

# **The APDVS Medical Student Curriculum**

The APDVS and contributors

1/31/23

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# Preface

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*APDVS* Association of Program Directors in  
Vascular Surgery

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This content was developed as part of the Association of Program Directors in Vascular Surgery's (APDVS) medical student curriculum. Each chapter covers a key domain of vascular surgery pathology and treatment and is associated with an Audible Bleeding episode which you can access from the link embedded in the text.

This eBook would not have been possible without the [Association of Program Directors in Vascular Surgery \(APDVS\)](#), the guidance and resources of [Dr. Sharif Ellozy](#) and [Dr. Adam Johnson](#), the [Audible Bleeding Vascular Surgery Exam Prep](#) project, and the [Audible Bleeding Team](#). This project is the direct result of [Dr. Chelsea Dorsey et al.](#)'s medical education research (see below) and the work of the [Society of Vascular Surgery's Resident Student Outreach Committee \(SVS RSOC\)](#).

Below, please see the two papers to understand the medical education need addressed by this eBook.

- [Vascular Surgery Curriculum for Medical Students: A National Targeted Needs Assessment](#)
- [The Value of a Vascular Surgery Curriculum for Clinical Medical Students: Results of a National Survey of Nonvascular Educators](#)

**Editors:** [The Association of Program Directors in Vascular Surgery](#) and [Ezra Schwartz](#).

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## Disclosures

This is an publication of the APDVS and therefore we share the same conflicts of interest. That noted, this eBook is the product of voluntarily donated time. This work has no financial backing and is not-for-profit. We are not receiving any funding or benefits from outside resources referenced. We include resources we believe will benefit you, the learner.

# Usage

This eBook is intended to be a medical student level, easily accessible review for vascular surgery rotations and general medical education. This eBook is paired with slide decks, pre- and post-reading questions and a teaching case produced by the APDVS in addition to podcast content produced by Audible Bleeding, a publication of the Society of Vascular Surgery.

We are also excited to include an open source annotations software called [hypothes.is](#). By creating a Hypothes.is account, you will be able to create notes in the eBook as you read. You can choose to make your notes private or public. As this eBook is a community-led initiative, please consider making your notes public if you feel they would benefit your peers or the editors of the book. Given the public nature of annotations, if you choose to post public notes, these annotations are regarded as contributions to the eBook and we expect readers and listeners to follow a [Contributor Code of Conduct](#). **We expect all participants, ourselves included, to maintain a safe space and behave professionally.**

Thanks to the flexibility and accessibility afforded as an eBook, we can (and will) update the book with extreme ease. Updates may include but are not limited to: slide decks, new pre- and post-reading questions, and relevant Audible Bleeding podcast episodes. **Please see the announcements page to stay up to date on developments.**

This eBook is free to the public and licensed under a Creative Commons Attribution-NonCommercial-NoDerivs 3.0 United States License.

## Additional Information

Please consider becoming a member of the [Society of Vascular Surgery](#). Benefits include free access to the Journal of Vascular Surgery, mentorship and scholarship opportunities, and more! A variety of [membership types](#) exist, including those for [medical students](#), general surgery residents, and integrated vascular trainees.

Visit the [ChooseVascular](#) website to learn why we chose vascular surgery and determine if you wish to choose vascular too! This site is complete with a student resources page, and information to pair you with a vascular surgery mentor or a vascular surgery interest group, this website is a wonderful introduction the work and community of vascular surgery.

# Comments, Questions or Contributions

Please visit our [github page](#) or [send the APDVS an email](#).

This book is built on [Quarto](#).

# Introduction

# Additional Vascular Surgery Educational Resources

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We hope you use this eBook as a review for your rotations, undergraduate medical licencing examinations, and entry to independent practice. This resource is a summary and is by no means comprehensive. That noted, in every chapter we have included links to additional resources if you wish to gain a more comprehensive understanding of the topics covered. As our mission is to make vascular surgery education accessible, we prioritize open access publications and free materials.

A few highly recommended additional resources include:

- The [Gore Medical Vascular and Endovascular Surgery Combat Manual](#) is an informative and entertaining read intended as a vascular surgery crash course for medical students, residents, and fellows alike. Highly accessible with a thoughtfully determined level of detail, but lacking in learning activities (e.g. questions, videos, etc.), this resource is a wonderful complement to the APDVS eBook.
- [TeachMe Surgery](#) is a student friendly online resource complete with short quizzes to help cement learning and recommended readings from the literature. Clear and concise, this is a great just-in-time learning resource.
- The [Audible Bleeding Medical Student Archive](#) contains podcast episodes tailored for medical students. We suggest paying special attention to the  *Holding Pressure*  series. Whereas this eBook is organized by disease,  *Holding Pressure*  episodes are organized by surgical procedure. These episodes contain a wealth of high-yield information and we will include links to  *Holding Pressure*  episodes throughout the eBook when applicable.
  - You can listen to Audible Bleeding episodes on [Apple Podcasts](#), [Spotify](#), [Google Podcasts](#), [Stitcher](#), or [Soundcloud](#)
- The [Audible Bleeding Exam Prep curriculum](#) and the associated podcast series. While this resource is intended for postgraduate trainees and fellows and therefore beyond the scope of this curriculum, it is a well authored, no-nonsense resource for those who wish to deepen their knowledge base. The accompanying podcast series also allows you to learn or study on the go!



- You can listen to Audible Bleeding episodes on [Apple Podcasts](#), [Spotify](#), [Google Podcasts](#), [Stitcher](#), or [Soundcloud](#)
- [All You Need to Know About Vascular Surgery: A Guide for Medical Students, Early Year Doctors and Allied Healthcare Professionals](#). The Journal of Vascular Societies Great Britain and Ireland (JVSGBI) has authored a concise, high-yield, well organized, and well illustrated eBook tailored to those learners and professionals with limited previous vascular surgery exposure. This resource is a wonderful primer for next-day activities as topics are covered in two pages. Please note, this eBook follows European guidelines and may present treatments not available in North America, however, this eBook remains **highly** relevant to your learning. Like the APDVS eBook, it is downloadable as a PDF.
- [VascularTraining.org](#) is an online repository of vascular surgery trainee resources compiled during the stages of the COVID-19 pandemic when providing traditional education was challenging and learning content was increasingly available online. Find videos, landmark and other informative papers, and device information here. Like the Audible Bleeding Exam Prep curriculum, the primary audience for this content is postgraduate trainees but is very accessible for undergraduate students who wish to deepen their knowledge base. This resource is password protected. The password is: *Vasceducation2021!*
- The **SVS Interactive Practice Guidelines Mobile App** has easy-to-find evidence-based guideline recommendations and easy-to-use clinical calculators (CEAP, VCSS, GLASS, Wifl, WVI CLTI Mortality Risk, Postthrombotic Syndrome score, VQI AAA Mortality Risk). For any trainee on vascular surgery rotations, this is an extremely helpful resource when looking for answers on the go! Find it here: [Apple App Store](#) or [Android Google Play](#)
- While tailored more towards general surgery trainees, [LearnAbdominal.com](#) provides a wonderful, student-friendly primer on reading abdominal CTAs. Scroll through the [Annotated teaching case](#) to review abdominal anatomy as seen on CT. Every organ is labelled! We also recommend viewing the [Evaluating the abdominal arteries](#) video lecture to familiarize yourself with abdominal vasculature as seen on CTA.

## Announcements & Case of the Week

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Please return to this page for updates on website, podcast, and activity developments.

### eBook Chapters

Claudication chapter hot off the press!

We are starting to post our very own illustrations!

Chapters in development include: - Patient History and Physical Examination of the Venous System - Peripheral artery disease (PAD) including topics such as acute limb ischemia (ALI) and chronic limb threatening ischemia (CLTI). - Aortic dissection - Thoracic outlet syndrome - Endovascular surgery basics - Vascular lab primer

### Podcast Episodes

The next podcast in development is an introduction to the APDVS curriculum.

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### Case of the Week

To harness the learning principles of [spaced practice](#), [retrieval practice](#), [interleaving](#) and [concrete examples](#), we invite students and residents to write a case of the week and submit it to APDVS for review.

Please try to build the case using material covered in the eBook. We ask you to write for the medical student level. Please include discussions to questions including why the correct answer is correct and why the incorrect answers are incorrect.

Once reviewed, we will post it here for the community! We hope you will take part in this mini-publication exercise!

Please submit your case to [APDVSebook@vascularsociety.org](mailto:APDVSebook@vascularsociety.org).

# Authors and Contributors

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This eBook is the result of a partnership between the APDVS and the Audible Bleeding Podcast, each of which is comprised of hard working individuals dedicated to medical education and the promotion of vascular surgery. The authors and contributors of this eBook have freely donated their time and experience to create this resource. Please follow them on twitter or other social media platforms to return this debt of gratitude and to, as Audible Bleeding says, “keep a finger on the pulse.”

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# **Approach to Vascular Patient**

# Social Determinants of Vascular Health

Abena Appah-Sampong, MD, Ezra Schwartz, MD,CM, MS, Sharif Ellozy, MD.

## Note

By the end of this chapter, students will:

- Review terms important to the discussion about social determinants of health.
- Be able to predict relevant questions to ask patients when obtaining a social history.
- Relate the importance of a thorough social history to the incidence, morbidity, and mortality of vascular disease.
- Recognize opportunities for one's biases to impact quality of care.

## Key Facts

1. Racial and ethnic minorities encounter social determinants of health and systemic racism that exacerbate the incidence, prevalence, and outcomes of many vascular diseases.
2. Addressing the challenges of healthcare deserts, particularly in rural areas, is vital for ensuring equitable access to vascular surgery care.
3. The underrepresentation of women in vascular surgery clinical trials highlights the imperative for concerted efforts to address gender disparities, enhance inclusion, and ensure findings are generalizable to broader populations.
4. Many veterans face socioeconomic, systemic, and mental health challenges stemming from their military service that challenge the management of chronic and complex vascular diseases.



## Introduction

*“Do we not always find the diseases of the populace traceable to defects in society?”* - Rudolf Virchow

Multiple studies have demonstrated significant disparities exist across the spectrum of vascular disease including prevalence, treatment, and disease outcomes. Furthermore, it has been demonstrated that social determinants of health (SDoH), comprising multiple factors including patient socioeconomic status, race or ethnicity, gender, and geographic location, can have potent effects on health outcomes. There are some estimates that SDoH account for 80-90 percent of the modifiable contributors to healthy outcomes for a population, and must be addressed in order to rectify persistent healthcare disparities. (Remington, Catlin, and Gennuso 2015)

Vascular surgeons treat patients with advanced atherosclerosis and diabetes, which are diseases that disproportionately affect vulnerable and socioeconomically disadvantaged patients. Given this reality, it is important for vascular surgeons to have a strong foundational understanding of the social determinants of health and health disparities that lead to unequal care among patients. This textbook chapter does not intend to be comprehensive, but rather an introduction to scope of health disparities in vascular surgery.

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## Definitions

**Health Disparities:** The preventable differences in the burden of disease, injury, violence, or opportunities to achieve optimal health that are experienced by socially disadvantaged populations. (“Health Disparities | DASH | CDC” 2023)

**Social Determinants of Health:** The circumstances in which people are born, grow up, live, work, age, and the systems put in place to deal with illness. (“Social Determinants of Health: Key Concepts,” n.d.)

**Structural Racism:** A system in which public policies, institutional practices, cultural representations, and other norms work in various, often reinforcing ways to perpetuate racial group inequity. (Allison et al. 2023)

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# Health Disparities in Vascular Surgery

## Race, Ethnicity, and Structural Racism

There is an abundance of literature documenting racial and ethnic disparities across the spectrum of vascular disease including thoracic and aortic aneurysms, carotid disease, and hemodialysis access. However, disparities spanning the disease continuum of peripheral artery disease (PAD) is one of the most robustly studied areas in surgery disparities research.

A scientific statement published by the American Heart Association outlines the scope of health disparities in PAD. Looking at prevalence, it has been demonstrated Black patients have a higher prevalence of PAD, even after controlling for traditional cardiovascular risk factors. Prevalence of PAD among other racial and ethnic groups is less documented, but limited evidence suggests higher rates of PAD among Cuban populations especially when compared to other Hispanic and Latinx groups. Risk factor control and preventative measures are important management tools to prevent the progression of PAD to critical limb threatening ischemia (CLTI) and limb loss. However, Black and Hispanic patients are more likely to present with hypercholesterolemia but less likely to be prescribed an aspirin or statin. (Allison et al. 2023)

Looking at PAD outcomes, studies have found that patients identifying as Black have nearly twofold greater rate of leg amputation, even after controlling for relevant confounders. Additionally, Black patients who undergo amputation are less likely to have any limb-related admission, suggesting less aggressive limb salvage among this population. Higher rates of diabetes and deep soft tissue infection are strongly correlated with the higher burden of amputation in Black patients. (Hackler, Hamburg, and White Solaru 2021)

Due to structural racism, racial and ethnic minority patients are often disproportionately burdened with socioeconomic barriers. One study evaluating the etiology of health care access among Hispanic patients found that Hispanic patients were more likely to delay medical care for socioeconomic reasons including inability to afford costs, time away from employment, and childcare issues. (Bazikian et al. 2023)

## Access and Care Delivery:

Millions of Americans live in communities considered ‘healthcare deserts,’ where they lack adequate access to important healthcare services including primary care, hospitals, emergency services, pharmacies, trauma centers, and low-cost health centers. (“Mapping Healthcare Deserts: 80,” n.d.) Many healthcare deserts are often concentrated in rural locations highlighting the unique geographical challenges that these patients face. Furthermore, certain vascular diseases including chronic limb threatening ischemia (CLTI) require specialized, multidisciplinary care for optimal management, but many communities are considered “vascular deserts” in which there is inadequate access to expert vascular surgery care. (DiLosa et al. 2023) Addressing the

challenges posed by healthcare and vascular deserts, particularly in rural areas, is crucial for ensuring equitable access to essential services. Continued investigation and investment into these issues are imperative to develop effective solutions that can bridge the healthcare gaps and provide optimal care.

### **Sexual and Gender Minorities:**

A growing body of research explores the impact of sex and gender on various aspects of vascular disease. A systematic review and meta-analysis of 236 studies demonstrates an association between female sex or gender and poorer outcomes in individuals with Abdominal Aortic Aneurysm (AAA) and Peripheral Artery Disease (PAD). (Lee et al. 2022) It has been demonstrated that women have higher risk of AAA rupture, prolonged lengths of stay, and decreased survival following both elective and emergent AAA repair. (Dillavou, Muluk, and Makaroun 2006) The lack of representation of women in large-scale vascular surgery clinical trials has also become an area of increasing concern, given the pivotal role these studies play in shaping the standard of care and management guidelines. Despite efforts to enhance women's participation in clinical trials, evidence suggests persistent underrepresentation in randomized control trials, potentially limiting the broader applicability of their findings. (Hoel et al. 2009) The multifactorial nature of this underrepresentation, influenced by factors like patient preference, bias, and local demographics, necessitates sustained efforts to achieve diverse recruitment and ensure a more comprehensive understanding of vascular disease across diverse patient populations.

### **Veteran Populations:**

Military veterans represent a unique population with distinct healthcare needs. While veterans may benefit from the ability to receive care through the Department of Veterans Affairs (VA) healthcare system, there remain notable challenges and disparities for this population. Barriers to medical treatment in the veteran population broadly include long wait times at VA facilities, mental health illness and stress related to previous deployments, challenges reintegrating into civilian life, and unmet basic needs such as housing and employment. (Misra-Hebert et al. 2015) Because of these barriers, veteran populations often suffer higher incidence of medical comorbidities compared to civilian population. Assari (2014) Looking at vascular pathologies, a large 2017 epidemiological study demonstrated that there is a higher incidence of PAD and associated mortality among veterans, with an estimated 30% of veterans dying within 3.8 years of a PAD diagnosis. (Willey et al. 2018) Additional studies have shown that demonstrated that mental illness may compound vascular comorbidities within the veteran population, as veterans suffering from comorbid depression face significantly higher risk of amputation and death from PAD. (Arya et al. 2018) Ultimately, understanding the complex social determinants and healthcare needs of veterans is essential for developing targeted interventions and support systems to improve the health and well-being of this vulnerable population.

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## Teaching Case

### Scenario

A 65 year old man is brought to the emergency room with altered mental status, fevers, and severe pain, swelling, and erythema in his right foot. On exam, the foot is warm to the touch and you observe a deep, ulcerating wound with surrounding necrotic tissue. His labs are notable for an elevated white count and HbA1c of 10. His daughter, who is the patient's legal medical decision maker, is at the patient's bedside and tells you that the patient was laid off from work 6-months ago and since then has not been able to afford insulin to manage his diabetes. You call your attending surgeon about the patient's concerning findings and recommend an emergency operation. Your attending sighs and says "I'll never understand what kind of person lets their disease get this bad."

*Case inspired by University of Michigan Cultural Complications Curriculum*

### Discussion Points

**i** N.B. There is no prepared answers for the questions below.

However, we feel this chapter contains all the necessary information to answer the questions. If not, please let us know!

1. How do socioeconomic factors impact a patient's ability to manage chronic conditions?
2. In this scenario, how might the attending surgeon's assumptions affect the quality of care provided?
3. Do surgeons have a responsibility in addressing underlying socioeconomic challenges that affect a patient's health?

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## Additional Reading

- [University of Washington Chapter on Taking a Social History](#)
- [University of Michigan Cultural Complications Curriculum](#)

## **Additional Resources**

### **Audible Bleeding Content**

- Audible Bleeding Exam Prep: [Social Deprivation in Vascular Surgery](#)
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# Patient History and Physical Examination of the Arterial System

Loay S Kabbani, MD, MHSA, FACS and Cassius Iyad Ochoa Char, MD, MS, RPVI

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*Listen to your patient, [they are] telling you the diagnosis.* - William Osler.

Specialized testing should always be guided by the initial clinical impressions.

Though the electronic medical record helps eliminate repetitive questions and tests, it tends to divert attention from the importance of talking to and examining the patient. Interacting with the patient and family during the history and physical is extremely important to **build rapport with the patients and their family.**

For vascular patients passing the “eyeball test” is important before discussing complex procedures. This includes knowing the level of independent living.

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## Patient History of the Arterial System

For vascular specialists, a focused arterial history should include the arteries of the neck, torso, and extremities. Many diseases are systemic. For example, atherosclerotic disease affects the carotid arteries, heart, upper extremities, abdominal blood vessels, and lower extremities. Patients with an abdominal aortic aneurysm may have synchronous peripheral aneurysms.



## Head and Neck History:

- The history should focus on the carotid artery disease—see [?@sec-carotidarterystenosis](#) for more detail—specifically, any history of stroke, transient ischemic attacks (TIA), or amaurosis fugax.
- Amaurosis fugax is described as a curtain or shade causing transient monocular blindness. This is usually caused by emboli from carotid disease on the ipsilateral side but may be caused by other embolic sources, migraine, or giant cell arteritis.
- Syncopal episodes are usually not vascular in nature. However, in patients with subclavian stenosis, syncope may be a manifestation of subclavian steal syndrome.
- History of radiation to neck, prior carotid interventions or prior surgery to the neck should be elucidated and prior studies should be obtained.

## Upper Extremity History:

- Evaluation of the upper extremities should focus on the timing (acute vs. chronic) and degree of ischemia (claudication vs. chronic limb-threatening ischemia).
- **Acute limb ischemia (ALI)** is a sudden occlusion of the blood supply with no time for collateral vessels to develop. It is characterized by the **6 Ps**. *Pulselessness, pain, pallor, paresthesia, poikilothermia (cold), and paralysis*. The most common etiology of acute limb ischemia is embolic. However, uncommon causes of ALI in the upper extremity may include thrombosis of a subclavian–axillary aneurysm.
- Intermittent hand coldness associated with pain and numbness may reflect Raynaud’s syndrome (associated with cold exposure) or vasospasm of small vessels due to conditions like frostbite or scleroderma.
- Raynaud’s phenomenon presents as episodes of vasospasm in response to cold or stress. During an attack, the patient describes the affected areas turning white, blue, and red associated with feeling cold and numb. As the circulation improves and the affected areas turn red, the patient may experience throbbing, tingling, and swelling.
- Episodes of ischemia to the digits may be persistent, severe and associated with underlying obliterative microangiopathy and manifest with pain and tissue loss (ulceration or gangrene).
- Chronic ischemia of the upper extremity is uncommon and can manifest with arm claudication (exertional fatigue). The etiology is most commonly proximal obstruction due to atherosclerotic disease in the subclavian artery. Uncommon causes can include Takayasu arteritis and thoracic outlet syndrome (TOS).
- Exertion of the arm causing posterior cerebral circulation symptoms (diplopia, dysarthria, dizziness, drop attacks, vertigo, syncope, and ataxia) may reflect a subclavian steal syndrome caused by subclavian artery stenosis proximal to the vertebral artery.

## Abdomen Arterial History:

- A majority of vascular pathology in the abdomen can be attributed to aneurysmal disease or atherosclerotic disease. Other etiologies of abdominal vascular diseases can include compression syndromes, embolisms, or dissections.
- Aneurysmal disease in the abdomen is usually asymptomatic.
- Patients with known abdominal aortic aneurysm (AAA) who present with flank or back pain should be presumed to have a ruptured aneurysm until proven otherwise.
- On the other hand, mesenteric atherosclerosis can present with a constellation of symptoms. The typical trifecta of symptoms is weight loss, sitophobia (fear of food), and postprandial pain. Atherosclerosis of the renal arteries, known as renal artery stenosis, may present as severe hypertension, especially in young adults.
- Aortoiliac occlusive disease may present with Leriche's syndrome. **Leriche's syndrome** is defined as bilateral hip and buttock claudication, absent femoral pulses, and impotence.
- Vascular etiologies of acute abdominal pain include ruptured AAA aneurysm, which presents as severe pain radiating to the back or acute mesenteric ischemia, which presents as pain out of proportion to physical exam

## Lower Extremities History:

- An evaluation of the lower extremities for peripheral artery disease should focus on the timing (acute vs. chronic) and degree of ischemia (claudication vs. chronic limb-threatening ischemia).
- **Acute limb ischemia (ALI)** is the sudden occlusion of the blood supply to a peripheral limb. It is typically characterized by the **6 Ps**. *Pulselessness, pain, pallor, paresthesia, poikilothermia (cold), and paralysis*.
- **Claudication** is defined as cramping pain in the leg induced by exercise and relieved by rest. It occurs after a fixed and reproducible distance and resolves with rest. The pain is described as discomfort, cramping, numbness, or tiredness in the legs. Claudication most commonly occurs in the calf muscles, but it can also affect the feet, thighs, hips, and buttocks. It is crucial to determine to what extent the patient's claudication affects their lifestyle during history taking.
- **Table 1** lists various differential diagnosis of leg pain, which can help differentiate claudication from pseudoclaudication and other types of leg pain. Table 1 is also helpful in differentiating between other types of leg pain, pseudoclaudication, and true claudication.
- **Chronic limb-threatening ischemia (CLTI)** is when a patient with PAD has rest pain or tissue loss. CLTI manifests as rest pain or tissue loss. Rest pain is characterized as pain in the dorsum of the foot and toes at rest (i.e., without exertion such as walking). The pain may worsen with leg elevation and many patients wake up in the middle of the

due the pain. They often state the pain improves by dangling their affected limb over the side of their bed, which leads to better blood flow to the foot thanks to gravity.

- Tissue loss is often present in the form of a nonhealing wound, ulcer, or gangrene anywhere along the toes, foot, or lower leg. It is imperative you determine how long the tissue loss has been present and if it has been improving. Often, patients with CLTI have multiple levels of arterial disease and are at a much higher risk of amputation compared to patients with simple claudication.

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## Physical Exam of the Arterial System

The arterial system is extensive, and a proper vascular exam should include the entire arterial system.

The exam starts with the nurse checking the vital signs and bilateral upper extremity blood pressures. A difference of > 10 mmHg between the upper extremities may indicate significant hemodynamic stenosis.

### **i** Key Notes

**Arterial Bruit:** Normal arterial flow is laminar and therefore silent. However, when blood flow becomes turbulent it can often be heard (bruit) or palpated (thrill). Although murmur and bruit are synonymous, audible turbulence originating from the heart is called a murmur. Outside of the heart, audible turbulence is referred to as a bruit. The presence of a bruit does not necessarily indicate significant arterial stenosis.

**Pulses are described as:**

- 0 or absent
- 1+ or diminished
- 2+ or normal
- 3+ or prominent or aneurysmal.
- 4+ or bounding or aneurysmal.

In patients with weak pulses, the examiner must take care not to confuse the patient's pulse with involuntary muscle twitches or their own pulse.

When a pulse is not palpated, a Doppler probe is used to assess the blood flow. The doppler signal can be triphasic, or biphasic, or monophasic.

**Doppler Signals:**

- Triphasic and biphasic doppler signals indicate good blood flow.

- Monophasic signal correlates with moderate to severe decrease in arterial blood flow.
- In severe ischemia, a soft continuous venous signal may be all that is heard.

Listen to examples of Doppler signals [here](#).

**On Exam:**

- Temperature changes may help demarcate the level of disease.
- Sensory loss may be present in acute ischemia, chronic limb threatening ischemia, and chronic neuropathy.

## Head and Neck Exam:

### Inspection:

- Pulsatile masses in the neck are usually tortuous carotid arteries mistaken for carotid aneurysms. Carotid aneurysms are usually near the carotid bifurcation, while tortuous carotid arteries are usually at the base of the neck. Carotid body tumors are also at the carotid bifurcation. Both carotid body tumors and aneurysms are not visible until they are large.
- If the patient complains of amaurosis fugax (i.e., transient vision loss), fundoscopy may reveal cholesterol plaques called Hollenhorst plaques. These are thought to originate from the carotid plaque.

### Palpation:

- The carotid pulse is palpated on the medial border of the sternocleidomastoid muscle. Carotid palpation is generally not performed routinely as it may cause a syncopal episode in elderly patients with sensitive carotid bulbs.
- A robust temporal pulse anterior to the ear is a sign of a patent common and external carotid artery sign.
- A large supraclavicular pulse may indicate an enlarged subclavian artery. Otherwise, the subclavian artery is usually not palpable.

### Auscultation:

- Auscultate the carotid arteries for bruits.

- Using the stethoscope bell, you can hear the S1, and S2 heart sounds in the carotid artery in the mid-neck. A bruit heard in the neck is not normal. This could be transmitted from the heart or could be from a kink or narrowing in the carotid artery. The carotid bruit is loudest in the mid neck over the carotid bifurcation. A heart murmur is loudest in the upper chest.
- The intensity of the bruit and pitch do not correlate with the severity of stenosis. A tight stenosis may have low flow and thus a faint bruit.
- When a carotid bruit is heard, only 25% will have significant stenosis (75% or greater), and 50% will not have any stenosis.

## Upper Extremity Exam:

### Inspection

- Pink fingertips with capillary refill times  $< 2$  seconds are a reliable sign of adequate perfusion. Ischemia in the extremities manifests as paleness with poor to no capillary refill. Chronic ischemia manifests with muscle atrophy.
- Raynaud phenomenon is characterized by a sharply demarcated triphasic color change after exposure to cold or emotional stress. First, the capillaries contract after the stressor, causing a characteristic white appearance. Then, as the capillaries open a little, deoxygenated blood re-perfuses sluggishly which leads to a hypoxic blue color of the distal extremity. Finally, when the capillaries recover and hyper-dilate, the affected limb becomes red and hyperemic. Raynaud's phenomena may occur idiopathically (Raynaud's Disease) or secondarily due to autoimmune disease. It is often provoked by emotional distress or exposure to the cold.

### Palpation

- Palpate the axillary artery in the upper arm in the groove between the biceps and triceps muscle.
- Palpate the brachial artery in the antecubital fossa just medial to the biceps tendon.
- Palpate the radial artery on the wrist's flexor surface just medial to the radial styloid.
- Palpate the ulnar artery on the wrist's flexor surface just medial to the distal ulna; it lies deeper than the radial artery and may not be palpable.
- Absent pulses should initiate a search for a cause such as proximal atherosclerotic stenosis in older adults or autoimmune disease such as Takayasu's in young females.
- Aneurysm of the subclavian artery and axillary artery (assessed above and below the clavicle) are difficult to palpate if small. Brachial artery aneurysms are usually pseudoaneurysms from trauma or arterial access. Ulnar artery aneurysm occurs from repetitive trauma in proximity to the hamate bone and manifests as hypothenar hammer syndrome.

## **Auscultation**

- Listen for a bruit in the supraclavicular fossa over the subclavian artery.
- When pulses are not palpable, a doppler is used to assess blood flow in the arteries.
- A blood pressure difference  $> 10$  mmHg reflects hemodynamically significant stenosis in the innominate, subclavian, or axillary arteries. In these situations, the higher blood pressure is reflective of the patient's actual blood pressure.

## **Chest and Abdominal Exam:**

### **Inspection**

- The aorta usually is typically not visible on the exam. However, a large aneurysm may be seen pulsating between the xiphoid and umbilicus, especially in thin patients.

### **Palpation**

- The aorta bifurcates at the level of the umbilicus. To palpate the aorta, press your fingers on both sides of the midline between the umbilicus and the xiphoid. To help relax the abdomen, ask the patient to bend their knees, flex their hips, and relax their abdominal muscle. The goal is not only to feel the aortic pulse but also to estimate the size of the aorta. In people without aortic aneurysms, a palpable aorta is often the size of the patient's thumb. A tender, enlarged, pulsatile abdominal mass may represent a symptomatic aortic aneurysm or inflammatory aneurysm.
- The sensitivity of palpation to detect an abdominal aortic aneurysm is low (29%) for small (3.0- 4cm) aneurysms. Moreover, even large aneurysms  $> 5$  cm may not be detected on physical exam (sensitivity of 76%). False positives can be found in elderly patients who have tortuous anterior placed aorta. It is important to mention that palpation of an abdominal aortic aneurysm is safe and has never been reported to precipitate aortic rupture. When an aortic aneurysm is identified, A complete peripheral arterial examination should be performed looking for evidence of distal embolization, ischemia or associated peripheral artery aneurysms (femoral, popliteal).
- The iliac arteries lie deep in the pelvis and are usually not palpable, even if aneurysmal.

### **Auscultation**

- Cardiac auscultation is performed to assess rate and rhythm with special attention to the presence of any arrhythmias, gallops, and murmurs.
- Bruits in the abdomen are associated with arterial stenosis. The origin of the bruit could be renal, mesenteric, or aortoiliac.

## Lower Extremity Exam:

### Inspection

- Pallor, cyanosis, and poor capillary refill are signs of chronic limb ischemia. Muscle atrophy, hair loss, and thick toenails may also be present.
- **Dependent rubor** and pallor with elevation indicate advanced peripheral occlusive disease. Dependent rubor is hyperemic erythematous discoloration of the limb in a dependent position (sitting or standing). That is, dependent rubor is due to maximally dilated capillaries and the effects of gravity. However, the limb becomes pale once the foot is elevated (the patient lies down). Dependent rubor is usually associated with rest pain and edema. It is frequently misdiagnosed as cellulitis.
- Ulcers need to be identified as neuropathic ulcers or ischemic ulcers. Neuropathic ulcers are at pressure points over the plantar aspect of the metatarsal head. Ischemic ulcers are more often towards the tip of the toes.
- *Livedo Reticularis*: Violaceous mottling of the skin with a reticular pattern of the skin of the arms and legs. The term “livedo racemosa” is used for cutaneous findings in inflammatory or thrombotic vascular disease patients.
- *Acrocyanosis*: is defined as bluish discoloration of the extremities due to high deoxygenated blood in the capillaries. It is a persistent disorder without episodic triphasic color response.
- Microembolic disease can manifest as blue toe syndrome/trash foot.
- Dry skin is present in chronic limb ischemia because the sebaceous glands function poorly without adequate blood flow.
- Edema is called “pitting” when the indentation persists after applying pressure to a small area. *Pitting edema* is associated with systemic diseases like heart failure, chronic kidney disease, hypoproteinemia, or local disease of the veins or lymphatic. Non-pitting edema is observed when the indentation does not persist. It is associated with myxedema, lipedema, and advanced lymphedema.
- *Claudication*: Patients with claudication may have no significant finding on inspection apart from muscle atrophy or hair loss.

### Auscultation

- Auscultate the femoral region for the presence of any bruits. Auscultation may also find continuous bruits which are likely due to an arterio-venous fistula.

### Palpation

- Femoral pulse: palpated under the inguinal ligament, two-finger breaths from the pubic tubercle.

- Popliteal pulse: with the patient’s knee flexed, both hands are wrapped around the knee, and the tips of the fingertips are pressed into the popliteal space (posteriorly). The pulse is located slightly lateral to midline. A normal popliteal artery may not be palpable.
- Dorsalis Pedis: palpated in the dorsum of the foot between the first and second extensor tendons. Located just lateral to the tendon of extensor hallucis longus. You can ask the patient to extend their great toe to identify the tendon. Posterior tibial: palpated by posterior to the medial malleolus. It is easier to palpate with the foot passively dorsiflexed.
- Peroneal artery: not palpable. However, it can be found with a doppler on the lateral aspect of the ankle
- When a pulse is not palpated, a doppler is used to assess the blood flow. The doppler signal can be triphasic, biphasic, or monophasic.
- Triphasic and biphasic doppler signals indicate good blood flow.
- Monophasic signals correlate with a moderate to severe decrease in arterial blood flow.
- In severe ischemia, a soft continuous venous signal may be all that is heard.
- Temperature changes may help demarcate the level of disease.
- Sensory loss may be present in acute ischemia and chronic neuropathy.

### Differential diagnosis of claudication

Table 1: **Table 1. Differential diagnosis of claudication** *Adapted from: Dormandy JA, Rutherford RB. Management of peripheral arterial disease (PAD). TASC Working Group. TransAtlantic Inter-Society Consensus (TASC). J Vasc Surg 2000; 31:S1.*

Condition	Location of pain or discomfort	Character of discomfort	Onset relative to exercise	Effect of rest	Effect of body position	Other features
Vascular claudication	Muscles of the buttock, thigh, or calf. Rarely the foot	most common is cramping. May complain of aching, fatigue, weakness, or pain	onset of pain is after some degree of exercise	Relieved by rest.	None	Reproducible



Condition	Location of pain or discomfort	Character of discomfort	Onset relative to exercise	Effect of rest	Effect of body position	Other features
Nerve root compression (eg, herniated disc)	Radiates down leg, usually posteriorly, usually from the back	Sharp lancinating pain. Electric.	Soon after, if not immediately after onset	Not quickly relieved. Often present at rest too.	Adjusting back position may relieve pain	History of back problems
Spinal stenosis	Hip, thigh, or buttock (within affected dermatome)	Motor weakness more prominent than pain	After standing for some length of time. Also may occur after walking confusing it with vascular claudication	Relieved by resting only if position changed	Relieved by lumbar spine flexion (sitting or stooping forward)	Frequent history of back problems, provoked by intraabdominal pressure
Hip arthritis / knee arthritis	Hip, thigh, buttocks. Or knees	Aching discomfort, usually localized to hip and gluteal region or knees	After variable degree of exercise. Or standing.	Not quickly relieved (and may be present at rest)	More comfortable sitting (ie, weight taken off legs)	Variable, may relate to activity level, weather changes. Tenderness when pressing on hip or knee area
foot arthritic, inflammatory processes	Foot, arch	Aching pain	After variable degree of exercise	Not quickly relieved (and may be present at rest)	May be relieved by not bearing weight	Variable, may relate to activity level

Condition	Location of pain or discomfort	Character of discomfort	Onset relative to exercise	Effect of rest	Effect of body position	Other features
Venous claudication	Entire leg, but usually worse in thigh and groin	Tight, bursting pain	After walking	Subsides slowly	Relief speeded by elevation of the extremity	History of iliofemoral deep vein thrombosis, signs of venous congestion, edema, venous stasis dermatitis or ulcers
Diabetic Neuropathy	numbness is sock like fashion in both feet	tingling, numbness.	constant	-	-	poorly controlled diabetes.
Night time cramps	foot, calf and thigh	crampy pain at night	not related to exercise	-	-	may be associated with use of diuretics.

# **Carotid Disorders**

# Carotid Artery Stenosis

Angela Kokkosis, MD, FACS and Michael Malinowski, MD, MEHP, FACS

## Note

By the end of this chapter, students will:

- Review the foundational knowledge to appraise asymptomatic and symptomatic carotid artery stenosis.
- Identify and describe risk factors, symptoms, and signs of carotid artery disease.
- Review and describe indications and contraindications for medical and surgical management of carotid artery stenosis.
- Propose next best steps in patient work-up and treatment of carotid artery disease.
- Describe the evidence-based outcomes of surgical management of carotid artery disease.

## Key Facts

1. Stroke is the leading cause of disability nationally and 3rd leading cause of death with a breakdown of 80% occlusive (ischemic) and 20% hemorrhagic.
2. Symptomatic carotid stenosis presents with only three symptoms including: stroke, transient ischemic attack (TIA) and amaurosis fugax (transient retinal ischemia).
3. Risk factors include: age, smoking, CAD, diabetes, hypertension, hyperlipidemia and genetic/family history.
4. Seminal studies include ACAS and NASCET. They define an 11% five-year risk of stroke for asymptomatic carotid artery stenosis (>60%) and 24% two-year risk of stroke for symptomatic disease (>70%), respectively.
5. The CREST landmark study associates carotid artery stenting with higher perioperative risk of stroke and carotid endarterectomy (CEA) with higher risk of myocardial infarction (MI) in symptomatic patients.
6. Carotid artery stenting (CAS) is indicated for recurrent stenosis after CEA, neck immobility, high carotid bifurcation, contralateral occlusion, high risk open surgical candidate due to cardiopulmonary comorbidity and neck radiation.

7. Transcarotid artery revascularization (TCAR) is available for direct common carotid delivery of stent while avoiding aortic arch manipulation and providing embolic protection through flow reversal.
8. There is a marginal benefit for intervention for asymptomatic carotid artery stenosis patients with significant cardiopulmonary disease due to risks of associated perioperative events.

#### Carotid Disease Slide Deck

Please find the slide deck corresponding to this eBook chapter [here](#).

Please find a video recording of Dr. Kokkosis and Dr. Malinowski's lecture using the above slide deck [here](#).

#### How We Suggest Using the Pre/Post Questions

The pre/post questions are listed below. They are all multiple choice questions with a single right answer. To best guide your learning, we have hidden the answers in a collapsible menu. Before reading the chapter, we suggest giving the questions a try, noting your answers on a notepad. After reading the chapter, return to the questions, re-evaluate your answers, and then open the collapsible menu to read the correct answer and discussion. Do not fret if you have difficulty answering the questions before reading the chapter! By the end of the chapter, we are certain you will have covered the knowledge necessary to answer the questions. There will be a teaching case at the end of the chapter. This is another opportunity to exercise your new knowledge!

## Pre/Post Questions

### Case Based Questions

1. A 75-year-old male smoke presents with recent visual changes to his right eye that occurred yesterday. The patient reports a shading of his visual field that resulted in momentary monocular blindness followed by return to normal vision. He has a carotid duplex showing >50% diameter reduction to his right internal carotid artery and >80% stenosis to his left internal carotid artery. *What treatment should be offered to his patient?*
  - A. Emergent DC cardioversion to treat any underlying arrhythmia.
  - B. Left carotid endarterectomy with shunt placement.
  - C. Left carotid TCAR.
  - D. Right carotid endarterectomy.

E. Placement on Apixaban and measurement of PF4 with medical management.

Answer

**D. Right carotid endarterectomy**

Discussion: The patient has evidence of amaurosis fugax with temporary monocular blindness as a sign of symptomatic right internal carotid artery disease. His left carotid artery is in a high-grade range and therefore is lower risk of stroke than the contralateral symptomatic lesion. There should be no immediate surgical treatment of the asymptomatic left carotid lesion. There is no evidence that the patient has an underlying arrhythmia that needs cardioversion and medical management with Apixaban and PF4 levels are unrelated to this patient's current pathology.

2. A 50-year-old female patient with >80% right internal carotid artery stenosis presents to clinic for her first postoperative visit after carotid endarterectomy (CEA). She has no interval neurologic events since her discharge, has a soft neck with a clean incision. During your neurologic exam you notice an unintentional, subtle tongue deviation to the side of surgery. *What is the most likely facial nerve involved in this finding?*

A. Vagus Nerve.

B. Hypoglossal Nerve.

C. Glossopharyngeal Nerve.

D. Long Thoracic Nerve.

E. Hering's Nerve.

Answer

**B. Hypoglossal Nerve**

Discussion: This patient has evidence of hypoglossal nerve neuropraxia which can occur as the result of injury or traction on the nerve during distal dissection of the internal carotid artery. Although both glossopharyngeal nerve injury and vagal injuries can also occur during CEA, the former causes oropharyngeal dysfunction with swallowing and the latter causes vocal cord paralysis leading to a hoarse voice. The long thoracic nerve is more related to thoracic outlet surgery and innervation of the serratus anterior. The Nerve of Hering is related to carotid sinus innervation and is unrelated to this clinical finding.

3. During the initial evaluation of a patient with high grade symptomatic carotid disease, you notice that the patient has internal carotid plaque on that side this is above the angle of the mandible at the 1st cervical vertebral body (C1). This appears to be too high to access through open surgery. The patient has no know history of coronary artery

disease, has a preserved ejection fraction and good functional status. He has a low-density lipoprotein level (LDL) of 200 mg/dL. *What is the best treatment option for this individual?*

- A. Transfemoral carotid artery angioplasty with placement on ASA only.
- B. Carotid artery enterectomy with shunt placement.
- C. Daily ASA therapy without any type of statin or antihypertensive therapy.
- D. Transcarotid artery revascularization (TCAR) surgery with dual antiplatelet therapy.
- E. No treatment is indicated.

Answer

**D. Transcarotid artery revascularization surgery with dual antiplatelet therapy.**

Discussion: This patient has symptomatic carotid stenosis with an overall optimal coronary health. Because his lesion is high at C1 and not surgically accessible, they would not qualify for a traditional carotid endarterectomy. Transfemoral carotid artery angioplasty is not indicated without stent placement, and daily ASA therapy without statin treatment for an LDL of 200mg/dL does not qualify as optimal medical management. Surgical treatment is indicated in this symptomatic patient since he has no significant cardiopulmonary disease and TCAR is the only option listed that could accomplish this outcome successfully.

4. A 60-year-old otherwise healthy woman, with no underlying comorbidities has a right carotid artery bruit on physical examination. She is concerned that she is at risk for stroke. She denies any episodes of vision changes, upper or lower extremity deficits, or speech impairments. She currently takes 81mg of aspirin daily, along with a multivitamin. *What is the next step in management for this patient?*
- A. No further treatment necessary.
  - B. Carotid duplex.
  - C. CT angiogram head and neck.
  - D. Neurology evaluation.
  - E. Addition of statin therapy.

Answer

**A. No further treatment necessary.**

Discussion: This patient has an incidental finding of a carotid bruit on physical exam,

however she has no risk factors for carotid disease (such as smoking, hyperlipidemia, smoking, family history, diabetes or hypertension). Additionally, she is neurologically asymptomatic. Therefore, carotid duplex, CTA, neurology evaluation, and the addition of statin therapy are not indicated. The prevalence of >75% carotid stenosis for those with a carotid bruit has been found to be very low at 1.2%.

5. A 65-year-old man who is right-handed is undergoing evaluation for a coronary artery bypass graft surgery (CABG). As part of his workup, a carotid duplex is performed which demonstrates a chronic right carotid occlusion and a >80% stenosis of his left carotid artery. He has no history of ocular or cerebrovascular events. He has hypercholesterolemia and well-controlled hypertension. *What is the next best step for this patient?*
- A. Proceed with the CABG as planned and continue medical therapy with aspirin and statin.
  - B. Left carotid endarterectomy before the CABG.
  - C. Place the patient on dual antiplatelet therapy, in addition to statin therapy.
  - D. Left TCAR (transcarotid stent) after the CABG.
  - E. Place the patient on anticoagulation.

Answer

**B. Left carotid endarterectomy before CABG.**

Discussion: It is standard of care to assess the carotid arteries prior to performing a CABG, with the goal of minimizing the risk of perioperative stroke. This patient has asymptomatic bilateral severe carotid disease (right occlusion and left >80% stenosis). Therefore, to reduce the risk of perioperative stroke, current guidelines recommend carotid revascularization prior or concomitant with the CABG. The patient does carry one high risk criterion for TCAR (coronary disease requiring revascularization), however this should not be performed after the CABG. Dual antiplatelet therapy is indicated in the event this patient undergoes carotid stenting, however medical management should not be the only management of his carotid disease. Lastly, anticoagulation has no role in atherosclerotic disease, such as carotid disease.

6. A 55-year-old woman has a past medical history of coronary artery disease status post coronary stenting in 2019, hypertension, hyperlipidemia, and previous smoking history of 60 pack-years. Her cardiologist sends her for a carotid duplex which demonstrates a 50-69% carotid stenosis on the left side, and mild atherosclerosis on the right side. She denies any prior signs or symptoms of stroke or transient ischemic attack. *How should this patient's carotid disease be managed?*



- A. Left carotid endarterectomy, along with aspirin/statin therapy.
- B. Left transfemoral carotid stent, along with dual antiplatelet/statin therapy.
- C. Aspirin and statin therapy **only**, and routine surveillance carotid duplexes.
- D. Aspirin, statin therapy, blood pressure management, and routine surveillance carotid duplexes.
- E. No further management is indicated.

Answer

**D. Aspirin, statin therapy, blood pressure management, and routine surveillance carotid duplexes.**

Discussion: This patient has asymptomatic 50-69% left carotid stenosis with the associated risk factors of vascular disease which include hypertension, hyperlipidemia, and smoking history. Medical therapy needs to be employed to reduce these factors, thus a single antiplatelet, statin, and blood pressure medication are indicated. Fortunately, the patient is not a current smoker, however if she was, then smoking cessation counseling would be added to her treatment plan. Carotid revascularization is indicated only for asymptomatic carotid stenosis that is >70% based on current guidelines.

## Operative Footage Questions

These questions are associated with the carotid endarterectomy (CEA) footage (**short version**) found at the bottom of the chapter.

1. What is the first muscle layer encountered in a CEA (i.e. the first muscle deep to skin)?
  - A. Sternocleidomastoid
  - B. Digastric
  - C. Platysma
  - D. Scalene

Answer

**C. Platysma**

Discussion: The platysma is the most superficial muscle in the neck. It covers most of the anterior and lateral aspect of the neck. It is the first muscle layer encountered in the neck during a CEA. It will be bisected and repaired upon neck closure. While the sternocleidomastoid is superficial, it is deep to the platysma. The sternocleidomastoid is an important landmark as it forms the anterolateral boundary of the carotid triangle.

You will dissect along its medial border and retract it laterally in order to access the carotid sheath. The digastric muscle is a small, “two-bellied” muscle located under the mandible. The posterior belly forms the superior border of the carotid triangle. It is often visualized in patients with high carotid bifurcations. The scalene muscles are deep to the sternocleidomastoid muscles and lateral to the cervical spine. They are not manipulated in a CEA.

2. What structure is **not** found in the carotid sheath?

- A. Internal jugular vein
- B. External jugular vein
- C. Common carotid artery
- D. Vagus nerve

Answer

**B. External jugular vein**

Discussion: The external jugular vein is not in the carotid sheath. It runs superficial to and obliquely across the sternocleidomastoid before passing to the posterior border of the sternocleidomastoid as it descends deep into the neck. The carotid sheath contains the common carotid artery anteromedially, the internal jugular vein anterolaterally, and the vagus nerve posteriorly.

3. In what order do you **unclamp** the carotid vessels at the end of a carotid endarterectomy?

- A. External carotid artery → common carotid artery → internal carotid artery
- B. Common carotid artery → internal carotid artery → external carotid artery
- C. Internal carotid artery → external carotid artery → common carotid artery
- D. Internal carotid artery → common carotid artery → external carotid artery

Answer

**A. External carotid artery → common carotid artery → internal carotid artery**

Discussion: In conventional CEA, the internal carotid artery (ICA), the common carotid artery (CCA), and the external carotid artery (ECA) are clamped in that order such that any atherosclerotic debris loosened during vessel manipulation and/or clamping embolizes into the ECA where it could cause extracranial ischemia rather than the ICA where it could cause intracranial ischemia (i.e. ischemic stroke). A useful mnemonic to remember

the clamping order is **ICE** for **ICA**, **CCA**, and **ECA**. The order when unclamping is the opposite, **ECI**. The rationale is similar. By unclamping the ECA first, remaining debris or debris loosened during removal of the clamps will flow up the ECA rather than the ICA. By unclamping the CCA second, and dislodged debris will flow down the open ECA rather than the ICA. The ICA is unclamped last. Options A and B are dangerous as they do not minimize the risk of embolus flowing into the ICA. Option C is simply incorrect.

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## Introduction

Hemispheric stroke related to carotid artery stenosis is a leading cause of both disability and death in the United States. Underlying etiologies for stroke include occlusive or hemorrhagic events with roughly 80% being related to occlusive pathology through embolus or in-situ thrombosis, the remaining 20% attributable to hemorrhage. Roughly 15% of stroke victims have a transient ischemic attack (TIA) that fully resolved prior to a later stroke event. Risk factors for carotid plaque formation are related to age, smoking, coronary artery disease, diabetes, hyperlipidemia, hypertension and family history of stroke. Due to carotid bulb anatomy, the most common area of plaque formation is within the proximal internal carotid artery. As plaque stenosis increases over time, the systolic velocity increases to maintain flow volumes which intensifies shear stress. This shear stress increases likelihood of plaque rupture, platelet aggregation and thromboembolization. There are multiple seminal studies that describe cohort comparisons of asymptomatic and symptomatic carotid artery stenosis with outcomes related to optimal medical management alone or as adjunct to surgical repair. Symptomatic carotid stenosis is described as carotid stenosis >50% with unilateral stroke, TIA or amaurosis fugax on the side of carotid disease. Amaurosis fugax is historically described as shade coming down across one eye on the side of stenosis to produce partial or complete, painless monocular visual loss related to transient retinal ischemia. Amaurosis fugax may be bilateral in the case of bilateral, symptomatic carotid artery stenosis.

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## Etiology

Atherosclerosis is the most common cause for the development of carotid artery disease. This process is defined by deposition of lipid-laden plaque at the carotid bifurcation, and potentially across a larger territory of the common carotid, external carotid, and internal carotid arteries. This plaque may contain varying degrees of calcification and/or thrombus. The mechanisms

by which atherosclerosis at the carotid bifurcation may lead to stroke or TIA are: occlusion (cessation of blood flow to the internal carotid artery) or embolization (plaque debris break off and travel through the internal carotid artery to the brain). There are various risk factors which may contribute to the degree of atherosclerosis and its progression. These include history of cigarette smoking, hyperlipidemia, coronary artery disease, diabetes, hypertension, advanced age, and family history of carotid disease or stroke.

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## **Diagnostics and Imaging**

Three primary imaging modalities are used to evaluate carotid artery stenosis with the lowest cost option being color flow duplex ultrasound (DSA) that allows a physician to determine peak systolic and end diastolic velocities throughout the carotid bifurcation. Based on the internally validated vascular laboratory criteria of the institution, these velocities can be correlated to ranges of degree of stenosis, with high grade stenosis defined as >70-80%. Since the modality is based primarily on velocity range, it cannot give exact stenosis such as 66%. The modality can also provide adjunct information about the blood flow waveforms in each arterial segment, as well as whether that flow is laminar or turbulent with utilization of color flow imaging. Limitations of this imaging include technician skill, inability to obtain optimal angle of Doppler interrogation for velocity determination, shadowing from heavily calcified lesions, poor visualization due to patient habitus and tortuosity.

Axial imaging options include both computed tomography angiogram (CTA) and magnetic resonance angiogram (MRA). Both of these options require some form of intra-arterial contrast, either iodinated contrast or gadolinium, respectively. However, they offer a fuller perspective of relevant anatomy and a more precise determination of stenosis within the limitations of the modality, with MRA often overestimating the degree of stenosis due to intrinsic properties of MRA imaging acquisition. Both CTA and MRA, although superior to DSA in determination of exact degree of plaque stenosis and arterial anatomy, sacrifice the physiologic information offered through DSA that speak to flow patterns, flow direction and turbulence. Axial imaging of CTA and MRA can define patency but do not speak to the dynamic nature of blood flow or directionality of flow.

Definitive determination of flow and directionality can be augmented to a carotid artery stenosis workup by diagnostic angiography. This requires femoral artery access and includes contrast administration, as well as a small risk of periprocedural embolization. However, it offers additional physiologic evaluation that might not be present in DSA and that is inherently lacking in CTA and MRA studies.

## Asymptomatic Carotid Artery Stenosis Screening

The 2021 SVS clinical practice guidelines outline the following recommendations. In asymptomatic patients who qualify for carotid artery stenosis screening, duplex ultrasound is the recommended choice over CTA, MRA, or other imaging modalities.

Routine screening is **not recommended** for clinically asymptomatic carotid artery stenosis for individuals **without significant risk factors** for carotid disease.

Screening is **recommended** for clinically **asymptomatic carotid artery stenosis in individuals** with significant risk factors\*\* for carotid disease. High-risk groups include:

- Patients with lower extremity peripheral artery disease (PAD)
- Patients undergoing coronary artery bypass surgery (CABG)
- Patients aged  $\geq 55$  years with at least two traditional atherosclerotic risk factors (hyperlipidemia, hypertension, etc.)
- Patients aged  $\geq 55$  years and active cigarette smoking
- Patients with diabetes, hypertension, or coronary artery disease (CAD)
- Patients with clinically occult cerebral infarction noted on brain imaging studies.

The presence of a carotid bruit increases the likelihood of detecting significant stenosis. Asymptomatic patients with an abdominal aortic aneurysm (AAA) or previous radiotherapy to the neck *who do not meet the criteria of any of the high-risk groups* above do not require screening. It has been shown that the prevalence of carotid stenosis increases proportionally with the number of risk factors present.

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## Treatment

### Carotid Artery Endarterectomy (CEA)

This procedure has been performed since the 1950s, either by plaque endarterectomy and patch angioplasty or primary arterial repair. To prevent arterial restenosis, patch angioplasty has become the standard of arterial closure after plaque removal. The procedure can involve cerebral monitoring including electroencephalography (EEG), transcranial Doppler (TCD) and stump pressure monitoring or be performed awake to directly monitor patient motor response. Endarterectomy and patch repair can be performed under a “clamp and sew” mentality or with an arterial shunt to maintain cerebral perfusion. Risks include cardiopulmonary risk of acute myocardial ischemia,  $<3\%$  perioperative risk of neurologic event, neck hematoma or cranial nerve injury of roughly 5-10% affecting the vagus, marginal mandibular, recurrent laryngeal or hypoglossal nerves.

## **Carotid Artery Stenting (CAS)**

### **Transfemoral Carotid Artery Stenting (TFCAS) with Embolic Protection**

Transfemoral stenting requires some type of protection from embolization including a distal internal carotid artery retrievable filter or flow arrest procedure to prevent cerebral embolization during stent placement with or without angioplasty.

### **Transcarotid Artery Revascularization (TCAR)**

Treatment of a carotid stenosis that avoids aortic arch manipulation involving direct common carotid artery exposure and sheath placement to allow for transcarotid stent delivery to the internal carotid artery. The common carotid artery sheath is connected to a femoral vein sheath so that the natural arterial pressure gradient reverses flow across the distal internal carotid artery driving blood and possible embolus into the arterial tubing circuit and across a filter before it reenters the venous circulation. As FDA approval for the device was delivered in 2016, the technology is less than 10 years old without robust long-term follow up data. This technique offers a lower perioperative stroke risk than transfemoral stenting, for multiple reasons including lack of transaortic arch manipulation and great vessel cannulation which can result in embolus prior to placement of an internal carotid artery embolic protection device.

## **Optimal Medical Management**

Understandably, optimal medical management requires full risk evaluation of the individual patient in question including other comorbidities, drug allergies, compliance, etc. We have listed a few broad recommendations to follow that offer general guidance surrounding the dynamic target of optimal medical management for arterial disease.

### **Antiplatelet Therapy**

- ASA offers a 22% risk reduction in major vascular events with no difference in protection based on dosage (81 versus 325 mg).
- Clopidogrel can be used as an adjunct or alternative to ASA, but the added benefit from dual antiplatelet combination in asymptomatic carotid artery stenosis is unproven.

### **Anticoagulants**

- Only useful for prevention of cardioembolic strokes due to arrhythmia or prosthetic valve.

## **Hypertension Treatment**

- Recommended blood pressure range of <130/80 with individual antihypertensive regimen based on other comorbidities and patient risk factors.

## **Diabetic Control**

- In accordance with best practice for diabetes management, the patient's hemoglobin A1c should be <7.0

## **Smoking Cessation**

- Treatments offered include nicotine replacement therapy (NRT), varenicline or bupropion as first line agents.

## **Hyperlipidemia Management**

- Regimen goals of LDL <100mg/dl, or <70mg/dl depending on risk profile.

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## **Outcomes and Surveillance**

### **Asymptomatic carotid stenosis**

- The historically touted Asymptomatic Carotid Atherosclerosis Study (ACAS) demonstrated that patients with >60% carotid stenosis who underwent CEA benefited significantly from stroke risk reduction at 5 years (5.1% for CEA vs. 11% for optimal medical therapy consisting of aspirin alone).
- More recent studies suggest that with the current optimal medical management, which consists of antiplatelet medication and statin therapy, 5-year stroke risk is highest in patients with >70% carotid stenosis, and therefore this patient population would benefit from carotid endarterectomy.
- Patients who are deemed high risk, either due to an anatomic (such as surgically inaccessible bifurcation or restenosis after previous CEA) or physiologic findings (congestive heart failure, severe coronary artery disease, or chronic obstructive pulmonary disease), may be considered for TCAR given the equivocal results of perioperative stroke or death at 1.3%, as compared to CEA.

- Asymptomatic patients **with significant risk factors** found to have moderate stenoses (50%-79%) should be followed every 6-months to detect disease progression. High risk patients with <50% stenosis can be followed-up annually.

## Symptomatic carotid stenosis

- Patients who have >50% carotid stenosis and have developed symptoms of TIA or stroke were found to benefit from CEA in the pivotal North American Symptomatic Carotid Endarterectomy Trial (NASCET) because of the significant 2-year stroke risk reduction as compared to optimal medical management (15.7% vs. 22.2%). An even greater stroke risk reduction was seen in patients with >70% carotid stenosis (9% CEA vs. 26% medical management).
- Current management of patients with symptomatic >50% carotid stenosis who are low/standard risk is carotid endarterectomy over transfemoral carotid stenting (TFCAS), as there are no studies to date which have shown benefit of TFCAS.
- Patients who are deemed high risk, as defined above, may be considered for TCAR over TFCAS due to the significantly lower incidence of in-hospital stroke and death (1.6% vs. 3.1%).
- Post-operative surveillance (by duplex ultrasound) after open (CEA) or endovascular (TF-CAS) repair of the carotid artery is strongly recommended by the SVS to monitor for signs of restenosis in the repaired artery or atherosclerotic disease progression in the unoperated, contralateral artery. Duplex ultrasound testing is recommended within 30 days of the procedure, then every 6 months for 2 years, then annually.
- Restenosis <50% warrants the regular surveillance protocol; 50-99% warrants closer follow-up, confirmation with a CTA, and possible angiographic evaluation; 100% restenosis warrants surveillance and medical treatment of the contralateral carotid artery.
- It should be noted, there is some debate as to the economic and medical value of continuing post-operative duplex ultrasound surveillance after successful CEA with patch closure when the immediate post-operative duplex was normal or showed minimal disease.

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## Teaching Case

### Scenario

An 81 year old male with a significant smoking history and prior three vessel CABG five years ago, presents with monocular right eye blindness that occurred two days ago. He has no prior ophthalmologic conditions and states that he describes the process of a veil coming down over



his right eye with resolution about a minute later with complete return of normal vision at that point. He denies any other symptoms during the event or since, such as motor or sensory deficits, speech, etc. He did not think much of the event but presented after his wife told him to see someone about the event.

## Exam

**HEENT:** No prior neck incisions, good cervical extension.

**Cardiac:** Regular rate and rhythm. Healed sternotomy scar.

**Pulmonary:** Clear to auscultation throughout.

**Abdominal:** Soft and nontender.

**Neurologic:** All cranial nerves 2-12 intact, no lateralizing deficits, 5/5 strength to all extremities.

**Ophtho:** No visual deficits at 20 feet from eye chart.

## Imaging

### Duplex Ultrasound (Peak Systolic Velocity/End Diastolic Velocity)

Location	Right	Left
Proximal ICA	540/240 cm/s	120/45 cm/s
Mid ICA	230/145 cm/s	119/37 cm/s
Distal ICA	240/110 cm/s	110/23 cm/s

**Duplex Report:** Based on color flow duplex imaging there is evidence of 80-99% stenosis of the right internal carotid artery segment and <50% stenosis to the contralateral side.

## Discussion Points

**i** N.B. There is no prepared answers for the questions below.

However, we feel this chapter contains all the necessary information to answer the questions. If not, please let us know!

1. Please explain the pathophysiology of the visual event for this patient? Describe why it can be termed amaurosis fugax. Ensure understanding that amaurosis fugax is the result of carotid plaque embolization to the retina.

2. Please list the patient's risk factors for carotid disease? What is best medical management to optimize these risk factors?
  3. Is this patient asymptomatic or symptomatic based on the clinical scenario presented?
  4. What next steps should be pursued to offer effective and timely treatment to this patient? Please discuss adjunct imaging such as CTA or MRA to determine anatomic characteristics of the lesions such as ulceration, vessel patency, level (accessible or high lesions), etc.
  5. What surgical managements could be suggested to this patient? Please include a discussion of carotid endarterectomy, transfemoral stenting or TCAR.
  6. What medications should be started in this scenario? Please consider ASA, Plavix, statin medications, etc.
  7. What are some possible relevant complications of surgical intervention, including periprocedural stroke risk?
- 

## Key Articles

1. Ricotta JJ, Aburahma A, Ascher E, Eskandari M, Faries P, Lal BK; Society for Vascular Surgery. [Updated Society for Vascular Surgery guidelines for management of extracranial carotid disease.](#) J Vasc Surg. 2011 Sep;54(3):e1-31.(Ricotta et al. 2011)
  2. AbuRahma AF, Avgerinos EM, Chang RW, Darling RC 3rd, Duncan AA, Forbes TL, Malas MB, Murad MH, Perler BA, Powell RJ, Rockman CB, Zhou W. [SOCIETY FOR VASCULAR SURGERY CLINICAL PRACTICE GUIDELINES FOR MANAGEMENT OF EXTRACRANIAL CEREBROVASCULAR DISEASE.](#) J Vasc Surg. 2021 Jun 18. (AbuRahma et al. 2022)
  3. [Endarterectomy for asymptomatic carotid stenosis.Executive Committee for the Asymptomatic Carotid Atherosclerosis Study.](#) JAMA 1995;273(18):1421-8. (Walker 1995)
  4. [Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade stenosis. North American Symptomatic Carotid Endarterectomy \(NASCET\) Trial Collaborators.](#) N Engl J Med 1991;325(7):445-53.(“Beneficial Effect of Carotid Endarterectomy in Symptomatic Patients with High-Grade Carotid Stenosis” 1991)
  5. Howard D.P.J., Gaziano L., Rothwell P.M.: [Risk of stroke in relation to degree of asymptomatic carotid stenosis: a population-based cohort study, systematic review, and meta-analysis.](#) Lancet Neurol 2021; 20: pp. 193-202.(Howard, Gaziano, and Rothwell 2021)
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## Additional Resources

### Audible Bleeding Content

- Audible Bleeding Exam Prep: [Cerebrovascular Chapter](#)
  - Audible Bleeding has an episode covering the NASCET trial. Listen to it below and find [additional information here](#), or find the episode wherever you listen to podcasts.
  - The Audible Bleeding Holding Pressure Series has an episode about carotid endarterectomy. The Holding Pressure Series is designed specifically for medical students! Listen to the episode below and find [additional information here](#), or find the episode wherever you listen to podcasts.
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### Websites

- TeachMe Surgery: [Carotid Artery Disease](#)
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### Serious Games

#### Touch Surgery Simulations.

- Must download the [Medtronic Touch Surgery mobile application](#) to access the modules. Available for Apple and Android mobile devices.
  - [Carotid Endarterectomy](#)
  - [Carotid Artery Stenting](#)
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### Gore Combat Manual

The [Gore Medical Vascular and Endovascular Surgery Combat Manual](#) is an informative and entertaining read intended as a vascular surgery crash course for medical students, residents, and fellows alike. Highly accessible with a thoughtfully determined level of detail, but lacking in learning activities (e.g. questions, videos, etc.), this resource is a wonderful complement to the APDVS eBook.

 Tip

Please see pages 119-125.

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## **Operative Footage**

Developed by the DeBakey Institute for Cardiovascular Education & Training at Houston Methodist. *YouTube account required as video content is age-restricted.* Please create and/or log in to your YouTube account to have access to the videos.

**Carotid Endarterectomy (Short Version)**

**Carotid Endarterectomy (Long Version) Part 1**

**Carotid Endarterectomy (Long Version) Part 2**

**Carotid Endarterectomy (Long Version) Part 2**

**Transcarotid Artery Revascularization (TCAR)**

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# **Aortic Disorders**

# Abdominal Aortic Aneurysm (AAA)

Erin K. Greenleaf, MD, MS, Jonathan Bath, MD, Dawn M. Coleman, MD, FACS and Ezra Schwartz, MD,CM, MS.

## Note

By the end of this chapter, students will:

- Review abdominal aortic anatomy and aneurysm pathophysiology.
- Recognize history, physical exam, and radiologic findings typical of patients presenting with an abdominal aortic aneurysm.
- Identify indications for intervention on abdominal aortic aneurysms and the evidence-based foundation for these recommendations.
- Describe the basic procedural steps of both an endovascular and an open surgical approach for abdominal aortic aneurysm repair.
- Delineate appropriate surveillance intervals for imaging an abdominal aortic aneurysm, both before and after intervention.

## Key Facts

1. Abdominal aortic aneurysms occur in 2-8% of the population in Western countries, with a slightly higher prevalence among males relative to females.
2. The average size of an abdominal aorta is approximately 2cm; hence, an aneurysm is diagnosed when the diameter of the aorta in this segment reaches 3cm (i.e. 1.5x normal diameter).
3. Risk factors for an abdominal aortic aneurysm growth include age, male sex, white race, smoking, atherosclerosis, family history of arterial aneurysms, and personal history of arterial aneurysms.
4. According to vascular surgery guidelines, elective repair of a fusiform abdominal aortic aneurysm is recommended once the aneurysm has reached 5.5cm diameter in males and 5 cm diameter in females, measured from outer wall to outer wall. Circumstances in which repair is indicated for smaller aneurysms include those associated with symptoms and/or rupture, saccular morphology, rapid ex-

pansion, or those in females (as mentioned above), immunosuppressed patients (i.e. chemotherapy, radiation therapy, or solid organ transplantation) and patients with aortopathies (e.g. Marfan Syndrome, Ehlers-Danlos Syndrome, etc.).

5. Nearly 80% of abdominal aortic aneurysms are currently repaired via an endovascular approach, with an open surgical approach generally reserved for patients in which a stent graft would perform poorly.
6. Outcomes following endovascular abdominal aortic aneurysm repair have improved over time, as experience has accrued with endovascular technique. This pattern of progressive improvement has not similarly been seen for open surgical repair.
7. Surveillance regimens for patients with unrepaired abdominal aortic aneurysms suggest imaging via ultrasound or computerized tomography (CT) scan every three years if the aneurysm is 3.0-3.9cm diameter, every year if the aneurysm is 4.0-4.9cm, and every six months if the aneurysm is 5.0-5.4cm.

### How We Suggest Using the Pre/Post Questions

The pre/post questions are listed below. They are all multiple choice questions with a single right answer. To best guide your learning, we have hidden the answers in a collapsible menu. Before reading the chapter, we suggest giving the questions a try, noting your answers on a notepad. After reading the chapter, return to the questions, re-evaluate your answers, and then open the collapsible menu to read the correct answer and discussion. Do not fret if you have difficulty answering the questions before reading the chapter! By the end of the chapter, we are certain you will have covered the knowledge necessary to answer the questions. There will be a teaching case at the end of the chapter. This is another opportunity to exercise your new knowledge!

## Pre/Post Questions

### Case Based Questions

1. A 75-year-old male with history of hypertension, hyperlipidemia, diabetes and degenerative disk disease presents to the emergency department with abdominal pain and a blood pressure of 90/60 mm Hg. The emergency department physician already obtained a CT scan showing what appears to be a contained rupture of an infrarenal abdominal aortic aneurysm. *What is the next step in management?*
  - A. Resuscitate the patient to ensure a systolic blood pressure of at least 120 mm Hg.
  - B. Two large bore IVs and admission to the ICU for closer monitoring.
  - C. Transfer to the operating room for emergent repair of the aneurysm.

- D. Corroboration of the CT scan with an abdominal ultrasound.
- E. Contact the next of kin to request their presence at bedside for a family meeting.

Answer

**C. Transfer to the operating room for emergent repair of the aneurysm.**

Discussion: **Explanations to come shortly**

2. An 83-year-old female presents to your clinic with a 5.1cm abdominal aortic aneurysm. She has a history of chronic tobacco abuse, hyperlipidemia, osteoarthritis, and renal cell carcinoma status post left nephrectomy. She denies abdominal pain and is without significant physical exam findings. *Based on her presentation, what is your recommendation to her?*
- A. Continue surveillance with imaging every 6 months until her aneurysm reaches 5.5cm in diameter.
  - B. Discuss repair of the aneurysm at this size.
  - C. Prescribe an antiplatelet, statin, and beta blocker prior to discussing operative repair of the aortic aneurysm.
  - D. Continue surveillance with imaging every 12 months until her aneurysm reaches 5.5cm in diameter.
  - E. Tobacco cessation should be confirmed prior to committing to aneurysm repair.

Answer

**B. Discuss repair of the aneurysm at this size.**

Discussion: **Explanations to come shortly**

3. A 68-year-old male with history of hypertension, hyperlipidemia, tobacco abuse, obesity, and gout is found to have a 5.9cm infrarenal abdominal aortic aneurysm with a 21mm neck, patent common femoral arteries, but highly calcified/nearly occluded bilateral iliac arteries. *Upon discussing repair with him in your surgery clinic, what would you advise the patient about his repair?*
- A. He should undergo endovascular repair because there is at least 20mm of neck below the lowest renal artery before the aneurysm begins.
  - B. He should undergo endovascular repair with percutaneous access because his common femoral arteries are widely patent.
  - C. He should undergo endovascular repair because this will minimize his inpatient stay while maximizing the primary patency of the stent graft.



D. He should undergo open repair because his diseased iliac arteries will preclude safe passage of sheaths and a device to the level of the aorta.

E. He should undergo open repair because this will minimize his short-term morbidity.

Answer

**D. He should undergo open repair because his diseased iliac arteries will preclude safe passage of sheaths and a device to the level of the aorta.**

Discussion: **Explanations to come shortly**

4. A 78-year-old female is referred by her primary care physician after she was incidentally found to have a saccular aneurysm immediately distal to her right renal artery, which is the lower of the bilateral renal arteries. *When documenting the location of this aneurysm, what terminology should be used to label it?*

A. Infrarenal

B. Juxtarenal

C. Suprarenal

D. Infrainguinal

E. No specific term exists

Answer

**B. Juxtarenal**

Discussion: **Explanations to come shortly**

5. Two days following an open transperitoneal repair of an infrarenal abdominal aortic aneurysm, an 81-year-old male with a history of a right colectomy for colon cancer has bright red blood in his stool. He remains in the ICU with a leukocytosis and appropriately tender abdomen without peritonitis. *What is the next best step in management?*

A. Broad spectrum antibiotics and flexible sigmoidoscopy.

B. Broad spectrum antibiotics and CTA.

C. Observation as this is likely related to his previous colon resection.

D. NPO order and immediate return to the operating room.

E. Stool softeners to mitigate the risk of hemorrhoids and anal fissures.

Answer

**A. Broad spectrum antibiotics and flexible sigmoidoscopy**

Discussion: **Explanations to come shortly**

6. A 70-year-old female is undergoing an endovascular repair of an abdominal aortic aneurysm in the operating room under general anesthesia and a completion aortogram was just taken. A late blush is noted in the sac, seemingly arising from a lumbar artery. *What kind of endoleak would this suggest is present?*
- A. Type Ia endoleak; blood is filling the sac from the proximal extent of the stent graft.
  - B. Type III endoleak; blood is filling the sac from the middle and can appear to be coming from a lumbar artery.
  - C. Type Ib endoleak; blood is filling the sac retrograde from the iliac arteries and so is only seen with a late aortogram view.
  - D. Type II endoleak; flow from a lumbar artery would cause a late blush and can be followed conservatively provided that the sac does not continue to expand.
  - E. No endoleak.

Answer

**D. Type II endoleak; flow from a lumbar artery would cause a late blush and can be followed conservatively provided that the sac does not continue to expand.**

Discussion: **Explanations to come shortly**

## Operative Footage Questions

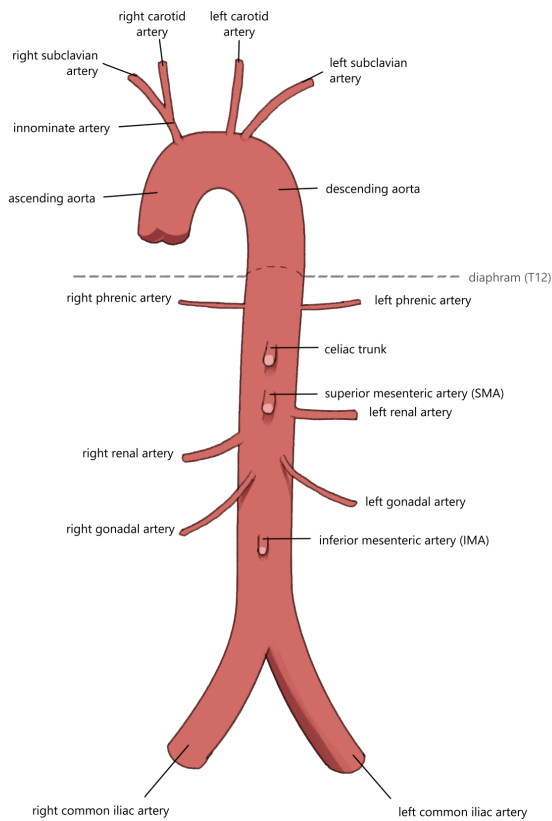
To come shortly. Stay tuned!

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## Introduction

The aorta is invariably the largest arterial conduit in the body, serving as the channel through which blood from the heart travels to every other body part. The wall of the aorta is comprised of three layers, which include the intima, media and adventitia. Its typical course begins at the distal side of the aortic valve and terminates at the aortic bifurcation, where it splits into

the two common iliac arteries. The thoracic aortic segment provides blood flow to the great vessels of the aortic arch, ensuring perfusion to the head and bilateral upper extremities via the innominate artery, the left carotid artery, and the left subclavian artery (in that order). The abdominal segment is a retroperitoneal structure that enters the abdominal cavity through the aortic hiatus of the diaphragm, at the level of T12. The branches of the abdominal aorta include, in descending order, the two inferior phrenic arteries, the celiac axis or trunk, the superior mesenteric artery (SMA), the renal arteries, the inferior mesenteric artery (IMA), and ultimately, the bilateral common iliac arteries. The branches of both the thoracic and abdominal aorta provide blood flow to areas of critically vital viscera.



APUS Medical  
Shuai Cui  
- Ivana Zhang -

Figure 1: Anatomically normal aorta with main and visceral branches.

While the aorta serves a critical role for the rest of the body, it can be the site of significant pathology. Aneurysms, which are common pathology in both the thoracic and abdominal aorta, are defined by an increase in arterial diameter to 1.5 times the normal diameter of the vessel. As

the diameter of the aortic aneurysm increases, the risk of rupture increases in direct proportion. For example, a patient with a 4cm abdominal aortic aneurysm (AAA) has an approximate 1-year rupture risk of 1%; a patient with a 5.5cm aneurysm has up to a 10% 1-year rupture risk. With the widespread use of cross-sectional imaging, many aortic aneurysms are discovered incidentally. Others are found on screening exams. The US Preventative Services Task Force recommends that men aged 65 to 75 years of age who have ever smoked tobacco undergo an abdominal ultrasound to screen for aneurysmal disease. Aortic aneurysms may also present symptomatically, with chest, abdominal and/or back pain and occasionally a palpable pulsatile abdominal mass, which is widely believed to represent an increasingly unstable aneurysm.

---

## **Etiology**

Certain risk factors, some modifiable and others non-modifiable (a.k.a. innate), predispose individuals to the development of aortic aneurysms. These include age, male gender, family history of aneurysms or aortopathies, and comorbidities including hypertension, hyperlipidemia, and peripheral vascular disease. Interestingly, female sex, African American race, and a history of diabetes have been found to be protective against the development and rupture of aortic aneurysms. A personal history of Marfan's syndrome, Loeys Dietz syndrome, or Ehlers Danlos Type IV (a.k.a. vascular Ehlers-Danlos or vEDS) is associated with a particularly elevated risk of aneurysmal degeneration. Medical providers should counsel and assist their patients to mitigate all potentially modifiable risk factors.

---

## **Diagnostics and Imaging**

When evaluating a patient with a suspected aortic aneurysm, a thorough history, physical examination, and imaging will help to narrow the differential diagnosis. Most often, patients present without symptoms; however, if symptomatic, typical complaints include chest, abdominal or back pain. Physical exam findings may not be overt but vital signs may provide a first clue in a symptomatic patient as heart rate and blood pressure might deviate from normal ranges. Patients with thoracic aortic aneurysms (TAA) often have no other easily identifiable physical exam findings aside from vital sign derangements in those with symptomatic or ruptured aneurysms. In thin patients with suspected AAA, the abdominal exam may demonstrate a pulsatile mass at or adjacent to the midline.

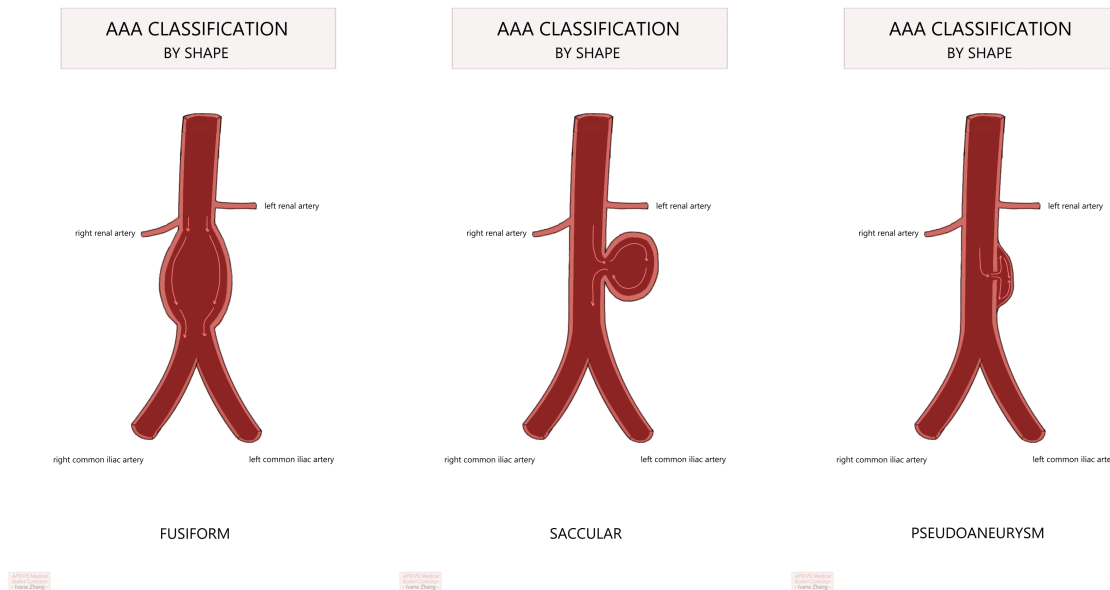


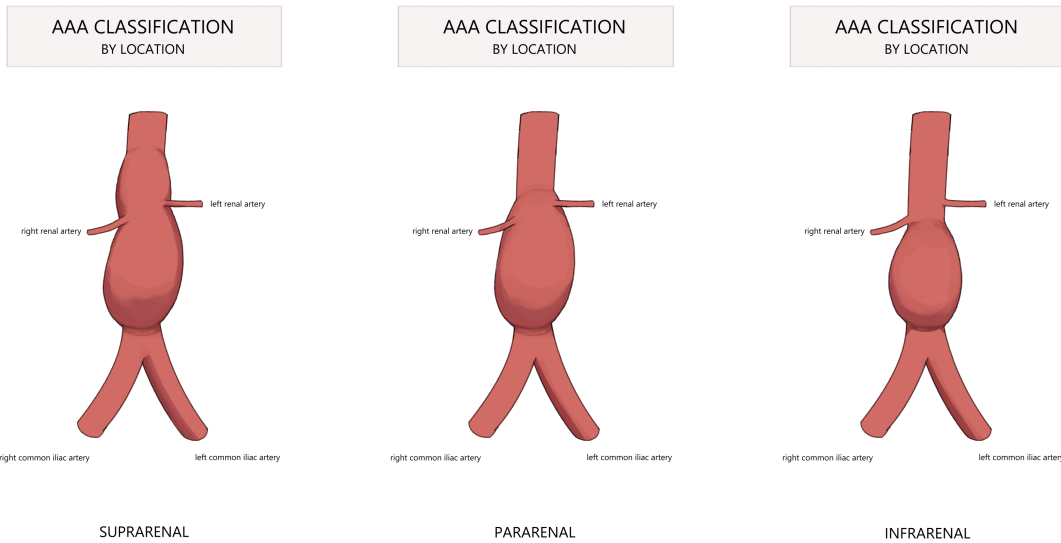
Figure 2: Aneurysm Topography

Although the history and physical exam are indispensable to patient evaluation, the diagnosis of an aneurysm is usually secured with imaging. The mainstay of imaging is computed tomography (CT), while ultrasound, magnetic resonance imaging (MRI), and angiography are useful modalities in non-urgent or non-emergent settings. The benefits of CT, particularly the richness of anatomic data obtained, must be weighed against the potential risks related to contrast administration and exposure to radiation. Since the 2019 statement from the U.S. Preventive Services Task Force regarding one-time screening with ultrasonography in men aged 65 to 75 years who have ever smoked, the use of ultrasound has a more prominent role in AAA screening. Equipped with history, exam, and imaging data, the diagnosis of an aortic aneurysm can usually be made.

When looking at an imaging study, abdominal aortic aneurysms are labeled in regard to their location relative to the renal arteries:

- Aortic aneurysms are termed “**suprarenal**” when their most proximal extent is above the level of the renal arteries. Suprarenal abdominal aortic aneurysms typically involve visceral branches of the aorta as well (i.e. the SMA or celiac trunk) and thus may also be classified as a Crawford type IV thoracoabdominal aortic aneurysm (TAAA)
- Aneurysms are labeled “**pararenal**” when the proximal extent of the aneurysm is at the level of the renal arteries and includes the origins of the renal arteries.

- Abdominal aortic aneurysms are termed “**juxtarenal**” when their most proximal extent is immediately below the take-off of the renal arteries.
- Lastly, aneurysms are labeled “**infrarenal**” when they begin below the renal arteries, often with a neck of non-dilated aorta between the lowest renal artery and the top of the aneurysm. As one might expect, the location of the aneurysm has implications for treatment options.



AAA anatomy description relative to the renal arteries.

**Tip**

Please see Figure 2 of [this American Heart Association publication](#) depicting the anatomic classifications of AAAs.

## Treatment

Once a diagnosis has been obtained, considerations for management follow. Similar to a thoracic aortic aneurysm (TAA), indications for repair are reflective of risk for rupture. The aim is to intervene when the rupture risk exceeds the risks posed by surgery. Intervention

is considered when an aneurysm reaches the size threshold of 5.5cm in males and 5cm in females or if the growth rate exceeds 5 mm in 6 months. Again, this threshold is modified in patients with a strong family history of aneurysmal disease or collagen vascular disorder, or when patients present symptomatically or with a ruptured aorta. The topography of the aneurysm must also be considered as a saccular aneurysm, an isolated pouch off the sidewall of the aorta, presents a much greater risk of rupture and should be fixed when identified.

On first evaluation, patients with AAAs should be medically optimized to minimize their perioperative risk of morbidity and mortality. Multiple factors must be considered when deciding between an open surgical approach and an endovascular one. While the prevalence of endovascular aortic repair (EVAR) has exponentially increased in the past two decades relative to open surgical repair (OSR), each aneurysm poses a unique set of potential constraints that suggest a preference for one type of repair over another.

Please see the Section section and watch the included videos.

## **Endovascular Aortic Repair (EVAR)**

Endovascular aortic repair (EVAR) is a novel approach to AAA treatment that has gained widespread popularity since its first successful use by Dr. Juan Parodi and Dr. Julio Palmaz in 1991. EVAR necessitates cross-sectional imaging to measure the AAA dimensions and determine the appropriate size of an aortic stent graft. Stent grafts are straight or bifurcated tubes with a wire framework covered by non-porous fabric. Simply, it is a tube that can be deployed from inside a vessel.

### **EVAR Procedural Steps**

A step-by-step guide of a typical, uncomplicated EVAR for an infrarenal AAA is listed below. Please see the Section section to a video guide of a typical EVAR.

1. Bilateral femoral access via percutaneous or open surgical exposure (a.k.a. “femoral cut-downs”)
2. Heparinization to achieve an activated clotting time (ACT) greater than 250 seconds.
3. Placement of stiff wires into the abdominal aorta and upsizing of sheaths into the access vessels.
4. Aortogram to visualize renal arteries and deployment of the endograft main body under the origins of the renal arteries.
5. Cannulation of contralateral limb gate using a wire and catheter with subsequent confirmation that both the wire and catheter are within the main body.

6. Aortoiliac angiogram to measure the distance from the contralateral gate to the contralateral hypogastric (i.e. internal iliac) artery.
7. Deployment of contralateral limb with the distal landing site just proximal to the hypogastric artery.
8. Partial deployment of the ipsilateral limb.
9. Aortoiliac angiogram to measure the distance from the ipsilateral gate of the main body to the ipsilateral hypogastric artery.
10. Deployment of the remainder of the ipsilateral limb.
11. Ballooning of the proximal and distal landing zones, and areas of stent overlap.
12. Remove wires and sheaths and close arteriotomies (often with closure devices).
13. Reverse heparin with protamine.
14. Apply pressure to surgical sites and monitor for hematoma formation.

### **EVAR Considerations and Planning**

The need for imaging can be a limiting factor such as when patients with a ruptured aneurysm are too hemodynamically unstable to stop at the CT scanner prior to repair or when a patient's kidney function is too impaired to tolerate a large contrast bolus.

If the situation is amenable to getting a CT angiogram to measure anatomic dimensions, several components of the aneurysm anatomy should be assessed to determine anatomic suitability for EVAR. These include the length, width, angulation of the aneurysm, and presence of disease at the aneurysm neck (i.e. the aortic segment between the lowest renal artery and the proximal extent of the aortic dilatation).

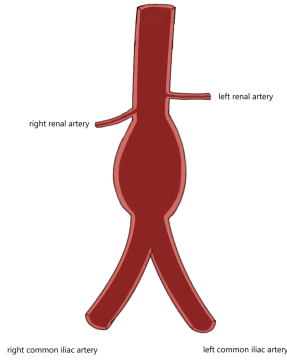
#### Tip

- See page 71 of the Gore Vascular/Endovascular Surgery Combat Manual in the additional resources ([?@sec-additionalresources](#)) for a **high-yield** EVAR planning and sizing considerations chart.
- Video Tutorial: Endovascular Aortic Repair Preoperative Sizing with Dr. Sharif Ellozy. *This is above the level of medical students*, but for those who are interested, watch it [here](#).

The access vessels, including the bilateral iliac and femoral arteries, must be healthy enough to allow sheaths, wires and catheters to pass through them. Calcification due to atherosclerosis can make arterial access difficult, and if vessels are so diseased as to be completely occluded, wires and devices may not be able to travel past the occlusion. Additionally, the iliac arteries must be within the appropriate diameter and length dimensions to accommodate the



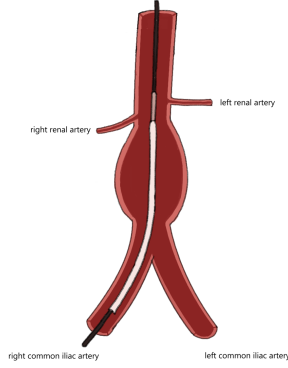
ENDOVASCULAR ANEURYSM REPAIR (EVAR)



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Figure 3: Fusiform infrarenal AAA

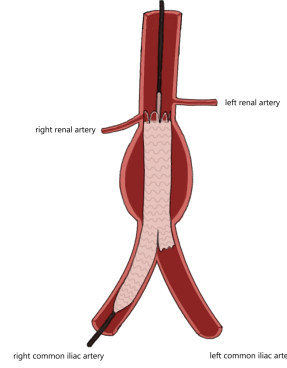
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Figure 4: Step 4.1

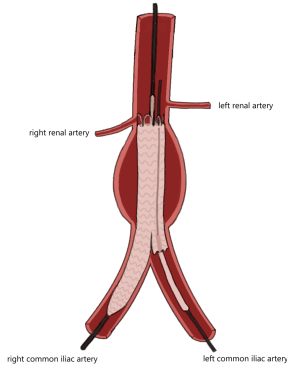
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Figure 5: Step 4.2  
EVAR Procedure

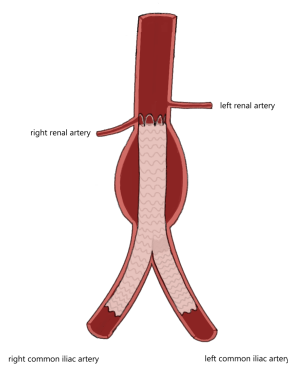
ENDOVASCULAR ANEURYSM REPAIR (EVAR)



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Figure 6: Step 5

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Figure 7: Step 7

chosen stent graft. Lastly, certain anatomic anomalies such as a horseshoe kidney must be evaluated to ensure the appropriateness of EVAR for repair. With recent innovations in endovascular devices and intra-operative imaging, many factors that were once contraindications to EVAR have now become considerations when planning for EVAR. This is to say, as technology advances, EVAR is increasingly possible and safe for patients with difficult anatomy and co-morbidities.

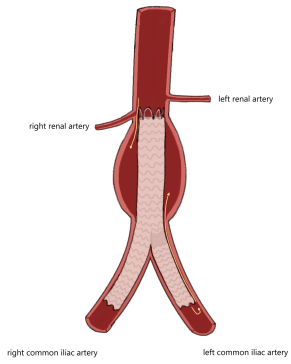
## **EVAR Complications**

Although a more recent development relative to open surgical repair (OSR), EVAR is not without risk of complications. EVAR may be complicated by injury to access vessels such as femoral occlusion or iliac avulsion (aka, “iliac-on-a-stick”), wire trauma from the relatively stiff wires that are used to deliver the stent graft, rupture of the aneurysm, and endoleak. Endoleak is the term used to describe ongoing blood flow in the aneurysm sac after stent graft placement (e.g. from lumbar arteries or the inferior mesenteric artery, inadequate proximal or distal endograft seal, a defect in the endograft, etc.). There are various types of endoleak and each has its own management approach.

Endoleaks are classified as follows:

- Type Ia: caused by an improper **proximal** endograft seal (i.e. the top of the graft is not well opposed to the abdominal aorta and blood can flow around it into the aneurysm sac).
- Type Ib: caused by an improper **distal** endograft seal (i.e. the distal iliac limb is not well opposed to the iliac artery and blood can flow retrograde into the aneurysm sac).
- Type II: caused by retrograde blood flow into the aneurysm sac from aortic side branches such as lumbar arteries or the IMA. This is the most common type and does not require intervention provided a stable aneurysm sac size is observed on follow-up. If the sac continues to expand, the feeding vessels may need to be occluded via embolization (e.g. coil embolization).
- Type III: caused by a defect in the endograft such as a fabric tear, or disconnection of the contralateral iliac limb from the main body of the graft.
- Type IV: caused by graft wall porosity. To explain graft wall porosity, imagine filling up a pillowcase with water. Even if the pillowcase has the tightest of weaves, eventually, water will leak *through* the pillowcase.
- Type V: increase in aneurysm size with no identifiable cause.

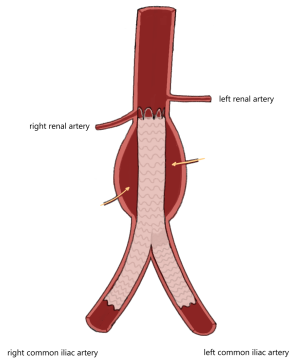
ENDOLEAK CLASSIFICATIONS



**TYPE I.**  
Proximal (top) or distal (bottom) graft attachment site leak.

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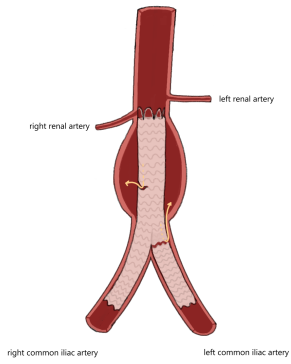
ENDOLEAK CLASSIFICATIONS



**TYPE II.**  
Retrograde flow into the aneurysm sac from aortic side-branches (e.g. lumbar or inferior mesenteric arteries).

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ENDOLEAK CLASSIFICATIONS

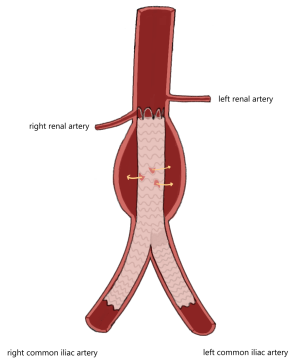


**TYPE III.**  
Graft defect from fabric tear or modular overlap disconnect.

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Endoleak classifications.

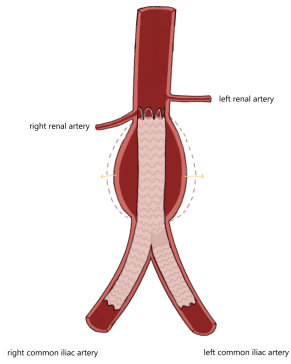
ENDOLEAK CLASSIFICATIONS



**TYPE IV.**  
Graft wall porosity.

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ENDOLEAK CLASSIFICATIONS



**TYPE V.**  
"Endotension." Unknown increase in aneurysm diameter with no identifiable leak.

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### Tip

Please click [here](#) to open an article by Dr. Tamer W. Kassem with a simple image depicting the endoleak classifications.

Additionally, one should monitor for abdominal compartment syndrome after repair of AAA rupture, as much of the large volume resuscitation can leak from the intravascular compartment into the interstitium of the abdominal tissues causing distention. In the event of rupture, the retroperitoneal hematoma can cause a mass effect and increase of abdominal pressures. Lastly, a well-known complication of EVAR is ischemic colitis, although it is only encountered in less than 3% of endovascular aortic repairs. This occurs when the colon becomes ischemic secondary to vascular hypoperfusion caused by a variety of quoted sources including atheroembolization and systemic hypotension. Albeit rare, ischemic colitis has a significant associated mortality risk and should be addressed promptly with bowel rest, antibiotic coverage, a flexible sigmoidoscopy, and potentially further intervention, if warranted. This complication is not limited to EVAR and has been identified postoperatively with the same incidence in OSR.

## **Open Surgical Repair (OSR)**

Open surgical repair (OSR) remains the traditional means of repairing AAA and is still the most appropriate modality when EVAR is not feasible for anatomic reasons. In the elective setting, the ideal patient is physiologically fit enough for OSR as it is a procedure with the potential for significant morbidity and a lengthy recovery.

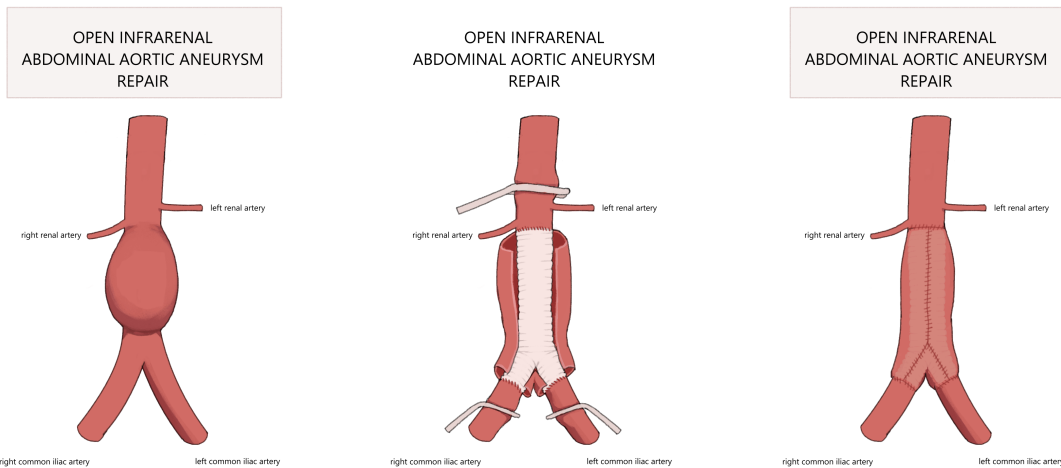
### **OSR Considerations and Planning**

OSR can be conducted via a transperitoneal or a retroperitoneal exposure. Other decision points for open repair include the following:

- **Location of the proximal clamp:** A clamp must be placed on the aorta in a location free of significant disease, with enough clearance below it to allow space for suturing the proximal anastomosis.
- **Configuration of the proximal anastomosis:** Anastomoses performed for aneurysmal disease are sewn in an end-to-end fashion, whereas aortas being reconstructed for occlusive disease can be performed either end-to-end or end-to-side.
- **Location of the distal anastomosis:** the end of the graft can be sewn to either the distal aorta, iliac arteries, or femoral arteries depending on the extent of the native vessel that needs to be excluded and the distal location where the vessel is healthy enough to clamp and receive sutures (e.g. is not heavily calcified).

- Management of the inferior mesenteric artery when an end-to-end anastomosis is created: The IMA can be ligated if collateral flow is adequate or reimplemented into the graft if collateral flow is inadequate.
- Management of aberrant intraperitoneal anatomy and pathology: Examples include adhesions or scarred tissue due to previous abdominal surgery, accessory renal arteries, horseshoe kidney, history of kidney transplant, and previous aortic replacement, among many other possibilities.

Depending on the urgency of repair, many of these considerations might only be entertained intraoperatively.



Open Aortic Repair (OAR)  
with suprarenal clamp.

## OSR Complications

Complications related to OSR differ from those of EVAR, owing greatly to the open abdominal exposure and the lack of intraluminal wire manipulation. Just as with most other laparotomies or open retroperitoneal dissections, incisional pain can be significant and cause atelectasis or other respiratory compromise. Additionally, ileus and wound infections can impair the return of proper bowel function. Similar to EVAR, ischemic colitis can manifest after OSR, particularly in the setting of rupture.

## Outcomes

Much experience has been gained since the first EVAR in 1990 and ample research has been dedicated to the comparison of EVAR to OSR. Multiple trials have demonstrated an early survival advantage for EVAR over OSR that tends to diminish over time. Certainly, the perioperative period for EVAR is notable for reduced morbidity and mortality relative to OSR with length of hospital stay significantly longer in those undergoing OSR. However, this early benefit is often balanced by a necessity for life-long follow-up including imaging at regular intervals and a tendency for re-interventions during the surveillance period (e.g. to manage endoleaks). Hence, the risk-benefit ratio must be weighed individually for each patient.

Contemporary management of abdominal aortic aneurysms is most often undertaken with EVAR. Endovascular repair has a third of the peri-operative all-cause mortality attributed to open repair (1.6% vs 4.8%, respectively). This survival benefit tends to dissipate by three to four years post-operatively. Moreover, the overall complication rate of EVAR in the perioperative period is up to 10%.

Among patients who undergo open abdominal aortic aneurysm repair, mortality in the immediate post-operative period can be as high as 5%. Of patients who survive, later death is typically related to cardiovascular disease burden. Complications arise in 9% of patients and most often include myocardial infarction, respiratory insufficiency, pneumonia, acute kidney injury, and ischemic colitis. Late Infection of the graft is possible, is challenging to manage, and occurs in less than 1% of patients.

---

## Surveillance

The Society for Vascular Surgery guidelines suggest that for patients found to have an abdominal aortic aneurysm, surveillance imaging should occur at intervals specific to the maximum diameter of their aneurysm.

- For patients with abdominal aortic aneurysms between 3.0 and 3.9cm in diameter, CT or ultrasound should be undertaken every 3 years.
- Patients with abdominal aortic aneurysms between 4.0 and 4.9cm in diameter, imaging is indicated every 12 months.
- When the aneurysm reaches a maximum diameter of 5.0 cm, imaging with CT is recommended at intervals of every 6 months.

After the abdominal aortic aneurysm has been repaired, ongoing surveillance is warranted to monitor for post-repair complications and the need for reintervention. As mentioned, endovascular repairs can develop endoleaks over time, which may necessitate another intervention if

the aneurysm sac is expanding. Therefore, patients who have undergone EVAR should undergo CTA at one month and 12 months post-operatively. A six-month scan is indicated in the presence of a persistent endoleak observed on the one-month scan. If the endograft is in a good position without complications, yearly interval follow-up with concomitant imaging is warranted. Open repairs can similarly develop problems that require repair, albeit with less frequency, and hence need surveillance imaging every five years.

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## Teaching Case

### Scenario

A 73-year-old male with a history of atrial fibrillation, diabetes, hypertension, ongoing tobacco use, hyperlipidemia, chronic kidney disease, and peripheral arterial disease undergoes a screening ultrasound of the abdominal aorta. The ultrasound shows a 5.6cm infrarenal fusiform aortic aneurysm extending into the proximal common iliac arteries. His primary care physician refers him to the vascular surgery clinic, where he presents without subjective complaints. CTA is recommended to assess the patient's anatomy.

### Exam

**HEENT:** No carotid bruits.

**Cardiac:** Irregularly irregular, no murmurs.

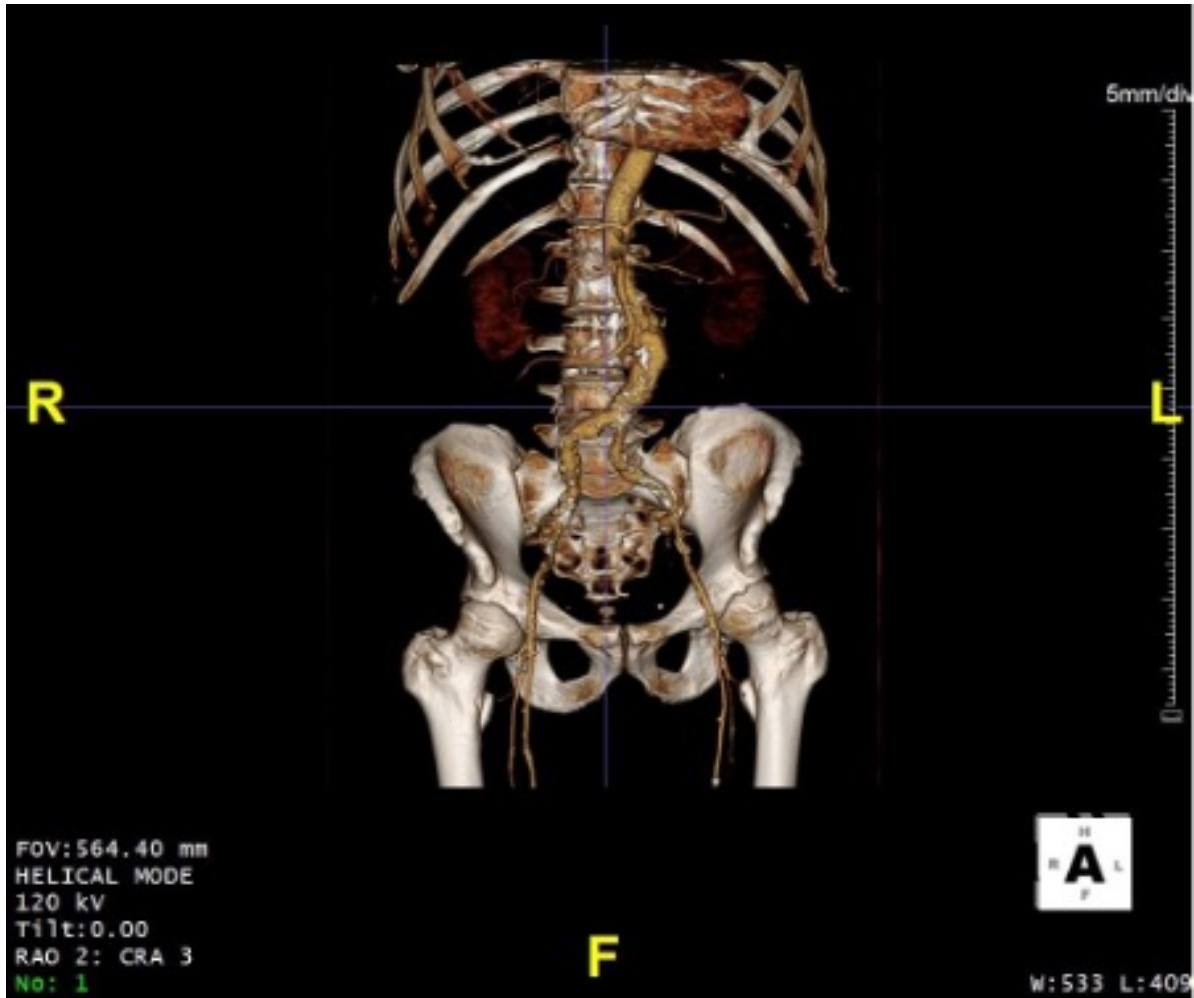
**Pulmonary:** Clear to auscultation in all lung fields.

**Abdominal:** Obese, soft, non-tender, no pulsatile masses felt.

**Vascular:** Palpable femoral pulses, multiphasic pedal signals bilaterally.

### Imaging

**CTA Abdo/Pelvis**



**CTA Report:** 5.6cm infrarenal bilobed aortic aneurysm with bilateral accessory renal arteries inferior to main renal arteries. 14mm infrarenal neck cranial to aneurysm. Widely patent iliac systems with non-diseased femoral arteries.

### Discussion Points

**i** N.B. There is no prepared answers for the questions below.

However, we feel this chapter contains all the necessary information to answer the questions. If not, please let us know!

1. What recommendations for medical management should be made to optimize this patient prior to surgical intervention?



2. What is the important information to ascertain from history, physical exam and imaging that can help you determine the best modality of treatment.
3. With what urgency should the patient be scheduled for operative repair?
4. What kind of repair would you suggest for this patient and why? Open or endovascular?
5. What complications are possible after an endovascular repair of the abdominal aorta? After an open repair?
6. Following repair, how should the patient be surveilled? With what frequency?
7. If the patient's aneurysm was 4.6cm in greatest transverse diameter, how often should he undergo imaging and follow-up?
8. What aortopathies and collagen vascular diseases would convince you to repair the aneurysm at a size less than 5.5cm?

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## Key Articles

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6. Darling, R. [Master Techniques in Surgery; Vascular Surgery: Arterial](#). 1st ed. Lippincott Williams & Wilkins, 2016. Print.(Darling and Ozaki 2016)
7. Qrareya M, Zuhaili B. [Management of Postoperative Complications Following Endovascular Aortic Aneurysm Repair](#). Surg Clin North Am. 2021 Oct;101(5):785-798. doi: 10.1016/j.suc.2021.05.020. Epub 2021 Jul 30. PMID: 34537143.(Qrareya and Zuhaili 2021)
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 Tip

Dr. Schanzer’s recent publication in the [New England Journal of Medicine \(NEJM\)](#) is particularly high yield. However, as a NEJM publication, the article is behind a paywall. We hope you are able to access this paper via your institution’s library.

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## Additional Resources

### Audible Bleeding Content

- Audible Bleeding Exam Prep: [AAA Chapter](#)
- Audible Bleeding has an episode covering the IMPROVE trial. Listen to it below and find [additional information here](#), or find the episode wherever you listen to podcasts.
- Holding Pressure Case Prep: EVAR. Listen to it [here](#), or find the episode wherever you listen to podcasts. **Be sure to take a quick look at the shownotes.**
- VSITE Review - AAA. Listen to it below and find [additional information here](#), or find the episode wherever you listen to podcasts.
- Video Tutorial: Dr. Kristina Giles - “Decision-Making and Treatment: Elective Infrarenal and Juxtarenal AAA.” Watch it [here](#).

- Video Tutorial: Open AAA repair: How I Do It with Dr. Ashlee Vinyard and Dr. John Eidt. Watch it [here](#).
  - Vascular Origin Stories: Bridging the Gap - The Fabric of Aortic Repair. Listen to it below and find [additional information here](#), or find the episode wherever you listen to podcasts.
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## Websites

- TeachMe Surgery: [Abdominal Aortic Aneurysm](#)
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## Gore Combat Manual

The [Gore Medical Vascular and Endovascular Surgery Combat Manual](#) is an informative and entertaining read intended as a vascular surgery crash course for medical students, residents, and fellows alike. Highly accessible with a thoughtfully determined level of detail, but lacking in learning activities (e.g. questions, videos, etc.), this resource is a wonderful complement to the APDVS eBook.

### Tip

Please see pages 63-75.

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## Operative Footage

Developed by the DeBakey Institute for Cardiovascular Education & Training at Houston Methodist. *YouTube account required as video content is age-restricted.* Please create and/or log in to your YouTube account to have access to the videos.

**Endovascular Aortic Repair (EVAR)**

**Open Surgical Repair (OSR) of a AAA, Transperitoneal Access**

**EVAR Explantation with Aortic Reconstruction**

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# **Lower Extremity Arterial Disease**

# Claudication

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## Note

By the end of this chapter, students will:

- Explain the definition, etiology, and pathophysiology of peripheral arterial disease and its relation to claudication.
- Identify the risk factors associated with claudication.
- Recognize the clinical manifestations and symptoms of claudication.
- Summarize the underlying vascular anatomy and physiology relevant to claudication.
- Outline the diagnostic approach and tests used to evaluate claudication.
- Summarize evidence-based outcomes of the management of claudication.

## Key Facts

1. Peripheral arterial disease (PAD), also known as peripheral vascular disease (PVD), is a condition characterized by atherosclerosis or narrowing of the arteries that supply blood to the extremities.
2. PAD affects approximately 202 million individuals worldwide.
3. Major risk factors for PAD include smoking, diabetes, and advanced age.
4. Claudication is the mildest symptomatic manifestation of peripheral arterial disease.
5. Claudication is defined as **reproducible** discomfort in a specific muscle group, typically in the lower extremities, that is **induced by exercise and relieved by rest**.
6. Patients with claudication have an overall 5-year mortality risk of 10-15%.
7. Approximately 2-3% of claudicants (i.e. patients experiencing claudication) will develop limb-threatening ischemia.

8. Claudication is managed conservatively with lifestyle modifications including exercise regimen, dietary changes, smoking cessation, and medical optimization to control hypertension, hyperlipidemia, and or diabetes.

#### Claudication Slide Deck

Please find the slide deck corresponding to this eBook chapter [here](#).

#### 💡 How We Suggest Using the Pre/Post Questions

The pre/post questions are listed below. They are all multiple choice questions with a single right answer. To best guide your learning, we have hidden the answers in a collapsible menu. Before reading the chapter, we suggest giving the questions a try, noting your answers on a notepad. After reading the chapter, return to the questions, re-evaluate your answers, and then open the collapsible menu to read the correct answer and discussion. Do not fret if you have difficulty answering the questions before reading the chapter! By the end of the chapter, we are certain you will have covered the knowledge necessary to answer the questions. There will be a teaching case at the end of the chapter. This is another opportunity to exercise your new knowledge!

## Pre/Post Questions

### Case Based Questions

1. A 70-year-old male, who has a history of heavy smoking (100 pack years), presents to the clinic for claudication-like symptoms. *What level of infrainguinal arterial occlusive disease is he most at risk for developing based on this history?*
  - A. Superficial femoral artery disease.
  - B. Deep femoral artery disease.
  - C. Anterior tibial artery disease.
  - D. Popliteal artery disease.
  - E. Posterior tibial artery disease.

#### Answer

#### **A. Superficial femoral artery disease.**

Discussion: The prevalence of peripheral artery disease in the general population is be-

tween 3-10%, and 15-20% in those over the age of 70. Risk factors for peripheral arterial disease (PAD) include age, male gender, hypertension, diabetes, smoking, dyslipidemia, family history and homocysteinemia. Heavy smokers have a propensity to develop superficial femoral artery disease (A). Patients with a history of diabetes have a tendency to develop infrapopliteal disease (C, E). Patients of nonwhite ethnicity and those with a history of chronic renal insufficiency are also at increased risk.

2. A 67-year-old female is being evaluated in the outpatient clinic for lower extremity pain associated with walking. Using a manual blood pressure cuff and hand-held Doppler, the following measurements are obtained:

Artery	Pressure (mm Hg)	
	Right	Left
Brachial	150	120
Dorsalis pedis (DP)	165	120
Posterior tibial (PT)	150	108

*What are the ankle-brachial indices (ABIs) of her lower extremities?*

- A. Right ABI 1.1; left ABI 0.8
- B. Right ABI 1.0; left ABI 1.0
- C. Right ABI 1.2; left ABI 0.9
- D. Right ABI 1.4; left ABI 1.0
- E. Right ABI 1.1; left ABI 0.7

Answer

**A. Right ABI 1.1; left ABI 0.8**

Discussion: Performing an ankle-brachial index (ABI) requires an appropriately sized manual blood pressure cuff and a hand-held, continuous-wave Doppler probe. The patient should be in the supine position and allowed to rest before the examination begins. The blood pressure cuff is placed above the ankle. The Doppler probe is used to locate the dorsalis pedis (DP) or posterior tibial (PT) pulse. The pressure cuff is inflated until the Doppler signal is obliterated, and then the cuff is then slowly released. The pressure at which the Doppler signal returns is the ankle pressure. The same process is repeated for both legs (DP and PT signals) and both arms. The arm pressure must be measured with the Doppler probe and the manual blood pressure cuff. To calculate an ABI, the higher ankle pressure (DP or PT) is divided by the highest arm pressure (left or right). Note that the higher arm pressure is used to calculate the ABI for both legs. For this specific



case, the highest arm pressure is 150. To calculate on the right,  $165/150=1.1$ , on the left  $120/150=0.8$ . A normal ABI is 0.9 to 1.2. Patients with claudication usually have an ABI ranging from 0.5 to 0.9, and those with ischemic rest pain or tissue loss usually have an ABI less than 0.5. *Sabiston Textbook of Surgery: The Biological Basis of Modern Surgical Practice, 20th ed. Ch. 62: Peripheral Arterial Disease. Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FGR. Inter-society consensus for the management of peripheral arterial disease (TASC II). J Vasc Surg. 2007;45:S5-S67.*

3. A 70-year-old male with poorly controlled diabetes and no smoking history presents to the clinic for an ischemic toe ulcer. *What level of infrainguinal arterial occlusive disease is he most at risk for developing based on this history?*
- A. Superficial femoral artery disease
  - B. Deep femoral artery disease
  - C. Iliac artery disease
  - D. Aortic disease
  - E. Tibial artery disease

Answer

**E. Tibial artery disease.**

Discussion: Risk factors for peripheral arterial disease include age, male gender, hypertension, diabetes, smoking, dyslipidemia, family history, and homocysteinemia. Individuals who are heavy smokers tend to develop superficial femoral artery disease, whereas those with diabetes and renal failure tend to develop infrapopliteal disease. Of the answers listed, only the tibial artery is infrapopliteal.

**The following prompt will be used for questions 4, 5, and 6.**

A 65-year old male with a history of hypertension, hyperlipidemia, and tobacco abuse presents to the vascular surgery clinic with complaints of pain in his legs. Patient states that the pain is crampy in quality, mostly in his calf muscles, it starts after he walks 4 blocks, and is worse on the right as compared to the left. He notices that when he stops walking, the pain resolves. This pain is keeping him from being able to participate in social events and spend quality time with his family.

4. *What is the name of the symptom the patient is experiencing?*
- A. Neuropathy
  - B. Claudication
  - C. Ischemic rest pain

- D. Bilateral sciatica
- E. Normal symptoms

Answer

**B. Claudication**

Discussion: Claudication is defined as reproducible pain or cramping of the lower extremities that occurs with ambulation and significantly improves or resolves at rest. Claudication can occur in any muscle group distal to the area of arterial stenosis and/or occlusion. As such, the calf muscles are the most common anatomic location to experience pain as they are distal in the lower extremity. As the patient does not report pain at rest, they do not have ischemic rest pain. Neuropathic pain or bilateral sciatica and claudication can be confused. Neuropathic pain is shocking in quality whereas claudication is describes as a cramp-type pain. Furthermore, claudication is absent at rest, triggered by exercise, and relieved by a return to rest. This pattern helps distinguish claudication from neuropathic pain. This patient also does not have normal pathology, as one should not have pain on ambulation and needs to be further evaluated.

5. *What would be the first step in confirming the diagnosis for this patient?*
- A. CT Angiography (CTA)
  - B. Doppler probe
  - C. Duplex Ultrasound
  - D. Angiogram
  - E. Ankle-Brachial Index and Pulse Volume Recording (ABI/PVR)

Answer

**E. ABI/PVR**

Discussion: Ankle Brachial Index (ABI) is a non-invasive method that measures the ratio of blood pressure in the lower legs to the blood pressure in the arms. Abnormal ABIs have been shown to have high specificity with identifying PAD. Patients who have claudication usually have ABI ranges from 0.5 to 0.9. Pulse volume recordings (PVR) use pressure cuffs to record the shape of a pulse. The type of waveform provides important information about the characteristics of blood flow. Waveforms can be used to diagnose and monitor arterial diseases, assess the severity of stenosis, and determine the effectiveness of treatment interventions. This patient likely has claudication based on the history and symptoms. The first non-invasive testing to confirm the diagnosis would be ABI/PVR. While a CTA would provide helpful information with regards to arterial anatomy and the extend of calcification, it cannot provide the physiologic information that one would ob-

tains an ABI/PVR. Furthermore, CTA requires contrast and radiation exposure. While used in measuring an ABI, a Doppler probe is not nearly as sensitive in identifying PAD and does not provide data helpful in creating a management plan. A Duplex Ultrasound would provide physiologic information as to the burden of disease. However, it is highly operator dependent and requires more resources. It may be considered as a second line investigation. While an angiogram is the gold-standard, it is an invasive technique that requires the use of contrast. As such, it is not a first line investigation.

6. *What would be the first step in treating this patient's symptoms?*

- A. Angiogram to identify targets for open surgical bypass.
- B. Angiogram with angioplasty/stent of any occluded vessels.
- C. Prescribe Cilostazol.
- D. Ensure medication optimization for his hypertension and hyperlipidemia, smoking cessation counseling, and exercise regimen.
- E. No treatments at this time, continue to monitor and come back to clinic in 3 months.

Answer

**D. Ensure medication optimization for his hypertension and hyperlipidemia, smoking cessation counseling, and exercise regimen.**

Discussion: The mainstay of treatment for claudication is conservative management, including medical optimization and lifestyle modification. The patient has hypertension and hyperlipidemia. He should be on appropriate antihypertensive to control the blood pressure. With regard to medical optimization, it is recommended that patients start on antiplatelet therapy (Aspirin 81 mg daily) and a high dose statin. Lifestyle modifications include smoking cessation, exercise training to develop collateral vessels, and diet modification to lower cholesterol intake. An appropriate exercise regimen is defined by at least 30 minutes of supervised ambulation/exercise at least 3 times per week. Patients should be followed closely with yearly ABIs by both Primary Care Physicians (PCPs) and Vascular Surgeons for progression of disease. While an angiogram allows for simultaneous diagnosis and treatment of PAD, it is an invasive technique that requires the use of contrast. Angiogram may be considered if conservative management fails. Cilostazol is an antiplatelet that has been shown to have beneficial effects for patients with intermittent claudication, however, it is not a first-line medication. Lastly, while follow-up is important in the management of PAD, conservative management should be initiated as soon as the diagnosis of PAD is made.

## Introduction

### Tip

The term peripheral arterial disease (PAD) is often used interchangeably with peripheral vascular disease (PVD) when referring to arterial blockages.

**Claudication is defined as cramping, pain, and/or discomfort in the hip, thigh, or calves that occurs with ambulation/exercise, improves shortly after cessation of activity, and recurs after a predictable amount of ambulation/exercise.**

Claudication is a symptom of peripheral arterial disease (PAD). Therefore, it is important to first understand PAD before addressing claudication. PAD is a chronic condition of arterial insufficiency due to the build-up of atherosclerotic plaque (or fatty deposits) in the arteries. PAD affects 8.5 million adults in the U.S. and 202 million adults worldwide, with approximately 15% of the population aged greater than 70 years old suffering from PAD. Risk factors for PAD include increasing age, hypertension, hyperlipidemia, renal disease, diabetes, and smoking. The risk factors for claudication are the same risk factors described for PAD, including: advanced age, race (non-hispanic blacks), male gender, smoking, diabetes mellitus, dyslipidemia, hypertension, renal insufficiency, and elevated homocysteine levels (hypercoagulable state). PAD has various distributions in the body including one or a combination of aortoiliac, femoral, and infrapopliteal disease. PAD severity falls along a spectrum. Some patients may be completely asymptomatic while others have nonhealing, chronically infected wounds requiring later limb amputation. Vascular claudication is the mildest symptomatic manifestation of the PAD spectrum.

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## Etiology

Prior to understanding the etiology of claudication and peripheral arterial disease. It is important to review the anatomy of the arterial blood flow of the lower extremity.

## Anatomy

The arterial anatomy of the lower extremity is a complex network of arteries that supply blood to the legs and feet. Please find an [image here](#) and a description below of the major arteries from the common femoral artery down to the tibioperoneal trunk, anterior tibial, and dorsalis pedis arteries:

- **Common Femoral Artery (CFA):** The common femoral artery is a large, thick-walled artery located in the groin. It is a continuation of the external iliac artery and bifurcates into the deep femoral artery (profunda femoris) and the superficial femoral artery.
- **Superficial Femoral Artery (SFA):** The superficial femoral artery is the largest branch of the common femoral artery. It descends along the anterior and medial aspect of the thigh and enters the adductor canal. As it descends, it eventually becomes the popliteal artery at the level of the knee joint.
- **Profunda Femoris (Deep Femoral) Artery (PFA):** The profunda femoris artery, also known as the deep femoral artery, is a significant branch of the common femoral artery. It typically arises about 1 to 2 cm below the inguinal ligament. The profunda femoris artery primarily supplies the muscles of the posterior and deep thigh, including the quadriceps muscles. It is also an important collateral pathway for blood flow in cases of arterial obstruction or occlusion in the lower extremity distal to the SFA/PFA bifurcation.
- **Popliteal Artery:** The popliteal artery is located behind the knee joint and is a continuation of the superficial femoral artery. It provides blood supply to the muscles and structures in the popliteal fossa and gives rise to several branches, including the anterior tibial, posterior tibial, and peroneal arteries.
- **Anterior Tibial Artery (AT):** The anterior tibial artery is the first branch off of the popliteal artery. It is NOT part of the tibioperoneal trunk (only includes the PT and peroneal arteries). The AT passes from behind the knee through the interosseous membrane of the leg and descends through the anterior compartment of the leg. It can be visualized as the most lateral tibial vessel during lower extremity angiogram run-offs. The AT runs along the front of the tibia and crosses the ankle joint to become the dorsalis pedis artery, supplying blood to the dorsum (top) of the foot.
- **Posterior Tibial Artery (PT):** The posterior tibial artery is another branch of the popliteal artery. Once the AT tibial branches from the popliteal, the remaining vessel is termed the tibioperoneal trunk. This trunk splits to give off the posterior tibial artery (PT) and the peroneal artery. The PT descends posteriorly in the leg and passes behind the medial malleolus (the bony prominence on the inner side of the ankle). It can be palpated on the medial aspect of the foot behind the medial malleolus. It continues into the sole of the foot and forms the plantar arch, contributing to the arterial supply of the sole of the foot and toes.
- **Peroneal Artery:** The peroneal artery, also known as the fibular artery, is the third major branch of the popliteal artery. As mentioned, it originated from tibioperoneal trunk along with the PT. The peroneal artery runs along the lateral side of the leg and supplies the lateral compartment muscles. In the ankle region, it anastomoses with the branches of the posterior tibial artery, providing collateral circulation.
- **Dorsalis Pedis Artery (DP):** The dorsalis pedis artery is a continuation of the anterior tibial artery. It extends along the anterior aspect of the ankle and runs atop the dorsum (top) of the foot. It can be palpated on the dorsum of the foot just lateral to the tendon of extensor hallucis longus. It plays a crucial role in supplying blood to the dorsal surface of the foot and the toes. The presence and palpability of the dorsalis pedis pulse are

often evaluated as part of a clinical assessment to determine arterial perfusion in the foot.

In summary, the common femoral artery is the arterial origin of the leg. It gives rise to the superficial femoral artery, and subsequently, the popliteal artery. The popliteal artery then divides into the anterior tibial, posterior tibial, and peroneal arteries, each supplying specific regions of the leg and foot. Understanding this anatomy is crucial for diagnosing and treating arterial diseases and vascular disorders in the lower extremities.

#### Tip

For a short, comprehensive, although advanced video lecture reviewing normal and diseased lower limb arterial anatomy, please see the video below.

#### Note

[Here](#) is a review of the lower extremity vascular anatomy and here are two sample diagnostic angiograms of these vessels ([Sample 1](#), [Sample 2](#)).

## Process of Atherosclerosis

As previously mentioned, atherosclerosis is a fundamental process that underlies the development of peripheral vascular disease in the lower extremities. The pathological process is precisely the same as with carotid disease leading to increasing stroke risk and coronary artery disease leading to increased risk of cardiac events.

Atherosclerosis is a progressive, inflammatory condition characterized by the accumulation of fatty deposits, cholesterol, inflammatory cells, and fibrous tissue within the inner layers of arteries, forming plaques. The process typically begins with damage or dysfunction of the endothelial lining of the arterial walls. This can be caused by risk factors such as hypertension, smoking, or high cholesterol levels. In response to endothelial injury, low-density lipoproteins (LDL) or “bad” cholesterol molecules penetrate the arterial wall and become oxidized. These oxidized lipids trigger an inflammatory response. The body’s immune system responds to the inflammatory signals by sending white blood cells (macrophages) to the site. These cells engulf the oxidized lipids and form foam cells within the arterial wall. The inflammation leads to the proliferation of smooth muscle cells. Over time, the combination of lipids, immune cells, and smooth muscle cell proliferation results in the formation of atherosclerotic plaques. These plaques narrow the arterial lumen, restricting blood flow. The build-up of plaques can lead to stiffening and loss of elasticity in the arterial walls, further diminishing the artery’s ability to dilate and maintain adequate blood flow.

As the arterial narrowing progresses, blood flow to the downstream tissues (e.g., muscles, nerves, and skin in the lower extremities) becomes compromised. This can lead to ischemia,

which manifests as claudication – a symptom characterized by intermittent pain, cramping, and fatigue in the muscles during physical activity that resolves once at rest. In severe cases, atherosclerosis can lead to critical limb threatening ischemia (CLTI), a condition marked by severe arterial blockages and tissue damage. CLTI can result in non-healing ulcers, gangrene, and, in the absence of intervention, limb amputation. Atherosclerotic plaques are also prone to rupture or ulceration, exposing their contents to the bloodstream. This can lead to the formation of blood clots (thrombosis) or embolization of plaque fragments to smaller arteries, causing acute blockages that may lead to acute limb ischemia (ALI). CLTI and ALI will be discussed in more detail in the subsequent PAD chapters.

**i** Note

The following lipid levels are considered to be “good” in healthy people:

- Total cholesterol: Levels below 200 mg/dL (5.2 mmol/L)
- LDL cholesterol: Levels below 130 mg/dL (3.4 mmol/L)
- HDL cholesterol: Levels above 40 mg/dL (1 mmol/L) in men and above 50 mg/dL (1.3 mmol/L) in women

The [American Diabetes Association \(ADA\) standards of care for diabetes](#) recommend that statin therapy should be initiated in individuals with diabetes and other atherosclerotic cardiovascular disease (ASCVD) risk factors.

Please see the [ASCVD risk calculator algorithm](#):

- For patients ages 40 years and older with diabetes: Use moderate-dose statin therapy.
- For patients ages 40 to 75 with diabetes and a higher cardiovascular risk (including those with one or more ASCVD risk factors): Use high-intensity statin therapy to reduce LDL cholesterol by 50% of baseline and target an LDL cholesterol goal of <70 mg/dL.
- For patients with diabetes who have already had an ASCVD event: High-intensity statin therapy is recommended to target an LDL cholesterol reduction of 50% from baseline and an LDL cholesterol goal of <55 mg/dL.

## PAD Risk Factors

All these risk factors increase rates of atherosclerosis and start to limit the blood flow to the lower extremities, attributing to claudication.

- **Smoking:** Smoking is a well-established major risk factor for PAD. It contributes to PAD through multiple mechanisms, including endothelial dysfunction, oxidative stress, inflammation, and platelet activation. The nicotine in tobacco constricts blood vessels

and promotes the formation of atherosclerotic plaques. Moreover, smoking cessation is the most effective intervention in reducing the progression of PAD.

- **Diabetes Mellitus:** Diabetes is closely associated with PAD due to hyperglycemia-induced endothelial dysfunction and microvascular damage. It accelerates atherosclerosis by promoting lipid abnormalities, systemic inflammation, and the development of neuropathy, which can mask or delay the recognition of PAD symptoms.
- **Hypertension:** Hypertension is a major contributor to PAD. Elevated blood pressure damages arterial walls, rendering them more susceptible to atherosclerosis. Hypertension also increases the risk of thrombosis and clot formation, further compromising arterial perfusion.
- **Hyperlipidemia:** High levels of low-density lipoprotein (LDL) cholesterol are a known risk factor for PAD. Elevated LDL cholesterol leads to the accumulation of lipid deposits in arterial walls, initiating the atherosclerotic process. Dyslipidemia management, particularly the reduction of LDL cholesterol, is essential in PAD prevention and management.
- **Age:** Age is an independent risk factor for PAD. As individuals age, atherosclerotic processes accumulate, contributing to a higher likelihood of PAD development.
- **Family History:** Genetic predisposition can influence an individual's susceptibility to PAD. A family history of vascular disease, particularly atherosclerosis, can confer a higher risk.
- **Obesity:** Obesity is one of the multifactorial contributors to the formation of PAD. Excess adipose tissue is associated with insulin resistance, chronic inflammation, and altered lipid profiles. These factors contribute to atherosclerosis and PAD development.
- **Physical Inactivity:** A sedentary lifestyle not only contributes to obesity but also reduces overall vascular health. Regular physical activity, conversely, is associated with better arterial function and can mitigate PAD risk.
- **Diet:** A diet rich in saturated and trans fats, along with a low intake of fruits and vegetables, promotes the development of atherosclerosis. In contrast, diets emphasizing heart-healthy components, such as whole grains, fruits, vegetables, and lean protein, are associated with reduced PAD risk.
- **Metabolic Syndrome:** Metabolic syndrome, which encompasses a cluster of conditions such as obesity, hypertension, hyperglycemia, and dyslipidemia, is a potent risk factor for PAD. The interplay of these factors significantly increases the risk of atherosclerosis and impaired arterial perfusion.
- **Gender and Menopause:** Men generally exhibit a higher prevalence of PAD, although the risk in women increases post-menopause. Reduced estrogen levels post-menopause contribute to endothelial dysfunction and alterations in lipid profiles, increasing susceptibility to PAD.



- **Race and Ethnicity:** Certain racial and ethnic groups, such as African Americans, are more prone to developing PAD. Genetic and environmental factors may contribute to these disparities.
- **Chronic Kidney Disease (CKD):** Reduced kidney function is associated with PAD, partly due to endothelial dysfunction and systemic inflammation. CKD patients are at increased risk of atherosclerosis, uncontrolled hypertension, thrombotic complications, and progressive PAD.

## Vessel Collateralization

Patients with peripheral arterial disease (PAD) often develop collateralization of blood vessels as a compensatory mechanism to improve perfusion to tissues affected by reduced blood flow. These new blood vessels, or collaterals, bypass or supplement the obstructed arteries and combat oxygen starvation of the tissues through “collateral circulation.” The main drivers for the development of collateral vessels in PAD are as follows:

- **Ischemia and Hypoxia:** In PAD, atherosclerosis narrows and sometimes occludes the arteries that supply blood to the limbs. The resulting reduced blood flow causes tissue ischemia (insufficient blood supply) and hypoxia (insufficient oxygen supply) to the tissues distal to the narrowing or occlusion. To combat this, the body initiates a response to create alternate pathways for blood flow.
- **Hypoxia-Inducible Factors (HIFs):** When cells are deprived of oxygen (as in the case of PAD), they activate hypoxia-inducible factors (HIFs). HIFs are transcription factors such as vascular endothelial growth factor (VEGF) and fibroblast growth factor (FGF). These factors promote the expression of genes involved in angiogenesis (the formation of new blood vessels) and arteriogenesis (the remodeling and enlargement of existing collateral vessels) through the stimulation of endothelial cells. Endothelial cells line blood vessels and play a central role in angiogenesis. They respond to these growth factors enabling endothelial cell proliferation and migration, which are essential for the formation of new capillaries in the direction of the hypoxic tissue.
- **Vasodilation:** When blood vessels are exposed to reduced oxygen levels, they often dilate in an attempt to increase blood flow. This vasodilation can help mobilize and enhance the development of collateral vessels.
- **Angiogenesis and Arteriogenesis:** Angiogenesis involves the formation of small capillaries, while arteriogenesis involves the enlargement and maturation of existing collateral vessels into larger arteries. These processes are driven by growth factors and signaling molecules like vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF), and various cytokines. As stated previously, they promote the proliferation and migration of endothelial cells and smooth muscle cells, leading to the formation of new blood vessels and the remodeling of existing ones.

- **Shear Stress:** As blood flows through collateral vessels, it exerts shear stress on the vessel walls. Shear stress is a mechanical force that can trigger the release of molecules like nitric oxide, which further stimulates vessel growth and remodeling.
- **Physical Activity:** Exercise and physical activity can also stimulate collateralization by increasing shear stress on the vessel walls, enhancing collateral vessel growth. Furthermore, physical activity increases oxygen demand of the active tissues creating a relative hypoxic environment, leading to collateralization/angiogenesis/arteriogenesis as described above. This is why dedicated exercise is an important aspect of treatment for claudication.

The development of collateral circulation in PAD is a gradual process that occurs over time in response to the chronic reduction in blood flow. While collateralization can help improve perfusion to some extent, it may not fully compensate for the lost function of the original, diseased arteries. In advanced stages of PAD, when the arterial obstruction is severe, collateralization may become insufficient, leading to continued symptoms of intermittent claudication, pain, and impaired wound healing.

### **Anatomic Distribution of PAD**

The distribution of disease in PAD varies among different risk groups such as smokers and diabetics. *Smokers tend to develop PAD predominantly in the infrainguinal arteries (CFA, SFA, PFA, popliteal artery), while diabetics tend to have higher infrapopliteal disease patterns (AT, TP, peroneal, DP).* (Conte et al. 2019a)

Smoking is a major risk factor for atherosclerosis, which is the primary underlying cause of PAD. Atherosclerosis tends to affect larger and proximal arteries, including the femoral and popliteal arteries, in smokers. The buildup of atherosclerotic plaques narrows these arteries, reducing blood flow to the lower extremities and causing symptoms like claudication.

Diabetes is associated with a specific type of arterial disease known as diabetic vasculopathy or microangiopathy. This condition leads to changes in the smaller blood vessels, including the tibial arteries and peroneal artery. The tibial arteries play a critical role in supplying blood to the feet, making them susceptible to diabetic vascular complications. Therefore, the microvascular changes associated with diabetes result in a distal distribution of arterial disease (i.e. toes and feet first).

## Diagnosics and Imaging

Measurement of the ankle-brachial index (ABI) is the primary method for establishing the diagnosis of PAD, however, a proper physical exam helps confirm the diagnosis of claudication.

### Peripheral Pulse Exam

In terms of physical exam, claudicants may exhibit signs of muscle wasting with thin, dry, and hairless skin, or even ulceration. These are all signs that blood flow is reduced to the point that there are not enough nutrients to feed these tissues. If ulceration or non-healing wounds are present, the patient likely has advanced peripheral arterial disease (i.e. critical limb ischemia). In addition, a lower extremity motor/sensory exam and peripheral pulse assessment is key.

#### **i** Note

Pulse exams study the presence, strength, and character of arterial blood flow and are graded on a scale from 0 to 4+. 0: non-palpable pulse 1+: diminished pulse 2+: normal pulse 3+: prominent pulse or aneurysmal. 4+: bounding pulse or aneurysmal.

Lower extremity peripheral pulse exams evaluate the dorsalis pedis (DP) and posterior tibial (PT) pulses using manual palpation. Patients with claudication may have pulse exams ranging from non-palpable to normal pulse. If patients have non-palpable pulses, then a continuous-wave Doppler probe can be used to assess blood flow.

### Ankle Brachial Index (ABI)

Ankle Brachial Index (ABI) is a non-invasive method that measures the ratio of blood pressure in the lower legs to the blood pressure in the arms. Performing an ABI requires an appropriately sized manual blood pressure cuff and a hand-held, continuous-wave Doppler probe. The patient should be in the supine position and allowed to rest before the examination begins. The blood pressure cuff is placed above the ankle. The Doppler probe is used to locate the dorsalis pedis (DP) or posterior tibial (PT) pulse, the cuff is inflated until the Doppler signal is obliterated, and then the cuff is then slowly released; the pressure at which the Doppler signal returns is the ankle pressure. The same process is repeated for both legs (DP and PT signals) and both arms. The arm pressure must be conducted with the manual blood pressure cuff that is placed above the target vessel and the Doppler probe that is placed below the cuff to evaluate blood flow. To calculate the ABI, the higher ankle pressure (DP or PT) is divided by the highest arm pressure (left or right).

ABI result of  $<0.9$  has shown to have high sensitivity and specificity with identifying PAD. Patients with claudication usually have an ABI ranging from 0.5 to 0.9, and those with ischemic rest pain or tissue loss usually have an ABI less than 0.5.

**i** Note

Below are the parameters used to diagnose PAD using ABI values.

1. ABI of  $>1.2$ : Calcification of arteries, non-compressible. Seen in diabetic patients.
  2. ABI of 0.9 – 1.2: Normal
  3. ABI of  $<0.9$ : Suggests arterial stenosis. (Khan, Farooqui, and Niazi 2008)
    - ABI 0.7-0.9 = mild PAD
    - ABI 0.4-0.7 = moderate PAD
    - ABI  $<0.4$  = severe PAD
- ABI 0.5 - 0.9: often causes claudication  
ABI  $<0.5$ : often causes ischemic rest pain/tissue loss

Below, we will review how to calculate the ABI for the right and left lower extremity using the following values:

Artery	Pressure (mm Hg)	
	Right	Left
Brachial	150	130
Dorsalis pedis (DP)	145	120
Posterior tibial (PT)	150	110

For both right and left lower extremities, the highest brachial pressure is 150 and will be used for comparison for the ankle/brachial index.

On the right lower extremity, using the DP systolic pressure the ABI is  $145/150 = 0.97$  and using the PT systolic pressure the ABI =  $150/150 = 1.0$ . So the Right ABI is 1.0, because we choose the highest of the two ankle pressures for the final value of the ABI.

On the left lower extremity, using the DP systolic pressure the ABI is  $120/150 = 0.8$  and using the PT systolic pressure the ABI =  $110/150 = 0.73$ . So the Left ABI is 0.8, because we choose the highest of the two ankle pressures for the final value of the ABI.

For this above example, R ABI = 1.0 and L ABI = 0.8. So this patient may have some peripheral vascular disease present in his left lower extremity as his ABI is less than 0.9.

**💡** Tip

[Here](#) is a video demonstrating how to obtain and calculate an ABI.

## Pulse volume recordings (PVR)

Pulse volume recordings (PVR) use pressure cuffs to record the shape of a pulse. The type of waveform provides important information about the characteristics of blood flow. These waveforms can be categorized as triphasic, bisphasic, or monophasic. Clinicians use these waveform patterns to diagnose and monitor arterial diseases, assess the severity of stenosis, and determine the effectiveness of treatment interventions.

Pulse volume recordings (PVR) use pressure cuffs to record the shape of a pulse. The type of waveform provides important information about the characteristics of blood flow. These waveforms can be categorized as triphasic, bisphasic, or monophasic. Clinicians use these waveform patterns to diagnose and monitor arterial diseases, assess the severity of stenosis, and determine the effectiveness of treatment interventions.

- **Triphasic Waveform:**

- A triphasic waveform consists of three phases: forward flow during systole, a brief reverse flow during early diastole, and a second forward flow component during late diastole.
- *Clinical Significance:* Triphasic waveforms are typically found in healthy, unobstructed arteries. They represent normal arterial blood flow. These waveforms are seen in arteries serving well-perfused tissues where resistance is low, and blood flow is unimpeded. Triphasic waveforms are commonly observed in the larger arteries of the extremities, such as the femoral and brachial arteries.

- **Biphasic Waveform:**

- A biphasic waveform displays two phases: an initial forward flow during systole (when the heart contracts and ejects blood) followed by brief reverse or “reflected” flow during early diastole.
- *Clinical Significance:* Biphasic waveforms are often associated with moderate arterial stenosis. The reverse flow component indicates some degree of obstruction. They can be observed in patients with early-stage PAD or moderate occlusions. Biphasic waveforms suggest that some blood is able to flow past the obstruction during systole but encounters resistance during diastole.

- **Monophasic Waveform:**

- A monophasic waveform consists of a single, continuous flow signal that moves in one direction. It typically lacks a distinct reverse or “reflected” flow signal.
- *Clinical Significance:* Monophasic waveforms are often seen in arteries downstream from significant stenosis or occlusion. In these cases, there is limited or no backflow during diastole (the relaxation phase of the cardiac cycle). They are commonly found in patients with advanced peripheral arterial disease (PAD) or severe arterial blockages, suggestive of reduced perfusion to the tissues served by the artery.

- **Flat-line:**
  - A flat waveform (i.e. no wave) does not necessarily indicate no blood flow. A flat waveform simply reflects the lack of pulsatile blood flow.

## Doppler

Doppler devices (handheld or otherwise) provide similar information to PVR studies. Instead of providing a visual representation of pulsatile flow, Doppler signals (sounds) are an auditory representation of flow. A triphasic pulse generates a triphasic signal (repeated sound), a biphasic pulse a biphasic signal, and a monophasic pulse a monophasic signal.

### Tip

[Here](#) is a video that demonstrates how to use a doppler probe and the signals associated with each waveform type. You can learn more about doppler probes and waveforms [here](#).

## Duplex Ultrasonography

Duplex ultrasonography (US) is another non-invasive imaging modality that can evaluate a patient's blood flow by measuring the speed at which blood travels through the vessel. In this way, a patient's hemodynamics can be assessed by identifying the location and degree of stenosis. Other imaging modalities such as a CT angiogram or invasive angiograms can also evaluate blood flow to the lower extremities.

A hematologic evaluation (including CBC, fasting blood glucose, creatinine, fasting lipid profile and urinalysis) is recommended in order to identify any associated comorbidities such as diabetes, hyperlipidemia, and chronic kidney disease that may also need to be addressed.

If patients have normal ABIs and pulse exams, it is important to consider other differentials of bilateral lower extremity pain include hematologic or neurogenic claudication (pain caused by lumbosacral nerve root impingement). Neurogenic claudication is positional, often worse when walking downhill and relieved by leaning forward (i.e. leaning on a walker).

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## Treatment

The mainstay of treatment for claudication is conservative management, including medical optimization and lifestyle modification. With regard to medical optimization, it is recommended

that patients start on antiplatelet therapy (Aspirin 81 mg daily) and a high dose statin. Patients without a prior history of congestive heart failure can also start on cilostazol (Pletal) to help with pain management. Patients with prior history of hypertension and diabetes should also be on appropriate antihypertensives to control their blood pressure and diabetes medications to control their blood sugar levels. Lifestyle modifications include smoking cessation, exercise training to develop collateral vessels, and diet modification to lower cholesterol intake. An appropriate exercise regimen is defined by at least 30 minutes of supervised ambulation/exercise at least 3 times per week. Patients should be followed closely with yearly ABIs by both Primary Care Physicians (PCPs) and Vascular Surgeons for progression of disease.

Below is an outline of appropriate treatment options for claudication:

### **Supervised Exercise Regimen**

Goal of at least 30 minutes of supervised walking exercise (at least 3 times a week)

### **Optimization of cardiovascular risk factors:**

**Hypertension** - Use of antihypertensive medications - Identify any underlying etiology of hypertension (ie renal artery stenosis, stressors)

**Hyperlipidemia** - Control of cholesterol levels - Diet/exercise modification - Use of medications: i.e. statins, anti-platelet agents

**Diabetes mellitus** - Control of blood glucose levels - Diet/exercise modification - Medications (i.e. antihyperglycemics)

**Smoking Cessation**

### **Pain Management**

**Anti-inflammatories** (ie acetaminophen, ibuprofen)

**Cilostazol**

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## Outcomes

When treated appropriately, most patients with claudication will not experience limb loss. Only 1-3% of claudicants develop critical limb ischemia and the complications associated with more severe PAD. While the majority of patients (70-80%) have stable claudication symptoms, approximately 20-30% of patients experience a worsening of their claudication. In terms of overall morbidity and mortality, patients with claudication have worse overall outcomes than the general population. 20% of patients with claudication will experience either a myocardial infarction (MI) or cerebrovascular accident (CVA) within their lifetime. Moreover, patients with diagnosis of claudication have a 5-year mortality rate of 10-15% due to the comorbidities associated with vascular disease. (Conte et al. 2015a)

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## Teaching Case

### Scenario

73 year old male with past medical history of hypertension, hyperlipidemia, and diabetes presents to clinic for pain in his lower extremities. Of note, he has smoked roughly one pack of cigarettes a day since he was 20 years old. He states that it is difficult for him to walk 3 blocks without having to stop which he finds particularly annoying. When asked where it hurts, he starts grabbing his calves. He describes an achy, cramping, and pain in his calves whenever he walks, mostly on the right side. He denies any new sores or wounds that are not healing.

### Exam

**HEENT:** pupils equal round reactive to light, no lymphadenopathy

**Cardiac:** Regular rate and rhythm (RRR)

**Pulmonary:** Clear to auscultation in all lung fields. No accessory muscle use.

**Abdominal:** Soft, non-tender, non-distended, no pulsatile masses felt.

**Vascular/Extremities:** Warm, no edema present. Minimal hair appreciated bilaterally. Non-palpable distal pulses. Bilateral femoral pulses appreciated. No wounds or sores noted.

### Imaging

**ABI:** Right: 0.7 Left: 0.65



## Discussion Points

**i** N.B. There is no prepared answers for the questions below.

However, we feel this chapter contains all the necessary information to answer the questions. If not, please let us know!

1. Please explain the pathophysiology for the patient's lower extremity pain and discomfort.
2. Please list the patient's risk factors for peripheral arterial disease. What is the best medical management to optimize the these risk factors?
3. What initial testing would you want to obtain and why?
4. What would be the first line of treatment for patients with symptomatic claudication after medical management?

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## Key Articles

1. Conte MS, Pomposelli FB, Clair DG, et al. Society for Vascular Surgery practice guidelines for atherosclerotic occlusive disease of the lower extremities: Management of asymptomatic disease and claudication. *Journal of Vascular Surgery*. 2015;61(3):2S-41S.e1. doi:10.1016/j.jvs.2014.12.009(Conte et al. 2015b)
2. Conte MS, Bradbury AW, Kolh P, et al. Global vascular guidelines on the management of chronic limb-threatening ischemia. *Journal of Vascular Surgery*. 2019;69(6):3S-125S.e40. doi:10.1016/j.jvs.2019.02.016(Conte et al. 2019b)
3. Gerhard-Herman MD, Gornik HL, Barrett C, et al. 2016 AHA/ACC Guideline on the Management of Patients With Lower Extremity Peripheral Artery Disease: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Circulation*. 2017;135(12). doi:10.1161/CIR.0000000000000470(Gerhard-Herman et al. 2017a)
4. Khan TH, Farooqui FA, Niazi K. Critical review of the ankle brachial index. *Curr Cardiol Rev*. 2008 May;4(2):101-6. doi: 10.2174/157340308784245810. PMID: 19936284; PMCID: PMC2779349.(Khan, Farooqui, and Niazi 2008)
5. McDermott MM, Criqui MH. Ankle-Brachial Index Screening and Improving Peripheral Artery Disease Detection and Outcomes. *JAMA*. 2018;320(2):143. doi:10.1001/jama.2018.8953(McDermott and Criqui 2018)

## Additional Resources

### Audible Bleeding Content

- Audible Bleeding Exam Prep: [Medical Management and Claudication Chapter](#)
  - Caitlin W. Hicks, MD, MS - Overuse of early peripheral vascular interventions in claudication. Listen to it below and find [additional information here](#), or find the episode wherever you listen to podcasts.
  - VSITE Review - Peripheral Arterial Occlusive Disease (Part One). Listen to it below and find [additional information here](#), or find the episode wherever you listen to podcasts.
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### Gore Combat Manual

The [Gore Medical Vascular and Endovascular Surgery Combat Manual](#) is an informative and entertaining read intended as a vascular surgery crash course for medical students, residents, and fellows alike. Highly accessible with a thoughtfully determined level of detail, but lacking in learning activities (e.g. questions, videos, etc.), this resource is a wonderful complement to the APDVS eBook.

 Tip

Please see pages 85-90.

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# Chronic Limb Threatening Ischemia (CLTI)

Nakia Sarad, DO, MS, Ezra Schwartz, MD,CM, MS, Chaitanya Dandu, MD, Marvin Chau, MD, Shannon McDonnell, BS, Bernadette Aulivola, MD, FACS, Kelly Kempe, MD, RPVI, FACS, Kaled Diab, MD, and Lindsey Korepta, MD, FACS.

## Note

By the end of this chapter, students will:

- Define chronic limb-threatening ischemia (CLTI) and summarize its etiology and pathophysiology.
- Identify the clinical features and diagnostic criteria used to diagnose CLTI.
- Elaborate on the complications and potential consequences of untreated CLTI.
- Outline a comprehensive history and physical examination to evaluate patients with suspected CLTI.
- Interpret diagnostic tests such as ankle-brachial index (ABI), Doppler ultrasound, and angiography to confirm the diagnosis.
- Develop and implement a management plan for patients with CLTI, including medical, endovascular, or surgical interventions.

## Key Facts

1. Chronic limb-threatening ischemia (CLTI) refers to a severe and advanced form of peripheral arterial disease (PAD) characterized by chronic inadequate blood supply to the lower extremities for **greater than 2 weeks**.
2. **CLTI presents as ischemic rest pain, tissue loss (e.g. ulcers, gangrene), or both.**
3. CLTI is associated with amputation, impaired quality of life, and mortality.
4. Rutherford Classification and Fontaine Grading are classification systems developed to categorize the degree of severity of CLTI, which also aids in management.
5. Newer classification systems including WiFi (Wound, Ischemia, and Foot Infection)

and GLASS (Global Limb Anatomic Staging System) aim to aid in prognostication and management of CLTI.

#### Claudication Slide Deck

Please find the slide deck corresponding to this eBook chapter [here](#).

#### 💡 How We Suggest Using the Pre/Post Questions

The pre/post questions are listed below. They are all multiple choice questions with a single right answer. To best guide your learning, we have hidden the answers in a collapsible menu. Before reading the chapter, we suggest giving the questions a try, noting your answers on a notepad. After reading the chapter, return to the questions, re-evaluate your answers, and then open the collapsible menu to read the correct answer and discussion. Do not fret if you have difficulty answering the questions before reading the chapter! By the end of the chapter, we are certain you will have covered the knowledge necessary to answer the questions. There will be a teaching case at the end of the chapter. This is another opportunity to exercise your new knowledge!

## Pre/Post Questions

### Case Based Questions

1. A 45-year-old male with a history of type 2 diabetes mellitus presents to the emergency department with wet gangrene of his first, fourth, and fifth digits of his right lower extremity. He has a right dorsalis pedis and posterior tibial artery pulse. *What level of amputation is appropriate in this situation?*
  - A. Transmetatarsal amputation
  - B. Below-knee amputation
  - C. Through-knee amputation
  - D. Above-knee amputation

#### Answer

#### **A. Transmetatarsal amputation (TMA).**

Discussion: This patient requires removal of the affected toes. However, if only the toes are removed, the remaining toes are subject to ulcer formation and or infection. If 3

or more toes are removed, the stability of the foot can be compromised and thus the correct amputation for him is a transmetatarsal amputation (TMA). He has adequate distal perfusion with palpable pedal pulses, and therefore, he will likely be able to heal from the amputation. A TMA will remove infected tissue and will provide him with the greatest amputation stump length. A below-knee amputation (BKA), through-knee amputation (TKA), and above-knee amputation (AKA) are too extreme for the burden of foot infection. Furthermore, the other alternatives are associated with greater morbidity and amount of energy required to ambulate, with the degree of severity increasing the more proximal the amputation (AKA is worse than BKA which is worse than TMA). *Lower extremity amputation: techniques and results. In: Cronenwett JL, Johnson KW, eds. Rutherford's Vascular Surgery. 8th ed. Philadelphia, PA: Elsevier; 2014.*

2. A 72-year-old male with diabetes presents to his PCP's office for a nonhealing ulcer to the plantar aspect of his right foot. Laboratory results and x-ray imaging are unremarkable. On examination, you note that his pulses are nonpalpable. Doppler examination reveals monophasic signals. He has necrotic, nonviable tissue to the base of the wound. There is no erythema or evidence of abscess. The patient has not been taking antibiotics. *What is the most likely cause of the non-healing in this scenario?*
- A. Pressure
  - B. Infection
  - C. Inadequate wound care
  - D. Peripheral vascular (arterial) disease

Answer

**D. Peripheral vascular (arterial) disease**

Discussion: This patient has nonpalpable pulses and monophasic doppler signals. Along with necrotic tissue to the wound base, these findings are consistent with peripheral artery disease (PAD). You may choose to order ankle-brachial index/pulse volume recording studies. He should be referred to a vascular specialist for further workup before any intervention is made. He does not have erythema or evidence of abscess on examination, and therefore it is not likely that primary infection is the cause of nonhealing. The scenario does not state what type of wound care the patient was receiving. In the presence of PAD, the type of wound care does not matter as much until the PVD is addressed first. Without adequate blood flow to a wound, the wound will not have the nutrients and cells necessary to heal. *Bader MS. Diabetic foot infection. Am Fam Physician. 2008;78(1):71-79.*

3. A 56-year-old male with hypertension, diabetes presents to the emergency department with redness and purulence draining from his right great toe. His heart rate is 110

beats/min, his blood pressure is 100/80 mm Hg, and his temperature is 38.5°C. Laboratory tests show a white blood count of 14,000/ $\mu$ L. An x-ray of his foot is performed, which demonstrates gas in his right great toe. *What is the best course of treatment?*

- A. Outpatient treatment with oral antibiotics and dressing changes.
- B. Inpatient treatment with intravenous antibiotics alone.
- C. Inpatient treatment with intravenous antibiotics and dressing changes.
- D. Inpatient treatment with intravenous antibiotics and emergent surgical debridement .

Answer

**D. Inpatient treatment with intravenous antibiotics and emergent surgical debridement.**

Discussion: This patient is septic from a diabetic wound infection. He is febrile and tachycardic, and he has relative hypotension. This type of patient should not be treated as an outpatient. The correct treatment course involves admission, broad-spectrum antibiotics, and source control with surgical debridement. Gas in the tissues on x-ray in the setting of an infection is an ominous sign and should always prompt surgical debridement. Hence, admission with intravenous antibiotics alone or admission with intravenous and dressing changes alone are inadequate management. *Hingorani A, LaMuraglia GM, Henke P, et al. The management of diabetic foot: a clinical practice guideline by the Society for Vascular Surgery in collaboration with the American Podiatric Medical Association and the Society for Vascular Medicine. J Vasc Surg. 2016;63(2S):3S-21S.*

4. A 72-year-old female with diabetes has a chronic right heel ulcer that persists despite local wound care. A magnetic resonance imaging scan demonstrates osteomyelitis of the calcaneus, and a metal probe inserted into the ulcer can “probe to bone.” She has a palpable ipsilateral dorsalis pedis pulse. *Which operation would be most appropriate?*
- A. Transmetatarsal amputation.
  - B. Below-knee amputation.
  - C. Local calcaneus excision with full-thickness skin flap.
  - D. Above-knee amputation.

Answer

**B. Below-knee amputation**

Discussion: Osteomyelitis of the calcaneus is a difficult condition to treat. The calcaneus is crucial for weight bearing and skin flaps are unlikely to heal in this location. The below-knee amputation will remove the infection and still provide the longest amputation

stump to decrease the work associated with ambulation. As such, an above-knee amputation is too aggressive. A transmetatarsal amputation will not remove the infected bone. *Lower extremity amputation: techniques and results. In: Cronenwett JL, Johnson KW, eds. Rutherford's Vascular Surgery. 8th ed. Philadelphia, PA: Elsevier; 2014. UpToDate (Publisher Subscription Required): Lower extremity amputation.*

5. A 70-year-old male with poorly controlled diabetes and no smoking history presents to the clinic for an ischemic toe ulcer. What level of infrainguinal arterial occlusive disease is he most at risk for developing based on this history?
- A. Superficial femoral artery disease
  - B. Deep femoral artery disease
  - C. Iliac artery disease
  - D. Aortic disease
  - E. Tibial artery disease

Answer

**E. Tibial artery disease**

Discussion: Risk factors for peripheral arterial disease include age, male gender, hypertension, diabetes, smoking, dyslipidemia, family history, and homocysteinemia. Individuals who are heavy smokers tend to develop superficial femoral artery disease, whereas those with diabetes and renal failure tend to develop infrapopliteal disease. The tibial artery is the only infrapopliteal vessel listed as an answer option. All other options are suprapopliteal.

6. A 75-year-old male with a history of migraines, diabetes, hyperlipidemia, hypertension, triple coronary bypass (CABG x3), recurrent cellulitis, glaucoma and benign prostatic hyperplasia (BPH) presents to your clinic due to pain. He has smoked approximately 2 packs per day for over 30 years. Despite smoking cessation counseling, he continues to smoke 1-2 cigarettes a day. He reports pain with walking for the past several years. After walking one city block, his calves begin to hurt. He intermittently develops toe sores and wounds that can take 1-2 months to heal. If the wound is larger, it can take 3-4 months to heal. *What risk factors does this patient have that contribute to the most likely disease process?*
- A. Migraines
  - B. BPH
  - C. Glaucoma

- D. Smoking
- E. Cellulitis

Answer

**D. Smoking.**

Discussion: Smoking contributes to worsening peripheral arterial disease (PAD). Peripheral arterial disease results when the arteries narrow due to plaque accumulation which prevents or decreases the amount of oxygen-rich blood reaching your legs. The chemicals in tobacco products increase the rate of plaque formation in arterial walls thereby narrowing the arterial lumen. Smoking also constricts blood vessels and causes blood to clot, ultimately putting PAD patients at a higher risk of further complications such as heart attacks, limb amputations, and death. Cellulitis and other wound complications can be an effect of peripheral arterial disease, but is not the cause of peripheral disease. The other options of migraines, BPH, glaucoma have no known associations with PAD.

7. A 74-year-old female with a past medical history of IDDM with ESRD on dialysis, hypercholesterolemia, hypertension, and previous MI is referred to you for evaluation of crampy calf pain for the last 2 months with onset upon physical exertion. It has since progressed to pain at rest. You perform ABIs which reveal critical limb ischemia. *Which of the following will you most likely observe when performing preoperative imaging of this patient prior to infrapopliteal bypass?*
- A. Critical occlusion of only the medial and lateral geniculate arteries.
  - B. Loss of enhancement in the region of the popliteal artery but reconstitution showing good distal vessel runoff.
  - C. Enhancement in the peroneal artery contributing to pedal circulation.
  - D. Complete lack of runoff past the region of the Hunter's canal.
  - E. Full enhancement in the posterior tibial artery ending in the region of the medial malleolus.

Answer

**C. Enhancement in the peroneal artery contributing to pedal circulation.**

Discussion: Because of its location in the deep compartments of the lower leg, the peroneal artery becomes the most likely artery to remain patent in diabetic patients with vascular disease. Therefore, when performing preoperative imaging to plan an infrapopliteal bypass, a more common target site for anastomosis is the peroneal artery. It is important to remember that it is necessary for the peroneal artery to have patency to the anterior or posterior tibial artery to perfuse the entire foot. Choice B is incorrect because this is



more of a presentation in a patient with acute occlusion of only the popliteal artery, as in a posterior knee dislocation. Patients with vascular disease tend to have more distal vessels affected before proximal. Choice A is incorrect because the geniculate arteries themselves are collaterals. Although there is a chance that these will be affected by vasculopathy, it is unlikely that only they will be affected. D is a representation of imaging in a patient with femoral artery disease. Choice E is a scenario that would rarely ever occur without proximal tibial artery being occluded.

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## Introduction

### Tip

The term peripheral arterial disease (PAD) is often used interchangeably with peripheral vascular disease (PVD) when referring to arterial blockages.

**Chronic limb-threatening ischemia (CLTI) is defined as the presence of peripheral arterial disease (PAD) with associated ischemic rest pain, gangrene, or lower limb non-healing ulceration for greater than 2 weeks duration**

The prevalence of PAD is approximately 6% in patients aged 40 years or older in the United States. Amongst patients with PAD, 11% of patients (approximately 2 million individuals) will present with CLTI within their lifetime.(Conte et al. 2019c)

CLTI has an increased risk of amputation, cardiovascular morbidity, and mortality.

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## Etiology

CLTI is a severe manifestation of peripheral arterial disease (PAD), a disease caused by the build-up of atherosclerotic plaque in the arteries. The pattern of occlusive disease can be found in one or a combination of locations including the aortoiliac or infrainguinal vessels (i.e. femoral, popliteal, and tibial arteries) with varying lesion lengths. The superficial femoral artery (SFA) is the most common location where lesions occur. Amongst diabetics, it is typical to find heavier disease burdens more distally, such as the infrapopliteal, tibial vessels (i.e. the tibial trunk, anterior tibial, posterior tibial, and peroneal arteries).

 Tip

[Here](#) is an image that demonstrates the various peripheral vessels that can be diseased and occluded in a patient with CLTI.

Risk factors for CLTI are the same as for PAD and include advanced age, race (with non-hispanic blacks having the highest risk), male gender, smoking, diabetes mellitus, dyslipidemia, hypertension, and renal insufficiency. It is important to note that any other etiologies including venous, traumatic, embolic, or nonatherosclerotic disease processes are NOT considered a part of chronic limb-threatening ischemia.

 Note

For a more comprehensive review of lower limb arterial anatomy and PAD risk factors, please see [?@sec-claudication](#).

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## Diagnosics and Imaging

The diagnosis of CLTI requires a prior diagnosis of PAD in association with ischemic rest pain or tissue loss. Pain should be present for more than two weeks and associated with at least one abnormal hemodynamic parameter.

A complete evaluation of patients with CLTI should include:

1. A good patient history
2. Physical examination
3. Noninvasive hemodynamic tests
  - Ankle-Brachial Index (ABI) and Pulse Volume Recording (PVR)
  - Arterial Doppler Ultrasound
4. Angiography
  - CTA (abd + pelvis + lower extremity with triple vessel runoff)
  - If necessary, invasive angiography

### Tip

For a comprehensive description of the arterial patient history and physical exam, please see [?@sec-patienthistoryandphysicalexaminationofheararterialsystem](#).

## Patient History

- Patients may state that they have persistent pain in their legs, even at rest.
- Rest pain and nonhealing wounds are highly indicative of CLTI.
- Patients may also share experiencing rest pain in bed relieved by dangling their feet off the side of their bed. By dangling their feet over the side of the bed, gravity increases blood flow through diseased and collateral vessels thereby reducing ischemia and pain.

## Physical Exam

Common physical exam findings on the lower extremities include:

- Cyanosis
- Cool Extremities
- Hair loss
- Dry skin
- Muscle atrophy
- Dystrophic toenails (thick toenails)
- Capillary refill > 5 seconds
- Non-palpable distal pulses
- Non-healing wounds

## Non-invasive Hemodynamic Testing

In terms of non-invasive testing, as stated above, a combination of ABI and PVR is the gold standard in establishing a diagnosis. As a review, the ABI is a non-invasive test that compares blood pressure in the ankle with blood pressure in the arm. ABIs are calculated by dividing the systolic ankle pressure by the highest systolic arm/brachial pressure (between the two arms).

An ABI value: -  $X > 1.2$  is non-compressible, calcification of arteries

- $0.9 < x < 1.2$  is normal
- $0.7 < x < 0.9$  reflects a mild disease burden
- $0.4 < x < 0.7$  reflects a moderate disease burden
- $x < 0.4$  reflects a severe disease burden

[Here](#) is an example of ABI/PVR recordings. Patient A (left) shows a normal ABI/PVR recording, while patient B (right) shows an abnormal ABI with diminished waveforms.

Arterial Doppler ultrasound can also be utilized to evaluate blood flow and detect arterial stenosis or occlusion.

For a definitive CLTI diagnosis, pain should be present for more than two weeks and associated with at least one of the following abnormal hemodynamic parametersI:

1. Ankle-brachial index (ABI)  $< 0.5$ .
2. Absolute toe pressure (TP)  $< 30$  mmHg.
3. Absolute Ankle Pressure (AP)  $< 50$  mmHg.
4. Transcutaneous pressure of oxygen (TcPO<sub>2</sub>)  $< 30$  mmHg.
5. Flat or minimal pulsatile volume recording (PVR) waveforms.

## Angiography

CT Angiography (CTA) with tri-vessel runoff (i.e. anterior tibial, posterior tibial, peroneal arteries) of lower extremities can help identify the location(s) of disease. However, this investigation requires a contrast load and should be carefully considered for those patients with renal disease.

Lastly, diagnostic angiography is an invasive imaging technique that provides detailed information about the arterial anatomy and helps identify specific sites of blockage or stenosis. Diagnostic angiography involves the patient undergoing a femoral artery puncture to access the arterial system. Next, a series of catheters and wires are used to deliver contrast. Fluoroscopic imaging of the lower limb reveals vessel anatomy and identifies areas of stenotic disease. As arterial access is already achieved, immediate intervention may be considered including angioplasty (i.e. ballooning of stenosis) or stenting.

## Classification Systems

### Tip

Below, we will introduce and summarize the most common classification systems. For a high-yield summary of all classification systems, please see this [paper](#). (Hardman et al. 2014)

### The Rutherford Classification and Fontaine Grading

- The Rutherford Classification is the mainstay of diagnosing the severity of CLTI, however, Fontaine grading can also be used.
- These grading systems are based on symptom presentation. Patients with CLTI usually present with Fontaine Grade 2 or higher or Rutherford Category 3 or higher.
- Minor tissue loss is considered defined as non-healing ulcers with focal gangrene that do not exceed the foot digit.
- Major tissue loss extends beyond the transmetatarsal level with the functional foot no longer salvageable. Patients with major tissue loss most often receive amputations.

Fontaine Grade	Rutherford Category	Clinical Description
0	0	Asymptomatic
	1	Mild claudication
1	2	Moderate claudication
	3	Severe claudication
2	4	Ischemic rest pain
3	5	Minor tissue loss
	6	Major tissue loss

### The Wound, Ischemia, and foot Infection (WIFI) classification

- The Wound, Ischemia, and foot Infection (WIFI) classification was proposed in 2014 as an integrated lower extremity wound classification and prognostication system. The WIFI classification was inspired by the tumor, node, metastasis (TNM) staging system for cancer.
- Incorporating the presence and severity of lower extremity wounds, ischemia, and infection, the classification system informs providers of the risk of lower extremity amputation and potential benefit of revascularization.
- For the limb in question, a severity grade of 0 to 3 is assigned to the severity and extent of wound, ischemia and foot infection, respectively, where 0 presents none, 1 mild, 2, moderate and 3 severe disease.

- Using the table [here](#), the combination of these three scores assigns the limb in question to one of four threatened limb clinical stages.
- While an oversimplification, the premise of the WIfI system is that the risk of amputation increases as the disease burden of the limb in question progresses from WIfI stage 1 (very low risk) to tage 4 (high risk).

Wound (ulcer or gangrene)	Ischemia (Toe Pressure/TcPO2 and ABI)	foot Infection
0: No ulcer or no gangrene	0: TP $\geq$ 60 mmHg, ABI $\geq$ 0.80	0: Not infected
1: Small ulcer and no gangrene	1: TP = 40-59 mmHg, ABI 0.60-0.79	1: Mild ( $\leq$ 2 cm cellulitis)
2: Deep ulcer or gangrene limited to toes	2: TP 30-39 mmHg, ABI 0.40-0.59	2: Moderate ( $>$ 2 cm cellulitis or involving structures deeper than skin and subcutaneous tissues)
3: Extensive ulcer or extensive gangrene	3: TP $<$ 30 mmHg, ABI $\leq$ 0.39	3: Severe (systemic response / SIRS)

 Tip

The SVS Guidelines Mobile App has an easy-to-use WIfI Calculator. - [Apple App Store](#)  
- [Android Google Play](#)

### The Global Limb Anatomic Staging System (GLASS) Classification

- The Global Limb Anatomic Staging System (GLASS) is a framework to estimate the success of lower limb revascularization based on the extent and the distribution of the atherosclerotic lesion. By categorizing the patient's lesion into one of three GLASS stages, the surgeon can predict the likelihood of immediate technical failure and one-year limb-based patency (LBP) following endovascular intervention. This information is extremely informative when planning a surgical intervention.
- To perform GLASS staging, the surgeon must obtain high quality imaging of the lower extremity to score the length and burden of disease femoropopliteal segment (FP) and infrapopliteal segment (IP).

Scoring System below (Wijnand et al. 2021)

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Femoro-Popliteal (FP)

Grading

---

0	Mild or no significant (<50%) disease
1	Total length SFA disease <1/3 (<10 cm); may include single focal CTO (<5 cm) as long as not flush occlusion; popliteal artery with mild or no significant disease
2	Total length SFA disease 1/3–2/3 (10–20 cm); may include CTO totaling <1/3 (10 cm) but not flush occlusion; focal popliteal artery stenosis <2 cm, not involving trifurcation
3	Total length SFA disease >2/3 (>20 cm) length; may include any flush occlusion <20 cm or non-flush CTO 10–20 cm long; short popliteal stenosis 2–5 cm, not involving trifurcation
4	Total length SFA occlusion >20 cm; popliteal disease >5 cm or extending into trifurcation; any popliteal CTO

---

Infra-Popliteal (IP)

Grading

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0	Mild or no significant (<50%) disease
1	Focal stenosis <3 cm not including TP trunk
2	Total length of target artery disease <1/3 (<10 cm); single focal CTO (<3 cm not including TP trunk or target artery origin)
3	Total length of target artery disease 1/3–2/3 (10–20 cm); CTO 3–10 cm (may include target artery origin, but not TP trunk)
4	Total length of target artery disease >2/3 length; CTO >1/3 (>10 cm) of length (may include target artery origin); any CTO of TP trunk

---

Trifurcation involvement is defined by disease that include the origin of either the anteriortibial or tibioperoneal trunk.

The combined scores of the FP and IP segments can then be used to determine the GLASS stage using the table below:

		FP Grade				
		0	1	2	3	4
IP Grade	0	NA	I	I	II	III
	1	I	I	II	II	III
	2	I	II	II	II	III
	3	II	II	II	III	III

FP Grade						
4	III		III	III	III	III

**Stage I: Average Complexity Disease:** immediate technical failure <10% AND >70% 12-month LBP. **Stage II: Intermediate Complexity Disease:** immediate technical failure <20% AND 12-month LBP 50–70%. **Stage III: High Complexity Disease:** immediate technical failure >20%; OR <50% 12-month LBP.

**i** Note

For a more complete explanation of the The Global Limb Anatomic Staging System (GLASS), please see [here](#).

**💡** Tip

The SVS Guidelines Mobile App has an easy-to-use GLASS Calculator. - [Apple App Store](#) - [Android Google Play](#)

### The Trans-Atlantic Inter-Society Consensus (TASC classification)

In 2000, a group of fourteen societies from North America and Europe representing a variety of disciplines including vascular surgery, internal medicine, interventional radiology, and cardiology convened to create a consensus in the classification and treatment of patients with peripheral artery disease (i.e. intermittend claudication, acute limb ischemia, and chronic limb ischemia).

The Trans-Atlantic Inter-Society Consensus (TASC) document focuses on individual arterial segments and classifies lesions into one of four large groups (TASC A through TASC D) according to the pattern of disease. Recommendations are then offered according to TASC Classification.

**i** Note

Please see these depictions of the TASC classification system: [Image 1](#) & [Image 2](#).

- TASC A lesions are those that should have excellent results from endovascular management alone.
- TASC B lesions are those that should have good results from endovascular management and endovascular interventions should be the first option offered to the patient.



- TASC C lesions are those that have better long-term results with open surgical management. Endovascular techniques should be reserved for patients who are poor surgical candidates.
- TASC D lesions are those that should be treated by open surgery.

*Important caveats:* - Developments in endovascular techniques and devices since the introduction of the TASC document have increased the role of endovascular surgery in treating TASC C and TASC D lesions.

- Many patients with CLTI present with multisegment disease thereby complicating the clinical decision-making usefulness of TASC recommendations. TASC is more reasonably useful for assessing lesion-specific device performance.

The consensus was updated in 2007 (TASC II) and included representatives from Australia, South Africa, and Japan.

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## Treatment

Treatment of CLTI includes conservative management and invasive intervention. Lifestyle modification is recommended and highly encouraged and includes smoking cessation, regular exercise, and a healthy diet to reduce cardiovascular risk factors.

### Conservative management

Pharmacotherapy with antiplatelet agents and statins plays a large role in helping prevent the progression of CLTI. Antiplatelet agents – such as aspirin or clopidogrel, reduce the risk of blood clots, while statins control cholesterol levels and reduce the progression of atherosclerosis. In some instances, vasodilators, such as cilostazol, can be utilized to improve blood flow and relieve symptoms, especially in those individuals experiencing ischemic rest pain.

### Invasive management

The mainstay of treatment modality in CLTI is revascularization with endovascular therapy, surgical bypass, or a combination of both.

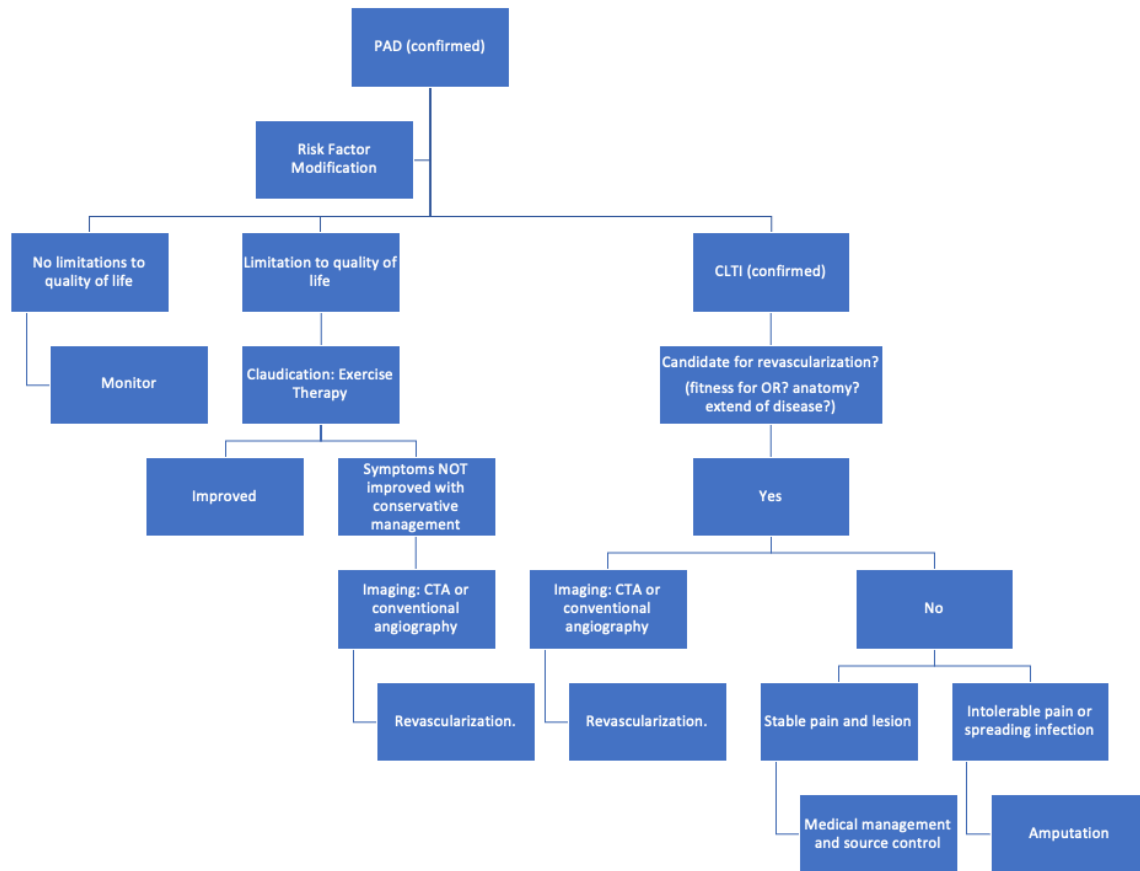
Endovascular revascularization is the least invasive approach to opening narrowed or blocked arteries. Using real-time contrast-enhanced images, the surgeon can perform angioplasty (i.e. ballooning of stenosis) stenting, and/or atherectomy (i.e. device mediated removal of the intimal layer and atherosclerotic material).

A surgical bypass, or extra-anatomic bypass, is the surgical creation of a new path for blood around the area of vessel stenosis/occlusion. Conduits for bypass can be artificial (e.g. PTFE or dacron grafts) or natural (e.g. autogenous or cadaveric saphenous vein grafts). In severe cases where limb salvage is not possible or limb-threatening infection persists even after attempts at revascularization, amputation may be necessary to preserve overall health and quality of life.

In addition to conservative management and surgical intervention, appropriate wound care is an important aspect of CLTI treatment. Surgical wound debridement, infection control, offloading techniques, and daily specialized wound care are integral in promoting healing and preventing amputation.

It is important to note that CLTI is a chronic disease, and patients may require multiple interventions, be on lifelong aspirin/statin therapy, and require close follow-up with routine imaging surveillance for the entirety of their lifetime. Moreover, vascular surgeons work with a team of physicians from other specialties – including primary care, cardiology, podiatry, nephrology, and endocrinology – in order to ensure that patients with CLTI are appropriately optimized given their respective comorbidities to improve overall outcomes. This integrative physician network to help manage CLTI patients is important as patency rates of stents and bypasses can be severely limited by a patient’s underlying disease burdens from uncontrolled hypertension, diabetes, renal insufficiency, and tobacco abuse.

Below is a sample algorithm for treatment management of a patient with PAD that progresses to CLTI. Candidacy for revascularization indicates that the patient is able to tolerate anesthesia and has suitable anatomy - including possible targets for bypasses or the ability to traverse lesions endovascularly.



[Here](#) is an image demonstrating angioplasty, atherectomy, and placement of stents.

[Here](#) is an image demonstrating surgical or extra-anatomic bypass.

## Outcomes

- Patients with CLTI have variable outcomes with regard to limb loss and overall mortality.
- Approximately 90% of all CLTI patients undergo a revascularization procedure (bypass or endovascular intervention) in their lifetime. (Conte et al. 2019d)
- For CLTI patients who do not undergo any revascularization, the natural history includes a 1-year major amputation and 1-year mortality rate of 22%. (Conte et al. 2019d)
- When looking at overall 1-year outcomes for all CLTI patients, 1-year amputation rates are at 15-20% and 1-year mortality is at 15-40%. (Conte et al. 2019d)

- Less than half of all CLTI patients (45%) will have both limbs after 1-year from initial time of diagnosis. These rates are even higher amongst the subset of CLTI patients who have diabetes. (Conte et al. 2019d)
- As the diagnosis of CLTI carries a high risk of mortality and morbidity even with vascular intervention to improve blood flow, there is a strong emphasis on preventing the progression of PAD to CLTI. (Conte et al. 2019d)

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## Surveillance

### Medical Treatment Surveillance

- Patients with CLTI have variable outcomes with regard to limb loss and overall mortality.
- After revascularization procedures, Patients remain on dual antiplatelet therapy (DAPT) for 1-6 months.
- Through DAPT has been show to have increased bleeding risk, it may result in increased survival and reduced risk of amputation. (Conte et al. 2019d)
- There is not a clear benefit to the use of of cilostazol, a PDE inhibitor, after endovascular interventions for CLTI. (Conte et al. 2019d)

### Endovascular Surveillance

- Early failure of endovascular procedures is common. As such, it is recommend that patients undergo duplex ultrasound surveillance and duplex guided interventions to prompt long-term patency. However, there are no set guidelines for how long and how frequently to perform surveillance.
- Recommended methods of surveillance include clinic visits, ankle-brachial indexes (ABIs), and duplex scans.
- Surveillance intervals can range from 3-6 months.
- Ultimately, there is no strong evidence of benefit of routine duplex surveillance after endovascular intervention of CLTI; however, patients with the characteristics below may benefit from duplex surveillance. (Conte et al. 2019d)

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#### Patient Risk Factors that may Prompt Duplex Surveillance

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Multiple failed angioplasties and failed bypasses

Severe ischemia

Unresolved tissue loss

Poor Inflow

## Vein and Prosthetic Bypass Surveillance

Surveillance of vein grafts after bypass surgery may solely be clinical and may include the vascular laboratory as well. The SVS recommends a 2 year surveillance program postoperatively to detect new symptoms, monitor changes in ABIs, and monitor for stenotic lesions within graft or at surveillance sites.

- Duplex surveillance and intervention based on duplex findings lead to better patency rates of the vein graft.
- Though there are no set guidelines for frequency of surveillance. Many studies in the literature often perform surveillance 30 days postoperatively and at 3-6 months intervals.
- Ultimately, though duplex surveillance does result in better patency of vein grafts, there is no clear evidence that duplex surveillance for CLTI results in clinical benefits for patients.

Prosthetic bypasses are known to fail more frequently and more early compared to vein bypasses. Similar to vein bypasses, the SVS does not have hard recommendations for when to perform duplex surveillance.

- Many studies perform duplex at 30 days postoperatively and at 6 month intervals.
- Despite this, the duplex itself does not always reliably detect intervenable lesions as it does in vein bypasses.

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## Teaching Case

### Scenario

A 73-year-old male with a significant smoking history and medical history including 4 vessel coronary bypass 10 years ago, hypertension, hyperlipidemia, and diabetes presents to clinic for pain in his lower extremities. He states that it is difficult for him to walk 3 blocks without having to stop. He locates the pain in his calves bilaterally. He endorses he has experienced this pain for over 5 years. He mentions that he previously had a wound that took almost 3 months to heal. He denies presence of new sores or wounds. He had a right lower extremity angiogram 2 months ago that failed to recanalize the superficial femoral artery (SFA).

## Exam

**HEENT:** No prior neck incisions, no lymphadenopathy.

**Cardiac:** Regular rate and rhythm (RRR), healed stereotomy scar.

**Pulmonary:** Clear to auscultation bilaterally, no increased work of breathing.

**Abdominal:** Soft, non-distended (ND), non-tender (NT)

**Vascular/Extremities:** Non-palpable lower extremity pulses and what appears to be a wound that is starting to form on the distal big toe of the right foot. He does have 2+ femoral pulses. On Doppler exam, he has monophasic dorsalis pedis (DP) and posterior tibial (PT) signals in his right lower extremity.

## Imaging

**Duplex Ultrasound:** On duplex ultrasound, he has a long segment occlusion of the superficial femoral artery (SFA) and on CTA he has two-vessel run off to the right lower extremity with well-formed collateralization.

## Discussion Points

**i** N.B. There is no prepared answers for the questions below.

However, we feel this chapter contains all the necessary information to answer the questions. If not, please let us know!

1. Please explain the pathophysiology of the patient's lower extremity pain and non-healing wounds.
2. Please list the patient's risk factors for Chronic Limb Threatening ischemia.
3. Explain why there is well-formed collateralization that was seen on CTA.
4. What next steps should be pursued to pursue treatment for the patient? Should the patient stop smoking?
5. What surgical treatment could be offered to this patient?
6. What additional imaging would help with surgical planning for the patient?

## Key Articles

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3. Blanchette, Virginie, Malindu E. Fernando, Laura Shin, Vincent L. Rowe, Kenneth R. Ziegler, and David G. Armstrong. Evolution of WIfI: Expansion of WIfI Notation After Intervention. *The International Journal of Lower Extremity Wounds*, 2022. doi:10.1177/15347346221122860. (Blanchette et al. 2022)
4. Conte MS, Bradbury AW, Kolh P, et al. Global vascular guidelines on the management of chronic limb-threatening ischemia. *Journal of Vascular Surgery.* 2019;69(6):3S-125S.e40. doi:10.1016/j.jvs.2019.02.016.(Conte et al. 2019e)
5. Gerhard-Herman MD, Gornik HL, Barrett C, et al. 2016 AHA/ACC Guideline on the Management of Patients With Lower Extremity Peripheral Artery Disease: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Circulation.* 2017;135(12). doi:10.1161/CIR.0000000000000470.(Gerhard-Herman et al. 2017b)
6. Mills JL, Conte MS, Armstrong DG, et al. The Society for Vascular Surgery Lower Extremity Threatened Limb Classification System: Risk stratification based on Wound, Ischemia, and foot Infection (WIfI). *Journal of Vascular Surgery.* 2014;59(1):220-234.e2. doi:10.1016/j.jvs.2013.08.003.(Mills et al. 2014)
7. Wijnand JGJ, Zarkowsky D, Wu B, van Haelst STW, Vonken EPA, Sorrentino TA, Pallister Z, Chung J, Mills JL, Teraa M, Verhaar MC, de Borst GJ, Conte MS. The Global Limb Anatomic Staging System (GLASS) for CLTI: Improving Inter-Observer Agreement. *J Clin Med.* 2021 Aug 4;10(16):3454. doi: 10.3390/jcm10163454. PMID: 34441757; PMCID: PMC8396876.(Wijnand et al. 2021)

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## Additional Resources

### Audible Bleeding Content

- Audible Bleeding Exam Prep: [CLTI Chapter](#)

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## **Gore Combat Manual**

The [Gore Medical Vascular and Endovascular Surgery Combat Manual](#) is an informative and entertaining read intended as a vascular surgery crash course for medical students, residents, and fellows alike. Highly accessible with a thoughtfully determined level of detail, but lacking in learning activities (e.g. questions, videos, etc.), this resource is a wonderful complement to the APDVS eBook.

 Tip

Please see pages 85-98.

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## **Operative Footage**

Developed by the DeBakey Institute for Cardiovascular Education & Training at Houston Methodist. *YouTube account required as video content is age-restricted.* Please create and/or log in to your YouTube account to have access to the videos.

### **External Iliac Artery Balloon Angioplasty and Stenting**

#### **Common Femoral Endarterectomy**

#### **Left Femoral to Peroneal Bypass by Reversed Ipsilateral Great Saphenous Vein (Surgical or Extra-anatomic Bypass)**

#### **Left Common Femoral Artery to Anterior Tibial Artery Bypass using PTFE Graft (Surgical or Extra-anatomic Bypass)**

#### **Below Knee Amputation (BKA)**

#### **Above Knee Amputation (AKA)**



# **Venous Disease**

# Chronic Venous Insufficiency (CVI)

Ethan Romero, BA, Mark Basiliou, BS, Emily Goodman, MD, and Chelsea Dorsey, MD.

## Note

By the end of this chapter, students will:

- Understand chronic venous disease etiology, risk factors, and classification schemes.
- Accurately describe venous disease.
- Describe the venous anatomy of the lower limbs.
- Outline a diagnostic approach and treatment plan for a patient with chronic venous disease.
- Describe treatment options for venous insufficiency including conservative and invasive measures.

## Key Facts

1. Chronic venous disease (CVD) is highly prevalent in the United States, affecting 25% of women and 15% of men, with 1% to 4% progressing to advanced stages such as chronic venous insufficiency (CVI) with venous ulcers.
2. The economic burden of CVD is substantial, with estimated annual healthcare costs in the US ranging from \$1.9 to \$3 billion, primarily impacting Medicare.
3. CVD arises from venous hypertension due to valvular incompetence, thrombotic obstruction, or extrinsic compression, leading to manifestations such as edema, hyperpigmentation, and venous ulcers.
4. Superficial, deep, and perforating veins play crucial roles in venous circulation, with dysfunctional valves in the great saphenous vein commonly causing significant venous reflux.
5. Duplex ultrasound is the primary diagnostic tool for CVD, assessing venous obstruction and reflux. The Clinical-Etiology-Anatomy-Pathophysiology (CEAP) classification system categorizes disease severity based on clinical manifestations and pathophysiology.
6. The Venous Clinical Severity Score (VCSS) complements CEAP for evaluating dis-

ease progression and treatment outcomes.

7. Management of CVD includes conservative approaches (compression therapy), pharmacological interventions, and various procedures including endovenous ablation, phlebectomy, and sclerotherapy.
8. Endovenous treatments such as radiofrequency ablation (RFA) and endovenous laser ablation (EVLA) are preferred over traditional surgeries due to lower morbidity and quicker recovery times.
9. Surgical interventions significantly improve quality of life and symptom relief compared to conservative therapies for severe CVD.
10. Long-term surveillance with duplex ultrasound is essential to monitor disease progression and recurrence post-treatment, with early intervention shown to accelerate ulcer healing and reduce recurrence rates.

### How We Suggest Using the Pre/Post Questions

The pre/post questions are listed below. They are all multiple choice questions with a single right answer. To best guide your learning, we have hidden the answers in a collapsible menu. Before reading the chapter, we suggest giving the questions a try, noting your answers on a notepad. After reading the chapter, return to the questions, re-evaluate your answers, and then open the collapsible menu to read the correct answer and discussion. Do not fret if you have difficulty answering the questions before reading the chapter! By the end of the chapter, we are certain you will have covered the knowledge necessary to answer the questions. There will be a teaching case at the end of the chapter. This is another opportunity to exercise your new knowledge!

## Pre/Post Questions

### Case Based Questions

1. A 55-year-old male presents with aching legs, swelling, and skin discoloration. Physical examination reveals dilated veins and hyperpigmentation. *Which diagnostic modality is most appropriate initially?*
  - A. Magnetic resonance imaging (MRI)
  - B. Venous duplex ultrasound
  - C. Arterial Doppler ultrasound
  - D. Computed tomography (CT) scan

Answer

**B. Venous duplex ultrasound**

Discussion: Venous duplex ultrasound is the initial diagnostic study of choice for evaluating chronic venous disease. It helps detect venous obstruction or reflux, which are critical in diagnosing and planning management for patients with symptoms like aching legs, swelling, and skin changes indicative of venous insufficiency. MRI, arterial Doppler ultrasound, and CT scan are not typically used as initial diagnostic tests for chronic venous disease.

2. A 40-year-old female presents with varicose veins that are causing discomfort. She has no skin changes or ulcers. *What is the most appropriate initial treatment for her condition?*
- A. Endovenous laser ablation (EVLA)
  - B. Sclerotherapy
  - C. Conservative therapy with compression stockings
  - D. High ligation and stripping of the great saphenous vein

Answer

**C. Conservative therapy with compression stockings**

Discussion: Conservative therapy with compression stockings is the initial treatment for patients with symptomatic varicose veins without skin changes or ulcers. This approach aims to relieve symptoms and may be sufficient for managing early-stage disease. EVLA, sclerotherapy, and surgical interventions like high ligation and stripping are considered for more advanced cases or when conservative measures fail.

3. A patient with chronic venous insufficiency (CVI) presents with a venous ulcer that has failed to heal with conservative measures. *What is the next appropriate step in management?*
- A. Initiate high-dose oral antibiotics
  - B. Perform surgical ligation of perforator veins
  - C. Consider endovenous ablation of refluxing superficial veins
  - D. Apply topical corticosteroids

Answer

**C. Consider endovenous ablation of refluxing superficial veins.**

Discussion: For patients with chronic venous insufficiency (CVI) and non-healing venous ulcers, consideration should be given to endovenous ablation of refluxing superficial veins

to reduce venous hypertension and promote ulcer healing. High-dose antibiotics, surgical ligation of perforator veins, and topical corticosteroids are not primary treatments for CVI-related ulcers.

4. A patient with chronic venous disease (CVD) is evaluated using the CEAP classification system and scored as C4b. *What does this classification indicate??*
- A. Active venous ulcer
  - B. Skin pigmentation or eczema
  - C. Lipodermatosclerosis or atrophie blanche
  - D. Telangiectasias or reticular veins

Answer

**C. Lipodermatosclerosis or atrophie blanche.**

Discussion: The CEAP classification C4b indicates the presence of lipodermatosclerosis or atrophie blanche, which are manifestations of chronic venous disease characterized by changes in the skin and subcutaneous tissue. Options A, B, and D correspond to other classifications within the CEAP system.

5. A patient undergoes endovenous laser ablation (EVLA) for symptomatic varicose veins. *Post-procedure, what is a potential complication that should be monitored using duplex ultrasound?*
- A. Allergic reaction to the laser
  - B. Endothermal heat-induced thrombosis (EHIT)
  - C. Superficial wound infection
  - D. Hypotension from anesthesia

Answer

**B. Endothermal heat-induced thrombosis (EHIT).**

Discussion: Endothermal heat-induced thrombosis (EHIT) is a potential complication of endovenous laser ablation (EVLA), where heat generated by the laser can extend thrombus into the deep venous system. Monitoring with duplex ultrasound post-procedure helps detect EHIT early. Allergic reactions, superficial wound infections, and hypotension are less common complications and managed differently.

6. A patient with chronic venous disease (CVD) undergoes surgical ligation and stripping of the great saphenous vein (GSV). *What is a known advantage of this procedure compared to endovenous ablation techniques?*

- A. Lower risk of procedural complications
- B. Shorter recovery time
- C. Less risk of recurrence
- D. Suitable for patients with deep vein thrombosis (DVT)

Answer

**C. Less risk of recurrence.**

Discussion: Surgical ligation and stripping of the great saphenous vein (GSV) have been associated with lower recurrence rates compared to endovenous ablation techniques. While endovenous ablation offers advantages such as shorter recovery times and lower procedural complications, it may have slightly higher recurrence rates in some studies. Surgical ligation and stripping are not suitable for patients with active deep vein thrombosis (DVT) and are generally reserved for superficial reflux.

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## Introduction

Chronic venous disease (CVD) is one of the most common chronic diseases in the United States. The more common manifestations of venous disease are [telangiectasia](#), reticular veins, and [varicose veins](#). Chronic venous insufficiency (CVI) is a term reserved for advanced chronic venous disease from functional abnormalities of the venous system, commonly manifesting with hyperpigmentation, edema, [lipodermatosclerosis](#), and venous ulcers. Common risk factors for CVI include family history of venous disease, increasing age, smoking, obesity, prolonged standing or sitting, history of venous thrombosis, pregnancy, history of lower extremity trauma, and surgery.

Venous disease is increasing in prevalence largely due to the aging global population. In the United States, more than 25 million adults (25% of women and 15% of men) are affected by CVD with a disproportionately greater incidence and prevalence reported amongst women at any age [Azar, Rao, and Oropallo (2022a)](Y. Kim et al. 2021a). Approximately 1% - 4% of the adult population (2.6% in women and 1.9% in men) is affected by more advanced stages of the disease including chronic, non-healing venous ulcers (Sidawy and Perler 2023a).

Socioeconomic factors also play a role in disease incidence and progression; patients without health insurance are 2.6 times more likely to have venous ulcers compared to insured patients

(Scott et al. 1995a). Furthermore, disadvantaged populations have increased rates of recurrent ulceration as well as delayed healing times. This is explained in part by the high cost of treatment for patients with CVI, though delayed treatment of advanced disease ultimately results in a greater overall cost burden (Y. Kim et al. 2021a).

When CVI progresses to its advanced stages and wounds develop it can negatively impact a patient's quality of life. It also creates an economic burden to society; not only are the direct healthcare costs high, but the severe symptoms and wounds from CVI can affect one's ability to contribute to the workforce. A review from 2019 estimated the global market for the treatment of varicose veins alone will increase by approximately 35% in 2021 when compared to 2016, increasing from \$290.59 million to \$396 million (Davies 2019a). Meanwhile, the management of venous ulcers in the United States has an estimated annual healthcare cost between \$1.9 and \$3 billion, with the greatest cost burden on the Medicare system (Sidawy and Perler 2023a).

Venous disease represents a unique challenge to our healthcare system, negatively affecting the quality of life and overall health of a large portion of the population. Recognizing the early signs of CVD and implementing timely interventions can drastically improve patient outcomes. Therefore, it is of the utmost importance that physicians and healthcare providers in all specialties are familiar with venous disease, so that it can be recognized and treated in its earlier stages.

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## Etiology

The peripheral venous system uses valves within the veins to prevent retrograde flow. Skeletal muscle contractions serve to pump blood forward, allowing travel against gravity and return to the heart. Patent veins, functioning valves, and muscle pumps are necessary for the venous system to operate properly.

The [veins of the lower extremities](#) are classified as superficial (above the muscular fascia), deep (below the muscular fascia), and perforating veins (penetrating the muscular fascia). The [superficial venous system](#) drains the cutaneous microcirculation and consists of the great saphenous vein (GSV) and small saphenous vein (SSV), collectively known as "truncal veins", in addition to several other accessory veins. [Deep veins](#) follow the course of corresponding arteries and consist of the plantar, tibial, popliteal, femoral, deep femoral, and common femoral veins. Perforating veins cross the fascial layer, connecting superficial and deep veins. Throughout the deep and superficial veins are several one-way valves that help blood travel toward the heart while preventing it from returning to the feet (Meissner 2005a).

CVD results from functional abnormalities of the venous system that cause elevated venous pressure, also known as venous hypertension. Valvular incompetence, thrombotic obstruction,

or extrinsic compression can all impede blood return and cause venous hypertension. Dysfunctional calf muscle pumps can also exacerbate the problem. Persistent venous hypertension can result in insufficiency and subsequent inflammation leading to edema, hyperpigmentation of the skin, subcutaneous tissue fibrosis (known as lipodermatosclerosis), and eventually, ulceration.

Incompetence of the valves in the superficial venous system allows for **retrograde flow** of blood, which is called “reflux.” Failure of valves located at the junctions of the deep and superficial systems can be a source of reflux leading to CVI. For example, the great saphenous vein (GSV) drains into the femoral vein at the **saphenofemoral junction**; a valve lies at this junction in 94% to 100% of individuals, and this is the most common site for clinically significant reflux (Moore, Lawrence, and Oderich 2019b). Obstruction of the deep venous system also limits the outflow of blood resulting from an intrinsic process such as deep vein thrombosis (DVT) or due to extrinsic compression. Deep venous outflow obstruction significantly contributes to the pathogenesis of secondary CVD and has a more rapid progression of the disease with a high rate of the development of ulcers (Eberhardt and Raffetto 2014a).

*Differential Diagnosis for Leg Swelling With or Without Pain*

Category	Condition	Key Features	Comments
Vascular	Chronic Venous Insufficiency (CVI)	Edema, skin changes (hemosiderin staining, lipodermatosclerosis), varicose veins	Symptoms worsen with standing, relieved by elevation; aching may be present
	Deep Vein Thrombosis (DVT)	Unilateral swelling, pain, warmth, erythema	Requires urgent evaluation due to risk of pulmonary embolism
	Lymphedema	Non-pitting edema, often unilateral, thickened skin	Often painless, may have history of trauma, infection, or malignancy
	Superficial Thrombophlebitis	Painful, red, cord-like structure along vein	Less severe than DVT; commonly occurs in varicose veins
Cardiac	Congestive Heart Failure (CHF)	Bilateral pitting edema, worse in the evening, dyspnea	Often associated with other signs of heart failure (e.g., orthopnea, paroxysmal nocturnal dyspnea)



Category	Condition	Key Features	Comments
Renal	Nephrotic Syndrome	Bilateral pitting edema, frothy urine, facial swelling	May present with proteinuria, hypoalbuminemia
Hepatic	Cirrhosis	Bilateral edema, ascites, jaundice	Portal hypertension leads to edema; may have history of liver disease
Endocrine	Hypothyroidism	Bilateral non-pitting edema, dry skin, hair loss	Often with fatigue, cold intolerance, weight gain
Musculoskeletal	Chronic Exertional Compartment Syndrome	Pain and swelling with exercise, relieved by rest	Typically bilateral, associated with physical activity
	Infection (Cellulitis, Abscess)	Unilateral swelling, erythema, warmth, tenderness	Fever and localized signs of infection
	Trauma (Sprain, Fracture, Hematoma)	Swelling, pain, bruising, decreased range of motion	History of injury; localized swelling
Malignancy	Lymphoma or Other Cancers	Painless swelling, often unilateral, systemic symptoms	May have “B” symptoms (fever, night sweats, weight loss); consider metastasis in older adults
Medication-Induced	Calcium Channel Blockers (e.g., Amlodipine) NSAIDs, Steroids, etc.	Bilateral swelling, more prominent in lower legs Bilateral or unilateral swelling, typically without significant pain	Medication history important; often no associated pain Common side effect of various medications
Pregnancy-Related	Pre-eclampsia	Bilateral swelling, headache, visual changes, hypertension	Requires urgent evaluation; occurs after 20 weeks of pregnancy

Category	Condition	Key Features	Comments
	Normal Pregnancy	Mild bilateral swelling, worse at end of day	Common in late pregnancy due to increased blood volume and pressure on pelvic veins

## Diagnosics and Imaging

The diagnosis of CVD is suggested by the presence of typical symptoms (aching, throbbing, fatigue, heaviness) and physical examination findings (varicose veins, edema, skin changes, and ulceration). Duplex ultrasound is the initial diagnostic study to evaluate for CVD. It aims to detect the presence of venous obstruction or venous reflux. It is reproducible, noninvasive, and inexpensive; it has largely replaced venography as the initial diagnostic imaging modality for CVD.

Venous duplex ultrasound should be performed for patients with significant symptoms and exam findings suggestive of venous obstruction or reflux, lower extremity ulcers, history of DVT presenting with persistent lower extremity symptoms (swelling, heaviness, pain), and for patients where the cause of leg swelling is undetermined. Venous duplex ultrasound is also helpful for planning interventions. Understanding the distribution and severity of venous reflux is essential. Given CVD can present with variable symptoms and signs, a standardized clinical classification system for diagnosing CVD is critical to understanding the natural history of the disease and how to best treat it. The Clinical-Etiology-Anatomy-Pathophysiology (CEAP) classification system (Fig. 1) is used to determine the complexity of the clinical manifestations of CVD, providing a reliable and reproducible classification system to describe the patient's current status. While CVD encompasses the full spectrum of abnormalities of the venous system, the designation of CVI is reserved for advanced venous disease-producing edema, skin changes, or venous ulcers (C3-C6) [Eklof et al. (2009a)](Lurie et al. 2020a).

Clinical Signs (C)	Etiology (E)	Anatomy (A)	Pathophysiology (P)
C0: No visible signs of venous disease	Ec: Congenital	As: Superficial	Pr: Reflux
C1: Telangiectasias or reticular veins	Ep: Primary	Ad: Deep	Po: Obstruction
C2: Varicose veins	Es: Secondary	Ap: Perforator	Pr,o: Reflux and Obstruction

Clinical Signs (C)	Etiology (E)	Anatomy (A)	Pathophysiology (P)
C3: Edema	En: No venous cause identified	An: No venous location identified	Pn: No pathophysiologic dysfunction identified
C4: Changes in skin and subcutaneous tissue secondary to CVD			
C4a: Pigmentation or eczema			
C4b: Lipodermatosclerosis or atrophie blanche			
C4c: Corona phlebectatica			
C5: Healed venous ulcer			
C6: Active venous ulcer			
C6r: Recurrent active venous ulcer			

Fig. 1: *CEAP Classification of Venous Disorders*

While the CEAP classification system is the global standard for describing clinical features of CVD, it is not sensitive to changes in disease over time and thus is a poor prognosticator of progression and treatment outcomes. In response, the Venous Clinical Severity Score (VCSS) (Fig. 2) was formulated as a complementary evaluative instrument based on ten categories graded on a scale of 0 to 3 for a final total score between 0 and 30 (Vasquez et al. 2010a).

Descriptor	Absent (0)	Mild (1)	Moderate (2)	Severe (3)
Pain	None	Occasional	Daily not limiting	Daily limiting
Varicose veins	None	Few	Calf or thigh	Calf and thigh
Venous edema	None	Foot and ankle	Below knee	Knee and above
Skin pigmentation	None or focal	Limited perimalleolar	Diffuse lower 1/3 calf	Wider above lower 1/3 calf
Inflammation	None	Limited perimalleolar	Diffuse lower 1/3 calf	Wider above lower 1/3 calf

Descriptor	Absent (0)	Mild (1)	Moderate (2)	Severe (3)
Induration	None	Limited perimalleolar	Diffuse lower 1/3 calf	Wider above lower 1/3 calf
No. active ulcer	None	1	2	3
Ulcer duration	None	< 3 mo	3-12 mo	> 1 y
Ulcer diameter	None	< 2 cm	2-6 cm	> 6 cm
Compression therapy	Not used	Intermittent	Most days	Full compliance

**Fig. 2: Revised Venous Clinical Severity Score (VCSS)**

## Treatment

Management of CVD requires an individualized approach which may include conservative, pharmacological, and/or interventional therapies. Surgical intervention for CVI focuses on treating refluxing axial veins and correcting the underlying chronic venous hypertension. Management decisions (Fig. 3) are guided according to a thorough evaluation of CEAP classification (e.g., varicose veins or CVI), source of reflux (superficial, deep, and/or perforator), vessel anatomy, and baseline candidacy for surgical intervention (Azar, Rao, and Oropallo 2022a).

CEAP classification	Recommended therapy
Asymptomatic C1	Conservative
Symptomatic C1	Conservative and/or sclerotherapy
Symptomatic C2-C6 with reflux	Superficial venous ablation, phlebectomy, or vein stripping and/or ligation

*Fig. 3: Therapy recommendations based on CEAP classification score*

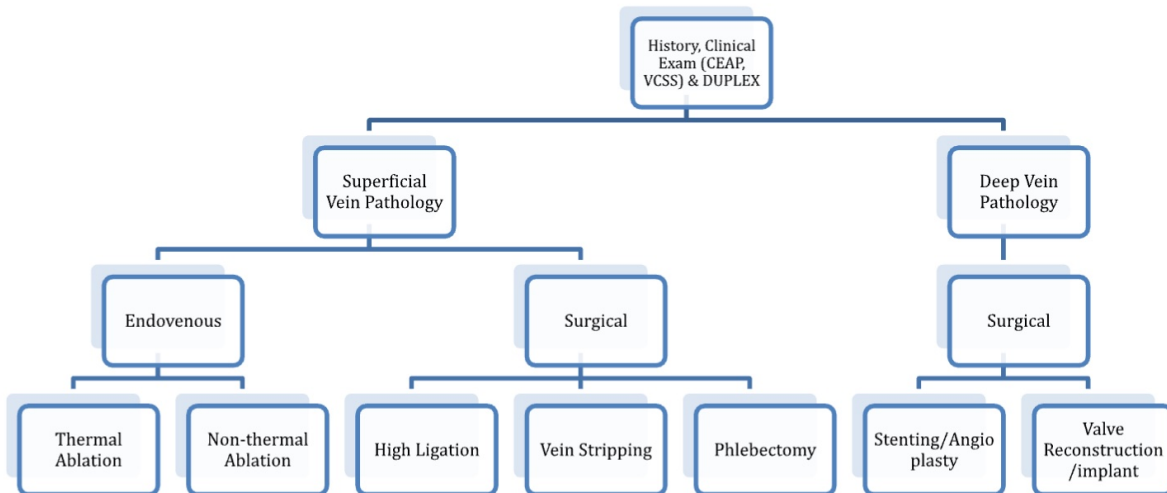
## Conservative Management

Initial therapy for the majority of patients with CVD is conservative treatment with compression and leg elevation. Compression therapy includes elastic stockings or wraps or pneumatic compression devices. Typically, 20-30 mmHg compression is used to treat symptomatic varicose veins, although higher levels can be used in patients with more advanced disease. Graduated compression stockings oppose tissue expansion during muscle contraction, narrowing the superficial veins' diameters, subsequently decreasing venous reflux and venous hypertension, which are two key elements in the pathophysiology of CVD (Gloviczki et al. 2024a).

Although compression stockings provide symptomatic relief for patients with varicose veins, a systematic review from 2009 found there is no evidence for prevention of recurrence or cessation of disease progression (Palfreyman and Michaels 2009a). The Society for Vascular Surgery, the American Venous Forum, and the American Vein and Lymphatic 2023 clinical practice guidelines for the management of varicose veins of the lower extremities recommends against a 3 month trial of compression therapy prior to intervention for patients with symptomatic varicose veins who are candidates for surgical intervention (Gloviczki et al. 2024a).

Despite the lack of evidence supporting the long term efficacy of compression therapy, insurance companies and the Centers for Medicare and Medicaid Services frequently require a 3 month trial of compression stockings before surgical intervention for C2 disease (Gloviczki et al. 2024a).

## Endovenous & Surgical Interventions



While conservative treatment is often offered as an initial step, it does not address the underlying pathology. Patients with symptomatic superficial reflux who are candidates for endovenous or surgical intervention should be offered early corrective therapy (Azar, Rao, and Oropallo 2022a). Surgical treatment of varicose veins or CVI due to superficial venous incompetence is accomplished by removing, ablating, or ligating the venous segment with reflux. Current options include but are not limited to the following: endovenous thermal ablation, chemical ablation, high ligation, ligation and stripping, phlebectomy, and sclerotherapy (Figs. 4, 5). The majority of these procedures are performed in a minimally invasive fashion under local anesthesia with or without sedation. For more invasive surgical interventions, general anesthesia may be utilized.

Endovenous Intervention	Vessel(s) Treated
Thermal ablation (RFA, EVLA)	Saphenous vein trunks, perforator veins
Chemical ablation (foam sclerosants)	Saphenous vein trunks/tributaries, perforator veins, varicose veins not connected to saphenous vein trunks
Mechanochemical ablation (MOCA) Adhesive closure	Saphenous vein trunks
Visual/Surface sclerotherapy	Varicose veins, reticular veins, telangiectasias
Angioplasty/stenting	Deep veins

Fig. 5: *Vessel-specific endovenous treatment modalities*

### Endovenous Ablation

Endovenous ablation is a minimally invasive procedure recommended for patients with ongoing symptoms of CVD with superficial venous reflux (Sidawy and Perler 2023b). Ablation is performed in an outpatient setting under local anesthesia using a variety of techniques including thermal, chemical, or mechanical methods to occlude the vessel and prevent retrograde flow. By reducing venous flow volumes, endovenous ablation treats underlying venous hypertension, thereby reducing the risk of harm to the cutaneous tissues which may result in edema, hyperpigmentation, lipodermatosclerosis, and ulceration (Hager et al. 2016a).

Endovenous treatments for varicose veins emerged as an alternative to open surgery and are now the standard of care due to improved safety and efficacy (Rautio et al. 2002a). The most widespread technologies used for the treatment of truncal vein reflux are radiofrequency ablation (RFA) and endovenous laser ablation (EVLA). Both techniques utilize [heat to induce closure](#) of the dysfunctional veins. RFA works by delivering radiofrequency energy, heating the vein wall to cause destruction of the endothelium, contraction of vein wall collagen, thrombus formation, and eventual fibrosis of the vein resulting in a durable ablation (Weiss, Feied, and Weiss 2001a). The radiofrequency ablation (RFA) catheter should be placed 2 cm from the saphenofemoral junction (SFJ) so as to prevent endothermal heat-induced thrombosis ([EHIT](#)). Similarly, EVLA delivers laser energy to an area, resulting ultimately in fibrosis and occlusion of the target vein (Perrin et al., n.d.a).

Non-thermal non-tumescent (NTNT) ablation methods offer alternatives to heat-based vein treatments, employing techniques such as mechanochemical ablation (MOCA), cyanoacrylate adhesive, and sclerosant foams. MOCA combines mechanical damage with the direct injection of a sclerosant to close the vein, enhancing the effectiveness of the chemical agent (Boersma et al. 2014a). Cyanoacrylate adhesive, injected directly into the vein, promotes vessel closure through inflammation and subsequent fibrotic occlusion. For larger vessels, chemical ablation via ultrasound-guided delivery of foam sclerosants like polidocanol and sodium tetradecyl sulfate (STS) is preferred (Bootun et al. 2017a).

## High Ligation & Stripping

Though minimally invasive endovenous methods are associated with reduced convalescence, recurrence, and morbidity compared to open surgery, great saphenous vein (GSV) ligation with or without stripping may be chosen for patients with reflux and varicose veins in the GSV distribution where ablation is not an option (Gloviczki et al. 2024a). High ligation of the GSV is accomplished via an oblique incision below the groin crease, centered over the SFJ which can be located and marked using duplex ultrasound. The GSV and its six main tributaries near its termination are circumferentially dissected and ligated. It is crucial to perform ligation of the GSV close to the femoral vein without impinging on and thus narrowing the femoral vein. Care must also be taken to not leave a long stump proximally because this increases the risk of venous thromboembolism.

Recurrence rates are reduced when GSV stripping is performed in addition to high ligation (HL&S) versus high ligation alone (Sidawy and Perler 2023b). After the GSV is ligated at the SFJ, a vein stripper is passed distally and a second incision is made near the knee. The GSV is then tied to the stripper and both the stripper and the vein are drawn out through the distal incision, stripping in a downward direction, avulsing its tributaries and removing it from circulation. Top-down passage of the stripper minimizes the potential for saphenous nerve injury as well as unintentional passage of the stripper into the femoral vein through a thigh perforator (Sidawy and Perler 2023b).

## Stab Phlebectomy

Stab phlebectomy, also known as ambulatory phlebectomy, is a technique used to remove residual superficial varicosities following endovenous ablation or high ligation and stripping (HLS) of the great saphenous vein (GSV) or small saphenous vein (SSV). These residual varicosities, if not addressed, can continue to drain via alternative pathways, remaining both symptomatic and cosmetically displeasing. The procedure involves making small 1-3 mm incisions through which [varicose veins are hooked, extracted, and removed](#). This method has largely replaced older techniques that left larger scars, as these smaller incisions typically heal with minimal scarring and do not usually require sutures. The phlebectomy not only improves aesthetic outcomes but also helps alleviate symptoms by removing venous pressure from untreated tributaries (Sidawy and Perler 2023b).

## Sclerotherapy

[Injection sclerotherapy](#) is a minimally invasive percutaneous technique that uses chemical irritants to close unwanted superficial veins. Primarily employed in the treatment of telangiectasias, reticular veins, and small nonaxial varicose veins less than 6 mm in diameter, sclerotherapy can address both symptomatic and asymptomatic veins. This method involves injecting

a sclerosant that damages the endothelial lining of the vein, leading to thrombosis and subsequent fibrosis, effectively eliminating the vein. While liquid sclerotherapy is typically used for smaller veins, larger varicosities may require foam sclerotherapy, which uses a foam-like consistency to enhance contact with the vein wall [Azar, Rao, and Oropallo (2022a)](Sidawy and Perler 2023b).

### **Interventions for Deep Venous Insufficiency: Stenting, Venoplasty, and Valvular Reconstruction**

Due to increased risks and technical challenges associated with the treatment of deep venous reflux, treatment is typically reserved for patients with ulcer recurrence refractory to therapies targeting superficial/perforator insufficiency (Sidawy and Perler 2023b). CEAP C4-C6 patients without superficial truncal reflux or those whose superficial treatment has failed may be offered stenting to correct deep venous obstruction.

Iliac and inferior vena cava (IVC) stenting remains somewhat controversial and is typically reserved for a specific subset of patients who are most likely to benefit from the procedure. These patients often include those with significant venous outflow obstruction, such as those suffering from May-Thurner syndrome, chronic venous insufficiency, or post-thrombotic syndrome that has not responded adequately to conservative treatments. It is now recognized that asymptomatic venous obstruction is far more common than previously thought, and there remains debate about which patients are suitable candidates for ilio caval stenting. In appropriately selected patients, ilio caval stenting can provide symptomatic relief with long-term patency rates >70% and relatively low DVT risk (Hwang et al. 2020a).

Patients with deep venous reflux due to congenital, primary, or secondary (post-thrombotic syndrome following DVT) valvular incompetence may be candidates for valvular reconstruction if symptoms recur following attempted conservative and/or endovenous therapies for superficial reflux. These patients usually have class C4b to C6 disease or disabling signs and symptoms such as severe edema (C3) (Sidawy and Perler 2023b). Treatment options focus on either correcting the anatomical defect of the incompetent valve via [internal valvuloplasty](#)/[external valvuloplasty](#), or by replacing the architecture via [valve transposition](#) or [vein transplantation](#) (Maleti and Perrin 2011a). Most recently, promising results from the [SAVVE trial](#) studying the use of a novel synthetic neo valve (VenoValve) represent an additional treatment modality for chronic venous insufficiency.

::: callout-tip

Video Tutorial: Diagnosis and Management of Deep Venous Incompetence with Dr. Ulises Baltazar



## Outcomes & Surveillance

### Compression Therapy vs. Surgical Intervention

- For patients with severe varicose veins (C2), surgical treatment (HL&S, phlebectomy) was associated with significant improvements to QoL, symptom relief, and patient satisfaction as compared to conservative treatment (lifestyle modification, leg elevation, and/or compression stockings) (Michaels et al. 2006a).
- In a randomized trial comparing compression to surgical stripping of the GSV in patients with C2-C3 venous disease, VCSS decreased from 4.6 to 3.5 in the compression group ( $P < 0.01$ ) and from 4.8 to 0.6 in the surgery group ( $P < 0.001$ ) (Sell et al. 2014a). Therefore, the Society for Vascular Surgery recommends against a three-month trial of conservative therapy prior to intervention for patients with C2 and C3 disease (Gloviczki et al. 2024a).
- In the landmark [ESCHAR trial](#), patients with C5-C6 disease randomized to HL&S and compression as compared to compression alone were found to have no significant differences in ulcer healing time (89% for the compression group and 93% for the compression plus surgery group at three years) but significantly lower rates of ulcer recurrence (56% for the compression group and 31% for the compression plus surgery group at 4 years,  $P < 0.01$ ).

### Endovenous Treatment of Saphenous Reflux

- Both EVLA and RFA are considered first-line therapies for the treatment of symptomatic saphenous reflux and are preferred over open surgery due to decreased postoperative morbidity and reduced convalescence time [Gloviczki et al. (2024a)](Sidawy and Perler 2023b).
- A meta-analysis of 64 studies comparing RFA, EVLA, foam therapy, or surgery for the treatment of truncal veins demonstrated higher success rates for RFA and EVLA (84% and 94%) as compared to surgery and foam sclerotherapy (78% and 77%) after 3 years (Nijsten et al. 2009).
- Patients with varicosities, enlarged reticular veins, or telangiectasias in addition to saphenous reflux benefit from simultaneous treatment with EVLA/RFA and ambulatory phlebectomy as opposed to staged treatment with improvement in early disease severity and QoL scores (Aherne et al. 2020a).
- Duplex ultrasound follow-up examination is recommended between 3-7 days following the procedure to assess for successful ablation as well as [endothelial heat-induced thrombosis](#) (extension of proximal thrombus into deep system) (Sidawy and Perler 2023b). EHIT may be treated with anticoagulants depending on the degree of extension (Sidawy and Perler 2023b).

## Disease Progression & Recurrence

- The [EVRA trial](#) demonstrates that patients who receive early endovenous ablation of superficial venous reflux with compression had shorter ulcer healing times (56 vs 82 days) as opposed to patients with compression therapy alone and deferred intervention (6 months) (Gohel et al. 2018a).
  - Patients receiving open or endovenous treatment of C2-C6 disease experience varicose vein recurrence rates of up to 35% at two-year follow-up and 65% at 11 years follow up (Wittens et al. 2015a). The majority of recurrent varicose veins are due to disease progression (development of venous reflux).
  - Long-term surveillance typically includes venous duplex ultrasound imaging, hemodynamic assessment, and reclassification by the CEAP/VCSS criteria.
- 

## Teaching Case

### Scenario

Mr. C.V.D is a 62-year-old male presenting to the vascular clinic with complaints of bilateral leg swelling, pain, and skin changes over the past year. He describes aching pain that worsens with prolonged standing and improves with leg elevation. He denies any recent trauma or surgery to his lower extremities. He reports a history of occasional night cramps in his calves and states that his symptoms have progressively worsened despite using over-the-counter pain relievers intermittently.

**PMHx:** Significant for hypertension, hyperlipidemia, and type 2 diabetes mellitus. He underwent coronary artery bypass grafting (CABG) surgery 7 years ago and takes aspirin 81 mg daily for cardiovascular protection. His diabetes is managed with metformin 1000 mg twice daily.

**PSHx:** He is a retired school teacher and leads a sedentary lifestyle. He does not smoke and consumes alcohol occasionally. He lives with his wife who helps him manage his medications and activities of daily living.

**FHx:** There is a family history of venous thromboembolism (VTE) on his father's side, but no history of chronic venous disease.

## Exam

Vitals: Blood pressure 138/82 mmHg, heart rate 72 bpm, respiratory rate 16 breaths/min, temperature 98.6°F (37°C).

**General:** Appears well-nourished and in no acute distress.

**Cardiac:** Regular rate and rhythm (RRR), clear S1 and S2, no murmurs.

**Pulmonary:** Clear to auscultation bilaterally, no increased work of breathing.

**Abdominal:** Soft, non-distended (ND), non-tender (NT), no palpable masses.

### Vascular/Extremities:

- Inspection: Bilateral pitting edema extending from mid-calf to ankles. Skin changes noted with hyperpigmentation, hemosiderin deposits, and mild venous eczema.
- Palpation: Mild tenderness over the posterior medial aspect of the left calf. No warmth or cords appreciated.
- Peripheral pulses: Palpable dorsalis pedis and posterior tibial pulses bilaterally.
- Neurological: Intact sensation and 2+ strength throughout lower extremities.

### Laboratory Findings:

- Complete Blood Count (CBC): Within normal limits.
- Comprehensive Metabolic Panel (CMP): Normal renal and hepatic function.
- Hemoglobin A1c: 6.7% (consistent with his known diabetes).
- D-dimer: Elevated at 600 ng/mL (normal < 500 ng/mL), suggesting ongoing low-grade thrombotic activity or venous insufficiency.

## Imaging

### Bilateral Venous Duplex Ultrasound

Impression: - Findings reveal bilateral superficial venous reflux in the great saphenous veins (GSVs) with retrograde flow into the femoral vein. - Deep venous system is patent with no evidence of acute or chronic deep vein thrombosis (DVT). - Perforator veins are dilated and incompetent bilaterally.

## Discussion Points

**i** N.B. There is no prepared answers for the questions below.

However, we feel this chapter contains all the necessary information to answer the questions. If not, please let us know!

1. Based on the patient's history and physical examination findings, what are the key diagnostic criteria for chronic venous disease (CVD) in this case?
  2. Describe the pathophysiology of chronic venous insufficiency (CVI) and how it leads to the clinical manifestations observed in this patient.
  3. What are the advantages of using the CEAP classification system in clinical practice? How does it help in categorizing and managing patients with chronic venous disease?
  4. Discuss the role of duplex ultrasound in the diagnosis and management of chronic venous disease. What information does it provide that is crucial for treatment planning in this case?
  5. What conservative measures would you recommend for this patient to manage symptoms and prevent disease progression? How would you tailor these recommendations based on the severity of their symptoms?
  6. Explain the rationale behind using compression therapy in the management of chronic venous disease. What are the different types of compression therapies available, and how would you decide the appropriate regimen for this patient?
  7. What are the indications for considering pharmacological interventions in patients with chronic venous disease? Which specific medications might be considered, and how do they work to improve symptoms?
  8. Discuss the potential complications associated with chronic venous disease, particularly in this patient with advanced symptoms. How would you monitor and manage these complications to optimize long-term outcomes?
  9. Describe the principles and techniques involved in endovenous ablation procedures for treating superficial venous reflux. How do these procedures compare to traditional surgical approaches?
  10. What are the key elements of a comprehensive management plan for this patient, considering both short-term symptom relief and long-term prevention of complications?
-

## Key Articles

### KEY ARTICLES

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## Additional Resources

### Audible Bleeding Content

- Audible Bleeding Exam Prep: [Venous Disease](#)- - Rouleaux Club Exam Prep - [Venous Disease](#)



## Websites

- TeachMe Surgery: [Varicose Veins](#)
  - TeachMe Surgery: [Deep Venous Insufficiency](#)
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## Gore Combat Manual

The [Gore Medical Vascular and Endovascular Surgery Combat Manual](#) is an informative and entertaining read intended as a vascular surgery crash course for medical students, residents, and fellows alike. Highly accessible with a thoughtfully determined level of detail, but lacking in learning activities (e.g. questions, videos, etc.), this resource is a wonderful complement to the APDVS eBook.

### Tip

Please see pages 165-175.

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### Tip

- Video Tutorial: Diagnosis and Management of Varicose Veins with Dr. Mitul Patel
  - Video Tutorial: Diagnosis and Management of Varicose Veins with Dr. Ruth Bush
- 

## Operative Footage

Developed by the DeBakey Institute for Cardiovascular Education & Training at Houston Methodist. *YouTube account required as video content is age-restricted.* Please create and/or log in to your YouTube account to have access to the videos.

**Endovenous Laser Ablation (EVLA/EVLT) of the Greater Saphenous Vein**

**Mechanochemical Endovenous Ablation (MOCA) of the Greater Saphenous Vein**

**Cyanoacrylate Adhesive (VenaSeal) Treatment of Greater Saphenous Vein**

# Miscellaneous Topics

# Basics of Endovascular Surgery (Endo Basics)

Jeremy Zack, Nitin Jethmalani, MD, Babak Abai, MD, FACS.

## Note

By the end of this chapter, students will:

- Be able to define endovascular surgery.
- Be able to elaborate on the key principles of arterial access, wires, catheters, and sheaths.
- Be able to outline the steps and associated risks of common endovascular surgeries.
- Be able to outline new developments in endovascular surgery.

## Key Facts

1. Endovascular surgery is a minimally invasive approach to treat a variety of vascular pathologies, including peripheral artery disease, aortic aneurysms, venous diseases, and others by deploying balloons, stents, and or endografts.
2. The common femoral artery (CFA) is the most common site of access for endovascular surgery due to its larger size and lower risk of complications relative to other sites of vascular access
3. Using the analogy of a train on a track passing through a tunnel, a catheter can be thought of as the train, a wire as the train track, and a sheath as the surrounding tunnel.
4. Wires are used to guide devices such as catheters and sheaths into a vessel.
5. Catheters are flexible, hollow tubes that are used in both the diagnosis and treatment of vascular disease. In an analogy of a train track, the catheter can be thought of as the train, the track is the wire, and the surrounding tunnel is the sheath.
6. Catheters are measured in French (1F = 0.33 mm) and the diameter corresponds to the outer diameter of the tube.
7. Sheaths are hollow tubes that provide conduit between outside the body and into the vessel, the “tunnel” in the train track analogy. Sheaths are used to advance

- and exchange catheters, wires, stents, balloons, and other devices.
8. Sheaths are measured in French (1F = 0.33 mm) and the diameter corresponds to the inner diameter of the tube.
  9. **A 6F catheter will pass through a 6F sheath.**
  10. Endovascular surgery is not without risks and complications. Complications include the formation of hematoma, pseudoaneurysm, retroperitoneal hematoma, fistula, dissection, thrombosis and embolism, and atheroembolism. Other complications include wire perforation vessel rupture.

### How We Suggest Using the Pre/Post Questions

The pre/post questions are listed below. They are all multiple choice questions with a single right answer. To best guide your learning, we have hidden the answers in a collapsible menu. Before reading the chapter, we suggest giving the questions a try, noting your answers on a notepad. After reading the chapter, return to the questions, re-evaluate your answers, and then open the collapsible menu to read the correct answer and discussion. Do not fret if you have difficulty answering the questions before reading the chapter! By the end of the chapter, we are certain you will have covered the knowledge necessary to answer the questions. There will be a teaching case at the end of the chapter. This is another opportunity to exercise your new knowledge!

## Pre/Post Questions

### Case Based Questions

1. A 77-year-old female with a past medical history of CAD, CHF with an EF of 14%, COPD, and a prior total thyroidectomy comes in for evaluation of carotid stenosis. She had a TIA with a right-sided facial droop two weeks ago and was briefly admitted to this hospital. She had a carotid US and was found to have an 85% left carotid stenosis. *Which of the following treatment modalities would be best suited for the patient?*
  - A. Open carotid endarterectomy
  - B. Transcarotid Artery Revascularization (TCAR)
  - C. No intervention because of her age
  - D. No intervention because of her medical comorbidities

Answer

**B. Transcarotid Artery Revascularization (TCAR)**

Discussion: Considering the patient has a symptomatic carotid artery lesion, her disease warrants treatment. This rules out answers C and D. TCAR is a safe, minimally invasive surgical technique used to treat carotid artery disease. TCAR and other endovascular procedures are often a safer option in the elderly, medically comorbid patients, especially those who have a higher risk of complications (such as those with prior neck surgery). In addition, TCAR can be performed under local anesthesia with sedation. This makes TCAR a safer option relative to open CEA, making B a better answer than C.

2. A 66-year-old male presents to the emergency room one week after a percutaneous coronary intervention via right groin access. He reports that over the last few days, he has noticed a bulge in his groin and when he touches it, it is painful and has a pulsatile nature. A complete exam reveals a pulsatile, tender mass in the right groin with no skin changes, but with a bruit on auscultation. *Which of the following complications is NOT associated with this patient's diagnosis?*
- A. Embolization to the lower extremity
  - B. Rupture of the mass
  - C. True femoral artery aneurysm
  - D. Compression of the femoral vein
  - E. Gangrene of the toes

Answer

**C. True femoral artery aneurysm**

Discussion: In the post-intervention patient who presents with a pulsatile tender mass at the intervention site with a bruit on auscultation, the most likely diagnosis is a femoral artery pseudoaneurysm. Risks of an untreated PSA include embolic events, distal ischemia including gangrene, rupture of the PSA, skin necrosis, and compression of surrounding structures including the femoral vein and nerve. Because a PSA is an outpouching of only 1 or 2 of the layers of the arterial wall, a true aneurysm (outpouching of all three layers of the artery, intima, media, and adventitia) is not associated with a femoral PSA.

3. A 42-year-old patient with a history of an arterial thrombus four years ago presents to the ER with severe right lower extremity pain. A CTA is obtained showing no atherosclerotic disease but with acute thrombus along the distal SFA and popliteal artery. You take the patient to the operating room for a lower extremity angiogram. *Which catheter would be most useful to help break up the clot over time?*

- A. Penumbra thrombectomy catheter
- B. Cragg Mcnamara Catheter
- C. Fogarty Catheter
- D. Peripherally inserted central venous catheter

Answer

**B. Cragg Mcnamara Catheter**

Discussion: In this case, the patient has an acute thrombus in her lower extremity arteries. Although there are many treatment options for this disease process depending on disease burden and characteristics, the question specifically asks about a catheter that can be used to break up the clot over time. The Cragg Macnamara catheter is a thrombolysis catheter that remains in place infusing “clot buster” medication (tPA) over a period of hours to days to dissolve the clot. A Penumbra thrombectomy catheter is used to suction blood clots out of a vessel. A Fogarty catheter can be used to manually withdraw thrombus from a vessel. A PICC line is used for long-term infusions to deliver medications to the central venous system and is not used intra-operatively for procedures.

4. A 70-year-old male is brought to the operating room for an EVAR procedure for his 6 cm infrarenal AAA. You have chosen a graft to use and the nurse is asking if there are any wires that you would like opened to deliver the device into the patient’s aorta. *Which of the following wires would be most useful for this task?*
- A. 0.018” flexible Terumo wire
  - B. 0.035” Amplatz wire
  - C. 0.035” floppy Glidewire
  - D. 0.014” Pilot wire

Answer

**B. 0.035” Amplatz wire**

Discussion: Vascular access wires each have a variety of uses determined by their stiffness, diameter, hydrophilicity, and tip shape. In this case, a stiff wire to pass a large aortic endograft over would be the most useful. The larger diameter wire will be stiffer, which rules out options “A” and “D”, which would both be used for getting access to smaller vessels. Option C is a floppy glide wire which can be useful for crossing lesions and getting up and over the aortic bifurcation. The 0.035” Amplatz wire is a stiff guidewire that will not buckle under the pressure of passing a large device into the aorta.

5. A 63-year-old smoker presents to the clinic with left lower extremity claudication and an ischemic ulcer. He is taken to the operating room for an angiogram via right groin access. *Which of the following steps is INCORRECT when obtaining arterial access?*
- A. Using anatomic landmarks to confirm cannulation through the inguinal ligament
  - B. Using ultrasound to ensure cannulation of the common femoral artery
  - C. Using fluoroscopy to ensure puncture at the level of the femoral head
  - D. Using the Seldinger technique for first access, making sure not to lose access to the artery during exchanges or wires and catheters.

Answer

**A. Using anatomic landmarks to confirm cannulation through the inguinal ligament**

Discussion: On first access for an angiogram with right groin access, the right common femoral artery should be accessed. The CFA begins below the level of the inguinal ligament, making answer A incorrect. One should avoid going through the inguinal ligament as native fascial planes are disrupted and it can make for challenging upsizing of devices and closure. Following identification of the inguinal ligament; ultrasound, fluoroscopy, and the Seldinger technique should be used as above to safely cannulate the CFA.

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## Introduction

Endovascular surgery is a minimally invasive approach to treat a variety of vascular pathologies including peripheral artery disease, aortic aneurysms, venous diseases, and more. Endovascular surgery is not limited to vascular surgery as a specialty and is also employed by interventional cardiologists, neurosurgeons, interventional radiologists, and others.

Over the past 30 years, endovascular surgery has dramatically changed the practice of vascular surgeons, decreasing patient morbidity and mortality in the immediate postoperative period and the length of hospital stay.

Understanding endovascular surgery as a medical student or junior resident can be a difficult task due to the abundance of wires, catheters, sheaths, and devices used and orienting oneself using fluoroscopic images. In this chapter, we will focus on developing a conceptual basis for endovascular surgery.

## Relevant Anatomy and Vascular Access

The common femoral artery (CFA) is the most common site of access for endovascular surgery due to its larger size and lower risk of complications relative to other sites of vascular access. Other sites of arterial access include the radial, brachial, and axillary artery in the upper extremity, as well as the popliteal and dorsal pedal arteries in the leg. The further away the vessel is from the aorta, the smaller the vessel lumen, and hence the smaller the diameter of devices that can be utilized in that artery.

The CFA is referred to as the external iliac artery proximal to the inguinal ligament and the superficial femoral artery (SFA) distal to the branching of the profunda femoris artery (PFA). The anatomic relationship of the CFA to the surrounding vessels can be remembered with the mnemonic “NAVeL”, femoral nerve, artery, vein, and lymph nodes from lateral to medial. The Femoral Triangle consists of these three vessels and nerves surrounded by the inguinal ligament superiorly, sartorius muscle laterally, and the adductor longus medially.

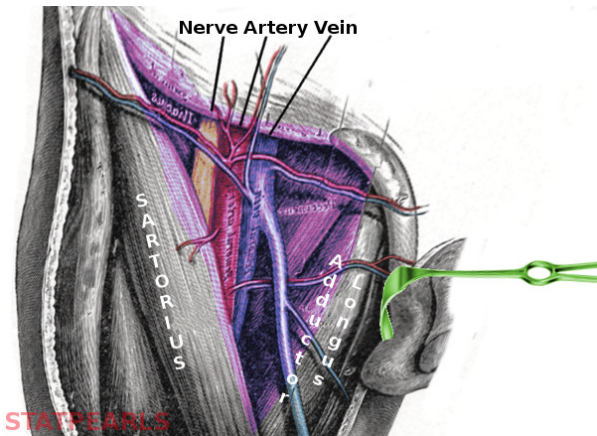


Figure 1: Femoral triangle anatomy courtesy of S Bhimji, MD.

Correct anatomic positioning for cannulation of the CFA can be achieved by palpating the point of maximum pulsation inferior to the inguinal ligament, a structure identifiable by tracing along the facial plane between the anterior superior iliac spine and pubic symphysis. The following imaging modalities assist with anatomic positioning:

- **Fluoroscopic guidance:** cannulation of the CFA should be attempted at the level of the middle to inferior femoral head.
- **Ultrasound (US) guidance:** identification of the PFA ensures cannulation is attempted of the CFA in place of the SFA. At the level where the great saphenous vein joins the femoral vein, US will show one large vessel surrounded by two smaller vessels, which can be remembered as the “Mickey Mouse Sign.”



 Tip

An in depth review of lower limb anatomy can be found in the APDVS Claudication Chapter or [here](#).

## Ultrasound Guided Access

There is emerging evidence to suggest real time identification of anatomic structures with US guidance may decrease the risk of access-related complications. A summary to the steps of US-guided access of the CFA are included below.

### Identifying the correct vessel:

1. Obtain baseline peripheral pulses.
2. Obtain a **micropuncture kit**, containing a hollow needle, guidewire, and catheter
3. Using the US probe, identify any plaque or calcification along the CFA or iliac artery that may change the site of access.
4. Identify the CFA, which can be differentiated from the femoral vein by its thick wall, resistance to compression, pulsatility, and relative lateral anatomic positioning. A video identifying the CFA through the “Mickey Mouse Sign” on US is available [here](#).

### Obtaining access through the Seldinger technique:

The Seldinger technique help obtain **safe** access to blood vessels. In contrast to previous methods, the Seldinger technique causes minimal damage to the blood vessel thereby allowing the puncture to be closed percutaneously, and avoids the risk of loss of access during the procedure through keeping at least one wire, catheter, or sheath at the site of interest in the body all times.

Steps to the Seldinger technique are as follows:

1. Cannulate the CFA by inserting the hollow needle at a 45-90 degree angle to the skin under US guidance, blood will flow out of the needle once inserted into the vessel.
2. Insert the guidewire into the vessel. Remove the hollow needle while keeping the guidewire in place, being careful not to allow the wire to withdraw as the needle is removed.
3. Insert the catheter over the guidewire.
4. Exchange the guidewire that came in the micropuncture kit with desired guidewire to begin the procedure.

 Tip

Please see [this video animation](#) of the Seldinger technique for vascular access.

## Closure and Closure Devices

Once the procedure is complete, percutaneous access will need to be closed. If relatively small catheters and sheaths were used during the procedure, generally below 6 French (1F = .33 millimeter), arterial access may be closed with manual compression by applying pressure with two fingers over the site of incision against the femoral head. Alternatively, certain indications, such as use of anticoagulation or large body habitus, may require closure devices even if larger catheters or sheaths were not used during the procedure.

There are several different types of closure devices. Each type can be differentiated by the use of suture or sealant to close the vessel.

A common closure device you are likely to encounter on a vascular surgery service is the **Perclose ProGlide** (Abbott Vascular, Chicago, IL), a suture-mediated closure device. Other closure devices include the **Angioseal** (Terumo, Tokyo, Japan), which deploys a bioabsorbable anchor to the arterial puncture site with surrounding collagen to promote closure, and the **MYNXGRIP** (Cordis, Miami Lakes, FL), which uses a balloon pressed against the site of arterial puncture and an adhesive sealant outside the vessel that dissolves slowly as the puncture site heals. Less commonly used closure devices include the **Angioseal** (Terumo, Tokyo, Japan), which deploys a bioabsorbable anchor to the arterial puncture site with surrounding collagen to promote closure. Other less commonly used closure devices include the **Manta**, **StarClose**, **Vascade**, and **Cardiva**.

 Tip

A step-by-step guide to deploying **Perclose devices** is shown in [this animated video](#) and listed below.

1. Exchange the procedural sheath for a guide wire so only the guide wire is left in the vessel.
2. Advance the flushed perclose device over the guidewire until the wire exit port is at skin level.
3. Remove the guidewire so only the perclose device is in the vessel and advance the perclose device until blood flows out of the marker lumen.
4. Lift the lever to open the foot of the device.
5. Insert the plunger down to insert needles, which will connect the threads of the perclose .
6. Remove the plunger, which pulls the thread taught, and cut the excess thread

Lower the lever to close the foot and retract the device until the guide wire access port is visible.

7. Reinsert guidewire and pull both threads taught.
8. Use the Snared Knot Pusher or Prostyle Suture Trimmer to advance the knot to the level of skin.
- 9 Pull the thread taught while removing perclose device.
9. Advance the knot over the guidewire and remove the guide wire once the bleeding is controlled.
10. Pull both threads tightly and remove the knot advancing device.

---

## Wires, Catheters, and Sheaths

### Tip

Using the analogy of a train on a track passing through a tunnel, the catheter can be thought of as the train, the wire as the train track, and the sheath as the surrounding tunnel.

## Wires

Wires are the first device inserted into a patient, have the lowest profile, and are variable in properties. Vascular access wires are made of a metal core and an outer wire wrapped around the metal core. They are used to guide devices such as catheters and sheaths into a vessel. Characteristics of wires include:

### Diameter

- Wire diameter is described in thousandths of an inch and typically range from 0.010-0.038”.
- Vascular access is typically obtained with a 0.018” wire and is exchanged for a smaller or larger profile as needed.
- The most common sizes used are 0.035”, 0.018” and 0.014”.

## **Stiffness**

- Wire stiffness is determined by the metal used as the core of the wire.
- The same wire will become stiffer with a larger diameter.
- Softer wires are used to manipulate catheters into vessels.
- Stiffer wires are needed to push larger devices, such as balloons or endografts.

## **Coating**

- Wires have a hydrophilic or hydrophobic coating.
- Hydrophilic wires have less friction when moving along the vessel wall and track more closely along the vessel lumen, increasing the risk of dissection.
- Hydrophilic wires are preferable in highly tortuous vessels.
- In contrast, hydrophobic wires keep more distance from the vessel wall and are less likely to dissect.

## **Tip Shape**

- The tip shape and consistency of the wire is important.
- Angled tips are used to navigate into side branches of vessels.
- Soft tips are designed to prevent trauma to the vessel wall.
- Weighted tips are used to cross occlusions.
- J-tips are used to prevent wiring of too small of vessels that may lead to trauma of end organs.

## **Length**

- Wires are measured in centimeters (cm) and typically range from 80 - 300 cm.
- Wires must not only be long enough to reach the target vessel from the point of access, but also need to be long enough to support exchanges outside of the body as needed.
- Care must be taken to avoid too long of a wire for its intended purpose as it is harder to control the end of the wire outside of the body.

## **Function**

Wires can be categorized by function. Please find a list of each category with common examples of specific wires below.

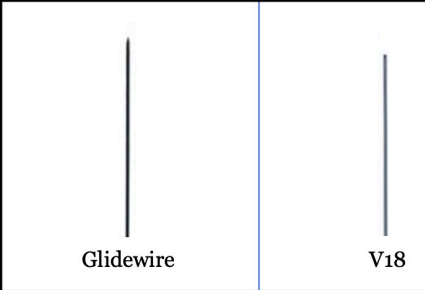

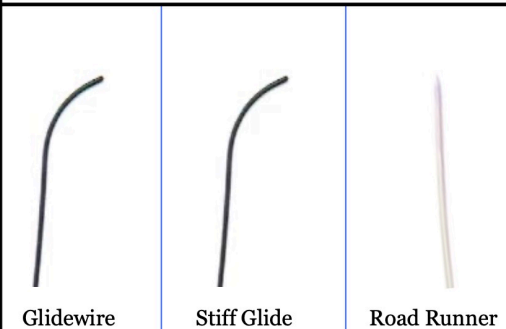
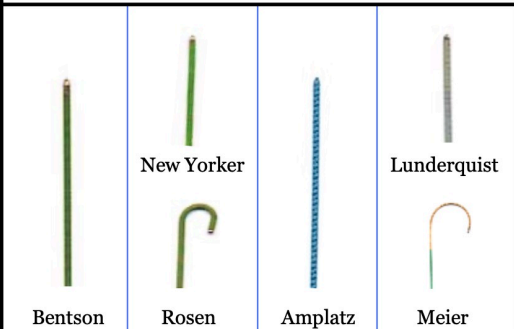
Diameter	Hydrophilic	Nonhydrophilic
0.018"	- ---Stiffness--- +	- ---Stiffness--- +
	 Glidewire                      V18	 Nitrex                              Cope
0.035"	- ---Stiffness--- +	- ---Stiffness--- +
	 Glidewire      Stiff Glide      Road Runner	 Bentson      New Yorker      Rosen      Amplatz      Lunderquist

Figure 2: Commonly used wires, courtesy of B. G. Northcutt, MD, A Shah, MD, Y Sheu, MD, L Carmi, MD.

**Access wires:**

- Short, typically 0.018” wires with a soft, non-directional tip to prevent any trauma to the vessel when entering, and a stiffer body to allow tracking of the introducer and sheath into the vessel.

**Guidewires:**

- Generally are characterized as pushable, hydrophilic wires with a floppy, curved or angled tips that are designed to track through tortuous vessels without causing blunt trauma to the vessel wall.
- Guidewires are also used for their directionality to cannulate branches.
- **Standard Glidewire (Terumo, Tokyo, Japan):** can be used as an access or guidewire. Hydrophilic coating, straight or angled tip, sized 0.018” - 0.035” in diameter and 150 cm or 260 cm in length.
- **Bentson (Cook Medical, Bloomington, IN):** flexible wire with a hydrophobic coating and curved tip. Used to access vessels that have a slight curve, or to introduce catheters or sheaths. Comes in 0.035” diameter and 150 cm or 260 cm in length.
- **Rosen (Cook medical, Bloomington, IN):** stiff hydrophobic wire with J tip. Often used as the first wire exchanged for the access wire. 0.035” long and 80 cm to 260 cm in length.

**Rail wires:**

- Used as a platform to support catheter and sheath exchanges, as well as to advance devices, such as stents or endografts.
- Rail wires come in a range of stiffness, from medium to very stiff.
- They may have a straight, floppy, or curved tip.

*Medium stiffness rail wires:*

- **Bentson (Cook Medical, Bloomington, IN):** flexible wire with a hydrophobic coating and curved tip. Used to access vessels that have a slight curve, or to introduce catheters or sheaths. Comes in 0.035” diameter and 150 cm or 260 cm in length.
- **Rosen (Cook medical, Bloomington, IN):** stiff hydrophobic wire with J tip. Often used as the first wire exchanged for the access wire. 0.035” long and 80 cm to 260 cm in length.

*High stiffness rail wires:*

- **Amplatz (Boston Scientific, Marlborough, MA):** stiff, hydrophobic, straight floppy or J-tip. 0.035” - 0.038” diameter and 75 cm - 260 cm in length.

- **Lunderquist (Cook Medical, Bloomington, IN):** one of the stiffest wires, hydrophobic, with floppy or curved tip. 0.035" diameter and 90 cm - 300 cm in length. Often used to advance large endografts.

## Catheters

Catheters are flexible, hollow tubes that are used in both the diagnosis and treatment of vascular disease.

### **i** Note

Catheters are measured in French (1F = 0.33 mm) and the diameter corresponds to the **outer diameter** of the tube.

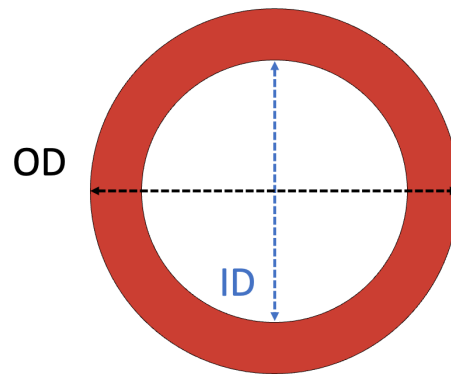


Figure 3: Outer diameter = OD, Inner diameter = ID.

Catheters vary by size, stiffness, hydrophilicity, radiopacity, and functionality, but can generally be described in two categories, flush catheters and selective catheters.

### Flush catheters:

- Contain multiple side holes just proximal to the end of the device to allow for the injection of contrast or other material, such as thrombolytics.
- **Omniflush (Angiodynamics, Latham, NY):** soft, flexible catheter with a radiopaque tip that is able to administer contrast to visualize vessels. [Omniflush device on back table](#)
- **Pigtail (Angiodynamics, Latham, NY):** Similar to the Omniflush with a looped end (Image YY).

## Selective catheters:

- Contain one hole at the distal end of the tube.
- Selective catheters come in many different shapes and sizes.
- Intuitively, the anatomy of the vessel to be tracked will determine the diameter and tip shape.
- When attempting to cross the aortic bifurcation into the contralateral common iliac artery, a catheter with more of a J-tip or “Shepherd’s hook” is preferable.
- When attempting to cannulate vessel branches with a more acute angle, a smaller angle catheter tip should be selected.
- Selective catheters can also be used to deliver contrast, but the contrast is only able to leave the catheter through the end of the device.
- Angiograms with selective catheters are more tailored to specific vessels, such as a renal or mesenteric artery.
- **Selective Glidecath (Terumo, Tokyo, Japan):** hydrophilic catheter available in 17 different shapes, but the 45 degree is most common. Comes in 4F or 5F. Offers great trackability and pushability. [This image](#) depicts some of the glidecath tip shapes.

## Sheaths

Sheaths are hollow tubes that provide a conduit between outer environment (i.e. outside the body) and the vessel. Returning to our train analogy, a sheath is the “tunnel” that the track and train run through.

Sheaths are used to advance and exchange catheters, wires, stents, balloons, and other devices. Sheaths can be divided into short sheaths for access and long sheaths for diagnostics and treatment. Long sheaths are flexible enough to track along different vessels, but are also stiff enough to support the advancement or exchange of devices.

### **i** Note

Sheaths are measured in French ( $1F = 0.33 \text{ mm}$ ) and the diameter corresponds to the **inner diameter** of the tube.

*As such, a 6F catheter will pass through a 6F sheath.*

Key points about sheaths:

- Sheaths are measured based on the inner diameter of the sheath, in contrast to catheters, which are measured based on their outer diameter.
- Sheaths contain a removable introducer, which is used to expand the tract from the outside into the vessel, and provide a smooth transition between the wire and the sheath.



- The side port on sheaths is used to flush the inner device with heparinized saline to prevent thrombus formation and distal embolization. It can also be used to inject contrast for diagnostic purposes.
- Sheaths come in variable diameters, lengths, and tips.
- Some sheaths, such as the **TourGuide (Medtronic, Minneapolis, MN)** are steerable. The operator can deflect the sheath tip up to 180 degrees. This is advantageous when cannulating branch arteries with sharp angles.

It is also helpful to understand what diameter of sheath can be inserted into different points of access as this will change the approach for the procedure. Though there is variation across patients, the table below is a useful starting point to understand what size sheath can be inserted in target access arteries.

Artery	Inner Diameter (mm)	Typical Access Sheath size (F)
Radial	2.3	4-6
Brachial		5-7
Axillary		5-14
Common Femoral	6.6	5-24
Popliteal	4.7	4-6
Pedal	2.1	4

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## Balloons, Stents, and Endografts

The delivery system for balloons consists of a catheter over the wire with the balloon built into the catheter tip. As such, advancing the balloon into place for angioplasty is no different from advancing a small catheter without a balloon.

Similar to balloons, stents are contained in catheters that are advanced into position over wires. The interventional component (i.e. stent) is near the tip of the catheter.

Both stents and balloons can elute drugs, most commonly *Paclitaxel*, an antiproliferative agent. Paclitaxel is lipophilic and remains active in the vessel wall following the procedure, preventing neointimal hyperplasia and improving patency. The impetus for using a drug-eluting stent or balloon in place of a standard stent or balloon is to decrease the chances of vascular remodeling following the procedure that may lead to restenosis.

In contrast to drug-eluting stents, drug-eluting balloons have the advantage of not leaving hardware in the vessel after intervention. Contrarily, drug-eluting stents remain in the vessel lumen after intervention, but are able to continue providing radial force.

## Balloons

Balloons are versatile endovascular tools and can be categorized by the degree to which they assume of the shape of the vessel.

**Compliant balloons:** - Compliant balloons inflate to a specific volume and *conform to the shape of the vessel*. - For example, the **Fogarty balloon** is a compliant balloon that can be used to remove thrombi in both open and endovascular procedures.

**Semi-Compliant balloons:** - These balloons have a slightly more rigid shape and therefore conform to a lesser degree to the shape of the container (i.e. vessel). - The **Coda balloon (Cook Medical, Bloomington, IN)** is semi-compliant and used to temporarily occlude large vessels.

**Non-Compliant balloons:** - Non-compliant inflate to a predetermined shape and size. - They are commonly used in angioplasty of peripheral and/or visceral stenosed vessels.

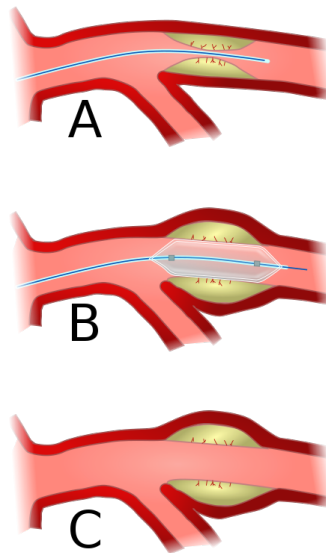


Figure 4: Angioplasty with a non-compliant balloon courtesy of J. Heuser, and Wikimedia.

Intraoperatively surgeons will often ask for the pressures of the balloon before using. Non-compliant balloons have a **nominal pressure**, which is the average pressure in atmospheres (atm) required to inflate the balloon to its *predetermined size*, and a **burst pressure**, where *99.9% of balloons will not burst if inflated to this pressure*.

## Stents

Stents may be covered or uncovered. **Covered stents** do not allow flow between the lumen of the stent and surrounding tissues, in contrast to **uncovered (bare metal) stents** which do allow flow through the stent.

Stents can be categorized by the means of deployment.

**Self-expanding stents** use a recoil mechanism during deployment to achieve a set diameter using stored energy from the packing of the stent.

Key characteristics of **self-expanding stents**:

- Flexible
- Low profile
- Deformable
- Low radial force (lower dissection risk)

**Balloon-expandable stents** are packaged around a balloon. The surgeon uses the balloon inside the stent to achieve a specific diameter based on the pressure used to inflate the balloon. As the diameter of the stent increases with higher balloon pressure, the length of stent decreases.

Key characteristics of **balloon-expandable stents**:

- Rigid
- Large profile
- Maintains shape over time
- More accurate deployment
- High radial force (higher risk of dissection)

### Tip

**Highly tortuous vessels** often require a **self-expanding stent** because of the increased flexibility needed to prevent kinking or in-stent restenosis.

**Heavily calcified vessels** often require the increased radial force of **balloon-expandable stents** to achieve full patency.

## Endografts

Endografts are tubes of fabric and interwoven metal used to exclude the diseased segment of an artery from circulation. The goal of an endograft is to prevent rupture or sac enlargement of an aneurysm by depressurizing the arterial walls.

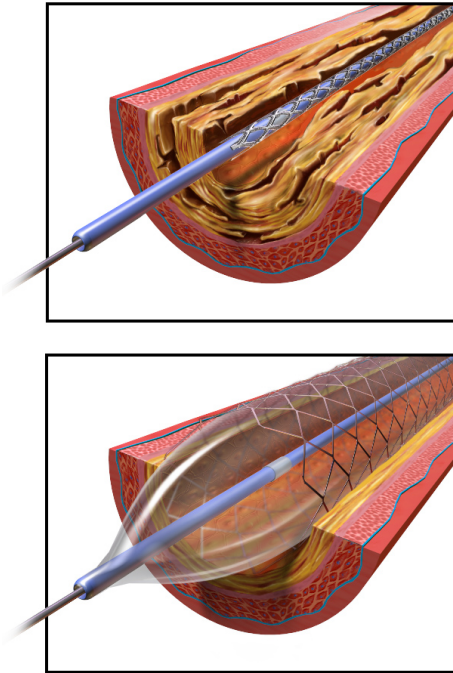


Figure 5: Balloon-expandable stent deployment courtesy of Blausen Medical. Retrieved on 22 February 20. Wikimedia.

Endografts are comparatively larger than stents and can require very large sheaths (>18F for endovascular aortic repair). To treat an aortic aneurysm with an endograft, there must be adequate sealing zones of healthy aorta above and below the aneurysm.

**i** Note

Please see the AAA Chapter for a full review of endovascular aortic repair (EVAR).  
Please see the TAA/TAAA Chapter for a full review of thoracic endovascular aortic repair (TEVAR)

For patients that have extension of the aneurysm into the visceral vessels of the aorta, the use of a conventional, non-fenestrated endograft would block blood flow to the branches of the diseased segment of the aorta, such as the kidneys or bowel. Treatment of thoracoabdominal or complex abdominal aortic aneurysms with endovascular repair requires a graft that allows flow to branches of the aorta. Some common techniques for maintaining visceral blood flow include :

**Fenestrated endografts** include small, circular holes in the main body of the endograft. The french word for window is “fenêtre.” When the fenestrations (or windows) are placed in line with visceral vessel origins, balloon-expandable stents can be used to bridge to the main endograft to the native renal or mesenteric arteries.

Additional characteristics about **fenestrated grafts**:

- They are ideal when the inner aortic diameter is small, making for a short distance (<5 mm) between the endograft and native visceral artery.
- They require the pathway between the endograft and the native artery to be close to perpendicular; that is, the pathway for the stent out of the graft cannot be at too large of an angle due to risk of stent fracture or kinking.
- The part of the stent inside the endograft must be flared out with a balloon to prevent endoleak from inside the endograft into the aneurysm sac.
- An of a commercially available fenestrated endograft is the Cook Medical Zenith Fenestrated (ZFen) AAA endograft. Click [here](#) to watch an animation of ZFen deployment.

**Branched endografts** have side branches built-into the main body of the graft in place of fenestration. These branches extend from the outer body of the main graft towards the target vessel.

Additional characteristics about **branched grafts**:

- They require a larger inner aortic diameter for more space between the endograft and target artery.
- Branches can receive self-expanding or balloon-expandable stents.

## Common Endovascular Procedures

Vascular surgeons perform a number of endovascular procedures. The most common include placement of an inferior vena cava (IVC) filter, diagnostic angiograms with angioplasty and stenting, fistulagrams, endovascular aneurysm repair (EVAR), and thrombectomy and thrombolysis. There are a number of basics that trainees should know before starting one of these procedures.

### IVC Filter Placement

- An IVC Filter is a metal cage-like device placed in the IVC to prevent clots from embolizing to the pulmonary vasculature.
- The hook at the superior aspect of the filter is used to aid with retrieval.
- The struts will catch clots and will anchor into the walls of the IVC to prevent migration.
- **Placement of an IVC filter can be controversial** but it is commonly used to prevent pulmonary embolism in patients who have a strict contraindication to anticoagulation.
- They are sometimes placed prophylactically in high-risk trauma patients.

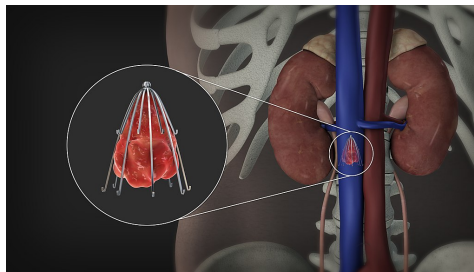


Figure 6: Inferior Vena Cava Filter courtesy of scientificanimations.com. Retrieved on 30 March. Wikimedia.

#### Procedure:

- Access is usually obtained via the right femoral vein as it gives direct access to the IVC.
- Ultrasound should be used to confirm that there is no thrombus at the access site - Access is then obtained via the Seldinger technique and a venacavogram is then obtained to assess the anatomy and the location of the renal veins
- The filter is generally placed just below the renal veins to prevent renal vein thrombus if filter thrombosis occurs.
- If a retrievable filter is placed, it should be removed as soon as the filter is no longer clinically indicated.
- Filter retrieval becomes more challenging the longer the filter remains in place.

- Filter retrieval is generally performed through the internal jugular vein in the neck to give access to the hook at the superior end of the filter, which can be snared and removed.

**i** Note

Please find a more detailed review of IVC filters [here](#)

## Lower Extremity Angiogram

A lower extremity angiogram is the starting point for a vascular surgeon to perform a number of different interventions including angioplasty, stenting, and thrombectomy. An angiogram is a procedure in which contrast dye is used to display the vasculature and its disease burden under X-ray.

**💡** Tip

Before an angiogram, one should be aware of all prior interventions a patient has had, both open and endovascular. It can be useful to review old operative reports and imaging studies in order to know the patient's current anatomy.

**💡** Tip

In addition, the patient's most recent imaging studies should be reviewed including ABI/PVRs, arterial duplex, and any axial imaging such as a CTA. Please see Claudication Chapter for a summary of ABI/PVR testing and interpretation.

Procedure:

- Access is typically obtained in the contralateral common femoral artery and a wire and catheter are passed up and over the aortic bifurcation to gain access to the operative (ipsilateral) side.
- A series of angiograms are taken to assess the level of disease and based on this a final operative plan will be made.
- There are multiple options for treatment including angioplasty, stenting, atherectomy, thrombectomy, and thrombolysis, to name a few.

Possible Interventions:

- **Angioplasty:** a balloon is advanced across the area of concern (usually atherosclerosis causing a flow limitation) and expanded to dilate the area of stenosis.

- **Stenting:** commonly after an angioplasty, a stent is placed to maintain patency of the area. There are multiple types of stents (covered above) that can be used depending on anatomic location, size, and indication.
- **Atherectomy:** a procedure where a calcified plaque is physically removed from the artery. This can be done with a cutting blade, a spinning drill-like device, or a laser.

### **i** Note

#### **Considerations for Atherectomy**

As plaque is broken into smaller pieces, there is a significant risk for **distal embolization** of plaque debris. To prevent this, an embolic protection device is often used which can filter blood and catch any smaller particulate matter that travels distally. Some of the devices include an aspiration device to remove clot and thrombus as it is removed.

There is ongoing research into the outcomes of atherectomy versus stenting alone, but generally, the long-term patency rate is similar. (“Atherectomy for Lower Extremity Intervention: Why, When, and Which Device?” n.d.)

- **Thrombolysis:** a procedure where the clot is dissolved often with tPA and heparin to treat an acute thrombotic event. Often, angiograms are performed for acute limb ischemia from a clot rather than atherosclerotic disease. A lysis catheter or Cragg-Macnamara catheter can be placed. The **Cragg-Macnamara catheter** is a long catheter with multiple side ports where medication can be infused over the length of a thrombosed artery. This catheter can be left in place for **24-48 hours** and the medication is allowed to “bust” the acute clot. After this, the patient will be brought back to the operating room to evaluate the progress of the clot dissolution. (Giannakakis et al. 2017)
- **Thrombectomy:** a procedure where acute or subacute thrombus is removed from a vessel. This can be accomplished in a variety of ways. Commonly, a suction thrombectomy system can be used to remove thrombus from a vessel using vacuum force for example using the **Penumbra system**. Alternatively, a mechanical thrombectomy can be performed where a Fogarty catheter is deployed past a thrombus, a balloon inflated at the end of the catheter, and then manually pulled back towards an arteriotomy and the clot then removed. (Karnabatidis et al. 2011)

## **Fistulograms**

A fistulogram is an angiographic study used to interrogate and troubleshoot a dysfunctional arteriovenous fistula.

Common complications of fistulas include:

- Anastomotic strictures.



- Inflow issues (such as atherosclerosis of the inflow artery).
- Outflow issues (such as central venous stenosis).
- Thrombosed fistula.

Access is obtained in different locations depending on the culprit lesions (the inflow artery, the fistula itself, the outflow veins) and can be treated with angioplasty and or stenting.

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## Risks

As with any invasive procedure, even a minimally invasive procedure comes with its own set of risks, and as the complexity of the procedure increases so does the risk.

The American Heart Association has put out guidelines to help stratify patients at risk for vascular complications.(Patel et al. 2010a)

**Low risk cases** were defined as those with a complication rate below 1%. Characteristics include:

- Diagnostic angiograms without interventions.
- Short operative times.
- Small sheaths (5 Fr).
- Minimal anticoagulation.
- Younger male patients with larger body habitus and normal renal function were at lowest risk of complications.

**Moderate risk cases** were defined as those with a complication rate of 1-3%. Risk factors include:

- Longer, more complex procedures.
- Involve larger sheaths (6-7Fr).

**High risk cases** were defined as those with a complication rate >3%. Risk factors include:

- Peripheral arterial disease patients
- Advanced age
- Female sex at birth
- Liver disease
- Coagulopathy
- Immunosuppression
- Status post valve replacement
- Renal dysfunction.
- Longer procedure times

- Larger sheaths (>8Fr), use of arterial closure devices
- More likely to involve anticoagulation

## Access Complications

Although each procedure comes with its own specific risks (e.g. stroke for a carotid artery stent, distal embolization for atherectomy etc), there are multiple complications that can occur with any endovascular procedure. The procedure specific complications will be explored in further detail in each chapter. Common access complications are listed below.

### Groin Hematoma

A groin hematoma is a collection of blood that has collected outside the lumen of the vessel, which often occurs after removal of the sheath.

Groin hematoma characteristics:

- Occur in about 1-3 % of cases
- Often due to a stick not directly over the femoral head in which case manual pressure may not provide adequate hemostasis.
- Hematomas are rarely ever operated on unless they cause skin compromise, severe pain, femoral nerve compression, or are rapidly expanding.
- Hematoma will generally be absorbed by the body.
- If they are operated on, there is a significant risk of infection (about 20%) and seroma formation.

### Pseudoaneurysms

A pseudoaneurysm is defined as a collection of turbulent blood flow contained only by one or two layers of the arterial wall as opposed to a true aneurysm where all three layers are dilated (**occur in less than 1% of endovascular cases**). They occur secondary to injury (iatrogenic or trauma).

**These must be managed urgently and treated appropriately to prevent significant morbidity and mortality.**

### Pseudoaneurysm presentation:

- Pulsatile often painful mass at a prior access site
- May lead to skin necrosis
- Bruit on auscultation

**Pseudoaneurysm risks:**

- Vessel rupture
- Thrombus formation with distal embolization
- Skin necrosis
- Nerve compression
- Venous compression

**Etiology:**

Most often, seen at the femoral artery as an access site complication from:

- Failed deployment of a closure device
- Laceration of the artery or branch by an access needle
- Inadequate pressure or length of pressure being held.

Patients who are obese, female, hypertensive or are on anticoagulation or antiplatelet therapy are at higher risk for femoral PSA.

**Workup:**

- Duplex ultrasound of the site.
- Pulse exam to ensure adequate distal perfusion.

**Management:**

Management is dependent on size and presentation of the pseudoaneurysm. In the past, all pseudoaneurysms were treated operatively, but with new advances, minimally invasive techniques have become more common with good outcomes.

- For a small asymptomatic pseudoaneurysm (less than 2cm), an appropriate first step would be observation and repeat duplex ultrasound in about one month.
- For a persistent pseudoaneurysm after one month, a larger/loculated asymptomatic pseudoaneurysm, or a pseudoaneurysm that presents with minor pain at the site, minimally invasive intervention with ultrasound guided compression or ultrasound guided thrombin injection is appropriate.
- For any pseudoaneurysm presenting with persistent hemodynamic instability, limb ischemia, skin necrosis, AV fistula, nerve compression, expanding hematoma, an urgent operation is required.
- Urgent open repair generally consists of gaining proximal and distal control and then primary repair of the arterial defect.
- Urgent endovascular management may also be entailed by deploying coils in the sac and placing a covered stent in the femoral artery, therefore thrombosing the pseudoaneurysm.

[Kassem et al. (2013)](Kassem and Elmahdy 2014)(Tulla, Kowalski, and Qaja 2024)

## **Retroperitoneal Hematoma**

A retroperitoneal hematoma is a collection of blood accumulating in the retroperitoneal space where the external iliac artery travels.

Key points:

- It is a rare complication occurring in about 0.5% of cases.
- Retroperitoneal hematomas are a dreaded complication as they come with high morbidity and mortality especially if not rapidly diagnosed.
- Retroperitoneal hematomas occur when the arterial stick in the artery during groin puncture is performed too high, above the level of the inferior epigastric artery.
- This leads to extravasation of blood into the retroperitoneal space after sheath removal.
- Retroperitoneal hematomas can usually be treated with a closure device if diagnosed intra operatively or a covered stent from the contralateral side.

## **Arteriovenous (AV) Fistula**

An arteriovenous fistula is an aberrant connection between an artery and a vein. Although commonly formed surgically for hemodialysis access, they can be caused iatrogenically during arterial access for endovascular intervention.

Key points:

- They are rare and occur in less than 1% of groin access cases.
- Occur during inadvertent puncture of the deep femoral artery and deep femoral vein.
- Usually no intervention is needed.

## **Dissection**

An arterial dissection is the separation of the layers of the arterial wall.

Key points:

- May occur naturally (often secondary to hypertension) or iatrogenically (in the setting of arterial puncture). This most often occurs in the setting of a posteriorly lying plaque in the artery where the wire can separate the posterior layers.
- To prevent dissection, do not apply too much forward pressure when advancing a wire during initial arterial access. Do not inject fluid (saline or contrast) unless there is blood return to ensure the needle or catheter tip are in the true lumen.

- Treatment can be conservative in the case of an iatrogenic dissection or treated with stent placement if the dissection limits blood flow. Intravascular ultrasound can be used to further assess the dissection.

### **Acute Thrombosis of Access Vessel**

Acute thrombus of the access vessel is when the artery where access is obtained completely “clots off.” It is an incredibly rare complication caused by incorrect deployment of vascular closure devices, access site dissection, or occlusive sheath placement in a small or diseased vessel.

## **Endovascular Surgical Complications**

### **Atheroembolism**

Atheroembolism is the dislodgement of a portion of a cholesterol plaque leading to distal embolization causing acute thrombosis or partial occlusion.

Key points: (Liew and Bartholomew 2005)

- Generally, when these come from the ascending aorta or arch, they will embolize to the cerebral and retinal vessels. On the other hand, when an embolus originates in the thoracic or descending aorta, it will likely embolize to the mesenteric, renal or lower extremity vessels.
- These emboli will then cause a further inflammatory response worsening outcomes.
- Outcomes are dependent on volume and size of emboli and can range from sub-clinical to fatal.
- Embolization is most common to the skin, kidneys, and GI tract though it is dependent on the site of the procedure.
- Unfortunately this condition is generally under diagnosed and challenging to treat. - Primary prevention is of utmost importance with good surgical technique.

### **Wire Perforation**

Wire perforation occurs when an angiographic wire creates a hole in a vessel.

Key points:

- Incredibly rare but treatable when diagnosed rapidly.
- Occurs when a large stiff hydrophilic wire is passed through a small vessel especially if not done under fluoroscopic guidance.
- Treatment involves transcatheter embolization of the perforation site.

## Vessel Rupture

Vessel rupture occurs when a device or balloon is oversized for a vessel and causes a “blowout” of a vessel.

Key points:

- Rupture can be caused by an oversized balloon or balloon expandable stent, by expanding a properly sized balloon in a heavily calcified vessel or by passage of a large device such as an EVAR device through a small vessel.
  - Rupture is associated with a high morbidity and mortality especially if there is any delay in diagnosis.
  - Immediately upon recognition of vessel rupture, a balloon should be inflated in the region to obtain adequate hemostasis. Simultaneously, supplies should be gathered for vessel repair.
  - Repair is usually done with a covered stent placed across the rupture. If adequate hemostasis cannot be obtained, open repair may be necessary.
- 

## Teaching Case

### Scenario

A 71 year old male with a past medical history of hypertension, hyperlipidemia, coronary artery disease with stent placement was diagnosed with acute type B aortic dissection three weeks ago. He was managed medically in the hospital and sent home after one week with blood pressure and pain well-controlled. An image from the CTA at time of diagnosis is seen below:

The proximal extent of the dissection was 3 cm distal to the left subclavian artery and the distal extent was 2 cm superior to the celiac artery, allowing good landing zones for an endograft. The left internal iliac artery is fully occluded, and the right internal iliac artery has roughly 50% stenosis. The left external iliac artery is heavily calcified and stenotic >50%. The right common and external iliac artery are patent without evidence of significant atherosclerosis. The patient is evaluated in the preoperative area prior to placement of a thoracic endograft in the hybrid operating room.



Figure 7: CTA

### Exam

**HEENT:** Pupils round and reactive to light, no lymphadenopathy

**Cardiac:** Regular rate and rhythm

**Pulmonary:** Clear to auscultation in all lung fields. No accessory muscle use.

**Abdominal:** Soft, non-tender, non-distended, no pulsatile masses felt.

**Vascular/Extremities:** Warm, non-edematous. Palpable pulses in all four extremities.

### Discussion Points

**i** N.B. There is no prepared answers for the questions below.

However, we feel this chapter contains all the necessary information to answer the questions. If not, please let us know!

1. What clinical risk factors make this patient a good candidate for endovascular vs. open repair?

2. The endograft used for this operation requires at least an 18F sheath, which site of access should be used to insert and implant the graft?
  3. What diameter and hydrophilicity of wire should be used to advance the thoracic endograft to the site of dissection?
  4. The attending surgeon requested mean arterial pressures to be at least 90 mmHg throughout the operation. Why would intraoperative hypotension be concerning in this patient? What anatomic factors make this patient at particular risk?
  5. If the patient developed pain at the site of access one week later, with a well-healing wound and palpable thrill on physical exam, what kind of access-related complication are you worried about and what would be your next step to confirm the diagnosis?
- 

## Key Articles

1. Patel MR, Jneid H, Derdeyn CP, Klein LW, Levine GN, Lookstein RA, White CJ, Yeghiazarians Y, Rosenfield K; American Heart Association Diagnostic and Interventional Cardiac Catheterization Committee of the Council on Clinical Cardiology, Council on Cardiovascular Radiology and Intervention, Council on Peripheral Vascular Disease, Council on Cardiovascular Surgery and Anesthesia, and Stroke Council. Arteriotomy closure devices for cardiovascular procedures: a scientific statement from the American Heart Association. *Circulation*. 2010 Nov 2;122(18):1882-93. doi: 10.1161/CIR.0b013e3181f9b345. Epub 2010 Oct 4. Erratum in: *Circulation*. 2010 Nov 2;122(18):e507. PMID: 20921445. (Patel et al. 2010b)
  2. Siracuse JJ, Farber A, Cheng TW, Rauli SJ, Jones DW, Kalish JA, Smeds MR, Rybin D, Schermerhorn ML; Vascular Quality Initiative. Common femoral artery antegrade and retrograde approaches have similar access site complications. *J Vasc Surg*. 2019 Apr;69(4):1160-1166.e2. doi: 10.1016/j.jvs.2018.06.226. Epub 2018 Dec 4. PMID: 30527937. (Siracuse et al. 2019)
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## Additional Resources

### Audible Bleeding Content

- Holding Pressure Case Prep: [Endovascular Basics](#)
- Audible Bleeding Exam Prep: [Endovascular Access](#)



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## Websites

- TeachMe Surgery: [Pseudoaneurysm](#)
- 

## Serious Games

### Touch Surgery Simulations.

- Must download the [Medtronic Touch Surgery mobile application](#) to access the modules. Available for Apple and Android mobile devices.
  - [Femoral Artery Access](#)
- 

## Gore Combat Manual

The [Gore Medical Vascular and Endovascular Surgery Combat Manual](#) is an informative and entertaining read intended as a vascular surgery crash course for medical students, residents, and fellows alike. Highly accessible with a thoughtfully determined level of detail, but lacking in learning activities (e.g. questions, videos, etc.), this resource is a wonderful complement to the APDVS eBook.

 Tip

Please see pages 25-41, 49-61.

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## Operative Footage

Developed by the DeBakey Institute for Cardiovascular Education & Training at Houston Methodist. *YouTube account required as video content is age-restricted.* Please create and/or log in to your YouTube account to have access to the videos.

**Ultrasound-Guided Femoral Access**

**ProGlide Vascular Closure Device Deployment**

**Inferior Vena Cava (IVC) Filter Placement**

**Standard IVC Filter Retrieval**

# Radiation and Radiation Safety

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## Note

By the end of this chapter, students will be able to:

- Recognize the fundamental types of ionizing radiation.
- Understand the sources of radiation in the vascular operating room.
- Recognize and practice radiation safety measures.
- Recognize adverse outcome of significant radiation exposure.

## Key Facts

1. The primary forms of ionizing radiation are alpha particles, beta particles, gamma rays, and x-rays.
2. In the operating environment, x-rays scatter leading to potential unexpected exposure.
3. Lead aprons and glasses are important in protecting radiosensitive organs in the body.
4. Proper shielding, minimizing duration of radiation use, and increasing distance from the source are important measures to reducing the amount of radiation exposure.
5. The side effects of prolonged radiation exposure to healthcare providers include various health issues such as cataracts and reproductive dysfunction.

## How We Suggest Using the Pre/Post Questions

The pre/post questions are listed below. They are all multiple choice questions with a single right answer. To best guide your learning, we have hidden the answers in a collapsible menu. Before reading the chapter, we suggest giving the questions a try,

noting your answers on a notepad. After reading the chapter, return to the questions, re-evaluate your answers, and then open the collapsible menu to read the correct answer and discussion. Do not fret if you have difficulty answering the questions before reading the chapter! By the end of the chapter, we are certain you will have covered the knowledge necessary to answer the questions. There will be a teaching case at the end of the chapter. This is another opportunity to exercise your new knowledge!

## Pre/Post Questions

### Case Based Questions

1. A 45 year old vascular surgeon with 10 years of professional practice is preparing to perform a left upper extremity fistulogram due to concerns for prolonged bleeding after hemodialysis. *What is the most appropriate PPE that the student should wear during the case?*
  - A. Full leaded face shield, lead apron, and personal radiation badge
  - B. Lead apron alone
  - C. Leaded safety glasses
  - D. Lower half of a 2-piece lead apron and thyroid shield
  - E. Lead apron, leaded safety glasses

#### Answer

##### **A. Full leaded face shield, lead apron, and personal radiation badge**

Discussion: The most appropriate PPE includes a full leaded face shield, lead apron, and radiation badge making answer a the correct answer. This will provide the most significant protection to the head and neck as well as vital organs of the body during the procedure. The radiation badge will help with long-term protection as it allows for cumulative tracking of exposure. A lead apron alone and or lead glasses alone is inadequate for optimal protection making answers b, c, and d incorrect. A full-face shield will be of greater protection than glasses which makes answer e incorrect.

2. A 25 year old medical student has been invited to observe endovascular stent placement for an infrarenal AAA. *In addition to wearing appropriate PPE, what can the medical student do to reduce their exposure during the case?*
  - A. Stand as close to the radiation source as possible.
  - B. Turn their back to the radiation source.

- C. Stand as far from the radiation source as possible.
- D. Wear a radiation badge.

Answer

**C. Stand as far from the radiation source as possible.**

Discussion: Increasing one's distance from the radiation source is a key measure to reduce radiation exposure. Radiation exposure decreases as the distance from the source increases. This phenomenon follows an inverse square law ( $1/d^2$ ). Standing close to the source will increase their radiation dose making answer a incorrect. If wearing a lead smock, turning one's back to the radiation source will render the PPE useless. If wearing a leaded vest and skirt, turning one's back has no effect. Wearing a radiation badge is beneficial for long-term monitoring of radiation exposure but does not affect the amount of radiation exposure.

3. A 55 year old vascular surgeon who has been practicing for 20 years has noticed some subtle changes in his vision. *What is the most likely underlying disease process?*
- A. Optical neuropathy
  - B. Glaucoma
  - C. Opacification of the lens
  - D. Retinal angiopathy

Answer

**C. Opacification of the lens**

Discussion: The lens is the most radiosensitive component of the eye. Lens opacification (i.e. cataracts) is the most common adverse effect of radiation exposure to the eye. There was no mention of increased intraocular pressure or abnormal fundoscopic exam making answers a, b, and d incorrect.

4. A 65 year old female is presenting to the hospital for endovascular repair of a complicated AAA. There is prolonged radiation time with a calculated absorbed dose of 3.5 Gy. *What is the most likely skin exam finding during the patient's hospital course?*
- A. Skin necrosis
  - B. Burn
  - C. No acute findings as the dose were too low
  - D. Erythema

Answer

**D. Erythema**

Discussion: With a dose of 3.5 Gy, the most likely result will be transient erythema between 2 to 24 hours from exposure. This can be found in table 2. Skin necrosis can occur, but it would need much higher doses making answer a wrong. It would be incorrect to say the dose is too low as 3.5 Gy making answer c invalid, and also below the threshold to cause a burn making answer b incorrect as well.

5. A 42 year old male on hemodialysis from a left upper extremity brachiocephalic fistula for CKD is seen for a fistulogram due the patient having trouble pulling during dialysis. The patient is placed in the supine position with left arm extended. The c-arm is initially perpendicular to the operating table. To achieve better views of the proximal vessels, the c-arm is rotated counterclockwise (from the perspective of looking from the patient's feet superiorly) so that the image intensifier is rotated more towards the patient's right shoulder. *What is the appropriate name for the c-arm position?*
- A. LAO (left anterior oblique)
  - B. RAO (right anterior oblique)
  - C. Cranial
  - D. Caudal
  - E. AP (anterior posterior)

Answer

**B. RAO (right anterior oblique)**

Discussion: When the image intensifier is rotated more towards the right shoulder, this is the RAO position making answer a correct. If the c-arm were rotated clockwise, with the c-arm over the left shoulder, this would be the LAO position. As no mention was made of rotating the c-arm towards the head or foot of the bed, answers c and d are incorrect. The AP position was the initial position of the c-arm when it is perpendicular to the OR table.

6. A 27 year old female who is a circulating nurse in the endovascular suite recently became pregnant. She has informed the radiation safety officer and staff of the endovascular suite. She is curious how best to monitor the radiation dose the fetus is receiving in utero. Where should the nurse place the fetal dosimetry badge?
- A. The dosimetry badge should be worn on the outside of the lead apron at chest level
  - B. The dosimetry badge should be worn under the lead apron around chest

- C. The dosimetry badge should be worn outside the lead apron at the abdomen level.
- D. The dosimetry badge should be worn under the lead apron at the abdomen level.

Answer

**D. The dosimetry badge should be worn under the lead apron at the abdomen level.**

Discussion: To monitor the dose to the unborn child, the dosimeter badge should be worn under the lead at the abdomen level, making answer d correct. We are concerned with the amount of radiation that is able to pass through proper PPE. Wearing the badge outside the lead apron will indicate the amount of radiation exposure in front of the shielding and not an approximate fetal dose making answers a and c incorrect. If the badge is worn at the level of the chest, the dose will also not be the most accurate, making answer b incorrect.

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## Introduction

The use of radiation is fundamental to a vascular surgeon's practice. Intraoperative fluoroscopy, computer tomography (CT), and plain film X-rays (to a lesser extent) are crucial tools in vascular surgery. The vascular surgery patient will likely undergo all these radiation-based imaging studies.

Vascular surgeons and other professionals (nurses, scrub technicians, imaging technicians, etc.) can be exposed to significant levels of radiation over a career of performing intraoperative fluoroscopy/angiography and endovascular surgery. As the medical community is well aware of these risks, many initiatives are underway to decrease the amount of radiation required to perform studies and interventions.

Vascular patients are also exposed to radiation, although often at higher doses over shorter periods of time. Educating oneself about radiation and radiation safety is essential. This chapter will explore the basics of radiation, radiation safety principles and measures, and potential adverse outcomes of significant radiation exposure.

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## Radiation Penetrance

A critical concept to understand when discussing occupational and patient exposure to radiation is penetrance.

- **Penetrance** refers to the ability of emitted radiation to penetrate into or through materials and is defined as the fraction of radiation passing through a given object (or the inverse of attenuation).
- The **density** of materials or tissues determines their **ability to attenuate or block X-rays**.
- Dense tissues (bone) or materials (lead) absorb a greater fraction of radiation while less dense tissues (skin) or materials (water) allow for deeper penetration.

This concept is especially relevant to vascular surgeons and their patients as an increasing proportion of vascular procedures are performed endovascularly under live fluoroscopy which utilizes X-rays.

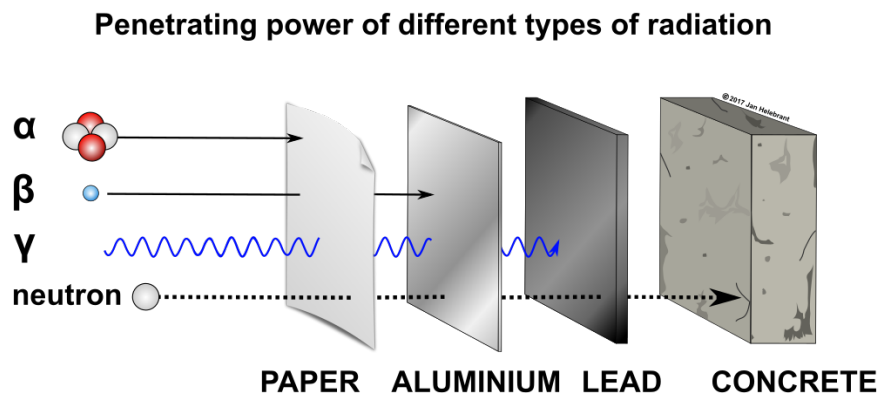


Figure 1: Penetrating power of different types of radiation - alpha, beta, gamma and neutrons courtesy of Openclipart and Juhele. *X-rays have similar penetrance to gamma rays.*

## Radiation types

Radiation can be broadly categorized into ionizing and non-ionizing forms. This chapter will focus on ionizing radiation, which is encountered by medical professionals in clinical settings.

Ionizing radiation falls within the higher frequency range of the electromagnetic spectrum. The commonly discussed types of ionizing radiation include X-rays, alpha particles, beta particles, gamma rays.



## Alpha Particles

Alpha particles, despite their high energy, have poor penetrance. As such, they do not penetrate the skin. However, they can be dangerous if inhaled or ingested.

## Beta Particles

Beta particles, have slightly more penetration than alpha particles.

They have some ability to penetrate the skin. However, they can be easily blocked by less dense shielding like aluminum.

Similar to alpha particles, beta particles can also cause harm if ingested or inhaled.

## Gamma Rays

Gamma rays possess significantly more energy and can penetrate through the human body and less dense shielding.

**Lead is effective in shielding against gamma rays due to its density.** Dense materials increase the likelihood of radiation interacting with subatomic particles, reducing the rays' passage through the material.

As gamma rays pass through the body, they cause ionizing damage to tissue and DNA and should be treated with caution.

## X-Rays

X-rays, widely used in healthcare, share similarities with gamma rays in their ability to penetrate tissue.

Their application in medical imaging includes plain X-ray films, CT scans, and fluoroscopy.

**Lead is effective in shielding against x-rays due to its density.**

The position of the x-ray emitting source of the the C-arm used for endovascular procedures is at the bottom of the arm, beneath the patient and the operating room table. **This is the location of highest exposure.**

As the X-ray source emits radiation, **radiation scatter occurs**, moving upward and outward from the source.

## Terminology

It is important to understanding the terminology related to radiation exposure.

- The **absorbed dose** refers to the energy from ionizing radiation per unit mass of exposed material, measured in **grays (Gy)**.
- The **equivalent dose** represents the mean absorbed dose of a specific organ or tissue, measured in **sieverts (Sv)**.
- The **effective dose**, also measured in **sieverts (Sv)**,\*\* accounts for total-body radiation exposure and is guided by the International Commission on Radiological Protection (ICRP) guidelines for comparing radiation effects on individuals.

X-ray technology for imaging comprises two critical components: the **radiation source**, where X-ray photons are generated, and the **detector**.

- The shape of the beam from the **source** is managed by the **collimator**.
- As X-ray beam passes through the patient, they are captured by the detector and an image is created by **image intensifier**.

In endovascular procedures using a C-arm, the detector portion (i.e. image intensifier) is above the patient, while the X-ray source is beneath the patient and table.

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## Principles for Patient Safety and Radiation Exposure

While any procedure that produces ionizing radiation will expose the patient to some degree of risk, it is the responsibility of the patient's surgeon to ensure that the risk of the procedure is minimized as much as possible without compromising the efficacy of the therapy.

Certain considerations exist for special patient populations including pregnant patients. These are discussed later in this chapter.

A number of core principles should be considered whenever a provider is considering recommending or performing a treatment with ionizing radiation including:

### Medical Necessity and Justification for the Procedure

The potential benefits of the procedure should outweigh the risks of not performing the procedure or performing an alternative procedure without radiation exposure.

## Exposure Optimization

The radiation dose delivered, particularly for intraoperative exposures, should be optimized to be as low as possible while still achieving adequate image quality for the procedure and decision making.

This includes fluoroscopy techniques such as reducing the time under fluoroscopy by using **pulsed fluoroscopy** as opposed to continuous scanning, **collimating the X-ray beam** to the area of interest and **narrowing the beam** to only scan structures necessary, **adjusting the frame rate of fluoroscopy** images when temporal resolution is not necessary.

It also includes **effective preoperative planning**, including properly positioning the patient for optimal imaging to reduce repeated attempts, and planning with previous imaging to ensure good understanding of the vascular anatomy and to form a procedural plan prior to exposing the patient to additional radiation.

## The ALARA Principles

ALARA stands for “*As Low As Reasonably Possible*,” and refers to the balance between minimizing radiation exposure to the patient without compromising the quality of care.

**The energy of the X-rays:** Higher energy X-rays will have greater penetrance and pass through thicker and deeper tissue more easily. Energy is typically adjusted during the procedure to optimize for the depth of the structures being imaged.

**The duration of exposure:** The dose of radiation delivered to the patient and the OR staff is directly correlated with the duration of exposure. This is why vascular surgeons try to limit the amount of time under fluoroscopy to the minimum required.

**The distance from the source:** The intensity of radiation exposure decreases with distance from the emitting source in an inverse square relationship. The patient and surgeons at bedside and closest to the C-arm, therefore, receive significantly greater doses of radiation as compared to OR staff further away.

## Patient Education and Informed Consent:

Patients should be informed about the risks of exposure to ionizing radiation, the benefits of the procedure, and the efforts made to reduce total dosage.

Patients should provide informed consent before any intervention whenever possible, and should be encouraged to ask questions and voice concerns prior to the initiation of treatment.

## Patient monitoring and dose recording:

During fluoroscopy-guided procedures, patient radiation doses should be monitored and recorded accurately to help understand the patient's cumulative radiation exposure.

In a hybrid OR, the total radiation is recorded on the large screen. If not, you can always ask the hybrid room technician.

## Radiation shielding:

The use of personal protective devices made of dense materials, such as lead aprons, thyroid collars, and other protective shields, serve to attenuate radiation.

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## Shielding

Shielding serves to protect vital organs from radiation exposure and damage. The most common type of shielding is lead, typically in the form of a skirt, vest, and thyroid shield worn beneath the surgical gown.

Lead aprons and other lead-lined materials are highly effective in protecting personnel from radiation exposure. Lead, being a very dense material, absorbs a significant portion of the radiation from X-ray beams that would otherwise reach the person wearing it. These lead protection devices are specifically designed to safeguard vital organs and those most sensitive to radiation exposure—such as organs not shielded by overlying bone. This includes the torso, reproductive organs, and the thyroid gland.

### **i** Note

Please find an example of lead [here](#):

Please find an example of a thyroid shield [here](#):

Reported effectiveness of worn lead varies. Some sources claim that a lead apron with a thickness of 0.5 mm can reduce radiation scatter by 90%, while others suggest a lesser reduction. However, there is a consensus that the apron should be at least 0.25 mm thick both in the front and back. (Cheon et al. 2018)

Additional shielding such as lead screens can be found in the OR to provide further protection against scattered X-rays.

**i** Note

Please find an examples of lead shields [here](#):

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## Radiation Badges

Radiation badges or “dosimeters” are personal wearable devices designed to measure the amount of radiation exposure an individual receives. They measure the total exposure accumulated over a set period of time. It’s important to note that these devices specifically measure ionizing radiation and not other forms.

There are several types of badges available. Film badges are commonly used in clinical practice. They are worn by a single person and are evaluated every 30 days. Other types of badges provide real-time quantification of radiation exposure, with readings recorded at the beginning and end of each shift. A radiation officer is typically tasked with tracking these exposure levels.

**i** Note

Please find more information about radiation badges [here](#):

When an individual’s exposure approaches a certain threshold, which is typically set below the maximum allowable dose for a year, measures are taken to further minimize their exposure. For instance, if an employee has reached a significant exposure level two months into the year, the radiation compliance officer would discuss strategies to reduce their exposure. Strategies include maintaining greater distance from radiation sources, rotating through cases more frequently, and avoiding involvement in procedures with a higher risk of elevated radiation levels.

If an employee surpasses their annual exposure limits, they are typically prohibited from participating in procedures involving radiation for the remainder of the year. The specific limits of exposure are detailed later in the chapter.

## Lead Glasses and Eye Protection

In the endovascular suite, it's common to see individuals wearing leaded glasses. A 2016 study compared different types of glasses and their effectiveness in reducing radiation exposure. The results showed that all leaded glasses outperformed non-leaded safety glasses, with an average reduction in dose of approximately **90.3%**. (Waddell et al. 2016) This study is just one of many that highlight the efficacy of lead glasses in minimizing eye exposure during procedures involving radiation.

Despite the significant reduction achieved with the use of **lead safety glasses**, there's a caveat regarding a false sense of security. Due to the scattering of radiation during procedures, the eye can be exposed to radiation at oblique angles (i.e. around the glasses), leading to increased exposure and limited reduction in dose. (Kirkwood et al. 2020) In such scenarios, the optimal solution for reducing eye exposure would be a **full visor or face shield with lateral eye shielding**. A study by Samara et al. demonstrated that the most substantial dose reduction was observed in the full visor group.(Samara et al. 2022)

**Wearing lead safety glasses during interventions is recommended** as it reduces radiation exposure and helps maintain eye health, particularly for individuals at risk of accumulating significant lifetime radiation exposure.

### **i** Note

Please find an examples of lead glasses [here](#):

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## Imaging Technique

Now that we've covered both radiation equipment and safety gear, it's crucial to underscore the importance of imaging technique. This concept is critical because it addresses the multifaceted aspects of minimizing radiation exposure during procedures.

The radiation dose depends on both the **intensity of imaging** and the **duration of fluoroscopic exposure**. (Rial and Vañó 2021) During endovascular procedures, the operating surgeon can control these factors by:

1. **Pulse imaging:** Instead of continuously scanning the patient, the surgeon can pulsate images (i.e. shoot several plain x-rays). This approach reduces scatter and lowers overall exposure. It's especially effective when high temporal resolution isn't necessary.
2. **Collimator use:** The surgeon can use the collimator to narrow the X-ray beam, focusing solely on the area of interest. This method effectively reduces both dose and scatter.

The image intensifier should be positioned closer to the patient to optimize image quality. Conversely, the X-ray tube should be placed further away from the area being imaged.

If the X-ray tube is too close, imaging quality can degrade and radiation dose increases. This strategic arrangement ensures that the image quality remains high while minimizing patient exposure to radiation.

 Tip

See a visual summary of correct and incorrect technique [here](#).

Another crucial aspect of radiation exposure is a person's **distance from the radiation source**. Radiation exposure decreases as the distance from the source increases. This phenomenon follows an **inverse square law** ( $1/d^2$ ). (J. H. Kim 2018)

 Note

Suppose a medical student is initially **one foot away** from a radiation source, and their exposure is measured at **500 mGy/min**. If they then move **ten feet away** from the source, their exposure would reduce significantly to **5 mGy/min**. This example vividly demonstrates why distance from the source plays a pivotal role in minimizing radiation exposure.

 Tip

See a visual summary of the inverse square law and distance from radiation source [here](#).

 Tip

If you are not scrubbed, the ideal place to stand is as far away from the x-ray source while making sure you can still see the images and interact with the surgical team.

When discussing the positioning of the C-arm, it's useful to become familiar with some common terminology. To improve visualization of the target area, the C-arm can rotate left and right, and cranially or caudally. **All terminology is relative to the radiation source (under the table) and not the intensifier (above the patient)**. Two frequently encountered positions are **RAO (right anterior oblique)** and **LAO (left anterior oblique)**.

 Tip

See a visual summary of C-arm rotation [here](#).

# Adverse Outcomes Given Prolonged Radiation Exposure

## Limits

Various governing agencies in the US and around the world have established limits on yearly radiation exposure.

Currently, the United States Occupational Safety and Health Administration, Nuclear Regulatory Commissions, and Department of Energy all stipulate a **yearly limit of 5,000 mrem/year (50 Sv)**. [“Radiation In Perspective” (n.d.)] (“Information for Radiation Workers,” n.d.)

Some agencies further specify this **limit to 1,250 mrem/quarter (12.5 Sv)**. (“Radiation Emergency Preparedness and Response - Response | Occupational Safety and Health Administration,” n.d.)

## Deterministic versus stochastic radiation effects

Before delving into the various risks associated with radiation exposure, it’s essential to provide a brief overview of the types of radiation effects. These effects can be broadly categorized into deterministic and stochastic effects.

**Deterministic effects**, also known as non-stochastic effects, occur when specific radiation dose thresholds are exceeded leading to adverse outcomes. They are **dose-dependent** and result in significant damage, such as cell death in a specific organ. Examples of deterministic effects include cataracts, infertility, and non-malignant skin lesions. (Weerakkody et al. 2008a) Making sure thresholds are not surpassed is pertinent not only for individuals working in the endovascular suite but also for ensuring patient safety.

The table below outlines the various adverse deterministic (non-stochastic) effects associated with radiation doses in comparison to the average exposure during endovascular aneurysm repair (EVAR). (Weerakkody et al. 2008a)

### Radiation absorbed dose and associated side effect compared to EVAR

System	Side Effect	Source	Dose (Gy)	Average EVAR dose (Gy) [31]
Integumentary	Early transient erythema	Stewart, et. al.	Single dose: 2	.85 median (interquartile range .51 - 3.74)
	Burns	Williams	Single dose: 5-10	”
Reproductive	Sterility	Williams	Single dose: 3-6	”



System	Side Effect	Source	Dose (Gy)	Average EVAR dose (Gy) [31]
Thyroid	Hypothyroidism	Nagayama	Cumulative dose High dose: 15-44 Low dose: variable	”
Ocular	Cataracts	Williams, et. al.	Single dose .5	”

The table above underscores a crucial consideration: deterministic effects may potentially arise in cases with prolonged run times. Endovascular aneurysm repair (EVAR) is a routine procedure for modern vascular surgeons. While the duration of these procedures may vary, considering the cited range of radiation dosages from 0.51 to 3.74 (Weerakkody et al. 2008a), it’s conceivable that patients could experience post-operative adverse events solely due to the effects of radiation exposure.

In contrast, stochastic effects result from repeated exposure to radiation levels below a certain threshold. A notable example of stochastic effects is cancer. Stochastic effects are probabilistic in nature. For example, the likelihood of developing leukemia after a single chest X-ray is low. However, if someone were to undergo a head-to-toe CT scan daily for a year, the probability of developing leukemia would significantly increase, even though there isn’t a direct correlation between the dose of radiation and the observed effect. (Hamada and Fujimichi 2014)

### The Eye and Radiation Exposure:

During radiological procedures, one organ that often goes unnoticed but is significantly exposed is the **eye**, specifically the **lens**.

The lens of the eye is particularly **sensitive to radiation** .

- **Cataracts: Prolonged exposure** to significant radiation levels can lead to **opacification of the lens** (i.e. cataracts)
- This gradual process ultimately results in the development of **cataracts**, with the most common subtype being **posterior subcapsular (PSC) cataracts**. (Loganovsky et al. 2020)
- A study conducted in 2013 examined the dose-effect relationship of radiation on the eyes of interventional cardiologists and electrophysiologists. The study found that up to 25% of the physicians involved had exceeded current exposure thresholds and were at risk of developing radiation-induced cataracts. (Jacob et al. 2012)

Other ocular pathologies associated with radiation exposure include:

- **Cataracts:** Prolonged exposure to radiation may lead to the development of cataracts, causing clouding of the lens.
- **Glaucoma:** Radiation exposure can contribute to glaucoma, a condition characterized by increased intraocular pressure.
- **Optic Neuropathy:** Damage to the optic nerve due to radiation exposure can result in vision impairment.
- **Retinal Angiopathy:** Radiation may impact retinal blood vessels, leading to angiopathy.
- **Dry Eye Syndrome:** Radiation exposure can cause dryness and discomfort in the eyes.

In recent years, there has been **growing research interest** in understanding the potential adverse effects of **long-term radiation exposure** on ocular health.

The increased understanding of these risks has prompted a greater emphasis on the **importance of safety equipment** to protect the eyes. Proper use of protective gear such as **lead glasses and face shields** is crucial for minimizing radiation exposure to ocular tissues.

### **The Skin and Radiation Exposure:**

Skin changes of varying severity are a well-documented adverse reaction to radiation exposure. These changes can range from mild redness and irritation to severe necrosis and the development of secondary skin cancers. Importantly, these changes may not always manifest immediately.

Adverse effects to the skin vary according to the cumulative dose of radiation. For example, at the large cumulative dose of 50 Gy, fibrosis may occur, presenting as induration, skin retraction, edema, restricted motion, ulceration, and necrosis. (Bennardo et al. 2021) This underscores the importance of understanding a patient's radiation exposure history, as it could increase their susceptibility to radiation-induced injury.

The most commonly encountered adverse skin effect is **acute radiation dermatitis**.

- Acute radiation dermatitis characterized by redness and ulceration, can occur with relatively low doses, such as **2 Gy**. [Stewart et al. (2012)](Bennardo et al. 2021)
- This level of exposure is concerning not only for individuals undergoing routine radiation therapy for cancer treatment but also for those in prolonged surgeries requiring fluoroscopy, as highlighted in Weerakkody's study on endovascular aneurysm repair (EVAR). (Weerakkody et al. 2008b)
- Symptoms of **acute radiation dermatitis typically appear within 2 to 24 hours** following exposure.

- As the radiation dose increases, the severity of side effects worsens, and the time to onset of these effects also lengthens. For instance, a single radiation dose of 10 Gy may result in dermal atrophy, but this may not be evident until more than 10 weeks after exposure. (Stewart et al. 2012)

This discussion doesn't cover all possible adverse effects related to skin radiation exposure. Rather, it provides an overview of common findings based on typical exposure scenarios in vascular surgery patients. Further information on the effects of single and cumulative radiation doses on the skin can be found in studies by Stewart ((Stewart et al. 2012)) and Bennardo ((Bennardo et al. 2021)), respectively.

## Thyroid Dysfunction, Thyroid Cancer, and Radiation Exposure

It is widely acknowledged that **radiation exposure during childhood** significantly increases the risk of developing **thyroid cancer**. Key risk factors in this context include both the **dose of radiation received** and the **age at the time of exposure**.(Iglesias et al. 2017) However, when it comes to establishing a similar relationship in **adulthood**, the evidence has been less straightforward.

In **October 2023**, **The Journal of Radiation Research** published a **systematic review and meta-analysis** specifically examining the **incidence of thyroid cancer following radiation exposure in adults**. While some studies suggested an **increased risk of cancer** in adults exposed to radiation, the relative risk at a **dose of 10 mGy** (milligray) was **deemed insignificant**. In other words, the risk did not show a significant elevation at this particular dose level.

While a definitive link between radiation exposure and thyroid cancer in adult populations remains elusive, several studies have **hinted at a potential association** between **low-dose ionizing radiation** and **thyroid dysfunction**.

- There have been reports of hypothyroidism marked by elevated levels of thyroid-stimulating hormone (TSH) and lower-than-average levels of free triiodothyronine (T3) and thyroxine (T4) compared to unexposed individuals. (Cioffi et al. 2020)

Given the potential implications of radiation exposure to thyroid dysfunction and cancer, additional research into the long-term effects of ionizing radiation exposure on thyroid function is warranted. Numerous studies have emphasized the need for further investigation in this area.

## Reproductive Function and Radiation Exposure

One of the significant concerns associated with radiation exposure is its impact on reproductive health.

In the **female reproductive system**, ionizing radiation primarily affects the **ovaries**, potentially leading to:

1. **Ovarian Insufficiency**: Prolonged exposure to radiation may result in **ovarian insufficiency**, affecting the ovaries' ability to function optimally.
2. **Delayed Puberty**: Radiation exposure can also contribute to **delayed puberty** in females.
3. **Infertility**: Ultimately, these effects may culminate in **infertility**.

Some sources suggest that **radiation to the head** during routine medical procedures could disrupt the **hypothalamic-pituitary-gonadal (HPG) axis**. (Marci et al. 2018) This disruption may lead to hormonal dysregulation, further impacting reproductive health.

Radiation exposure to **male reproductive organs** can disrupt spermatogenesis, depending on the stage of sperm development. The damage from ionizing radiation may lead to decreased sperm motility, increased fragmentation, and DNA alterations. (Wdowiak et al. 2019)

## **Hematologic Disease and Radiation Exposure**

Prolonged low-dose radiation exposure has the potential to cause serious hematological effects. Numerous studies, including those conducted outside the medical field, have examined hematologically consequences of employees at risk of radiation exposure.

A particularly noteworthy study, published in 2015, involved an international cohort of observers within the nuclear industry. Their research revealed compelling evidence linking low-dose radiation exposure to the development of non-chronic lymphocytic leukemia (CLL). (Leuraud et al. 2015)

- Specifically, the study highlighted a **direct correlation** between cumulative red bone marrow dose over a two-year period and the excess relative risk of developing leukemia.
- Their findings indicated that the relative risk of leukemia mortality was approximately 2.96 per Gy of exposure (RR = 2.94 per Gy).

While we won't delve into the intricate details of these findings in this chapter, they underscore the critical importance of considering lifelong radiation exposure and the need for effective protective measures.

## Pregnant Populations

When it comes to **radiation exposure during pregnancy**, there are important guidelines and considerations to keep in mind.

- To monitor the radiation dose absorbed by the fetus, a dosimeter or radiation badge is typically worn under the lead apron at the level of the abdomen.
- This dosimeter is checked monthly, and the absorbed dose is calculated accordingly.
- **Fetal Harm Threshold:** Ensure that the radiation dose does not exceed **0.5 mSv per month** or a maximum fetal dose of **5 mSv** throughout the entire pregnancy

The following table adapted from a study by Vu et al. ((Elder and Vu 2013)) summarizes the deterministic effects of radiation during pregnancy.

Deterministic effect	Gestation (weeks)	Threshold dose (mGy)
Embryonic death	3 – 4	100 – 200
Major malformations	4 – 8	250 – 500
Growth retardation	4 – 8	200 – 500
Irreversible whole body growth retardation	8 – 15	250 – 500
Severe mental disability	8 – 15 >16	60 – 500 >1,500
Microcephaly	8 – 15	>20,000
Decrease in intelligence quotient	>16	>100

### Lead Thickness Considerations for Pregnant Providers:

- **Minimum Thickness:** Pregnant individuals should wear a lead apron with a minimum thickness of **0.5 mm**. This ensures proper coverage of the abdomen, shielding the fetus from radiation exposure.
- **Double-Thickness Option:** Alternatively, a double-thickness lead apron (1.0 mm) can be worn. According to Vu et al. ((Elder and Vu 2013)), this reduces the absorbed dose from **9 mrem** to **3 mrem**.
- **Weight Consideration:** However, it's essential to recognize that wearing a thicker lead apron may increase the risk of **back injury** due to the added weight. Balancing protection and comfort is crucial.

As always, healthcare providers should adhere to the ALARA principles outlined above (i.e. maintaining a maximum reasonable distance from the source, using the collimator, pulse imaging, etc.).

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## Teaching Case

### Scenario

A 75-year-old male is scheduled for an angiogram of the left lower extremity to investigate and potentially treat claudication symptoms. The attending vascular surgeon, with over 25 years of experience in endovascular therapy, takes precautionary measures by wearing a thyroid shield, prescription glasses, and a 2-piece lead apron. However, due to challenges in navigating the stenosed arterial system, the fluoroscopy time during the procedure was extended. Subsequently, the calculated radiation dose amounted to 3 Gy.

### Discussion Points

**i** N.B. There is no prepared answers for the questions below.

However, we feel this chapter contains all the necessary information to answer the questions. If not, please let us know!

1. What could be done to reduce the dose of radiation to the surgeon?
2. What could be done to reduce the dose of radiation to the OR staff?
3. What gaps are present in the surgeon's PPE?
4. What are some potential adverse effects of prolonged radiation exposure to the surgeon?
5. Given the level of radiation, what adverse effects might be noted in the patient?
6. If the case were to progress longer, what might be the subsequent adverse outcomes to the patient?

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## Key Articles

1. Mitchell EL, Furey P. Prevention of radiation injury from medical imaging. *J Vasc Surg.* 2011 Jan;53(1 Suppl):22S-27S. doi: 10.1016/j.jvs.2010.05.139. Epub 2010 Sep 16. PMID: 20843625. (Mitchell and Furey 2011)

## Additional Resources

### Audible Bleeding Content

- Audible Bleeding Exam Prep: [Radiation Safety](#)
  - Special Topic: Occupational Hazards for the Vascular Surgeon. Listen to the episode below and find additional information [here](#), or find the episode wherever you listen to podcasts.
  - JVS Author Spotlight - Jasmine Bhinder, MD. Listen to the episode below and find additional information [here](#), or find the episode wherever you listen to podcasts.
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### Gore Combat Manual

The [Gore Medical Vascular and Endovascular Surgery Combat Manual](#) is an informative and entertaining read intended as a vascular surgery crash course for medical students, residents, and fellows alike. Highly accessible with a thoughtfully determined level of detail, but lacking in learning activities (e.g. questions, videos, etc.), this resource is a wonderful complement to the APDVS eBook.

#### Tip

Please see pages 43-46.

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# Anonymous Feedback

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We welcome suggestions for updates. We want to hear from you! Please help us help you!

To this end, please complete this anonymous [Google Form](#) to send us any feedback you may have.

Thank you for your continued support!





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