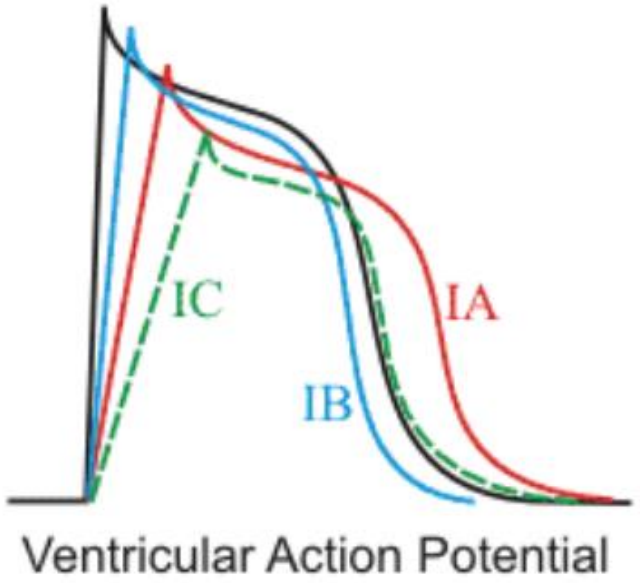
The Year in Review Series: Case 6. Palpitations
Case-based NBME review



Howard J. Sachs, MD
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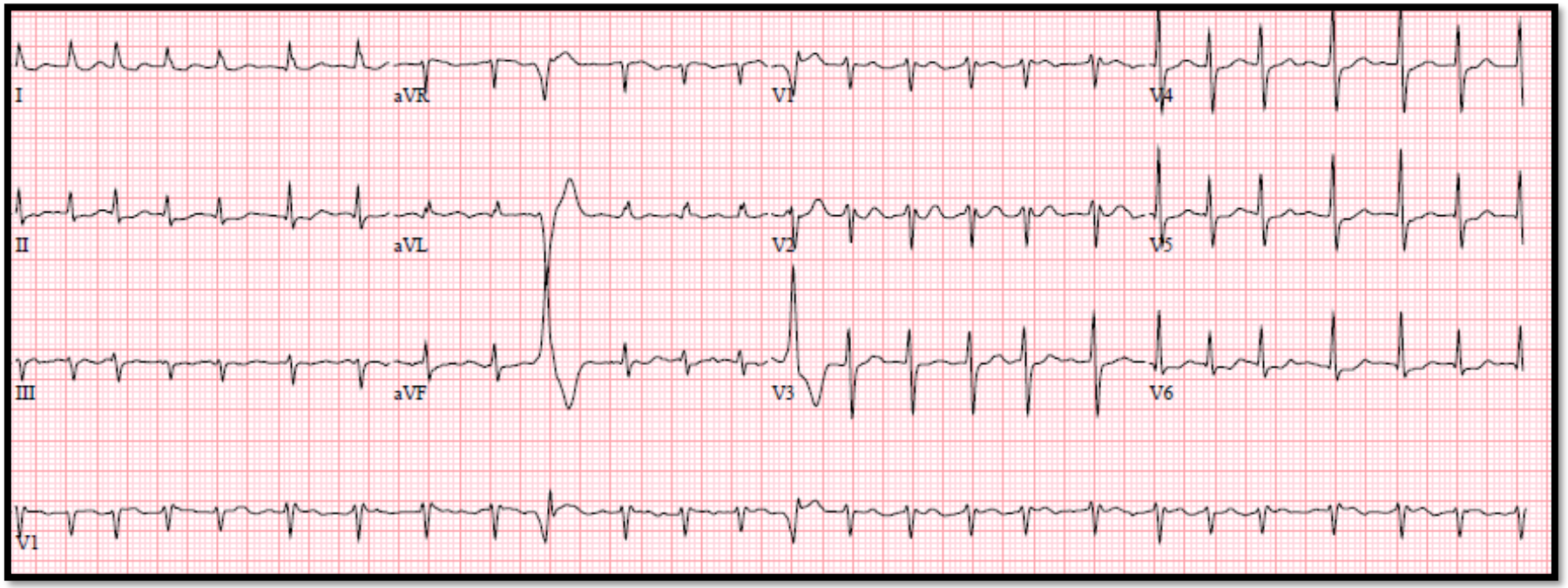
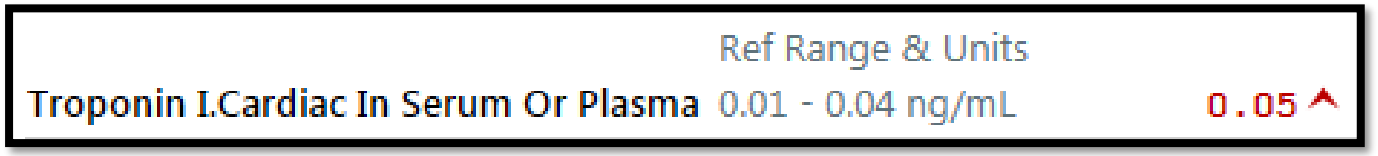
85 y.o. woman presents for evaluation of weakness and lightheadedness. Of course, it's my first patient on a busy Monday morning. PE: 124/82; pulse 157 and irregularly irregular; lungs – clear; cardiac – S1 and S2 are normal. No S3 or JVD. Choose which of the following are the principle determinant of her rapid ventricular response?



- Sinus node automaticity
- Atrioventricular node refractoriness
- Stimulation of beta-1 adrenoreceptors
- Left venticular end-diastolic volume
- Conduction through Purkinje fibers

Question 1 of 4

A repeat EKG obtained 6 hours later shows no evidence of ischemia with unchanged ST segments. As part of her evaluation, cardiac biomarkers are obtained (see graphic). She reports never experiencing exertional chest pain symptoms. To further evaluate the troponin elevation, she is referred for a nuclear perfusion stress test. The study is interpreted as normal.



	Ref Range & Units	
Troponin I.Cardiac In Serum Or Plasma	0.01 - 0.04 ng/mL	0.05 ^

Which of the following are most likely to explain her myocardial injury?

- Coronary artery occlusion with thrombosis
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Question 3 of 4

Select the mechanism of action of the agent and potential changes seen on the EKG for the agent that was most likely used to treat her underlying rhythm disturbance?

- Blocks the sodium channel during phase 0 of the action potential with possible widening of the QRS complex
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85 y.o. woman is diagnosed with atrial fibrillation. Her daughter who attended the visit saw a TV commercial talking about the risk of blood clots. After reviewing the pros and cons of therapy and the multiple agents available, the patient is started on rivaroxaban.

At her follow up visit, my MD-PhD student finally shows up for a clinic and orders a host of blood tests to assess her compliance. Assuming she is compliant, which pattern is most consistent with her therapeutic agent?

PT	aPTT	Thrombin Time	Bleeding Time
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Increase	Increase	Increase	Normal
Increase	Increase	Normal	Increase
Increase	Increase	Normal	Normal
Normal	Normal	Normal	Increase

Question 4 of 4

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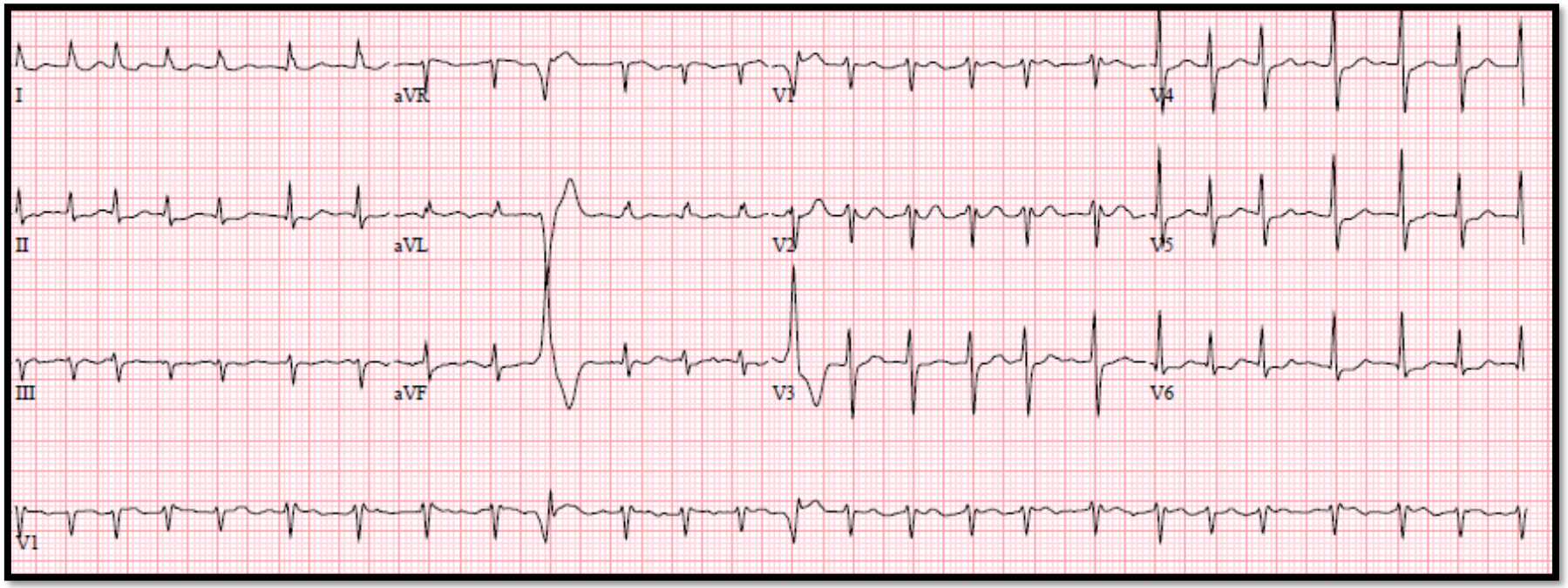

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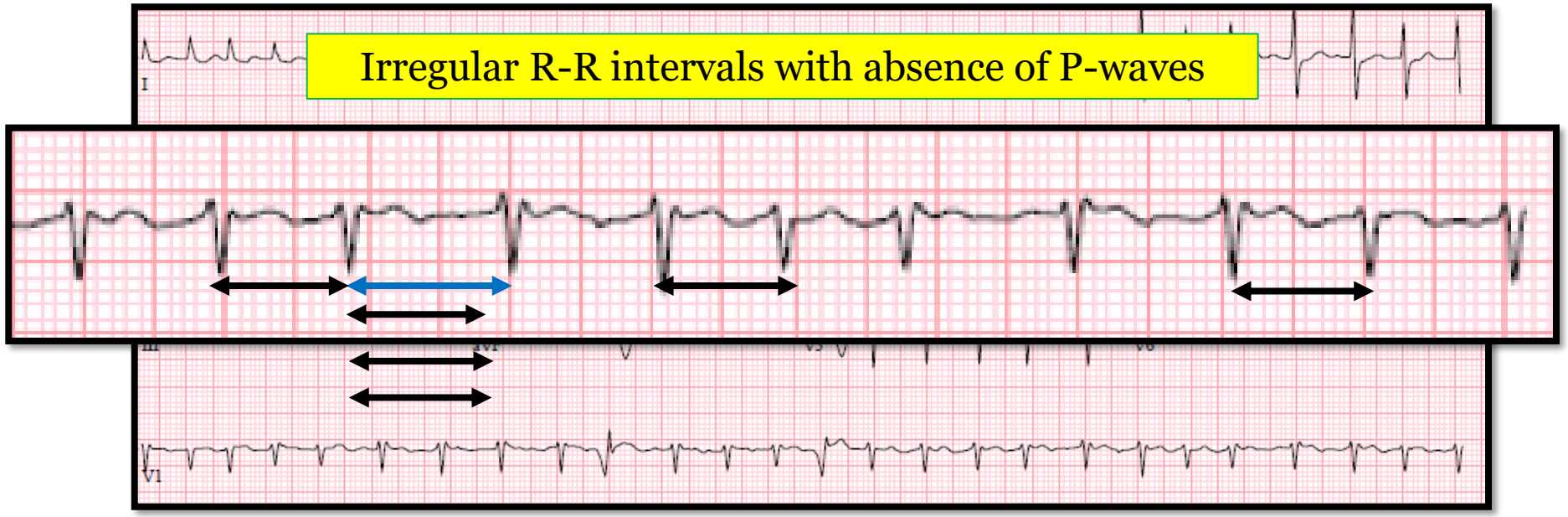

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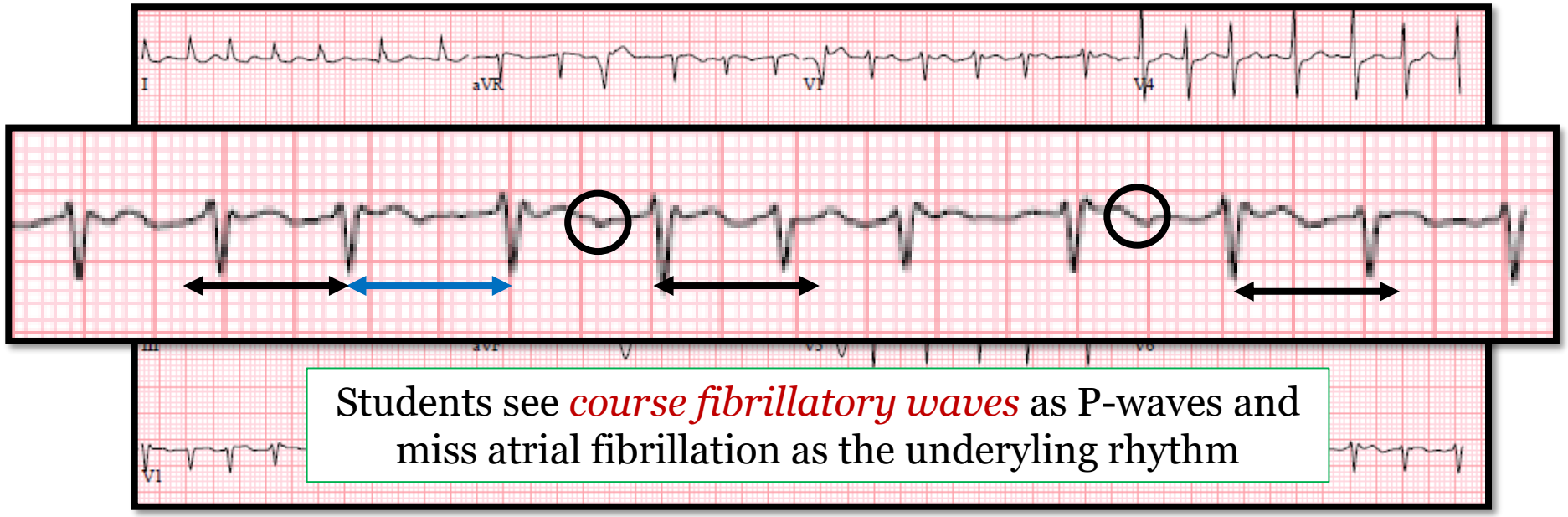

Students see *course fibrillatory waves* as P-waves and miss atrial fibrillation as the underyling rhythm

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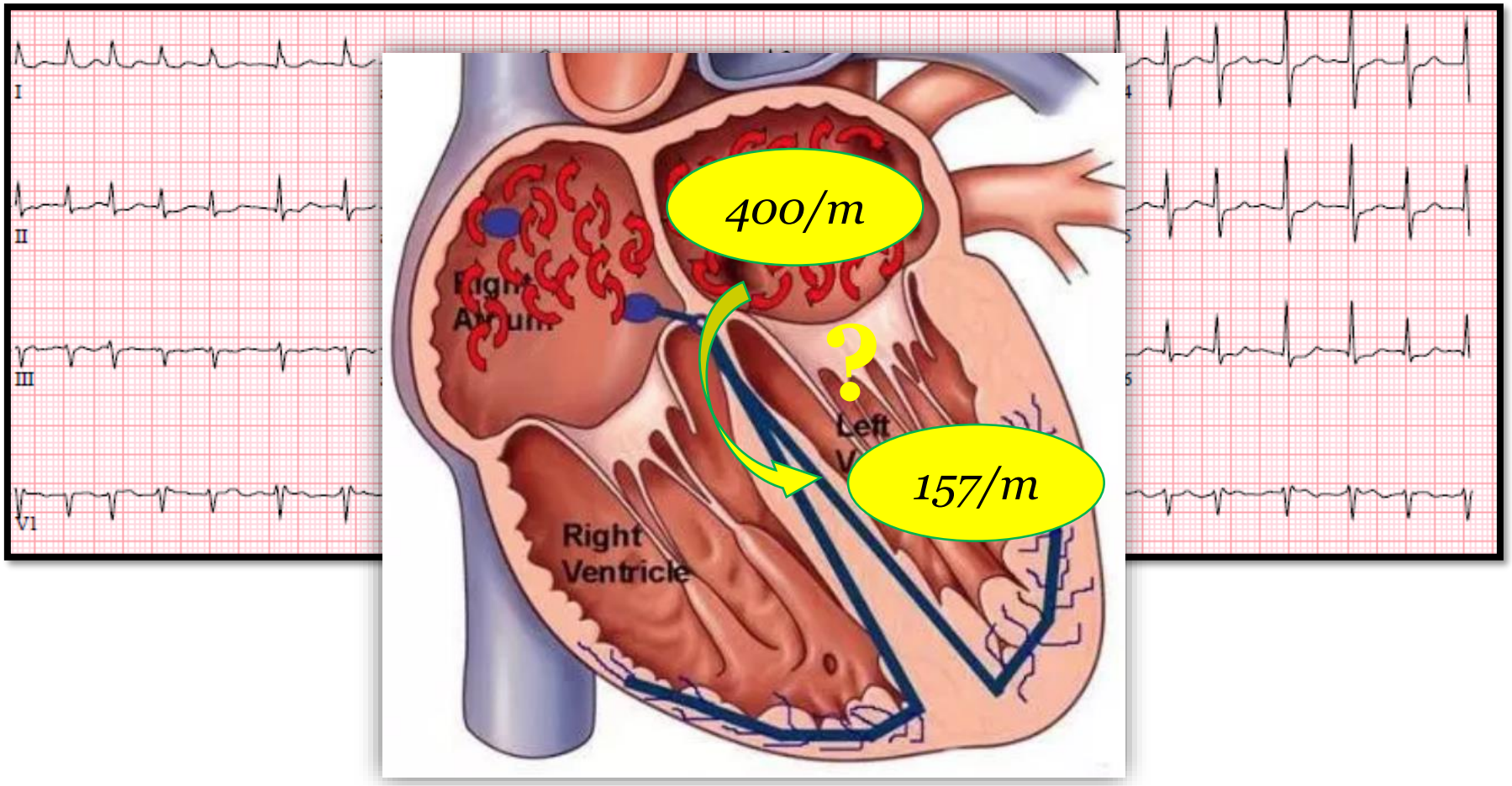

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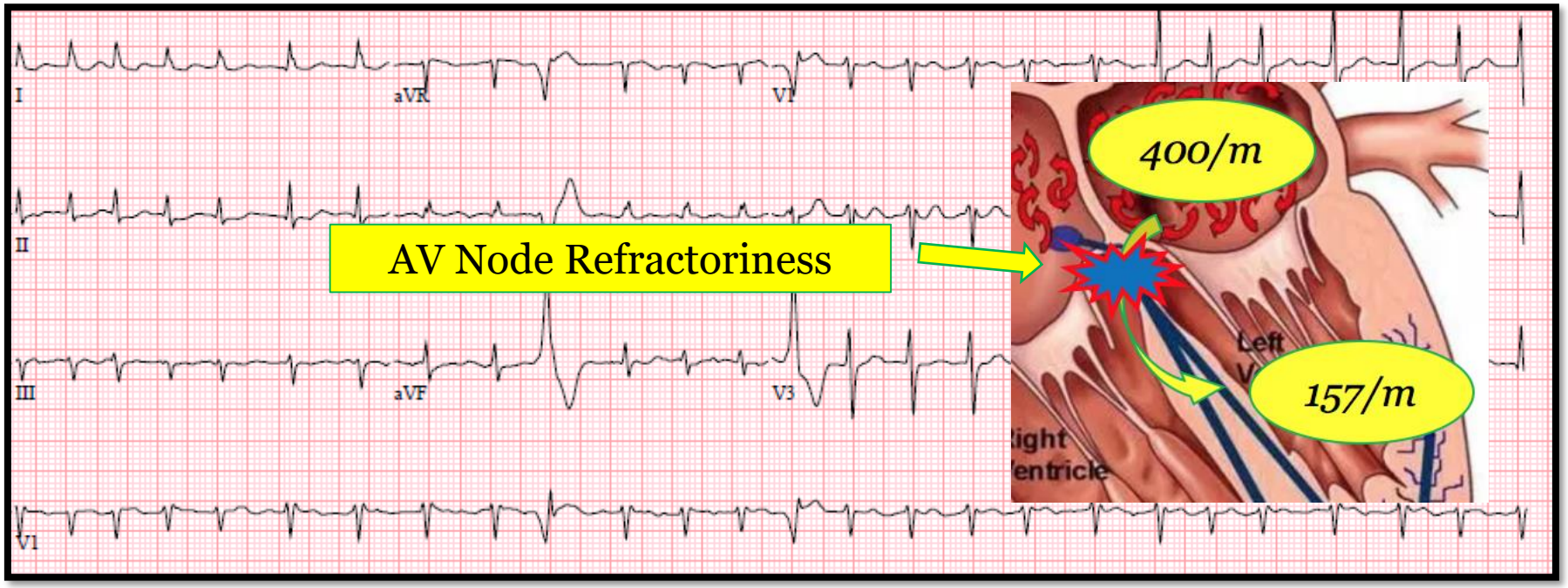

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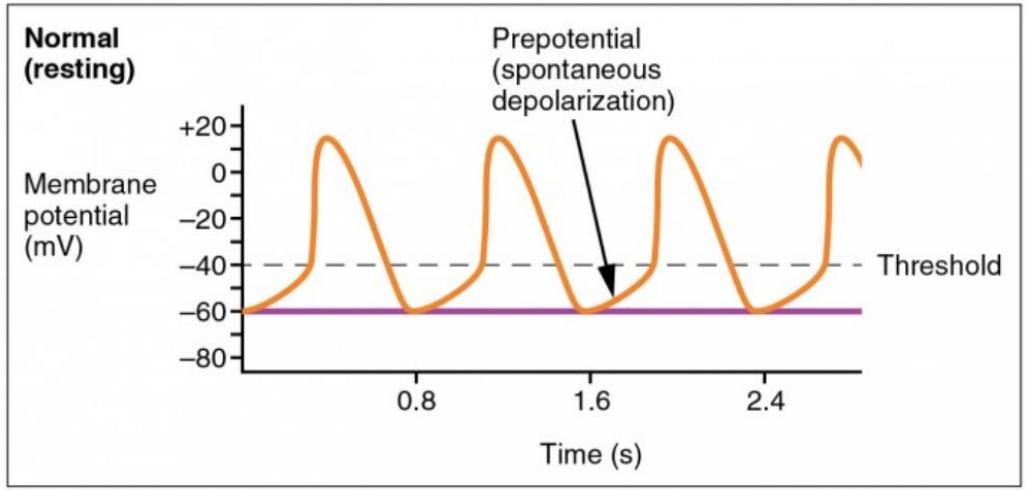
Prolong AV Node Refractory Period:

- Beta-blockers
- CCB (verapamil, diltiazem)
- Digoxin
- Adenosine
- Carotid massage



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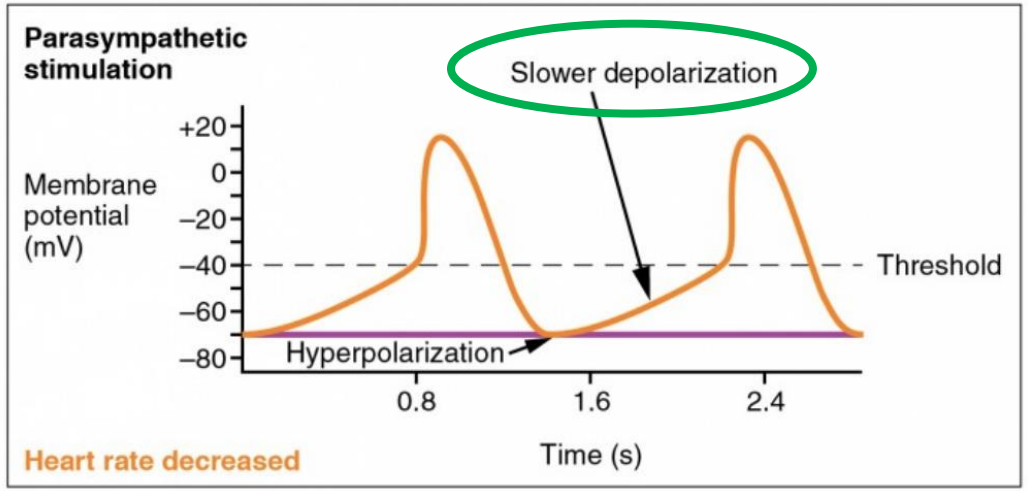

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- Sinus node automaticity: a fib is not generated by the sinus node
- Stimulation of beta-1 adrenoreceptors: *would increase conduction*
- Left venticular end-diastolic volume: *will increase but this doesn't dictate HR*
- Conduction through Purkinje fibers: doesn't govern HR

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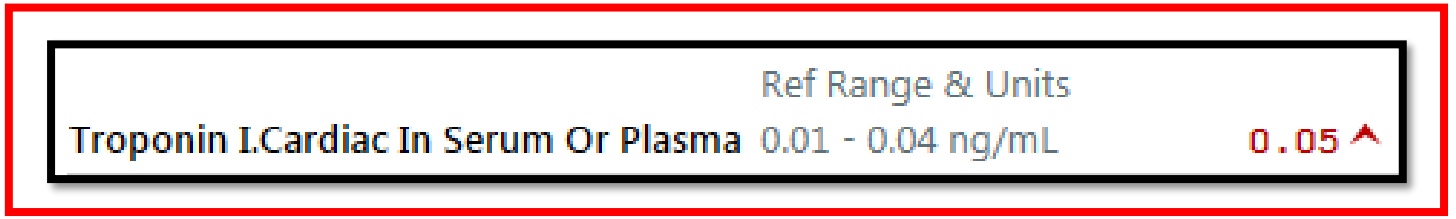

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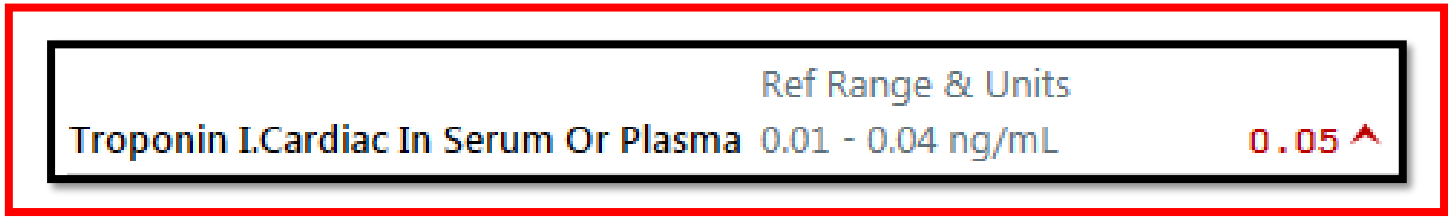

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Demand Ischemia:

- Imbalance (mismatch) between oxygen supply and demand
- *May occur with or without occlusive CAD*
- Increased frequency ➛high sensitivity Troponin assays

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Which of the following are ***most likely to explain her myocardial injury***?

- ~~*Coronary artery occlusion*~~ with thrombosis (*refers to STEMI*)
- ~~*Plaque rupture with partially occluding thrombosis*~~ (*refers to NSTEMI or Unstable Angina*)
- **Diastolic duration**
- ~~*Ventricular wall stress during rapid ejection phase*~~: (*physiologically correct but not basis for injury*)
- ~~*Neurohumoral response generating increased afterload*~~: (*physiologically correct but not basis for injury*)

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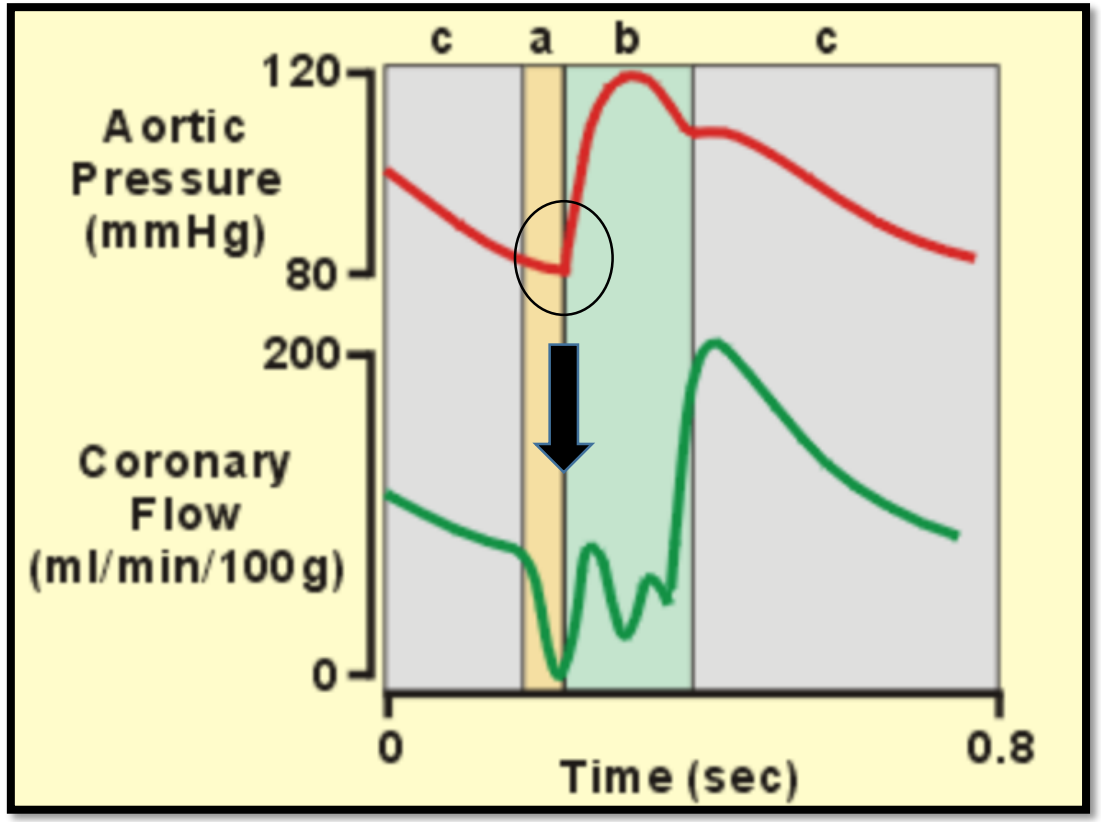
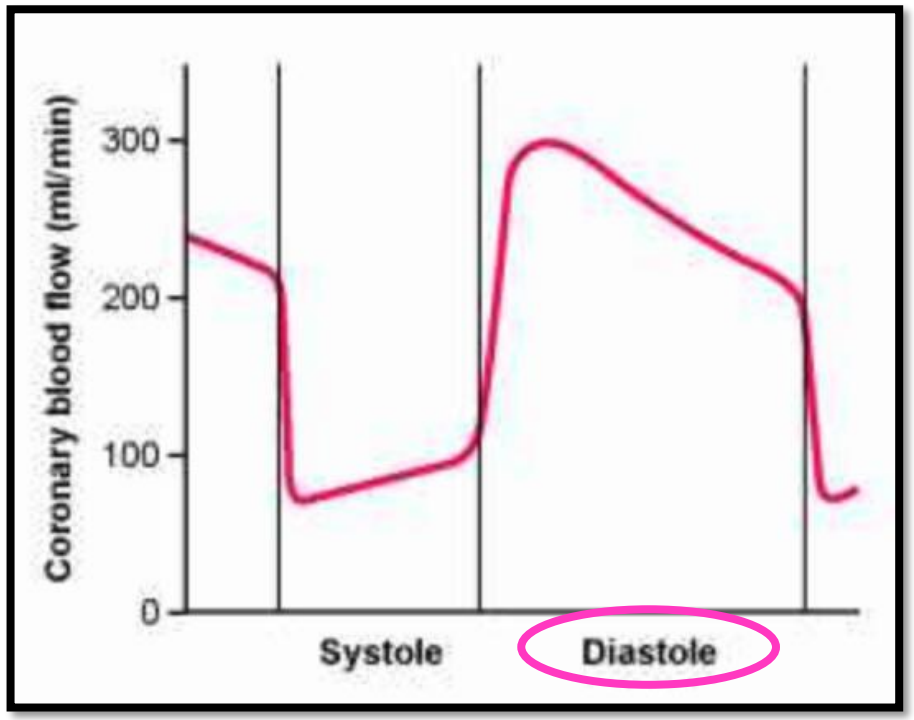
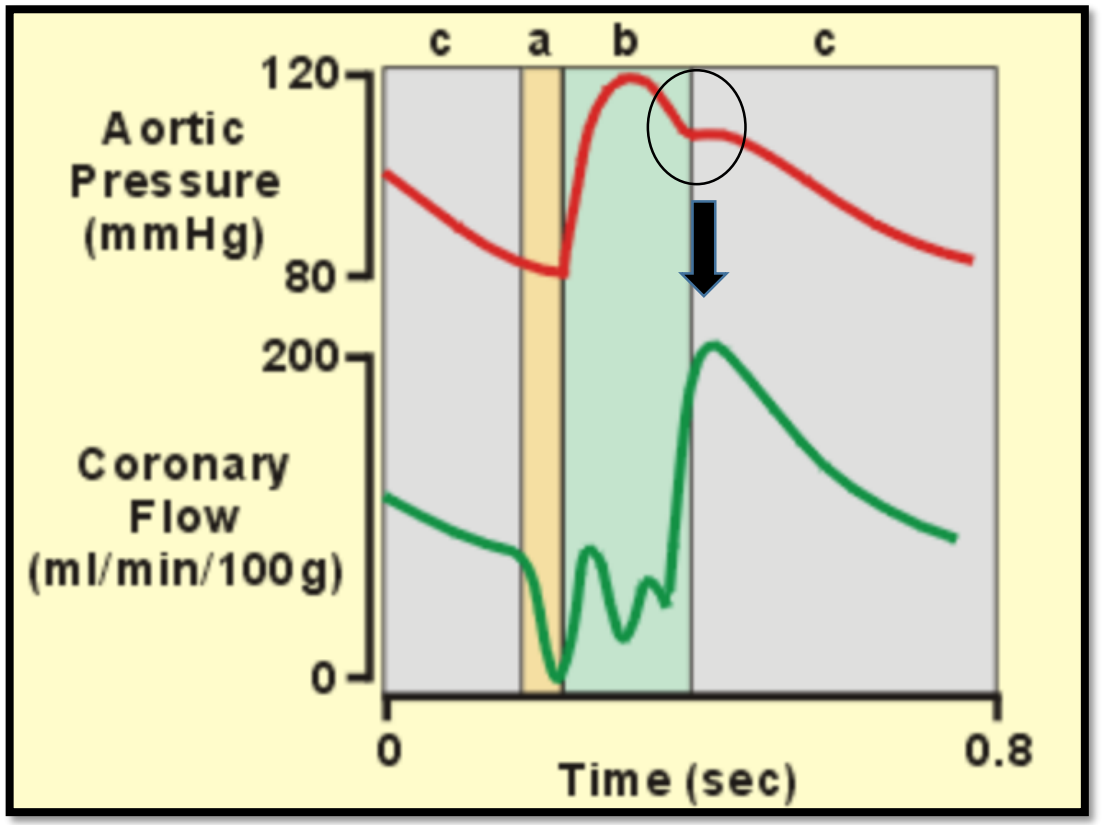
Which of the following are most likely to explain her **myocardial injury**?

- **Diastolic duration: coronary vessels fill during diastole;** *eliminate diastole, eliminate coronary filling*







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FEV1	68%
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Interstitial Changes (*and nodular densities*)
ILD: PE almost always includes dry crackles

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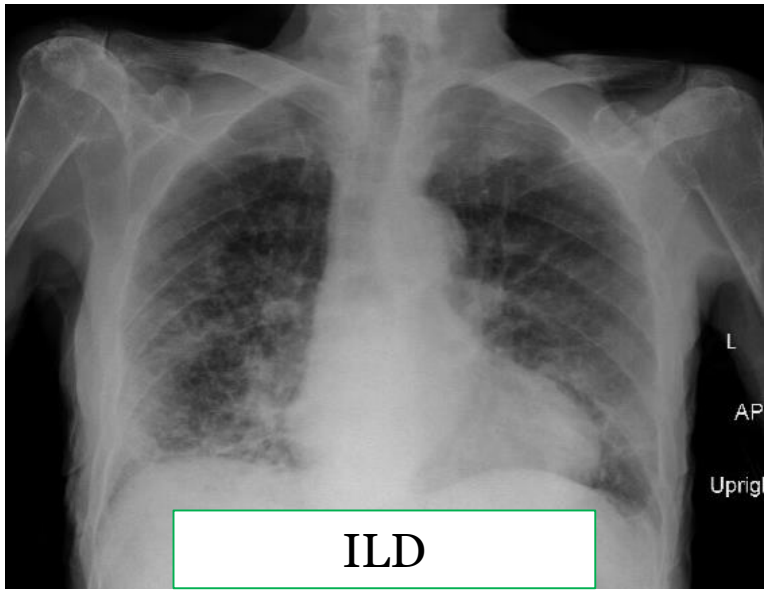

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Restriction
- Decreased Airflow
- Normal Ratio

Restriction
- Decreased lung volumes

Impaired Gas Exchange

1. Pulmonary vascular disease
2. Disorders of the interstitium
3. Loss of alveolar surface area

Clinical correlation with history, clinical exam and imaging is required.

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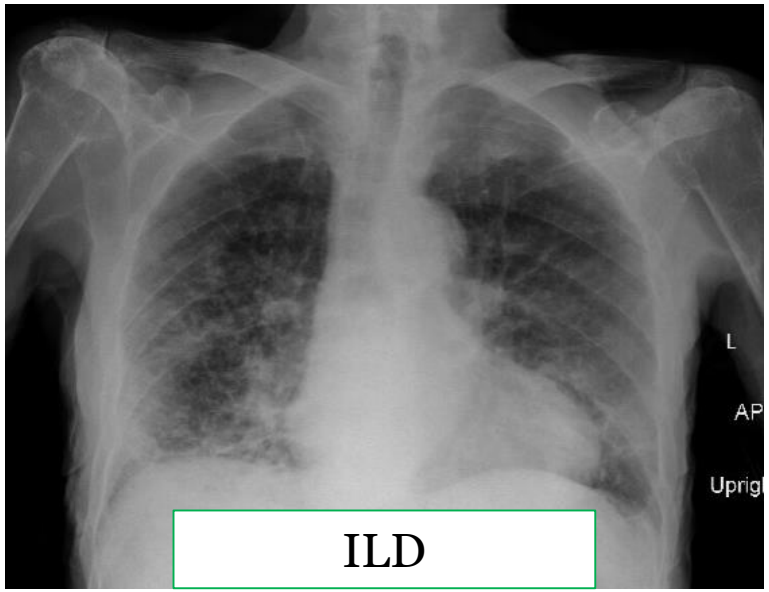

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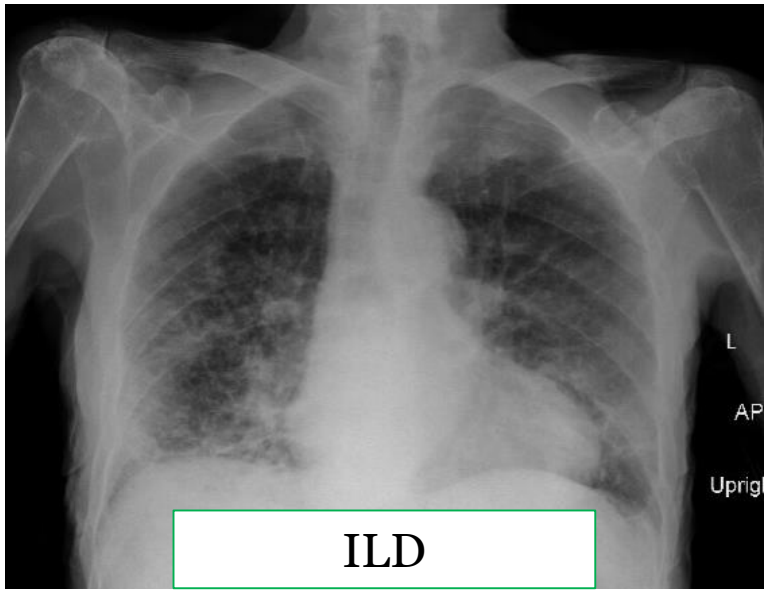

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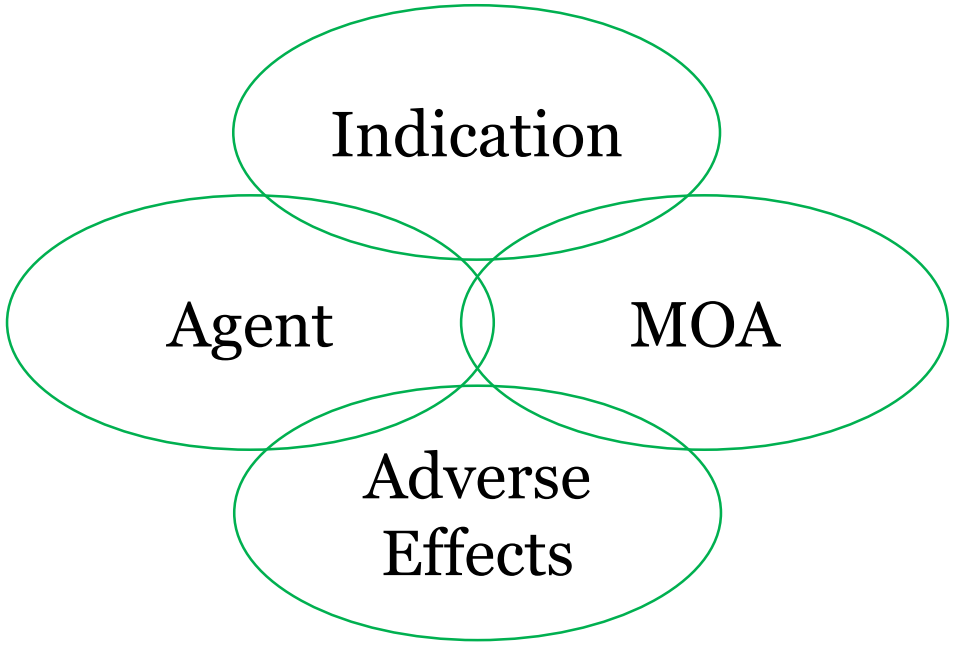
Pharmacology Fun



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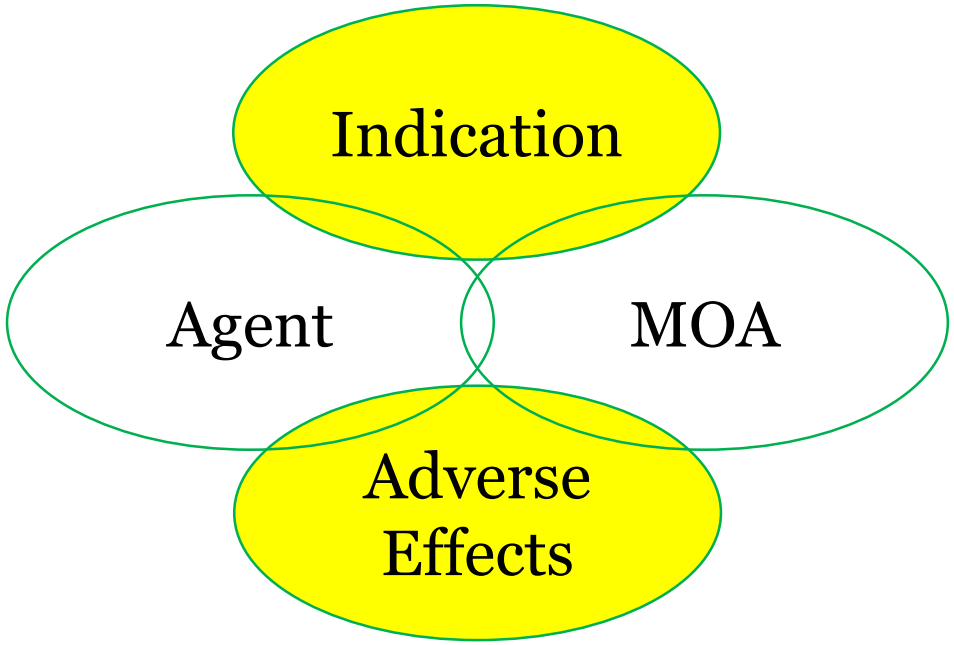

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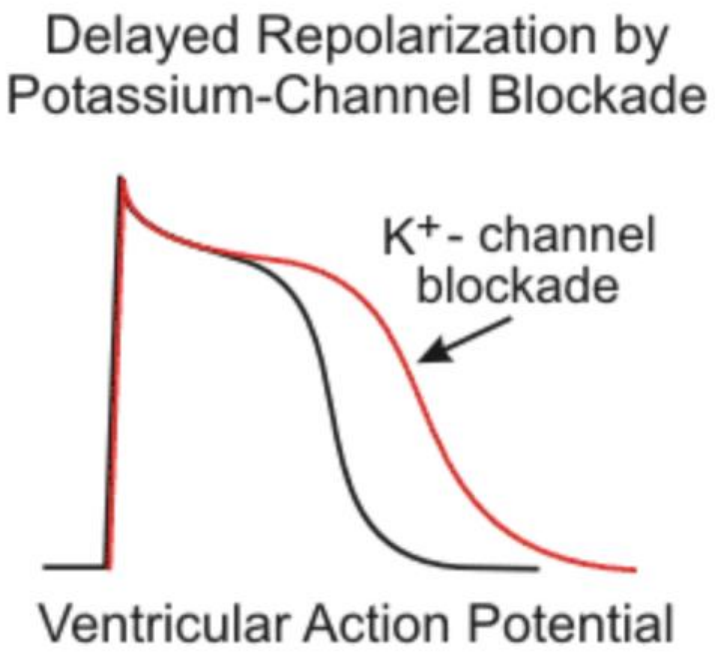
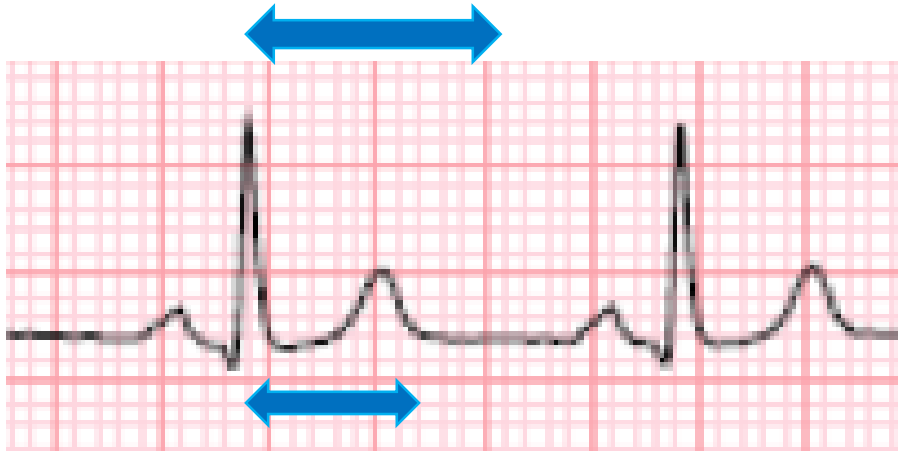


Prolongs Repolarization



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- Blocks the **potassium channel during phase III** of the action potential with **possible prolongation of the QT interval**
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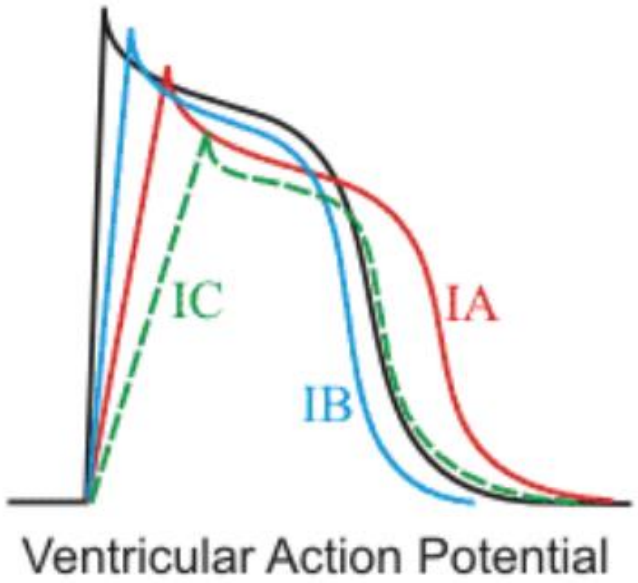
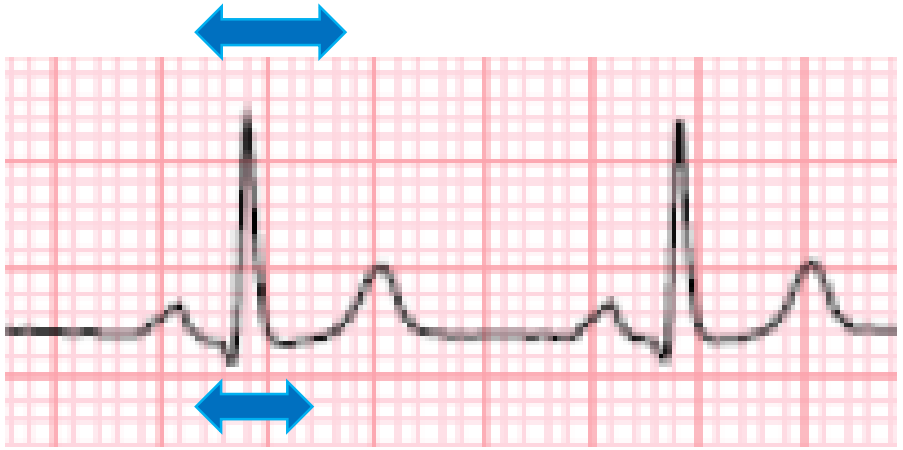
1a (*e.g. procainamide*)



Prolongs Depolarization → Widening of QRS complex



Select the **mechanism of action of the agent** and ***potential*** **changes seen on the EKG** for the agent that was most likely used to treat her underlying rhythm disturbance?

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- *Blocks the potassium channel during phase III of the action potential with possible prolongation of the QT interval*
- Blocks AV nodal conduction with narrowing prolongation of the PR interval
- Blocks the alpha-1 subunit of the L-gated voltage channel with prolongation of the QT interval: CCB don't prolong (or cause ILD)

PR interval represents conduction through the AV node

Select the **mechanism of action of the agent** and ***potential*** **changes seen on the EKG** for the agent that was most likely used to treat her underlying rhythm disturbance?

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85 y.o. woman is diagnosed with atrial fibrillation. Her daughter who attended the visit saw a TV commercial talking about the risk of blood clots. After reviewing the pros and cons of therapy and the multiple agents available, the patient is started on **rivaroXaban**.

At her follow up visit, my MD-PhD student finally shows up for a clinic and orders a host of blood tests to assess her compliance. Assuming she is compliant, which pattern is most consistent with her therapeutic agent?

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Platelet number

Platelet function

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Bleeding Time:

- Qualitative *platelet* disorders: vWD, Bernard-Soulier, Glanzmann's, ASA/NSAIDs
- Quantitative *platelet* disorders: thrombocytopenia (<30,000)

85 y.o. woman is diagnosed with atrial fibrillation. Her daughter who attended the visit saw a TV commercial talking about the risk of blood clots. After reviewing the pros and cons of therapy and the multiple agents available, the patient is started on **rivaroXaban**.

At her follow up visit, my MD-PhD student finally shows up for a clinic and orders a host of blood tests to assess her compliance. Assuming she is compliant, which pattern is most consistent with her therapeutic agent?

PT	aPTT	Thrombin Time	Bleeding Time
Increase	Normal	Normal	~~Increase~~
Increase	Increase	Increase	Normal
Increase	Increase	Normal	~~Increase~~
Increase	Increase	Normal	Normal
Normal	Normal	Normal	~~Increase~~

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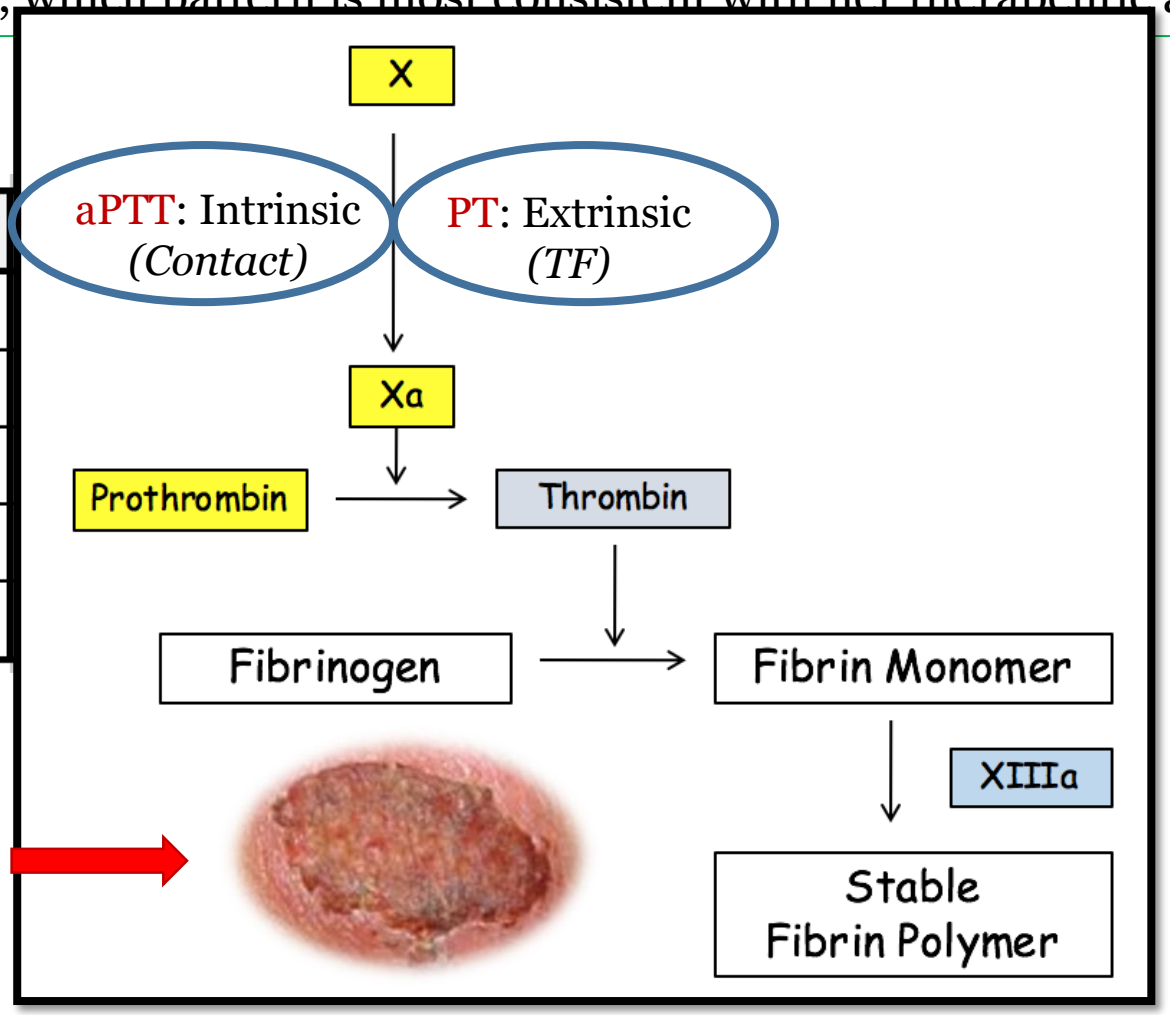
PT	aPTT
Increase	Normal
Increase	Increase
Increase	Increase
Increase	Increase
Normal	Normal



Secondary Hemostatic Plug
(i.e. a 'scab')

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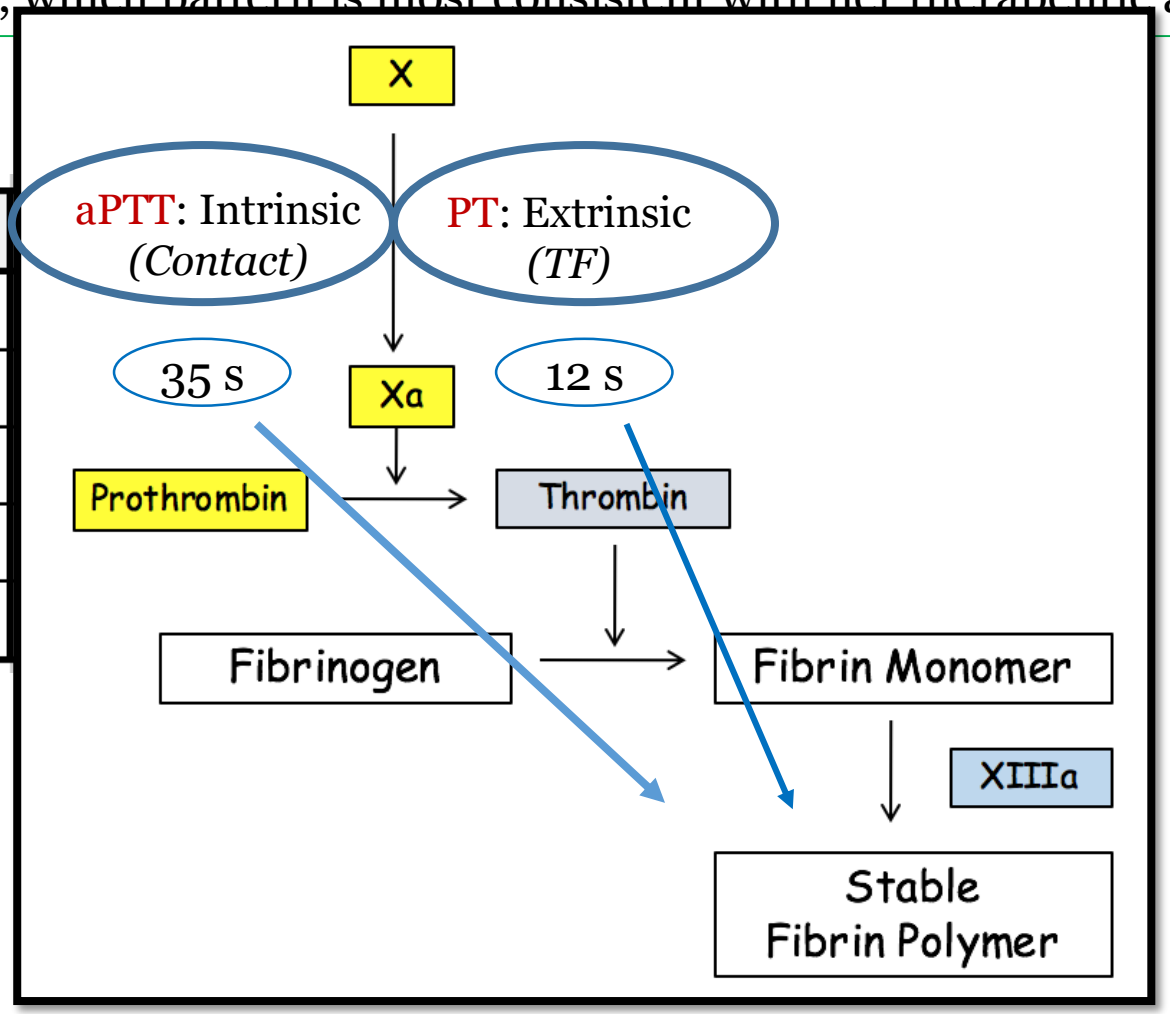
PT	aPTT
Increase	Normal
Increase	Increase
Increase	Increase
Increase	Increase
Normal	Normal



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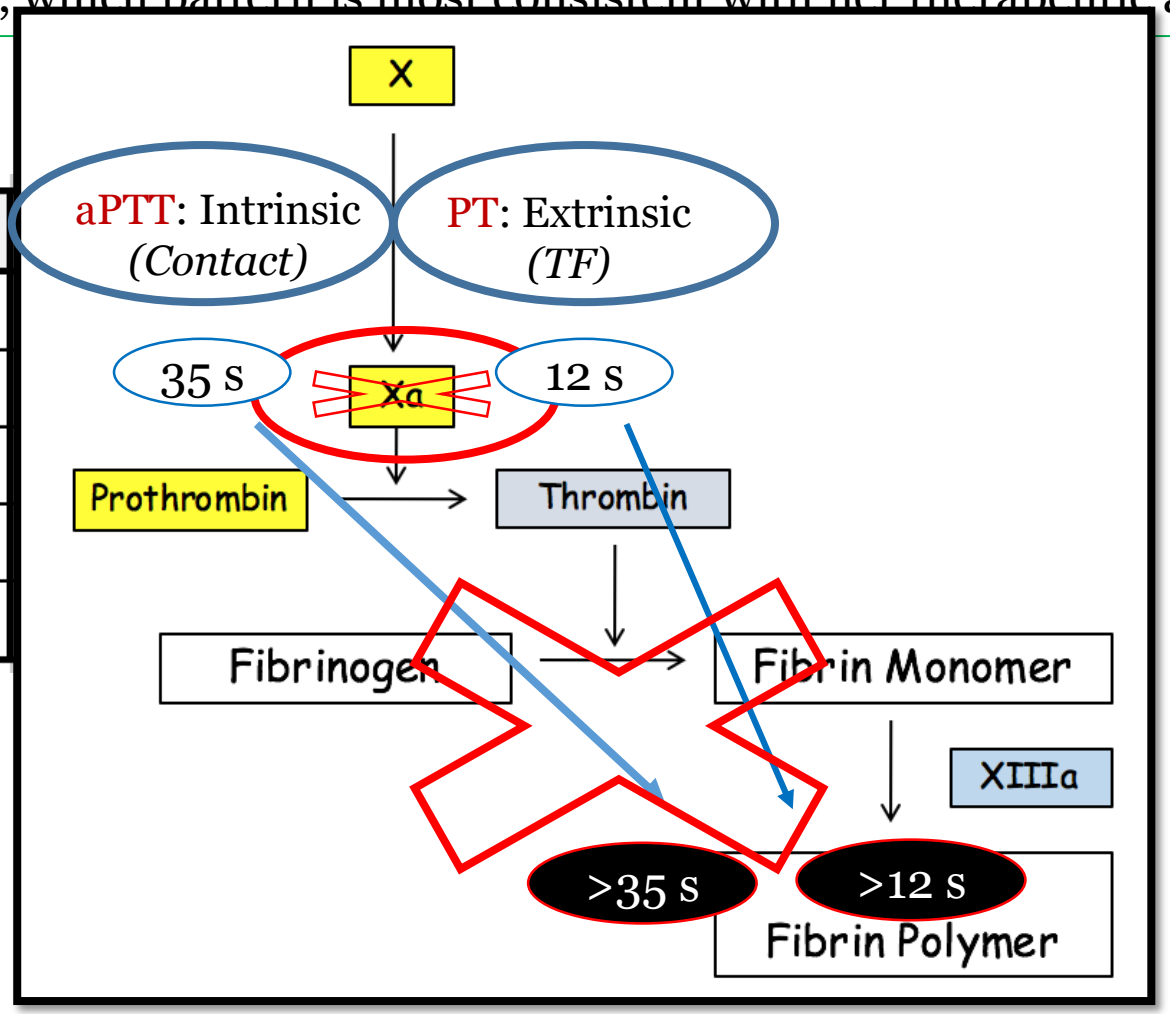
Increase	Normal
Increase	Increase
Increase	Increase
Increase	Increase
Normal	Normal



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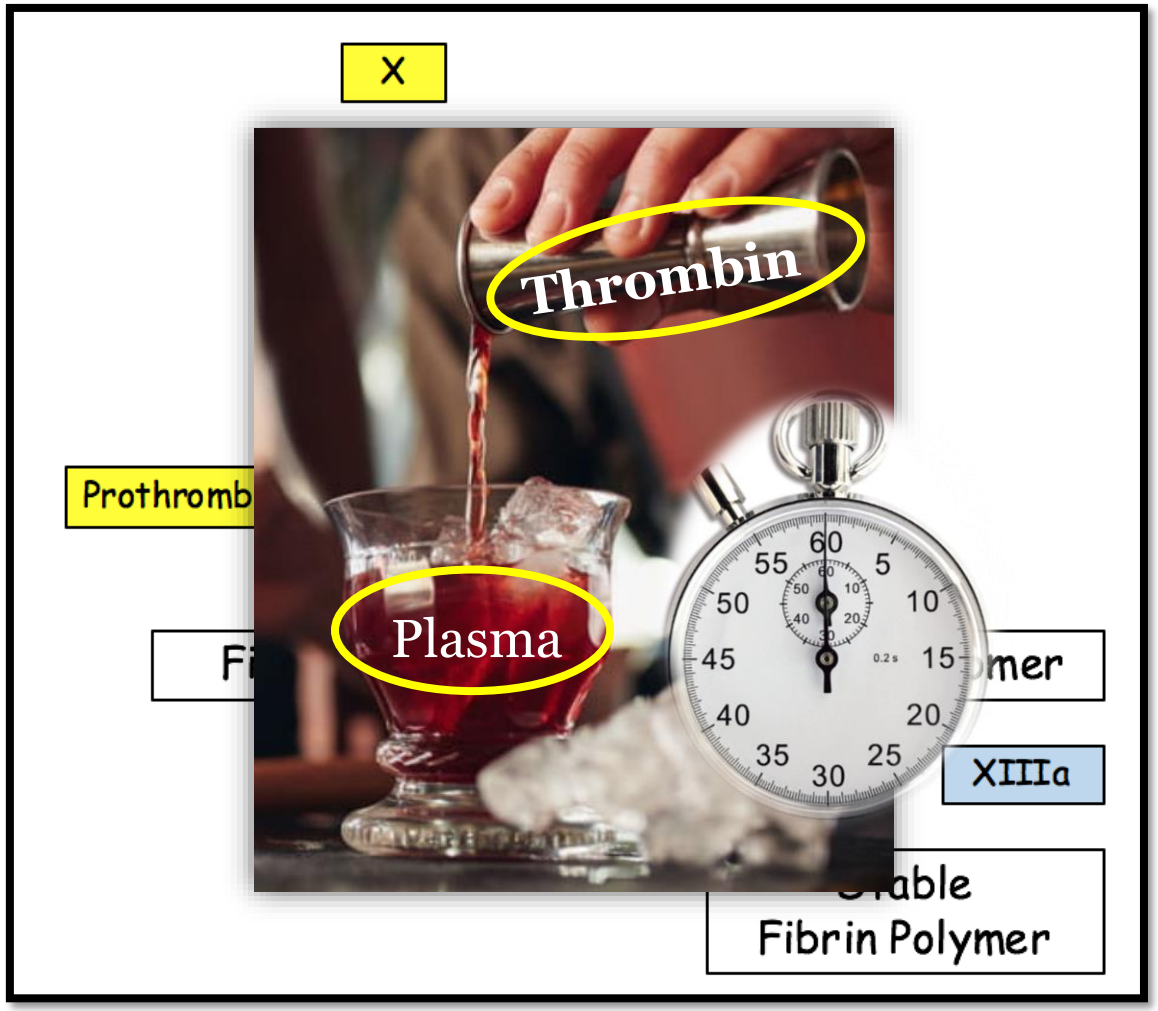
Increase	~~Normal~~	Normal	~~Increase~~
Increase	Increase	Increase	Normal
Increase	Increase	Normal	~~Increase~~
Increase	Increase	Normal	Normal
~~Normal~~	~~Normal~~	Normal	~~Increase~~

Thrombin Time:

1. Mix Thrombin with patient plasma.
2. Measure time to clot.



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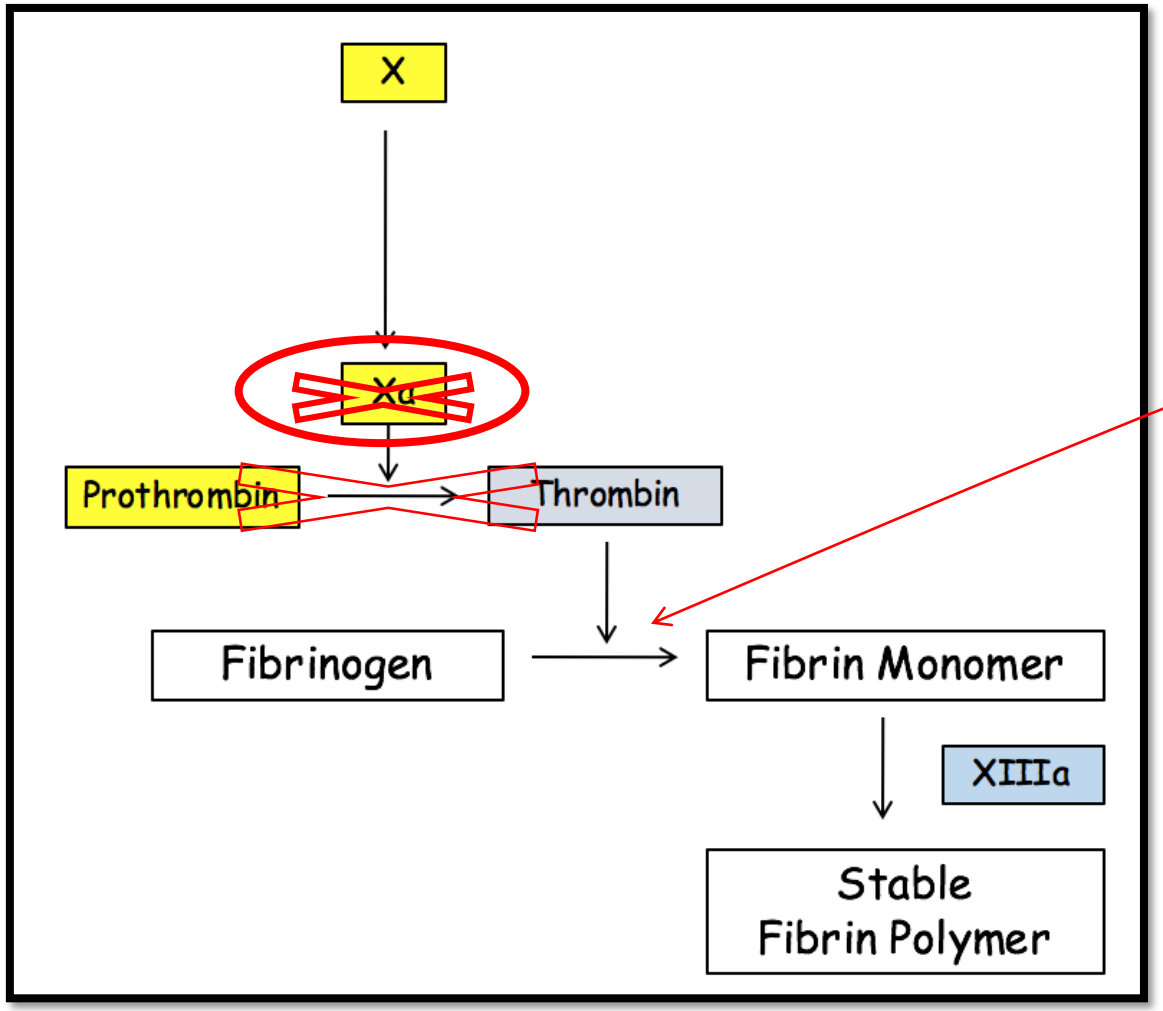

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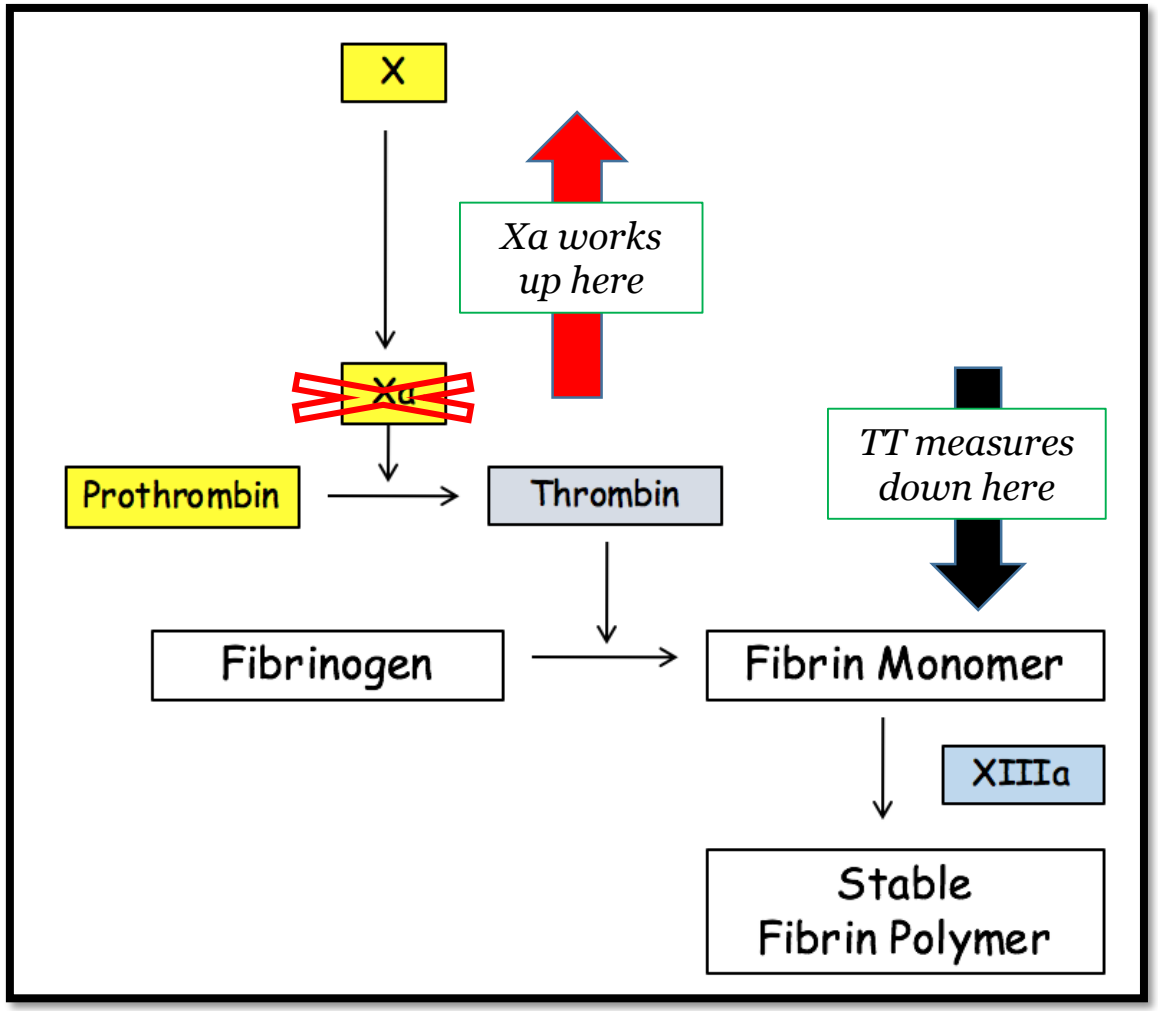
T	Thrombin Time	Bl
nal	Normal	
ase	Increase	
ase	Normal	
ase	Normal	
nal	Normal	

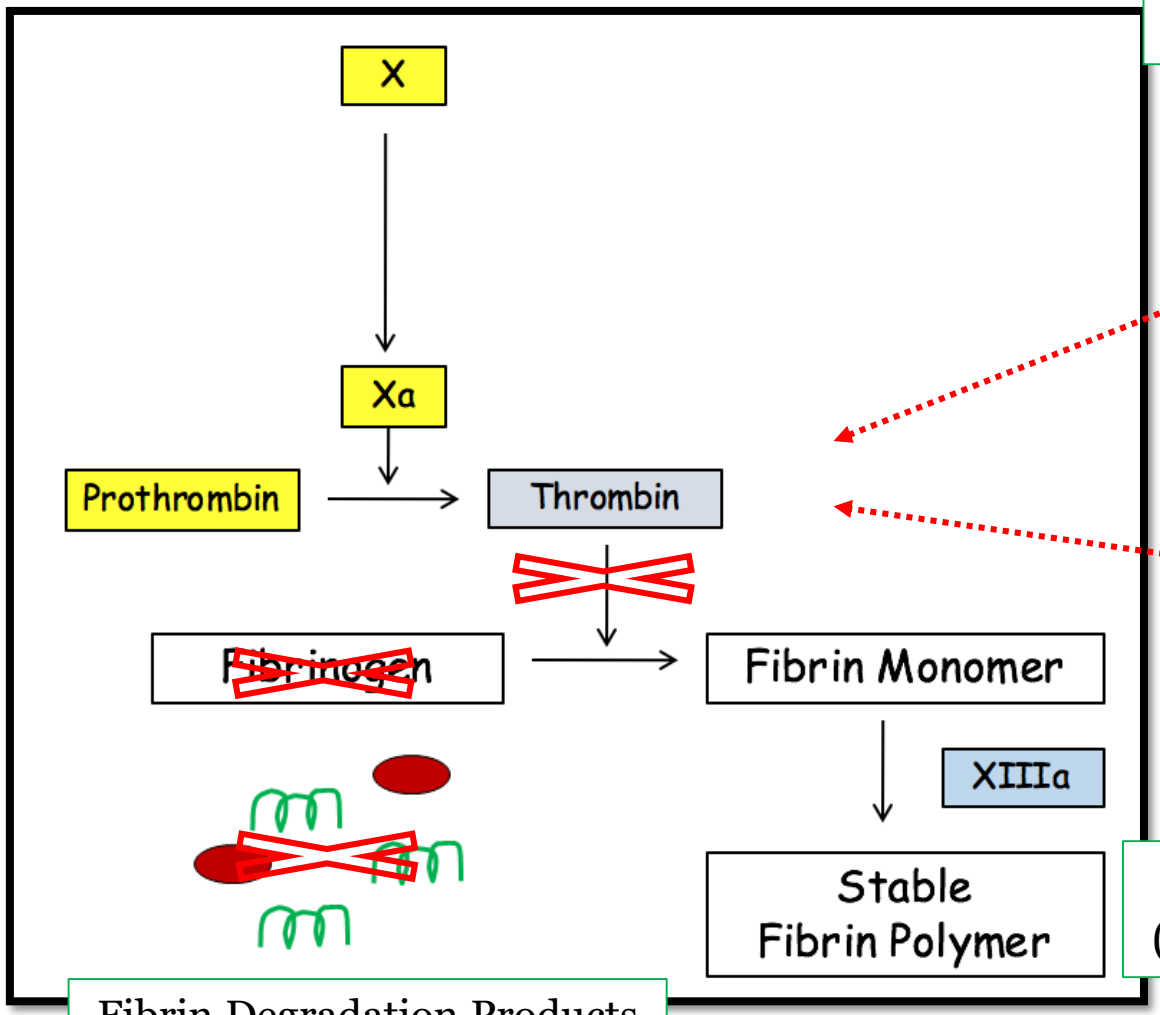


Thrombin Time:

1. Mix Thrombin with patient plasma.
2. Measure time to clot.

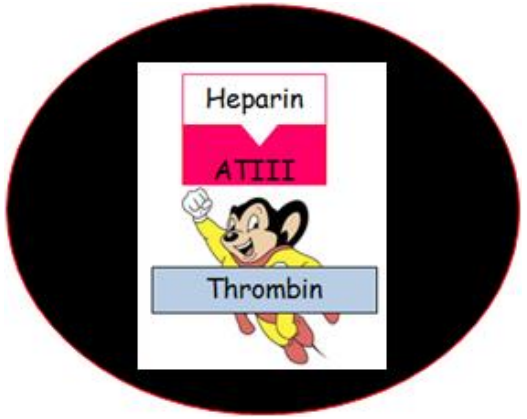
Direct *Thrombin* Inhibitors:

- Dabigatran
- Hirudin



Fibrin Degradation Products

DTI



Heparin
(bound to ***Anti-Thrombin*** III

Elevated Thrombin Time:

- No Fibrinogen
- FDP (*DIC, tPA rx*)
- DTI
- IV heparin

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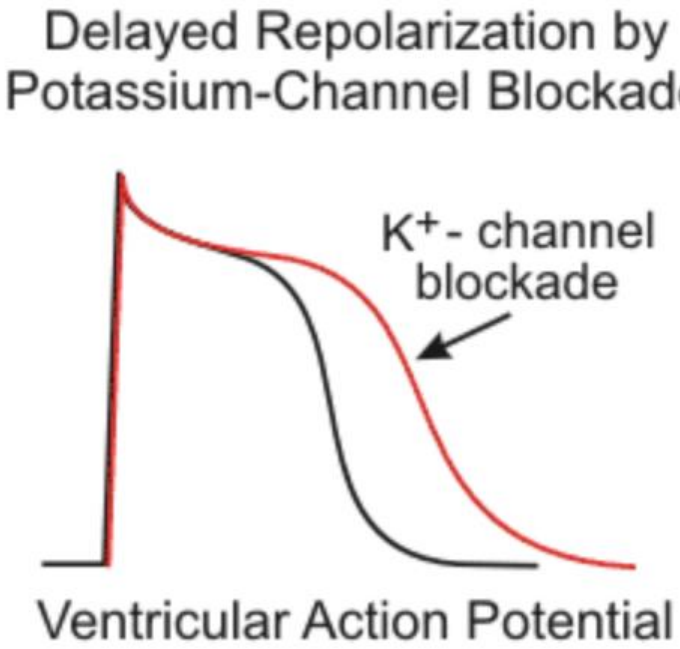
PT	aPTT	Thrombin Time	Bleeding Time
Increase	Normal	Normal	Increase
Increase	Increase	Increase	Normal
Increase	Increase	Normal	Increase
Increase	**Increase**	**Normal**	**Normal**
Normal	Normal	Normal	Increase

What Just Happened?

- Atrial Fibrillation
  - Ventricular response is dictated by AV node refractory period
- Myocardial Injury
  - Mechanism other than occlusive CAD (*demand ischemia*)
  - Inadequate coronary artery filling in fast a fib (impaired diastolic duration)
- Amiodarone & Pharmacology Fun
  - Mechanism of Action: Potassium channel blockade ➛ QT prolongation (not QRS widening)
  - AE: interstitial fibrosis/pneumonitis, blue-gray skin, corneal deposits, thyroid dysfunction
- Anti-coagulation Therapy and Laboratory Parameters
  - Xa inhibitor: elevation of PT, aPTT; normal thrombin time
  - Thrombin time: fibrinogen, FDP, direct thrombin inhibitors (but not Xa inhibition)

The Year in Review Series: Case 6. Palpitations
Case-based NBME review



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