# Common Questions in Vascular Medicine

**(and one cardiologist's humble, relatively non-scientific, and completely editorialized guide on what to do about them)**

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No Disclosures

# Outline

I. **What is Vascular Medicine?**

II. Vascular Medicine Questions
   A. The Clotting/Bleeding Ones
      1) PEs
      2) DVTs
      3) Bleeders who Clot and Clotters who Bleed
   B. The Dissection Ones
   C. The PAD Ones
   D. The “Weird” Ones

# What is Vascular Medicine?

- Anything related to the vasculature

- Sub-field of Cardiology ("cardioVASCULAR medicine") but draws on several specialties with lots of collaborative work:
  - Internal Medicine
  - Cardiology
  - Hematology
  - Pulmonology
  - Rheumatology
  - Dermatology
  - Neurology
  - Vascular Surgery
  - Cardiac Surgery
  - OB/GYN
  - Neurosurgery
  - Hand Surgery

- We are often good old-fashioned internists and diagnosticians

# What is Vascular Medicine?

- A very Brigham story

- Vascular Medicine initially founded as a Section in the Department in 1984

- Several leaders in Cardiology got their start in Vascular Medicine:
  - Victor Dzau
  - Joseph Loscalzo
  - Peter Libby
  - Others went on to lead Vascular Medicine sections and divisions across the country
- The tradition has lived on 40 years later, and encompasses clinical care, research (basic, translational, and clinical) and education (dedicated vascular medicine fellowship)

# What is Vascular Medicine?

- Vascular medicine topics are not like other parts of cardiology like prevention, heart failure, coronary disease, etc
  - There's often a dearth of data
    - Guidelines are only guidelines
    - (If there are even guidelines available to begin with…)
  - Creates a lot of variability in practice patterns
    - Rely weighing risks/benefits, understanding pathobiology of disease, and sound logic/common "medical" sense

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# PEs

- The big question is: Intervention or No Intervention
- We most commonly use the ESC classification system

| Early mortality risk | | Indicators of risk | | | |
|---|---|---|---|---|---|
| | | Haemodynamic instability[a] | Clinical parameters of PE severity and/ or comorbidity: PESI class III–V or sPESI ≥I | RV dysfunction on TTE or CTPA[b] | Elevated cardiac troponin levels[c] |
| High | | + | (+)[d] | + | (+) |
| Intermediate | Intermediate–high | - | +[e] | + | + |
| | Intermediate–low | - | +[e] | One (or none) positive | |
| Low | | - | - | - | Assesment optional; if assessed, negative |

- **High-Risk**: Hemodynamic instability + imaging confirming or strongly suspecting PE (old AHA nomenclature: “massive PE”)
  - cardiac arrest
  - obstructive shock (systolic BP <90 mmHg despite an adequate filling status, in combination with end-organ hypoperfusion)
  - persistent hypotension (systolic BP >=40 mmHg for >15 min, not caused by new-onset arrhythmia, hypovolemia, or sepsis)

*Konstantinides et al 2019 EHJ 00:1-19*

# PEs

- The big question is: Intervention or No Intervention
- We most commonly use the ESC classification system
- Intermediate-Low risk and Low risk patients can be managed without Vascular Medicine input

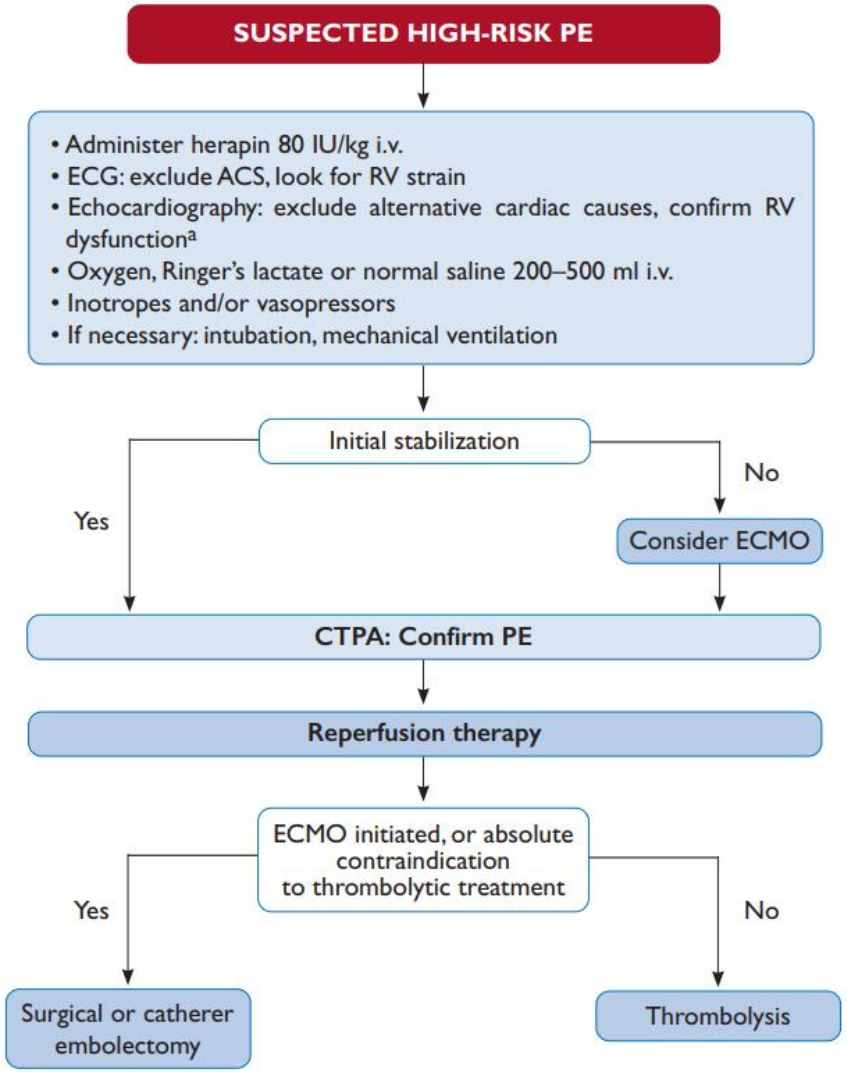
| Early mortality risk | | Indicators of risk | | | |
|---|---|---|---|---|---|
| | | Haemodynamic instability[a] | Clinical parameters of PE severity and/ or comorbidity: PESI class III–V or sPESI ≥I | RV dysfunction on TTE or CTPA[b] | Elevated cardiac troponin levels[c] |
| High | | + | (+)[d] | + | (+) |
| Intermediate | Intermediate–high | - | +[e] | + | + |
| | Intermediate–low | - | +[e] | One (or none) positive | |
| Low | | - | - | - | Assesment optional; if assessed, negative |

- **Intermediate-High Risk**: (old AHA nomenclature: "submassive PE")
  - Clinical features (PESI/sPESI)
  - RV dysfunction by CT-PE (RV/LV ratio > 0.9) or TTE (enlarged RV, McConnell's sign, RV/LV > 1, TAPSE < 16, S' < 10-11)
  - Elevated troponin
  - NOTE: elevated NTproBNP > 600 may also provide additional prognostic value

*Konstantinides et al 2019 EHJ 00:1-19*

# PEs

- High-Risk PE management:
  - Code PE team:
    - For ED patients (and perhaps Ortho patients)
    - Not other inpatients
    - ED physician, cardiac surgery, and vascular medicine (or pulm vasc)
    - Procedure or medical therapy
    - ECMO is a consideration but used less often here so far




*Konstantinides et al 2019 EHJ 00:1-19*

# PEs

- High-risk PE management:
  - Emergent embolectomy? ECMO?
  - If no, use tPA, unless any bleeding contraindications
    - "Full dose": 100 mg infusion over 2 hrs
    - "Half dose": 50 mg over 2 hours
    - "Code dose": 100 mg IV push
  - "Contraindications" are mostly based on experience from thrombolytics in STEMI
  - Possible surgery/ECMO after tPA (as a bailout)?

| Absolute Contraindications |
|---|
| • Any prior intracranial hemorrhage |
| • Known intracranial malformation/neoplasm |
| • <3 months from ischemic stroke |
| • Suspected aortic dissection |
| • Intracranial/intraspinal surgery within 2 months |
| • Recent (<3 mo) closed head/facial trauma |
| • Bleeding diathesis |

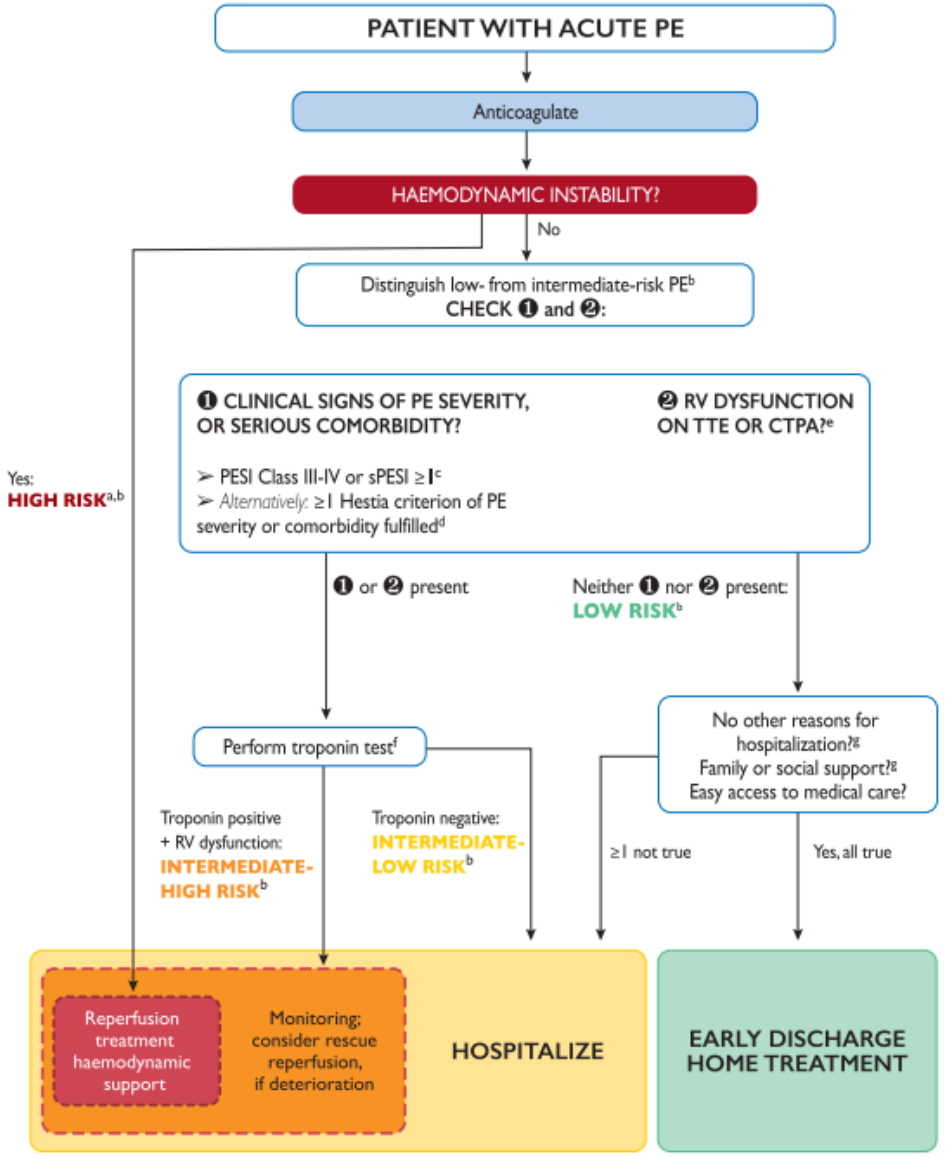
| Relative Contraindications |
|---|
| • >75 years of age |
| • Current anticoagulants |
| • Pregnancy |
| • Cardiopulmonary resuscitation > 10 minutes |
| • Recent internal bleeding (2-4 weeks) |
| • Uncontrolled hypertension (180/110 mmHg) |
| • Remote ischemic stroke |
| • Major surgery within 3 weeks |

- AHA PE scientific statement: *"The clinician is in the best position to judge the relative merits of fibrinolysis on a case-by-case basis."*
- AHA 2013 STEMI guidelines: *"Viewed as advisory for clinical decision making and may not be all-inclusive or definitive."*

*Jaff et al 2011 Circulation 123(16):1788-1830; O'Gara et al 2013 Circulation 127:e362-e425*

# PEs

- Intermediate-High Risk PE:
  - Code PE team variably gets activated
  - Procedure vs heparin (or enoxaparin)?
    - Catheter-based thrombectomy (FlowTriever) most common; less ultrasound-assisted catheter-directed tPA (EKOS)
    - Sometimes both
  - Does every intermediate high-risk patient “need” a procedure (Emergent? Urgent? Ever?)
  - Reality is there is plenty of grey
    - Theory: Early intervention may help prevent long-term complications and improve recovery
    - Reality: Never convincingly shown, but hints have been scattered throughout the literature



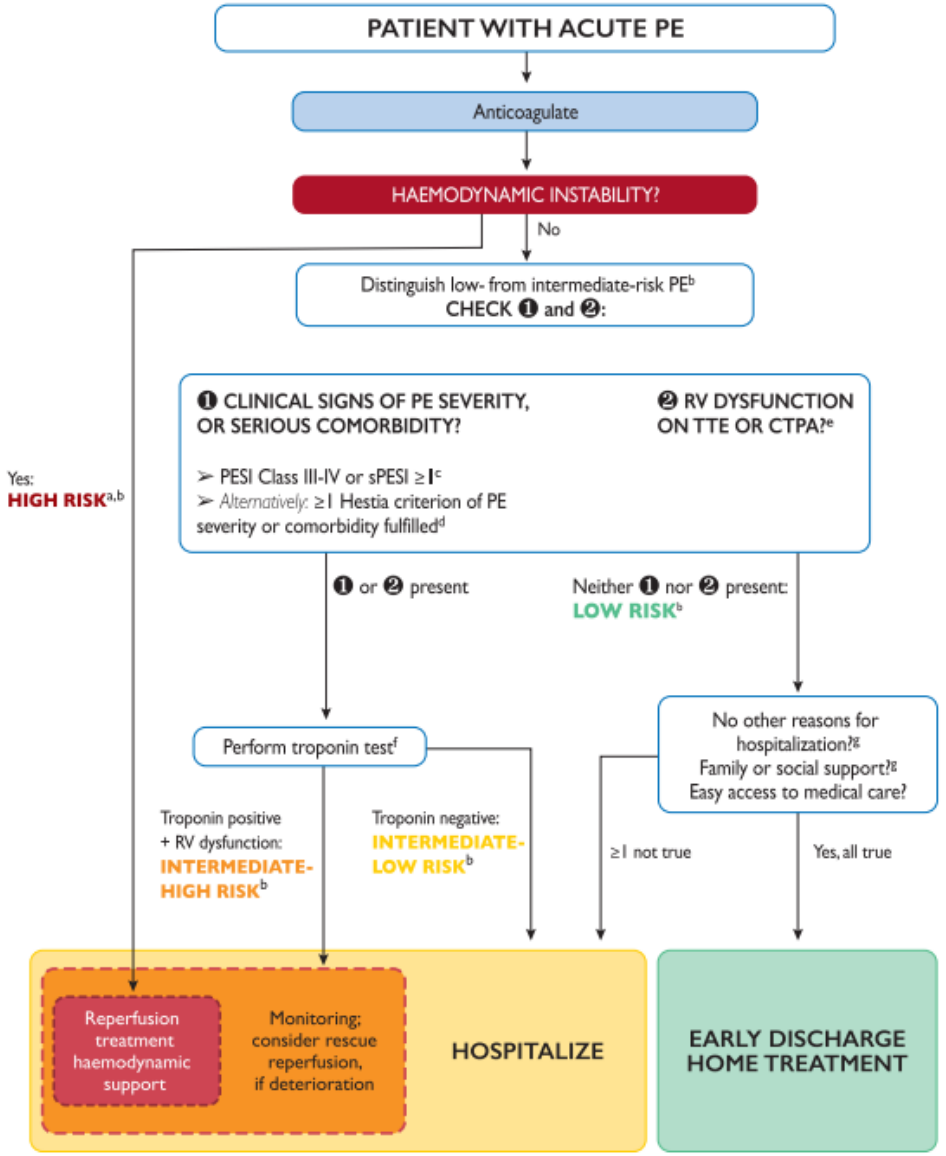

*Konstantinides et al 2019 EHJ 00:1-19*

# PEs

- How to deal with the grey zone?
- Flowtriever vs EKOS vs reduced-dose lytics vs heparin?

- Clinical gestalt
  - Borderline to frankly tachycardic (HR >90-95?)
  - Diaphoresis?
  - Sense of anxiety/unease?
  - BOVA score? sPESI? Shock index?
  - Respect syncope as a presenting symptom

- Think about a procedure in someone you want to get better faster
- In general, can often wait until the daytime but clinical scenario may prompt watching overnight in CCU/MICU




*Konstantinides et al 2019 EHJ 00:1-19*

# PEs

- Intermediate-High Risk PE: The Limited Data
  - ULTIMA trial (2014): 59 patients randomized to EKOS (n=30) or UFH (n=29) and assessed at 24 hr and at 90 days

| | Baseline | | 24 h | | Difference: Baseline vs 24 h | | 90 days | | Difference: Baseline vs 90 d | |
|---|---|---|---|---|---|---|---|---|---|---|
| | USAT | Heparin | USAT | Heparin | USAT | Heparin | USAT | Heparin | USAT | Heparin |
| RV/LV ratio, mean±SD | 1.28±0.19 | 1.20±0.14 | 0.99±0.17 | 1.17±0.20 | 0.30±0.19 | 0.03±0.16 | 0.92±0.15 | 0.96±0.16 | 0.35±0.22 | 0.24±0.19 |
| n | 26 | 29 | 28 | 28 | 25 | 28 | 26 | 27 | 23 | 27 |
| Between-group comparison | *P*=0.07 | | *P*=0.001 | | *P*<0.001 | | *P*=0.36 | | *P*=0.07 | |
| Within-group comparison | NA | | NA | | *P*<0.001 | *P*=0.31 | NA | | *P*<0.001 | *P*<0.001 |
| RV systolic dysfunction, n | | | | | | | | | | |
| None/mild/moderate/severe | 0/4/5/16 | 0/5/11/13 | 5/10/10/2 | 1/9/7/11 | 1.1±0.8* | 0.3±0.4* | 19/5/0/0 | 10/15/1/1 | 2.2±0.9* | 1.5±0.9* |
| Between-group comparison | *P*=0.37 | | *P*=0.01 | | *P*<0.001 | | *P*=0.003 | | *P*=0.01 | |
| Within-group comparison | NA | | NA | | *P*<0.001 | *P*=0.02 | NA | | *P*<0.001 | *P*<0.001 |

- The data for FlowTriever: it does what it says it can do: take out clot and decrease RV size at 48 hours… but WITHOUT killing people! (10% MAE at 48 hrs) Or causing massive bleeding! (1% at 48 hrs)
  - Yay FDA approval!

*Kucher et al 2014 Circ 129:479-86; Tu et al 2019 JACC Cardiovasc Interv 12:856-69*

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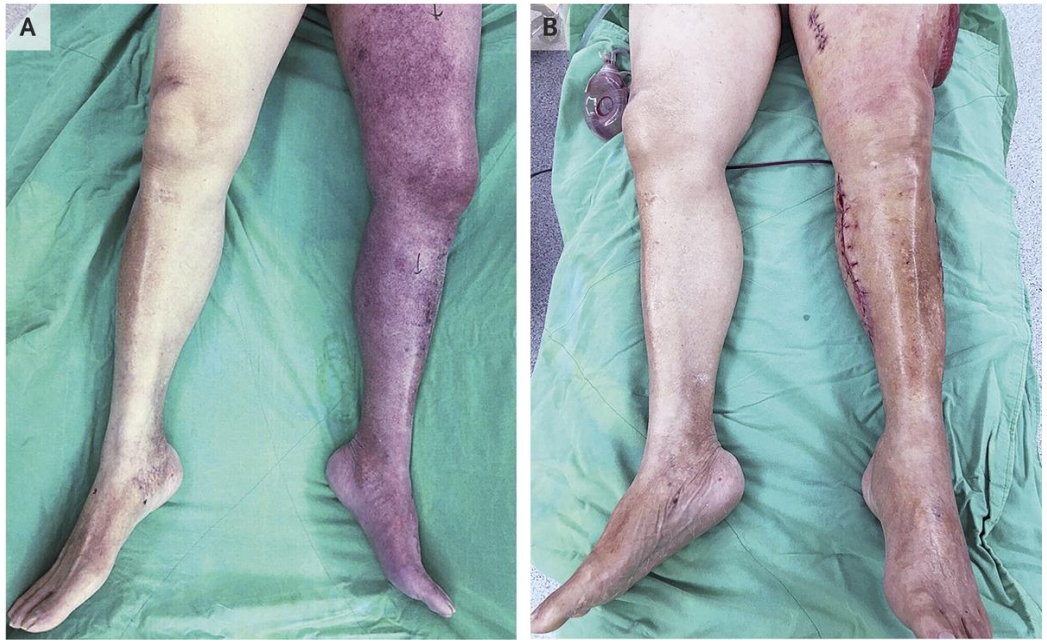
# DVTs

- My patient has a DVT but I can't anticoagulate! Help!!
  - Where is the DVT?
  - Above the knee vs below the knee
  - IVC filter if above the knee, typically IVC filter to bridge the time until can safely start full-dose anticoagulation
  - Usually ok with prophylactic-dose heparin 24-48 hours after a bleed/surgery
    - If post-surgery, surgical team will drive the ship on that
    - The service which "owns" the organ (or the surgeon/proceduralist who operated) determines bleeding risk
- Don't put in an IVC filter if you can anticoagulate!
  - "Prophylactic IVC filters" have fallen out of favor
  - No improvement in recurrent PE rates, similar bleeding profile, and increased chance of long-term complications (primarily recurrent DVT) if filter is left in

*Konstantinides et al 2019 EHJ 00:1-19*

# DVTs

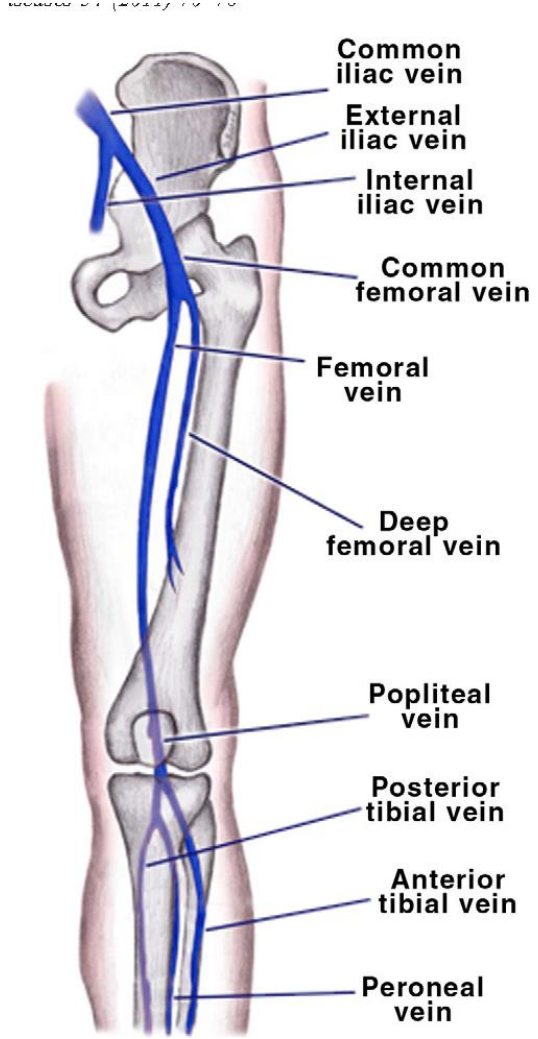
- Concern for phlegmasia cerulea dolens
  - Pain, edema, signs of arterial insufficiency, and characteristic cyanosis
    - impending venous gangrene
  - Phlegmasia alba dolens: white instead of cyanotic; usually a precursor
  - Indication for procedural intervention
    - thrombectomy +/- catheter-directed thrombolysis
    - May also need to address compartment syndrome
  - Often (but not always) encountered in conjunction with mechanical venous obstruction (pregnancy, mass, May-Thurner)




*Lai and Huang 2018 NEJM 378:658*

# DVTs

- Consider procedures in patients who have iliofemoral DVT and fail anticoagulation
  - ATTRACT trial showed patients with above-knee DVT undergoing thrombolysis + AC had the same incidence of post-thrombotic syndrome as AC alone
  - But patients with iliofemoral DVTs (as opposed to femoro-popliteal DVT) undergoing thrombolysis had reduced severity of post-thrombotic syndrome (based on a sub-study of ATTRACT)




*Vedantham et al 2017 NEJM 377:2240-52; Comerota et al 2019 Circulation 139:1162-73*

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# Bleeders who Clot and Clotters who Bleed

- No great data on how to approach these patients
  - “You anticoagulate until they bleed, and then you stop. When they stop bleeding, you anticoagulate again.” – David Faxon, M.D.

- Bleeders:
  - Hold AC until bleeding stops
  - Can restart with prophylactic dosing first
  - Can do low-goal heparin drip (aPTT 50-70) before going to standard goal (aPTT 60-80)
  - Titrate up enoxaparin, guided by anti-Xa levels (e.g., 40 daily, then 40 BID, then 0.5 mg/kg BID, then 1 mg/kg BID)

- Clotters:
  - Change anticoagulants
    - Dabigatran may be slightly more “bleedy” than other DOACs
    - Change to warfarin (enoxaparin) and shoot for higher INR (anti-Xa)
    - Don’t forget about fondaparinux; perhaps another more “bleedy” agent

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# The Dissection Ones

- Most common: Aortic dissections
  - Typically present as Code Aorta
  - ED physician, Cardiac Surgery, Vascular Surgery, Vascular Medicine
  - OR vs medical management

- Type A:
  - FIRST: HR < 60-65, typically with esmolol
  - SECOND: Systolic BP < 120
  - 98% of the time, cardiac surgery will take emergently to the OR
- Type B:
  - FIRST: HR < 60-65, typically with esmolol
  - SECOND: Systolic BP < 120
  - 98% of the time, they will be admitted to the CCU

**Table 4.** Management and Outcomes of Acute Aortic Dissection

| | Type A (n = 289) Management, No. (%) | | Type B (n = 175) Management, No. (%) | |
|---|---|---|---|---|
| | Surgical | Medical | Surgical | Medical |
| No. | 208 (72) | 81 (28) | 35 (20) | 140 (80) |
| In-hospital mortality | 54 (26) | 47 (58) | 11 (31.4) | 15 (10.7) |
| Total* | 101 (34.9) | | 26 (14.9) | |

*Hagan et al JAMA. 2000; 283:897-903*

# The Dissection Ones

- Rarely, type B patients may need a procedure:
  - Complicated type B dissections
  - Typically becomes apparent over 24-72 hours
  - Will often consider stent graft over open procedure for these (both done by Vascular Surgery) but case-dependent

## What Makes a Type B Dissection Complicated?

- Impending rupture
- Refractory hypertension
- End-organ ischemia
- Spinal cord ischemia/paraplegia
- Extension of dissection
- Localized pseudoaneurysm
- Continued, intractable pain

*DeSanctis et al NEJM 1987; 317:1060-1067*
*Fann and Miller Ann Vasc Surg 1995; 9(3):311-323*

# The Dissection Ones

Branch vessel (non-aortic) dissections:

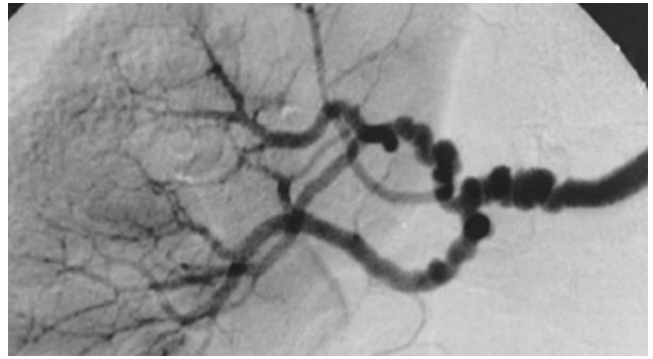
- Non-inflammatory arteriopathies:
  - Fibromuscular dysplasia (FMD)
  - Spontaneous coronary artery dissection (SCAD)
  - Segmental arterial mediolysis (SAM)

- Manifestations of a common underlying pathophysiology?
- Or multiple pathophysiologies with final common phenotypes?

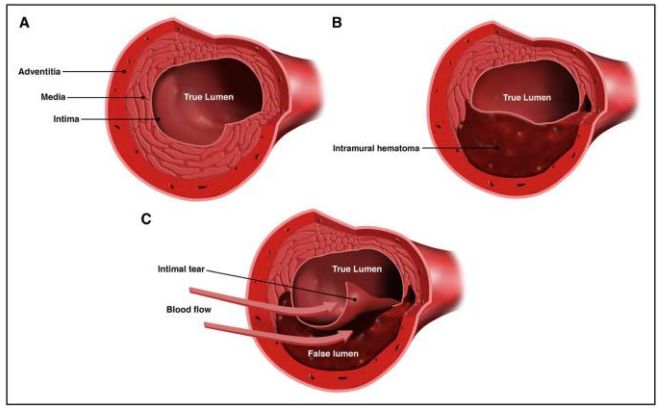
- Need to rule out inflammatory arteriopathy (vasculitis)

# The Dissection Ones

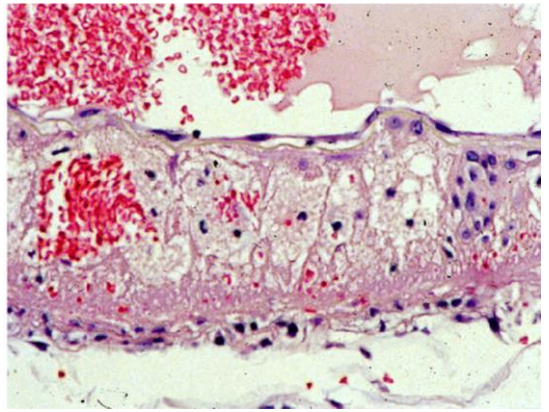
- Fibromuscular dysplasia (FMD)
  - Non-atherosclerotic, non-inflammatory arterial vascular disease, with classic “string of pearls” anatomy on imaging



- Spontaneous coronary artery dissection (SCAD)
  - Epicardial coronary dissection or intramural hematoma (IMH) that is not a result of trauma, inflammation, or iatrogenic injury




- Segmental arterial mediolysis (SAM)
  - Least defined; path shows multifocal mediolytic lesions at the media-adventitia border



# The Dissection Ones

- Non-Aortic Dissections:
    - CTA imaging (if not done already) for better anatomic definition
    - Head-to-pelvis imaging once to identify other areas that may need surveillance (aneurysms, silent/prior dissections)
    - HR <60-65 and systolic BP < 120 mmHg
    - For FMD and SAM, we will often anticoagulate for 1-3 months, although data are sparse for this

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# The PAD Ones

- PAD often is not an acute, inpatient consult question
  - ED consults often go to Vascular Surgery

- The 5 P's of acute limb ischemia:
  - Pain
  - Pulselessness (yes, you have to palpate AND doppler)
  - Pallor
  - Paresthesia
  - Paralysis
  - Poikilothermia – ie cool/cold extremity
- Surgical Emergency: revasc or amputate

# The PAD Ones

- Critical Limb-Threatening Ischemia
  - Limb pain for > 2 weeks
  - Non-healing wounds or gangrene
  - Objectively demonstrated occlusive arterial disease
    - ABI, MRA (better than CTA due to less issue with calcium artifact)

- Most often Vascular Surgery ends up dealing with this as well; interventional cardiology may also consider percutaneous options
  - BEST-CLI (2022):
    - If someone has appropriate venous conduit, surgical revascularization is better than endovascular approach
    - If there is no appropriate venous conduit, then both are options

*Farber et al 2022 NEJM 387:2305-16*

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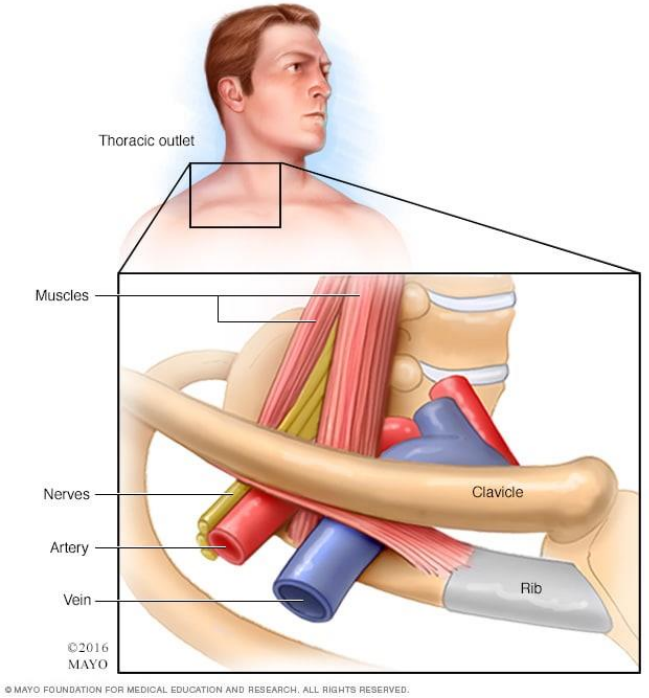
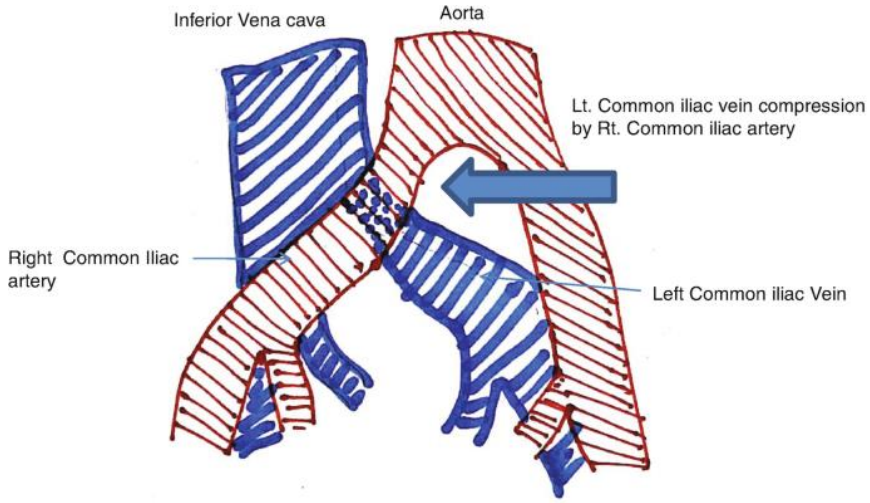
# The “Weird” Ones

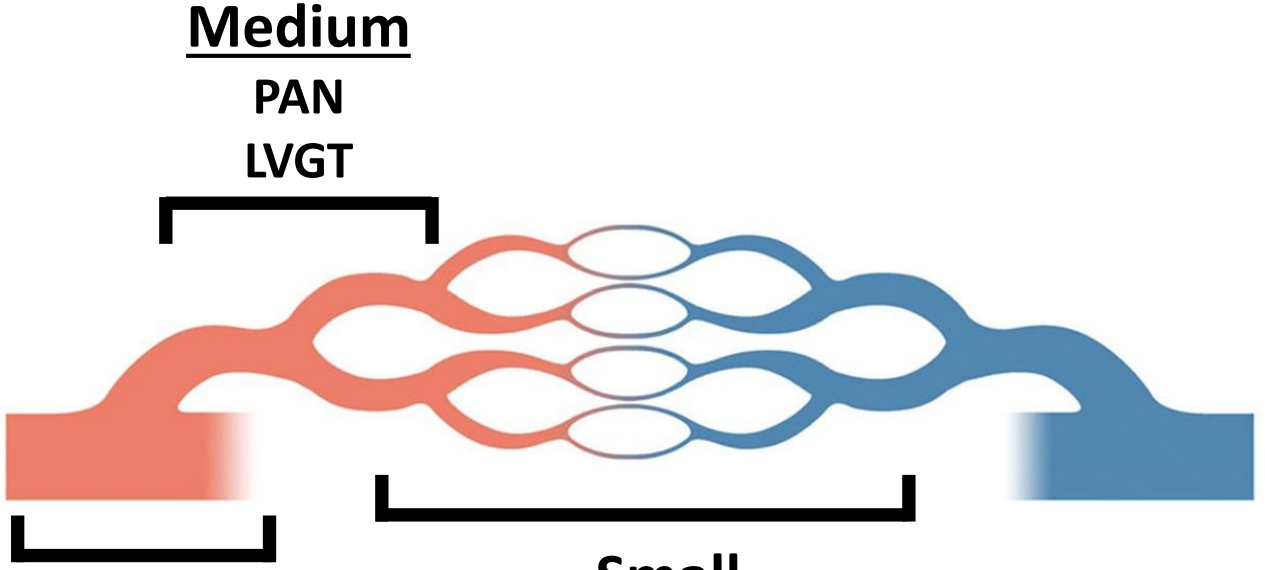
- Clots in Odd Places
  - Abdominal venous thrombosis:
    - Recent surgery and associated inflammation
    - Pregnancy
    - APLS
  - Venous stenosis/compression leading to clots
    - Paget-Schroetter Syndrome
    - Thoracic Outlet Syndrome
    - May-Thurner Syndrome
      - May-Thurner anatomy is very common (20-25% of all people)



# The “Weird” Ones

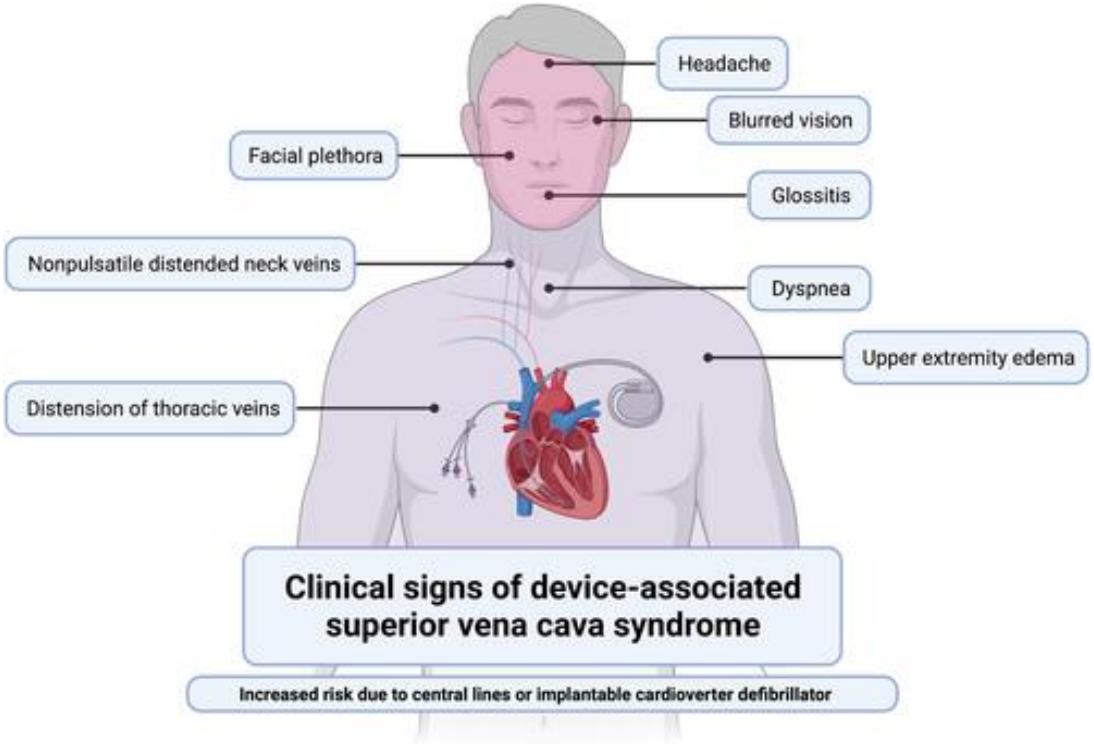
- Vasculitis
  - Large/medium vessel
    - Imaging
    - Rheumatology evaluation
  - Small vessel
    - Rheumatology evaluation
    - Microvascular dysfunction?
      - Severe Raynauds
      - Vasodilator therapy
      - Alpha blocker, PDE5i, maybe CCB
      - Low-dose epoprostenol infusion




**Variable:** IgG4-related disease, Behcets, Erdheim-Chester

# The “Weird” Ones

- SVC Syndrome
  - What’s causing the blockage?
  - Usually compression or device-related obstruction with clot
  - Anticoagulate; consider stenting?
    - Interventional vs Vasc Surg
- Arterial/venous abnormalities
- Issues with AV fistulas
  - Usually vascular surgery is involved as well
- Tumor Thrombus (RCC)



# Thank You!

**As my ACS used to say, “Never stop THINKING!”**

**Questions?**