DVT PROPHYLAXIS AND THE TRANSITION HOME
No Disclosures
1) Understand the mechanism of delayed DVT
2) Describe the options for home prophylaxis
3) Discuss timing and management of outpatient treatment
Nearly all hospitalized patients have at least one risk factor for VTE
- 40% of pt’s will have more than one
- Surgical patients have a 10-40% risk of VTE, higher in some sub populations
  - Total Joints (40-60%)
  - Major Trauma (10-70%)
  - Cancer surgery (20-40%)
- VTE can lead to PE
  - #1 source of in-hospital mortality
UNDERSTANDING HYPERCOAGULABILITY

Virchow’s Triad

Flow

Blood

Vasculature
UNDERSTANDING HYPERCOAGULABILITY

- **Clotting Factors**
  - Intrinsic pathway (XII→XI→X)
  - Extrinsic pathway (TF&VII)
- **Platelets**
- **Leukocytes**
- **RBC’s**
- **[cells/platelets] and [factors], as well as their state of activation affects clotting system wide**
UNDERSTANDING HYPERCOAGULABILITY

Blood

Contact activation (intrinsic) pathway
Damaged surface

Tissue factor (extrinsic) pathway
Trauma

TFPI

Prothrombin (II)

Xa

Thrombin (IIa)

Fibrinogen (I)

Fibrin (Ia)

Cross-linked fibrin clot

VIIIa

VIIa

VII

Common pathway

Active Protein C

Protein S

Protein C + thrombomodulin

XIIa

XIa

IXa VIIIa

IX

X

XI

XII
Studied through deficiency models:

- Factor Deficiencies
  - Protein C, Protein S, Antithrombin
    - Risk can increase 10-fold during these deficiency states
  - Factor VIII or IX (Hemophilia A or B)

- [fibrin] affects the strength and distensibility of clots.
UNDERSTANDING HYPERCOAGULABILITY

- **Sheer Stress**
  - Radial forces between fluid layers

- **Sheer Rate**
  - Velocity difference between fluid layers
  - Arteries 500-1500/s, Veins 10-100/s

- **"Wall Sheer Stress"**
  - Tangential F of fluid on endothelium
  - Arteries adapt to stay @ 5-20 dynes/cm²
UNDERSTANDING HYPERCOAGULABILITY

DELIVERY
- Clotting factors
- Cells & Platelets
- Nutrients:
  - Oxygen
  - Glucose
  - Proteins

REMOVAL
- Clotting factors
- Activated cells & platelets
- Waste products
  - Chemokines
  - Fibrin monomers
  - CO2
  - Activated Thrombin (Xa)
UNDERSTANDING HYPERCOAGULABILITY

- Valves or Stenosis can create recirculation zones
  - Low sheer force
  - Decreased oxygen delivery
  - Promotes P-selectin expression and platelet aggregation or monocyte-platelet aggregates
  - These aggregates are activated and can promote thrombosis in areas of injury or recirculation zones
- Low sheer rates can change endothelial activity (↓KLF):
  - Decrease thrombomodulin
  - Increased VCAM-1
  - Increased TF
UNDERSTANDING HYPERCOAGULABILITY

- **Local injury**
  - Allows exposure of sub-endothelial tissue (TF)
  - Activation and recruitment of endothelium, platelets, and leukocytes
  - Creates an area of active coagulation
  - Ideally, coagulation should stop after the area of injury (Protein C & S, thrombomodulin)

- **Alterations to endothelial expression occur:**
  - Systemic inflammation
    - Infection – Bacterial lipopolysaccharide
    - Cytokines – IL-1, TNF-\(\alpha\)
  - Result in TF synthesis, decreased thrombomodulin
Stasis behind vein valve leaflets
- Decreased oxygen tension
- Activation/inflammation of the endothelium

Recruitment of monocytes and platelets
- increased [microparticles (MP)]
- Increased exposure of tissue factor (TF)
- Activation of Extrinsic Pathway

Activation of granulocytes & platelets
- Exposure of DNA, RNA, and inorganic phosphates
- Activation of factor XII $\rightarrow$ Intrinsic pathway

UNDERSTANDING HYPERCOAGULABILITY

FLOW

VASCUKATURE

BLOOD
SURGERY & TRAUMA = INFLAMMATION
Injury

Activation of the Coagulation Cascade

Expression of tissue factor

Disseminated intravascular coagulation

Insult

Activation of macrophages, neutrophils, dendritic cells, T cells

Release of reactive oxygen species by activated granulocytes

Endothelial damage

Microcirculatory dysfunction

Tissue Hypoxia

Tissue Damage

Organ dysfunction

Death

Susceptibility to Infection

Signal Amplification

Hyper-inflammation

TNFα
IL-1β
IL-6
IL-8
PAF
C5a
MIF
HMGB1

Anti-inflammatory cytokines, apoptosis, and anergy of immune cells

Cardiovascular dysfunction

Apoptosis

Figure 146-1 Critical Care
When severe Trauma combines with shock, there is a profound response from Protein C

- Coagulopathy of Trauma
- High transient levels of APC
- Early active bleeding in up to 1/3rd of trauma patients
- Depletion of protein C in this early cascade
- Long term deficiency of protein C during recovery
SURGERY & TRAUMA = INFLAMMATION

HOSPITAL STATISTICS 2011

Count of Total Trauma Patients
with
Count with DVT / PE Diagnosis & Count with VTE Prophylaxis Medication Administration

<table>
<thead>
<tr>
<th>Month</th>
<th>Total Count of Trauma Patients</th>
<th>Count of Trauma Patients with Diagnosis of DVT/PE</th>
<th>Count of Trauma Patients Receiving VTE Prophylaxis Medications</th>
</tr>
</thead>
<tbody>
<tr>
<td>12/2010</td>
<td>15</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>1/2011</td>
<td>52</td>
<td>23</td>
<td>14</td>
</tr>
<tr>
<td>2/2011</td>
<td>45</td>
<td>27</td>
<td>23</td>
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<tr>
<td>3/2011</td>
<td>48</td>
<td>20</td>
<td>27</td>
</tr>
<tr>
<td>4/2011</td>
<td>45</td>
<td>2</td>
<td>20</td>
</tr>
<tr>
<td>5/2011</td>
<td>67</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>6/2011</td>
<td>56</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>7/2011</td>
<td>63</td>
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<td>6</td>
</tr>
<tr>
<td>8/2011</td>
<td>94</td>
<td>18</td>
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</tr>
<tr>
<td>9/2011</td>
<td>69</td>
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<tr>
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<td>21</td>
</tr>
<tr>
<td>12/2011</td>
<td>45</td>
<td>3</td>
<td>19</td>
</tr>
</tbody>
</table>
HOSPITAL STATISTICS 2011

Rate of Occurrence of Diagnosis of DVT / PE
&
Rate of VTE Prophylaxis Medications Administered

% of Trauma Patients Receiving VTE
% of Trauma Patients with Diagnosis of DVT/PE

- % of Trauma Patients Receiving VTE Prophylaxis Medications
- % of Trauma Patients with Diagnosis of DVT/PE
Goal to decrease the rate of DVT/PE

Improve Compliance of DVT prophylaxis in All Trauma Patients

Establish a Hospital Pathway to improve DVT prophylaxis in other populations
- Orthopedics
- Gynecology and Colorectal Cancers
## Thrombosis Risk Factor Assessment

### Each Risk Factor Represents 1 Point
- Age 41-60 years
- Acute myocardial infarction
- Swollen legs (current)
- Congestive heart failure (< 1 month)
- Varicose veins
- Medical patient currently at bed rest
- Obesity (BMI > 25)
- History of inflammatory bowel disease
- Minor surgery planned
- History of prior major surgery (< 1 mo)
- Sepsis (< 1 month)
- Abnormal pulmonary function (COPD)
- Serious lung disease including pneumonia (< 1 month)
- Oral contraceptives or hormone replacement therapy
- Pregnancy or postpartum (< 1 month)
- History of unexplained stillborn infant, recurrent spontaneous abortion (≥ 3), premature birth with toxemia or growth-restricted infant
- Other risk factors

### Each Risk Factor Represents 2 Point
- Age 61-74 years
- Central venous access
- Arthroscopic surgery
- Major surgery (> 45 min)
- Malignancy (present or previous)
- Laparoscopic surgery (> 45 minutes)
- Patient confined to bed (> 72 hours)
- Immobilizing plaster cast (< 1 month)

**Subtotal:** (0-16)

### Each Risk Factor Represents 3 Point
- Age 75 years or older
- Family history of thrombosis*
- History of DVT/PE
- Positive Prothrombin 20210A
- Positive Factor V Leiden
- Positive Lupus anticoagulant
- Elevated serum homocysteine
- Heparin-induced thrombocytopenia (HT)
  (Do not use heparin or any low molecular weight heparin)
- Elevated anticardiolipin antibodies
- Other congenital or acquired thrombophilia

*most frequently missed risk factor

### Each Risk Factor Represents 5 Point
- Stroke (< 1 month)
- Multiple trauma (< 1 month)
- Elective major lower extremity arthroplasty
- Hip, pelvis or leg fracture (< 1 month)
- Acute spinal cord injury (paralysis) (<1 month)

**Subtotal:** (0-25)

---

### TOTAL RISK FACTOR SCORE: (0-88)

---

### FACTORS ASSOCIATED WITH INCREASED BLEEDING

Patient may not be a candidate for anticoagulant therapy & SCDs should be considered. Active Bleed, Ingestion of Oral Anticoagulants, Administration of glycoprotein IIb/IIIa inhibitors, History of heparin induced thrombocytopenia

### CLINICAL CONSIDERATIONS FOR THE USE OF SEQUENTIAL COMPRESSION DEVICES (SCD)

Patients with Severe Peripheral Arterial Disease, CHF, Acute Superficial DVT may not be a candidate for SCDs & alternative prophylactic measure should be considered.
<table>
<thead>
<tr>
<th>Total Risk Factor Score</th>
<th>Risk Level</th>
<th>Incidence of VTE</th>
<th>Prophylaxis Regimen</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-1</td>
<td>Low Risk</td>
<td>2%</td>
<td>☐ Early ambulation</td>
</tr>
<tr>
<td>2</td>
<td>Moderate Risk</td>
<td>10-20%</td>
<td>Choose the following medication <strong>OR</strong> compression devices:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>☐ Sequential Compression Device (SCD)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>☐ Heparin 5000 units SubQ BID</td>
</tr>
<tr>
<td>3 or more</td>
<td>High Risk</td>
<td>20-80%</td>
<td>☑ SCD’s PLUS</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Choose ONE of the following medications</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>☐ Enoxaparin (Lovenox) 40 mg SubQ BID for trauma patients</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>☐ Enoxaparin (Lovenox) 40 mg SubQ daily for general surgery patients</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>☐ Other:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><strong>If epidural present, substitute heparin 5000 units SubQ every 12 hours. Can begin</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><strong>enoxaparin 2 hours after epidural discontinued.</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(Pharmacy to adjust dosing of medications for renal insufficiency)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(Please refer to Dosing Guidelines on the back of this form)</td>
</tr>
</tbody>
</table>
**GOALS OF TREATMENT**

- **Acute Treatment**
  - Short term immobility
  - Major Surgery
  - Cancer

- **Long Term Tx**
  - Prolonged Immobility
  - Spinal Cord Injury
  - Known DVT

- **In Hospital LMWH**

- **DC home on 2 weeks of LMWH**
  - Goal is a total of 3-4 weeks of LMWH

- **Consider long term adjuncts**
  - Compression Stockings
  - Aspirin
  - Warfarin
CONCLUSIONS

- Trauma Patients are susceptible to altered coagulation
- Goal is to decrease DVT and PE by
  - Increasing mobility
  - Mechanical prophylaxis
  - Chemical prophylaxis in the hospital and after d/c
- Treatment can be ended
  - At one month
  - When patients are fully mobile
  - Once systemic inflammation has subsided
QUESTIONS?