INTRODUCTION TO HEMOSTASIS

PRIMARY HEMOSTASIS - BLOOD VESSELS AND PLATELETS

WHAT IS HEMOSTASIS?

THE MAINTENANCE OF
CIRCULATORY BLEEDING AND
CLOTTING THROUGH A BALANCED
PROCESS OF COAGULATION AND
FIBRINOLYSIS.



HEMOSTASIS IS THE HIGHLY INTEGRATED AND REGULATED INTERACTION OF:

MAJOR SYSTEMS

- BLOOD VESSELS
- o Platelets
- COAGULATION PROTEINS
- FIBRINOLYSIS
- SERINE PROTEASE INHIBITORS

MINOR SYSTEMS

- KININ SYSTEM
- COMPLEMENT SYSTEM

TWO STAGES:

PRIMARY

• INITIAL RESPONSE TO VASCULAR INJURY - THE PLATELET PLUG SECONDARY

 ENZYMATIC ACTIVATION OF THE COAGULATION PROTEINS TO PRODUCE FIBRIN FROM FIBRINOGEN.

COAGULATION





In platelet rich plasma, with few space occupying RBCs, contraction may be to as little as 10% of the original clot volume.

Actin in the filipods and myosin in the platelet body contract, shortening the fibrin strands and shinking the platelet-fibrin plug. This process utilizes large amounts of energy (plateletATP) and Ca++. Add more Ca++ !!!!

STEPS 1-3 ARE PRIMARY HEMOSTASIS. STEPS 4 AND 5 ARE SECONDARY HEMOSTATIS

BEFORE WE GET INTO HEMOSTASIS LET'S TALK About the vascular system.

• TUNICA INTIMA

- INNER MOST
- SINGLE ENDOTHELIAL LAYER
- TUNICA MEDIA
 - MIDDLE LAYER
 - ELASTIC AND SMOOTH MUSCLE
- TUNICA ADVENTITIA
 - OUTER MOST
 - FIBROUS CONNECTIVE TISSUE



VESSEL SIZE COMPARISON

VESSEL	ST/E	VESSEL	ST/E
Aorta	25 MM	VENA CAVA	20 MM
ARTERY	4MM	VEIN	5 MM
ARTERIOLE	30 J M	VENULE	20 µ m
PRE SPHINCTER CAPILLARY	35 µ m	CAPILLARY	8 J M

ROLE OF INTACT ENDOTHELIUM



IT IS THROMBORESISTANT!

PLATELET FACTS

- 1 MEGAKARYOCYTE = 1000-2000 Platelets
- MATURATION TIME = 5 DAYS
- LIFE SPAN = 7-10 DAYS
- BM TO SPLEEN = 2 DAYS
 - CIRCULATING POOL/ACTIVE SPLENIC POOL
- 70% PLATELETS IN CIRCULATION
- 30% PLATELETS IN SPLEEN FOR EXCHANGE
- 150-450 x 10⁹/L @ 2-4 MICRONS



PLATELET STRUCTURE

- PERIPHERAL ZONE
 - GLYCOCALYX
 - PLATELET MEMBRANE
 - 0CS
- SOL-GEL ZONE
 - MICROTUBULES AND MICROFILAMENTS
- ORGANELLE ZONE
 - GRANULES
 - MITOCHONDRIA
 - DENSE TUBULAR SYSTEM







WHAT HAPPENS WHEN A BLOOD VESSEL Is injured?

- VASOCONSTRICTION
- DIVERSION OF BLOOD FLOW
- INITIATION OF CONTACT ACTIVATION OF PLATELETS
- CONTACT ACTIVATION OF COAGULATION CASCADE

WHAT'S RELEASED FROM DAMAGED VESSELS?

COAGULATION	ANTICOAGULANT	FIBRINOLYSIS	ANTIFIBRINOLYSIS
COLLAGEN VWF	HEPARIN SULFATE PGI ₂	T-PA	PAIS
TISSUE FACTOR	TFPÍS Thrombomodulin		

HOW DO PLATELETS PARTICIPATE IN HEMOSTASIS?

- PROVIDE NEGATIVELY CHARGED PHOSPHOLIPID SURFACE FOR FACTOR X AND PROTHROMBIN ACTIVATION
- RELEASE SUBSTANCES THAT MEDIATE VASOCONSTRICTION, PLATELET AGGREGATION, COAGULATION, AND VASCULAR REPAIR
- PROVIDING SURFACE MEMBRANE GLYCOPROTEINS TO ATTACH TO OTHER PLATELETS VIA FIBRINOGEN, COLLAGEN, AND SUBENDOTHELIUM

ACHIEVE THIS VIA:

- SHAPE CHANGE
- ADHESION
- AGGREGATION
- SECRETION

PLATELET FUNCTION

- SHAPE CHANGE PSEUDOPOD FORMATION AND CONTRACTIONS SIGNAL INTRACELLULAR ACTIVATION
- ADHESION TO SITE OF INJURY VIA GPIB AND VWF
- AGGREGATION PLATELETS STICK TO ONE ANOTHER VIA GPIIB/IIIA AND FIBRINOGEN
- SECRETION RELEASE OF GRANULE CONTENTS AMPLIFIES THE PLATELET RESPONSE



Activated platelets show little cell feet called pseudopodia.







IN SUMMARY



CREDITS

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- TEXTBOOK: MCKENZIE, S.B., & WILLIAMS, J.L (2015). <u>CLINICAL LABORATORY HEMATOLOGY</u>, PEARSON EDUCATION INC. ISBN 978-0-13-307601-1.