massive PE
Management

Dr LUBNA HWEJEH

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اليوم العلمي لقسم الداخلية

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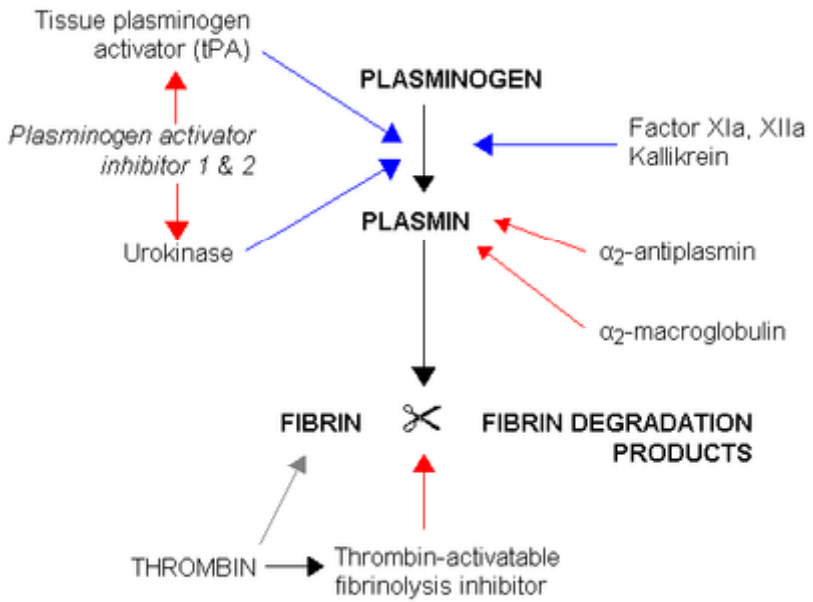
ALL DATA COLLECTED FROM

- ATS REVIEW
- UPTODATE

Thrombolytic agents

- activate **plasminogen** to form **plasmin**, resulting in the accelerated **lysis** of thrombi (fibrin clots).



Fibrinolysis

Plasmin is an important enzyme ,

- **present in blood** that
- degrades many blood **plasma proteins**, including **fibrin clots**.
- The degradation of fibrin is termed fibrinolysis.
- In humans, the plasmin **protein** is encoded by the PLG gene.

The **PLG gene** provides **instructions for making a protein called plasminogen**, which is **produced in the liver**.

Enzymes called plasminogen activators convert plasminogen into the protein plasmin, which **breaks down** another protein called **fibrin**. Oct 1, 2019

thrombolytic agents have been used in a variety of thrombotic disorders including

- **acute myocardial infarction**,
- acute pulmonary embolism (**PE**),
- and deep vein thrombosis (**DVT**).

Indication thrombolytic therapy

Potential indications for thrombolytic therapy in venous thromboembolism

Presence of hypotension related to PE*
Presence of severe hypoxemia
Substantial perfusion defect
Right ventricular dysfunction associated with PE
Extensive deep vein thrombosis

* This indication is widely accepted; the other potential indications require careful review of relative contraindications to thrombolytic therapy.

Contraindications to thrombolytic therapy

Contraindications to thrombolytic therapy

History of hemorrhagic stroke
Active intracranial neoplasm
Recent (<2 months) intracranial surgery or trauma
Active or recent internal bleeding in prior 6 months
Bleeding diathesis
Uncontrolled severe hypertension (systolic BP >200 mmHg or diastolic BP >110 mmHg)
Nonhemorrhagic stroke within prior 2 months
Surgery within the previous 10 days
Thrombocytopenia (<100,000 platelets per mm^3)

Table 10-16. Therapy for Massive VTE

Potential Indications for Therapy	Indications for Therapy	Relative Contraindications to Therapy	Absolute Contraindications to Therapy
Severe hypoxemia	Persistent hypotension	Severe uncontrolled HTN (BP > 180/110)	Previous hemorrhagic stroke at any time; other stroke or cerebrovascular events within 1 year
Large perfusion defect on V/Q scan	Shock	Hx of prior CVA/intracranial disease not covered in absolute contraindications	Known intracranial neoplasm
Extensive embolic burden on CT	Massive proximal LE thrombus associated with severe swelling or limb ischemia	Current use of anticoagulants; known bleeding diathesis	Active internal bleeding
RV dysfunction		Recent trauma (within 2–4 weeks); prolonged CPR (> 10 minutes) or major surgery within 3 weeks	Suspected aortic dissection
Free-floating RA or ventricular thrombus		Noncompressible vascular punctures	
PFO		Recent (2–4 weeks) internal bleeding	
CPR		Pregnancy, PUD	
		For streptokinase: prior exposure (especially within 5 days–2 years) or prior allergic reaction	

BP, blood pressure; CPR, cardiopulmonary resuscitation; CT, computed tomography; CVA, cerebrovascular accident; DVT, deep venous thrombosis; HTN, hypertension; Hx, history; LE, lower extremity; PFO, patent foramen ovale; PUD, peptic ulcer disease; RA, right atrial; RV, right ventricular; V/Q, ventilation-perfusion; VTE, venous thromboembolism.

The following are situations during which clinicians typically contemplate thrombolysis:

- Severe **hypoxemia**
- Large **perfusion defect** on V/Q scan
- **Extensive embolic burden** on computed tomography (CT)
- **Right ventricular dysfunction**
- **Free-floating right atrial** or ventricular thrombus
- Patent foramen ovale
- **Cardiopulmonary resuscitation**

- **The value of thrombolysis** to treat acute PE in the **other settings** is **uncertain** and must be determined on a case-by-case basis.

- Despite the **lack of evidence** that **systemic thrombolysis improves mortality**,
- most clinicians **accept hypotension due to an acute PE (massive PE) as an indication for thrombolysis**
- because **successful therapy can be lifesaving** .

Thrombolytic therapy carries a significant risk for massive bleeding.

Therefore, **its use must be weighed carefully against the risk of complications.**

For each patient, the

- **indications** and
- **potential benefits** must be carefully weighed against the
- **risk of adverse events**.
- and the **patient's values and preferences** should be considered.

The evidence indicates that **thrombolytic therapy leads to :**

- **early hemodynamic improvement,**
- **reduce the frequency of recurrent thromboembolism**.
- **Thrombolytic therapy has not been proven to improve mortality** or
- but at a cost of **increased major bleeding**.

Few trials have evaluated

- **the effects of thrombolytic therapy in hemodynamically unstable patients,**
- **those that did found a consistent trend toward improved mortality.**

A **meta-analysis** that included those trials did a subgroup analysis of

154 patients with **massive PE** and found that **systemic thrombolytic therapy**

- **decreased the composite endpoint of death**
- **and recurrent thromboembolism** (9.4 versus 19 % , odds ratio 0.45, 95% CI 0.22-0.92).

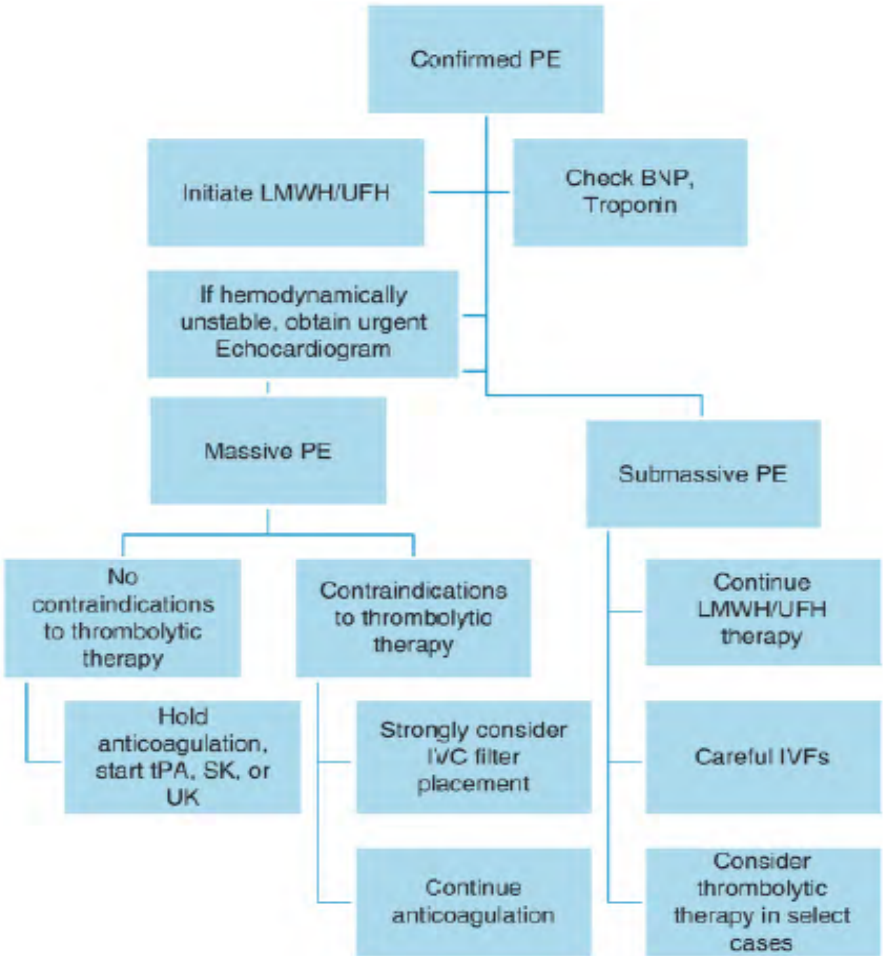


Figure 10-8. Management algorithm for massive PE.
BNP, brain natriuretic peptide; IVC, inferior vena cava; IVF, intravenous fluid; LMWH, low-molecular-weight heparin; PE, pulmonary embolism; SK, streptokinase; tPA, tissue plasminogen activator; UFH, unfractionated heparin; UK, urokinase.

Heparin therapy for venous thrombosis and pulmonary embolism.

Raskob GE[1], Carter CJ, Hull RD.

Author information

1 Department of Medicine, Chedoke-McMaster Hospital, Hamilton, Ontario, Canada.

Abstract

Intravenous heparin is the initial treatment of choice for most patients with acute pulmonary embolism or proximal deep vein thrombosis. The primary objective of initial heparin therapy in such patients is to prevent recurrent venous thromboembolism. The efficacy of intravenous heparin for this purpose has been established by randomized clinical trials in patients with pulmonary embolism, and more recently, in patients with proximal vein thrombosis. Heparin is given as an initial intravenous bolus of 5000 units, followed by a maintenance dose of 30,000-40,000 units per 24 h by continuous intravenous infusion. A recent randomized trial in patients with proximal vein thrombosis indicates that failure to achieve an adequate anticoagulant response (APTT greater than 1.5 times control) is associated with a high risk (25%) of recurrent venous thromboembolism. Intravenous heparin administered in doses that prolong the activated partial thromboplastin time (APTT) to 1.5 or more times the control value is highly effective, and associated with a low frequency (2%) of recurrent venous thromboembolism. Heparin is continued for 7-10 days, overlapped with warfarin sodium during the last 4-5 days. Multiple randomized clinical trials indicate that this approach is highly effective. An alternative approach is to commence heparin and oral anticoagulants together at the time of diagnosis, and to discontinue heparin on the fourth or fifth day. A recent randomized trial in patients with submassive venous thrombosis or pulmonary embolism suggests that 4-5 days of initial heparin therapy is effective and safe, but this approach must be evaluated by further randomized trials before it is recommended for patients with extensive proximal vein thrombosis.(ABSTRACT TRUNCATED AT 250 WORDS).

PMID: 3061531

[Indexed for MEDLINE]

Troponin

- Serum troponin I and troponin T are
- **elevated in 30 to 50 %** of patients who have a **moderate to large** PE

- Results are given in nanograms per milliliter (ng/mL). The **normal range** for **troponin** is between **0 and 0.4 ng/mL**.

Troponin, or the **troponin complex**, is a complex of three regulatory proteins (troponin C, troponin I, and troponin T) that is integral to muscle contraction[2] in skeletal muscle and cardiac muscle, but not smooth muscle.

Blood troponin levels may be

used as a diagnostic marker for stroke, although the **sensitivity of this assay is low.**

Assays of cardiac-specific troponins I and T are extensively used as diagnostic and prognostic indicators in the management of **myocardial infarction** and acute coronary syndrome.[4]

- The magnitude of the **elevation of BNP (**or its precursor, **N-terminal pro-brain natriuretic peptide NT-proBNP)**
- in patients with PE appears to **correlate with the risk of subsequent complications and prolonged hospitalization**.
- This suggests that **BNP and NT-proBNP may have a prognostic role in PE,**

Critical Care BMC

- **Elevated** brain natriuretic peptide (**BNP**) **levels can** help to identify patients with acute **pulmonary embolism** at **high risk of short-term death and** adverse outcome events.

- Although **elevated BNP levels** have a **high sensitivity** to detect patients at risk of death, the **specificity is low.**

- **levels** below **100 picograms per milliliter** (pg/ml) rule out CHF
- (the cutoff is 200 pg/ml for patients with kidney failure).
- Feb 1, 2007

A **normal level** of NT-proBNP,

- based on Cleveland Clinic's **Reference Range** is:
- **Less than 125 pg/mL** for patients aged 0-74 years.
- **Less than 450 pg/mL** for patients aged 75-99 years.

Brain natriuretic peptide (BNP) is a

- natriuretic hormone
- initially identified in the brain but
- released primarily from the heart,
- particularly the **ventricles**. Cleavage of the prohormone proBNP produces biologically active 32 amino acid BNP as well as biologically inert 76 amino acid N-terminal pro-BNP (NT-proBNP).Mar 2, 2017

Atrial natriuretic peptide (ANP) is a

- hormone that is released from myocardial cells in the atria and in some cases the ventricles in response to volume expansion and possibly increased wall stress [1]. ANP circulates primarily as a 28 amino acid polypeptide, consisting of amino acids 99-126 from the C-terminal end of its prohormone, pro-ANP.

- **The release of both ANP and BNP** is
- increased in heart failure (HF**), as ventricular cells are recruited to secrete both ANP and BNP in response to the high ventricular filling pressures** .
- The **plasma concentrations of both hormones are increased in patients with asymptomatic and symptomatic left ventricular dysfunction, permitting their use in diagnosis**

Brain natriuretic peptide

- **predicts right heart failure** in patients with **acute pulmonary embolism**.
- Plasma levels of brain natriuretic peptide (**BNP**) are increased in:
- patients with left **heart failure**.
- In patients with severe pulmonary embolism (PE),
- primary **right ventricular** (RV) dysfunction is frequent.

AMERICAN FAMILY PHYSICIAN

The Role of BNP Testing in Heart Failure

JENNY DOUST, B.M.B.S., FRACGP, *University of Queensland, Brisbane, Australia*
RICHARD LEHMAN, B.M.B.C.H., MRCAP, *Banbury, Oxfordshire, United Kingdom*
PAUL GLASZIOU, M.B.B.S., PH.D., FRACGP., *University of Oxford, Oxford, United Kingdom*

Am Fam Physician. 2006 Dec 1;74(11):1893-1900.

Brain natriuretic peptide (BNP) levels are simple and objective measures of cardiac function. These measurements can be used to diagnose heart failure, including diastolic dysfunction, and using them has been shown to save money in the emergency department setting. The high negative predictive value of BNP tests is particularly helpful for ruling out heart failure. Treatment with angiotensin-converting enzyme inhibitors, angiotensin-II receptor blockers, spironolactone, and diuretics reduces BNP levels, suggesting that BNP testing may have a role in monitoring patients with heart failure. However, patients with treated chronic stable heart failure may have levels in the normal range (i.e., BNP less than 100 pg per mL and N-terminal proBNP less than 125 pg per mL in patients younger than 75 years). Increases in BNP levels may be caused by intrinsic cardiac dysfunction or may be secondary to other causes such as pulmonary or renal diseases (e.g., chronic hypoxia). BNP tests are correlated with other measures of cardiac status such as New York Heart Association classification. BNP level is a strong predictor of risk of death and cardiovascular events in patients previously diagnosed with heart failure or cardiac dysfunction.

Critical Care BMC

Abstract

Go to:

Introduction

The relationship between brain natriuretic peptide (BNP) increase in acute pulmonary embolism (PE) and the increase in mortality and morbidity has frequently been suggested in small studies but its global prognostic performance remains largely undefined. We performed a systematic review and meta-analysis of data to examine the prognostic value of elevated BNP for short-term all-cause mortality and serious adverse events.

Table 10-15. Clinical Markers in PE

Test	Comments
BNP	6x increased mortality when BNP > 100, 16x increased mortality when NT-proBNP > 600
Troponin	Increased risk of short-term mortality (OR 5.24, 95% CI 3.28–8.38) or death due to PE (OR 9.44, 95% CI 4.14–21.49)
RV dysfunction	Incongruent findings on mortality effect in 2 meta-analyses
Concomitant DVT	Correlated with both increased all-cause mortality (adjusted HR 2.05, 95% CI 1.24–3.38) and increased PE-specific mortality (adjusted HR 4.25, 95% CI 1.61–-1.25)

BNP, brain natriuretic peptide; CI, confidence interval; DVT, deep venous thrombosis; HR, hazards ratio; NT, N-terminal; OR, odds ratio; PE, pulmonary embolism; RV, right ventricular.

Thrombolytic therapy guidelines

Guidelines for thrombolytic therapy in venous thromboembolism

Clear documentation of PE (or DVT)*
Potential contraindications carefully reviewed
Delivery by peripheral intravenous infusion
Initiate or continue other supportive therapy
Discontinue heparin during thrombolytic infusion

* High-probability ventilation-perfusion scan or positive pulmonary arteriogram.

Bleeding

Systemic thrombolytic therapy increases the risk of major bleeding.

occurs most commonly **at sites of invasive procedures** such as

- pulmonary **arteriography** or
- **arterial puncture**.

In a **meta-analysis** of 11 studies (**847 patients**)

- that compared **systemic thrombolytic therapy to anticoagulation alone in acute PE**,
- **thrombolytic therapy increased major bleeding**

(**9.0 versus 5.7 %** , relative risk 1.63, 95% CI 1.00-2.68).

There was a large difference in the

- **frequency of major bleeding** among patients defined as having a **high risk** **for bleeding versus those defined as having a low risk** for bleeding (6.2 versus 0.1%).

- Few **studies** have sought to identify **risk factors** for **bleeding** during **thrombolytic therapy.**

Known risk factors for major bleeding following thrombolytic therapy for acute pulmonary embolism.

Demographic characteristics

Advanced age (>75 years)

Female gender

African American race

Low body weight (risk inversely related with each 10 kg below 100 kg)

Medical history

Acute myocardial infarction

Hypertension (poorly controlled at baseline)

Aortic dissection

Acute pancreatitis

Dementia

Cardiopulmonary resuscitation exceeding 10 minutes

Surgical history

Major surgery within prior 3 weeks

Bleeding history

Stool occult positive

Stool occult positive

Internal bleeding in previous 4 weeks

Gastrointestinal bleeding in prior 3 months

Laboratory

Elevated bilirubin (> 3 mg/dl)

Coagulopathy (defined as INR >1.7)

Invasive device

Presence of intra-aortic balloon pump

Femoral venous access

Independent predictors of major hemorrhage

- **administration of catecholamines for systemic arterial hypotension** (odds ratio 115, 95% CI 9.4-1411),
- **malignancy** (odds ratio 16, 95% CI 3.2-80),
- **diabetes mellitus** (odds ratio 9.6, 95% CI 1.7-54), and
- an elevated international normalized ratio **(INR)** (odds ratio 6, 95% CI 1.5-22).

- patients with **significant or refractory bleeding are typically transfused**
- ten units of **cryoprecipitate** and
- two units of **fresh frozen plasma**, then reassessed.

Cryoprecipitate (cryo) contains

- a concentrated subset **of FFP** components including **fibrinogen**, **factor VIII** coagulant, **vonWillebrand factor**, and **factor XIII.**

- protamine sulfate should be considered to **reverse the effect of any heparin** that may remain in the patient's plasma.

In a **retrospective analysis** of 104 patients with acute PE who received alteplase,

20 patients (**19%**) had **major bleeding**.

The principal **site of bleeding was**

- **unknown in nine patients** (**45%**),
- **gastrointestinal** in six patients (**30%**),
- **retroperitoneal** in three patients (**15%**),
- **intracranial** in one patient (**5%**), and
- **splenic** in one patient (**5%**).

- **Unnecessary invasive procedures**

  (particularly **arterial punctures**) should be **minimized** while thrombolytic therapy is being considered.

- **Bleeding from vascular puncture sites should be controlled with manual compression followed by a pressure dressing.**

Administration

- Once the decision to administer thrombolytic therapy has been made**, the thrombolytic agent should be administered via a peripheral intravenous catheter.**

- **Anticoagulant therapy** is generally discontinued during the thrombolytic infusion.

- Common thrombolytic regimens include:
- **tPA – Administer 100 mg intravenously over two hours.**
- **Streptokinase – Administer 250,000 units intravenously over the initial 30 minutes, then 100,000 units/hour for 24 hours**.

What is the FDA recommended regimen of streptokinase for thrombolytic therapy of pulmonary embolism (PE)?

Updated: Dec 31, 2017 | Author: Wanda L Rivera-Bou, MD, FAAEM, FACEP; Chief Editor: Erik D Schraga, MD more...

References

Share Feedback

Answer

The FDA-approved streptokinase regimen for PE consists of 250,000 U as a loading dose over 30 minutes, followed by 100,000 U/hr over 12-24 hours.

- **Monitor closely** for hypotension, anaphylaxis, asthma, and allergic reactions.
- **Mild adverse reactions** may respond favorably to a **decreased infusion rate.**

- **Urokinase – Administer 4400 units/kg intravenously over the initial 10 minutes, then 4400 units/kg per hour for 12 hours.**

- Numerous **thrombolytic** agents and regimens have been directly **compared** in randomized trials, but **superiority of any thrombolytic agent or regimen over another has not been established.**

- **Studied regimens** include
- **tPA** administration over **15 minutes** or two hours,
- **urokinase** administration **over two hours** or 24 hours, and
- **streptokinase** administration **over two hours**, 12 hours, or 24 hours

The evidence suggests that

- shorter infusions ≤2 hours **achieve more rapid clot lysis** and are
- associated with **lower rates of bleeding** than longer infusions ≥12 hours.

- **An activated partial thromboplastin time (aPTT) should be measured when infusion of the thrombolytic therapy is complete.**
- **Heparin** should be resumed without a loading dose when the aPTT is **less than** twice its upper limit of normal(80 sec).
- (A typical **aPTT value** is 30 to 40 seconds).

Partial Thromboplastin Time, Activated

Updated: Jul 30, 2019 | Author: Muhammad Bader Hammami, MD; Chief Editor: Eric B Staros, MD more...

Reference Range

Partial thromboplastin time (PTT) and activated partial thromboplastin time (aPTT) are used to test for the same functions; however, in aPTT, an activator is added that speeds up the clotting time and results in a narrower reference range. The aPTT is considered a more sensitive version of the PTT and is used to monitor the patient's response to heparin therapy.

The reference range of the aPTT is 30-40 seconds.[1, 2, 3]

The reference range of the PTT is 60-70 seconds.

- If the **aPTT** exceeds this value, the test should be repeated **every four hours** until it is less than twice its upper limit of normal,
- **at which time heparin should be resumed**.
- **Coagulation assays** are unnecessary during infusion of the thrombolytic agent since thrombolytic agents are administered as fixed doses.

Medscape

Partial Thromboplastin Time, Activated

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The reference range of the aPTT is 30-40 seconds.[1, 2, 3]

The reference range of the PTT is 60-70 seconds.

In patients receiving anticoagulant therapy, the reference range is 1.5-2.5 times the control value in seconds.

Critical values that should prompt a clinical alert are as follows:

- aPTT: More than 70 seconds (signifies spontaneous bleeding)
- PTT: More than 100 seconds (signifies spontaneous bleeding)

The one **exception** is that the

- thrombin time may be measured **four hours after the initiation of SK**, since the presence of **antibodies** to this drug may impair its therapeutic effect.

Prolonged APTT may indicate:

- **use of heparin** (or contamination of the sample)
- **antiphospholipid antibody** (especially lupus anticoagulant, which paradoxically increases propensity to thrombosis)
- coagulation factor deficiency ,**hemophilia**
- **sepsis** : coagulation factor consumption
- presence of antibodies against coagulation factors (factor inhibitors)

adverse effects
that are specific for certain thrombolytic agents.

As an example, **streptokinase is associated with allergic reactions and hypotension:**

- **Streptokinase (SK)** is antigenic and can cause **immunologic sensitization** and **allergic reactions,** particularly with repeat administration.

- **Major reactions are rare, with anaphylaxis occurring in < than 0.5 % of patients.**

- **However, less severe symptoms such as shivering, pyrexia, or rash, may occur in up to 10 % of patients.**

- The efficacy of SK is not reduced by an allergic reaction; however,

- **anti-SK antibodies remain elevated for up to 7.5 years after treatment**,
- suggesting that a **suboptimal response and/or an allergic reaction may occur even if SK is readministered many years later.**

- **Hypotension** may occur during **streptokinase** infusion (particularly if the infusion rate is increased above 500 units/kg per min).

The decreased blood pressure usually responds to

- cessation or slowing of the infusion,
- intravenous fluids, or
- vasopressors

Bolus infusion

- Bolus infusion of thrombolytics **appear to be effective without excess bleeding complications.**
- However, it has **not** been directly compared to a two hour infusion of tPA.

- **Trials** comparing the regimens are **necessary before routine bolus infusion** replaces the more conventional two hour regimen.
- **An exception is that bolus infusion of thrombolytic therapy is indicated for patients with imminent or actual cardiac arrest** .

Catheter-directed

- Thrombolytic agents have been **infused directly into the pulmonary artery via a pulmonary arterial catheter** .
- **There is no evidence that intrapulmonary arterial infusion confers greater benefit than peripheral venous infusion**,
- **but it is associated with bleeding at the catheter insertion site.**

When there are contraindications to thrombolysis

(or thrombolysis fails to induce clinical improvement),

- catheter or **surgical embolectomy** may be warranted if the necessary resources and expertise are available.
- The **decision** of whether to pursue one of these approaches should be based on **local expertise.**

Table 10-13. Therapies for VTE

Treatment	Mechanism	Monitoring	Reversal Agent	Comments
Heparin	Binds AT III and factor Xa	PTT levels	Protamine	Can cause Type II HIT when Ab to PF4 form; treat with argatroban.
Enoxaparin	Binds factor Xa >>> AT III	Factor Xa levels	Protamine	Recommended agent in malignancy and pregnancy; monitor levels. Do not use if CrCl < 30 or weight > 150 kg.
Warfarin	Blocks vitamin K epoxide: Slow decrease in factors II, VII, IX, X, and thrombin	INR	Vitamin K, FFP, prothrombin complex	Category X; OK during lactation; multiple drug interactions.
Argatroban	Direct thrombin inhibitor	PTT	None	Primarily used for HIT.
Dabigatran	Oral factor IIa inhibitor	None	None	FDA approved, noninferior to warfarin; lower overall bleeding but increased major bleeding.
Rivaroxaban	Oral factor Xa inhibitor	None	None	FDA approved; equivalent bleeding risk; long-term studies forthcoming.
Thrombolytic therapy: streptokinase, urokinase, tPA	Increased plasminogen increases plasmin, which breaks down fibrin.	None	None	None proven superior; tPA preferred given shortest infusion time. No mortality benefit. Indicated for massive PE.
IVC filter	Placed when a contraindication exists to anticoagulation or if poor cardiopulmonary reserve after PE.		Retrievable or permanent	No short-term or long-term demonstrable mortality benefit. Increased risk of DVT.
Compression stockings	Reduces symptoms of post-thrombotic syndrome.			Pharmacologic therapy for post-thrombotic syndrome not recommended (rutosides, hidrosmin).

Ab, antibodies; AT, antithrombin; CrCl, creatinine clearance; DVT, deep venous thrombosis; FFP, fresh frozen plasma; HIT, heparin-induced thrombocytopenia; IVC, inferior vena cava; PE, pulmonary embolism; PF, platelet factor; PTT, partial thromboplastin time; tPA, tissue plasminogen activator; VTE, venous thromboembolism.

Key Fact

Protamine should be given only when resuscitation techniques and treatment of anaphylactic and anaphylactoid shock are readily available. Reduce the rate of administration in cases of bradycardia, dyspnea, and hypotension. Administration that is too rapid may cause severe hypotension.

Length of Treatment

- The length of optimal therapy for PE has been studied extensively.
- **The ACCP Antithrombotic Guidelines** published in 2012 discuss this issue and are

Table 10-17. Length of Treatment for VTE

Event	Duration of Therapy	Comments
First VTE	Provoked: 3 months Unprovoked: at least 3 months	Consider risk/benefit for extended therapy LMWH indicated over VKA if patient has active malignancy Consider D-dimer testing within 4 weeks of discontinuation of therapy
Recurrent VTE	Low bleeding risk: extended (> 1 year) High bleeding risk: 3 months	Consider IVC filter placement if recurrent despite anticoagulation

IVC, inferior vena cava; LMWH, low-molecular-weight heparin; VKA, vitamin K antagonists; VTE, venous thromboembolism.

- a **provoked DVT** is one that is usually caused by a known event (surgery, hospital admission).

Use of Inferior Vena Cava Filters

Inferior vena cava (IVC) filters should be considered in patients with

- **acute PE and absolute contraindications to anticoagulation,** as well as
- in patients with **recurrent PE despite therapeutic anticoagulation**.


THANK
YOU