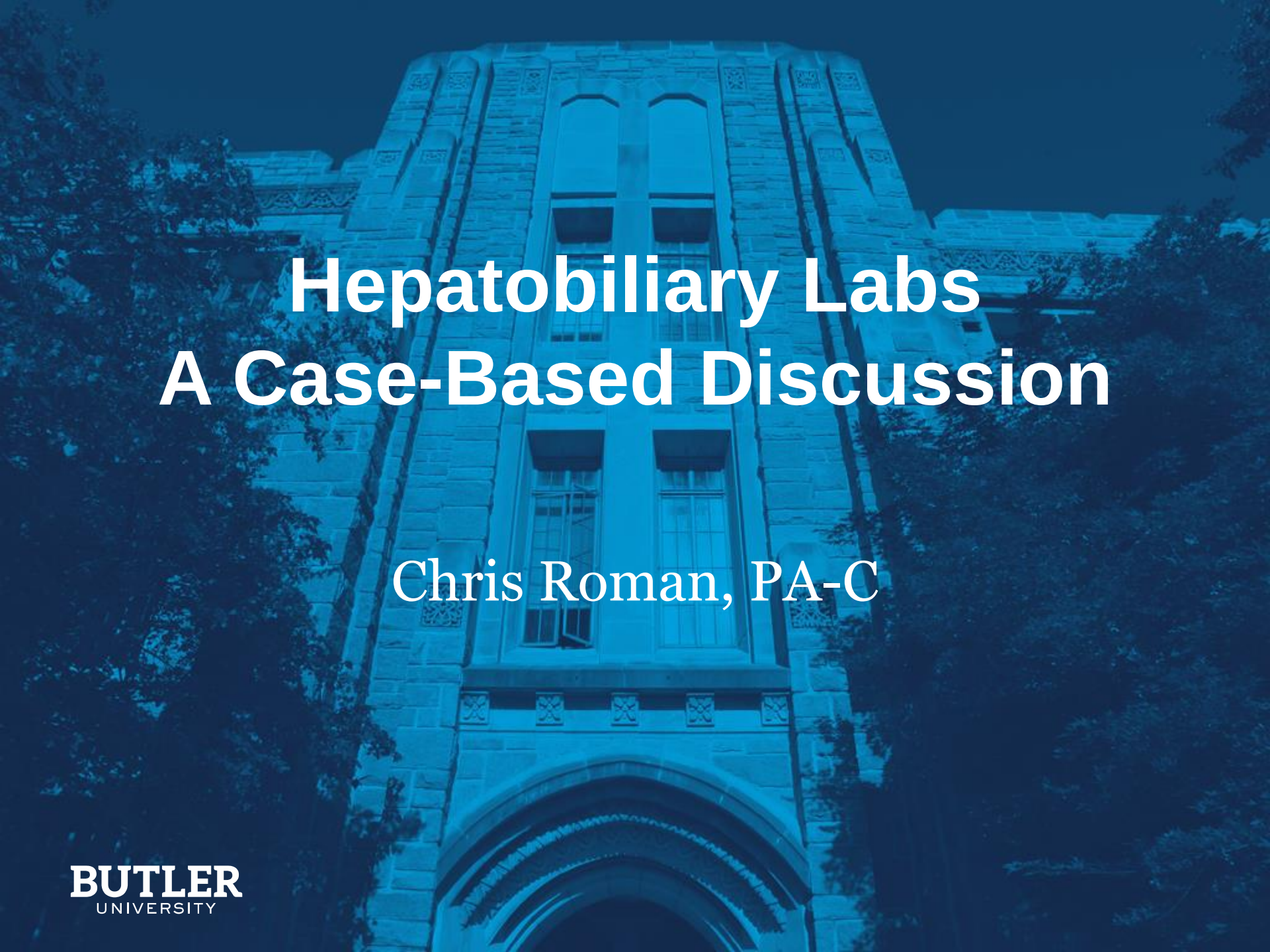

Hepatobiliary Labs
A Case-Based Discussion

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Case 1

- 43yo female with cc of “severe acid reflux”
  - 2 weeks of intermittent burning abd pain
    - No relief with TUMS
  - ↓ appetite
  - Dark urine
  - New onset generalized pruritus

Questions

1. What causes the pruritus in cases of hyperbilirubinemia?
   a. Basophil stimulation
   b. Bile salt deposition
   c. Histamine overproduction
   d. Hyperactive macrophages

2. Where is the first place where icterus may appear?
   a. Sclera
   b. Sublingual
   c. Subungal

Case 1

- She is indeed icteric, with modest RUQ discomfort
  - Also uncomfortable through epigastrum and LUQ
- Nonfasting labs are here:

CMP	RESULT	REF RANGE
Gluc	105 mEq/L	65-99
Na	139 mEq/L	135-145
K	4.9 mEq/L	3.5-5.0
Cl	100 mEq/L	97-108
CO2	22 mEq/L	20-32
Ca	9.3 mg/dL	8.7-10.2
Cr	0.9 mg/dL	0.75-1.27
BUN	7 mg/dL	6-24
GFR	73 mL/min/1.73 m^2	>59
PROT	7.7	6.0-8.5
ALBUMIN	4.1	3.5-5.5
BILI	3.6	0-1.2
ALK PHOS	247	25-150
AST	106	0-40
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3. What do you is more likely to have caused this pattern of labs?

a. Cholestatic/gallbladder issue
b. Hepatocellular damage
c. Could be either
d. Unsure

Case 1

Cholestatic

- Alkaline Phosphatase synthesis increases with obstruction
  - Biliary dz
  - Obstruction (tumor)
  - Cholestasis

Hepatocellular

- Direct damage/lysis of hepatocytes
  - AST and ALT are intracellular enzymes
    - Released into blood with cell death

Case 1

4. At this point, are you more likely to pursue
    a. Additional labs
    b. Imaging

- US shows mild bile duct dilation but no mass or stones
- What are you most worried about?
  - Answer in chat
- What is your next move?
  - Answer in chat

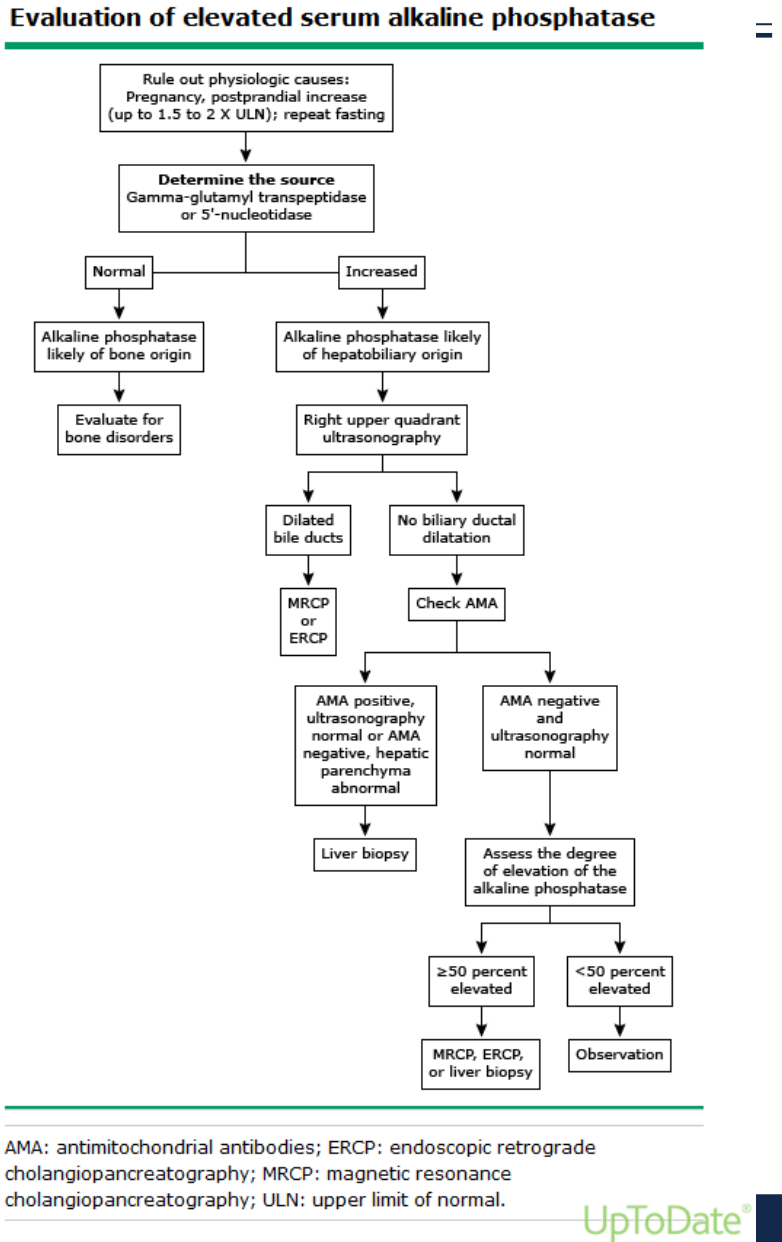
Case 1

- Clinical course
  - Admitted
  - CT unrevealing
  - HIDA
    - Nonvisualized biliary tree after 90m
      - Obstruction
  - MRCP
    - No mass, stones
- Diagnosis
  - Drug-induced cholestasis
    - Recent Amox-Clav for respiratory infection
- Outcome
  - Slow recovery
  - Don’t give her penicillins

Alkaline Phosphatase

- Produced with
  - Disturbed metabolism
    - Liver
  - Stimulated growth
    - Placenta, growing bone

1. Elevated levels of GGT in a pt with elevated Alk Phos suggests:
   a. Bony dz
   b. Hepatobiliary dz
   c. Unsure



Low Alkaline Phosphatase

- Not usually a big deal
- Can be present in
  - Hypothyroidism
  - Pernicious anemia
  - Zinc deficiency
  - Congenital hypophosphatemia

Case 2

- Herman is a 50yo male taking 80mg daily of atorvastatin
  - Increased from 20mg after he received a diagnosis of stable angina from his cardiologist
- All LFTs were WNL when this change was made 3 months ago

Case 2 – Asymptomatic labs drawn yesterday

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Gluc	105 mEq/L	65-99
Na	139 mEq/L	135-145
K	4.9 mEq/L	3.5-5.0
Cl	100 mEq/L	97-108
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ALBUMIN	4.8	3.5-5.5
BILI	0.5	0-1.2
ALK PHOS	110	25-150
AST	80	0-40
ALT	175	0-55

1. Is routine monitoring of LFTs needed in pts on statins?
   a. Yes
   b. No
   c. Unsure

Case 2

CMP	RESULT	REF RANGE
Gluc	105 mEq/L	65-99
Na	139 mEq/L	135-145
K	4.9 mEq/L	3.5-5.0
Cl	100 mEq/L	97-108
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2. At what level of LFT elevation would you adjust statin therapy?

a. 1.5x
b. 2x
c. 2.5x
d. 3x
e. 4x

What adjustment would you make?

-Answer in chat.

Statin-Induced Liver Damage

- Uncommon
  - 0.5-3.0% have persistent elevations
    - Usually in 1st 3 months
    - Dose dependent
- More severe liver injury is more rare
  - Drug interactions more common than purely statin-induced dz

- Recommendations
  - Check baseline when starting statin therapy
    - Not needed for dose Δ
  - Routine monitoring of LFTs not needed (FDA)
    - Ditto for CK levels
- Change Rx or lower dose in patients with ALT > 3x normal on 2 occasions

Case 3

- 44yo male with abnormal LFTs drawn 6 months ago.
- His serum aminotransferase levels were two times normal at that time and remain unchanged after repeat testing.
- On further questioning, he denies regular alcohol use but states that he used to inject heroin.
- Currently, he reports some fatigue but says he feels well otherwise.

Case 3

- You order serologic testing, which reveals
- HBsAg-positive
- Anti-HBs-negative
- Anti-HBc-positive IgG
- Anti-HCV-negative

1. What is his diagnosis?
   a. Active HBV
   b. Past HBV
   c. SP immunization for HBV
2. What percentage of pts with acute HBV develop chronic infection?

   Answer in chat

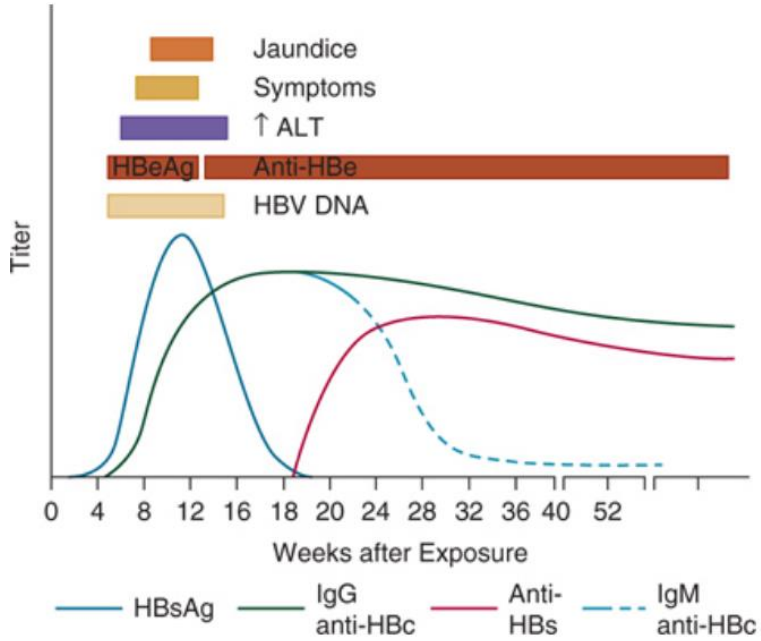
Case 3 - HBV

- DNA virus transmitted via bodily fluids
  - Blood, sexual contact most common
- Onset often insidious
  - Incubation 5 wks-6mos

- COMMON
  - ~80K new cases in US per year
    - Declining w vaccination!
  - >2M carriers in US
    - Asians/Pacific Islanders are 50% of chronic HBV cases (only 5% of pop.)
  - 5000 US deaths per annum
  - Globally, carrier rate as high as 33%
    - Can be asymptomatic!

Case 3 - HBV

- Vaccine very effective!
- About 5 of pts exposed will develop chronic dz
  - Of these
    - 2/3 have benign course
    - 1/3 develops cirrhosis
- Viral load needed to determine level of infection



Case 3 - HBV

3. What other virus(es) should you test for?

Answer in chat ☺

- HBV is not curable
  - Not everyone gets Rx
- Rx intended to
  - Prevent progression
  - Minimize further damage
- Goals
  - Clear viremia
  - Loss of detectable antigen
  - Seroconversation to anti-HBV antibodies

Case 4 - Hyperbilirubinemia

- CMP drawn on a 19yo healthy female with fatigue
  - Normal except Total Bilirubin of 1.8mg/dl
    - RR 0.2-1.2mg/dl

1. What test do you call the lab and request they add?
   a. D-dimer
   b. Direct bilirubin
   c. GGT
   d. LDH

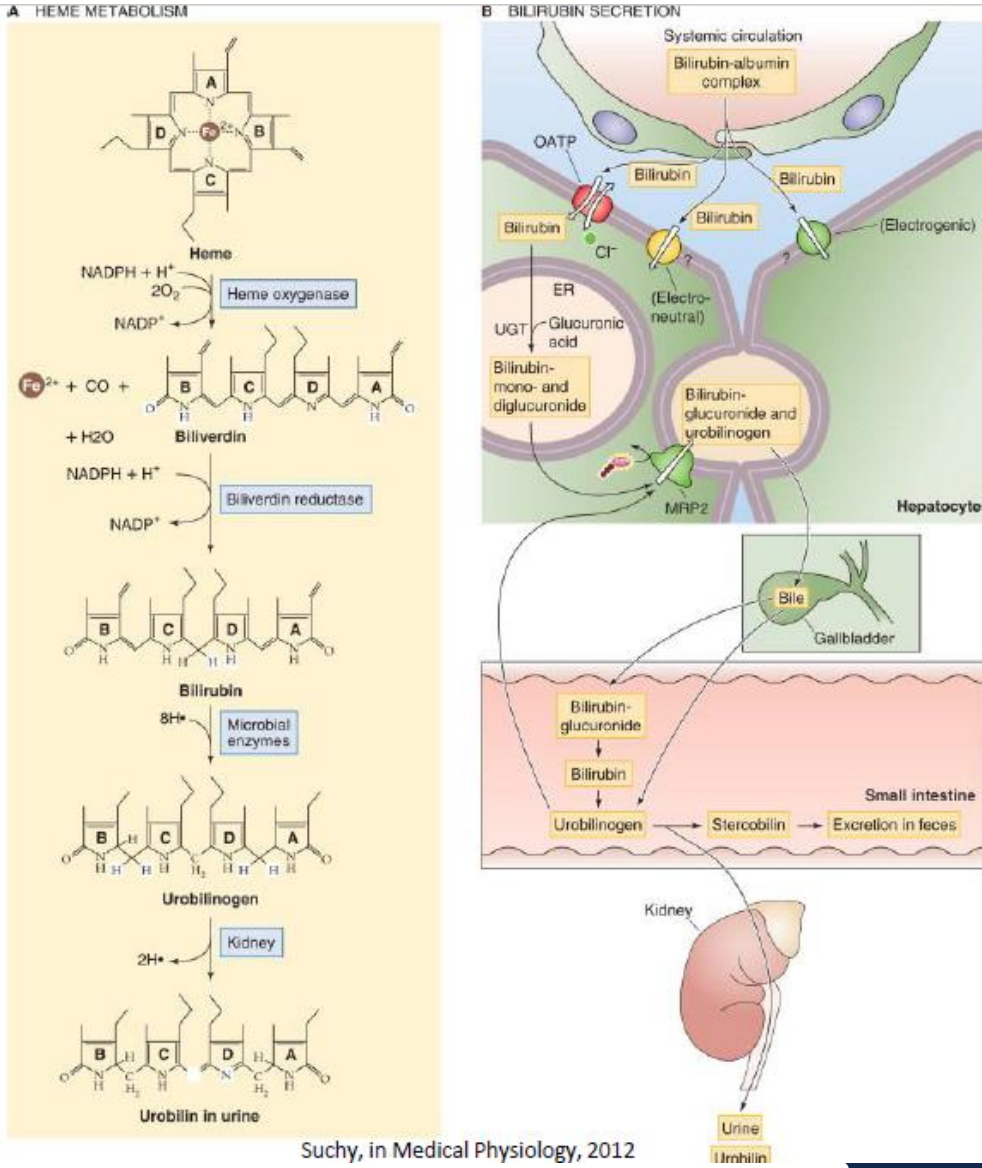
Case 4 - Hyperbilirubinemia

- CMP drawn on a 19yo healthy female with fatigue
  - Normal except Total Bilirubin of 1.8mg/dl
    - RR 0.2-1.2mg/dl
- Direct bilirubin is 0.6mg/dl (0.0-0.5)

2. This patient will likely appear jaundiced
   a. True
   b. False
3. This lab abnormality is most likely due to an issue with:
   a. Blood
   b. Gallbladder
   c. Kidney
   d. Liver

Case 4 - Hyperbilirubinemia

- Bilirubin metabolism
  - Conjugated in liver
  - "Indirect" is pre-hepatic, i.e., from RBC lysis
  - "Direct" is post-hepatic



Suchy, in Medical Physiology, 2012

Hemolysis

- Hemolysis=premature destruction of erythrocytes (RBC)
- Anemia can arise if hematopoiesis cannot keep up with RBC loss
  - Reticulocytosis and premature ("nucleated") RBC with release of immature RBCs
- Variety of causes
  - Genetic
    - e.g., G6PD deficiency
  - Immune disorder
    - e.g., autoimmune hemolytic anemia
  - Chemical/drug reaction
    - e.g., ribavirin
  - Physical damage to RBC
    - e.g., prosthetic heart valve
  - Infections
    - e.g., CMV

Questions?