Enhancing patient outcomes with sequential compression device therapy

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<table>
<thead>
<tr>
<th>Vessel wall damage</th>
<th>Hypercoagulability</th>
<th>Venous stasis</th>
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<tbody>
<tr>
<td>• Damage activates intrinsic pathway of clotting cascade.</td>
<td>• Blood coagulation becomes abnormal, with increased clotting factors and presence of tissue plasminogen activator, plasmin, and heparin.</td>
<td>• Blood pools in veins, allowing static blood to clot.</td>
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<td>• Platelet adhesion and aggregation occur.</td>
<td>• Fibrinolysis decreases, allowing continued clot formation.</td>
<td>• Clot may enlarge and enter venous system, then travel to lungs, resulting in PE.</td>
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<tr>
<td>• Clot forms at injury site as vascular system tries to repair injury.</td>
<td>• Coagulation modulators that halt clot formation (protein C, protein S, and antithrombin III) are absent.</td>
<td>• Venous hypertension causes one-way venous valves to fail.</td>
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Conditions that may cause endothelial damage include:
• venipuncture (as from venous-access catheters, I.V. therapy, trauma, or surgery)
• other invasive procedures
• orthopedic, abdominal, and thoracic surgery,

Conditions that may cause hypercoagulability include:
• burns
• surgery
• trauma
• infection or sepsis
• inflammatory disease
• chronic diseases
• recent myocardial infarction
• elevated lipid levels
• stroke
• atrial fibrillation
• cancer
• estrogen or hormone therapy
• certain medications
• pregnancy
• less than 1 month postpartum
• age older than 40.

Conditions that may cause venous pooling include:
• smoking
• heart failure
• polycythemia
• blood dyscrasias
• nephrotic syndrome
• limited mobility or bedbound status
• obesity
• pregnancy
• recent travel with prolonged sitting.