Case Presentation

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- Chest xray
- CT head
- CTA chest
- ICU : IV heparin , PTT 60-80, Warfarin
- HOD#2 : Transferred to regular floor, weaned off O₂
- Venous Dupplex neg
- HOD#5 : D/C home on warfarin.
Management and prevention of Pulmonary Embolism

Georges E Al-Khoury, MD
Pulmonary Embolism

- The diagnosis and treatment of pulmonary embolism demand an interdisciplinary approach, combining medical, surgical, and radiologic specialties.
- Despite substantial advances, mortality and recurrence rates remain high.
Epidemiology and Pathophysiology

- PE ranges from incidental, clinically unimportant thromboembolism to massive embolism with sudden death.
Epidemiology and Pathophysiology

- Thrombi form in the deep veins of the legs, pelvis, or arms, may dislodge and embolize to the pulmonary arteries with potentially serious consequences.
Pathophysiology

- ↑ PVR : (PA obstruction , Platelets → serotonin ).
- Alveolar hyperventilation : (↑ Alveolar dead space ↓ V/Q )
- ↓ Pulmonary compliance: (Reflex bronchoconstriction , lung edema ).
- Dilatation, dysfunction, and ischemia of the RV: (↑ RV afterload ).
Pulmonary embolism and deep venous thrombosis should be considered part of the same pathological process.

- 40% of pts who had DVT w/o symptoms of PE had evidence of PE on lung scanning.

- 29% of pts w PE had abnormalities on ultrasonographic studies of leg veins.

(Moser, Frequent asymptomatic pulmonary embolism in patients with deep venous thrombosis. JAMA 1994)

(Turkstra FDiagnostic utility of ultrasonography of leg veins in patients suspected of having pulmonary embolism. Ann Intern Med 1997)
Incidence, Mortality, and Recurrence of VTE

- More than 250,000 pts are hospitalized annually in the US.
- More common in men.
- For each 10-year increase in age, the incidence x2.
Incidence, Mortality, and Recurrence

- The three-month mortality rate (10% - 17.5%).
- Overall, men had higher fatality rates than women (13.7% vs. 12.8%).
- Blacks had higher fatality rates than whites (16.1% vs 12.9%).
Right Ventricular Function

- The mortality rate at one year was three times higher in pts with RV dysfunction than in those with nl RV function.

(Ribeiro A, Echocardiography Doppler in pulmonary embolism: right ventricular dysfunction as a predictor of mortality rate. Am Heart J 1997)
Rudolf Virchow (1821-1902)

- Professor of Pathology at Wurzburg and then Berlin
- Rudolf Virchow, the brilliant 19th century pathologist, was the first to recognize that blood clots in the pulmonary artery originate as venous thrombi.
- He stated: "The detachment of larger or smaller fragments from the end of the softening thrombus which are carried along by the current of blood and driven into remote vessels. This gives rise to the very frequent process on which I have bestowed the name of Embolia."
Virchow’s Triad

- Hypercoagulability
- Stasis
- Injury to the vessel wall
## Risk Factors for VTE

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>Hypercoagulability</th>
<th>Stasis</th>
<th>Trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous VTE</td>
<td>√</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Major surgery</td>
<td></td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Cancer</td>
<td>√</td>
<td>√</td>
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<tr>
<td>Obesity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trauma</td>
<td></td>
<td></td>
<td>√</td>
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<tr>
<td>Fracture (hip or leg)</td>
<td></td>
<td>√</td>
<td>√</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>√</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td></td>
<td></td>
<td>√</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroke</td>
<td></td>
<td></td>
<td>√</td>
</tr>
<tr>
<td>Estrogen therapy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prolonged immobilization</td>
<td></td>
<td></td>
<td>√</td>
</tr>
<tr>
<td>Burns</td>
<td></td>
<td></td>
<td>√</td>
</tr>
</tbody>
</table>
Risk Factors/ Surgery

- Surgery predisposes pts to PE, even as late as one month postoperatively.
- 25% of the cases of PE occurred between the 15th and 30th postoperative day and 15% were detected more than 30 days postoperatively.


- In a Swiss study, PE after discharge occurred a median of 18 days postoperatively and led to an overall increase of 30% in the rate of postoperative PE.

Risk Factors/ Surgery

- Twentyfold increase in the odds of being diagnosed with VTE.
- PE may account for 10% of all postoperative deaths following total hip arthroplasty
# Type of surgery as a risk factor for DVT

<table>
<thead>
<tr>
<th>Type of surgery</th>
<th>Incidence of DVT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orthopedic surgery</td>
<td>50%–60%</td>
</tr>
<tr>
<td>Oncologic surgery</td>
<td>29%</td>
</tr>
<tr>
<td>General surgery</td>
<td>25%</td>
</tr>
<tr>
<td>Neurosurgery</td>
<td>22%</td>
</tr>
<tr>
<td>Gynecologic surgery</td>
<td>16%</td>
</tr>
<tr>
<td>Urologic surgery</td>
<td>5%</td>
</tr>
</tbody>
</table>
Risk Factors / Genetic

- Genetic predisposition appears to explain only about one fifth of cases of PE.
Risk Factors/ Pregnancy

- From 1979 to 1986, 2726 pregnancy-associated deaths were reported in the United States.
- For women whose pregnancies resulted in a live birth, thrombotic pulmonary embolism was the leading cause of death.
- The risk of PE among users of oral contraceptives is about three times the risk among nonusers.
- Hormone-replacement therapy doubles the risk of VTE.
- (the risk is higher near the start of therapy than after long-term use.

(Vandenbroucke JP. Risk of venous thrombosis with hormone-replacement therapy. Lancet 1996;
Risk Factors/ Cancer

- Neoplastic cells can generate thrombin or synthesize various procoagulants.
- Occasionally, previously unsuspected cancer is identified in patients with newly diagnosed venous thrombosis.
- Presence of a malignancy is a potent risk factor that increases the risk of postoperative symptomatic VTE by at least twofold.
### Relative risk of DVT for the most common thrombophilic disorders

<table>
<thead>
<tr>
<th>Relative risk of DVT</th>
<th>Thrombophilic disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor V Leiden</td>
<td>X 5-80</td>
</tr>
<tr>
<td>Prothrombin 20210 mutation</td>
<td>X 3</td>
</tr>
<tr>
<td>Protein C and S deficiency</td>
<td>X 7</td>
</tr>
<tr>
<td>Antithrombin deficiency</td>
<td>X 5</td>
</tr>
<tr>
<td>Hyperhomocysteinemia</td>
<td>X 2-4</td>
</tr>
<tr>
<td>Antiphospholipid antibody syndrome</td>
<td>X 1-2</td>
</tr>
</tbody>
</table>
Thrombophilia/ Factor V Leiden

- Resistance to activated protein C inherited as an autosomal dominant trait.
- The point mutation (the substitution of adenine for guanine) in the gene coding for coagulation factor V that is responsible for activated protein C resistance.
- Glutamine replaces arginine at position 506, thereby making activated factor V more difficult for activated protein C to cleave and inactivate.
- Most common in Europe and least common in Africa and Southeast Asia.
- Factor V Leiden also appeared to increase the risk of recurrent PE after discontinuation of anticoagulation by a factor of 2 to 4.
Diagnosis

- The accurate detection of PE remains difficult, and the differential diagnosis is extensive.
- PE can accompany as well as mimic other cardiopulmonary illnesses.
- The optimal strategy is an integrated diagnostic approach that includes a methodical history taking and physical examination, supplemented by selective testing when appropriate.

<table>
<thead>
<tr>
<th>TABLE 1. DIFFERENTIAL DIAGNOSIS OF PULMONARY EMBOLISM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumonia or bronchitis</td>
</tr>
<tr>
<td>Asthma</td>
</tr>
<tr>
<td>Exacerbation of chronic obstructive pulmonary disease</td>
</tr>
<tr>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>Pulmonary edema</td>
</tr>
<tr>
<td>Anxiety</td>
</tr>
<tr>
<td>Dissection of the aorta</td>
</tr>
<tr>
<td>Pericardial tamponade</td>
</tr>
<tr>
<td>Lung cancer</td>
</tr>
<tr>
<td>Primary pulmonary hypertension</td>
</tr>
<tr>
<td>Rib fracture</td>
</tr>
<tr>
<td>Pneumothorax</td>
</tr>
<tr>
<td>Costochondritis</td>
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<tr>
<td>Musculoskeletal pain</td>
</tr>
</tbody>
</table>
Diagnosis

- Dyspnea is the most frequent symptom.
- Tachypnea is the most frequent sign.
- The presence of dyspnea, syncope, or cyanosis usually indicates a massive PE.
- Pleuritic pain, cough, or hemoptysis often suggests a small embolism near the pleura.
Diagnosis

- On Ph Ex, findings of RV dysfunction:
  - Bulging neck veins with v waves
  - Left parasternal lift
  - Accentuated pulmonic component of S2
  - Systolic murmur at the left lower sternal border that increases in intensity during inspiration.
Clinical Recognition of Pulmonary Embolism

- Signs and symptoms of PE are nonspecific
- The clinical recognition of PE is inaccurate.
- The majority of cases of PE detected postmortem were not diagnosed (or treated) prior to death.
- Sensitivity 25% - 45%


### Clinical Diagnosis of Pulmonary Embolism vs Angiographic Diagnosis, PIOPED.

<table>
<thead>
<tr>
<th>Clinical Probability of PE Prior to / Scan or Pulmonary Angiography</th>
<th>PE Documentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clini<strong>c</strong>al diagnosis</td>
<td>10%</td>
</tr>
<tr>
<td>Pulmonary angiography</td>
<td>68%</td>
</tr>
<tr>
<td>Clinical Probability of PE Prior to / Scan or Pulmonary Angiography</td>
<td>64%</td>
</tr>
<tr>
<td>Pulmonary angiography</td>
<td>30%</td>
</tr>
<tr>
<td>Clinical Probability of PE Prior to / Scan or Pulmonary Angiography</td>
<td>26%</td>
</tr>
<tr>
<td>Pulmonary angiography</td>
<td>9%</td>
</tr>
</tbody>
</table>
The most frequent electrocardiographic abnormality is T-wave inversion in the anterior leads, especially leads V1 to V4.

Inferoposterior ischemia due to compression of the right coronary artery by the right ventricle as a result of pressure overload.

New-onset right-bundle branch block or atrial fibrillation is uncommon.

The most specific ECG abnormality S1Q3T3 pattern, was reported to be due to acute cor pulmonale secondary to acute massive pulmonary embolism.
Diagnosis/ Chest film

- Focal oligemia (Westermark's sign)
- Peripheral wedge-shaped density above the diaphragm (Hampton's hump)
- Enlarged right descending pulmonary artery (Palla's sign)
IMAGING OF PULMONARY THROMBOEMBOLISM
/ Chest radiography

- Insensitive and nonspecific
- Even pts with life-threatening PE may have a nl chest radiograph.
- True positive rate 33%.
IMAGING OF PULMONARY THROMBOEMBOLISM
/ Chest radiography

- Dilatation of the pulmonary artery proximal to the site at which the embolus has lodged
- Loss of local lung volume caused by pulmonary embolism is shown by areas of linear atelectasis, or by the position of fissures and/or elevation of the hemidiaphragm
- Pleural effusions occur in approximately half of all cases of acute PE
- **Pulmonary infarction** may result in a large bloody pleural effusion that clears slowly.
Dilatation of the pulmonary artery proximal to the site at which the embolus has lodged.
Typically, an established pulmonary infarct is a shallow hump-shaped lesion (alliteratively referred to as a Hampton’s hump) with its base applied to a pleural surface.
Aseptic cavitation of pulmonary infarcts is rare (and data from various studies suggest that it develops in very few (less than 5%) infarcts.
Normal ABG values do not rule out the diagnosis of PE and cannot accurately discriminate between pts who require further investigation.

Hypoxemia or Hypocapnia may increase the physician's level of diagnostic suspicion, but these findings are not specific for PE.

(Stein PD, Arterial blood gas analysis in the assessment of suspected acute pulmonary embolism. Chest 1996)
Diagnosis of DVT / Venography

- Venography became the "gold standard" for the diagnosis of DVT just as selective pulmonary angiography became the "gold standard" for the diagnosis of pulmonary embolism.

- DVT can be recognized by the presence of constant filling defects, abrupt cutoffs, nonfilling of the entire deep system, or portions thereof, and/or demonstration of collateral flow.
Diagnosis of DVT / compression ultrasonography

- Accurate method to detect proximal DVT.
- Sensitivity 95.2%
- Specificity 98.6%.
- Compression ultrasonography is less sensitive in detecting DVT below the knee
- In pts with silent DVT: sensitivity 62%
The high sensitivity is not matched by a high specificity for the diagnosis of PE.

A normal ventilation–perfusion scan excludes PE with a probability approaching 95%.

In the multicentre PIOPED:
- Intermediate-probability scans: 33% had PE
- High-probability scan: 85% had PE
A normal finding is capable of excluding the diagnosis of embolism.
High-probability radionuclide scan for pulmonary embolism.

The cardinal sign of pulmonary embolism is an underperfused part of the lung on perfusion scanning (typically, the defect is segmental) while the ventilation scan remains normal—the so-called ‘mismatched perfusion defect’.
Diagnosis/ CTA

- This approach is best suited for identifying PE in the proximal pulmonary vascular tree.
- If the CT findings are normal in the presence of a high index of clinical suspicion, contrast pulmonary angiography that focuses on the distal pulmonary vasculature should be performed.
The range of nondiagnostic spiral CTs for PE is low (2% - 9%) is similar to that of pulmonary arteriography which has a nondiagnostic rate of approximately 3%.
Thromboembolic filling defects in pulmonary arteries of both lower lobes in a patient with renal cell carcinoma. Note right rib metastasis.
Chest computed tomographic scan with contrast, demonstrating extensive embolization involving the right main, upper lobe, and lower lobe pulmonary arteries.
Pulmonary infarction.
(A) Typical CT appearance of a pulmonary infarct: a pleurally-based truncated cone containing an air bronchogram.
(B) Ten weeks later the pulmonary infarct is clearing by retraction and resolution around its edges.
The multiplanar capability of MRI may clarify questionable signs of embolism.

Flow studies can estimate the haemodynamic effect of PE and coexisting pulmonary hypertension.

MRI offers the ability to identify the PE and its source, for example the pelvic veins or deep veins of the legs, in a single examination.
MRI demonstrates PE and DVT.
### Accuracy of Cross-Sectional Imaging for Detecting Pulmonary Embolism

<table>
<thead>
<tr>
<th>Technique</th>
<th>No. of Patients</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>PPV</th>
<th>NPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>CT</td>
<td>517</td>
<td>159/187</td>
<td>315/330</td>
<td>159/174</td>
<td>315/343</td>
</tr>
<tr>
<td></td>
<td></td>
<td>85%</td>
<td>95%</td>
<td>91%</td>
<td>92%</td>
</tr>
<tr>
<td>MR</td>
<td>147</td>
<td>64/72</td>
<td>68/75</td>
<td>63/71</td>
<td>68/76</td>
</tr>
<tr>
<td></td>
<td></td>
<td>89%</td>
<td>91%</td>
<td>89%</td>
<td>89%</td>
</tr>
</tbody>
</table>
Diagnosis/ Pulmonary Angiography

- Definitive method of diagnosing PE
- Relatively safe (mortality <1%)
- Too expensive to be considered a cost-effective first-line investigation in all cases of suspected PE
- The two major angiographic signs of acute PE are:
  1. An intraluminal filling defect within an opacified pulmonary artery
  2. Complete occlusion of a pulmonary arterial branch.
IMAGING OF PULMONARY THROMBOEMBOLISM
/Pulmonary arteriography

- An intraluminal filling defect within an opacified pulmonary artery
Complete occlusion of a pulmonary arterial branch. The abrupt occlusion of an artery is a nonspecific sign which may be seen in a variety of conditions, including an organized previous embolus, in situ thrombosis, mediastinal fibrosis, occlusion from direct involvement by neoplasm, or inflammatory disease (such as granulomatous disease).
When consecutive patients with PE undergo echocardiography, about 40% have abnormalities of the RV.

Transthoracic echocardiography is particularly useful in critically ill patients suspected of having pulmonary emboli and can help identify right ventricular pressure overload as well as myocardial infarction, dissection of the aorta, or pericardial tamponade, which may mimic pulmonary embolism.

An echocardiogram showing RV hypokinesis combined with positive findings on ultrasonography of the legs is virtually pathognomonic of PE.
Diagnosis/ Echocardiography

- Diastolic and systolic bowing of the interventricular septum into the LV (RV volume and pressure overload)
- Impaired LV relaxation
- Dilated RV markedly hypokinetic, with little change from diastole to systole
The plasma d-dimer ELISA is highly sensitive but not specific for the diagnosis of PE, with a high negative predictive value.

A finding of > 500 ng/ml of d-dimer is present in > 90% of pts with PE.

Normal d-dimer level provides reassurance in more than 90% of cases that PE is not present.
Natural History of DVT

- 132 pts undergoing surgery without prophylaxis:
- DVT occurred in 40 pts (30%), began in the calf in the majority of pts
- Thrombi lysed spontaneously in 14 of the 40 pts
- In 17 pts thrombi remained in the calf
- In 9 pts, the thrombi extended into the popliteal or femoral veins
- PE occurred in 4 of these 9 pts.

(Kakkar, VV, Howe, CT, Flanc, C, et al Natural history of postoperative deep-vein thrombosis. Lancet 1969;6,230-232)
Natural History of DVT

- Without treatment, approximately 20 to 25% of calf vein thrombi extend into the popliteal and femoral veins, causing proximal DVT.
- Without treatment, approximately half of patients with proximal DVT develop PE.

Natural History of Pulmonary Embolism

Incidence of Pulmonary Embolism Per Year in the United States

- Total Incidence: 630,000
- Survival > 1 hour: 563,000 (89%)
  - Diagnosis not made: 400,000 (71%)
    - Survival: 280,000 (70%)
    - Death: 120,000 (30%)
  - Diagnosis made therapy instituted: 163,000 (29%)
    - Survival: 150,000 (92%)
    - Death: 13,000 (8%)
- Death within 1 hour: 67,000 (11%)

Progress in Cardiovascular Diseases, Vol. XVII, No. 4 (January/February), 1975
Natural History of Pulmonary Embolism

- The mortality rate of patients treated for pulmonary embolism has decreased from 8% to < 5%.
- The majority of deaths due to PE (ie, > 90%) occur in pts who are not treated because the diagnosis is not made.
- Less than 10% of all PE deaths occur in patients in whom treatment is initiated.
- Improved treatment will have a minimal impact on the number of deaths due to PE.
- Improved diagnosis, and most importantly, more effective prevention of DVT present the greatest opportunities to prevent fatal PE.
Prevention

- Prevention of pulmonary embolism is of paramount importance because the disorder is difficult to detect, and treatment of established pulmonary embolism is not universally successful.
Risk Factors/ Surgery

- The risk of symptomatic VTE is directly related to:
  (1) The type of surgery being performed
  (2) Presence of other risk factors for VTE
  (3) Duration and extent of postoperative immobilization
  (4) Use or nonuse of specific thromboprophylactic measures.
### Prophylaxis and treatment of deep vein thrombosis in general surgery/ Table 3. Thromboembolic risk stratification for surgery patients

<table>
<thead>
<tr>
<th>Risk Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low risk</td>
<td>Uncomplicated surgery in patients aged &lt;40 years with minimal immobility postoperatively and no risk factors</td>
</tr>
<tr>
<td>Moderate risk</td>
<td>Any surgery in patients aged 40–60 years, major surgery in patients &lt;40 years and no other risk factors, minor surgery in patients with 1 or more risk factors</td>
</tr>
<tr>
<td>High risk</td>
<td>Surgery in patients aged &gt;60 years, major surgery in patients aged 40–60 years with 1 or more risk factors</td>
</tr>
<tr>
<td>Very high risk</td>
<td>Major surgery in patients aged &gt;40 years with previous venous thromboembolism, cancer or known hypercoagulable state, major orthopedic surgery, elective neurosurgery, multiple trauma, or acute spinal cord injury</td>
</tr>
</tbody>
</table>
# Risk stratification for thromboembolism after surgery

<table>
<thead>
<tr>
<th>Level of risk</th>
<th>Incidence of proximal DVT%</th>
<th>Incidence of PE %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>0.4</td>
<td>&lt; 0.5</td>
</tr>
<tr>
<td>Moderate</td>
<td>2-4</td>
<td>1-2</td>
</tr>
<tr>
<td>High</td>
<td>4-8</td>
<td>2-4</td>
</tr>
<tr>
<td>Highest risk</td>
<td>10-20</td>
<td>4-10</td>
</tr>
</tbody>
</table>
Early ambulation should be a routine part of postoperative care for all patients, unless an absolute contraindication exits.

Early postoperative ambulation is acceptable as VTE prophylaxis for patients undergoing low-risk surgical procedures such as general, gynecologic, and urologic surgery.
DVT prophylaxis and anticoagulation in the surgical patient/ Nonpharmacologic prophylaxis/ Elastic stockings

- Improved venous flow and reduced vessel wall damage caused by the passive venous dilation that occurs during surgery.
- The relative risk reduction: 60%
- Improperly fitted stockings: ↑ venous pressure below the knees and ↑ risk of VTE.
- Should be applied preoperatively and continued throughout the hospital and rehabilitation period.
- Other modalities are more effective in high risk surgery.
- Recommended as an adjunct for all moderate or higher-risk pts.
DVT prophylaxis and anticoagulation in the surgical patient/Nonpharmacologic prophylaxis/Intermittent pneumatic compression devices

- The principal mechanism is likely a direct effect of pumping venous blood, thereby reducing stasis.
- It is also possible that there is promotion of clearance of prothrombotic clotting factors and an increase in local plasminogen activators leading to enhanced fibrinolysis.
- May not be effective in obese individuals, perhaps because of failure to transmit sufficient pressure to the deep veins.
- Excellent safety profile, no known complications.
- Only effective if used continuously while patients are nonambulatory.
- They may be used as the primary prophylaxis modality in many surgical settings.
- Presumed “additive” prophylactic effect when used in conjunction with pharmacologic methods.
DVT prophylaxis and anticoagulation in the surgical patient/ Nonpharmacologic prophylaxis/ Intermittent pneumatic compression devices

- Not recommended as the only thromboprophylactic modality:
  1. highest-risk general surgery pts
  2. high-risk urologic surgery pts
  3. orthopedic surgery pts undergoing hip or knee surgery.

- Method of choice when pts are at increased risk for bleeding with anticoagulants.

- Solo thromboprophylaxis in moderate to high-risk gynecologic surgery.

- No difference between IPC devices and LMWH in surgery for presumed gynecologic malignancy.
DVT prophylaxis and anticoagulation in the surgical patient/Nonpharmacologic prophylaxis/IVC filters

- The currently accepted indications for (IVC) filters include:
  1. An absolute contraindication to anticoagulation,
  2. Life-threatening hemorrhage on anticoagulation,
  3. Failure of adequate anticoagulation.

- ↓ the incidence of PE to 0.3–3.8% in pts with a contraindication to anti-coagulation.

- Prophylactic filter is not recommended simply because a pt is undergoing a procedure associated with a high incidence of VTE.
DVT prophylaxis and anticoagulation in the surgical patient/Nonpharmacologic prophylaxis/IVC filters

- The risks of IVC filter placement:
  1. Migration of the filter
  2. Recurrent DVT
  3. IVC thrombosis
  4. Postphlebitic syndrome
DVT prophylaxis and anticoagulation in the surgical patient/Pharmacologic prophylaxis/ *Low dose unfractionated heparin*

- Very effective, clearly reduces the incidence of fatal postoperative PE
- 68% to 76% risk reduction
- SQ 5000 (IU) Q12 hours or 5000 IU Q8 hours, the first dose given 2 h preop
- Equivalent efficacy when compared low-dose LMWH in general surgery pts with a moderate increase in the risk of bleeding (LDUH)
- Modestly higher incidence of bleeding compared with IPC devices.
- The risks of LDUH include: excess bleeding heparin-induced thrombocytopenia (HIT).
DVT prophylaxis and anticoagulation in the surgical patient/Pharmacologic prophylaxis/

Low dose unfractionated heparin

- One of the recommended medical prophylactic agents for most general surgical procedures, high-risk urologic or gynecologic surgery pts with or w/o nonpharmacologic methods.

- Not the agent of choice for very high-risk procedures (less effective compared with LMWH in very high-risk orthopedic or neurosurgical procedures).
DVT prophylaxis and anticoagulation in the surgical patient/ Pharmacologic prophylaxis/ Aspirin

- Not recommended as sole prophylaxis for any surgical procedure.
- May have a role for VTE prophylaxis among hip fracture pts, may be postdischarge prophylaxis if no other medical agent is used.
DVT prophylaxis and anticoagulation in the surgical patient/ Pharmacologic prophylaxis/ Warfarin

- Very high-risk pts with lower-extremity orthopedic and neurologic surgery.
- One of the principal prophylactic agents in hip fracture repair, total hip arthroplasty, and total knee arthroplasty.
- Low dose 10–14 days preop targeting an INR < 1.5, and then increasing the dose postop to a target INR of 2.5.
- The night before surgery or immediately after surgery (lower incidence of bleeding complication but a higher incidence of asymptomatic thrombosis, particularly in calf veins).
Pharmacologic prophylaxis/ Low molecular weight heparin

- LMWHs safe, and effective alternatives:
- Modestly lower incidence of bleeding compared with LDUH in general surgery
- The incidence of heparin-induced thrombocytopenia is 3.5% with UFH but only 0.6% with LMWH
- Less osteoporosis.
- Reduced number of daily administrations without the need for monitoring
- LMWH The agent of choice In trauma surgery, if the risk of bleeding is low.
- Enoxaparin 30 mg q 12 hr (60 mg/day)
Prophylaxis and treatment of deep vein thrombosis in general surgery

- The new anticoagulant molecules fondaparinux and ximelagatran seem to have similar efficacy as LMWH for the treatment of VTE, but their efficacy in prophylaxis is increased 2-fold when compared with LMWH.
- Inhibition of factor Xa mediated by antithrombin.
Timing of prophylaxis

- In most patients, it is appropriate to initiate VTE prophylaxis as soon as the risk of developing thrombosis begins.
- For trauma patients, this means as soon as they are hospitalized.
- For elective surgery patients, it is as soon as they are taken to the operating room.
- For recently immobilized patients, it may be prior to admission to the hospital.
- Stockings and IPC devices should be initiated preoperatively as soon as the risk of immobility increases, then continued during the procedure and throughout the hospital stay.
- The clinical practice in North America tends to be to dose LMWH postoperatively, whereas in European countries it is begun preoperatively.
Duration of prophylaxis

- Extended prophylaxis is important for patients undergoing lower-extremity orthopedic surgery, particularly total hip arthroplasty.
- Extended prophylaxis for 30–42 days was associated with a significantly lower incidence of symptomatic VTE (1.3%) than placebo (3.3%).
- LMWH (enoxaparin 40 mg or dalteparin 5000 IU) or warfarin (target INR = 2.5).
# Evidence-based use of antithrombotic prophylaxis in general surgery

<table>
<thead>
<tr>
<th>Risk Level</th>
<th>Anticoagulation Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low risk</td>
<td>Early mobilization</td>
</tr>
<tr>
<td></td>
<td>LDUH (5000 IU 12 hourly starting 2 hours before surgery) or</td>
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<tr>
<td></td>
<td>LMWH (&lt;3400 anti-Xa IU daily)</td>
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<tr>
<td></td>
<td>or</td>
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<tr>
<td>Moderate risk</td>
<td>ES (compression elastic stockings) or</td>
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<tr>
<td></td>
<td>IPC (intermittent pneumatic compression) or</td>
</tr>
<tr>
<td></td>
<td>LMWH (&gt;3400 anti-Xa IU daily) plus ES</td>
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<tr>
<td></td>
<td>or</td>
</tr>
<tr>
<td>High risk</td>
<td>LDUF (5000 IU eight hourly starting two hours before surgery) plus ES or</td>
</tr>
<tr>
<td></td>
<td>IPC if anticoagulation contraindicated</td>
</tr>
<tr>
<td></td>
<td>Perioperative warfarin (INR 2-3)</td>
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<tr>
<td></td>
<td>or</td>
</tr>
<tr>
<td>Very high Risk</td>
<td>LMWH (&gt;3400 anti-Xa IU daily) plus ES</td>
</tr>
</tbody>
</table>
Both UFH and LMWH can be used as effective prophylaxis against postoperative thromboembolic complications after general surgery.

The optimal prophylaxis in general surgery seems to be the combination of graded compression stockings and LMWH.
Heparin constitutes the cornerstone of management. It accelerates the action of antithrombin III, thereby preventing an additional thrombus from forming and permitting endogenous fibrinolysis to dissolve some of the clot.

A bolus of unfractionated heparin (usually 5000 to 10,000 U) followed by a continuous infusion (initiated at a dose of 18 U/kg/h but not exceeding 1600 U per hour)
Recently, inpatient administration of LMWH has been shown to be as safe and effective as unfractionated heparin to treat hemodynamically stable PE.
For patients with objectively confirmed nonmassive PE, short-term treatment with SC LMWH, or IV UFH

In patients with acute nonmassive PE, LMWH recommended over UFH.

Initiation of VKA together with LMWH or UFH on the first treatment day and discontinuation of heparin when the INR is stable and > 2.0.
For patients who are hemodynamically unstable
- Both streptokinase and urokinase have similar thrombolytic effects
- Unsettled issue is the use of thrombolytic agents in hemodynamically stable patients with echocardiographic evidence of RV dysfunction
- No difference was detected in clinically relevant outcomes such as the death rate or the resolution of symptoms between patients receiving thrombolytic therapy and those receiving anticoagulant therapy alone.
The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy: Evidence-Based Guidelines/ Guidelines/ 4.0 Initial Treatment of Acute PE / 4.3 Catheter extraction or fragmentation for the initial treatment of PE

- **Against** use of mechanical approaches for most pts with PE.

- Use of mechanical approaches in selected highly compromised pts who are unable to receive thrombolytic therapy or whose critical status does not allow sufficient time to infuse thrombolytic therapy.
Catheter extraction or fragmentation for the initial treatment of PE

- A cap device balloon-tipped steerable catheter permits suction extraction of PE under fluoroscopy. Venturi effect at the catheter tip using jets of high-speed saline solution, recoiled rotating pigtail.
- In a series of 26 patients undergoing catheter embolectomy, extraction was successful in 23 patients, with a mortality rate of 27%.
- A report of catheter embolectomy in 18 patients with a 28% mortality rate has also been published.
Catheter extraction or fragmentation for the initial treatment of PE

- The pigtail catheter shaft is rotated manually, and the embolus is fragmented by mechanical action of the recoiled pigtail. During rotation, the pigtail is slowly advanced and withdrawn over the stationary guide wire within the embolic occlusion.
Catheter extraction or fragmentation for the initial treatment of PE

- Prefragmentation complete occlusion of the left pulmonary artery.
- Pigtail rotation catheter in place, from left femoral approach
Catheter extraction or fragmentation for the initial treatment of PE

- After embolus fragmentation: partial recanalization and intraluminal fragments.
Catheter extraction or fragmentation for the initial treatment of PE

- Final control angiography 3 days later (central venous digital subtraction angiography) after additional thrombolysis with 70 mg of plasminogen activator.
Pulmonary embolectomy continues to be performed in emergency situations when more conservative measures have failed.

If it is attempted, the candidate should meet the following criteria:
(1) massive PE (angiographically documented if possible)
(2) hemodynamic instability (shock) despite heparin and resuscitative efforts;
(3) failure of thrombolytic therapy or a contraindication to its use.

Operative mortality in the era of immediately available cardiopulmonary bypass has ranged from 10 to 75% in uncontrolled retrospective case series.
Placement of an IVC filter:
(1) contraindication for anticoagulation
(2) complication of anticoagulantion
(3) recurrent thromboembolism despite adequate anticoagulation
Patients with acute PE require long-term anticoagulant treatment to prevent a high frequency (20 to 50%) of symptomatic extension of thrombosis and/or recurrent VTE.
Long-term treatment with a VKA for at least 3 months for patients with a first episode of PE secondary to a transient (reversible) risk factor.

Treatment with a VKA at least 6 to 12 months for patients with a first episode of idiopathic PE.