GE Healthcare

TiP-TV™ Training in Partnership Program Supplement and Test for Imaging Professionals

US: DVT – The Silent Killer

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1.0 ASRT-approved Category A+ CE Credit

imagination at work

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Program Summary

This page provides an overview of the program content and learning objectives. Please refer to the Table of Contents for a detailed list of the topics covered. We encourage you to file a copy of this Program Summary and the Table of Contents with your continuing education certificate. We also recommend you provide your manager with a copy of this information as a record of your educational achievement.

Program Description

Beginning as blood clots forming in the veins of the lower extremities, deep vein thrombosis, or DVT, can break free and travel to the lungs causing pulmonary embolism. It can also be lethal. We explore the causes, treatment, and imaging of DVT and how the sonographer plays a role in saving these patients' lives.

Program Objectives

By the end of this program, the viewer should be able to:

- Identify the risk factors associated with the development of deep venous thrombosis.
- Describe the differences between deep venous thrombosis, superficial thrombophlebitis, and pulmonary embolism.
- Recognize the clinical signs and symptoms associated with deep venous thrombosis.
- Describe the anatomical and pathological elements of thrombosis.
- Discuss the methods of treating deep venous thrombosis.

Target Audience

Course objectives for this program specifically target vascular sonographers. While not limited to this audience group, the technical content is most effective when applied to people with this training.

Registered radiologist assistants, other sonographers and medical personnel may also benefit from viewing this program.

NOTE: Regardless of your imaging specialty, you may apply for continuing education credit. Refer to the Continuing Education Credit page for additional information.

Continuing Education Credit

1.0 ASRT-approved Category A+ CE Credit

NOTE: Effective February 1, 2005, the ARDMS accepts credits for ASRT-approved CE activities. ARDMS registrants may claim ASRT-approved Category A credit to meet their CE requirements. For more information, visit: www.ardms.org

Continuing Education Credit and Video File Download

Online Process for CE Credit (hls.gehealthcare.com)

In order to receive continuing education credit, you must log into the GE Healthcare Learning System (HLS) and complete all of the required steps. Please refer to the online TiP-Ed Online Quick Start User Guide (click the User Guides link on the HLS Welcome page) for additional information on how to use the GE HLS as needed.

1. **View the entire program video** online or download the video file for later viewing (refer to the process below). This supplement is *not* intended to replace watching the video.
2. Go to the GE HLS web site at **hls.gehealthcare.com** and complete the **feedback form**.
   - NOTE: The Feedback Form link is not activated until the View Video Now module has been completed.
   - This provides valuable information regarding your thoughts on the program's quality and effectiveness.
3. Complete the **program post-test** without aids or assistance of any kind; this is an *individual effort*.
   - You have up to three attempts to successfully complete the test with a minimum passing score of 75% (ASRT and CBRN approved programs) or 80% (SNMMI-approved programs).
   - The post-test measures knowledge gained and/or provides a self-assessment on a specific topic.
4. Upon successful completion of the online CE information, you can instantly print a **certificate**.

Video Download Process

For programs with an original start date of September 1, 2008 or later, the GE HLS includes an option to download the program video file. You can then watch the program on your personal computer or transfer the video file to your portable video player for viewing.

NOTE: Please refer to the **Video Download Quick Start Guide** for complete details (click the User Guides link on the GE HLS Welcome page).

1. With the desired program in your GE HLS Learning Plan, launch the program content to view the Online Content Structure. In the Video Download (Optional) area, click the Download Video to View Later link.
2. Save the video file on your personal computer, using your existing video download software.
3. View the program on your personal computer or transfer it to your portable video player for later viewing.
4. After viewing the entire program, log into the HLS and complete the CE activities as noted above.

Continuing Education Credit Eligibility — Important Notice!

A GE Healthcare TiP-Ed Online course may be available in several different formats, such as an online web course or CD/DVD. You may be able to receive CE credit only once for a particular course, regardless of the format in which it was viewed. If you have already received credit for a course, you are encouraged to contact your CE certification organization (ARRT, NMTCB, ARDMS, CBRN, etc.) to determine if you can repeat this course for CE credit.

Thank you for choosing GE Healthcare as your continuing education partner. We hope you will join us for other TiP-Ed Online programs in the future. For more details and program schedule information, please visit our education web site (**www.gehealthcare.com/education**).

Please forward any questions or comments to: **geeducation@ge.com**

Blood and Vessel Conditions

Thrombosis

Composed of fibrin and blood cells, thrombi may form in veins, arteries, capillaries, and in the heart. Because the relative proportion of fibrin and blood cells depends on the hemodynamic factors of the vessel, the proportions differ between arterial and venous thrombi. Arterial thrombi form in high flow conditions and are composed mainly of platelets tied together by fibrin strands, the insoluble protein formed from fibrinogen.

Platelets play a crucial part in the normal arterial blood-clotting process. First, single platelets bind to the site of a damaged artery, which is referred to as adhesion. Then platelets bind to each other in a process called activation. Activation can be stimulated by factors released by the damaged endothelial cells and by thrombin, also released during the blood-clotting process. When platelets become activated, they release agents that attract and activate any surrounding platelets. The last step of this process is the coagulation of fibrin strands, which stabilizes the clot or thrombus. Thrombus is supposed to dissolve as part of the healing process called fibrinolysis.

In contrast, venous thrombi are composed of blood cells with a large amount of fibrin, and form in areas of stasis or low flow. Venous thrombi contain relatively few platelets, and they typically attach loosely to the vessel wall and tend to build proximally over time. Activation of blood coagulation agents is the critical instigator in the pathogenesis of venous thromboembolism, which can be lethal.

Thrombophlebitis

Thrombophlebitis, or superficial phlebitis, is inflammation and clotting in a superficial venous system, such as the greater and lesser saphenous veins near the skin in the lower extremities. Even a slight injury can cause inflammation in a vein. Symptoms associated with superficial phlebitis include pain along the course of the inflamed vein, mild to moderate edema in the affected extremity, and a noticeable increase in temperature in the affected extremity. The symptoms arise as the thrombi develop. Thrombi associated with superficial phlebitis rarely dislodge to form an embolus due to the lack of surrounding muscle contractions. Often, a prominent, tender, cord-like subcutaneous vein may be palpable.

Deep Venous Thrombosis (DVT)

Deep venous thrombosis is thrombus that develops within the deep venous system, typically in the lower extremities. DVT can produce long-term complications due to venous hypertension by damaging the venous valves as thrombus develops. DVT can form following trauma, surgery, or due to prolonged immobilization. Certain people also have inherited or acquired factors that put them at increased risk.

Dr. Rudolf Virchow, a German pathologist, discovered the connection between DVT and pulmonary embolism. He also devised a three-pronged formula to explain the cause of thrombosis. Today, this formula is commonly known as "Virchow's Triad."

Virchow's Triad

- **Endothelial damage:** Injury to the inner lining of the vessel wall.

- **Venous stasis:** Stopped or slow flow of blood through the vessel.
- **Hypercoagulability:** An often genetic, abnormal blood-clotting response.

Pulmonary Embolism (PE)

Pulmonary embolism is the blocking of a pulmonary artery by an embolus, a free-floating portion of clot or thrombus. The clinical presentation of PE is commonly non-specific and may mimic many other acute cardiorespiratory illnesses. Pulmonary embolism from an upper extremity source is increasingly being identified.

Indications of PE include:

- Shortness of breath
- Light-headedness
- Chest pain (usually pleuritic)
- Hemoptysis (coughing up of blood)
- Pre-syncope or syncope (loss of consciousness)
- Shock
- Hypoxemia (low arterial oxygen)
- Arrhythmia, especially atrial fibrillation
- Fever
- Right heart failure
- Pulmonary hypertension

The following is the path of a lower extremity deep vein thrombus (starting at the superficial femoral vein) breaking free to form a thromboembolism as it travels through the venous system and terminates in the lung:

- Superficial femoral vein
- Common femoral vein
- Iliac vein
- Inferior vena cava
- Right atrium of the heart
- Right ventricle of the heart
- Pulmonary trunk
- Pulmonary arteries (right or left)
- Lung

Risk Factors

Venous Stasis

Venous stasis generally occurs in patients who are bedridden or patients whose mobility has been dramatically affected. Venous stasis may form when an interruption occurs in the normal muscle pumping action, or when a defect develops within the valves of the venous system. Studies have shown that the factor of hypercoagulability is required along with venous stasis to promote thrombi formation.

Factors that can lead to venous stasis include:

- Immobility
- Debilitating medical conditions
- Stroke
- Myocardial infarction
- Heart failure
- Obesity
- Valvular incompetency
- Anesthesia/surgery
- Age (>70 years)

Economy Class Syndrome

Due to the prolonged immobility common to international travel, being still for long periods of time can lead to venous stasis. This increases the traveler's chances of developing a blood clot, which can lead to a life-threatening pulmonary embolism. Some case studies report individuals experiencing symptoms of DVT or pulmonary embolism soon after deplaning, while others may not have a problem until days or even weeks after their flights.

There is some discussion about whether it is the actual flying that is the culprit. Studies have shown that long periods of immobility by any means of travel can lead to the formation of DVT. But why traveling? Typically a person traveling by vehicle is stuck in a seated position (legs lower than the heart) for hours at a time. When it comes to flying, add the dehydrating effects of drinking alcohol, and the risk increases.

Surgery

Deep vein thrombosis can occur in patients undergoing reconstructive or total hip replacement surgery. Usually age and history of previous DVT or PE indicate a higher risk. Hip injuries, hip or knee replacement surgery, pelvic or abdominal surgery, and even lower limb amputations can have a post-operative risk of DVT. The risk of thromboembolism is increased in procedures that last longer than 30 minutes. The longer the patient is immobile and the more complex the surgery, the higher the risk of the patient developing DVT.

The incidence of DVT rises sharply in patients confined to bed for more than one week. The immobility of a lower extremity alone is enough to increase the frequency of thrombosis.

Other Risk Factors

Certain factors make the occurrence of DVT more likely. The people at most risk are those who are age 40 years and older and/or are obese, and those who have already had DVT. Several inherited conditions, such as hypercoagulability, make the blood more likely to clot than usual, increasing the risk.

Intravenous (IV) needles may also cause DVT in the implanted vein. The continuous damage to the inner lining of the venous wall promotes the formation of a clot. The use of IV needles in drug use has the same risk. Typically, the veins that are affected by IV needles are the veins in the upper extremities and the jugular veins.

Other factors include:

- Major injuries or paralysis
- Cancer and cancer treatments
- Pregnancy and childbirth
- Taking birth control pills that contain estrogen
- Hormone replacement therapy (HRT)
- Other circulation or heart problems

Statistics

Here are some statistics on DVT and PE that help demonstrate the impression these conditions are making on the medical scene.

Heart Disease and Stroke Statistics – 2004 Update, The American Heart Association

- Death occurs within 1 month in 6% of those diagnosed with DVT.
- Incidence of DVT in the 30 to 49 age range was 2 to 3 per 10,000 people.
- Incidence of DVT in the 70 to 79 age range was 20 per 10,000 people.
- Death occurs within 1 month in 12% of those diagnosed with PE.
- 2001 data showed that 54.6% Americans did not meet physical activity recommendations.
- 2003 data showed that 68.7% of those over 18 years of age did not engage in regular leisure activity.
- 2001 data showed that 64.5% of Americans are considered overweight by the body mass index (BMI).

Imaging

Ultrasound

An ultrasound examination is commonly used to diagnose DVT. Since it is a very hands-on procedure, patients with significant pain or discomfort may not be good candidates for the full exam. The basics of the procedure consist of compressions of the deep veins in the transverse plane in conjunction with longitudinal color flow and Doppler analysis of normal and augmented venous flow.

Depending on the protocol of the facility and the symptoms the patient presents, unilateral or bilateral limbs can be scanned. The lower limb may be scanned to the popliteal level only or more distally to include the deep calf veins. Also, the sonographer may scan proximally from the common femoral vein to include the iliac veins and the inferior vena cava. The upper extremities may be scanned at the point of the wrist and proximally to the subclavian vein, including the superficial venous system. The jugular vein may be scanned if a jugular vein thrombus is suspected, for example, from an IV entry point (see Figures 1 and 2).

Figure 1 Thrombus Formation in Jugular Vein – Transverse View

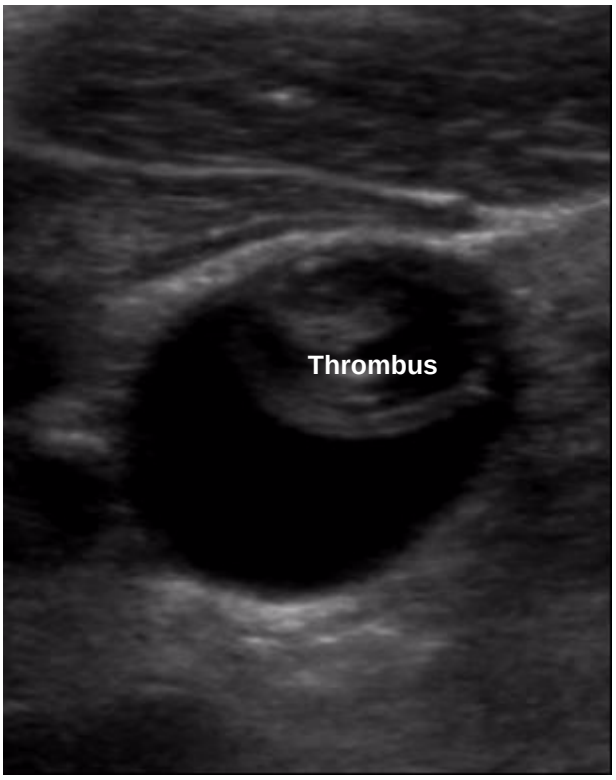
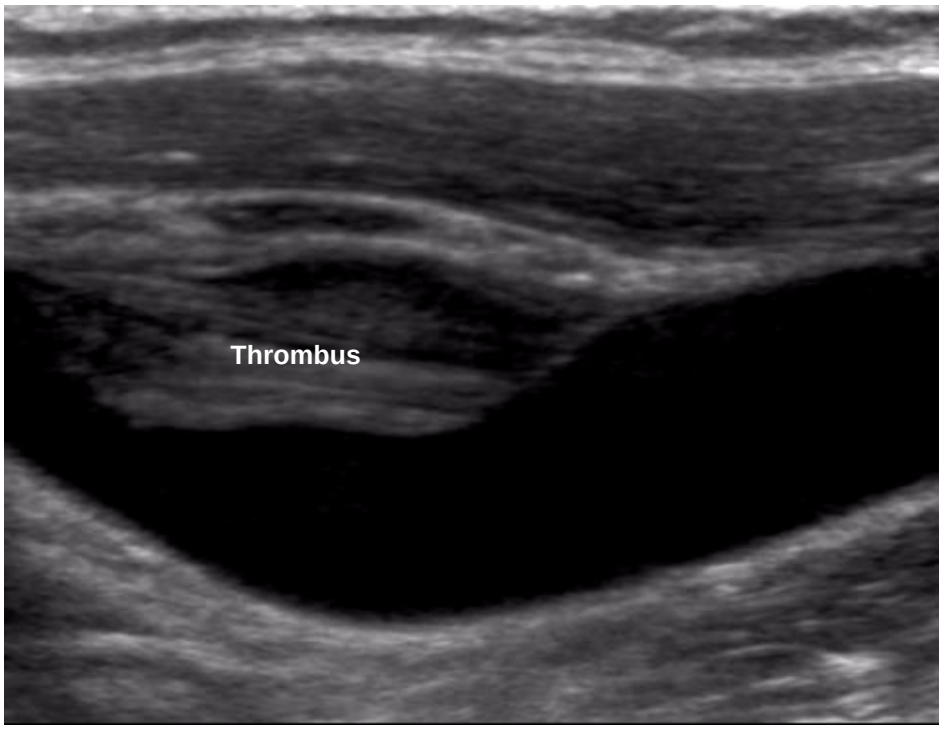


Figure 2 Thrombus Formation in Jugular Vein – Longitudinal View



Other Techniques

If PE is suspected, the following exams may help determine the diagnosis:

- **D-dimer blood test:** While most patients with PE and DVT do have an elevated D-dimer test result, the D-dimer result may also be elevated in many other conditions, such as injury, surgery, cancer, or inflammatory diseases. Therefore, a positive test result is not helpful. A negative result, however, helps to rule out PE.
- **Contrast-enhanced spiral computed tomography (CT) of the chest:** The CT scan may also include the lower extremities to help rule out DVT.
- **Ventilation perfusion (VQ) lung scan:** A commonly requested nuclear medicine examination, this scan can be ordered to study both airflow and blood flow in the lungs.
- **A chest x-ray (CXR):** A CXR can also be ordered. However, while often abnormal in PE cases, it rarely helps to confirm or exclude PE.

Reflux

As we walk, blood is pumped proximally in the veins by the calf muscles toward the heart. Gravity intervenes and tries to pull the blood back down as the leg muscles relax. Valves are located throughout the veins of your legs and throughout your body to prevent the reversal or downward flow of blood, which is referred to as venous reflux. Venous reflux is associated with chronic venous insufficiency as well as varicose veins.

Reflux leads to the bulging and stretching of the vein walls due to incompetent valves that allow blood to flow in the opposite direction. This can lead to venous hypertension, which is the fundamental cause of all the problems associated with varicose veins or chronic venous insufficiency. While venous reflux may occur in the deep venous system, the tortuous manifestation of a varicose vein is typically reserved for the superficial venous system.

Venous reflux disease can be tested with duplex ultrasound. By squeezing or augmenting the calf or thigh or having the patient bear down (Valsalva maneuver), Doppler waveform analysis can show any significant reversed flow. A slight amount of reflux is considered normal as it is the function of the venous valve closing. Flow reversal for greater than 1 second is indicative of valvular incompetence.

Treatment

Deep venous thrombosis can be treated by utilizing a variety of techniques, including:

- Compressive stockings
- Patient education
- Physical therapy
- Prophylaxis
- Pharmacological therapy
- Surgery

All methods are in an effort to relieve symptoms and decrease the risk of pulmonary embolism. Recurrent thrombi development is always a concern.

If the patient has a history of chronic edema or venous insufficiency, compressive stockings can be utilized to help control swelling. Physical therapy for an immobilized patient is also an important parameter in helping to prevent the development of a deep venous thrombosis. By moving a patient's extremities through their range of motion, this encourages the normal muscle pumping action of everyday activity, such as walking, and helps to promote venous flow.

Patients who are bedridden should be placed in an anti-embolic position. The hips and knees are flexed approximately 30 degrees and the legs are elevated higher than the level of the heart, similar to the Trendelenburg position. Adequate hydration is extremely important, either through intravenous fluids or oral intake, to help ensure that blood viscosity is not compromised. Warm, moist heat can also be applied to any affected areas to help promote venous dilation and flow.

Pharmacological Therapy

Thrombolytic Therapy

The goal of thrombolytic therapy is to dissolve the thrombi, especially in pulmonary embolism, with pharmacological agents.

Thrombolytic therapy (clot-dissolving medication) includes:

- Streptokinase
- Urokinase
- Alteplase (t-PA, a genetically engineered tissue plasminogen activator)

Streptokinase and urokinase both work in similar ways. They react with plasminogen to produce the proteolytic enzyme called plasmin (which breaks down proteins) that dissolves the fibrin in thrombi (fibrinolysis). Contraindications to these agents include any internal bleeding and/or a severe cerebrovascular or intracranial procedure within the prior two months. When applied to pulmonary embolism, the rapid fibrinolysis should quickly result in increased cardiac output, improved oxygenation, and decreased pulmonary hypertension.

Anticoagulation Therapy

After the thrombolytic agent is discontinued, anticoagulation is begun. Anticoagulation therapy (clot-preventing medication) includes:

- Heparin standard (administered intravenously)
- Low molecular weight heparin (Lovenox®, administered by injection)
- Warfarin (Coumadin®, administered orally)

Subcutaneous low molecular weight heparin is often substituted for intravenous heparin in many circumstances and can be administered in an outpatient manner. Anticoagulation therapy may also be given as a prophylaxis to prevent DVT during an upcoming surgery.

Intravenous heparin is usually prescribed to begin the anticoagulant treatment. It is given as a continuous drip. The goal of the treatment is to maintain the clotting time at twice normal, and to prevent further clotting and shift the body's natural balance to thrombolysis. The partial thromboplastin time (PTT) can be used to monitor heparin therapy. It is performed several times the first few days and then once daily. The goal in the monitoring is to keep the PTT in a therapeutic range greater than 1.5 times and less than 2.5 times normal. Heparin is usually prescribed for several days to several weeks. Heparin treatment can be overlapped with the initial doses of the oral anticoagulants, such as warfarin, to help prevent the potential of future clotting.

Filters

In patients who cannot tolerate anticoagulation therapy, an inferior vena cava (IVC) filter may be installed. This device, placed in the abdomen, is designed to block large clots from traveling into the pulmonary vessels. The filters may be installed through the common femoral vein or the brachial vein.

FDA Approved IVC Filter Devices

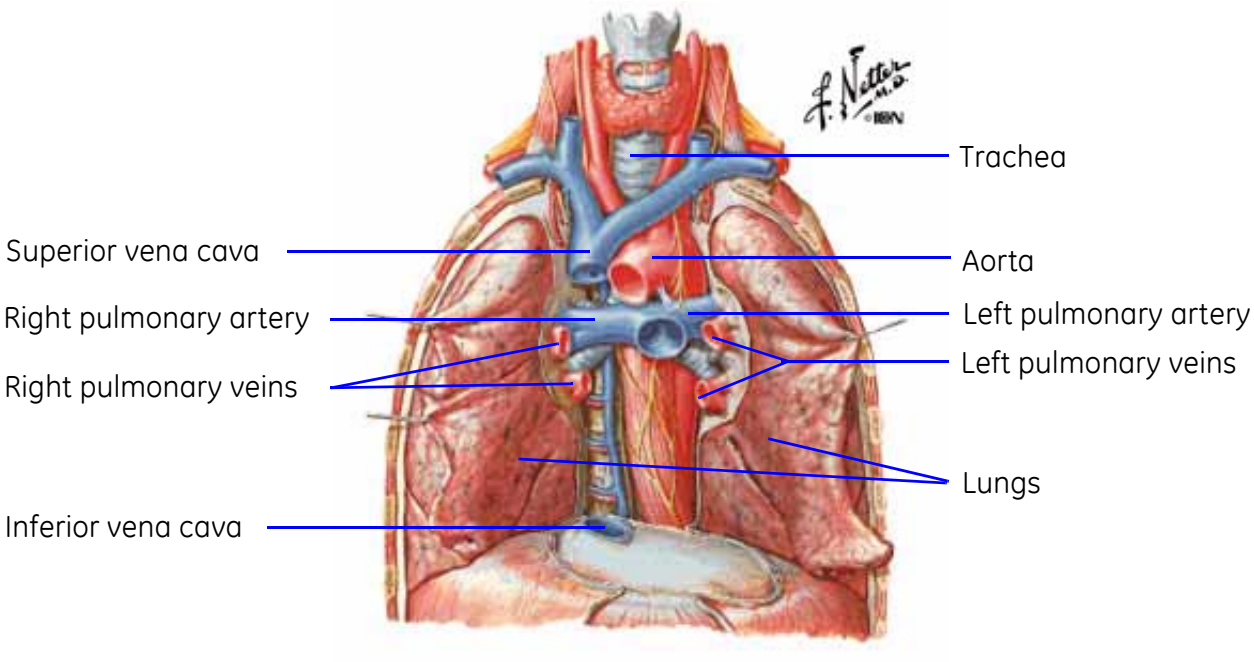
- Stainless steel Greenfield® Vena Cava Filter (Medi-Tech; Boston Scientific)
- Titanium Greenfield® Vena Cava Filter (Medi-Tech)
- Gianturco-Roehm Bird's Nest® Vena Cava Filter (Cook)
- Vena Tech™ LP Vena Cava Filter (B. Braun Medical)
- The Simon-Nitinol Filter® (Nitinol Medical Technologies; Bard)

Embolectomy

Another interventional means of treatment is an embolectomy, a procedure to remove the clot in the pulmonary arterial system with a catheter or by surgery.

Appendix A: Anatomy

Figure 3 Thoracic Anatomy



Appendix B: Presenter Biographies

Samuel Zachary Goldhaber, M.D. – Director, Venous Thromboembolism Research Group, Brigham and Women's Hospital, Cardiovascular Division, Boston

Dr. Samuel Goldhaber earned his medical degree from Harvard Medical School in 1976. He served his residency and fellowship at the Peter Bent Brigham Hospital/Harvard Medical School. Dr. Goldhaber is board certified in Cardiovascular Disease and Internal Medicine. He is currently the Director of the Venous Thromboembolism Research Group at Brigham and Women's Hospital and Associate Professor of Medicine at the Harvard Medical School.

Dr. Goldhaber has authored more than 125 articles, published numerous periodicals and text book chapters, and has edited several books. His clinical interests include anticoagulation, antiplatelet therapy, deep vein thrombosis (DVT), general clinical cardiology, pulmonary embolism, and thrombolytic therapy. He also received the Daniel D. Federman Outstanding Clinical Educator award at the Harvard Medical School from the class of 2000.

Marie Gerhard- Herman, M.D. – Medical Director, Vascular Diagnostic Laboratory at Brigham and Women's Hospital, Boston

Dr. Marie Gerhard-Herman is a cardiovascular physician with particular expertise in clinical vascular ultrasound imaging and vascular medicine. She is the Medical Director of the Vascular Diagnostic Laboratory at Brigham and Women's Hospital. She teaches an ongoing course titled "Current Practice of Noninvasive Vascular Imaging." She also directs the General Clinical Research Center (GCRC) Ultrasound Laboratory and works with many investigators who are interested in vascular physiologic testing.

Dr. Gerhard-Herman was recognized by Brigham and Women's Physician Organization with a Physician Recognition Award, one of two, for outstanding clinical leadership. In 2004, she was again one of two who received the William Proctor Harvey Teaching Award, at the American College of Cardiology, given for exceptional talent and dedication to teaching.

Jean M. Alessi, RVT, RDMS – Technical Director, Vascular Diagnostic Laboratory at Brigham and Women's Hospital, Boston

With 12 years of ultrasound experience, Jean Alessi is the Technical Director of the Vascular Diagnostics Laboratory at the Brigham and Women's Hospital. Her responsibilities include maintaining the quality control program and procedure volume statistics consistent with Joint Commission on Accreditation of Healthcare Organizations (JCAHO) and The Intersocietal Commission for the Accreditation of Vascular Laboratories (ICAVL) standards.

Jean was the President of Greater Boston Vascular Technologists from 1999 to 2001. She has eight published articles to her credit in the *Journal of Vascular Technology*, the *Video Journal of Color Flow Imaging*, and others. Jean also has written several abstracts and a chapter titled "Vascular Imaging" in the *Atlas of Cardiac Imaging*.

Andrew Stonefield – GE Healthcare Ultrasound TiP-TV Program Manager

Andrew worked as a Registered Vascular Sonographer with Medicalab, Inc. throughout New England and acquired diverse experience in imaging technology and techniques. Following that position, he worked as an applications consultant for MEDITECH, Inc., where he was responsible for the training of several hospital teams in the United States and Canada in integrated radiology department software.

Andrew joined GE Healthcare as a contractor in 2000 and worked with the ultrasound marketing team producing web content for various ultrasound products and service technical training programs. In 2002, he became the Program Manager of Clinical Ultrasound TiP-TV programs and related eLearning products. In addition, he has responsibility for product offerings for the GEMS IT, OEC, and Lunar businesses.

Andrew earned a bachelor's degree in biology from Coastal Carolina University and is currently working on a Master of Business Administration degree at the Keller Graduate School of Management. He has earned both Telly and Communicator awards in broadcasting.

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Appendix C: Resources

Hirsh, Jack, M.D., et al. *Guide to Anticoagulant Therapy: Heparin.* The American Heart Association. © 2001.

Davidson, Bruce L., M.D., MPH. *Controversies in Pulmonary Embolism and Deep Venous Thrombosis.* American Academy of Family Physicians. © 1999.

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Appendix D: Post-Test

LMS Course Number: 2723

To be eligible for CE credit, you MUST view the video presentation first. Then complete the post-test on the GE Healthcare Learning System **(hls.gehealthcare.com)** by the due date listed online.

1. Venous thrombi are mostly comprised of all of the following, EXCEPT _____.
   a. platelets
   b. coagulating agents
   c. blood cells
   d. fibrin
2. Arterial thrombi form in high flow conditions and are composed mainly of platelets tied together by fibrin strands, the insoluble protein formed from _____.
   a. blood plasma
   b. angiopoietin
   c. amino-acid
   d. fibrinogen
3. DVT can be a significant threat to cause a pulmonary embolism when located in any of the following vessels, EXCEPT the _____ vein.
   a. greater saphenous
   b. popliteal
   c. superficial femoral
   d. common femoral
4. _____ is NOT a factor in Virchow's Triad.
   a. Hypercoagulability
   b. Venous stasis
   c. Endothelial damage
   d. Angiogenesis
5. Pulmonary embolism is the blocking of a _____ artery by an embolus, a free-floating portion of clot or thrombus.
   a. systemic
   b. pulmonary
   c. aterioles
   d. carotid
6. Which of the following pathways is consistent with a thromboembolism originating from the leg?
   a. Common femoral vein, inferior vena cava, left atrium, pulmonary artery
   b. Superficial femoral vein, common femoral vein, inferior vena cava, pulmonary vein
   c. Superficial femoral vein, common femoral vein, right atrium, pulmonary artery
   d. Popliteal vein, common femoral vein, pulmonary vein, bronchial tube

7. Symptoms of acute pulmonary embolism include all of the following, EXCEPT _____.
   a. light-headedness
   b. shortness of breath
   c. chest discomfort or pressure
   d. pain in the left arm
8. Air travel is the only form of transportation that can lead to the formation of DVT.
   a. True
   b. False
9. All of the following are risk factors of pulmonary embolism, EXCEPT _____.
   a. atherosclerosis
   b. immobilization
   c. a history of deep vein thrombosis
   d. obesity
10. Clotting is an abnormal process in the body.
    a. True
    b. False
11. Venous stasis can be caused by _____.
    a. valvular incompetency
    b. inferior vena cava filters
    c. superficial phlebitis
    d. intermittent claudication
12. The risk of thromboembolism is increased in surgical procedures that last longer than _____ minutes.
    a. 20
    b. 30
    c. 45
    d. 60
13. An intravenous drug user may get DVT due to _____.
    a. the coagulating effects of heroin
    b. the drug paralyzing the venous wall muscle
    c. repetitive needle damage to the venous wall
    d. the repetitive use of a tourniquet before injections
14. The people at most risk of DVT are those who are age _____ years and older and/or are obese, and those who have already had DVT.
    a. 20
    b. 30
    c. 40
    d. 50
15. _____ is an effective way to identify deep vein thrombosis.
    a. Compression while scanning
    b. Obtaining segmental pressures
    c. Using coded harmonics
    d. Continuous wave Doppler

16. Besides ultrasound compression of the lower extremity deep veins, what other noninvasive imaging technique is commonly used to diagnose pulmonary embolism?
    a. Lower extremity x-ray
    b. Embolectomy
    c. Spiral computed tomography
    d. Pneumoplethysmography
17. Venous reflux disease can be tested with _____.
    a. Infrared thermography
    b. Fluoroscopy
    c. MRI
    d. duplex ultrasound
18. All of the following are ways to prevent the formation of DVT, EXCEPT _____.
    a. keeping your feet raised
    b. breathing deeply
    c. walking
    d. drinking plenty of non-alcoholic fluids
19. All of the following are used to treat blood clots, EXCEPT _____.
    a. warfarin
    b. low molecular weight heparin
    c. tissue plasminogen activator (t-PA)
    d. steroids
20. _____ is a DVT treatment option for a patient who cannot have anticoagulation therapy.
    a. Tissue plasminogen activator (t-PA)
    b. Streptokinase
    c. Inferior vena cava filter
    d. Low molecular weight heparin