

Individual Session Template

Please Save These Documents as “Session Name_Session Authors Name_Session Date”

Session Author’s Name:	Tracy Fulton
Session Presenter’s Name (if panel, lecture, etc)	
Course Title:	Cardiovascular

Session Title:	Biochemistry Coagulation and Hemostasis
Session Format:	Lecture
Discipline(s):	Biochemistry
Organ System(s):	Cardiovascular
Length:	50min
Related Course Objective(s):	<ol style="list-style-type: none"> 1) Assess patient’s condition and determine the appropriate therapeutic treatment needs for cardiovascular conditions (i.e. immediate medical care, self-care, and management by pharmacist) utilizing knowledge of anatomy, pathology, pathophysiology, pharmaceutical chemistry, pharmacology, behavioral and social sciences, as well as current evidence (i.e. literature review) 2) Assess current treatment, recommend alternative treatment and, when appropriate, prescribe optimal treatment for a patient with cardiovascular disease using knowledge of pathophysiology, pharmaceutical chemistry, pharmacology, behavioral and social sciences, as well as current evidence (i.e. literature review).
UCSF 49 Cluster or Syndrome:	Relevant tpx topics: ischemic heart disease, VTE, afib
Work to do prior to this session (pre-reading)	Biochemistry Coagulation Cascade Video (30min)

Description/notes:
Lecture will...

After this session, students should be able to meet the following <i>objectives</i>*:)
BC 112: Hemostasis 1 & 2 11/21/2016
<ul style="list-style-type: none"> • Describe how clots are broken down. • Describe the causes of hemophilia A, hemophilia B, and von Willebrand's disease. • Describe the sequence of events that leads to formation of a normal blood clot at a site of vascular damage. • Draw a picture of a platelet plug and indicate the names, location and function of key proteins and small molecules that participate in platelet aggregation. • Explain the difference between the intrinsic, extrinsic and common clotting pathways. • Explain the role of vitamin K in the production of functional clotting factors. • Explain why patients with liver disease are at increased risk of aberrant excessive bleeding. • For each of the following contributors to excessive bleeding, list and describe two possible causes of the condition: reduced platelet number, defective platelet function, and clotting factor abnormalities

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- Make a diagram of the coagulation cascade and be able to show the location within the cascade of tissue factor, factor V, factor VII, factor VIII, factor IX, factor X, thrombin, and fibrin.
- Name four substances that inhibit clotting and describe their anti-clotting action.
- Name three substances that trigger platelet degranulation.

Relevant Medications

- Aspirin
- Anticoagulants: warfarin, unfractionated heparin, enoxaparin, dalteparin, fondaparinux, bivalirudin, argatroban, dabigatran, rivaroxaban, apixaban, edoxaban
- Fibrinolytics (alteplase, tenecteplase)
- P2Y12 Antagonists: clopidogrel, prasugrel, ticlopidine, cangrelor
- GPIIb/IIIa inhibitors: abciximab, eptifibatide, tirofiban
- Vorapaxar

Will this session spiral? If so, where and how?:

The content will spiral to Arrhythmia (CV Block), VTE (Pulmonary Block)

If this is a case or patient presentation, what are the demographics/characteristics?

Potential link to Bridges Theme, Inquiry, or CMC, etc

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Assessment Ideas *(feel free to jot down creative assessment ideas here):*

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