelling, pain and discoloration of the skin. A rusty discoloration of the skin, caused by iron deposits from old blood, is often the first sign, followed by chronic swelling. In severe cases, the skin can break down, allowing an **ulcer** (open sore) to form. After one year, only 2 to 3 percent of people develop these ulcers; by contrast, after five years, as many as 10 to 20 percent may suffer from such an ulcer on their legs. Not surprisingly, **post-phlebitic (post-thrombotic) syndrome** is more likely to develop after DVT that was associated with symptoms as opposed to a DVT that was diagnosed by imaging alone. (See Questions 80 and 81 at the end of this document before the Glossary.)

12. What is pulmonary embolism?

The pulmonary embolism (PE) process was first described by Rudolph Virchow, the famous nineteenth-century German pathologist who called the traveling clots, *embolia*. The terms "embolus" (plural: emboli) and "embolism" are still used to describe a clot or part of a clot that has formed in one site and traveled to another part of the body. When a clot wedges itself in one of the pulmonary arteries or its branches, it is called PE.

A very large PE can block the entire trunk of the pulmonary artery (before it branches into the right and left pulmonary arteries) and cause death instantly. Pulmonary emboli that are not quite so large may block an entire right or left pulmonary artery, stopping the blood flow to an entire lung and--especially if the person already has lung or heart disease--causing death. Smaller emboli may block smaller branches of the pulmonary artery with varying consequences. When the blood supply to a small “end-artery” at the edge of the lung is blocked, oxygen to that part of the lung is cut off, and the cells in that part of the lung begin to die, resulting in **pulmonary infarction** (that is, death of lung tissue). When blood flow is blocked within a larger branch of the pulmonary artery, the normal exchange of oxygen for carbon dioxide does not take place and the entire body is affected. Pulmonary infarctions usually result from smaller clots and are unlikely to be fatal.

The likelihood of death from PE depends largely on the size of the PE. If the main pulmonary artery is completely blocked, the right ventricle (the chamber of the heart that pumps blood into the lungs) cannot get the blood into the lungs; this “right ventricular failure” then leads to death from PE. The age and health of the affected individual are also critical factors. When the person already has lung or heart disease, PE may have a more dramatic impact. While the death rate from PE may be as high as 25 percent in sick, hospitalized patients, the rate in young healthy individuals is closer to 1 percent.

13. What are the symptoms of PE?

While the vast majority of pulmonary emboli are believed to originate in the deep veins of the body, fewer than 30 percent of individuals who experience PE have symptoms of DVT. Instead, the most common symptoms are shortness of breath and chest pain. In the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study, a large study conducted by the National Heart Lung and Blood Institute of the National Institutes of Health, 73 percent of patients with PE experienced shortness of breath, 66 percent experienced chest pain, 37 percent experienced cough, and 13 percent coughed up blood. During physical examination, 70 percent had rapid breathing and 30 percent had a rapid heart rate. When doctors listened to the study participants with a stethoscope, half had abnormal sounds in their lungs and one fourth had abnormal sounds in their hearts. Fourteen percent had a fever.

In some cases, PE may occur very suddenly, without any warning. The symptoms of PE, when noted, are also very nonspecific. For example, shortness of breath and chest pain may occur with pneumonia, bronchitis, or other lung or heart problems. Chest pain may be caused by a number of problems, including muscle strain, heart problems, lung infections, stomach problems such as acid reflux or hiatal hernia, or even anxiety. Whenever a patient has symptoms such as shortness of breath or chest pain, especially when the person has other risk factors for DVT (such as recent surgery, admission to the hospital for a medical disease, or recent immobility), PE should be considered as a possibility.

14. How is PE diagnosed?

A number of different things may alert the physician that PE may be present (See Question 13). When it is suspected, testing must be considered.

Pulse Oximetry

Often, the first test performed when PE is suspected is a blood oxygen level. The simplest way to measure the blood oxygen level is with a pulse oximeter. **Pulse oximetry** is a noninvasive way (does not involve a blood draw or needle stick) to monitor the percentage of hemoglobin that is saturated with oxygen. Hemoglobin is the unique molecule in red blood cells that has the ability to carry oxygen. The pulse oximeter consists of a probe or sensor plus a computer. The probe, which looks like a padded clothespin, is placed on a relatively thin part of a person's body, such as a finger or earlobe.

Both red and infrared light are then transmitted through the tissue by the probe. Based on the absorption of the red and infrared light caused by the difference in color between the hemoglobin saturated with oxygen (red) and unsaturated hemoglobin (blue), the computer can estimate the proportion of hemoglobin that is oxygenated. The pulse oximeter then displays this result as a percentage. A blood oxygen saturation level less than 95 percent is abnormal. It may be explained by a lung or heart problem already present, such as emphysema or pneumonia, or by PE (or both).

Arterial Blood Gas

A more precise measurement of blood oxygen level is obtained from a sample taken directly from an artery with a needle or a thin tube (catheter). An **arterial blood gas (ABG)** measures the levels of both oxygen and carbon dioxide in the blood to determine how well the lungs are working. While most blood tests are performed on samples taken from a vein, an ABG is performed on a sample taken from an artery. In most cases, the artery in the wrist is used for this purpose, but other arteries may be used. The levels of blood gases are measured as partial pressures in units of millimeters of mercury (mm Hg). A partial pressure of oxygen less than 80 mm Hg is abnormal.

Chest X-Ray

A chest x-ray cannot prove that PE is present or absent because clots do not show up on x-ray. Nevertheless, a chest x-ray is a useful test in the evaluation for PE because it can find other diseases, such as pneumonia or fluid in the lungs, that may explain a person's symptoms. Occasionally, when pulmonary infarction occurs, the x-ray may suggest this diagnosis, although more testing is necessary to prove it with certainly. A normal or negative chest x-ray with a low, otherwise unexplained blood oxygen level, however, raises the suspicion for PE.

Ventilation-Perfusion Scan (VQ Scan)

A **VQ lung scan** may be a useful test to determine whether a person has experienced PE. This test evaluates both air flow (V = ventilation) and blood flow (Q = perfusion) in the lungs. About one hour before the test, a slightly radioactive version of the mineral technetium mixed with liquid protein is administered through a vein to identify areas of the lung that may have reduced blood flow. Multiple images are taken from different angles, using a special camera that detects radioactivity. For half of the images, the person breathes from a tube that has a mixture of air, oxygen, and a slightly radioactive version of the gas xenon, which reveals air flow in different parts of the lung. For the other half of the images, the camera tracks the technetium, which reveals blood flow in different parts of the lung. PE is suspected in areas of the lung that have significant "mismatches"—that is, good air flow but poor blood flow.

Except for the minor discomfort of having an intravenous catheter placed, A VQ lung scan is painless and usually takes less than an hour. The exposure to radioactivity from the test is very minor and results in no side effects or complications.

A radiologist interprets the images from the VQ lung scan and decides whether the probability of a PE is high, low, or intermediate. If the probability is high, the diagnosis is made. If the probability is low or intermediate (that is, nondiagnostic), or if the VQ scan cannot be interpreted clearly, other testing must be considered. Even when PE is ultimately proven to be present, the VQ scan may be nondiagnostic. If clinical suspicion is low and the VQ scan reveals a low probability of PE, generally no further testing is needed. A normal VQ scan means PE is not present.

Spiral Computed Tomography of the Chest

An alternative to the VQ scan is a spiral computed tomography (CT) of the chest. A spiral CT of the chest uses special equipment to obtain multiple cross-sectional x-ray images of the organs and tissues of the chest. CT produces images that are far more detailed than those available with a conventional x-ray. Many different types of tissues—including the lungs, heart, bones, soft tissues, muscles, and blood vessels--can be seen. When PE is suspected, contrast dye (usually iodine dye) is administered through a vein to make the blood vessels stand out.

During the spiral CT, radiation is emitted from a rotating tube. Different tissues absorb this radiation differently. During each rotation, approximately 1,000 images are recorded, which a computer then reassembles to produce a detailed image of the interior of the chest. The x-ray rotates as the patient passes through the CT scanner in a spiral path—hence the term "spiral" CT. The amount of radiation exposure is relatively low, and the procedure is not invasive.

Pulmonary Angiogram

If the VQ scan interpretation is low, intermediate or uncertain probability of PE, or if the spiral CT is normal yet the symptoms are still suspicious, then the definitive test is a **pulmonary angiogram**. An angiogram is an invasive test that uses x-rays to reveal blockages or other abnormalities within the veins or arteries. Contrast dye (usually iodine dye) helps blood vessels show up clearly on x-rays. During an angiogram, contrast dye is injected into a blood vessel, and its path is tracked by a series of x-rays.

A pulmonary angiogram examines the arteries that carry blood from the heart to the lungs and is performed to see if PE is present. Using x-rays in real-time (fluoroscopy), the radiologist inserts a catheter into a vein and advances it until it reaches the vena cava (the very large vein that carries blood to the heart). Next, the radiologist advances the catheter still farther into the right side of the heart and finally into the pulmonary artery, the large artery that carries blood to the lungs. The radiologist directs the tip of the catheter into the different branches of the right and left pulmonary arteries and injects the contrast dye, which illuminates the arteries on x-ray. If PE is present, it will show up as a blockage.

Risks associated with a pulmonary angiogram include the possibility of damage caused by the catheter, bleeding, and an allergic reaction to the contrast dye. The amount of radiation from the x-rays is too small to cause any harm.

Echocardiogram

An echocardiogram is an ultrasound of the heart. Doppler ultrasound, B-mode ultrasound, and M-mode ultrasound (a rapid sequence of B-mode images that allows motion to be visualized) are combined to give information about the size of the heart, the function of the valves, and the strength of the heart muscle. (Duplex ultrasound is discussed in more detail in Question 9 – see below.) The echocardiogram can spot areas of the heart that are not working well. When patients with a PE have an echocardiogram, about 40 percent will be found to have abnormalities of the right side of the heart, particularly the right ventricle. While an echocardiogram is not actually used to diagnose a PE, it can identify strain on the right side of the heart caused by a large PE as well as certain heart problems that may imitate a PE.

Initial Treatment

15. What are the goals of treatment of DVT?

The goals of treatment of DVT are fourfold: (1) to prevent DVT from growing in size, (2) to prevent DVT from recurring, (3) to prevent PE from developing, and 4) to minimize complications.

17. What is heparin?

Heparin is a naturally occurring anticoagulant. While Dr. Jay McLean is often given credit for discovering heparin while he was a medical student, it was his mentor and laboratory director at Johns Hopkins University, Dr. William Howell, and another medical student, Emmett Holt, who first isolated this natural anticoagulant from dog liver in the 1920s and coined the term "heparin." Later in the decade, at the University of Toronto, heparin was first purified in sufficient quantities from beef liver, and later beef lungs and intestines, to treat humans. Heparin is still derived mainly from beef and sometimes pork sources. It cannot be taken by mouth, but must be administered directly into a vein or injected underneath the skin.

18. How does heparin work?

During the formation of a clot, factor X and factor V work together to convert **prothrombin** (factor II) to **thrombin.** Thrombin is the clotting factor that converts fibrinogen to fibrin. Fibrin is the mesh that holds platelets firmly in place during the formation of a clot. (See Question 3 below.) Antithrombin is a natural blood thinner that blocks the conversion of factor X to its active form, factor Xa, and also blocks the conversion of prothrombin to thrombin, thereby preventing the conversion of fibrinogen to fibrin. Heparin works by multiplying the action of antithrombin 1,000-fold.

19. What is the difference between heparin and low-molecular-weight heparin?

Low-molecular-weight heparin (LMWH) is made from standard or “unfractionated” heparin that has been chemically cut or cleaved. Consequently, these partial heparin molecules are of lower weight and behave differently than standard heparin. They have relatively more activity against factor Xa and relatively less activity against prothrombin. LMWH has a longer duration of action than standard heparin and is less likely to result in bruising, bleeding, or a decrease in the number of platelets (a rare complication that develops in some patients who are treated with heparin). Because LMWH behaves differently from standard heparin, when it is monitored, different tests than those for standard heparin must be used.

20. How is the dose of heparin or LMWH determined?

When LMWH is used to treat DVT or PE, the amount of enoxaparin or tinzaparin prescribed is based on the patient’s weight. The initial dose of enoxaparin used to treat DVT or PE is 1 milligram of drug per kilogram of body weight (1 kilogram = 2.2 pounds) given every 12 hours. The initial dose of tinzaparin used to treat DVT or PE is 175 international units (IU) of anti-0factor Xa activity per kilogram of body weight given every 12 hours. (Tinzaparin doses are not calculated in milligrams.) These doses of enoxaparin and tinzaparin are considered “full doses.” Lower does are used to prevent DVT or PE.

One advantage of LMWH over standard heparin is that when full doses of LMWH are used to treat DVT or PE, blood levels do not usually have to be monitored. Monitoring is recommended in patients who have certain other conditions such as morbid obesity, kidney disease, or pregnancy. When monitored, LMWH levels are checked with a blood test that measures the medication's activity against clotting factor Xa. Standard heparin is monitored with a different blood test, the activated partial thromboplastin time.

A summary chart comparing four recent oral anticoagulants is available through Clot Connect. To view it, please click here.

21. What is the activated partial thromboplastin time?

The activated partial thromboplastin time (aPTT) test is a blood test that measures the length of time (in seconds) that it takes for clotting to occur when certain substances are added to the liquid portion of blood in a test tube. A normal result requires normal levels of clotting factors VIII, IX, XI, and XII; pre-kallikrein and high-molecular-weight kininogen; and factors V and X, prothrombin, and fibrinogen. The aPTT is used not only to detect clotting factor deficiencies, but also used to monitor heparin's effectiveness.

***Question 3: How and why does blood clot?**

Because normal blood flow is necessary to supply oxygen to organs and extremities and to carry carbon 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.

This plug remains just a clump of platelets until a mesh or net made of a substance called **fibrin** surrounds these cells. Fibrin is a 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. A **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). Blood vessel injury, interruption of blood flow, and the increased tendency toward blood clotting are, therefore, commonly called **Virchow's triad**. Virchow did describe the process whereby some clots detach from the subendothelium, travel through larger blood vessels, and become lodged in smaller, remote blood vessels.

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).

*** Question 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 duplex ultrasound 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 imaging (MRI)**. 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 then taken, 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 stops, 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 that holds the platelets firmly in place within a clot, is a solid that is formed from fibrinogen, a specialized protein (clotting factor) found in blood (See Question 3). These **f**ibrinogen molecules are strung together end-to-end and cross-linked within 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, so the **D-dimer** is a fragment of cross-linked fibrin that consists 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.

*** Question 80: Can DVT cause permanent damage?**

Most patients recover from DVT without significant problems or complications. Even patients who have very large clots with significant leg pain and swelling generally recover. Longer-term problems may occur, however.

The veins in the body have valves in them to keep blood flowing in the right direction. When a blood clot forms in the leg veins, it often forms around these valves. In some patients, the valves may become damaged so that the blood settles in the legs and doesn't move forward toward the heart as it should. Over months to years, this venous stasis (sluggish or nonexistent blood flow) may cause swelling in the legs and discoloration of the skin. Pain can develop as well. Of course, this post-phlebitic or post-thrombotic syndrome does not occur in everyone. (See Question 11.) It is variable in severity but can be quite disabling. Because of swelling and poor circulation, 10 to 20 percent of these patients develop ulcers in the skin, which sometimes require surgery.

Some patients develop these chronic problems without even having obvious DVT, presumably because the DVT was not associated with any symptoms when it occurred. That is, long-term problems may sometimes develop in patients who never even had their DVT diagnosed. Significant obesity may make these chronic leg problems even worse. Fitted elastic compression stockings may help to alleviate the symptoms from post-phlebitic syndrome.

*** Question 81: how can post-phlebitic or post-thrombotic syndrome be prevented?**

You can reduce your risk of developing post-phlebitic or post-thrombotic syndrome by wearing fitted knee-high elastic compression stockings that exert pressure at the knee. While the duration for which they should be worn is unknown, their use for the first two years after a DVT is suggested.

Glossary

Chronic thromboembolic pulmonary hypertension (CTEPH): High blood pressure on the lungs that occurs in a small percentage of patients who have had pulmonary embolism. The most common symptom is shortness of breath. This problem usually progresses but may be cured with surgery.

Ulcer: A chronic (long-standing) open sore.

Post-phlebitic (post-thrombotic) syndrome: Long-term, recurring leg symptoms that affect some patients as a result of permanent injury to veins and their valves from DVT.

Pulmonary infarction: The death of a small area of lung resulting from pulmonary embolism that occurs in a small, dead-end pulmonary artery. Pulmonary infarction often causes pain the chest or back.

Pulse oximetry: A noninvasive (no blood needed!) method of monitoring the percentage of hemoglobin that is saturated with oxygen. A low saturation may be caused by a number of lung or heart diseases, including pulmonary embolism.

Arterial blood gas (ABG): A technique used primarily to measure the oxygen level of blood with precision. It is obtained from a sample taken directly from an artery with a needle or a thin tube (catheter).

VQ lung scan: A test to evaluate both air flow (V = ventilation) and blood flow (Q = perfusion) in the lungs to determine whether a person has experienced a pulmonary embolism.

Pulmonary angiogram: The most definitive test to diagnose PE. Pulmonary angiogram is an "invasive" test, requiring injection of a dye through a catheter (IV line) into the body. Because newer tests such as CT scanning are now available, pulmonary angiography is rarely needed today.

Low-molecular-weight heparin (LMWH): Chemically cut or cleaved heparin. LMWH lasts longer, must be monitored differently, and generally has fewer side effects than standard heparin.

Heparin: An anticoagulant medicine ("blood thinner") that is routinely prescribed for the treatment of clotting disorders, including treatment of clots in the coronary arteries (causing heart attack), clots in the blood vessels of the brain (leading to stroke), clots that occur in the leg veins (deep vein thrombosis), and clots that obstruct blood flow to the lungs (pulmonary emboli).

Prothrombin: A protein in the blood that is essential for the formation of a blood clot. The active form of prothrombin is called thrombin.

Thrombin: the clotting factor that converts fibrinogen to fibrin.

Activated partial thromboplastin time (aPTT): A blood test that measures the length of time (in seconds) that it takes for clotting to occur when certain substances are added to the liquid portion of blood in a test tube. The aPTT is used not only to detect clotting factor deficiencies, but also used to monitor heparin's effectiveness.

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.

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."