Surgical Bleeding and Blood Replacement

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Introduction

► Hemostasis defined by Virchow as the balance among blood flow, humoral factors, and cellular elements of the vascular system.

► Two coagulation pathways
  - Intrinsic
  - Extrinsic

► Platelets play a vital role early in hemostasis with the formation of the platelet plug

► Platelets release factors that promote hemostasis at the site of injury

► The intrinsic and extrinsic pathways lead to formation of Xa which starts the common pathway to coagulation
Introduction

- Negatively charged phospholipid phosphatidylserine is found on the inner leaflet of mammalian cells.
- Collagen or thrombin exposure changes the distribution of phospholipids to the external leaf.
- This provides a pro-coagulant surface for the various steps to take place.
- This also selects the activation to the site of injury.
FIGURE 30-2 Coagulation cascade. Protease network in coagulation, fibrinolysis, and kallikrein-kinin systems. HMWK, high-molecular-weight kininogen; AT-III, antithrombin III; TF, tissue factor; TFPI, tissue factor pathway inhibitor; HC-II, heparin cofactor II; FDPs, fibrinogen degradation products; PAI, plasminogen activator inhibitor; sc-uPA, single-chain urokinase plasminogen activator; APC, activated protein C; tPA, tissue plasminogen activator.
Intrinsic Pathway

- Factor XII becomes activated in the contact phase of coagulation
  - Combines with XI, prekallikrein, and high molecular weight kininogen
  - Come together on the highly negatively charged surfaces experimentally
- Factor XII is then activated by an unknown mechanism
- Factor XIIa converts prekallikrein to kallikrein
- Kallikrein converts factor XII to XIIa
- XIIa converts XI to XIA
- XIa converts IX to IXA
Intrinsic Pathway

► IXa with its cofactor VIII plus calcium and phospholipid membranes form the “tenase” complex
  ▪ This complex converts X -> Xa
  ▪ Xa activates the common pathway

► This complex is enhanced by two mechanisms
  ▪ The phospholipid membrane allows the enzymes to become more easily saturated
  ▪ Helps localize coagulation response to where it’s needed
Extrinsic Pathway

- Circulating factor VII encounters tissue factor and activates

- Tissue factor
  - Transmembrane glycoprotein normally expressed by fibroblast like cells that surround the blood vessel
  - Endothelium shields circulating blood from exposure to tissue factor
  - Activated monocytes, atherosclerotic plaques, and activated endothelial cells express tissue factor

- Factor VII
  - Weak procoagulant
Extrinsic Pathway

► Factor VII
  - 10,000,000 fold increase in activity when bound to tissue factor
  - How VII activated unknown (activation by Xa)
  - Both VII and VIIa bind to tissue factor
  - VIIa activates Xa

► IX activated by VII showing a cross activation of the two pathways

► Activation of X by the IXa/VIII complex is 50 times greater than the activation by VII/TF
Common Pathway

► Factor Xa
  - Combines with Va, calcium, and the phospholipid membrane to form prothrombinase complex
  - Converts prothrombin to thrombin

► Factor Va
  - Factor Xa and Va are present in stoichiometric amounts and cause an alteration in the binding site of Xa to increase the catalytic efficiency
  - Binds to prothrombin and sequesters it to the site of the prothrombinase complex
  - Produces a 300,000 fold increase in rate of prothrombin conversion

► Factors V and VIII are activated by proteases but are not active proteases themselves
Common pathway

► Thrombin and Fibrin
- Cleaves the soluble protein fibrinogen to produce the insoluble fibrin monomer
- Factor XIIIa cross links these monomers and allows formation of the meshwork of the thrombus
- Thrombin activates
  - Factors XII, XI, VII, and V
  - Activates platelets
  - Activates Protein C
  - Stimulates endothelial cells to produce plasminogen inhibitor
Role of Platelets

- Disc shaped, anuclear particles that circulate in a nonadhesive state in the undamaged circulation

- Changes in the platelet surface in the activated vs inactive state
  - Inactive- mostly phosphatidylcholine
  - Activated- mostly phosphatidylserine

- Contain a contractile system and storage granules
  - α granules contain platelet factor 4, thromboglobulin, PDGF, P-selectin, fibrinogen, factor V, vWF
  - β granules contain ATP, ADP, and serotonin
Role of Platelets

- First step toward platelet aggregation is adhesion
- Aggregation prevented by
  - Heparan sulfate- activates antithrombin
  - Thrombomodulin- activates protein C
  - PAI- induces fibrin degradation
  - TFPI- inhibits TF
  - Prostacyclin I2- raises CAMP levels and NO levels
- Injured endothelium promotes adhesion of platelets
- Platelet adhesion promotes activation
- Thrombin is the most potent aggregation factor for platelets
Other Factors

► Platelet integrins
  - GP Ib- vWF
  - GP Ia/IIa- collagen
  - GP IIb/IIIa- fibrinogen and fibronectin (most abundant)

► Leukocytes
  - Express minimal amounts of procoagulant activity normally
  - Monocytes express TF
  - Contain XI-VIII receptors which allows intrinsic pathway activation
  - Linked to thrombosis in sepsis

► Endothelium
  - Important in the regulation of coagulation
  - Undamaged
    - Thrombomodulin, fibrinolytic mediators, prostaglandins, NO, TFPI
  - Damaged
    - TF, PAI, vWF, procoagulant proteins
Endogenous Inhibitors

► Antithrombin
  ▪ Serine protease inhibitor (SERPIN)
  ▪ Primary inhibitor of coagulation
  ▪ Targets most coagulation proteases, plasmin, and kallikrein

► Heparin cofactor II
  ▪ Resembles antithrombin
  ▪ Only has activity against thrombin

► Protein C
  ▪ Keeps blood in fluid state
  ▪ Activated when thrombin binds to thrombomodulin
  ▪ Cleaves membrane bound Va and VIIIa
  ▪ Needs Protein S and factor V as cofactors
History and Physical Exam

► Detailed bleeding history
  - ? Bleeding after dental procedures, minor cuts, previous OR, prolonged menses, easy bruising, nose bleeds
  - Family history

► Physical Exam
  - Few true physical signs
  - Splenomegaly, hepatomegaly, hemarthroses, petechia (plt) or ecchymosis (coag)
Diagnostic Testing

- Bleeding is common
- Diagnosis of the underlying reason is vital
- Test of coagulation
  - PT
    - Extrinsic pathway
    - Measured by subjecting citrated plasma to TF, phospholipids, and calcium
    - Vitamin K dependent factors- II, VII, XI, X, V
    - INR
    - Corrected with FFP and/or Vitamin K
    - Can be elevated with high doses of heparin
  - aPTT
    - Intrinsic pathway
    - Unfractionated Heparin
    - Not used for low molecular weight Heparin
Diagnostic Testing

- **ACT**
  - Gross measurement of aPTT
  - Exposure to diatomaceous earth
- **Thrombin Time**
  - Thrombin induced conversion of fibrinogen to fibrin
  - Useful in monitoring thrombolysis/DIC
  - Rarely used
- **Bleeding Time**
  - Time needed for a superficial wound to clot
  - Tests mainly platelet function/number
  - Done by making a controlled wound with a template
- **Other tests**
  - Euglobulin Clot Lysis Time- time needed for clot to lyse in a test tube
  - Thromboelastography- blood placed in oscillating chamber allows complete evaluation of blood clotting ability
  - Platelet aggregability
  - Fibrinogen level
Causes of Bleeding

- Coagulopathic bleeding Congenital
  - Platelet Disorders
    - Rare
    - Divided into problems of adhesion, aggregation, secretion, and procoagulant activity
    - Treatment is platelets or DDAVP

- Von Willebrand Disease
  - Quantitative or qualitative defect of vWF
  - Carrier for factor VIII
  - Most commonly inherited bleeding disorder (incidence 1-2%)
  - Easy bruising, mucosal bleeding, menorrhagia, epistaxis, etc...
  - Treatment rarely required but if bleeding then DDAVP or factor VIII/vWF concentrate

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**Table 33-3** Inherited Platelet Disorders

<table>
<thead>
<tr>
<th>DISORDER</th>
<th>INHERITANCE</th>
<th>DEFECT</th>
<th>CAUSE</th>
<th>TREATMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bernard-Soulier syndrome</td>
<td>AR</td>
<td>Platelet adhesion</td>
<td>GP Ib-IX-V deficiency</td>
<td>Platelet transfusion</td>
</tr>
<tr>
<td>Glanzmann’s thrombasthenia</td>
<td>AR</td>
<td>Platelet aggregation</td>
<td>αIIbβ3 integrin</td>
<td>Platelet transfusion</td>
</tr>
<tr>
<td>Gray platelet syndrome</td>
<td>AR</td>
<td>Platelet secretion (alpha granule)</td>
<td>ξ-granule proteins are absent</td>
<td>Platelet transfusion, DDAVP</td>
</tr>
<tr>
<td>Quebec platelet disorder</td>
<td>AD</td>
<td>Platelet secretion (alpha granule)</td>
<td>Alpha granule proteins are degraded</td>
<td>Platelet transfusion, DDAVP</td>
</tr>
<tr>
<td>Wiskott-Aldrich syndrome</td>
<td>XR</td>
<td>Platelet secretion (dense granule)</td>
<td>Defect in dense granule secretion due to absence of WASP</td>
<td>Platelet transfusion, DDAVP, cryoprecipitate</td>
</tr>
<tr>
<td>Hermansky-Pudlasky syndrome</td>
<td>AR</td>
<td>Platelet secretion (dense granule)</td>
<td>Ceroid-lipofuscin-like lysosomal storage disease</td>
<td>Platelet transfusion, DDAVP, cryoprecipitate</td>
</tr>
<tr>
<td>Chédiak-Higashi syndrome</td>
<td>AR</td>
<td>Platelet secretion (dense granule)</td>
<td>Presence of giant inclusion bodies</td>
<td>Platelet transfusion, DDAVP, cryoprecipitate</td>
</tr>
</tbody>
</table>

AD: autosomal dominant; AR, autosomal recessive; DDAVP, desmopressin acetate (1-deamino-8-arginine vasopressin); GP, glycoprotein; XR, X-linked recessive.
### Table 33-4  Inherited Defects of Coagulation Factors

<table>
<thead>
<tr>
<th>DISEASE</th>
<th>FACTOR DEFICIENCY</th>
<th>INHERITANCE</th>
<th>DIAGNOSIS</th>
<th>TREATMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemophilia A</td>
<td>VIII</td>
<td>XR</td>
<td>Specific factor assay</td>
<td>Factor VIII</td>
</tr>
<tr>
<td>Hemophilia B</td>
<td>IX</td>
<td>XR</td>
<td>Specific factor assay</td>
<td>Factor IX</td>
</tr>
<tr>
<td>Hemophilia C</td>
<td>XI</td>
<td>AR</td>
<td>Specific factor assay</td>
<td>Factor XI</td>
</tr>
<tr>
<td>Fibrinogen disorders</td>
<td>I</td>
<td>Variable</td>
<td>Fibrinogen levels and analysis</td>
<td>Cryoprecipitate</td>
</tr>
<tr>
<td>Abnormal prothrombin</td>
<td>II</td>
<td>AR</td>
<td>Specialized assays</td>
<td>Variable replacements</td>
</tr>
<tr>
<td>Parahemophilia</td>
<td>V</td>
<td>AR</td>
<td>Specific factor assay</td>
<td>FFP</td>
</tr>
<tr>
<td>Factor VII deficiency</td>
<td>VII</td>
<td>AR</td>
<td>Specific factor assay</td>
<td>Factor VII</td>
</tr>
<tr>
<td>Stuart-Prower factor deficiency</td>
<td>X</td>
<td>AR</td>
<td>Specific factor assay</td>
<td>FFP, prothrombin complex concentrates</td>
</tr>
<tr>
<td>Hageman factor deficiency</td>
<td>XII</td>
<td>AR</td>
<td>Specific factor assay</td>
<td>Factor XIII, cryoprecipitate</td>
</tr>
<tr>
<td>Factor XIII deficiency</td>
<td>XIII</td>
<td>AR</td>
<td>Specific factor assay</td>
<td></td>
</tr>
</tbody>
</table>

AR, autosomal recessive; FFP, fresh frozen plasma; XR, X-linked recessive.

### Table 33-5  Characteristics of the Various Clotting Factors Required for Safe Surgical Hemostasis

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>IN VIVO HALF-LIFE</th>
<th>LEVEL REQUIRED FOR OPERATIVE HEMOSTASIS</th>
<th>STABLE IN PLASMA IF</th>
<th>BEST OPTIONS FOR REPLACEMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>3-4 days</td>
<td>100 mg/dL</td>
<td>4°C</td>
<td>FFP, cryoprecipitate</td>
</tr>
<tr>
<td>II</td>
<td>2-5 days</td>
<td>20-40%</td>
<td>4°C</td>
<td>FFP, concentrates</td>
</tr>
<tr>
<td>V</td>
<td>15-36 hr</td>
<td>&lt;25%</td>
<td>Frozen</td>
<td>FFP, platelets</td>
</tr>
<tr>
<td>VII</td>
<td>4-7 hr</td>
<td>10-20%</td>
<td>4°C</td>
<td>Concentrates, FFP</td>
</tr>
<tr>
<td>VIII</td>
<td>9-18 hr</td>
<td>≥85%</td>
<td>Frozen</td>
<td>Concentrates, cryoprecipitate, FFP</td>
</tr>
<tr>
<td>IX</td>
<td>20-24 hr</td>
<td>≥50%</td>
<td>4°C</td>
<td>Concentrates, FFP</td>
</tr>
<tr>
<td>X</td>
<td>32-48 hr</td>
<td>10-20%</td>
<td>4°C</td>
<td>FFP, prothrombin complex concentrates</td>
</tr>
<tr>
<td>XI</td>
<td>40-80 hr</td>
<td>15-25%</td>
<td>4°C</td>
<td>Concentrates, FFP</td>
</tr>
<tr>
<td>XII</td>
<td>48-52 hr</td>
<td>None</td>
<td>4°C</td>
<td>Not necessary</td>
</tr>
<tr>
<td>XIII</td>
<td>12 days</td>
<td>&lt;5%</td>
<td>Frozen</td>
<td>Concentrates, cryoprecipitate, FFP</td>
</tr>
<tr>
<td>vWF</td>
<td>Few hours</td>
<td>25-50%</td>
<td>Frozen</td>
<td>Concentrates, cryoprecipitate, FFP</td>
</tr>
</tbody>
</table>

FFP, fresh frozen plasma; vWF, von Willebrand's factor.

Causes of Bleeding

► Coagulopathic bleeding congenital cont’d
  ▪ Hemophilia
    ► A or B
    ► Hallmark is repeat bleeding into joints and muscles
    ► Levels
      ▪ <1% severe
      ▪ 1-5% moderately severe
      ▪ 6-25% mild
    ► Treat with factor replacement
    ► If immunity develops to exogenous factors → activated factor VII
  ► A
    ▪ Factor VIII
    ▪ X-linked recessive
    ▪ 1 in 5000 men affected
    ▪ 3% prevents spontaneous hemorrhage
    ▪ 30% for mild bleeding, 50% for major bleeding
    ▪ 80-100% during OR and 30% post op for 2 weeks
Causes of Bleeding

- **Hemophilia cont’d**
  - B
    - Accounts for 20% of hemophilia
    - X-linked recessive
    - Indistinguishable from hemophilia A
    - 20-30% levels for minor bleeding
    - 50-100% for 2 weeks post op

- **Acquired disorders of hemostasis**
  - Liver disease (decreased prothrombin, V, VII, X)
  - EtOH (thrombocytopenia)
  - Hypersplenism (thrombocytopenia)
Treatments

► Whole Blood
  - Occasionally used in the military, not readily available here

► PRBC
  - Stored @ 4 degrees Celsius up to 5 weeks
  - Restore oxygen carrying capacity
  - Transfuse to 7 mg/dL minimum

► FFP
  - Replaces all coagulation factors, but not as rich in factor VIII
  - Can be stored frozen for up to 12 months at -30 degrees Celsius
  - Useful in elevated PT
  - Useful when specific factor not available
Treatments

- **Platelets**
  - Prophylactic in massive hemorrhage
  - Contain a substantial amount of FFP and V
  - Need 20/mcL minimum for normal hemostasis 50-70/mcL for active bleeding

- **Cryoprecipitate**
  - Rich in VIII, vWF, fibrinogen, and fibronectin
  - Most commonly used to increase fibrinogen
  - Can be stored at -30 degrees Celsius for 12 months

- **Desmopressin**
  - Synthetic vasopression
  - Increases release of factor VIII and vWF
  - Improves platelet adhesion
Treatments

► Vitamin K
  - Carboxylates already synthesized factors stored in hepatocytes
  - Slower more durable correction

► Protamine Sulfate
  - Positively charged protein that reverses the effect of negatively charged heparin
  - 1mg/100u heparin
  - Can cause hypotension, pulmonary HTN, anaphylaxis, death
  - Derived from Salmon Semen

► Antifibrinolytic agents
  - i.e. Amicar
  - Block plasminogen primarily or the effect of plasmin on fibrinogen and fibrin

► Specific factors
Transfusion Reactions

► Febrile Transfusion Reactions
  - Most common
  - Treated with antipyretics and antihistamines
  - Removal of white cell debris from PRBC, plt and FFP reduces risk
  - Can be pre-treated if pt has history

► Hemolytic Transfusion Reaction
  - STOP administration of blood
  - Return to lab for repeat crossmatch
  - May require pressors to support BP, maintenance of renal perfusion, management of DIC
  - Treat with volume support first, pressors if needed, diuretics to maintain UO, and HD if renal failure
Transfusion Reactios

► Infection

- Hep C – 1 in 1,390,000
- Hep B – 1 in 200,000-500,000
- HIV – 1 in 2,000,000
- HTLV - <1 in 2,000,000
- West Nile Virus (11 documented cases)
- Syphilis – none in 30 years
- Chagas Disease – extremely low, red cross qualifies each donor rather than each donation for negativity
- Bacterial infection most common with plt transfusion

► Volume Overload
Transfusion Reactions

► Massive Transfusion
  - Coagulopathy, hypothreemia, citrate toxicity (liver dysfxn), electrolyte abnormalities (hyperkalemia, acidemia, hypocalcemia)

► TRALI
  - Acute lung injury developing within 6 hours of transfusion
  - Rapid onset of tachypnea, cyanosis, dyspnea, fever
  - Acute hypoxemia (paO2/FiO2 <300)
  - Wedge pressure < 18mmHg
  - Treatmens: aggressive resp support, may need mechanical ventilation
  - Leading reported cause of fatal transfusion reactions in the US in 2003/4
Hypercoagulable States

► Congenital disorders
  - Activated protein C resistance most common
    - Most common cause of APCR is Factor V leiden deficiency
  - ATIII deficiency
  - Protein C & S deficiency
  - Hyperhomocysteinemia

► Acquired disorders
  - Decreased production (liver failure)
  - Ineffective fibrinolysis
  - High levels of clotting factors (upregulated during stress)
  - Thrombocytosis
  - Antiphospholipid syndromes
  - Chronic cases of DIC
  - Hyperhomocysteinemia (in pts with renal failure)
Hypercoagulable States

► Diagnostic Evaluation
  ▪ Activated protein C resistance test
  ▪ Antithrombin III activity assay
  ▪ Proteins C & S activity
  ▪ Antiphospholipid antibody
  ▪ Prothrombin activity (screening for prothrombin 20210)
  ▪ Serum homocystine level

► Management
  ▪ Therapeutic anticoagulation for VTE (heparin/coumadin/antiplatelet)
  ▪ Treat hyperhomocystenemia
  ▪ DVT prophylaxis for high risk patients
Spleen

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Objectives

- Anatomy & Physiology
- Surgical disorders
- Consequences of Splenectomy
- Complications
Anatomy

- Embryology – develops from the dorsal mesogastrium by the 6th gestational week
- Receives 5% cardiac output
- Dual arterial/venous supply (splenic vessels and short gastric vessels)
  - Splenic artery – branch of the celiac
  - Short gastrics – from left gastroepiploic artery
Anatomy

- LUQ bound by the diaphragm and rib cage
- Intimately assoc w/ pancreas, stomach, left kidney, colon and diaphragm
- Multiple ligaments: splenorenal, gastroplenic, splenocolic and splenophrenic ligaments
Anatomy

► Accessory spleens: most commonly found in the splenic hilum, followed by the splenocolic ligament, gastrocolic ligament, splenorenal ligament and omentum.
  ▪ Important to know when performing splenectomy for hematologic disorders

► Polysplenia: multiple small spleens, no normal spleen
  ▪ Assoc w/ cardiac defects, situs inversus, biliary atresia

► Asplenia: absence of spleen
  ▪ Lethal condition assoc w/ cardiac defects and situs inversus

► Splenogonadal Fusion
  ▪ Rare disorder. Splenic tissue found in scrotum, attached to testicle.
Physiology

► Functions
  - Hematopoiesis
  - Blood filtering
  - Immune modulation

► Structure
  - Blood enters spleen through central arteries
  - Branch to trabecular arteries – white pulp
  - Then goes to the marginal zone (sinuses) and directed either to the red pulp or back to white pulp
Physiology

► White Pulp
  - Surrounded by lymphatic sheaths (T-lymphocytes and macrophages) that process soluble antigens
  - Some goes into lymphatic follicles, where B-lymphocytes can proliferate. Plasma cells are also found here.

► Red Pulp
  - Reticular network, no endothelial cells, moves slowly through numerous macrophages, then enters sinuses
  - Antibody-sensitized and particulate material removed
Physiology

- Filters and sequesters abnormal and aged erythrocytes, granulocytes and platelets
- Nearly 350 L/day filtered through spleen
- Immune function – reticuloendothelial system
  - Specific
    - Antigen processing and antibody production.
    - Largest producer of IgM
  - Non-specific
    - Clearance of opsonized particles and bacteria by splenic macrophages
    - Production of opsonin (properdin, tuftsin, fibronectin)
Surgical Disorders of the Spleen

► Splenic Rupture
  ▪ Trauma
  ▪ Spontaneus
  ▪ Iatrogenic injury

► Hematologic Disorders
  ▪ Hematolytic anemias
  ▪ Hereditary spherocytosis
  ▪ Thalassemias
  ▪ ITP
Surgical Disorders of the Spleen

- Hypersplenism from other Diseases
  - Inflammation
  - Infiltrative diseases
  - Congestion

- Leukemia and Lymphoma

- Other Diseases
  - Splenic abscess
  - Primary and metastatic tumors
  - Cysts
  - Splenic artery aneurysm
  - Isolated gastric varices
Surgical Disorders of the Spleen

► Splenic Trauma
  ▪ Most commonly injured organ after blunt trauma
    ► Usually has associated injuries (rib fractures, TBI, ortho injuries, liver injuries)
  ▪ Also common in penetrating trauma
  ▪ Management based on hemodynamic stability of the patient.
Splenic Trauma

► ATLS protocol for all patients

► Physical Exam
  - Unreliable in trauma patients
  - 20% of patients with splenic injuries have rib fx
  - LUQ pain/tenderness
  - Kehr’s sign – referred pain in left shoulder
  - Balance’s sign – percussion dullness to left flank
Splenic Trauma

► FAST
- Quick, good in “unstable” patients
- Non-specific – tells you fluid or no fluid
- Unstable pt with positive fast = OR

► CT
- Better for stable patients, more specific, diagnostic test of choice
- Allows for grading of injury and measurement of hemoperitoneum
- Able to diagnose other injuries as well
Splenic Trauma

- Grading scale to determine severity and uniformity of diagnosis
- No rule about certain grade needing splenectomy, but some generalizations
  - Elderly more likely to fail non-op
  - Higher grades more likely to fail non-op
  - Large amt of hemoperitoneum more likely to fail non-op

<table>
<thead>
<tr>
<th>Grade*</th>
<th>Injury type</th>
<th>Description of injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Hematoma</td>
<td>Subcapsular, &lt;10% surface area</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Capsular tear, &lt;1cm parenchymal depth</td>
</tr>
<tr>
<td>II</td>
<td>Hematoma</td>
<td>Subcapsular, 10%-50% surface area; intraparenchymal, &lt;5 cm in diameter</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Capsular tear, 1-3cm parenchymal depth that does not involve a trabecular vessel</td>
</tr>
<tr>
<td>III</td>
<td>Hematoma</td>
<td>Subcapsular, &gt;50% surface area or expanding; ruptured subcapsular or parenchymal hematoma; intraparenchymal hematoma ≥ 5 cm or expanding</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>&gt;3 cm parenchymal depth or involving trabecular vessels</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Laceration involving segmental or hilar vessels producing major devascularization (&gt;25% of spleen)</td>
</tr>
<tr>
<td>V</td>
<td>Laceration</td>
<td>Completely shattered spleen</td>
</tr>
<tr>
<td></td>
<td>Vascular</td>
<td>Hilar vascular injury with devascularizes spleen</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple injuries up to grade III.
Hematologic Disorders

► Hypersplenism vs splenomegaly
  ▪ Hypersplenism – excess fxn of spleen and causes cytopenia (anemia, leukopenia, thrombocytopenia)
  ▪ Splenomegaly (anatomic enlargement of the spleen)

► Hereditary Spherocytosis
  ▪ Autosomal dominant
  ▪ Deficiency in spectrin and makes rigid RBC which become sequestered in the red pulp
  ▪ Splenectomy to prevent anemia (wait until age 5, if possible)

► Metabolic hemolytic anemia
  ▪ Pyruvate kinase deficiency, G6PD deficiency, etc
  ▪ Not responsive to splenectomy
Hematologic Disorders

► Sickle Cell
  - Autosomal recessive
  - Rigid cells at low O2 sats
  - Also lead to increased viscosity, stasis and thrombocytosis
  - Usually infarct spleen and become functionally asplenic
  - Splenectomy may be beneficial during hemolytic crisis and splenomegaly

► Thalassemias
  - Major (homozygous beta thalassemia) – reduces transfusion requirements, splenomegaly and rupture
  - Minor (heterozygous beta thalassemia) – decreases transfusion requirements and issues with iron overload
Hematologic Disorders

► Thrombocytopenia
  ▪ Splenectomy only appropriate for idiopathic immune mediated thrombocytopenia (cause can’t be found)

► ITP
  ▪ Usually after an acute viral infection
  ▪ Women > men
  ▪ Steroids first
  ▪ If no response, may benefit from splenectomy
  ▪ Better response to splenectomy if good response w/ steroids, but recurrence once steroids are tapered.
Hematologic Disorders

► TTP
  - Disease of arteries or capillaries
  - Fevers, purpura, hemolytic anemia, neurologic manifestations, renal disease
  - Plasmapheresis is treatment

► HIV assoc Thrombocytopenia
  - Splenectomy if AIDS and symptomatic thrombocytopenia resistant to medical management
Hypersplenism from other diseases

► Congestive splenomegaly
  ▪ Usually as result of liver failure
  ▪ Treatment of portal hypertension
  ▪ Splenectomy contra-indicated as one of the treatments for portal hypertension is splenorenal shunt

► Infiltrative Splenomegaly
  ▪ e.g Gaucher’s disease
    ▶ Partial splenectomy or embolization used to treat symptoms (hypersplenism an pain from splenomegaly)

► Felty’s Syndrome
  ▪ RA pts with leg ulcers and assoc splenomegaly ane neutropenia
  ▪ Splenectomy controversial as results are unpredictable
Hematologic Malignancies

► Acute Leukemia
  ▪ Not indicated

► Chronic Leukemia
  ▪ Rarely for hypersplenism or sx of splenomegaly

► Leukemic reticuloendotheliosis
  ▪ Hairy cell leukemia
  ▪ For palliation of cytopenia and sx of splenomegaly with advent of medications

► Hodgkin’s disease
  ▪ Not routinely part of staging laparotomy anymore because of newer imaging techniques

► Non-hodgkin’s lymphoma
  ▪ Rarely indicated for hypersplenism or sx of splenomegaly
**Splenectomy**

- Midline incision, left subcostal incision (Kehr’s incision), or laparoscopic
- Mobilized from its retroperitoneal attachments bluntly
- Splenocolic, splenophrenic and splenorenal ligaments divided with electrocautery
- Short gastric arteries individually ligated in the gastrosplenic ligament near the spleen
- Splenic artery and vein are then individually ligated close to the spleen
Consequences and Complications

► Transient leukocytosis and thrombocytosis
  - WBC increases by avg of 50% from baseline
  - Usually normalizes within 5-7 days
  - Plt increases by avg 30%
  - Usually normalizes within 2 wks

► Postsplenectomy sepsis
  - Higher incidence in children (2-4% vs 1-2% in adults)
  - Hematologic disorders at highest risk
  - Strep pneumo most common organism (H.flu, N.meningitidis, beta-hemolytic strep, S.aureus, E.coli, Pseudomonas)
  - Pts progress rapidly (within hours) to sepsis and even death despite appropriate abx
  - Waterhouse-Friderichsen’s Syndrome not uncommon
Consequences and Complications

► Encapsulated organisms
  - Vaccinate against S.pneumo, H.flu, N. meningiditidis
  - Before surgery appx 1 week, if possible

► Atelectasis
  - Most common complication, from discomfort related to upper abd incision
  - Pulmonary toilet

► Subphrenic abscess
  - Can develop assoc pleural effusion
  - Fluid collects in splenic fossa. Can become infected
  - No role for routine drain
Consequences and Complications

► Pancreatic injury
  ▪ 1-3% of patients
  ▪ Increased risk of abscess
  ▪ Can have pancreatic fistula, local pancreatitis, pancreatic pseudocyst
  ▪ Sx similar to subphrenic abscess

► Stomach injury
  ▪ Usually related to where short gastric arteries were ligated
  ▪ Subphrenic abscess or gastrocutaneous fistula
  ▪ Some surgeons advocate keeping NG for a couple days, but no data to support
Questions?