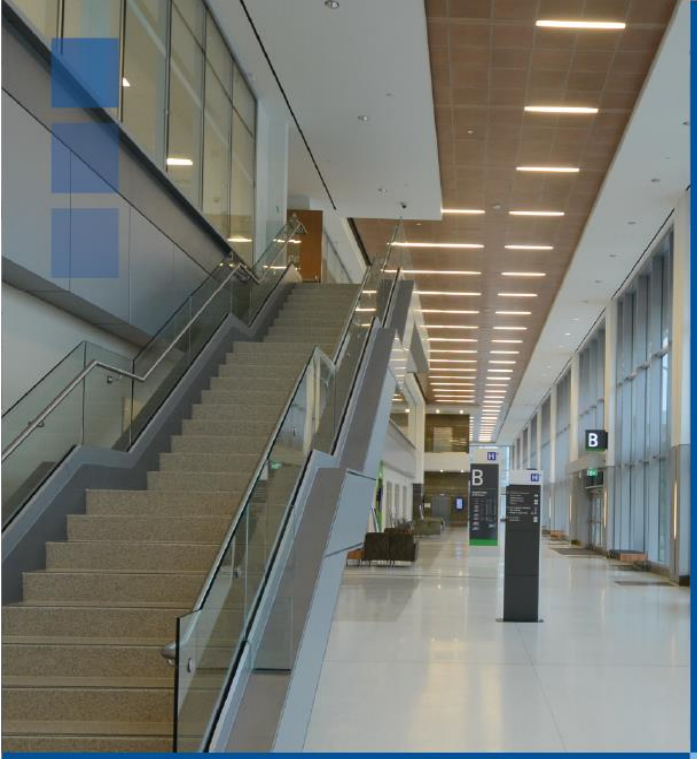


Dealing with the Abnormal CBC:

An approach to anemia and thrombocytopenia for the family physician

Vighnesh Bharath
Dept. of Hematology/Oncology
HRH 21st Annual Clinical Day
Dec. 8, 2018

HUMBER RIVER HOSPITAL

Patient Care Reinvented.

Disclosures

- No conflicts of interest
- No relationships with commercial/financial interests

Objectives

- Develop an approach to the assessment of anemia and thrombocytopenia
- Understand the various common (and dangerous) causes of anemia and thrombocytopenia
- Recognize which tests/investigations are important in the initial evaluation of an anemic or thrombocytopenic patient
- Outline an approach to the management of various important causes of anemia, including iron deficiency and hemolytic anemia
- Formulate an approach to the management of common causes of thrombocytopenia, particularly immune thrombocytopenia (ITP)
- Identify the “red flags” in an anemic or thrombocytopenic patient (i.e. hematologic emergencies), thus knowing when to refer to a hematologist

Question 1

- A 35F presents with an incidentally discovered Hb 105. MCV is 65, ferritin 7. She reports heavy menstrual bleeding. She has been on ferrous gluconate 300mg PO TID for 6 months, and is compliant. As usual, you wonder about absorption issues and bleeding elsewhere. For now, how would you manage her iron?

- A) Double the dose of ferrous gluconate
- B) Switch to feramax 150mg PO daily
- C) Give venofer 300mg IV x3 doses, each a week apart
- D) No change; reassess in 3 months

Question 2

- A 47F presents with a Hb of 84. She has a history of well-controlled lupus on plaquenil. She is asymptomatic but exam reveals mild jaundice. Given the results below, which of the following is NOT a possible explanation for her anemia?
  - MCV 102
  - Cr 154, TSH normal, SPEP normal
  - Ferritin 250 (high), TIBC low, transferrin-sat normal
  - Retics 150 (high), indirect bili high, LDH high, haptoglobin low

- A) Lupus nephritis causing decreased epo production
- B) Warm autoimmune hemolytic anemia
- C) Anemia of chronic disease due to lupus
- D) Plaquenil causing a sideroblastic anemia

Question 3

- Which of the following mechanisms is NOT a typical cause of thrombocytopenia?

- A) Renal disease causing uremic platelet dysfunction
- B) Severe B12 deficiency leading to a thrombotic microangiopathy picture
- C) Liver disease causing decreased thrombopoietin production and hypersplenism
- D) Antiplatelet antibodies binding to platelets, causing immune destruction in the spleen

Question 4

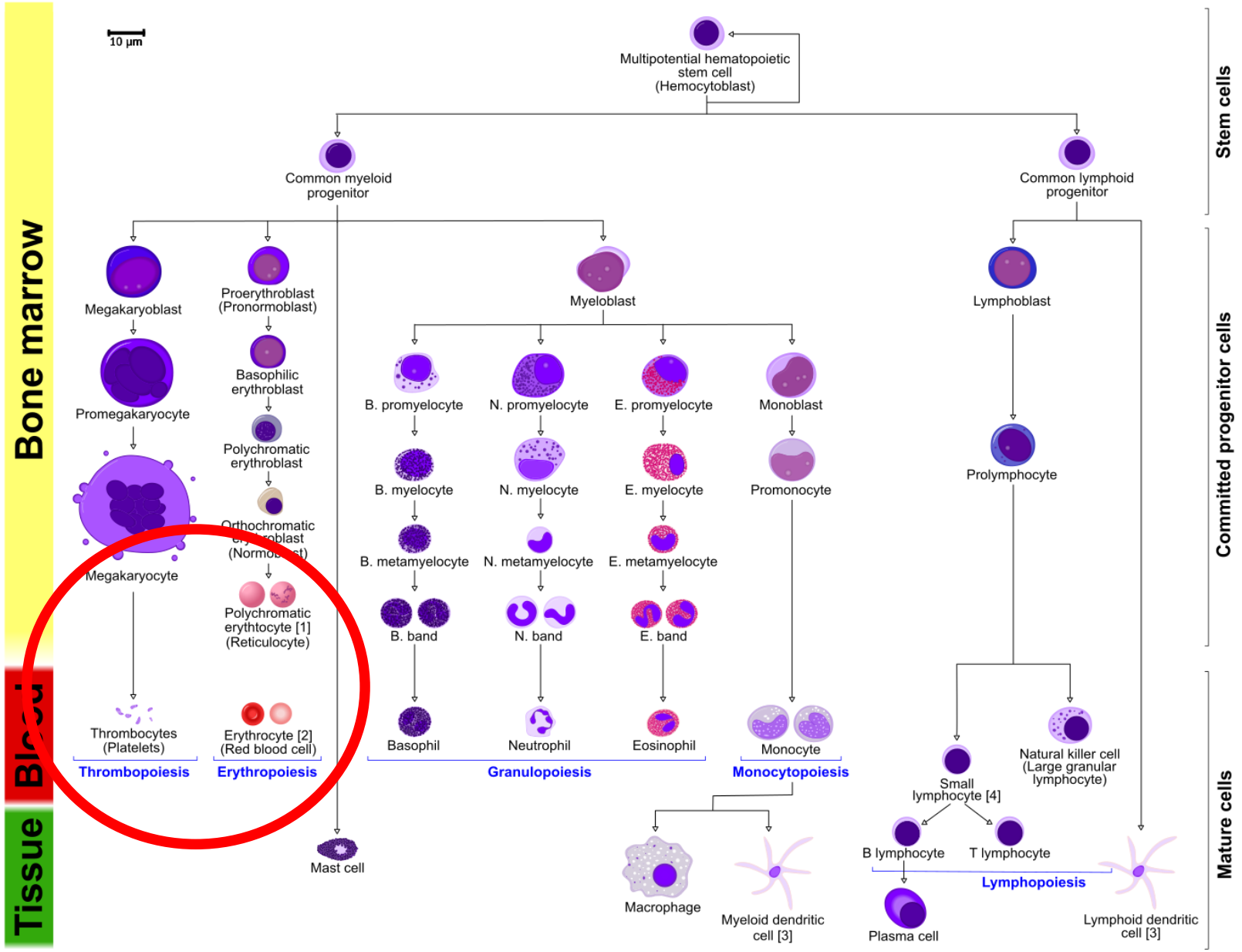
- A 56M presents with an isolated plt count of 25. He is otherwise healthy and on no regular meds. CBC, Cr, liver enzymes, B12/folate, ANA, and abdo U/S are all normal. You suspect ITP. He has petechiae on his legs bilaterally. No upcoming procedures. What is the most reasonable next step?

- A) Check Hep C, HIV +/- H pylori. If negative, treat as ITP with steroids as 1st line.
- B) Admit to hospital. Give IVIg 1g/kg x2 doses, 2 days apart.
- C) Check Hep C, HIV. Transfuse 1 adult dose plts. Treat with IVIg and steroids.
- D) Check Hep C, HIV +/- H pylori. Watch and wait. No indication for acute treatment.

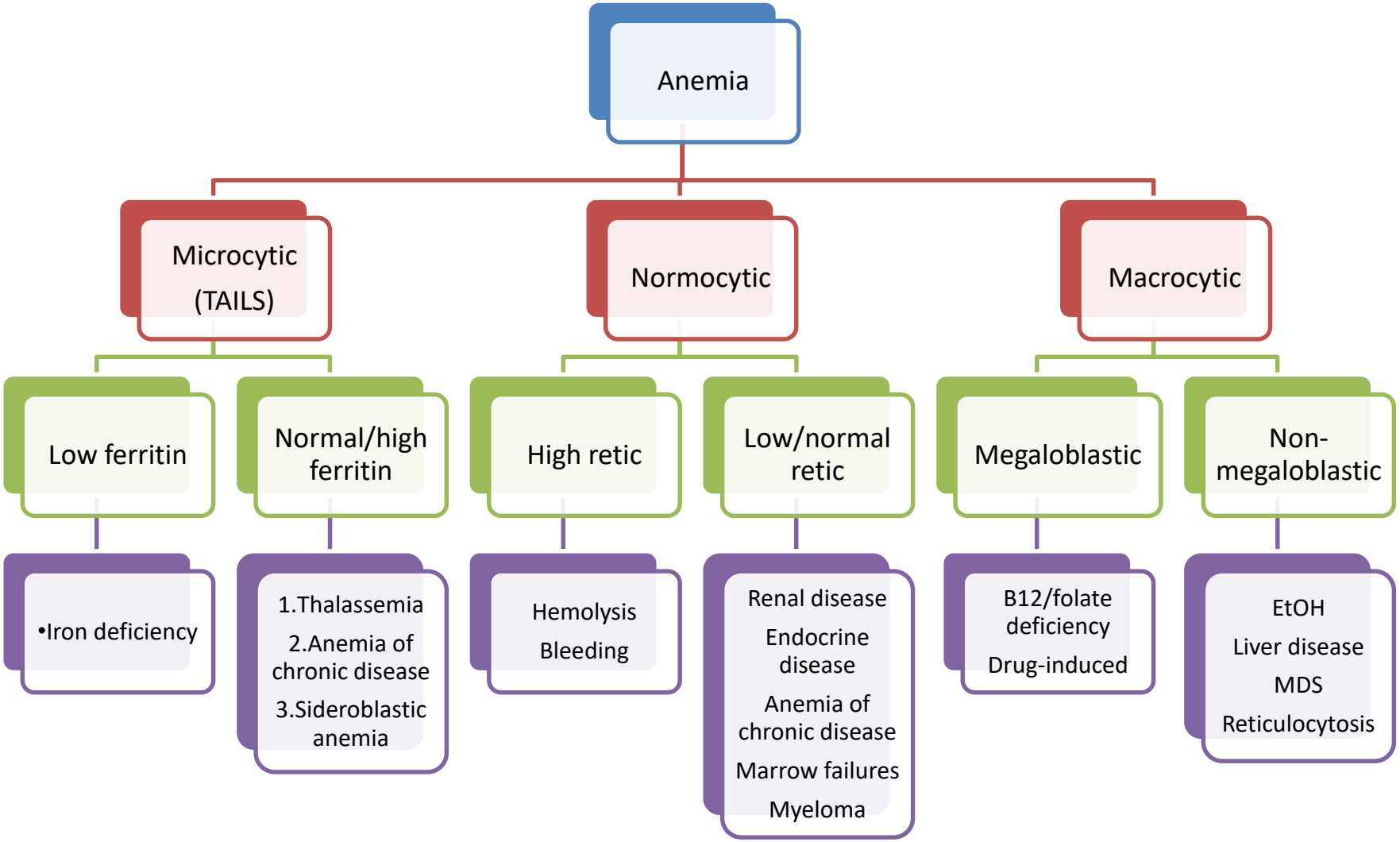
Agenda

- Approach to and evaluation of anemia
- Investigation and management of anemia
- Causes of anemia
  - Iron deficiency
  - Thalassemia
  - Anemia of chronic disease
  - Hemolytic anemia
  - Anemia of renal failure
  - B12 deficiency
  - Miscellaneous anemias
  - Pregnancy
- Anemia red flags
- Approach to and evaluation of thrombocytopenia
- Investigation and management of thrombocytopenia
- Causes of thrombocytopenia
  - ITP
  - TMA
  - Pregnancy
- Thrombocytopenia red flags

Hematopoiesis



Approach to Anemia



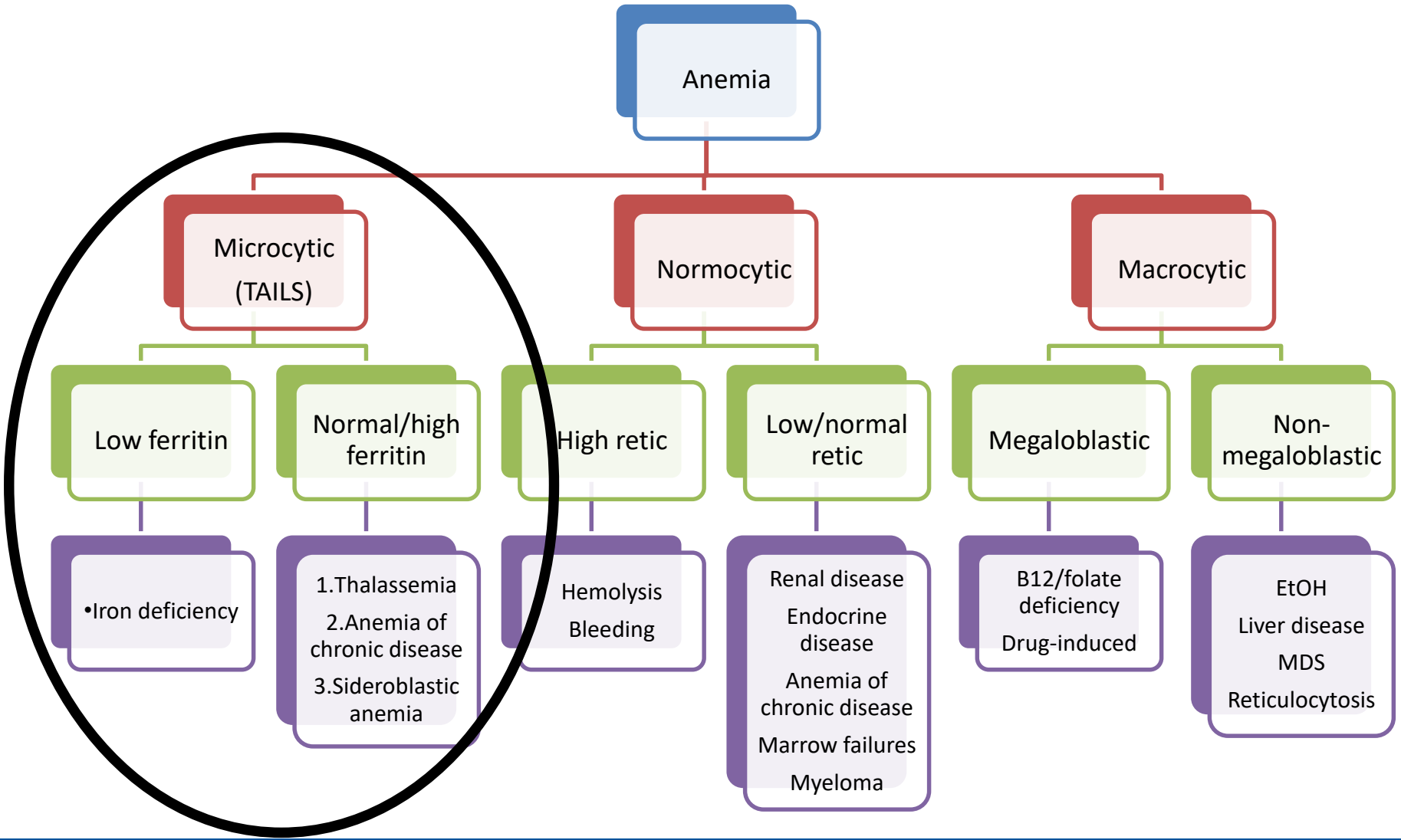
Investigation of Anemia

- What is the MCV?
- Microcytic → ferritin, TIBC → +/- HBEP
- Normocytic → retic → bleeding vs. hemolytic w/u OR Cr, SPEP, TSH, ESR/CRP +/- bone marrow biopsy
- Macrocytic → retic, B12, folate, LFTs (+ any drugs?) → +/- bone marrow biopsy
- If normo/macrocytic + remains unexplained (esp. significant <100 and transfusion-dependent) → bone marrow biopsy

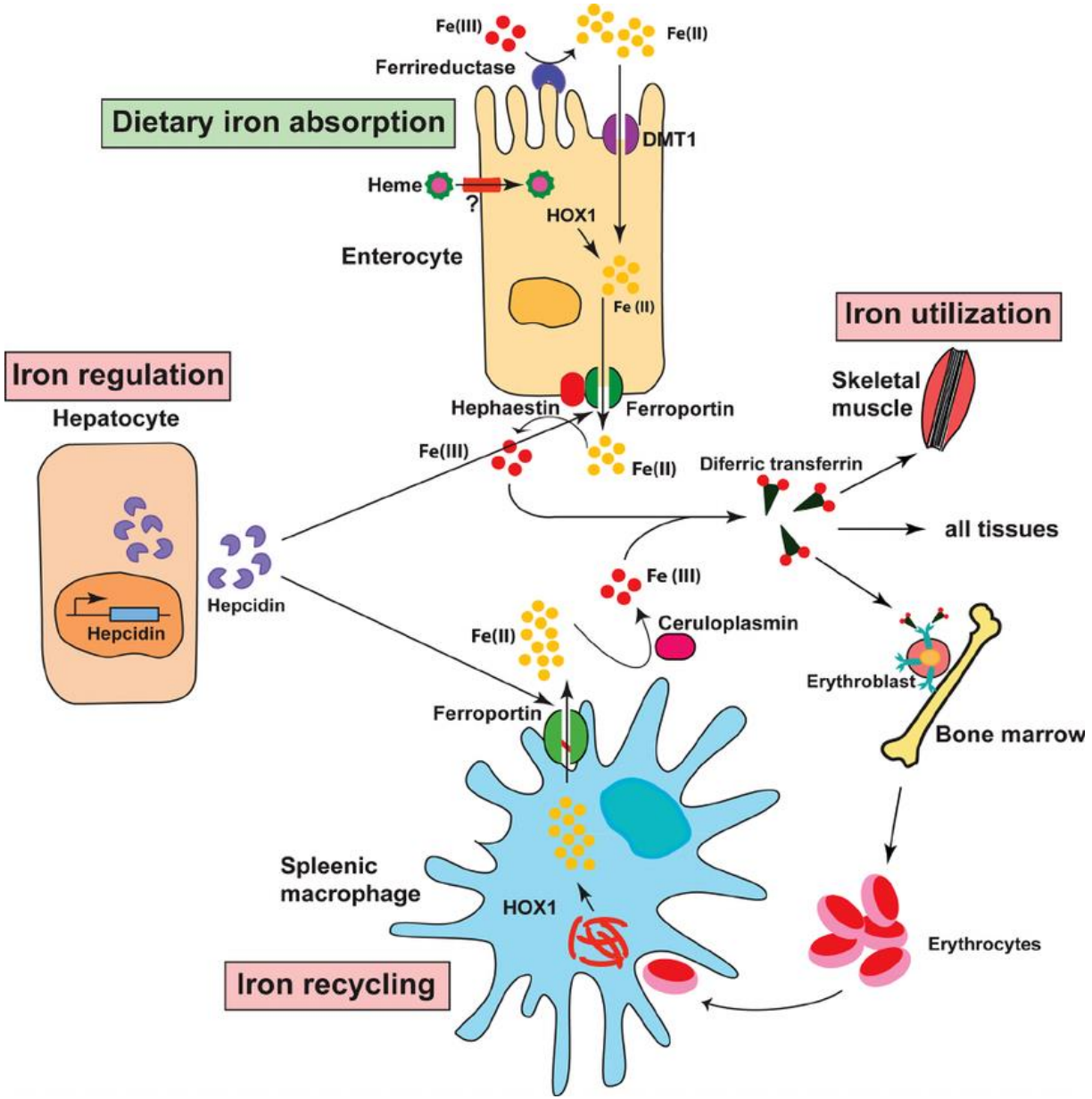
General Management of Anemia

- Manage the underlying cause!
- Transfusion practices (Bloody Easy 4)
  - Generally if Hb <60-70 (post-op: Hb <70-80)
  - Hb <70-90 w/ CV disease or symptoms (ACS, CAD, impaired oxygen delivery)
  - Likely NO if Hb >90
- Iron (PO, IV)
- Erythropoietin – kidney disease, malignancy on chemo, MDS

Approach to Anemia



Iron Metabolism



Iron Deficiency

- Daily iron requirement is 10-20mg from diet and 20-25mg recycled (non-heme 10% absorbed, heme a bit more)
- Etiology
  - Not enough in: diet, malabsorption (duodenal disease, congenital)
  - Too much out: bleeding! – usually GI, menstrual
  - Need more: rapid growth (peds), epo, pregnancy
- Clinical
  - Symptoms of anemia
  - Pica (pagophagia [ice eating], etc.), restless legs
  - Stomatitis, angular chelitis, glossitis, Plummer-Vinson (dysphagia due to esophageal webs, glossitis, IDA)
  - Koilonychia, hair loss

Iron Deficiency

- Investigations
  - Ferritin (<15-30 most specific), low serum iron, low t-sat, high TIBC
  - Low retics, high RDW, thrombocytosis
  - Smear: microcytic, hypochromic
  - Look for cause: endoscopy, celiac, gyne consult
- Order of events
  - Low iron in BM/liver/spleen (low ferritin) → stores go lower (high TIBC, low t-sat) → iron-restricted erythropoiesis (microcytic) → further depletion (anemia)

Iron Deficiency

- Management
  - Need ~10 mg/d elemental iron in adults (replacement dose usually 100-200mg/d bc ***only absorb 10%***)
  - 300-325mg tabs of ferrous gluconate/sulfate/fumarate have elemental iron of ~30/60/90mg and are taken TID/TID/BID, respectively
  - Iron polysaccharides (feramax; ferric 3+) – 150mg tab = 150mg elemental iron
  - Heme iron polypeptide (ex. proferrin – 11mg, elemental iron 11mg); absorbed better through unknown GI mechanism
  - IV iron: venofer; usually 300mg weekly x3 doses
    - Typically if not responding to >/=3 months of 2 orals
- Side effects: N/V, constipation, epigastric discomfort, darker stools (25% cannot tolerate)
  - Side effects: sulfate > gluconate/fumarate > feramax
  - IV: arthralgias/myalgias
    - Contraindications: anaphylaxis/hypersensitivity, iron overload, 1st trimester pregnancy (safety NYD), decompensated liver disease, active infection

Iron Deficiency

Commonly Used Iron Replacement Therapies	Dose mg	Elemental mg
Ferrous gluconate	300	35
Ferrous sulfate	300	60
Ferrous fumarate (Palafer®)	300	100
Polysaccharide-iron complex (FeraMax®)	150	150
Polysaccharide-iron complex (Triferex®)	150	150
Proferrin	398	11

Iron Deficiency

- Instructions
  - Better absorption w/ empty stomach, but better tolerated w/ food
  - Ascorbic acid (vit C) can help absorption (take w/ orange juice)
    - Acidic environment prevents conversion to ferric (Fe3+) which is not as readily absorbed as ferrous (Fe2+)
  - Absorption decreased by: tannins (tea), antacids, calcium, bran, whole grains (if taken concurrently)
- Response
  - Retics within 7-10d
  - Hb response within 2 weeks
  - Ferritin is last; only once additional iron repletes body stores
  - Continue treatment for 3 months after Hb normalizes and underlying cause fixed; to replete stores fully
  - Failure to respond? → consider bleeding, poor compliance, poor absorption (celiac, H pylori, atrophic gastritis, etc.), inadequate replacement dosing

Thalassemia

- African, Mediterranean, Southeast Asian
- Quantitatively decreased synthesis of structurally normal globins
  - Alpha (DNA deletions) and beta (point mutations)
- Decreased production of one globin causes excess of other globin (imbalance) → excess unpaired globin is unstable and precipitates → oxidative damage to RBCs and precursors in marrow → death/hemolysis → results in severe microcytic anemia → compensation w/ erythroid expansion

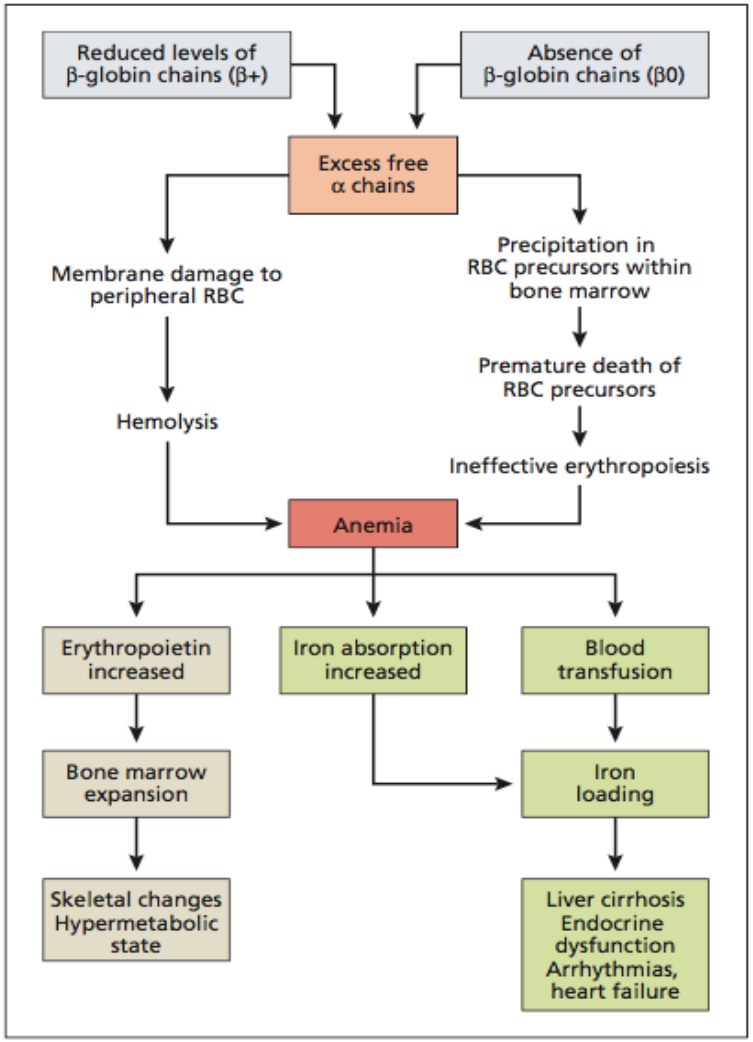


Figure 7-3 Pathophysiology of β-thalassemia. Effects of excess production of free α-globin chains in β-thalassemia. Adapted with permission from Viprakasit V and Origa R. In: *Guidelines for the Management of Transfusion Dependent Thalassaemia (TDT)*. 3rd ed. Nicosia, Cyprus: Thalassaemia International Federation; 2014.

Thalassemia

CLASSIFICATION OF β THALASSEMIA(genetic)

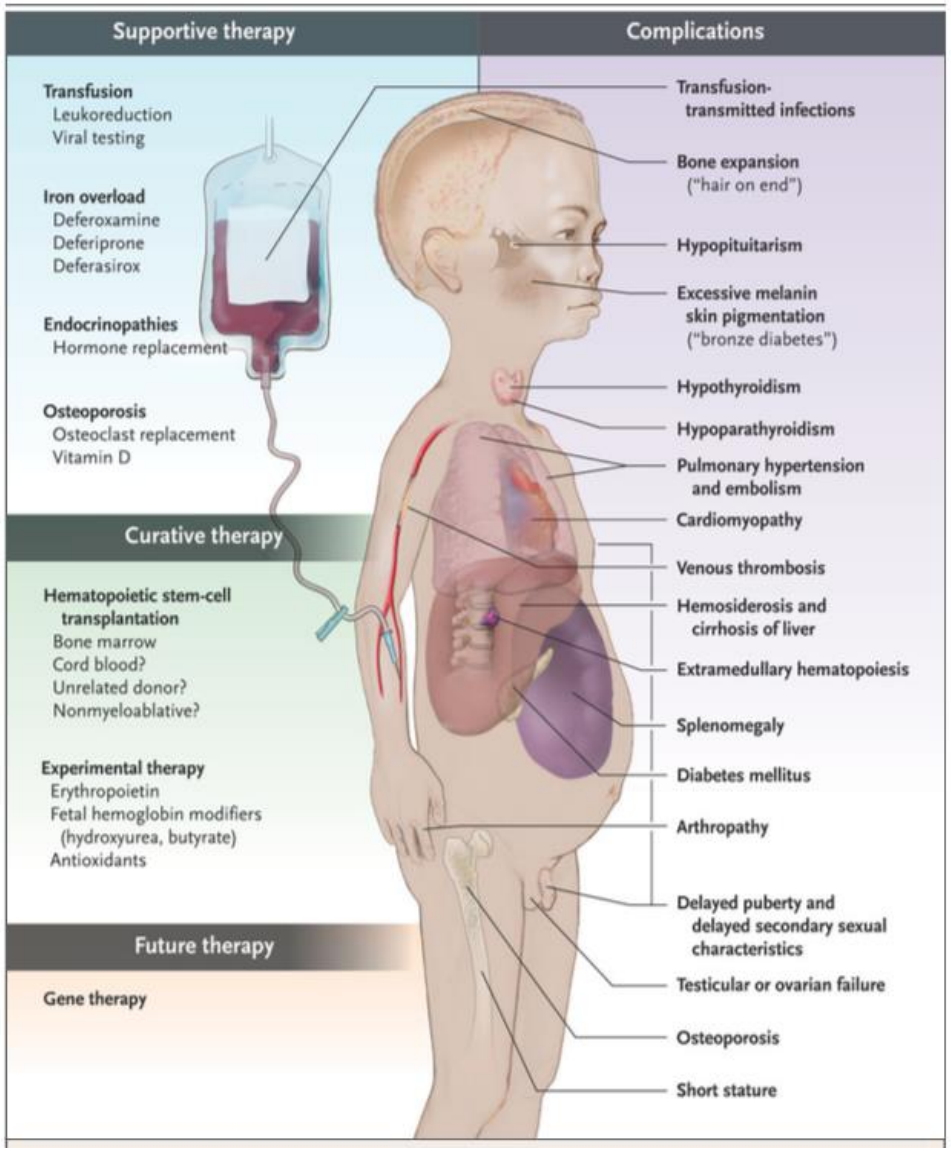
CLASSIFICATION	GENOTYPE	CLINICAL SEVERITY
β thal minor/trait	β/β+, β/β0	Silent
β thal intermedia	β+ /β+, β+/β0	Moderate
β thal major	β0/ β0	Severe

ALPHA THALASSEMIA - CLASSIFICATION

CLINICAL CLASSIFICATION	GENOTYPE	NO. OF GENES PRESENT
Silent carrier	aa/- a	3 genes
a thalassemia trait	- a/- a or aa/- -	2 genes
Hemoglobin H disease	-a/- -	1 gene
Hb Barts / Hydrops fetalis	- -/- -	0 genes

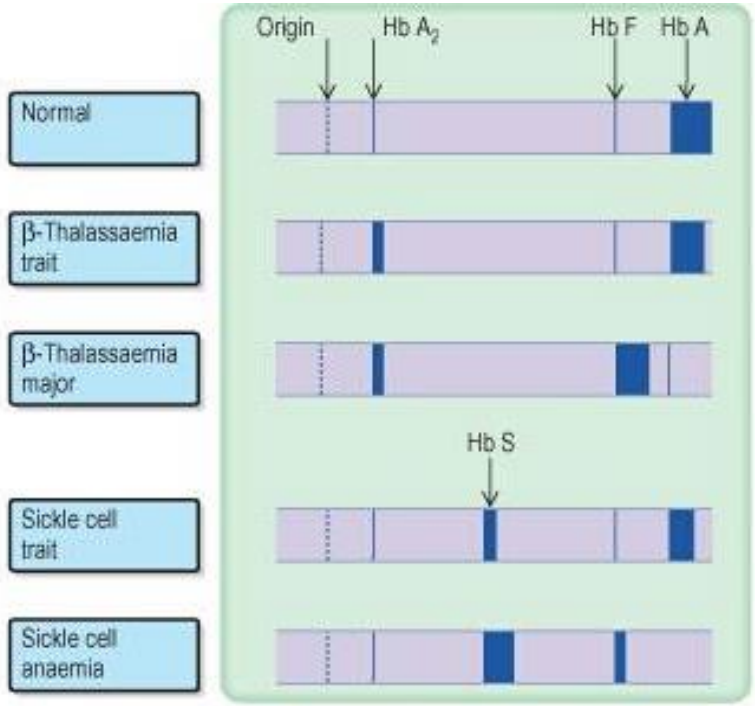
Thalassemia

- Usually asymptomatic if minor/carrier/trait
- Clinical (thal major)
  - ANEMIA + HEMOLYSIS
  - ERYTHROID EXPANSION: skeletal, splenomegaly, increased GI iron absorption
  - IRON OVERLOAD (disease + transfusions): multi-organ damage



Thalassemia

- Investigations
  - Hb ranges from 90-normal
  - Smear: microcytic/hypochromic RBCs, target cells, basophilic stippling
  - MCV <70, retics mildly elevated
  - Mentzer index: MCV/RBC<13 (>15 suggests IDA)
  - HbEP: variable A (absent in homozygous B0), A2 elevated >3.5 (usually 4-7%), F increased
    - Usually NORMAL in alpha thalassemia → genetic testing



Thalassemia

- Management
  - General: genetic counseling / family planning, avoid iron supplementation, supportive care / treat complications
  - Beta
    - Trait: nothing
    - Intermedia/major: regular transfusions, iron chelation
  - Alpha
    - 1-2 gene deletion: nothing
    - 3-gene deletion (Hb H disease): same as beta intermedia
    - Hydrops fetalis: intrauterine transfusions

Anemia of Chronic Disease

- Etiology: malignancy, autoimmune, chronic infection, prolonged illness
- Pathophysiology
  - Cytokines reduce erythroid proliferation, decrease epo, decrease RBC survival
  - IL-6 increases hepcidin → inhibits ferroportin → inhibits dietary iron absorption and macrophage iron recycling → functional iron deficiency
- Lab
  - Hb 70-110, low retics, normochromic normocytic anemia
  - May become more severe and hypochromic/microcytic over time
  - Low-normal serum iron, TIBC low-normal, t-sat low, ferritin normal-high
  - Elevated ESR/CRP
- Management
  - Treat underlying disorder
  - Epo +/- iron may be beneficial in some patients (epo<500)

IDA vs. AoCD

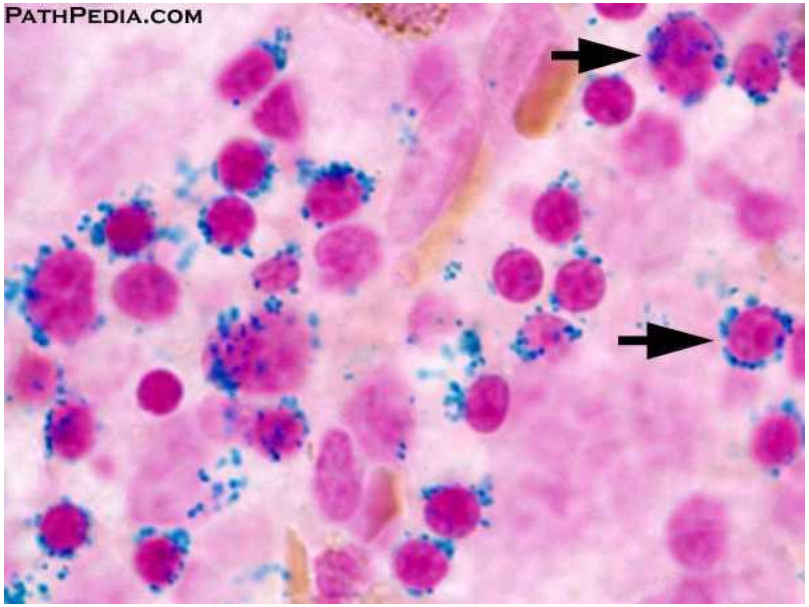
Table 3. Serum Levels That Differentiate Anemia of Chronic Disease from Iron-Deficiency Anemia.*

Variable	Anemia of Chronic Disease	Iron-Deficiency Anemia	Both Conditions†
Iron	Reduced	Reduced	Reduced
Transferrin	Reduced to normal	Increased	Reduced
Transferrin saturation	Reduced	Reduced	Reduced
Ferritin	Normal to increased	Reduced	Reduced to normal
Soluble transferrin receptor	Normal	Increased	Normal to increased
Ratio of soluble transferrin receptor to log ferritin	Low (<1)	High (>2)	High (>2)
Cytokine levels	Increased	Normal	Increased

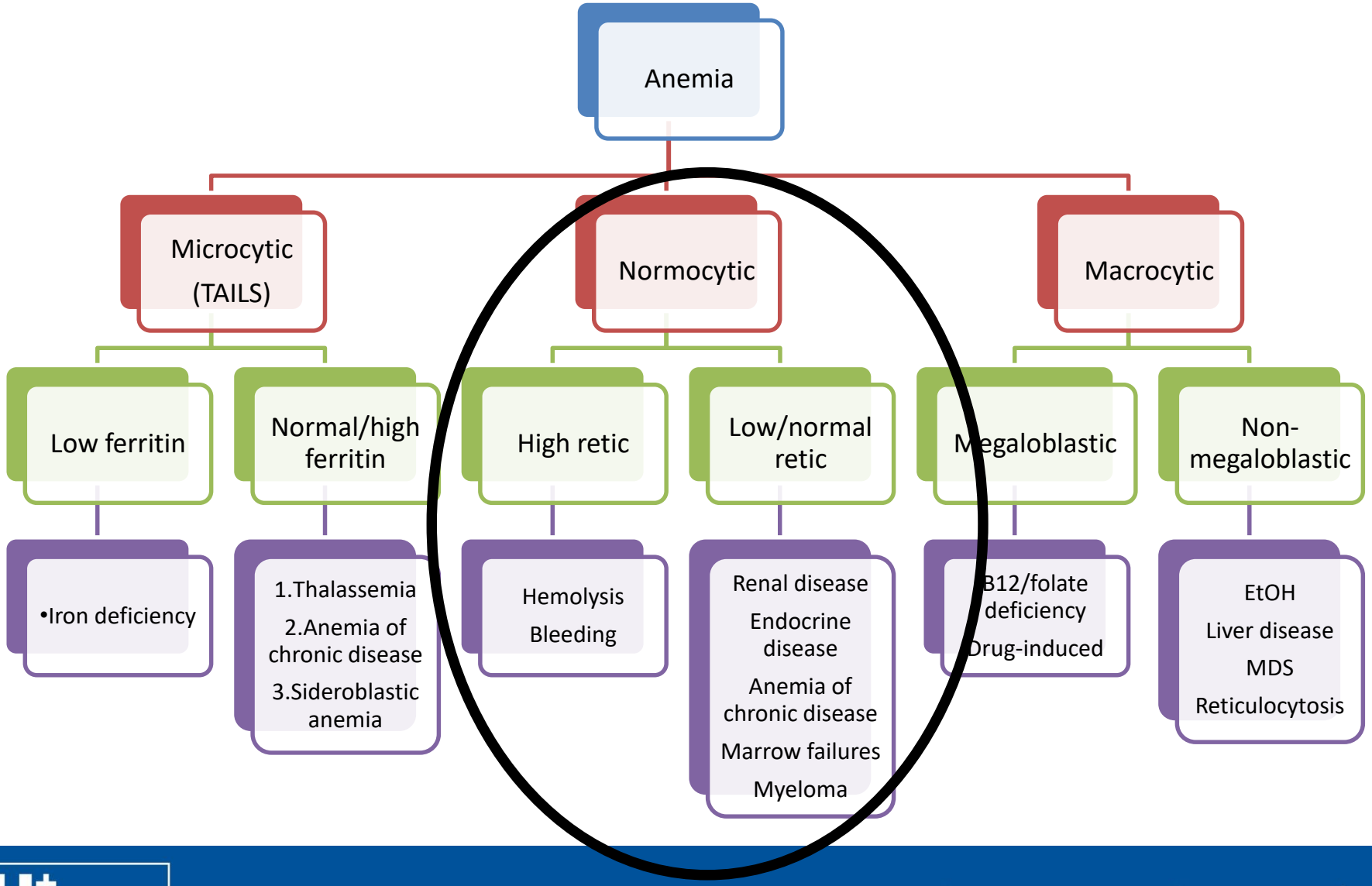
* Relative changes are given in relation to the respective normal values.
† Patients with both conditions include those with anemia of chronic disease and true iron deficiency.

Other Causes of Microcytic Anemia

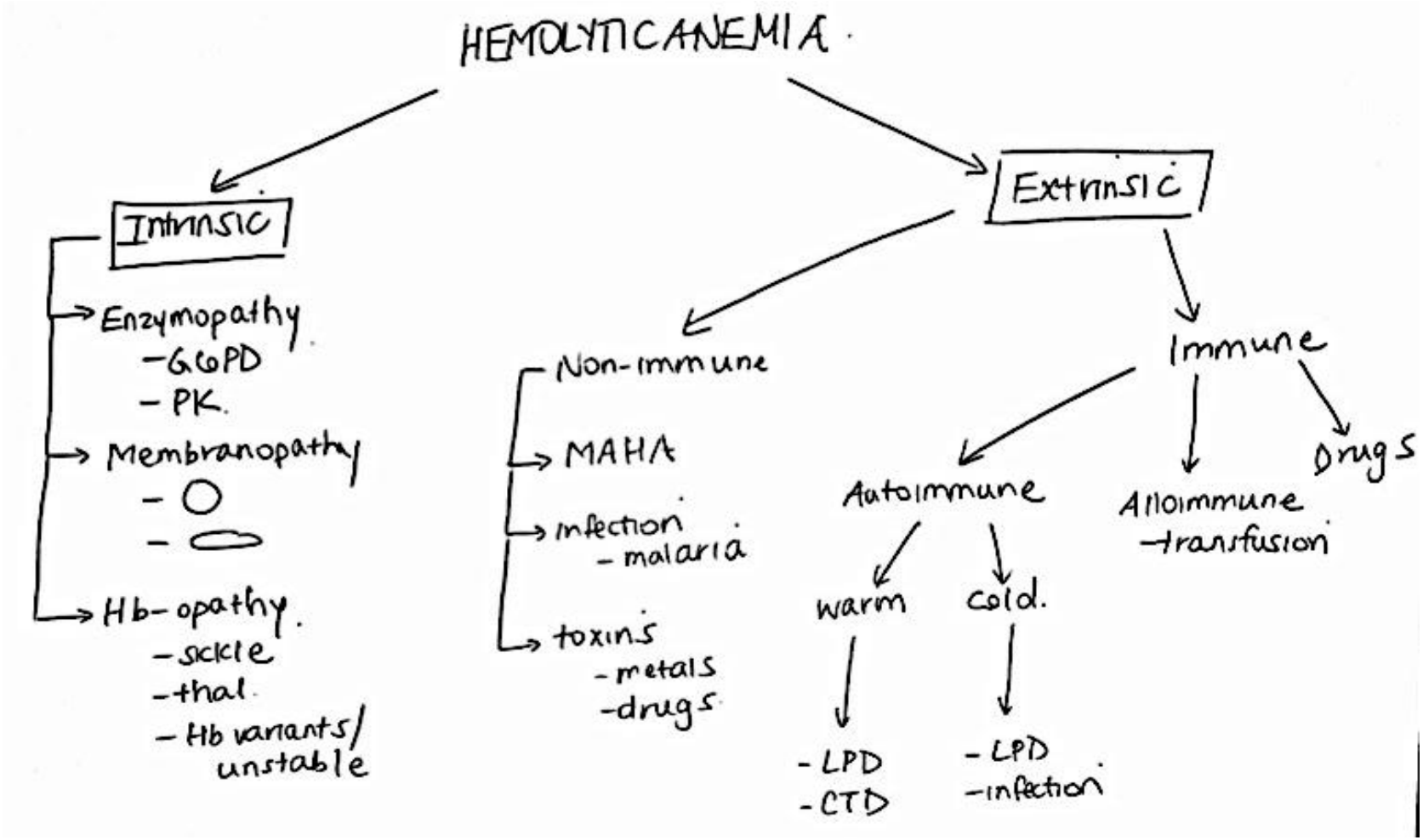
- Sideroblastic Anemia
  - Heterogenous group of congenital and acquired disorders w/ ring sideroblasts (erythroid precursors w/ excess mitochondrial iron that surrounds/rings nucleus)
  - Insufficient production of protoporphyrin to *utilize iron* delivered to erythroblasts
  - Etiology
    - Congenital (many)
    - Acquired: MDS, EtOH, drugs (isoniazid, linezolid), lead poisoning, copper deficiency
  - Treat underlying cause



Approach to Anemia



Approach to Hemolysis



WAIHA and CAIHA/CAD

- Clinical: anemia symptoms, jaundice, underlying cause, hepatosplenomegaly
- Lab: low Hb, high LDH, high indirect bili, high retic, low haptoglobin, DAT+
- Treat underlying cause!
- Warm AIHA
  - IgG autoantibodies bind RBC at 37 deg → extravascular hemolysis in spleen → spherocytes
  - DAT+ w/ IgG +/- C3
  - Tx: steroids → splenectomy → rituximab
- Cold AIHA / Cold agglutinin disease (CAD)
  - IgM autoantibodies bind RBC at <37 deg and fix complement → agglutination → intravascular hemolysis
  - DAT+ w/ C3, cold agglutinin screen, thermal amplitude, agglutination on smear
  - Tx: cold avoidance → rituximab

Anemia of Renal Failure

- Pathophysiology
  - Decreased epo, blood loss from dialysis or uremic plt dysfunction (although normal plt count), more iron utilization from epo tx
- Treatment (KDIGO 2012)
  - Epo (typically when Hb <100)– but replace iron first (if t-sat <=25% or ferritin <=200, non-dialysis)
  - Target Hb 110-120
  - Transfusions PRN
  - *Look for other causes of anemia* if unresponsive

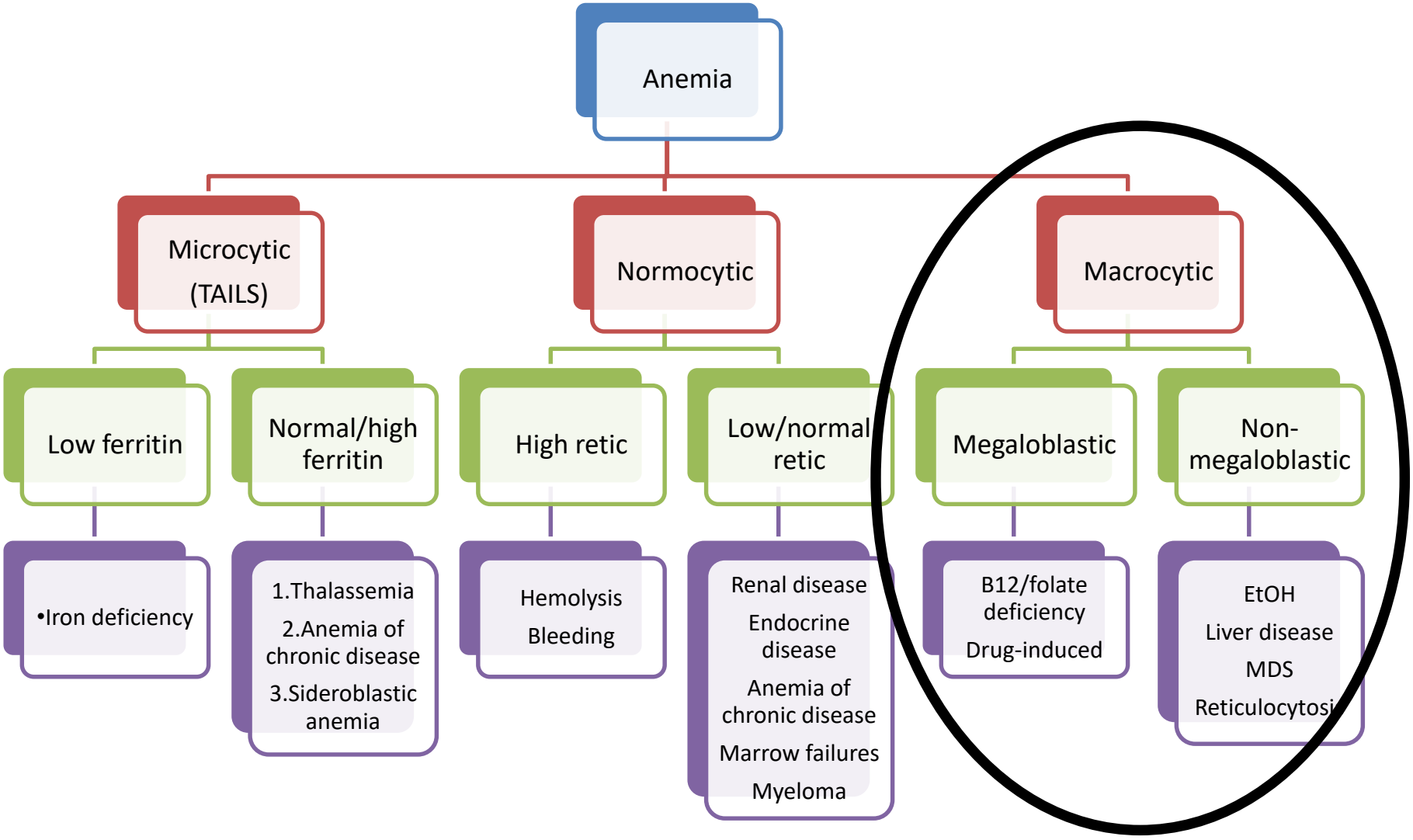
Other Causes of Normocytic Anemia

- Bleeding (esp. w/ unexplained iron deficiency)
- Anemia of chronic disease
- Marrow failure (underproduction due to malignancy, infiltration, infection, drugs, etc.)
  - Including multiple myeloma
- Endocrine disease (hypothyroidism)
  - Decrease in RBC mass, hypoproliferation
  - Pernicious anemia (usually macrocytic)

A Note on Myeloma

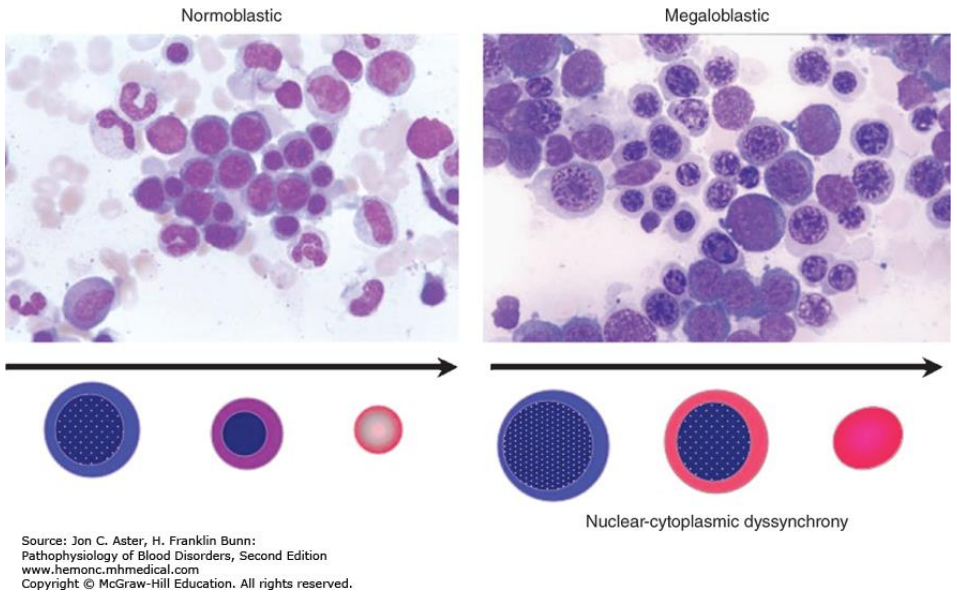
PLASMA CELL DYSCRASIA	MGUS	Smoldering Multiple Myeloma	Multiple Myeloma
M-protein	<30g/L	≥30g/L **OR** urinary M-protein ≥500mg/24h	–
BM clonal plasma cells	<10%	10-60%	≥10% or extramedullary plasmacytoma (+Bx)
End-organ damage or myeloma-defining events	None	None	≥1 CRAB **or** BM ≥60% **or** FLC ratio ≥100 **or** >1 focal lesion on MRI (≥5mm)

Approach to Anemia



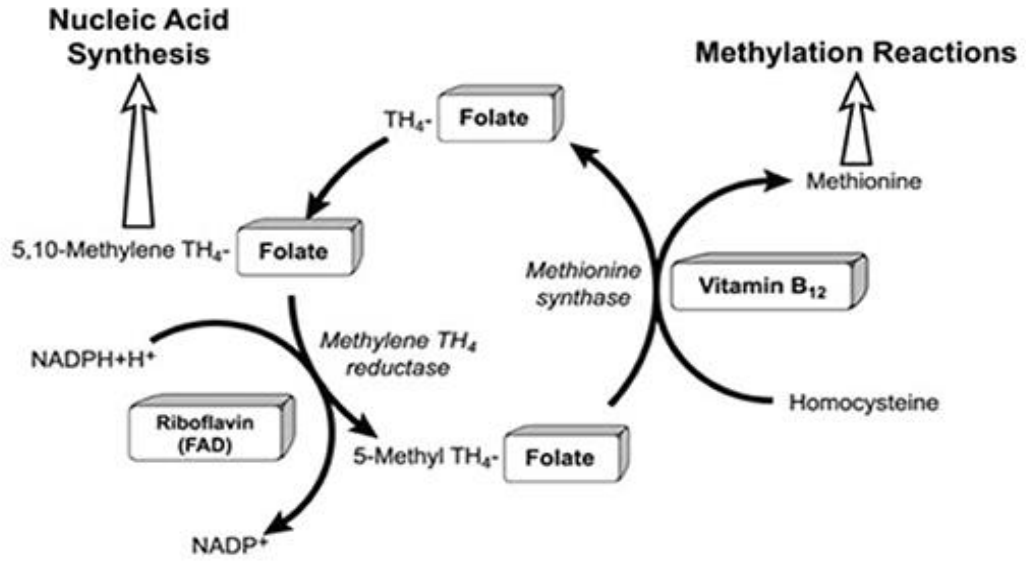
Megaloblastic Anemia

- Differentiate
  - MEGALOBLASTIC: big marrow precursors and circulating mature RBCs
  - MACROCYTIC: big mature circulating RBCs only
- Etiology: B12/folate deficiency, drugs
- Pathophysiology
  - Impaired DNA synthesis in hematopoietic cells → dyssynchrony b/w nuclear and cytoplasmic maturation → "big" erythroid cells in marrow and blood



B12 Metabolism

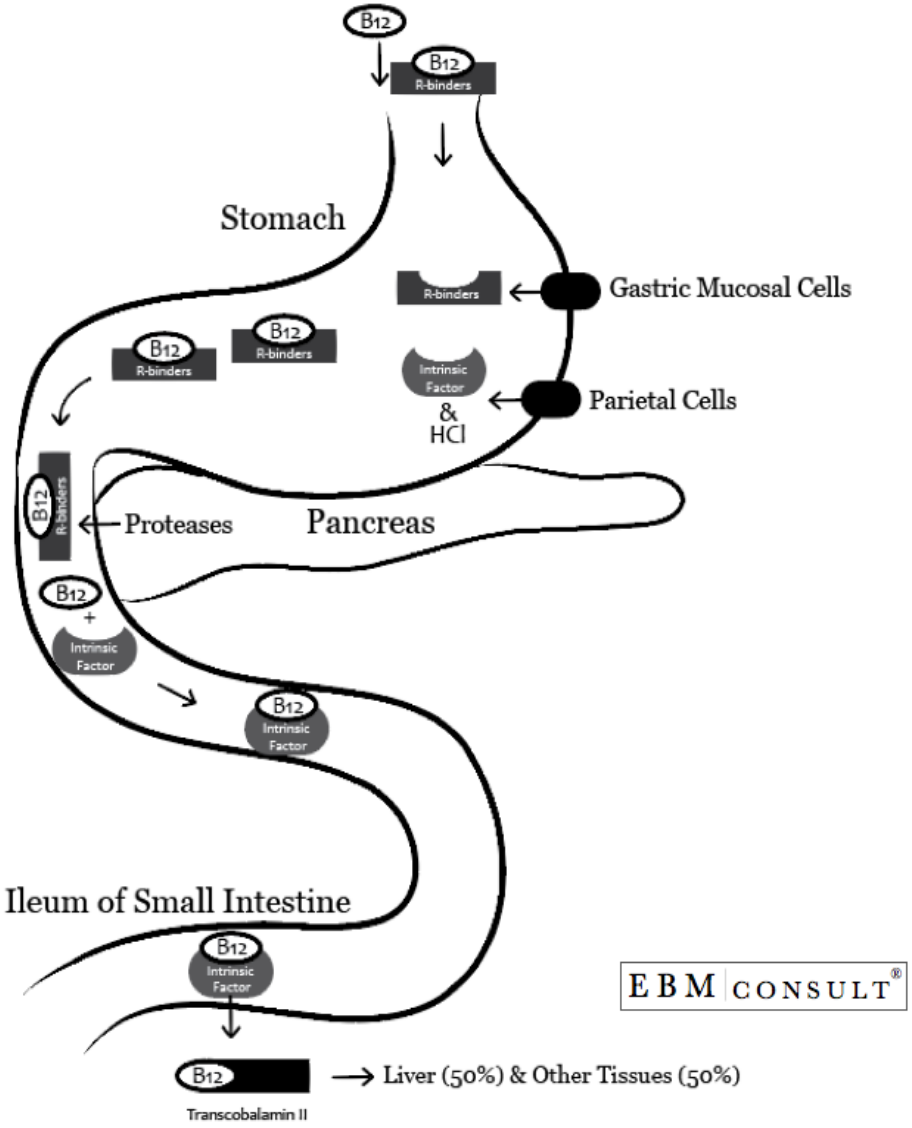
Figure 3. Vitamin B_{12} and Nucleic Acid Metabolism



5,10-Methylene tetrahydrofolate (TH_4) is required for the synthesis of nucleic acids, while 5-methyl TH_4 is required for the formation of methionine from homocysteine. Methionine, in the form of S-adenosylmethionine, is required for many biological methylation reactions, including DNA methylation. Methylene TH_4 reductase is a flavin-dependent enzyme required to catalyze the reduction of 5,10-methylene TH_4 to 5-methyl TH_4.

B12 Deficiency

- Etiology
  - Low intake (vegan)
  - Poor absorption
    - Low stomach acid
    - Low intrinsic factor (pernicious anemia)
    - Low pancreatic enzymes
    - Terminal ileal disease
    - Congenital
    - Medications
  - Defective transport



B12 Deficiency

- Clinical
  - Anemia symptoms
  - Neurologic (mainly gait, vibration/proprioception)
  - Neuropsychiatric
- Investigations
  - B12 <200 pg/mL
  - Elevated MMA/homocysteine
  - Anti-IF and anti-parietal cell antibodies
  - Blood smear: hypersegmented neutrophils
- Treatment
  - Treat underlying cause
  - B12 replacement – 1000mcg oral or IM
    - ++symptoms: IM daily x1 week → weekly x4 → monthly
    - OR oral: 1000-2000 mcg/day
    - Monitor for hypokalemia

Other Causes of Macrocytic Anemia

- Folate deficiency: very rare
- Drugs: methotrexate, hydroxyurea, anticonvulsants, septra, HIV meds, chemo
- Reticulocytosis
- MDS
- Liver disease
- EtOH
- Autoimmune hypothyroidism + pernicious anemia

Miscellaneous Anemias

- Anemia of malignancy (usually normocytic)
  - Low epo, increased hepcidin, bleeding / iron deficiency, chemoradiotherapy, marrow infiltration, hemolysis, nutritional
  - Treat w/ IV iron, epo, transfusions
- Anemia of liver disease (usually macrocytic)
  - Underproduction, blood loss, hemolysis (Zieve), nutritional, EtOH, viral hepatitis, therapy
  - Treat w/ transfusions, nutrition, EtOH cessation, etc.
- Anemia of the elderly (usually normocytic)
- Anemia of pregnancy

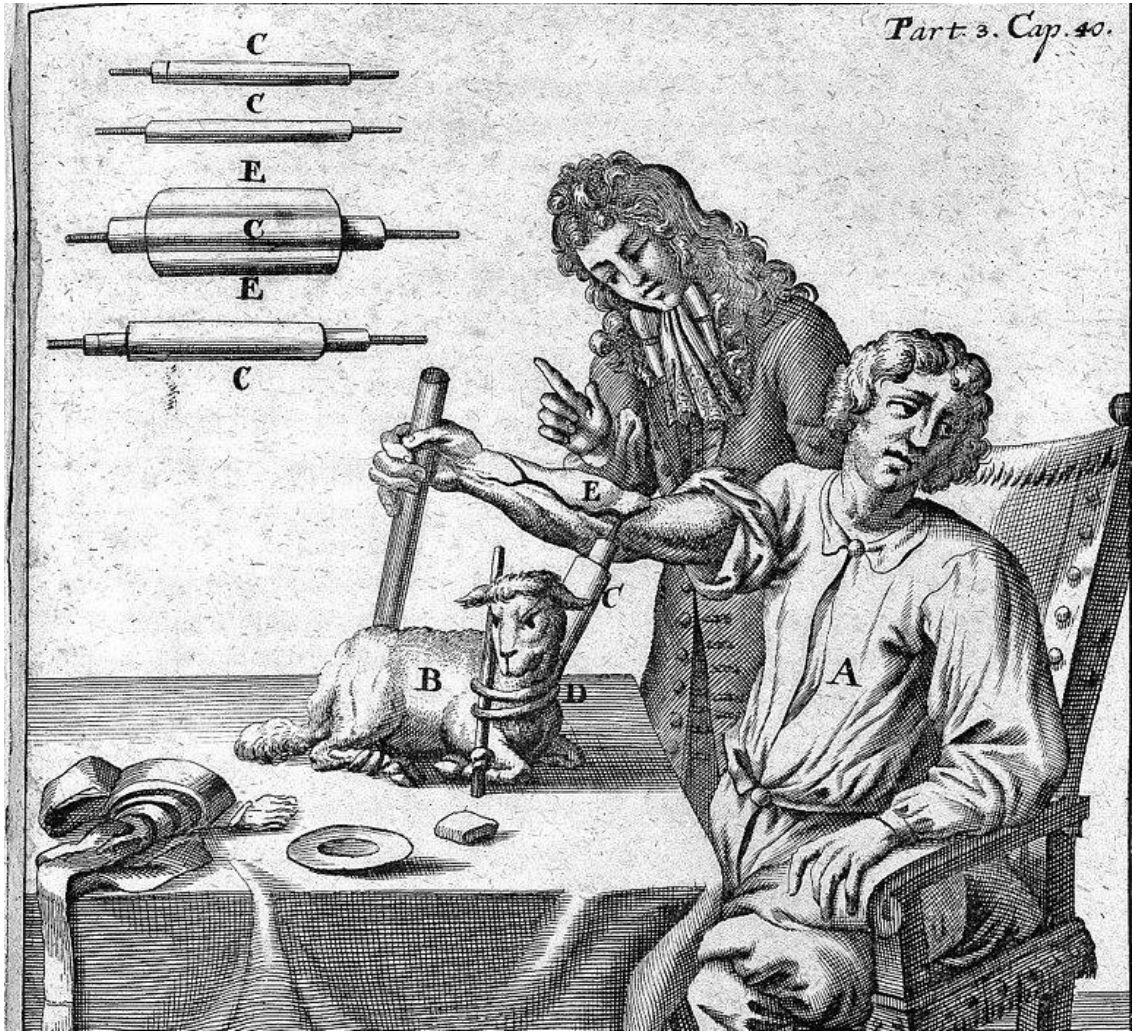
Pregnancy + Anemia

- Physiologic normocytic anemia due to increased plasma volume and dilution
- Pathologic anemia
  - Iron deficiency most common
  - Increased folate requirements
  - MAHA: preeclampsia/eclampsia, HELLP, AFLP
  - All other usual causes
- More fetal distress and perinatal complications

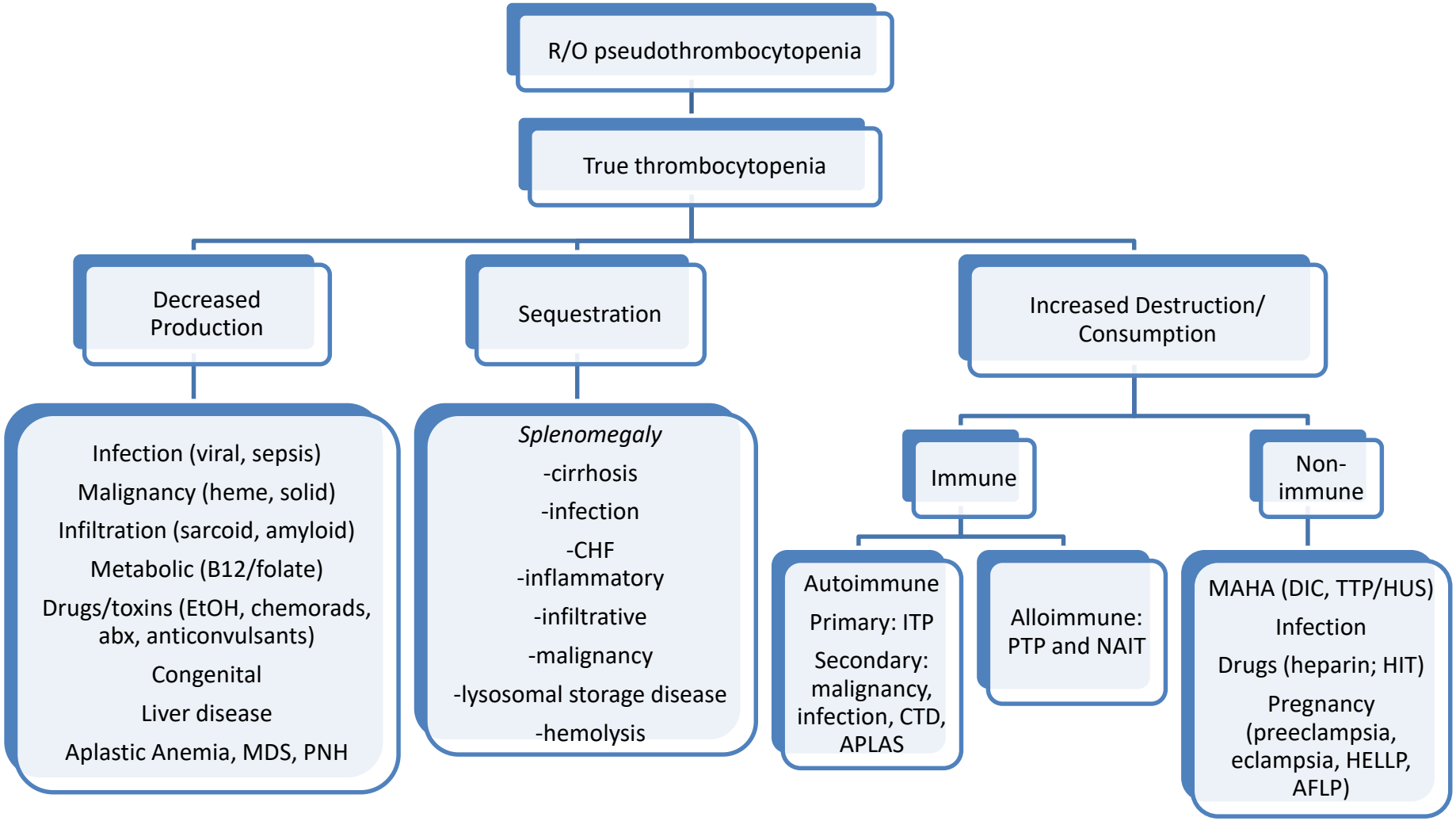
Anemia Red Flags

- Red Flags
  - Acuity
  - B symptoms / CRAB features
  - Other cytopenias
- When to refer
  - Refractory iron deficiency
  - Clinically significant thalassemia
  - Hemolysis
  - Suspect bone marrow failure
  - Suspect malignancy
  - Unexplained
  - Other unexplained cytopenias

Trivia: 1667



Approach to Thrombocytopenia



Investigation & Management of Thrombocytopenia

- Investigation
  - Very dependent on clinical picture
  - General: CBC, blood smear, viral (Hep B/C +/- HIV and H pylori), B12, liver enzymes, Cr, abdo U/S; +/- ANA, fibrinogen
- Management
  - Treat underlying cause
  - Supportive: transfusion if <=10 or ++bleeding, IVIg and steroids for ITP
  - Discontinue/hold offending agents and anticoagulation

Platelet Cutoffs

- When to transfuse? (Bloody Easy 4)
  - General: plt <10
  - Minor procedure (ex. CVC): plt <20
  - Surgery: plt <50
    - Neurosurgery or head trauma: plt <100
  - Anticoagulation: plt <30
  - LP / epidural: plt <50 (some say <100)
  - ITP + bleeding: plt <50
    - Generally ITP does not need transfusion
  - Plt dysfunction w/ bleeding: anytime

Immune Thrombocytopenia (ITP)

- Diagnosis of exclusion
- Antiplatelet antibodies bind to plts → platelets cleared by spleen
- Clinical: mucocutaneous bleeding, petechiae (<0.3cm), purpura (0.3-1cm), ecchymosis (>1cm)
- Spontaneous severe bleeding at plt <10
- BMBx not indicated unless clinically indicated to r/o another cause

ITP

- Management
  - Asymptomatic + plt >30: watch and wait
  - ++Symptomatic or plt <=30: treat
    - 1st -line: steroids (pred 1mg/kg w/ taper OR dex 40mg x4d only)
    - 2nd-line: splenectomy → rituximab → TPO agonists → other immunosuppressants
  - Adjunct
    - IVIg for rapid increase
    - Hold blood thinners?
    - Upcoming surgery?
    - Pregnancy implications

Thrombotic Microangiopathy (TMA)

- Spectrum of disease involving microangiopathic hemolysis, both congenital and acquired
- Prototype: TTP/HUS
  - Pentad: thrombocytopenia, MAHA, renal failure, fever, neuro symptoms
  - Treat w/ urgent PLEX, steroids
- DIC
  - Disorder of too much clotting and fibrinolysis
  - High INR/PTT, low fibrinogen, low platelets
  - Treat w/ blood products (FFP, cryo, plts) as needed

Pregnancy + Thrombocytopenia

- Physiologic: 10% decrease in normal pregnancy
- Pathologic
  - Gestational thrombocytopenia: 2nd/3rd trimester, benign, plt not <70, no tx (resolves postpartum)
  - ITP: same tx; beware cutoffs for planned delivery/epidural
    - Possibility of neonatal thrombocytopenia
  - MAHA
    - Severe preeclampsia/eclampsia
    - HELLP, AFLP
    - TTP/HUS, DIC
  - All other usual causes

Thrombocytopenia Red Flags

- Red flags
  - Significant bleeding
  - Thrombosis
  - Other cytopenias (esp. concurrent hemolytic anemia)
  - B symptoms
  - Acuity
- When to refer
  - Unexplained (?ITP)
  - Concern for TMA or HIT
  - Unexplained splenomegaly or concern for malignancy
  - Thrombosis
  - Other unexplained cytopenias

Question 1

- A 35F presents with an incidentally discovered Hb 105. MCV is 65, ferritin 7. She reports heavy menstrual bleeding. She has been on ferrous gluconate 300mg PO TID for 6 months, and is compliant. As usual, you wonder about absorption issues and bleeding elsewhere. For now, how would you manage her iron?

- A) Double the dose of ferrous gluconate
- B) Switch to feramax 150mg PO daily
- C) Give venofer 300mg IV x3 doses, each a week apart
- D) No change; reassess in 3 months

Question 1

- A 35F presents with an incidentally discovered Hb 105. MCV is 65, ferritin 7. She reports heavy menstrual bleeding. She has been on ferrous gluconate 300mg PO TID for 6 months, and is compliant. As usual, you wonder about absorption issues and bleeding elsewhere. For now, how would you manage her iron?

- A) Double the dose of ferrous gluconate
- B) Switch to feramax 150mg PO daily
- C) Give venofer 300mg IV x3 doses, each a week apart
- D) No change; reassess in 3 months

Question 2

- A 47F presents with a Hb of 84. She has a history of well-controlled lupus on plaquenil. She is asymptomatic but exam reveals mild jaundice. Given the results below, which of the following is NOT a possible explanation for her anemia?
  - MCV 102
  - Cr 154, TSH normal, SPEP normal
  - Ferritin 250 (high), TIBC low, transferrin-sat normal
  - Retics 150 (high), indirect bili high, LDH high, haptoglobin low

- A) Lupus nephritis causing decreased epo production
- B) Warm autoimmune hemolytic anemia
- C) Anemia of chronic disease due to lupus
- D) Plaquenil causing a sideroblastic anemia

Question 2

- A 47F presents with a Hb of 84. She has a history of well-controlled lupus on plaquenil. She is asymptomatic but exam reveals mild jaundice. Given the results below, which of the following is NOT a possible explanation for her anemia?
  - MCV 102
  - Cr 154, TSH normal, SPEP normal
  - Ferritin 250 (high), TIBC low, transferrin-sat normal
  - Retics 150 (high), indirect bili high, LDH high, haptoglobin low

- A) Lupus nephritis causing decreased epo production
- B) Warm autoimmune hemolytic anemia
- C) Anemia of chronic disease due to lupus
- D) Plaquenil causing a sideroblastic anemia

Question 3

- Which of the following mechanisms is NOT a typical cause of thrombocytopenia?

- A) Renal disease causing uremic platelet dysfunction
- B) Severe B12 deficiency leading to a thrombotic microangiopathy picture
- C) Liver disease causing decreased thrombopoietin production and hypersplenism
- D) Antiplatelet antibodies binding to platelets, causing immune destruction in the spleen

Question 3

- Which of the following mechanisms is NOT a typical cause of thrombocytopenia?

- A) Renal disease causing uremic platelet dysfunction
- B) Severe B12 deficiency leading to a thrombotic microangiopathy picture
- C) Liver disease causing decreased thrombopoietin production and hypersplenism
- D) Antiplatelet antibodies binding to platelets, causing immune destruction in the spleen

Question 4

- A 56M presents with an isolated plt count of 25. He is otherwise healthy and on no regular meds. CBC, Cr, liver enzymes, B12/folate, ANA, and abdo U/S are all normal. You suspect ITP. He has petechiae on his legs bilaterally. No upcoming procedures. What is the most reasonable next step?

- A) Check Hep C, HIV +/- H pylori. If negative, treat as ITP with steroids as 1st line.
- B) Admit to hospital. Give IVIg 1g/kg x2 doses, 2 days apart.
- C) Check Hep C, HIV. Transfuse 1 adult dose plts. Treat with IVIg and steroids.
- D) Check Hep C, HIV +/- H pylori. Watch and wait. No indication for acute treatment.

Question 4

- A 56M presents with an isolated plt count of 25. He is otherwise healthy and on no regular meds. CBC, Cr, liver enzymes, B12/folate, ANA, and abdo U/S are all normal. You suspect ITP. He has petechiae on his legs bilaterally. No upcoming procedures. What is the most reasonable next step?

- A) Check Hep C, HIV +/- H pylori. If negative, treat as ITP with steroids as 1st line.
- B) Admit to hospital. Give IVIg 1g/kg x2 doses, 2 days apart.
- C) Check Hep C, HIV. Transfuse 1 adult dose plts. Treat with IVIg and steroids.
- D) Check Hep C, HIV +/- H pylori. Watch and wait. No indication for acute treatment.

Bottom Line

- Approach to anemia is based on MCV
  - Microcytic: iron def, thal (**look for CAUSE of IDA)
    - Iron supplementation – adequacy, logistics, monitor
    - Thal: usually no treatment needed unless severe
  - Normocytic: bleed? hemolyzing? → marrow
    - Usually refer to hematologist for hemolysis or marrow
    - GI referral for bleeding
  - Macrocytic: retics? B12/folate? liver? → marrow
- Approach to thrombocytopenia is based on production, sequestration, and destruction
  - ITP is diagnosis of exclusion; first line tx is steroids
  - Dangerous: TMA/MAHA
- Red flags: acuity, B symptoms, other cytopenias, thrombosis, etc.
- Treatment is based on underlying cause + transfusions/supportive

Thank you!