Splenectomy

- **1549** – First reported by Zacarello in Italy

- **1816** – First in North America, O’Brien
  - Victim stabbed in LUQ while committing a rape

- **1826** – Quittenbaum, 1st elective splenectomy
  - Portal HTN

Table 53.1. HISTORICAL MILESTONES IN SURGERY OF THE SPLEEN

<table>
<thead>
<tr>
<th>Year</th>
<th>Description</th>
</tr>
</thead>
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<tr>
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</tr>
<tr>
<td>1991</td>
<td>Laparoscopic splenectomy</td>
</tr>
</tbody>
</table>
Splenectomy

- **1866** – Bryant, 1\textsuperscript{st} splenectomy for leukemia
- **1908** – Johnson reports mortality of 87.7\% in 49 patients with leukemia
- **1916** – Kaznelson reports good results for thrombocytopenic purpura

Table 53.1. HISTORICAL MILESTONES IN SURGERY OF THE SPLEEN

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<td>Laparoscopic splenectomy</td>
</tr>
</tbody>
</table>
Splenectomy

- **1952** – OPSS reported
- **1962** – Christo in Brazil: Splenic salvage prevents OPSS
- **1991** – Laparoscopic splenectomy reported by four different groups

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<td>First report of splenic salvage procedure for trauma</td>
</tr>
<tr>
<td>1991</td>
<td>Laparoscopic splenectomy</td>
</tr>
</tbody>
</table>
Anatomy

Tail of Pancreas
- Direct contact with spleen in 30%
- Otherwise within 1 cm

Figure 53.1. Anatomic relation of the spleen to the liver, diaphragm, pancreas, colon, and kidney. The stomach is sectioned to illustrate the anatomic relations in situ.
**Suspensory Ligaments**

- Gastrosplenic Ligament
  - Short Gastric Vessels
  - Gastroepiploic Vessels

- Splenorenal Ligament
  - Splenic Vessels
  - Tail of Pancreas

- Splenocolic Ligament

- Phrenicocolic Ligament
  - Phrenicosplenic ligament
Blood Supply

-Superior Polar Artery may arise directly from Celiac trunk ("Duplicate artery")
-Splenic artery may arise directly from Aorta, SMA, MCA

Figure 53.3. The arterial blood flow to the spleen is derived from the splenic artery, left gastroepiploic artery, and short gastric arteries (vasa brevia). The venous drainage into this portal vein is also shown.
70% - Distributed
- Branches originate 3-13cm from hilum
- Transverse Anastomoses – why embolization / clipping may fail
- Pancreatica Magna – embolic debris from angio may cause pancreatitis

30% - Magistral
- Branches originate within 3.5 cm of hilum
- L Gastroepiploic artery – Most varied of splenic branches (72% arise several cm from splenic artery proximal to terminal branching)

Figure 1 Shown are (a) the distributed type and (b) the magistral type of splenic vascularization.
Splenic blood flow accounts for what percentage of total portal blood flow in normal subjects?

A. 10%
B. 20%
C. 30%
D. 40%
E. 50%
F. Beats the Fick out of me
Vascular anatomy of the spleen is:

A. Segmental end arteries with somewhat discrete segments

B. An open system of sinusoids without segments
Pseudo-Segmental Blood Supply

- End Arterioles
  → “White Pulp”
    - T-Cells
    - B-Cells

- No true capillaries
  → “Red Pulp”
    - Venous Sinusoids
    - Splenic Cords of Billroth
      - Macrophages
      - Dendritic Cells
The spleen is an important storage site for:

A. RBC’s
B. WBC’s
C. Platelets
D. All of the Above
Table 53.2. NORMAL FUNCTIONS OF THE SPLEEN

<table>
<thead>
<tr>
<th>HEMATOLOGIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Culling or destruction of senescent erythrocytes</td>
</tr>
<tr>
<td>Pitting or removal of cytoplasmic inclusions in erythrocytes</td>
</tr>
<tr>
<td>Reservoir for platelets and granulocytes</td>
</tr>
<tr>
<td>Hematopoiesis—during fetal life and in conditions associated with bone marrow destruction</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>IMMUNOLOGIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Filtration and trapping of circulatory antigens</td>
</tr>
<tr>
<td>Lymphocyte stimulation and proliferation</td>
</tr>
<tr>
<td>Antibody production in germinal follicles</td>
</tr>
<tr>
<td>Production of opsonins: tuftsin and properdin</td>
</tr>
</tbody>
</table>
White Pulp
- Opsonins – IgM, IgG, tuftsin, properdin
- T-Cells

Red Pulp (75% of parenchyma)
- Phagocytosis
- “Pitting” damaged RBCs, WBCs
- Opsonized Pathogen Clearance
Immunologic “Anatomy”
The spleen is usually enlarged in all EXCEPT:

- A. ITP
- B. Hemolytic Anemias
- C. Gaucher’s Disease
- D. Chronic Lymphocytic Leukemia (CLL)
- E. Spleen is enlarged in all the above
Hypersplenism

• “Hematologic effects of splenomegaly”

• Enhanced capacity of the enlarged spleen:
  ▪ Pooling,
  ▪ Sequestering, and
  ▪ Destroying blood cells

• Results in reduction of blood cell counts
  ▪ Bone marrow function usually normal
  ▪ Sometimes by sequestration alone
  ▪ Cytopenia corrected following splenectomy
Hypersplenism

Platelet Sequestration

Palpable spleen is at least twice its normal size
Hypersplenism –– Many Etiologies

Table 53.5. **CAUSES OF HYPERSPLENISM**

I. Primary diseases of blood cells; normal spleen
   A. Congenital
      i. Erythrocyte abnormalities
         a. Hereditary spherocytosis
         b. Hereditary elliptocytosis
         c. Glucose-6-phosphate dehydrogenase deficiency
         d. Pyruvate kinase deficiency
      ii. Hemoglobin abnormalities
         a. Thalassemia major
         b. Sickle cell anemia (eventually results in splenic infarction and hyposplenism)
      iii. Platelet abnormalities
         a. Wiskott-Aldrich syndrome
   B. Acquired
      i. Autoimmune hemolytic anemia
      ii. Autoimmune neutropenia—Felty's syndrome
      iii. Immune thrombocytopenic purpura
      iv. Thrombotic thrombocytopenic purpura

II. Primary disorders of the spleen
   A. Neoplastic
      i. Hairy cell leukemia
      ii. Chronic lymphocytic leukemia
      iii. Chronic myelogenous leukemia
      iv. Non-Hodgkin's lymphoma
   B. Cellular infiltration (hematopoiesis)
      i. Agnogenic myeloid metaplasia
      ii. Mastocytosis
      iii. Chédiak-Higashi syndrome
   C. Metabolic infiltration
      i. Gaucher's disease
      ii. Sarcoidosis
   D. Vascular
      i. Splenic vein thrombosis
      ii. Portal vein hypertension (cirrhosis)
Hypersplenism

• Why Splenectomy in Hypersplenism?
  ▪ Treat Splenomegaly
    • Compressive symptoms
    • Risk for splenic injury if active
  ▪ Improve blood counts – RBC’s, Platelets
  ▪ Temporize underlying condition
    • Rarely curative, but an adjunctive therapy
    • Failed medical management
    • Reduce number of required transfusions
    • Pain or abcess secondary to splenic infarction
      (sickle cell, thalassemia)
  ▪ Staging Procedure
Splenectomy is the treatment of choice for hydatid cysts of the spleen:

A. True
B. False
C. Both
D. None of the above except maybe C

- Typically unilocular cyst in the spleen < 5% in USA
- Serology confirms Dx
- Open splenectomy
Splenic Neoplasms and Cysts

- **NHL** - Most common 1° and 2° splenic tumor

- True Cysts (20%)
  - Epidermoid (15-20%)
    - Benign, Young
    - Compression Sx’s
    - Hemorrhage, rupture, infection
  - Echinococcal Cysts (<5%)
    - Serology diagnostic

- Pseudocysts – (80%)
  - Splenic trauma

---

**Table 1 Classification of Splenic Neoplasms and Cysts**

<table>
<thead>
<tr>
<th>NEOPLASMS</th>
<th>Malignant</th>
<th>Primary (non-Hodgkin’s lymphoma)</th>
<th>Metastatic (lung, colorectal, breast, melanoma)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Benign</strong></td>
<td></td>
<td>Hemangioma</td>
<td>Lymphangioma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hamartoma</td>
<td>Littoral cell angioma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Peliosis of the spleen</td>
<td>Hemangioendothelioma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Angiomyolipoma</td>
<td>Hemangiopericytoma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Inflammatory pseudotumor</td>
<td></td>
</tr>
<tr>
<td><strong>CYSTS</strong></td>
<td></td>
<td><strong>True</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Parasitic (echinococcal)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nonparasitic</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Congenital</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neoplastic (epidermoid, dermoid, lymphangioma, cavernous angioma)</td>
<td></td>
</tr>
<tr>
<td><strong>False</strong></td>
<td></td>
<td>(pseudocyst)</td>
<td></td>
</tr>
</tbody>
</table>
All are associated with congenital spherocytosis EXCEPT:

A. Leg Ulcers
B. Pigment gallstones
C. Jaundice
D. Increased osmotic fragility of RBC’s
E. Splenomegaly
F. Cholesterol gallstones
Hereditary Spherocytosis

- Autosomal Dominant
  - Most common hemolytic anemia in North Europeans
- Defect in Spectrin
- Shortened RBC Life
  - Unable to pass through splenic sinusoids
  - Splenomegaly
  - Pigmented Gallstones
- “Anemic Crisis” following viral Sx
Gaucher’s disease

Fig. 20.12 Gaucher’s disease: (a) bone marrow aspirate—a Gaucher cell with ‘fibrillar’ cytoplasmic pattern; (b) spleen histology—pale clusters of Gaucher cells in the reticuloendothelial cords.
Splenic Abcess

- Uncommon
  - Male > Female 2:1

- Bimodal Distribution for Primary Abcess
  - < 40 years
    - Immunosuppressed
    - IV Drug Abuse
    - Multiloculated
  - > 70 years
    - DM, Septic Focus
    - Unilocular

- Secondary Abcess
  - Infected hematoma (trauma)
  - Infected splenic infarct
  - Contiguous infection (pancreatitis, perinephric)

- Treatment
  - Broad-spectrum Abx
    - GPC, GNB, Anaerobes
    - May be complicated by recurrent abcess
  - Percutaneous Drainage
    - Thin fluid
  - Drainage in OR / Splenectomy
Platelet count following splenectomy for ITP will rise significantly within:

A. 1-2 Days
B. 1-2 Weeks
C. 1 Month
D. 1 Year

s/p Splenectomy for trauma – platelets rise within 7-10 days
Immune Thrombocytopenic Purpura (ITP)

• Highest incidence in women aged 15-50
• Usually Idiopathic
• May be seen in conjunction with:
  ▪ SLE, HIV, CLL, Hodgkin’s Dz, or autoimmune hemolytic anemia
• Normal platelet lifespan from 7-10 days to 5 hrs
  ▪ Total platelet turnover 5x normal
• May remit and relapse over time
  ▪ Autoantibodies directed against platelet surface Ag
**Immune Thrombocytopenic Purpura (ITP)**

- Medical treatment successful in only about 15% of patients
- Splenectomy successful in:
  - 66% of patients initially with full response
  - Additional 15% with partial response
  - 15% of these will relapse with 1 year, can relapse years later
- **Indications for Splenectomy in ITP:**
  - Persistent platelet count < 80,000/mm³ despite therapy
  - Recurrence of thrombocytopenia after tapering or discontinuation of steroids
  - Emergent splenectomy only if evidence of ICH
  - Children: Indicated if no remission after 1 year
All the following can help reduce intraoperative bleeding during splenectomy for ITP, EXCEPT:

A. Preop Steroid Administration
B. Preop use of IV IgG (gamma-globulin)
C. Intraop administration of Platelets before splenic hilum is controlled
D. It doesn’t matter because “If you ain’t wreckin’, you ain’t racin’”

- Preop steroids only depending on the patient – either for stress dose if on steroids or to help increase platelet levels
- Preop IV IgG to help boost Platelet levels in thrombocytopenic patients
Mechanism and Treatment of ITP

- Steroids
- IV IgG
- Antimitotics, Colchicine
Splenectomy for ITP
Hyposplenism - Peripheral Blood Smear

Table 3 Changes in Peripheral Red Blood Cells after Splenectomy

<table>
<thead>
<tr>
<th>DESCRIPTION</th>
<th>CAUSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Howell-Jolly bodies</td>
<td>Nuclear remnant</td>
</tr>
<tr>
<td>Target cells</td>
<td>Immature cells</td>
</tr>
<tr>
<td>Stippling</td>
<td>Inclusions</td>
</tr>
<tr>
<td>Acanthocytes</td>
<td>Spurred cells</td>
</tr>
<tr>
<td>Heinz bodies</td>
<td>Denatured hemoglobin</td>
</tr>
<tr>
<td>Pappenheimer bodies</td>
<td>Iron granules</td>
</tr>
</tbody>
</table>

Normoblast (nucleated RBC)  Howell-Jolly body  Siderotic granules (Pappenheimer bodies)  Target cell
Incidence of overwhelming post-splenectomy sepsis is LEAST following splenectomy for:

A. ITP  
B. Congenital Hemolytic Anemia  
C. Acquired Hemolytic Anemia  
D. Trauma  

Trauma –
- 20% of patients with capsule disruption develop splenosis
Splenosis
Accessory spleens are found in about:

A. 2% of cases
B. 20% of cases
C. 80% of cases
D. 100% of cases when really searched for
E. All cases of Splenosis

-Hypersplenism / ITP – Searching for and resecting any accessory spleens part of the procedure
Accessory Spleens

- Usually on Left side
- Never below Left Ovary or Testicle

**Figure 6** Laparoscopic splenectomy: lateral approach. (a) Accessory spleens are known to occur at specific sites. (b) Shown is an accessory spleen.
Immunizations should be given:

A. Prior to splenectomy
B. Intraop, before clamping splenic artery
C. Prior to discharge
D. Within 2-4 weeks following splenectomy

- Best to give before splenectomy – better immune response
- Immune suppressed on steroids for ITP - BEFORE
- Prior to trauma lap if possible - otherwise ASAP
- Definitely before D/C on Trauma patient – poor followup
Post-Splenectomy Sepsis

- Overwhelming Post-Splenectomy Sepsis
  - a.k.a. OPSS, OPSI

- Caused by encapsulated bacteria
  - *Streptococcus pneumoniae*
  - *Haemophilus influenzae*
  - *Neisseria meningitidis*

- 80% of cases occur within 2 years of splenectomy
Post-Splenectomy Sepsis

- Occurs more often in children and immunocompromised adults
  - Splenectomy reserved for after age 4

- Rare but usually fatal
  - 50-60% mortality even with treatment
  - Splenic salvage highly desirable when safe
    - Selective nonoperative management (i.e. Trauma)
    - Operative splenorrhaphy
    - Intentional splenosis when appropriate
Table 53.4. **GUIDELINES FOR PREVENTION OF POST-SPLENIC SEPSIS**

- Vaccinate with polyvalent pneumococcal vaccine at least 10 to 14 days before splenectomy, if possible.
- For high-risk patients (immunosuppressed, children <10 years of age), meningococcal vaccine and Haemophilus influenzae vaccine.
- Antibiotic prophylaxis for children <5 years of age.
- Early antibiotic treatment for initial signs of infection.
- Medi-Alert bracelet.

* Pre-op for distal pancreatectomy in case splenectomy performed
Splenic Trauma

• Treatment has come full circle
  ▪ 1890’s – Nonoperative Management
  ▪ 1900’s – Operative Management

• OPSS – Not as likely following Trauma
  ▪ Partial Splenectomy
  ▪ Accessory Spleens
  ▪ Splenosis
Splenic Trauma

- Most frequently injured intraabdominal organ in blunt trauma
- Fractured left ribs and pulmonary contusion most common associated injuries
- Hematuria most common nonspecific finding
  - Renal injury
Splenic Trauma - Diagnosis

- **Old:** Peritoneal Lavage
  - >100,000 RBC’s/HPF
  - Food Particles
  - Very Sensitive
  - Many False +’s

- **Intermediate:** CT Scan

- **New:** FAST
  - Looking for Blood only
  - **NOT** looking for organ detail!
  - Wait for foley: may get more detail if bladder full
# AAST Splenic Injury Scale

## Table 1: AAST Organ Injury Scales for Liver, Biliary Tract, Diaphragm, and Spleen

<table>
<thead>
<tr>
<th>Injured Structure</th>
<th>AAST Grade</th>
<th>Characteristics of Injury</th>
<th>AIS-90 Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spleen*</td>
<td>I</td>
<td>Hematoma: subcapsular, nonexpanding, &lt; 10% surface area&lt;br&gt;Laceration: capsular tear, nonbleeding, &lt; 1 cm parenchymal depth</td>
<td>2</td>
</tr>
<tr>
<td>Spleen*</td>
<td>II</td>
<td>Hematoma: subcapsular, nonexpanding, 10%-50% surface area; intraparenchymal, nonexpanding, &lt; 5 cm in diameter&lt;br&gt;Laceration: capsular tear, active bleeding, 1-3 cm parenchymal depth, not involving a trabecular vessel</td>
<td>2</td>
</tr>
<tr>
<td>Spleen*</td>
<td>III</td>
<td>Hematoma: subcapsular, &gt; 50% surface area or expanding; ruptured subcapsular hematoma with active bleeding; intraparenchymal, &gt; 5 cm or expanding&lt;br&gt;Laceration: &gt; 3 cm parenchymal depth or involving trabecular vessels</td>
<td>3</td>
</tr>
<tr>
<td>Spleen*</td>
<td>IV</td>
<td>Hematoma: ruptured intraparenchymal hematoma with active bleeding&lt;br&gt;Laceration: laceration involving segmental or hilar vessels producing major devascularization (&gt; 25% of spleen)</td>
<td>4</td>
</tr>
<tr>
<td>Spleen*</td>
<td>V</td>
<td>Laceration: completely shattered spleen&lt;br&gt;Vascular: hilar vascular injury that devascularizes spleen</td>
<td>5</td>
</tr>
</tbody>
</table>

*Advance one grade for multiple injuries, up to grade III.

*Advance one grade for bilateral injuries, up to grade III.

AAST—American Association for the Surgery of Trauma
Figure 8  (a) The first step in mobilizing the spleen is to make an incision in the peritoneum and the endoabdominal fascia, beginning at the inferior pole and continuing posteriorly and superiorly. (b) The correct plane of dissection is between the pancreas and Gerota's fascia.
Figure 53.12. (A, B) A bleeding spleen can be mobilized rapidly in most patients by blunt dissection of the lateral attachments. (C) The splenic hilum can then be controlled quickly.
Splenic repairs (splenorrhaphy) are usually more successful in:

A. Women than in men  
B. Men than in women  
C. Adults than in children  
D. Children than in adults

**Splenic conservation historically performed in Children:**
- More likely to develop OPSS, saving splenic function protective  
- Capsule more amenable to repair (adults tear)  
-Lower arterial pressure in splenic arterial system – easier hemostasis
Other Methods:

Figure 53.13. (A) Techniques to suture superficial splenic lacerations. (B) Technique to control bleeding after hemispleenectomy. The sutures can be interlocked. (C) A polyglycolic acid mesh sheet or mesh bag can be applied to a spleen from which the capsule has been stripped.
Nonoperative Management

• With careful patient selection, success rate now approaches 95% (85-95%)
  ▪ Hemodynamic stability
  ▪ No contrast ‘pooling’ on CT
  ▪ No other intraabdominal injuries requiring laparotomy

• Follow frequent serial vital signs and H/H
  ▪ Treat persistently falling RBC with pRBC’s
  ▪ Rebleeding most likely within 1st 48 hours
  ▪ Likely failure if patient requires ≥ 2 u pRBCs
    • If still falling after 2u, consider angio for embolization
    • If hypotension develops, consider angiography
Nonoperative Management

- Follow-up CT scans rarely necessary
  - Indicated for falling BP or H/H during observation
  - **Grade I-II:** rarely show progression of lesion or other complications on CT. No need for repeat CT scan if H/H and vitals stable
  - **Grade III:** CT’s on case-by-case basis
  - Consider U/S for monitoring if necessary
  - Contact Sports: Complete resolution on CT required before can return to activity

- 2-5% of patients treated nonoperatively will develop parenchymal infarction or infection
Table 1 Criteria for Nonoperative Management

Hemodynamic stability
Computed tomography scan documentation of injury
Absence on computed tomography scan of active intrasplenic bleeding ("pooling of contrast")
Absence on computed tomography scan of other intraabdominal injuries requiring operative intervention
Limitation of splenic-related blood transfusion (<2 units)
Nonoperative Management

• Delayed Rupture
  ▪ 75% occur within 2 weeks in several series
    • Hematoma liquifying?
  ▪ Can occur anytime! (1 month – years)
  ▪ Actual incidence of delayed rupture very low
  ▪ Need to inform patients of this prior to D/C
Splenectomy is appropriate for:

A. Esophageal varices secondary to cirrhosis
B. Fundal varices secondary to splenic vein thrombosis
C. Both A and B
D. None of the Above

Splenic Vein Thrombosis –
- “Sinistral” paradoxic LEFT sided portal Hypertension
- Short Gastric vv → Fundal/Esophageal vv → Azygous V
- Gastric varices in 80%, Esophageal varices in 30-40%
# Elective Splenectomy

## Table 53.10. OPERATIVE INDICATIONS FOR SPLENECTOMY (TOTAL OR PARTIAL)

<table>
<thead>
<tr>
<th>Disease</th>
<th>Splenectomy required</th>
<th>Partial splenectomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hereditary spherocytosis</td>
<td>Always</td>
<td>Yes</td>
</tr>
<tr>
<td>Hereditary elliptocytosis</td>
<td>Sometimes</td>
<td>Yes</td>
</tr>
<tr>
<td>Thalassemia</td>
<td>Sometimes</td>
<td>Yes</td>
</tr>
<tr>
<td>Sickle cell anemia</td>
<td>Rarely</td>
<td>No</td>
</tr>
<tr>
<td>Wiskott-Aldrich syndrome</td>
<td>Sometimes</td>
<td>No</td>
</tr>
<tr>
<td>Autoimmune hemolytic anemia</td>
<td>Usually</td>
<td>No</td>
</tr>
<tr>
<td>Autoimmune neutropenia</td>
<td>Sometimes</td>
<td>No</td>
</tr>
<tr>
<td>Immune thrombocytopenic purpura</td>
<td>Usually</td>
<td>No</td>
</tr>
<tr>
<td>Thrombotic thrombocytopenic purpura</td>
<td>Sometimes</td>
<td>No</td>
</tr>
<tr>
<td>Hairy cell leukemia</td>
<td>Rarely*</td>
<td>No</td>
</tr>
<tr>
<td>Chronic lymphocytic leukemia</td>
<td>Sometimes</td>
<td>No</td>
</tr>
<tr>
<td>Chronic myelogenous leukemia</td>
<td>Sometimes</td>
<td>No</td>
</tr>
<tr>
<td>Non-Hodgkin's lymphoma</td>
<td>Sometimes</td>
<td>No</td>
</tr>
<tr>
<td>Agnogenic myeloid metaplasia</td>
<td>Sometimes</td>
<td>Yes</td>
</tr>
<tr>
<td>Mastocytosis</td>
<td>Rarely</td>
<td>No</td>
</tr>
<tr>
<td>Gaucher's disease</td>
<td>Rarely*</td>
<td>Yes</td>
</tr>
<tr>
<td>Hodgkin's disease</td>
<td>Rarely*</td>
<td>No</td>
</tr>
<tr>
<td>Splenic vein thrombosis</td>
<td>Always</td>
<td>No</td>
</tr>
<tr>
<td>Splenic abscess</td>
<td>Usually</td>
<td>No</td>
</tr>
<tr>
<td>Splenic cyst</td>
<td>Rarely</td>
<td>Yes</td>
</tr>
<tr>
<td>Echinococcal cyst</td>
<td>Always</td>
<td>No</td>
</tr>
</tbody>
</table>

*Splenectomy rarely indicated in current practice because of effective medical therapy.

*Splenectomy rarely indicated because of change in current therapy.
Figure 53.15. Technique for elective splenectomy. (A) The inferior pole is reflected laterally by the assistant's fingers to expose the lower edge of the hilar peritoneal envelope. (B) The hilar peritoneum is opened, inferiorly to superiorly in this case. (C) Individual vessels are identified and ligated with sutures.
Staging Laparotomy for Hodgkin’s Disease

- Indicated in Stage Ia-IIa patients where results may affect medical management (radiation vs chemotherapy)

- Very rarely performed anymore

- No evidence staging laparotomy affects outcome, despite different treatment modalities

Table 53.7. ANN ARBOR STAGING SYSTEM FOR HODGKIN’S DISEASE

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Single nodal group involved</td>
</tr>
<tr>
<td>II</td>
<td>Two nodal groups on the same side of the diaphragm</td>
</tr>
<tr>
<td>III</td>
<td>Nodal and/or splenic involvement on both sides of the diaphragm</td>
</tr>
<tr>
<td>IV</td>
<td>Extraneal (E) involvement (e.g., liver, bone marrow, skin)</td>
</tr>
<tr>
<td>A</td>
<td>No constitutional symptoms</td>
</tr>
<tr>
<td>B</td>
<td>Constitutional symptoms of weight loss, fevers, night sweats, and pruritus</td>
</tr>
</tbody>
</table>

Figure 53.8. The tissues to be removed or to undergo biopsy in a staging laparotomy for Hodgkin’s disease. Splenectomy, liver biopsy, and lymph node sampling in the specific sites are shown. Bone marrow biopsy can be performed if necessary.
Anticoagulation should be considered following splenectomy:

A. If the WBC remains elevated > 1 month
B. If the Platelet count rises ≥ 1,000,000
C. Patient has persistent anemia following splenectomy for hemolytic anemia
D. Following splenectomy for ITP and the platelet count rises to ≥ 1,000,000

- Hypercoaguable state – High risk for portal vein thrombosis
  - Warm Agglutins (IgG) vs Cold Agglutinins (IgM)
  - Anemia by IgM Ab’s generally not improved by splenectomy
- 15-20% of patients have persistent elevation of WBC post-splenectomy
# Lap vs Open Splenectomy

## Table 2 Collected Series Comparing Laparoscopic (LS) and Open Splenectomy (OS)

<table>
<thead>
<tr>
<th>STUDY</th>
<th>NUMBER</th>
<th>LOS (DAYS)</th>
<th>LIQUIDS</th>
<th>TIME (HR)</th>
<th>COST ($ \times 10^3)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LS</td>
<td>OS</td>
<td>LS</td>
<td>OS</td>
<td>LS</td>
</tr>
<tr>
<td>Freidman, 1997</td>
<td>63</td>
<td>74</td>
<td>3.5</td>
<td>6.7*</td>
<td>1.5</td>
</tr>
<tr>
<td>Brunt, 1996</td>
<td>26</td>
<td>20</td>
<td>2.5</td>
<td>5.8*</td>
<td>3.4</td>
</tr>
<tr>
<td>Janu, 1996</td>
<td>14</td>
<td>47</td>
<td>2.6</td>
<td>3.6</td>
<td>3.1</td>
</tr>
<tr>
<td>Rhodes, 1995</td>
<td>24</td>
<td>11</td>
<td>3</td>
<td>7*</td>
<td>2.0</td>
</tr>
<tr>
<td>Yee, 1995</td>
<td>25</td>
<td>25</td>
<td>5.1</td>
<td>6.7</td>
<td>3.3</td>
</tr>
<tr>
<td>Sclhinkert, 1995</td>
<td>7</td>
<td>14</td>
<td>2.1</td>
<td>5.0*</td>
<td>2.5</td>
</tr>
<tr>
<td>Total (mean)</td>
<td>159</td>
<td>191</td>
<td>3.1</td>
<td>5.8</td>
<td>2.8</td>
</tr>
</tbody>
</table>

*LOS, Length of stay.

*p < .05 compared with LS.*
Laparoscopic Splenectomy - Indications

- ITP \( \rightarrow \) **Most Common**
- Hereditary Spherocytosis
- TTP
- Autoimmune Hemolytic Anemia
- Lymphoma
  - Hodgkin’s Dz (Staging)
  - Non-Hodgkin’s (hypersplenism)
- Leukemia (hypersplenism)
- Hemoglobinopathies
  - Thalassemia, Sickle Cell
- Splenic Abcess
- Splenic Cyst
- Gaucher’s Dz (storage)
- Felty’s Syndrome
  - RA neutropenia
  - Splenomegaly
- Myelofibrosis
  - Extramedullary hematopoiesis
- Splenic Infarct
- AIDS Thrombocytopenia
- Hypersplenism
  - Portal HTN, SLE, or Sarcoid
## Table 2  Clinical Results of Laparoscopic Splenectomy

<table>
<thead>
<tr>
<th>Authors</th>
<th>N</th>
<th>ITP/Non-ITP</th>
<th>Conversion Rate (%)</th>
<th>OR Time (min)</th>
<th>Morbidity (%)</th>
<th>Mortality (%)</th>
<th>Length of Stay (days)</th>
<th>Accessory Spleen Present (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All diagnoses</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Katkhouda et al (1998)(^\text{37})</td>
<td>103</td>
<td>67/36</td>
<td>3.9</td>
<td>161</td>
<td>6</td>
<td>0</td>
<td>2.5</td>
<td>16.5</td>
</tr>
<tr>
<td>Targarona et al (2000)(^\text{35})</td>
<td>122</td>
<td>54/68</td>
<td>7.4</td>
<td>153</td>
<td>18</td>
<td>0</td>
<td>4.0</td>
<td>12</td>
</tr>
<tr>
<td>Park et al (2000)(^\text{38})</td>
<td>203</td>
<td>129/74</td>
<td>3.0</td>
<td>145</td>
<td>9</td>
<td>0.5</td>
<td>2.7</td>
<td>12.3</td>
</tr>
<tr>
<td>Poulin et al (2001)(^\text{36})</td>
<td>100</td>
<td>50/50</td>
<td>8.0</td>
<td>180</td>
<td>15</td>
<td>4</td>
<td>3.0</td>
<td>25</td>
</tr>
<tr>
<td>ITP</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trias et al (2000)(^\text{39})</td>
<td>48</td>
<td>—</td>
<td>4.2</td>
<td>142</td>
<td>12</td>
<td>N/A</td>
<td>4.0</td>
<td>11</td>
</tr>
<tr>
<td>Poulin et al (2001)(^\text{38})</td>
<td>51</td>
<td>—</td>
<td>3.9</td>
<td>160</td>
<td>5.9</td>
<td>0</td>
<td>2.0</td>
<td>32</td>
</tr>
<tr>
<td>Malignancy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schlachta et al (1999)(^\text{40})</td>
<td>14</td>
<td>—</td>
<td>21</td>
<td>239</td>
<td>18</td>
<td>9</td>
<td>3.0</td>
<td>—</td>
</tr>
<tr>
<td>Trias et al (2000)(^\text{39})</td>
<td>28</td>
<td>—</td>
<td>14*</td>
<td>171</td>
<td>28</td>
<td>N/A</td>
<td>5.5</td>
<td>—</td>
</tr>
</tbody>
</table>

\(^*\) 71% required accessory incision because of spleen size.
Laparoscopic Splenectomy

- **Patient Positioning**
  - Usually Lateral
  - Anterior approach for large spleens (>25-30cm)

- **Trocar placement**
Figure 4 Laparoscopic splenectomy: lateral approach. Shown is standard trocar placement. Four trocars are used. In most cases, the procedure is begun without the posterior trocar in place.

Table 1 Classification of Spleens According to Spleen Length*

<table>
<thead>
<tr>
<th>Spleen Class</th>
<th>Spleen Length</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal-size spleen</td>
<td>7–11 cm</td>
</tr>
<tr>
<td>Moderate splenomegaly</td>
<td>12–20 cm</td>
</tr>
<tr>
<td>Massive splenomegaly</td>
<td>21–30 cm</td>
</tr>
<tr>
<td>Megaspleen</td>
<td>&gt; 30 cm</td>
</tr>
</tbody>
</table>

*Spleen length is defined as interpole length, measured along a straight line connecting the two poles.
Laparoscopic Splenectomy

• Patient Positioning
  ▪ Usually Lateral
  ▪ Anterior approach for large spleens (>25-30cm)

• Trocar placement

• Search for accessory spleens
  ▪ Resect prior to splenectomy – difficult to locate following splenectomy
  ▪ Wash out and recover splenic fragments after splenectomy if necessary