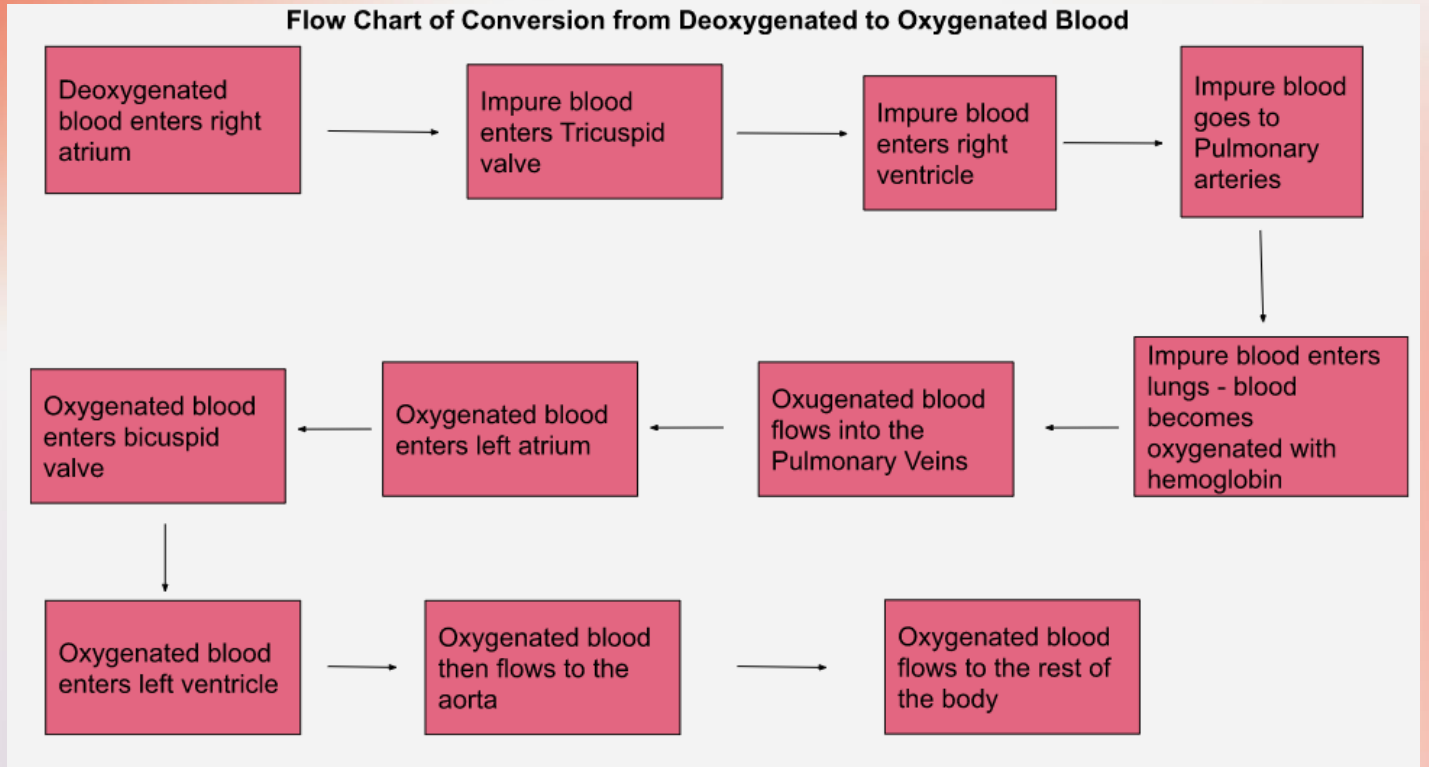
# Pathophysiology of PE

Richa Mahajan and Mala Niverthi, The GTF Group
Rajan Memorial Lecture January 8, 2023

# Introduction

- Pulmonary Embolism (PE) is one the most dangerous conditions that occurs to nearly 900,000 people every year in the United States.
- PE is caused by the embolus traveling from the DVT site to the lungs.
- According to Dr. Joseph Caprini, PE is one of the most preventable conditions.
- We researched into the various aspects of pathophysiology of PE.

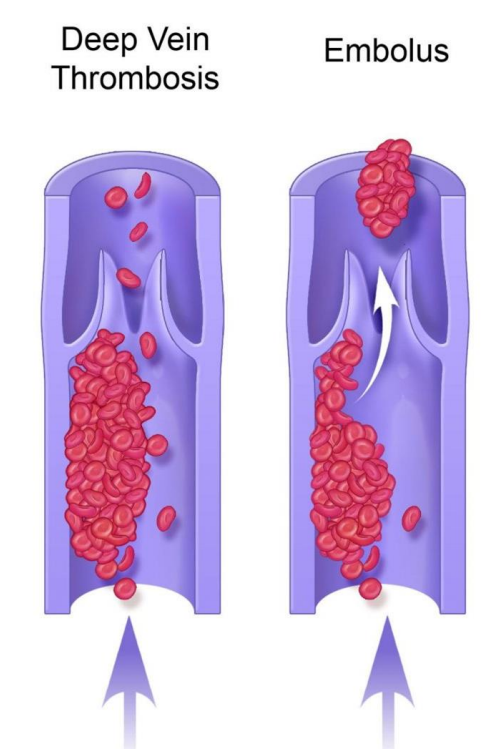
# Figure 1: Physiology of Pulmonary Circulation

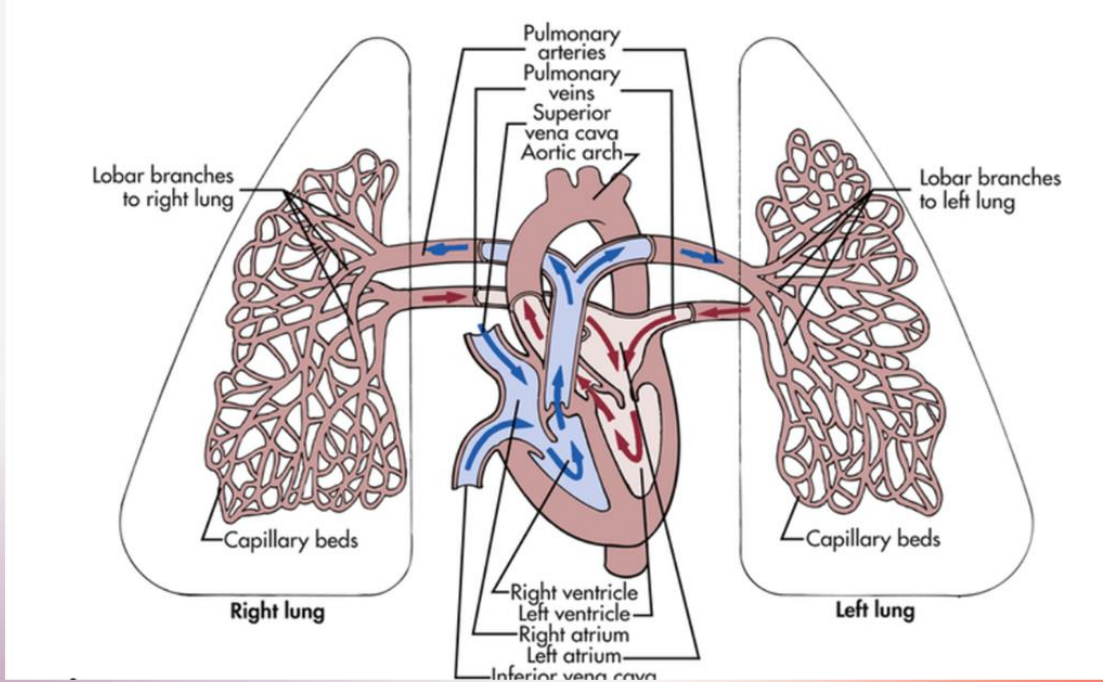


# Three mechanisms

1. Ventilation, the movement of air into and out of the lungs (breathing).
2. Diffusion, the movement of gases between air spaces in the lungs and the bloodstream.
3. Perfusion, the movement of blood into and out of the capillary beds of the lungs and into the body organs and tissues.

# Figure 2: Formation of embolus & Pulmonary Embolism



# Hemodynamic Decompensation

1. In normal lungs, ventilation and perfusion are well matched.
2. PE: Transfer of oxygen is impaired when alveolar ventilation to pulmonary capillaries is reduced relative to blood flow.
3. Physical obstruction of blood flow.
4. Release of humoral factors, such as serotonin from platelets, thrombin from plasma, and histamine from tissue.
5. PE increases pulmonary vascular resistance due to vasoconstriction caused by hypoxia.
6. The mean pulmonary artery pressure can be doubled to approximately 40 mm Hg.

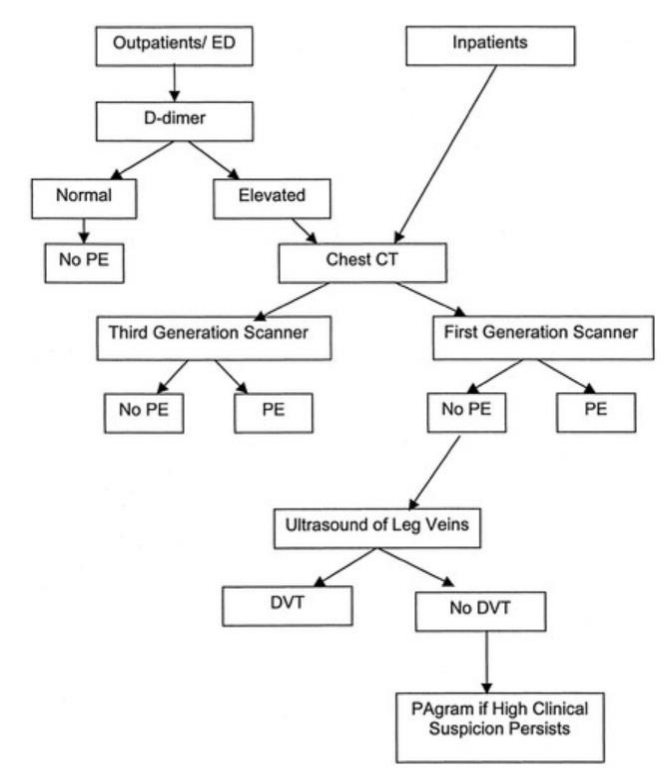
# Hemodynamic Decompensation, contd.

6. Under extreme circumstances in patients with chronic thromboembolic pulmonary hypertension, the pulmonary arterial pressure can exceed the systemic arterial pressure.
7. Most patients maintain a normal systemic arterial pressure for 12 to 48 hours and may give the impression of being hemodynamically stable.
8. Right ventricular enlargement attributable to pressure overload causes a leftward shift of the interventricular septum, which is a manifestation of interventricular dependence.
9. Right ventricular contraction continues even after the left ventricle starts relaxing at end-systole.

# Hemodynamic Decompensation, contd.

10. The interventricular septum flattens during systole and then bulges toward the left ventricle, with paradoxical septal motion that distorts the normally circular left ventricular cavity.
11. There is diastolic left ventricular impairment, attributable to septal displacement, reduced left ventricular distensibility, and impaired left ventricular filling during diastole. Left atrial contraction has a greater than normal contribution to left ventricular filling
12. Gas exchange acute PE impairs the efficient transfer of oxygen and carbon dioxide across the lung
13. Decreased arterial PO2 (hypoxemia) and an increase in the alveolar-arterial oxygen tension gradient are the most common gas exchange abnormalities.

# Figure 3: Diagnosis of PE



# Conclusions

PE is one of the deadliest preventable thrombotic conditions. PE is caused by an embolus traveling from DVT to the lungs. It is therefore important to be aware of the pathophysiology of PE so we can actively work to lower the incidence rate of PE.

# Acknowledgements

We would like to thank Dr. Laddu and the GTF Board for guiding us through this project as well as our parents for their constant encouragement.

# References


- Goldhaber, Samuel Z., Elliott, Gregory C., Acute Pulmonary Embolism: Part I: Epidemiology, Pathophysiology, and Diagnosis. Review: Clinical Cardiology: New Frontiers. November 13, 2022.

- Riedel, Martin. (2001 February 1). Acute pulmonary embolism 1: pathophysiology, clinical presentation, and diagnosis. BMJ. November 13, 2022.

- Yang, P., Li, H., Zhang, J., Xu, X. (2021, June 29). Research progress on biomarkers of pulmonary embolism. Wiley Online Library. November 13, 2022.