Clinical Pearls:

1. A **deep vein thrombus (DVT)** can “grow up” to become a pulmonary embolism (PE), but is part of the same spectrum of venous thromboembolism.

2. **Pathophysiology**: PE causes exponential increase in pulmonary vascular resistance. The thin-walled right ventricle (RV) fails. Left ventricle (LV) fails in sequence dropping cardiac output and leading to coronary ischemia of RV (even if no underlying stenosis).

3. **Risk stratification**: High if hypotension present. Low if minimal symptoms, and lack of RV strain on imaging. Intermediate-low if less severe RV dysfunction and patient looks well. Intermediate-high if more severe RV strain, increase RV:LV diameter, patient “looks ill”, high clot burden, or sPESI score = 1 or higher.

4. **sPESI**: age >80; h/o cancer, CHF, chronic lung disease; HR >110 bpm; systolic BP <100 mmHg; SaO2 <90%. Zero = low risk. One or above = high risk.

5. **Treatment of low or intermediate-low risk patients**: Anticoagulation with parenteral agent, vitamin K antagonist with a parenteral anticoagulant bridge, or a direct acting oral anticoagulant (DOAC).

6. **Treatment of intermediate-high risk patients**: Consider IV unfractionated heparin for easy on/off since these patients might need more advanced therapy if they become unstable or their symptoms fail to improve on anticoagulation. -Dr Friedman’s expert opinion.

7. **Treatment of high risk PE** (previously called “massive”) requires advanced therapies (see below).

8. **Advanced therapies for PE**: Systemic thrombolytics, catheter-directed thrombolytics, thrombectomy (either surgical or catheter directed).

9. **IVC filters**: Still controversial. Main indication is patient with DVT who cannot receive anticoagulation. Some experts place temporary filter if “large clot burden” and “low cardiopulmonary reserve”, but long term benefit has not been shown and future DVT risk increase [add citation]. Don’t forget to remove it!

10. **When can I discharge my patient with PE?** Patient is off oxygen, HR and BP are stable. Symptoms have improved and patient can tolerate ambulation. -Dr Friedman’s expert opinion.

11. **How should I handle subsegmental PE?** The evidence is not clear. Most patients still need treatment. Consider watchful waiting in patients without risk factors for VTE, who are hemodynamically stable with good RV function, no other clot burden and reliable follow up. -Dr Friedman’s expert opinion.
In-Depth Show-Notes:

**Some Basic Background**

A *venous thromboembolism (VTE)* is an aggregation of platelets and blood cells to form a plug somewhere in a vein.

**Deep vein thrombosis (DVT)** is when a clot occurs in a “deep” vein (as opposed to a superficial vein) and can cause pain, redness, swelling, and pitting edema. They can sometimes be “provoked” by an underlying cause: hypercoagulability disease, inflammation from cancer or infection, some medications, prolonged immobility (e.g. long flights or surgery recovery), or trauma (e.g. some type of venous catheter or IV). DVTs are confirmed using ultrasound.

A *pulmonary embolism (PE)* is a more concerning form of VTE or when a DVT migrates to block the pulmonary artery, the vessel going to the lungs. This can present with shortness of breath, rapid breathing, rapid heart rate, low oxygen saturation, and sometimes fever. The inflammatory response to a PE can sometimes cause hemodynamic instability (i.e. hypotension, shock, and death). PEs are confirmed using contrast enhanced CT of the chest, or sometimes a “V/Q scan.”

**PE and DVT are part of a spectrum**

- Best thought of as “same disease with different manifestations.” DVTs “grow up” to be PEs. In PE dopplers, show ½ have DVT in leg, and the other ½ likely had a DVT that embolized.

**Deep Venous Thrombosis (DVT)**

- Treatment is to alleviate symptoms but also prevent progression to PE
- **Phlegmasia (cerulea dolens):** Rare, severe DVTs w/associated with severe pain, swelling/edema & limb cyanosis. High risk of massive PE or may result in tissue necrosis
- **Post-Thrombotic Syndrome:** DVT may not fully resolve and can result in chronic symptoms

**Pulmonary Embolism (PE)**

**Cardiac Effects of PE**

- PE leads to increased pulmonary vascular resistance (exponential worsening, not linear)
- As clot burden worsens, PVR increases, the right ventricle (RV)’s thin walls can fail
- RV failure -> Decreased LV output -> hypotension and shock -> RV ischemia
**Classification of Pulmonary Embolism**

- Hemodynamics are critical in outcomes so classification is based on hemodynamics
- **Low Risk**: no evidence off heart strain or hemodynamic instability
- **Intermediate Risk** (previously “sub-massive”): Presence of RV dysfunction (such as septal bowing visualized on echo or CT) and/or evidence of myocardial injury (ischemic EKG changes, elevated cardiac troponin, elevated BNP) without systemic hypotension.
- **High Risk** (previously “massive”): Patients with persistent shock / hypotension as a result
- [European Society of Cardiology Guidelines](https://www.scar guidelines.org) use a different classification to risk-stratify PE which includes “low-intermediate” and “high-intermediate” risk. Summary Table [here](https://www.scar guidelines.org).

**Clinical Evaluation of a patient with PE**

- sPESI (Simplified PE-Severity Index) score shown alongside the original PESI score

**Heart rate**: excellent barometer for risk *(expert opinion)*

**Blood pressure**: extreme worry if hypotension / shock. Stop calculating risk factors and do something!

**Echocardiography**: looking for right ventricular strain:
- Marked RV dilation is concerning.
- LV under-filling may suggest impending hemodynamic collapse
- **McConnell’s Sign**: RV dysfunction with akinesia of the mid-free wall

**Biomarkers**:
- Elevated troponin and BNP tend to be seen in patients with worse outcomes
- Elevated Lactate: This can be a poor prognostic sign

**The “eyeball” test**: this should be taken seriously even in absence of objective findings

**CT Scan**: Great for making diagnosis, but it’s not just about the diagnosis
- Can sometimes see RV dilation before the echocardiogram
- Central clots and clot burden tend to lead to worse outcomes

*Is there residual DVT?* Anecdotal evidence suggests patients with high-residual clot burden may do worse. *-Dr Friedman on The Curbsiders #92*

**History of syncope** suggests patient had enough RV obstruction to drop their cardiac output

**Who is low risk?**

- A young person, short of breath with PE, but normal vital signs and no need for oxygen is likely a patient who can be started on anticoagulation and discharged home!
- **Hestia Criteria**: Consider inpatient admission if any one of the criteria are true

**Pulmonary Embolism Treatment**

**Management of the intermediate-low or intermediate-high risk PE**

- Most intermediate-low will be fine with single agent anticoagulation
- Consider more therapy for those with: borderline hypotension, possible RV dysfunction, large clot burden, no symptom improvement after 24 hours of anticoagulation
Choices for anticoagulants
1. Low-molecular weight heparin (LMWH) e.g. Lovenox
   a. Predictable, quick, safe
2. Unfractionated heparin
   a. For higher risk patients
   b. Consider if thinking catheter directed lysis (easier peri-procedural management)
3. Target-specific oral anticoagulation
   a. Low-risk / Low Hestia score

Thrombolysis/clot retrieval
1. Systemic thrombolytics
   a. If catheter directed therapy is not readily available or for emergent treatment
2. Catheter-directed thrombolysis (ULTIMA, Seattle II, PERFECT Registry)
   a. Performed by interventional radiology or interventional cardiology
   b. Catheters placed in sheathes, run directly into the clot(s) and used to administered a smaller total amount of TPA
3. Clot- retrieval (Thrombectomy)
   a. Indication: absolute contraindication to low doses of TPA (e.g intracranial malignancy with known hemorrhage)

IVC Filters
- Not much more additional benefit to anticoagulation
- Consider in someone who cannot get anticoagulation or in sick patients with lots of residual clot on anticoagulation
- Remember to go back and get the filter out! Not a long term solution: IVC filters migrate, can allow for clot propagation, can pierce through the IVC

When to discharge?
- Off oxygen? BP & HR okay? Symptom improvement? Ambulatory?
- Ensure patient education

What to do with the incidental PE?
Isolated, subsegmental PE - not great evidence either way. Some studies say watchful waiting is OK.
- Ensure hemodynamic stability, good RV function, no other clot burden and reliable follow-up before deciding to NOT treat
Lobar or segmental clots (e.g. found incidentally in COPD patients) should not be ignored.

The PERT Team (Pulmonary Embolism Response Team)
Multidisciplinary group of PE experts who work together to help decide the best care for a given patient

Goals: Listeners will develop a standardized approach to risk stratification and management of patients presenting to the hospital with pulmonary embolism.
Learning objectives:
After listening to this episode listeners will…
1. Recognize the common signs and symptoms of PE/DVT.
2. Recall the risk factors for PE/DVT.
3. Explain the pathophysiology of PE and the “inflammatory milieu” associated with PE.
4. Stratify risk in patients with PE using clinical judgement and various scoring criteria.
5. Determine appropriate care setting e.g. internal medicine vs. ICU
6. List anticoagulation options and how to choose an appropriate one
7. Counsel patients regarding anticoagulation & contraindications to anticoagulation.
8. Know what to look for during a patient’s admission / when to seek expert consultation (pulmonary/ICU vs IR).
9. Decide what, if any work up, should be done in a patient found to have a PE (additional imaging, labs. etc).
10. Know the basics regarding IVC filters: when they are indicated, risks and benefits, management after hospital discharge.

Disclosures: Dr Friedman and The Curbsiders report no relevant financial disclosures

Time Stamps
- 5:10 - Start of Interview / Introduction
- 10:00 - Case Presentation - Introduction of DVT / PE Spectrum and Pathophysiology
- 16:18 - How to Triage and Work-Up Pulmonary Embolism (CT, Echo, troponins, “eyeball”)
- 23:30 - Other ways to risk-stratify (Other CT findings, clot burden)
- 28:45 - What does a low-risk patient look like?
- 30:35 - When to consider treatment modalities other than general anticoagulation
- 35:12 - Anticoagulation treatment options
- 36:38 - Next step intensive interventions after anticoagulation
- 42:00 - Evidence behind catheter-directed lysis
- 44:05 - IVC Filter discussion
- 47:22 - Discharge criteria for PE
- 50:00 - Incidental PEs
- 52:40 - Interdisciplinary PERT Team

Links from the show:
1. Recommended Medical “App:” UpToDate
2. Oren Book mention: The Righteous Mind, Thinking Fast and Slow
3. MDCalc: Simplified PE-Severity Index (sPESI) score
4. MDCalc: Original PESI score


8. MDCalc: [Hestia Criteria](#).


**Other Recommended Resources:**

1. [JAMA - Does This Patient Have Pulmonary Embolism?](http://thecurbsiders.libsyn.com/92-pulmonary-embolism-for-the-internist)

2. [ATS Reading List on Pulmonary Embolism](http://thecurbsiders.libsyn.com/92-pulmonary-embolism-for-the-internist)

3. Chaterjee **Meta-analysis:** Thrombolytics reduce mortality in submassive PE

**Permalink URL:** [http://thecurbsiders.libsyn.com/92-pulmonary-embolism-for-the-internist](http://thecurbsiders.libsyn.com/92-pulmonary-embolism-for-the-internist)