Contemporary Management in Vascular Surgery

Moderator:

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Center for Cardiovascular Health

What's New in the Treatment of Iliofemoral Deep Vein Thrombosis (DVT)

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Learning Objectives

- Review recent evidence and guidelines/position updates for iliofemoral DVT treatment
- Summarize new endovascular devices and techniques (lytic sparing approaches)
- Review clinical evidence and what to watch out for
- Translate evidence into practical patient selection and post procedure care
- Clinical case

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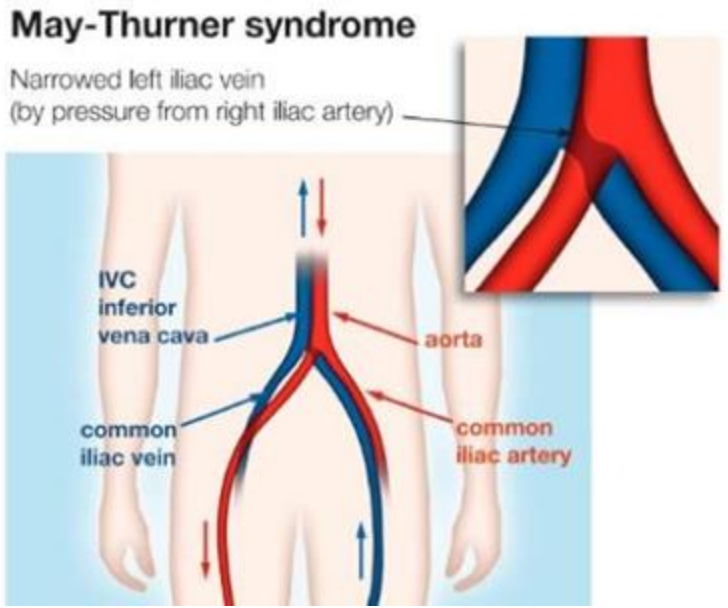
Why Iliofemoral DVT is Different?

- Proximal thrombosis of iliac - femoral veins
- Risk factors:
  - Bed rest /inactivity
  - Surgery
  - Trauma
  - Pregnancy
  - Hypercoagulability (genetic, cancer)
  - Iliac vein compression (aka May-Thurner syndrome)
- Phlegmasia cerulea dolens
- Pulmonary embolism
- Post thrombosis syndrome (PTS)



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Post Thrombotic Syndrome (PTS)

- PTS Iliofemoral DVT -> PTS:
  - Venous outflow obstruction
  - Residual thrombus
  - Chronic valvular damage
  - Venous claudication:
    - Leg pain, fatigue, swelling, skin changes, ulcers
  - 20-50%, >10% severe, lifelong condition, affects QOL, no effective treatment
  - Lack of endoluminal recanalization ~ 6 months is a predictor of PTS

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Current Clinical Guidelines

CHEST (2021):

- 3 months of DOAC over VKA therapy
- Discourages routine IVC filter use
- Anticoagulation preferred over intervention

Society for Vascular Surgery (SVS):

- CDT or MT may reduce PTS risk (Level A, class IIa)
- Aims to restore venous patency, preserve endothelial function, and improve long-term outcomes

Society of Interventional Radiology (SIR):

- Endovascular thrombus removal acceptable for selected patients with severe symptoms, extensive thrombus, low bleeding risk, and early presentation

Iliofemoral DVT Treatment Options

Anticoagulants

DOAC[1]

Prevent thrombin from acting on fibrinogen to produce fibrin

- Direct Thrombin Inhibitors
  - Ex: Pradaxa
- Factor Xa Inhibitors
  - Ex: Xarelto

Warfarin[2]

Prevents creation of thrombin by inhibiting vitamin K-dependent clotting factors II, VII, IX, and X

Heparin[3]

Prevents thrombin from producing fibrin by activating anti-thrombin III, which inactivates thrombin and Factor Xa

Thrombolytics[4]

Clot-busting drugs work by use of plasmin (which is the body's way of degrading fibrin)

1. Kustos, S et al. Direct-Acting Oral Anticoagulants and Their Reversal Agents– an Update. Medicines (Basel). 2019 Dec; 6(4): 103.
2. Hirsh, J et al. Mechanism of Action and Pharmacology of Unfractionated Heparin. Arteriosclorosis, Thrombosis, and Vascular Biology. Vol. 21, No. 7. July 2001.
3. Incampo, F et al. Effect of warfarin treatment on thrombin activatable fibrinolysis inhibitor (TAFI) activation and TAFI-mediated inhibition of fibrinolysis. J Thromb Haemost. 2013 Feb;11(2):315-24.
4. Collen, D et al. Fibrin-Selective Thrombolytic Therapy for Acute Myocardial Infarction. Circulation. Vol. 93, No. 5. March 1996.

The majority of venous clot is lytic-resistant by the time of treatment

>70% of clot removed at **time of treatment** is resistant to lytics (non-fibrin).[1]

Clot formation

DAY 3	DAY 7	DAY 15	DAY 21
Fibrin-Rich Matrix[2]	~20% Collagen[2]	>50% Collagen[2]	~80% Collagen[2]

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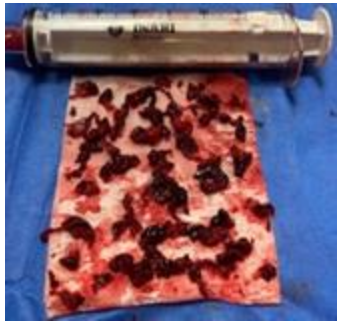
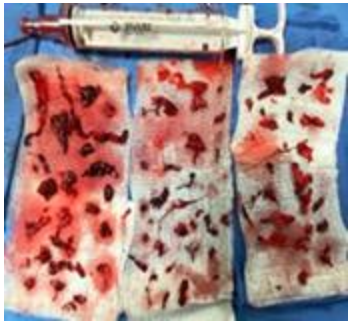
1. Silver, et al. Histopathologic analysis of extracted thrombi from deep venous thrombosis and pulmonary embolism: Mechanisms and timing. Catheter Cardiovasc Interv. 2021 Jun 1;97(7):1422-1429.
2. Czaplicki, et al. Can thrombus age guide thrombolytic therapy? Cardiovasc Diagn Ther. 2017 Dec;7(Suppl 3):S186-S196.

Most venous clot does not respond to thrombolytics[1,2]

Acute → Chronic

Lower extremity thrombus





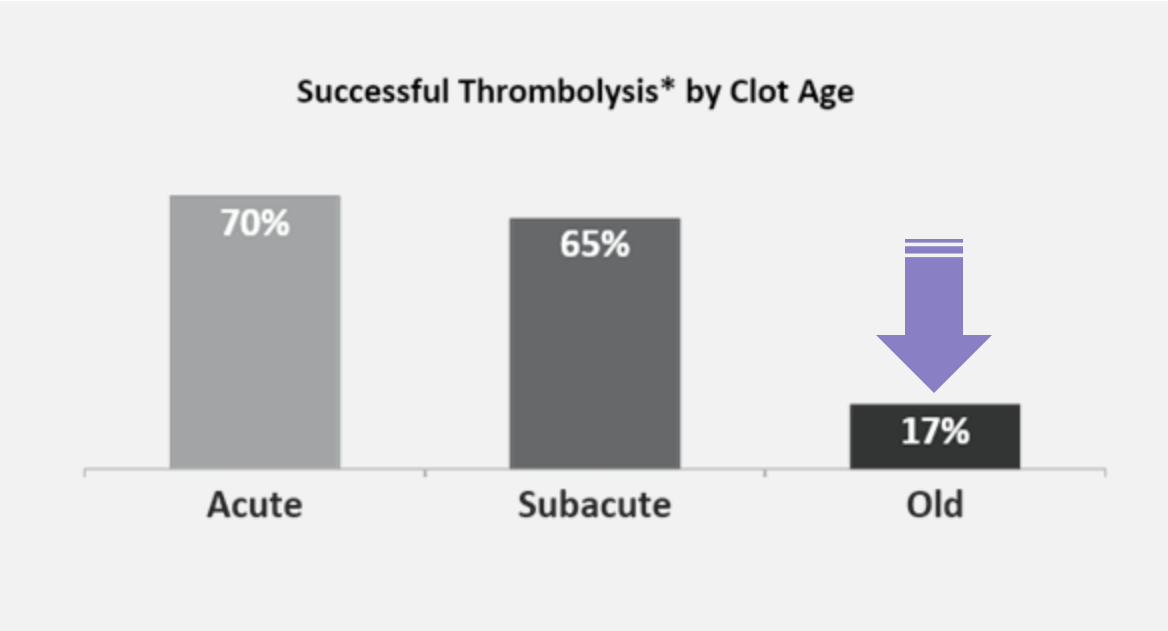

Pulmonary thrombus




1. Goldhaber SZ, et al. Randomized controlled trial of tPA in proximal DVT. *Am J Med*. 1990; 88: 235-240.

Lysis fails a significant proportion of time, especially in older, more chronic DVT

Arnoldussen, et al. 2022[1]*: Sub-analysis of the CAVA trial (n=56). Clinical impact of assessing thrombus age using magnetic resonance venography prior to catheter-directed thrombolysis.*



DVT thrombus is commonly older than suggested by symptom duration.

CDT was less successful in cases with old thrombus.

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*Defined as regained venous patency of > 90% on control angiography, performed every 24h.

1. Arnouldussen, et al. European Radiology 2022 Jul; 32(7):4555–4564

Residual thrombus is common after conservative treatment

Up to

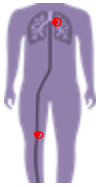
50%

of DVT patients have **residual vascular obstruction (RVO)** after AC or tPA[1-6]

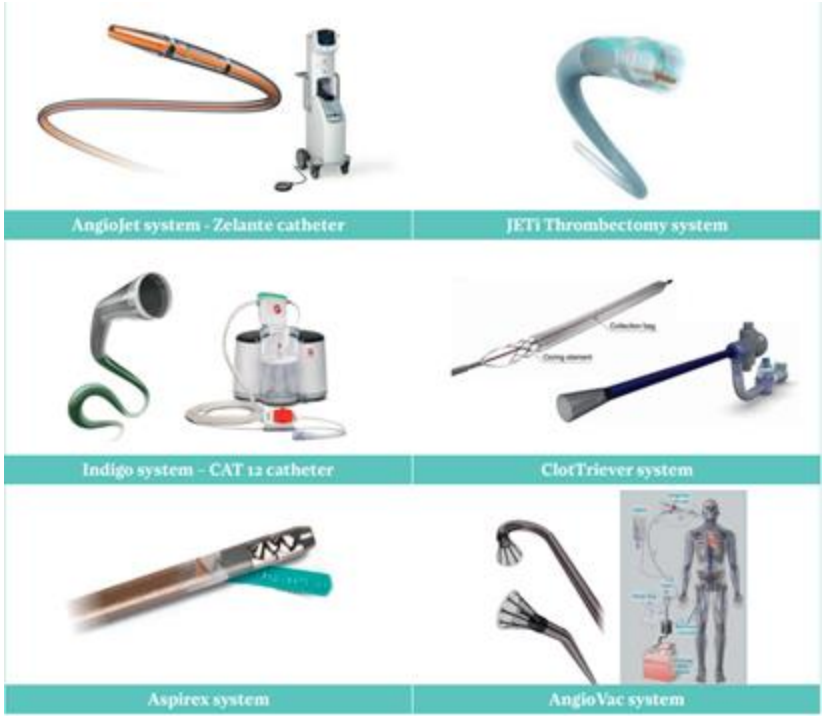
Study	Year	Patients (N)	Treatment	% with RVO	Time for Assessment	Threshold
Young[1]	2006	316	AC	**55%**	60 mo.	n/s
Yoo[2]	2018	156	AC	**61%**	8 mo.	> 40%
Aziz[3]	2012	75	CDT	**11%**	36 mo.	> 50%
Prandoni[4]	2002	313	AC	**13%**	72 mo.	n/s
Dronkers[5]	2018	2,684	AC	**36%**	6 – 72 mo.	n/s
Avgerinos[6]	2019	142	CDT, PMT + Stenting	**67%**	Index	> 50%

1. Young et al., Post-treatment residual thrombus increases the risk of recurrent deep vein thrombosis and mortality. J Thromb Haemost 2006; 4: 1919–24.
2. Yoo et al., Presence and degree of residual venous obstruction on serial duplex imaging is associated with increased risk of recurrence and progression of infrainguinal lower extremity deep venous thrombosis. Copyright 2018 by the Society for Vascular Surgery. Published by Elsevier Inc.
3. Aziz et al., Predictors of Outcome in Iliofemoral Deep Venous Thrombosis Treated by Catheter-directed Thrombolysis. 2012 European Society for Vascular Surgery. Published by Elsevier Ltd.
4. Prandoni et al., Residual Venous Thrombosis as a Predictive Factor of Recurrent Venous Thromboembolism. Ann Intern Med. 2002;137:955-960.
5. Dronkers et al., Predicting Post-Thrombotic Syndrome with Ultrasonographic Follow-Up after Deep Vein Thrombosis: A Systematic Review and Meta-Analysis. Thromb Haemost 2018;118:1428–1438.
6. Averginos et al., Outcomes and predictors of failure of iliac vein stenting after catheter-directed thrombolysis for acute iliofemoral thrombosis. Copyright 2018 by the Society for Vascular Surgery. Published by Elsevier Inc.

Treatment of DVT evolved to definitive mechanical catheter intervention

	Anti-Coagulation **(AC) Only**	AC + **Thrombolytics (Lytics)**	AC + **Definitive Catheter Intervention**	
 **Myocardial Infarction**	AC alone	AC + **Thrombolysis**	AC + **POBA & DES**	
 **Stroke**	AC alone	AC + **Systemic Lytics**	AC+ **Stentrievers & Aspiration Thrombectomy**	
 Expected Path for **VTE (DVT & PE)**	AC alone	AC+ **Systemic & Catheter-directed Lytics**	AC+ **Pharmacomechanical thrombectomy**	

New Devices and Techniques



Pharmacomechanical thrombectomy and venous stenting

Evolving Clinical Evidence

- **ATTRACT trial (2017):** Anticoagulation vs. pharmacomechanical catheter-directed thrombolysis (PCDT) in 692 acute DVT patients; 311 iliofemoral DVT patients showed lower PTS and improved QOL with PMT
- **ZelanteDVT Single**-Session Study (2024): High technical success, symptom resolution, and ~91% primary patency at 1 year
- Multiple registries and institutional studies on mechanical thrombectomy device safety and effectiveness, but lack long-term data
- **DEFIANCE** randomized industry sponsored trial: Inari ClotTriever vs anticoagulation alone, results will clarify the role of mechanical thrombectomy in PTS outcomes

Patient Selection

- Thrombus acuity <21 days, recent symptoms
- Type of patient - young, active, generally healthy
- Severity of symptoms - proximal DVT, severe edema, venous claudication, phlegmasia
- Temporal evolution of symptoms
- Underflying ethology - iliac vein compression
- **Avoid:**
  - Active bleeding, recent major surgery/stroke, high bleeding risk, limited life expectancy and functional capacity
- Multidisciplinary decision making is recommended

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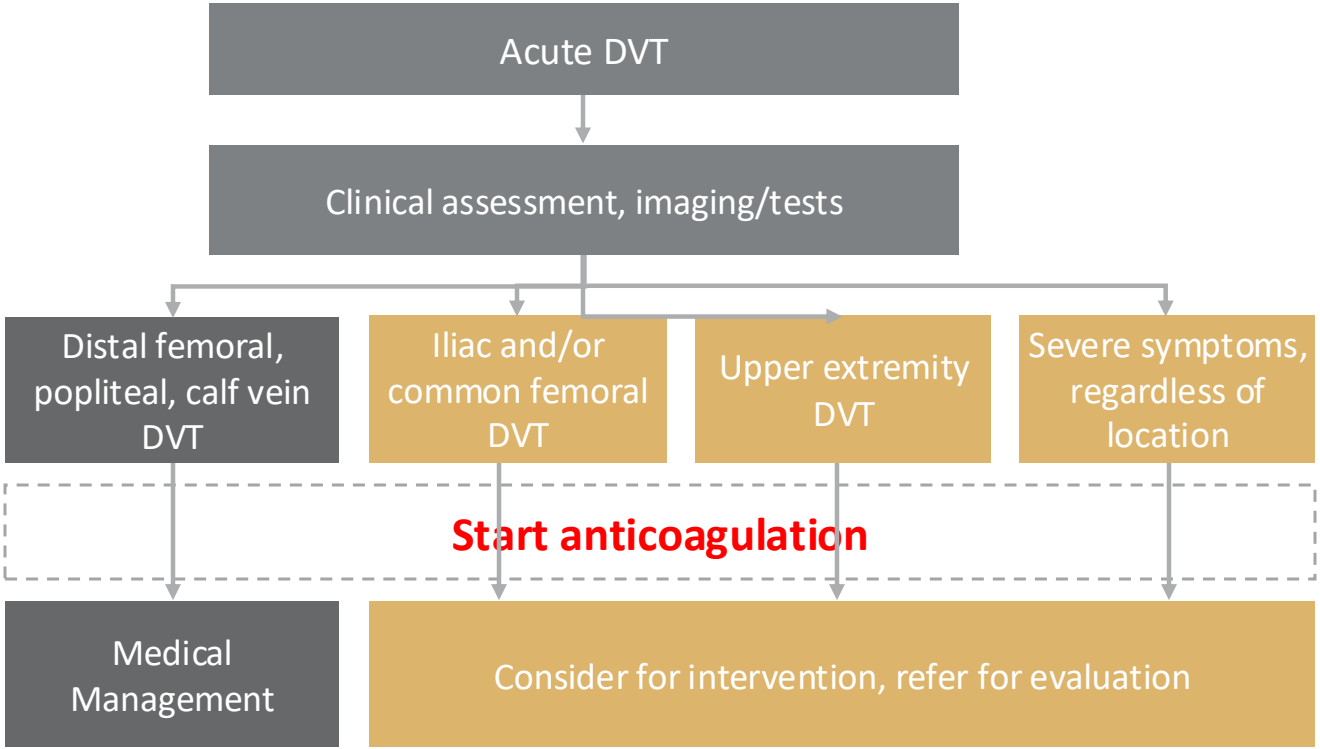
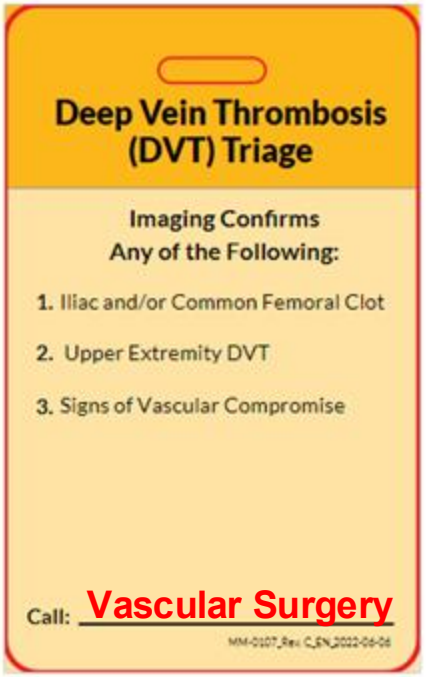
Post Procedure Care

- DOAC is the preferred treatment unless contraindicated
- Treatment typically lasts 3-12 months, with longer durations for unprovoked, recurrent, or stented cases
- Antiplatelet therapy may be included, especially if a venous stent is placed
- This area of practice is still evolving, leading to variations in treatment
- 20-30 mmHg compression is recommended to reduce swelling
- Follow-up includes duplex ultrasound at 1, 6, and 12 months, then annually
- Cross-sectional imaging for complex iliocaval reconstructions

DVT Triage


Acute DVT
Clinical assessment, imaging/tests
Distal femoral, popliteal, calf vein DVT
Iliac and/or common femoral DVT
Upper extremity DVT
Severe symptoms, regardless of location
Start anticoagulation
Medical Management
Consider for intervention, refer for evaluation


Deep Vein Thrombosis (DVT) Triage
Imaging Confirms Any of the Following:
1. Iliac and/or Common Femoral Clot
2. Upper Extremity DVT
3. Signs of Vascular Compromise
Call: Vascular Surgery

Clinical Case

- 54yo F with RLE pain and swelling
- R common -external iliac- common femoral vein thrombus on US and CT venogram
- 7 Passes with Inari ClotTriever Bold to remove acute and chronic thrombus, 90% clearance
- No lytics, 25cc EBL
- 16X120 & 14X60 Venovo stents relieve compression seen on IVUS from the external to the common Iliac Vein
- 1.5 year patency and symptom relief

Key Takeaways

- Treatment of iliofemoral DVT evolved towards endovascular options in selected patients
- Lytic sparing devices reduce complications, hospital stay and might improve long term outcomes
- Randomized evidence is pending (DEFIANCE) and will help guide future standard of care
- Unanswered questions: timing, durability, optimal anticoagulation after stenting

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Updates in the Treatment of Lower Extremity Wounds: Is it Arterial or Venous? A Stepwise Approach

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St. Michael Medical Center

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Wounds That Won’t Heal:
A Vascular Surgeon’s Perspective

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Disclosures

None

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What is a wound?

Noun: “an injury to the body (as from violence, accident or surgery) that typically involves laceration or breaking of a membrane (such as the skin) and usually damage to the underlying tissues”

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Merriam-Webster English Dictionary

Normal wound healing phases

- Hemostasis
- Inflammation
- Proliferation
- Epithelialization/Remodeling

- Typically heals in 4 weeks or less

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Chronic wounds

- Lack of healing after 4 weeks – 3 months

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Why do chronic wounds matter?

- Since 2014, chronic wound treatment costs Medicare upwards of $20 billion annually
    - Shift from hospital-based to office-based care (and therefore costs)
- 10.5 million people in the United States affected by chronic wounds
- Up to 85% of amputations are preceded by chronic wounds
- Venous ulcers affect 1-3% of the U.S. population
- Chronic wounds significantly affect on quality of life

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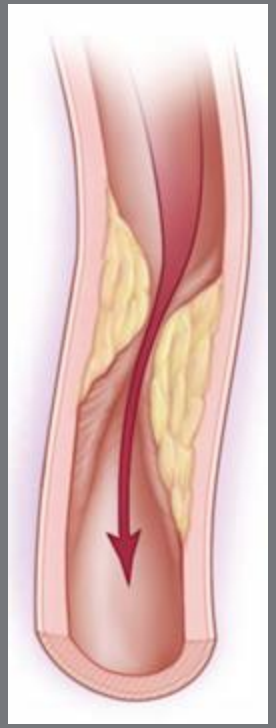
Sen, C. K. (2025). Human Wound and its Burden: Updated 2025 Compendium of Estimates. *PubMed*. https://doi.org/10.1177/21621918251359554
Millan, S. B., Gan, R., & Townsend, P. E. (2019). Venous ulcers: Diagnosis and treatment. *PubMed*, *100*(5), 298–305. https://pubmed.ncbi.nlm.nih.gov/31478635
Bowers, S., & Franco, E. (2020). Chronic Wounds: evaluation and management. *PubMed*, *101*(3), 159–166. https://pubmed.ncbi.nlm.nih.gov/32003952

Etiology	Example	1st Diagnostic Investigation
Infectious	Fungal, mycobacterial, bacterial	Wound bx & cx, CBC, CSR, X-ray
Malignant	Basal cell CA, squamous cell CA	Wound Bx
Arterial insufficiency – macro	Atherosclerosis (PAD), thromboembolic dz	Duplex & physiologic arterial studies (ABI)
Arterial insufficiency - micro	DM, Raynaud's, Buerger's,	$TcPO_2$, toe pressure/TBI
Venous insufficiency – deep & superficial	DVT, extrinsic compression, valvular reflux	Duplex venous studies
Lymphatic obstruction	Lymphatic insufficiency, malignancy, secondary to an "overload state"	H&P
Hematologic abnormality	PCV, Sickle cell	CBC, iron studies
Collagen vascular disorder	Scleroderma, lupus, Wegener	Rheumatologic work-up
Excessive pressure	Neuropathy (diabetic, alcoholic), paraplegia, bone spurs	Neuro exam (monofilament & vibratory), x-ray

Sidawy, A. N., & Perler, B. A. (2018). Rutherford's vascular surgery and endovascular therapy. In *Elsevier eBooks*. https://ci.nii.ac.jp/ncid/BB26259348

Vascular Diagnoses

Lower Extremity Peripheral Arterial Disease - PAD



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Lower Extremity Peripheral Arterial Disease (PAD)

- 8-12 million Americans affected
  - 3-10% of the American population
- Increasing prevalence with increasing age
  - Affects 15-20% of patients older than 70
- Fewer than 40% will report symptoms

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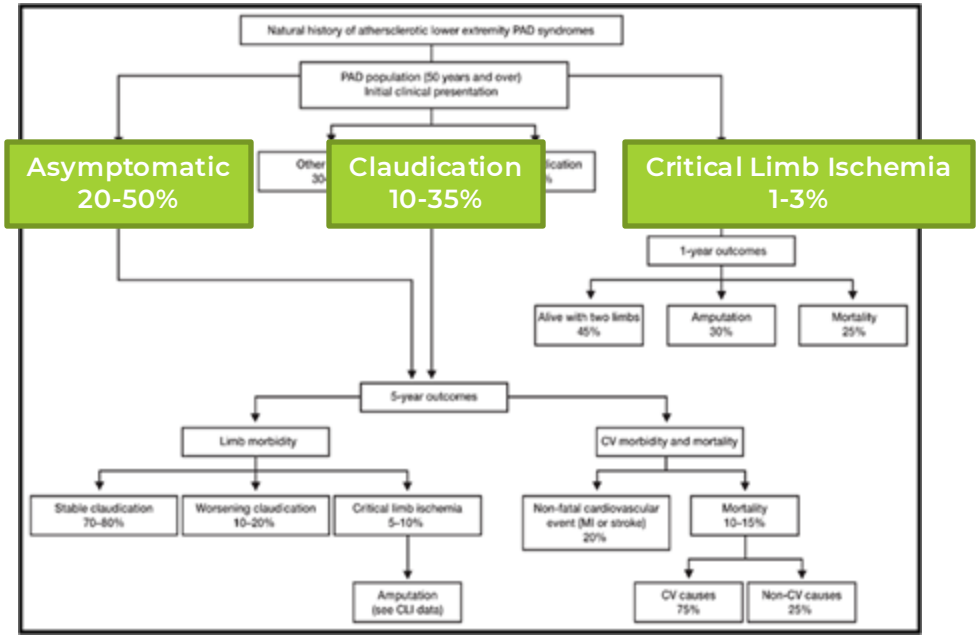
Conte, Michael S., et al. “Society for Vascular Surgery Practice Guidelines for Atherosclerotic Occlusive Disease of the Lower Extremities: Management of Asymptomatic Disease and Claudication.” *Journal of Vascular Surgery*, vol. 61, no. 3, 2015.
Hirsch AT, et al. JAMA 2001;286:1317-24

PAD – Natural History



Five-year history of a claudicant

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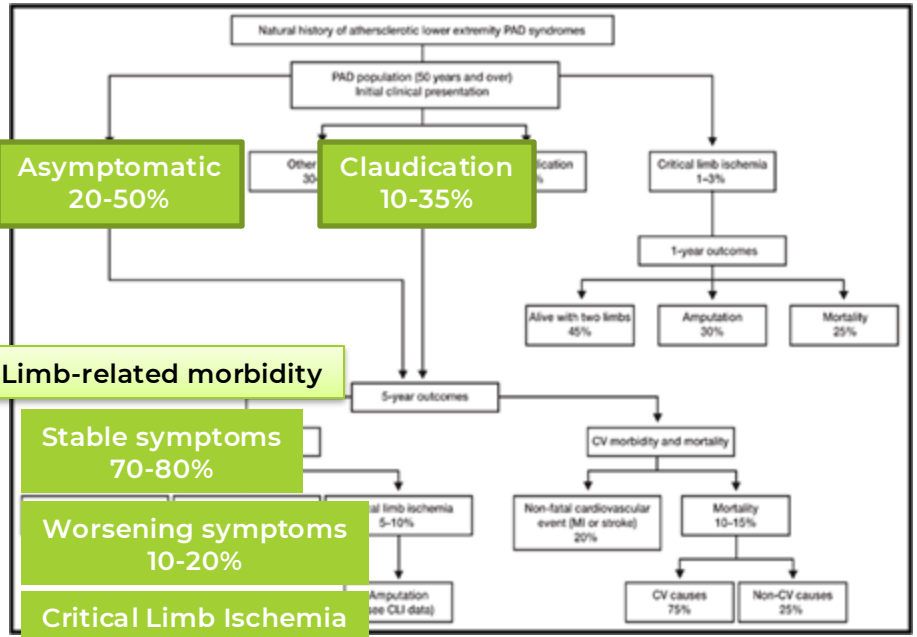
Norgren, L, et al. J Vasc Surg 2007; 45 Suppl S: S5-67

PAD – Natural History



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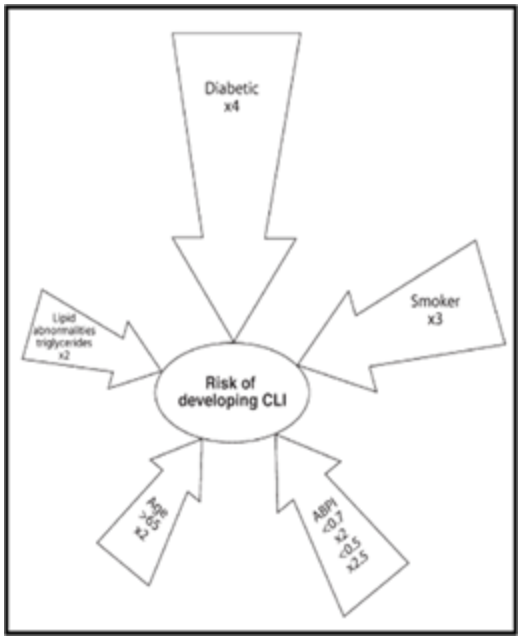

Magnitude of the effect of risk factors in PAD patients

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Norgren, L, et al. J Vasc Surg 2007; 45 Suppl S: S5-67

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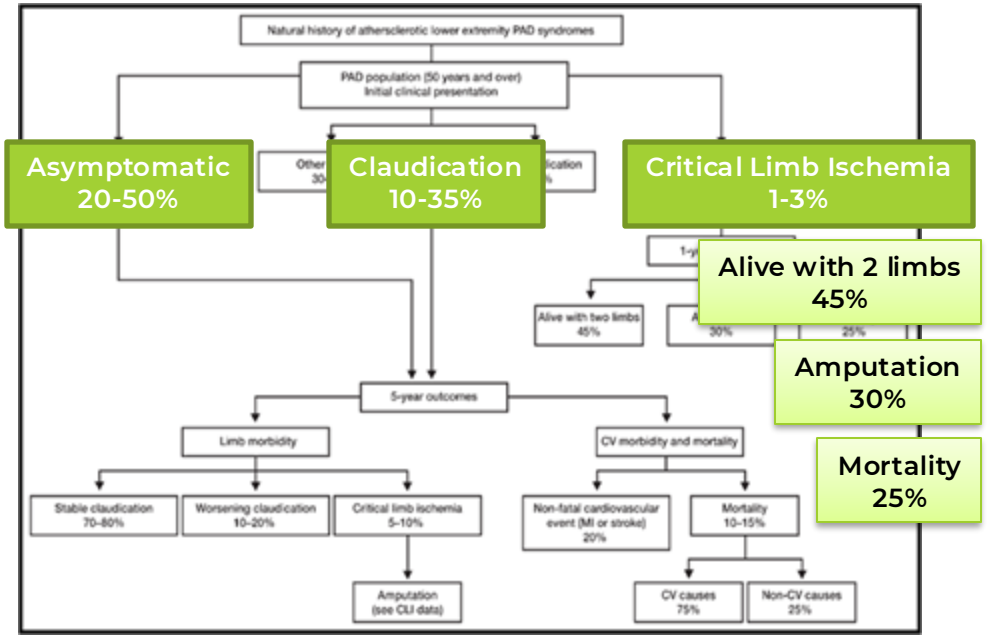

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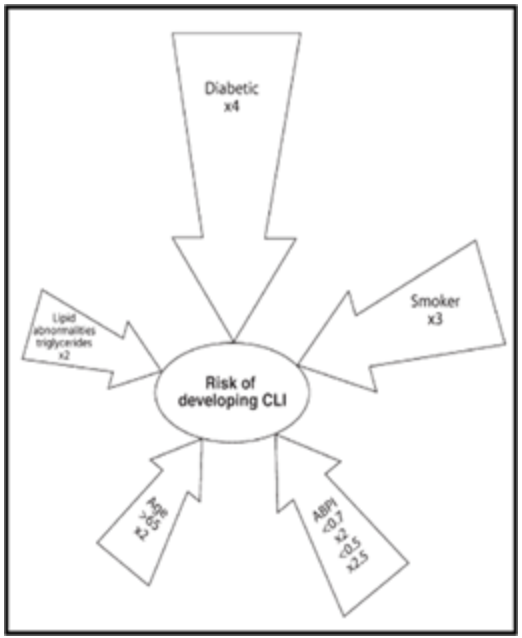

Magnitude of the effect of risk factors in PAD patients

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Norgren, L, et al. J Vasc Surg 2007; 45 Suppl S: S5-67

What is Critical Limb Ischemia (CLI)?

- Ischemic rest pain
- **Tissue loss/non-healing ulcer**

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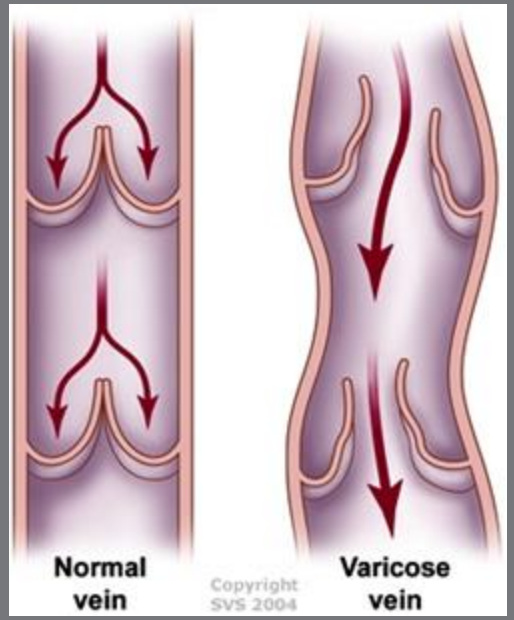
Vascular Diagnoses

Venous Disease – Venous Insufficiency and Varicose Veins



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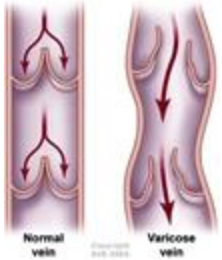
Venous Insufficiency



- Can occur in anyone
  - Major genetic component
  - More common in women
- Varicose veins are present in up to 35% of people in the U.S.

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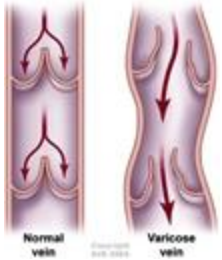
https://vascular.org/patient-resources/vascular-conditions/varicose-veins

Venous Insufficiency - symptoms



- Leg swelling/edema
- Pain, itching, burning
- Leg heaviness/tiredness
- Skin discoloration
- **Ulcerations**

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https://vascular.org/patient-resources/vascularcondtions/varicose-veins

What differentiates ulcers of arterial vs. venous etiologies?

	Arterial	Venous
History	History suggestive of PAD, claudication, rest pain	History of varicosities, DVT, known venous insufficiency
Classic site	Toes, foot, ankle, pressure points	“gaiter” distribution
Edges	“punched out” appearance	Sloped, shallow
Wound bed appearance	Varying degrees of slough & necrotic tissue	Often covered with slough
Exudate level	Low	High
Pain	Painful, even without infection (variable with associated neuropathy)	Pain associated with infection & edema
Edema	Not common	Common
Associated features	Shiny, thin skin, hairlessness	Lipodermatosclerosis, hemosiderosis

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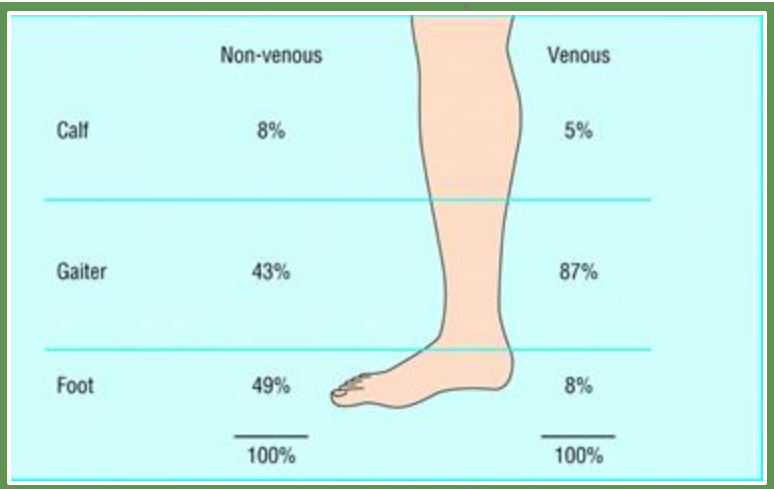
Grey, J. E., Harding, K. G., & Enoch, S. (2006). Venous and arterial leg ulcers. *BMJ*, *332*(7537), 347–350. https://doi.org/10.1136/bmj.332.7537.347
Singer, A. J., Tassiopoulos, A., & Kirsner, R. S. (2017). Evaluation and management of Lower-Extremity ulcers. *New England Journal of Medicine*, *377*(16), 1559–1567. https://doi.org/10.1056/nejmra1615243

Arterial

Venous



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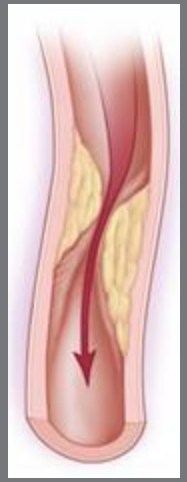
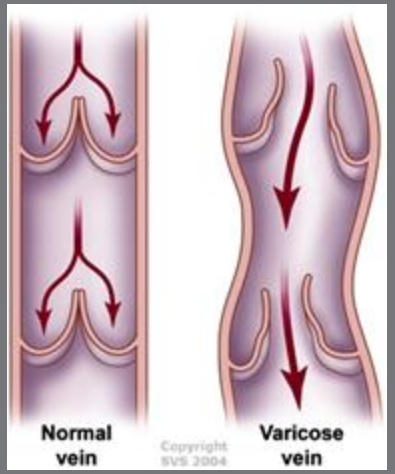
London, N. J. M. (2000). ABC of arterial and venous disease: Ulcerated lower limb. *BMJ*, *320*(7249), 1589–1591. https://doi.org/10.1136/bmj.320.7249.1589
Singer, A. J., Tassiopoulos, A., & Kirsner, R. S. (2017). Evaluation and management of Lower-Extremity ulcers. *New England Journal of Medicine*, *377*(16), 1559–1567. https://doi.org/10.1056/nejmra1615243

Vascular Evaluation

Lower Extremity Peripheral Arterial Disease - PAD

Venous Disease – Venous Insufficiency and Varicose Veins





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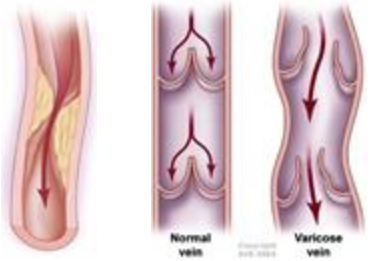
Evaluation



- Exam:
  - Description of limb
  - Pulse exam – palpation & doppler
    - Groins to feet
- Studies:
  - ABIs/TBIs/$TcPO_2$
  - +/- Arterial duplex
  - +/- Venous insufficiency duplex

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Inflow Before Outflow

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Non-healing lower extremity ulcer

↓

ABI/TBI

Recommendations for Resting ABI and Additional Physiological Testing (Continued)		
COR	**LOE**	**Recommendations**
2a	**B-NR**	10. In patients with CLTI with nonhealing wounds or gangrene, it can be useful to use toe pressure/TBI with waveforms, $TcPO_2$, SPP, and/or other local perfusion measures to determine the likelihood of wound healing without or after revascularization.[13,14,28,33-36,38]

Gornik, H. L., Aronow, H. D., Goodney, P. P., Arya, S., Brewster, L. P., Byrd, L., Chandra, V., Drachman, D. E., Eaves, J. M., Ehrman, J. K., Evans, J. N., Getchius, T. S., Gutiérrez, J. A., Hawkins, B. M., Hess, C. N., Ho, K. J., Jones, W. S., Kim, E. S., Kinlay, S., . . . Wilkins, L. R. (2024). 2024 ACC/AHA/AACVPR/APMA/ABC/SCAI/SVM/SVN/SVS/SIR/VESS Guideline for the Management of Lower Extremity Peripheral Artery Disease: A report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation, 149*(24). https://doi.org/10.1161/cir.0000000000001251
Hedayati N, Carson JG, Chi Y-W, Link D. Management of mixed arterial venous lower extremity ulceration: A review. *Vascular Medicine*. 2015;20(5):479-486. doi:10.1177/1358863X15594683

Non-healing lower extremity ulcer

ABI/TBI

Evidence of venous insuff/edema & ABI>0.5 (>40mmHg) – start compression therapy

ABI<0.5 (<40mmHg) – more urgent revascularization

ABI >0.7

ABI <0.7

Treat underlying conditions

Additional imaging – arterial duplex, CTA, angiogram

Revascularization

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Circulation

CLINICAL PRACTICE GUIDELINES

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Developed in Collaboration With and Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation, American Podiatric Medical Association, Association of Black Cardiologists, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine, Society for Vascular Nursing, Society for Vascular Surgery, Society of Interventional Radiology, and Vascular & Endovascular Surgery Society

CLTI	Severe clinical subset of PAD.
	Among patients with known PAD, incidence of CLTI estimated to be between 11% and 20%.[16–18]
	Manifests as ischemic rest pain, nonhealing wounds/ulcers, or gangrene with symptoms present for >2 wk.[19]
	Responsible for most major and minor limb amputations related to PAD.[16,17]
	Historically estimated 1-y mortality rate of 25%-35% and 1-y rate of amputation up to 30% among patients presenting with CLTI.[20,21]
	Lower rates of mortality and amputation reported in a recent RCT of patients with CLTI undergoing revascularization.[22]
	Ischemic rest pain often affects the forefoot and is worsened with limb elevation and relieved by dependency.
	Among vascular specialists, the Fontaine[23] and Rutherford[24] classification systems are most commonly used to categorize severity of CLTI.
	The WIfI classification estimates risk of lower extremity amputation according to wound extent, severity of ischemia, and presence of foot infection and has been shown to correlate with clinical outcomes.[18,25–28]

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Revascularization Goals for CLTI		
1	B-R	1. In patients with CLTI, surgical, endovascular, or hybrid revascularization techniques are recommended, when feasible, to minimize tissue loss, heal wounds, relieve pain, and preserve a functional limb.[1–14]
1	C-EO	2. In patients with CLTI, an evaluation for revascularization options by a multispecialty care team is recommended before amputation (Table 15).
Revascularization Strategy for CLTI		
1	A	3. In patients undergoing surgical revascularization for CLTI, bypass to the popliteal or infrapopliteal arteries (ie, tibial, pedal) should be constructed with autogenous vein if available.[14–20]

2a	B-NR	6. In patients with CLTI for whom a surgical approach is selected and a suitable autogenous vein is unavailable, alternative conduits such as prosthetic or cadaveric grafts can be effective for bypass to the popliteal and tibial arteries.[21–26]
2a	B-NR	7. In patients with CLTI and nonhealing wounds or gangrene, revascularization in a manner that achieves in-line blood flow or maximizes perfusion to the wound bed can be beneficial.[27–33]

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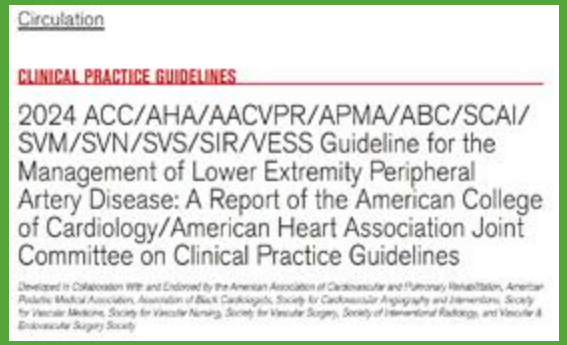
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SOCIETY FOR VASCULAR SURGERY® DOCUMENT

The Society for Vascular Surgery Lower Extremity Threatened Limb Classification System: Risk stratification based on Wound, Ischemia, and foot Infection (WIfI)

Joseph L. Mills, Sr, MD,[a] Michael S. Conte, MD,[b] David G. Armstrong, DPM, MD, PhD,[c] Frank B. Pomposelli, MD,[b] Andres Schanzer, MD,[d] Anton N. Sidawy, MD, MPH,[e] and George Andros, MD,[f] on behalf of the Society for Vascular Surgery Lower Extremity Guidelines Committee, *Tucson, Ariz; San Francisco and Van Nuys, Calif; Brighton and Worcester, Mass; and Washington, D.C.*

W: Wound/clinical category
SVS grades for rest pain and wounds/tissue loss (ulcers and gangrene):
0 (ischemic rest pain, ischemia grade 3; no ulcer) 1 (mild) 2 (moderate) 3 (severe)

Grade	*Ulcer*	*Gangrene*
0	No ulcer	No gangrene
Clinical description: ischemic rest pain (requires typical symptoms + ischemia grade 3); no wound.		
1	Small, shallow ulcer(s) on distal leg or foot; no exposed bone, unless limited to distal phalanx	No gangrene
Clinical description: minor tissue loss. Salvageable with simple digital amputation (1 or 2 digits) or skin coverage.		
2	Deeper ulcer with exposed bone, joint or tendon; generally not involving the heel; shallow heel ulcer, without calcaneal involvement	Gangrenous changes limited to digits
Clinical description: major tissue loss salvageable with multiple (≥3) digital amputations or standard TMA ± skin coverage.		
3	Extensive, deep ulcer involving forefoot and/or midfoot; deep, full thickness heel ulcer ± calcaneal involvement	Extensive gangrene involving forefoot and /or midfoot; full thickness heel necrosis ± calcaneal involvement
Clinical description: extensive tissue loss salvageable only with a complex foot reconstruction or nontraditional TMA (Chopart or Lisfranc); flap coverage or complex wound management needed for large soft tissue defect		

a, Estimate risk of amputation at 1 year for each combination

	Ischemia – 0				Ischemia – 1				Ischemia – 2				Ischemia – 3			
W-0	VL	VL	L	M	VL	L	M	H	L	L	M	H	L	M	M	H
W-1	VL	VL	L	M	VL	L	M	H	L	M	H	H	M	M	H	H
W-2	L	L	M	H	M	M	H	H	M	H	H	H	H	H	H	H
W-3	M	M	H	H	H	H	H	H	H	H	H	H	H	H	H	H
	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3

b, Estimate likelihood of benefit of/requirement for revascularization (assuming infection can be controlled first)

	Ischemia – 0				Ischemia – 1				Ischemia – 2				Ischemia – 3			
W-0	VL	VL	VL	VL	VL	L	L	M	L	L	M	M	M	H	H	H
W-1	VL	VL	VL	VL	L	M	M	M	M	H	H	H	H	H	H	H
W-2	VL	VL	VL	VL	M	M	H	H	H	H	H	H	H	H	H	H
W-3	VL	VL	VL	VL	M	M	M	H	H	H	H	H	H	H	H	H
	f-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3

fI, foot Infection; I, Ischemia; W, Wound.

Premises:

1. Increase in wound class increases risk of amputation (based on PEDIS, UT, and other wound classification systems)
2. PAD and infection are synergistic (Eurodiale); infected wound + PAD increases likelihood revascularization will be needed to heal wound
3. Infection 3 category (systemic/metabolic instability): moderate to high-risk of amputation regardless of other factors (validated IDSA guidelines)

Four classes: for each box, group combination into one of these four classes

Very low = VL = clinical stage 1
Low = L = clinical stage 2
Moderate = M = clinical stage 3
High = H = clinical stage 4
Clinical stage 5 would signify an unsalvageable foot

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W-0	VL	VL	L	M	VL	L	M	H	L	L	M	H	L	M	M	H
W-1	VL	VL	L	M	VL	L	M	H	L	M	H	H	M	M	H	H
W-2	L	L	M	H	M	M	H	H	M	H	H	H	H	H	H	H
W-3	M	M	H	H	H	H	H	H	H	H	H	H	H	H	H	H
	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3

b, Estimate likelihood of benefit of/requirement for revascularization (assuming infection can be controlled first)

	Ischemia – 0				Ischemia – 1				Ischemia – 2				Ischemia – 3			
W-0	VL	VL	VL	VL	VL	L	L	M	L	L	M	M	M	H	H	H
W-1	VL	VL	VL	VL	L	M	M	M	M	H	H	H	H	H	H	H
W-2	VL	VL	VL	VL	M	M	H	H	H	H	H	H	H	H	H	H
W-3	VL	VL	VL	VL	M	M	M	H	H	H	H	H	H	H	H	H
	f-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3

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I: Ischemia
Hemodynamics/perfusion: Measure TP or $TcPO_2$ if ABI incompressible (>1.3)
SVS grades 0 (none), 1 (mild), 2 (moderate), and 3 (severe).

Grade	*ABI*	*Ankle systolic pressure*	*TP, $TcPO_2$*
0	≥0.80	>100 mm Hg	≥60 mm Hg
1	0.6-0.79	70-100 mm Hg	40-59 mm Hg
2	0.4-0.59	50-70 mm Hg	30-39 mm Hg
3	≤0.39	<50 mm Hg	<30 mm Hg

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fI: foot Infection:
SVS grades 0 (none), 1 (mild), 2 (moderate), and 3 (severe: limb and/or life-threatening)
SVS adaptation of Infectious Diseases Society of America (*IDSA*) and International Working Group on the Diabetic Foot (IWGDF) perfusion, extent/size, depth/tissue loss, infection, sensation (*PEDIS*) classifications of diabetic foot infection

Clinical manifestation of infection	*SVS*	*IDSA/PEDIS infection severity*
No symptoms or signs of infection	0	Uninfected
Infection present, as defined by the presence of at least 2 of the following items: • Local swelling or induration • Erythema >0.5 to ≤2 cm around the ulcer • Local tenderness or pain • Local warmth • Purulent discharge (thick, opaque to white, or sanguineous secretion)	1	Mild
Local infection involving only the skin and the subcutaneous tissue (without involvement of deeper tissues and without systemic signs as described below). Exclude other causes of an inflammatory response of the skin (eg, trauma, gout, acute Charcot neuro-osteoarthropathy, fracture, thrombosis, venous stasis)		
Local infection (as described above) with erythema >2 cm, or involving structures deeper than skin and subcutaneous tissues (eg, abscess, osteomyelitis, septic arthritis, fasciitis), and No systemic inflammatory response signs (as described below)	2	Moderate
Local infection (as described above) with the signs of SIRS, as manifested by two or more of the following: • Temperature >38° or <36°C • Heart rate >90 beats/min • Respiratory rate >20 breaths/min or $PaCO_2$ <32 mm Hg • White blood cell count >12,000 or <4000 cu/mm or 10% immature (band) forms	3	Severe[a]

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W-2	L	L	M	H	M	M	H	H	M	H	H	H	H	H	H	H
W-3	M	M	H	H	H	H	H	H	H	H	H	H	H	H	H	H
	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3

b, Estimate likelihood of benefit of/requirement for revascularization (assuming infection can be controlled first)

	Ischemia – 0				Ischemia – 1				Ischemia – 2				Ischemia – 3			
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W-1	VL	VL	VL	VL	L	M	M	M	M	H	H	H	H	H	H	H
W-2	VL	VL	VL	VL	M	M	H	H	H	H	H	H	H	H	H	H
W-3	VL	VL	VL	VL	M	M	M	H	H	H	H	H	H	H	H	H
	f-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3	fI-0	fI-1	fI-2	fI-3

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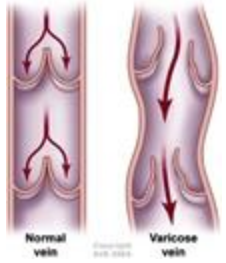
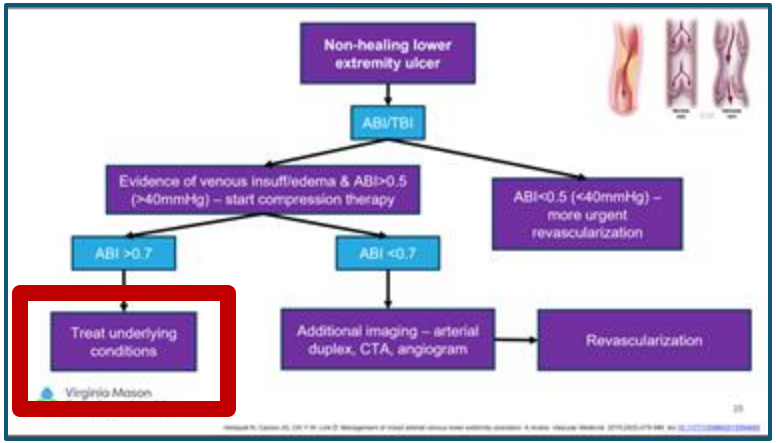
Venous Insufficiency - Evaluation

Class	Description
Clinical[a]	
C0	No visible or palpable signs of venous disease
C1	Telangiectasias or reticular veins
C2	Varicose veins
C2r	Recurrent varicose veins
C3	Edema
C4	Changes in skin and subcutaneous tissue due to chronic venous insufficiency
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





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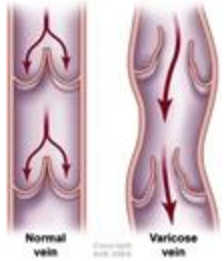
Venous Insufficiency - Management



- Retrograde flow in superficial vein (GSV or SSV) >500ms

- Compressive therapy
- “Early” ablative therapy

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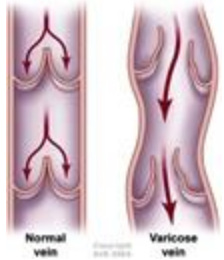
https://vascular.org/patient-resources/vascularcondtions/varicose-veins

ORIGINAL ARTICLE

A Randomized Trial of Early Endovenous Ablation in Venous Ulceration

Manjit S. Gohel, M.D., Francine Heatley, B.Sc., Xinxue Liu, Ph.D., Andrew Bradbury, M.D., Richard Bulbulia, M.D., Nicky Cullum, Ph.D., David M. Epstein, Ph.D., Isaac Nyamekye, M.D., Keith R. Poskitt, M.D., Sophie Renton, M.S., Jane Warwick, Ph.D., and Alun H. Davies, D.Sc., for the EVRA Trial Investigators*



- Intervention within 2wks of randomization vs 6mo or after ulcer healing
- Early intervention group
  - More healed ulcers
  - Faster healing
  - Higher rate of healing at 24wks post-randomization
  - More ulcer-free days in year after intervention

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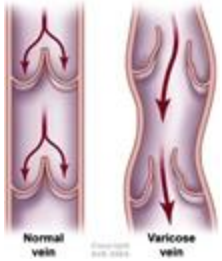
Gohel, M. S., Heatley, F., Liu, X., Bradbury, A., Bulbulia, R., Cullum, N., Epstein, D. M., Nyamekye, I., Poskitt, K. R., Renton, S., Warwick, J., & Davies, A. H. (2018). A randomized trial of early endovenous ablation in venous ulceration. *New England Journal of Medicine*, *378*(22), 2105–2114. https://doi.org/10.1056/nejmoa1801214

Venous Insufficiency - Management



- Retrograde flow in superficial vein (GSV or SSV) >500ms

- Compressive therapy
- “Early” ablative therapy
- Wound care – including skin substitute grafting

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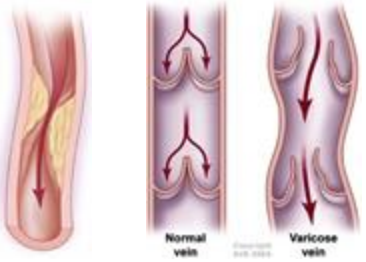
https://vascular.org/patient-resources/vascularcondtions/varicose-veins

Management

Etiology	Example	1st Diagnostic Investigation
Infectious	Fungal, mycobacterial, bacterial	Wound bx & cx, CBC, CSR, X-ray
Malignant	Basal cell CA, squamous cell CA	Wound Bx
Arterial insufficiency – macro	Atherosclerosis (PAD), thromboembolic dz	Duplex & physiologic arterial studies (ABI)
Arterial insufficiency - micro	DM, Raynaud's, Buerger's,	$TcPO_2$, toe pressure/TBI
Venous insufficiency – deep & superficial	DVT, extrinsic compression, valvular reflux	Duplex venous studies
Lymphatic obstruction	Lymphatic insufficiency, malignancy, secondary to an "overload state"	H&P
Hematologic abnormality	PCV, Sickle cell	CBC, iron studies
Collagen vascular disorder	Scleroderma, lupus, Wegener	Rheumatologic work-up
Excessive pressure	Neuropathy (diabetic, alcoholic), paraplegia, bone spurs	Neuro exam (monofilament & vibratory), x-ray

Sidawy, A. N., & Perler, B. A. (2018). Rutherford's vascular surgery and endovascular therapy. In *Elsevier eBooks*. https://ci.nii.ac.jp/ncid/BB26259348



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Management

Circulation

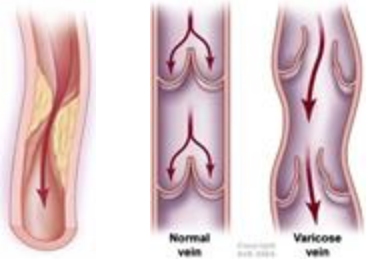
CLINICAL PRACTICE GUIDELINES

2024 ACC/AHA/AACVPR/APMA/ABC/SCAI/SVM/SVN/SVS/SIR/VESS Guideline for the Management of Lower Extremity Peripheral Artery Disease: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines

Developed in Collaboration With and Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation, American Podiatric Medical Association, Association of Black Cardiologists, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine, Society for Vascular Nursing, Society for Vascular Surgery, Society of Interventional Radiology, and Vascular & Endovascular Surgery Society

Recommendation for Team-Based Care for CLTI
Referenced studies that support the recommendation are summarized in the Online Data Supplement.

COR	LOE	Recommendation
1	B-NR	1. In patients with CLTI, a multispecialty care team should evaluate and provide comprehensive care with goals of complete wound healing, minimizing tissue loss, and preservation of ambulatory status.[1,2]



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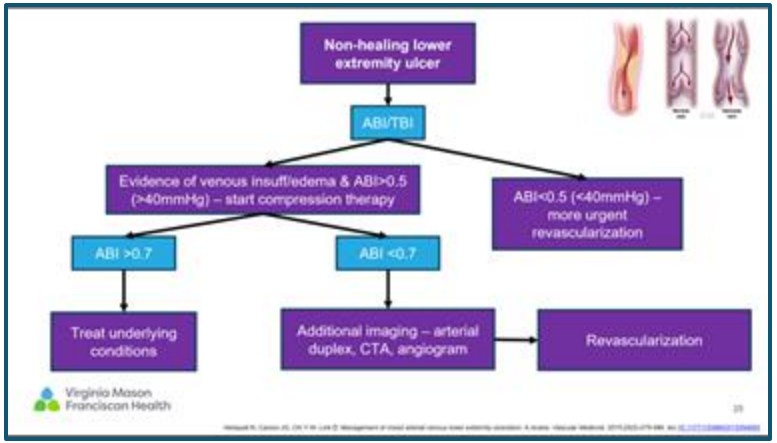
Gornik, H. L., Aronow, H. D., Goodney, P. P., Arya, S., Brewster, L. P., Byrd, L., Chandra, V., Drachman, D. E., Eaves, J. M., Ehrman, J. K., Evans, J. N., Getchius, T. S., Gutiérrez, J. A., Hawkins, B. M., Hess, C. N., Ho, K. J., Jones, W. S., Kim, E. S., Kinlay, S., . . . Wilkins, L. R. (2024). 2024 ACC/AHA/AACVPR/APMA/ABC/SCAI/SVM/SVN/SVS/SIR/VESS Guideline for the Management of Lower Extremity Peripheral Artery Disease: A report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation*, *149*(24). https://doi.org/10.1161/cir.0000000000001251

Management



Revascularization Strategy for CLTI

1	A	3. In patients undergoing surgical revascularization for CLTI, bypass to the popliteal or infrapopliteal arteries (ie, tibial, pedal) should be constructed with autogenous vein if available.[14–20]

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Gornik, H. L., Aronow, H. D., Goodney, P. P., Arya, S., Brewster, L. P., Byrd, L., Chandra, V., Drachman, D. E., Eaves, J. M., Ehrman, J. K., Evans, J. N., Getchius, T. S., Gutiérrez, J. A., Hawkins, B. M., Hess, C. N., Ho, K. J., Jones, W. S., Kim, E. S., Kinlay, S., . . . Wilkins, L. R. (2024). 2024 ACC/AHA/AACVPR/APMA/ABC/SCAI/SVM/SVN/SVS/SIR/VESS Guideline for the Management of Lower Extremity Peripheral Artery Disease: A report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation*, *149*(24). https://doi.org/10.1161/cir.0000000000001251

Diabetic Foot Ulcers

Etiology	Example	1st Diagnostic Investigation
Infectious	Fungal, mycobacterial, bacterial	Wound bx & cx, CBC, CSR, X-ray
Malignant	Basal cell CA, squamous cell CA	Wound Bx
Arterial insufficiency – macro	Atherosclerosis (PAD), thromboembolic dz	Duplex & physiologic arterial studies (ABI)
Arterial insufficiency - micro	DM, Raynaud's, Buerger's,	$TcPO_2$, toe pressure/TBI
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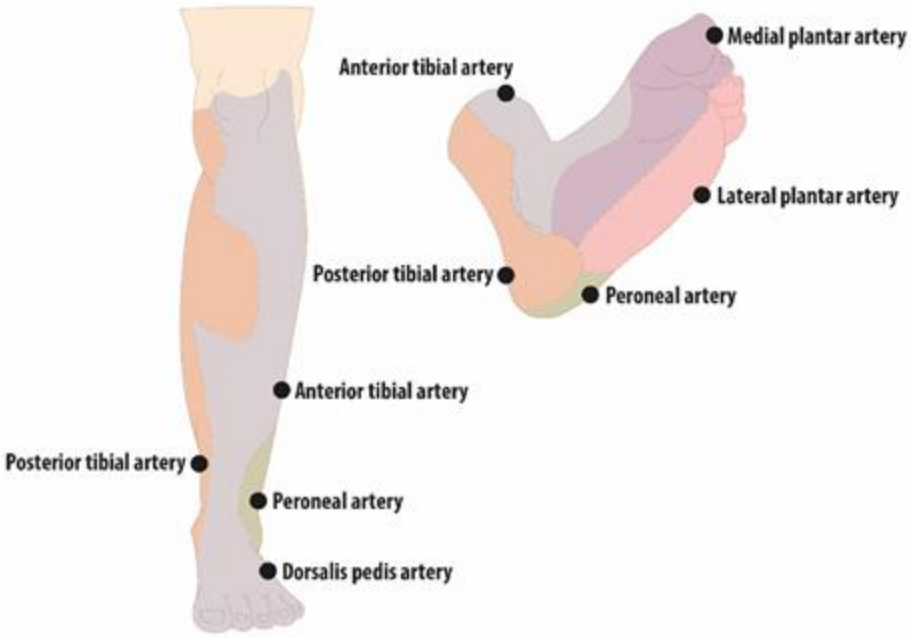
Lymphatic obstruction	Lymphatic insufficiency, malignancy, secondary to an "overload state"	H&P
Hematologic abnormality	PCV, Sickle cell	CBC, iron studies
Collagen vascular disorder	Scleroderma, lupus, Wegener	Rheumatologic work-up
Excessive pressure	Neuropathy (diabetic, alcoholic), paraplegia, bone spurs	Neuro exam (monofilament & vibratory), x-ray

- Multidisciplinary care
- ?Hyperbaric oxygen therapy
- Skin-substitute grafts
- Negative pressure wound therapy



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Fitridge, R., Chuter, V., Mills, J., Hinchliffe, R., Azuma, N., Behrendt, C., Boyko, E. J., Conte, M. S., Humphries, M., Kirkey, L., McGinigle, K. C., Nikol, S., Nordanstig, J., Rowe, V., Russell, D., Van Den Berg, J. C., Venermo, M., & Schaper, N. (2023). The intersocietal IWGDF, ESVS, SVS guidelines on peripheral artery disease in people with diabetes mellitus and a foot ulcer. *Journal of Vascular Surgery*, *78*(5), 1101–1131. https://doi.org/10.1016/j.jvs.2023.07.020

Key Takeaways

- **Chronic wounds impose a significant cost on individuals and society**
- **Often multifactorial etiologies – require multidisciplinary approach for best outcomes**
- **Screening studies have value – don’t take short-cuts**

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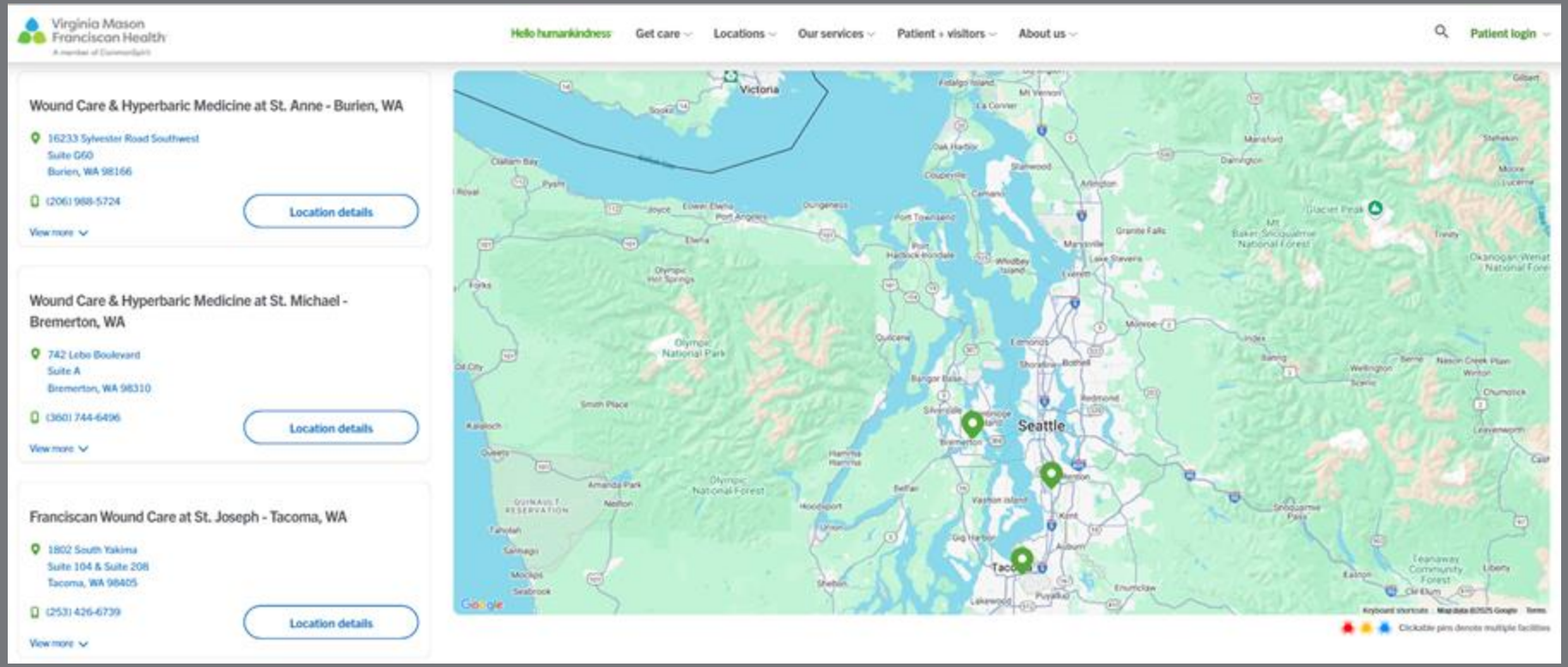

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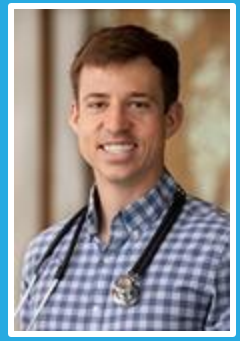

Sam Godin, ARNP

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Thank you.

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Thoracic Outlet Syndrome (TOS): A Current Review in the Diagnosis and Treatment

Matthew Bennett, MD, RPVI

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Center for Cardiovascular Health

Disclosures

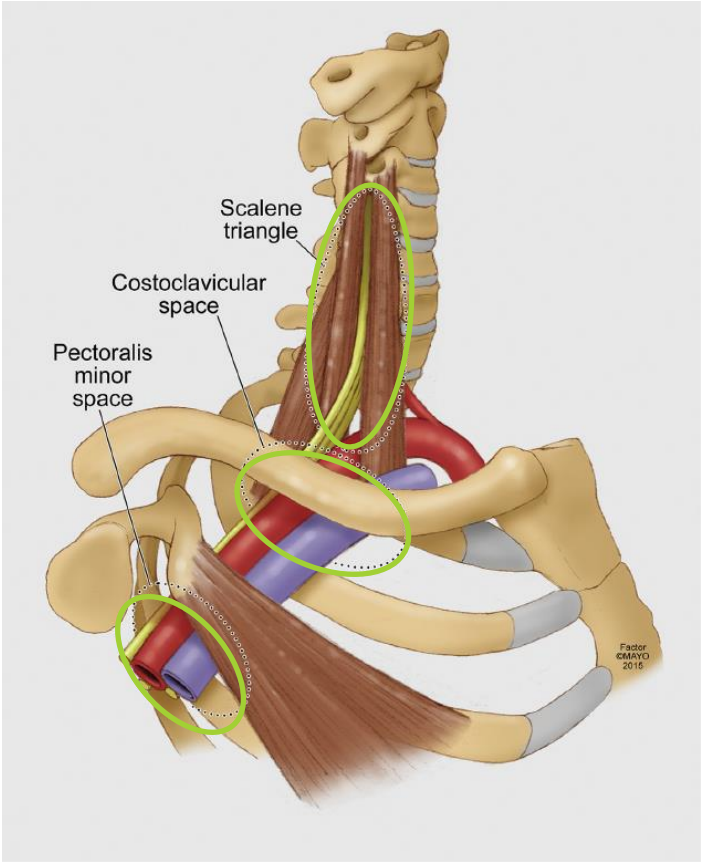
- None

Background

- Thoracic outlet syndrome refers to the collection of diseases involving the arterial, venous, or nervous structures as they traverse the thoracic outlet
- Classified as neurogenic (nTOS), arterial (aTOS), or venous (vTOS)
- Can cause significant upper extremity pain and dysfunction, typically in younger patients, and can cause lifelong disability

DiLosa KL, Humphries MD. Epidemiology of thoracic outlet syndrome. Semin Vasc Surg. 2021 Mar;34(1):65-70.

Anatomy



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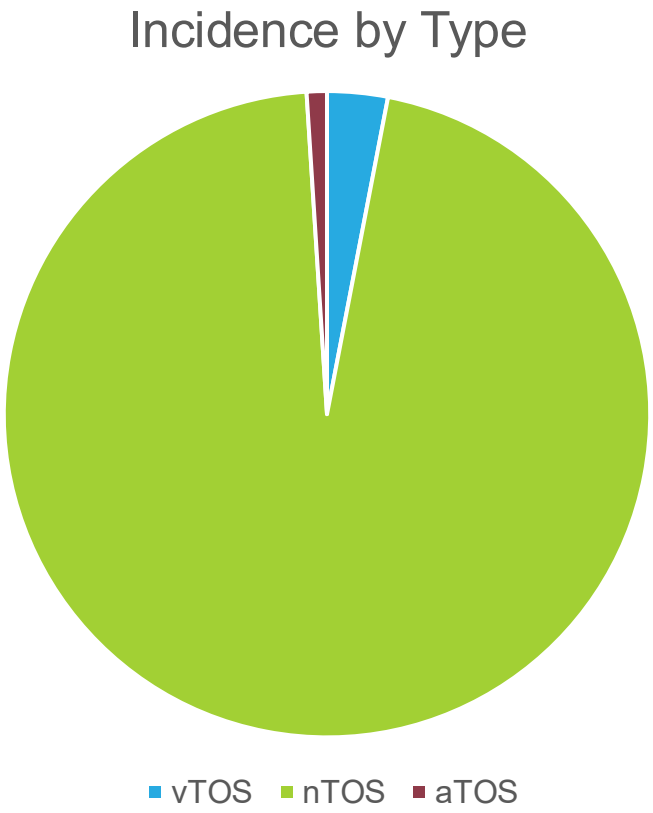
Illig KA, Donahue D, Duncan A, Freischlag J, Gelabert H, Johansen K, Jordan S, Sanders R, Thompson R. Reporting standards of the Society for Vascular Surgery for thoracic outlet syndrome. J Vasc Surg. 2016 Sep;64(3):e23-35.

Epidemiology



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DiLosa KL, Humphries MD. Epidemiology of thoracic outlet syndrome. Semin Vasc Surg. 2021 Mar;34(1):65-70.

Epidemiology: nTOS

- Typically ages 30-40
- Predominately women
- Frequent history of neck trauma: auto accident with hyperextension or whiplash, repetitive stress injuries
- Community prevalence anywhere from 1 – 80,000 per 1,000,000

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DiLosa KL, Humphries MD. Epidemiology of thoracic outlet syndrome. Semin Vasc Surg. 2021 Mar;34(1):65-70.

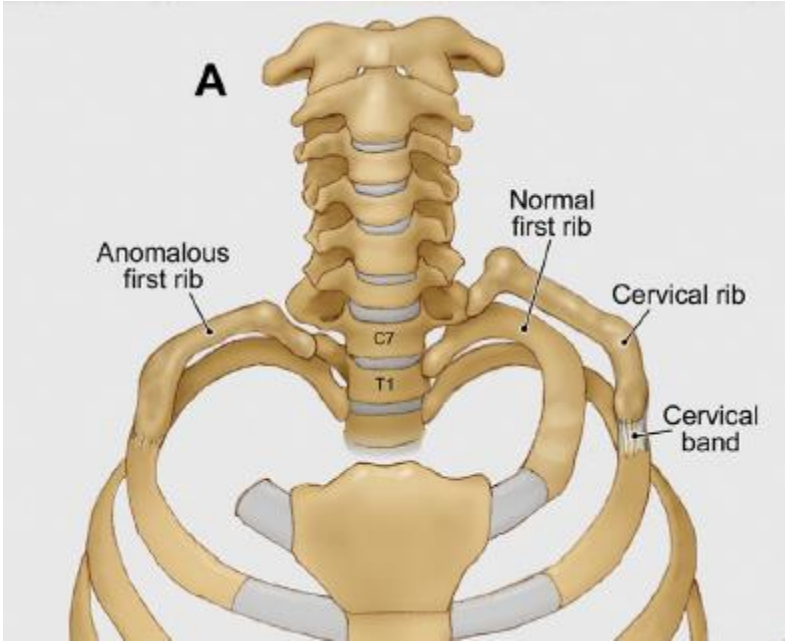
Epidemiology: vTOS

- Most common ages 20-30, historically 2:1 male prevalence, though now closer to 1:1
- Also known as effort-induced thrombosis or Paget-Schroetter syndrome
- Associated with repetitive overhead shoulder movements (e.g. manual labor or certain athletes)
- Typically unilateral involving dominant arm

DiLosa KL, Humphries MD. Epidemiology of thoracic outlet syndrome. Semin Vasc Surg. 2021 Mar;34(1):65-70.

Epidemiology: aTOS

- Almost always associated with cervical ribs, anomalous first ribs, or congenital muscular bands
- More common in women (higher prevalence of cervical ribs)
- Most common cause of acute upper extremity arterial thrombosis for those under 40



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DiLosa KL, Humphries MD. Epidemiology of thoracic outlet syndrome. Semin Vasc Surg. 2021 Mar;34(1):65-70.
Illig KA, Donahue D, Duncan A, Freischlag J, Gelabert H, Johansen K, Jordan S, Sanders R, Thompson R. Reporting standards of the Society for Vascular Surgery for thoracic outlet syndrome. J Vasc Surg. 2016 Sep;64(3):e23-35.

Diagnosis: nTOS

Table 3 – Society for Vascular Surgery guidelines for diagnosing neurogenic thoracic outlet syndrome.

Findings	History	Examination
Local	Irritation or inflammation in the scalene triangle or pectoralis insertion site Referred pain in areas near the thoracic outlet, chest wall, axilla, upper back, shoulder, trapezius, neck, or head	Pain on palpation to previously stated areas
Peripheral	Arm or hand numbness, pain, paresthesia, vasomotor changes, and weakness Symptoms exacerbated by movements that narrow the thoracic outlet or stretch the plexus	Palpation of the affected area reproduces peripheral symptoms Provocative maneuvers including elevated arm stress test and upper limb tension test reproduce symptoms
Absence of other reasonably likely diagnoses that might explain most of the symptoms	—	—
Response to a properly performed test injection is positive	—	—

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Betancourt A, Benrashid E, Gupta PC, McGinigle KL. Current concepts in clinical features and diagnosis of thoracic outlet syndrome. Semin Vasc Surg. 2024 Mar;37(1):3-11.

nTOS: Upper Limb Tension Test (ULTT)

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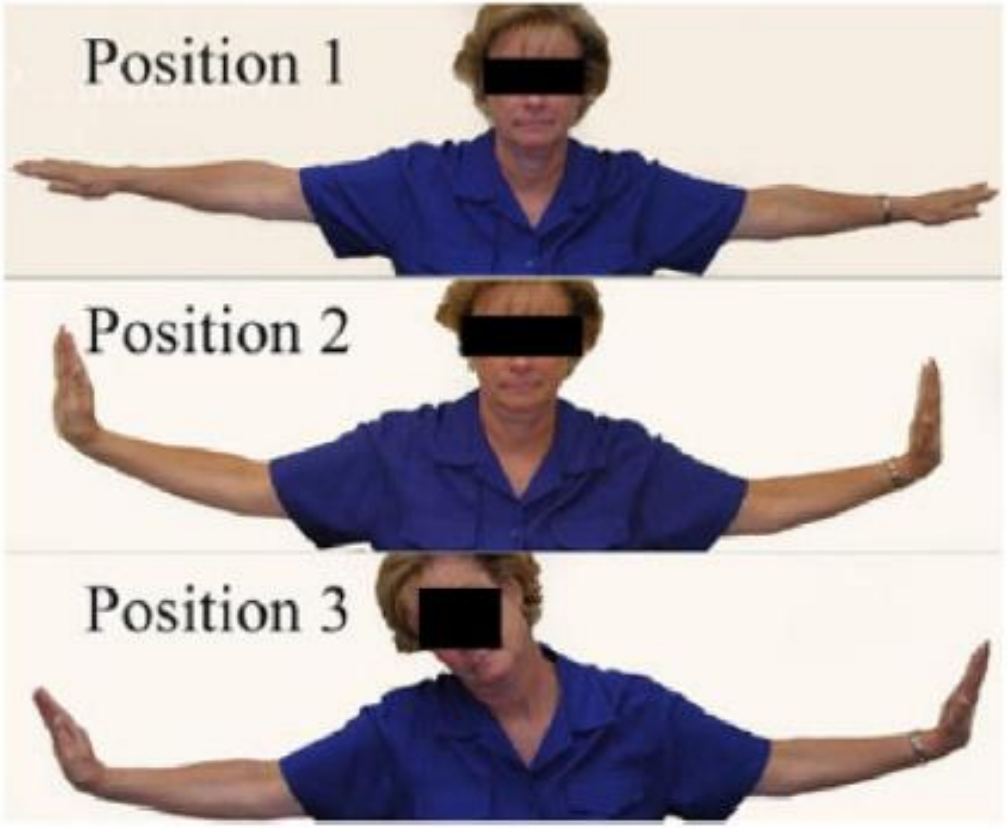
Betancourt A, Benrashid E, Gupta PC, McGinigle KL. Current concepts in clinical features and diagnosis of thoracic outlet syndrome. Semin Vasc Surg. 2024 Mar;37(1):3-11.

nTOS: Elevated Arm Stress Test (EAST)



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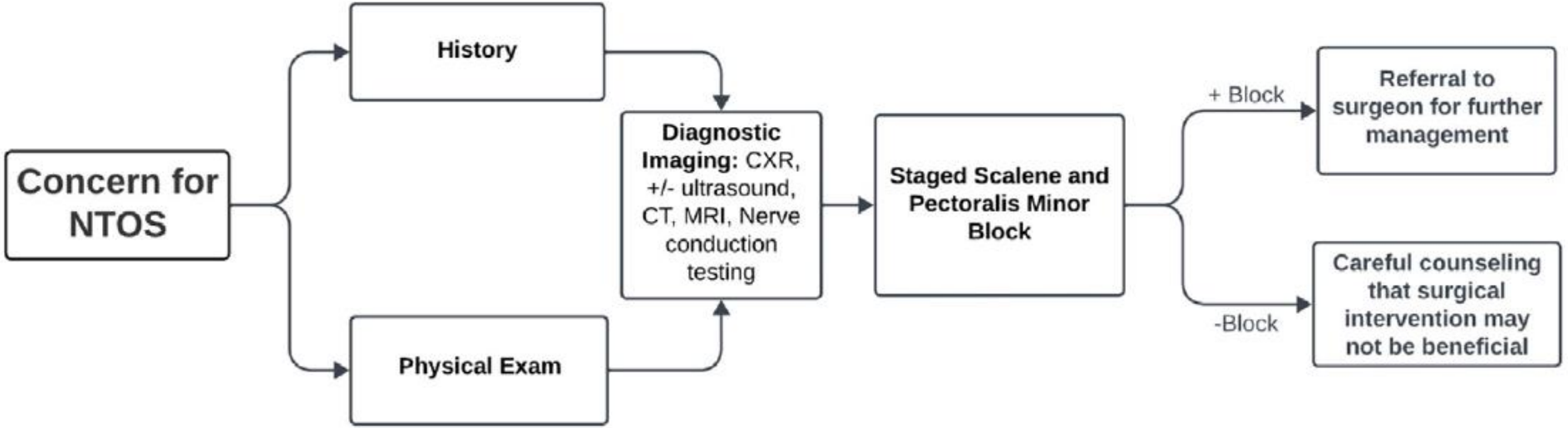
Betancourt A, Benrashid E, Gupta PC, McGinigle KL. Current concepts in clinical features and diagnosis of thoracic outlet syndrome. Semin Vasc Surg. 2024 Mar;37(1):3-11.

Diagnosis: nTOS



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Betancourt A, Benrashid E, Gupta PC, McGinigle KL. Current concepts in clinical features and diagnosis of thoracic outlet syndrome. Semin Vasc Surg. 2024 Mar;37(1):3-11.

Diagnosis: vTOS

- Acute: DVT symptoms: pain, swelling, discoloration; in severe cases, phlegmasia with cyanosis or pallor
- Chronic: swelling and pain with use of the extremity. Chest wall collaterals may be present.
- Duplex ultrasound for first line imaging. CT venogram or MR angiography may help delineate anatomy for surgical planning
- Venography can be diagnostic and potentially therapeutic

Betancourt A, Benrashid E, Gupta PC, McGinigle KL. Current concepts in clinical features and diagnosis of thoracic outlet syndrome. Semin Vasc Surg. 2024 Mar;37(1):3-11.

Diagnosis: aTOS

- Requires documented injury to the subclavian artery, either stenosis/occlusion or post-stenotic dilatation/aneurysm, or evidence of ischemia with arterial compression
- Arm claudication or rest pain, coolness, paresthesia, digital ulceration or gangrene
- Exam may reveal diminished pulses, particularly with provocative maneuvers, pulsatile mass in supraclavicular fossa, bruit
- Ultrasound for initial imaging, CT angiogram may assist with surgical planning

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Betancourt A, Benrashid E, Gupta PC, McGinigle KL. Current concepts in clinical features and diagnosis of thoracic outlet syndrome. Semin Vasc Surg. 2024 Mar;37(1):3-11.

Intervention: nTOS

- Initial treatment of nTOS is conservative with physical therapy (generally 4-6 months)
- Goal of improving posture to decompress the scalene triangle and costoclavicular space
- Anti-inflammatory medication and muscle relaxants useful adjunct
- Anterior scalene Botox injections can provide temporary relief (~3 months)
- Scalene or pectoralis minor muscle blocks can provide relief, and also positively predict response to surgery

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Capodosal G, Holden D, Maloy W, Schroeder JD. Thoracic Outlet Syndrome. Curr Sports Med Rep. 2024 Sep 1;23(9):303-309.
McIntosh E, Tripathi RK, Ohman JW. Neurogenic thoracic outlet syndrome and controversies in diagnosis and management. Semin Vasc Surg. 2024 Mar;37(1):20-25.

Surgical Decompression: nTOS

- Will optimally involve anterior scalenectomy, mobilization and resection of the middle scalene, and extensive resection of the first rib
- Brachial plexus neurolysis
- Supraclavicular, transaxillary, or infraclavicular approaches, depending on needs for resection and reconstruction
- Robotic first rib resection also seems to offer similar outcomes
- Most trials seem to support better improvements in upper extremity symptoms with surgery as opposed to ongoing physical therapy
- Recurrence rates may be from 5 – 30%

McIntosh E, Tripathi RK, Ohman JW. Neurogenic thoracic outlet syndrome and controversies in diagnosis and management. Semin Vasc Surg. 2024 Mar;37(1):20-25.
Reyes M, Alaparthi S, Roedl JB, Moreta MC, Evans NR, Grenda T, Okusanya OT. Robotic First Rib Resection in Thoracic Outlet Syndrome: A Systematic Review of Current Literature. J Clin Med. 2023 Oct 23;12(20):6689.

Surgery vs Physical Therapy

Miscellaneous

Eur J Vasc Endovasc Surg (2022) 64, 119—127

RANDOMISED CLINICAL TRIAL

Surgery Versus Continued Conservative Treatment for Neurogenic Thoracic Outlet Syndrome: the First Randomised Clinical Trial (STOPNTOS Trial)☆

Jens Goeteyn [a], Niels Pesser [a], Saskia Houterman [b], Marc R.H.M. van Sambeek [a,c], Bart F.L. van Nuenen [d], Joep A.W. Teijink [a,e,*]

[a] Department of Vascular Surgery, Catharina Hospital, Eindhoven, The Netherlands
[b] Department of Education and Research, Catharina Hospital, The Netherlands
[c] Department of Biomedical Technology, University of Technology Eindhoven, Eindhoven, The Netherlands
[d] Department of Neurology, Catharina Hospital, Eindhoven, The Netherlands
[e] CAPHRI School for Public Health and Primary Care, Faculty of Health, Medicine and Life Sciences, Maastricht University, The Netherlands

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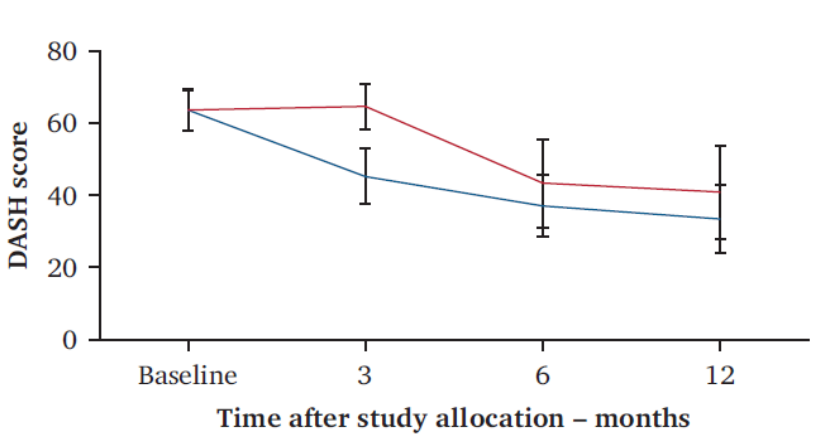
Surgery vs Physical Therapy

- 24 patients randomized to surgery, 22 to conservative management
- Significant improvement in disability scores in surgical group compared to physical therapy alone
- Overall failure rate still 19.56% at one year



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Surgical Treatment: aTOS

Table 2 – Scher classification for management of arterial thoracic outlet syndrome.

Stage	Description	Recommended management
0	Asymptomatic compression	Observation with noninvasive surveillance
I	Artery stenosis with minor dilation	Thoracic outlet decompression
II	Artery with intimal damage and/or aneurysmal disease and/or mural thrombus	Thoracic outlet decompression with arterial reconstruction
III	Distal embolization	Thoracic outlet decompression with arterial reconstruction and combination of embolectomy, lytic catheter and/or anticoagulation

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Potluri VK, Li RD, Crisostomo P, Bechara CF. A review of arterial thoracic outlet syndrome. Semin Vasc Surg. 2024 Mar;37(1):12-19.

Surgical Treatment: aTOS

- Similar decompression including resection of cervical rib or other bony abnormality if present
- Initial thrombolysis or thrombectomy particularly if patient presents with ischemia
- Will frequently require arterial reconstruction or bypass graft
- PTFE generally preferred for reconstruction, though no studies directly comparing to autologous graft
- Patency rates for reconstruction generally very high – 90 to 100% at several years from surgery

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Dalio MB, Filho ERDS, Barufi MB, Ribeiro MS, Joviliano EE. Contemporary Management of Arterial Thoracic Outlet Syndrome. Ann Vasc Surg. 2021 Jul;74:42-52.
Potluri VK, Li RD, Crisostomo P, Bechara CF. A review of arterial thoracic outlet syndrome. Semin Vasc Surg. 2024 Mar;37(1):12-19.

Surgical Treatment: vTOS

- Anticoagulation alone will have higher failure rate than thrombectomy/thrombolysis
- Initial removal of acute thrombus (thrombolysis most widely reported, though there are newer endovascular therapies which likely provide similar results)
- Followed by decompression of thoracic outlet with venoplasty or angioplasty of residual stenosis within the subclavian vein

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Karaolanis G, Antonopoulos CN, Koutsias SG, Giosdekos A, Metaxas EK, Tzimas P, de Borst GJ, Geroulakos G. A systematic review and meta-analysis for the management of Paget-Schroetter syndrome. J Vasc Surg Venous Lymphat Disord. 2021 May;9(3):801-810.e5.

Surgical Treatment: vTOS

A systematic review and meta-analysis for the management of Paget-Schroetter syndrome

Georgios Karaolanis, MD,[a] Constantine N. Antonopoulos, MD,[b,c] Stylianos G. Koutsias, MD,[a] Alexandros Giosdekos, MD,[c] Efstathios K. Metaxas, MD,[d] Petros Tzimas, MD,[e] Gert J. de Borst, MD,[f] *and* George Geroulakos, MD,[c] *Ioannina, Athens, and Piraeus, Greece; and Utretch, The Netherlands*

- 25 studies, 1511 patients
- With first rib resection, 97.89% vein patency and 98.02% symptom relief rate at final follow-up, compared to 64.6% patency and 70.65% symptom relief rate for thrombolysis alone

Thank You

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What is Thoracic Branch Endoprosthesis (TBE)?

Amit S. Kainth, MD, FACS, RPVI

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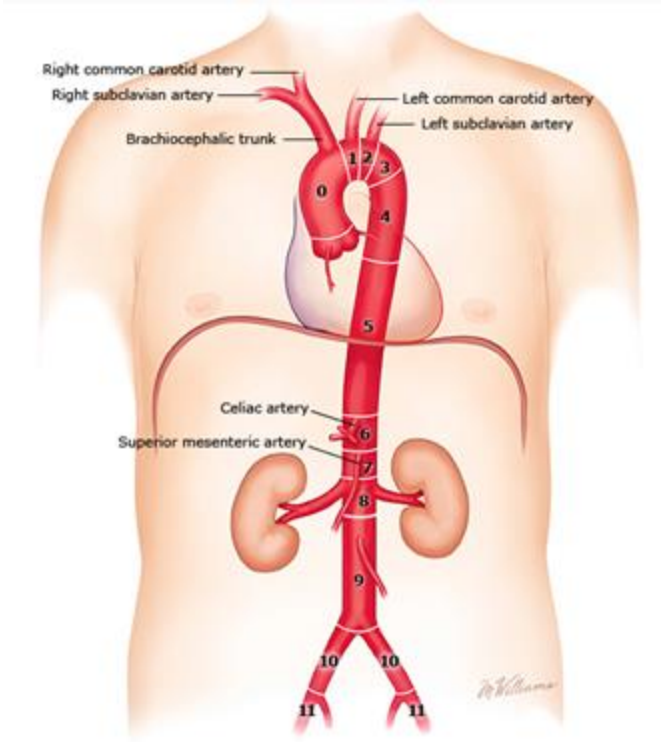
Disclosures

- NO RELEVANT DISCLOSURES

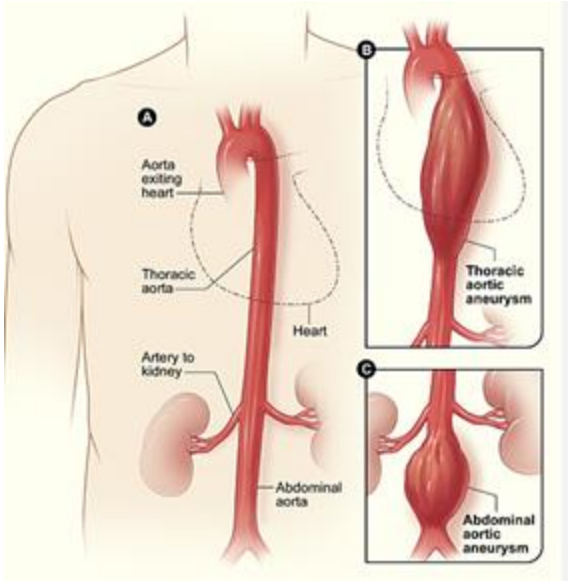
Thoracic Aorta

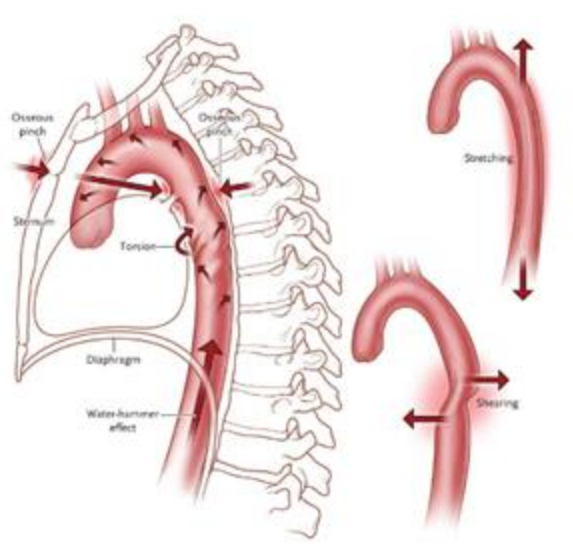
- The aorta originates immediately beyond the aortic valve and ascends initially, then curves, forming the aortic arch, and descends caudally adjacent to the spine.
- The ascending thoracic aorta gives off the coronary arteries
- The aortic arch branches are typically the brachiocephalic trunk (branches to the right common carotid and right subclavian arteries), left common carotid, and left subclavian arteries

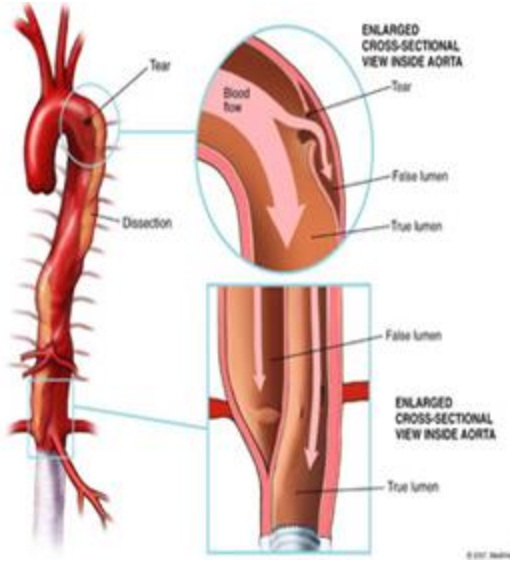


Aortic Pathologies

- Aortic pathologies requiring repair include aortic aneurysms, blunt aortic injury, aortic dissection, and penetrating aortic ulcer, among others







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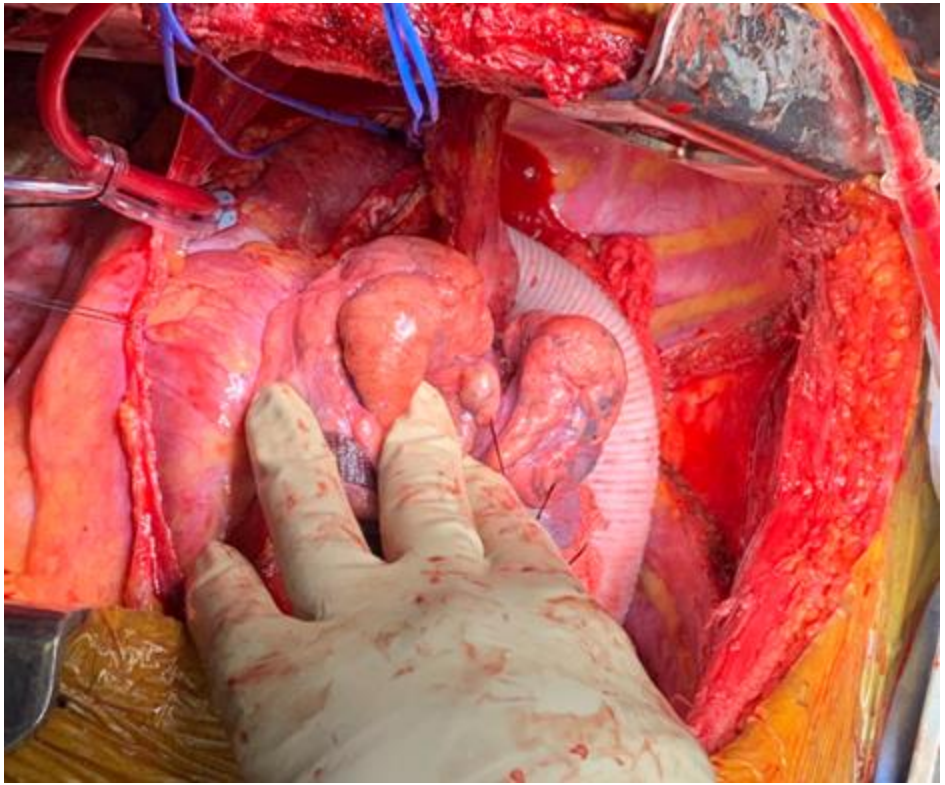
Open Thoracic Aortic Repair

- Open thoracic aneurysm repair remains the gold standard for thoracic aneurysm treatment but is associated with significant morbidity and mortality resulting in relatively poor long-term survival and prolonged hospitalization



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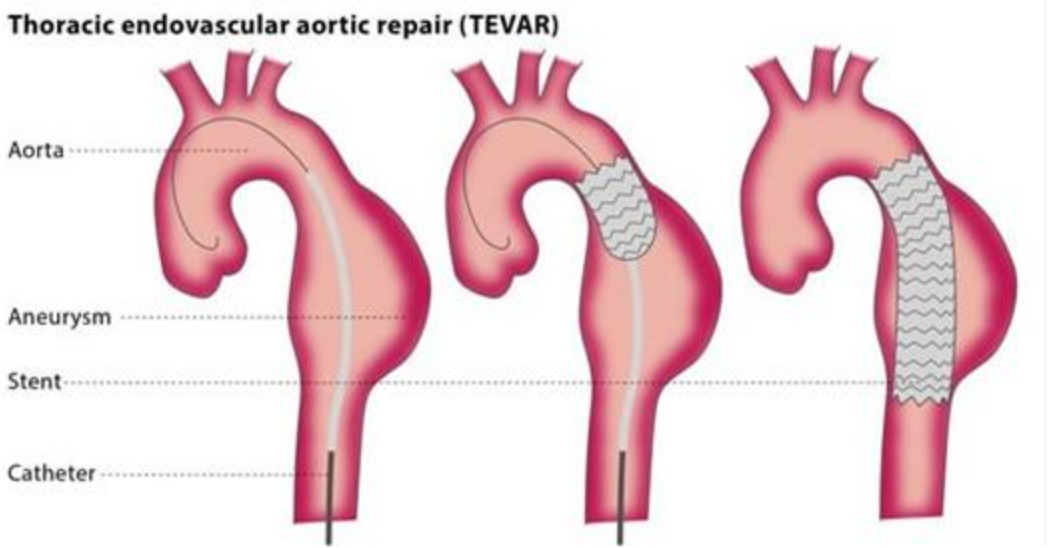
TEVAR

- Thoracic endovascular aortic repair (TEVAR), refers to a minimally invasive approach that involves placing a stent-graft in the thoracic or thoracoabdominal aorta.
- TEVAR was initially used to provide treatment to patients who were not considered to be surgical candidates, but it is now the preferred technique for treatment due to improved outcomes compared with open thoracic aortic surgery.



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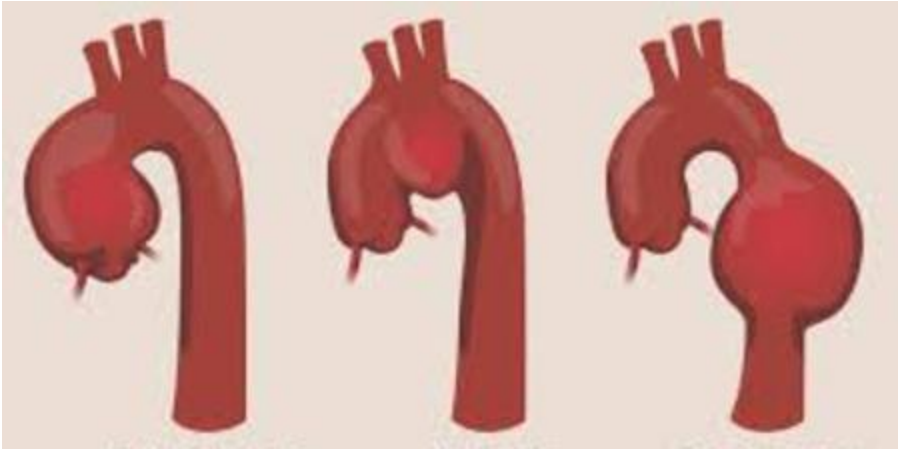
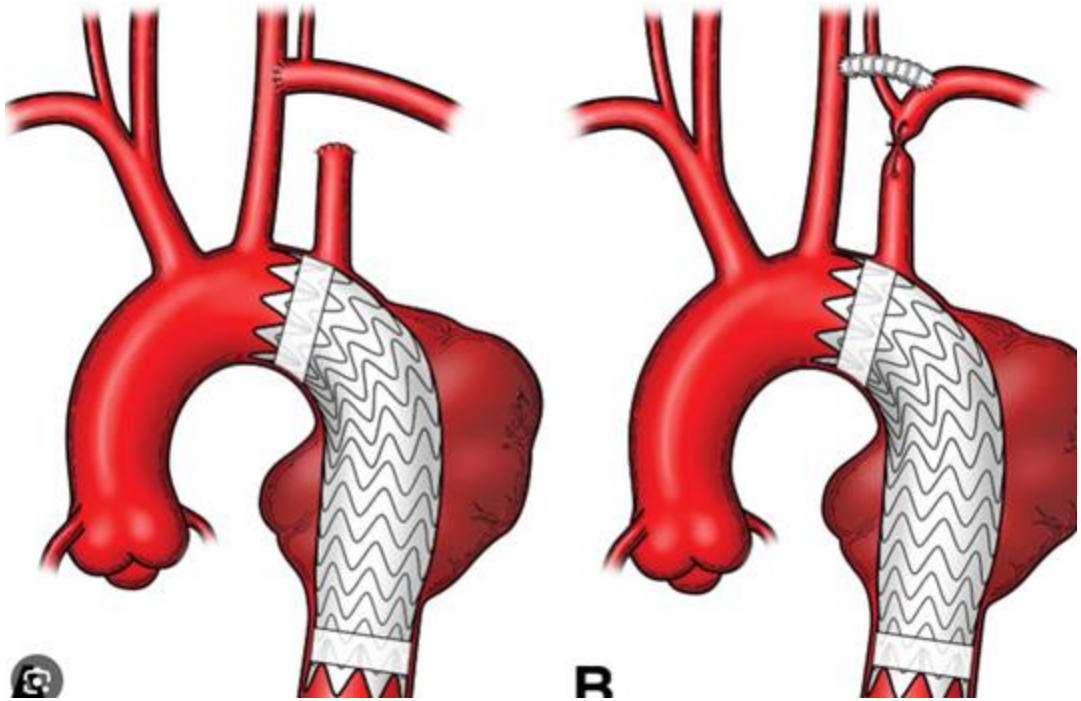
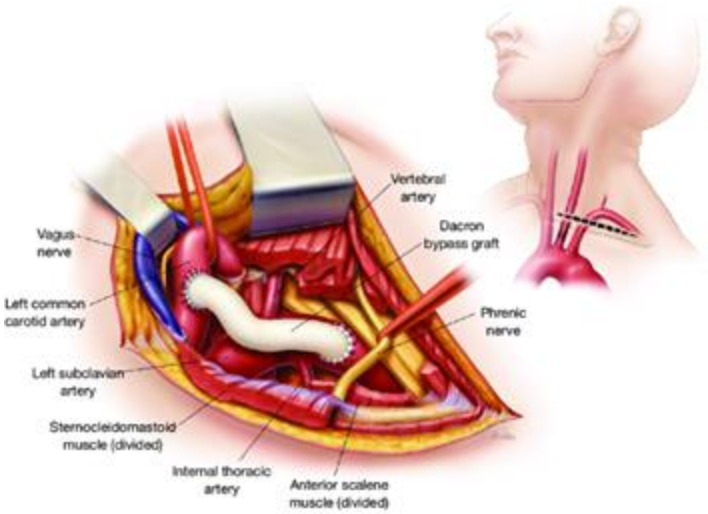
Complex Pathology

- Traditionally, when the pathology extends to the aortic arch vessels, additional procedures need to be done in order to perform a thoracic stent graft



Carotid Subclavian Bypass/ Transposition





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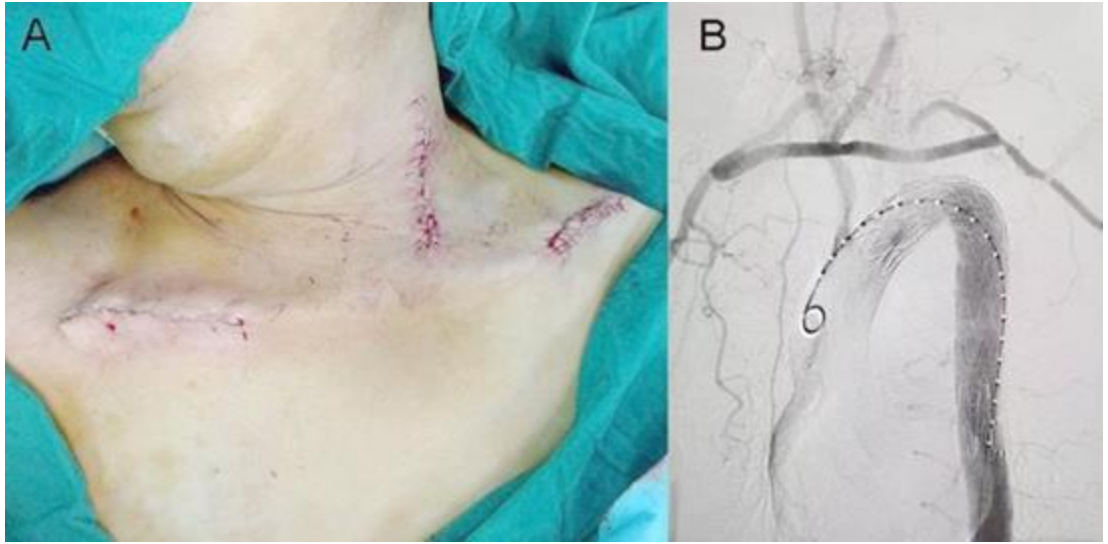
Complex Arch Debranching



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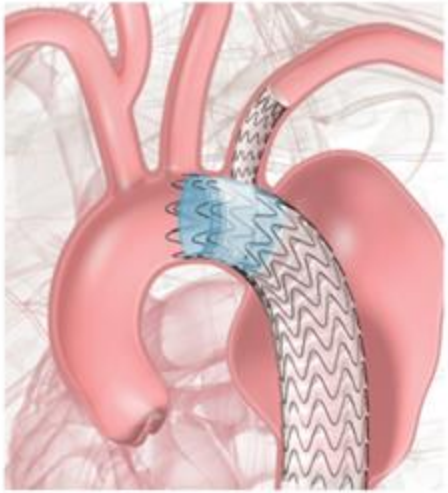
Thoracic Branch Endoprosthesis

- Thoracic Branch Endoprosthesis provides endovascular repair of lesions of the aortic arch and descending thoracic aorta, while maintaining flow into a single aortic arch branch vessel



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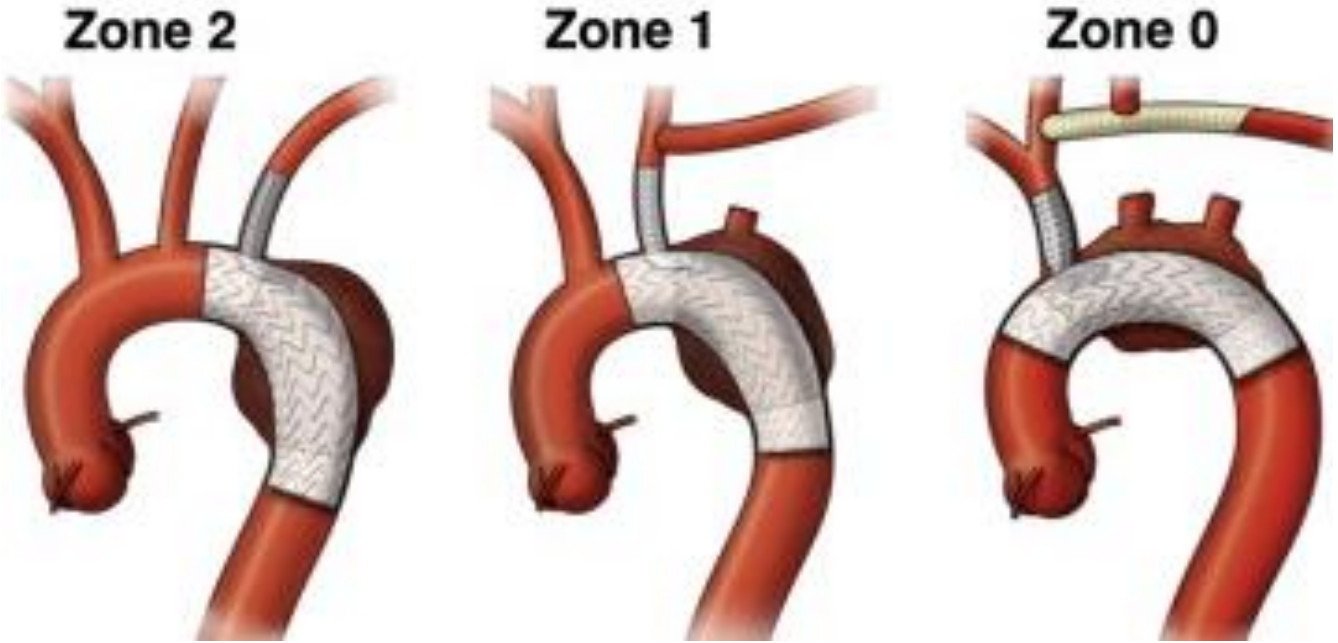
TBE + Extra Anatomic



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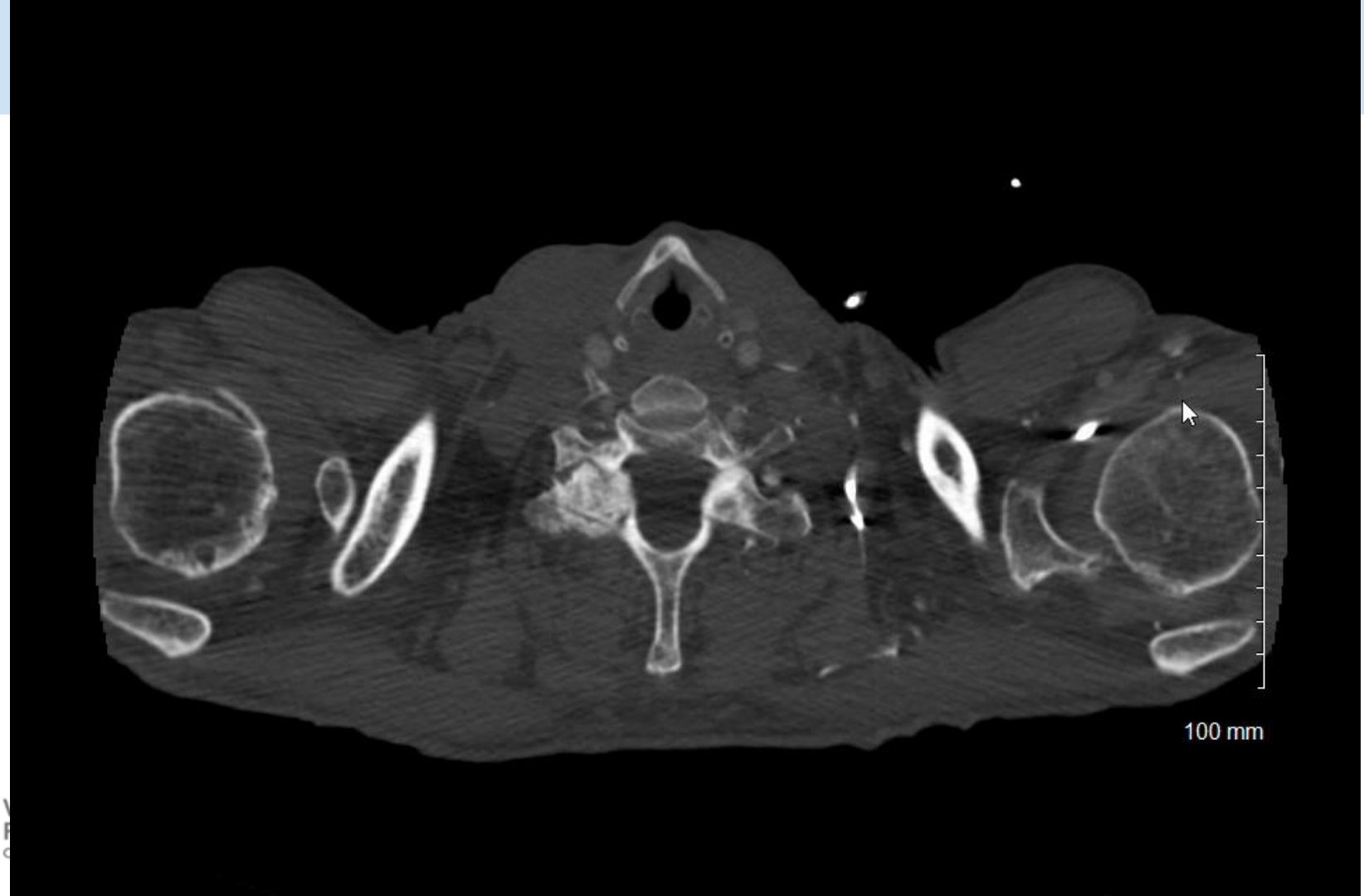
Case Presentation

- 65-year-old male with a history of a chronic type B thoracoabdominal aortic dissection likely secondary to cocaine use. Initially found in February 2024 and was medically managed at an outside hospital.

- Surveillance imaging revealed revealed rapid aneurysmal degeneration likely due to medical non compliance and continued hypertension. Aorta measuring 49mm which was previously 42mm four months prior.


100 mm

Procedure

Patient underwent:

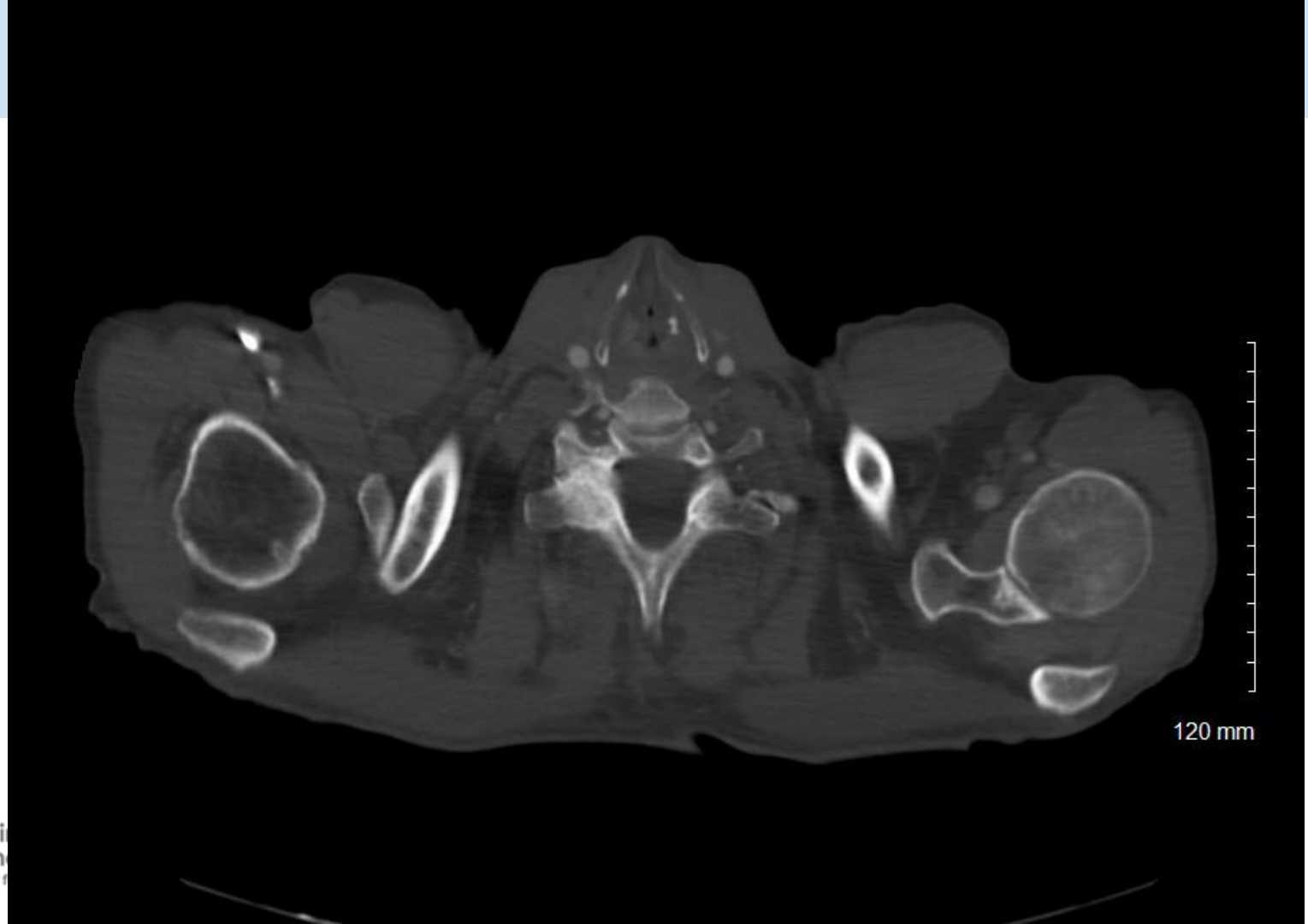
1. Ultrasound guided access of the left common femoral artery and left radial artery
2. Intravascular ultrasound
3. Thoracic endovascular aortic dissection repair with thoracic branch endoprosthesis (TEVAR-TBE)
4. Left subclavian artery stenting
5. Percutaneous closure of the left common femoral artery

Case time: 99 minutes

Fluoroscopy: 20.4 minutes, 379 mGy

Estimated blood loss: 100ml

Contrast: 55ml


120 mm

Post Operative Course

- Patient admitted overnight
- Discharged postoperative day 1
- Follow up most recently 3 months postoperatively and doing well

Conclusions

- Although open thoracic aortic repair remains the gold standard repair, endovascular aortic repair has far supplanted open repair due to significantly improved outcomes.

- We are continuing to push boundaries with regards to zone 0, 1 and 2 repairs in conjunction with extra-anatomic bypass.

- New generation devices are on the horizon to help cardiac and vascular surgeons treat complex aortic arch pathology.

Thank You

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