

# Venous Thrombosis and Pulmonary Embolism

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## ABSTRACT

Venous thrombosis and pulmonary embolism (PE) (collectively venous thromboembolism [VTE]) is an enormous health concern worldwide [1]. There are approximately 1,000,000 VTE cases per year in both the United States and European Union, with a yearly mortality rate of about 300,000 in each [2].

**Keywords:** Venous Thrombosis, Embolism, Pulmonary Embolism

## INTRODUCTION

### SYMPTOMS

DVT mainly affects the massive veins within the lower leg and thigh, nearly always on one side of the body at a time. The clot can block blood flow and cause:

- Leg pain or tenderness of the thigh or calf
- Leg swelling (edema)
- Skin that feels warm to the touch
- Reddish discoloration or red streaks

PE, or embolism, are often fatal and occurs when the DVT breaks free from a vein wall and blocks some or all of the blood supply to the lungs, causing:

- Unexplained shortness of breath
- Rapid breathing
- Chest pain anywhere under the skeletal structure (may be worse with deep breathing)
- Fast heart rate
- Light headedness or passing out.

### DIAGNOSIS

There are other conditions with signs and symptoms almost like those of DVT and PE. For example, muscle injury, cellulitis (a bacterial skin infection), and inflammation (swelling) of veins that are slightly below the skin can mimic the signs and symptoms of DVT. It is important to understand that attack and pneumonia

can have signs and symptoms almost like those of PE. Therefore, special tests which will search for clots within the veins or within the lungs (imaging tests) are needed to diagnose DVT or PE [3].

## TREATMENT

### Anticoagulants

Anticoagulants (commonly mentioned as “blood thinners”) are the medications most ordinarily used to treat DVT or PE. Although called blood thinners, these medications don't actually thin the blood. They reduce the ability of the blood to clot, preventing the clot from becoming larger while the body slowly reabsorbs it, and reducing the risk of further clots developing [4].

The most frequently used injectable anticoagulants are

- Unfractionated heparin (injected into a vein),
  - Low relative molecular mass heparin (LMWH) (injected under the skin), and
  - Fondaparinux (injected under the skin).
- Anticoagulants that are taken orally (swallowed) include
- Warfarin,
  - Dabigatran,
  - Rivaroxaban,
  - Apixaban, and
  - Edoxaban.

All of the anticoagulants can cause bleeding, so people taking them need to be monitored to stop unusual bleeding.

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## Thrombolytics

Thrombolytics (commonly referred to as “clot busters”) work by dissolving the clot. They have a higher risk of causing bleeding compared to the anticoagulants, so they are reserved for severe cases [5].

## RISKS

VTE risk increases with age, cancer diagnosis, pregnancy, oral contraceptive use, recovery from surgery, bed rest for medical illness (e.g., heart failure, COPD, neurologic disorders, etc.) [5]. and in individuals with hereditary thrombophilia stemming from common and uncommon gene mutations (e.g., Factor V Leiden and natural anticoagulant deficiencies, respectively) [1]. VTE is also a frequent and devastating complication in patients with other hematologic diseases such as sickle cell anaemia, thrombotic thrombocytopenia purpura, haemolytic uremic syndrome, and paroxysmal nocturnal hemoglobinuria and autoimmune diseases such as antiphospholipid syndrome [7].

Enhanced VTE risk assessment and the development of advanced pharmaceutical agents with better safety profiles and routes of administration has led to improved VTE diagnosis and prevention for many patients. As a result, VTE recurrence has been reduced by the maximum amount as 98 per cent. In addition the recent prophylactic regimens for total knee replacement have decreased the incidence of VTE down to one in every 25 operations [8].

Notwithstanding the progress in VTE diagnosis and prevention, effective treatment of this disease remains an on-going challenge. Anticoagulation has been the mainstay of VTE therapy since the 1930s when heparin was introduced. While newer pharmaceutical agents improve VTE outcomes, nearly all current therapies have the potential to cause bleeding. The development of therapeutics with novel mechanisms of action is needed to determine if thrombosis can be prevented and treated with agents that do not cause bleeding. To do so, the underlying pathophysiology of VTE needs to be better understood at genetic, protein, and cellular levels [9].

Improved understanding of the underlying pathophysiology would help advance assessment tools for predicting VTE risk within the unselected (i.e., low-risk) population and reduce the

incidence of primary VTE. Enhanced understanding would also improve the pharmaceutical armamentarium with the event of novel drugs that are effective for prophylaxis and treatment and should reduce or eliminate the danger of bleeding. Such drugs would likely have high impact for treating VTE in patients across a good spectrum of diseases. Understanding this balance may also provide critical insights into common arterial thrombotic disorders such as myocardial infarction and ischemic stroke, which remain significant public health concerns, and less common haemostatic disorders such as von Willebrand disease and haemophilia A, B and C [10].

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