Pathophysiology of DVT

Ishaan Dhaneshwar and Shriya Sawant, the GTF Group
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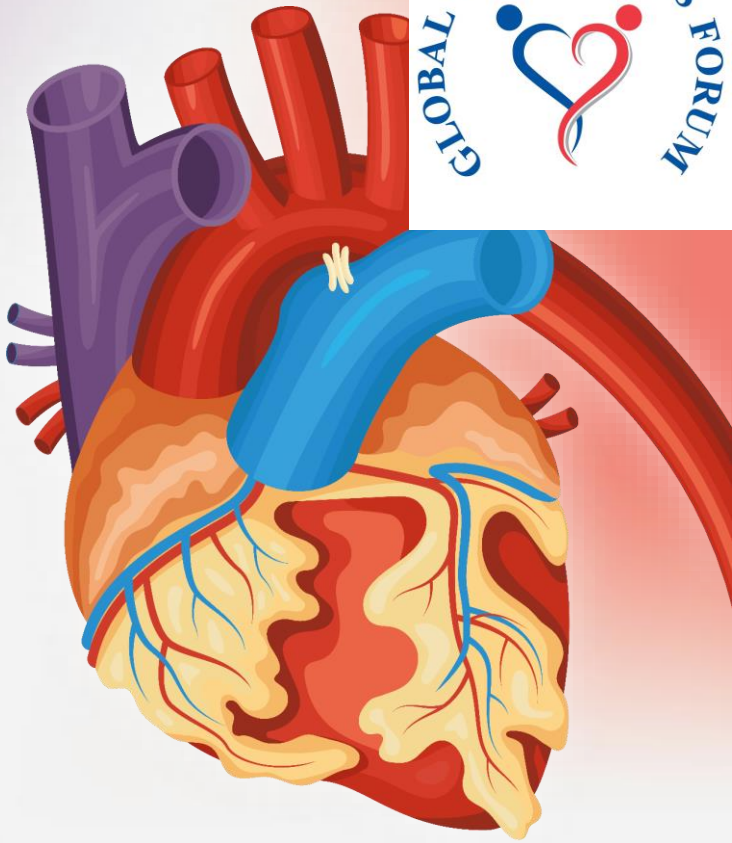
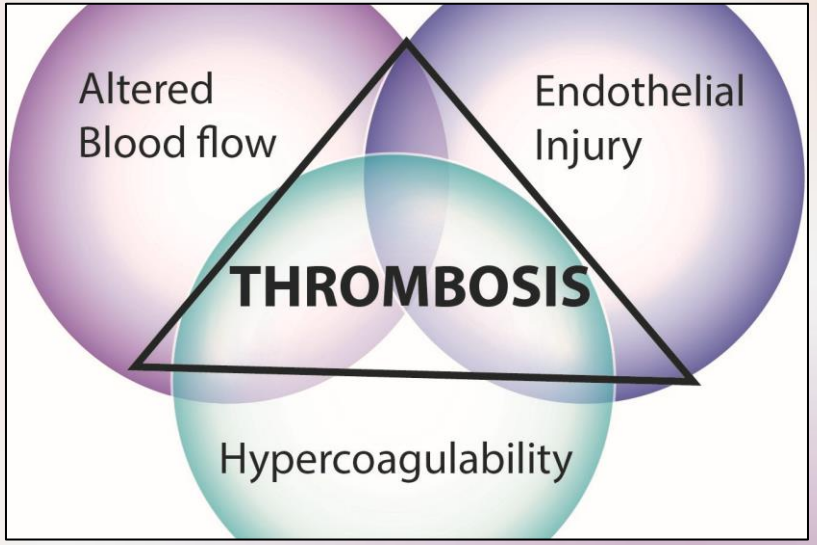


Introduction

- DVT is one of the most common and serious forms of VTE.
- The incidence of DVT is 200,000 cases per year in the US alone.
- Roughly 50% of all DVT cases result in a PE.
- We already know the triggers that can produce DVT.
- Today, we are going to take a deeper look at the pathophysiology of DVT.

Professor Rudolf Virchow (1821–1902)

Virchow's Triad



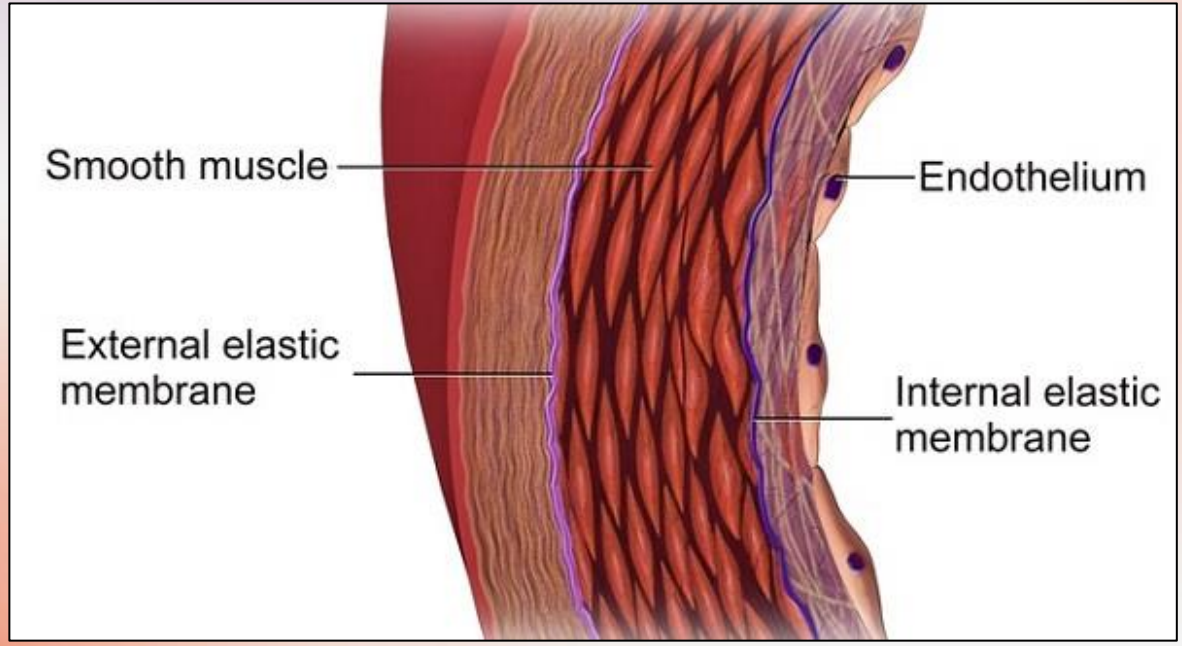
Virchow’s Triad, continued

- Virchow’s classic triad for thrombus formation consists of venous stasis (stagnation), vessel wall damage, and a hypercoagulable state.
- Prolonged, cramped sitting during long-distance travel interferes with venous flow in the legs creating venous stasis.
- Seat-edge pressure to the popliteal area (the back area between the leg and the thigh) of the legs can aggravate venous stasis as well as contribute to vessel wall damage.

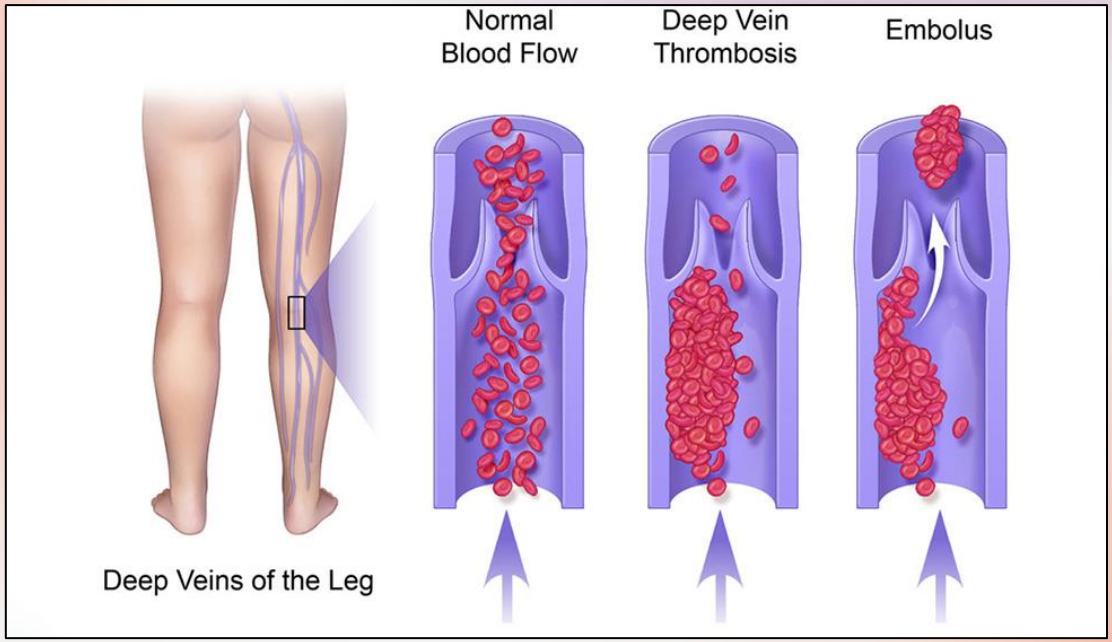
Virchow's Triad, continued

- Studies of the pathophysiologic mechanisms for the increased risk of VTE after long-distance travel have indicated that venous stasis appears to play a major role.
- Other factors specific to air travel may result in activation of coagulation particularly in travelers with preexisting risk factors for VTE.
- Activation of coagulation may result from an interaction between cabin conditions (such as hypobaric hypoxia) and individual risk factors for VTE.

The Blood Vessel



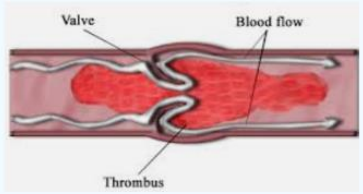
DVT


Normal
Blood Flow
Deep Vein
Thrombosis
Embolus
Deep Veins of the Leg

Formation of Thrombus and Embolus

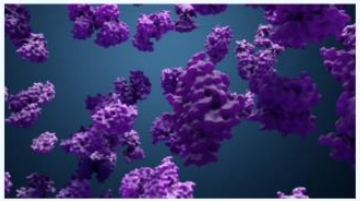
Formation of a Blood Clot

1. Blood clogs the vein



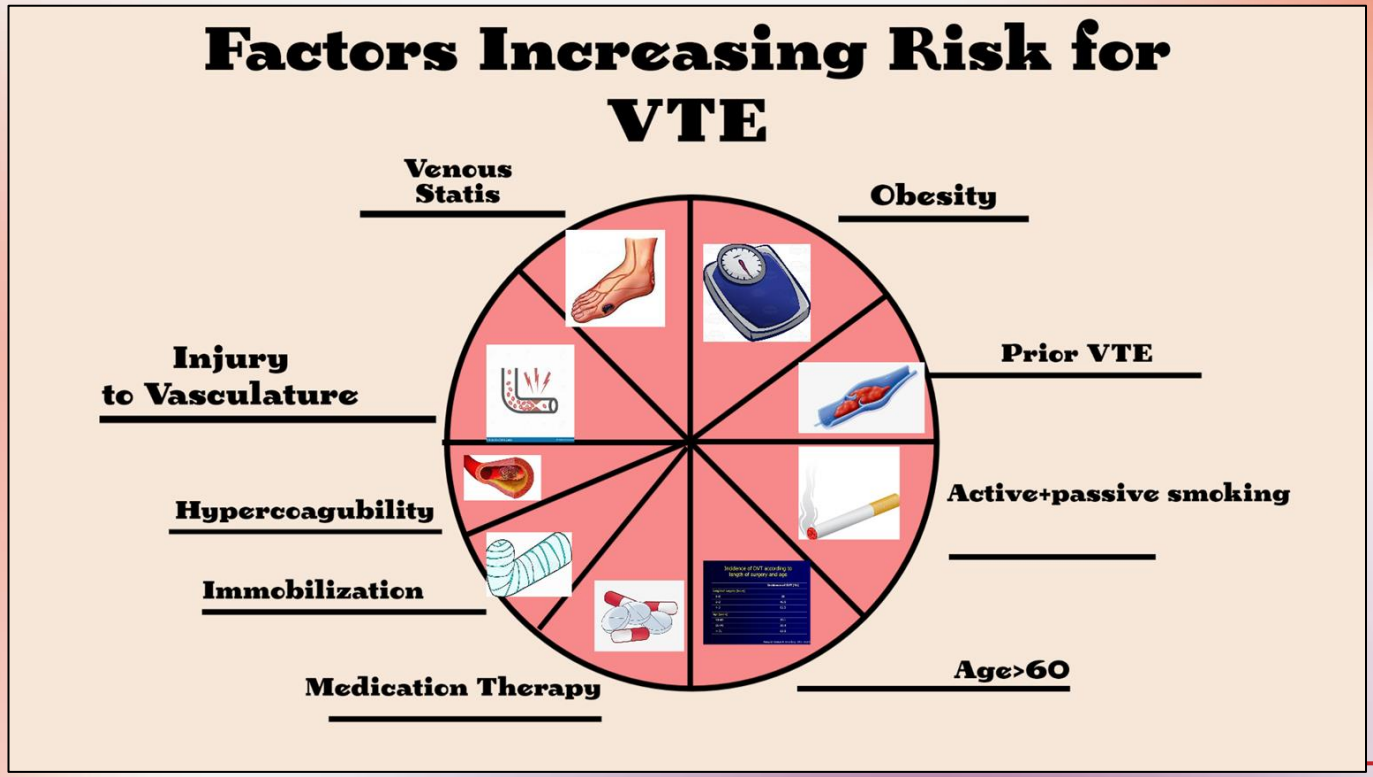
2. Hypoxia

3. Release of Enzymes



4. Symptoms of DVT Arise

Factors Increasing Risk of VTE



Pathogenesis of DVT

Inappropriate thrombus formation is a disruption of homeostasis and may result from an alteration in any of the factors listed below. The dominant influence, and the one factor that by itself can lead to thrombosis, is endothelial injury.

Endothelial Injury: Endothelial injury causes subendothelial collagen exposure and platelet adherence, among other changes; many factors can contribute to the injury, including hypertension, vasculitis, scarred valves, bacterial endotoxins, cholesterolemia, and chemicals from cigarette smoke.

Abnormal (reduced) blood flow

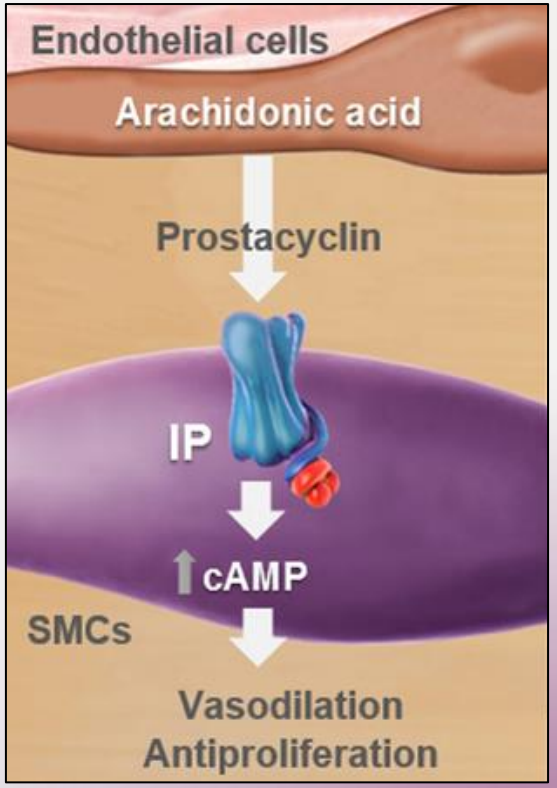
Stasis can cause endothelial injury, predisposing a patient to thrombosis.

Pathogenesis of DVT- Continued

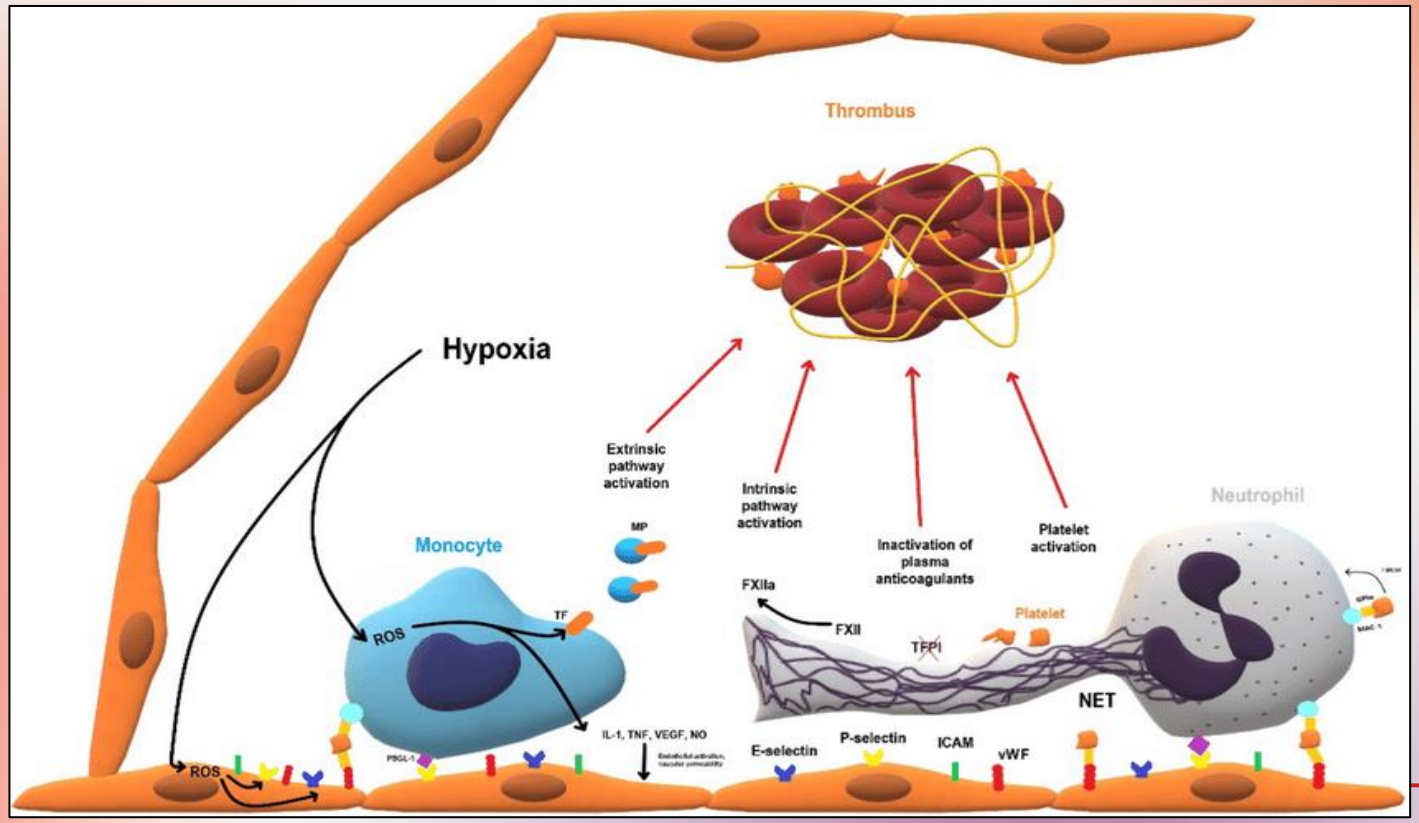
Hypercoagulability (increase in the tendency of the blood to clot)

- An increased tendency of the coagulation pathway that places the patient at risk for thrombosis.
- Advancing age, surgery, fractures, burns, myocardial infarction, and cancer can cause hypercoagulability
- Increased hypercoagulability causes an increase in platelet aggregation, which results in:
- Reduction in prostacyclin, a potent vasodilator (dilates the vessel) and inhibitor of platelet aggregation that is released by the endothelium.

Prostacyclin Pathway



Process of Thrombosis



Conclusions

- DVT is a global problem affecting all ages, races, and genders.
- DVT does not discriminate and needs to be acknowledged as a major health threat.
- In this presentation, we have explained the pathophysiology of DVT.
- Virchow's Triad plays an important role in the genesis of DVT.
- The primary problem with DVT is sluggish circulation, followed by hypoxia, endothelial injury, and the release of enzymes.
- To understand the causes, symptoms, and management of DVT is the responsibility of every citizen.

Acknowledgments

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- A special thank you goes out to our viewers for listening to us today.

References

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