Surgery

2nd stage 2nd course

B L E E D IN G D IS O R D E R S

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HEMOSTASIS

- VASCULAR PHASE
- 2. PLATELET PHASE
- 3. COAGULATION PHASE
- 4. FIBRINOLYTIC PHASE

VASCULAR PHASE

WHEN A BLOOD VESSEL IS DAMAGED, VASOCONSTRICTION RESULTS.

PLATELET PHASE

PLATELETS ADHERE TO THE DAMAGED SURFACE AND FORM A TEMPORARY PLUG.

COAGULATION PHASE

THROUGH TWO SEPARATE
PATHWAYS THE CONVERSION OF
FIBRINOGEN TO FIBRIN IS
COMPLETE.

FIBRINOLYTIC PHASE

ANTICLOTTING MECHANISMS ARE ACTIVATED TO ALLOW CLOT DISINTEGRATION AND REPAIR OF THE DAMAGED VESSEL.

HEMOS TAS IS

DEPENDENT UPON:

- Vessel Wall Integrity
- Adequate Numbers of Platelets
- Proper Functioning Platelets
- Adequate Levels of Clotting Factors
- Proper Function of Fibrinolytic Pathway

LABORATORY EVALUATION

- PLATELET COUNT
- BLEEDING TIME (BT)
- PROTHROMBIN TIME (PT)
- PARTIAL THROMBOPLASTIN TIME (PTT)
- THROMBIN TIME (TT)

PLATELET COUNT



100,000 - 400,000 CELLS/MM3

< 100,000

Thrombocytopenia

50,000 - 100,000

Mild Thrombocytopenia

< 50,000

Sev Thrombocytopenia

BLEEDING TIME

PROVIDES ASSESSMENT OF PLATELET COUNT AND FUNCTION

NORMAL VALUE
2-8 MINUTES

PROTHROMBIN TIME

- Measures Effectiveness of the Extrinsic Pathway
- Mnemonic PET

NORMAL VALUE 10-15 SECS

PARTIAL THROMBOPLASTIN TIME

Measures Effectiveness of the Intrinsic Pathway

Mnemonic - PITT

NORMAL VALUE 25-40 SECS

THROMBIN TIME

- Time for Thrombin To Convert

 Fibrinogen

 Fibrin
- A Measure of Fibrinolytic Pathway

NORMAL VALUE 9-13 SECS

So What Causes Bleeding Disorders?

- VESSEL DEFECTS
- PLATELET DISORDERS
- FACTOR DEFICIENCIES
- OTHER DISORDERS

VESSEL DEFECTS

- VITAMIN C DEFICIENCY
- BACTERIAL & VIRAL INFECTIONS

ACQUIRED

P LATELET D IS O R D E R S





INADEQUATE NUMBER OF PLATELETS

ADEQUATE NUMBER BUT ABNORMAL FUNCTION

- DRUG INDUCED
- BONE MARROW FAILURE
- HYPERSPLENISM
- OTHER CAUSES



.Alcohol

.Thiazide Diuretics



BONE MARROW FAILURE

- Viral Infections
- Nutritional Deficiencies
- Chemotherapy & Radiation Therapy
- Infiltration of Abnormal Cells
 - Aplastic Anemia
 - Leukemia
 - Metastatic Cancer

HYPERSPLENISM

- Increase in Size Leads to Destruction of Platelets
- Associated with Portal Hypertension Seen in Patients with Cirrhosis

- OTHER CAUSES
 - Lymphoma
 - HIV Virus
 - Idiopathic Thrombocytopenia Purpura (ITP)

- UREMIA
- INHERITED DISORDERS
- MYELOPROLIFERATIVE DISORDERS
- DRUG INDUCED



ASPIRIN

IRREVERSIBLY BINDS TO THE PLATELET FOR ITS ENTIRE LIFESPAN (7-10 DAYS)



NSAIDS

REVERSIBLY BINDS TO THE PLATELET FOR A LIMITED TIME PERIOD (APPROX 6 HOURS)

FACTOR DEFICIENCIES

(CONGENITAL)







FACTOR DEFICIENCIES

- HEMOPHILIA A (Classic Hemophilia)
 - 80-85% of all Hemophiliacs
 - Deficiency of Factor VIII
 - Lab Results Prolonged PTT

- HEMOPHILIA B (Christmas Disease)
 - 10-15% of all Hemophiliacs
 - Deficiency of Factor IX
 - Lab Test Prolonged PTT

FACTOR DEFICIENCIES

VON WILLEBRAND'S DISEASE

Deficiency of VWF & amount of Factor VIII

Lab Results - Prolonged BT, PTT

(ACQUIRED)



ORAL ANTICOAGULANTS

- × COUMARIN
- **X** HEPARIN



LIVER DISEASE



MALABSORPTION



BROAD-SPECTRUM ANTIBIOTICS

ORAL ANTICOAGULANTS

Coumarin Prevents Thromboembolic Events & is a Vit K Antagonist. Monitored by PT times.

Heparin Therapy is Monitored by PTT times.

MALABSORPTION

Various Intestinal Diseases Will Interfere w/ Bile Acid Metabolism.

Bile Acids are Required for Vit K Absorption so You Will See a Deficiency in Vit K Dependent Coagulation Factors (II,VII,IX,X).

LIVER DISEASE

Jaundice Results in Malabsorption of Vit K.

Liver Disease can Result in Reduced Production of Coagulation Factors (I,II,V,VII,IX,X).



Change in Intestinal Flora which Might Decrease Vitamin K Production.

Vitamin K is Necessary for the Liver to Produce Coagulation Factors II,VII,IX,X.