# THROMBUS

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**Primary Disciplinary Field(s): Pathology**, **Hematology**, **Cardiology**

## 1. Core Definition

A thrombus is defined as a pathological, solid mass formed from blood constituents--primarily platelets, fibrin, and trapped cellular elements--that develops within the circulatory system, specifically within a blood vessel (artery, vein, or capillary) or a chamber of the heart. Crucially, a thrombus remains fixed at its site of origin, adhering to the vascular wall. The process of its formation is termed **thrombosis**. Thrombosis represents a failure of the normal hemostatic mechanism, which is designed to arrest bleeding (hemostasis) only following injury, rather than initiating inappropriate clotting in intact vessels. When a vessel is damaged, hemostasis is protective; when thrombosis occurs spontaneously or due to underlying pathology, it becomes detrimental, leading to obstruction of blood flow (ischemia) or detachment, which carries a much wider systemic risk.

The clinical distinction between a thrombus and an **embolus** is vital for diagnosis and treatment. A thrombus is stationary; an embolus is any intravascular solid, liquid, or gaseous mass carried by the blood to a distant site from its point of origin. While a piece of detached thrombus is the most common form of embolus, the term also encompasses fat droplets, air bubbles, atherosclerotic debris, or tumor fragments. When a thrombus breaks free and travels downstream until it lodges in a smaller vessel, blocking perfusion, it is then referred to as a thromboembolus, and the condition is termed an **embolism**. Thus, a thrombus poses a localized risk of occlusion, but an embolus poses a systemic or organ-specific risk of infarction.

## 2. Pathophysiology and Formation

The mechanistic basis for thrombus formation is encapsulated by **Virchow's Triad**, a foundational principle in vascular pathology. This triad identifies three primary factors--endothelial injury, alterations in blood flow (stasis or turbulence), and **hypercoagulability**--which, either individually or in combination, predispose an individual to thrombosis. Endothelial injury, often caused by hypertension, toxins (like cigarette smoke), or inflammation, exposes the highly thrombogenic subendothelial matrix, particularly collagen, which triggers the immediate adherence and activation of platelets and the initiation of the coagulation cascade. This injury is typically the dominant factor in arterial thrombosis.

Alterations in normal, laminar blood flow play a critical role, especially in venous thrombosis. **Stasis**, or sluggish blood flow, prevents the rapid washout of activated clotting factors and the inflow of natural clotting inhibitors, allowing local factor concentrations to reach a critical threshold

necessary for fibrin polymerization. Stasis is common in situations such as prolonged bed rest, immobilization, or heart failure. Conversely, **turbulence**, often seen downstream from atherosclerotic plaques, contributes to thrombosis by causing endothelial damage and creating countercurrents or eddies that slow local flow, further promoting platelet aggregation and activation.

The third component, **hypercoagulability** (or thrombophilia), refers to any disorder of the blood that predisposes it to clot inappropriately. This can be inherited (e.g., Factor V Leiden mutation or deficiencies in natural anticoagulants like Protein C or S) or acquired (e.g., malignancy, oral contraceptive use, or conditions causing elevated fibrinogen levels). In hypercoagulable states, the balance between procoagulant and anticoagulant forces is shifted heavily toward clot formation, meaning a thrombus can form with minimal or no discernible endothelial injury or stasis, though these factors often synergize to produce severe clinical outcomes.

## 3. Classification and Types

Thrombi are typically classified based on their location, which dictates their underlying cause, structure, and clinical consequences. **Arterial thrombi** usually form in medium to large arteries, such as the coronary or cerebral arteries. They are almost universally initiated by the rupture of an atherosclerotic plaque, leading to rapid, high-shear flow platelet aggregation. Because of the high flow velocity in arteries, these clots are often rich in platelets and appear pale, leading to the designation of "white thrombi." They are the primary cause of acute arterial occlusion, resulting in conditions such as myocardial infarction (heart attack), ischemic stroke, and critical limb ischemia.

In contrast, **venous thrombi** primarily form in low-flow environments, most commonly in the deep veins of the legs (Deep Vein Thrombosis or **DVT**). Due to the predominant role of stasis and the slower coagulation process, venous thrombi contain a larger proportion of entrapped red blood cells and fibrin relative to platelets, giving them a dark red appearance, hence the term "red thrombi." The primary danger of venous thrombi is not local tissue ischemia, but rather their propensity to detach and travel to the lungs, causing a potentially fatal **pulmonary embolism** (PE).

Further sub-classification exists based on morphology and location. **Mural thrombi** are non-occlusive thrombi that adhere to the wall of a large vessel or heart chamber, often over an area of myocardial infarction or aneurysm; they are highly prone to embolism. **Occlusive thrombi** completely fill the lumen of the vessel, halting blood flow. Additionally, the microscopic appearance can reveal **Lines of Zahn**, which are characteristic laminations seen in thrombi formed in flowing blood, consisting of alternating layers of pale platelet/fibrin aggregates and darker layers of red blood cells. The presence of these lines distinguishes an ante-mortem (during life) thrombus from a post-mortem clot.

## 4. Clinical Significance and Impact

The morbidity and mortality associated with thrombi are immense, as thrombosis underlies the majority of acute cardiovascular diseases worldwide. The impact of a thrombus is two-fold: immediate local injury due to **ischemia**, and remote injury due to **embolism**. In the arterial system, thrombi are responsible for acute syndromes where oxygen supply is critically compromised. For example, a coronary thrombus leads to necrosis of the heart muscle (myocardial infarction), while a cerebral thrombus causes tissue death in the brain (ischemic stroke). The speed and extent of the thrombus formation determine the severity of the resulting infarction.

In the venous system, the localized impact of DVT includes pain, swelling, and potentially chronic venous insufficiency (post-thrombotic syndrome). However, the systemic significance is far greater. A DVT in the leg can travel through the major veins, pass through the right side of the heart, and become lodged in the pulmonary circulation, causing a **pulmonary thromboembolism (PE)**. PE is a leading cause of sudden, unexpected death, as it severely impedes gas exchange and places a massive strain on the right ventricle of the heart. Therefore, the management of venous thrombosis is often aggressively focused on preventing the embolic event.

## 5. Diagnosis and Management

The diagnosis of a thrombus relies on a combination of clinical risk assessment and diagnostic imaging. For DVT, initial assessment often involves clinical prediction rules (such as the Wells Score) combined with measurement of **D-dimer**, a fibrin degradation product that is elevated when a significant clot is present and being broken down. Definitive diagnosis usually requires imaging, such as Doppler ultrasound for venous thrombosis, or angiography (CT or conventional) for arterial blockages. Diagnosis of acute arterial thrombosis often requires immediate intervention based on clinical presentation due to the rapid onset of tissue necrosis.

The treatment for thrombosis is centered on three main strategies: immediate dissolution of the existing clot, prevention of further clot growth, and long-term prevention of recurrence. **Thrombolytic agents** (e.g., tissue plasminogen activator or tPA) are drugs that actively dissolve the fibrin network of the thrombus; these are used urgently in acute, severe cases like massive PE or certain strokes. **Anticoagulants** (such as heparin or warfarin/DOACs) do not dissolve the existing clot but inhibit the formation of new fibrin, preventing the thrombus from growing larger and reducing the risk of embolism.

In certain scenarios, mechanical intervention is necessary. **Thrombectomy** involves the surgical or catheter-based removal of the thrombus, commonly employed in acute arterial occlusions (e.g., stroke intervention) or when thrombolysis is contraindicated. For patients with recurrent DVT or those who cannot tolerate anticoagulation, **inferior vena cava (IVC) filters** may be placed temporarily to physically catch emboli originating from the lower extremities before they reach the

lungs, though the use of these filters is often debated due to potential complications.

## Further Reading

Thrombus (Wikipedia)
Virchow's Triad (Wikipedia)
National Heart, Lung, and Blood Institute (NHLBI) on Thrombosis