

How to interpret abnormal coagulation studies in critically ill?

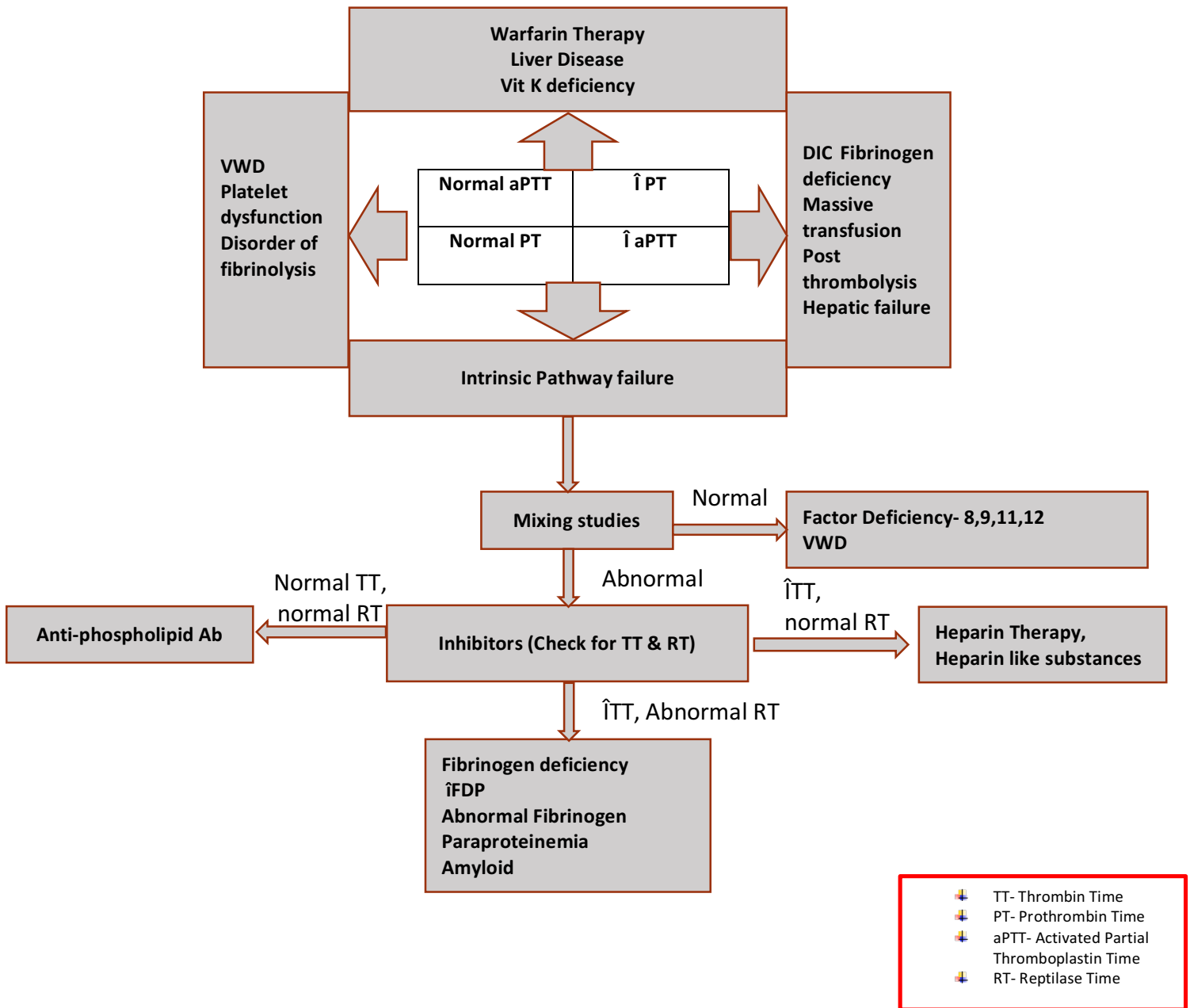
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Frontiers in Intensive care

Interpretation of Abnormal Coagulation studies



Coagulation Tests:-

Sl No	Test	How is it measured	Significance	Interpretation
1	TT (Thrombin Time)	<ul style="list-style-type: none"> Addition of thrombin to patient's platelet poor plasma. Thrombin converts Fibrinogen to Fibrin Very rapid (TT= 13-15 sec) 	<ul style="list-style-type: none"> It is a measure of final step of coagulation cascade - Fibrinogen to Fibrin AT-III is the natural antagonist of this step. Heparin causes a prolonged TT (as it potentiates AT-iii by 1000 times) Warfarin- does not affect TT. 	<ul style="list-style-type: none"> Low Fibrinogen level Abnormal Fibrinogen Paraproteinemia Amyloidosis Drugs- <ul style="list-style-type: none"> ◇ Heparin ◇ Direct Thrombin inhibitors- Argatroban, Dabigatran, Hirudin
2	PT (Prothrombin Time)	<ul style="list-style-type: none"> External "Tissue factor or Thromboplastin and Calcium" are added to plasma and time to clot is measured. This initiates the extrinsic pathway, which proceeds to common pathway 	<ul style="list-style-type: none"> Represents the Extrinsic and common pathway function. It tests the function of Vit K dependent factors- II, VII & X (Not IX though Vit K dependent) 	<ul style="list-style-type: none"> Warfarin therapy Vit K deficiency Liver failure Deficiency of factors (VII, II, X, Fibrinogen,V) Presence of inhibitors of the factors Fibrinogen abnormalities (Hypo/Dys/Afibrinogenemia) Dilutional coagulopathy
3	aPTT (Activated partial Thromboplastin Time)	<ul style="list-style-type: none"> Phospholipid (cephalin), contact activator (Kaolin/silica/ellagic acid) & calcium (all pre-warmed to 37°C) are added to platelet poor plasma. Time taken from the addition of calcium to the formation of clot is measured as aPTT. 	<ul style="list-style-type: none"> Represents the intrinsic or common pathway function. It is increased with either factor deficiency or presence of factors inhibitors or both The distinction can be made by "Mixing studies" 	<ul style="list-style-type: none"> Deficiency of factors <ul style="list-style-type: none"> ◇ VIII,IX,XI,XII ◇ VWD (Factor VII deficiency) ◇ Dilutional coagulopathy Inhibition of factors <ul style="list-style-type: none"> ◇ Heparin ◇ Antiphospholipid syndrome
4	Mixing studies	<ul style="list-style-type: none"> Patient's plasma & normal plasma are mixed with 1:1 ratio. 		If the aPTT is not corrected with in a stipulated time (usually 3-4 sec) then- <ul style="list-style-type: none"> An inhibitor of the coagulation factor Antiphospholipid Ab
5	PTT (Partial Thromboplastin Time)	<ul style="list-style-type: none"> No activator is added to speed up the clotting 	<ul style="list-style-type: none"> Same as aPTT but reported to be less sensitive 	
6	ECT (Ecrine Time)	<ul style="list-style-type: none"> "Ecarin" activates prothrombin bypassing intrinsic or extrinsic pathway. It differentiates the cause for 	<ul style="list-style-type: none"> Used to measure activity of direct thrombin inhibitors i.e Argatroban, Dabigatran 	If abnormal along with a ↑ aPTT- direct thrombin inhibitor present. If normal with an abnormal aPTT- Heparin or heparin like substances present.

		<ul style="list-style-type: none"> • ↑ aPTT (factor deficiency Vs presence of inhibitors of coagulation factor) 		
7	RT (Reptilase Time)	<ul style="list-style-type: none"> • "Reptilase" converts fibrinogen to fibrin 	<ul style="list-style-type: none"> • It is not affected by any human haemostatic mechanism which includes activity of AT-III, direct thrombin inhibitors i.e Argaroban, Hirudin. 	<ul style="list-style-type: none"> • ↑ only in disorders related to <ul style="list-style-type: none"> • fibrin function ◇ Dysfunctional fibrin ◇ Paraproteinemia ◇ Amyloidosis • Fibrin concentration ◇ Hypofibrinogenemia

References-

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3. Practical-Haemostasis.com