

## CLINICAL PROBLEM SET # 12

<b>Patient</b>
Male, 52 years old
<b>Chief Complaint</b>
"I'm here to have my filling done. Last time, my jaw was numb for many hours afterwards and I could not talk with anyone. I have a job interview this afternoon and really want that job. So, I hope you have a trick, Doc, for the numbness to go away quickly."
<b>Background and/or Patient History</b>
Cardiac Arrhythmia (atrioventricular reentrant tachycardia), the onset coincided with: Myocardial Infarction 5 years ago; Epilepsy;  Abuses alcohol (> 10 years); Smokes tobacco (30 pack-years)  Medications: Dilantin® (Phenytoin) Quinidine
<b>Current Findings</b>
Cariou tooth #3, restorable. Temp: 98.5 F BP: 115/65 mmHg HR: 72 bpm  For local anesthesia, the patient received one cartridge of 2% Lidocaine with 1:50,000 Epinephrine. The procedure was completed in 30 minutes. To shorten the duration of anesthesia in consideration of the patient's needs, he received one cartridge of Oraverse® (Phentolamine, 0.4 mg). Within a minute, the patient reported chest pain, feeling dizzy and shortness of breath. The EKG revealed a 2 <sup>nd</sup> degree atrioventricular block (prolonged P-R intervals and selected P waves not followed by QRS complexes - 'skipped beats') and the heart rate of 55 bpm.

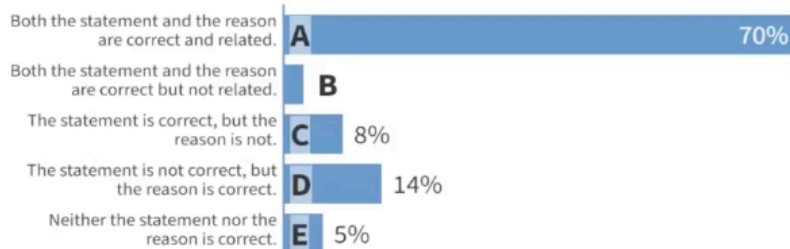
1. What are the expected systemic effects of Lidocaine, Epinephrine and Phentolamine?
2. What was the most likely mechanism of the 2<sup>nd</sup> degree heart block and bradycardia in this patient's case?
3. What is a potential link between the dental treatment this patient received and the dizziness, chest pain and shortness of breath that he is experiencing? Please explain the underlying mechanism.
4. Could any of the patient's other medications have evoked, or contributed to, the incident? If so, please indicate which medication and explain the underlying mechanism of interaction.

**Email # 1**

**(regarding one of the small group quiz questions)**

“...an increase in extracellular potassium causes a decrease in Digoxin efficiency.  
However, (...) extracellular potassium increases with only potassium-sparing diuretics.  
Do all diuretics increase extracellular potassium?...”

**Diuretics can decrease the efficacy of Digoxin because binding of Cardiac Glycosides to  $\text{Na}^+/\text{K}^+$ -ATPase is dependent on extracellular potassium concentrations.**



For glycosides if you increase EC K you decrease binding of glycosides to Na/K ATPase so you decrease effectiveness of glycosides  
Some diuretics increase and some decrease, but the questions says CAN decrease not WILL necessarily  
Furosemide would not be true here

**Email # 2**  
**(Lecture 11 LO #2)**

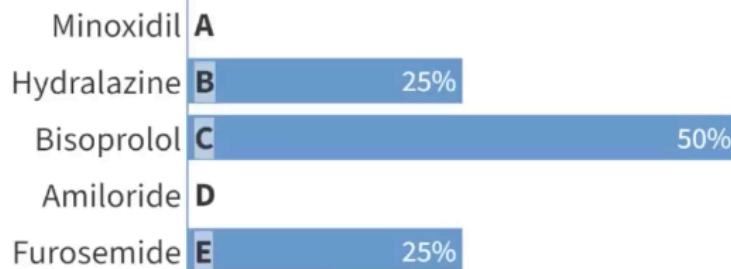
(...) let me know where I can find the information for the following learning objective for Lecture 11(...)

Be able to explain why calcium channel antagonists can aggravate Angina and how to prevent the aggravation.

Respond at [PollEv.com/agnieszka](https://PollEv.com/agnieszka)

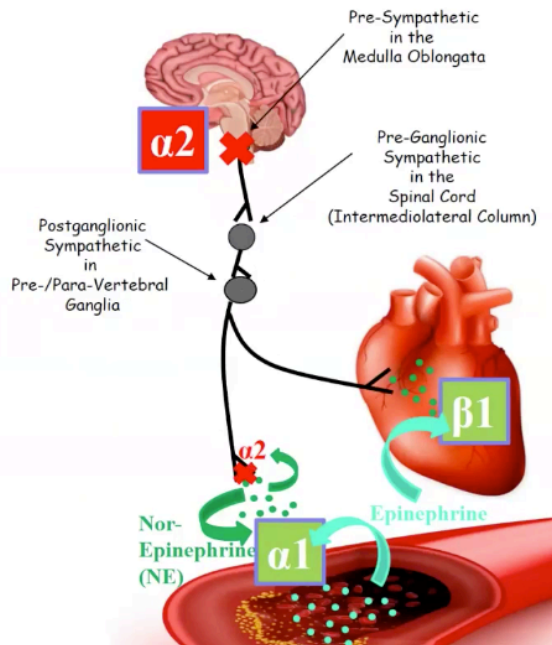
Text **AGNIESZKA** to **22333** once to join, then **A, B, C, D, or E**

**An effective treatment to prevent aggravation of myocardial ischemia by Verapamil would likely include:**



**Email # 3**  
**(Lecture 10 Question)**

At the beginning of the lecture you discussed the alpha-2 adrenergic receptors and mentioned that they are located on pre-sympathetic neurons. However, in the diagram it appears they are found on the post-sympathetic neurons.



Alpha 2 receptors by themselves cause inhibition so if you put alpha 2 blockers you will actually increase activity of the neuron; for anti-hypertensive effect we want to inhibit the sympathetic system so we have two options. Activate the receptor that causes inhibition or inhibit the receptor that causes activation, meaning contraction.

**Please identify the correct match concerning anti-arrhythmic drugs.**

Class I – Calcium channel block	<b>A</b>
Class II – Sodium channel block	<b>B</b>
Class III – Action Potential Prolongation	<b>C</b>
Class IV – Beta-adrenergic block	<b>D</b>
Class V – Alpha-adrenergic block	<b>E</b>

Answer: C

**The likely successful treatment option for bradyarrhythmias would be:**

Bisoprolol	<b>A</b>
Amiodarone	<b>B</b>
Lidocaine	<b>C</b>
Quinidine	<b>D</b>
Implanted pacemaker	<b>E</b>

Answer: E

No good way to treat bradyarrhythmias pharmacologically

**The analgesic effectiveness of Codeine is likely to be compromised if the patient is chronically treated with:**

Labetalol	<b>A</b>
Verapamil	<b>B</b>
Amiodarone	<b>C</b>
Quinidine	<b>D</b>
Lidocaine	<b>E</b>

Answer: D

**Please identify the drug that is most likely to prevent arrhythmias triggered by EMOTIONAL STRESS.**

Lidocaine	<b>A</b>
Sotalol	<b>B</b>
Quinidine	<b>C</b>
Dopamine	<b>D</b>
Hydralazine	<b>E</b>

Answer: B

Emotional stress, sympathetic system activated, so you want to inhibit sympathetic system

**Bradycardia is a likely effect of treatment with the following drugs, EXCEPT ONE. Please identify the EXCEPTION.**

Sotalol	<b>A</b>
Hydralazine	<b>B</b>
Amiodarone	<b>C</b>
Propranolol	<b>D</b>
Quinidine	<b>E</b>

Answer: B

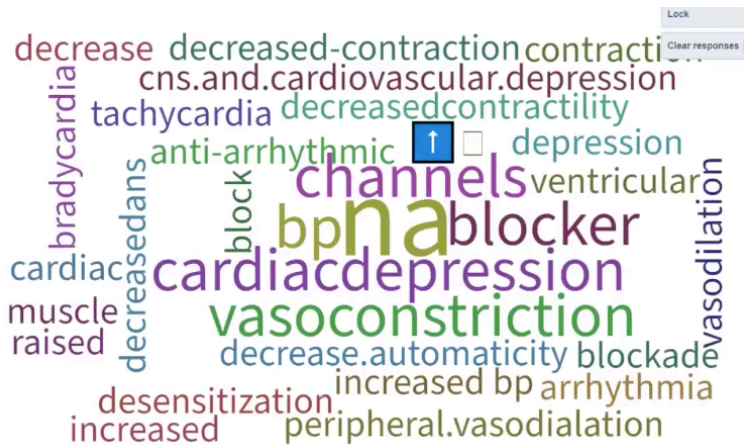
Decreases BP, unloads baroreceptors, and increases HR

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Limiting factor for lidocaine metabolism is blood flow. Systemic conditions of lidocaine under normal conditions are not expected to be severe because it is very quickly metabolized



**In a SHORT PHRASE: What are the expected SYSTEMIC effects of Epinephrine?**

A word cloud of cardiovascular-related terms. The most prominent word is 'vasoconstriction' in large purple letters. Other visible words include 'increased heart rate', 'vascular resistance', 'increased hr', 'vasodilation', 'flow', 'vasoconstrictor', 'hr', 'rate', 'heart', 'higher', 'blood', 'dizziness', 'tachycardia', 'cerebral', 'sympathomimetic', 'decreased diastole', 'increased contractility', and 'hypertension'. A small cartoon person icon is positioned near the center of the word cloud. A 'Clear response' button is located in the top right corner.

Patients on beta blockers are at risk because of vasoconstrictive effect of epinephrine  
Tachycardia as well



**In a SHORT PHRASE: What are the expected SYSTEMIC effects of Phentolamine?**

It is non-selective alpha antagonist. Alpha 1 causes vasodilation...

cardiac depression  
vasodilation  
effects vasodilator  
vasodilation epinephrine  
anti-adrenergic reverse vasodilate  
increase blood-flow  
cardiac depression  
bradycardia

Indirect effect on cardiac depression

**Please list TWO FACTORS/DRUGS involved in the mechanism of the patient's heart block.**

Directly or indirectly.

lidocaine/phentolamine  
propanolol excitement  
quinidine dilantin. quinidine  
mi cardiac  
phentolamine  
channel  
epinephrine block  
labetalol  
sodium dilantin  
arrhythmia phenytoin  
lidocaine & re-entry

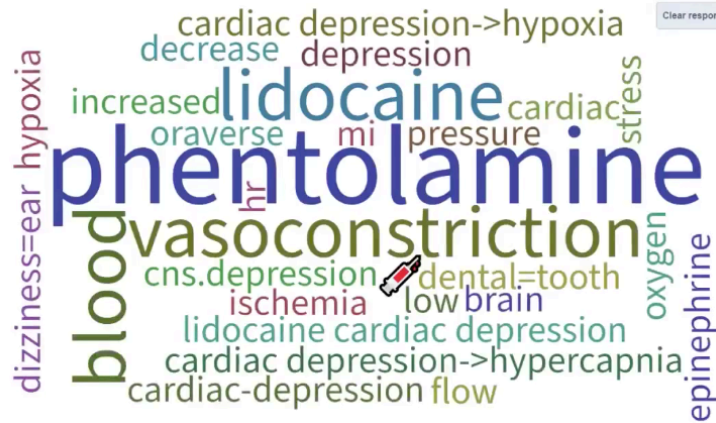
Phentolamine is indirectly, this would be OraVerse.

Lidocaine directly.

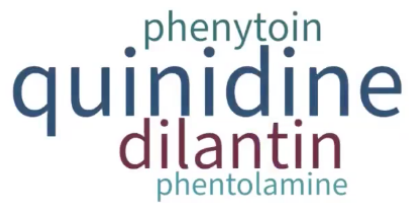
Quinidine.

Alcohol abuse causing liver damage, can't metabolize lidocaine as quickly

Lock  
Clear response



**In a SHORT PHRASE: Which of the patient's long-term medication(s) could have contributed to the incident?**



**The following statements regarding Lidocaine are true, EXCEPT one. Please identify the EXCEPTION.**

It is a Class I Anti-Arrhythmic Drug and blocks voltage-gated sodium channels.

When used in dentistry, it can lead to a life-threatening cardiac depression.

One of its side effects could be ventricular tachyarrhythmia.

In cardiology, Lidocaine is used primarily to treat bradyarrhythmias.

Both C and D constitute exceptions, i.e. neither statement applies to Lidocaine.

Answer: D

**Anti-arrhythmic drugs may trigger arrhythmias because anti-arrhythmic drugs can promote re-entry by changing tissue properties.**

Both the statement and the reason are correct and related.

Both the statement and the reason are correct but not related.

The statement is correct, but the reason is not.

The statement is not correct, but the reason is correct.

Neither the statement nor the reason is correct.

Answer: A